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JOURNAL OF BANGLADESH COLLEGE OF PHYSICIANS AND SURGEONS

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EDITORIAL

Medical Ethics : Our duty now

The word ethics is from Greek word ethos, the manner and habits of man or of animals. It indicates the rules or principles which govern right conduct. In case of medical ethics, for a long time till date, it applies about the values and guidelines that should govern decisions in medicine. From time immemorial, several opinions had come in different countries all centered around it (ethics), as an issue of right and wrong. There are several definations. Some wants to relate it with professionalism. These are close to one another and there is need of relationship.

History of Medical Ethics e.g. Hippocrates (460-377 BC), code of Hammurabi (about, 2200 BC) Thomas percival (1803, Geneva Declaration 1948) etc. are there. Even one can see the changes of Hippocratic oath as the old Hippocratic Oath –425 BC and a new Hippocratic Oath –1998 AD[1]. Many of us read different books of western ethics. But our social environment, educational systems, religious beliefs and feelings, cultural heritage, available resources etc, are different.

As such the two could not be same in several aspects and should be so.

The codes or systems of western countries are not made applicable or accepted by the people of eastern parts.

We have less formal teaching programme about medical ethics in undergraduate education. It is encouraging to see that BMDC has published the code of Medical Ethics and is distributing it to the doctors[2]. The oath is taken by the members and fellows of BCPS during convocation. Another step is its inclusion in the text books as chapters[3,4].

Ethics in medicine was complementary to the teaching of symptoms and signs in Islamic University of Medina, Kufa, Bagdad, Bokhara, Samarkand from 7th century onwards. Between 8th century and the end of the 15th century Universities of Cordova, Toledo, Almeria, Eadiz etc. taught ethics along with medicine to their students.

From the relegions points of view ethics has got very high value. ‘On the day of Judgement the weightiest item is one’s balance shall be one’s good ethics, and nothing shall weight more than that’- asserts the Prophet (P,BHU) in one hadiath[5].

The Quranic verse wa mun uhyaahaa fakurrama uhannaasa jamee 'aa' (And he who saveth one life is as if he saveth the whole mankind –5 : 32) affirms that medical ethics occupy a position among the noblest of human morals[5].

The old Indian Medical Ethics are the Caraka Samhita and the Susruta Samhita. In C. Samhita the physicians were told to lead a life a celibate, speak only the truth, eat no meat, be free from envy and carry no arms. Confucian ethics had different understanding. The centre of each person’s life is not himself or herself but the family[6,7].

Going through the different books of different times and of different countries, the truth is ‘doctors belong to fraternity of noblest of professions’.

Because of different factors between east and west, as mentioned earlier, any attempt for adaptation of western ideas of medical ethics in Asia will face problems. Transferring an idea is like transplantation. Rejection is expected. We above two very important points in our civilisation- ‘benevolence’ and compassion. Previously we had the impression that bioethics was a western product. But these sort of ideas are now rapidly changing. Several countries like Japan, China etc are preparing the ethics as per their own need. American Academy of Orthopaedic Surgeons have their ethics[8].

We the Asians should interpret the principles of biomedical ethics from the perspective of Asian belief. We should remember the following points while discussing the ethics - a) moral bindings, b) legal bindings, c) conventional rules and d) codes of conduct.

Moral Bindings

For example, we should take the history very cordially and examine the patients very gently keeping in mind that he has reached me as he is in distress. In this doctor patient relationship, the patients should always feal that he is in the hand of a relation. We should have sufficient time to listen to the patients. Humane touch is mandatory.

Legal Bindings

Physician should introduce himself to the patient and also record the details of patient with date, time and signature. Maintenance of privacy is a must . Confidentiality to be maintained and informed consent for examination and any procedure to be taken.

Conventional Rules

In an era with society of progressively loosing human values altention is to be paid in all respects in particular to confidentiality. One should honour the comment of other doctor (colleagues). Patients' findings, clinical datas, diagnosis and prognosis should not be discussed in front of patients (if consent was not taken)[8]. Higher education or study on a topic needs due permission from Ethical Committee which has again rules of its own.

Codes of Conduct

The crafts of medical science is as old as mankind. We should believe the changes of codes over the years but always for the welfare of the patients. A global or universal code of medical ethics also seems paradoxical in the era of pluralism and postmodernism[9]. In 'the moral foundation of medical leadership the professional virtues of the physician as fiduciary of the patients- the detail description has been made[10].

Violation of Ethics (Unacceptable Parts)

There are certain topics which embarrass a physician.

Debates about public health ethics, equity and justice were discussed in "International debate about the right to health"[11]. Here few of those are mentioned. They need discussion and each may be a chapter for solution and decision :

Diagnostic procedures, private practice, gratification from pharmaceutical industries, self-promotion, medical certificate, public hospital vs private consultation, decision about right or wrong treatment, chair-related post holding with special motives, accepting gifts, lunch and dinner, ethics in medical journalism (code of Ethics for Medical Press)[5] etc.

Oath

Oaths are commitments. This is true for other professionals also. Several aspects are included in it. For us the following are few of those aspects

1. Confidence,
2. Physical, Mental, Social and spiritual help,
3. Help for healing
4. Obligations (Positive and negative)

Examples of positive obligations are

a) Always to promote health and happiness,
b) To inform dangers / risk of illness to a near one of patients (fatality, incurability, disabilities, complications etc),
c) Informed consent, record maintaining, postmorten, confidentiallty.
d) Social and legal obligations

Examples of negative aspect are :

a) Not to indulge in malpractice, any type of social, illegal professional practice.
b) Never to have unusual personal relationship, black mailing, distortion of wittiness / medicolegal records,
c) Never to cause personal, social embarrassment..

Health is a vital concern of every human being. Technology being its entrée into human activities long ago. The future sends ethical signals in two ways-man's hope to do and to be something; and signal of consequence. "There is no profession", Says Poul Ramsey, "that comes close to medicine in its concern to inculcate, transmit and keep in constant repair its standards governing the conduct of its members"[12].

Physicians were thought to be man of wisdom – as such they were called, Hakeem derives from Hikmat or wisdom. Now also knowledgeable persons are doing this job. We should have relink our past and recent past, do self analysis, disuss among us and have necessary alterations and additions. The

editors/authors of 'Medical Ethics in the Contemporary Era' have righty mentioned in the preface of the book'. The effort may prove to be a candle attempting to burn on a windy night to dissipate darkness to identify a path for the lonely wonderer.

AKM Mahbubur Rahman
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Professor of Surgery (Retd.)

(J Bangladesh Coll Phys Surg 2004; 22 : 79-81)

References :

1. Hope RA, Longmore JM, Mc Mamus SK, Wood-Allum CA : Hippocratic Oaths : old and new in Oxford Handbook of clinical Medicine (4th Ed) 1998 : Oxford University Press : XIV=XV.
2. Bangladesh Medical and Dental Council : Code of Medical Ethics : Printed by Quality Printing Ltd. 6 Rajani Bose Lane, Dhaka : 1-8.
3. Russel RCG, Williams NS, Bulstrode CJK (Editors) : Surgical Ethics in Baily and Love's Short Practice of Surgery (24th Ed) 2004 : Arnold, A member of the Holder Headline Group, London : 251-256.
4. Way LW, Doherth GM: Legal Medicine for the Surgery in Current Surgical Diagnosis and Treatment (11th Ed) 1994 : Int. Ed. 2003 : 72-85.
5. Khalid JA: Intraprofessional and Socio-economic Ethics of Medical Practice in Jaida HS, Jafary MH, Niaz-Anwar IL, Jawaid SA (Editors) : Medical Ethics in the Contemporary Era, 1995 : Royal Book Co. Korachi : 125-146 and Ethics in Medical Journalism SA Jawaid : 171-179.
6. Tai MC, Lin CS : Developing a culturally relevant bioethics for Asian people: J Med Ethics 2001; 27 : 51-54.
7. Bishop WJ: The Early History of Surgery: Hale London 1960 in Burnand KG, Young AE: The new Air's Companion in Surgical Studies (2nd ed) 1998: Churchill Livingstone; London: 1.
8. Code of Medical Ethics and Professionalism for Orthopaedic Surgeons by American Academy of Orthopaedic Surgeons, revised in May, 2002.
9. Neitzke G: Global aspects of Medical ethics: conditions and possibilities: Wein Med. Wochenschr, 2001; 151(9-10) : 108-12.
10. Chervenak FA, McCullough LB: the moral foundation of Medical leadership: the professional virtues of the physician as fiduciary of the patient : Am J Obstet. Gynecol, 2001 Apr : 184(5) : 875-9.
11. Ehglish V, Romano-Critchley G, Sommeriville A and Gardner L : Ethics Briiefings : J Med Ethics 2000; 26 : 473-4.
12. Voux K, Biomedical Ethics Mortality for the New Medicine 1976 : Harper and Row, Publishers, London : 3-9 and 37-45.

ORIGINAL ARTICLES

Study to Document Pre Admission Risk Factors for Development of Severe Malaria and the Spectrum of It and Outcome in Different Categories of Hospitals in Malaria Endemic Zone of Bangladesh

EB YUNUS

Summary :

As per inclusion and exclusion criteria based on World Health Organization formulated case definitions 1303 cases were screened. Out of these 909 severe malaria cases were selected at 3 different categories of hospitals of Chittagong zone. Hospitals were one Tertiary Health Care, 2 Secondary Health Care and 4 Primary Health Care providers which were, Chittagong Medical College Hospital, District General Hospitals and Thana Health Complex Hospitals respectively. The study area is the most malaria endemic zone of Bangladesh at the south-eastern part of the country. In this zone a National Malaria Control Program based on WHO sponsored country specific `Early Diagnosis Prompt Treatment' strategy has been in operation since 1994. In view of the high mortality and morbidity in presence of good care providing network and availability of effective drugs, this study envisaged documenting some preadmission factors in order to describe and if possible relate their influence on the outcome of SM cases. Moreover it tried to document the pattern and outcome of severe malaria cases at different tiers of hospitals. It was done over a period of 6 months in 1996 covering peak and off-peak seasons of malaria transmission. Data collector, who were designated Medical Officers of respective centers, were trained through workshop on the study, protocol, definitions, diagnosis, management and documentation. It was found that males in their 3rd decades were most frequent, significant numbers of pregnant cases, delays in initiating appropriate treatment, failure to recognize and prior use of first line antimalarials and significant mortality. The clinical patterns were different amongst different categories of hospitals, with severe ones were more at tertiary health care center with highest fatality, but as a whole all patterns are more frequent at secondary health care facility.

We conclude that more awareness and orientation training are needed both for the community and the professionals working in the endemic zone to ensure early diagnosis and prompt treatment. Secondary Health Care Hospitals should be more equipped for handling severe malaria cases and Chittagong Medical College Hospital should be made as a center of excellence for the same as it is the only tertiary health care provider at the perimeter of the malaria endemic zone of Bangladesh. Malaria issue should be incorporated in national MCH program. Further studies are needed including those directed to KAP.

(J Bangladesh Coll Phys Surg 2004; 22 : 83-88)

Introduction

Mortality from malaria is often due to severe and complicated form. The World Health Organisation (WHO) had a target to reduce the malaria specific mortality by at least 25% by the year 2000 and 50% by 2010 by application of new strategy for control of malaria. Mortality due to malaria[1] remained high (around 25%) among severe malaria (SM) cases in Chittagong Medical College Hospital (CMCH), which is the only tertiary health care setup at the perimeter of most malarious zone of Bangladesh, Chittagong[2]. Till recently quinine resistance has not been documented in the malaria-affected areas of the country. Even then the mortality is found to be quite high in comparison to other malaria-affected countries. It is conceivable that there are some risk factors operating at the community and the sojourn till attendance at the appropriate facilities. SM is a medical emergency demanding immediate management and, appropriate care can only be provided at hospital setting, the lowest tier is the primary health care (PHC) one. Most of the patients of cerebral malaria admitted in CMCH have been found to have fever for about a week before hospitalisation and about 85% patients after a period of unconsciousness for more than 24 hours[3]. On the other hand there are some subtle SM criteria which can easily be overlooked like 'prostration' if one is not used to these. Obviously these factors influence the outcome even after appropriate treatment. Therefore it is imperative to describe the similar risk factors as far as possible to identify the modifiable aspects, which can be picked up by the national control program for prudent intervention.

Address of Correspondence : Dr. Emram Bin Yunus, Associate Professor of Nephrology, Chittagong Medical College, Chittagong.

Aims of the study

This study has been done to describe various risk factors for the development of SM prior to admission in hospital and, to document the clinical patterns of SM at different categories of hospitals in Bangladesh.

Experimental Design and Methods :

All cases of SM admitted were documented in a Tertiary Referral Hospital, Chittagong Medical College Hospital (Tertiary Health Center - THC); two Secondary Health Care Facilities, Khagrachari and Bandarban District Hospitals (Secondary Health Center - SHC); and, 4 Primary Health Centers: Ramu, Teknaf, Matiranga and Fatikchari Thana Health Complex Hospitals (Primary Health Center - PHC); in high risk endemic area of Bangladesh, the southern and eastern part of the country. The period of study was six months from May to October 1998, which included pre-monsoon and post-monsoon. Diagnosis and management of SM were based on WHO guidelines customized in the 'National Guidelines for Clinical Management of Malaria[1]. An intensive care with minimum facilities required for nursing care, clinical monitoring, laboratory service and follow-up was established within the existing setups of CMCH and other study site hospitals. Ethical clearance was taken from the Ethical Review committee of CMC&H. Written informed consent was taken from the responsible person of each patient after elaborating the purpose of the study, the procedures and the implications. Attending doctors recorded base line data from tile patients on admission to hospital and ensured 12 hourly follow-up subsequently. For diagnosis of malaria thick and thin blood films were done for every patient. Depending on availability of blood glucose monitoring, renal and hepatic function assessment, chest x-ray, ECG and CSF study (in cerebral malaria cases only) were also done as indicated. The story preceding hospital admission was noted from tile accompanying person or patient (when able to communicate reliably) in a pre-designed proforma. A five day-long training course was arranged for twenty Medical Officers selected from all the participating hospitals to document the cases and manage them uniformly. All patients of SM were treated with parenteral quinine to be followed by oral formulation of the same as depicted by the national guidelines. Data were double entered and analysed utilizing EPINFO 6 software to determine the risk factors related to the development of SM. The different parameters of SM were compared in respect of different levels of care.

Results

Out of total screened 1308 cases, 909 fulfilled the selection criteria and enrolled for the study of which 58 patients died. Of these 339 (37.4%) cases admitted at PHCs, 382 (42.0%) at SHCs and 188 (20.6%) at THC. The male female ratio was 1.9 and mean age was 21.4 (± 14.4 SD, 95% CI 20.5 - 22.3) and maximum was 95, minimum 0.2 years with median value of 20 and mode 25. Of the female patients 39 (12.7.5.) cases were pregnant. 47.7% of cases had some form of antimalarials prior to admission within previous 2 weeks with a mean period of 2.4 (± 2.7) days. The case fatality rate was 6.4% and recovery without sequele 82.2%. But 1.4% cases recovered with some form of sequele. The outcomes of the other cases (N=91, 10%,) were not known because they left hospital before attaining the outcome.

Table – I

Description: Patient Profile of SM cases N=909

Particulars	Total 909 (100%), PHCs 339 (37.4%), SHCs 382 (42.0%), THCs 188 (20.6%)
Sex: Male N (%)	601(66%)
Male female ratio	1.9
Pregnancy N.('%)	39(12.7%)
Mean Age (SD, 95% CI), Maximum, Minimum, Median and Mode	21.4 (14.4, 20.5-22.3) 95 0.2 20.0 25.0
Within 2wks AM usage N (%)	434(47.7%)
Period of prior AM usage Days (SD)	2.4 (2.7)
Case fatality	58(6.4%)
Recovery without sequele	747(82.2%)
Recovery with sequele	13(1.4%)

AM=Antimalarials, PHC=Primary Health Center, SHC=Secondary Health Center, THC=Tertiary Health Center

On admission-duration of illness, severe symptoms, inability to eat/drink (NPO: Non Per Os) and prostration were longest at the THC, and most of them were lowest at SHCs. The travel time was lowest at SHCs followed by THC and longest at the PHCs. The hospital stay was longest at THC followed by SHCs. These durations saving NPO were significantly different between different categories of hospitals.

All patterns of presentation of SM were found in cases at district hospitals (SHCs). Amongst the types as predominant presentation severe prostration ranked the top followed by hypoglycemia, unarousable coma. convulsion, severe anemia, impaired consciousness, hyperpyrexia and hyperparasitemia. Some cases of hemoglobinuria, jaundice, abnormal behavior, pulmonary edema, acute renal failure, shock, DIC/bleedings and acidosis were also encountered. Many of the cases had more than one type (7S.4%). Severe types of SM predomiently unarousable coma were more in tertiary care hospital (THC).

Table-II

Description: Different parameters with respect to different durations at different categories of hospitals

Particulars	Total N=909	PHC N=339	SHC N=382	THC N=188	P
Duration: Illness Days (SD)	6.2 (5.5)	5.7(3.4)	5.0 (3.9)	9.7 (8.9)	< 0.001
Duration: Severe symptoms Hours (SD)	41.3 (51.3)	37(48)	37(33)	59(78)	< 0.001
Duration: NPO Hours (SD) N	35(60) 293	26(21)49	31 (53) 102	41 (72) 142	> 0.1
Duration: Impaired Consciousness hours (SD) N	30.7 (54.8) 317	14 (14) 35	24(27) 142	42(78) 126	< 0.01
Duration: Prostration hours (SD) N	37(39) 448	34(39) 309	32(28) 58	70(57)81	< 0.001
Travel time: To reach hospital hours (SD) N	2(3.2)	3.2 (4.4)	1.0 (1.9)	1.9 (2.1)	< 0.00 l
Duration: Hospital stay Hours (SD)	107(76)	81 (43)	110(54)	147 (12G)	< 0.001

PHC=Primary Health Center, SHC=Secondary Health Center, THC=Tertiary Health Center

Table-III

Predominant Presentations of patients of SM by categories of hospitals

Presentations	Total = 909	PHC=339*	SHC=382*	THC=188*	P*
Severe prostration N(%)	379 (41.7)	248 (73.2)	101 (26.4)	30(16.0)	< 0.001
Hypoglycemia N(%)	135 (14.9)	9(2.7)	123 (32.2)	3 (1.6)	< 0.001
Unarousable coma N (%)	115 (12.7)	7 (2.1)	17(4.5)	91 (48.4)	< 0.001
Convulsion N(%)	99 (10.9)	23 (6.8)	54 (14.1)	22(11.7)	< 0.05
Severe anemia N(%)	53 (5.8)	10(2.9)	27(7.1)	16(8.5)	< 0.05
Impaired consciousness N(%)	44(4.8)	20(5.9)	16(4.2)	8(4.3)	NS
Hype pyrexia N(%)	39(4.3)	15 (4.4)	18(4.7)	6(3.2)	< 0.05
Hyperarasitemia N(%)	14(1.5)	4(1.2)	8(2.1)	2(1.1)	> 0.5
Hemoglobinuria N(%)	8(0.9)	0	6(1.6)	2(1.1)	NS
Jaundice N(%)	8 (0.9)	0	2(0.5)	6(3.2)	NS
Abnormal behavior N(%)	7(0.8)	I (0.3)	6(1.6)	0	NS
Pulmonaryedema N (%)	3 (0.3)	1 (0.3)	2(0.5)	0	NS
Acute renal failure N (%)	2(0.2)	0	2(0.5)	0	NS
Shock N(%)	2(0.2)	0	0	2(1.1)	NS
DIC/Bleedings N(%)	1 (0.1)	1 (0.3)	0	0	NS
More than one	678 (78.4%)	233 (8.7%)	353 (92.4%)	92(48.9%)	< 0.001

PHC=Primary Health Center, SHC=Secondary Health Center, THC=Tertiary Health Center

Table-IV

Outcome pattern of SM categories of hospitals N=909

Outcome	Total = 909 N (%)	PHC =339 N(%)	SHC=382 N(%)	THC N=188 N(%)	
Full Recovery	747 (82.2)	286 (84.4)	321 (84.0)	140 (74.5)	P > 0.05
Recovery with sequele	13(1.4)	9(2.7)	2(0.5)	2(1.1)	P < 0.05
Death	58(6.4)	5 (1.5)	19(5.0)	34 (18.1)	P < 0.01
Not known	91 (10.0)	39 (11.5)	40 (10.4)	12(6.4)	P < 0.05

PHC=Primary Health Center, SHC=Secondary Health Center, THC=Tertiary Health Center

747 (82.20%) cases recovered fully. But 13 cases (1.4%) had some sequele after recovery. The outcome of 91 (10%) cases was not known as they left the hospital without advice and or referred to other facilities. Case fatality was highest at THC followed by SHCs. But there was also differences of various outcome patterns among various categories of hospitals both significant and non significant as well.

Discussions

We have described the patient profile and time lines and presentations of patients admitted with SM in defferent categories of hospitals in high risk areas in Bangladesh for malaria. There is in operation the 'National Malaria Control Program' to control the malaria as per the WHO guidelines, which emphasizes on early diagnosis on clinical criteria and treatment with affordable and readily available 'chloroquine' as first line agent for uncomplicated malaria and parental quinine for severe category. For a successful campaign the need is to describe relevant influencing factors for appropriate addressing.

Patients were documented in three different categories of hospitals. Most cases were male of the age group of third decade. This signifies the sections of population who were economically active and are most exposed to the vector were affected. Therefore malaria is not only a health problem rather a developmental economic issue[4]. A significant number of patients were pregnant. Pregnancy is a susceptible state for SM, and carries more risk for case fatality. Studies in Thailand revealed that SM is 3 times more common amongst pregnant women[5]. The morbidity is also high in pregnant state as well. The 'National MCH Program' should develop guidelines for malaria with pregnancy and incorporate them in field operation.

The National Guidelines for Malaria emphasizes use of chloroquine as first line antimalarials (AMs) for all fever cases without focal signs for any other febrile illness in endemic zone. But it was found that more than half of the enrolled cases didn't get any AM prior to development of SM. This issue call for more awareness of the people and professional in this regard. The over all case fatality was 6.4%. In a hospital-based series in Papua-New-Guinea it was found that the mortality was 18%[6]. On the other hand the same was found in an ICU in Singapore to be 12.5%[7]. Compared to these, the mortality in our series is low. One of the reasons for this is the non homogeneity of study stations plus less severe patterns in most cases. About 1.4% of cases developed some form of sequele. Various neurological sequele, mostly psychiatric were described in cases of cerebral malaria but the association with malaria was doubtful[8]. So SM is not only regrettable for high mortality but also for lasting morbidity and or infirmity with related social and economic consequences. A study dedicated to describe the sequele is warranted in this behalf.

SM is a medical emergency, which needs immediate management in a hospital setup. The different durations, which were documented by this study from the onset to hospital admission, will provide some important insight. These durations: onset of illness, severe symptoms, impaired consciousness, prostration, travel time, and hospital stays are all found to be in appropriately prolonged leading to delay in starting specific management. The delay may be important contributing factor to influence the outcome. The travel time to hospital was found to be comparatively less if the commuter mechanism of the country is considered. Therefore the delays are possibly due to lack of awareness about the condition and consequences. Other authors in this region of Asia also observed this[9]. Even one can speculate this lack of awareness is also prevailing amongst the practicing and operational health care personnel. A KAP study is needed to describe the situation prevailing among people and professionals.

Detail examination of different durations by different categories of hospitals provided that these are more prolong in THC. This is possibly because of the fact that patients developing complications over time and more serious cases are used to seek admission at THC and or being referred from PHCs and SHCs. But surprisingly the travel time was lowest at the SHCs that should be supposed to be in between PHCs and THCs. This needs further study. But all the durations were found to be significantly different between different categories of hospitals saving duration of prostration.

Most of the patients had more than one types or presentation of SM, types as described by WHO. But out of these in descending order the most frequent types were: Severe prostration, Hypoglycemia, Unarousable coma, Convulsion, Severe anemia, Impaired consciousness, Hyperpyrexia and Hyperparasitemia. But in a small number of cases jaundice, abnormal behavior, pulmonary edema, shock and DIC/bleedings were also present. There was a striking difference between the THC and PHCs for different types with SHCs in between encountering most of the types that the other two lacked in this series. But it was found that more severe types like unarousable coma and impaired consciousness were more frequent at THC. This feature emphasizes that the center of attention for SM management should be more focused to SHCs through making them more efficient in all respect.

The outcome data revealed that the case fatality was highest at THC followed by SHCs. This is lowest at the PHCs. As more severe cases with more serious types were conglomerated naturally and or by referral to THC and SHCs obviously the fatality rate was higher. So these centers should be upgraded and refurnished so that critical cases can be appropriately and comprehensively managed. A good number of cases, with insignificant difference between all categories of hospitals, left the hospital before the attainment of outcome. So outcome of these cases were unknown and therefore acting as an artefact for general implication of outcome data. The interpretation of those therefore needs some reservation for this reason.

Conclusion :

It can be concluded that more awareness and orientation training are needed both for the community and the professionals working in the endemic zone to ensure early diagnosis and prompt treatment. Secondary Health Care Hospitals should be more equipped for handling severe malaria cases and Chittagong Medical College Hospital should me made as a center of excellence for the same as it is the only Tertiary Health Care Provider at the perimeter of the malaria endemic zone of Bangladesh. Malaria issue should be incorporated in National MCH program. Further studies are needed to explore the situation critically.

Acknowledgement & Conflicts of Interest

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References

1. M&PDC Unit of DGHS and WHO. (1994). Recommendations of the workshop on "Malaria Clinical Case Definition - Their use for Early Diagnosis and Prompt Treatment (EDPT) and Epidemiological surveillance".
2. Rhaman MR, Faiz MA, Das JC, Yunus EB: A prospective documentation of prognostic factors of severe malaria among adult patients in Chittagong Medical College Hospital, Bangladesh. JCMCTA 1996;7(S3):32-45.
3. Faiz MA, Rhaman MR, Hussain MA, Rashid HU: Factors contributing to outcome in cerebral malaria. Bang Med Res Court Bull (in press).
4. Brundfand GH: Roll Back Malaria answering the call. 1998 http://rbm.who.int/docs/rbm brochure.htm
5. Luxemburg C, Riccf F, Nostcn F, Raimond D, Bathed S, White NJ:The epidemiology of severe malaria in an area of low transmission in Thailand. Trans R See Trop Med Hyg 91 : 256-62
6. Brown N: Severe malaria in children at Port Moresby General Hospital, Papua New Guinea. Trop Geogr Med 1995; 47 : 107-10.
7. Kool KL, Tan WL, Eng P, Ong YY: Malaria requiring intensive care. Ann Acad Med Singapore 1995 May 27 : 353-7.
8. Grag RK, Karak B, Misra S: Neurological manifestations of malaria : an update. Neurol India 1999 Jun 47 : 85-91
9. Kochar D, Kumawat BL, Karan S, Kochar SK, Karan Agarwal RP: Severe and complicated malaria in Bikaner, Western India. Southeast Asian J Trop Med Public Health 1997; 28(2) : 259-267.

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Evaluation of Menstrual Pattern in Young College Girls

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Summary

With a view to cast a glance on menstrual pattern, a descriptive cross-sectional study was carried out among 158 purposely selected young college girls of Sylhet MAG Osmani Medical College and Sylhet Nursing Institute through self-administered structured questionnaire. Age of the respondents was between 18 - 26 years with a mean of 22.4 years. Minimum age at menarche was 10 years, while maximum age was 16 years with mean 12.8 years and median 13 years. It was observed that as many as 125 (79.11 %) respondents had regular menstrual cycle, whereas 33 (20.89%) had irregular cycle. Menstrual flow was average in 129 (81.65%), scanty in 8 (5.06%) and heavy in 21 (13.29%) respondents. At least 116 (73.42%) respondents conceded that they had painful menstruation (dysmenorrhoea) with a varying degree of severity. Of them, as many as 60 (51.72%) needed medical intervention either by analgesic and / or antispasmodic. About 57 respondents has family history of dysmenorrhoea.

To establish relationship between dysmenorrhoea and its family history, conduction of a large scale study has been suggested.

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Introduction :

Menstruation is a periodic and cyclical shedding of progestational endometrium accompanied by loss of blood. This peculiar function only present in women and in higher apes[1]. Menstruation is the visible manifestation at the conclusion of one cycle of hormonal activity, marks the beginning of the next[2]. It needs coordinated interplay of hypothalamo-pituitary- ovarian axis, functioning ovary, responsive endometrium and presence of patent utero-vaginal canal for the onset of menstruation. The first 4 - 5 days of menstrual cycle is menstrual phase with shedding of two third to four fifth of endometrium. The remaining days consists basically of a proliferative and secretary phases. Menarche, the first menstrual period of life, usually occurs between the ages of 10 - 16 years, the average being 13.5 years. The age at menarche varies to some extent with family, race, social class, family size, birth order, environment, diet and general health but not with climate.[3] Menstruation tends to occur earlier in the higher social classes and in urban surroundings probably reflecting general health. It is more closely related to bone age than to chronological age. For the past couple of decades, the age of menarche is gradually declining with improvements of nutrition and environmental condition[3].

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During active reproductive life menstruation occurs at approximately 28 days interval, but 21 - 35 days is accepted as normal. Duration of menstrual period between 2 - 7 days is accepted as normal, but it must be individualized. Nonetheless, there is wide variation in duration and amount of blood loss. Menstrual flow varies from 50 - 80 ml with an average of 45 ml. In practice, however, menses lasting more than seven days, or occur at an interval of 21 days or less, or which are subjectively thought to be heavy are considered as excessive[3].

In teenage or in nullipara, menstruation may be associated with tolerable colicky pain at the beginning of mense due to uterine contraction. Pain of sufficient magnitude incapacitating the day-to-day activities is called dysmenorrhoea. Nearly 50 per cent women experience some discomfort in relation to menstruation. But in 5-10% individuals severe pain incapacitate them for several hours in each month.[1]

First period of life is usually anovular, followed by irregular ovulation. Moreover, it takes about 2 years for regular ovulation to occur. Anovulatory cycles can result in excessive bleeding. This is typically found in the post-pubertal teenager with an immature hypothalamopituitary- ovarian axis.[1]

This study was conducted among 158 young college girls to throw light on the menstrual pattern and problem related to menstruation.

Materials and Methods :

This descriptive cross-sectional study was conducted among 158 purposively selected young college girls of Nursing Institute, Sylhet, and undergraduate students of different session of Sylhet MAG Osmani Medical College, Sylhet. The study period was four months ranging from March 2002 to June 2002. Age at menarche, menstrual cycle with duration, menstrual flow including associated complaint such as dysmenorrhoea were the study variables. Data were collected through self-administered structured questionnaire.

Menstrual cycle was considered as regular one when it was within 21 - 35 days with a mean of 28 ± 2 days, while it was considered irregular when it was less than 21 days or more than 35 days. Menstrual flow was considered as scanty, average and heavy one based on number of sanitary towels used per day as mentioned by the respondents (1- 2, 3 -5 and > 5 sanitary towel per day as scanty, average and heavy menstrual flow respectively). Painful menstruation with pain ranging from dull ache to spasmodic of varying intensity was categorized as dysmenorrhoea. Data were analyzed manually and with the help of scientific calculator.

Results :

A total of 158 college girls were selected purposively and interviewed by self-administered structured questionnaire. Of them, 128 was from Sylhet MAG Osmani Medical College and 30 from Nursing Institute, Sylhet. Age of the respondents was between 18 - 26 years with a mean of 22.4 years. Of them, 9 (5.70 %) were married and 149 (94.30%) were single. Shortest age at menarche was 10 years, while highest age was 16 years with a mean of 12.8 years and median 13 years. Onset of menarche in 64 (40.51%) respondents was at the age of 13 years. Menstrual cycle was regular in 125 (79.11%) respondents, whereas 33 (20.89%) had irregular cycle (Table-I). Concerning menstrual period, it was found that a good number of respondents (148) had period within 2 to 7 days (Table- II).

Table- I

Menstrual cycle N=158

Menstrual cycle	Frequency	Percentage
Irregular	33	20.89
Regular	125	79.11
Total	158	100

Table II

Duration of Menstrual period N=158

Menstrual cycle	Duration in days		
Type	1 Day	2-7 Days	>8 Days
Regular	1	120	4
Irregular	2	28	3
Total	3	148	7

It was revealed that in 129 (81.65%) respondents menstrual flow was average, while in 8 (5.06%) it was scanty and was heavy in 21 (13.29%) respondents (Table-III). As discussed in materials and methods; menstrual flow was considered as scanty, average and heavy one based on number of sanitary towels used per day as mentioned by the respondents (1- 2, 3 -5 and > 5 sanitary towel per day as scanty, average and heavy menstrual flow respectively). At least 116 (73.42%) respondents disclosed that they had painful menstruation (Table- IV). Regarding severity, it was found that in 56 (48.27%) respondents pain was mild, whereas in 47 (40.51%) respondents it was moderate and in 13 (11.20%) pain was severe in intensity (Fig.-1). Those who had dysmenorrhoea, as many as 60 (51.72%) needed medical intervention either by analgesic and/or antispasmodic (Fig.-2). Moreover, at least 57 (49.14 %) respondents with dysmenorrhoea had family history of dysmenorrhoea (Table-V). However, the relationship either between type of menstrual cycle and dysmenorrhoea (x^2, df-1, 1.5; $P > 0.05$) or between dysmenorrhoea and family history was statistically insignificant. (x^2, df=1, 2.69; $P > 0.05$).

Table III

Menstrual bleeding by type of cycle N=158

Menstrual cycle	Menstrual bleeding		
Type	Average	Scanty	Heavy
Regular	106	6	13
Irregular	23	2	8
Total	129(81.65%)	8(5.06%)	21(13.39%)

Table- IV

Types of menstrual cycle with dysmenorrhoea N=158

Menstrual cycle Type	Dysmenorrhoea	
	Present	Absent
Regular	89	36
Irregular	27	6
Total	116(73.42%)	42(26.58%)

x^2, df=l, 1.5; P > 0.05

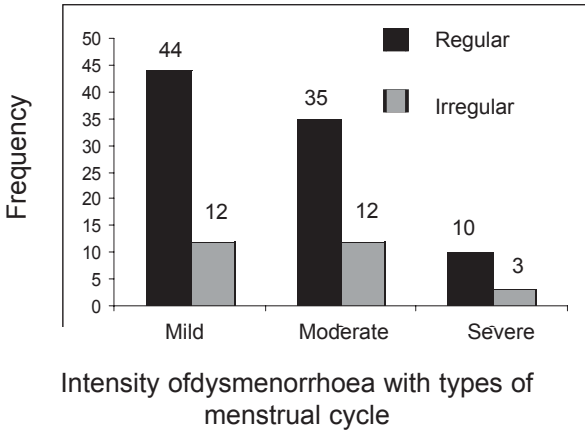

Fig.-1 : *Multiple bar diagram showing severity of dysmenorrhoea with type of menstrual cycle*

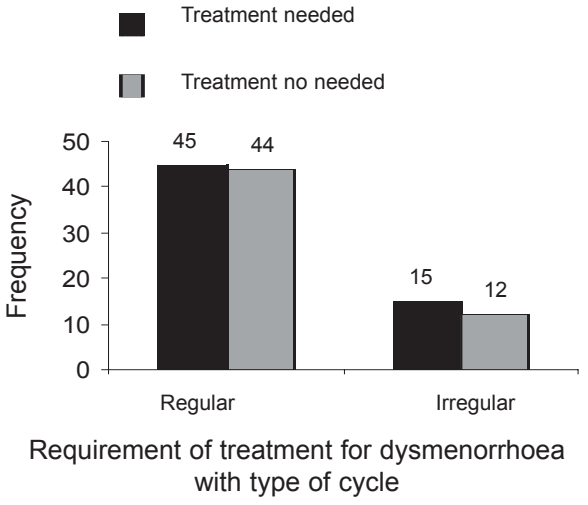

Fig.-2 : *Multiple bar diagram showing requirement of treatment for dysm enorrhoea with type of cycle*

Table-V

Dysmenorrhoea with family history N=116

Dysmenorrhoea with type of cycle	Family history of dysmenorrhoea	
	Present	Absent
Regular	40	49
Irregular	17	10
Total	57(49.14%)	59(50.86%)

x^2, df=l, 2.69; P > 0.05

Discussion :

In this study the age at menarche was 10 - 16 years with a mean of 12.8 years. This is in line with statement made by Datta.[3] In a significant number of respondents, age of menarche was 13 years which is indicated by findings of study conducted by Chowdhury et. al.[4] and corroborated by Datta,[3] and Jeffcoate[1] as well. Median age at menarche was 13 years which is very much related to findings of study conducted by Grover et. al., Singh et. al. and Hedge et. al.[5]

In respect of regularity of menstrual cycle, it was revealed that it was regular in 125 (79.11%) respondents, whereas 33 (20.89%) had irregular cycle (Table-III). As many as three respondents had menstrual cycle over ninety days. Regarding menstrual flow, it was found that it was average in 129 (81.65%) respondents, while it was scanty in 8 (85.06%) and heavy in 21 (13.29%) respondents. This is more or less similar to the findings of study conducted by Chowdhury et al.[5]

A substantial number of respondents (116, 73.41%) disclosed that they had dysmenorrhoea with various degree of severity. Furthermore, majority of them (60, 37.97%) required medical intervention either with analgesic and/ or antispasmodic. This can be compared with the study findings of Chowdhury et al.[5] where only 21.3% needed medical intervention. Moreover, at least 57 (49.14%) respondents with dysmenorrhoea had positive family history.

Conclusion :

This descriptive, cross-sectional study with sample size 158 was conducted to shed light on menstrual pattern and its related problem. To explore facts regarding menstruation however needs large group study. As incidence of dysmenorrhoea is affected by social status, age, occupation and family history[1]. It

has been suggested to test relationship between type of menstruation and dysmenorrhoea, and dysmenorrhoea with family history through large group study.

Aknowledgement

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References :

1. Neerja Bhatta (Revised and updated). Menstruation and other cyclical phenomena, In Jeffcoates Principles of Gynaecology, 6th international ed. Arnold 2001: 81-86 : 88-99.
2. I.D. Cooke Menstrual cycle and ovulation, In Dewhurts Textbook of Obstetrics and Gynaecology for postgraduates, 6th edition, Blackwell Science Ltd 1999 : 28.
3. D.C. Datta. Menstruation, In Text Book of Gynecology, 3rd edition, New Central Book Agency, Calcutta 2001 : 74-88.
4. Chowdhury T.A., Akhter S. Survey of dysmenorrhoea in a group of college girls at Dhaka city. Journal of BCPS.1985; 3(l) : 12 - 16.
5. Usha R, Krishna and Vinita Salvi. Adolescent and pediatric gynecological problems, In Ratnam SS, Bhasker Rao K, Arulkumaran S, eds. Obstetrics and Gynaecology for postgraduates, Vol-2, 1st edition, Orient longman ltd. 1994 : 293-301.

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Study of Protein Level of Cerebrospinal Fluid (CSF) in Motor Neuron Disease

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Summary:

A prospective study of cerebrospinal fluid (CSF) protein level of motor neuron disease (MND) patients was carried out in Medicine Department of Sir Salimullah Medical College, Dhaka and Sher-e-Bangla Medical College, Barisal. The duration of study was from July 1, 2000 to December 31, 2002. One hundred forty MND patients between 25 to 82 years were studied. Among them 89 were male and 51 were female. The mean age (with SD) of the patients was 53.12(±13.94) years (male 54.90(±12.32) and female 53.40(±14.52)). Clinical typing showed 67 (47.85%) had amyotrophic lateral sclerosis (ALS), 58 (41.42%) had amyotrophic lateral sclerosis with probable upper motor neuron signs (ALS-PUMNS), 11 (7.85%) had progressive muscular atrophy (PMA) and four (2.88%) had progressive bulbar palsy (PBP). Maximum patients were between 36 and 60 years and only 14% patients presented before 35 years of age. Cerebrospinal fluid was collected from each subject and protein was estimated. Ninety- eight patients (70%) had cerebrospinal protein level less than 50 mg/dl, 35 (25%) had protein between 50 to 74 mg/dl and seven (5%) had more than 75 mg/dl.

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Introduction:

Motor neuron disease is one of the important debilitating diseases of mankind. It is a neurodegenerative disease. Motor neuron disease can briefly be described as a complex condition resulting in muscular weakness and wasting without sensory changes[1]. Motor neurons, grouped as upper and lower motor neurons, are nerve cells that transmit signals for movement from the brain and spinal cord to muscle fibres. Motor neuron disease is characterized by progressive deterioration of and loss of these neurons. The loss of nerve stimulus to specific muscle results in atrophy and progressive weakness leading to paralysis. Progressive muscular wasting disease was first described in 1850 by Aran and in 1860 by Duchenne. In 1869, the French neurologist Jean-Martin Charcot described a unique condition characterized by deterioration of both upper and lower motor neurons and this condition was termed amyotrophic lateral sclerosis[2]. If only lower motor neuron involvement is evident, the condition is termed progressive muscular atrophy[3]. Clinical manifestations of amyotrophic lateral sclerosis are largely dependent on the degree to which the upper or lower motor neurons are affected[4].

Amyotrophic lateral sclerosis is nearly always progressive and eventually leads to death. Common cause of death is respiratory failure or cardiac arrythmias. There are some rare reports of patients in whom the disease is stable[5]. There are three types of ALS – (i) Sporadic, (ii) Familial and (iii) Western Pacific variant. Individuals who have no family member with this condition are said to have sporadic or classic ALS. It constitutes 90% of all ALS cases. The age of onset of sporadic ALS commonly between 55 and 75 years[6,7] and more in females than males[8] (M:F = 1:1.6), but recent studies have suggested that the sex difference is decreasing[9]. Familial ALS can be defined as two or more cases occurring in the same family. About 5% to 10% of ALS cases are familial[10] and related to mutation in Cu/Zn superoxide dismutase gene[11]. There is little information about total CSF protein content in several important general reviews of CSF of MND[12-15].

Many fields of MND had been explored by many researchers. Till now we do not know it's etiology. Several simple facts are not yet known. How often is the total CSF protein content abnormally increased? This simple question is not satisfactorily answered till this moment. No work had been done in this field in our country. This has stimulated to do this study among the Bangladeshi MND patients.

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The terminologies used in this study needs clarification

1. Nomenclature:

i. Motor neuron diseases (pleural): any disease in which the primary pathology is believed to affect the perikaryon of motor neurons and the manifestations are usually paretic but some conditions are ascribed to neural over activity (cramps, twitching of muscles, spasm or persistent abnormal posture of limbs or trunk).

ii. Pure motor neuropathy: the term is used when it is believed that the primary disorder affects axons of peripheral nerve which is defined by non-clinical criteria, such as electrophysiology, histopathology or other laboratory tests.

iii. Neuronopathy: the term is used to describe the disease of the neurones when, after all tests, it is still not possible to assign the primary pathology either to perikaryon, peripheral nerve or both[16].

2. Obligatory criteria for motor neuron disease:

i. Symptoms must include focal weakness of upper motor neuron type i.e. spastic gait or limb clumsiness of corticospinal tract disorder.

ii. There must be signs of lower motor neuron lesion i.e. evidence of focal weakness and wasting of muscles. Focal wasting is needed to differentiate neurogenic atrophy of muscle from generalized wasting or gaunt appearance after weight loss or dietary restriction. Other lower motor neuron lesion signs, not mandatory but helpful, including loss of tendon reflexes and fasciculation. "Diminished tendon reflexes" do not count because "diminished" is difficult to define. "Lost" means that the patellar reflexes are absent with reinforcement and ankle jerks are absent in the kneeling position.

iii. Unequivocal upper motor neuron signs include positive Hoffmann signs with flexor plantar response. Though hypertonicity is a feature of upper motor neuron lesion, yet it is difficult to define objectively on clinical ground and is not under consideration.

3. Definitions of some clinical syndromes:

i. Progressive muscular atrophy (PMA): lower motor neuron signs only.

ii. Amyotrophic lateral sclerosis (ALS): lower motor neuron signs and unequivocal upper motor neuron signs.

iii. Amyotrophic lateral sclerosis with probable upper motor neuron signs (ALS-PUMNS): over active tendon reflexes in limbs with weak, wasted and twitching muscles but no clonus or plantar extensor.

iv. Progressive bulbar palsy (PBP): a syndrome in which dysarthria and dysphagia are predominant symptoms and in which there is weakness, wasting and faciculation of tongue. There may or may not be concomitant upper motor neuron signs.

v. Primary lateral sclerosis (PLS): a syndrome characterized by upper motor neuron signs and no lower motor neuron signs. It is uncertain whether PLS is a form of MND[16]. Rarely this syndrome may evolve to full ALS[17].

Materials and methods:

Selection of patients: The daily admission register of Medicine Department of Sir Salimullah Medical College and Mitford Hospital, Dhaka and Sher-e-Bangla Medical College Hospital, Barisal were screened continuously for patients who were thought to have disease related to ALS or PMA. The study period was from July 1, 2000 to December 31, 2002. In the present study all patients had lower motor neuron signs. Patients with PLS were excluded. Patients were divided into four groups: ALS, ALS-PUMNS, PMA and PBP.

Exclusion criteria: A total of 260 patients were suspected to have MND according to admission register. They were examined thoroughly to judge whether inclusion criteria were met. Of the 260 patients, so selected, 70 were rejected because they did not meet the clinical diagnostic criteria. These included 40 patients with spastic paraparesis or paraplegia without evidence of lower motor neuron signs and eight patients who met established criteria for the diagnosis of PLS. Eleven patients had disorders unrelated to MND. Charcot-Marie-Tooth disease, lumbosacral plexopathy, post-polio muscle atrophy, progressive supranuclear palsy, benign fasciculation, pseudobulbar palsy related to multiple

strokes were also excluded. In six patients the diagnosis of MND was indeterminate. Five patients with MND who had coexistent sensory motor neuropathy were excluded.

Inclusion criteria: Out of 190 patients who met the criteria, 50 were excluded because they did not have full laboratory studies. Cerebrospinal fluid protein estimation was done in all the remaining 140 patients for the study purpose. Following were the inclusion criteria:

Age between 21 and 80 years of any sex; patients with upper motor neuron lesion signs (hypertonia, hyperreflexia and extensor plantar response); patients with lower motor neuron lesion signs (flaccid paralysis, muscle wasting and fasciculation) and patients with both upper motor neuron lesion signs and lower motor neuron lesion signs.

Results:

Clinical syndromes: Out of 140 patients who met inclusion criteria, there were 89 men with mean age (±SD) 54.9 (±12.32) years with age ranging from 25 to 82 years. There were 51 women with mean age (±SD) 53.4 (±14.52) in years with age ranging from 15 to 85 years. Sixty seven patients had ALS, 58 had ALM-PUMNS, 11 had PMA and four had PBP. Eleven (12.36%) of the 89 male patients and eight (15.68%) of the 51 female had symptoms before of age 39 years. Fasciculation was the major symptom which was clinically evident in 126 (90%) of the 140 patients. The remaining 14 patients without clinical fasciculation were of PMA six, ALS four and ALS-PUMNS four.

Cerebrospinal fluid protein content: Out of 140 patients, CSF protein value was greater than 50 mg/dl in 35 (25%). In seven (5%), the CSF protein value was greater than 75mg/dl. Rest 98 (70%) had CSF protein less than 50 mg/dl (ranging from 15 to 50mg/dl).

Table – I

Distribution of patients according to sex (n=140).

	Number	%	Mean age (years)
Male	89	63.57	54.90
Female	51	36.43	53.40
Male : Female	1.74:1		

Table – II

Distribution of MND patients according to clinical types (n=140).

Clinical types	Number	%
ALS	67	47.85
ALS-PUMNS	58	41.42
PMA	11	7.85
PBP	04	2.88

Table – III

Presentation of the patients before 35 years.

Symptoms before 35	Number	%
Male	11	12.36
Female	08	15.68

Table – IV

Distribution of the patients according to CSF protein value (n=140)

CSF protein value	Number	%
<50mg/dl	98	70
50 to 74mg/dl	35	25
>75mg/dl	07	05

Discussion:

This study was carried out to know the CSF protein level of Bangladeshi MND patients. The study subjects were taken from the medicine department of Sir Salimullah Medical College and Mitford Hospital, Dhaka and Sher-e-Bangla Medical College Hospital, Barisal. During the study period, 140 patients who fulfilled the diagnostic criteria of MND were evaluated.

This study revealed that majority of the patients were between 36 to 60 years. This finding correlates with the finding of a study in Bangladesh[18] but does not correlates with the European studies[6,7]. However, the finding is also similar to an Indian study by K Sood in 1990[19].

The male female ratio of the study subjects was 1.74:1 which is similar to different studies[9,10,20].

This study revealed that the mean age (with SD) of the male was 54.9 (±12.32) years and that of female was 53.4 (±14.52) years. This finding correlates with that of an Indian[19] and a Mexican study[21]. Motor

neuron disease patients were divided into ALS, ALS-PUMNS, PMA, and PBP. It was found that patients having ALS was 47.85%, ALS-PUMNS was 41.42%, PMA was 7.85% and PBP was 2.88%. This is similar to the finding of an American study[22]. It also correlates with that of Indian studies[19,23] but differs from other studies[24,25]. This dissimilarity is probably due to geographical variation, which could also be supported by similarity with the Indian study[19].

The study subjects of this series presented before 35 years in 14% and the rest 86% presented after that age. This picture corroborates with that of Younger's[22] and Ashraf's studies[18].

Seventy percent patients of this study had CSF protein value less than 50 mg/dl, 25% had between 50 to 74 mg/dl and the rest 5% had above 75mg/dl. This finding is similar to that of study in Columbia where they showed that CSF protein content was greater than 50mg/dl in 25% cases and 5% patients had CSF protein more than 75gm/dl[22]. Finding of current study is also similar to the finding of a very old study by Guiloff in 1953[26]. In another study of living MND patients, CSF value over 75gm/dl occurred in 5% of the patients[27] which is similar to this finding but does not corroborate with other studies[28].

In conclusion it may be said that CSF protein of MND patients usually do not rise but 30% of patients may show raised CSF protein. Sometimes the value may be more than 100mg/dl. This finding is similar to the other studies but the age of the patients are younger in Bangladesh which is similar to that in many Indian studies.

References:

1. Brown RH Jr. Motor neuron disease and the progressive ataxia. In: Isselbacher KJ, Martin JB, Braunwald E, Wilson JD, Fausi AS, Kasper DL, (editors). Harrison's Principles of Internal Medicine. New York: McGraw-Hill, 1994 pp 2280-86.
2. Bobowick AR, Brody JA. Epidemiology of motor neuron disease. New Eng J Med 1973; 288 : 1047-55.
3. Rowland LP, ShneiderNA. Amyotropic lateral sclerosis. New Eng J Med 2001; 344 : 1788-700.
4. Swash M. ALS 2000; the past points to the future. Amyotrophic Lateral Sclerosis and Other Motor Neuron Disorders 2001; 2 (Suppl) 1:S 3-9.
5. Mulder DW, Kurland LT. Motor neuron disease: epidemiologic studies. Adv Exp Med & Biol 1987; 209 : 325-32.
6. Eisen A. Amyotropic lateral sclerosis is a multifactorial disease. Muscle and Nerve 1995; 18 : 741-52.
7. Chio A. Risk factors in the early diagnosis of ALS: Europian epidemiological studies. Amyotrophic Lateral Sclerosis and Other Motor Neuron Disorders 2000; 1 (Suppl) 1: S 13-8.
8. Kondo K. Motor neuron disease: changing population patterns and clues for etiology. In: Schoenberg B, (editor). Advances in Neurology. New York: Raven Press, 1978 pp 509-43.
9. Maasilt P, Jokelainen M, Loytonen M, Sabel CE, Gatrell AC. Mortality from amyotropic lateral sclerosis in Finland, 1986-1995. Acta Neurol Scand 2001; 104: 232-35.
10. Rowland LP. Ten central themes in decade of ALS research. Adv Neurol 1991; 56 : 3-23.
11. Cudkowicz ME, Mc Kenna Yasek D, Sapp PE, Loytonen M, Sabel CE. Epidemiology of mutations in superoxide dismutase in amyotropic lateral sclerosis. Ann of Neurol 1997; 41 : 210 –21.
12. Friedman A, Freedman D. Amyotropic lateral sclerosis. J Nerve Ment Dis 1950; 3 : 1-18.
13. Castaigne P, Lhermitte R, Schuller E, Rouques C. Less Protein du liquide cephalo-rachidien au cours de la sclerose laterale amyotrophique. Rev Neurol (Paris) 1971; 125 : 393-400.
14. Brownell B, Oppenheimer D R, Trevor Hughes J. The central nervous system in motor neuron disease. J Neurol Neurosurg Psychiatry 1970; 33 : 338-57.
14. Lawyer T, Netsky MG. Amyotrophic lateral sclerosis – a clinico-anatomic study of fifty-three cases. Arc Neurol Psychiat 1953; 69 : 171-92.
16. Rowland LP. Peripheral Neuropathy, motor neuron disease or neuropathy? In: Battistin L, Hashim G, Lajtha A (editors). Clinical and Biological Aspects of Peripheral Nerve Disease. New York: Alan R Liss, 1983 pp 27-41.
17. Younger DS, Chou S, Oppenheimer DR, Hays AP, Schuller E, Castaigne P. Primary lateral sclerosis. Arch Neurol 1988; 45: 1304-07.
18. Ashraf Ali. Study of trace elements in MND patients. Thesis for MD in Neurology, IPGMR, Dhaka.
19. K Sood, D Nag, SV Chandra. Role of aluminium in sporadic motor neuron disease. Indian J Med Res (B) 1990; 36 : 9-12.
20. Olivares L, Esteban ES, Alter M. Mexican resistance to amyotrophic lateral sclerosis. Arch Neurol 1972; 27: 397-02.
21. E Otero Siliceo, Mencoa NA, Vazquel TC. Frequencies of motor neuron disease in a Mexico city referral center. La Revista de Investigacion Clinica 1997; 49(6) : 445-48.
22. DS Younger, LP Lowland, N Latov, Schuller E, W Sherman, M Pesce, D Nag. Motor neuron disease and amyotrophic lateral sclerosis: relation of high CSF protein content to paraproteinemia and clinical syndrome. Neurology 1990; 40 : 595-99.
23. Caroscio JT, Mulvihil MN, Sterling R, Abra B. Amyotrophic lateral sclerosis- it's natural history. Neurol Clin 1987; 5 : 11-17.
24. Mitsumoto H, Hanson MR, Chand DA. Amyotrophic lateral sclerosis-recent advances in pathogenic and therapeutic trials. Arch Neurol 1988; 45 : 489-99.
25. Swash M, Schwatz MS. What do we really know about amyotrophic lateral sclerosis. J Neurol Sci 1992; 113 : 4-16.
26. Guilloff RJ, McGregor B, Blackwood W, Parre E. Motor neuron disease with elevated cerebrospinal fluid Protein. J Neurol Neurosurg Psychiatry 1980; 43 : 390-96.
27. Shy ME, Rowland LP, Smith T, Abra B, McGregor B, Loytonen M. Motor neuron disease and plasma cell dyscrasia. Neurology 1986; 36: 1429-36.
28. Swank RL, Putnam TJ. Amyotrophic lateral sclerosis. Arc Neurol Psychiat 1943; 49 : 151-77.

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Benign Lesions Causing Facial Disfiguration

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Summary:

This retrospective study was aimed to find out diseases although benign but causing facial disfiguration. Lesions affecting the mid-face region were more (44.72%) in the study. Among 123 patients, the commonest disease was mandibular osteomylitis with discharging sinus due to dental infection. The disease profile included congenital anomalies, inflammatory as well as neoplastic lesions.

The treatment was surgical excision & repair with or without reconstruction. The outcome of the treatment was normal to accepted facial configuration in 92 (75%) patients. Cases with late presentation, extensive disease and fibrosis with repeated infection had residual deformity. The causes of late presentation are ignorance, poverty, lack of education, fear of surgery and overall poor facilities. In rural Bangladesh health education, poverty alleviation, adequate orientation, proper referral system and skilled manpower development will improve the situation.

(J Bangladesh Coll Phys Surg 2004; 22 :97-99)

Introduction :

During the course of evolution from the pre-human to modern humans, the face became smaller in relation to the overall size of the head[1]. While brain and braincase tripled in volume, the jaws became shorter and the teeth simpler in from and smaller in size. In consequence, the face receded beneath the forehead. Thus the modern human face exhibits an essentially vertical profile, in marked contrast to the protruding facial muzzle of the gorilla, the chimpanzee, and to a lesser extent, extinct hominids. The recession of the tooth-bearing portion of the jaws beneath the forehead left two distinctively human features: a prominent, projecting nose and a clearly defined chin[1].

The face grows more slowly than the nasal passages and the tooth eruption. Viewed in profile, the face at birth is less than one-fifth of the braincase; by adulthood it has increased to nearly half. Facial dimensions increase most in depth, next in height (length), and least in width; and in general to a greater extent in males than in females' .

In aesthetic point of view, human being do not want any scar in their face. Everyone is concerned with his or her impressive facial outlook. In this retrospective study, we found some diseases although benign causing facial disfiguration.

Materials & Method:

This retrospective study was done in the otolaryngology department of two-referral hospitals, Sir Salimullah Medical College & Mitford Hospital, and Institute of Post Graduate Medicine & Research, and a Private Hospital, ENT Hospital from January,1997 to December 2002. Patients admitted in the otolaryngology department with benign lesions having facial disfiguration were included in the study. Cases with malignant neoplasm were not included in the series.

Results:

Patients of second and third decade suffered than the other age groups.

Table I

Age incidence n=123

Age in years	No of Patients	Percentage
0-10	18	14.63
11-20	42	34.15
21-30	21	17.07
31-40	11	08.94
41-50	17	13.82
51 & above	14	11.38

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Table II

Sex distribution n=123

Sex	No.	Percentage
Male	82	66.70
Female	41	33.30

Male and female ratio is 2:1

Table III

Lesions affecting different facial regions were as follows:

Affected area	No	Percentage
Lower face region	38	30.99
Mid-face region	55	44.72
Lateral face region	30	24.29

Table IV

Lesions affecting lower face region (Mandibular and associated area) n=38

Lesions	No	Percentage
Mandibular osteomylitis with sinus	21	55.26
Haemangioma of the lower lip	04	10.53
Adamantinoma of the mandible	12	31.58
Fibromyxoma of the mandible	01	02.63

Table V

Diseases of the mid-face region (nose and paranasal sinuses) n=55

Lesions	No	Percentage
Tumour		
Inverted papilloma	15	27.27
Nasopharyngeal angiofibroma	07	12.72
Ossifying fibroma	04	07.27
Haemangioma	02	03.64
Osteoma of the fronto-ethmoidal complex	03	05.45
Myxoma of the maxilla	01	01.82
Dental cyst	08	14.54
Granulmatous lesions		
Rhinosporidiosis	02	03.64
Giant cell reparative granuloma	01	01.82
Miscellaneous		
Fibrous dysplasia of maxilla	07	12.72
Frontal sinus mucocele	03	05.45
Frontal sinus osteomylitis	01	01.82
Nasal vestibulitis with stenosis	01	01.82

Table VI

Lesions in the lateral face region (parotid and auricular area) n=30

Lesions	No	Percentage
Parotid swelling	14	46.67
Pre-auricular sinus with scar	09	30.00
Keloid of the pinna	03	10.00
Perichonidritis of the pinna	03	10.00
Accessory auricle	01	03.33

Treatment of the cases was total surgical excision & repair with or without reconstruction.

Table VII

Results of surgeries (n=123)

Comments	No.	Percentage
Normal facial configuration	55	44.72
Accepted facial configuration	37	'30.18
Residual deformity	30	24.29
Mortality	01	0.81

Discussion:

This is a retrospective study aimed to observe how benign lesions causing facial deformity, which are preventable with early diagnosis and proper treatment. In our study it is revealed that second and third decades are more vulnerable of these diseases. Male outnumbered female and male female ratio was 2:1 in the study.

Lesions affecting the mid-face region (nose and paranasal sinus) were more in number 55 (44.72%), followed by 38 (30.99%) in lower face region (mandibular and associated area) and 30 (24.29%) in lateral face region (parotid and pre-auricular area) respectively.

Acquired diseases were common than congenital lesions in the series, which correlates with other studies[2,3,4]. Among disease profile mandibular osteomylitis with discharging sinus in the neck due to dental infection was highest in this observation, which has significant correlation with another report[3].

Treatment given was surgical in all the cases. Reconstruction was done according to the requirement[5]. The outcome of operations was normal

to satisfactory facial configuration in 92 (75%) cases. Residual deformity was noted in the rest of the cases. Patients with late presentation, extensive disease, previous inadequate removal, fibrosis due to repeated infection had residual deformity. Causes of late presentation are ignorance, poverty, lack of education, fear of surgery and overall poor treatment facilities outside the cities.

One patient died due to anaesthetic complication during surgery. He was a case of very extensive juvenile nasopharyngeal angiofibroma.

It is concluded from this study that early diagnosis and proper treatment of benign lesions can prevent facial deformity. Health education, adequate orientation, proper referral system and development of skilled manpower will improve the situation.

References:

1. Encyclopedia Britannica Library 2003, face pp…..
2. Jafek BW, Nahum AM, Butler RM and Ward PH. Surgical treatment of juvenile nasopharyngeal angiofibroma. Laryngoscope 1973; 83 : 707-720.
3. Carl W and Sullivan MA. Dental abnormalities & bone lesions associated with familial adenomatous polyposis: report of cases. Journal of American Dental Association 1989; 119 : 137-139.
4. Hjorting-Hansan E. Benign tumours of jaws. Current opinion of dentistry 1991, 1 : 296-304.
5. Bingham BJG and Hawthrone MR. Synopsis of operative ENT Surgery, Butterworth-Heinemann, First ed. Oxford, 1992; 313-324.

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HbA1c in Diagnosis of Diabetes Mellitus without Ketonuria in Young Adult

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Summary:

Glycated protein HbA1c was tested as an alternate tool to measurement of blood glucose to diagnose diabetes mellitus in different studies. It was mostly discarded because of low sensitivity. A significant proportion of young adult diabetic patients in this country presents with very high blood glucose without ketonuria.They are lean and have persistent hyperglycemia for months. HbA1c was studied for the diagnosis of 242 consecutive cases without ketonuria at the under 30 clinic of Bangladesh Institute for Research and Rehabilitation for Diabetes, Endocrine and Metabolic disorders (BIRDEM).The subjects had high HbA1c (8.7 to 9.5%) and fasting blood glucose (13.05 to 14.75 mmol/L) respectively at 95% confidence interval.HbA1c was found to have a positive correlation with fasting blood glucose(r=0.686,p.000).HbA1c > 6.5% showed 85% sensitivity for diagnosing this subset of diabetes mellitus.Therefore,HbA1c can be used as a diagnostic tool in young adult diabetics.

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Materials and Method:

Consecutive 242 young (age < 30 years) diabetes mellitus cases without ketonuria were recruited at the under 30 clinic of Bangladesh Institute for Research Rehabilitation of Diabetes Endocrine and Metabolic disorders(BIRDEM).HbA1c was measured by HPLC (high performance liquid chromatography) along with the blood glucose values during diagnostic oral glucose tolerance test(OGTT) i.e. fasting blood glucose(FBG) and 2 hours after oral glucose load(2HG).Other variables studied were age,BMI,sex,family history of diabetes mellitus(DF), pancreatic calcification(PC), signs of nutritional deficiencies(ND), presentation of diabetes mellitus(TS), blood cholesterol(Chol) and triglycerides(TG). Statistical analysis was done to see correlation between blood glucose and HbA1c and also to see diagnostic sensitivity of HbA1c for diabetes mellitus.

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Result:

a).Distribution of subjects according to their age, BMI, cholesterol and TG levels(n=242).

Age(years):Mean 24.26 :SD 4.32 and 95% CI:23.71 to 24.80

BMI: Mean 20.77 : SD 00 and 95% CI : 20.14 to 21.41

Cholesterol(mg/dl):Mean 189.72(5.69 mmol/l):SD 55.92 (1.67)and 95% CI :182.64 to 196.79(5.47-5.90)

Triglycerides(mg/dl):Mean 202.89(2.02 mmol/l):SD 147.73 and 95% CI:184.19-221.60 (1.84-2.21).

Table-1

Distribution of subjects according to their age,BMI,cholesterol and TG levels(n=242)

Parameter	Mean with SD	95% confidence interval
Age (years)	24.25; SD 4.32	23.71 to 24.80
BMI	20.77; SD 00	20.14 to 21.41
Cholesterol(mg/dl)	189.72; SD 55.92	182.64-196.79
Triglyceride(mg/dl)	202.89; SD 147.73	184.19-221.60

b). Distribution of patients according to their HbA1c, FBG,2HG levels(n=242)

FBG (mmol/L):Mean 13.90:SD 6.71(Range 4-45.0):95% CI:13.05-14.75

2HG (mmol/L):Mean 21.82: SD 7.41 (Range 11.1-55.0) and 95% CI :20.88-22.76

HbA1c(%) : Mean 9.16 :SD 3.21(Range 3.6-21.6) and 95% CI:8.76-9.57

Table-II

Distribution of patients according to their HbA1c, FBG,2HG levels (n=242)

Parameters	Range with SD	Mean	95% CI
FBG mmol/L	4- 45.0 ;SD 6.71	13.90	13.05 -14.75
2HG mmol/L	11.1- 55.0;SD 7.41	21.82	20.88-22.76
HbA1c (%)	3.6-21.6;SD 3.21	9.16	8.76-9.57

c) Distribution of patients according to Sex, DF, PC, ND, and TS (n=242)

Male:113(46.69%),Female 129 (53.30 %),DF 117 (48.35%),PC 22 (9.09%),ND150(61.98%) and TS 169 (69.83%) cases.

Table-III

Distribution of patients according to their HbA1c, FBG,2HG levels(n=242)

Particulars	Number	Frequency(%)
Male	113	46.69
Female	129	53.30
DF	117	48.35
PC	22	9.09
ND	150	61.98
TS	169	69.83

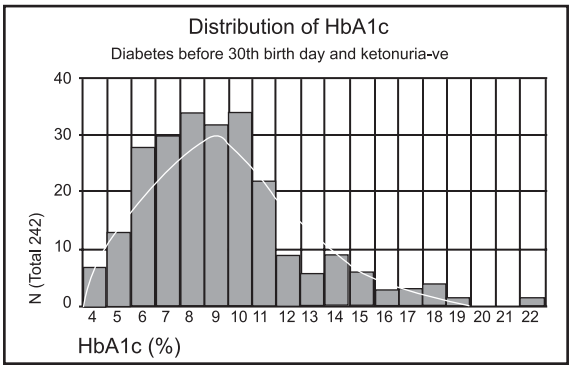

Fig-1. *Distribution of HbA1C among the subjects*

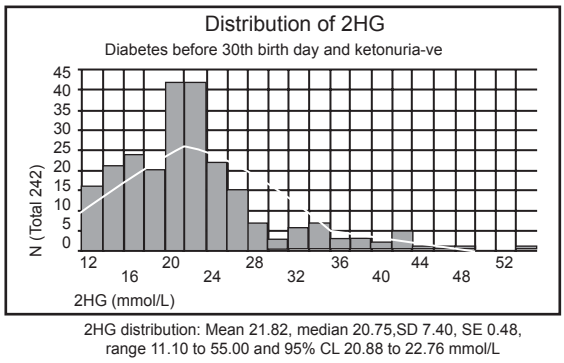

Fig-3 : *Distribution of blood glucose two hours after glucose load(2HG) among the subjects*

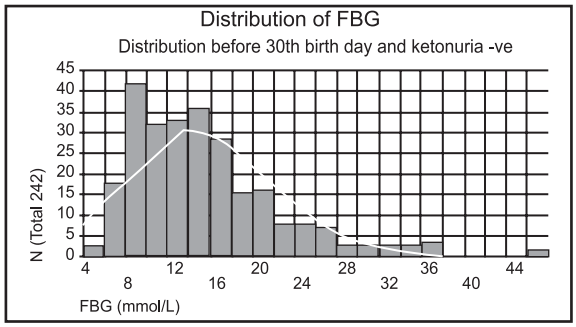

Fig-2 : *Distribution of fasting blood glucose (FBG) among the subjects*

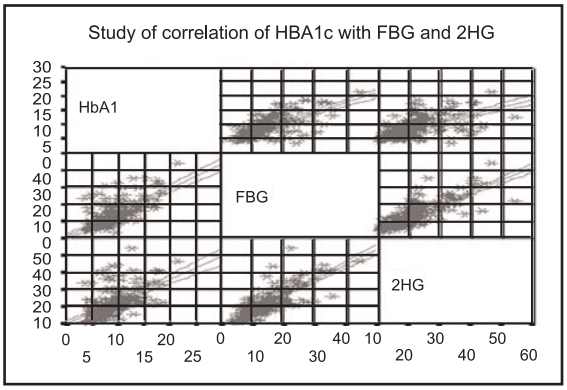

Fig-4 : *Correlation of HbA1c level with fasting blood glucose(FBG) and blood glucose levels two hours after glucose load(2HG)*

Table-IV

Study of diagnostic sensitivity

Evaluation of sensitivity of FBG(cut off 7.0 mmol/l),2HG cut off 11.1 mmol/L) and HbA1C(cut off 6.5%) as diagnostic tool of diabetes mellitus.(n-242)

Test	Diagnosis (+ve)	Diagnosis (- ve)	Total	Sensitivity (%)
FBG > 7.0 mmol/L	222	20	242	92
2HG > 11.1 mmol/L	242	0	242	100
HbA1C > 6.5%	206	36	242	85

Sensitivity of FBG (cut off 7.0 mmol/L), 2HG (cut off 11.1mmol/L) and HbA1C (cut off 6.5%) documented in 92%,100% and 85% respectively to diagnose DM.

Discussion:

Adult haemoglobin is heterogeneous, and in addition to unmodified haemoglobin (HbA0) there are minor components that are negatively charged-these are called HbA_1a, HbA_1b, and HbA_1c in order of their elution of ion-exchange chromatography[1]. Rah bar was the first to show in 1968 that these minor haemoglobins are elevated in diabetes. Since these are posttranslational modifications formed by the slow non-enzymatic attachment of glucose to haemoglobin over lifetime of the red cell, the degree of haemoglobin glycation can be used as an index of average glycaemia over the preceding weeks and months.Glycated haemoglobin (GHb), previously called 'glycosylated haemoglobin' and sometimes 'glycohaemoglobin' has been used for this purpose since 1970s and has been the cornerstone of assessment of glycaemia control in all major trials testing the links between control and complications, including the Diabetes Control and Complications Trial(DCCT) in type-1 diabetes and United Kingdom Prospective Diabetes Study(UKPDS) in type-2 diabetes. Three species of GHb are measured in clinical practice .HbA1c is the component present in largest amount (60-80%) and is often measured on it's own; it results from the attachment of glucose to the N-terminal amino acid valine of the B chain of haemoglobin. The monitoring of diabetic patients by evaluating glycated protein levels is now widely accepted and performed. HbA_1c is a glycated protein used to measure the integrated glycemic control in the preceding 2-3 months with extra weighting for preceding one month. There are more than 30 commercially available analytical methods for determination of glycated hemoglobin.Ion exchange chromatography, both low pressure and high performance liquid chromatography (HPLC) measures HbA1c; electrophoretic methods have been less used in recent years to measures HbA_1. The micro chromatographic version of the HPLC is the technique most frequently used in clinical practice[2]. Ito C et al in their study for prevalence of DM in population using $HbA_1c \geq 6.1\%$ has shown the association between HbA1c and FBG or 2hPG. High correlation were demonstrated among all the three measure FBG, 2hPG, HbA_1c[3]. In a study in Miyako island Japan among 2,621 health check-up participants, 34.9% of the subjects with newly diagnosed diabetes were identified by blood glucose (BG) alone and 33.% were diagnosed by HbA1C alone, combination of BG and HbA!c resulted in considerable increase in newly diagnosed diabetes cases.[4] A US study of using GHb(HbA1C) in screening undiagnosed diabetes concluded that GHb is a highly specific and convenient alternative to fasting plasma glucose for diabetes screening. A GHb value of 2 SD above the normal mean could identify a high proportion of individuals with undiagnosed diabetes who are at risk for developing diabetes complications[5]. Perry et al showed in a study that HbA1C measurement improves the detection of type-2 diabetes in high risk individual. They have shown that diagnosis based on FBG criteria are relatively less sensitive in detection of early type-2 diabetes in

high risk individuals.HbA1c measurement improves the sensitivity of screening in high-risk individuals[6] .All these studies support our findings of sensitivity HbA1c as initial test for detection of diabetes mellitus. In this study HbA_1c was shown to have 85% sensitivity (table-4).

HbA1c in the diagnosis of diabetes of young cases without spontaneous ketonuria showed positive correlation with blood glucose and a cut off at 6.5% was 85% sensitive to diagnose diabetes. So HbA1c can be used as a diagnostic tool with fairly good sensitivity in our young population.

References:

1. Allen DW, Schoroeder WA,Balog J.Observations on the chromatographic heterogeneity of normal adult and foetal human hemoglobin. Jam Chem Soc 1959;80:1628-34.
2. Pickup JC. Diabetic control and it's measurements. In :Pickup JC,Williums G,eds.Text book of diabetes.3rd edn. Oxford.Blackwell, 2003. p34.3-34.53.
3. Ito C, Maeda R , S,Sasaki H,Harada H . Correlation among fasting glucose, two hours plasma glucose level in OGTT and HbA1C. Diabetes Res Clin Pract 2000;50:225-30.
4. Takahshi Y, Noda M, Tsugane S, Kuzuya T, Ito C,kadowaki T.Prevalence of diabetes estimated by plasma glucose criteria combined with standered measurement of HbA1c among health check-up participants on Miyako island. Diabetes Care 2000; 23: 1092-6.
5. Rohlfing CL, Little RR, Wiedmeyer HM, England JD, Madsen R, Harris MI et al. Use of GHb(HbA1C) in screening for undiagnosed diabetes in US population. Diabetes Care 2000;23:187-91.
6. Perry RC, Shanker RR , Fineberg N, McGill J, Baron AD. Early Diabetes Intervention Program (EDIP). Diabetes Care 2001;24:465.

REVIEW ARTICLES

Angiotensin Converting Enzyme (ACE) inhibitors and Anogiotensin II Receptor Blockers (ARBs) in renal disease

S AHMED

Summary:

Chronic Kidney disease is emerging as a new health problem. Therapy with angiotensin converting enzyme inhibitors (ACEI) and angiotensin II receptor blockers (ARBs) have shown improvement in patients with hypertension, proteinuria and chronic renal failure. This improvement in GFR, fall in plasma renin activity (PRA) is due to improved sympathetic activity, improved endothelial function, reduced inflammation or combination of these factors. Several large scale, prospective randomized studies with clinical end point have strongly suggested that both ACEI or ARBs can slow progression of chronic glomerulonephritis alone. However, beneficial effect are much pronounced in combined group. Both ACEI & ARBs can prevent the progression from microalbuminuria to overt albuminuria in both type I and type II diabetes. When progression of renal disease is used as end point, protection has been demonstrated with ACEI for type I, but not type II diabetes. In type II only ARB have shown to slow progression to ESRD. Combination of ACEI and ARB is superior than maximum ACEI dose in type I diabetes. Whereas calcium channel blocker (CCB) and ARB combination is better option for type II diabetes. Patients treated with ACEI or ARBs should be monitored for hypertension, decrease GFR and hyperkalaemia. These two drugs should not be used in pregnancy for risk of foetal abnormality.

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Introduction:

Angiotensin converting enzyme inhibitors (ACEI) and angiotensin II receptor blockers (ARBs) are currently recommended for management and prevention of renal disease. In 1898, Tiegerstedt and Berg man found that crude saline extracts of kidney contained a pressor substance which is termed renin[1]. Renin exists in both inactive (prorenin) and active renin form. Active renin is a product primarily if not exclusively of the kidneys. Active renin is formed in the secretary granules of the juxtaglomerular cells and renin has a half life of 80 minutes or less in the circulation. Renin acts on the basic substrate angiotensinogen, circulating (a_2 globulin synthesized in liver to form the decapeptide angiotensin I. Angitensin I is then transformed to the octapeptide angiotensin II by angiotensin coverting enzyme (ACE). Angiotensin II cause deleterious effect such as vasoconstriction, salt retenion, inflammation, fibrosis and increased oxidative stress. Most of the converting enzyme that form angiotensin II in circulation is located in the endothelial cells particularly the pulmonary vascular endothelium. This angiotensin II was synthesined by Schwyer and Bumpus in 1957. Later, ACEI and ARB were developed in the year 1982 & 1988 respectively[2,3].

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Factors leading to progressive renal insufficiency

Patients with almost any form of renal disease are at risk of progressive decline renal functions over variable period of time. Risk factors are -

1) Persistent immune insult to glomerulus or disease gene abnormality
2) Coexisting modifiers of risks like infection, obstruction & drugs to the kidney
3) Systemic hypertension and glomerular capillary hypertension and hyperperfusion leading to enhanced internal traffic of protein & structural injury.
4) Proteinuria.
5) Low number of nephrons caused by congenital or acquired nephropathy,
6) Hyperlipidemia,
7) Metabolic factors like phosphate, calcium & urate depositon[4].

Systemic Hypertension

The mechanism by which systemic hypertension play role in progression of renal disease include injury to preglomerular arteries, leading to glomerular ischaemia with progressive luminal narrowing and hence fall in glomerular blood flow. Renal vessels and glomeruli appear susceptible to adverse effect of systemic hypertension when proteinuria or when there is glomerular inflammation and vascular damage. One of the cellular mechanisms of hypertensive renal injury is mediated by elevated angiotensin II[5].

Angiotensin II mediated renal injury

Angitensin II can be viewed as central molecule in several of the processes involved in chronic renal injury. When the number of nephrons is reduced to critical extent there is compensatory increase in renal plasma flow and increase in amount of filtrate in each remaining nephrons. Increase flow rates result from dilatation of afferent arteriole to a greater extent than efferent arteriole which is under the influence of angiotensin II. These changes increase glomerular capillary pressure with subsequent increase in single nephron glomerular filtration rate (GFR). This adaptive response to maintain GFR in the failing kidney may be maladaptive response in long term, cause renal functional and structural damage.

In kidney, local generation of anginotensin II is formed in excessive amount during haemodynamic injury to the endothelium and physical stretching of tuft. Angiotension II induced increased glomerular capillary pressure impairs the parmeselective selective function of glomerular filtration barriers leading to proteinuria and chronic parenchymal injury. Elevation in glomerular capillary pressure do enlarge the; c radius of the pores in glomerular membrane. Excessive angiotensin II formation or action may promote functional & structural injury. Other mechanism of injury include increase influx of macromolecule into mesangium, thereby inciting cytokines release, glomerular cell proliferation and macrophage influx and mesangial matrix formation. Angitensin II may directly induce expression of transforming growth factors B (TGF-B) responsible for extracellular matrix overproduction. Glomerular hypertensin is associated with change of endothelial cell function & structure which is potential cellular source of TGF-B and growth factor (platelet derived growth factor, PDGF) and basic roblast growth factor (BFGF).[6] Thus breach of the glomerular barrier to protein is perhaps most threatening consequences of increased glomerular capillary pressure. Glomerular hypertrophy which develops during adaptive response of the kidney to nephron loss precedes and may predispose to glomerular scarring. Therefore, angiotensin II is responsible for chronic renal injury by several mechanims. Angiotensin II is rapidly metabolized by various peptidases and half life is 1-2 minutes. Through several mechanisms, angiotensin II alter peripheral resistance, renal function and finally cardiovascular structure.

Physiology in RAS.

Juxtraglomerular apparatus (JGA) is located in vascular pole of glomerulus where distal tubule comes in contact with parent glomerulus. It has two components namely vascular & tubular component. Vascular component consists of terminal portion of afferent arteriole, initial portion of efferent arteriole and extraglomerular mesangial region. Tubular component consists of cells of distal tubule in contact with afferent arteriole, which become tall & columnar termed macula densa. Extraglomerular portion contain lucis cells bounded by macula densa, specialized region of afferent & efferent arteriole & intraglomerular mesangial cells. Myoepithelial cells or granular cells of afferent arteriole known as juxtraglomerular cells (JG cells) produce renin. The lucis cells, the JG cells and macula densa constitute JGA[7].

Control of Renin secretion

Renin secretion is regulated by two local pathway in the kidney and third one acts through central nervous system (CNS). First pathway of renin secretion is intrarenal mechanism by macula densa. Increased Nacl flux in macula densa inhibit and decrease Nacl flux which stimulate renin secretion. Second pathway for renin secretion is intrarenal baroreceptor pathway in which decreased tension in afferent arteriolar wall increase and increased tension decrease renin secretion.

Negative feedback of renin secretion

Increase renin secretion enhance the formation of angiotensin II and angiotensin II stimulates

angiotensin subtype I (AT I) receptors to juxtra glomerular cells to inhibit renin release. This mechanism has been termed short loop negative feedback mechanism. Agiotensin II induced increase blood pressure inhibit renin release by: a) activation of high pressure baroreceptor thereby reducing renal sympathetic tone b) Increased pressure in preglomerular vessel (c) reducing Nacl absorption in proximal tubule (pressure natriuresis) which increase tubular delivery of NaCl at macula densa. This is long loop negative feedback mechanism of renin secretion. Angiotensinogen is primarily synthesised in liver. Synthesis is stimulated by inflammation, estrogen, glucocorticoid, thyroid hormone, insulin, angiotensin II and in pregnancy. During pregnancy plasma level of angiotensinogen increase several fold due to estrogen[8].

Angiotensin converting enzyme (ACE):
This octoenzyme is located in endothelial cells, much abundant in lung and also in all other tissues[9]. ACE is identical to kinase which inactivate bradykinin and other potent vasodilators peptide.

Alternative pathway for angiotensin pathways
Some tissues contain nonrenin angiotensin processing enzymes that convert angiotensinogen to angiotensin I or directly to angiotensin II. Chymase possibly mast cell derived contributes to local tissue conversion of angiotensin I to angiotensin II particularly in heart and kidneys[10].

Local tissue RAS
Besides tradition circulating renin of renal origin, circulating angiotensinogen of hepatic origin, there are also extrinsic & intrinsic local renin angiotensin system found in brain, pituitary, blood vessel, heart, kidney and adrenal glands. This local production of angiotensin influences vascular, cardiac and renal function & structure[11].

Angiotensin II Receptors
The effect of angiotensins are exerted by two subtypes of receptors now designated AT, and AT_2 [12,13]. AT, receptor has high affinity for losartan and most biological effects are mediated through this receptor where as AT, has low affinity for losartan and poorly defined but may exert antiproliferative, proapoptotic and vasodilator effect.

Clinical Pharmacology of ACEI & ARBs

Currently 12 ACE inhibitors are approved for use (eleven marketed) include captropril, lisinopril, enalapril benzepril, fasinopril, moexipril, perindropril. FDA approved ARBs are candesartan cilexetil, irbesartan , lasartan, telmisartan, valsartan and eprosartan .

ACEI and ARBs in chronic kidney diseases in Adults

Chronic kidney disease (CKD) is defined as either kidney damage or GFR < 60ml/min 1.73 m^2 for ≥ 3 months. Kidney damage is defined as pathologic abnormalities or markers of damage, including abnormalities in blood or urine tests or imaging studies[14]. Adverse outcomes of chronic kidney disease can often be prevented or delayed through early detection and treatment. Earlier stages of chronic kidney disease can be detected through routine laboratory measurements. According to the K/DOQI CKD Classifications

Stages of chronic Kidney disease[14] are

Stage	Description	GFR ml/min/1.73 m^2
1	Kidney damage with normal or ?GFR	≥90
2	Kidney damage with mild ?GFR	60-89
3	Moderate ?GFR	30-59
4	Severe ?GFR	15-29
5	Kidney failure	<15 or dialysis

Mechanism of effects of ACE Inhibitors and ARBs to slow progression of CKD[15]

1. ACE inhibitors and ARBs reduce blood pressure
Hypertension accelerates the progression of kidney disease. The extent of blood pressure lowering correlates the activity of renin angiotensin system (RAS). ACEI and ARBs have greater antihypertensive effect in condition in which pressure is maintained through stimulation of RAS. By cotrast they have a lesser antihypertensive effect in condition in which pressure is maintained through ECF volume overload and concomitant suppression of RAS . CKD is associated with both stimulation of RAS and ECF volume overload. ACEI & ARBs reduce intraglomerular pressure as well as systemic blood

pressure which contributes to their beneficial effect of slowing the progression of kidney disease.

2. ACEI& ARBs reduce proteinuria:

Proteinuria is associated with faster progression of kidney disease. In controlled trial in CKD, ACE /< inhibitors & ARBs reduce proteinuria by approxiamtely 35 to 40 % which is greater than other antihypertensive agent. The nondihydropyridine agents such as verapamil and diltiazem have significant antiproteinuric effect in diabetic but not in nondiabetic kidney disease. The dihydropyridine agent such as amlodipine and nifidipine generally have no consistent effect on protein excretion. Both ACE inhibitors and ARBs reduce glomerular permeability barrier to proteins and limit proteinuria and filtered protein dependent inflammatory signals. The beneficial effects of ACE inhibitors on progression of kidney disease appears to be greater than expected due to their antiproteinuric effect.

3. ACEI & ARBs slow the progression of kidney disease by "class effect" mechanism in addition to antihypertersive and antiproteinuric effects.

The mechanism for this effects include (1) decrease glomerular intracapillary pressure (2) reduction in permselectivity (3) alteration in the function of mesangial cells (4) interfering with angiotensin mediated generation of free radical formation.

Adverse effect of ACEI & ARBs:

The incidence of adverse effects varies from 5 to 20%. Adverse effects are hypotension, worsening kidney function (acute renal failure), hyperkalaemia, cough, angioneurotic oedema, skin rash, neutropenia, agranulocytosis, fetal abnormalities.

Clinical Studies:

Meta analysis of randomised clinical trials suggest ACEI have substantial beneficial effect in delaying progression of renal disease and ACEIs are more effective than other antihypertensive agents. ACEI reduce urinary total protein and favourable impact on long term renal functions[16,17]. ARBs are alternative drugs inhibiting renin angiotensin system and has renoprotective activity. Observation of one study, use of ACEI and ARBs alone or in combination showed combination of drugs cause significantly a greater antiproteinuric effect. In this study losartan 25 mg and enalapril 10 mg was used alone and in combination on 17 patients in each group for 3 months with chronic glomerulonephritis (Ccr 36-93 ml/min). Reduction of proteinuria in losartan group is 25.35%, in enalapril group is 45.07% and enalapril + losartan group is 65.96% (P=0.0/09, combined group vs. losartan group). Treatment with losartan is associated with less fall in GFR. Decreased blood pressure was pronounced in combined group[18]. Observation from another study described fasinopril 20 mg/day, lrbesartan 150 mg /day and, combination of these two drugs in 3 groups of patients with chronic glomerulonephritis (Ccr 40-106 ml/min) was used. Preliminary results from after 6 weeks showed reduction of proteinuria is much greater in combined group[19]. Similar result was shown by another study that co-administration of losartan and enalapril exerts additive antiproteinuric effect in (IgA) nephropathy. Enalapril and losartan administration alone reduced proteinuria by same extent, but no further reduction when doses were doubled[20].

Chronic kidney disease (CKD) is emerging as a new health pandemic. Underlying the global rise in CKD is an increase in diabetic nephropathy which is the leading cause of end stage renal disease (ESRD). In terms of renal protection, there are ample data to support a role for both ACEI and ARBs to prevent the progression from microalbuminuria to overt albuminuria in both type I and type II diabetes. However, when progression of renal disease is used as an end point, protection has been demonstrated with ACEI only in type I but not for type II diabetes. In the later group, only ARB have been shown to slow progression of ESRD[21]. Cardiovascular protection effect of ACEI in high risk population is widely appreciated. Most head to head comparisons between ACEI and ARB have yeilded comparable cardiovascular protective effect with ARBs being associated fewer side effects [21].

Losartan (angiotensin II antagonist) has an antihypertensive effect equivalent to ACE inhibitors, however its role in microvascular complication is not yet known. However, in one study showed losartan remarkably improves albuminuria, benefit in autonomic or peripheral neuropathy in normotensive type II diabetes seen over 12 weeks[22]. Several clinical trials have established the benefits of ACEI & ARBs

in patients with diabetes. ACEI have shown to delay renal decline in patients with type I diabetes, whereas protective effect with type II diabetes is less clear. The ARBs have shown to provide significant benefits with type II diabetes, both early (microalbuminuria) and late (proteinuria) stages of renal decline. In the Irbesartan Diabetic Nephropathy Trial (IDNT) and the reduction of end points in NIDDM with the angiotensin II antagonist losartan(RENNAL) study, ARB therapy significantly reduced the progression of overt nephropathy (composite of doubling of serum creatinine, ESRD and death) benefit that has not been shown for ACE inhibitor.

Addition of ARB with maximized ACE inhibitor

Prolonged angiotensin coverting enzyme ACE inhibitors therapy lead to angiotensin I accumulation which may escape ACE inhibition generate angiotensin II, stimulate angiotensin II subtype AT, receptor and exerts deleterious renal effects in patient with chronic renal disease like vasoconstriction, salt retension, inflammation and fibrosis and enhance the activity of central & peripheral sympathetic activity. Furthermore, pathway other than ACE may be responsible for angiotensin II generation particularly in tissues of blood vessels. ARBs can overcome these shortcomings of ACEI by antagonizing the AT1 receptors.

In addition, because AT2 receptor mediate the beneficial effects of angiotensin II, by blocking ATI receptors with ARBs, angiotensin II would be available to stimulate the AT2 receptors[24].

Combination treatment with ACEI & ARBs safely retards progression of non diabetic renal disease compared with monotherapy of each drug at its maximum dose[25]. The benefit of combination therapy of its antiproteinuric effect was different between 1gA and diabetic nephropathy over the 12 weeks trial. 24 hours urinary total protein excretion rate was significantly reduced by combination therapy in patients with IgA nephropathy but no reduction with diabetic nephropathy with combination therapy[26] however, one study showed dual blockade of renin angiotensin system is superior to maximal recommended dose of ACE inhibitions with regard to lowering of albuminuria and blood pressure in type I patients with diabetic nephropathy. Another study suggests that combined antihypertensive therapy with either a calcium channel blocker (CCB) plus an ARB or an ACEI plus ARB exerts an antiproteinuric effect in patients with type II diabetic nephropathy with mild renal impairment. ACEI and ARB combination had more profound effect, but it was associated with increase in serum potassium concentration and worsening of renal anaemia. Thus, combination of CCB and ARB should be first line for antihypertensive therapy in those overt type 2 diabetic nephropathy[28]. To sumerise the combination therapy ACEI & ARBs showed benefical effect in both non diabetic and type I diabetic nephropathy patients. In type II diabetic nephropathy ARB plus CCB combination is better option of combination treatment.

Patient treated with ACE inhibitor & ARBs

Patient treated with ACEI & ARBs should be monitored for hypertension, decrease GFR and hyperkalaemia[15]. At initiation and increase in dose of ACE inhibition or ARB, the level of blood pressure, GFR and serum potassium should be measured to establish a "baseline" or "new baseline". Transient abrupt decrease in blood pressure occur in about 2.5% of patients. Clinician should be cautious to lower systolic blood pressure below 110 mm Hg. Causes of hypotension in CKD are excessive dose of antihypertensive agents, extracellular fluid depletion (diuretics), cardiovascular diseases (myocardial infarction, heart failure, arrhythmia, valvular disease, pericarditis), neurological diseases, liver disease, haemorrhage & sepsis[15].

An early decrease in GFR is defined as a decrease in GFR by more than 15% from baseline within 4 weeks after initiation of ACEI or ARB. The reported incidence varies from 4% to 17% and most common causes are ECF volume depletion, excessive dose of ACEIs or ARBs and concomitant use of diuretics or NSAIDs[15]. If GFR decreases by more than 30% over base line, the dose of ACEI or ARB may be reduced and GFR reassessed. If GFR does not return to baseline within appropriate interval ACEI or ARBs should be discontinued with alternative antihypertensive agents. Hyperkalaemia is defined when single measurement of serum potassium > 5 mEq/1, > 6 mEq/L, or persistant or single increase of

0.5 mEq/1 above baseline. Reported incidence of hyperkalemia ranges from 1% to 62.5% of patients. Causes of hyperkalaemia in CKD are food, acidosis, hyperglycaemia, hyperproteinemic hyperaldosteronism, oliguria and drugs[15].

Conclusion

Chronic kidney disease is emerging as new health pandemic. Emerging evidences strongly suggest that inhibition of renin-angiotensin-aldosterone system confers significant renal and cardioprotection for patient with CKD. Several large scale, prospective randomized studies with clinical end point have been performed with ACEI or ARBs alone or in combination both in non diabetic and diabetic renal disease.

Both ACEls or ARBs can slow progression of chronic glomerulonephritis alone. However, recent studies suggest beneficial effect are much pronounced in combined group. Both ACEI & ARB can prevent the progression from microalbuminuria to overt albuminuria in both type I and type II diabetes. However when progression of renal disease is used as an end point, protection has been demonstrated with ACEI for Type 1 not Type II diabetes. In type II only ARB have been shown to slow progression to ESRD. Combination of ACEI and ARB is superior than maximum ACEI dose in Type I diabetes. Whereas calcium channel blocker (CCB) and ARB combination is better option for Type II diabetes. Patients treated with ACEI or ARB should be monitored for hypertension, decreased GFR and hyperkalaemia. These two drugs should not be used in pregnancy for risk of foetal abnormality.

References:

1. Jackson E.K. Renin and angiotensin, In Joel C. Hardman, Lee E Lionbird (eds) Goodman & Gilman's The Pharmacologcal basis of Therapeutics. Tenth ed, New York, Chicago, 2001, p. 809.
2. Furakawa, Y, Kistiomoto S, Nishikawa. Hypotensive imidazole derivatives and hypotensive imidazole 5 acetic acid derivatives, patents issued to Takeda chemical indurtries Ltd on July 20, 1982 and October 19, 1982, respectively, US. patents 4340598 and 4355040. Osaka, Japan, 1982.
3. Carini. D, Duncia, JV. Angiotensin II receptor blocking imidazoles. European patent application 0253310, 1988. Page ---
4. Abbate M, Remuzzi G. Progression of renal insufficiency. In Massry SG, Glassock RJ (eds), Massry and Glassock's Text book of Nephrology, 4th ed. Lippincott Williams & Wilkins, Philadelphia, 2001, p. 1210.
5. Remuzzi G. Bertani T. Pathophysiology of progressive nephropathies N. Engl. J. Med. 1998; 339 : 1448-1456.
6. Ketteler M, Noble NA, Border WA. Transforming growth factor beta and angiotensin II, the missing link from glomerular hyperfiltration to glomerulosclerosis. Annu Rev. Physiol. 1995; 57 : 279-295.
7. Madsen KM, Aisher CC. Anatomy of the Kidney, Brenner BM & Rector FC (eds), The kidney, 4th ed, WB Saunders, Philadelphia, 2004, p15-19.
8. Compbell DJ, Habener JF. Angiotensinogen gene is expressed and differentially regulated in multiple tissue of rat. J. din. Invest. 1986; 78 : 31-39.
9. Soubrier F, Alhenc Gelas F, Hubert C et. al. Two putative active centres in human angiotensin I converting enzyme revealed by molecular cloning. Proc. Natl. Acad. Sci. USA, 1988; 85 ; 9386-9390.
10. Akasu M, Urata H, Kinoshita A. Difference in tissue angiotensin II forming pathways by species and organs in vitro. Hypertension, 1998 ; 32 : 514-520.
11. Danser AHJD, Koning MMG, Admiraol PJJ. et al.Production of angiotensins I and II at tissue sites in intact pigs. Am. J. Physiol, 1992; 263 : H427-H42a.
12. Sasaki K, Yamano Y, Bardhan S. Cloning and expression of a complementary DNA encoding an bovine adrenal angiotensin I type receptor. Nature 1991; 351 : 230-233.
13. Mukoyama M, Nakajima M, Horiuchi M. Expression cloning of type 2 angiotensin II receptor reveal unique class of seven - transmembrane receptors. J. Biol. Chem. 1993; 268 : 24539-24542.
14. Clinical Practice Guidelines for Chronic Kidney disease : Evaluation, classification and stratification. Am. J. Kidney Dis. : 2002 ; 39 (2), S17-31.
15. K/DOQ1. Clinical Practice Guidelines on hypertension and Antihypertensive agents in chronic kidney diseases. Guideline II : use of angiotensin converting enzyme inhibitors and angiotensive receptor blockers in CKD. Internet. www. kidney.Org.
16. Remuzzi G, Tognoni G. Randomised placebo-controlled trial of effect of ramipril on decline in glomerular filtrate rate and risk of terminal renal failure in proteinuric, non-diabetic nephropathy. The Lancet. 1997; 349 : 185763.
17. Giatras I, Lan J. Levey A Effect of Angiotensin Converting Enzyme Inhibitors on progression of nondiabetic renal disease : A meta analysis of randomized trails. Ann. Intern. Med. 1997; 127 : 337-345.
18. Jylickil, Rutkowski P, Renke M, Rutkowskib. Renoprotective effects of small doses of lo sartan and enalapril patients with primary glomerulonephritis. Am. J. Nephrol. 2002; 24(4) : 356-362

19. Fessai, Paolo, Mati, Hams-Peter. Additive antiproteinuric effect of combined ACE inhibition and angiotensin Il receptor blockade. J. Hypertens. 2002; 20(1) : 125-130.

20. Russo D, Minutolo R, Pisani A. Coadministration of losartan and enalapril exerts additive antiproteinuric effect in 1gA nephrophathy. Am. J. Kidney Dis. 2001; 38 (1) : 1825.

21. Berl T. Angiotensin Converting enzyme inhibitors versus AT, receptors antagonist in cardiovascular and renal protection : the case for AT, receptor antagonist. J. Am. Soc. Nephrol. 20041 15 : S71 6.

22. Kubbu S. Agarwal Sk. Prakash A. Effect of losartan on albuminuria, peripheral and autonomic neuropathy in normotensive microalbuminuric type 2 diabetes. Neurol India, 2003; 15 (3) : 3558.

23. Raij L. Recommendations for the management of special populations : renal disease in diabetes. Am. J. Hypertension. 2003; 16 : S46-S49.

24. Agarwal R. Add on angiotensin receptor blockade with maximized ACE inhibition. Kidney Intern. 2001; 59 : 2282-2289.

25. Nakao N. Yoshimura A, Mortia H. Combination treatment of angiotensin II receptor blocker and angiotensin converting enzyme inhibitor in non diabetic renal disease (COOPERATE) : a randomised controlled trial. Lancet 2003 ; 361(9352) : 117-124.

26. Kim MJ, Song J.H, Suh JH. Additive antiproteinuric effect of combination therapy with ACE inhibitator and angiotensin II receptor antagonist differential short term response in IgA nephropathy and diabetic nephropathy. Yonsei M.J. 2003 ; 44 (3) : 463-472.

27. Jacobsen P. Anderson S. Rossing K. Dual blockade of the renin-angiotensin system versus maximal recommended dose of ACE' inhibition in diabetic nephropathy. Kidney Int. 2003 ; 63 (5) : 1874-1880.

28. Kuriyama S. Tomonari H. Takudome G. Antiproteinuric effect of combined antihypertensive therapies, in patients with overt type 2 diabetic nephropathy. Hypertens Res. 2000; 25 (6) : 849-855.

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Pulmonary Valvuloplasty: Analysis of Fifteen Cases

NN FATEMA

Summary :

Fifteen cases of severe pulmonary valve stenosis (PS) had undergone cardiac catheterization and pulmonary valvuloplasty in Cardiac Catheterization Laboratory (CCL) of Combined Military Hospital (CMH) Dhaka from July 2001 to December 2002. 14 cases (93.33%) had excellent result and only one case had unsatisfactory result. In that case valvuloplasty was done on three occasions and each time she developed severe stenosis 3-6 months within the procedure. Now she is waiting for surgery.

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Introduction :

Pulmonary valve stenosis (PS) is a condition where pulmonary valve cannot open normally. Isolated pulmonary valve stenosis is a relatively common anomaly, with a prevalence estimated as high as 10 percent of all congenital heart disease[1-2]. Majority of patients with valvar pulmonary stenosis are asymptomatic and discovered only during routine examination[3]. Mild stenosis usually improves with growth but severe stenosis often becomes worse[3]. Balloon valvuloplasty is the non-surgical approach for treatment which was first described in 1979[4]. The technique is relatively straight forward and the result is excellent[5-6]. In this study 15 cases of severe pulmonary valve stenosis were treated with balloon valvuloplasty with excellent out come.

Materials and Methods :

This is a retrospective study carried out in the cardiac catheterization laboratory (CCL) of Combined Military Hospital (CMH) Dhaka over a period of 30 months from July 2001 to December 2002. Total twentyeight cases were prepared for pulmonary valvuloplasty but 13 cases were postponed after cardiac catheterization because of presence of subvalvular stenosis along with valvular PS or dysplastic pulmonary valve. The inclusion criteria for the patients were (a) severe or critical pulmonary valve stenosis (b) isolated valvular stenosis without any association with subvalvular or supravalvular stenosis (c) Absence of other cardiac lesions which necessitates open-heart surgery. Patients with dysplastic pulmonary valve and mild to moderate pulmonary stenosis were excluded from the study. Patients who had mild pulmonary valve stenosis had pressure gradient of < 40 mmHg across pulmonary valve and those who had moderate stenosis had gradient of 40 – 60 mmHg across the pulmonary valve. Patient with severe pulmonary stenosis had peak pressure gradient (PPG) of >60 mmHg across the pulmonary valve. Patient with critical pulmonary stenosis had severe pulmonary stenosis along with serious symptoms. The 15 cases who had pulmonary valvuloplasty were referred to the paediatric cardiology out patient department by the paediatricians. Out of 15 cases 8 cases had incidental findings of murmur when they reported to the paediatricians for other reasons. Five cases had exertional dyspnoea, one had chest pain and another one with critical PS had cyanosis.

Before cardiac catheterization complete workup of the patients were done. Chest X-ray, electrocardiogram (ECG) and Doppler echocardiography was done and patients who fulfilled the selection criteria were selected for cardiac catheterization. After cardiac catheterization 13 cases were excluded and 15 were finally selected for valvuloplasty on the same sitting. Sizes of the balloons were selected after measuring the pulmonary valve annulus from the Right Ventricular (RV) angiogram in lateral view. After valvuloplasty patients were kept in observation for 24 hours and then they were discharged with an appointment for echocardiography and follow up at 1, 3, 6, 12, 18, 24 months interval and yearly thereafter. So far follow up upto 6 months has been completed in all cases.

Procedure

Equipments:

Balloon – weight, age and pulmonary valve annulus determined the size of balloons.

Guide wire – super stiff guide wire was used.

Preparation of balloon – balloons were prepared by aspiration and flushing. The air inside the balloon and vent were replaced with contrast solution. Dilution of 1:8 of omnipaque 350 and saline used. Right ventricular (RV) angiogram was performed first to see pulmonary valve annulus.

Procedure – Exchange wire was inserted through Goodale Lubin (GL) catheter and advanced into distal

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pulmonary vessels. Catheter and sheath were removed together keeping the wire in position. The prepped balloon was inserted over the wire. Balloon was advanced to the pulmonary valve. Coordination with wire was maintained to prevent arrhythmia and damage to the distal pulmonary vessels. The balloon was inflated quickly. Inflation was stopped when the waist of the balloon disappeared. Gentle traction was given to keep the balloon in the annulus. The balloon then quickly deflated and withdrawn slightly to allow perfusion. Procedure was repeated 2-3 times before removing balloon. Balloon was then withdrawn and catheter reintroduced to pulmonary artery over the guide wire along with sheath. Withdrawal gradient was recorded across pulmonary valve and RV angiogram repeated to see the result. All the patients had out patient appointment at regular interval to see the result of the procedure.

Result :

Out of fifteen patients ten were female and five were male. Age varied from 26 days to 25 years. Right ventricular (RV) pressure was <100 mmHg in 8 cases and it was 100 mmHg or above in 7 cases. Pressure gradient across pulmonary valve was between 60 to 80 mmHg in eight cases. Pressure gradient was >80 mmHg in rest 7 cases. In 10 cases pressure gradient across pulmonary valve dropped to 30 mmHg or less after valvuloplasty. In other 5 cases pressure gradient was more than 30 mmHg (table I). Table II showed symptoms on presentation. Most of the patients (53.33%) were asymptomatic. Table III showed out come of treatment. In all 15 cases immediate result was satisfactory but 6 months follow up showed restenosis in one case. Results were considered satisfactory when pressure gradient across pulmonary valve was reduced to 40 mmHg or less. One patient who developed restenosis at 6 months follow up was a neonate with critical PS. Her valvuloplasty was performed as a life saving intervention and immediate result was good. This procedure has saved her life at that time when surgery was not possible on her. Out of 15 cases 14 had peak pressure gradient of <40 mmHg in echocardiography at 6 months follow up and one had PPG >60 mmHg across pulmonary valve.

Table-I

Patient data and analysis. n= 15

Serial No	Age	Sex	Pressure inside right ventricle	Pulmonary stenosis gradient	Pulmonary valve annulus	Balloon size gradient	Post balloon
01	9½ yrs	F	75 mmHg	60 mmHg	16.5 mm	18x4	4 mmHg
02	8 yrs	F	85 mmHg	75 mmHg	17 mm	18x3	15 mmHg
03	16 yrs	F	85 mmHg	65 mmHg	15.5 mm	18x3	26 mmHg
04	7 yrs	M	75 mmHg	55 mmHg	13 mm	15x4	20 mmHg
05	3 yrs	F	80 mmHg	62 mmHg	13 mm	15x4	20 mmHg
06	3 yrs	F	160 mmHg	144 mmHg	10 mm	12x4	35 mmHg
07	5 yrs	M	110 mmHg	88 mmHg	9.5 mm	12x4	No gradient
08	25 yrs	M	80 mmHg	60 mmHg	16.5 mm	18x3	35 mmHg
09	3 months	F	76 mmHg	55 mmHg	12 mm	15x4	30 mmHg
10	8yrs	M	200 mmHg	180 mmHg	13 mm	15x4	38 mmHg
11	1½ yrs	F	105 mmHg	85 mmHg	13 mm	15x4	20 mmHg
12	26 days	F	102 mmHg	88 mmHg	7 mm	8x2	135 mmHg
13	1 yr	F	84 mmHg	70 mmHg	12 mm	14x3	30 mmHg
14	2 yrs	F	115 mmHg	96 mmHg	10 mm	12x3	20 mmHg
15	2 8 mo	M	100 mmHg	82 mmHg	13 mm	15x4	35 mmHg

Table II

Symptoms on presentation: n = 15

Symptoms	No of patient	Percentage
Asymptomatic	08	53.33
Exertional dysponea	05	33.33
Cyanosis (mild)	01	6.64
Chest pain	01	6.64

Table III

Outcome of treatment: n = 15

Outcome	Immediate	6 months after procedure
Satisfactory	15 (100%)	14 (93.33%)
Not satisfactory	0	1 (6.64%)

Note: Satisfactory result means PPG of 40 mmHg or less across pulmonary valve after valvuloplasty. Immediate pressure gradients were recorded in the cath lab after valvuloplasty.

Discussion

Balloon valvuloplasty for congenital pulmonary valvular stenosis is a safe and effective procedure and it is the initial treatment of choice[7]. Outcome for balloon valvuloplasty for critical PS in newborn babies is also excellent. One study conducted in Taiwan showed 79% definitive success of balloon valvuloplasty in neonates with critical PS[8]. In our study we had one patient with critical PS. She developed recurrent pulmonary stenosis and after repeating the procedure three times, she was referred for surgical valvotomy. Immediate result was satisfactory in this baby in every attempt but she developed restenosis within 6 months of the procedure. This procedure saved her life in every occasion when she was severely symptomatic and surgery was not available for her in any cardiac centre of Bangladesh. Sex distribution of patient is about equal, but in our study female were predominant (table I). Growth and development in patient with pulmonary stenosis is usually normal[3]. We had not seen any cases of growth failure in our study. Symptoms are rare in infants, with a notable exception in those with critical pulmonary stenosis[9]. In this study one patient had critical PS and she prsented with cyanosis in neonatal period. Most of our patients (53.33%) were asymptomatic (table II). Although immediate and intermediate term results after balloon dilatation of congenital stenotic lesions of the heart in children are well studied, long term rsults have not been documented[10]. In our study, follow up echocardiography at 6 months showed good result in 93.33% cases (table III). One study conducted in Osaka, Japan showed decrease of mean pressure gradient immediately after balloon valvuloplasty (BVP) from 61± 27 mmHg to 28 ± 20 mmHg and the reduced gradient continued at follow up in most cases[7]. We did only one adult valvuloplasty in this study who was 25 years old and he had excellent result till 6 months follow up. One study conducted on 34 cases of adult showed good medium term result[11]. Late result was shown good in another study in adult[12]. In our series no complications were encountered. But complications like acute pulmonary oedema, significant pulmonary valve incompetence following oversized pulmonary balloon valvuloplasty is quite common[13-14]. A long term follow up study in USA showed, six out of 107 consecutive patients undergone balloon valvuloplasty developed increasing pulmonary valve incompetence during follow up period of 0.5 to 10 years (mean 7.2years). In our series none of the patient had developed pulmonary valve incompetence.

The outcome of balloon valvuloplasty for critical pulmonary stenosis in young infants were studied in National Taiwan University Hospital, Taipei[15]. Out of 34 infants, procedure was accomplished in 28 patients and failed in six who subsequently required surgery. The study concluded that balloon valvuloplasty is the procedure of choice for critical pulmonary stenosis[15]. Another study conducted in King Faisal Specialist Hospital & Research Centre, Riyadh, KSA showed that phentolamine can improves clinical out come after balloon valvuloplasty in neonates with severe pulmonary stenosis[16].

Conclusion :

It will take time to get long-term result on our patients. Short term (6 months) follow up with Doppler echocardiography showed excellent outcome in all except one though immediate result was good in all cases. The baby who had restenosis, also got the benefit of the procedure which saved her life for time

being and gave her a chance to travel abroad later for surgical valvotomy. It may be concluded that balloon valvuloplasty is the procedure of choice for severe and critical pulmonary valve stenosis. Surgery should be reserved for those with unsuccessful balloon valvuloplasty.

References

1. Abrahams DG, Wood P. Pulmonary stenosis with normal aortic root. Br. Heart J 1951; 13 : 519
2. Campbell M. Simple pulmonary stenosis, Pulmonary stenosis with a closed ventricular septum. Br. Heart. J 1954; 16 : 273.
3. Albert P. Rocchini MD, George C. Emmanouilides MD. Pulmonary stenosis in: GC Emmanouilides, Hugh D Allen, Thomas A. Riernen Schneider, Hower P. Gutgessel, (editors). Moss and Adams Heart disease in infant children and adolescents, Fifth edition. Baltimore: William and wilkins, 1995. PP 930 - 960.
4. Semb BHK, Tjonneland S, stake G, Aaby holm G. "Balloon valvulotomy" of congenital pulmonary valve stenosis with tricuspid valve insufficiency. Cardiovasc Radiol 1979; 2 : 239 - 241.
5. Kan JS, White RI, Mitchell SE, Gardner TJ. Percutaneous balloon valvuloplasty: a new method for treating congenital pulmonary valve stenosis. N Engl J Med 1982; 307 : 540 - 542.
6. Rocchini AP, Kveselis DA, Crowley D, Dick M, Rosenthal A. Percutaneous balloon valvuloplasty for treatment of congenital pulmonary valve stenosis in children. J Am coll cardiol 1984; 3 : 1005 - 1012.
7. Echigo - S. Balloon valvuloplasty for congenital heart disease: Immediate and long term results of multi institutional study. Pediatr - Int. 2001; 43 (5) : 542 - 7.
8. Wang JK, Wu MH, Lee W-L, Cheng CF, Lue H-C. Balloon dilatation of critical pulmonary stenosis. Int -J - cardiol 1999; 69 (1) : 27 - 32.
9. Hanley FL, Sade RM, Freedom RM, Blackstone EH, Kirklin JW. Outcome in critically ill neonates with pulmonary stenosis and intact ventricular septum: A multi institutional study. J Am coll cardiol 1993; 23 : 183 - 192.
10. Roa PS. Long term follow up results after balloon dilatation of pulmonic stenosis, aortic stenosis and coarctation of aorta: a review. Preg cardiovasc Dis. 1999; 42 (1) : 59 - 74.
11. Bhatia A, Bhatia V, Batra J, Mahajan D-S, Batra K-S. Congenital valvular aortic and pulmonary stenosis. J Assoc-Physicians India 1998; 46 (6) : 566 - 7.
12. S A Sheikholeslami, F, Firoozi I, Azarnik, H. Late results of balloon pulmonary valvuloplasty in adults. AM J cardiol. 1998; 82 (3) : 398 - 400.
13. Walker CP, Beteman CJ, Rigby ML, Brookes CI. Acute pulmonary oedemia after percutaneous balloon valvuloplasty for pulmonary valve stenosis. J cardiothorac vase Anesth 2001; 15 (4) : 480 - 2.
14. Berman W, Fripp R R, Raisher BD, Yabek S M.Pulmonary valve incompetence following pulmonary valvoplasty. Catheter cardiovasc interv 1999; 48 (1) : 61 - 5.
15. Wang JK; Wu MH; LeeWL; Cheng CF; Lue HC. Balloon dilatation for critical pulmonary stenosis. Int. J Cardiol, 1999; 69(1) : 27-32.
16. Galal O, Kalloghlian A, Pittappilly BM, Dzimiri N. Phentolarnine improves clinical outcome after balloon valvoplasty in neonates with severe pulmonary stenosis. Cardiol - Young 1999; 9 (2) : 127-8.

CASE REPORTS

Rhino Cerebral Mucormycosis – A Case Report

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Summary

A 42-year-old man who was on long term steroid therapy for bone marrow failure developed left orbital swelling and blindness along with prolonged fever. Neurological examination revealed proptosis of the left eyeball with complete ptosis and periorbital edema. There was ulceration of both lips with crust formation. There was total ophthalmoplegia with dilated pupil on left side. There was impairment of sensation over the distribution of trigeminal nerve. Fundoscopy showed papilloedema on the left side. Left sided orbital debridement and exenteration was done. The pathological diagnosis was mucormycosis. Systemic administration of amphotericin B was started immediately. On 25th day of starting therapy the patient suddenly started to deteriorate and died on the next day possibly due to rapid extension of the disease. Rhino cerebral mucormycosis is a rapidly progressive fatal disease. Successful treatment seems to be based on early diagnosis, control of underlying disease, radical surgical resection, and systemic administration of amphotericin B. Mucormycosis should be considered as differential diagnosis of orbital cellulitis or orbital apex syndrome.

(J Bangladesh Coll Phys Surg 2004; 22 : 115-118)

Introduction:

Mucormycosis (Zygomycoosis) is an opportunistic fungal infection caused by filamentous fungus of the order mucorale,[1-2] the rhino-cerebral being the commonest one. The site of onset is the nasal mucosa and the disease extends rapidly to the palate, orbit and brain. Risk factors for mucormycosis include hematological malignancy, namely leukemia and lymphoma, diabetes mellitus especially those with ketoacidosis[2]. The use of steroids and immunosuppressive agents in bone marrow and solid organ transplantation[3], broad-spectrum antibiotics and cytotoxic chemotherapy, and dialysis for uraemic patients, particularly with desferrioxamine therapy, are also known predisposing causes[1,4-5]. Thus, the infection is often an indication of a serious predisposing condition. Malnutrition in children in developing countries also carries a risk of mucormycosis with gastrointestinal involvement, and cerebral mucormycosis has been reported in intravenous drug user via the hematogenous route[5]. Mucormycosis has very high mortality rate of at least 50%[6]. Pulmonary and gastrointestinal disease has an even higher mortality rate due to late diagnosis. Lethal mucormycosis has been described in an otherwise healthy man[7-9]. Here, a case of rhino-cerebral mucormycosis in a patient on steroid therapy for bone marrow failure is presented. Possibly this is the second case reported from Bangladesh[10].

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Case Report:

A 42 year old machine man of a daily newspaper weighing 60 kg presented with progressively increasing fever, anorexia and weakness for 15 days. He was treated with a 10 days course of ciprofloxacin along with antipyretics without any response. When he was hospitalized he was found moderately anaemic. There was no lympadenopathy. Liver and spleen were not palpable. Systemic examination revealed no abnormal findings. Laboratory examinations revealed: Hemoglobin 8 gm/dl, ESR 135 mm in first hour, WBC count 1000/cumm, with neutrophil 26% and lymphocyte 68%, platelet count 60000/cumm, and peripheral blood film showing pancytopenia. He was not diabetic and fasting and post prandial blood sugar were 5.6 and 7.9 mmol/l respectively. His liver and kidney function tests were within normal limits. Urine analysis revealed no abnormality and blood culture revealed no growth. Bone marrow study revealed dry tab. Later on trephine biopsy of bone marrow showed evidences of progressive marrow failure. He was started Inj. Dexamethasone 2mg IV six hourly along with Inj. Ceftriaxone and Inj. Gentamicin. Five units of blood were transfused.

After two weeks when the patient was transferred to Bangabandhu Sheikh Mujib Medical University Hospital he was still febrile but complaining of discharge from nose that was initially clear but later becoming blackish. Subsequently, he started to feel mild pain in left eye with dimness of vision. Very shortly he developed swelling of left orbit and completely lost his vision. He was also complaining of loss of sensation over left side of face. He didn't loose consciousness. He was found mildly anaemic, febrile (temperature 39^0C), non-icteric and there was no significant lymphadenopathy. His thyroid and testes were normal.

There was proptosis of the left eyeball with complete ptosis and periorbital edema (Figure-I). Both the lips were ulcerated with crust formation. There was complete ophthalmoplegia with dilated pupil on the left side. There was impairment of sensation over the distribution of trigeminal nerve. Fundoscopy showed dilated fixed pupil, diffuse retinal edema, and multiple hemorrhagic spots in the fundus associated with papilloedema on the left side. The patient was fully conscious and well oriented. Examination of nose and nasopharynx under general anesthesia revealed red and necrotic turbinate with extensive crusting over the left nasal cavity. There was no evidence of nasopharyngeal carcinoma. After removal of the crust, tissue under the crust was taken for histopathological and mycological study. MRI of the brain showed left-sided proptosis with ethmoidal sinusitis. CT scan of nose and paranasal sinuses showed evidence of inflammatory lesions involving left retro-orbit, left side of the face, and left maxillary, ethmoidal and cavernous sinus. During this period of illness he was on steroid in various doses and routes; and he was taking prednisolone 30 mg daily when he was hospitalized. After admission in the hospital his hematological profile was as follows: WBC count 12,000/cu mm with neurtrophil 90% and lymphocyte 08%, hemoglobin 13 gm/dl, ESR in first hour 05 mm, platelet count 200 x 10^9/l, peripheral blood film showing neutrophilic leuocytosis. Isotope bone scan was normal. Fasting and postprandial blood sugar, liver function and renal function tests were within normal limits. Histopathology of tissue obtained from nasal mucosa showed fungi with broad aseptate hyphae, branching at right angles and forming terminal spore (Fig.-2). These were also present within blood vessels (Fig.-3). Culture of the tissue in Sabourd's Agar (SDA) media yielded growth of mucor on the frothy day which was further confirmed under microscope (Figure 4). Injection Amphotericin B 100 mg mixed with 500ml of dextrose in aqua per day was started. A team of otolaryngologist, ophthalmologist and neurosurgeon did surgical exploration. All the sinuses were cleared of the crusts. Left-sided orbital debridement and exenteration was done and ethmoidal sinus was also removed (Figure 5). The tissue obtained also showed the presence of the fungi, characteristics of mucor both on culture and histologically. After starting the antifungal therapy the patient was improving well. He started to feel sensation in the left side of face. The drug showed no side effects. His blood count, renal and hepatic functions were also

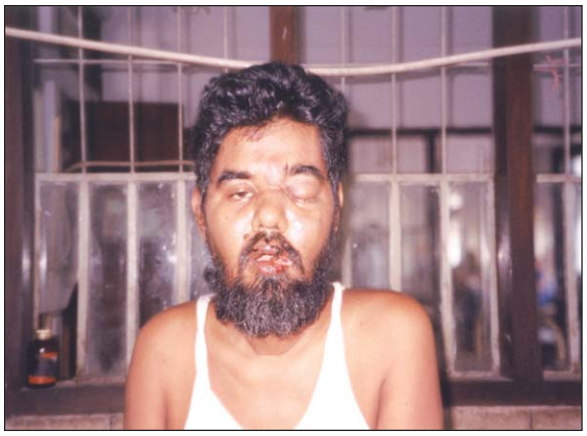

Fig.-1 : *Clinical view of the patient's face*

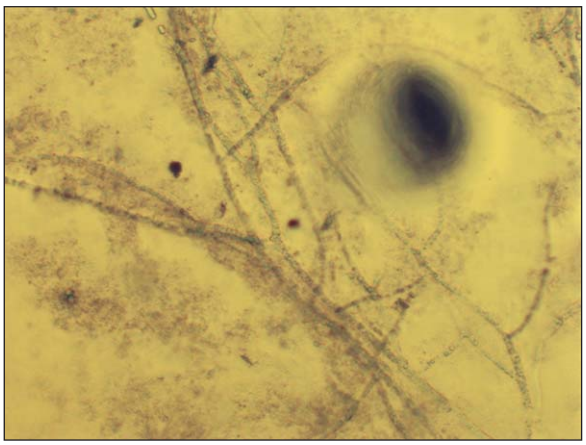

Fig.-2 : *Nasal tissue stained with lactophenol blue showing aseptate ribbon like branching hypae many are at right angle.*

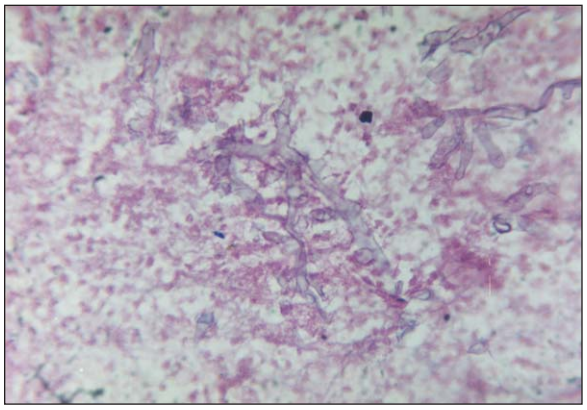

Fig.-3 : *Hypae invading the blood vessel*

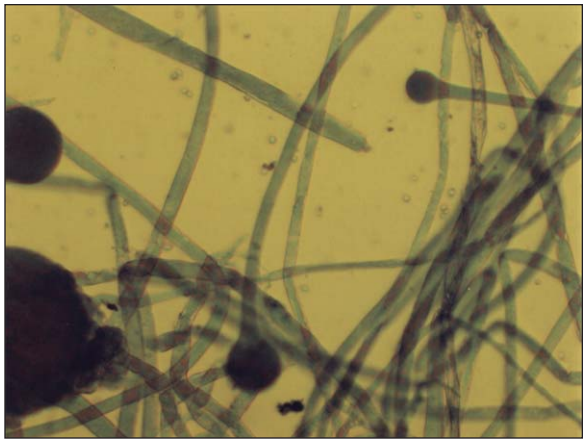

Fig.-4 : *Growth of mucor in Sabourd's Agar Media and stained in lactophenol blue.*

Fig.-5 : *Necrotic tissue material after debridement.*

within normal limits. On the 25th day of starting Amphotericin the patient started to deteriorate with acute respiratory distress. At that time his ECG, serum CPK, serum electrolytes were within normal limits. Emergency chest X-ray PA view showed patchy infiltrate on both lung fields. Arterial blood gas analysis showed mild hypoaxemia with normal carbon-dioxide. Parenteral Ceftriaxone 2 gm was given. On the following day the patient expired.

Discussion:

Mucormycosis has growing importance because of the increasing population of immunocompromised patients. Due to the high mortality and morbidity rates of the fungal infection in this group of patients, a high index of suspicion is warranted in the relevant clinical situation. Early diagnosis by an aggressive investigative approach to obtain tissue biopsy and early surgical intervention, combined with systemic antifungal therapy will optimize the treatment outcome[11]. Correction of the underlying risk factors is essential to achieve a cure. This patient suffered from rhino-cerebral mucormycosis, commonly referred as mucormycosis, the infection spreading from the left maxillary and ethmoidal sinus to the left orbit. The term rhino-cerebral indicate sinus involvement but does not always means that central nervous system involvement has occurred[12]. The immunodeficiency caused by prolonged steroid therapy on the setting of progressive marrow failure favored the growth of such opportunistic fungus in this case. Mucormycosis can present either as craniofacial mucormycosis which manifest as low grade fever later turning to high grade, dull sinus pain, nasal congestion with thin bloody nasal discharge, and unilateral generalized reduction of ocular motion, chemosis and proptosis. Invasion of globe and ophthalmic artery may lead to blindness. Haematogenous spread may occur to brain or lung or other organs. It may affect the gastrointestinal system causing multiple ulcers, which may perforate. This opportunistic mucor commonly attack patients suffering from severely debilitating disease like uncontrolled diabetes mellitus, patient with organ transplantation taking immunosuppressive drug, hematological malignancy, steroid therapy, uraemia, or receiving long term desferrioxamine therapy. Its presentation can be confused with those of sinusitis, carotid or cavernous sinus thrombosis or even viral infection and is often missed at early presentation[13-14].

Classical predisposing factors, typical clinical pictures and isolation of the specific mucor fungus

from the smear and histopathological section of the tissue are sufficient for diagnosis of the case. If any patient presents with the above features at the background of immunosuppression, as in this case, mucormycosis should be suspected strongly. Amphotericin B is the most reliable antifungal agent that is effective against mucormycosis. The usual recommended dose is 1 mg/kg/day, but a higher dose of 1.5 mg/kg/day was used for the reported case because of the serious infection[15]. The duration of treatment is three to six weeks. But the patient died on the 25th day of starting therapy, even after withdrawal of steroid much earlier, surgical debridement and good hematological condition.

Mucormycosis has a very high mortality rate. There is no report of survivors of mucormycosis before the availability of Amphotericin B in 1950s. Mortality rate among the patients with invasive sinus disease without cerebral involvement may be as high as 50-80%. If infection spreads to the brain case fatality rate may exceed 80%[12]. Deaths in this case in spite of early diagnosis and institution of Amphotericine B in higher doses might be due to extension of the disease due to inadequate debridement. Ferry et al described 11 deaths out of 16 after treatment with surgical debridement along with intravenous Amphotericin B[16].

So, a high index of suspicion is needed, in appropriate clinical settings, to diagnose and aggressively treat mucormycosis in view of the high mortality rate for susceptible patients.

References:

1. Sugar AM. Mucormycosis. Clin Infect Dis 1992; 14(Suppl 1): 126S-9S
2. Lehrer RI, Howard DH, Sypherd PS, Edwards JE, Segal GP, Winston DJ. Mucormycosis. Ann Intern Med 1980; 93 : 93-108
3. Boelaert JR. Mucormycosis (zygomycosis): is there news for the clinician? J Infect 1994; 28(suppl 1) : 1S-6S
4. McNab AA, Mckelvie P. Iron overload is a risk factor for zygomycosis. Arch Ophthalmol 1997; 115 : 919-21.
5. Abe F, Inaba H, Katoh, T, Hotchi M. Effects of iron and desferrioxamine on Rhizopus infection. Mycopathologia 1990; 110 : 87-91.
6. Eisen D. Mucormycosis. htts: www.emedicine.com med tosic1513.htm
7. Castelli JB, Pallin JL. Lethal rhinocerebral phycomycosis in a healthy adult: a case report and review of the literature. Otolaryngology 1978; 86 : 696-703.
8. Bhattacharyya AK, Deshpande AR, Nayak SR, Kirtane MV, Ingle MV, Vora IM. Rhinocerebral mucormycosis: an unusual case presentation. J Laryngol Otol 1992; 106 : 48-9.
9. Sharma RR, Pawar SJ, Delmendo A, Lad SD, Athale SD. Fatal rhino-orbito-cerebral mucormycosis in an apparently normal host: case report and literature review. J Clin Neurosci 2001; 8 : 583-6.
10. Rafiqueuddin AKM, Ahasan HAMN, Chowdhury MAJ, Quashem A, Kundu NC. Mucormycosis (zygomycosis)- case report. Trop Doct 1994; 24 : 41.
11. Forteza G, Burgeno M, Martorell V, Sierra I. Rhinocerebral mucormycosis. Presentation of two cases and review of the literature. J Craniomaxillofac Surg 19881; 16 : 80-4.
12. Earhart KC, Baugh WP. Rhinocerebral Mucormycosis. http://www.emedicine.com/med/topic2026.htm
13. Hendrickson RG, Olshaker J, Duckett O. Rhinocerebral mucormycosis: a case of a rare, but deadly disease. J Emerg Med 1999; 17 : 641-5.
14. Onyango JF, Kayima JK, Owen WO. Rhinocerebral mucormycosis: case report. East Afr Med J 2002; 79 : 390-3.
15. Yeung CK, Cheng VCC, Lie AKW, Yuen KY. Invasive disease due to Mucorales: a case report and review of the litarature. HKMJ 2001; 7 : 180-8
16. Ferry AP, Abedi S. Diagnosis and management of rhino-orbitocerebral mucormycosis (phycomycosis). A report of 16 personally observed cases. Ophthalmology 1983; 90 : 1096-04.

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Post Traumatic Female Urethral Stricture : A Case Report with Review of Current Literature

MZH BHUIYAN[a], M SIRAJ[b], MF ISLAM[c], KMH TAWHID[d],

Summary :

A girl of nine years age presented with a suprapubic catheter and she had been with it for two years. Immediately after an accidental blunt abdominal trauma, she was managed by laparotomy, bowel repair, enterostomy and suprapubic cystostomy (SPC). After closure of enterostomy, it was tried many times to re-establish the patent lower urinary tract. She refused to go to school as she was ashamed of her body image. After multiple procedures over a span of six months, it was possible to provide a catheter free life to the girl.

(J Bangladesh Coll Phys Surg 2004; 22 : 119-121)

Introduction :

Historically, post-traumatic female urethral stricture has been considered virtually a non existent condition[1]. In recent years, more cases of female urethral trauma have been reported and the estimated incidence in association with pelvic fracture is 4.6 to 6%[2]. The rarity of female urethral injury with pelvic fracture can be attributed to its relative mobility, short length and the absence of rigid attachment to the pubic bone[3]. Accurate pre-operative diagnosis could be possible only in 50% of cases[4]. Initial minor voiding symptom, easy catheterization and overall low index of suspicion is the main cause to overlook or delay in diagnosis[1]. The delay in diagnosis or its management significantly increases the morbidity.

Case Report : A girl of nine years age with history of blunt abdominal trauma two years back, was referred to Urology department of Bangladesh Medical College Hospital, Dhaka. She was treated before as a case of acute abdomen with pelvic fracture and soft tissue injury of vulva and perineum. She underwent emergency laparotomy to repair rectal injury, protective proximal sigmoid colostomy, SPC and perineal wound toileting. She recovered from her injury but removal of suprapubic catheter was not possible because of post-traumatic complete urethral stricture. After colostomy closure and stabilization of pelvic fracture, she was referred to the department of Urology. As per record she was evaluated thoroughly; diagnosed as a case of post-traumatic complete proximal urethral stricture with associated malunited pelvic fracture, distal urethro-vaginal fistula (Figure–1). It was attempted many times to re-establish normal urethral passage. The parents were counselled regarding the delayed management of post-traumatic complete female urethral stricture, the outcome of which is completely different to that of male. The parents were afraid of incontinence of their daughter following urethral reconstruction. Her parents consented after counselling and the girl was admitted under our care in Bangladesh Medical College Hospital, Dhaka. She was evaluated under anaesthesia (EUA) with colposcopy, simultaneous rigid paediatric cystoscopy, urethroscopy and flexible cystoscopy through suprapubic cystostomy route. It was seen that there was complete proximal urethral

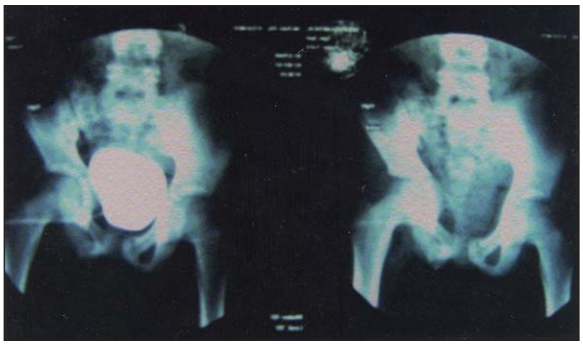

Fig. – I : *Pre-operative antegrade cystogram, showing healed mal united pelvic fracture, Urinary bladder is well outlined and no contrast material has passed through the urethra.*

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stricture more then 1 cm in length with immediate distal urethro-vaginal fistula. The region of internal urethral meatus was totally smooth and it was not well identified. "Core-through visual internal urethrotomy'' was not possible either due to failure of identification of the internal urethral meatus accurately through suprapubic flexible cystoscope or the Xenon cold light may have been inadequate to pass through long dense fibrous urethral stricture segment. In the second procedure the urethro-vaginal fistula was repaired through vaginal route preceded by an episiotomy like incision. Finally the urinary bladder was explored adequately. The land mark of internal urethral meatus was totally absent, the region was smooth and epithelialised. It was identified with the help of other important landmark like ureteric orifices & trigone and was confirmed by urethral dilator. A circular disk of thick tissue about 1.5×1.5 cm was excised with electosurgery unit to reconstruct the internal urethral meatus. Vesico-urethral continuity was then established (OIU) with the help of a paediatric urethrotome. The neo-urethral passage was then calibrated upto 16 Fr. after keeping a safety wire in situ. Finally, otis urethrotomy (20 Fr.) was done in 3 and 9'o clock position and the urethra was stented with a 12 Fr. Foly's catheter. After three weeks a second look urethrocystoscopy was done to observe and confirm a satisfactorily epithelialised urethra and the bladder neck. Otis urethrotomy was repeated in the same session and the urethral catheter was continued for another 3 weeks. After a prolonged period of complete inactivity the urinary bladder was given full training supported with uro-selective anti-cholinergic drug (oxybutanine hydrochloride). It required two weeks to achieve almost its normal capacity, to reduce nocturnal frequency, to ensure a sound sleep at night. Before making her catheter free the importance and effectiveness of intermittent self dilation (ISD) of urethra in preventing the recurrence of urethral stricture was counselled to both the patient and the parents. Day after removal of catheter ISD was demonstrated to the mother and it has been ensured in every follow-up. At the third month of ISD, the post-operative outcome appeared excellent confirmed by uroflowmetry and contrast imaging study (retrograde cystogram; Figure–2 and micturating cysto-urethrogram (Figure– 3).

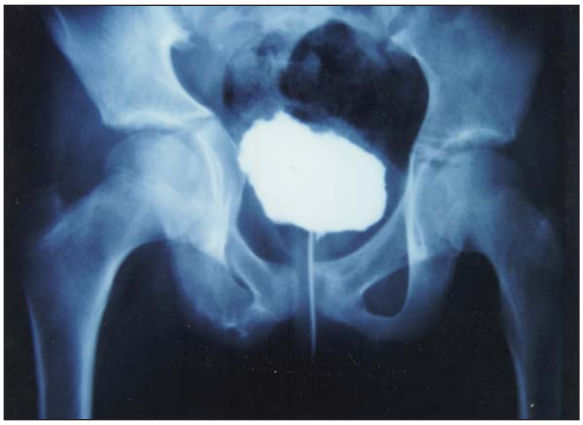

Fig. – 2 : *Post-operative retrograde cystogram showing healed mal-united pelvic fracture, contrast material is in the urethral catheter and urinary bladder is well outlined.*

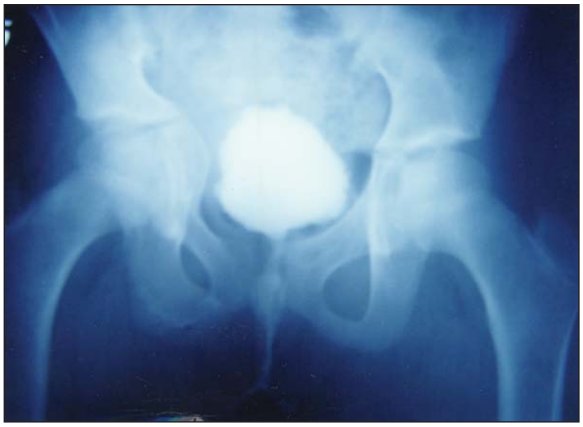

Fig. – 3 : *Post-operative micturating cystourethrogram showing healed mal-united pelvic fracture, urinary bladder is well outlined and there is free passage of contrast material through the urethra.*

Discussion :

As late as 1965 female urethral injuries associated with pelvic fracture for all practical purposes were nonexistent[2]. Literature review had shown that till 1980 only 30 cases were reported[5]. In recent years more cases of female pelvic fracture with associated urethral injuries are reported. The excellent modern acute care facilities have resulted in increased survival of severely injured patients and is causing an apparent increase in the incidence of this previously rare entity[4]. Both male and female urethral injuries in association with pelvic fracture may results from following causes [9].

Upward displacement of hemi-pelvis specially pubic rami resulting traction injury to the anterior and posterior urethral ligaments.

Sudden diastases of pubic symphysis damaging the posterior pubourethral ligament and urogenital diaphragm.

Bony spicule causing direct trauma to the urethra.

Overall, the risk of post-traumatic urethral injury is greatly influenced by sex, age and the types of pelvic fracture[6]. The incidence is 4.6 to 6%[4] in case of female and 9.9%[7] in case of male. High risk pelvic fracture and incidence of urethral injury is common in younger age group than in older patients[6]. Partial female urethral injury is most common, (at 12'o clock position in variable length) but the complete urethral injury involves the bladder neck and proximal urethral region, and is not seen to involve distal 5 mm[4, 8] .

The low incidence in female is due to short urethral length, relative increase mobility as well as absence of rigid attachment of it to pubic bone as in male[2]. Obviously pelvic fracture as they are intimately related to urethral injuries may be classified as no risk group :– isolated fractures of acetabulam, ilium and sacrum, low risk group :– single or ipsilateral ischiopubic rami fracture, high risk group :– straddle and Malgaigne fractures[2].

Accurate clinical diagnosis of female urethral injury may be looked over in 50% of cases[4] specially when the patient gets multiple injuries that require urgent attention for other critical problem. In case of incomplete urethral injury which is more common, initial catheterization may be easier. So, the gynaecologists promptly repair the vaginal injury while the need for examination of urinary tract or need for urological consultation is not given adequate attention. A high index of suspicion is mandatory for the diagnosis of female urethral injury when there is pelvic fracture. Inability to void, hematuria, urethral bleeding, vaginal bleeding and laceration, labial oedema are common presenting features. Normal voiding and continence may be intact when urethral injury is in distal to external sphincter[2].

The diagnostic reliability of the contrast urethrography in case of partial urethral longitudinal tear is still controversial. The double balloon urethrography (one outside the urethra another inside the bladder) proved to be beneficial[1]. However, the role of urethroscopy is generally recommended[4].

The management options differ in male and female urethral injuries. For proximal urethral injury gold standard management in male is immediate suprapubic cystostomy and delayed urethral reconstruction[1]. The similar procedure in female urethral injury may lead to urethrovaginal fistula, urethral stenosis or complete urethral stricture. The delayed reconstruction presents a practical problem[4] as in this reported situation. For proximal urethral injuries immediate urethral re-alignment around a stent through suprapubic route is recommended. Vaginal injury is to repaired simultaneously to prevent future urethrovaginal fistula or vaginal stenosis. Distal urethral injury is approached through vaginal route and the aim of management is to maintain adequate external meatus around a stent with or without proximal urethral advancement[3].

In this case it was initially managed by general surgeons and major attention was given to laparotomy and management of bowel injury. Urethral injury was managed by suprapubic cystostomy. Post-traumatic female urethral injury is rare in Bangladesh. It is not surprising that practical experience in this regard is limited even among the urologist. The girl was referred to urologist after complete recovery following staged management of bowel injury. The morbidity of delayed presentation of post-traumatic female urethral injury is well documented in international literature. Similar morbidity was also seen in this case.

References :

1. Lev RY, Mor Y, Golomb J, Leibovitch I and Ramon J. Missed female urethral injury complicated by myonecrosis of the thigh. J Urol 2001; 165:1216.
2. Koraitim MM. Pelvic fracture urethral injuries : The unresolved controversy. J Urol 1999; 161 : 1433-41.
3. Pokorny M, Pontes JE and Pierce JM Jr. Urological injuries associated with pelvic trauma. J Urol 1979; 121 : 455.
4. Perry MO, Husmann DA. Urethral injuries in female subjects following pelvic fractures. J Urol 1992; 147 : 139.
5. Petil U, Nesbitt R and Meyer R. Genitourinary tract injuries due to fracture of the pelvis in females : sequelae and their management. BJU 1982; 54 : 32.
6. Koraitim MM, Morzouk ME, Atta MA and Orabi SS. Risk factors and mechanism of urethral injury in pelvic fracture. BJU 1996; 71 : 876.
7. Wilkinson FOW. Rupture of the posterior urethra. Lancet 1961; 1 : 125.
8. Pode D and Shapiro A. Traumatic avulsion of female urethra : a case report. J Trauma 1990; 30 : 235.

COLLEGE NEWS

(J Bangladesh Coll Phys Surg 2004; 22 : 122-126

EXAMINATION NEWS :

FCP'S Part I, FCPS Part II and MCPS Examinations of July, 2004 were held as scheduled. Result were announced in time. 3053 candidates appeared in FCPS Part - I Examination of July, 2004, of which 1033 candidates came out successful. Subject-wise results are as follows :

FCPS PART I EXAMINATION

Subject	No of candidates appeared	Passed
Medicine	956	363
Surgery	571	285
Paediatrics	338	110
Obst. & Gynae	660	102
Ophthalmology	82	35
Otolaryngology	76	25
Psychiatry	16	03
Anaesthesiology	61	23
Radiology	66	15
Radiotherapy	15	04
Dermatology & Venereology	73	36
Physical Medicine	19	11
Dental Surgery	51	13
Family Medicine	04	O1
Haematology	28	05
Biochemistry	05	-
Microbiology	17	02
Histopathology	15	-
Total	3053	1033

385 candidates appeared in FCPS Part II Examination in different subjects. Candidates who satisfied the board of examiners are as follows:

Roll No.	Name of candidate	Graduated from	Speciality
002	Dr. Mohammad Jashim Uddin	MAG osmani Medical College	Medicine
005	Dr. Md. Nur-A-Alom Khan	Rajshahi Medical College	Medicine
007	Dr. Rajib Nayan Chowdhury	Mymensingh Medical College	Medicine
011	Dr. Harendra Nath Sarker	Sher-e-Barngla Medical College	Medicine
013	Dr. Manash Saha	Dhaka Medical College	Medicine

Roll No.	Name of candidate	Graduated from	Speciality
023	Dr. Wasim Md. Mohosin Ul Haque	Sir Salimullah Medical College	Medicine
035	Dr. Md. Shahabul Huda Chowdhury	Chittagong Medical College	Medicine
038	Dr. Md. Abdur Rouf	Chittagong Medical College	Medicine
045	Dr. Md. Ismail Chowdhury	Dhaka Medical College	Medicine
064	Dr. Mukhlesur Rahmarn	Dhaka Medical College	Medicine
065	Dr. Rajaslush Chakrabortty	Dhaka Medical College	Medicine
066	Dr. Mohammad Abdullah Al Hasan	MAG Osmani Medical College	Medicine
068	Dr. Mohammad Kumruzzaman Sarker	Chittagong Medical College	Medicine
071	Dr. Iqbal Murshed Kabir	Dhaka Medical College	Medicine
074	Dr. Abdul Ahad Mohammed Ryhan Uddin	USTC. Chittagong	Medicine
076	Dr. Abdul Momen	Sir Salimullah Medical College	Medicine
078	Dr. Md. Imrul Hasan	Mymensingh Medical College	Medicine
080	Dr. A.K.M. Shaheen Ahmed	MAG Osmani Medical College	Medicine
082	Dr. A.K.M. Monwarul Islam	Dhaka Medical College	Medicine
093	Dr. S. M. Mizanur Rahman	Mymensingh Medical College	Medicine
102	Dr. S.A.M.M.A. Hafiz	Chittagong Medical College	Medicine
103	Dr. Mir Jakib Hossain	Chittagong Medical College	Medicine
104	Dr. Mohammad Mamunur Rashid	Sir Salimullah Medical College	Surgery
105	Dr. Mohd. Abdus Samad Azad	Chittagong Medical College	Surgery
106	Dr. Md. Mashiur Arefin	Sir Salimullah Medical College	Surgery
107	Dr. A.S.M. Zahidur Rahman	Khulna Medical College	Surgery
108	Dr. Md. Anowar Hossain	Rangpur Medical College	Surgery
113	Dr. Md. MUstafi zur Rahman	Sher-e-Bangla Medical College	Surgery
116	Dr. Deh Prosad Paul	Khulna Medical College	Surgery
132	Dr. Md. Aminul Islam	Rangpur Medical College	Surgery
134	Dr. Md. Fazal Naser	Dhaka Medical Colelge	Surgery
138	Dr. Abul Kheire Mohammed Minhai Uddin Bhuiyan	Mymensingh Medical College	Surgery
139	Dr. Prosenjit Sanyal	Rajshahi Medical College	Surgery
143	Dr. Ahmad Seraji	Dhaka Medical College	Surgery
144	Dr. Mohammad Ibrahim Khalil	Chittagong Medical College	Surgery
145	Dr. Md. Monirul Ahsan	Sher-e-Bangla Medical College	Surgery
146	Dr. Abu Hassan Md. Rafiqul Bari	Sher-e-Bangla Medical College	Surgery
152	Dr. Md. Mohsin Uddin	Dhaka Medical College	Surgery
154	Dr. Md. Farruque Hossain	Rajshahi Medical College	Surgery
160	Dr. Samiron Kumar Mondal	Rajshahi Medical College	Surgery
161	Dr. Shamim Hassan	Dhaka Medical College	Surgery
163	Dr. Mohammad Akram Hossain	Mymensingh Medical College	Surgery

Roll No.	Name of candidate	Graduated from	Speciality
173	Dr. Md. Shahidul Islam	Dhaka Medical College	Surgery
174	Dr. Md. Nasir Uddin	Rajshahi Medical College	Surgery
176	Dr. Mahbuba Begum	Dhaka Medical College	Surgery
180	Dr. Swapan Kumar Biswas	Sir Salimullah Medical College	Surgery
181	Dr. Subinoy Krishna Paul	Mymensingh Medical College	Surgery
196	Dr. Md. Habibur Rahman	Rajshahi Medical College	Surgery
199	Dr. Md. Mizanur Rahman	Sher-e-Bangla Medical College	Paediatrics
201	Dr. Mohammad Imrul Kayes	Chittagong Medical College	Paediatrics
207	Dr. Md. Rukunuzzaman	Mymensingh Medical College	Paediatrics
211	Dr. Sanat Kumar Barua	MAG Osmani Medical College	Paediatrics
214	Dr. Rukhsana parvin	Dhaka Medical College	Paediatrics
232	Dr. Md. Khoybar All	Dhaka Medical College	Paediatrics
233	Dr. Md. Ibrahim Khalil	Sir Salimullah Medical College	Paediatrics
251	Dr. Mohammad Imnul Islam	MAG Osmani Medical College	Paediatrics
252	Dr. Khaleda Khanam	Sher-e-Bangla Medical College	Obstetrics & Gynaecology
255	Dr. Tahmina Begum	Mymensingh Medical College	Obstetrics & Gynaecology
259	Dr. Humaira Alam	Mymensingh Medical College	Obstetrics & Gynaecology
260	Dr. Nadira Khan	Sir Salimullah Medical College	Obstetrics & Gynaecology
261	Dr. Ayesha Rahim	MAG Osmani Medical College	Obstetrics & Gynaecology
262	Dr. Ismatara Bina	USSR	Obstetrics & Gynaecology
266	Dr. Nasima Shaheen	Mymensingh Medical College	Obstetrics & Gynaecology
173	Dr. Syeda Ummay Kulsum	Mymensingh Medical College	Obstetrics & Gynaecology
276	Dr. Atashi Saha	Sher-e-Bangla Medical College	Obstetrics & Gynaecology
285	Dr. Sharif Masuma Ismat	Sher-e-Bangla Medical College	Obstetrics & Gynaecology
287	Dr. Hosna Zari Tahmina	Rajshahi Medical College	Obstetrics & Gynaecology
292	Dr. Florida Rahman	Sir Salimullah Medical College	Obstetrics & Gynaecology
296	Dr. Musarrat Suoltana	Dhaka Medical College	Obstetrics & Gynaecology
298	Dr. Nargis Sultana	MAG Osmani Medical College	Obstetrics & Gynaecology
299	Dr. Sartaj Begum	MAG Osmani Medical College	Obstetrics & Gynaecology
306	Dr. Saika Shaheed	Mymensingh Medical College	Obstetrics & Gynaecology
309	Dr. Aziza Begum	Rangpur Medical College	Obstetrics & Gynaecology
312	Dr. Salina Akhter	Mymensingh Medical College	Obstetrics & Gynaecology
314	Dr. Fahmida Khan	Dhaka Medical College	Obstetrics & Gynaecology
316	Dr. Moshammat Zebunnesa	Rangpur Medical College	Obstetrics & Gynaecology
317	Dr. Masuda Islam Khan	Mymensingh Medical College	Obstetrics & Gynaecology
319	Dr. Shila Rani Das	Dhaka Medical College	Obstetrics & Gynaecology
320	Dr. Saila Parvin	Dhaka Medical College	Obstetrics & Gynaecology
321	Dr. Shila Sen	Mymensingh Medical College	Obstetrics & Gynaecology

Roll No.	Name of candidate	Graduated from	Speciality
328	Dr. Sulfikar Hasan	Dhaka Medical College	Ophthalmology
332	Dr. Md. Ismail Hossain	Mymensingh Medical College	Ophthalmology
337	Dr. Muhammmad Ziaul Karim	Mymensingh Medical College	Ophthalmology
340	Dr. Abul Kalam Azad	Rajshahi Medical College	Ophthalmology
341	Dr. Md. Arif Hossain Bhuyan	Mymensingh Medical College	Otolaryngology
350	Dr. Kabir Ahmed	MAG Osmam Medical College	Otolaryngology
351	Dr. Md. Saiduzaman	IPGMR	Otolaryngology
362	Dr. Chowdhury Md. Ikramul Latif	MAG Osmani Medical College	Psychiatry
365	Dr. Shahnaz Afroza	Mymensingh Medical College	Anaesthesiology
367	Dr. Begum Marjan Mohol Choudhury	MAG Osmani Medical College	Anaesthesiology
369	Dr. Shyama Prosad Mitra	Rajshahi Medical College	Anaesthesiology
371	Dr. Md. Abdul Kader	Mymensingh Medical College	Anaesthesiology
378	Dr. Md. Enayet Karim	Sir Salimullah Medical College	Radiology
379	Dr. Md. lqbal Hossain	Mymensingh Medical College	Radiology
381	Dr. Md. Nasir Uddin	Sir Salimullah Medical College	Radiology
384	Dr. Md. Abul Kashem Chy.	Chittagong Medical College	Dermatology & Venereology
386	Dr. Md. Abdul Latif Khan	IPGMR	Dermatology & Venereology
389	Dr. Mohammed Yousuf	Chittagong Medical College	Physical Medicine
393	Dr. Md. Salahuddin Shah	Mymensingh Medical College	Haematology

203 candidates appeared in MCPS Examinations in different subjects. Candidates who satisfied the board of examiners is as follows.

Roll No.	Name of candidate	Speciality
008	Dr. Salauddin Mamun Chowdhury	Medicine
013	Dr. Debashis Kumar Gupta	Medicine
019	Dr. Zahir Uddin Mahmud Illius	Medicine
040	Dr. Dewan Azmal Hussain	Medicine
041	Dr. Md. Mahbubul Alam	Medicine
050	Dr. Mahbub Murshed	Surgery
054	Dr. Proshanta Kumar Biswas	Surgery
060	Dr. Qazi Mahbub-Ul-Alam	Surgery
080	Dr. Shahara Haque	Paediatrics
082	Dr. Md. Nazrul Islam	Paediatrics
085	Dr. Zakia Afroze	Obstetrics & Gynaecology
093	Dr. Mahmuda Begum	Obstetrics & Gynaecology
095	Dr. Mina Humayun Kabir	Obstetrics & Gynaecology
107	Dr. Jamila Khatun	Obstetrics & Gynaecology
108	Dr. Suchanda Das	Obstetrics & Gynaecology

Roll No.	Name of candidate	Speciality
110	Dr. Qamar Naz Banu	Obstetrics & Gynaecology
1 13	Dr. Mst. Mamataz Begum	Obstetrics & Gynaecology
1 15	Dr. Ruma Sen Gupta	Obstetrics & Gynaecology
121	Dr. Kamala Kanta Barman	Obstetrics & Gynaecology
129	Dr. Shirin Akhter	Obstetrics & Gynaecology
130	Dr. Kazi Farzana Huq	Obstetrics & Gynaecology
136	Dr. Afroza Khan	Obstetrics & Gynaecology
140	Dr. Asifa Ali	Obstetrics & Gynaecology
146	Dr. Syed Muhammed Tipu Sultan	Otolaryngology
151	Dr. Ratan Chowdhury	Otolaryngology
160	Dr. Md. Shafiqul Islam	Anaesthesiology
165	Dr. Md. Shafiqul Islam	Anaesthesiology
168	Dr. Manisha Paul	Anaesthesiology
173	Dr. Mohammad Abdul Karim Miah	Anaesthesiology
175	Dr. Md. Jahid Hossain	Anaesthesiology
193	Dr. Mushtaq Ahmed	Forensic Medicine
194	Dr. Kh. Abdul Karim	Forensic Medicine
201	Dr. Md. Golam Rasul	Family Medicine
208	Dr. Shahana Begum	Clinical Pathology
210	Dr. Md. Abul Hossain Miah	Clinical Pathology
211	Dr. Jarina Begum	Clinical Pathology
215	Dr. Md. Abdul Mazid Sardar	Clinical Pathology