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Address for Correspondence: Prof. Sharmeen Yasmeen, Professor & Head, Dept. of Community Medicine
Bangladesh Medical College, House # 34, Road # 14A, Dhanmondi R/A, Dhaka-1209
Phone: (88-02) 44812101-6, Fax: (88-02) 9125655
E-mail: sharmeenbmc@yahoo.com, Web: www.bmcjournal.org

Editorial

Integrated vector management for prevention of Dengue in Bangladesh

Chaklader MA[a], Yasmeen S[b]

Dengue is a public health problem in many tropical and subtropical countries, particularly in urban and semi-urban areas, where most outbreaks have been reported. Many factors have influenced the global rise of dengue, including population growth, high population density, unplanned rapid urbanization and construction, climate change, absence of reliable piped water, and ineffective vector control strategies. The rapid global spread of dengue is also associated with increased human mobility through air travel; 75% of the global dengue burden lies in Southeast Asia and the Western Pacific region. The incidence of overall global dengue virus (DENV) infection has also increased rapidly in the last two decades; 505,430 cases were reported in 2000, while over 2,400,138 and 3,312,040 cases have been reported in 2010 and 2015, respectively. The number of deaths has also increased from 960 to more than 4032 between 2000 and 2015. Each year, an estimated 100–400 million infections occur, and over 80% of these infections are generally mild and asymptomatic. In line with global trends, the incidence of dengue has also dramatically increased in Bangladesh.

Dengue is a viral infection transmitted to humans through the bite of infected mosquitoes and the primary vectors that transmit the disease are *Aedes aegypti* mosquitoes and, to a lesser extent, *Aedes albopictus*.

Dengue virus (DENV) has four serotypes (DENV-1, DENV-2, DENV-3, DENV-4) and it is possible to be infected by each. Infection with one serotype provides long-term immunity to the homologous serotype but not to the other serotypes; sequential infections put people at greater risk for severe dengue. Many DENV infections produce only mild illness; over 80% of cases are asymptomatic. There is no specific treatment for dengue; however, timely detection of cases, identifying any warning signs of severe dengue infection, and appropriate case management are key elements of care to prevent patient death and can lower fatality rates of severe infection to below 1%.

Dengue was first recorded in the 1960s in Bangladesh (then East Pakistan) and was known as “Dacca fever”. The establishment of the *Aedes aegypti* mosquito vector and urban cycles have made dengue endemic in Bangladesh. The growth factor of dengue cases since 2010 appeared to be linked to regional rainfall patterns (May to September) and is coincidental with higher environmental temperatures. Bangladesh's climate conditions are becoming more favorable for the transmission of dengue and other vector-borne diseases like malaria and chikungunya due to excessive rainfall, waterlogging, flooding, rise in temperature and the unusual shifts in the country's traditional seasons.

Between 1 January and 20 November 2022, a total of 52 807 dengue cases including 230 related deaths (case fatality rate = 0.44%) were reported by the Ministry of Health & Family Welfare (MOHFW) The cases were confirmed either by non-structural protein (NS1) diagnostic kits or by Immunoglobulin M (IgM) tests. According to information available for 40% of reported cases (n=20 982) the median age is 25 years (range: 0 – 89) with males accounting for 60% of the cases. This is the second highest annual number of cases since 2000, the highest having occurred in 2019, when 101 354 cases including 164 deaths were reported.

The most affected division is Dhaka, accounting for 70.6% of cases and 60.4% of deaths. Dhaka city, the largest city in Bangladesh, located in Dhaka division, has reported 64.5% (n= 34 071) of the total number of cases. Other affected divisions include Chattogram division (13.2% of cases and 24.8% of deaths) and Khulna division (5.5% of cases and 4.8% of deaths).

The high incidence of dengue cases this year is taking place in the context of an unusual amount of rainfall since June 2022, accompanied by high temperatures and high humidity which have resulted in an increased mosquito population throughout Bangladesh.

The proximity of mosquito vector breeding sites to human habitation is a significant risk factor for dengue virus infection. Although dengue does not directly spread from human-to-human, *Aedes* species mosquitoes can become infected after biting dengue-infected individuals, thus creating a cycle of transmission capable of spreading dengue and leading to clusters of cases.

The prevention and control of dengue depends on effective vector control. WHO promotes a strategic approach known as Integrated Vector Management (IVM) to control mosquito vectors, including the mosquito genus *Aedes* (the primary vector for dengue). IVM should be enhanced to remove potential breeding sites, reduce vector populations, and minimize individual exposure. This should involve vector control strategies for larvae and adults (i.e., environmental management and source reduction, biological control, and chemical control measures), as well as strategies for protecting people and households. Bangladesh should implement the IVM strategy developed in 2021.

Vector control activities should focus on all areas where there is a risk of human-vector contact (place of residence, workplaces, schools and hospitals). Vector control activities can include covering, draining, and cleaning household water storage containers on a weekly basis. Space spraying with insecticide can be deployed as an emergency measure. Chlorination and application of suitable larvicides/

insecticides for water storage in outdoor containers should also be considered.

Personal protective measures during outdoor activities include the topical application of repellents to exposed skin or on clothing, and the use of long sleeve shirts and pants. Indoor protection can include the use of household insecticide aerosol products or mosquito coils. Window and door screens, as well as air conditioning, can reduce the probability of mosquitoes entering the house. Insecticide-treated nets offer good protection to people against mosquito bites while sleeping during the day. Since *Aedes* mosquitoes are active at dawn and dusk, personal protective measures are recommended particularly at these times of day.

Timely access to appropriate clinical management are key elements of care to reduce the risk for severe dengue complications and deaths due to dengue. Case surveillance should continue to be enhanced in all affected areas and across the country. Where feasible, resources should be allocated to the strengthening of laboratory sample referral mechanisms for the confirmation and sub-typing of the dengue virus.

In a bid to arrest the upsurge of dengue in our country in recent times rather than to focus primarily on patient-centred, curative and medicine-intensive disease management, approaches to improve environmental health and to manage vector habitats should be prioritized. Dengue vector control is an effective tool in reducing *Aedes* mosquito populations, particularly when control strategies utilize a community-based and integrated approach, combined with educational programmes to increase knowledge, awareness, attitudes and practices of people in the local community.

Taking a holistic perspective, for effective management of dengue, the coordinated and multidisciplinary efforts of different government and non goverment departments with regard to sanitation, urban development and education are essential. Increasing community awareness is also important, which can be done through local visits community healthcare workers, radio broadcasts with public/religious leaders and healthcare professionals to encourage the use of preventive methods, and TV as well as social media, particularly in urban areas. Moreover, local communities must be engaged to take active responsibility for their own protection by supporting elimination of Aedes breeding sites and taking personal measures towards prevention of infection, such as use of mosquito repellents.

a. Dr. Mainul Alam Chaklader; MPH, MBBS
Associate Professor, Department of Community Medicine
Bangladesh Medical College, Dhaka, Bangladesh
Email: dr.macmisha@gmail.com

b. Prof. Dr. Sharmeen Yasmeen; M. Phil, MPH, MBBS
Professor and Head, Department of Community Medicine
Bangladesh Medical College, Dhaka, Bangladesh

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Antimicrobial susceptibility pattern of *Escherichia coli* from clinical specimens in a tertiary care hospital, Dhaka, Bangladesh

Begum T[a], Chowdhury A[b], Ahmed SR[c], Iqbal H[d], Ahmed M[e], Nahar A[f]

Abstract

Background: *Escherichia coli* is one of the leading causes of mortality & morbidity throughout the world. The improper use of antibiotics leads to the emergence of drug resistant pathogens, which is a great public health challenge.

Objective: To determine antimicrobial susceptibility pattern of *Escherichia coli* from various clinical specimens in a tertiary care hospital, Dhaka, Bangladesh.

Methods: This retrospective cross-sectional study was conducted at Holy Family Red Crescent Medical College laboratory between July 2020 to June 2021 on 287 isolates of *E. coli* from various clinical specimens (urine, blood, sputum, wound swab/ pus) of patients. Written consent was taken from the concerned authority. The samples were processed according to the standard conventional methods and the isolates were identified by standard biochemical tests. Antimicrobial susceptibility testing was performed on Muller-Hinton agar using Kirby-Bauer disc diffusion method. The antibiotic discs were selected according to the protocol recommended by Clinical Laboratories Standards Institute (CLSI) guideline. Data ware analyzed by SPSS version 18**.**

Results: A total 287 (36.46%) *E. coli* were isolated from 790 of various clinical specimens collected from the patients of all age & both sex group attending various inpatient and outpatient departments. Out of 287 *E. coli* strains maximum sensitivity was shown against meropenem 90.94%, gentamicin 78.75%, nitrofurantoin 77.35% & amikacin 71.43%.

Conclusion: Nitrofurantoin, gentamicin & amikacin are suitable treatment options because of their high sensitivities and affordability. Although meropenem showed high sensitivity, its use should be restricted due to its status as a reserve drug. It is recommended to regular monitor antimicrobial susceptibility pattern for *E. coli* in order to improve treatment modalities and reduce drug resistance.

Keywords: *E. coli*, Meropenem, Gentamicin, Amikacin, Nitrofurantoin.

a. Dr. Taslima Begum; M. Phil, MBBS
Associate Professor, Department of Microbiology
Holy Family Red Crescent Medical College and Hospital, Dhaka

b. Dr. Afifah Chowdhury; MBBS
Ex- Intern, Bangladesh Medical College Hospital, Dhaka

c. Dr. Samia Rasna Ahmed; MBBS
Medical Officer, Department of Gastroenterology, Bangladesh Medical College Hospital, Dhaka

d. Dr. Hasina Iqbal; M. Phil, MBBS
Professor, Department of Microbiology
Popular Medical College, Dhaka

e. Dr. Mostaque Ahmed; M. Phil (Virology), FCPS
Professor & Head, Department of Microbiology
Popular Medical College, Dhaka

f. Dr. Azizun Nahar; M. Phil (BSMMU), MBBS
Associate Professor, Department of Microbiology Bangladesh Medical College, Dhaka

Correspondence to:
Dr. Taslima Begum; M. Phil, MBBS
Associate Professor, Department of Microbiology
Holy Family Red Crescent Medical College and Hospital, Dhaka
E mail: pannataslima@gmail.com

Introduction:

E. coli is a Gram-negative, rod-shaped, motile, oxidase negative, facultative anaerobic bacteria under the family Enterobacteriaceae.[1] It is a normal flora of the human and animal gastrointestinal tract, but can also be found freely in soil, water, and vegetation and can be a potential threat for various infections. *E. coli* is responsible for a wide variety of infections including both hospital and community onset infections.[2] *E. coli* is the most frequent causes of Urinary Tract Infection (UTI) and is among the most important pathogens causing Blood Stream Infections (BSI), nosocomial pneumonia, meningitis, wound infection & otitis media.[3-5] *E. coli* is a major cause of waterborne and foodborne human diarrhea worldwide, especially in developing countries, causing significant number of deaths, particularly in children under five-years-old.[6]

Urinary tract infection (UTI) is the most common infection of human population. Most susceptible groups are neonates, girls, young women and men. In case of adults, it occurs more commonly in women than men due to short length of female urethra, which is closer to anus. Therefore, up to 40% women develop UTI at least once during their lifetime; a significant number of these women suffer

recurrent UTI.[7] Around 90% *E. coli* infection was acquired from human community compared with 50% of nosocomial UTI.[8]

The improper use of antibiotics leads to the emergence of drug resistant pathogens, which is a great challenge to the health services. Different studies have been conducted time to time across the globe to assess the bacterial profile and the antibiotic susceptibility pattern in different samples. These studies help clinicians to decide on empirical treatment of patient before the results of confirmatory laboratory cultures.[9]

The emergence of multidrug- resistant *E. coli* is a growing problem around the world.[10] Treatment failure resulting in increased morbidity, mortality & cost of health services. Regular monitoring of antibiotic sensitivity report provides data for antibiotic therapy and resistance control. Thus, in this study, the aim is to determine antimicrobial susceptibility pattern of *E. coli* from various clinical specimens in a tertiary care hospital in Dhaka, Bangladesh

Materials & Methods:

The present study is a retrospective observational study and was conducted at Holy Family Red Crescent Medical College laboratory between July 2020 to June 2021. During the study period 287 isolates of *E. coli* were isolated from various clinical specimens (urine, blood, sputum & wound swab/ pus) obtained from the OPD & IPD patients of Holy Family Red Crescent Medical College Hospital, Dhaka, Bangladesh. Written consent was taken from the concerned authority. All isolates of *E. coli* isolated from various clinical samples were included in this study. All other bacteria except *E. coli* were excluded from the study.

The samples were processed according to the standard conventional methods and the isolates were identified by standard biochemical tests. Antimicrobial susceptibility testing was performed on Muller-Hinton agar using Kirby-Bauer disc diffusion method.[11] Antibiotic discs were selected according to the protocol as recommended by Clinical Laboratory Standards Institute (CLSI) guideline.[12] Nitrofurantoin was used only in urine samples. Data was analyzed by SPSS version 18.

Results:

A total 287 (36.46%) of *E. coli* were isolated from 790 clinical specimens, collected from inpatient and outpatient department patients from all age groups and both sex groups.

Table 1: Age and sex distribution of isolated *E. coli* (n=287)

Gender	Children up to 18 yrs No.	Adult patients No.	Total No. (%)
Male	32	92	124 (43.20%)
Female	14	149	163 (56.80%)
Total	46 (16.03%)	241 (83.97%)	287 (100%)

In Table 1, 241(83.97%) adult patients had *E. coli* infections in comparison to child group 46(16.03%). The highest number of female patients 163(56.80%) were suffered from *E. coli* infections than the male patients 124 (43.20%).

Table 2: *E. coli* isolated from different specimens (n=787)

Samples	No. of samples	Total Culture positive (%)
Urine	562	243 (43.24%)
Wound swab/pus	102	26 (25.49%)
Blood	85	10 (11.76%)
Sputum	38	8 (21.05%)
Total	787	287 (36.46%)

A total of 787 different kinds of samples were analyzed for isolation, identification and susceptibility pattern of *E. coli*. Among them culture positive *E. coli* was 287 (36.46%). Of these positive cases the isolation rate of *E. coli* is highest 243 (43.24%) in urine, followed by 26 (25.49%) in wound swab/ pus, (11.76%) in blood and 8 (21.05%) in sputum (Table 2).

Table 3: Antibiotic sensitivity pattern of *E. coli* (n=287)

Antibiotics	Disc potency	Sensitive	Percentage
AMC	30 µg	135	47.03%
CXM	30 µg	66	22.99%
CRO	30 µg	92	32.05%
CAZ	30 µg	78	27.17%
CFM	30 µg	126	43.90%
F/M	30 µg	222	77.35%
CIP	5 µg	116	40.42%
GN	10 µg	226	78.75%
AK	30 µg	205	71.43%
SXT	25 µg	157	54.70%
MEM	10 µg	261	90.94%

AMC- Amoxicillin/clavulanic acid, CXM- Cefuroxime, CRO- Ceftriaxone, CAZ - Ceftazidime, CFM- Cefepime, F/M- Nitrofurantoin, CIP- Ciprofloxacin, GN- Gentamicin, AK- Amikacin, SXT- Co- trimoxazole, MEM- Meropenem.

Out of 287 *E. coli* strains maximum sensitivity was shown against meropenem 90.94%, gentamicin 78.75%, nitrofurantoin 77.35% & amikacin 71.43%, followed by co-trimoxazole 54.70%, amoxicillin – clavulanic acid 47.03%, cefepime 43.90% and ciprofloxacin 40.42%. Very low sensitivity was found in ceftriaxone 32.05%, ceftazidime 27.17% & cefuroxime 22.99% (Table 3).

Discussion:

E. coli is one of the most common causative agents of bacterial infections. An antimicrobial resistance pattern of

E. coli is a great threat that leads to prolonged hospitalization and treatment failure. Therefore, this study aimed to determine antimicrobial susceptibility pattern of *E. coli* from various clinical specimens. In our study, adult patients 241(83.97%) had *E. coli* infections in comparison to child group 46(16.03%). *E. coli* infection is higher in female patients 163(56.80%) were suffered from *E. coli* infections than the male patients 124(43.20%) (Table 1). Similar observation was seen in a study where *E. coli* was common in female 58.06 % as compare to male 41.96%.[13] The isolation rate of *E. coli* in this study was 287(36.46%) (Table 2) which is inconsistent with findings reported by other researchers where they found 75.32% and 12.84% isolation rate of *E. coli*.[14,15]A low isolation rate of *E. coli* 14.2% was also identified in another study and samples were urine ear discharge, wound swab & eye discharge.[16] It may be variation of disease in different area.

In this present study, we found that meropenem was highly sensitive in 90.94% of cases. This is due to its limited use as it is reserve drug and expensive. Similarly, gentamicin (78.75%), amikacin (71.43%) and nitrofurantoin (77.35%) have good susceptibility rates, which may also be due to their limited use. Conversely, 2nd, 3rd, 4th generation cephalosporins, amoxicillin-clavulanic acid and cotrimoxazole due to their overuse (Table 3). Nitrofurantoin is relatively cheap, easily available drugs and showed higher sensitivity though it has some side effects. Ciprofloxacin showed low sensitivity due to overuse and misuse of this drug (Table 3). In a study, out of 62 *E. coli* strains maximum sensitivity was shown against nitrofuratoin 82.5% followed by meropenem 79.03%, ciprofloxacin 61.30%, amikacin 56.45%. Cefepime, ceftazidime, ceftriaxone showed lower sensitivity like 43.54%, 38.71% & 37.09%.[13] In another study from Bangladesh, *E. coli* showed highest susceptibility rates to meropenem, and amikacin (>90%), high susceptibility rates to amoxicillin-clavulanic acid, ceftazidime, and gentamycin (>70%).[15]

Conclusion:

Nitrofurantoin, gentamicin & amikacin are suitable treatment options because of their high sensitivities and affordability. Although meropenem showed high sensitivity, its use should be restricted due to its status as a reserve drug. It is recommended to regular monitor antimicrobial susceptibility pattern for *E. coli* in order to improve treatment modalities and reduce drug resistance.

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Original Article

Analysis of bleeding time and clotting time with ABO blood group among healthy adults: a cross-sectional study

Akter R[a], Musharraf I[b], Faruky SB[c], Akter T[d], Azim SF[e], Akhter QS[f]

Abstract

Background: Bleeding time (BT), clotting time (CT) and blood grouping are routinely done in physiology laboratory as part of haematology experiments. BT, CT and blood grouping are essential for surgeons and anesthetists prior to do any surgical procedure because of their association with hemostasis.

Objective: To determine the BT and CT and analyze their association with ABO blood group among healthy adults.

Methods: This cross-sectional study was conducted in the department of Physiology, Dhaka Medical College, Dhaka from July 2016 to June 2017. The study included 150 healthy subjects, who were 1st year MBBS students from 18 – 21 years' age group. After taking the informed consent bleeding time, clotting time and blood grouping of the subjects were determined. All collected data were documented in a prefixed questionnaire. Data analysis was done by SPSS (Statistical Package for Social Sciences) Version 22. Comparison between blood groups and BT, CT and their association was done by unpaired Students t-test.

Results: In this study mean age of the study subjects was 19.50 ± 0.82 years and 71(47.3%) were male & female were 79 (52.7%). Majority of the subjects had blood group B 57(38%) followed by O group 49(32.7%). The mean (±SD) bleeding time was 2.95 ± 1.00 minutes. The mean clotting time was 6.82 ± 0.85 minutes. BT and CT of blood group A was 2.53 ± 0.72 minutes & 6.52 ± 0.72 mintes, blood group B was 2.53 ± 0.90 minutes & 6.41 ± 0.51 minutes, blood group O was 3.85 ± 0.55 minutes & 7.58 ± 0.80 minutes and blood group AB was 2.20 ± 1.39 minutes & 6.33 ± 0.52 minutes. Statistically significant differences ($p \leq 0.001$) were observed among the bleeding and clotting time of O blood group with other blood groups (A, B and AB).

Conclusion: Blood group B is most common among the study subjects. Bleeding time and clotting time are significantly higher in O blood group than other blood groups (A, B, AB). Blood grouping, bleeding time & clotting time is mandatory before any surgical procedures because of their association with thrombosis & epistaxis.

Keywords: Bleeding time, Clotting time, Blood grouping, Medical students.

a. Dr. Raheena Akter; M. Phil, MBBS
Assistant Professor, Department of Physiology
Bangladesh Medical College, Dhaka

b. Dr. Ishtiaque Musharraf; MD (Nephrology), MBBS
Resident Physician, National Institute of Kidney Diseases and Urology, Dhaka

c. Dr. Salma Binte Faruky; M. Phil, MBBS
Associate Professor, Department of Physiology
Bangladesh Medical College, Dhaka

d. Dr. Tahmina Akter; M. Phil, MBBS
Assistant Professor, Department of Physiology
Dhaka Medical College

e. Dr. Sabrina Fahmida Azim; M. Phil, MBBS
Assistant Professor, Department of Physiology
Kumudini Women's Medical College, Mirzapur

f. Dr. Qazi Shamima Akhter; M. Phil, MBBS
Professor & Head, Department of Physiology
Dhaka Medical College, Dhaka

Correspondence to:
Dr. Raheena Akter; M. Phil, MBBS
Assistant Professor, Department of Physiology
Bangladesh Medical College, Dhaka
Email: raheenadr@gmail.com

Introduction:

Bleeding time is the time between the puncture of the blood vessel to the stoppage of bleeding. Normal bleeding time is 3 to 4 minutes. Bleeding time is affected by platelet function and activation, aggregation and coagulation. Bleeding time is increased in thrombocytopenia, disseminated intravascular coagulation (DIC) and also in Bernard-Soulier disease which is a rare autosomal recessive disorder. Clotting time is the time between the puncture of the blood vessel to the formation of the fibrin thread. Normal clotting time is 5 to 8 minutes. Clotting time is affected by clotting factors. Defect or absence of any clotting factor causes increase in clotting time.[1,2]

Determination of hematological parameters like bleeding time, clotting time and blood grouping is essential for surgeons and anesthetists prior to do any surgical procedure. Correlation of these parameters like BT, CT and blood grouping is important in case of epistaxis, thrombosis etc. Some studies have found that epistaxis in blood group O is more common as compared to other ABO blood groups.[3,4]

According to some researchers, the ABO blood group system influences the bleeding time and clotting time. They stated that vWF gene have been influenced by gene locus of ABO blood group on chromosome 9q34.[5,6] Person with blood group O has longer bleeding and clotting time compared to other ABO blood groups because of lower expression of Von Willebrand factor (VWF) in them. These individuals with O blood group have lower plasma VWF level due to faster hepatic clearance leading to shorter plasma half-life of VWF.[7] Von Willebrand factor is a large glycoprotein which is synthesized by Weibel-Palade bodies in the endothelial cells and alpha granules of megakaryocytes.[8] It is involved in hemostasis. Von Willebrand factor has two major roles in hemostasis. First, it helps in platelet adhesion and platelet aggregation. Second, VWF is the specific carrier of factor VIII (Anti-Hemophilic factor) in plasma. It protects factor VIII from proteolytic degradation and prolonging its half-life in circulation. Thus, effectively localizing it at the site of vascular injury .[9] So, the aim of this study was to determine the BT and CT and analyze their association with ABO blood group among healthy adults.

Materials & Methods:

TThis cross-sectional study was conducted in the Department of Physiology, Dhaka Medical College, Dhaka from July 2016 to June 2017. The study was conducted on 150 healthy subjects who were 1st year MBBS students with 18 – 21 years' age group. Bleeding time, clotting time and blood grouping were determined after taking informed consent from them. Statistical analysis was done by student's unpaired t test. ANOVA and Post Hoc test was done for assessing relationship between BT, CT and blood groups. Ethical clearance was taken from Research Review Committee and Ethical Review Committee of Dhaka Medical College, Dhaka. Study subjects were selected on the basis of inclusion criteria like age-group from 18-21 years, both sex and students with normal BMI. Exclusion criteria included: history of bleeding and clotting disorder, history of taking drugs that may influence bleeding/clotting time, eg; Warferin, Aspirin, Clopidogrel or NSAID, rare blood groups, suffering from any acute or chronic disease, abnormal BMI and h/o abnormal function of liver.

Study procedure

After selection of the subjects, the nature, purpose and benefit of the study was explained to each subject in details. They were encouraged for voluntary participation. They were allowed to withdraw from the study whenever they feel like. Informed written consent was taken from the participants. Before collecting blood sample, detailed family and medical history was taken. Anthropometric measurements of the subjects were done and blood pressure was measured. All the information's was recorded in a prefixed questionnaire. Blood grouping, bleeding time and clotting time were done in Department of Physiology, Dhaka Medical College, Dhaka. Height and weight were recorded in centimeter (cm) & in kilogram (kg). Body mass index of the subjects was calculated from measured height and weight using standard formula. BMI = Weight in kg/ Height in m². For data analysis SPSS (Statistical Package for Social Sciences) Version 22 was used. Results were presented as mean ± standard deviation (mean ± SD). Comparison between two groups was done by unpaired Students t-test, where applicable p value <0.05 was accepted as level of significance.

Results:

This study was conducted on 150 1st year medical students who were in 18-21 years' age group. Male was 71(47.3%) and female was 79(52.7%).

Table 1: General characteristics of the study subjects (n=150)

Parameters	Mean±SD	Range (min-max)
Age (years)	19.50 ± 0.82	18.00- 21.00
Height (cm)	161.46 ± 8.68	148.00- 179.50
Weight (kg)	59.25 ± 11.57	33.50- 96.00
BMI (kg/m^2)	20.69 ± 3.86	18.13- 24.73

Table 1 shows the age range of the study subjects was 18-20 years. The mean (± SD) age of the study group was 19.50±0.82 years. The mean (±SD) height of the study group was 161.46 ± 8.68 cm. The mean (±SD) weight of study subjects was 59.25±11.57 kg. The mean (± SD) BMI of the study group was 20.69 ± 3.86 kg/m2. All the values were within normal range.

Table 2: Blood group distribution of the subjects (n=150)

Blood group	No. of subjects	Percentage
A	38	25.3
B	57	38.0
O	49	32.7
AB	06	4.0
Total	150	100

Table 2 shows majority 57(38%)of the subjects had blood group B, followed by O group 49(32.7%), A group 38(25.3%). Only 6(4%) were in AB group.

Table 3: Bleeding time and clotting time of the subjects (n=150)

Parameters	Mean±SD	Range (min-max)
Bleeding time (min)	2.95 ± 1.00	1.00-4.30
Clotting time (min)	6.82 ± 0.85	6.00- 10.00

Mean bleeding time was 2.95 ± 1.00 minutes and mean clotting time was 6.82 ± 0.85 minutes as shown in Table 3.

Table 4: Bleeding and clotting time of the study subjects in different blood groups (n=150)

Blood group	Bleeding time (min.)	Clotting time (min.)
A (n=38)	2.53 ± 0.72	6.52 ± 0.72
B (n=57)	2.53 ± 0.90	6.41 ± 0.51
O (n=49)	3.85 ± 0.55	7.58 ± 0.80
AB (n=6)	2.20 ± 1.39	6.33 ± 0.52

Table 4 shows that the blood group O was found to have bleeding time 3.85 ± 0.55 minutes and that of clotting time were 7.58 ± 0.80 minutes. The blood group B had bleeding time 2.53± 0.90 minutes and that of clotting time was 6.41 ± 0.51 minutes, blood group A had bleeding time 2.53± 0.72 minutes and that of clotting time was 6.52 ± 0.72minutes, AB blood group had bleeding time 2.20± 1.39 minutes and that of clotting time was 6.33 ± 0.52 minutes.

Table 5: Statistical analysis between BT, CT and different blood groups

Blood groups	*p* value	
	Bleeding time	Clotting time
A vs B	0.996	0.366
A vs O	<0.001	<0.001
A vs AB	0.365	0.542
B vs O	<0.001	<0.001
B vs AB	0.415	0.737
O vs AB	<0.001	<0.001

Table 5 shows statistically significant differences ($p \leq 0.001$) among the bleeding and clotting time of O blood group with other blood groups (A, B and AB).

Discussion:

The present study was undertaken to observe blood group distribution and its relationship with bleeding time and clotting time. For this study, a total number of 150 male and female students with age ranging from 18 to 21 years were considered. In the study group, height and weight were measured to calculate their body mass index (BMI). Blood group was determined to observe its distribution. Bleeding time and clotting time were estimated to see its relationship with blood group.

In the present study, all the parameters in adult healthy subjects were within reference value, similar findings were observed by the various investigators from different countries .[2,1,10,11] In this study, age and BMI of all the subjects were almost similar (Table-1). The blood group B was most common in study subjects (Table 2). This finding agreed with the study of many researchers of different country.[10,12,13] On the contrary, Akhter et al., Mridha and Jena found different distribution among blood groups. This disagreement in findings might have occurred due to racial and geographical variation. [11,14]

In the present study, the mean BT and the mean CT were longer in blood group O (Table-4). Almost similar types of results of bleeding time and clotting time in blood group O were found by many researchers.[1,2,10-12] However, some researchers found prolonged bleeding time in blood group B and blood group AB .[15,16,17,8] This dissimilarity might be due to variation in plasma vWF level which was genetically determined. On the other hand, a study showed that CT was longer in blood group B.[18] Prolonged CT in AB blood group was also found.[15] These findings might have occurred due to variation of plasma vWF in different race. Here, in this study, statistically significant differences ($p \leq 0.001$) among the bleeding and clotting time of O blood group with other blood groups (A, B and AB) were found (Table-5). Similar finding was found only in case of clotting time where it was statistically significant only between group O & group A, with higher value in group O but in bleeding time no statistical significance was found.[19] In a study, bleeding time of >4 minutes was found in both O and B group but the result was not statistically significant (p=0.85) & clotting time of >6 minutes was found again in both O and B groups and no statistical difference was found (p=0.96).[20] Clotting time was found more in blood group AB and bleeding time in blood group B than other blood groups which was statistically significant.[16]

In this study the sample size was less, so further research should be performed with larger sample size. Analysis with other blood group system may also necessary because we only considered ABO blood group. Plasma vWF levels should be estimated to rule out any reasons for the difference clotting and bleeding time among ABO blood groups. Further studies can be conducted using other test method such as estimation of prothrombin time, activated partial thromboplastin time and determination of coagulation factor which should give more accurate results.

Conclusion:

After analyzing the results of our study, it is concluded that blood grouping, bleeding time & clotting time is mandatory before any surgical procedures because of their association with thrombosis & epistaxis. Blood group B is most common among the study subjects. Bleeding time and clotting time are significantly higher in O blood group than other blood groups (A, B, AB). This study will help to bring awareness among the individuals about their inherited risk of bleeding tendency.

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Factors influencing developmental outcome following developmental therapy in children with cerebral palsy

Arefin KE[a], Lucky MA[b], Bhuiyan ME[c], Karim ME[d], Ali ML[e], Rahman MM[f]

Abstract

Background: Cerebral palsy (CP) describes a group of motor syndromes resulting from disorders of early brain development. In Bangladesh a single approach therapy has been adopted in many medical institutes as developmental therapy where a developmental therapist assesses the child and provides physiotherapy, occupational therapy, cognitive stimulation, feeding therapy, speech and language therapy to the children according to the need by a holistic approach.

Objective: This study was designed to determine the factors influencing developmental outcome following 6 months developmental therapy in children with cerebral palsy.

Methods: A quasi-experimental study was conducted in the department of Paediatrics, Bangabandhu Sheikh Mujib Medical University on 135 children with CP aged 6 months to 3 years. The motor, mental and behavioral functions of the selected children were initially measured by Bayley Scales of Infant Development II. They were reassessed after 3 months and 6 months following "developmental therapy". Only 62 (45.9%) children have completed the 2 follow-up sessions and hence were evaluated against their baseline motor, mental and behavior score. Data analysis were done by Friedman test, Wilcoxon signed rank test, Chi square test, Fisher's exact test and Unpaired T test. For above tests, significance level α was set at 5% so *p* value <0.05 was considered as significant.

Results: Among 135 children only 62 children completed 2 follow-ups. Mean age of 62 children was 15±8.4 months; male was 66% and female was 44%. After developmental therapy for 6 months 46.8% the children had improved motor skills, 40.3% mental skills and about 69 % behavioral skills; presence of epilepsy, microcephaly and significant illness affected the outcome. Early age, duration and intensity of developmental therapy showed positive impact on the outcome in children with cerebral palsy.

Conclusion: Developmental therapy should be started sooner after the diagnosis of a child with CP and intensity of therapy should as much as possible.

Keywords: Cerebral palsy, Developmental outcome, Developmental therapy.

a. Dr. Khondoker Ehsanul Arefin; MD, MPH, MBBS
Associate Professor, Department of Paediatrics
Bangladesh Medical College, Dhaka

b. Dr. Mohsina Akter Lucky; MD, MPH, MBBS
Associate Consultant, Department of Paediatrics, MEDIX,
United Health Care, Dhaka

c. Dr. Md Elias Bhuiyan; FCPS, MRCP, MBBS
Assistant Professor, Department of Medicine
Bangladesh Medical College, Dhaka

d. Dr. Mohammad Enamul Karim; MD, FCPS, MBBS
Assistant Professor, Department of Medical Gastroenterology
Sheikh Russel National Gastroliver Institute and Hospital,
Mohakhali, Dhaka

e. Dr. Md. Liaquat Ali; MD, MBBS
Associate Professor, Department of Paediatrics
North Bengal Medical College, Sirajganj

f. Prof. Dr. Md. Mizanur Rahman; FCPS, MBBS
Professor of Paediatric Neurology (Retd.), Department of
Paediatrics, Bangabandhu Sheikh Mujib Medical University
(BSMMU), Dhaka.

Correspondence to:
Dr. Khondoker Ehsanul Arefin; MD, MPH, MBBS
Associate Professor, Departmen of Paediatrics
Bangladesh Medical College, Dhaka
Email: drarefin@yahoo.com

Introduction:

Cerebral palsy (CP) describes a group of motor syndromes resulting from disorders of early brain development.[1] The prevalence of cerebral palsy in the world is about 1.6/1000 live birth.[2] In Bangladesh the prevalence is about 3.4/1000 live birth.[3]

Various combination therapies are now practiced in abroad in different names. In Bangladesh a combination therapy named developmental therapy has been advocated for the management of these children. Developmental therapy is the combination of physiotherapy, occupational therapy, cognitive stimulation, and speech and language therapy. It is principally a home based therapy where mother or caregiver plays a role of therapist. She comes to the centre with her children periodically for therapy and learns how to give the therapy to her children at home.[4] It is very much effective where parents are used as therapist. Maximum effectiveness is achieved when parental skills are increased.[5,6] There are some factors that might influence the developmental outcome following developmental therapy. Studies showed that age, epilepsy, intensive therapy, awareness about the condition of CP interfered developmental outcome.[7-11]

There are limited published evidence in this aspect so the present study was conducted on children with CP to find out the associated factors that may influence the developmental outcome following developmental therapy.

Materials & Methods:

It was a quasi-experimental study done in Child Neurology Centre of Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh. A total of 135 children with CP from 6 months to 3 years of age were taken as study population**.** Convenience sampling method was applied. Informed written consent was taken from the caregiver/parents of the patients. Ethical clearance was obtained from the institutional ethical committee of the university. Children with CP who have visual, hearing impairment and who had prior developmental therapy were excluded from the study. Categories of CP and their associated problems like epilepsy, behavioral problem were assessed and recorded. Any illness which occurred in a single or multiple episodes causing interruption of developmental therapy for 15-30 days (total) within 6 months' period were considered as significant illness which included respiratory infection and diarrhoeal diseases. After giving primary management of associated problems by pediatricians, their motor, mental and behavioral status were assessed by the investigators assisted by the psychologist at every visit at three-month interval. After first assessment, counseling of the parents was done. Parents counseling was the major part of the management of children with CP because they would have been the central focus part of the developmental therapy as they had to give the therapy their children at home regularly. Data were entered and analyzed using the software "Statistical Package for Social Science" (SPSS version 18.0 for Windows, IBM, Chicago, IL). Friedman test, Wilcoxon signed rank test, Chi square test, Fisher's exact test and Unpaired T test were done. For above tests significance level α was set at 5% so *p* value <0.05 was considered as significant.

Assessment Tools

Assessment was done by Bayley Scales of Infant Development II (BSID II)[12], administration time for under 14 months was 25-35 minutes and above 15 months up to 60 minutes. The improvement of motor, mental and behavioral functions were assessed as follows;

Firstly, the deviations between the observed scores of the children at baseline and the reference median scores of normal children (obtained from BSID II) of the same age were calculated. The deviations were then converted into percentage. Similarly, the percentage of deviation after 6 months of therapy was calculated with respect of normal children (obtained from BSID II) of the same age. The percentage of deviation after 6 months of therapy was then subtracted from that of the baseline. If the percentage of gap was reduced by >5, the child was considered as "improved" and If the percentage of gap was reduced by ≤5, the child was classified as "not improved".

Follow Up

Parents with each child was asked to come for developmental therapy at 1-month interval. Their motor, mental and behavioral status was assessed by the principal investigator assisted by developmental psychologist in every 3 months' interval and improvement of outcome were assessed. The growth and development and general health measures were monitored by the principal investigator. If any problem or illness was identified in any visit; steps were taken to conduct appropriate investigations (USG, CT or MRI of brain, EEG, Complete blood count etc.) and to provide intervention, advice and specific counseling to the parents. Total 6 month follow up was done.

Results:

A total of 135 children with CP attended the Pediatric Neurology outpatient department but only 62 (45.9%) completed 2 follow up sessions and hence were evaluated against their baseline motor, mental and behavior score.

Table 1: Demographic characteristics of children with CP (n=62)

Characteristic	Frequency	%
Age in months		
06-18	36	58.07
19-36	26	41.93
Mean age in months	15±8.4	
Age range in month	6-36	
Sex		
Male	41	66
Female	21	34
Monthly Income		
< 6000 tk	39	62.9
6000-12000 tk	16	25.8
>12000 tk	7	11.3
Mothers' education		
Illiterate	5	8
Primary education	32	51.6
SSC and HSC	17	27.4
Graduate and above	8	12.9

Baseline characteristics shows most of the children 36(58.07%) in this study were between 6-18 months; mean age was 15.37± 8.4 months. Over two- third 41(66%) were male (male: female ratio 2:1). In terms of income by parents, over two-third 39(62.9%) had earnings of <6000 taka. Mother's education level demonstrated that about 51.6% of the mothers had primary level educated as shown in Table 1.

Table 2: Distribution of children by developmental outcome (n=62)

Outcome	Frequency	Percentage
Motor function improved	29	46.8
Mental function improved	25	40.3
Behavioral function improved	43	69.3

Table 2 shows that nearly half 29(46.8%) the children had improved motor skills, 25(40.3%) improved mental skills and about 43(69 %) improved behavioral skills.

Table 3: Outcome following developmental therapy (n=62)

Outcome variables	Evaluation at			
	Baseline	Month 3	Month 6	*p* value
Motor score	29.0	34.50	48.0	< 0.001
Mental score	60	68	72.50	< 0.001
Behavior score	91	97.50	104	< 0.001

Table 3 shows that as outcome of developmental therapy median motor score of 62 children was increased slowly from 29 at baseline to 34.50 at month 3 and to 48 at month 6 (p<0.001). The mental score was increased from 60 at baseline to over 68 at 3 and 72.50 at 6 months (p<0.001). Significant improvement was also noted at 6 month in behavior score.

Friedman test was done to see if there was any significant difference among the baseline, 3 months and 6 months' score of motor, mental and behavioral function.

Table 4: Association between demographic variables and motor outcome (n-62)

Demographic variables	Outcome		*p*-value
	Improved (n = 29)	Not improved (n = 33)	
Age (months)			
06-18	21 (72.4)	15 (41.7)	0.032
19-36	08 (27.6)	18 (69.3)	
Sex			
Male	18 (62.1)	23 (69.7)	0.527
Female	11 (37.9)	10 (30.3)	
Socioeconomic status			
Poor	19 (65.5)	20 (60.6)	0.589
Middle class & rich	10 (34.5)	13 (39.4)	
Residence			
Urban	15 (51.7)	11 (33.3)	0.143
Rural	14 (48.3)	22 (66.7)	

Data were analyzed using Chi-square Test. Figures in the parentheses denote corresponding percentage.

Association between 4 demographic variables and motor outcome following developmental therapy reveals only age was found to be associated with outcome of developmental therapy (Table 4).

Table 5: Anthropometric variables and developmental outcome (n=62)

Anthropometry	Developmental outcome					
	Motor		Mental		Behavior	
	Improved	Not improved	Improved	Not improved	Improved	Not improved
3rd degree malnutrition* n_1=5	2 (40)	3 (60)	1 (20)	4 (80)	4 (80)	1 (20)
p value	**1.0**		**0.640**		**0.511**	
Microcephaly # n_2=22	5 (22.7)	17 (77.3)	4 (18.1)	18 (81.9)	9 (40.9)	13 (59.1)
p value	**0.005**		**0.008**		**0.001**	

* Data were analyzed using **Fisher's Exact Test**

Data were analyzed using **Chi-square Test**; Figures in the parentheses denote corresponding percentage.

Of the two anthropometric variables, the frequency of microcephaly was 22 and 3rd degree malnutrition was 5 out of 62 children. Microcephaly was less in the children who exhibited significant improvement 5 (22.7 %) in motor function compared to those who did not show any improvement 17(77.3%) where p=0.005. In mental outcome the frequency of microcephaly was much less in the children who exhibited significant improvement 4 (18.1%) compared to those who did not show any improvement 18 (81.9 %) where p=0.008. Regarding behavioral outcome, the frequency of microcephaly was less in the children who exhibited significant improvement 9 (40.9 %) compared to those who did not show any improvement 13(59.1 %) where p = 0.001.

Differences were also compared with nutritional status. As sample size was small for undernourished group, no statistical significant differences were found (Table 5).

Table 6: Influence of epilepsy on developmental outcome (n=62)

Epilepsy	Developmental outcome					
	Motor		Mental		Behavior	
	Improved	Not improved	Improved	Not improved	Improved	Not improved
Present	2 (40)	3 (60)	1 (20)	4 (80)	4 (80)	1 (20)
Absent	5 (22.7)	17 (77.3)	4 (18.1)	18 (81.9)	9 (40.9)	13 (59.1)
p value	**0.001**		**0.041**		**0.025**	

Data were analyzed using Chi-square Test; Figures in the parentheses denote corresponding percentage.

Majority of the children 14 (87.5 %) who had epilepsy did not improve in motor function following "developmental therapy" (p= 0.001). Similar result was evident in mental function where 81.2% did not show any improvement (p=0.04). Regarding behavioral function improvement was seen more of the epileptic children than that of motor and mental function. However, there was significantly more improvement in children without epilepsy (p= 0.02) as shown in Table 6.

Table 7: History of significant illness that interfered with developmental therapy and developmental outcome

History of significant illness	Developmental outcome					
	Motor		Mental		Behavior	
	Improved	Not improved	Improved	Not improved	Improved	Not improved
Present	05 (26.4)	14 (73.6)	4 (21.1)	15 (78.9)	11 (57.7)	08 (42.3)
Absent	24 (55.8)	19 (44.1)	21 (48.8)	22 (51.1)	35 (81.3)	08 (18.6)
p value	**0.032**		**0.040**		**0.002**	

Data were analyzed using Chi-square Test; Figures in the parentheses denote corresponding percentage.

Table 7 shows 19 children had different significant illness that interfered with developmental therapy. Of them only 5 (26.4%) had improved motor function during study period, whereas 43 children did not suffer from any significant illness, amongst them 24 (55.8%) had improved motor function (p= 0.032). In mental outcome 4 (21.1%) children who suffered from significant illness had improved during study period. Whereas 43 children who did not suffer from any significant illness, amongst them 21 (48.8%) had improved in mental function (p = 0.040). In behavioral outcome 11 (57.7%) children had improved during study period suffered from significant illness, whereas 43 children did not suffer from any significant illness, amongst them 35 (81.3) children had improved the behavioral function (p = 0.002).

Table 8: Impact of duration of therapy at home on developmental outcome

Duration of therapy in a week	Developmental outcome					
	Motor		Mental		Behavior	
	Improved	Not improved	Improved	Not improved	Improved	Not improved
Mean hours	8.18	4.97	8.56	5.06	7.45	4.26
SD	3.91	2.62	3.57	2.99	3.64	2.55
p value	**< 0.001**		**< 0.001**		**< 0.001**	

Data were analyzed using unpaired t-Test and were presented as mean ± SD.

The mean duration of developmental therapy in a week was significantly longer as 8.18 hours, 8.56 and 7.45 hours in children who demonstrated improvement in motor, mental and behavioural function ($p < 0.001$) respectively than that in children who did not show any improvement in the above three areas with less duration of developmental therapy as shown in Table 8.

Discussion:

Total 135 cases were included initially in the study. The guardians (particularly mothers) were asked to bring their children at 3 monthly intervals for assessment up to 6 months following therapy. Only 62 (45.9%) completed 2 follow-up sessions and hence were evaluated against their baseline motor, mental and behavior functions.

After giving "developmental therapy", nearly half (46.8%) of the children were improved in motor function, 40.3% in mental function and about 69 % in behavioral functions (Table 03). The behavioral function was improved more in relation to the mental function. This might be explained that the children were neglected before "developmental therapy". When they got extra care from their mother and family members the behavioral function was the first thing that changed in higher proportion.

Similar findings were found in a retrospective study done by Jahan et al. to evaluate the effectiveness of developmental therapy in children with cerebral palsy and other disabilities. Total 100 children were included in the study. The result showed most of the children improved their function slowly in various aspect of development. The maximum improvement was seen in gross motor function (36%), 12% in cognitive function and least in fine motor function (7%). The behavioral function was not assessed in the above study.[13]

In the present study, age group from 6 to 18 months showed more improvement after developmental therapy compared to the age group from 19 to 36 months group (Table 04). Hong shows similar findings in a study which was designed to identify factors influencing the short-term effect of intensive therapy on gross motor function in children with cerebral palsy. The study included 145 cases who received 8 weeks intense therapy. Measurements of gross motor functions were performed using the gross motor function measure-88 (GMFM-88) and gross motor function classification system (GMFCS), and were obtained at the start and end of the course. Result showed that age ≥ 36 months were significantly associated with a poor response after intensive therapy.[8]

Microcephaly was present in 22 (35.4 %) children. Majority of the children who did not show any improvement had microcephaly (Table-5) compared to those who improvement in motor, mental and behavioral function ($p < 0.01$). There are some studies that support these findings. Gordon showed that postnatal etiologies of microcephaly and infants with comorbid epilepsy had worse outcomes.[10] Messerschmidt did a case control study to observe the neurodevelopmental outcome in preterm babies suffering from disrupted cerebellar development. Thirty-one preterm patients with disrupted cerebellar development were taken as cases and a control group of thirty-one gender and gestational age matched premature infants with normal cerebellar development were included in the study. CP was diagnosed in 48% of affected patients later on whereas none of the patients of the control group had CP. Microcephaly was significantly related to disrupted cerebellar development and poor neurodevelopmental outcome.[14] Watemberg et al. did a study to assess the clinical impact of microcephaly among children with developmental disabilities, they reviewed 1393 patients from birth to 5 years of age. Mental retardation was significantly more common among microcephalic patients with cerebral palsy than among normocephalic ones ($p = 0.004$).[15]

One-fourth of the children 16(25.8 %) had history of epilepsy. Majority of the children who had epilepsy did not improve in motor, mental and behavior function following "developmental therapy" compared to children who showed improvement (Table-6). The association between ongoing seizures and cognitive and behavioral regression has been demonstrated convincingly.[9,11,13,16,17,18] Moreover antiepileptic drugs have a detrimental effect on the central nervous system and may affect mood, cognitive and behavior function.[16] Though the epilepsy affect only cognition and behavior function in the above studies but in the present study mental and behavior along with motor outcome was also influenced by epilepsy.This might be explained that epilepsy interfered "developmental therapy" in our children.

In the present study recurrent seizure (epilepsy), recurrent respiratory tract infection and dirrhoea, which were considered as significant illness influenced the developmental outcome. Over one-third of the children who did not improve in motor and mental function and over half of the children who did not improve in behavior function suffered from significant illnesses during study period ($p < 0.05$) shown in Table-7.This may suggest that the significant illness might interfered "developmental therapy" and then developmental outcome.

The present study considered the duration of "developmental theapy" at home as an important associated factor that influenced developmental outcome.The mean duration of "developmental therapy" in a week was significantly longer in children who demonstrated improvement in motor, mental and behavioral function than those who did not show improvement ($\boldsymbol{p} < 0.001$) according to Table-8. There are some studies those suggest the benefit of intensive therapy for CP children. Tsorlakis et al. did a randomized control trial to examine the effect of neurodevelopmental therapy (NDT) and differences in its intensity on gross motor function of 34 children with cerebral palsy. Participants were assigned randomly to two groups, Group A was designed to receive NDT for 16 weeks, twice weekly, for 50 minutes each session. Group B was the intensive group and followed the NDT program for 16 weeks, five times weekly, for 50 minutes each session. Result showed children in group B performed better and showed significantly greater improvement than those in group A ($\boldsymbol{p} < 0.05$).[19] This study underlined the need for intensive application of the therapy.

Stiller did a study to compare the effects of intensive therapy, conductive education and special education services for a group of 19 children diagnosed with cerebral palsy. Results of this study found that physical gains were greatest in children who received intensive physical, occupational, and speech therapy over a five-week period. In addition, children in all groups showed some improvement in physical functioning in spite of the short duration of the study.[20] This also confirm that duration and intensity of developmental therapy affect the outcome significantly.

Conclusion:

The present study observed that presence of epilepsy, microcephaly or significant illness that interfered "developmental therapy" may affect the outcome. Early age, duration and intensity of developmental therapy has positive impact on the outcome. Despite of the high attrition rate (54%), short intervention period for evaluation and variability of response from the parents, it could be concluded that developmental therapy should be started sooner after the diagnosis of a child with CP and intensity of therapy should as much as possible. To lower attrition rate, parents need counseling, regular updating regarding the evaluation report of the children and subsidization of financial charges for service delivery might be other important aspects to be addressed.

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Original Article

Serum ferritin level of type 2 diabetic patients in a tertiary care hospital in Dhaka city

Khondker F[a], Khanduker S[b], Ahmed R[c], Biswas S[d]

Abstract

Background: Serum ferritin, a marker of body iron stores, has been implicated in the pathogenesis of type 2 diabetes mellitus (T2DM). However, the relationship between serum ferritin levels and T2DM remains unclear, particularly in the Bangladeshi population.

Objective: To determine association between serum ferritin levels and T2DM.

Methods: This case-control study conducted at the Department of Biochemistry, Sir Salimullah Medical College and Mitford Hospital, Dhaka, Bangladesh over a period of 2 years. It included 92 Bangladeshi adults, with 46 T2DM patients and 46 age and gender-matched controls. Serum ferritin, fasting plasma glucose (FPG), and other biochemical parameters were measured. Correlations between serum ferritin and other variables were assessed using Pearson's correlation test, and risk assessment was conducted using odds ratios (OR).

Results: Significantly higher serum ferritin levels were found in T2DM patients compared to controls (197.97 ± 75.99 µgm/L vs. 64.24 ± 27.83 µgm/L; $p < 0.001$). Serum ferritin levels were positively correlated with FPG ($r = 0.637$, $p = 0.001$) in T2DM patients, but not in controls. Participants with serum ferritin levels ≥151 µgm/L had an OR of 11.64 for T2DM ($p < 0.001$). No significant correlations were observed between serum ferritin and age or BMI in either group.

Conclusion: The present study supports the association between elevated serum ferritin levels and type 2 diabetes mellitus *(*T2DM*)*. Higher serum ferritin levels were linked to poorer glycemic control and increased T2DM-related complications. The relationship between serum ferritin and T2DM appears to be independent of BMI and age. Further research is needed to confirm these findings and investigate the underlying mechanisms.

Keywords: Serum ferritin, Type-2 Diabetes Mellitus, Obesity, Fasting plasma glucose.

Introduction:

Type 2 diabetes mellitus (T2DM) is a global public health concern, affecting millions of people worldwide.[1,2] The International Diabetes Federation (IDF) estimates that approximately 536 million adults were living with diabetes in 2021, and this number is projected to rise over 700 million by 2045. [2, 3] The increasing prevalence of T2DM poses significant challenges to healthcare systems, as it is associated with increased morbidity, mortality, and reduced quality of life.[4,5] Among the various forms of diabetes, T2DM constitutes about 90% of all cases.[6,7] Prevalence is particularly high in Asia, that is 60% or more [8,9] Changes in dietary habits and sedentary lifestyles are some of the contributing factors for this desease.[10,11] The escalating diabetes epidemic in Bangladesh demands urgent attention to identify the underlying risk factors and implement appropriate interventions.[12] T2DM is a complex metabolic disorder characterized by insulin resistance and relative insulin deficiency.[13,14] Various genetic, environmental, and lifestyle factors have been implicated in the development and progression of T2DM. Some of the established risk factors include obesity, physical inactivity, family history of diabetes, and age.[15-17] Additionally, ethnicity also plays a crucial role in T2DM, with South Asians being at a higher risk than their Caucasian counterparts.[18-20] Serum ferritin, an iron-storage protein, has recently emerged as a potential biomarker for T2DM.[21,22] Elevated serum ferritin levels have been reported in several studies as a risk factor for the development and progression of T2DM, independent of traditional risk factors such as body mass index (BMI) and family history.[23] Iron overload, which can result in increased serum ferritin levels, has been associated with

a. Dr. Farhana Khondker; MBBS, MD
Associate Professor (CC), Department of Biochemistry
Anwer Khan Modern Medical College, Dhaka

b. Dr. Sadia Khanduker; MBBS, MD
Associate Professor, Department of Biochemistry
Bangladesh Medical College, Dhaka.

c. Dr. Rumana Ahmed; MBBS, MD
Associate Professor, Department of Biochemistry
Ahsania Mission Medical College, Dhaka

d. Dr. Sumona Biswas; MBBS, MD
Associate Professor, Department of Biochemistry
Laboratory Service Division, Sheikh Hasina National Institute of Burn and Plastic Surgery, Dhaka.

Correspondence to:
Dr. Farhana Khondker; MBBS, MD (Clinical Biochemistry)
Associate Professor (CC), Department of Biochemistry
Anwer Khan Modern Medical College, Dhaka
Email: farhanakhondker1977@gmail.com

insulin resistance and impaired glucose metabolism. [24-25] Furthermore, excess iron may promote oxidative stress, leading to tissue damage and exacerbating the complications associated with T2DM. The disease is associated with various microvascular and macrovascular complications, including retinopathy, nephropathy, neuropathy, and cardiovascular diseases.[26,27] These complications can result in long-term disability, reduced life expectancy, and increased healthcare costs. So, the high prevalence of T2DM in Bangladesh and the potential link between serum ferritin levels and the disease, is imperative to investigate the relationship between these factors. The present study aimed to assess serum ferritin levels in T2DM patients and explore the potential association with various clinical and demographic factors. Understanding the role of serum ferritin in T2DM may provide valuable insights into the pathophysiology of the disease and contribute to the development of targeted interventions to improve the management and prevention of T2DM in this vulnerable population.

Materials & Methods:

This case-control study was conducted at the Department of Biochemistry, Sir Salimullah Medical College and Mitford Hospital, Dhaka, Bangladesh. The study duration was 2 years, from July 2013 to June 2015. During this period, a total of 92 participants were divided into case and control groups based on the individual inclusion and exclusion criteria. Forty-six patients who were diagnosed with type 2 diabetes following the ADA criteria were selected as the case group, while another 46 age and gender matched non-diabetic participants were selected as the control group. Purposive sampling technique was used for the selection of the participants. Age of the present study participants ranged between 35 to 65 years, Patients with chronic kidney disease, liver disease, anemia, ailments associated with altered serum ferritin levels and other chronic diseases were excluded from the study. Informed consent was obtained from all the participants regarding their participation in the present study, and ethical approval was also obtained from the ethical review committee of the study hospital. Information was collected and recorded in a structured interview schedule. The collected and analyzed data were expressed as the mean ± SD (standard deviation). Data had been analyzed by SPSS (Statistical Package for Social Sciences) computed program [*version 20*]. Students 't' test, Pearson's Correlation test, Chi Square test & Odds Ratio was used to do the statistical analysis. A two-tailed *p*-value of <0.05 was considered statistically significant.

Results:

Table 1: Distribution of participants by baseline demographic characteristics

Characteristics	Case (n=46)	Control (n=46)
Age (mean±SD)	54.91±6.46	53.19 ±7.31
Gender		
Male	26 (56.5%)	27 (58.7%)
Female	20 (43.5%)	19 (41.3%)
Anthropometric measurements		
Weight (kg)	74.689 ±11.90	72.77±11.92
Height (cm)	163.15 ± 10.80	163.28± 9.68
BMI (kg/m²)	27.95±2.21	27.19±2.98

The mean age of the case group was 54.91 ± 6.46 years, while the control group's mean age was slightly lower at 53.19 ± 7.31 years. Gender distribution was similar in both groups, with males constituting 56.5% (n=26) of the case group and 58.7% (n=27) of the control group. Females accounted for 43.5% (n=20) of the case group and 41.3% (n=19) of the control group. The case group's mean weight was 74.689 ± 11.90 kg, while the control group's mean weight was slightly lower at 72.77 ± 11.92 kg. Height measurements were nearly identical, with the case group's mean height at 163.15 ± 10.80 cm and the control group's at 163.28 ± 9.68 cm. The mean BMI of the case group was 27.95 ± 2.21 kg/m², higher than the control group's mean BMI of 27.19 ± 2.98 kg/m. ²

Table 2: Biochemical parameters of the study groups

parameters	Case (n=46)	Control (n=46)	*p* value
	Mean+SD	Mean+SD	
FPG (mmol/L)	7.88±1.57	5.09±0.65	0.001
S. Ferritin (µgm/L)	197.97±75.99	64.24±27.83	0.001
Hb (gm%)	13.07±1.27	13.42±0.98	0.726

The case group had significantly higher mean FPG (7.88 ± 1.57 mmol/L) and HbA1c (8.53 ± 2.31%) levels compared to the control group's mean FPG (5.09±0.65 mmol/L) A substantial difference in serum ferritin levels was observed between the groups, with the case group's mean level (197.97±75.99 µgm/L) being significantly higher than the control group's (64.24±27.83 µgm/L; *p*-value <0.001), suggesting a potential association with T2DM. However, mean hemoglobin levels were comparable between the case group (13.07±1.27 gm%) and the control group (13.42 ± 0.98 gm%; *p*-value = 0.726).

Table 3: Association of serum ferritin level with gender among the study subjects

Gender	Serum Ferritin Levels		*p* value
	Case (n=46)	Control (n=46)	
	Mean+SD	Mean+SD	
Male	216.36 ±68.34	68.62 ±30.20	**<0.001**
Female	176.08 ±80.40	58.02 ±23.45	

**showing that the ferritin level is higher in DM patients irrespective of sex

In male participants, the mean serum ferritin level in the case group was significantly higher at 216.36 ± 68.34 µgm/L compared to the control group, which had a mean level of 68.62 ± 30.20 µgm/L (*p*-value < 0.001). Similarly, female participants in the case group exhibited a higher mean serum ferritin level of 176.08 ± 80.40 µgm/L compared to the control group, which had a mean level of 58.02 ± 23.45 µgm/L.

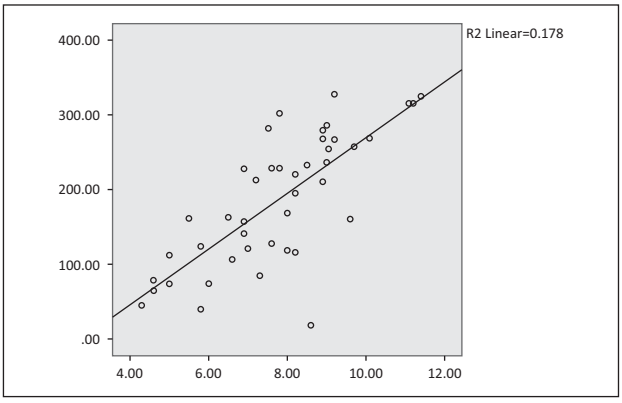

Figure 1: Correlation between serum ferritin and FBG in cases

The given graph clearly represents that the two variables, serum ferritin and Fasting blood glucose levels are positively and significantly correlated, among diabetic patients.

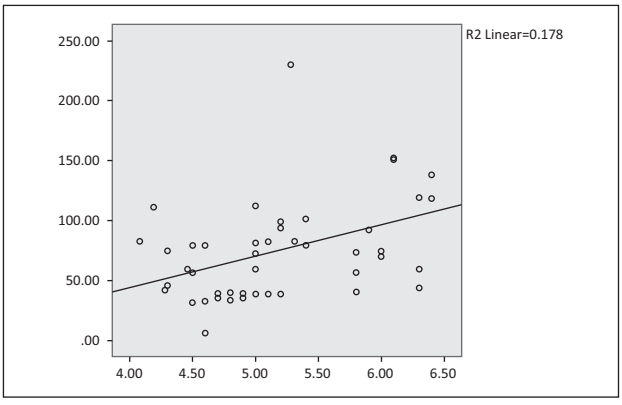

Figure 2: Correlation between serum ferritin and FBG in controls

The given graph shows that the control group participants had positive correlation between serum ferritin and FBG, but this correlation was not significant (Fig-2).

Table 4: Risk measurement of serum ferritin in type 2 DM

Serum Ferritin (µgm/L)	Case (n=46)	Control (n=46)	OR	*p* value
≥151	34 (73.91%)	9 (19.57%)	11.64	11.64
≤150	12 (26.09%)	37 (80.43%)		

Table 4 shows in the case group, 34 participants (73.91%) had serum ferritin levels ≥151 µgm/L, while only 9 participants (19.57%) in the control group had levels within the same range. The odds ratio (OR) for having type 2 DM with a serum ferritin level ≥151 µgm/L was calculated to be 11.64, with a *p*-value <0.001, indicating a statistically significant association between elevated serum ferritin levels and the presence of type 2 DM. In contrast, among the subjects with serum ferritin levels ≤150 µgm/L, only 12 participants (26.09%) were in the case group, whereas 37 participants (80.43%) were in the control group.

Discussion:

The present study aimed to investigate the potential association between serum ferritin levels and type 2 diabetes mellitus (T2DM) in our study subjects. The study found significant association in fasting plasma glucose (FPG) and serum ferritin levels between the case and control groups, while hemoglobin levels remained similar between the groups. The demographic characteristics and anthropometric measurements of the participants were comparable, minimizing potential confounding factors. We have selected the subjects after matching in respect to age and BMI (Table 1) A study by Jin et al. (2015) found that the association between serum ferritin and T2DM was maintained after adjusting for BMI and other potential confounders.[28]

The results revealed a substantial difference in serum ferritin levels between the case and control groups, with the case group exhibiting significantly higher levels of serum ferritin.(Table 2) This finding is in line with previous studies that have reported an association between elevated serum ferritin levels and T2DM.[29,30] For instance, a study conducted by Jung et al. (2013) found that higher serum ferritin levels were significantly associated with an increased risk of T2DM in a Korean population,[31] and a meta-analysis by Jin et al. (2015) confirmed this association across various populations.[28] The exact mechanism underlying this association is not yet fully understood. However, it has been suggested that elevated serum ferritin levels might reflect increased body iron stores, which could contribute to insulin resistance and impaired glucose metabolism.[32] Iron overload has been shown to induce oxidative stress and inflammation, leading to pancreatic β-cell dysfunction and insulin

resistance and impaired glucose metabolism, which are key features of T2DM.[24,25] Furthermore, iron may interfere with glucose metabolism by inhibiting glucose uptake and glycogen synthesis in skeletal muscles.[33] Notably, the study found a significant positive correlation between serum ferritin and FPG levels in the case group, while no significant correlations were observed in the control group. This association is similar in both sex, so it's not affected by sex.(Table 3) Elevated serum ferritin levels have been linked to increased oxidative stress and inflammation, which may contribute to the development and progression of T2DM and its complications.[29] A study by Son et al. (2019) found that higher serum ferritin levels were associated with an increased risk of developing T2DM-related complications, such as cardiovascular disease and nephropathy.[34] The study also assessed the risk of T2DM in relation to serum ferritin levels, finding that participants with serum ferritin levels ≥151 µgm/L had an odds ratio of 11.64 for having T2DM compared to those with levels ≤150 µgm/L. This indicates that higher serum ferritin levels are associated with a significantly increased risk of T2DM (Table 4). This finding is consistent with other studies that have reported similar associations between serum ferritin levels and T2DM risk.[34,35]

Conclusion:

In conclusion, this case-control study in a selected study subjects of Bangladesh, provides evidence supporting an association between elevated serum ferritin levels and type 2 diabetes mellitus (T2DM). Importantly, the association between serum ferritin levels and T2DM seems to be independent of sex, BMI and age. Further research is required to confirm these findings and explore the underlying mechanisms considering a large sample size.

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Haemodynamic effects of intravenous bolus of carbetocin and oxytocin on women undergoing caesarean section

Khan MRH[a], Devnath CK[b], Sumon RA[c], Taskin R[d], Ibrahim K[e]

Abstract

Background: Postpartum hemorrhage (PPH) is still the leading cause of maternal mortality in poor countries. There are many pharmacological options for the management of postpartum hemorrhage, oxytocin being the first line of treatment. There is as yet no evidence about the safety and efficacy of using carbetocin, an oxytocin agonist, in patients with PPH.

Objective: The objective of this study is to compare the haemodynamic effects of carbetocin and oxytocin given as intravenous bolus on women undergoing caesarean section.

Methods: This randomized control trial was conducted on 60 women more scheduled for Cesarean delivery in a selected teaching hospital. Patients were selected according to inclusion criteria. They were divided equally into two groups where Group-A (n1 = 30) patients received i.v. oxytocin 10 U/ml (0.5 ml) + 4.5 ml saline and Group-B (n2 = 30), received i.v. carbetocin 100 µg/ml (1 ml) + 4 ml saline. Then haemodynamic parameters were compared. Main outcome variables were Systolic blood pressure (SBP), Diastolic blood pressure (DBP), Mean blood pressure (MAP), Heart rate (HR).

Results: The haemodynamic parameters at baseline were almost alike between two groups. After giving test drugs, haemodynamic parameters compared among 1 minute to 15 minutes and also observed similar result between two groups. No PPH found in carbetocin group. PPH ignored bleeding up to 800-1000ml in both groups. 3 cases of PPH found in oxytocin group, which was not statistically significant.

Conclusion: No significant difference in haemodynamic changes found in using carbetocin and oxytocin intravenously after caesarian section.

Keywords: Haemodynamic effects Carbetocin, Oxytocin, Postpartum haemorrhage, Caesarian section.

Introduction:

Oxytocin is the most commonly used uterotonic agent in obstetrics. It is routinely administered after both normal and operative delivery to initiate and maintain adequate uterine contractility for minimizing blood loss and preventing postpartum hemorrhage.[1, 2] Oxytocin injection (synthetic) acts on the smooth muscle of the uterus to stimulate contractions; response depends on the uterine threshold of excitability. It exerts a selective action on the smooth musculature of the uterus, particularly toward the end of pregnancy, during labor and immediately following delivery. Oxytocin stimulates rhythmic contractions of the uterus, increases the frequency of existing contractions and raises the tone of the uterine musculature. It has a very short half-life of 4 to 10 minutes.[3, 4] Carbetocin is a long-acting synthetic oxytocin analogue,[5, 6, 7] 1-deamino-1-monocarbo-(2-O-Methyltyrosine)-oxytocin, firstly described in 1987. It has a half-life of 40 minutes (around 4–10 times longer than oxytocin) and uterine contractions occur in intravenous.

Administration of optimal dosage of 100 µg.[8] A single dose of carbetocin has been hypothesized to act as a 16 hours' intravenous oxytocin infusion regarding the increase in uterine tone and the reduction of the risk of PPH in elective caesarean section. Carbetocin binds to oxytocin receptors present on the smooth musculature of the uterus, resulting in rhythmic contractions of the uterus, increased frequency of existing contractions, and increased uterine tone. The oxytocin receptor content of the uterus is very low in the non-pregnant state, and increases during pregnancy,

a. Dr. Md. Rafiqul Hasan Khan; MCPS, FCPS
Associate Professor of Anaesthesiology
Bangladesh Medical College & Hospital, Dhaka

b. Dr. Chanchal Kumar Devnath; DA, FCPS
Consultant-Anaesthesia and Neuro-ICU, BRB Hospital, Dhaka

c. Dr. Rajib Ahsan Sumon; DA
Consultant of Critical Care Medicine
Bangladesh Medical College Hospital

d. Dr. Roksana Taskin; MBBS
Resident Surgeon (Gynae & Obs), Monowara Orthopedics and General Hospital, Dhaka

e. Prof. Kamal Ibrahim; FCPS, FICS
Ex-Head, Dept. Of Anaesthesiology
Bangladesh Medical College Hospital, Dhaka

Correspondence to:
Dr. Md. Rafiqul Hasan Khan MCPS, FCPS.
Associate Professor, Department of Anaesthesiology
Bangladesh Medical College and Hospital, Dhaka
Email: azad300769@gmail.com

reaching a peak at the time of delivery. J. S. Thomas[9] et al found marked cardiovascular changes occurred in the bolus dose oxytocin; the heart rate increased. The mean arterial pressure decreased. Giovanni Larciprete[10] et al recorded that regarding the haemodynamic effects, both drugs have a hypotensive effect, but a greater reduction in blood pressure within the oxytocin group. This study is undertaken to evaluate the impact of carbetocin versus oxytocin for relevant maternal haemodynamic parameters in a non-invasive set-up during caesarean delivery in a randomized control trial.

Materials & Methods:

This was a prospective randomized control study to compare haemodynamic effects of carbetocin and oxytocin given as intravenous bolus on women undergoing caesarean section in inpatients department of Gynaecology and Obstetrics of Bangladesh Medical College Hospital, Dhaka, during the period of 1st January 2016 to 30th June 2016. Ethical clearance was taken from the concerned authority. A total number of 60 patients were included in this study. These patients were divided into two groups consisting of 30 patients each, allocated randomly. Thirty patients received i.v. oxytocin 10 U/ml (0.5 ml) + 4.5 ml saline was considered as Group-A and rest 30 patients received I.V. carbetocin 100 µg/ml (1 ml) + 4 ml saline was considered as Group-B. Inclusion criteria were ASA grading I & II, age between 18-35 years, undergoing elective caesarean section under Sub arachnoid block & gestational age 36 weeks or more. Exclusion criteria were- ASA grading III & lV, emergency caesarean section, reported adverse reactions to any of the drugs included in the study, known hypertension (pregnancy induced hypertension), preeclampsia and eclampsia, placenta previa, placenta accrete, preoperative systolic blood pressure less than 90 mm of Hg, if required general anaesthesia due to failure of spinal anaesthesia, patient refusal, patients receiving sympathomimetic or sympatholytic drugs. Main outcome variables were Systolic blood pressure (SBP), Diastolic blood pressure (DBP), Mean blood pressure (MAP), Heart rate (HR) were recorded just before giving the spinal anesthesia with the patient in a left lateral position. With the woman in a sitting position, spinal anesthesia was induced in the L3–L4 intervertebral space, and bupivacaine 12.5 mg were injected through a 27-Gauge spinal needle concomitantly i.v. infusion of Hartmann's solution (37°C, 10 ml/kg) were started. During surgery, the patient was supine with an operating wedge under her right hip (19°). The level of anesthesia was tested by cold sensation 5 minutes after spinal anesthesia as well as by pinching with surgical tweezers before skin incision. Either the study or control drug accordingly to injected slowly, over the course of 60 seconds, starting when the baby's head and shoulders were delivered. Systolic blood pressure, Diastolic blood pressure, Mean blood pressure and Heart rate were monitored before injection control or study drug and 1, 3, 5, 7, 9,11,13 and 15 minutes after injection. Patients were in close monitoring up to 24 hours after caesarean section. Hypotension was treated with i.v. Ephedrine and intravenous fluid infusion.

Statistical data analysis

All data presented as (mean±SD). Analysis of data was done by unpaired t-test & chi-square test. A p value <0.05 accepted as statistically significant. Statistical analysis was done by Statistical Package for Social Science (SPSS) for Windows version 20.0

Results:

Table 1: Demographic characteristics of patients (N=60)

	Group-A (Mean±SD)	Group-B (Mean±SD)
Age (Year)	26.5±4.5	27.2±4.8
Gestational age (weeks)	38.6±1.6	38.9±1.6
Weight (Kg)	59.3±3.0	59.9±2.3

Table 1 shows demographic characteristics of the study patients, it was observed that mean age was found in 26.5±4.9 years in Group-A and 27.2±4.8 years in Group-B. The mean gestational age was found 38.6±1.6 weeks in Group-A and 38.9±1.6 weeks in Group-B. The mean (SD) weight of the study patients were 59.3 ± 3.0 Kg in Group-A and 59.9 ± 2.3 Kg in Group-B, majority patients were in 55-60 kg weight group. The difference was not found statistically significant (p>0.05).

Table 2: Pre-operative assessment of patient

	Group-A No (%)	Group-B No (%)
Gravida-Multi	20 (66.7)	22 (73.3)
ASA G-I	18 (60)	21 (70)
ASA G-II	12 (40)	9 (30)

Table 2 shows Pre-operative assessment of the study patients. Majority 20(66.7%) patients were multi gravida in Group-A and 22 (73.3%) in Group-B. The ASA Grade I was 18(60%) in Group-A and 21(70%) in Group-B. The ASA Grade II was 12(40%) in Group-A and 9(30%) in Group-B. The difference was not statistically significant (p>0.05) between two groups. That is the groups were homogenous.

Table 3: Haemodynamic mean parameters comparison at different time after injection of drugs

	Baseline	1 min	3 min	5 min	7 min	9 min	11 min	13 min	15 min
Group-A									
SBP	117.3±7.8	116.7±7.5	111.5±11.7	109.±7.36	107.5±5.4	106.5±4.5	107.3±4.5	105.0±8.1	104.0±11.1
DBP	76.3±5.6	75.7±6.8	72.8±8.6	54.5±3.8	74.0±7.9	74.8±3.6	71.3±3.6	70.1±4.3	65.30±9.1
MAP	81.3±5.6	88.9±7.0	86.4±5.8	73.3±5.4	74.2±6.2	73.3±5.8	74.7±4.2	69.9±5.3	67.9±5.3
HR	85.7±3.92	89.5±8.4	92.3±10.6	98.0±2.8	91.8±5.5	88.1±7.5	91.8±5.2	93.2±2.3	92.5±3.0
Group-B									
SBP	120.0±6.9	119.7±6.7	113.2±10.	108.±6.9	108.7±4.3	107.6±3.6	106.9±3.6	105.±7.4	108.5±11.4
DBP	78.7±4.3	77.8±4.9	73.5±8.9	73.7±8.1	55.0±3.6	73.3±4.9	72.3±4.9	74.9±4.8	67.5±8.4
MAP	81.7±4.1	86.4±4.4	86.3±6.2	72.0±6.0	74.7±7.3	73.4±6.2	74.3±5.3	74.8±6.4	70.8±6.4
HR	87.7±5.2	86.9±9.5	91.3±11.5	97.6±2.2	99.4±4.4	87.5±6.6	90.1±2.1	90.4±3.5	89.3±2.60

Table 3 shows, in Group-A: SBP decreases from baseline to 15 min gradually. DBP also follows the same pattern, but it shows a drop at 5 min reading, then again rises, and after that gradually decreases to 15 min reading. MAP rises from baseline to 1min reading, then gradually decreases to 15 min reading. HR rises from baseline to 5 min reading, then gradually decreases to 15 min reading with a drop at 9 min reading.

In Group-B: SBP decreases from baseline to 15 min gradually. DBP also follows the same pattern, but it shows a drop at 7 min reading, then again rises, and after that gradually decreases to 15 min reading. MAP rises from baseline to 1min reading, then gradually decreases to 15 min reading. HR rises from baseline to 7 min reading, then gradually decreases to 15 min reading with a drop at 9 min reading.

The mean difference of all haemodynamic parameters were not statistically significant (p>0.05) in unpaired t-test.

Table 4: Mean and Range of SBP and DBP of Group-A and Group-B.

	Group A		Group B	
	Mean	**Range**	**Mean**	**Range**
SBP	109.4 mm Hg	125.1-92.9 mm Hg	110.8 mm Hg	126.9-97.1 mm Hg
DBP	70.5 mm Hg	50.7-81.9 mm Hg	71.8 mm Hg	51.4-83 mm Hg

Mean SBP in Gr-A and Gr-B is 109.4 mm Hg and 110.8 mm Hg; mean DBP is 70.5 mm Hg and 71.8 mm Hg in in both groups respectively.

Table 5: Changes in MAP

MAP	Group-A	Group-B
Decrease in MAP	24 mmHg	12 mmHg
Range of Decrease in MAP	19-32 mmHg	8-18 mmHg

Decrease in MAP in group A and group B is 24 mmHg and 12 mmHg respectively. In group A and in group B range of decrease is 19-32 mmHg and 8-18 mmHg.

Table 6: PPH between Group-A and Group-B

Status of PPH	Group-A	Group-B
PPH	3 cases (10%)	00

PPH ignored bleeding up to 800-1000ml in both groups. three cases of PPH found in oxytocin group, which was not statistically significant. PPH was 10% in Group-A (Table 6).

Discussion:

This prospective randomized control study was carried with an objective to compare the haemodynamic effects in blood pressure and heart rate in carbetocin and oxytocin group after caesarean section.

In this current study in Table-1, it was observed that mean age was found in Group-A 26.5±4.9 years and 27.2±4.8 years in Group-B. No significant differences were found between group A and group B. Similarly, Mohamed et al.[11] found non-significant difference between oxytocin group and carbetocin group as regards age, where mean of group A was 26.97 years and 26.09 years in group B. Fahmy et al.[12] observed that the mean age was found 25.4 ±4 years in group A and 24.5 ±3 years in Group-B, which are closely resembled with the present study. On the other hand, Holleboom et al.[13] had observed the mean age was 33.3±4.6 years in Oxytocin group and 33.0±4.6 years in Carbetocin group. Similarly, Uy et al.[14] observed at baseline, there was no significant difference between oxytocin and carbetocin in terms of mean age, where mean was 31 years and 30 years respectively. The higher mean age may be due to geographical variations, racial, ethnic differences, genetic causes, different lifestyle and increased life expectancy may have significant influence in their study patients.

In this present study it was observed that the mean gestational age was found 38.6±1.6 weeks in Group-A and 38.9±1.6 weeks in Group-B. The mean gestational age was not statistically significant (p>0.05) between two groups, which is consistent with Uy et al.[14] study, where the investigators found there was no significant difference between oxytocin and carbetocin in terms of mean gestational weeks (P>0.05). Holleboom et al. [13] found that the mean gestational age was found 38.8±1.0 weeks in Oxytocin group and 38.9±1.0 weeks in carbetocin group. The difference was not statistically significant (p>0.05) between two groups, which is consistent with the present study. Similarly, Larciprete et al.[10] and Reyes et al.[15] had observed the identical mean gestational age of their studied patients, thus support the present study.

In this current study, it was observed that the mean (SD) weight of the study patients were 59.3 ± 3.0 Kg in group-A and 59.9 ± 2.3 Kg in group-B, majority patients were in 55-60 kg weight group. No significant differences were found between Group-A and Group-B, which is similar with Kim et al. [16] study.

In this current study it was observed that majority 66.7% patients were multi gravida in Group-A and 73.3% in Group-B (Table -2). The difference was not statistically significant (p>0.05) between two groups. Similarly, Uy et al. [14] found at baseline, there was no significant difference between oxytocin and carbetocin in terms of parity (p>0.05). Holleboom et al. [13] undertook a study and observed multigravida 23.1% and 28.3 in oxytocin and carbetocin group respectively. The difference was not statistically significant (p>0.05) between two groups.

In this current study, it was observed that the ASA Grade was almost two third 60% belonged to grade I in group A and 70% in groups B. The difference was statically not significant (p>0.05) between two group. Bhattacharya et al.[1] observed that 77.50% had ASA grade I and 25.0% ASA grade II in Group-A. In Group-B, 77.5% patients had ASA grade I and 22.5% ASA Grade II, which is consistent with the current study.

In this current study in Table-3, it was observed that the haemodynamic parameters result at baseline and found that the mean (SD) heart rate was 85.73.92 beats/min range from 80 to 90 beats/ min in group-A and 87.75.2 beats/min range from 81 to 92 beats/ min in group-B. The mean (SD) systolic BP was 117.37.8 mmHg in Group-A and 120.06.9 mmHg in Group-B. Similarly, the mean (SD) diastolic BP was 76.35.6 mmHg in Group-A and 78.74.3 mmHg in Group-B. The mean difference of all haemodynamic parameters at preoperative period were not statistically significant (p>0.05) in unpaired t-test.

Fahmy et al.[12] observed the mean heart rate was found 76.2±5.16 (beats/min) in Group-A and 75.9±4.9 (beats/min) in Group-B. In another study Mohamed et al[11]. (2015) observed that the mean systolic blood pressure 110.1 (mm Hg) with the range from 80-130 (mmHg) in oxytocin group and 98.8 (mmHg) with the range from 80-130 (mmHg) in carbetocin group and the mean diastolic blood pressure 71.7 (mmHg) with the range from 50-90 (mmHg) in oxytocin group and 65.4 (mmHg) with the range from 40-80 (mm Hg) in carbetocin group, which are consistent with the current study (Table 4). Similar findings also observed by Larciprete et al[10] after giving test drugs, parameters compared between 1 minute to 15 minute.

The current study (Table 5**)** demonstrated an average decrease in MAP of 24 mmHg ranged from 19 to 32 mmHg in group A, whereas in group B average decrease in MAP of 12 mmHg range from 8 to 18 mmHg. Whilst this magnitude of decrease in MAP may be well tolerated normally. In the present study it was observed that the changes in heart rate were significantly higher in group A with compared to group B. However, the gentler increase of heart rate in group B is preferable clinically. Thomas et al.[9] (2007) found in their study that the decrease in MAP of 8(8.7) mmHg and the small increase in HR are certainly clinically preferable, which is closely resemble with the present study.

Uy et al.[14] found systolic and diastolic BP were not significantly different immediate post intervention between the two groups, immediate post-operative and while recovering between groups were also not significant. Attilakos et al.[5] showed there were significant changes with time for each of systolic blood pressure and diastolic blood pressure respectively but no significant main effect of group, which are similar with the current study.

In this current study (Table 6), it was observed that all patients had bleeding up to 800-1000ml in both groups. Holleboom et al.[13] showed the blood loss were also comparable in both groups. In another study Larciprete et al.[10] reported that there was no significant difference in the amount of estimated blood loss and the incidence of primary post-partum haemorrhage in both groups. In fact, the investigators did not demonstrate any difference in the amount of blood loss after caesarean section. In another study Uy et al.[14] found that the estimated blood loss was significantly lower in the carbetocin group.

In this current study (Table 6**)**, it was observed that 10.0% patients had primary PPH in Group-A and not found in Group-B. PPH was comparatively higher in group A, but the difference was not statistically significant **(**p>0**.**05**)** between two groups**.** Similarly, Holleboom et al.[13] found PPH 8.4% and 7.8%, in oxytocin and carbetocin group respectively. In another study Larciprete et al.[10] reported that there was no significant difference in the amount of estimated blood loss and in the incidence of primary post-partum haemorrhage in both groups.

Conclusion:

This study was undertaken to evaluate the Haemodynamic effects of carbetocin and oxytocin given as intravenous bolus on women undergoing caesarean section. Age, parity, gestational age, weight and indication of C/S were almost similar between two groups. All patients had almost stable haemodynamic status in carbetocin group and oxytocin group. Fewer side effects and no primary PPH observed in carbetocin group. Therefore, it can be concluded that both drugs have comparable haemodynamic effects and are uterotonic drugs with an acceptable safety profile for prophylactic use. Minimal differences in the recovery phase are in keeping with the fact that carbetocin has an extended half-life compared with oxytocin. Single dose of carbetocin appears to be more effective than oxytocin for several hours on uterine contraction.

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Delirium: An underdiagnosed clinical condition in the elderly

Haq AI[a], Nahar A[b], Hasnat S[c], Aziz S[d], Akhtaruzzaman M[e], Arafat MA[f], Sumon RA[g]

Case vignette

A 70 year old male patient of CKD, hypertension and DM, fever with no previous H/O psychiatric disorder is referred to psychiatry dept. of Bangladesh Medical College Hospital with the complaints of agitation, restlessness, outgoing tendency, and insomnia. The patient developed the symptoms two days after admission. Patient's family members complained that the patient remains drowsy and nonresponsive throughout the day. But after sunset he patient became agitated, restless and was unable to sleep. The patient failed to identify his close relatives and wanted to go to his home. On physical examination, temperature was raised (102^0F), and there was tachycardia, tachypnea and hypertension. Other systemic examination revealed no abnormality. In mental state examination, the patient was restless, inattentive, disoriented and agitated, appeared disheveled. He was picking his clothes, bed sheets etc. Initial laboratory tests demonstrate leukocytosis, raised creatinine and blood urea nitrogen, microscopic examination of urine showed increased pus cells, urine culture showed growth of *E. coli* and Hemoglobin A_{1c} was above normal.

Introduction:

Delirare, a Latin word means 'to go out of the furrow', that is, to deviate from a straight line, to be deranged.[1] One of the component of geriatric symptomatology - delirium also has an important prognostic role in older patients with acute illnesses. In everyday practice most of the junior doctors and nurses have lack of knowledge and training about this syndrome.[2] In clinical practice delirium is underdiagnosed. This review will focus on the predisposing factors, epidemiology, outcomes, pathophysiological mechanisms, tools for screening and diagnosis, prevention and treatment recommended. We hope it will provide a guideline for our clinicians to diagnose and to improve the quality of care provided to the older patients.

a. Dr. Arman Ibne Haq; FCPS (Psy), MBBS
Assistant Professor, Department of Psychiatry
Bangladesh Medical College, Dhaka

b. Dr. Azizun Nahar; M. Phil (BSMMU), MBBS
Associate Professor, Department of Microbiology
Bangladesh Medical College

c. Dr. Shahed Hasnat; M. Sc (UK), MBBS
Assistant Professor, Department of Microbiology
Bangladesh Medical College

e. Dr. Muhammed Akhtaruzzaman; MD, MBBS
Associate Professor, Department of Cardiology
Bangladesh Medical College Hospital, Dhaka

f. Dr. Mohammad Ali Arafat; MS, MBBS
Junior Consultant, Department of Urology,
Bangladesh Medical College Hospital, Dhaka

g. Dr. Rajib Ahsan Sumon DA, MBBS
Consultant, ICU, Bangladesh Medical College & Hospital, Dhaka

Correspondence to:
Dr. Arman Ibne Haq; FCPS (Psy), MBBS
Assistant Professor, Department of Psychiatry
Bangladesh Medical College
Email: arman 0913@gmail.com

Definition:

Delirium is a severe neuropsychiatric syndrome characterized by the acute global impairment of consciousness (clouding of consciousness) which ultimately leads to altered level of attention, awareness and other cognitive functions.[3]

Etiology of Delirium:

Delirium can be triggered by a single cause, but it may be multifactorial, resulting from the interaction between predisposing and precipitating factors. The higher the burden of the predisposing factors, the lower the magnitude of the precipitating factors required to cause delirium.[4,5] Multiple predisposing factors such as old age, pre- existing cognitive impairment, previous history of delirium, comorbid conditions (DM, HTN) have important role in delirium etiology.

Precipitating Factors: These factors are present just before the onset of delirium. Important precipitating factors include -

- Infection- septicemia, pneumonia, UTI, typhoid fever.
- Organ failure- respiratory, hepatic, cardiac and renal failure.
- Medications- diuretics, digoxin, broad spectrum antibiotics, narcotic analgesics, benzodiazepines, anticholinergic drugs, steroid, illicit drug use like alcohol intoxication and withdrawal.
- Neurological- CVD, epilepsy, head injury, space occupying lesion.
- Urinary catheterization.
- Constipation.
- Sleep deprivation.
- Electrolyte imbalance- hyponatremia, hypernatremia, hypomagnesaemia.
- Visual impairment.
- Hearing impairment in elderly patients.[4,5]

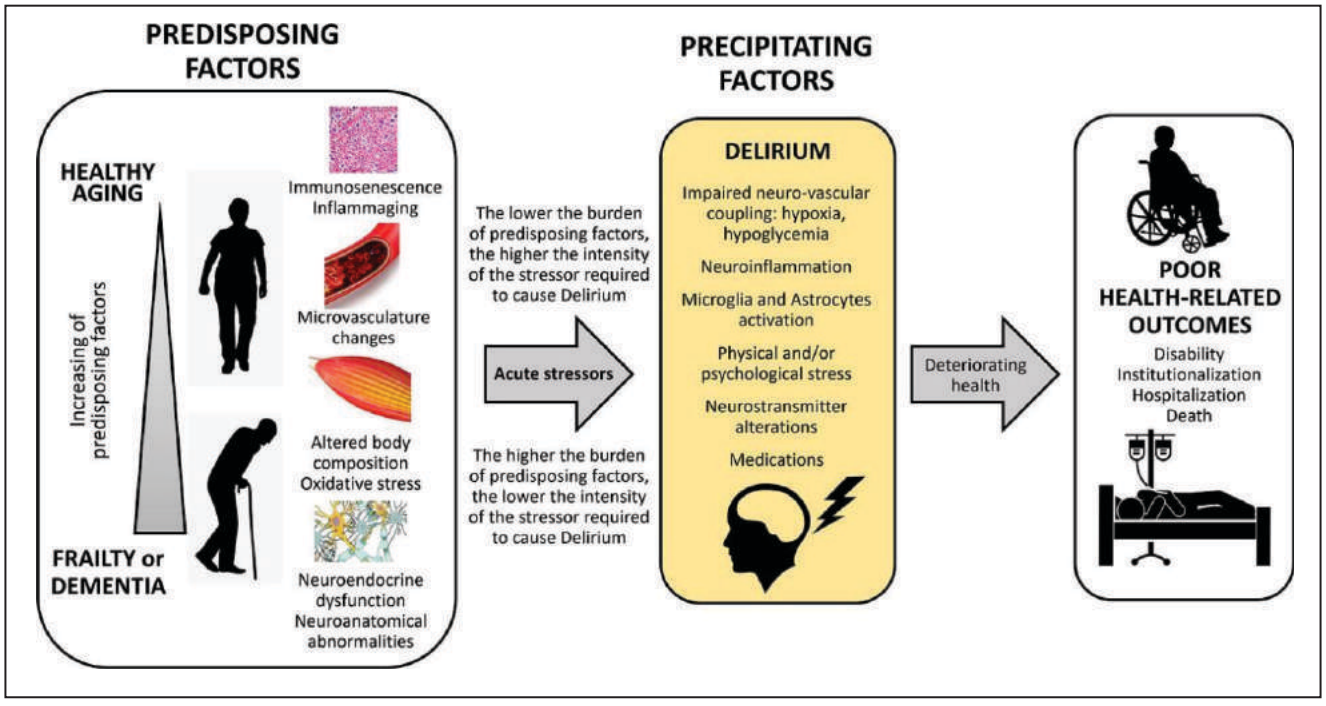

Figure 1: Interaction of predisposing and precipitating factors in the development of delirium (Source: Giuseppe Bellelli, Justin S. Brathwaite and Paolo Mazzola. Delirium: A marker of vulnerability in older people. Frontiers in Aging euroscience 2021; volume 13 | article 626127. doi 10.3389/fnagi.2021.626127)

A systematic review and meta-analysis was done on 2338 patients including 11 articles identified dementia with visual impairment, illness severity, urinary catheterization, low albumin levels, and increased length of hospital stay as risk factors for delirium.[6] Another systemic review on patients with hip fracture also had cognitive impairment and dementia which were prominent risk factors for delirium.[7] Patients from ICU had same risk factors.[8] Some authors suggest that SARSCoV-2 infection may be a potential cause of delirium in admitted patiens.[8]

Epidemiology:

Prevalence of delirium varies considerably by patient group and setting. It is common in hospitalized older adults. The prevalence of delirium was found 23% with a meta-analysis of 33 studies of hospitalized inpatients.[9]

Delirium prevalence was 24% of patients undergoing coronary artery bypass grafting.[10] Delirium occurrence might be even higher in the ICU. A systematic review of studies from North and South America, Europe and Asia reporting a pooled high prevalence 31.8% of delirium in ventilated and non-ventilated intensive care unit (ICU) patients.[11] Delirium prevalence (25%) is also common after acute stroke.[12] Almost two-thirds of 65 years old admitted patients had subsyndromal delirium in North America.[13]

Pathophysiology:

Pathophysiology of delirium is complex and results from a diverse range of pathobiological processes. Delirium can be conceptualized as a final common pathway resulting from multiple factors that lead to a state of impaired brain function.[14]

Systemic inflammation, metabolic derangement and hypoxia may impair brain function directly or indirectly through production of cytokines. The cytokines cause damage to the blood brain barrier and activation of microglia. The microglia in turn causes release of chemical mediators which disrupts normal neuronal function leading to aberrant neurotransmitter activity. Decreased synthesis of acetylcholine cause impairment of alertness, attention, memory and REM sleep.[15] Other neurotransmitters implicated in delirium include melatonin, dopamine, norepinephrine, glutamate, histamine, 5-hydroxytryptamine, and/or gamma-aminobutyric acid.[16]

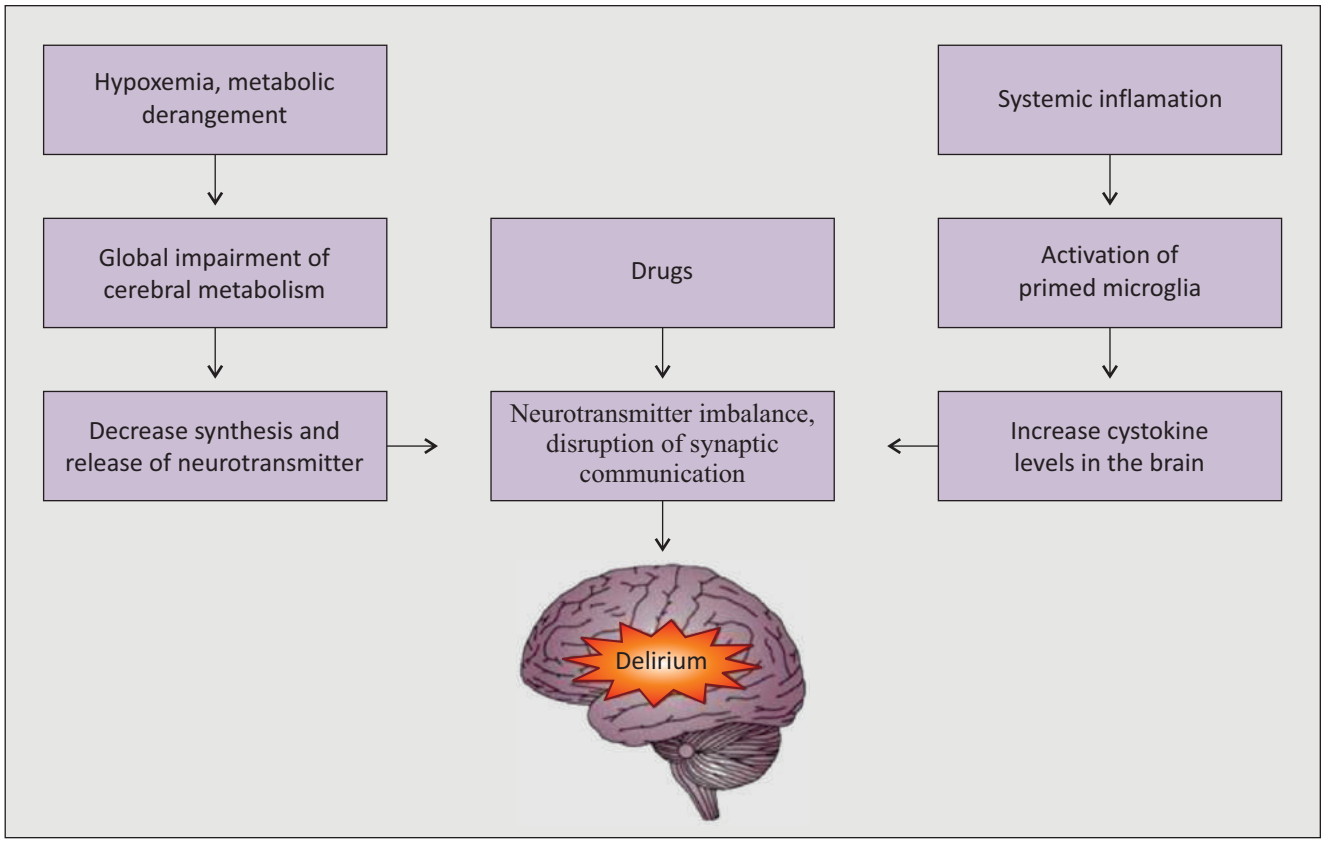

Figure 2: Pathophysiology of delirium (Source: Tamara G. Fong, Samir R. Tulebaev & Sharon K. Inouye . Delirium in elderly adults: diagnosis, prevention and treatment. Nature Reviews Neurology 2009; volume 5, pages 210-220.)

Neural Circuitry

Delirium is associated with aberrant resting-state neural interactions between the suprachiasmatic nucleus (the biological master clock) and cortical regions.[17]. Delirium is associated with decreased functional connectivity between suprachiasmatic nucleus with the posterior cingulate cortex (alertness and attention), parahippocampal gyrus (memory).[18]

Clinical features:

Delirium episodes are usually acute, transient and fluctuate in severity. Most of the episode of delirium lasts for few days but 20-30 % individuals episodes persists for weeks or months.[19-21] Some patients have delirium features but they do not present all the criteria of delirium, this condition is described as 'subsyndromal delirium.' Symptoms and signs vary widely - between patients, in the same patient at different times of day- typically being worse at night.

Features are usually of three types:

A) Hyperactive type- Hyperactive, restless, irritable, psychotic symptoms (visual and auditory hallucinations), transient delusions, anxiety, depression, emotional lability.

B) Hypoactive type- Hypoactive, psychomotor retardation, perseveration and abnormally drowsy

C) Third subtype (mixed)- The patient switches rapidly between hyper and hypoactive states. [13,22]

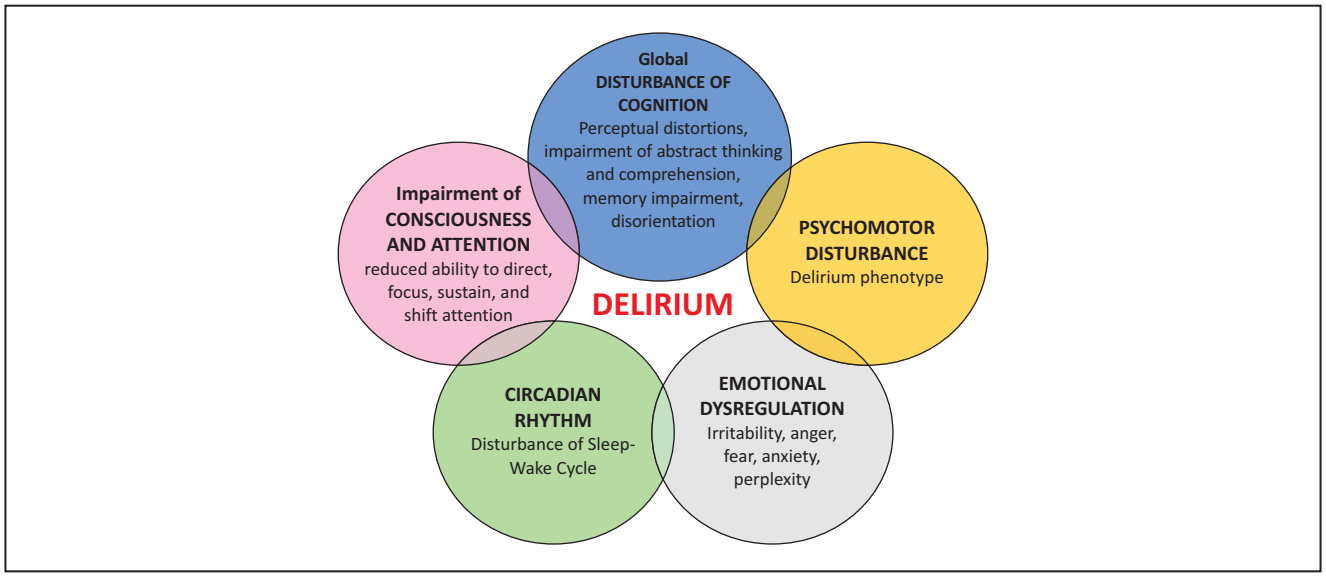

Figure 3: Clinical features of delirium (Source: José R. Maldonado. R. Delirium pathophysiology: An updated hypothesis of the etiology of acute brain failure. International journal of Geriatric Psychiatry 2017. https://doi.org/10.1002/gps.4823)

Diagnosis:

History

Previous history of delirium, H/O dementia, H/O co-morbid physical conditions, detailed medication history, H/O any psychiatric disorder.

Patient interview

Significant findings- Appearance- patient may be drowsy/ restless/agitated, speech- may be incoherent, consciousness- altered, attention- ↓ ability to focus, sustain, shift, orientation- disoriented, memory- impaired.

Physical findings

Parameter	Findings	Clinical implication
Pulse	Bradycardia	Hypothyroidism, raised ICP
	Tachycardia	Hyperthyroidism, infection, HF
Temperature	Fever	Sepsis, vasculitis, thyroid storm
BP	Hypotension	Shock, hypothyroidism, Addison's disease
	Hypertension	Encephalopathy, ICSOL
Respiration	Tachypnea	DM, Pneumonia, HF, Fever, Metabolic acidosis
	Shallow	Alcohol or other substance intoxication
Carotid vessels	Bruits or slow pulse	TIA
Neck	Rigidity	Meningitis, SAH
Eyes	Dilated pupil	Autonomic over activity, DTs, anxiety
	Papilloedema	HTN, encephalopathy, ICSOL
Mouth	Tongue, cheek laceration	GTCS
Thyroid	Enlarged	Hyperthyroidism
Heart	Arrhythmia, congestion, cardiomegaly	Heart disease

Assessment scale

Confusion Assessment Method (CAM) and Richmond Agitation and Sedation scale (RASS).[23]

Confusion Assessment Method (CAM)

The diagnosis of delirium by CAM requires the presence of both features *A* and *B*		
CAM Confusion Assessment Method	**A. Acute onset** and **Fluctuating course**	Is there evidence of an acute change in mentalstatus from patient baseline? Does the abnormal behavior: ‣ come and go? ‣ fluctuate during the day? ‣ increase/decrease in severity?
	B. Inattention	Does the patient: ‣ have difficulty focusing attention? ‣ become easily distracted? ‣ have difficulty keeping track of what is said?
	C. Disorganized thinking	Is the patient's thinking ‣ disorganized >incoherent For example does the patient have ‣ rambling speech/irrelevant conversation? ‣ unpredictable switching of subjects? ‣ unclear or illogical flow of ideas?
	And the presence of either feature *C* or *D*	
	D. Altered level of consciousness	Overall, what is the patient's level of consciousness: ‣ alert (normal) ‣ vigilant (hyper-alert) ‣ lethargic (drowsy but easily roused) ‣ stuporous (difficult to rouse) ‣ comatose (unrousable)

Laboratory investigations

CBC, blood glucose, serum electrolytes, blood urea nitrogen and S. creatinine, blood C/S, liver function test, thyroid function test, urinalysis- R/M/E, C/S, Toxicology, CSF study, serum B12 and folate levels, HIV- ELISA, Serologic test for syphilis, EEG, CT scan of brain, MRI of brain.

Treatment:

- ☐ Maintaining quiet, well lighted room, staff continuity, adequate nutrition, normal sleep- wake cycle, limiting visits.
- ☐ Symptomatic treatment-
  - ✓ Non pharmacological intervention-
    - ▪ Effective communication and reorientation.
    - ▪ Reorientation during the day- Physical therapy and early mobilization.
  - ✓ Pharmacological intervention-
    - ▪ Agitation, restlessness- Low dose antipsychotics→ risperidone, quetiapine, haloperidol.
    - ▪ Insomnia→ Choose light sedation like short acting benzodiazepines, melatonin, low dose quetiapine.
- ☐ Treatment of underlying cause- exclusion of hypoglycemia or hyperglycaemia, review of medication list, removal of unnecessary devices or tubes.

How can you differentiate Delirium from Dementia?

Characteristics	Delirium	Dementia
Onset	Acute	Insidious
Duration	Days to weeks	Months to years
Course	Fluctuate, short term	Progressive
Consciousness	Impaired	Clear
Attention	Inattention	Normal
Memory	Impaired	Impaired
Reversibility	Usually	Not
Orientation	Impaired	May be
Hallucination	May be present	Usually absent
Delusion	Transient	Usually absent

Prognosis:

Symptoms recede over 3-7 day period after identification and removal of causative factors. But the mortality rate is high in ensuing year indicating serious nature of associated medical condition. Nearly half of the delirium patients are discharged with persistent symptoms, among them 20-40% still have delirium at 12 months. These persistent symptoms patients outcome are worse than patients who fully recover by point of discharge.[24]

Differential diagnosis:

Dementia- especially frontal lobe

Psychiatric disorders- MDD, Psychosis, Bipolar disorder

Aconvulsive status epilepticus

Akathisia

Conclusion:

In elderly patients delirium is often undiagnosed or misdiagnosed. Early diagnosis and multidisciplinary approach is necessary for effective treatment of delirium. Although most of the delirium cases are acute and transient. Regular follow up is required to reduce the mortality resulting from comorbid conditions.

Recommendation:

- Training of doctors and nursing staffs in diagnosis and management of delirium.
- Developing multidisciplinary approach to deal with delirium cases.
- Effective communication and psycho education of family members.

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Crohn's disease in a patient with pre-existing Ankylosing Spondylitis- A case report

Karim ME[a], MahbubI[b], Paul S[c], Arefin KE[d], Bhuiyan ME[e], Jubaere AKM[f], Parveen R[g], Rahim S[h]

Abstract

Crohn's disease is an inflammatory bowel disease which may be present in association with Ankylosing Spondylitis. Here, we are presenting a case of 31-year-old male who presented to us with abdominal pain and unexplained anaemia at Sheikh Russel National Gastroliver Institute and Hospital, Dhaka in 2022. . He was previously diagnosed as Ankylosing Spondylitis. With the help of history, physical examination, endoscopic immunological and radiological examination he has been diagnosed as a case of Crohn's disease along with pre-existing Ankylosing Spondylitis.

Keywords: Ankylosing Spondylitis, Crohn's disease.

Introduction:

The diagnostic criteria of Ankylosing Spondylitis (AS) are in accordance to the modified New York criteria that include both clinical and radiological aspects.[1]The spondyloarthritis includes sacroiliitis, inflammatory back pain, asymmetric oligoarthritis predominantly of the lower limbs, enthesitis, dactylitis and extra-articular manifestations such as gut inflammation, psoriasis and anterior uveitis. Clinical criteria included: low back pain and stiffness for >3 months, which improve with exercise and not relieved by rest, limitation of motion of the lumbar spine in both the sagittal and frontal planes, and restriction of chest expansion relative to normal values corrected for age and sex. Radiological criterion is bilateral sacroiliitis grade ≥2 or unilateral sacroiliitis grade 3 to 4. A definite AS is yielded by the radiological criterion and at least 1 clinical criterion.[2,3]

The diagnostic criteria of Crohn's Disease (CD) include the following aspects:a history of abdominal pain, vomiting, diarrhea, weight loss or rectal bleeding; radiologic findings of stricture or fistula formation, mucosal cobble stoning, or deep ulceration; endoscopic appearances of cobble stoning, linear ulceration, or skip lesions; and pathologic confirmations of transmural inflammation or noncaseating epithelioid granulomas.[4]

The Patient having CD with Extra Intestinal Manifestations (EIM) has been associated with increased morbidity and worse quality of life compared to their counterparts without EIMs.[5,6] Several studies have estimated the occurrence of Spondyloarthropathies (SpA) in patients with IBD ranging between 1-15% confirming that SpA is the most frequent extra-intestinal manifestation in patients with IBD.[6-11]

Case Presentation:

A 31 year gentleman having Ankylosing spondylitis for 15 years presented with complaints of recurrent episodes of abdominal pain and repeated blood transfusion for the last 1 year. The pain was located at periumblical region, sudden in onset, mild to moderate intensity, cramping in nature without radiation, aggravated after meal and relieved by oral medication and/or induced vomiting. Each episode of pain occurred 2-3 days apart and persisted for 3-7 hours. Pain was associated with vomiting which occurs 2/3 hours after meal. Vomitus contains partially digested bile stained

a. Dr. Mohammad Enamul Karim; MBBS, FCPS, MD
Assistant Professor, Department of Medical Gastroenterology
Sheikh Russel National Gastroliver Institute and Hospital, Mohakhali, Dhaka

b. Dr. Imteaz Mahbub; MBBS, MD
Registrar, Department of Medical Gastroenterology
Sheikh Russel National Gastroliver Institute and Hospital, Mohakhali, Dhaka

c. Dr. Subrata Paul; MBBS, FCPS
Medical Officer, Department of Medical Gastroenterology
Sheikh Russel National Gastroliver Institute and Hospital, Mohakhali, Dhaka

d. Dr. Khondoker Ehsanul Arefin; MBBS, MPH, MD
Associate Professor, Department of Paediatrics
Bangladesh Medical College, Dhaka

e. Dr. Md Elias Bhuiyan; MBBS,MRCP, FCPS
Assistant Professor, Department of Medicine
Bangladesh Medical College, Dhaka.

f. Dr. A.K.M Jubaere; MBBS, MD
Registrar, Department of Medical Gastroenterology
Sheikh Russel National Gastroliver Institute and Hospital, Mohakhali, Dhaka.

g. Dr. Rezina Parveen; MBBS, MD
Assistant Registrar, Department of Medical Gastroenterology
Sheikh Russel National Gastroliver Institute and Hospital, Mohakhali, Dhaka.

h. Prof.Dr. Sayeda Rahim; MBBS, MD
Professor, Department of Medical Gastroenterology
Sheikh Russel National Gastroliver Institute and Hospital, Mohakhali, Dhaka.

Correspondence to:
Dr. Mohammad Enamul Karim; MBBS, FCPS, MD
Assistant Professor, Department of Medical Gastroenterology
Sheikh Russel National Gastroliver Institute and Hospital, Mohakhali, Dhaka
Email: mdenkarim@gmail.com

food particles but not mixed with blood. During attack of pain, he did not notice abdominal distension or any visible lump. He had history of alteration of bowel habit for same duration in the form of loose stool. Loose stool occurred 3/4 times a day and continued for 2/3 days with 2/3 weeks interval. Amount of stool was scanty, liquid in consistency (Bristol stool chart: Type 6). The patient occasionally passed black tarry stool for the last 3 months. He gave no history of steatorrhoea. Loose motion was not related to milk or other foods. He also complained of weight loss about 12 kg over last 8 months. Weight loss was not associated with anorexia but he was afraid of taking food as abdominal pain was aggravatedafter meal.This patient had no history of fever, cough, skin rash, red eyes, oral ulcer, night sweats,heat intolerance, tremor, any contact with known TB patient. His bladder habit was normal.

He was diagnosed as Ankylosing spondylitis 15 years back at the age of 16 years on the basis of inflammatory type of back pain, asymmetrical oligoarthritis of large joints with relevant investigations. He suffered from GBS (Guillain Barre Syndrome) 4 years back; now on recovery state with some degree of weakness in both lower limbs.

He visited physician for several times and hospitalized only for blood transfusion not for abdominal pain/or loose stool. He got physiotherapy for residual weakness of GBS and 7 units of blood transfusion from December 2020 to January 2021.

He is 5th issue of non-consanguineous parents. All of his brothers are in good health. There is no such type of illness in his family. No family history of GI malignancy, IBD and liver diseases.His maternal uncle had similar type of spinal gesture problem.

He is non-smoker, non-alcoholic and nor having history of taking betel leaf and nut. He belonged to a middle class family.He completed graduation in political science. He has medicine business. He was prescribed Sulfasalzine and NSAIDs for ankylosing spondylitis. He took Sulfasalzine irregularly and stopped it after taking for initial 4 years. But he has been using diclofenac in the form of tablets occasionally to relieve back pain. He often takes Ciprofloxacin and Metronidazole in the tablet form for relieving loose motion. Recently he has taken anti-H pylori medication but his symptoms persisted.

On physical examination, the patient looked ill having BMI-17.He was anaemic. The power of the lower limbs decreased and Schober's test positive with restrictive body movement.Rest of the general and systemic examinations were found normal.

His initial investigations after admission were as follows: Haemoglobin 6.1 gm/dL with low MCV and MCH, RDW CV-23%, Neutrophil-68%, Lymphocyte-22%, Monocytes-07%, Eosinophil -03%, Platelet count-8,85,000/mL, ESR-58, RBS-6.4, S.creatinine -1.03 mg/dl, CRP - 48mg/dL, SGPT- 21 U/L , S. Albumin-3.15 gm/dL, S. TSH- 2.75 pg/mL, S. Electrolytes, S.lipase are within normal range, HLA B27 – Positive, Occult blood test-Positive, fecal calprotectin-1105 mcg/mg, MT- Negative, .

X-rays

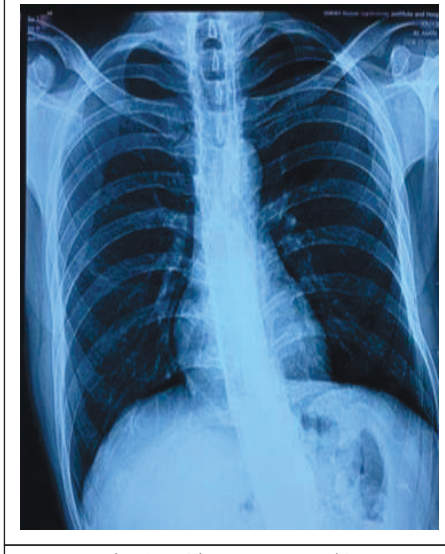


Fig 1a: Chest X-ray P/A:

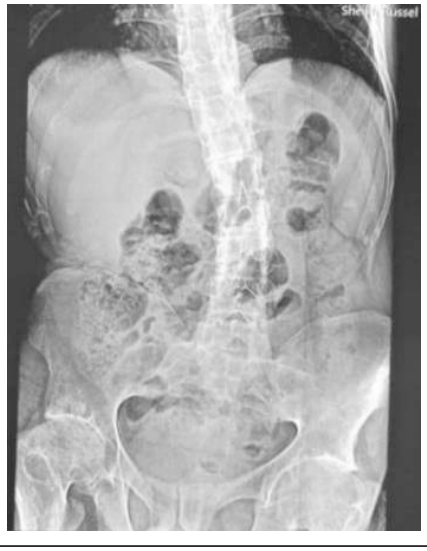


Fig 1b: X-ray of spine

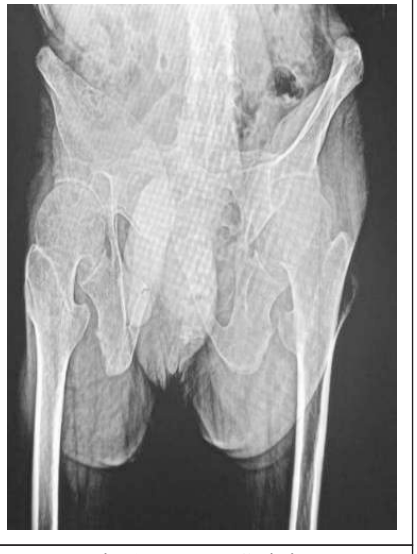


Fig 1c: X-ray SI joint

Chest X-ray was Normal **(**Fig 1a**)**, X-ray spine showed Bamboo spine and Scoliosis **(**Fig 1b**)** and X-ray of SI joints showed Sacroiliitis **(**Fig 1c**)**.

Endoscopy of upper GI tract

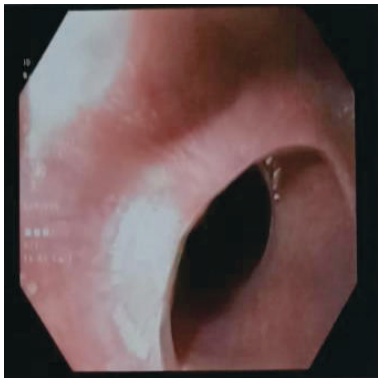


Fig 2a: Oesophageal web

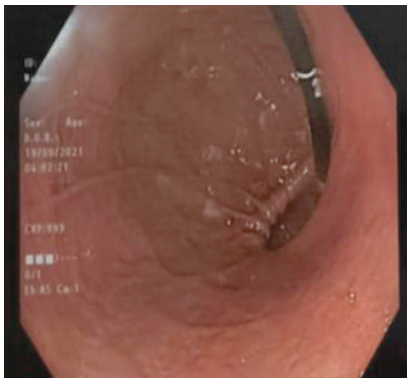


Fig 2b: Hiatus hernia

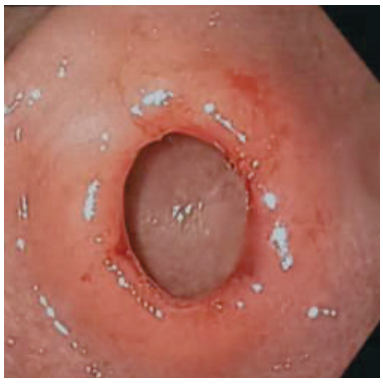
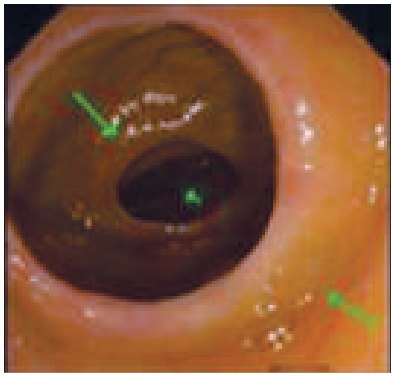


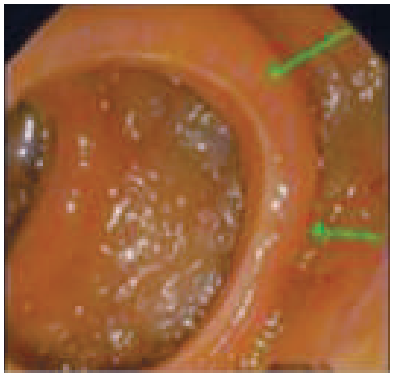
Fig 2c: Ulcer at rim of pylorus

It showed oesophageal web **(**Fig 2a), hiatus hernia **(**Fig 2b), and gastric ulcer with deformed bulb **(**Fig 2c**).** Biopsy was taken from the ulcers at stomach which revealed gastritis**.**

Single Balloon Enteroscopy

Multiple ring like ulcers (Fig 3) at mid ileum.





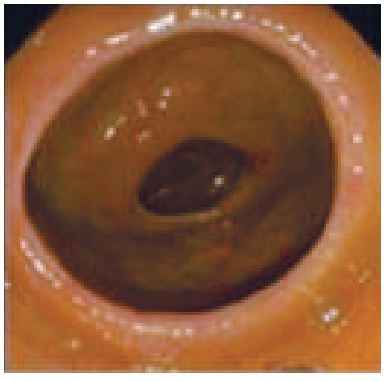
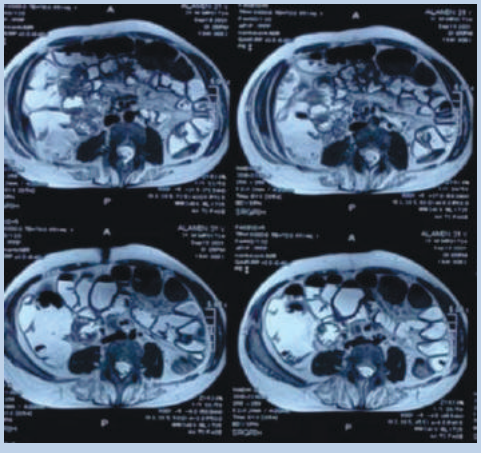


Fig 3: Single balloon enteroscopy showing multiple ring like circular ulcer and partial narrowing of the lumen



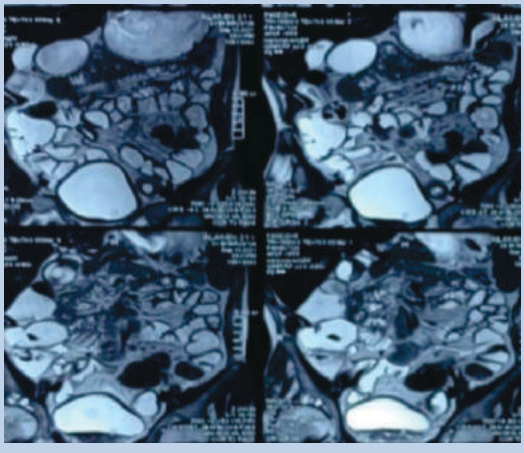


Fig 4:MR enterography shows mid ileal luminal narrowing and stricute

On the basis of history, clinical examination and relevant investigation, the patient is diagnosed as Crohn's Disease with Ankylosing spondylitis.

Discussion:

Concurrent incidence of Crohn's disease and Ankylosing spondylitis are not uncommon. It varies in different part of the world ranges from 1-15%.[6,7,8,9,10,11]In our case, the patient presented oligoarthritis and diagnosed as Ankylosing spondylitis earlier. After long 15 years later he had developed loose motion, abdominal pain, anaemia and diagnosed as Crohn's Disease. Song Liu et al described the symptoms of AS appeared earlier than CD in majority of patients, suggesting that CD is presumptively secondary to AS.[12]

The type of CD of this patient according to the Montreal classification is A2,L1+ L4,B2. A stands for the age of the patient. Age below 17 is A1, Age 17-40 is A2 and age above 40 will be titled as A3. The disease location is classified into L1 (terminal ileum), L2 (colon), L3 (ileo-colon), and L4 (upper gastrointestinal tract). The disease behavior is classified into B1 (inflammatory),B2 (stricturing), B3 (penetrating), and P (perianal lesion).Worth mentioning, the presence of chronic ileal lesions might be apredictor of an aggressive evolution of the spondyloarthropathy (SpA).[13]

Our patient presented with recurrent episodes of abdominal pain for 1 year which was similar to the data shown by Perler et al. According to that study, the most common presenting symptoms in CD found based on disease location at time of diagnoses were abdominal pain (82.14%) and tiredness/fatigue (72.41%) for ileal CD.[14]

Typical endoscopic findings in CD included discontinuous distribution of longitudinal ulcers (defined as ≥4 to 5 cm ulcers in the Japanese criteria), cobblestone appearance, and/or small aphthous ulcerations arranged in a longitudinal fashion.[15]In our patient, multiple ring like ulcers at mid ileum was found with histopathology showing active and chronic inflammation.

Fecal calprotectin (FC) is a biological marker of intestinal inflammation which was present in this patient. FC is one of the major proteins found in the cytosol of inflammatory cells.[16]The utility of FC as a surrogatemarker for endoscopic lesions in adults with IBD and found that cut-off value > 250 µg/g yielded sensitivity 71% and specificity 100% in distinguishing active and not active endoscopic mucosal disease.[17]In our case we found calprotectin level of 1105 mcg/gm which indicates active CD.

MR enterography is more effective than ultrasonogram in the evaluation of gastrointestinal tract, perianal region and complications in case of Crohn's disease.[18]Moreover, Lee et al have demonstrated that the effectiveness of MR enterography is comparable to that of CT enterography and also it has the advantage of not using ionizing radiations.[19]Aphthoid and deep ulceration, wall thickening (greater than 4 mm), intramural and mesenteric edema, stratified enhancement pattern of bowel wall, increased mesenteric vascularity, reactive lymphadenopathy are the common findings in MR Enterography in active Crohn's disease.[20] In MR Enterography, we found two short segment mild bowel wall thickening, mild luminal narrow ingand stricture formation at mid ileum and minimally englarged lymph nodes without central necorsis in our case.

IBD, however, precedes the development of spondylitis in most cases. On the other hand, a small proportion (<5%) of patients with established spondyloarthropathy can develop classic IBD within 10 years of the primary diagnosis: 80% of patients develop Crohn's disease and 20% ulcerativecolitis.[21] Same thing was found in our case. Patient was previously diagnosed as Ankylosing Spondylitis and now diagnosed as Crohn's disease.

It could be speculated that SpA and CD probably should be considered as distinct phenotypes of a common immune mediated inflammatory disease pathway rather than as separate disease entities and that ileitis of SpA might actually represent subclinical Crohn's disease.[22]

Treatment of overlapping Crohn's disease and Ankylosing spondylitis is not satisfactory sometimes. Low doses of systemic steroids and selective COXIBs may be used as a "bridge therapy" to oral SSZ (2–3 g/day). Inadequate response or intolerance to SSZ, biologic treatment (specific anti-TNFα) should be started.[23,24]

Conclusion:

Simultaneous presence of Ankylosing Spondylitis and Crohn's disease is rare. But they can overlap as both have immune mediated pathophysiology. So, extensive investigation is needed to diagnose vice versa.

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Adolescent adenomyosis- Curse or can cure?

Joty FS[a], Rahman A[b], Hossain M[c], Adhikary A[d]

Abstract

Juvenile cystic adenomyosis is a condition that affects nulliparous women between the ages of 13 and 20; the real incidence of this condition is unknown because so few cases have been reported. Adenomyosis appears to be quite uncommon before the age of 20, yet young women are primarily affected by the cystic type. Here, we report a 17 years old unmarried girl, admitted at CARe Hospital, Dhaka. She had a history of severe and worsening dysmenorrhea, cramps, and steadily increasing monthly flow since menarche, all of which had a negative impact on her day-to-day activities. Myometrial hyperechoic regions, uterine wall asymmetries, and intra-myometrial cystic regions were all identified by ultrasound. To confirm the diagnosis, magnetic resonance imaging (MRI) was then suggested. It described a weakly defined junctional zone of the endometrium with discontinuous variability of the surrounding myometrium, suggesting adenomyosis. The patient used Dienogest for a year after a regimen of continuous combined oral contraceptives (COC) pills containing mefenamic acid for pain relief for 6 months. Future fertility is a problem in this population, and managing the uterus's ongoing alteration is difficult.

Keyword: Adolescent, Adenomyosis, MRI.

Introduction:

Infrequently, severe dysmenorrhea in adolescents is caused by adenomyosis. This benign uterine condition causes globular enlargement of the myometrium with some cysts containing extravasated, hemolyzed red blood cells and siderophages.[1] It is characterized by endometrial glands and stroma that are deeper than 2.5 mm in the myometrium, as well as varying degrees of adjacent myometrial hyperplasia.[2] Juvenile cystic adenomyosis is a condition that affects nulliparous women between the ages of 13 and 20; the real incidence of this condition is unknown because so few cases have been reported. Adenomyosis appears to be quite uncommon before the age of 20, yet young women are primarily affected by the cystic type.[3,4] The aim of this case report is to draw attention to severe primary or secondary dysmenorrhea in adolescents and to emphasize the importance of being thorough when evaluating the underlying cause because the majority of instances go undiagnosed. Even though adenomyosis is rare, it should be remembered that if it is discovered, a doctor should be consulted to address the available treatments and any fertility issues.

Case Presentation:

Here, we report a 17-year-old unmarried girl who had a history of severe and worsening dysmenorrhea, cramps, and steadily increasing monthly flow since menarche, all of which had a negative impact on her day-to-day activities. She first used analgesics and Tranexamic acid to stop the bleeding and agony. She had Ultrasonography since her dysmenorrhea was uncontrollable, and the results showed that she had adenomyosis, myometrial hyperechoic areas, asymmetry in the uterine wall, and intramyometrial cystic areas (Fig 1). To confirm the diagnosis, magnetic resonance imaging (MRI) was then suggested. It described a weakly defined junctional zone of the endometrium with discontinuous variability of the surrounding myometrium, suggesting adenomyosis (Fig 2). The patient used Dienogest for a year after a regimen of continuous combined oral contraceptives (COC) pills and Mefenamic acid for pain relief for 6 months.

a. Dr. Fahmida Sharmin Joty; MBBS, DGO, FCPS
Assistant Professor, Department of Obstetrics & Gynaecology, Bangladesh Medical College, Dhaka

b. Dr. Ashrafur Rahman; MBBS, FCPS
Assistant Professor, Department of Surgery, National Institute of Cancer Research and Hospital (NICRH)

c. Dr. Munmun Hossain, MBBS, M. Phil (Physiology)
Assistant Professor, Department of Physiology
Shahabuddin Medical College, Dhaka

d. Dr. Alpana Adhikary; MBBS, FCPS
Associate Professor, Department of Obstetrics & Gynaecology, Shaheed Suhrawardy Medical College, Dhaka

Correspondence to:
Dr. Fahmida Sharmin Joty; MBBS, DGO, FCPS;
Assistant Professor, Department of Obstetrics & Gynaecology
Bangladesh Medical College, Dhaka
Email: joty2113@gmail.com

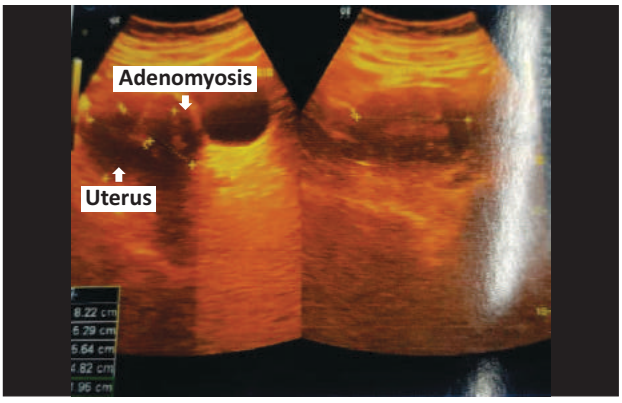


Fig 1: USG shows myometrial hyperechoic areas, asymmetry in the uterine wall, and intra-myometrial cystic areas.

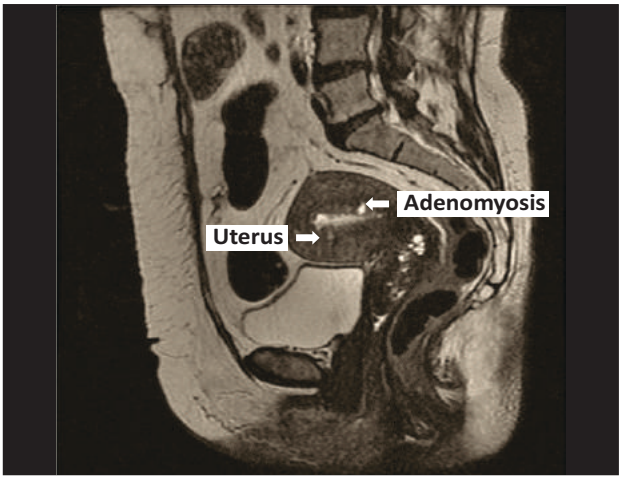
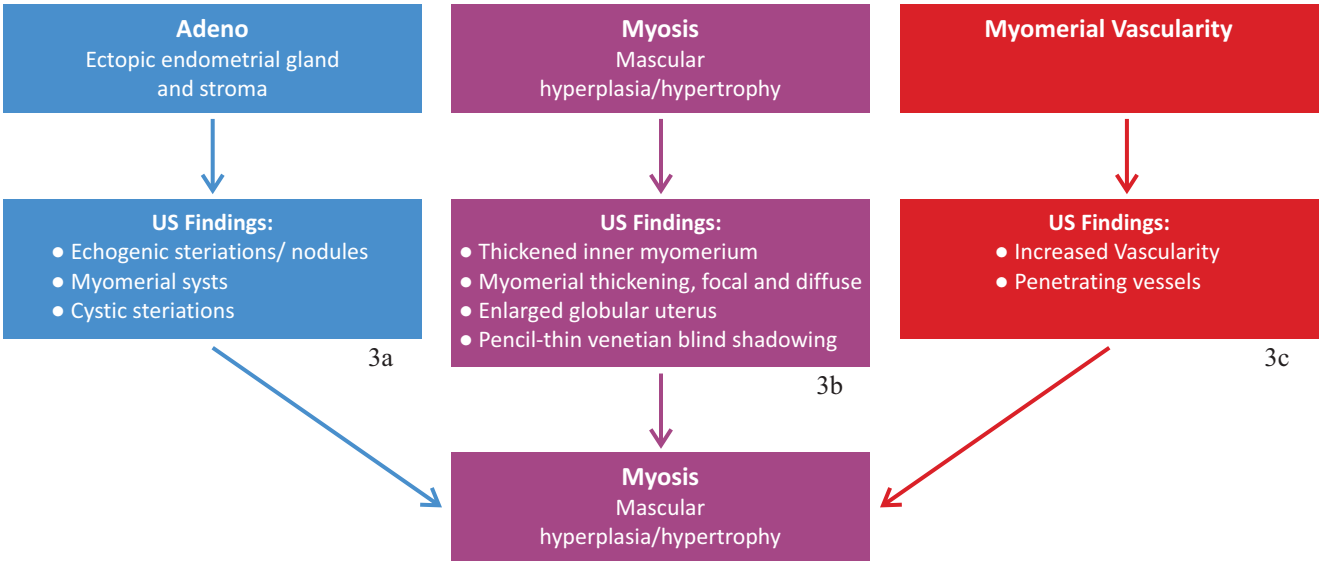


Fig 2: MRI shows weakly defined junctional zone of the endometrium with discontinuous variability of the surrounding myometrium, suggesting adenomyosis.

Discussion:

Discussing adenomyosis in young individuals, especially teenagers, is essential as it presents unique challenges and considerations. Despite the condition mainly affecting adult women in their third or fourth decade of life, adenomyosis is an uncommon but potential cause of dysmenorrhea and pelvic pain in young patients, as exemplified by the case of the 17-year-old girl mentioned. Although adenomyosis should still be considered in the differential diagnosis, endometriosis may be the most frequent cause of secondary dysmenorrhea in younger women.[5] Here in this case, the initial thought was primary dysmenorrhea most probably due to endometriosis. The invasion of endometrial tissue may not necessarily be mediated mechanically; it could result from endometrial-myometrial dysfunction, according to evidence of adenomyosis early in reproductive life and in the absence of prior surgery or pregnancy.[6] Now a day, transvaginal US is regarded as the main imaging technique for adenomyosis diagnosis. But as the girl was unmarried we could not do this. According to the histology of adenomyosis, US results can be categorized into three groups: (a) ectopic endometrial glands and stroma (Fig 3a), (b) muscle hyperplasia/hypertrophy (Fig 3b), and (c) enhanced vascularity (Fig 3c).



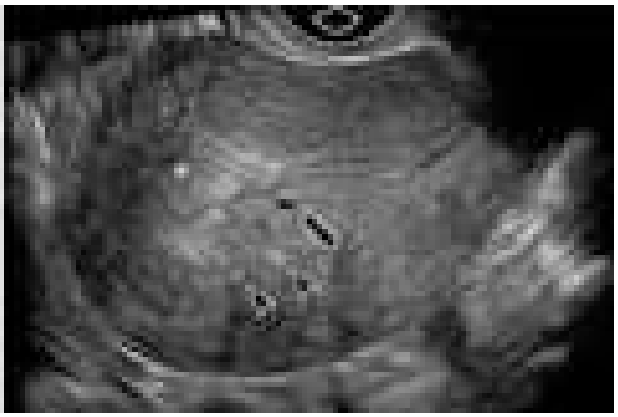


Fig 3a: USG finding of endometrial gland

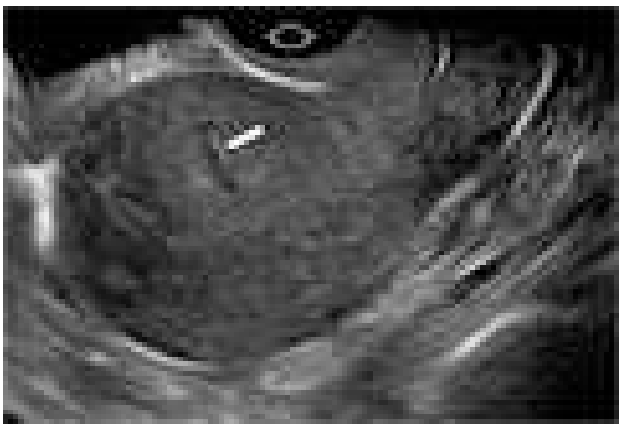


Fig. 3b: USG finding of myometrium

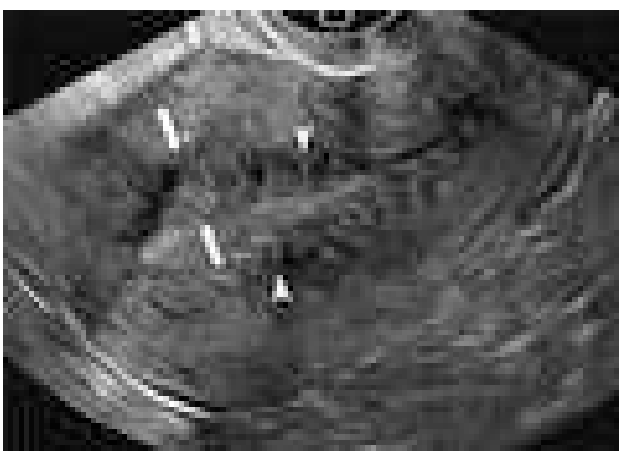


Fig. 3c: USG finding of myometrial vascularity

Echogenic nodules and striations that protrude from the endometrium into the myometrium are the outward signs of ectopic endometrial glands. Myometrial cysts and fluid-filled striations may be observed on ultrasound when the glands are fluid-filled. Focused or widespread myometrial thickening and globular uterine enlargement, frequently with thin "venetian blind" shadows, are caused by muscular hyperplasia and hypertrophy. When these data are combined, a heterogeneous myometrium with blurred endometrial borders is the consequence. Adenomyosis causes an increase in uterine vascularity, which is shown on color Doppler US as a pattern of penetrating vessels. Obtaining cine clips and coronal reformatted images, both of which can survey the entire endometrial-myometrial border, and performing saline-infusion sonohysterography, in which ectopic glands frequently fill with either air or fluid, are additional US techniques that are useful in the diagnosis of adenomyosis.[7] Since MRI has demonstrated potential advantages over TVUS in specific circumstances and because inter-observer variability has been reduced by specified diagnostic criteria, MRI is more warranted than TVUS.[8]

Diagnostic criteria for adenomyosis in magnetic resonance imaging (MRI)	
1	Thickening of the junctional zone, with a thickness exceeding 12 mm
2	Large, rand asymmetric uterus, with a maximum junctional zone thickness of at least 12 mm and punctate high-intensity myometrial foci
3	Bright foci on T2-weighted images, which represent foci of heterotopic endometrial tissue, cystic dilatation of endometrial glands or hemorrhagic foci

Although adenomyosis has typically been diagnosed histo-pathologically after surgery, in the majority of instances, MRI can provide a precise diagnosis of adenomyosis. Inhibiting ovulation, ceasing menstruation, and achieving a stable steroid hormone milieu are the goals of medical treatment.[9] Disease stability and remission prospects are provided by both medicinal and surgical therapy. Initial therapy for focal adenomyosis involves hormonal suppression with COC, especially for the female adolescent in whom preserving fertility is paramount. Adolescents may represent a subset of patients with adenomyosis whose lesions are completely treatable with hormonal therapy.[10] For adenomyosis, the goal of therapy is important, and can include symptom relief and possibly increased fertility. The therapeutic goal of medical treatment is not lesion resorption: Lesions survive any drug, at any dose, for any period of use, and come back after treatment discontinuation. Therefore, treatment should be tailored to the specific symptom or the special request of the individual patient. The findings of systematic literature studies have repeatedly shown that, if amenorrhea is achieved, there are no statistically significant variations in

pain relief across the many medications that are currently available, but that tolerability, side effects, and costs do differ significantly.[9] A thicker junctional zone, which alters the expression of molecules that modulate endometrial receptivity, has an impact on implantation with or without IVF.2 Adenomyosis and fertility may be related via several processes. Anatomical deformation of the uterine cavity may impact fertility by preventing sperm from migrating to the tubal ostia and preventing the transfer of embryos. It was shown that macrophage density was higher in adenomyosis, and macrophages are known to produce pro-inflammatory cytokines and reactive oxygen species that can harm developing embryos. Progesterone and estrogen receptor expression, including estrogen receptor-, can vary as a result of the inflammatory cytokine IL-6, which modulates receptivity.[10]

Conclusion:

In conclusion, this case highlights a rare but little-known factor that can afflict young nulliparous women and produce both primary and secondary dysmenorrhea. Even in cases where TVUS is normal, MRI can help with diagnosis. In this population, maintaining fertility is the main goal, hence COC hormonal suppression is used as the initial therapy for localized adenomyosis. Due to the few findings in the literature on the management of adenomyosis in adolescents, more research is required. Clinicians can better educate their patients by explaining the connection between adenomyosis and infertility to them. The first stage in your patients' fertility journey may be to accurately diagnose their condition using ultrasound.

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Bronchogenic cyst: A case report

Khanam R[a], Jahan F[b]

Abstract

Bronchogenic cysts are rare congenital malformations which arise from abnormal budding of the primitive tracheobronchial tube. It can localize to either the mediastinum or lung parenchyma. Bronchogenic cysts are often solitary and rarely multiple. Most bronchogenic cysts do not show any symptoms. When it compresses the surrounding structures then symptom arises. Infections involving bronchogenic cysts are often polymicrobial. Gram-positive, gram-negative, and mycobacterial infections have been reported, though frequently a pathogen is not identified. We represent the case of a 20 years' female who was diagnosed incidentally. She underwent lobectomy with culture positive for Klebsiella Spp. The patient recovered following a course of antibiotic.

Keywords: Bronchogenic cysts, Emphysematous bullae, Mediastinal mass, Lobectomy.

Introduction:

Bronchogenic cysts arise during the process of the development from the abnormal or late budding of the ventral lung bud or the tracheobronchial tree. Most of the bronchial branches are formed within the 15th week of development in fetal life, but they continue to divide and completed in eighth year. Bronchogenic cysts are found in babies or infants but can be detected in fetus or in stillbirths also. About 10–15% of all primary mediastinal masses are bronchogenic cysts. It can be classified as either intrapulmonary or mediastinal. Overall, 72% of bronchogenic cysts produce some symptoms, but 90% of mediastinal type bronchogenic cysts are reported to be asymptomatic.[1] Mediastinal type bronchogenic cysts are classified into five types: para-tracheal, carinal, hilar, para-oesophageal, or miscellaneous.[2] The para-tracheal or carinal types can produce symptoms such as dyspnea or chest pain, due to compression of the trachea or bronchi. A giant carinal type of mediastinal type bronchogenic cysts can compress the left atrium of heart due to its proximity to the heart. On histopathologic examination the cyst is lined by ciliated, secretory respiratory epithelium with cartilage, smooth muscle, fibrous tissue fibrous tissue and mucus gland. The cavity of the bronchogenic cyst may be filled with fluid or air or both. It is due to communication with the tracheobronchial tree.[3] Usually, the bronchogenic cysts don't produce any symptoms but it may produce pressure symptoms to the surrounding structures. Due to workload or on exertion the patient might complain of heaviness in chest specially.

a. Dr. Rehana Khanam; M. Phil (Path), MBBS
Associate Professor, Department of Pathology
Bangladesh Medical College, Dhaka

b. Dr. Fauzia Jahan; M. Phil (Path), MBBS
Professor (CC) of Pathology, Bangladesh Medical College, Dhaka

Correspondence to:
Dr. Rehana Khanam; M. Phil (Path), MBBS
Associate Professor of Pathology
Bangladesh Medical College, Dhaka
Email: rehana-khanam@hotmail.com

The electrocardiogram can reveal left atrial overload.[1] The echocardiography and computed tomographic (CT) scan can find out the exact cause of the atrial overload. It can affect any compartment of the mediastinum but most common radiological position is in the subcarinal region of the lung. The CT report shows well-defined, rounded, non-enhancing cystic masses. The appearance of the fluid can vary from water density to higher one according to secretion type. Some bronchogenic cysts may have flecks of calcium within the fluid, the so-called milk of calcium.
Magnetic resonance imaging (MRI) appearance may vary according to the nature of the fluid of the cyst. In T1-weighted imaging it can be low (grey to black) or high (white to grey) signal intensity. In T2-weighted imaging it is bright (white) signal intensity. In T1-weighted images, fat, proteinaceous and hemorrhagic fluids appear in white and water appears in low signal intensity. In T2-weighted images water, with or without proteins, appears in high signal intensity.[4]

Case Presentation:

A female of 22 years having B-negative blood group was admitted to surgery department of Dhaka Medical College Hospital with chest pain and shortness of breath for one year. Her shortness of breath was associated with exertion and it was relieved by taking rest. The patient has been suffering for mild chest pain and was accidentally diagnosed as having right sided emphysematous bullae. On admission the patient had mild fever. But she did not complain of any cough or sputum production. On laboratory investigations her routine hematological test, electrolytes creatinine and TSH were within normal limit. Her tuberculin test came out to be negative. Her culture was positive for Klebsiella Spp. Her chest x-ray revealed large right sided emphysematous bullae. Her CT scan shows right sided emphysematous bulla with contralateral shifting of mediastinum and streaky fibrotic change in right upper lung presumably as a sequel of old healed tuberculosis. After all preoperative assessment right lower lobectomy was done (Fig: 1,2). Post-operative chest X-ray reveals satisfactory lung expansion but there was clotted

blood inside the pleural cavity. So for fibrinolysis, Injection Streptokinase, 500,000 IU was given through ICT (intercostal drainage tube) for 6 days. Injection Streptokinase clot cleared. Post operative wound was healthy, tube was removed. Histopathology report revealed inflamed bronchogenic cyst with bronchiectasis (Fig:3).

Discussion:

Bronchogenic cysts are rare congenital anomalies that arise in early gestation as a result of abnormal budding of the developing respiratory system. As it is a space occupying lesion in the mediastinum, so the appearance of the symptoms of the patients depend on the position and most importantly the size of the cyst. As they enlarge, they may produce symptoms by compressing the surrounding structures. My patient developed chest pain and shortness of breath for one year. Bronchogenic cysts predominantly occur in males and are often solitary and rarely multiple.[5,6]

Typically, bronchogenic cysts have ciliated epithelium with cartilage, smooth muscles and mucus glands similar to the bronchial walls. Bronchogenic cysts lack alveolar structures because the abnormal budding occurs before the formation of the alveoli. Intrathoracic bronchogenic cysts have two radiographic classifications based on location: Mediastinal and Intrapulmonary.[2] Mediastinal bronchogenic cysts appear as smooth, rounded, thin-walled, unilocular, sharply circumscribed structural opacities in the mediastinum adjacent to the tracheobronchial tree, often below the carina. In 80% cases their location is in the middle mediastinum, 17% in the posterior and 3% in anterior mediastinum. Intrapulmonary bronchogenic cysts are mostly found in the lower lobes in the medial third of the parenchyma same as this case. They vary in size. Bronchogenic cysts are found in other location like subcutaneously and in the pericardium, thymus, cervical region, diaphragm, retro-peritoneum, abdomen, esophagus and sternum.[4,5,6]

Approximately 75% of bronchogenic cysts do not show any symptoms. The occasional symptoms such as cough, chest pain, dyspnea, or fever may develop.[7] The size, location and compression of the surrounding structures by the cyst are important factors in producing symptoms. If the bronchi are affected recurrent respiratory infections may occur; superior vena cava syndrome occurs due to involvement of superior vena cava. If the bronchogenic cyst is located near the pulmonary artery/atrium hypoxemia, pulmonary hypertension, or atrial fibrillation may develop. Additional complications include infection of the cyst contents, fistulae to surrounding structures in the chest, cyst rupture, and hemorrhage into the cyst cavity.[7,8,9]

Superimposed cyst infection is usually the result of communication with the tracheobronchial tree. Intra-parenchymal bronchogenic cysts are more likely to have a connection with the tracheobronchial tree than mediastinal cysts and are thus more prone to infectious complications.[4,6] Mediastinal cysts, on the other hand, rarely fistulize with the bronchi or lung parenchyma and thus seldom become infected. In one of the largest reported series of 86 patients, St-Georges et al. found only one infected mediastinal cyst; the source of infection was unknown as this cyst was not fistulized.[7]

Cyst recurrence following incomplete excision as has been reported as well as malignant transformation into adenocarcinoma, squamous cell carcinoma, or carcinoid tumors.[7,9] As mentioned above, several cases of bronchogenic cyst recurrence after incomplete surgical resection have been reported. Recurrence of the cyst usually takes years to appear.[10,11]

Conclusion:

Asymptomatic patients with known bronchogenic cysts may eventually develop symptoms and serious, life-threating complications. The cysts may become infected, either by spread of bacteria from the adjacent tracheobronchial tree or by hematogenously. Mediastinal cysts are usually affected through haematogenous route. In addition to appropriate antimicrobial therapy, prompt surgical excision is necessary. For symptomatic bronchogenic cysts, complete surgical excision is usually the goal, but trans-bronchial needle aspiration (TBNA) is an alternate method of decompression that can instantly relieve the pressure on surrounding structures.

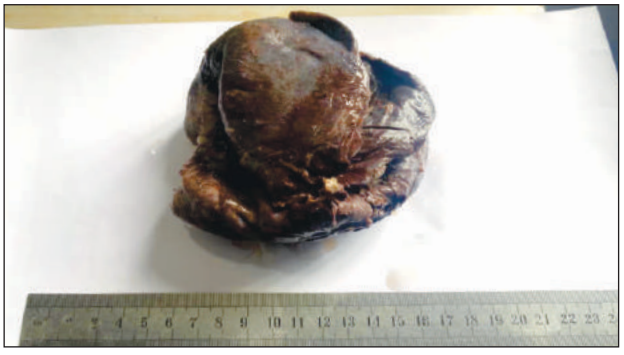

Fig 1: Resection specimen of broncogenic cyst.

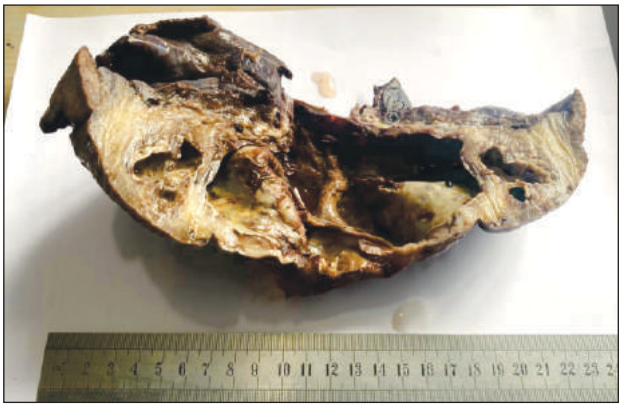

Fig 2: Resection specimen of cut section of broncogenic cyst.

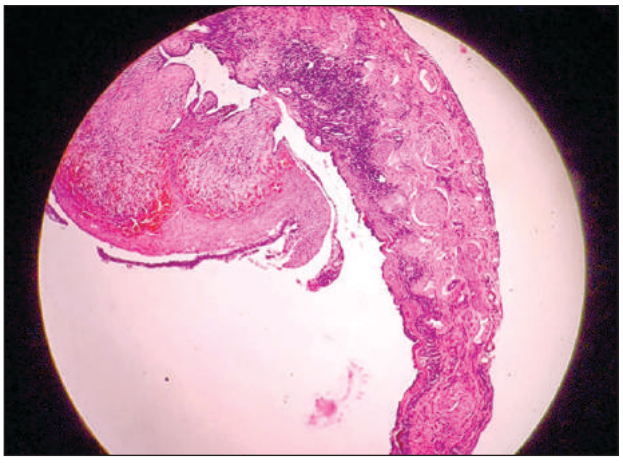

Fig 3: Cystic structure partly lined by atrophic respiratory epithelium. The wall has been infiltrated by many acute and chronic inflammatory cells. Focal area shows hemorrhage. The lining epithelium is focally ulcerated.

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College News

College Events:

- The National Mourning Day was observed on 47th death anniversary of Father of Nation Bangabandhu Sheikh Mujibur Rahman in Bangladesh Medical College and Hospital on 15th August, 2022. Teachers, doctors, nurses, students of BMC & BMCH, officials & staffs of BMSRI participated in that event.

- 51st Victory Day of Bangladesh was celebrated in Bangladesh Medical College and Hospital premises on 16th December 2022. Teachers, doctors, nurses, students of BMC & BMCH and officials & staffs of BMSRI participated in that event.

- Commencement ceremony of newly admitted students of BM-36 was held on 1st August, 2022 at the BMC auditorium. Total 120 students were admitted in the session of 2021-2022. Hon'ble Minister, Ministry of Agriculture, Government of Peoples' Republic of Bangladesh Dr. Md. Abdur Razzak MP, Chairman, E.C, BMSRI was present as Chief Guest of that ceremony.

Seminar/Workshops:

- Seminar on "NAFLD" was held on 11.08.2022. The speaker was Prof. Dr. Faizul Islam Chowdhury, Professor, Dept. of Medicine, Anwar Khan Medical College.

- CME on "Training on Teaching Methodology and Assessment (Phase – II)," Topic: "Effective teaching and learning" was held on 23.08.2022. The presenter was Dr. Khondoker Ehsanul Arefin, Associate Professor, Dept. of Paediatrics, BMC.

- CME on "Educational objectives" was held on 30.08.2022. The presenter was Prof. Dr. Md. Dabir Hossain, Professor, Dept. of Medicine, BMC.

- CME on "Lesson plan" was held on 11.09.2022. The presenter was Prof. Dr. Sharmeen Yasmeen, Professor and Head, Dept. of Community Medicine, BMC.

- CME on "Effective delivery of lecture" was held on 20.09.2022. The presenter was Prof. Dr. Rehnuma Tasnim Chowdhury, Professor and Head, Dept. of Pharmacology and Therapeutics, BMC.

- Seminar on "Celebration of prostate cancer awareness month -September" was held on 25.09.2022. The speakers were Prof. Dr. Abdus Salam, Ex-Chairman and Professor, Dept. of Urology, BSMMU, Prof. Dr. Md. Fakrul Islam, Professor and Chief Consultant, Dept. of Urology, BMCH and Prof. Dr. Zafor Md. Masud, Professor and Head, Dept. of Oncology, BMC

- CME on "Small group teaching" was held on 27.09.2022. The presenter was Dr. Sharmila Huda, Associate Professor, Dept. of Pharmacology and Therapeutics, BMC

- CME on "Assessment: Concept, principles and methods" and "SAQ-MCQ" was held on 15.10.2022. The presenters were Prof. Dr. Zafor Md. Masud, Professor and Head, Dept. of Oncology, BMC and Prof. Dr. Md. Mizanur Rahman, Professor and Head, Dept. of Opthalmology, BMC.

- CME on "OSCE - OSPE" was held on 20.10.2022. The presenter was Prof. Dr. Syed Khalid Hasan, Professor, Dept. of Surgery, BMC.

- CME on "Structured oral examination" was held on 30.10.2022. The presenter was Dr. Sadia Saber, Assistant Professor, Dept. of Medicine, BMC.

- CME on "Integrated Teaching" was held on 10.11.2022. The presenter was Prof. Dr. Mahfuja Rahman, Professor, Dept. of Biochemistry, BMC.

- CME on "Effective use of teaching material" was held on 15.11.2022. The presenter was Dr. Sultana Jebunnahar, Associate Professor, Dept. of Gynae & Obs., BMC.

- Celebration of "World Antibiotic Awareness Week" was held from 18th to 24th November, 2022. A series of seminar on "Antimicrobial resistance and present scenario in BMC," and, "Awareness on antimicrobial resistance" was organized jointly by Dept. of Microbiology and Dept. of Pharmacology & Therapeutics on 19.11.2022. The speakers were Prof. Dr. Farhana Alamgir Moony, Professor and Head, Dept. of Microbiology, BMC and Dr. Sharmila Huda, Associate Professor, Dept. of Pharmacology & Therapeutics, BMC. Panelists were Prof. Dr. Sharmeen Yasmeen, Professor and Head, Dept. of Community Medicine, BMC and Prof. Dr. Md. Dabir Hossain, Professor, Dept. of Medicine, BMC. Other events were as follows:

i) Opening ceremony – Principal, BMC and Director, BMCH were present and delivered speech among the participants. Leaflets were distributed to the common people by them to create awareness.

ii) Rally of doctors, interns and students of BMC and BMCH.

iii) Poster presentation by students at common room and photo session.

iv) Leaflet distribution among hospital nurses and counseling.

v) Quiz contest participated by the students.

vi) Leaflet distribution among outdoor and indoor patients and counseling.

vii) Raising awareness among school students regarding antibiotic resistance.

viii) Distribution among pharmacy sales persons and medical representatives.

ix) Debate competition by students.

x) Announcement of results of the competition, crest and certificate giving ceremony and photo session.

- CME on "Educational Management" was held on 29.11.2022. The presenter was Prof. Dr. Md. Tarek Alam, Professor and Head, Dept. of Medicine, BMC.
- CME on "Integrity in Medical Education" was held on 29.11.2022. The presenter was Prof. Dr. Paritosh Kumar Ghosh, Principal, BMC and Professor and Head, Dept. of Pathology, BMC

Participation in the International Conferences/Seminars/Workshop/ Congress/Meetings:

- Prof. Dr. M. Touhidul Haque, Professor and Head, Dept. of Cardiology, BMC attended 7th International SPACE Cardiology from 7th to 11th October, 2022 in Switzerland.
- Dr. Rezwanur Rahman, Associate Professor, Dept. of Nephrology, BMC attended the 59th Annual Nephrology Congress organized by European Renal Association (ERA) from 19th to 22nd May, 2022 in France.
- Prof. Dr. Md. Ashraful Islam, Professor and Head, Dept. of ENT, BMC attended Diploma Ceremony on 7th September, 2022 in Glasgow, UK.
- Prof. Dr. Zafor Md. Masud, Professor and Head, Dept. of Oncology, BMC attended the ESMO Congress 2022 from 9th to 13th September, 2022 in Paris, France.
- Dr. Jamal Uddin, Professor and Head, Dept. of Dermatology, BMC attended 31st EADV Congress from 7th to 11th September, 2022 in Milan, Italy.
- Dr. Hasan Khalid Md. Munir, Assistant Professor (RS), Dept. of Orthopaedics, BMC attended the Combined Meeting of TOSSM & APKASS 2022 from 29th to September to 1st October, 2022 in Thailand.
- Dr. Halima Begum, Associate Professor, Dept. of Radiology & Imaging, BMC attended the training at Focus Diagnostics from 11th to 17th September, 2022 in Hyderabad, India.
- Dr. Saikat Barua, Associate Professor C. C, Dept. of Radiology & Imaging, BMC attended the training at Focus Diagnostics from 11th to 17th September, 2022 in Hyderabad, India.
- Dr. Md. Saydur Rahman, Professor C. C, Dept. of Orthopaedics, BMC attended the 42nd SICOT Orthopaedic World Congress from 28th to 30th September, 2022 in Kuala Lumpur, Malaysia.
- Prof. Dr. Md. Khaled Noor, Professor of Neonatology, Dept. of Paediatrics, BMC attended the 8th International Congress on Probiotics, Prebiotics, Postbiotics in Paediatrics from 15th to 17th September, 2022 in Valencia, Spain.
- Dr. Fahmida Sharmin Joty, Assistant Professor, Dept. of Gynae & Obst., BMC attended RANZCOG International Fellowship Program (IFP) from 4th to 7th October, 2022 in Australia.
- Dr. A. T. M. Zulfiqur Rahman, Assistant Professor, Dept. of Orthopaedics, BMC attended the AO Trauma Masters Course-Management of Pelvic and Acetabular Fractures (with Anatomical Specimen) from 7th to 10th October, 2022 in Dubai, United Arab Emirates.
- Dr. Sadia Saber, Assistant Professor, Dept. of Medicine, BMC attended International Conference on Global Healthcare and Nutrition from 17th to 18th November, 2022 in Paris, France.
- Dr. Mohammad Aftab Haleem, Assistant Professor, Dept. of Neuromedicine, BMC attended the 14th World Stroke Congress from 26th to 29th October, 2022 in Singapore.
- Dr. Muhtamim Chowdhury, Assistant Professor, Dept. of Neurosurgery, BMC attended the 2022 CNS Annual Meeting from 8th to 12th October, 2022 in California, USA.
- Prof. Dr. Md. Tarek Alam, Professor and Head, Dept. of Medicine, BMC attended the 26th Congress of the Asian Pacific Society of Respirology (APSR 2022) from 17th November to 20th November, 2022 in Seoul, South Korea.
- Dr. Yasmin Aktar, Assistant Professor, Dept. of Endocrinology, BMC attended the International Diabetes Federation (Congress 2022) from 5th to 8th December, 2022 in Lisbon, Portugal.
- Dr. Rezwanur Rahman, Associate Professor, Dept. of Nephrology, BMC attended 52nd Annual National Conference – (ISNCON 2022) from 1st to 4th December, 2022 in India.
- Dr. Sonia Mahjabin, Assistant Professor, Dept. of Nephrology, BMC attended 52nd Annual National Conference – (ISNCON 2022) from 1st to 4th December, 2022 in India
- Dr. Muhammed Akhtaruzzaman, Associate Professor, Department of Cardiology, BMC attended 74th Annual CSI Conference Chennai 2022 by the Cardiological Society of India from 8th to 11th December, 2022 in India.

New Promotions in BMC:

- Dr. Syed Khalid Hasan, Professor, Department of Surgery.

- Dr. Mushtaque Ahmed Rana, Professor, Department of Gastroenterology.
- Dr. Farhana Hossain, Associate Professor, Department of Anatomy
- Dr. Nargis Sultana, Assistant Professor, Department of Anatomy

New Appointments in BMC:

- Dr. Md. Masud Rana, Assistant Professor, Department of Urology.
- Dr. Rafa Faaria Alam, Registrar, Department of Medicine.
- Dr. Oyeshi Bhattacharjee, Lecturer, Department of Physiology.
- Dr. Sumayya Binte Abdur Razzaque, Lecturer, Department of Physiology.
- Dr. Tania Mawla, Lecturer, Department of Physiology.

Prepared by:
Shahana Akter Dalia
Senior Admin. Assistant
Bangladesh Medical College

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