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EDITORIAL

A Radical Approach to Medical Education - A British Perspective

This review is informed by current postgraduate training of doctors in the UK, emphasising changes as to how this training is being delivered, and the objectives in ensuring that doctors are prepared to meet the challenges of patient and community expectations, and are responsive to the requirements of a modern society and concerns regarding the sometimes conflicting demands of autonomy, the wish to do good but not harm, respect for beliefs, equity and equality. There is also a need to ensure that medical talent is not wasted and that the skills and knowledge of trainees are developed and utilised efficiently..

There is therefore a requirement that educational training opportunities should be maximised, and that training should have primacy over service delivery for trainees, and that there should be a clear curriculum for trainees with clear achievable outcomes. Trainees should be imbued with a culture of lifelong learning, especially with the rapid development of medical knowledge and clinical care. Indeed, this vast expansion of medical knowledge makes it impossible for trainees to memorise and recall as perhaps previously possible, and although a knowledge of certain basic principles and the ability to deal with common practical issues remain, depending on the stage and speciality of the trainee, of equal importance is to develop the skills to locate and evaluate knowledge, and apply appropriately to the clinical situation[1].

The traditional scientific background to medical training remains essential, particularly for those who are likely to pursue a career in academic medical science. However, to improve population health, a broader skill development is required to integrate with other health professionals, appreciate the perspective of patients, and how their social situation inter-relates to their illness, and understand the ethical context in which doctors work. Indeed, a broader understanding of health, rather than just illness is required.

This has consequences for medical personnel of all grades and other health care personnel. In particular, senior staff have a professional commitment to training their junior staff (as expected by all the Speciality Colleges), and accept that more clinical care will need to be provided by seniors. To achieve this, the pyramidal structure of medical careers, needs to be inverted, with an expansion of senior doctors, and maximisation of the utilisation of the clinical skills of other health care workers. This will ensure that patients are cared for by staff appropriately trained and skilled, for the situation that they are in. Streamlining medical training ensures that trainees are efficiently developed to their potential with minimum wastage of talent.

A curriculum based on continuous formative competency-based assessment, to enable trainees to address deficiencies of specific competencies, as they progress, does not necessarily remove the requirement for summative assessment, or examinations, as a benchmark of standards prior to entering independent practice, or more specialised training, but should include assessment of practical skills, such as interaction with patients, and efficient use of resources. For example, a viva question might relate to a diagnostic clinical scenario, where the answer should not be a text-book list, but the probable diagnoses in that particular patient, given their age, sex, environment and other features specific to that patient. Management should also be discussed within that context.

To avoid wastage of medical trainees as a resource, failure to progress to the next stage should be the exception rather than the rule. Well-structured formative training, with appropriate career advice will achieve this. This requires that trainees are supervised clinically, and also in respect of their personal development.

Training needs to be closely integrated with service provision, stand alone courses have a value, but should be mainly concerned with aspects of the curriculum, or occasionally with particular interests of the trainee,

whilst viva skills, and other life skills such as interview techniques, should be a small part of the educational process. The prime objective should be to train doctors skilled in the arts and science of medicine, able to play a role in modern medical practice, to interact constructively with patients, and be able to relate to other health professionals, and appreciate their responsibility to society rather than produce doctors who can pass examinations, but have not developed other essential personal and professional skills.

Advantages of a competency-based formative assessment education:

For trainees:

A clear career pathway, of defined duration, meeting the achievable aspirations of the trainee.

Confidence in possessing fundamental practical and other skills.

Satisfaction as a team member.

Flexibility to respond to the rapidly developing scientific aspects of medicine, new diseases, and previously unfamiliar social and public health situations.

More time-efficient training, less time spent on non-educationally-useful activities.

For trainers:

The opportunity to bring out the best in trainees, and ensure a legacy of a well- trained medical workforce.

Share and experience new approaches to patient care with trainees.

For patients:

The reassurance of being cared for by a well-trained medical team of sufficient seniority.

Medical and other carers within the team, who are responsive to all aspects of the patient's concerns.

For Public Health:

Trainees responsive to the social, environmental and Public Health aspects of health and disease.

For Government:

A cost-effective training programme, minimising waste, producing doctors fit forpurpose, who are flexible and responsive to the Health needs of society, and work constructively with colleagues and other health professionals.

Disadvantages (possible or perceived):

Trainees:

Frustration for those who wish to proceed quickly to more specialist training[2].

Trainers and senior doctors:

More time required for training (formal and opportunistic).

Perception that trainees are inadequately trained (at least within their speciality – speciality skills develop later), and require more support.

More hands on care, including emergency care, required from Consultants.

Patients:

Less continuity of care from junior doctors, due to shorter rotations, and educational requirements. The European working time directive, which is not unrelated to providing good education, is a major factor in the UK, affecting continuity of care.

Government:

More medical or other health care staff required to provide routine care.

More equitable care provision will identify funding gaps.

Postgraduate Medical Training in the United Kingdom

In 2000, following a commitment to modernise the Senior House Officer (SHO) grade, a report "Unfinished Business" was presented by Liam Donaldson, Chief Medical Officer, outlining the unsatisfactory situation of the SHOs, in respect of training and career prospects[3]. Many trainees (including a disproportionate number of non-UK graduates) were stuck at this grade, with little supervision or training and poor prospects for furthering their career. As a consequence, a new system of recruitment and training, Modernising Medical Careers (MMC), was introduced, commencing in 2005.

Trainees, preference being given to European Union medical students (a requirement of UK membership of the European Union), but mainly new UK graduates, were appointed to two-year Foundation Training Programmes, through a standardised recruitment and assessment system[4]. Rotations consisted of four months in different specialities, to include at least General Medicine.

Achievement of the national curriculum competences had to be demonstrated each year to progress to the next year. There was also a formal teaching programme, and opportunistic teaching was encouraged. The aim was for trainees to develop generic skills and competencies, and knowledge from undergraduate years in the first year, the second year was to demonstrate an ability to assess and manage the acutely ill patient, and to improve generic skills of teamwork, time management, communication and self-directed learning. Particularly in the second year, there was an opportunity to be placed in health care settings other than hospitals, such as public health, primary care and academia.

Therefore, despite rotating through different specialities, the standard curriculum competences had to be achieved.

In addition, the Programme was to provide trainees with the opportunity to sample specialities that they might favour as a career, and to broaden the outlook of those determined to follow a specific career pathway before they entered speciality training. These aims could be in conflict, and in reality, there were practical issues since placements were based on existing posts, there was some funding for additional innovative placements and primary care, but insufficient to achieve everything hoped for in the initial plans.

The second year in particular, has come under considerable criticism from many trainees and senior staff, because of the lack of specialist skills they possess for a particular placement. Some rotations lack flexibility and some trainees are impatient to experience their intended speciality, particularly as the selection process for specialities begins early in the second year. These trainees are now allowed and sometimes encouraged to take initial specialist College examinations early in their first year. It is likely that the conflict between the Colleges and many trainees, and the Educational principles behind the Foundation Programme, will reach a compromise, by the second year becoming "themed", that is consisting largely of a particular speciality. This is in conflict with the objectives of producing doctors with a broad-based understanding of and commitment to health care within society.

Specialist training also has a curriculum for each speciality, in many cases there is the opportunity for even more specialised experience towards the end. Most Speciality Programmes now run for seven or eight years (longer than originally hoped for by Government). Some specialities have only one appointment process at the beginning, others such as medicine, have two or three years core speciality training, followed by a further competitive application for more specialist training, such as Cardiology[5].

There is opportunity for non-European Union doctors to enter specialist training within the UK, but this not easy, and discussion is not within the scope of this Editorial.

Trainees with particular issues, such as a disability, have, at least theoretically, the option of more flexibility within their programmes. Flexible training should add more equity and diversity to the mix of trainees. Overall, doctors should mirror in other ways the make-up of society, such as similar proportions in sex ratio, ethnicity and social class. Equal sex ratio has been achieved, if not exceeded in the UK, some, but not all, ethnic groups are proportionally represented, but with social class origin, the disadvantaged are poorly represented[6]. These issues can be ameliorated by flexible training, although the solution lies with Medical School selection and pre-University education.

Summary

The advantages and problems of competency-based medical training have been discussed, as developed in the UK. Although only postgraduate and pre-specialist training has been described, training is regarded as a continuum, from entering medical school to completion of medical practise, and the issues of continuous professional development and revalidation relate to this. The UK model will continue to develop, and whilst responding positively to constructive criticism from within the profession, hopefully, will not be deterred from ensuring that doctors are suitably trained to meet the expectations of civilised society, unfortunately, the experiences of the regressive changes to the second Foundation year highlights that impetus for radical change could be lost. Implementation, especially within a resource-poor environment, may well be costly, and change will be threatening to more established clinicians, but streamlined training, producing doctors better able to face the challenges ahead, and avoid the wastage of trainees who fail to complete, or have very prolonged unsatisfactory training, has great advantages to trainees and society, compared to a traditional highly competitive pyramidal career structure. It has been suggested that excellence is being sacrificed to deliver competence, but a well-structured programme can produce both, including high academic achievement.

Fenk, Chen, Bhutta et al[7], in a report commissioned by The Lancet, proposed a global approach to reform medical education. Commenting on the failure of medical care to address the needs of most populations, they supported competency-based curricula, responsive to changing needs and local contexts, and promotion of inter-professional education, especially for training in generic skills. Information technology can now support learning across national boundaries, and professions, which could lead to internationally recognised common accreditation. Increasingly, in some countries, medical training is moving towards this goal, being patient-centred but with a commitment to the needs of society, in others there is still a way to go[8.] To achieve a global health care system, responsive to need, will require considerable maturity from politicians, the professions and the public. Enthusiasm by medical students may well help to push this agenda forward[9].

(J Bangladesh Coll Phys Surg 2011; 29: 58-61)

R H Robson

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Microbial Isolates from Patients and their Antibiogram at the Tertiary care Burn Unit in Bangladesh

H BEGUM[a], M QUAMRUZZAMAN[b], M TALUKDAR[c]

Summary

Infection and antibacterial resistance are important issue in severe burn. This prospective study was carried out in 112 patients who were enrolled from July 2007 to June 2008 in DMCH burn unit (only dedicated burn facility in Bangladesh with a mean annual admission of 869). The aim of this study were to investigate the profile of microorganism and resistance to antimicrobial agents; individuals who were admitted more than 5 days, with partial or full thickness burn developed clinical signs and symptoms of wound infection or pneumonia or septicaemia were included in this study. Nearly 50% of participants were aged 11-30 yrs, the most common type of burn was flame burn and females were the common victims. Bacterial isolates were found in 104 (92.85%) samples and eight (08) swabs were sterile (7.14%). Pseudomonas species was the commonest pathogens (46.42%) followed by Proteus species (21.41%) and Klebsiella species (19.6%). Multiple organisms were found in 38 samples(33.92%). Antibiogram results obtained from ten antimicrobial agents demonstrated that Imipenem was the most efective agent, followed by amikacin and ceftazidime (92.3%, 52.8% and 38.46% sensitive respectively). Hundred percent (100%) of Proteus species were sensitive to imipenem, then amikacin 58.33%; they were highly resistant to cephalosporins (nearly 100%) and ciprofloxacin (83.33%). The resistance of Pseudomonas aeruginosa to anti-pseudomonas agents were exceptionally high. Newer drugs were found to be effective.

(J Bangladesh Coll Phys Surg 2011; 29: 62-66)

Introduction:

Burn wounds are never sterile, even in the presence of topical agents or systemic antibiotics.[1] Severe burn caused complex changes in homeostasis that can hardly be compared with other traumas or disorders and mortality is relatively common in the early phase. Infection is an important cause of mortality in burn, if the burn exceeds 40% of the total body surface area (TBSA). Wound infection will be one of the main complications[2]. Sepsis is the leading cause of death in patient with large burns.[3] 75% of all deaths following burn injury are related to infection[4]. Infection in the wound prolongs the healing process; treatment includes rational antibiotic administration, removal of necrotic tissues, sufficient blood and oxygen supply to the wound and good nutritional support in burn victims.[5] Individual hospital units will notice a change in their common pathogens over time. Infetions with Pseudomonas organisms began to be seen in increasing numbers.[6] From 11% to 30% of burns are contaminated by microorganism of the gastrointestinal tract, skin and upper respiratory system, including Pseudomonas aeruginosa, Staphylocoocus aureus, Escherichia coli, Klebsiella spp, Enterococcus spp and Candida spp.[7] The organism most often involved in wound infection particularly in the first week is Staphylocoocus aureus.[8] Infection with gram-negative organism is more evident after the 1st week. Pseudomonas organism is present on the wounds of approximately 25% of burn patients.[1] Enterococci and Candida albicans are now seen with increasing frequency, each being found in the wounds of about 50% of burn patients[1]. The rate of nosocomial infections are higher in burn patients due to various factors like nature of burn injury itself, immunocompromised status of the patients, invasive diagnostic and therapeutic procedures and prolonged ICU stay. In addition, cross-infection results between

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different burn patients due to overcrowding in burn wards. Antimicrobial resistance is a great problem in infectious disease. In burn units, because of the wide use of antibiotics and particularly the empirical administration of broad-spectrum antimicrobial, this problem is worse than in other hospital department.[9] Complicating this high rate of infection is the fact that the spectrum of bacterial isolate varies with time and geographical area.[10]

The present prospective study was conducted to determine the bacteriological profile and the resistance pattern in the burn unit of DMCH, a tertiary referral center, Bangladesh, which forms the basis for modification of drug regimen strategy.

Material and Methods:

This prospective cross-sectional study involved 112 patients who were enrolled from July 2007 to June 2008 in burn unit, DMCH, which is the only dedicated burn facility in Bangladesh with a mean annual admission of about 869. It receives all severe burn cases and more than 90% of burn cases in Bangladesh.

Early excision and skin grafting is practiced in our unit as soon as the patients are surgically fit and repeated till all areas of the body are grafted. Superficial burns not requiring surgery are dressed with duoderm to permit moist wound healing. Extent and severity of burns are calculated by assessing total percentage of body surface area (TBSA) burnt. Individuals who were admitted more than 5 days, with partial or full-thickness burn who developed clinical signs and symptoms of wound infection or pneumonia or septicaemia were included in this study.

Demographic and clinical data including gender, age, degree of burn, TBSA burnt, cause of burn and antibiotic therapy were collected for each participant.

According to the clinical status of the participants, appropriate samples including wound swab, urine and blood samples were taken. The first swab was obtained from deep areas of the burn before any cleaning and transferred to the laboratory by sterile test tube. Blood and urine samples were collected from individuals with signs and symptoms of septicaemia or pneumonia. Blood sampling was repeated three times.

Descriptive statistics were used for demographic and bacteriological profiles of each case; χ^2 testing compared the microorganisms sensitivity and resistance patterns to the examined antimicrobial; $p<0.05$ was considered significant.

Result:

A total 24, 354 patients were attended in DMCH burn unit from July 2007 to June 2008, of which 2376 (198/month) acute burn patients were admitted; 1613 (67.88%) patients were discharged with or without some residual deformity; 547 (23.02%) patients were expired; 557(23.56%) patients leave the hospital with or without consent of the authority. (Table-I).

Table-I

Burn & Plastic Surgery Unit

Burn patients hospital outcome and pattern :

Month	Total Patients Attended Emergency & OPD	Total Pateints attended				Total Patients admitted	Discharged (%)	DORB & Absconded	Death (%)
		Flame Burn	Scald Burn	Electric Burn	Chemical Burn				
July'07	1496	90	90	65	06	95	97	17	24
August'07	1829	145	137	10	04	60	87	06	24
September'07	1487	132	86	39	04	72	104	14	31
October'07	1477	93	49	37	05	80	134	31	35
November'07	1731	85	87	74	02	245	122	21	34
December'07	1833	112	96	68	07	317	207	54	51
January'08	2774	90	90	65	06	260	136	105	83
February'08	3348	145	137	10	04	314	128	96	89
March'08	3351	132	86	39	04	274	114	65	62
April'08	1537	93	49	37	05	221	171	65	41
May'08	1552	89	47	40	03	219	161	41	43
June'08	1939	88	50	49	04	219	152	45	30
Total	24354	1294	1004	533	54	2376	1613 (67.88%)	550 (23.56%)	547

A total 112 people were included in this study. Their characterstics including gender, distribution of age, cause of burn, and TBSA burnt are presented in Table - 2. Nearly 50% of the participant were aged 11 to 30 yreas. The most common type of burn was flame burn and females were the common victims.

Bacterial isolate were found in 104 (92.85%) samples and only eight (08) swabs were sterile 7(14%). Pseudomonas species was the commonest pathogen isolated (46.42%) followed by proteus species (21.4%), Klebsiella species (19.6%), Providencia (19.6%), E.coli (14.2%), Acinetobacter (12.5%) and Staphylococcus aureus (7.1%). Multiple organism were found in 38 samples (33.92%), None of the 5 blood sampls contained them. Bacterial isolate were found in only in 9 urine samples (8.03%) of which pseudomonas species was the commonest (3.57%), as shown in Table-III.

Antibiogram of Pseudomonas Aeruginosa and Proteus species to 10 antimicrobial agents including carbapenems (Imipenems), Cephalosporins, fluoroquinolones, aminoglycosides and netilmicin are presented in Table-4. The most effective agent against P. aeroginosa was Imipenem (92.30%). After that, Amikacin and ceftazidime were the most effective agents at 53.80% and 38.46% respectively. Most isolates of P.aeroginosa were resistant to ampicillin (100%), cephalexin (96.15%), cefixime (92.30%) and ceftriaxone (88.46), 100% bacterial isolates of Proteus spp. were sensitive to imipenem, then amikacin 58.33%; they are highly resistant to cephalosporins (nearly 100%) and ciprofloxacin (83.33%).

Table-II

Characterstics of burn patients (n-112)

Age Of the Patients			Gender of the patients				Causes of Burn			TBSA % of burn	
Age in yrs	No	%	Male	Female	Type	Number	%	% of burn	No	%	
0-10	26	19.6	No.	%	No.	%	Scald	30	26.78	<10	14	12.5
11-20	20	14.28	40	35.71	72	64.28	Flame	50	44.64	11-20	20	17.85
21-30	38	32.14					Electric	22	19.64	21-30	32	28.57
31-40	10	8.9					Gas Explosion	10	8.9	31-40	28	25
>40	18	16.07					Chemical	0	0	41-50	10	8.9
										>50	8	7.14

TBSA = Total Burn Surface Area

Table-III

Microorganisms isolated (n=112)

	Sample	Positive	No growth	Microorganism isolated						
Wound swab	112	104 92.85%	08 7.14%	Pseudo. 52 (46.42%)	Kleb. 22 (19.6%)	Proteus 24 (21.4%)	E. coli 16 (14.2%)	Staph. 08 (7.1%)	Acinetobacter 14 (12.5%)	Providencia 22 (19.6%)
Blood	05	No growth								
Urine	112	09 (8.03%)	103 (91.9%)	4 (3.57%)	1 (0.89%)	2 (1.78%)	2 (1.78%)	-	-	-

Combined growth —— 38 (33.92%)

Table-4a

Antibiogram for Pseudomonas spp. (n=52).

Antibiotic	Sensitive	Intermediate	Resistance
Imipenem	48 (92.30%)	-	4 (7.6%)
Ciprofloxacin	6 (11.3%)	-	46 (88.46%)
Amikacin	28 (53.8%)	2 (3..8%)	22 (42.33%)
Gentamycin	6 (11.53)	2 (3.8%)	44 (84.61%)
Ceftazidime	20 (38.46%)	2 (3.8%)	30 (57.69%)
Ceftriaxone	2 (3.8%)	4 (7.6%)	46 (88.46%)
Cefixime	4 (7.6%)	-	48 (92.30%)
Cephalexin	2 (3.8%)	-	50 (96.15%)
Ampicilin	-	-	52 (100%)
Netilmicin	16 (30.76%)	-	36 (69.23%)

Table-4b

Antibiogram for Proteus spp. (n=24).

Antibiotic	Sensitive	Intermediate	Resistance
Imipenem	24 (100%)	-	-
Ciprofloxacin	-	4 (16.66%)	20 (83.33%)
Amikacin	14 (58.33%)	2 (8.3%)	8 (33.33%)
Gentamycin	4 (16.66%)	-	20 (83.33%)
Ceftazidime	8 (33.33%)	-	16 (66.66%)
Ceftriaxone	2 (8.3%)	2 (8.3%)	20 (83.33%)
Cefixime	-	-	24 (100%)
Cephalexin	-	-	24 (100%)
Ampicilin	-	-	24 (100%)
Netilmicin	8 (33.33%)	-	16 (66.66%)

Discussion:

Burn patients constitute a subset of patients particularly prone to infection. In the present study the most commonly isolated organisms from burn patients were Pseudomonas species followed by Proteus spp. and Klebsiella spp. and the high resistance rate of P. aeruginosa to common anti Pseudomonas agents. Compared with other studies, it was highly corresponds with the study of G. Khorasania et. al [2]. Like our study Agnihotri et al[11] reported that 96% of swab were positive and P aeruginosa was found to be the most common isolates (59%). These results contrast to the study of Mehta Manjula et al[12]. there was high incidence of Staph aureus isolation next to Pseudomonas. They also found a changing trend in burn bacteriology, it was decreased for Pseudomonas spp. Staph. aureus and Proteus to increase for Klebsiella species. The prevalance of Staph. aureus was very low in our study (only 7.1%). Another significant difference of our study results to the other studies is that the prevalance of Acinetobacter spp. is very low (12.5%). There was significant rise in the isolation rate of Acinetobacter spp. over the last five to eight years as stated by Sengupta et al[13]. Acinetobacter spp. are emerging as an important cause of nosocomial infection in burn units. There are a number of factors which may contribute to this increase like its presence as a normal skin commensal and its easy spread due to multi drug resistance in a hospital settings.

G Khorasani et al[2] found high prevelance of Citrobacter freundii in their study and they found citrobacter in all clinical samples except blood.

The high prevelance of P.aeruginosa is in agreement with other studies [2,11,12,13,14] and may be explained by the fact that this opportunistic microorganism grows mainly in moist body areas, such as burn wounds[15] and also by prolonged hospital stay and the administration of broad spectrum antibiotics in burn cases. A burn represents a site susceptible to opportunistic colonisation. The situation of burn victims with P. aeruginosa infection is particularly problematic, since this organism is inherently resistant to many drugs and is able to acquire resistance to all effective antimicrobial drugs.[16] The change in the pattern of bacterial resistance in the burn unit has importance both for clinical settings and epidemiological purpose. We saw a significantly high percenatge of resistance among gram-negative bacilli to aminoglycosides like gentamicin and amikacin, ciprofloxacin, amoxicillin, cefotaxime' and ceftriaxone. This alarming trend was seen for both Enterobacteriaceae group and Pseudomonas spp. as seen in the study of Mehta et al[12]. A similar report of multi-drug resistant gram-negative bacilli was also reported by Singh *et al*[17]. In comparison, imipenem and combination of drugs were found to be effective. This could be due to the reason that these are reserve drugs and used as last options for multi drug resistant bacteria in our hospital settings. Such high antimicrobial resistance is probably promoted due to selective pressure exerted by an bacteria due to numerous reasons like non adherece to hospital; antibiotic policy and excessive and indiscriminate use of broad spectrum antibiotics.

These multidrug resistant strains establish themselves in the hospital environment in areas like sinks, taps, railing, mattress, toilets and thereby spread from one patient to another. The implementation of a strict infection control strategy with a more rational use of antibiotics, including topical formulations and antimicrobial rotation, has been proposed to prevent the high incidence of multi-drug resistant strains of microorganism, particularly of P. aeruginosa in burn units[18].

Conclusion:

Routine microbiological surveillance and careful in vitro testing prior to antibiotic use and strict adherence to hospital antibiotic policy may help in the prevention and treatment of multi-drug resistant pathogens in burn infection.

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Review on Treatment of Gout & Hyperuricemia

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Abstract

In the last few decades, both hyperuricemia and gout have increased markedly. Recent studies show new concept into the transporters that handle uric acid in the kidney as well as possible links between these transporters & hyperuricemia. There are changes in the treatment of established hyperuricemia. Febuxostat and PEGuricase are two novel treatments that have been evaluated and shown to be highly effective in the management of hyperuricemia. Monosodium urate (MSU) crystals are the inducers of inflammation. Within the joint, they trigger a local inflammatory reaction, neutrophil recruitment, and the production of proinflammatory cytokines as well as other inflammatory mediators. The uptake of MSU crystals by monocytes involves interactions with components of the innate immune system. The inflammatory effects of MSU are IL-1-dependent and can be blocked by IL-1 inhibitors. These advances in the understanding of hyperuricemia and gout provide new therapeutic targets for the future.

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Introduction

Hyperuricemia and gout is a common metabolic disorder all over the world as well as in Bangladesh[1,2]. Among the countries of the West, in the United States, the overall prevalence of gout and hyperuricemia was 41 per 1000 in 1999 and in the UK the overall prevalence of gout is 1.4%[3,4].

All patients with gout have hyperuricemia (supersaturation of serum for urate) at some point in their disease. However, most hyperuricemic individuals never experience a clinical event resulting from urate crystal deposition. Thus, the diagnosis of gout focuses on the fundamental pathophysiologic events defining the clinical state: tissue deposition of urate crystals; and the accompanying inflammatory and degenerative consequences. Within this framework, hyperuricemia is viewed as a necessary but not sufficient precondition for the development of urate crystal deposition disease, and is to be distinguished from gout, the clinical syndrome. Gout is a common medical problem, affecting at least 1 percent of men in Western countries, with a male: female ratio ranging from 7: 1 to 9: 1. Statistically normal uric acid levels in men and premenopausal women (7 mg per deciliter [416 ìmol per liter] and 6 mg per deciliter [357 ìmol per liter], respectively) are close to the limits of urate solubility (approximately 7 mg per deciliter at 37°C) in vitro, imposing a delicate physiologic urate balance. Hyperuricemia is central to gout but does not inevitably cause disease.

The classic symptom of gouty arthritis is recurrent attack of acute, markedly painful monoarticular or oligoarticular inflammation[77], but polyarticular gout and chronic arthritis can occur. Definitive diagnosis requires the direct identification of urate crystals in the joint and the exclusion of infection. Serum urate levels are frequently normal during attacks of acute gout. The accuracy of clinical diagnosis without crystal confirmation[5] is uncertain. For this reason diagnosis in our country is still a challenge as polarized microscopy is still not widely available.

Asymptomatic hyperuricemia is the term applied to settings in which the serum urate concentration is

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elevated, but neither symptoms nor signs of urate deposition have occurred. Although gout may develop in a hyperuricemic individual at any point, it is likely that two-thirds or more of hyperuricemic individuals will remain asymptomatic. [6-10]

Asymptomatic hyperuricemia alone has not been related to the development of clinically significant renal disease in large cohorts and in itself is not an indication for treatment unless rises above 12 mg/dl. [11] It remains uncertain whether gout and hyperuricemia are independent risk factors for vascular disease in humans. Large epidemiologic studies have shown that hyperuricemia is associated with an increased incidence of CHD and increased mortality in those with and without preexisting CHD[12-15].

Dietary association and contributory factors as long been discussed with gout. Many scientific studies have also been performed. Purine content of food has been the centre of discussion. Higher levels of meat and seafood consumption are associated with an increased risk of gout, whereas a higher level of consumption of dairy products is associated with a decreased risk[16-18]. however strict dietary restriction is no longer recommended.

The goal of therapy in an acute gout attack is prompt and safe termination of pain and disability. While symptoms will resolve without therapy within a few days to several weeks, symptoms improve more quickly with administration of any of a broad array of anti inflammatory drugs, with the most prompt and complete resolution when the treatment is introduced earlier[19,20].

Our aim & objective is to aware physicians & patients regarding pitfalls in the management of Hyperuricemia & gout. They also should be aware of several newer approaches that are emerged in the recent past.

Materials and methods

The material for this review was taken mostly from Rheumatology textbooks & electronic journal "Uptodate." To collect publication PubMed and the Cochrane database of systematic reviews was used. Some other relevant references were collected from personal database of papers on gout.

Clinical Features and Natural Course

There are four phases of gout characterized by asymptomatic hyperuricemia, recurrent attacks of acute arthritis, intercritical gout, and chronic tophaceous gout. Ten to fifteen percent of all the patients having hyperuricemia develop clinical gout[21,22]. After many years of asymptomatic hyperuricemia, patients may experience their first attack of a painful gouty arthritis. A family history of gout is identified in 10–25% of patients. Female patient accounts for just 5–8% of total gout cases. In female, during reproductive age gout is rare; however incidence is equal in both sexes during post menopausal period. Early onset in female is mostly associated with the use of cyclosporine, diuretic, or renal insufficiency, and may present with polyarticular attack[23]. The primary manifestation of acute gouty attack is very painful arthritis, usually monoarticular at first but later may be polyarticular and accompanied with fever. The pain peaks within 1–2 days and usually subsides within 3–10 days even without treatment but in rare occasion may last for a couple of weeks.

First metatarsophalangeal joint (podagra) comprises for at least 50% of initial attack. Eighty percent of patients experience an acute attack of first MTP in their life time. Following the first metatarsophalangeal, the sites of initial attack are, in order of frequency, the insteps, ankle, heel, knee, wrist, finger, and elbow joints. Notably, the proportion of initial ankle joint attacks recently has been increasing[24,25]. The hallmark of gout is the acute attack of inflammatory monoarthritis. Early attacks tend to subside spontaneously, and most of the patients do not have residual symptoms until the next episode. As acute gout tends to be recurrent, a history of previous self-limiting episodes is a helpful point for clinical diagnosis. Atypical presentations of gout include polyarthritis, inflammation of the periarticular structures and febrile reactions. Provocative factors include trauma, alcohol ingestion (particularly beer consumption), initiating urate-lowering drugs, excess purine in the diet, or surgery. The periods between gouty attacks are called interval (or intercritical) gout[77]. In most patients, second attacks occur within 6 months to 2 years. In the series described by Yu, 61% of patients had recurrence within the first year, 27% in 1 to 2 years, 5% in 2 to 5 years, 3% in 5 to 10 years, and 4% had experienced no recurrence at ten or more years. Thus, urate lowering drug is not recommended in first gouty attack rather patient should be kept under observation and if attack is frequent i.e. more than two attacks per year, then urate lowering drug should be started[26,27]. The natural course

of gout remains unclear because there was only one report in the pretreatment era by Gutman. Gutman found severe gout with 44% of 10 Gout and hyperuricemia patients developed tophi, and 30% of patients had demonstrable tophi 5 years after diagnosis of gout, which increased to 50% by 10 years, and up to 72% by 20 years. In the followup series of Gutman, reported by Yu in 1984, there were still 34% of gout patients with tophi[34,28]. In a recent study in Taiwan, 21.1% of hospital patients and 12.7% of community outpatients with gout had tophi[29]. In another large outpatient series reported by Chan, approximately 9% of gout had tophi[30]. Other large series reports on proportions of tophacious gout varied widely. Most reports included only hospital cases, and most patients were post treatment[31,66]. The 1977 American Rheumatism Association classification criteria provide a clinical diagnostic guide for gout. The presence of characteristic urate crystals in joint fluid, or a tophus demonstrated to contain urate crystals either chemically or by a polarized light microscopy means, or the presence of Six or more of the following criteria are needed to make a diagnosis: More than one attack of acute arthritis, Maximum inflammation developed within one day, Attack of monoarthritis, Redness over joints, Painful or swollen first metatarsophalangeal joint, Unilateral attack on first metatarsophalangeal joint, Unilateral attack on tarsal joint, Tophus (proved or suspected), Hyperuricaemia, Asymmetric swelling within a joint on radiograph, Subcortical cysts without erosions on radiograph, Joint fluid culture negative for organisms during attack.[33] The symptom free interval between acute attacks may last a few weeks to several years.

The development of subcutaneous tophi before first gouty attack is very rare. Invisible intraarticular tophi may present earlier than first gouty attack, which was induced by crystal shedding[34,35]. Extracellular urate crystals can be found in more than two thirds of aspirates from previously affected joints during asymptomatic intervals, while less than 5% of asymptomatic hyperuricemia subjects without gout have such crystals[36,37]. Gout can be diagnosed based on clinical features alone, but definite diagnosis requires aspiration and examination of synovial fluid, with identification of the causative crystals[38]. The main differential diagnosis of gout includes septic arthritis, cellulitis, pseudogout, palindromic rheumatism, bunion, seronegative spondyloarthropathy, and rheumatoid arthritis. In case of prolonged attack, one should consider infection (septic arthritis or concomitant septic and gouty arthritis)[39], loss of the golden time of initiation of colchicines (within 48-72 hours) for acute attack, drug-related serum urate level fluctuation, mixed crystals (monosodium urate and calcium pyrophosphate dihydrate) deposition disease, or reconsider if it is really gouty arthritis. Although hyperuricemia is a necessary condition for the development of gout, it is worthwhile to note that around 30% of gout patients had normal serum urate levels during acute gouty attack[40-43]. Tophi deposits are well known to cause joint destruction, gouty nephropathy, carpal tunnel syndrome, limited joint range of motion and joint deformity[46], spinal cord compression[47], concomitant with septic infection[48], and associated with life threatening necrotizing fasciitis[48].

Risk factors for Gouty arthritis

Risk factors other than inherited abnormality are obesity (excessive weight gain specially in youth), moderate to heavy alcohol intake, high blood pressure, and abnormal kidney function. Thiazide diuretics, low dose aspirin, niacin, cyclosporine, anti-tuberculous drugs (pyrazinamide, ethambutol) elevated serum uric acid level can also lead to gout. Diseases including leukaemia, lymphoma, and hemoglobinopathies are associated with raised uric acid in the blood. Dehydration, injury to the joint, fever, excessive eating, heavy alcohol intake, and recent surgery have been reported to precipitate gouty attack in patients at risk of developing gout[16-18].

How to diagnose?

Gout is suspected when a patient reports a history of attacks of painful arthritis, particularly at the base of the toes. Ankles and knees are the next most commonly involved joints in gout. Gout usually attacks one joint at a time, while other arthritis conditions, such as systemic lupus and rheumatoid arthritis, usually attack multiple joints simultaneously.

The most reliable test for gout is finding uric acid crystals in a sample of the joint fluid obtained by joint aspiration (arthrocentesis). Arthrocentesis is a common office procedure performed under local anesthesia. Using sterile technique, fluid is withdrawn (aspirated) from the inflamed joint using a syringe and needle. The joint fluid is then analyzed for uric acid crystals and for infection. Shiny, needle-like uric acid crystals are best

viewed with a special polarizing microscope. The diagnosis of gout can also be made by finding these urate crystals from material aspirated from tophi nodules and bursitis fluid.[34-37]

Sometimes patients with a classic history and symptoms of gout can be successfully treated and presumed to have gout without undergoing arthrocentesis. However, establishing a firm diagnosis is still preferable since other conditions can mimic gout. These include another crystal-induced arthritis called pseudogout, psoriatic arthritis, rheumatoid arthritis, and even infection in the joint.

X-rays can sometimes be helpful and may show tophi-crystal deposits and bone damage as a result of repeated inflammations. X-rays can also be helpful for monitoring the effects of chronic gout on the joints[44]. The characteristic radiography of gout comprises well-defined, punched-out erosions with overhanging edges, with preservation of the joint space, asymmetrical involvement, and soft tissue nodules shadow[44]. On magnetic resonance imaging, tophi generally have low signal intensity on both TI- and T2-weighted images and a variable enhancement pattern[45,46].

Treatment of gout and hyperuricemia

Treatment of Acute Gouty Arthritis

Acute gout is a self-limiting condition typically lasting less than one week, but treatment ensures pain relief and speeds recovery. The sooner drug treatment is started, the quicker the response. As gout is likely to recur, giving patients a supply of NSAIDs or colchicine to start treatment at the onset of the next episode is important. The mainstay of treatment during an acute gouty attack is the administration of non-steroid antiinflammatory drugs (NSAIDs), colchicine, or corticosteroids depending on the Comorbid conditions of the patient[78]. The basic principles of gout treatment are: (1) terminate acute attack as promptly and gently as possible, (2) prevent recurrence of acute gouty arthritis, (3) prevent or reverse complications resulting from deposition of monosodium urate in the joints, kidneys, or elsewhere, (4) prevent or reverse associated conditions such as obesity, hyperlipidemia, or hypertension, (5) prevent the formation of uric acid urolithiasis. The drug of choice for acute attack in most patients would be a NSAID provided that there were no contraindications. Cyclooxygenase-2 highly selective inhibitors are probably equally effective as traditional NSAIDs but have less short-term gastrointestinal toxicity[51]. Anti-inflammatory drugs should be gradually tapered when improvement occurs. The second choice drug is colchicine because of its narrow therapeutic index[52]. The choice of NSAID versus colchicine depends on individual assessment of cardiovascular, gastrointestinal, and renal risk factors. The American Association of Poison Control Centers toxic exposure system recorded 33 colchicine-related deaths from 1985 to 1997[53]. Owing to its narrow therapeutic margin, intravenous colchicine are no more recommended. Oral colchicine is more effective when administered in the first 24 hours after onset of an acute attack, before phagocytosis establishes itself. A standard oral regimen is 0.5 mg each hour or 1.0 mg every 2 hours until pain relief or side effects (vomiting or diarrhea) occur, or until a maximum of 6 mg is taken (but less than this amount of colchicine in those with renal insufficiency and in the elderly). However, up to 80% of patients are unable to tolerate an optimal dose owing to gastrointestinal side effects. A useful alternative approach is to add low doses of oral colchicine (0.6 mg qd–bid) as an adjunct to another better-tolerated primary treatment approach (e.g. NSAIDs). The administration of low daily dose of colchicines provides effective prophylaxis against further acute attacks[54]. Corticosteroids were advised when colchicine and NSAIDs are ineffective or are contraindicated during gouty attack. Intraarticular corticosteroids are usually reserved for patients suffering attacks of one or two large joints who also had contraindication or intolerance to NSAIDs or colchicines. In patients with polyarticular gout in whom other treatments are difficult, systemic corticosteroids are an option. Oral prednisolone (20–30 mg/day initially, then taper the dose gradually) or equivalent is also effective[55]. High doses of steroids have been reported to be associated with higher rates of rebound gouty attack.

Treatment of Chronic Gout

Gout is characterized by recurrent, painful attacks of acute arthritis. If left untreated, gout may become chronic and tophaceous depositions may result in destructive arthritis and loss of joint function. The pharmacological treatment of hyperuricemia in gout is generally very effective. The main reason for treatment failure is poor compliance, and patient education

improves compliance. Patients should be instructed that allopurinol or uricosuric drugs have no role in the treatment of the acute gouty attack, and that urate lowering drugs do not have anti-inflammatory properties. Drugs used to lower serum urate are aiming to reduce serum urate level to below the solubility limit (usually set at < 6 mg/dl for non-tophaceous and < 5 mg/dl for tophaceous gout patients). Urate-lowering therapy is a long term commitment and patients who have had just one or two acute episodes are unlikely to be compliant[56]. Recurrence is possible if treatment is intermittent or is withdrawn after apparent good control[57-58]. Current evidence does not support treating asymptomatic hyperuricemia[39,59,60]. Since only approximately 0% of asymptomatic hyperuricemic subjects develop gouty arthritis and the risk of urolithiasis in asymptomatic hyperuricemia subjects is small (just one case per 295 subjects each year), the risk of azotemia (follow up eight years) in asymptomatic hyperuricemia is 1.8% (2/113), compared with 2.1% (4/193) for the control subjects. The workup of gout and asymptomatic hyperuricemic subjects includes defining the cause of the hyperuricemia, which may disclose some other important diseases besides gout. Indications for the initiation of urate-lowering drug therapy are controversial but most physicians consider drug therapy in the event of two or more acute attacks of gout per year, tophi, bone destruction, uric acid urolithiasis, or to prevent acute uric acid nephropathy[32].

Urate-lowering Drug

Serum urate levels can be reduced by using medication that increases renal excretion of uric acid (uricosuric drug) or decreases uric acid production (xanthine oxidase inhibitor). Xanthine oxidase inhibitors (allopurinol, febuxostat) reduce uric acid production through competitive inhibition of xanthine oxidase, which converts hypoxanthine to xanthine and xanthine to uric acid. With rigorous medical compliance, allopurinol shrinks tophi and in time can lead to their disappearance. Resorption of extensive tophi requires maintaining a serum uric acid below 5 mg/dL, which may be achievable only with concomitant use of allopurinol and a uricosuric agent. Surgical excision of large tophi offers mechanical improvement in selected deformities. Allopurinol is usually initiated at a single daily dose of 100 mg and gradually increased[81]. Most patients require 300 mg/day, and the maximal dose is 800 mg/day. Oxipurinol is the major metabolite of allopurinol and has a half-life ranging from 18 to 30 hours, thus allopurinol can be given once daily. Since oxipurinol is largely excreted in the urine, its half-life is prolonged in patients with renal insufficiency and the dose of allopurinol thus should be reduced in patients with renal insufficiency[61]. If allopurinol and azathioprine are used together, the doses of azathioprine should be reduced by 75% as both drugs are metabolized by xanthine oxidase[62]. Uricosuric drugs (probenecid, sulfinpyrazone, and benzbromarone) increase urinary uric acid excretion by inhibiting renal tubular urate reabsorption. Uricosuric drugs are risky if urinary urate excretion is >800 mg/day while on a normal diet, and are contraindicated in individuals with uric acid calculi. Urine alkalinization can be indicated in a few high risk patients, particularly in those with a history of uric acid urolithiasis, and uricosuric drugs are absolutely required. Giving sodium bicarbonate at a dose of one gram three to four times daily, or giving potassium citrate, minimizes crystallization, which is likely to occur in acidic urine. It is important to ensure adequate urine amount and fluid intake to reduce the risk of uric acid stone formation. The transient increase in uric acid excretion during uricosuric drugs treatment may lead to the development of uric acid urolithiasis in a small proportion of gout patients. To avoid this complication, uricosuric drugs may be initiated at low doses and gradually increased as necessary. Precautions with uricosuric drugs include maintaining a daily urinary output of 2000 mL or more in order to minimize the precipitation of uric acid in the urinary tract. This can be further prevented by giving alkalinizing agents (eg, potassium citrate, 30–80 mEq/d orally) to maintain a urine pH of above 6.0. Uricosuric drugs are avoided in patients with a history of uric acid nephrolithiasis. Aspirin in moderate doses antagonizes the action of uricosuric agents, but low doses (325 mg or less per day) do not; doses greater than 3 g daily are themselves uricosuric. Probenecid and sulfinpyrazone might be ineffective in patients with creatinine clearance of below 30–50 ml/min, while benzbromarone (not available in the United States) might be ineffective given creatinine clearance of below 25 ml/min[63]. Probenecid is usually initiated in doses of 250 mg twice a day, and increased over several weeks to the dose necessary to achieve the target goal (serum urate < 6.0 mg/dl). A total dose of 1 g/d is appropriate for roughly 50% of patients, and the

maximal dose should not exceed 3 g/d. Because the half-life is 6 to 12 h, probenecid should be administered in two or four evenly spaced doses per day. Sulfinpyrazone is usually started at a dose of 50 or 100 mg twice a day and gradually increased to a maintenance dose of 300 to 400 mg/d, administered in three or four doses. The maximal dose is 800 mg/d. Benzbromarone is usually started at 50 mg/day, and the maximal dose is 100 mg/day (maximal dose is 150–200 mg/day for unmicronized benzbromarone). Benzbromarone associated fulminant hepatic failure has been reported[64,65]. Urate lowering drugs are of equal effectiveness. However, drug safety is of paramount importance in choosing hypouricemic agent. Almost all currently available urate-lowering agents have side effects, which are sometimes severe and life-threatening. Although the risk is very low, it exists and continuously happens[66,67]. Choosing the most appropriate urate-lowering agent for a patient may avoid unnecessary complication. Treatment with urate-lowering drugs should be carefully evaluated and considered on an individual basis. Generally, uricosuric drugs such as probenecid, sulfinpyrazone, or benzbromarone, are safer than allopurinol[66,67]. Whether febuxostat[40], a novel nonpurine selective inhibitor of xanthine oxidase, is safer than allopurinol but requires large scale post-marketing surveillance. Febuxostat is superior to allopurinol both in short and long term treatment in lowering serum uric acid and resolving tophi[79]. Febuxostat is approved for use in European countries at 80 and 120 mg daily. The FDA approved febuxostat for use in the USA in February, 2009[80]. Side effects of febuxostat include rash and elevation of hepatic enzymes, diarrhoea, and arthralgia may occur[80]. Benzbromarone and allopurinol have the advantage of once daily dosing. Benzbromarone have been reported to be associated with hepatotoxicity[64,65] and have been withdrawn from certain European countries, either by the government or by pharmaceutical company policy[66]. This has lead to allopurinol becoming the only urate-lowering medication available for gout in the Netherlands, and whether this action increases allopurinol-related morbidity and mortality deserves further observation. Allopurinol has been considered the drug of choice for hyperuricemia by some[67] because it can be conveniently administered once daily, and might prevent urolithiasis. The use of allopurinol should have indication to avoid the unnecessary, rare but potentially life threatening complications[52,53]. Specific indications for choosing allopurinol over a uricosuric drug include: (1) increased urinary uric acid excretion (>800 g/d on a general diet), (2) impairment of renal function with creatinine clearance of less than 30–50 mL/ min, (3) uric acid urolithiasis, and (4) gout not controlled by uricosuric drugs because of ineffectiveness or intolerance. Severe allopurinol hypersensitivity is more likely to occur in patients with renal impairment or in those receiving thiazide diuretics or ampicillin. Hung et al. indicated that HLA-B*5801 allele is a genetic marker for severe allopurinol hypersensitivity syndrome[70]. If uricosuric drugs are contraindicated and the reaction is mild, patient can be desensitized by administering an initial dose of 25–50 ìg of allupurinol. Febuxostat and oxipurinol are other alternative approaches. Allopurinol and a uricosuric drug may be used simultaneously in a few patients who cannot be controlled with a single medication. Uricase catalyses the conversion of urate to more soluble allantoin and is very effective at reducing urate level[71], but is potentially antigenic and contraindicated in G-6-P D deficiency. Pegluticase, a formulation of uricase with polyethylene glycol reduces antigenicity and prolongs the half-life of uricase, and may have a role in difficult gout patients in the future.

Treating Asymptomatic Hyperuricemia

Antihyperuricemic drug therapy for the great majority of individuals with asymptomatic hyperuricemia is not justifiable by risk/benefit analysis. As mentioned above, gouty arthritis is readily treatable and reversible if it occurs. Similarly prophylaxis against stone disease is not warranted in most individuals, but therapy should be started after discovery of a stone. The primary therapeutic modality in this setting is urinary alkalinization with potassium citrate or potassium bicarbonate, not allopurinol. Specific circumstance that warrant at least consideration for the institution of antihyperuricemic treatment in asymptomatic subjects is persistent hyperuricemia in the infrequent patients with sustained serum urate concentrations greater than 13 mg/dL (773 µmol/L) in men and 10 mg/dL (595 µmol/L) in women. These high values may carry some nephrotoxic risk, perhaps related to the likelihood of some component of uric acid overproduction. This recommendation does not generally apply to patients with heart failure who may develop marked hyperuricemia due to renal hypoperfusion and reduced

urate excretion. Such patients typically have advanced heart failure with limited life expectancy (unless they undergo transplantation) and are therefore at low risk for chronic urate nephropathy. Excretion of urinary uric acid in excess of 1100 mg (6.5 mmol) daily is associated with a 50 percent risk of uric acid calculi[72,73]. Management of these individuals should begin with dietary purine restriction. Allopurinol should be used if dietary restriction does not reduce uric acid excretion to less than 1000 mg/day (5.9 mmol/day). The dose should be adjusted to reduce uric acid excretion below 800 mg/day (4.8 mmol/day). Patients about to receive radiotherapy or chemotherapy that is likely to result in extensive tumor cytolysis should be treated to prevent acute uric acid nephropathy and other manifestations of tumor lysis syndrome [74]. Preventive therapy in patients at risk includes intravenous hydration and either allopurinol or rasburicase (recombinant urate oxidase).

The Role of Diet Control in Gout and Hyperuricemia

Previous studies have shown that purine rich diet produces only a minor and transient serum urate increase of approximately 1–2 mg/dl. Conversely, an iso caloric purine free diet for 7–10 days achieves a slight (1– 2 mg/dl) reduction in serum urate. Recent 14 Gout and hyperuricemia data confirm the long-held association of gout and hyperuricemia with high intake of meat, seafood, and alcohol (especially beer), but not with "high-purine vegetables" such as beans, peas, and lentils[72-75]. Gouty patients are advised to avoid foods or drinks known to precipitate acute attacks, such as excess meat, seafood, and beers. Crash dieting and fasting should be avoided as they can also precipitate acute attacks. Patients should also receive specific management and instruction for the associated cardiovascular disease risk factors and metabolic syndrome. Dessein et al. demonstrated that increasing protein and unsaturated fat intake while restricting carbohydrates significantly reduces uric acid, serum cholesterol, triglyceride, weight and frequency of gouty attacks[76]. Furthermore, identification of elevated mean serum urate levels in general population during recent decade merits further investigation and lifestyle modification.

In 2006, European league against rheumatism(EULAR) published evidence based recommendations for gout(general, acute management, and chronic management)[81] which are as follows

EULAR evidence based recommendations for gout (general, acute management, and chronic management)[81].

1 Optimal treatment of gout requires both non-pharmacological and pharmacological modalities and should be tailored according to:

(a) specific risk factors (levels of serum urate, previous attacks, radiographic signs)

(b) clinical phase (acute/recurrent gout, intercritical gout, and chronic tophaceous gout)

(c) general risk factors (age, sex, obesity, alcohol consumption, urate raising drugs, drug interactions, and comorbidity)

2 Patient education and appropriate lifestyle advice regarding weight loss if obese, diet, and reduced alcohol (especially beer) are core aspects of management

3 Associated comorbidity and risk factors such as hyperlipidaemia, hypertension, hyperglycaemia, obesity, and smoking should be addressed as an important part of the management of gout

4 Oral colchicine and/or NSAID are first line agents for systemic treatment of acute attacks; in the absence of contraindications, an NSAID is a convenient and well accepted option

5 High doses of colchicines lead to side effects, and low doses (for example, 0.5 mg three times daily) may be sufficient for some patients with acute gout

6 Intra-articular aspiration and injection of long acting steroid is an effective and safe treatment for an acute attack

7 Urate lowering therapy is indicated in patients with recurrent acute attacks, arthropathy, tophi, or radiographic changes of gout.

8 The therapeutic goal of urate lowering therapy is to promote crystal dissolution and prevent crystal formation; this is achieved by maintaining the serum uric acid below the saturation point for monosodium urate (360 mmol/l)

9 Allopurinol is an appropriate long term urate lowering drug; it should be started at a low dose (for example, 100 mg daily) and increased by 100 mg every 2–4 weeks if required; the dose must be adjusted in patients with renal impairment; if allopurinol toxicity occurs, options include other

xanthine oxidase inhibitors, a uricosuric agent, or allopurinol desensitisation (the latter only in cases of mild rash)

10 Uricosuric agents such as probenecid and sulphinpyrazone can be used as an alternative to allopurinol in patients with normal renal function but are relatively contraindicated in patients with urolithiasis; benzbromarone can be used in patients with mild to moderate renal insufficiency on a named patient basis but carries a small risk of hepatotoxicity

11 Prophylaxis against acute attacks during the first months of urate lowering therapy can be achieved by colchicine (0.5–1 mg daily) and/or an NSAID (with gastro-protection if indicated)

12 When gout associates with diuretic therapy, stop the diuretic if possible; for hypertension and hyperlipidaemia consider use of losartan and fenofibrate, respectively (both have modest uricosuric effects)

Prognosis

Without treatment, the acute attack may last from a few days to several weeks. The intervals between acute attacks vary up to years, but the asymptomatic periods often become shorter if the disease progresses. Chronic gouty arthritis occurs after repeated attacks of acute gout, but only after inadequate treatment. The younger

the patient at the onset of disease, the greater the tendency to a progressive course. Destructive arthropathy is rarely seen in patients whose first attack is after the age of 50.

Patients with gout are anecdotally thought to have an increased incidence of hypertension, renal disease (eg, nephrosclerosis, interstitial nephritis, pyelonephritis), diabetes mellitus, hypertriglyceridemia, and atherosclerosis.

Conclusion

Only 15% of all hyperuricemia develop gout. Asymptomatic hyperuricemia usually does not require treatment. Gout is rare in reproductive age of women. S. uric acid may be normal during acute attack. Strict dietary restriction is no longer recommended. Rapid lowering of uric acid is not recommended. Allopurinol should not be prescribed during acute attack. Allopurinol if prescribed should be continued for indefinite period. Colchicine also indicated as an adjunct during initiation of allopurinol therapy. Oral colchicine is effective for acute gout but frequently causes unpleasant side effects. For patients who cannot take NSAIDs or colchicines and who are not candidates for intraarticular corticosteroid injection because of polyarticular disease, oral glucocorticoids are recommended. Prednisolone in doses of 30 to 50 mg/day (or other equivalent glucocorticoid) for one to two days, then taper over seven to ten days. Treatment options in patients who are unable to take oral medications include intraarticular or intravenous glucocorticoids, intramuscular or subcutaneous ACTH, and in locales where it is still available, cautious use of intravenous colchicine. For patients unable to take oral medications, with only one or two actively inflamed joints, and in whom infection has been ruled out, intraarticular injection of glucocorticoids may be used. For patients with polyarticular involvement, existing or easily established intravenous access, and no contraindications to glucocorticoids, systemic administration of a parenteral glucocorticoid also may be used. The dose and frequency depend upon the agent chosen. Patients who are taking antihyperuricemic therapy due to previous episodes of acute gout should be warned that antihyperuricemic therapy alone is not effective. Therapeutic recommendations for acute attacks in these patients are the same as in patients not taking antihyperuricemic therapy. If indicated, patient should be put on hypouricemic drug e.g allopurinol and should be continued for life long. Interleukin-1 inhibition has been reported to be effective in some cases but randomized controlled trials are needed to better assess the usefulness and safety of this approach and it is not recommended at this time. Newer agents like Febuxostat and Pegloticase seems to be promising.

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Pulmonary Hydatid Cysts and Tuberculosis in a Child – A Case Report

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Summary:

A 7 years old male child presented with history of cough, fever, haemoptysis and chest pain for 2 years. On examination he was moderately pale and wasted. Respiratory system examination revealed features of consolidation in both lungs. His provisional diagnosis was pulmonary tuberculosis. TC was 1500/cumm ESR-70mm in first hour, MT was negative and sputum for AFB was also negative. Radiological finding of chest revealed two large well defined dense opacities in both mid and lower zones of both lungs and there was no calcification or air fluid level. CT scan of chest showed large irregular enhancing mass lesion having air fluid level in right lower zone, well defined cystic lesions in both lungs, no calcification was seen. On the basis of x-ray and CT scan report we reviewed our diagnosis as pulmonary hydatid disease. Tablet albendazole was started preoperatively. The cyst was removed surgically and specimen was sent for histopathology. Report showed hydatid cyst and pulmonary tuberculosis. So confirmed diagnosis was Hydatid cyst and pulmonary tuberculosis.He was treated with antitubercular drugs and continuation of tablet Albendazole for 6 months. He was followed up regularly and was doing well.

(J Bangladesh Coll Phys Surg 2011; 29: 102-105)

Introduction:

Hydatid disease (Echinococcus) is one of the most widespread, serious human cestode infection in the world[1,2] .Its Public health problems occur in many areas, including counries of Central America and South America, Western and Southern/Southeastern Europe, the Middle East and North Africa, sub-Saharan countries, Russia and China[2,3]. Annual incidence rates of diagnosed human cases per100.000 inhabitants vary widely, from less than 1 case per100,000 to high levels [2, 3]. It is a zoonosis that is transmitted from domestic and wild members of canine family, who become infected after eating infected viscera and the host of the small adult worm. It is generally caused by the larval stage of dog tapeworm E.granulosus and man can become the intermediate host through contact with infected dogs or by ingesting contaminated food.[5,6] Domestic animals such as sheep, goats, cattle and camels ingest Echinococcus granulosus eggs while grazing. Humans are accidental host and are usually infected with intermediate stage of the parasite by ingesting food or water contaminated with eggs or by direct contact with dogs[3,4] There are many studies of Hydatid disease in adult but only few articles regarding the rate and pattern of involvement in children have been published[5,6]. The liver and lungs are most frequently involved organs. Pulmonary disease appears to be more common in younger individuals but bilateral pulmonary involvement is relatively rare [2,4,7] In a large series of 527 cases of hydatid disease from India, lungs were involved in 29% cases[5,6]. In countries like India having high prevalence of pulmonary tuberculosis, an association of these two diseases,has not been frequently reported in medical litertaure[8, 9]

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Case Report:

A seven years old boy weighing 19 kg, 3rd issue of nonconsanguinous parent hailing from Sirajgonj was admitted in the Paediatric ward of Shahid Suhrawardy Medical college and Hospital with the complaints of fever for 2 years, cough for 1 year, chest pain and difficulty in breathing for 2 months. According to statement of grandmother the child was reasonably well 2 years back then he developed fever which was intially irregular and low grade in nature but gradually became high grade and continuous for 4 months. Fever was relieved by taking anti-pyretic. He also developed cough for 1 year which was productive in nature, colour of the sputum was whitish, thin in consistency and was not foul smelling. There was history of hemoptysis for few occasions in last 2 months. He also developed difficulty in breathing.and chest pain. He had no history of contact with TB patient. He came from poor socioeconomic family, with poor housing and sanitation. Both parents were active and healthy. He was completely immunized according to EPI schedule. His milestone of development was age appropriate

On general examination, he was ill looking, moderately pale, not icteric, not cyanosed. Temperature was 102^0 F and BCG mark was present. Lymphnodes were not enlarged. He was mildly stunted and moderately wasted according to WHO standard. His respiratory rate was 48/min, apex beat normal in position, breath sound vesicular and diminished in lower zone in both lungs and crepitation was present in mid and lower zone of both lung fields. Other systems revealed no abnormalities. Provisionally he was diagnosed as a case of pulmonary tuberculosis.

Investigations showed – Haemoglobin-10.8 gm/dl, ESR 70 mm in first hour, TC 11500/cumm, DC- N 72%, L 18%, M 02%, E 10% and PBF–microcytic hypocromic anemia. Radiological finding of chest showed – two large well defined dense opacities in both para cardiac regions in the lungs, there was no calcification or air and fluid levels fig 1).Ultrasonogram of the chest and hepatobiliary system showed multiple cystic area in chest cavities of both sides; the larger one is about (7.5X5 cm) detected in the left chest cavity, a hypoechoic area with irregular margin measuring (1.56X1.64) is seen in inferior aspect of right lobe of the liver (Fig-2). CT scan of the chest showed large irregular enhancing mass lesion having air fluid level in right lower zone, well defined cystic lesion in both lungs, no calcification was seen(fig -3), Total circulating Eosinophil count was 2400/cumm

Fig.-1: *Patient picture*

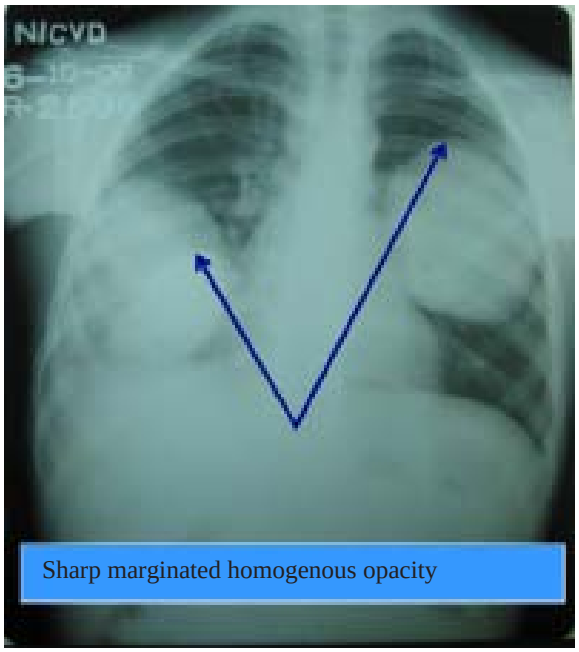

Fig.-2: *X- ray chest: Two large well defined dense opacities in both paracardiac regions. There was no calcification or air and fluild level.*

Beside nutritional support, broad spectrum antibiotics given, because of fever and neutrophilic leucocytosis. Albendazole (15mg/kg) and hematinics were given as his PBF showed microcytic hypocromic anemia. His fever subsided but chest pain and difficult breathing persisted. He was refered to chest specialist and surgery was done in two settings (fig 4 first operation was excision of hydatid cyst through extended right posterior lateral thoracotomy followed by left upper lobectomy was done after one month. On histopathology– specimen of the lung tissue (fig-5), presents large area of caseating tubercles and section made from the cyst wall of laminated layer of hyalinized material.Diagnosis-

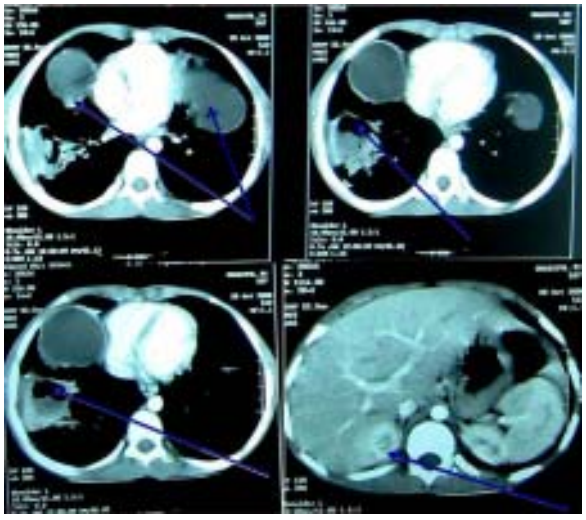

Fig.- 3: *CT Scan of Chest: showed large irregular enhancing mass lesion having air fluid level in right lower zone, well defined cystic lesions in both lungs,no calcification was seen.*

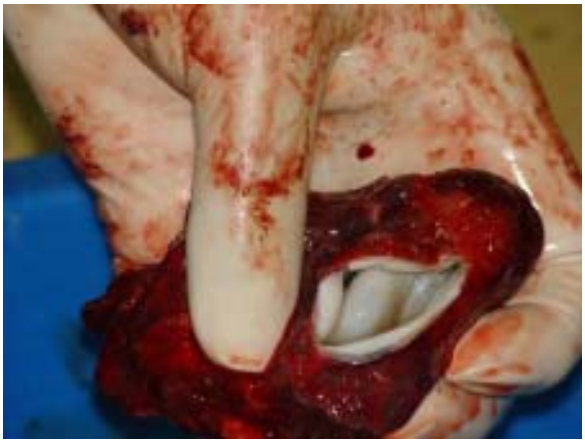

Fig.- 4: *Section of Lung showing hydatid cyst after operation*

ganulomatous inflammation, tuberculosis and hydatid cyst. He was treated with antitubercular drugs for 6 months with the doses of tab INH-10 mgkg, Rifampicin 10 mg/kg, Pyrazinamide 35 mg/kg daily(2HRZ+10 HR) along with albendazole 15 mg/kg daily for 6 months. He was clinically improved like weight gain, fever subsided and Hb increased after one month.

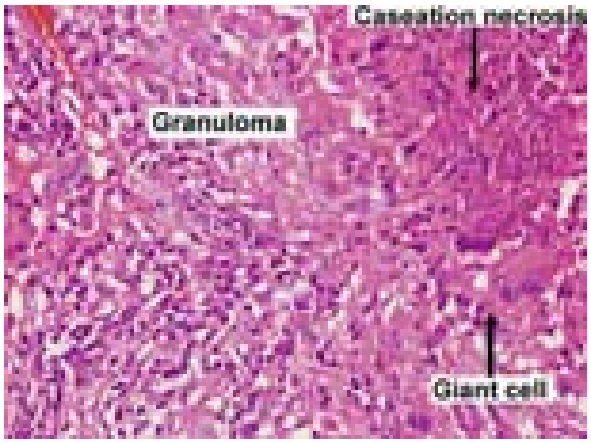

Fig.-5: *Histopthology: of the lung specimen: granulomatous inflammation, area of caseating tubercles and section made from the cyst wall of laminated layer of hyalinized material. Diagnosis: Tuberculosis and hydatid cyst.*

Discussion

Hydatid disease is an extensive epidemiological problem in developing countries like Iran, Egypt, and China[3,4,.5]. The hydayid cyst in the liver is most common but can also be found in the lungs, kidney, spleen, nervous system and bone[4,5]. The frequency of hydatid disease was higher in male (58%) than female (42%)[4,5]. Signs and symptoms depend on cysts size and location which was shown in different studies as cough (92%), fever(89%), chest pain(42%), breathlessnass(48%) and haemoptysis[4,5] Hydatid disease is a rare cause of hemoptysis and rare presentation of paediatric patient.The incidence of haemoptysis in children may be as high as 38% [5,6,7], which is also present in our case. The most frequent affected organ in children is lung (77%) whereas in adult liver is commonly affected organ[2,4, 5]. Similar finding was present in other studies The parasite is often acquired in childhood, but liver cysts require many years to become large enough to be detected or cause symptoms. Single organ involvement is present in *85-90%* and more than 70% harbours a

solitary cyst [6.7,.8]. In children, the lungs appear to be the most common site as in the present case whereas 70%of the adults have disease in the right lobe of the liver[5, 6.] It seems that in children scolices has more ability to pass from liver barriers than adults. It may be due to low density of the liver in children[9,10]. But combined lung and liver involvement is more frequent in children than adult (18% vs 4%)[8,9]. Present case pulmonary hydatid disease affect both the lung like other studies the right lung in 60% cases, *30%* multiple pulmonary cyst and about 4% bilateral cyst[3,8]. Although many patients are asymptomatic, some may occasionally expectorate the contents of cyst or develop sign and symptoms related to compression of surrounding structures. Most intact lung cysts are discovered incidentally on chest radiographs[9,10]. Studies showed well defined opacities in both paracardiac regions and there were no calcification or air fluid levels. CT scan of mediastinum revealed multiple large well defined intrapulmonary cystic masses in both lower lobes[11,12]. Similar finding was present in our case. Detection of antibody directed against specific Echinococcal antigen is useful in confirming a diagnosis but the false negative rate may be as high as *50%* in cystic hydatid disease of the lungs[4, 6]. ELISA for echinococcus was not done in our case because this is not specific and patient was poor. Few case reports were published hydatid disease with tuberculosis. Present case is also an example of this rare association. Some studies described hydatid disease as primarily a surgical disease[13,14] Conservative surgical treatment like cystotomy, capitonage and other lung preserving surgical approaches like segmentectomy, lobectomy, wedge resection and enucleution were carried out in 65 of 72 children (90%)[10,11,12]. However medical treatment may also be successful by benzimidazole drugs. Mebendazole and albendazole are the only antihelmenthic effective against cystic echinococcosis and probably treated before surgery[3,8,10], which was followed in our case. More recently Keramidas et al treated 36 children with pulmonary echinococcus cysticus with oral mebendazole or albendazole, During this treatment 11 patients developed complications requiring surgery[10,11]. Surgical complications are less frequent in children and their outcome is better[9,12]. Recently few literatures highlight on percutaneous drainage of hydatid cyst popularly known as PAIR(Puncture Aspiration, Instilation of scolicidal agent and Reaspiration) has gained acceptance.This procedure is minimally invasive, cost effective, involves reduced hospital stay and less morbidity and mortality than surgery[4,5].

Conclusion:

Hydatid disease of lungs is not so uncommon in Paediatric practice, but bilateral involvement is rare. Pulmonary tuberculosis is a co-existence with pulmonary hydatid cyst. Surgery is the mainstay of treatment for most patients with pulmonary hydatid disease in children but medical therapy for inoperative cyst with albendazole, mebendazole also suggestive.

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Scar Endometriosis, An Uncommon Entity

L SAHA

Summary:

Scar endometriosis is an uncommon condition where there is presence of endometrial tissue in the abnormal sites. Generally this is a benign lesion although malignant transformation is possible. Commonest site is at the site of laparoscopy done for non gynaecologic indications[2]. Extrapelvic sites are fairly uncommon, among which abdominal wall may also be affected[3]. This case of scar endometriosis was diagnosed after 2 years of caesarean section. Patient presented with pain and swelling below the umbilicus. Pain had a distinct relationship with the menstrual cycle. Swelling appeared later and was slowly increasing in size. There was a high index of suspicion based on history and clinical findings. Wide excision was done and histopathogy reported the lesion as scar endometriosis.

(J Bangladesh Coll Phys Surg 2011; 29: 99-101)

Introduction:

Presence of functioning endometrium (glands and stroma) in sites other than uterine mucosa is called endometriosis. It is not a malignant condition although malignant transformation is possible[1].

The most common location is within the pelvis and has been reported to occur in as many as 44% of women undergoing laparoscopy for non-gynaecological symptoms[2].

However, extra pelvic endometriosis is a fairly uncommon disorder and difficult to diagnose. The various sites for extra pelvic endometriosis are bladder, kidney, bowel, omentum, lymph nodes, lungs, pleura, extremities, umbilicus, hernial sacs, and abdominal wall[3].

Abdominal wall endometriomas often develop in previous surgical scars but there is a case report of a spontaneous occurrence also[4]. Majority of the scar endometriosis have been reported after obstetrical or gynaecological procedures such as cesarean delivery, hysterotomy, hysterectomy, episiotomy, and tubal ligations[5,6,7] but few cases are also reported following appendicectomy, in the laparoscopic trocar tract, amniocentesis needle tract. Scar endometriosis patients are often referred to the general surgeons because the clinical presentation suggests a surgical cause. In a study by Blanco et a[l8] the diagnosis was initially confused with inguinal hernia, incisional hernia and abdominal wall tumor.

Here is the report of a case of scar endometriosis following cesarean section over a period of 2 years. This paper may play some role to increase the awareness among the clinicians regarding this rare condition, which is often misdiagnosed and hence delays the patients cure.

Case History:

A 28 year old lady, working as an assistant nurse, married for 7 years, having one child of 3 years and 6 months old referred to a gynaecologist with the complaints of localized painful swelling below umbilicus for 2 years. At first she had pain below umbilicus. Pain increased during the period of menstruation which became intolerable on the first day, decreased from the 2nd day onwards. But thereafter she had low grade pain in the area throughout the cycle. After eight months she noticed a swelling below umbilicus. Swelling was gradually increasing in size. Her obstetric history revealed delivery of a male baby by cesarean section due to primi gravida with breech presentation. She also complained of low grade dysmenorrhoea. She was found to be with good general health, not pale, normo-tensive. A swelling of 4 cm diameter, circumscribed, tender, not red or warm, was found 2 cm below the umbilicus and 5 cm above the cesarean scar. Swelling was non reducible, not fixed with the overlying skin but slightly fixed with underlying structure. On per vaginal examination uterus was found bulky, antiverted and mobile. Based on characteristic history and examination findings, there was a high index of suspicion of endometriosis considering other possibilities like hematoma, granuloma,. Patient was

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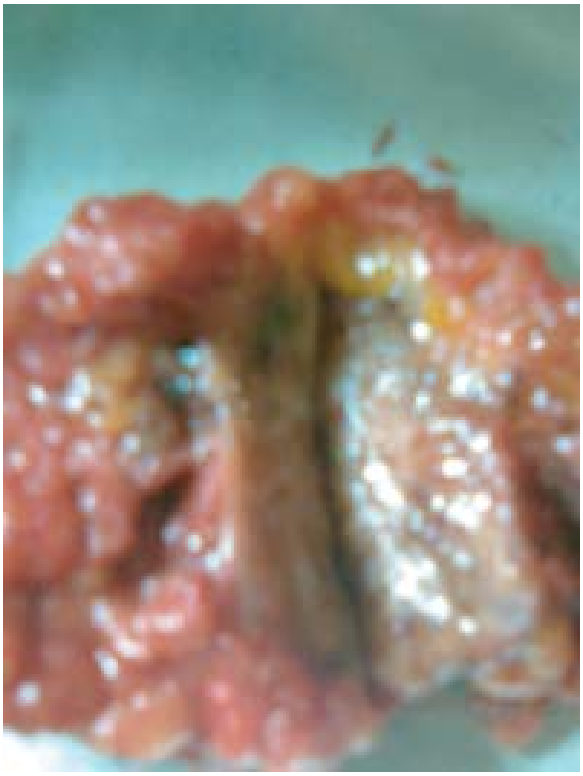

Fig.-1: *Gross appearance of the lesion (after surgical excision)*

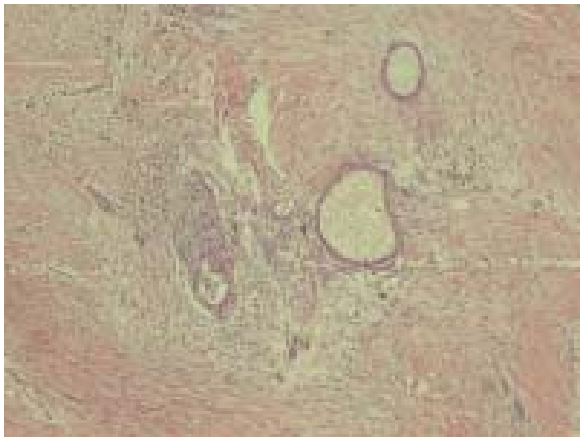

Fig:-2: *Microscopic appearance of the lesion showing fibromuscular tissue containing endometrial gland and stroma*

advised excision biopsy but initially she refused and preferred medical treatment if any. A trial of medical treatment was given with progesterone and analgesic for one month, as the patient had associated dysmenorrhoea. However, medical trial did not bring any benefit for the patient except slight improvement of dysmenorrhoea..

Finally wide excision of the swelling was done and histopathology reported the case as scar endometriosis. The patient is kept under regular follow-up.

Discussion:

Endometrioma is a well-circumscribed mass of endometriosis. Abdominal wall endometrioma presents as a painful swelling resembling surgical lesions such as hernias, hematomas, granulomas, abscess and tumors. So that is why these cases first report to general surgeons. This case was reported to the gynaecologist as the patient had exaggerated pain during menstruation.

Scar endometriosis most commonly occurs after operation on the uterus and tubes. Incidence of scar endometriosis following hysterotomy is 1.08-2% where as after cesarean section the incidence is 0.03-0.4%.[7] The reason for higher incidence after hysterotomy has been given as the early decidua has more pleuripotential capabilities and can result in cellular replication producing endometriomas.

Time interval between operation and presentation has varied from 3 months to 10 years in different series. The etiology of abdominal wall endometrioma is thought to be a result of transportation of endometrial tissue during surgical procedures and subsequently stimulated by estrogen to produce endometriomas. The simultaneous occurrence of pelvic endometriosis with scar endometriosis is infrequent[7]. This patient also did not have associated pelvic endometriosis. Preoperative diagnosis is difficult to make and sometimes the diagnosis is made after excision only. This particular case was diagnosed after excision histopathology.

Various diagnostic methods have been described in the literature. Till recently the use of ultrasonography (USG) have hardly been reported in detail and anecdotal reports have described it as nonspecific, can give a varied picture of hypo echoic mass with scattered internal echoes. Recently a large series of 12 patients where USG and color Doppler substantially contributed to the correct preoperative diagnosis[9] and it is suggested that sonographic and color doppler when combined with clinical data may substantially contribute to the preoperative diagnosis. FNAC has been reported to be accurate in diagnosis but in a recent report by Dwivedi et al. FNAC was not diagnostic in any of the four patients who underwent this procedure[10,11]. Anecdotal studies have mentioned the use of computed tomography (CT)

and magnetic resonance imaging (MRI) in making a diagnosis. CT usually shows a solid, well-circumscribed mass. MRI can be more helpful when the lesion is small because of its high spatial resolution, furthermore it perform better than CT scan in detecting the planes between muscles and abdominal subcutaneous tissue.[12] However, imaging technique was not used as a diagnostic tool in this case.

Treatment of choice is wide excision of the lesion and may sometimes require mesh placement which was not done in this patient. Medical treatment with the use of progestogens, oral contraceptive pills, and danazol is not effective and gives only partial relief in symptoms. Goel et al [13] reported failure of medical treatment in their two cases as in this case. Recently there has been report of use of gonadotrophin agonist but only with the prompt improvement in symptoms with no change in the lesion size[14].These patients need to be followed up because of the chances of recurrence, which require re-excision. In cases of continual recurrence possibility of malignancy should be kept in mind. To prevent the occurrence of scar endometriosis it has been suggested that at the end of surgery especially on uterus and tubes, the abdominal wall wound should be cleaned thoroughly and irrigated vigorously with high jet solution before closure[15].

Conclusion

One should have a high index of suspicion of scar endometriosis when a woman presents with a painful swelling in the abdominal scar especially with a history of previous gynaecological or obstetrical surgery. This condition can be confused with other surgical conditions. Efforts should be made to make a preoperative diagnosis with the help of imaging techniques and FNAC. Medical treatment is not helpful. Wide excision is the treatment of choice. Patient should be followed-up for recurrence.

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A Case of Diffuse Cutaneous Leishmaniasis in a HIV Positive Patient

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Summary:

A cultivator of 30 years of age presented with fever, cough, diarrhoea, anorexia and weight loss for 6 months and papulonodular skin lesions for one month. Skin lesions appeared on the face, first over the left cheek and gradually involved whole of his face, extremities and external genitalia sparing the trunk. Skin biopsy from the nodule showed collection of histiocytes, lymphocytes & plasma cells with plenty of LD bodies inside the histiocytes. Screening test for HIV was positive and it was confirmed with western blot. Probably this is the first case Leishmaniasis/ HIV co infection reported from Bangladesh.

(J Bangladesh Coll Phys Surg 2011; 29: 106-108)

Introduction:

Leishmania/human immunodeficiency virus (HIV) coinfection is emerging as an increasingly frequent and extremely serious new disease. Although many reports have described the association of visceral leishmaniasis and HIV, cutaneous leishmaniasis is associated with HIV is very uncommon[1,2]. Diffuse cutaneous leishmaniasis may be a common clinical manifestation when leishmaniasis associated with HIV infection[3]. Leishmaniasis covers three well-individualized clinical variants, each due to individual species found in different geographic areas, viz, visceral, cutaneous and mucocutaneous. It is transmitted by female Phlebotomus sandflies. Human immunodeficiency virus (HIV) infection is increasing worldwide and several reports indicate a rising trend of VL / HIV co-infection, modifying the traditional anthroponotic pattern of VL transmission[4]. Observed clinical forms of cutaneous leishmaniasis are: papulo-nodular, ulcerative, infiltrative , lepromatous and diffuse, psoriasis-like, cheloid, histioid or kaposi-like. Some patients presented with more than one clinical form[5]. L. major is responsible for typical cutaneous leishmaniasis but particular clinical forms have been described in immunodeficient patients, especially with diffuse cutaneous involvement. Here we reported a patient with diffuse cutaneous leishmaniasis with AIDS from Bangladesh.

Case Report:

A cultivator of 30 years of age, from Sylhet, Bangladesh was admitted in department of medicine, Sylhet MAG Osmani Medical College Hospital on 26th April'09 with the complaints of fever, cough, diarrhoea, anorexia and weight loss for 6 months and papulonodular skin lesions for one month.

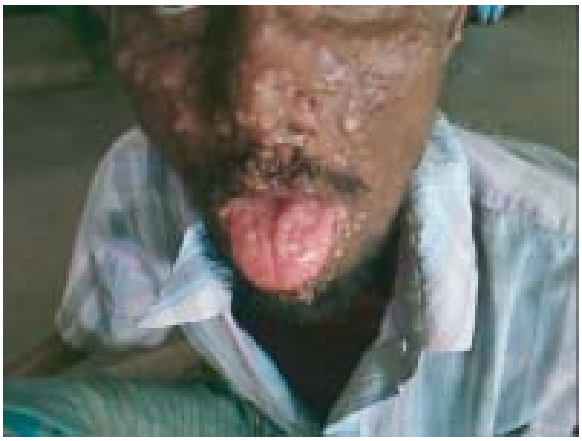

Fig.-1: *Papulonodular lesions concentrated in central part of face*

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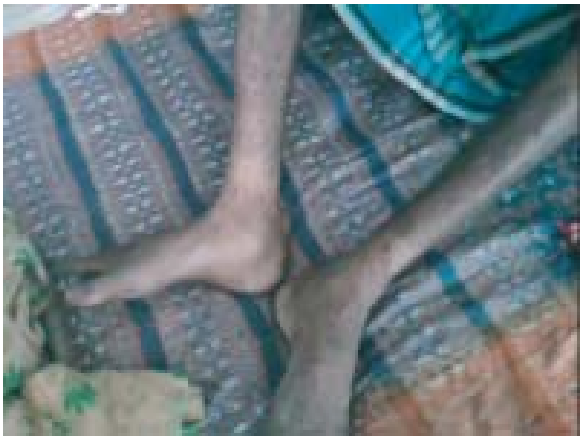

Fig.-2: *Skin lesions also involving extremities*

He stated that his fever was low grade, continuous and associated with cough with mucopurulent sputum and occasional haemoptysis. He also complained of frequent loose stools. He had significant weight loss over last six months. For the last one month he had noticed multiple papulo-nodular skin lesions appearing on the face, first over the left cheek and gradually involved whole of his face, extremities and external genitalia sparing the trunk.

Examination revealed he was grossly emaciated, body temperature of 101°F, pulse rate 88/min, respiratory rate 20 breaths/min, and a blood pressure of 90/50 mm of Hg. He was severely anaemic with mild oedema. Multiple light brown papulo-nodular skin lesions involving the face and both upper and lower extremities were noted. The nodules were more concentrated in central part of face and there was involvement of ears also (figure-1). Few of the nodules near the nose were crusted. The extremities were mainly involved by papules than nodules (figure-2). Trunk was relatively free .There were multiple hypo & hyper pigmented patches over the abdomen. Sensations were intact over these areas. Examination of oral cavity revealed multiple erosions in the inner cheek and tongue and a whitish coating extending from dorsum of tongue up to oropharynx. There was no organomegaly. Complete blood count (CBC) showed a raised ESR 60 mm/1st hour; a hemoglobin of 6.5 g/dl, with white cell count 5000/cmm, differential count N-50% & L-45% and a platelet count160,000/cmm. The peripheral blood picture, RBS, renal function & tuberculin test were unremarkable. The chest X- ray, ECG, USG of whole abdomen did not show any abnormalities. Sputum for AFB & slit skin smear for leprosy were also negative.

Skin biopsy from the nodule showed collection of histiocytes, lymphocytes & plasma cells with plenty of LD bodies inside the histiocytes. Screening test for HIV is positive and it was confirmed with western blot.

Discussion:

Leishmaniasis/human immunodeficiency virus (HIV) coinfection is emerging as an increasingly frequent and extremely serious new disease although it is very uncommon[1,3]. Human immunodeficiency virus (HIV) infection is increasing worldwide and several reports indicate a rising trend of VL / HIV co-infection, modifying the traditional anthroponotic pattern of VL transmission.[4] Here we reported a patient with diffuse cutaneous leishmaniasis with HIV from Sylhet, Bangladesh. Barro-Traoré F et al reported A 38-years old HIV-positive man presenting with generalized, copper-coloured, painless, infiltrated, itching, papulonodular lesions present over the previous 10 months.[6] The case we reported here also presented with multiple light brown papulo-nodular skin lesions involving the face and both upper and lower extremities. Some patients may present with more than one clinical form.[6] This patient also presented with more than one lesion.

Although India is one of the countries having the largest burden of Leishmaniasis; nevertheless, there are very few HIV & leishmania co-infection cases reported till date.[4] Same comment is true for Bangladesh. Probably this is the first case of leishmaniasis & HIV co infection reported from Bangladesh. Most of the HIV infected persons in Bangladesh were ex- workers in other countries specially middle east. This is the first case that never travelled outside the country.

Conclusion:

HIV is spreading alarmingly in Bangladesh especially in Sylhet. Most of them are ex workers in Middle eastern countries. This patient never travelled outside. This means he acquired infection inside country and indicates HIV is spreading in between our population. HIV & leishmaniasis co-infection is a very rare occurrence and it modifies the presentation of leishmaniasis. Physicians should remain cautious and vigilant regarding this co-infection where HIV infection is common.

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Metastatic Jaw Swelling as the Manifestation of Leiomyosarcoma of Uterus- A Case Report

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Summary:

Metastatic tumor in oral region is uncommon and may occur in the oral soft tissues or in the Jaw bone. Because of their rarity, metastasis in oral cavity are challenging to diagnose and treat. Oral metastasis is associated with poor prognosis. This case report is of a 45 year old female with a small pedunculated swelling on the left side of the hard palate in the molar region for 30 days. Incisional biopsy revealed metastatic leiomyosarcoma with possible primaries in the uterus. Metastasis in the right lung and liver was also diagnosed. Palliative chemotherapy was started but the patient died after two weeks of diagnosis after receiving the first cycle of chemotherapy.

(J Bangladesh Coll Phys Surg 2011; 29: 96-98)

Introduction:

Metastasis to the oral cavity is uncommon and constitutes about 1 % of all oral malignant tumor[1,2,3,4]. About 1 % of the malignant tumors of the body metastasize to the oral cavity[5]. In the absence of any other metastasis isolated tumor seedling of oral tissues are extremely rare and constitutes 0.1%[6,7]. This metastatic jaw tumor usually comes form lung, breast, genital organ, prostate, thyroid, kidney, bone and adrenals. Intra abdominal leiomyosarcoma commonly metastasize into the liver (65%), peritoneum (21%), lymph nodes (6%), bone (6%) and lung (2%)[8]. This report describes a case of Leiomyosarcoma of uterus metastasized, to the soft tissue of upper jaw which is very rare.

Case report:

Mrs. Rahima khatun, a 45 years old woman from Pabna reported to the Oral and Maxillofacial Surgery department of Dhaka Dental College and Hospital with the complaints of a swelling on the right side of the posterior palate for 1 month with masticatory problem. The swelling was growing rapidly and was associated with pain. She had history of bleeding from the mass during mastication. Past medical history revealed the history of hysterectomy 5 months back in a private clinic of Pabna on the imaging based diagnosis of Fibroid uterus but no excisional biopsy was done. She had severe low back pain, pain on the right leg, chest heaviness but no cough or hemoptysis. She had the history of betelnut chewing, 4-5 leaves per day, for the last 10 years.

Physical examination showed an obese and anxious looking middle aged lady with moderate anemia. Her recorded blood pressure and pulse rate was 160/110 mm of Hg and 80 beats/ minute respectively. No sign of ascites or clubbing was seen but her right leg was edematous. No extra oral asymmetry was seen due to the swelling. Intra orally a soft mass was seen on the right side of the palate in the molar area which was pedunculated, pinkish in color, surface was smooth but ulcerated, non tender, measuring about 4cm X 2.5 cm. Regional lymph node was not palpable. On examination, the chest percussion note was dull over the lower right lung and the liver was palpable which was about one

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finger breadth from the right costal margin. Orthopantomogram showed soft tissue shadow with no underlying bony erosion. ESR was 25 mm in first hour and liver function test showed normal enzyme level with INR of 1.25. Incisional biopsy reported spindle cells arranged in interlacing fascicles compatible with leiomyosarcoma suggesting metastasis with possible primary in uterus. Ultrasonography of whole abdomen revealed enlarged grossly heterogenous liver with multiple space occupying lesion (secondaries) with absence of uterus. Chest radiograph showed dense oval opacities in right para cardiac region and coin shadow ws seen in the right lower region of the lung with pleural effusion. X-ray lumbosacral spine showed lumbo sacral spondylitis. CT guided FNAC or liver biopsy was not performed due to poor general condition of the patient. Patient died after first cycle of chemotherapy that is two weeks after diagnosis.

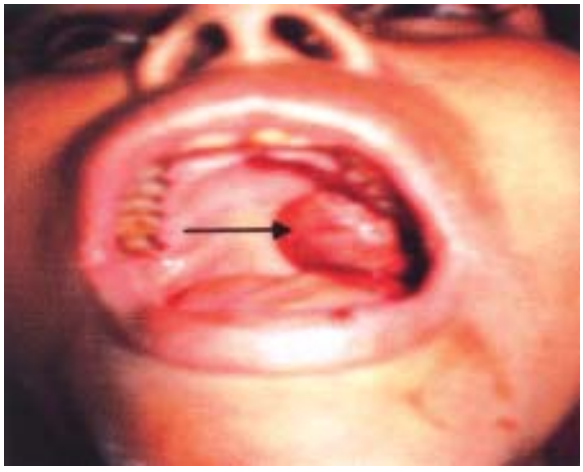

Fig-1: *Intra oral photograph the swelling indicated with arrow*

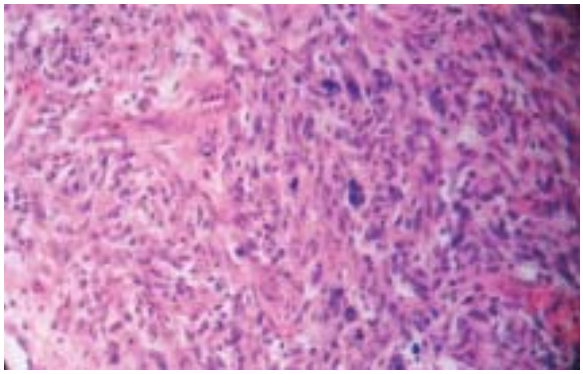

Fig.-2: *Low power microscopic view of oral lesion*

Discussion:

Tumors that metastasize to the oral soft tissues are very rare. The majority of the oral metastasis occur in the jaw bone (90%) and only 5% in the oral soft tissues[5]. Of the oral soft tissues 5% occur in the tongue, 4% in the gingiva and cheek and 1 % elsewhere[5]. Oral metastasis arises as a result of secondary spread from other metastatic lesions especially the lungs. In about 30% of oral metastasis, lung is the first site of metastatic disease. In such cases, the tumor cells bypass filtration by the lungs. Any increase in intrathoracic pressure directs blood flow into the valveless vertebral venous plexus from the azygous and caval venous system. This accounts for the increased occurrence of metastases from lung in the head neck area and axial skeleton[9]. Pathogenesis of metastasis to oral soft tissue is due to the rich capillary network of chronically inflammed mucosa, especially of the gingiva that can trap malignant cells. These capillaries contain fragmented basement membrane through which tumor cell can easily penetrate[10]. For the most metastatic jaw tumor the primary tumor is in the breast (24%), genital organs (17%), lung (1296), kidney and bone 10% each[10]. The report describes a case of metastatic leiomyosarcoma of oral cavity with possible primary in the uterus which is very rare. Metastasis from leiomyosarcoma to the head-and-neck, and, to the palate in particular, is unusual[11]. We had to depend on the histopathology of the presented oral lesion and other history and metastatic feature for the diagnosis of primary site. CT guided FNAC and liver biopsy may have helped for diagnosis. But in this reported case it was not done due to poor general condition of the patient. Oral metastasis is considered a late complication and is commonly associated with multiple organ metastases. Oral metastasis can grow rapidly causing pain, difficulty in chewing, dysphagia, disfigurement and intermittent bleeding, leading to poor quality of life[1,12-16]. Oral metastasis is an ominous prognostic sign and is associated with poor prognosis with a median survival of 4 months[12-16]. So treatment is aimed at palliation of symptoms. In this case the patient was given chemotherapy and the lesion was reduced in size after the first cycle of chemotherapy but the patient died after

2 weeks of diagnosis. From the reported case it is recommended that all the oral lesions should be correlated with the thorough clinical examination and investigation of the general body condition especially when suspected for metastatic jaw swelling and any resected specimen must be sent for histopathology which may limit the subsequent disease sequele.

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Surgery at Cranio-vertebral (CV) Junction: Our Experience of 32 Cases

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Summary

Cranio vertebral (CV) junction is one of the critical sites for surgery. It's anatomy, physiological aspects and pathological involvement varies in a wide range of margins. Common problems are developmental anomalies, traumatic involvement, inflammatory, infective and neoplastic lesion. Management of these problems varies a lot from each other. Aim of the article is to overview the pathologies in this area and to study presentations, investigations, surgical procedures and results of these pathologies.

We prospectively analyzed 32 cases of Cranio-vertebral (CV) region surgery in the Department of Neurosurgery Dhaka Medical College Hospital and Mitford Hospital, Dhaka, from 2000 to 2008.

In our series, male and female ratio was 7.2:1. Pathologies were atlanto- axial dislocation (AAD), Chiari malformation type –I, schwannoma, meningioma, hydatid cyst and tuberculosis. Common clinical findings were- neck pain, quadriparesis, quadriplegia, hand atrophy, autonomic dysfunction and hypertension. Various types of surgical procedures were done in this series according to the pathology. Death was in 01 case, neurological deterioration seen in one case, 2 cases were neurologically stable and 28 cases (87.5%) improved neurologically where one was non useful improvement(Frankel grade-C).

Complete pre operative radiological study is a very important adjunct for a successful surgical result. Proper evaluation of patients with selection of appropriate surgical procedures along with safe surgical techniques are the necessary things for successful surgery in this area.

(J Bangladesh Coll Phys Surg 2011; 29: 78-84)

Introduction

Cranio vertebral (CV) junction is anatomically and functionally very complex zone. Anatomical variations of neurovascular and skeletal components in this site are quite common. Here, many types of developmental anomalies, traumatic fracture& dislocation, inflammatory, infective and neoplastic lesions are common lesions. Management of these problems varies a lot from each one another. Therefore without adequate background knowledge and surgical skill, surgery at this site is dangerous. Proper anatomical, physiological, pathological, radiological knowledge and surgical skill is preliminary things before surgical visit in this critical site. Aim of the article is to overview the surgical pathologies in this area along with their clinical presentations, investigations, surgical procedures & results as well as to share our experiences to our colleagues.

Methods:

We prospectively analyzed 32 cases of Cranio-vertebral (CV) region surgery in the Department of Neurosurgery Dhaka Medical College Hospital and Mitford Hospital, Dhaka, from January'2000 to July'2008.The complete history was taken regarding neck pain, weakness, sensory dysfunction and autonomic dysfunction. Then neurological findings of preoperative and postoperative last follow up were recorded for analysis. Neurological status was assessed by Frankel grading system. Improved was defined as improved neurological status at the last follow-up compared with the preoperative neurological status. Average duration of follow up is 11.4 months (range - 4.5 months to 96 months)

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Observations and Results:

Total No of cases-32. Sex- male: 28(87.5%) and female: 04(12.5%). Average age-37.27 years, range-20 to 65 years**.**

Pathologies in this area were-

Atlanto- axial dislocation (AAD)- 19 cases(59.3%)

*traumatic-05 cases (15.62%)

*developmental-07 cases (21.87%)

*spontaneous-06 cases (18.75%)

*Psoriatic arthritis-01case (3.12%)

Chiari malformation type –I: 04 cases (12.48%; Table-2,case no-1,2,3 & 4)

Schwannoma- 04 cases (12.5%; Table-2, case no-7,8,9 & 10)

Meningioma- 02 cases (6.25%; Table-2, case no-11 &12)

Hydatid cyst- 01 case (3.37%; Table-2, case no-13)

Tuberculosis- 02 cases (6.25%; Table-2, case no-5 & 6)

Clinical features were-
Neck pain- 24 cases (75%),
Quadriparesis-27cases (84.3%)
Quadriplegia-04 cases (12.5%)
Hand atrophy-04 cases (12.5%)
Autonomic dysfunction-05 cases (15.62%)
Hypertension-01 case (3.12%).
Features of skeletal dysplasia-04 cases (12.5%)
Respiratory difficulty-02 cases (6.25%)

Investigations done in these patients-
Plain x-ray anterior-posterior & neutral lateral view were done in all cases (100%); Flexion & extension lateral view and open mouth views in 26 cases (81.25%). MRI done in all 32 cases (100%). CT scan done in 07 cases (21.87%).3-D CT scan was done in 02 cases (6.25%).

Operative procedures done in these cases were-
Reduction and bilateral C1C2 transarticular screw fixation with fusion by bone grafting-09 cases (28.12%;Table-1,case no-1,2,3,4,12,13,16,17&19)

Reduction and unilateral C1C2 transarticular screw fixation with fusion by bone grafting-01 case (3.12%; Table-2, case no.-6).

Sublaminar wiring and occipitocervical fusion-2 cases (6.25%; Table-1,case no-10 &11).

Stabilization by U loop and wire followed by occipito-cervical fusion-02 cases (6.25%;

Table-1, case no-14&18)

Posterior fossa decompression with removal of C1 posterior arch and duraplasty-04 cases (12.5%; Table-2, case no-1,2,3 & 4)

Removal of tumour/cyst through posterior/posterior-lateral approach-07 cases (21.86%; Table-2, case no-7,8,9,10,11 & 13).

Posterior stabilization by loop/wiring/transarticular screw + occipito-cervical/atlanto-axial fusion and transoral decompression-03 cases[9.37%; Table-1,case no-1,5&15(Figure-1&2)]

Peroperative reduction and C1C2 lateral mass screw& plate stabilization and fusion-03 cases (9.37%; Table-1, case no-7,8 & 9).

Decompression by removing posterior margin of foramen of magnum with preparation of C1 from occiput followed by U loop-wire stabilization and bony fusion between prepared C1 and C2 along with transoral decompression –one case(3.12%;Table-1,case no-6).

Transoral drainage of abscess-01 case (3.12%; Table-2, case no-5).

Complications -

Death-01 case (3.12%; Table-1, case no-10)

Transient CSF fistula-01 case (3.12%; Table-2, case no-9)

Palatal incompetence (Following transoral decompression, improved after 06 months)-01case (3.12%; Table1, case no-15)

Persistent neck pain-03 cases (9.37%; Table-1, case no-1, 5 &17)

Persistent spasticity-02 cases (6.25%; Table-1, case no-1 &5)

Neurological deterioration-01 case (3.12%; Table-2, case no-9)

Break of one Transarticular C1C2 screw followed by neurological deterioration-01case (3.12%; Table-1, case no-1; Figure-3 F). Here neurological stability was made by transoral decompression)

Results of CV junction surgery-
Complete or near complete neurological recovery (Frankel grade-E +/- persistent hand atrophy)-14 cases (43.75%)

Improved but not normal neurostatus-
Frankel grade-C (not useful)-01 case (3.12%; Table-2, case no-4)

Frankel grade-D (useful)-13 cases (40.62%)

Stable (peroperative and postoperative Frankel grade-E)-02 cases (6.5%; Table-1, case no-13 and Table-2, case no-5)

Deterioration-01case (3.12%; Table-1, case no-9, preoperative Frankel grade-D, postoperative grade-C)

Death-01 case (3.12%; Table-1, case no-10).

In table -1 and table-2 clinical profile, surgical procedures and outcome of CV junction surgery cases are shown.

Table-1

Age, sex and pathological profile as well as pre and post surgical neurological status in AAD

No & sex	Age (yrs)	Diagnosis	Preop Frankel Grade	Final outcome & Frankel Grade
1.M	45	Traumatic AAD	(A)	Improved but can just walk with support; persistent spasticity& pain (D-)
2.M	40	AAD	(D)	Full recovery(E)
3.M	38	AAD	(D)	Full recovery(E)
4.M	26	Tr.AAD &Odontoid #	(C)	Improved(D)
5.M	65	BI with cervical C3-4 disc prolapse	(C)	Improved but persistant spasticity & pain (D)
6.M	36	Fixed AAD& BI with occipitalization of C1	(C)	Improved.(D)
7.M	35	Fixed AAD	(C)	Improved(D)
8.M	43	Platybasia with BI with syrinx	(C)	Improved(Grade-D)
9.M	28	Sk. dysplasia with AAD	(D)	Immediate post operatively patient deteriorated then improved but not upto preoperative state.(C)
10M	56	AAD	(A)	Respiration stabilized,but no neurological recovery, later expired.
11M	45	AAD	(C)	Improved but restricted neck movements(D)
12.M	28	Tr. dens # with AAD	(C)	Complete recovery(E)
13.M	39	AAD	(E)	Neck pain gone(E)
14.M	21	Sk. dysplasia with AAD	(D)	Improved except incontinence of urine(E)
15.M	25	SkDysplasia with HTN with AAD Fixed	(D)	Improved completely but palatal /nasal regurgitation, improved later(E)
16.M	44	AAD	(D)	Improved(E)
17.M	34	AAD Tr.	(C)	Improved(D)
18.F	41	AAD with Psoriatic arthritis	(D)	Improved with persisted neck pain(E)
19..M	55	Tr AAD	.(C)	Improved(D)

[No-Number, yrs-years, C/F-clinical features, M-male, F-female, Tr-traumatic, Preop-Preoperative ,AAD-atlantoaxial dislocation, BI-basillar invagination, Sk-skeletal, #-fracture, HTN-hypertension, Frankel grading-A,B,C,D&F].

Table-II

Age, sex and pathological profile as well as pre and post surgical neurological status in other pathologies of CV junction (excluding AAD)

N& sex	Age (yrs)		Preop Frankel grade	Final outcome & Frankel grade
1.M	42	Chiari -1	(C)	Improved except hand atrophy(D)
2.F	36	Chiari 1 with syrinx	(D)	Improved(E)
3.M	20	Chiari-1	(C)	Improved except hand atrophy(D)
4.M	28	Chiari-1	(B+)	Improved (C)
5.M	25	AAJ TB with retropharyngeal abscess	(E)	Improved(E)
6.M	38	Tubercular AAD	(D)	Complete recovery(E)
7.M	35	FM and high cervical Schwannoma	(C)	Complete recovery(E)
8.M	27	FM Schwannoma	(C)	Improved(Postop. CSF fistula)(D)
9.M	39	C1C2 schwannoma	(D)	Complete recovery(E)
10.F	44	FM and high cervical schwannoma	(D)	Complete recovery(E)
1M	30	FM and high cervical meningioma	(C)	Improved(D)
1M	50	C1C2 meningioma	(D)	Complete recovery(E)
13.F	21	C1C2 hydatid cyst	(B)	Recurrence of cyst that regressed with albandazole(E)

[No-Number, yrs-years, C/F-clinical features, M-male, F-female, Preop-Preoperative ,AAD-atlantoaxial dislocation,AAJ-atlantoaxial joint, FM-foramen magnum,TB-tuberculosis, Frankel grading-A,B,C,D&F].

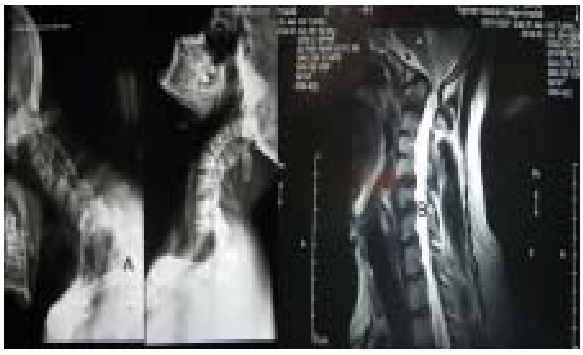

Fig.-1: *A-x-ray cervical spine flexion & extension view, B-MRI saggital section T1W image showing cranio-vertebral anomaly with irreducible atlanto-axial dislocation(AAD)*

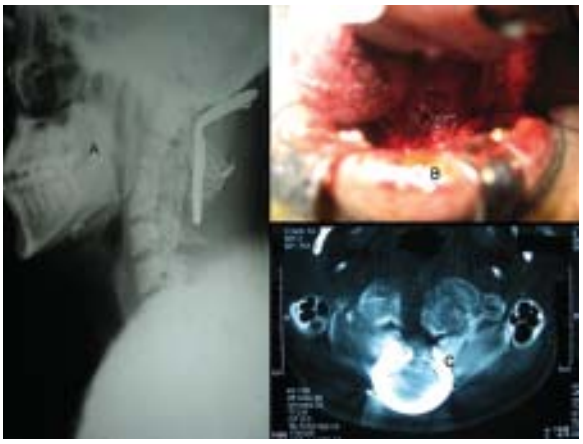

Fig.-2: *A-x-ray after posterior stabilization, B-peroperative view of closure of pharyngeal wound after transoral odontoidectomy, C-CT Scan of CV junction axial view showing absence of dens.*

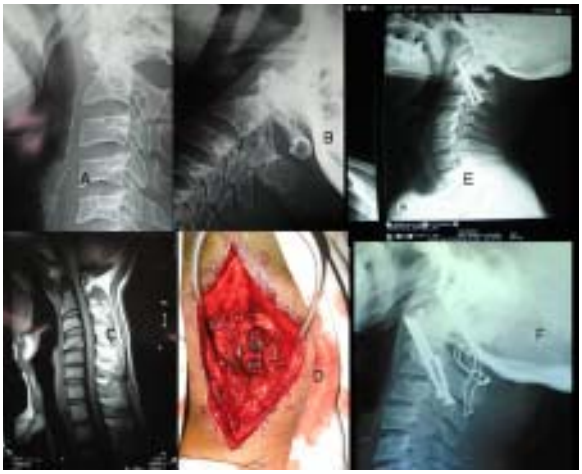

Fig.-3: *A-X-ray controlled flexion lateral view, B-x-ray extension lateral view, C-MRI T1W saggital image of cervical spine showing reducible AAD. D-peroperative picture of c1 c2 transarticular screw fixation. E-postoperative x-ray showing bilateral c1c2 transarticular screw. F-X-ray showing hardware failure in a different case(broken one screw)*

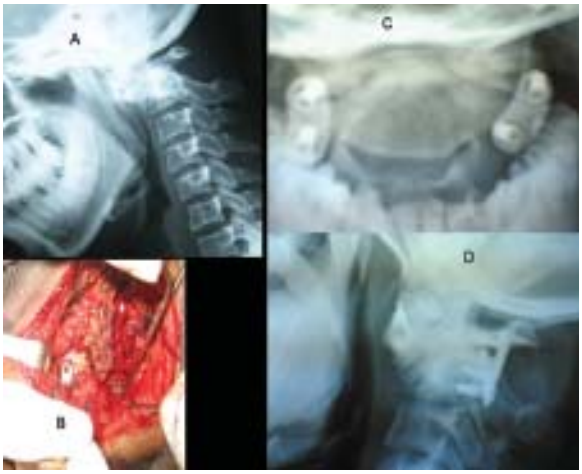

Fig.-4:*A-X-ray CV junction lateral view showing non reducible AAD, B-peroperative picture of C1C2 lateral mass plate and screw fixation, C-x-ray CV junction open mouth view showing lateral mass plate and screw, D-X-ray CV junction lateral view showing C1C2 lateral mass plate and screw in non reducible AAD with bone fusion.*

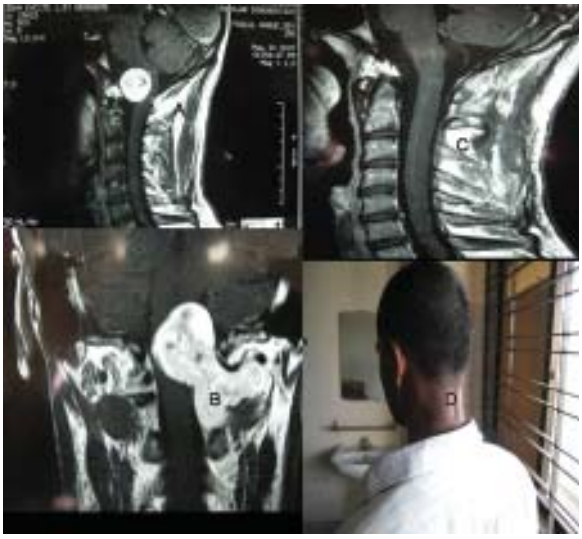

Fig.-5: *A-MRI saggital section T1W image, B-MRI coronal section T1W image of CV junction showing schwannoma. C-Postoperative MRI of CV junction*

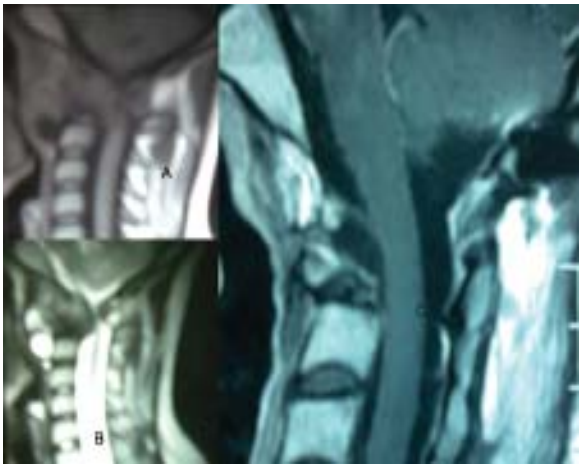

Fig.-6: *A-MRI of CV junction saggital section T1W image, B-MRI of CV junction saggital section T2W image showing multiple cystic lesion compressing cervico- medullary junction. C-Postoperative MRI saggital section T1W image showing no residual cyst.*

Discussion:

Various types of abnormalities can affect[1] cranio vertebral junction. Common pathologies include-developmental anomalies i.e. atlanto-axial dislocation (AAD), basilar invagination (BI), Chiari malformation, syringomyelia etc; traumatic i.e. AAD, fracture of C1, C2, odontoid fracture etc; inflammatory-rheumatoid arthritis; infective-tuberculosis; neoplastic-schwannoma, meningioma, cordoma, bony tumour etc.[1,2,3,4,5,6,7,8,9,10,11,12] In our study, we can find the similar picture. Here we found a very rare condition that is hydatid cyst (Table-2,case no-13). This was a

known case of hydatid cyst and she underwent surgery at CV junction in abroad 1 year back; after 1st operation she improved but later she developed quadriplegia. In our another case (Table-1,case no-19), we found AAD associated with psoriatic arthritis. Young people are commonly affected by CV junction pathologies. In this series common age is 25 to 45years. Common mode of presentation is history of trauma, neck pain, features of myelopathy i.e .quadriparesis or quadriplegia, hand muscle atrophy, autonomic dysfunction etc.[1,2,3,4,5,8,] Plain x-ray is very important to see bony alignment ,AAD dislocation and its reducibility. CT scan gives much better information than plain x-ray but usually done when x-ray picture is not clear.[2,3,4,5] We have done plain x-ray in all cases but CT scan in 04 cases only. MRI is essential to see neural pathology and other soft tissue pathology. We have done MRI in all cases. We also used per operative radio-imaging to see the reduction, alignment of atlanto-axial joint & bones and also to see the tract of screw.

We put three traumatic AAD patient under tong traction for a period of 7-15 days. Surgical procedures varies according to the pathology in CV junction.[2,4,5,7]

When AAD is reducible assessed by preoperative flexion and extension lateral view x-ray and lateral masses of C1 and C2 are intact (not destroyed by pathological process), bilateral transarticular lateral mass screw fixation is done. Transarticular screw fixation is one of the important method in the treatment of craniocervical instability.[13,14,15] It gives immediate stability to C-V junction and patient can be mobilized on immediate post operative period. Peroperative mal tracking of screw with vertebral artery injury or spinal cord damage which are potentially dangerous complications.[13,14,15] Restriction of neck mobility is not so prominent here. To prevent long term hardware failure usually C1 C2 posterior bony fusion done along with the screw. Reduction and bilateral C1C2 transarticular screw fixation with fusion by bone grafting done in 09cases in our series (Figure-3). Reduction & unilateral C1C2 transarticular screw fixation with fusion by bone grafting done in 01 case due to destruction of opposite lateral mass by tubercular lesion. In one of our cases one sided screw was broken six month after operation (Table-1,case no-1;Figure-3F) and there was neurological deterioration with severe neck pain, for which we have to go for transoral odontoidectomy; patient improved after anterior decompression.

Posterior fossa decompression with removal of C1 posterior arch +/-C2 laminectomy and duraplasty is the usual treatment in type-I Chiari malformation.[16] If syrinx is associated with the malformation some advocate syringosubarachnoid shunt. We did not perform any shunt in presence of syrinx.

When per operative reduction of AAD is not possible or transarticular screw fixation or lateral mass screw fixation is not possible then posterior stabilization by loop/wiring + occipito-cervical/atlanto-axial fusion with or without transoral decompression is needed.[1,2,3,4,5,7,13] After posterior stabilization we did transoral decompression in-03 cases.

C1C2 lateral mass screw& plate stabilization and fusion is one of the important techniques described by Goel et al, is used for treating reducible AAD or reducible basilar invagination and other craniovertebral pathologies.[17,18] We used this technique in three (9.09%) cases of AAD. In one case we failed to reduce AAD(only partial reduction was possible peroperatively by traction and effort of joint distraction) where stabilization was done by C1C2 lateral mass plate and screw with bone fusion and advised for transoral decompression on separate occasion. Postoperatively patient improved neurologically and refused second operation (Figure-4).

In one case we found BI with occipitalization of C1 and morphological dysgenesis and hyperplasia of C2.In such a case options are- preparation of C1 from occiput +/- decompression by removing posterior margin of foramen of magnum followed by occipito-axial fusion/ C1-C2 lateral mass plate & screw fixation with or without transoral decompression.[19,20] Here decompression by removing posterior margin of foramen of magnum with preparation of C1 from occiput followed by U loop-wire stabilization and bony fusion between prepared C1 and C2 was done.In this case we went for transoral odontoidectomy on a separate setting.

Tumors in this area is usually removed through posterior, posterolateral, lateral or far lateral approaches.[8,9,10,11,12] We used posterior & posterior-lateral approach only (Figure-5).

In the case of hydatid cyst we removed the cysts through posterior approach and she recovered, completely after

8 months cyst recur with some neurological deterioration, we put her on anti helminthic; cyst disappeared and she recovered again(Figure-6). She got married 2 months back but she is still on albandazole.

In our series one patient expired from severe high cervical spinal injury with respiratory distress before operation. Another patient expired three months after operation from complication of quadriplegia (bedsore, urinary tract infection and respiratory tract infection) who had quadriplegia with respiratory distress preoperatively. Postoperatively respiration was stable but there was no neurological improvement.

One patient deteriorated immediate postoperatively who later improved but not up to preoperative state. This was due to iatrogenic damage. In one case of CV junction schwannoma we faced post operative CSF fistula that was managed by lumbar drain.

The ultimate result of CV junction surgery is in favour of surgeon when appropriately chosen surgical technique is applied by a safe surgeon.[13] The rate of post surgical success varies according to pathologies along with severity & duration of neurodeficit . In our series the neurological improvement was 93.75% (in spite of improvement one patient was non ambulant), deterioration in 3.12% and death in 3.12%.

Conclusion:

Surgery at CV junction is a very challenging task even for an experienced skullbase surgeon. Proper anatomical, physiological, pathological, radiological and surgical knowledge (i.e. Proper evaluation of patients with proper selection of surgical procedures along with safe surgical techniques) is essential for managing these pathologies in this surgically complex site.

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"Dredging Method"- A Conservative Surgical Approach for the Treatment of Ameloblastoma of Jaw

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Summary:

Ameloblastoma is an aggressive benign odontogenic tumor of jaws with different clinical features and histologic patterns. The resection of mandible in growing young patient is associated with number of complications such as loss of jaw bone support, deformity, dysfunction and psychological distress even after reconstruction. An alternative conservative surgical procedure "Dredging Method" is a procedure which can eradicate the tumor as well as restore the normal contour and function of jaw. The procedure was followed in twenty four cases of histologically confirmed mandibular ameloblastoma in Bangladeshi patients, with recurrence of three cases in an average of two years follow up. The recurrent cases could be handled easily due to early detection in regular follow up.

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Introduction

Ameloblastoma is a locally invasive benign epithelial odontogenic tumor with different clinical characteristics and histological patterns. This tumor occurs mostly in the mandible[1-10] and is slow growing and histologically benign in nature[3-5, 9]. Resection of the mandible has been the principle treatment of ameloblastoma as the chance of recurrence is extremely high if it is treated by an inadequate procedure[3-6, 9, 10]. In most of the cases, the lesion is very extensive at the time of treatment because the tumor is painless and shows a slow and expansive growth. The resection of mandible including condyle and wide anterior region in growing young patient is associated with number of complications such as loss of jaw bone support, deformity, dysfunction and psychological distress even after reconstruction[4, 8].

The present paper introduces an alternative conservative procedure "Dredging Method" which eradicates the tumor and restores the normal contour and function of jaw in order to overcome these disadvantages. This article also describes the outline of the "Dredging Method" and presents the clinical feature of ameloblastoma treated by conservative procedures.

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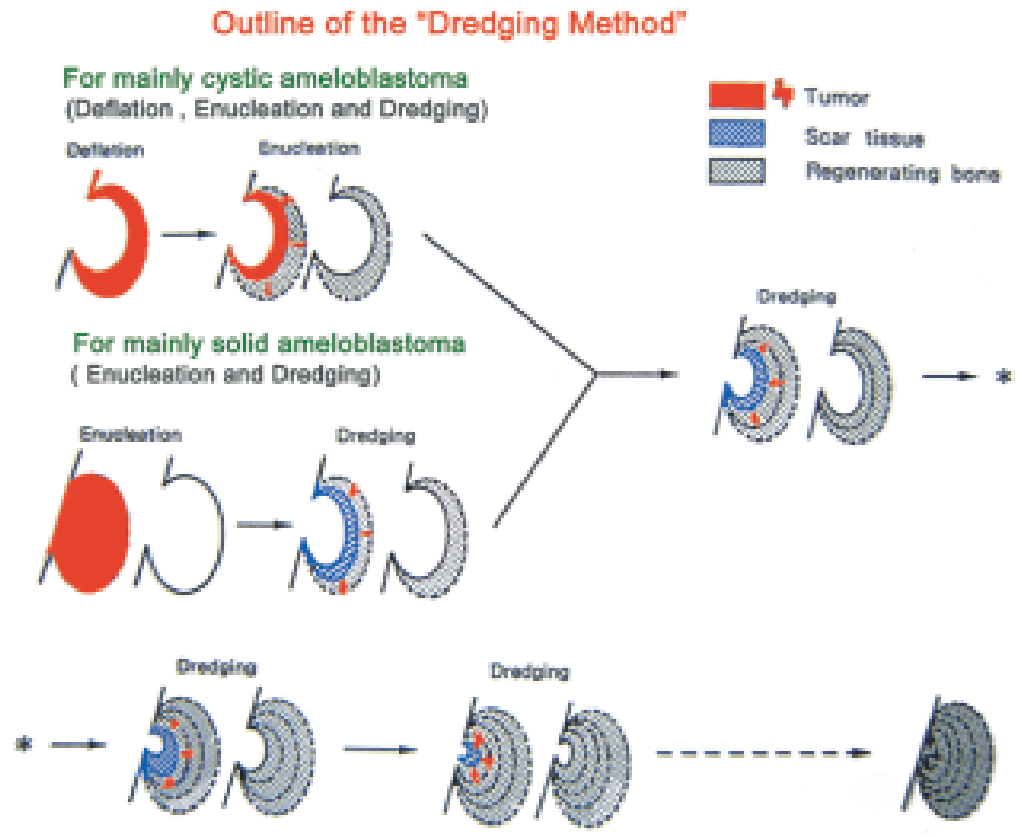
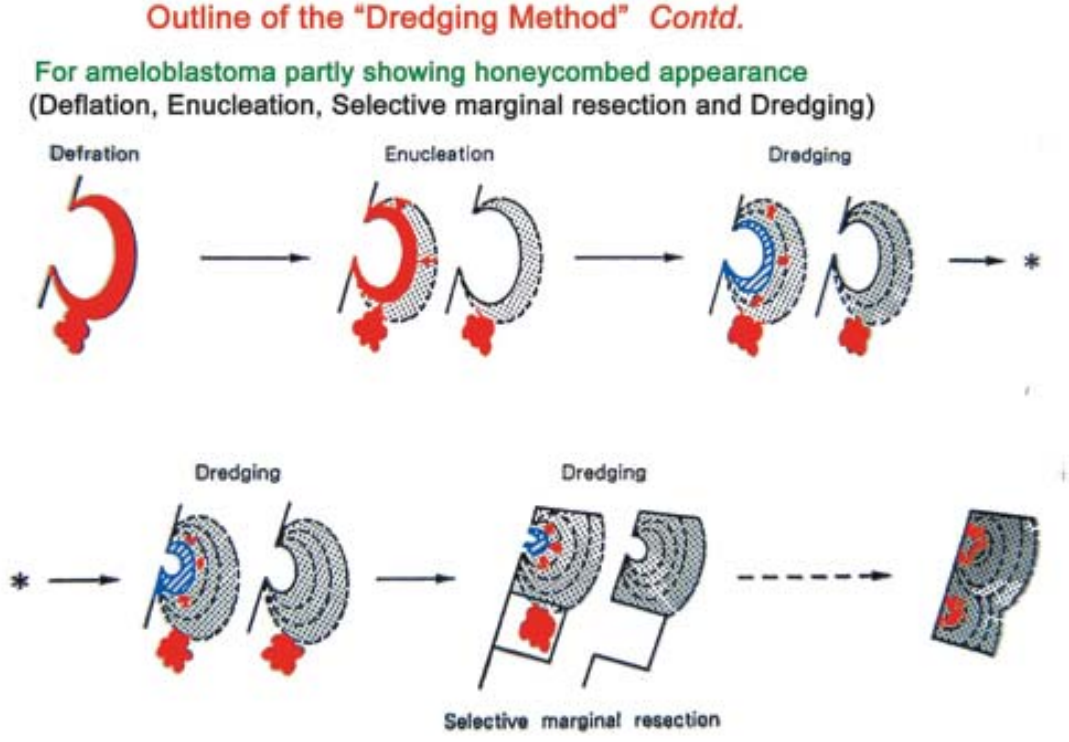
Materials and Methods:

A retrospective study had been conducted on patients who attended department of Oral & Maxillofacial Surgery in Dhaka Dental College & Hospital and a private clinic, Dhaka from August 2001 to April 2008. The study included 24 patients who were selected by convenient sampling. All patients were diagnosed as ameloblastoma histologically and subsequently treated by "Dredging Method".

"Dredging Method" is a conservative surgical procedure in which, after deflation and enucleation or only enucleation, repeated dredging is applied to accelerate new bone formation by removing out the scar tissue from the bony cavity. Deflation is used in large cystic lesion, where portion of the cystic wall, overlying bone and mucoperiosteum are removed in order to release intracystic pressure and facilitate the formation of a clear bony outline[2, 10]. Enucleation is done after formation of clear bony outline; on the other hand enucleation is done directly in solid ameloblastoma. After enucleation the tumor is removed completely along with a portion of surrounding healthy bone, and then the bony cavity is kept open. The procedure is followed by repeated dredging out of the scar tissue that fill up the bony cavity and prevent the bone formation. Dredging is applied in 2-3 months interval to accelerate new bone formation and elimination of tumor cell nests. Histological examinations for all specimens are mandatory to ensure elimination of residual tumor cells and prevention of recurrence. In "Dredging Method" the follow up begins when the tumor cells are not identified in microscopic examinations of the scar tissues removed by 2 consecutive dredging. Continuous and regular follow up is an essential part of the treatment.


Outline of the "Dredging Method"
For mainly cystic ameloblastoma
(Deflation , Enucleation and Dredging)
Deflation
Enucleation
Tumor
Scar tissue
Regenerating bone
For mainly solid ameloblastoma
(Enucleation and Dredging)
Enucleation
Dredging
Dredging
Dredging
Dredging


Outline of the "Dredging Method" Contd.
For ameloblastoma partly showing honeycombed appearance
(Deflation, Enucleation, Selective marginal resection and Dredging)
Defration
Enucleation
Dredging
Dredging
Dredging
Selective marginal resection

The above figures outlined the Dredging Method. In unicystic ameloblastoma deflation (marsupialization) is initially done to reduce tumor size which is followed by enucleation after adequate bone formation. Then dredging is applied repeatedly after every 2-3 months. In solid ameloblastoma, initial step is enucleation with removal of some peripheral bone followed by repeated dredging. In lesions having some honeycomb appearances in radiographs are treated by deflation, enucleation of total lesion with marginal resection of the area having honeycomb appearances. The total procedure ends up with repeated dredging to clear the tumor cells as well as to assist in sequential bone formation.

Results:

In the study period, a total of 88 cases of histologically diagnosed ameloblastoma were treated by different surgical approaches. Out of 88 cases 24 were treated by Dredging method and rest of the cases were treated by enucleation, marginal resection and segmental resection with or without condyle (Table-1). The male and female ratio of treated cases (by dredging method) was 5:7. Most of the cases (66.66%) were within the age of 10-20 years (Table-2). In the Dredging patients, 18 cases (75%) were treated by deflation, enucleation and dredging in which tumor cells were identified in dredged out tissues in 5 cases (Table-3) and recurrence was seen in 1 case (Table-4). 6 (25%) out of 24 cases were treated by enucleation and dredging in which tumor cells were identified in dredged out tissues in 2 cases (33.3%) (Table-3) and recurrence was seen in 2 cases (33.3%) (Table-4).

Table-I

Treatment methods and no of cases treated (August 2001-April 2008)

Method of Treatment	No of Case
Enucleation	12
Deflation Enucleation & Dredging	18
Enucleation and Dredging	06
Marginal mandibular resection	10
Segmental mandibular resection	12
Mandibular resection including condyle`	33
Total	88

Table-II

Age and sex distribution of patients underwent Dredging Technique (n=24)

Age Range	Male	Female	Total
10-15	4 ((16.7%)	4 (16.7%)	8 (33.3%)
16-20	3 (12.5%)	5 (20.835)	8 (33.3%)
21-25	3 (12.5%)	4 (16.7%)	7 (30.4%)
26-30	0 (0%)	1 (04.250	1 (04.2%)
Total	10 (41.7%)	14 (58.3%)	24 (100%)

Table-III

Clinical Evaluation by Identification of tumor cell nests in dredged out scar tissue

Treatment Method	Identified	Not Identified	Total
Deflation, Enucleation, Dredging	5	13	18
Enucleation, dredging	2	4	6

Table-IV

Clinical evaluation of cases treated by conservative treatment methods

Treatment Method	No Recur	Recur	Lost FU	Total
Deflation, Enucleation, Dredging	14	1	3	18
Enucleation, dredging	4	2	0	6\
Total	18	3	3	24

Case – 1a: Radiographs show gradual bone formation and achievement of normal mandibular contour in sequential steps of "Dredging Method" in a 10 years old girl

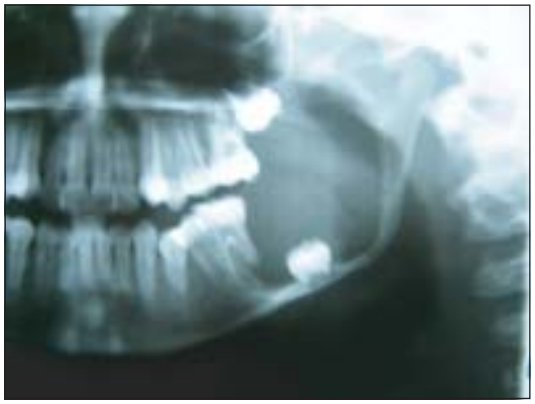


Fig-1: *Orthopantomogram shows unicystic lesion in left ramus and adjacent body of mandible*

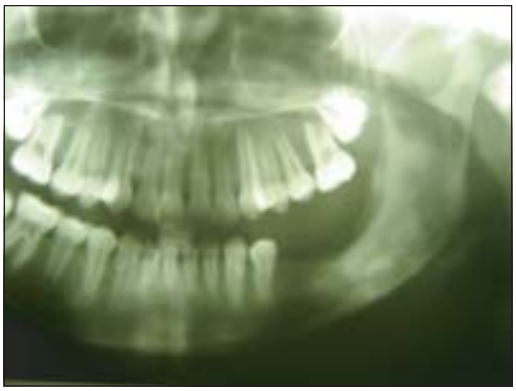


Fig-2: *Orthopantomogram shows bone formation in left ramus and body of mandible 2 months after enucleation*

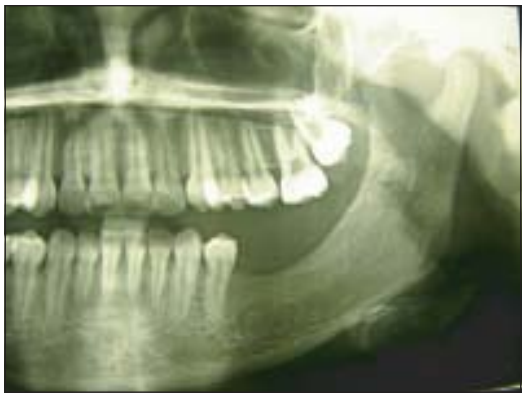


Fig-3: *Orthopantomogram shows normal bone contour with cortical outline without evidence of recurrence 6 months after 2nd dredging (12 months after enucleation)*

Case – 1b: Clinical achievement of normal facial contour by "Dredging Method" in a 10 years old girl.

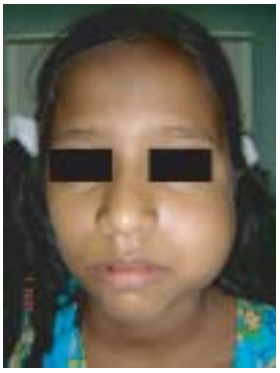


Fig-4: *Photograph shows clinically evident expansile swelling (ameloblastoma) in left mandible*

Fig-5: *Photograph shows almost normal facial contour 2 months after enucleation*

Fig.-6: *Photograph shows normal facial appearance 12 months after enucleation*

Case-2: Orthopantomograms show gradual bone formation and achievement of normal mandibular contour in sequential steps of "Dredging Method" in a 13 years old boy.

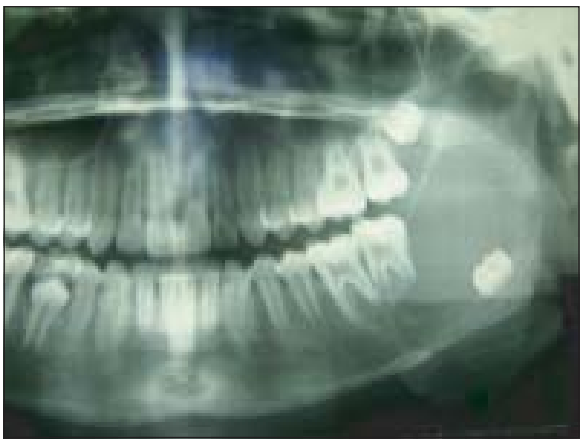


Fig.-7: *Cystic lesion in left ramus of mandible*

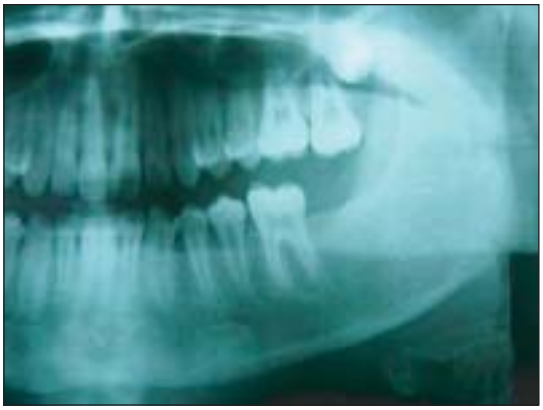


Fig.-8: *Bone formation in ramus 6 months after enucleation*

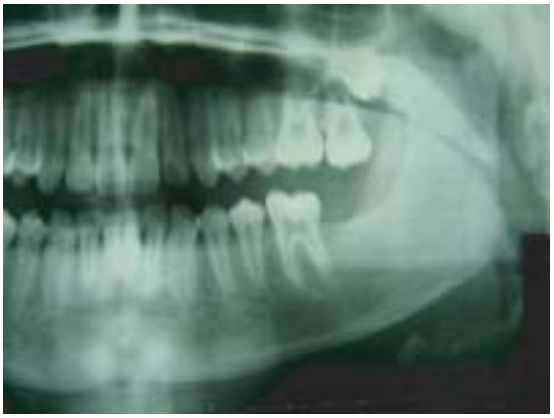


Fig.-9: *Normal cortical outline with no evidence of recurrence in 20 months follow up*

Discussion:

The contour of the face and oral cavity is directly related to the function and facial aesthetics. So, treatment of disease of the oral cavity becomes inadequate if it causes deformity of face. Deformity of the oral cavity causes functional inconvenience, aesthetic dissatisfaction and mental agony. So, the purpose should be – correction of disorder as well as to restore normal contour and function of jaw[4,12]. Considerations should be given to the age of patient, site, nature, extension of lesion. Dredging Method is considered to fulfill the purposes. It is seen that after deflation and enucleation the tumor cells are identified in the scar tissue within the bony cavity which is the cause of recurrence. So the scar tissue should be dredged out repeatedly to prevent the recurrence as well as to accelerate new bone formation. We got very low recurrence by this technique as also seen by other authors[13,14]. Follow up of these patients started when tumor cells were not identified in two consecutive microscopic examinations of dredged tissues. But often dredging is continued only for restoration of bony defect. For the treatment of ameloblastoma, a continuous and regular follow up is an essential part[15, 16]. The authors recommended that this new technique should not be applied if the patient is not totally motivated for long term duration of follow up.

Conclusion:

A good result could be achieved in surgical treatment of ameloblastoma by systematic application of the technique -"Dredging Method". This procedure not only helps in acceleration of new bone formation at surgical areas but also helps in eliminating the tumor cell nests in the scar tissue. The facts indicate the essential role of dredging subsequent to deflation and enucleation. Ameloblastoma has usually variable clinical and histological patterns, and so the suitable treatment method from various procedures; i.e., deflation, enucleation, dredging, and different types of resection, should be chosen for successful outcome of this lesion.

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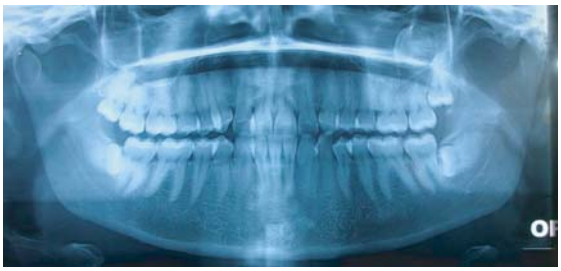
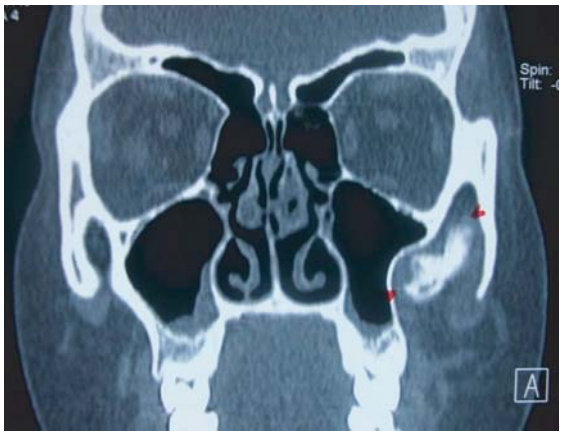
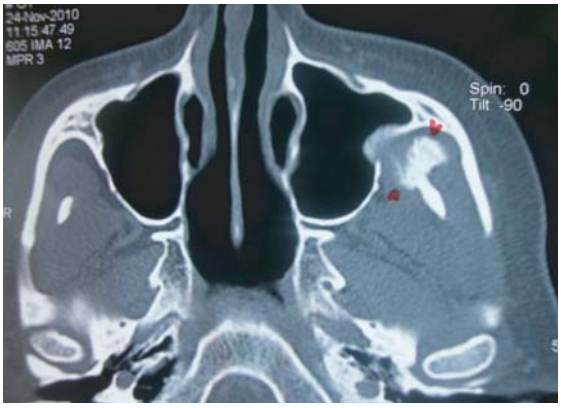
Images in Medical Practice
A Patient with Rare Cause of Restricted Mouth Opening

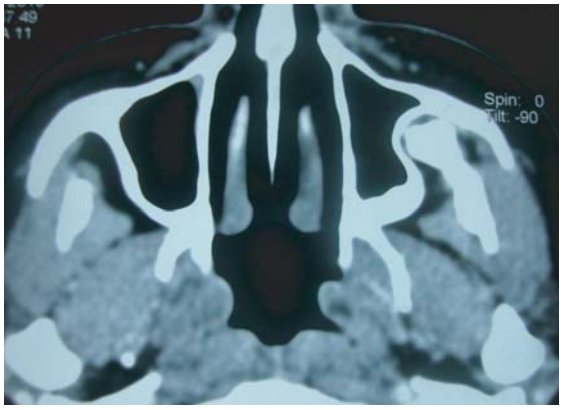
S. M. ANWAR SADAT[a], SUFIA NASRIN RITA[b], MAHFUJUL HAQ KHAN[c]

(J Bangladesh Coll Phys Surg 2011; 29: 109-110)









A 30-year-old female patient presented with gradual inability to open her mouth for last eight months without any history of accidental injury to face, dental pain or associated fever. She was quite healthy without any habit of betel quid chewing. Clinical examination of patient revealed inadequate mouth opening with inter incisal opening of 12 mm, restricted movement of both TMJs in all dimensions with no tenderness or palpable swelling in TMJs areas and over the surfaces of mandible and maxillae. Intra-oral examination revealed unerupted all 3rd molars with normal dento-alveolar and mucosal contours as seen through limited mouth opening. Orthopantomogram of mandible and maxillae showed deep seated horizontal impaction of lower 3rd molars and vertical impaction of upper 3rd molars without any evidence of bony lesion in both lower and upper jaws. Suspecting any pathology in coronoid process, pterygomaxillary fissure, infra-temporal and temporal spaces, a CT Scan of maxillofacial region was advised

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c. Dr. Mahfujul Haq Khan, BDS, DDS (BSMMU), PhD (Japan), Post Doc JSPS, Fellow (Japan), Associate Professor and Consultant, Department of Dentistry, BIRDEM Hospital & Ibrahim Medical College, Dhaka

which showed a mushroom shaped bony lesion attached to the tip of coronoid process which compressed the lateral wall of the left maxillary antrum with remodeling of bony wall of the antrum. Lucent areas with surrounding osteosclerotic change were seen at bony outgrowth. Considering the possibility of osteochondroma, the area was exposed through intra-oral approach under general anesthesia and the mass was removed with a part of coronoid process. Histologically the lesion was confirmed as osteochondroma. After one and half month follow up, patient's mouth opening is 25 mm normally and 30 mm with stretching. Considering the experience of such case, tumor or tumor like lesions can be suspected as one of the causes of trismus or ankylosis (extra-articular) of TMJ which is usually not considered in daily practice.

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COLLEGE NEWS

Examination news:
Results of FCPS Part-I, FCPS Part-II and MCPS examination held in January 2011 are given bellow:

3161 candidates appeared in FCPS Part-I examination held in January 2011 of which 512 candidates came out successful. Subject wise results are as follows:

Sl No.	Subject	Appeared	Passed	% of Pass
1	Medicine	838	182	21.72
2	Surgery	497	41	8.25
3	Paediatrics	358	120	33.52
4	Obs. and Gynae	726	119	16.39
5	Otolaryngology	82	3	3.66
6	Ophthalmology	82	5	6.10
7	Psychiatry	16	7	43.75
8	Anaesthesiology	75	12	16.00
9	Radiology	59	0	0.00
10	Radiotherapy	29	5	17.24
11	Dermatology and Venereology	61	10	16.39
12	Physical Medicine & Rehabilitation	23	2	8.70
13	Dentistry	279	5	1.79
14	Family Medicine	2	0	0.00
15	Haematology	15	1	6.67
16	Microbiology	7	0	0.00
17	Histopathology	12	0	0.00
	Grand Total	3161	512	16.20

The following candidates satisfied the Board of Examiners and were declared to have passed the FCPS examinations held in January 2011 subject to confirmation by the council of Bangladesh College of Physicians and Surgeons.

Roll No.	Name	From where Graduated	Subject
017-8701	Dr. Tanjima Parvin	Dhaka Medical College, Dhaka	Cardiology
017-8704	Dr. Mohammed Ali	Sher-E-Bangla Medical College, Barisal	Urology
078-7002	Dr. Kamal Krishna Karmakar	Rangpur Medical College, Rangpur	Anaesthesiology
078-7004	Dr. Md Nurul Islam	Mymensing Medical College, Mymensing	Anaesthesiology
078-7005	Dr. Md Sazzad Hossain	Dhaka National Medical College, Dhaka	Anaesthesiology
078-7006	Dr. M M Shahidur Rahman	Jahurul Islam Medical College, Bajitpur	Anaesthesiology
078-7008	Dr. Mohammed Mohiuddin Shoman	Sir Salimullah Medical College, Dhaka	Anaesthesiology
078-7009	Dr. Moumita Talukder	Sir Salimullah Medical College, Dhaka	Anaesthesiology

Roll No.	Name	From where Graduated	Subject
078-7010	Dr. Reza Ershad\	Mymensing Medical College, Mymensing	Anaesthesiology
078-7013	Dr. Salma Jabeen	Dhaka Dental College, Dhaka	Conservative Dentistry and Endodontics
078-7034	Dr. Mohammed Ziaur Rahman Bhuiyan	Chittagong Medical College, Chittagong	Dermatology and Venereology
078-7040	Dr. M Morsed Zaman Miah	Rajshahi Medical College, Rajshahi	Haematology
078-7042	Dr. Mohammed Abdullah Al Anis	Chittagong Medical College, Chittagong	Haematology
078-7048	Dr. Shamoli Yasmin	Mymensing Medical College, Mymensing	Histopathology
078-7049	Dr. Taslima Hossain	Dhaka Medical College, Dhaka	Histopathology
078-7063	Dr. Tabassum Samad	Mymensing Medical College, Mymensing	Medicine
078-7116	Dr. Md Anwar Hossain	Sher-E-Bangla Medical College, Barisal	Medicine
078-7153	Dr. Md Abu Bakar Siddique	Sir Salimullah Medical College, Dhaka	Medicine
078-7190	Dr. S.M.Rezaul Irfan	Shahid Ziaur Rahman Medical College, Bogr	Medicine
078-7238	Dr. Mohammad Faisal Ibn Kabir	Dhaka Medical College, Dhaka	Medicine
078-7252	Dr. Mohammad Abdur Rahman	Faridpur Medical College, Faridpur	Medicine
078-7301	Dr. Poly Sengupta	Chittagong Medical College, Chittagong	Medicine
078-7357	Dr. Bolai Chondro Sarker	Dhaka Medical College, Dhaka	Medicine
078-7366	Dr. Md Monirul Hoque	Sir Salimullah Medical College, Dhaka	Microbiology
078-7376	Dr. Anowara Begum	Chittagong Medical College, Chittagong	Obst and Gynae
078-7380	Dr. Afroza Sultana	Armed Forces Medical College, Dhaka	Obst and Gynae
078-7381	Dr. Afroza Sultana	Rajshahi Medical College, Rajshahi	Obst and Gynae
078-7394	Dr. Fatima Wahid	Khulna Medical College, Khulna	Obst and Gynae
078-7396	Dr. Fatema Yasmin	MAG Osmani Medical College, Sylhet	Obst and Gynae
078-7407	Dr. Farzana Haseen	Chittagong Medical College, Chittagong	Obst and Gynae
078-7411	Dr. Farhana Khatoon	Sir Salimullah Medical College, Dhaka	Obst and Gynae
078-7412	Dr. Farhana Karim Satu	Sher-E-Bangla Medical College, Barisal	Obst and Gynae
078-7417	Dr. Fakhrun Nessa Manna	MAG Osmani Medical College, Sylhet	Obst and Gynae
078-7418	Dr. Fahmida Zesmin	Sher-E-Bangla Medical College, Barisal	Obst and Gynae
078-7421	Dr. Eva Rani Nandi	Sher-E-Bangla Medical College, Barisal	Obst and Gynae
078-7442	Dr. Sanjida Rahman	Rajshahi Medical College, Rajshahi	Obst and Gynae
078-7453	Dr. Nasreen Akhter	Rajshahi Medical College, Rajshahi	Obst and Gynae
078-7457	Dr. Mst Shahana Pervin	Rangpur Medical College, Rangpur	Obst and Gynae
078-7467	Dr. Rakhi Debi	Dhaka Medical College, Dhaka	Obst and Gynae
078-7479	Dr. Parveen Shahida Khanum	Rangpur Medical College, Rangpur	Obst and Gynae
078-7481	Dr. Nusrat Hossain	Dhaka Medical College, Dhaka	Obst and Gynae
078-7492	Dr. Neher Banu	Rangpur Medical College, Rangpur	Obst and Gynae
078-7517	Dr. Nahid Akhter	Sher-E-Bangla Medical College, Barisal	Obst and Gynae
078-7519	Dr. Murshida Pervin	Sir Salimullah Medical College, Dhaka	Obst and Gynae
078-7526	Dr. Mahmuda Begum Shoma	MAG Osmani Medical College, Sylhet	Obst and Gynae
078-7531	Dr. Mahfuja Asma	Sir Salimullah Medical College, Dhaka	Obst and Gynae
078-7545	Dr. Khadija Rahman Shilpi	Dhaka Medical College, Dhaka	Obst and Gynae
078-7571	Dr. Hasina Akhter	Sir Salimullah Medical College, Dhaka	Obst and Gynae
078-7579	Dr. Fazle Noor-E-Tawhida	MAG Osmani Medical College, Sylhet	Obst and Gynae
078-7586	Dr. Khandker Rokhsana Momtaz	Rajshahi Medical College, Rajshahi	Obst and Gynae

Roll No.	Name	From where Graduated	Subject
078-7590	Dr. Ishrat Sharmin	Dhaka Medical College, Dhaka	Obst and Gynae
078-7617	Dr. Samiya Alam	Sir Salimullah Medical College, Dhaka	Obst and Gynae
078-7631	Dr. Sabiha Sultana	MAG Osmani Medical College, Sylhet	Obst and Gynae
078-7636	Dr. Runa Akhter Dola	Sir Salimullah Medical College, Dhaka	Obst and Gynae
078-7638	Dr. Rumana Afroz	Dhaka Medical College, Dhaka	Obst and Gynae
078-7643	Dr. Rubaiya Ferdousi	Mymensing Medical College, Mymensing	Obst and Gynae
078-7646	Dr. Rooh-E-Zakaria	Mymensing Medical College, Mymensing	Obst and Gynae
078-7650	Dr. Rinku Rani Das	Chittagong Medical College, Chittagong	Obst and Gynae
078-7651	Dr. Rina Ghose	Rangpur Medical College, Rangpur	Obst and Gynae
078-7656	Dr. Mst. Umma Salma Chowdhury	Sir Salimullah Medical College, Dhaka	Obst and Gynae
078-7660	Dr. Mst. Nargish Khanam	Rajshahi Medical College, Rajshahi	Obst and Gynae
078-7662	Dr. Mst. Kulsum Akhter	Rangpur Medical College, Rangpur	Obst and Gynae
078-7678	Dr. Mosammath Nazma Begum	Dinajpur Medical College, Dinajpur	Obst and Gynae
078-7679	Dr. Mosammath Khadiza Mamdu	Sir Salimullah Medical College, Dhaka	Obst and Gynae
078-7680	Dr. Mosammat Shahina Begum	Dhaka Medical College, Dhaka	Obst and Gynae
078-7683	Dr. Mortuza Begum	Rangpur Medical College, Rangpur	Obst and Gynae
078-7684	Dr. Monogna Chitralekha Kundu	Rajshahi Medical College, Rajshahi	Obst and Gynae
078-7685	Dr. Monira Najnin	Rajshahi Medical College, Rajshahi	Obst and Gynae
078-7715	Dr. Sharbari Dey	Faridpur Medical College, Faridpur	Obst and Gynae
078-7719	Dr. Shilpi Saha	Shahid Ziaur Rahman Medical College, Bogra	Obst and Gynae
078-7720	Dr. Shirin Aktar Jahan	Rajshahi Medical College, Rajshahi	Obst and Gynae
078-7724	Dr. Sohely Nazneen	Sir Salimullah Medical College, Dhaka	Obst and Gynae
078-7741	Dr. Tanvina Akhter	Sir Salimullah Medical College, Dhaka	Obst and Gynae
078-7746	Dr. Taimoon Nahar Khanom	Rajshahi Medical College, Rajshahi	Obst and Gynae
078-7752	Dr. Umme Salma	Sir Salimullah Medical College, Dhaka	Obst and Gynae
078-7754	Dr. Umme Jesmin Sultana	Rajshahi Medical College, Rajshahi	Obst and Gynae
078-7755	Dr. Umme Hafsa Zakiatul Husna	Sir Salimullah Medical College, Dhaka	Obst and Gynae
078-7761	Dr. Shirin Akhter	Mymensing Medical College, Mymensing	Obst and Gynae
078-8287	Dr. Ratna Majumder	Dhaka Medical College, Dhaka	Obst and Gynae
078-7767	Dr. Syeda Najme Ara Mukhtar	Mymensing Medical College, Mymensing	Ophthalmology
078-7777	Dr. Farzana Sohel	Comilla Medical College, Comilla	Ophthalmology
078-7788	Dr. Md Emranul Islam	Sir Salimullah Medical College, Dhaka	Ophthalmology
078-7792	Dr. Chandana Sultana	Foreign Medical College	Ophthalmology
078-7798	Dr. Md. Golam Rosul	Sher-E-Bangla Medical College, Barisal	Ophthalmology
078-7803	Dr. Quazi Sindhi	Dhaka Dental College, Dhaka	Oral and Maxillofacial Surgery
078-7809	Dr. Tanzila Rafique	Pioneer Dental College, Dhaka	Orthodontics and Dentofacial Orthopaedics
078-7810	Dr. Hasnat Jahan	Dhaka Dental College, Dhaka	Orthodontics and Dentofacial Orthopaedics
078-7812	Dr. Md. Nazmul Hasan	Pioneer Dental College, Dhaka	Orthodontics and Dentofacial Orthopaedics
078-7813	Dr. Saeed Hossain Khan	Bangladesh Dental College, Dhaka	Orthodontics and Dentofacial Orthopaedics
078-8292	Dr. Mohammed Monuwarul Islam	Dhaka Dental College, Dhaka	Orthodontics and Dentofacial Orthopaedics

Roll No.	Name	From where Graduated	Subject
078-7814	Dr. Ali Imam Ahsan	Rajshahi Medical College, Rajshahi	Otolaryngology
078-7817	Dr. Shaker Ahmed	Mymensing Medical College, Mymensing	Otolaryngology
078-7819	Dr. Sanjoy Banerjee	Jahurul Islam Medical College, Bajitpur	Otolaryngology
078-7820	Dr. S M Masudul Alam	Moulana Bhasani Medical College, Dhaka	Otolaryngology
078-7822	Dr. K M Nurul Alam	Sir Salimullah Medical College, Dhaka	Otolaryngology
078-7825	Dr. Mohammad Aktaruzzaman	Chittagong Medical College, Chittagong	Otolaryngology
078-7828	Dr. Md. Zahidul Islam	Mymensing Medical College, Mymensing	Otolaryngology
078-7830	Dr. Md Mahmudul Huq	Khulna Medical College, Khulna	Otolaryngology
078-7832	Dr. Muhammad Mukhlesur Rahman	Sir Salimullah Medical College, Dhaka	Otolaryngology
078-7833	Dr. Mohammad Nasimul Jamal	Chittagong Medical College, Chittagong	Otolaryngology
078-7841	Dr. Mazharul Alam Siddique	Mymensing Medical College, Mymensing	Otolaryngology
078-7843	Dr. Mohammad Rokan Uddin Bhuiyan	Chittagong Medical College, Chittagong	Otolaryngology
078-7845	Dr. Husne Qumer Osmany	Sir Salimullah Medical College, Dhaka	Otolaryngology
078-7848	Dr. Syed Farhan Ali Razib	Rajshahi Medical College, Rajshahi	Otolaryngology
078-7852	Dr. Shegufta Rahman	Sir Salimullah Medical College, Dhaka	Paediatrics
078-7866	Dr. A R M Sakhawat Hossain Khan	Dhaka Medical College, Dhaka	Paediatrics
078-7886	Dr. Nusrat Farooq	haka Medical College, Dhaka	Paediatrics
078-7887	Dr. Nurunnaher		Paediatrics
078-7888	Dr. Nazmul Haque	Dhaka Medical College, Dhaka	Paediatrics
078-7916	Dr. Md. Azmal Hoque	Rajshahi Medical College, Rajshahi	Paediatrics
078-7927	Dr. Md Javed Iqbal	Dhaka Medical College, Dhaka	Paediatrics
078-7939	Dr. Jannatul-Ferdous	Sher-E-Bangla Medical College, Barisal	Paediatrics
078-7941	Dr. Husnea Ara Khan	Sir Salimullah Medical College, Dhaka	Paediatrics
078-7944	Dr. Gulshan Akhtar	Faridpur Medical College, Faridpur	Paediatrics
078-7957	Dr. Shahana Parveen	Dhaka Medical College, Dhaka	Paediatrics
078-7964	Dr. Rumana Shelim	Dhaka Medical College, Dhaka	Paediatrics
078-7965	Dr. Rubaiya Islam	Dhaka Medical College, Dhaka	Paediatrics
078-7983	Dr. Mohammad Shafiqul Alam Chowdhury	Chittagong Medical College, Chittagong	Paediatrics
078-8005	Dr. Mohammed Shawkat Hossain	MAG Osmani Medical College, Sylhet	Physical Medicine & Rehabilitation
078-8008	Dr. Md Zahirul Hoque Bhuiyan	Chittagong Medical College, Chittagong	Physical Medicine & Rehabilitation
078-8010	Dr. Mohammad Abdur Rahim	Chittagong Medical College, Chittagong	Physical Medicine & Rehabilitation
078-8011	Dr. Mohammad Nazim Uddin	Chittagong Medical College, Chittagong	Physical Medicine & Rehabilitation
078-8015	Dr. Abdullah Al Mamun Hussain	Rajshahi Medical College, Rajshahi	Psychiatry
078-8016	Dr. Sohel Hasan Chowdhury	Sir Salimullah Medical College, Dhaka	Psychiatry
078-8019	Dr. Tariqul Islam	Dhaka Medical College, Dhaka	Radiology & Imaging
078-8020	Dr. Afroza Parvin	Sir Salimullah Medical College, Dhaka	Radiology & Imaging
078-8049	Dr. Rivu Raj Chakraborty	Chittagong Medical College, Chittagong	Surgery
078-8053	Dr. Nurul Quayum Mohammad Musallin	Comilla Medical College, Comilla	Surgery
078-8082	Dr. Mohammad Rashedul Hasan	Rajshahi Medical College, Rajshahi	Surgery
078-8094	Dr. Mohammad Jahangir Hossain	Chittagong Medical College, Chittagong	Surgery
078-8097	Dr. Mohammad Israt Faisal	Rangpur Dental College, Rangpur	Surgery
078-8121	Dr. Md. Mozzammel Haque	Sir Salimullah Medical College, Dhaka	Surgery
078-8163	Dr. Md Shahinur Rahman	Sir Salimullah Medical College, Dhaka	Surgery
078-8167	Dr. Md Morfudul Islam	Sher-E-Bangla Medical College, Barisal	Surgery
078-8170	Dr. Md Mahboob Hasan	Mymensing Medical College, Mymensing	Surgery
078-8179	Dr. Mahamud Riyad	Sir Salimullah Medical College, Dhaka	Surgery

The following candidates satisfied the board of examiners and are declared to have passed the Preli-FCPS-II exam held in January 2011 subject to confirmation by the council of Bangladesh College of Physicians and Surgeons.

Roll No.	Name	From where Graduated	Subject
012-8409	Dr. Md.Mahbubur Rahman	Rajshahi Medical College, Rajshahi	Preli – Medicine
012-8432	Dr. Tarun Kumar Roy	Faridpur Medical College, Faridpur	Preli – Paediatrics
012-8433	Dr. Kamrun Nahar	Dhaka Medical College, Dhaka	Preli - Paediatrics
012-8434	Dr. Mohammad Mozibur Rahman	MAG Osmani Medical College, Sylhet	Preli – Paediatrics
012-8436	Dr. Ujjal Barua	Comilla Medical College, Comilla	Preli – Surgery
012-8441	Dr. Most. Afroza Nazneen	Rajshahi Medical College, Rajshahi	Preli – Surgery
012-8444	Dr. Mohammad Rabiul Karim Khan	Rajshahi Medical College, Rajshahi	Preli – Surgery
012-8448	Dr. Mohammad Azad	Mymensing Medical College, Mymensing	Preli – Surgery
012-8450	Dr. Md. Shahjahan	Chittagong Medical College, Chittagong	Preli – Surgery
012-8451	Dr. Md. Nazmul Islam	Comilla Medical College, Comilla	Preli – Surgery
012-8457	Dr. Jamal Uddin Ahmad	Rajshahi Medical College, Rajshahi	Preli – Surgery
012-8459	Dr. Harun-Or-Rashid	Comilla Medical College, Comilla	Preli – Surgery
012-8461	Dr. Muhammed Shariful Islam	Comilla Medical College, Comilla	Preli – Surgery
012-8462	Dr. Mahbubul Alam	Chittagong Medical College, Chittagong	Preli – Surgery
012-8467	Dr. Md. Mohiur Rahman Khan	Kuban State Medical Institute, Russia.	Preli – Surgery

The following candidates satisfied The board of examiners and are declared to have passed MCPS examination held In January 2011 subject to confirmation by the council of Bangladesh College of Physicians and Surgeons

Roll No.	Name	From where Graduated	Subject
078-9008	Md Tarikul Alam	Rangpur Medical College, Rangpur	Anaesthesiology
078-9009	Mohammad Hosne Mobarak	Dinajpur Medical College, Dinajpur	Anaesthesiology
078-9017	Md. Muniruzzaman	Sher-E-Bangla Medical College, Barisal	Anaesthesiology
078-9024	A. N. M. Musa Siddik	Rangpur Medical College, Rangpur	Dental Surgery
078-9028	Israt Shilpi	Bangladesh Medical College, Dhaka	Dermatology and Venereology
078-9030	A T M Rezaul Karim	Rajshahi Medical College, Rajshahi	Dermatology and Venereology
078-9031	Md. Shirajul Islam Khan	Rangpur Medical College, Rangpur	Dermatology and Venereology
078-9034	Md. Shah Zaman	People's Friendship University of Russia	Dermatology and Venereology
078-9039	Md Amiruddin	Rangpur Medical College, Rangpur	Forensic Medicine
078-9040	Md Maksud	Mymensing Medical College, Mymensing	Forensic Medicine
078-9046	Md Kamrul Hasan	Rangpur Medical College, Rangpur	Medicine
078-9065	Mohammad Najim Uddin	Chittagong Medical College, Chittagong	Medicine
078-9067	Md. Aynul Islam	Rajshahi Medical College, Rajshahi	Medicine
078-9070	Mamunur Rashid Shikder	Sir Salimullah Medical College, Dhaka	Medicine
078-9077	Jayanta Banik	Dhaka Medical College, Dhaka	Medicine
078-9131	Nitai Chandra Ray	Mymensing Medical College, Mymensing	Medicine
078-9138	Fahima Akte	MAG Osmani Medical College, Sylhet	Obst and Gynae
078-9139	Taleya Chowdhury	Chittagong Medical College, Chittagong	Obst and Gynae

Roll No.	Name	From where Graduated	Subject
078-9143	Luna Laila	Chittagong Medical College, Chittagong	Obst and Gynae
078-9145	Nazma Mazumder	MAG Osmani Medical College, Sylhet	Obst and Gynae
078-9147	Farzana Fahmin Chowdhury	Chittagong Medical College, Chittagong	Obst and Gynae
078-9151	Rabeya Sultana Jolly	Sir Salimullah Medical College, Dhaka	Obst and Gynae
078-9153	Rokeya Khatun	Khulna Medical College, Khulna	Obst and Gynae
078-9158	Samina Sultana	Chittagong Medical College, Chittagong	Obst and Gynae
078-9167	Mst. Mahfuza Begum	Rajshahi Medical College, Rajshahi	Obst and Gynae
078-9173	Rafia Sultana	Dhaka Medical College, Dhaka	Obst and Gynae
078-9182	Mst. Jesmin Akter	Sher-E-Bangla Medical College, Barisal	Obst and Gynae
078-9187	Afroza	Community Based Medical College, Mymensing	Obst and Gynae
078-9193	Farah-Naz Mabud	Fatima Jinnah Medical College, Lahore, Pakistan.	Obst and Gynae
078-9202	Farhana Tabassum	Rangpur Medical College, Rangpur	Obst and Gynae
078-9205	Nusrat Jahan	Dinajpur Medical College, Dinajpur	Obst and Gynae
078-9207	Nasrin Jahan Baker	Sir Salimullah Medical College, Dhaka	Obst and Gynae
078-9208	Lutfa Begum Lipi	Sir Salimullah Medical College, Dhaka	Obst and Gynae
078-9213	Mahbuba Akter	Moulana Bhasani Medical College, Dhaka	Obst and Gynae
078-9218	Sanchita Bhowmik	Chittagong Medical College, Chittagong	Obst and Gynae
078-9222	Ishrat Jahan	Dhaka Medical College, Dhaka	Obst and Gynae
078-9223	Sabiha Yeasmin	Dhaka Medical College, Dhaka	Obst and Gynae
078-9225	Mst Rahima Khatun	Dhaka Medical College, Dhaka	Obst and Gynae
078-9229	Shameema Hossain Khan	Sir Salimullah Medical College, Dhaka	Obst and Gynae
078-9235	Uma Nag	Mymensing Medical College, Mymensing	Obst and Gynae
078-9243	Md Shahin Akhter	Rangpur Medical College, Rangpur	Ophthalmology
078-9249	Mohammad Amir Abdulla-Hel Azam	Khulna Medical College, Khulna	Ophthalmology
078-9251	Md Lutful Kabir	Sher-E-Bangla Medical College, Barisal	Ophthalmology
078-9254	Mousumi Malakar	Medical College for Women and Hospital, Dhaka	Otolaryngology
078-9260	Mahbubul Alam Choudhury	Rajshahi Medical College, Rajshahi	Otolaryngology
078-9261	Miraj Ahmed	Sir Salimullah Medical College, Dhaka	Otolaryngology
078-9264	Md Zahidul Islam	Shahid Ziaur Rahman Medical College, Bogra	Otolaryngology
078-9339	Md Abul Bashar	Sher-E-Bangla Medical College, Barisal	Otolaryngology
078-9274	Imamuddin Md Abu Kawsar Mir	MAG Osmani Medical College, Sylhet	Paediatrics
078-9280	Md Abu Sayed Munsi	MAG Osmani Medical College, Sylhet	Paediatrics
078-9282	Md Rokibul Islam	Chittagong Medical College, Chittagong	Paediatrics
078-9284	A. N. M. Shahidul Islam Bhuiyan	Chittagong Medical College, Chittagong	Paediatrics
078-9288	Mushfique Mahmud	Bangladesh Medical College, Dhaka	Psychiatry
078-9291	Md Abu Saleh Esha	Rajshahi Medical College, Rajshahi	Radiology & Imaging
078-9295	Md. Tahidur Rahman	Rangpur Medical College, Rangpur	Surgery
078-9300	Md.Shafiqul Islam	Chittagong Medical College, Chittagong	Surgery
078-9304	Md. Sharifuzzaman	Sir Salimullah Medical College, Dhaka	Surgery
078-9325	Mohammad Rezaul Karim	Sir Salimullah Medical College, Dhaka	Surgery
078-9330	Ganesh Kumar Agarwala	Rangpur Medical College, Rangpur	Surgery

FROM THE DESK OF EDITOR in CHIEF

(J Bangladesh Coll Phys Surg 2011; 118

Dear readers, another successful year has just passed with the journal of the college maturing under the supervision of the immediate past Editor-in -Chief Prof. Quazi Tariqul Islam, who left me with the task of continuing the momentum of this prestigious journal. The first meeting of the journals new committee was held on 3rd of May, 2011 which was chaired by the Chairperson Prof. Chowdhury Ali Kawser. The agenda of the meeting included introduction of the new committee along with publication of the 1st issue of the year April 2011. Four issues of the Journal will be published in a year now onwards. The meeting was attended by all the members of the committee and ended with a jovial mood with the members looking forward to meet the challenges ahead.

Prof. HAM Nazmul Ahasan
Editor-in-Chief

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FROM THE DESK OF EDITOR in CHIEF

(J Bangladesh Coll Phys Surg 2011; .....)

The editorial board meeting was held on 29th December, 2010 and chaired by Professor AKM Mahbubur Rahman. This meeting was the last formal board meeting of this editorial board (2009-2011).

In the meeting a decision has been taken that review & revises of guideline for the authors and also for reviewer in needed. A Committee will do this task as soon as possible.

A total of 5 web sites and 5 online indexing have been done so far for the journal.

This Journal Committee (2009-2011) has tried their best to do for the quality improvement of the Journal as well as international link with websites online database.

We always invite constructive criterion of our work so that we can improve and render more for the journal.

Professor Quazi Tarikul Islam

NAME OF THE REVIEWER OF ARTICLES IN THIS ISSUE

(J Bangladesh Coll Phys Surg 2011; 29: 56)

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Obituary

The following Fellows who died between February 2011 to April 2011

Professor A. Rab Bhuiyan

Professor A. Rab Bhuiyan died on 17th February, 2011.He was awarded honorary fellowship in Radiotherapy in 2006 from Bangladesh College of Physicians and Surgeons (BCPS).

Professor Md. Nurun Nabi

Professor Md. Nurun Nabi died on 7th March, 2011. He was awarded fellowship without examination in Medicine in 1974 from Bangladesh College of Physicians and Surgeons (BCPS).

Professor M. Alauddin

Professor M. Alauddin died on 4th April, 2011. He was awarded fellowship without examination in Otolaryngology in 1994 from Bangladesh College of Physicians and Surgeons (BCPS).

Journal of Bangladesh College of Physicians and Surgeons
Vol. 29, No. 2, April 2011

Comparative Study of Stone Pulverization and Clearance Rate between Patients Treated by ESWL Under Spinal Anesthesia in Comparison with ESWL Under Sedation and Analgesia

MA HOSSAIN

Summary:

This interventional (quasi) comparative clinical study was conducted on patients with renal stone to find out the stone pulverization and clearance rate in patients treated by ESWL under spinal anesthesia and treated by ESWL under sedation and analgesia.

Selected patients were grouped as 'Group-A' for ESWL under spinal anesthesia & 'Group-B' for ESWL under sedation & analgesia. Immediate stone clearance was much higher in Group-A (96.7%) than that of Group-B (66.7%). Although both groups demonstrated 100% clearance after 3rd follow up. In this study different numbers of shock waves were given for stone pulverization as some stones were soft, hard or very hard. Under sedation and analgesia patients could not tolerate more shock waves and stayed long time on table in targeted position due to pain. But under spinal anesthesia more shock waves application was possible. This study outcome suggest that ESWL under spinal anesthesia is a better option than ESWL under sedation and analgesia.

(J Bangladesh Coll Phys Surg 2011; 29: 67-71)

Introduction:

Stone formation in the kidney is one of the oldest and widespread disease known to human beings. Calculi have been found in the pelvis, in the bladder of an Egyptian mummy estimated to be in 4800 BC[1]. The history of stone disease implies that many diverse factors might be involved in its causation like heredity, environment, age, sex, urinary infection, metabolic diseases, and dietary excesses or deficiencies[2].

It has been estimated that in United Kingdom the incidence of urinary stone disease is about 2-3%. Male to female ratio is 3:1. Stone disease is also common in Bangladesh, more in northern part of the country[3]. Revolutionary changes occurred in the field of management of renal stone in last 20 years[4]. Treatment of stone disease moved dramatically from an open operative procedure to endoscopic, minimally invasive and non-invasive methods[2]. Among those non invasive procedures ESWL is more popular. Treatment of renal stone depends on stone size, composition, position, degree of obstruction, presence of infection, single kidney, abnormal anatomy and functional status of the kidneys[1]. Management of renal and ureteral calculus disease has dramatically changed after Introduction of extracorporeal shockwave lithotripsy (ESWL) in 1980[4]. Success of ESWL depends on stone size, composition, location, excretory function of the kidneys, position of the patient, shock wave lithotripsy rate and energy level.

Principle:

The abrupt release of energy in a small space (air or water) generates high-energy amplitudes, which is called shockwaves. The physical laws of acoustics regulate the propagation and transmission of shockwaves through water or media of similar density (e.g. soft tissues). The passage of a shockwave through substances of differing acoustic impedance generates compressive stresses at the boundary surface. If the tensile strength of the encountered object (e.g. a stone) is overcome by the produced stress, the anterior surface of the stone crumbles. Part of the energy of the shockwave crossing to the posterior surface of the stone is reflected, causing fragmentation and ultimately implosion of the stone by increasing the tensile stress on the fragment. The ultimate goal of ESWL is the creation of stone fragments that are smaller than 1 mm, which can pass spontaneously and painlessly from the urinary tract[4].

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ESWL under sedation and analgesia causes pain which hampers proper fragmentation. When patient gets pain, he/she moves & target become displaced. Also energy can not be increased due to excessive pain. Even adequate number and rate of shock wave can not be exerted due to pain. On the basis of the result of the study done in different parts of the world, the present study also has been designed to compare the effectiveness of stone pulverization and clearance rate between patients treated by ESWL under spinal anesthesia in comparison with ESWL done under sedation and analgesia.

To my knowledge, no such study has been conducted in Bangladesh. Hence, this study has been designed to find out the stone pulverization and clearance rate in patients treated by ESWL under spinal anesthesia or treated by sedation with analgesia.

Materials & Methods:

Type of study

It is an interventional quasi experimental study.

Place of study

Department of Urology

National Institute of Kidney Diseases & Urology

Sher-E-Bangla Nagar, Dhaka-1207, Bangladesh.

Duration of study

July 2005 to July 2006.

Study population

Patients presenting with loin pain and / or haematuria due to renal stone at the Urology Outpatient Department of National Institute of Kidney Diseases & Urology and fulfilling patient's selection criteria mentioned below were included as study population.

Sampling technique & sample size

Sampling technique: Random sampling (Lottery Method)

Sample size was taken conveniently.

Sample size: 100

Group A (ESWL under spinal anesthesia): 50 patients

Group B (ESWL under sedation & analgesia): 50 patients

A total of 120 patients were considered for inclusion, but 10 were excluded before randomization. 110 patients were randomized by lottery into two groups namely group-A for 'ESWL under spinal anesthesia' and group-B for 'ESWL under sedation & analgesia'. After randomization four patients in the group A were withdrawn from the study by own and six (four in group-A, two in group-B) failed to attend follow-up visits. Thus total 100 patients, 50 in each group completed this study.

Patient's Inclusion criteria

1) Renal stone size < 3 cm
2) Well excreting kidneys without any congenital anomalies of the genitourinary tract.
3) For inferior calyceal stone wide infundibulopelvic angle (> 45^0).

Patient's exclusion criteria

1. Acute urinary tract infection
2. Uncorrected bleeding disorders
3. Pregnancy
4. Uncorrected obstruction distal to the stone
5. Orthopedic or spinal deformities
6. Renal ectopia, or renal malformations (including horseshoe and pelvic kidneys)

Procedure

From July 2005 to July 2006 one hundred patients of renal stone were selected according to inclusion and exclusion criteria from the urology outpatient department of National Institute of Kidney Diseases and Urology. Diagnosis was confirmed by history, physical examination, USG, plain X-ray KUB region and IVU. Size of the stone was measured by scale from 100% film of digital X-ray. After sampling of patients, group-A were selected for ESWL under spinal anesthesia and Group-B were selected for ESWL under sedation and analgesia. Follow up given at three weeks interval. Digital plain X-ray KUB, Urine culture and sensitivity, and in some cases USG were done. In group-B 50 mg pethidine given intravenously in all cases. One anesthetist was present in all cases in both groups. Re-ESWL done in all cases of residual stone. Four patients in group-A needed second session ESWL under spinal anesthesia. Twenty patients in group-B needed second session ESWL under sedation and analgesia. Only one patient needed third session ESWL in group-A and five patients in group-B for complete clearance. All data were collected in a pre-designed and pre-tested data collection

sheet. Data were processed and analyzed using software SPSS-12. Results were correlated with other study done in different parts of the world.

Observations and Results:

Total 100 subjects were selected for the study, 50 were in Group-A and 50 were in Group-B. The findings of the study derived from data analysis are presented below:

IVU findings:

Mean size of the stone of group A patients observed in IVU was 2.01 (±.58) cm and group B patients was 1.97 (±.61) cm. Mean stone size of all the present study population was 1.99 cm. Statistically no significant difference was observed (p>.05). The results shown in Table I, demonstrates that most of the stone were within 21 – 25 mm(32%), & 16-20 mm (30%) in Group-A and 16-20 mm (28%), & 21-25 mm (26%) in Group-B

Table-I

Size of the stone

Size of the stone in mm	Group	
	Group A	Group B
5-10	3 (6%)	4 (8%)
11-15	7 (14%)	8 (16%)
16-20	15 (30%	14 (28%)
21-25	16 (32%)	13 (26%)
26-30	9 (18%)	11 (22%)
Total	50 (100%)	50 (100%)

Position of the stone are shown in Table II. Statistically no significant difference was observed in terms of position of the stone (p>.05).

Table-II

Position of the stone of both groups on IVU

Position of the stone	Group A	Group B	df	p value
Upper calyx	16 (32%)*	15 (30%)	3	.979
Middle calyx	14 (28%)	15 (30%)		
Lower calyx	6 (12%)	5 (10%)		
Pelvis	14 (28%)	15 (30%)		
Total	50 (100%)	50 (100%)		

Energy level:

Most of the stones were pulverized at energy level 7 and 8 in group A and 6 and 7 in group B. Due to excessive pain energy level could not be exerted beyond 7 in group B. In group A energy level could be exerted at 8 in 16 patients. Statistically significant difference was observed in terms of energy level of both groups (p<.0001). These results are shown in Table III.

Table-III

Energy level for complete pulverization in both groups

Energy level	Group		df	p value
	Group A	Group B		
5	0 (.0%)*	10 (20%)	3	.0001
6	11 (22%)	32 (64%)		
7	23 (46)	8 (16%)		
8	16 (32)	0 (0%)		
Total	50 (100)	50 (100%)		

Number of session:

Complete clearance of stone occurred in 46 patients in group A and 30 patients in group B after 1st session. In group A only 3 patients needed 2nd session but in group B 2nd session needed for 15 patients. In group A only one patient needed 3rd session but in group B 3rd session needed for 5 patients for complete clearance of stone. Mean number of session for full clearance of stone of group A was 1.1 ±.364 and group B was 1.5±.678 (p<.001).

These results are shown in Table IV.

Table-IV

Total number of session for full clearance of stone of both groups

No. of session	Group		df	p value
for full clearance	Group A	Group B		
1	46 (92%)	30 (60%)	2	.001
2	3 (6%)	15 (30%)		
3	1 (2%)	5 (10%)		
Total	50 (100%)	50 (100%)		

Side effects:
In the operation table no patients of group A had experienced pain whereas 15 (30%) patients of group B had experienced excessive pain (p<.0001).

Nausea was reported significantly high in group B than group A (p=.046). Vomiting and steinstrasse observed more in patients of group B and haematuria more in group A. Statistically no significant difference was observed in terms of vomiting, steinstrasse and haematuria between groups. Side effects due to anesthesia and analgesia were observed only in the patients of group A. Out of all patients only two patients of group A had hypotension and headache. These results are shown in table V and VI.

Table-V

Side effects due to operation procedure (ESWL)

Side effects due	Group		p value
to operation	Group A	Group B	
Pain	0 (.0%)	15 (30%)	.0001
Nausea	2 (4%)	8 (16%)	.046
Vomiting	1 (2%)	4 (8%)	.359
Steinstrasse	2 (4%)	4 (8%)	.674
Haematuria	10 (20%)	6 (12%)	.275

Table-VI

Side effects due to anaesthesia and analgesia

Side effects due	Group		p value
to anaesthesia	Group A	Group B	
Hypotension	2 (4%)	0 (.0%)	.475
Headache	2 (4%)	0 (.0%)	.475

Complete clearance of stone: (Seen by 100% film of digital plain X-ray of KUB region).

In 1st follow up complete clearance of stone was seen in 46 patients of group A and 30 patients of group B. In 2nd follow up 3 patients of group A and 15 patients of group B showed complete clearance of stone. In 3rd follow up one patient in group A and 5 patients in group B showed complete clearance of stone. Significant difference was observed statistically (p=.001). These results are shown in table VII.

Table-VII

Complete clearance of stone in follow up.

Follow up for	Group		df	p value
clearance of stone	Group A	Group B		
1st follow up	46 (92%)	30 (60%)	2	.001
2nd follow up	3 (6%)	15 (30%)		
3rd follow up	1 (2%)	5 (10%)		
Total	50 (100%)	50 (100%)		

Discussion:
Total 100 patients were selected for this study, 50 were in Group-A and 50 in Group-B. Age range for group A was 21 to 89 years and in Group-B was 21 to 87 years. The mean age (± SD) of Group-A and Group-B were 46.06 ± 15.85 and 44.98 ± 14.71 years respectively. Mean size of the stone of group A patients observed in IVU was 2.01 (±.58) cm and of group B patients was 1.97 (±.61) cm (p>.05). Mean stone size of this present study was 1.99 cm. One British study showed mean stone size found 9 ± 4 mm and 1.07 cm[6], which does not correlate with this study. Present study demonstrates that most of the stone were within 21 – 25 mm (32%) in Group-A followed by 16-20 mm (30%) and 16-20 mm (30%) in Group-B.

IVU showed that statistically no significant difference was observed in terms of position of stone (p>.05).

In this study different numbers of shock wave were given for stone pulverization as some stone were soft, hard and very hard. Under sedation and analgesia patients could not tolerate more shock wave and stay long time on table in targeted position due to pain. But under spinal anesthesia more shock wave application was possible. In Group-A highest numbers of shock wave (3000 – 3500) were given in 20 (40%) patients. In Group-B highest numbers of shock wave (2000 – 2500) were given in 24 (48%) patients. Statistically significant difference was observed in terms of given shock wave of both groups (p=.0001). Mean shock wave was applied for group A 2810 ±436.12 and group B 2215± 476.52. (mean shock wave for all patient was 2512.5 ± 544). The mean number of shock waves was 2879 ±1415; (median of 3000; range of 900-5600) in a British study

conducted by Ather[6]. Das G et al found that for the complete clearance of stone a mean of 1200 shocks (range 100-4000) was needed at each procedure[5]. It is revealed that most of the stones were pulverized at energy level 7 and 8 in group A and 6 and 7 in group B. Due to excessive pain energy level could not be exerted beyond 7 in group B. In group A energy level could be exerted at 8 in 16 patients (p=.0001).

From the present study it is revealed that complete clearance of stone has occurred in 46 patients (92%) in group A and in 30 patients (60%) in group B after 1st session. In group A only 3 patients needed 2nd session but in group B 2nd session needed in 15 patients. In group A only one patient needed 3rd session but in group B 3rd session needed for 5 patients for complete clearance of stone. In group A subsequent sessions were also performed accordingly under spinal anesthesia and in group B under sedation and analgesia (p=.001). Mean number of session for full clearance of stone of group A was 1.1 ±.364 and group B was 1.5±.678[6].

In the operation table no patients of group A of present study had complaints of pain whereas 15 (30%) patients of group B had complaints of pain (p=.0001).

Nausea was reported significantly high in group B than group A (4% vs 16%,p=.046). However vomiting, Stainstrasse and haematuria rates were similar in both groups.

In a study in King Abdul Aziz University Hospital, Saudi Arabia, 2006 May. 64 patients underwent ESWL under spinal anesthesia and they showed that successful stone fragmentation and clearance was 90%[7].

Another study published in Canadian Journal of Anesthesia in 1997, which was done in the Department of Anesthesiology, Hadassah University Hospital, Jerusalem, Israel. That study showed that ESWL was done in continuous spinal anesthesia and successful pulverization and clearance rate was 95%[8].

In another study published in BJU in 2001, which was done in the department of urology, King Abdul Aziz Hospital, Saudi Arabia. That study showed that when ESWL was done under sedation and analgesia successful pulverization and clearance rate was 64%[9].

All these studies show that they are comparable with my study in terms of outcome in the form of stone pulverization and clearance rate. As far as outcome is considered it is seen that both study group experienced a favourable result. But in relative terms the outcome of Group-A was much better than that of Group-B. However data required validation by other studies conducted around the world on the same issue. The present study is by far the first study conducted in Bangladesh.

Conclusion:

From this study it is concluded that ESWL under spinal anesthesia permits more total shock wave and desired energy level which is more effective for pulverization and clearance of renal stone than ESWL under sedation and analgesia. So ESWL should be done under spinal anesthesia to make it more effective and tolerable to the patient.

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LETTER TO THE EDITOR

About review article "Biological agent in the treatment of Rheumatoid arthritis" (Journal Vol. 29. No. 1, 2011 Page 27-31)

To
Editor-in-Chief
Journal of Bangladesh College of Physician and Surgeon

The review article titled "Biological Agents in the Treatment of Rheumatoid arthritis" (January Vol. 29, No. 1, 2011 page 27-31) in the previous issue of your journal was an interesting read. The context covered a lot of information regarding the biologics used in RA and had a lot of information regarding individual agents on how to administer and their side effects. In spite we believe that a few more inclusion and clarification would have been nice.

The article reviewed a number of papers but it was not mentioned what were the selection criteria for the articles the authors went through. The search criteria and the mesh or key words used for generating the search along with the data bases such as Pubmed or Cochrane were also not mentioned.

Comparison of DMARD and Biologics has been looked into in a number of studies. Where some were concern with the appropriate timing of starting these agents, others were concern with the suitable candidates for biologics. Comparison of individual biologics with each other has also been seen in a good number of articles.' The authors didn't give enough information in this regard in the discussion.

The criteria for starting biologics in RA patient had been set by ACR which has changed over the years and the recent guideline came in 2008 suggesting set criteria for early, intermediate and late RA. In early RA Anti-TNFα are only advocated to a patient with high (stage C) disease activity and had never received DMARD, While in intermediate group it is advised in whom a prior MTX therapy showed a poor response. [2]

The article overlooked a very important perspective of biologics in our context, which are the chances of reactivation of tuberculosis and other infections with the use of certain biologics and ways to minimize them.

Overall it was a good informative article with the exception of the omissions made above.

Dr. Md. Titu Miah & Dr. K.F.M. Ayaz
Assistant Professor
Department of Medicine, Dhaka Medical College.

Reference:

1. Current evidence for the management of rheumatoid arthritis with biological disease-modifying antirheumatic drugs: a systematic literature review informing the EULAR recommendations for the management of RA. Nam J L, Winthrop K L, van Vollenhoven R F, Pavelka K, et al. *Ann Rheum Dis 2010;69:976-986.*

2. American College of Rheumatology *2008* Recommendations for the Use of Nonbiologic and Biologic Disease-Modifying Antirheumatic Drugs in Rheumatoid Arthritis Saag K G., Teng G G,. Patkar N M, Anuntiyo J, et al. *Arthritis & Rheumatism (Arthritis Care & Research) 2008; 59 (6) :762-784*

Author's Reply

It was nice to get a feed back on our paper. In our review we were mainly concern with the dosage schedule and administration of the drugs. The basic reason behind was to make our physicians accustomed to these agents. The comparison with DMARDs was not in the purview of our review article though it's true that comparison data would have been more informative and helpful to our readers. Regarding the search criteria we mainly went through the pubmed using the key words 'biological agents', 'rheumatoid arthritis', 'management'. The articles were chosen based on the context and mostly clinical trials were reviewed. Guidelines were also taken into account. We had tried to cover those areas of management of RA that is still in its infancy in Bangladesh and hope that it would generate enough enthusiasm among the aspiring physicians to come forward and take steps to implement studies on biological agents in our country. Finally we would like to thank you for the additional information you have provided in your letter.

Dr. SM Kamal & Prof. MA Bakar
Assoc. Professor, Medicine Khulna Medical College &
Professor of Medicine Khulna Medical College