ISSN: 2226-7964

PRIME MEDICAL JOURNAL

Vol. 6 No: 2 July-2016

An official organ of Prime Medical College

ISSN: 2226-7964

PRIME MEDICAL JOURNAL

Vol-6 No: 2 July - 2016

ISSN 2226-7964

Printed at:
Prime printers
Prime Medical College Campus , Rangpur.

Request for re-print:
Editor-in-Chief
Prof. Dr. Nawazesh Farid
Prime Medical College
Pirjabad, Badargonj RoadRangpur.
Cell: 01819119577
E-mail : pmcrang@gmail.com

Informations about Prime Medical College, Rangpur

Prime Medical College is one of the best and largest private medical college in Bangladesh. It was established in 2008. The ideas of establishing this Medical College is to provide standard Medical Education and Health Services to the people at an affordable cost.

The objectives of the institute are :

- To promote and provide education in Medical Science and to Provide training in different discipline of medicine recognized by the postgraduate institutes and universities.
- To conduct research work on the diseases prevalent in the country.
- To conduct research on medical education with the aim of uplifting the quality and standard of medical education in the country.
- To produce and provide skilled manpower in the medical, nursing and paramedical fields.
- To provide quality medical care and heath services to the people at reasonable cost.

The first and foremost objective of establishment of this medical college is to offer MBBS degree under Rajshahi University of Bangladesh and to provide good quality medical graduates, who can fulfill the need of health care prevailing in the country.

Editorial

Undergraduate Medical education in Bangladesh- our responsibilities.

Mohammad Mushfiqur Rahman

Through its journey from the prehistoric era, medicine has evolved itself into a social system heavily bureaucratized and politicalized and truly commercialized. Medical education has also traversed a long way from the ancient medicine in India, China, Egypt, Mesopotamia and Greece to the Middle Ages and finally to the present day medicine. Now colonization made a great change in our medicinal practice[1]. We lost our glory of contribution to medical science by the celebrated authorities of our own.

Throughout the colonial rule of two hundred years, we had no choice other than to accept the westernized medicinal practice. In fact, during this period, Europe and America experienced a revolutionized stage of development in culture, science and industry[2]. In recent years, medical education in developed and developing countries has undergone profound changes. To cope with these changes, medical schools around the world have attempted to make their curricula more meaningful and relevant to the needs of the time and to produce doctors oriented to the real needs of the community[3].

Medical education in Bangladesh has also experienced many changes and challenges. In Bangladesh a set up for undergraduate medical education was continued from pre-liberation period up to 1988. Then a great change was brought about in curriculum in 1988 by making it more community oriented[4]. In Bangladesh, a community-oriented and competency-based undergraduate medical curriculum was implemented in 2002 with an aim to produce need-based doctors for the community[5,6]. However, it is necessary that the curriculum should be further reviewed and improved.

AUTHOR'S AFFILIATION
Dr. Mohammad Mushfiqur Rahman
Associate Professor
Department of Surgery
Prime Medical College, Rangpur

The process of medical education in Bangladesh began with the establishment of the Mitford Medical School and Hospital at Dhaka in the early 20th century. The medical education system inherited the typical features of colonial education, which was very much on the traditional pattern: lecture-based, teacher-centered, discipline-based, examination-driven and hospital oriented. Till 1988, there was no formal medical curriculum except a syllabus, published by Bangladesh Medical and Dental Council (BMDC). Centre for medical education (CME) was set up at Dhaka in 1983 by an UNDP-funded Project and a national curriculum was developed for undergraduates in 1988. This is the first scientifically developed curriculum in the country with the active involvement of the medical teachers. In 1992, the Further Improvement of Medical College Project was set up under the Fourth Health and Population Project, supported by the World Bank with ODA (Overseas Development Administration) and IDA (International Development Association) components, in order to re-orient and strengthen medical education in the country[7].

The outcome is not frustrating, rather encouraging, as an "educational environment for change" has been established in the arena of medical education of Bangladesh[8]. The positive aspects are: i) growing realization to review existing curriculum[9,10,11,] ii) availability of a sufficiently large group trained teachers in medical education[12,] iii) availability of resources, expertise procedures and guidelines[9,10,11,] iv) presence of an organizational and operational framework of the Quality Assurance Scheme, both nationally and locally in the medical colleges[13] and v) commitment to improve the medical education in the current Health and Population Sector Program (HPSP)[14.]

The Bangladeshi medical students are no less meritorious than those of the other western or neighboring countries and also no less studious or industrious, though the outcomes are not satisfactory; but why? Possibly there are a number of causes of weaknesses.

One of the important cause is the education model that we are running that we need to change the hundred years old model to Integrated Medical Curriculum (IMC). Successful implementation of integrated curriculum may face serious obstacles. Secondly, we are to introduce psychosocial and community issues in more detail with practical exposure in field practice as in primary health care at community level15. The issues include economic, behavioral and community factors which influence a patient in his or her compliance and/or response to disease and wellbeing. The students use that understanding to collect and incorporate appropriate psychosocial, cultural, family and community data into an appropriate patient care plan1. Thirdly, assessment of competence and performance of a medical student16. Assessment of competence should provide insight into actual performance, as well as the capacity to adapt to change, find and generate new knowledge and improve overall performance. At last, the quality and integrity of the medical educators and trainers are questioned. How many of us (teachers) maintain honesty in teaching and assessment? How many of us envision involving the students in research addressing our commonly encountered health problems? It is of utmost importance to investigate and evaluate the assessment mechanism and the integrity and honesty of the assessors (teachers).

Above all, we need dedicated resources, dedicated teachers and even dedicated and committed leadership to bring real changes in medical education of Bangladesh. Are we really ready to do that?

REFERENCES

1. Syeed MA. Medical eduation in Bangladesh – Past, present & future. Ibrahim Med. Coll. J.2010; 4(2): i-ii.
2. Bonner TN. Becoming a Physician: Medical Education in Britain, France, Germany, and the United States, 1750-1945. Oxford, Oxford University Press, 1995.
3. Majumder AA, D'Souza U, Rahman S. Trends in Medical Education: challenges and directions for needbased reforms of medical training in south-east Asia, Indian J Med Sci. 2004; 58(9): 369-380.
4. Karim A, Haque M. Assessment system in Pharmacology – Does it reflect educational objectives and community health needs? Bangladesh J PhysiolPharmacol. 1996; 12(2): 65-67.
5. Ali T, Begum N, Begum AA, Shamim KM, Ferdousi S, Bennoor KS. Summative Assessment (Written) in Undergraduate Physiology Curriculum in Bangladesh. Reflection of Educational Objective. J Bangladesh Soc Physiol 2008; 3: 61-65.
6. Curriculum for Under-graduate Medical Education in Bangladesh 2002. Approved by Bangladesh Medical & Dental Council.
7. Majumder MAA. Medical Education in Bangladesh: Past Success, Future Challenges. Bangladesh Medical Journal 2003; 32: 37-39.
8. Majumder MAA. A review of the undergraduate medical curriculum. Bangladesh Medical Journal 2002; 34: 47-49.
9. Further Improvement of Medical Colleges Project in Bangladesh: Progress report. Dhaka: FIMC Project; 1996.
10. Present Status of the Project: Report to Executive Steering Group. Dhaka: FIMC Project; 1998.
11. QAB. Quality assessment and audit review (QAAR): Report on a formal review visit to Dhaka, Chittagong and Rangpur Medical Colleges. Dhaka: Quality Assurance Body (QAB), CME; 1998.
12. CME. Annual Report 1999. Dhaka: Centre for Medical Education; 2000.
13. FIMC Project. Quality Assurance Handbook. Dhaka: FIMC Project; 1998.
14. MOH&FW. Health and Population Sector Program (HPSP): 1998-2003. Program Implementation Plan. Part-I. Dhaka: MOH&FW; 1998.
15. Fragstein MV, Silverman J, Cushing A, Ouilligan S, Salisbury H, Wiskin C. UK consenus statement on the content of communication curricula in undergraduate medical education. Medical Education 2008; 42: 1100-1107.
16. Ronald M, Epstein MD. Assessment in Medical Education. N Engl J Med 2007; 356: 387-396.

Original article

Study on Serum Thyroxine Hormone level in menopausal women.

Mahfuza Pervin[1], Chandra Rani Sarkar[2], Neaz Ahmed[3], A. T. M Zoadur Rahim Zahid[4].

ABSTRACT

INTRODUCTION: *The incidence of thyroid disease increases in women particularly during menopause, where they experience estrogen dominance. Increased prevalence of subclinical thyroid disease has been observed in this group of people, which is about 23.2% and among them 73.8% were hypothyroid. So, there may be relationship between estrogen dominance and hypothyroidism.* ***OBJECTIVE:*** *To observe serum thyroxine hormone level in menopausal women.* ***METHODS:*** *This cross sectional study was conducted from July 2014 to December 2015 in the Department of Physiology, Rangpur Medical College, Rangpur. For this 54 menopausal women were studied and they were compared with 52 apparently healthy reproductive age women. The serum thyroxine hormone level was measured in each subject. For statistical analysis independent sample "t" test was performed by computer based software SPSS- 17.0 version for windows.* ***RESULTS:*** *Mean serum total thyroxine level was lower menopausal women then healthy reproductive aged women and the difference was not statistically significant ($p>0.05$). Again, mean serum free thyroxine level was decreased in menopausal women than those of control subjects, which was highly significant ($p<0.001$).* ***CONCLUSION:*** *From this study we can conclude that serum thyroxine hormone level decreases in menopausal women, which indicates hypothyroidism in menopausal women.*

KEY WORDS: Thyroxine, Menopause, Hypothyroidism.

INTRODUCTION

Menopausal women may be presented with signs and symptoms of hypothyroidism, which are often atypical. Therefore, thyroid hormone deficiency may not be recognized during menopause[1]. Again, in postmenopausal women increased incidence of diseases like atherosclerosis, myocardial infarction, osteoporosis and some cancer has been observed and subclinical hypothyroidism is the independent risk factor for these diseases[2]. In women, at this stage of life cessation of menstruation as well as "Depletion of Ovarian follicle" occurs, which leads to decrease in ovarian hormones level. Natural menopause occurs at or after 40 years of age[3]. In the Western world, the most typical age range for menopause (last period from natural causes) is between 40 to 61years and the average age for last period is 51 years[4].

During menopause, hormone levels normally fluctuate and decline. These hormonal imbalance acts as a trigger for thyroid problems. Women body has a delicate balance of hormone such as estrogen and progesterone which can be upset during menopause and which are associated with hypothyroidism[5]. Physiological changes in thyroid hormone concentration might be related to the changes in the overall physical functions in the body[6]. The main changes regarding thyroid physiology are reduction of thyroid iodine uptake, free thyroxine and free triiodothyronine synthesis and catabolism of free thyroxine. Menopausal symptoms may be related to imbalances in the thyroid and adrenal

AUTHOR'S AFFILIATION

1. *Dr. Mahfuza Pervin
   Assistant Professor of Physiology
   Prime Medical College, Rangpur
2. Prof. Dr. Chandra Rani Sarkar
   Head of the Department of Physiology
   Rangpur Medical College, Rangpur.
3. Dr. Neaz Ahmed
   Associate Professor & head of Biochemistry
   Rangpur Medical College, Rangpur .
4. Dr. A. T. M Zoadur Rahim Zahid
   Associate Professor of of Physiology
   Rangpur Medical College, Rangpur .

***for correspondence**

glands that interact with lowered ovarian hormone levels. These occur most often when the ovaries cease producing the same amount of ovarian hormones. It has also been observed that menopausal symptoms are more intense in patients with hypothyroidism. If hypothyroidism remains undetected and untreated, it can lead to serious health hazard[7]. In this perspective we have observed serum thyroixine hormone level in menopausal women, which would help the physician for early detection and better management of thyroid dysfunction in menopausal women in order to improve their quality of life and also to develop awareness among the general people.

MATERIALS AND METHODS

This cross-sectional study was carried out in the Department of Physiology, Rangpur Medical College, Rangpur, from July 2014 to December 2015. A total number of 106 apparently healthy women subjects were included in this study. Among them 54 menopausal women aged 40-55 years were selected as experimental group (Group B) and 52 women aged 20-35 years were selected as control group (Group A). All the experimental subjects were selected from surrounding community. Subjects, who had at least 12 consecutive months of amenorrhoea from last menstrual period, were selected as study subjects. Induced menopause by chemotherapy, pelvic radiation, bilateral oophorectomy, hysterectomy, menopausal women below 40 years and above 55 years, reproductive aged women below 20 years and above 35 years and subjects suffering from chronic diseases like liver disease, diabetes mellitus, pregnancy and kidney disease were excluded from the study. Study protocol was approved by ethical committee of Rangpur Medical College, Rangpur. After selection of the subjects objectives and benefits of this study were explained to each subject and an informed written consent was taken. Standard questionnaire were filled up after taking history and thorough clinical examinations. 5 cc blood was collected from ante-cubital vein of each subject to estimate serum total thyroxine(T4) and serum free thyroxine (FT4) hormone levels by ELISA method and also to estimate fasting blood sugar, serum creatinine level and serum ALT level by Analyzer machine in the Department of Biochemistry, Rangpur Medical College, Rangpur to exclude diabetes mellitus, liver diseases or renal impairments. All data were recorded systematically in a preformed history sheet and all statistical analysis were done by computer using the software SPSS 17.0 version for windows. Comparison of serum thyroxine hormone level in reproductive age women and menopausal women were done by unpaired students 't' test. p value < 0.05 was accepted as level of significance.

RESULTS

In this study mean age ±SD were 49.32 ± 2.02 and 25.84 ± 3.29 years in Group B (experimental) and group A (Control) respectively (Table I).

Table I: Distribution of subjects according to age.

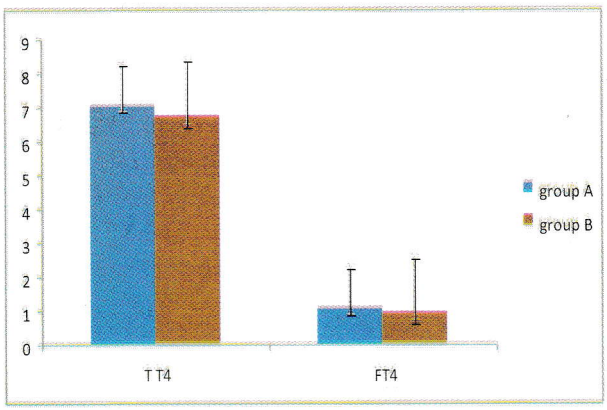
Groups	Age (yrs)
Group A (n=52)	(25.84 ± 3.29) (20 to 35)
Group B (n=54)	(49.32 ± 2.02) (40 to 55)

n= number of subjects in each group.
Group A = Control; Group B = Experimental
Figures in perenthesis indicates ranges

The mean ± SD serum total thyroxin levels were 6.709 ± 1.694 µg/dl in group B (experimental) and 7.044 ± 1.253 µg/dl in group A (control). The mean value of serum total thyroxin level was lower in experimental group than that of control group but the differences was not statistically significant ($p>0.05$).

The mean ± SD serum free thyroxine levels were 0.882 ± 0.235 ng/dl in group B (Experimental).1.051 ± 0.145 ng/dl in group A (Control). The mean value of serum free thyroxine level was decreased which is highly significant ($p<0.001$) in experimental group than that of control group (Fig 1).

Fig 1: Distribution of subjects according to mean serum total thyroxine and free thyroxin level.



TT4= Mean± SD total thyroxine
FT4= Mean± SD serum free thyroxine

DISCUSSION

In our study we have observed the serum thyroxine hormone level in menopausal women and also compared this value with apparently healthy reproductive aged women.

In this study we have observed decreased level of mean serum total thyroxine level and mean serum free thyroxine level in menopausal women than those of reproductive age group. Incase of total thyroxin level the difference was not statistically significant but incase of free thyroxin level the difference was statistically significant. These findings are consistent with those reported by some reasearchers [8, 9,10,6,11,12.]

In menopause estrogen dominance occurs when estrogen level remains high along with normal level of progesterone or low level of progesterone. Again, normal level of estrogen along with low level of progesterone can be said as estrogen dominance. Estrogen dominance causes thyroid imbalances where estrogen may affect the conversion of T4 into T3 and resulting low T3 level. It can also block the uptake of iodine by thyroid follicle during thyroid hormones synthesis. Again, estrogen dominance increases thyroid binding globulin level in blood, which usually carries thyroid hormones to the cells. Free thyroid hormone levels becomes low due to increased thyroid binding globulin[8]. All these factors leads to hypothyroidism. Some Researchers reported that when estrogen is not properly counterbalanced with progesterone, it can block the action of the thyroid hormones. So the thyroid hormones become ineffective and hypothyroidism occur[9]. The mean serum thyroxine level significantly decreased in menopausal women which might be due to decrease T4 turnover rate, decline in T4 distribution space, increase in T4 degradation rate and alterations in TBG concentrations[10.] Again significantly decreased serum thyroxine level in menopausal women which might be due to mild to moderate decrease in function of thyroid gland may occur with advancing age even in apparently healthy subjects. In aged women involves progressive loss of cells, reduced metabolic activities and decreased efficiency of many functions of different organs. Aging is not solely an intrinsic process other environmental factors such as lack of exercise, poor diet and genetic factor is also to play role in causing physiological changes in aging process[6]. In, addition significantly decreased serum thyroxine level in menopausal women which might be due to changes in binding proteins, alteration in metabolic clearance rates, production and degradation of these hormones with increasing age[11.] Again found significantly decreased serum thyroxine level in menopausal women which might be due to increased serum level of TBG which causes decrease serum level of free thyroxin hormone level[12.]

CONCLUSION

It can be concluded that both serum total thyroxine and free thyroxin level decreases in menopausal women. However, the exact mechanism can not be elucidated from this type of study. Further study by estimation of serum antithyroid antibody along with ovarian hormones and serum thyroid binding globulin level in menopausal women can give more conclusive findings.

REFERENCES

1. Sawin CT, Chopra D, Azizi F, Mannix JE, Bacharach P. The aging Thyroid: increased prevalence of elevated serum thyrotropin levels in the elderly. JAMA Network 1979; 242 (3): 247-250.
2. Joshi SA, Bhalerao A, Somalwar S, Jain S, Vaidya M, Sherawat N et al. Screening of peri- and postmenopausal women for hypothyroidism. Available form: sulabhaajoshi@gmail.com.
3. Jeffcoate's N. Menopause. In: Kumar P and Malhotra N. 7th ed. Principles of gynaecology. India: Jaypee Brothers; 2008. 862-865.Menopause. Wikipedia, the free encyclopedia. Available form: //J:\menopause.htm.
4. Menopause. Wikipedia, the free encyclopedia. Available form://J:\menopause.htm.
5. Nita G, Singh J, Sodhi KS and Ashima B. Evaluaion of subclinical hypothyroidism in women of postmenopausal age group. Advance researches in biological sciences 2012; 4(1):20-22.
6. Rahman S, Jahan N and Sultana N. Age related change in thyroid function. J Bangladesh Soc Physiol. 2012 Dec; 7(2): 72-77.
7. Valencia HM, Perez CN, Zarate A, Basurto L, Apolinar ML, Ruiz M, Vargus C Vargus A et al. Hypothyroidism associated to menopause symptoms worsening change with thyroid substitution therapy. Ginecol Obstet Mex. 2008; 76(10): 571-75.
8. Gutner M. How estrogen dominance can ruin your thyroid. Thyroid autoimmunity, Hashimoto's disease and hypothyroidism in women. Outsmart disease blog Jan 8, 2013; 1-6.
9. Estrogen Dominance: A true balancing act-blog-Dr. Christiane Northrup. Available form: http://www.facebook.com/DrChristianeNorthrup.
10. Lipson A, Nickoloff EL, Hsu TH, Kasecamp WR, Drew HM, Shakir R et al. A study of age-dependent changes in thyroid function tests in adults. J Nucl Med. 1979; 20: 1124-1130.
11. Khamees SI. The effect of age on thyroid and thyroid stimulating hormones levels in healthy Iraqi females. 829-836. Available form: http://www.cbe – journal.com.
12. Bottiglioni F, Aloysio D, Nicoletti G, Mauloni M, Mantuano R, Capelli M et al. A study of thyroid function in the pre- and post-menopause. Maturitas 1983; 5:105- 114.

Original article

Comparison of clinical and laboratory parameters between Type 1 and Type 2 Diabetes Mellitus in children and adolescents in a diabetic care centre of Bangladesh.

Sultana Tahmina Huq[1], M.A. Muttalib[2], Bedowra Zabeen[3], Asma Ul Hosna[4].

ABSTRACT

BACKGROUND: *Though T1DM is a disease of children and adolescents but the incidence of T2DM is increasing in our country. The clinical and laboratory findings are quite different between the type 1 and type 2 diabetic children and adolescents.* ***OBJECTIVE:*** *The objective of our study is to compare the clinical and laboratory parameters in children and adolescents with type 1 and type diabetes mellitus.* ***METHODS:*** *The study was conducted in the Department of Biochemistry of BIRDEM General Hospital from July 2015 to June 2016. A total of 512 subjects of aged 10-18 years were selected from the outpatient Department of CDiC, BIRDEM-2 Hospital according to inclusion and exclusion criteria. Type 1 and Type 2 Diabetes Mellitus were diagnosed based on the World Health Organization (WHO) / ISPAD criteria in children and adolescents. Written consent was taken from patients and parents and they were interviewed. A structured questionnaire was filled up for each study subjects including data about current age, sex, duration of diabetes, age at onset and family history of diabetes. Laboratory parameters done for this study were- fasting blood glucose, glycated hemoglobin and lipid profile. For group comparison, we used the student's t- test. All statistical tests were considered significant at the level of ≤5%. Statistical analysis was performed with SPSS for Windows version 20.* ***RESULTS:*** *Among the 512 subjects, significant difference was observed in age at diagnosis (11.1 ± 3.5 vs. 12.2 ± 2.6; p<0.001). We also observed that mean duration of diabetes mellitus was significantly higher in type 1 diabetes mellitus than type 2 diabetes mellitus(5.2 ± 2.7 vs. 3.5 ±2.3; p<0.001). Significant difference was also observed in age at visit between the two groups (16.2 ± 2.3 vs. 15.8 ± 2.2; p<0.001). The study also revealed that BMI was higher in Type 2 diabetes mellitus than in Type 1 diabetes mellitus (25.9± 5.2 vs. 20.2 ± 4.2kg/m2, p<0.001). There was also significant difference in blood pressure in type 1 and type 2 diabetic children and adolescence, systolic blood pressure(SBP) 102.4±12.8 vs. 111.6±15.3 mm of Hg and diastolic blood pressure(DBP) 69.1±10.3 vs. 73.6±10.8 mm of Hg, p<0.001. The study also revealed that waist circumference between the two groups was significant 70.9±11.4 vs. 85.6±14.2cm, p<0.001).* ***CONCLUSION:*** *In our study, we found that most of the clinical and laboratory findings were increased in T2DM than T1DM.*

KEY WORDS: Type1DM, Type2DM, children and adolescents.

INTRODUCTION

Now a days, diabetes mellitus has become a serious chronic disorder of childhood and represents a major public health problem[1,2] . The global incidence of type1 diabetes is increasing worldwide, at an annual rate of 3-5%, particularly in children under the age of 5 years and this trend might lead to a significant health burden[2]. The International Diabetes Federation (IDF) Atlas estimated the incidence of type1DM (T1DM) in Bangladesh that is about 4.2 cases of T1DM/100,000 children (0-14 years)/year, in 2013. This report indicated that Bangladeshis are more prone to develop diabetes from its early age[3]. Again, T2DM is no longer a disease of adults; it has become a disease of children and adolescents too[4]. This group of people is becoming an increasingly important public health concern throughout the world due to its increasing incidence[5]. In a study done in CDiC of BIRDEM-2 General Hospital, in children the prevalence of newly diagnosed with type 2

diabetes mellitus was 8%[6]. The increase of type 2 diabetes has been attributed to obesity, lack of physical activity, improper diet, family and medical history[7]. Although type 1 diabetes can occur at any age, it is predominantly seen in children and young adults[8]. But type 2 diabetes rarely occurs before the age of 15[9]. As because in T2D comorbid characteristics of insulin resistance are commonly present at diagnosis or gradually appear in the course and also acute onset is absent or symptoms not found at early stage like T1D, they should be screened earlier than T1D[6]. Duration of diabetes is difficult to determine in type 2 diabetes due to long asymptomatic period[10]. Overweight patients with type2 diabetes were 15 times more likely to develop cardiovascular events[11]. Many studies have demonstrated that the atherosclerotic process begins in childhood in association with high blood cholesterol levels. Severe obesity is associated with the premature onset of type 2 diabetes in adolescence and dyslipidemia is generally found in overweight/obese children[12]. Hypertension (HTN) is uncommon in the paediatric population. However, HTN is more common in children with T2DM children than T1DM13. This study was conducted to compare the clinical as well as laboratory status of type1 and type 2 diabetic children and adolescents people of Bangladesh. To the best of our knowledge, this study was not conducted previously elsewhere in our country. This study may help the clinicians for early diagnosis, screening and also to prevent further complications in children and adolescents with type 1 and type 2 diabetes.

MATERIALS AND METHODS:

This study was carried out in the period from July 2015 to June 2016 in the Department of Biochemistry of BIRDEM General Hospital. It included 512 subjects of aged 10-18 years from the outpatient Department of CDiC, BIRDEM-2 Hospital. Among the subjects 214 were male and 298 were female. Written consent was obtained for each subject & their parents after approval of the study by the ethical review committee of the Bangladesh Diabetic Samity (BADAS). Type 1 and Type 2 Diabetes Mellitus were diagnosed based on the World Health Organization (WHO) / ISPAD criteria in children and adolescents. Among the 512 subjects 317 were type1 & 195 were type 2 diabetic children and adolescents. The classical symptoms of type 1 diabetes are-polyuria, polydipsia, weight loss over 2-6wk, severe dehydration, shock, diabetic ketoacidosis, fasting insulin <25μIU/ml. Features suggesting of type 2 diabetes are-age above 10 year, overweight or obesity, strong family history of type 2 diabetes, acanthosis nigricans, metabolic syndrome, polycystic ovarian syndrome, fasting insulin >25μIU/ml[9]. Height, weight and waist circumference were recorded and body mass index (BMI) scores were calculated as weight (kg)/height (m^2). BMI was classified using the Center for Disease control and Prevention growth chart (2000 CDC Growth Charts for the United States: Methods and Development). Blood pressure was measured on the right arm, with the subject seated after 10 minutes rest using a standard clinical mercury sphygmomanometer with an appropriate cuff size. Patients were diagnosed as hypertensive who had blood pressure >95th percentile according to center for disease control for BP for age and height percent. Glycemic control was assessed by fasting blood glucose and glycosylated hemoglobin (HbA_{1c}).Laboratory assessment was done for fasting lipid profile including: serum total cholesterol (TC), serum triglyceride (TG), low density lipoprotein-cholesterol (LDL-C) and high density lipoprotein-cholesterol (HDL-C) were estimated. Cut points for abnormal lipid levels (TC≥200 mg/dl, LDL cholesterol≥130 mg/dl, Triglyceride≥150 mg/dl and HDL cholesterol <40 mg/dl in male and <50 mg/dl in female) were taken from the Third Report of the National Cholesterol Education program and the American diabetes Association. For group comparison, we used the student's t- test. All statistical tests were considered significant at the level of ≤5%. Statistical analysis was performed with SPSS for Windows version 20.

RESULTS

Clinical characteristics of T1DM and T2DM patients were shown in table I. Significant difference in age at diagnosis was observed among the groups (11.1 ± 3.5 vs. 12.2 ± 2.6; p<0.001).Again mean duration of diabetes mellitus was significantly higher in type 1 diabetes mellitus than type 2 diabetes mellitus(5.2 ± 2.7 vs. 3.5 ±2.3, p<0.001). Significant difference in age at visit was also observed between the two groups (16.2 ± 2.3 vs. 15.8 ± 2.2; p<0.001). Regarding BMI, it was higher in Type 2 diabetes mellitus than in Type 1 diabetes mellitus (25.9± 5.2 vs. 20.2 ± 4.2kg/m2, p<0.001) and there was also significant difference in blood pressure in type 1 and type 2 diabetic children and adolescence, systolic blood pressure (SBP) 102.4±12.8 vs. 111.6±15.3 mm of Hg and diastolic blood pressure(DBP) 69.1±10.3 vs. 73.6±10.8 mm of Hg, p<0.001. The study also revealed that waist circumference between the two groups was significant, p<0.001.

Table II: Comparison of Clinical Characteristics of patients with Type 1 and Type 2 diabetes mellitus.

Variables	Type 1 (n = 317) Mean ± SD	Type 2 (n =195) Mean ± SD	p value
Age at diagnosis (years)	11.1 ± 3.5	12.2 ± 2.6	<0.001
Duration of DM (years)	5.2 ± 2.7	3.5 ± 2.3	<0.001
Age at visit(years)	16.2 ± 2.3	15.8 ± 2.2	<0.05
BMI(kg/m^2)	20.2 ± 4.2	25.9 ± 5.2	<0.001
WC(cm)	70.9 ± 11.4	85.6 ± 14.2	<0.001
Systolic BP (mm Hg)	102.4 ± 12.8	111.6 ±15.3	<0.001
Diastolic BP (mm Hg)	69.1 ± 10.3	73.6 ±10.8	<0.001

*Statistical analysis was done by Student's t-test to compare among the groups
n= Number of subjects.

Laboratory parameters of the study participants

Table-II showed the laboratory characteristics of Type 1 diabetes mellitus and Type 2 diabetes mellitus. The mean fasting blood glucose was higher in T2DM than in T1DM 11.3 ± 3.8 vs. 10.9 ± 4.1mmol/l, but the difference was not significant, p=0.266. Mean value of HbA_{1c} was significantly (p<0.001) higher in T2DM than in T1DM 10.8 ± 3.1 vs. 9.5 ± 2.5. Patients with T2DM had higher total cholesterol, LDL-cholesterol and triglyceride than in T1DM which was not statistically significant. HDL cholesterol was higher in Type 1 diabetes mellitus than Type 2 diabetes mellitus and that was not statistically significant (p> 0.05).

Table II: Comparison of Laboratory Characteristics of patients with Type 1 and Type 2 Diabetes mellitus.

Variables	Type 1 (n =317) Mean ± SD	Type 2 (n=195) Mean ± SD	p value
FBS(mmol/l)	10.9 ± 4.1	11.3 ±3.8	0.266^{ns}
HbA_{1c} (%)	9.5 ± 2.5	10.8 ±3.1	<0.001
Total Cholesterol(mg/dl)	169.4 ±41.1	175.5 ±41.7	0.106^{ns}
LDL Cholesterol(mg/dl)	103.4 ±39.4	109.8 ±36.3	0.068^{ns}
Triglyceride(mg/dl)	142.5 ±66.2	145.8 ± 70.7	0.596^{ns}
HDL Cholesterol(mg/dl)	43.2 ± 20.8	39.1 ± 17.8	0.022

*Statistical analysis was done by Student's t-test to compare among the groups
n= Number of subjects;ns= Not significant (p>0.05)

DISCUSSION

The present study was conducted to see the comparison of clinical parameters in type 1 and type 2 diabetes mellitus in children & adolescents. A total of 512 subjects were taken in our study among them 317 were type 1 and 195 were type 2 diabetic children and adolescents.

In our study, the mean age at diagnosis was statistically higher in T2DM than in T1DM. The findings are consistent with other studies[14,15,16]. But different studies showed higher age at diagnosis in type 1 diabetes mellitus than compared with type 2 diabetes mellitus[9,11]. Our study showed longer duration of DM in T1DM than T2DM. Many studies showed that duration of DM in type 1 diabetes mellitus was longer in type 2 diabetes mellitus [14,17,18]. But others showed longer duration of DM in type 2 diabetes mellitus than compared with type 1 diabetes mellitus[16,19]. In the present work mean age at visit was significantly higher in T1DM than T2DM.Similar observation was made by others[18]. This can be explained as type 1 diabetes is prepubertal and type 2 diabetes is postpubertal we found age at visit higher in type 1 diabetes mellitus than type 2 diabetes mellitus. In different studies showed that T1DM may be pubertal. However, others showed that age at visit was higher in T2DM than T1DM because type 2 diabetes mellitus is asymptomatic for long time and its onset is slow or insidious[9,11,14]. Our study showed that mean BMI(kg/m2) in type 2 diabetic patients was significantly higher than compared with type 1 diabetic patients. This is consistent with other studies[11,15,16,18-21]. It can be explained because T2DM presents with obesity, overweight or metabolic syndrome. In a study showed that childhood obesity had increased rapidly in Asian countries[22,23]. Other study showed higher mean waist circumference in T2DM than T1DM[21]. In our study, mean waist circumference was statistically higher in T2DM than T1DM. A study done in Thailand showed that T2DM patients had lower mean fasting blood glucose levels than T1DM patients[15]. But our finding was not consistent with this study. In the present study, fasting blood glucose was higher in T2DM than T1DM which was not significant. As most of the T1DM patients were already diagnosed and having insulin where as majority of T2DM patients were newly diagnosed, so we found fasting blood glucose higher in T2DM than T1DM. Elevated blood pressure is documented as the most significant contributing factor in the pathogenesis and progression of diabetic nephropathy in both type 1 and type 2 diabetic patients. In the present study, both systolic and diastolic blood pressure was significantly higher in T2DM than T1DM. The findings are in agreement with other studies in which they showed that blood pressure was higher in

T2DM than compared with T1DM[14,16,17]. In different studies showed that lower mean HbA_{1c} level in T2DM than compared with T1DM[9,15,16]. But in a previous study showed higher mean HbA_{1c} level in T2DM than T1DM[14]. The present study was also consistent with that study. In our study, we found that there was higher mean HbA_{1c} level in type 2 diabetes mellitus than type 1 diabetes mellitus which was statistically significant. Our study showed that total cholesterol was higher in type 2 diabetes mellitus than compared with type 1 diabetes mellitus which was not statistically significant. In a previous study showed that higher total cholesterol in T2DM than compared with T1DM[17]. In the present study, we observed that higher LDL-cholesterol in T2DM than compared with T1DM. Another study also showed higher LDL-cholesterol in T2DM than compared with T1DM[17]. The present study also revealed that triglyceride was more in type 2 diabetes mellitus than compared with type 1 diabetes mellitus.In another study, triglyceride was also higher in T2DM than compared with T1DM[21]. We also observed that in our study, higher HDL-cholesterol in type 1 diabetes mellitus than compared with type 2 diabetes mellitus. The study also showed higher HDL-cholesterol in type 1 diabetes mellitus than compared with type 2 diabetes mellitus[21]. This could be explained by the fact that poor glycemic control among those with T2DM contributes substantially to a high lipid profile.

This study has some limitations. Firstly, the study was cross-sectional in nature; therefore, causality cannot be inferred. Secondly, the study was done in a single center of Bangladesh. Auto antibodies cannot be done in our set up, so patients are diagnosed by clinical characteristics. Another study could be done by doing auto antibodies and C-peptide in patients with type 1 and type 2 diabetes mellitus. Longitudinal prospective study could be done to evaluate the risk of chronic complications.

CONCLUSION

In our study, we found that most of the clinical and laboratory findings were increased in T2DM than T1DM. These findings might facilitate the clinicians to early screening, prongnosis,follow up and as well as reduce the development of chronic complications in both types of diabetes mellitus in children and adolescents.

REFERENCES

1. Craig M.E, Hatersley A, Donaghue K.C. Definition, epidemiology and classification of diabetes in children and adolescents. Pediatr Diabetes, 2009; 10 (Suppl 12): 3-12.
2. Patterson CC, Dahlquist G.G, Gyurus E, Green A, Soltesz G. Incidence trends for childhood type 1 diabetes in Europe during 1989-2003 and predicted new cases 2005-20: a multicentre prospective registration study. Lancet 373, 2009; 2007-2033.
3. Sayeed M.A, Rahman M.A, Fayzunnessa N , Khanam P.A, Tanjima B. Childhood diabetes in Bangladesi population. Journal of diabetes mellitus, 2013; 3:1: 33-37.
4. Basma A.Ali, Samir T, Abdullah, Ahlam M.A, Hussein M.M. The frequency of Type 2 diabetes mellitus Among diabetic children in El Minia Governorate, Egypt. Sultan Quboos Univ Med J, 2013; 13(3): 399-403.
5. Rosenbloom AL, Janet H.S, Amemiya S, Zeitler P, Klingensmith GJ. Type 2 diabetes in children and adolescents, ISPAD Clinical Practice Concensus Guidelines 2009 Compendium. Pediatric Diabetes, 2009; 10(Suppl.12): 17-32.
6. Zabeen B, Jebun Nahar, Samin Tayyeb , Fauzia Mohsin, Nazmun Nahar, Kiswar Azad. Characteristics of children and adolescents at onset of type 2 diabetes in a tertiary hospital in Bangladesh. Indian Journal of Endocrinology and Metabolism, 2016; 20:18-22.
7. Jordan D N and Jordan J L. Pediatric type 2 diabetes mellitus complications systemic review of the literature. Journal of Diabetes Research and Clinical Metabolism, 2012; 1-5.
8. Valma H, Niina L, Marjatta K and Per-H group. Age at onset of type 1 diabetes in parents and recurrence risk of offspring diabetes. Diabetes journals .org, 2010; 59: 210-214.
9. Hiroki Y, Okudaira M, Toshika O, Akiko S, Junnoske M. Higher incidence of diabetic nephropathy in type 2 than type 1 diabetes in early onset diabetes in Japan. International Society of Nephrology, 2000; 58: 301-311.

10. Maike C.E, Maria EC, Janine H, Albert KFC, Neville S et al. Prevalence of diabetes complications in adolescents with type 2 compared with type 1 diabetes. Diabetes care, 2006; 29: 6:1300-1306.
11. Andrea O.Y, Eric S.H. Lau ,Wing-Yee So, Ronald C.W. Ma, Alice P.S.Kong. Prospective study on the incidences of Cardiovascular-Renal complications in Chinese patients with young-onset Type 1 and Type 2 Diabetes, Diabetes Care, 2014; 37.
12. American Diabetes Association. Management of dyslipidemia in children and adolescents with diabetes. Diabetes care, 2003; 26:7.
13. Deovina N J and James L J. Pediatric type2 diabetes mellitus complications: a systemic review of the literature. Diabetes research & clinical metabolism, 2012; 1-5.
14. Yoo EG, Choi IK and Kim DH. Prevalence of Microalbuminuria in Young patients with type1 and type2 diabetes mellitus, Journal of Pediatric endocrinology and Metabolism, 2004; 17: 1423-1427.
15. Trepatchayakorn S, Supornsik V, Wacharasindhu S, Aroonparkmongol S, Sahakitrungruang T. Trends and characteristics of childhood diabetes in a tertiary care center in Thailand, 2014; 8: 707-715.
16. Lutale JJK, Thordarson H, Abbaas ZG and Vetvik K. Microalbuminuria among type 1 and type 2 diabetic patients of African origin in Dar Es Salaam, Tanzania. BMC Nephrology, 2007; 8: 2: 1-8.
17. Constantino MI, Molyneaux L, Gisler FL, Tprac M, Saeed AA. Long-term complications and mortality in young-onset diabetes, Diabetes Care, 2013; 36: 3: 863-3869.
18. Eppens MC, Craig ME, Cusumano J, Hing S. Prevalence of diabetes complications in adolescents with type 2 compared with type 1 diabetes, Diabetes Care, 2006; 29:6: 1300-1306.
19. Maahs DM, Beverly M.S, Ronny A.B, Lawrence D, Hirsch I et al. Higher prevalence of elevated albumin excretion in youth with type 2 than type 1 diabetes, Diabetes Care, 2007; 30: 10: 2593-2598
20. Son MK, Yoo HY, Kwak BO, Park HW, Kim KS. Regression and progression of microalbuminuria in adolescents with childhood onset diabetes mellitus, Annals of Pediatric Endocrinology and Metabolism, 2015; 15-20.
21. Rubeaan KA. National surveillance for type 1, type 2 diabetes and prediabetes among children and adolescents: a population-based study (SAUDI-DM), BMJ, 2015; 1-7.
22. Yoon KH, Lee JH, Kim JW, Cho JH, Choi YH. Epidemic obesity and type 2 diabetes in Asia, Lancet, 2006, 368: 1681-8.
23. Wang Y, Lobstein T. Worldwide trends in childhood overweight and obesity, Int J Pediatr Obes, 2006; 1: 11-25.

Original article

Study on serum glucose levels in tobacco chewer and smoker subjects in Rangpur district.

Masuma Begum[1], Chandra Rani Sarkar[2], A.T.M Zoadur Rahim Zahid[3], Neaz Ahmed[4].

ABSTRACT

BACKGROUND: *In Bangladesh the prevalence of smoking among men is 48.3% and women is 20.9%. Active smoking could be independently associated with glucose intolerance, impaired fasting glucose, and type 2 diabetes.* ***OBJECTIVES:*** *To assess the effects of tobacco consumption on fasting serum glucose level, serum glucose level 2 hours after ingestion of 75 gm glucose.* ***METHODS:*** *This cross sectional study was conducted from July 2015 to June 2016 in the Department of Physiology, Rangpur Medical College, Rangpur. For this a total number of 100 male age & socioeconomic condition matched subjects were selected, among them 50 were apparently healthy non-tobacco chewer non-smoker subjects (group A), 50 were apparently healthy tobacco chewer smoker subjects (group B). The subjects were selected from Rangpur district. The study parameters were fasting serum glucose level and serum glucose level 2 hours after ingestion of 75 gram glucose. For statistical analysis Unpaired student's 't' test was performed by computer based software SPSS- 17.0 version for windows.* ***RESULTS:*** *Fasting serum glucose level and serum glucose level 2 hours after ingestion of 75 gm glucose were significantly ($p<0.001$) higher in tobacco chewer smoker subjects as compared with the healthy control subjects.* ***CONCLUSIONS:*** *The increased level of fasting serum glucose and serum glucose level 2 hours after ingestion of 75 gram glucose in tobacco user is the evidence of development of type 2 diabetes mellitus due to tobacco consumption.*

KEY WORDS: Tobacco, smoking, chewing, fasting serum glucose, serum glucose 2 hours after ingestion of 75 gm glucose.

INTRODUCTION

Smoking is one of the leading causes of diseases, disability and premature death in the world where nearly 5 million people die annually from tobacco related illness. In Bangladesh, more than 57000 people die each year from tobacco related diseases. About twenty million people use tobacco in its one form and about 5 million people use the other form of tobacco. In our country the prevalence of smoking among men is 48.3% and in women is 20.9%[1]. It has been estimated that tobacco-attributable deaths will be double from 3.4 million to 6.8 million in low and middle income countries by 2020[2].

A good number of primary studies have assessed the association between smoking and incidence of glucose abnormalities. They have suggested that active smoking could be independently associated with glucose intolerance, impaired fasting glucose, and type 2 diabetes. Therefore smoking may be a modifiable risk factor for causing type 2 diabetes mellitus[3-7].

Diabetes mellitus is characterized by uncontrolled hyperglycemia, which leads to life threatening complications due to acute metabolic aberrations[8]. A total of 366 million

AUTHOR'S AFFILIATION
1. *Masuma Begum
   Assistant Professor of Physiology
   Prime Medical College, Rangpur.
2. Prof. Chandra Rani Sarkar
   Head of the Department of Physiology
   Rangpur Medical College, Rangpur.
3. A.T.M Zoadur Rahim Zahid
   Associate Professor of Physiology
   Rangpur Medical College,Rangpur.
4. Neaz Ahmed
   Assistant Professor and Head
   Department of Biochemistry,
   Rangpur Medical College, Rangpur

*** for Correspondence**

people around the world had diabetes mellitus in the year 2011 and the number will be 522 million by the year 2030. In 2011 about 4.6 million people died due to diabetes related complications[9,10] like cardiovascular and renal complications and both of which may have relation with cigarette smoking[11].

The use of tobacco has increased the risk of type 2 diabetes in a graded manner. The association was independent of age, systolic blood pressure, alcohol consumption, BMI and physical activity. Daily smoking less than 20 cigarettes causes the multifactor elevation of the health risk about 30%, and those who smoked 20 cigarettes or more daily, the risk will be increased by 65%[12,13].

Cigarette smoking causes transient elevation in blood glucose concentration and may also influence insulin sensitivity. Independent of the body size, people who smoke, has a higher transient increase in blood glucose concentration after an oral glucose challenge test. They also observed higher insulin resistance in smoker than non-smokers, suggesting a potential risk of diabetes[14].

To the best of our knowledge few researches have been done on the smokers to observe the effects of smoking on glycemic control in our country. As many people in our country are smoker, so we have selected this group of people to assess the effects of tobacco consumption on fasting serum glucose level and serum glucose level 2 hours after ingestion of 75 gm glucose.

MATERIALS and MATHODS

This cross-sectional analytical study was conducted in the Department of Physiology, Rangpur Medical College, Rangpur from July 2015 to June 2016. A total number of 100 male subjects, aged 35-45 years were included in this study from different community of Rangpur district. Among them 50 non-tobacco chewer non-smoker subjects were included in control group and was denoted as group A & 50 tobacco chewer and smoker subjects were included in experimental group and denoted as group B. They were age and socio-economic condition matched. History of smoking average 10 sticks of cigarettes and or chewing tobacco regularly minimum 5 times in a day for five years was taken as inclusion criteria. Study was carried out with prior protocol approved by the thesis protocol review committee and ethical committee of Rangpur Medical College. Before collection of sample all the subjects was informed about the objectives of the study and then informed written consent was taken from each of the study subjects. A standard questionnaire was filled up after taking history and thorough clinical examinations. Obese & known diabetic subjects and subjects with liver, kidney, heart, lungs and other diseases were taken as exclusion criteria. None had donated or received blood in last 3 months. Serum creatinin & ALT level was estimated to exclude kidney and liver diseases respectively.

For calculation of BMI weight in kg & height in m2 of each subject was taken by measuring tape and medical weighing maching. All the subjects were advised to be in overnight (8-10 hrs) fasting state then to attend next day at 8.00 A.M. in the Department of Physiology of Rangpur Medical College. Three (3) ml of blood was collected from ante-cubital vein from each subject under all aseptic precaution by a disposable syringe. The needle was detached from the nozzle and then the blood was immediately transferred into a de-ionized test tube with a gentle push to avoid hemolysis & blood was kept in standing position till formation of clot. Serum was separated by centrifuging the blood at 3000rmp for 5 minutes. The clear supernatant was taken and kept in ependrops. All tests were carried out as early as possible.

For collection of next sample 75gm glucose dissolved in 250 ml of distilled water was ingested to each subject and directed to come after 2 hours. Just after 2 hours 3 ml blood was collected and prepared for centrifuge for estimation of serum glucose as early as possible.

All data were recorded systematically in a preformed history sheet and all statistical analysis was done by computer using the software SPSS 17.0 version for windows. Fasting serum glucose, serum glucose level 2 hours after ingestion of 75 gm glucose levels were compared between the groups by unpaired student's 't' test. In the interpretation of results, < 0.05 level of probability (p) was accepted as significant.

RESULTS

The mean age of control subjects (group A) was 36.60±2.14761 years and the tobacco chewer and smoker subject (group B) was 42.24±3.47915 years. The mean height was 1.68 m in group A & 1.66 m in group B and mean weight was 61.6 kg in group A & 66.3 kg in group B. The mean BMI were 21.82 Kg/m2 & 23.66 Kg/m2 in group A & group B respectively (Table I).

Table I: Distribution of subjects according to mean age, height, weight and BMI.

Groups	Age (years)	Height (m)	Weight (Kg)	BMI (Kg/m^2)
Group A (n=50)	36.60±2.14761 (35-45)	1.68	61.6	21.82
Group B (n=50)	42.24±3.47915 (35-45)	1.66	66.3	23.66

Group A: control (nontobacco chewer & nonsmoker)
Group B: experimental (tobacco chewer & smoker)
n= number of subjects. m= meter
Figures in parenthesis indicates ranges.

The mean ± SD fasting serum glucose level was 4.9020 ± 0.55420 mmol/L in group A(control) & 7.4520 ± 0.78409 mmol/L in group B(tobacco chewer & smoker). The mean fasting serum glucose level was significantly ($p<0.001$) higher in tobacco chewer & smoker subjects (group B) than control subjects (group A). The mean ±SD serum glucose level 2 hours after ingestion of 75 gm glucose was 6.0220 ± 0.65971 mmol/L in group A(control), 10.6740 ± 1.56033 mmol/L in group B tobacco chewer & smoker subjects. Again, the mean serum glucose level 2 hours after ingestion of 75 gm glucose was significantly ($p < 0.001$) higher in group B (tobacco chewer & smoker) subjects than group A(control) (Table II).

Table II: Mean ± SD serum fasting glucose & serum glucose level 2 hours after ingestion of 75gm glucose in group A & group B subjects.

Variables	Group A (n=50)	Group B (n=50)
Fasting serum glucose (mmol/L)	4.9020 ± 0.55420 (3.60 – 5.80)	7.4520 ± 0.78409*** (5.10 – 9.60)
Serum glucose level 2 hours after ingestion of 75gm glucose (mmol/L)	6.0220 ± 0.65971 (4.80 – 9.50)	10.6740 ± 1.56033*** (6.60 – 17.50)

Data were expressed as mean± SD. Unpaired student's 't' test was done.
Group A : control (non tobacco chewer & smoker)
Group B : experimental (tobacco chewer & smoker)
n= number of subjects.
*** = P <0.001
Figures in parenthesis indicates ranges.

DISCUSSION

The present study was carried out to compare the fasting serum glucose and serum glucose level 2 hours after ingestion of 75 gm glucose in between tobacco chewer smoker and non tobacco chewer non smoker control subjects. The mean fasting serum glucose level and serum glucose level 2 hours after ingestion of 75 gm glucose were significantly higher in tobacco chewer smoker subjects than those of control subjects. Similar observations were made by some other investigators[5,15-17].

It has been suggested that dyslipidemia occurs in tobacco users due to qualitative and quantitative alteration of lipids or lipoproteins metabolism. As a result there was more acetyl COA concentration leading to increase fatty acid and cholesterol synthesis. This increase cholesterol decreases insulin receptor sensitivity[8,18].

Again, cigarette smoking has harmful effects on the pancreas as it enhances ethanol-induced pancreatic injury leading to impaired beta cell function and insulin receptor sensitivity. Smokers also have increased hepatic lipase activity, vascular changes and reducing blood flow to skeletal muscles contribute to insulin resistance. Degree of insulin resistance is also associated with the amount of cigarette smoked[12,18]. Moreover, acute bouts of smoking provoked hyperglycemia, hyperinsulinemia and elevated blood pressure. Cigarettes contain multiple noxious substances such as nicotine, cadmium, a strong biological marker of smoking, is linked to increased risk of diabetes[5,15].

Some authors reported that increased serum glucose levels in cigarette smoking might be due to greater intraabdominal fat mass or larger upper body fat distribution leading to increased insulin resistance, raised plasma glucose concentrations and overt diabetes[18-21]. Smoking might reduce insulin sensitivity due to nicotine which stimulates the secretion of insulin-antagonising hormones such as cortisol, catecholamines and growth hormone[22].The biological plausibility that smoking might lead to insulin resistance or inadequate compensatory insulin secretion, impaired glucose tolerance due to direct effect on beta cells and chronic pancreatitis[23].

From this above discussion it may be concluded that the higher fasting serum glucose and serum glucose level 2 hours after ingestion of 75 gm glucose in tobacco chewer smoker subjects may be due to nicotine, which stimulates release of adrenaline and noradrenaline from the adrenal medulla by sympathetic stimulation. Adrenaline acts through the β1 receptor leads to lipolysis in adipose tissues, leading to the increased serum concentration of free fatty acids, cholesterol, triglyceride and LDL-C. Serum HDL-cholesterol is decreased may be due to increased hepatic lipase enzyme activity in smoker. Smoking may reduce insulin sensitivity partly due to nicotine which stimulates the secretion of insulin-antagonising hormones such as cortisol, catecholamines and growth hormone.

However, the exact mechanism cannot be elucidated from this type of study. Estimation of serum nicotine level, serum epinephrine and nor-epinephrine levels, HDL-cholesterol level, serum cortisol and growth hormone levels, plasma free fatty acid level, fasting serum insulin level, insulin sensitivity and insulin resistance can be done in same study population with large sample size, which may help us to understand the basic mechanisms of insulin resistance due to smoking.

CONCLUSION

From this study it may be concluded that the increased level of fasting serum glucose and serum glucose level 2 hours after ingestion of 75 gram glucose in tobacco user is the evidence of development of type 2 diabetes mellitus due to tobacco consumption. In this respect we recommend to build awareness among the tobacco users so that they may refrain from tobacco consumption in order to reduce mortality and morbidity caused by type 2 diabetes mellitus.

REFERENCES

1. Khan FI, Afrin S, Huq ME, Zaman UKZ and Rahman MR. Socio demographic factors related to smoking among rural adolescent. Delta Med Col J. 2014; 2(2): 58-63.
2. Global Adult Tobacco Survey: Bangladesh report 2009 [Internate]. Available from:http://zunia.org/post/global-adult-to bacco-survey-bangladesh-report- 2009.
3. Mahsud MAJ, Khan A and Hussain J. Hematological changes in tobacco using type 2 diabetic patients. Gomal Journal of Medical Sciences. 2010; 8: 8-11.
4. Uchimoto S, Tsumura K, Hayashi T, Suematsu C,Endo G, Fujii S et al. Impact of cigarette smoking on the incidence of type 2 diabetes mellitus in middle- aged Japanese men: the Osaka Health Survey. Diabet Med. 1999; 16(11): 951-5.
5. Wannamethee SG, Shaper AG and Perry IJ. Smoking as a modifiable Risk Factor type 2 Diabetes in middle-aged men. Diabetes Care. 2001; 24(9): 1590-1595.
6. Yeh HC, Duncan BB, Schmidt MI, Wang NY and Brancati FL. Smoking, smoking cessation, and risk for type 2 diabetes mellitus. Annals of Internal Medicine. 2010; 152: 10-17.
7. Nakanishi N, Nakamura K, Matsuo Y, Suzuki K and Tatara K. Cigarette smoking and risk for impaired fasting glucose and type 2 diadetes in middle-aged Japanese men. Ann Intern Med. 2000: 133(3): 183-91.
8. Aski BS, Meti V, Walvekar SS, Devararnavadagi BB and Kashinath RT. The smokeless tobacco induced effect on biochemical parameters levels in type 2 diabetes mellitus subjects. Research Journal of Pharmaceutical, Biological and Chemical Sciences.2014; 5(3): 1203-1208.
9. Khalid N, Khan EA , Saleem S, Tahir A, Mahmood H and Saleem S. Prevalance and associated factors of cigarette smoking among type 2 diabetes patients in Pakistan. International Journal of Collaborative Research on Internal Medicine and Public Health. 2014; 6(4): 73-88.
10. Zhang L, Curhan GC, Hu FB, Rimm EB and Forman JP. Association between passive and active smoking and incident type 2 diabetes in women. Diabetes Care.2011; 34(4): 892-897.
11. Al-Delaimy WK, Willett WC, Manson JE, Speizer FE and Hu FB. Smoking and mortality among women with type 2 diabetes. Diabetes Care. 2001 december; 24: 2043-2048.
12. Patja K, Jousilahti P, Hu G, Valle T, Qiao Q and Tuomilehto J. Effects of smoking, obesity and physical activity on the risk of type 2 diabetes in middle-aged Finnish men and women. Journal of Internal Medicine. 2005; 258: 356-362.
13. Xie X, Liu Q, Wu J and Wakui M. Impact of cigarette smoking in type 2 diabetes development. Journal of Acta Pharmacologica Sinica. 2009; 30: 784-787.
14. Jyothirmayi B, Kaviarasi S and William E. Study of glycated hemoglobin in chronic cigarette smokers. International Journal of Pharmaceutical and Clinical Research. 2013; 5(1): 4-6.
15. Foy CG, Bell RA, Farmer DF, Junior DC and Wagenknecht LE. Smoking and incidence of diabetes among U.S.adults. Diabetes Care. 2005; 28: 2501-2507.
16. Eliasson M, Asplund K, Nasic S and Rodu B. Influence of smoking and snus on the prevalence and incidence of type 2 diabetes among men: the northern Sweden MONICA study. Journal of Internal Medicine. 2004; 256: 101-110.
17. Rimm EB, Chan J, Stampfer MJ, Colditz GA and Willett WC. Prospective study of cigarette smoking, alcohol use, and the risk of diabetes in men. BMJ Clinical Research. 1995; 310: 555-559.
18. Radzeviciene L and Ostrauskas R. Smoking habits and risk of type 2 diabetes: A case-control study. Diabetes & amp; Metabolism. 2009; 35: 192-197.
19. Eliasson M, Asplund K, Nasic S and Rodu B. Influence of smoking and snus on the prevalence and incidence of type 2 diabetes among men: the northern Sweden MONICA study. Journal of Internal Medicine. 2004; 256: 101-110.
20. Rimm EB, Chan J, Stampfer MJ, Colditz GA and Willett WC. Prospective study of cigarette smoking, alcohol use, and the risk of diabetes in men. BMJ Clinical Research. 1995; 310: 555-559.
21. Jee SH, Foong A W, Hur NW and Samet JM. Smoking and risk for diabetes incidence and mortality in Korean men and women. Diabetes Care. 2010; 33(12): 2567-2572.
22. Soulimane S, Simon D, Herman WH, Lange C, Lee CMY, Colagiuri S et al. HbA1c, fasting and 2 h plasma glucose in current, ex- and never- smokers: a meta-analysis. Diabetologia. 2014; 57: 30-39.
23. Saeed AA. Association of tobacco products use and diabetes mellitus-results of a national survey among adults in Saudi Arabia. Balkan Med J. 2012; 29: 247-51.

Primary Testicular Lymphoma in young male: A Case Report.

Asma Ul Hosna[1], Shahina Akther[2], Muhammed Imran Uddin[3], Md.Sahadat Hossain[4].

ABSTRACT

Primary testicular lymphoma is a rare non-Hodgkin's lymphoma, which accounts for 1% of all non-Hodgkin's lymphoma. It usually seen in people around 50 years and rarely involves young adult. A 26 year young patient, presented with painless swelling in his left testis for fifteen days, came to our hospital on 22 th January 2016. Ultrasonogram report of inguino-scrotal region revealed a hypoechoic well-circumscribed mass within left the testicular parenchyma. Right testis was normal on ultrasonoram. Histopathological examination revealed diffuse large B-cell lymphoma and the tumor was limited to the testicular parenchyma and lymphovascular invasion was absent. We examined the patient properly and there was no secondary involvement. Orchidectomy followed by chemotherapy with prophylactic radiotherapy to other testis is the treatment modality of this patient.

KEYWORDS: Lymphoma, Testis, B-cell lymphoma, Orchidectomy.

INTRODUCTION

Malignant lymphomas comprise 5% of all testicular malignancies. It is the most common testicular tumor in elderly persons but it can occur in any age group including children[1]. Primary testicular lymphoma is an intermediate or high grade lymphoma and most commonly large B cell type[2]. Testicular lymphoma carries a poor prognosis compared to other extranodal lymphomas[3,4]. HIV infection is a risk factor for aggressive NHL. Most of the patient presented with a firm, painless, unilateral testicular or scrotal swelling[2,5]. Though testicular lymphoma has no standard treatment due to their rare occurrence; orchidectomy followed by chemotherapy and radiotherapy is using now a days[6].

CASE PRESENTATION

A 26 year young patient came to our hospital on 22th January 2016 with the complaint of swelling in his left testis for fifteen days. On physical examination, 2×1.5 cm firm, painless mass was palpable in his left testis, and the contralateral testis and other structures were found normal. Regarding ultrasonogram report a hypoechoic well-circumscribed mass was seen within the testicular parenchyma and rest of the abdominal organ was normal. After carrying out necessary preoperative preparations for the patient in whom AFP, beta-HCG, Complete blood count, Random blood sugar, Serum creatinine, Chest x-ray levels were normal, left radical inguinal orchiectomy was performed under general anesthesia. Histopathological examination revealed diffuse large B-cell lymphoma (Figure 1) and reported that the tumor was limited to the testicular parenchyma, presence of necrosis (Figure 2) and lymphovascular invasion was not observed. According to the Histopathology the case was diagonosed

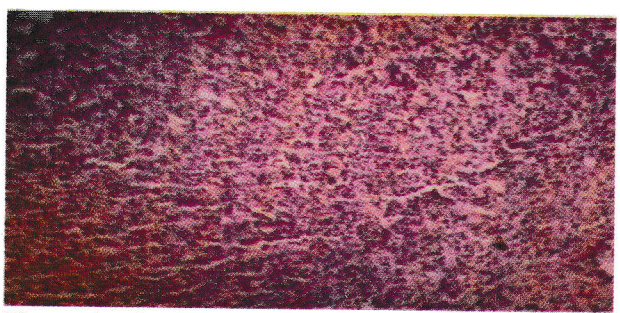


Figure 1: photomicrograph of testicular lymphoma showing atypical round tumor cells in the testis tissue, having lymphoid cell traits (HE×100x).

AUTHOR'S AFFILIATION

1. *Dr. Asma Ul Hosna
   Assistant Professor of Pathology,
   Prime Medical College, Rangpur.
   Contact No: 01681313387.
   Email: asma32nd@gmail.com
2. Dr. Shahina Akther
   Professor (C.C) of Gynae and Obstetrics,
   Prime Medical College, Rangpur
3. Dr. Muhammed Imran Uddin
   Medical Officer of Microbiology,
   Rangpur Medical College, Rangpur.
4. Dr. Md.Sahadat Hossain
   Resident. Dept. of Biochemistry
   BIRDEM Genaral Hospital, Dhaka

*** for correspondence**

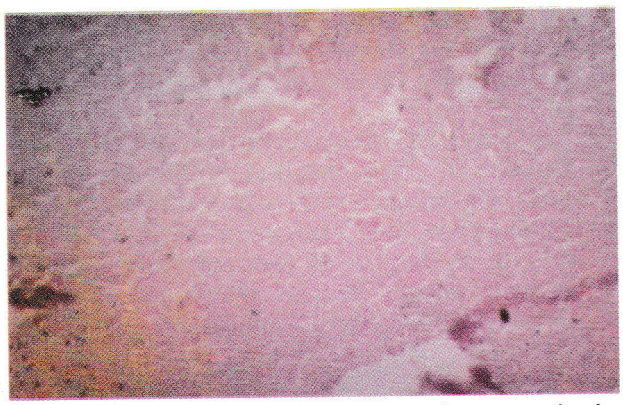


Figure 2: photomicrograph of necrosis in testicular lymphoma (HE×100x).

DISCUSSION

Testicular lymphoma was first reported by Malassez and Curling in 1866[7,8]. The mean age at presentation was 60 years, but the recent published cases of concerned patients were found younger than the past and considered that this fact had a positive effect on the outcome of the patients[9]. Our case is 26 years aged young male, who presented with the complaints of painless testicular swelling for fifteen days. In primary T-cell lymphomas, most reported cases are of T-cell/Natural Killer (NK) cell lymphoma with very few cases of peripheral T-cell lymphoma(NOS)[10,11,12]. T-cell NHL is now considered to be the most common bilateral tumor of the testes, with reported incidence of bilateral metachronous testicular involvement of 35% and bilateral synchronous testicular involvement of 3%. Other authors have reported the incidence of bilateral involvement of about 10-40%[13]. It's etiologies includes prior trauma, chronic orchitis, undescended testicles, and filariasis of the spermatic cord[14]. Histologically, 80-90% of the rarely seen primary testicular lymphomas are diffuse large B-cell lymphomas[15]. In our case we also found diffuse large B-cell lymphoma. Classification of primary testicular lymphoma that was modified by the Nordic Lymphoma Group is as follows: stage I: unilateral testis involvement with or without epididymis or cord involvement; stage II: abdominal and pelvic lymph node involvement; and stage II-IV: distant metastasis[16]. It has been reported that a primary tumor larger than 9 cm, epididymis, presence of spermatic cord and bilateral testis involvement, vascular invasion, advanced age, high LDH levels, presence of B symptoms, high International Prognostic Index (IPI) score, and left testis involvement are factors associated with poor prognosis[17]. Nevertheless, young age, localized tumor, presence of sclerosis, small size of the tumor, low histologic grade and no epididymis or spermatic cord involvement are indicators of good prognosis[18]. In our case, 26 years young male having 2×1.5cm painless mass and on histological examination we found atypical lymphoid cell with presence of necrosis in testicular tissue. The epididymis and spermatic cord were free of the tumor. No lymphovascular invasion was seen. According to the prognostic criteria our patient has comparatively good prognosis. Immunohistochemistry has the role for confirmation the diagnosis of lymphoma.

CONCLUSION

Primary testicular lymphoma is a disease with a poor Prognosis. It occurs usually in elderly but young person is not spared. Therefore, if any young patient come with painless testicular mass, then he should be tested for primary testicular lymphoma and surgeon should rule out that it is not secondary NHL. Moreover, he should be examined for any secondary involvement. This type of patient also requires joint action of urologist, pathologist and oncologist.

REFERENCES

1. Rosai J. Urinary tract, surgical pathology. 9th ed, stlouis USA MOSBY, 2004; PP-1442.
2. Sebnem IG, Didem K, Mahmut Y. Primary testicular diffuse large B-Cell lymphoma: A Case Report. World J Oncol 2013; 4(1): 61-65.
3. Lagrange JL, Ramaioli A, Theodore CH et al. Non-Hodgkin's lymphoma of the testis: A retrospective study of 84 patients treated in the French anticancer centres. Ann Oncol 2001; 12: 1313-19.
4. Touroutoglou N, Dimopoulos MA, Younes A et al. Testicular lymphoma: late relapses and poor outcome despite doxorubicin-based therapy. J Clin Oncol 1995; 13: 1361-67.
5. Chayan YC, Andrew W, John FS. Primary testicular lymphoma. Blood Journal 2014; 123(4): 486-493.
6. Filiz V, Seckin C, Guray S, Mine H, Nur AS, Murat T. Primary testicular lymphoma. J Nat I

Medn Assoc. 2007; 99: 1277-1282.
8. Crocetti E, Capocaccia R, Casella C. Cancer trends in Italy: figures from the cancer registries (1986-1997). Epidemiol Prev 2004; 28(2 Suppl): 1-6.
9. Harnly ME, Swan SH, Holly EA. Temporal trends in the incidence of non-Hodgkin's lymphoma and selected malignancies in a population with a high incidence of acquired immunodeficiency syndrome (AIDS). Am J Epidemiol 1988; 128(2): 261-7.
10. Gundrum JD, Mathiason MA, Go RS. Adult diffuse large B cell lymphoma of the testis: Analysis of the surveillance Epidemiology and end results data base from 1980-2004. J Clin Oncol 2008; 26: 19524.
11. Ferry JA, Harris NL, Young RH. Malignant lymphoma of the testis, epididymis, and spermatic cord. A clinicopathologic study of 69 cases with immunophenotypic analysis. Am J Surg Pathol 1994; 18: 376–90.
12. Coupland SE, Foss HD, Assaf C. T-cell andT/natural killer-cell lymphomas involving ocular and ocular adnexal tissues: a clinicopathologic, immunohisto-chemical, and molecular study of seven cases. Ophthalmology 1999; 106: 2109–20.
13. Bavai PD, Roger JA, Ariño AB. Primary testicular lymphoma. Report of two cases and bibliographic review. Arch Esp Urol 2008; 61: 527–31.
14. Tepperman BS, Gospodarowicz MK, Bush RS, Brown TC. Non-Hodgkin lymphoma of the testis. Radiology. 1982; 142(1): 203-8.
15. Zicherman JM, Weissman D, Gribbin C, Epstein R. Best cases from the AFIP: primary diffuse large B-cell lymphoma of the epididymis and testis. Radiographics 2005; 25: 243-248.
16. Salem YH, Miller HC. Lymphoma of genitourinary tract. J Urol 1994; 151: 1162-1170.
17. Ergün O, Koşar A, Baş E, Bircan S, Alanoğlu EG. Primer testis lenfoması: Bir olgu sunumu ve rituximab ile kombinekemoterapinin etkinliği. Turkiye Klinikleri J Med Sci 2011; 31: 748-751.
18. Shahab N, Doll DC. Testicular Lymphoma. Semin Oncol 1999; 26: 259-269.
19. Mlika M, Chelly I, Benrhouma M, Haouet S, Horchani A, Zitouna MM et.al. A Primary Testicular Diffuse Large B-cell Lymphoma Belonging to the Germinal Center B-cell-like Group. J Clin Med Res 2010; 2: 47-49.

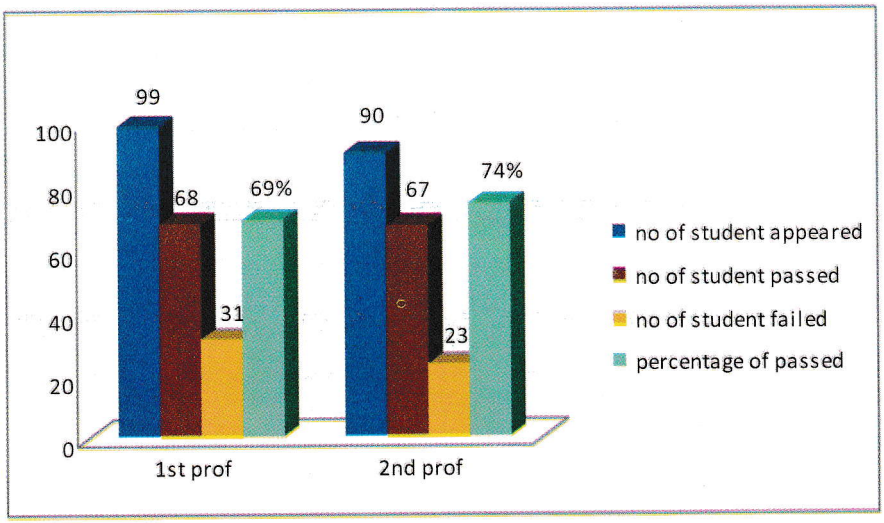
RESULTS OF PROFESSIONAL EXAMINATIONS

1st and 2nd Professional MBBS Examinations were held in May, 2016. The number of students appeared in; total number of students passed, total failed and percentages of pass in the 1st and 2nd Professional MBBS examinations are shown in the following table and figure (Table I & Figure 1).

Table I: Result of 1st and 2nd Professional MBBS Examinations in May, 2016.

Exam yeaar	exam name	no of student appeared	no of student passed	no of student failed	percentage of passed
May/16	1st prof	99	68	31	69%
	2nd prof	90	67	23	74%

Figure 1: Result of 1st and 2nd Professional MBBS Examinations in May, 2016.



Forwarding letter for submission of article Prime Medical Journal

To
The Editor-In-Chief
Prime Medical Journal
Prime Medical College, Rangpur.

Sub : Submission of manuscript

Dear Sir,
I/We are submitting our manuscript titled .....in your journal. This article has not been published or submitted for publication elsewhere. There is no Conflict of interest between the authors by.....1.....2
.....
.....3.....4.....etc

for publication in your journal. The article has not been published or subjected for publication elsewhere.

We believe that article may be of value to the professionals engaged in different fields of Medicine. We are submitting 2 copies of manuscript along with an electrinic version (CD) .

We, therefore, hope that you would be kind enough to consider our manuscript for publication on your journal as Orginal Articles/Review Articles/Special Article/Case Report/Letter to Editor.

Thanks and best regards

Signature of author/authors

Information for the contributors

The Prime Medical Journal in Published twice in a year in the month of January & July. The Journal Publishes Orginal articles, Review Articles, Case Reports, Procedures, Letter to the Editors etc. in all branches of medical Science.

Editoral scope:

- The Prime Medical Journal (PMJ) is intended to promote publication of concise scientific article based on the study in all fields of medical and health sciences.
- Submitted manuscripts should not be previously published or accepted for publication eleswhere.
- All Submitted articles will undergo double blind peer review as per recommendations by subject specific experts selected by editors.
- Reviewed manuscripts will be sent to the corresponding author for correction if it is necessary.
- Acceptance is based on significance, orginality, clarity and fulfillment of the criterias of a publication policy of this journal.
- Selection of the reviewed and accepted manuscripts intended for publication on a paricular issue will be decided by Editorial Board.
- The Editor- in- Chief will take of final decision regarding acceptance.
- Rejected manuscript will be retuned if accompanied by stamped & self-addressed envelop.
- Upon acceptance for publication the copy right of the paper automatically belong to the PMJ and will not be published elsewhere either in part or whole without written permission of copyright holder.
- Review article should be written by a subject expert

Ethical aspects :

- Manuscripts based on the study should be conducted according to the ethical standards laid down in the 1994 Declaration of Helsinki rervised in 2000.
- Manuscript must contain a statement in the method section that all human subjects involved in studies have been approved by appropriate ethical committee after careful exmination of the ethical aspects.
- Permission of the patients or their families to be taken to publish photographs of the patients where identiy is not disguised.
- Author should obtain written permission to reporoduce any table, illustration from any other source.
- Information of the individaual article are the responsibility of the outhor (s), the editoral board bears no liability what so ever for consequences of any such inaccurate and misleading information, opinion or statement.

Manuscript Sumission :

Manuscripts Prepared following the "Uniform Requirements for Manuscripts to Biomedical Journals" is acceptable to this journal for publication. The authors are requested to strictly follow the lines below for submisson of manuscript to PMJ for publication. The follwing documents with manauscripts are to be submitted for publication.

- A Covering letter adderssed to the Editor-in-Chief of the journal (Sample given at pg. no. 29).
- Abstract and key words in the first page followed by the text.
- Authors must submit 2 hard copies of all documents and one copy in electronic form preferably written in a IBM compatible CD with adequate labeling.
- In special case, submission through E-mail with file attachment of all document is acceptable.

Covering letter:

- All authors must sign after seeing the manuscript with the statement that they are the only authors.
- The corresponding author should mention the contribution of each author to the work.
- It should contain a declaration that this manuscript has not been submitted elsewhere or not under consideration in any journal.
- It should cleary indicate the publication type (Orginal/Review/Case report/Letter etc.)
- It should also mention the expected benefit of the medical science from publishing of this article.

Authors are requested to submit new and revised manuscript to:

Editor-in-Chief
Prof. Dr. Nawazesh Farid
Prime Medical College
Pirjabad, Badargonj Road, Rangpur.
Cell: 01819119577
E-mail :pmcrang@gmail.com.

Executive Editor
Prof. Dr. Latifa Afrin Dill Naher
Prime Medical College
Pirjabad, Rangpur.
E-mail : afrin_latifa2007@yahoo.com
Cell: 01768-794920

- **Manuscript Organization:**
- **Typing**
- Double Spaced throughout with justified and 2.5 cm margins in left & top.
- Font type is Times New Roman with size 12.

Printed on a good quality A4 80 gm white paper on one side of paper.

Manuscript should have uniform style, correct journal format, carefully proofed for grammar, spelling and punctuation.

Manuscript format

In general, original article should be divided into following sections: Title page, Abstract, Text, Tables with titles and foot notes, alternatively Graphs with title and Illustrations with legends. Each of the secitons is to start on a separate page. Wpages should be numbered consecutively beginning from the abstract.

Title Page:

- Title of the article (Not exceeding 60 characters).
- Names of all authors with their designation and institutional affiliations with name of the department and institute where the study was undertaken.
- Name of the corresponding author with contact address, telephone number, E-mail address.
- Disclosure of confilct of interest (if any).
- Disclosure of source of funding or sponsor.

Abstract :

- Structured with headings (Background, Objectives, Methods with statistical anlaysis, Result and Conclusion).
- Authors name should not be given.
- Preferably within 250 words.
- Avoid abberviations in the title and abstract except standard abbreviation.
- A non stuctured abstract is suggested for review article and case report.

Text:

- Text should be arranged into Introduction, Materials & Methods, Results, Discussion, Acknowledgement & References (IMRDAR).

Introducation :

- Statement of the problem with a short discussion of its importance and significance.
- Review of the literatiure related to the problem with pertinent reference.
- Objectives/hypothesis/benefits expected stated in 1-2 paragraph.

Materials & Methods :

- Study type, place and time .
- Description of study variables.
- Description of study subjects and grouping.
- Selection cariteria.
- Approval of the study involving human subjects by ethical review committee and description of the ethical aspects in such study.
- Descripation of procedure, methods, apparatus, drugs or chemicals as applicable.
- Descripation of statistcal procedure with enough detail to enable a knowledgeable reder with access to the original data to verify the reported results.

Result:

- Present result in logical sequence in text, table and Illustration with most important finding first.
- Describe without comment.
- Restrict number of table and figure needed to support assessment of paper.
- Do not duplicate data in table and figure.

Table :

- Simple self explanatory with brief title, not duplicate in text.
- Each table should be numbered in Romans and printed in separate page
- Do not use internal horizonatal and vertcal rules.
- Uses of many tables are not encouraged.

Illustration :

- All illustrations must be numbered consecutively in English numerals as they appear in the text.
- Submit print photograph of each Illustration along with its electronic file.
- Figure number, title of manuscript, name of the corresponding author and arrow indicating top should be written on a sticy label on the back of each photograph.
- Scanned picture, graph, chart with title and figure number should be printed on separate page and its original data presentation file should be inserted in the CD along with text.

Legend :

- Must be typed in seperate sheet of paper.
- Photomicrograph should indicate the magnification, internal scale and method of staining.
- All drugs should be menitoned in their generic from. The commercial name may be used in parenthesis.

Acknowledgement:

- Individuals, Institutions, Sponsors, Organizations of bodies can be acknoweledged in the article for their contribution or financial or any form of assistance the work.

References:

- For reference, use author number style (Vancouver) which is based on an ANSI standard adapted by the National Libary of Medicine (NLM).
- References should be numbered consecutively in the order on which they are first mentioned in the text.
- Identify reference in the text, tables and legend by English numerals in superscript.
- All citations to electronic references should be presented in numbered referencs following the text.

The titles of the journals should be abbreviated as:

- Coding to the style used in Index Medicus.
- Write names of 6 author followed by et al, if authors number is more than six.
- The reference list is also checked by the editorial staff or reviewer, so it is the responsibility of authior to provide accurate information.

Standard journal artcle:
Example:

Khalil M, Chowdhury MAI, Rahman S, Sultana SZ, Rahman MM et al. Splenic mass and its relation to age, sex and height of the individual in Bangladeshi People. J Bangladesh Soc Physiol 2008;3(1): 71-78.

Journal article with organization as author:

American diabetes Association. Diabetes Update. Nursing, 2003 Nov: Suppl;19-20.

Journal article with multiple organization as author:

American Dietetic association;Dietitians of Canada; Position of Dietetic association and Dietitians of Canada Nutrition and Women's health. JAm Diet Assoc 2004 Jun; 104(6): 948-1001.

Journal article with Governmental body as author:

National Institute on Drug Abuse (US); Caribbean Epidemiology Centre; Pan American Health Organization ; World Helth Organization. Building a Collaborative research agenda; drrug abuse and HIV/AIDS in the Caribbean 2002-2004. West Indian Med J. 2004 Nov; 53 suppl 4; 1-78.

Standard book with intitials for authors:

Eyre HJ, Lange DP, Morris LB, Informed decisions: the complete book of cancer diagnosis, treatment and recovery 2nd ed. Atlanta: American Cancer Society ; 2002.768p.

Contributed chapter of a book :
Rojko JL, Hardy WD. Feline lukemia virus and other retroviruses. In: Sherding RG, editor . The cat; diseases and clinical management. New york: Churchil Livingstone; 1989. p 229-332

Conference Proceedings :
Pacak K, Aguilera G, Sabban, E, Kvetansky R, editors. Stress: Current neuroendocirne and genetic approaches. 8th Symposium on Catecholamines and Other Neurotansmitters in stress: 2003 Jun 28-July 3; Smolenice Castle (place of confernce), Slovakia. New york (Place of Publication), New York Academy of Sciences (pubilisher) ; 2004 Jun. 590p.

Scientific and Technical Reports:
Page E, Harney JM. Health hazard evaluation report. Cincinnati (OH) (Place of publication; National Institute for Occupational Safety and Health) (US) (Publisher); 2001 Feb. 24p (Total number of pages). Report No: HETA2000-0139-2824.

Dissertation & Thesis:
Entire Reference
Kempner JL, Aching heads. making medicine gender and legitimacyin headache (title) [dissertation] [Philadelphia] University of Pennsylvania; 2004-271p.
Alam M. Study of Heart Rate Variability in Adolecent Athletes [M Phil Thesis]. [Dhaka] Bangabandhu Sheikh Mujib Medical University; 2008

Part of Dissertation & Thesis:
Makckwski MP. Human factors: aeropace medicine and the origins of manned space flight in the United States [dissertation]. [Tempe (AZ)]: Arizina State University; 2002 May. Part 2, Space medicine ; p. 188-377.
Alam M. heart Tate Variability in Adolescent Athletes [M Phil thesis]. [Dhaka (Bangladesh)]. Bangabandhu Sheikh Mujib Medical University; 2008 July. Appendix (name of the part 4 (number of the part), Classification of physical Activity Intensity (Tit of the Part). p.7 (Location of the Part).

Standard jouranal article on the Internet:
Kaul S, Diamond GA. Good enough.: a primer on the analysis and interpretation of noninferiority trials. Ann Intern Med [Internet]. 2006 July 4 [cited 2007 Jan 4] ; 145 (1) : 662-9, Available from:http:// www.annals. org/cgi/reprint/145/1/52.pdf

Jouranl article on the Internet with organization (s) as author:
National osteoporosis Foundation of South Africa. Use of generic alendronate in treatment of osteroporosis. S Afr Med J [Inernet]. 2006 Aug [cited 2007 Jan 9] ; 9 (8);696-7.Avilable from:http:/blues.sabinet.coza/WebZ/Authorize?

Journal article on the Internet with governmental body as author:
Centers for Disease Control and Prevention (US), National center for HIV/AIDS, Hepatitis, STD, and detection and control of tuberculosis in correctional and detection facilities: recommendations from CDC. Endorsed by the American correctional Association. MMWR R Rep[Internet]. 2006 July 7 [cited2007Jan9]; 55(RR-9]; 1-44. Avilable from: http/www.cdc.gov/mmwr/preview/mmwrhtml/rr5509al.htm.

Journal article on the Internet with no author:
Prevention stategies for Asthma-secondary prevention. CMAJ [Internet]2005 Sept[cited2007 jana5]; 173(6Suppl); S25-7. Available from;http//www.cmij.ca/content/full/173'6__supp1/s25.

Journal article on the Internet without standard volume, issue or article number:
jacobs JL, Lee MT, Lindberg M, kamin C. Problem based learning, multimedia paucity of behavioral issue learninig Med Educ. Online [Interner]. 2005[cited2005]: [5p]. Available from:http:www.med-ed-online.org/pdf/10000006.pdf.

Corrigendum

We are extremely sorry to declare that the author's affiliations of the article "Comparison of Clinical and laboratory parameters between Type 1 and Type 2 Diabetes Mellitus in Children and Adolescents in a Diabetic care centre of Bangladesh" in volume 6 number 2 July'2016 page number 42 has been dropped.

Please read the author's affiliations as mentioned here.

Authors Affliation

1. *Dr. Sultana Tahmina Huq,
   Assistant Professor,
   Department of Biochemistry,
   Prime Medical College, Rangpur.
   farihalipon@gmail.com.
   Mobile: 01723021647.
2. Prof. Dr. M.A. Muttalib,
   Professor and Head,
   Department of Biochemistry,
   BIRDEM, Dhaka.
3. Dr. Bedowra Zabeen,
   Consultant, CDiC,
   BIRDEM 2, Dhaka.
4. Dr. Asma Ul Hosna,
   Assistant Professor,
   Department of Pathology,
   Prime Medical College, Rangpur
   * for corrospondence