ISSN 2518-3249

Vol. 5 No. 1
January 2019

Barind Medical College Journal

Children with epilepsy need proper assessment for associated comorbidities. Treatment of these coexisting disorders is very important to achieve the best seizure control and overall quality of life for the patients and their families.

See Original Article Page 05

OFFICIAL JOURNAL OF
BARIND MEDICAL COLLEGE
Recognised by BM&DC

BARIND MEDICAL COLLEGE JOURNAL (BMCJ)

Official Journal of Barind Medical College

Volume 5 Number 1 **January 2019**

Contents

BARIND MEDICAL COLLEGE JOURNAL (BMCJ)

Volume 5 Number 1 January 2019

Official Journal of Barind Medical College

Paulus Aegineta (625– 690)

Paul of Aegina or Paulus Aegineta (625 – 690) was a 7th-century Byzantine Greek physician as born in Aegina in 625. He was educated at the University of Alexandria, and grew to become a famous Greek physician. “He is the father of early medical books medical encyclopedia." He also provided us a very early description of asthma. He became a very skilled surgeon who provided many achievements in the surgical process. He was among the first to describe a process called bronchotomy, which was an old term for tracheotomy. Some consider him the originator of plastic surgery.

Published By
Barind Medical College
Rajshahi, Bangladesh

Annual Subscription
Tk. 200/- for local subscribers
US$ 20 for overseas subscribers

BMCJ, a peer reviewed biannual medical journal, is the official journal of Barind Medical College, Rajshahi, Bangladesh.

Physiotherapy in Bangladesh : A bleak situation

Md. Anayet Ullah

Professor, Department of Community Medicine, Barind Medical College, Rajshahi, Bangladesh.

Correspondence to : MA Ullah md.anayet_u.@yahoo.com

Cite this as: BMCJ 2016; 2(2):1-4

Received : 8 December 2018

Accepted : 19 December 2018

Physiotherapy is a type of therapy that can help ill, injured or disabled people recover movement and function to their full potential.[1] Physiotherapy or physical therapy is a branch of rehabilitative medicine aimed at helping patients maintain, recover or improve their physical abilities.[2] It promotes healthy lifestyles, treats, and prevents many problems caused by illness, injury, pain, disease, age, and inactivity. Physiotherapists work in many diverse settings, including hospitals, emergency medical teams, community settings, hospices, nursing homes, health centers, education, and research.

Concomitant with the aging of population in Bangladesh is a significant rise in the prevalence of chronic diseases like arthritis, stroke, Neuromuscular diseases etc. This in turn has increased the need for physiotherapists and physical therapy services by all health agencies. The unprecedented need for services may outstrip the capabilities of the existing medical facilities.[3]

According to the WHO, 15% of the world's population suffers some form of disability and 80% of these can be found in developing countries.[4,5] Gupta et al. estimates that 92% of the disease burden in the world is related to causes requiring health professionals associated with physical rehabilitation.[6] Noncommunicable diseases (NCDs), including injuries, now account for 60% of deaths in Bangladesh.[7] A 2010 government survey found a high incidence of smoking (26%), diabetes (3.9%), hypertension (17.9% stage I, 5.5% stage II), and low physical activity (27%) within Bangladesh.[8] These are all contributing factors to NCDs such as cancer, chronic lung diseases, stroke, and other cardiovascular disease, which fall under the top five causes of death in the country.[7] The size and skill of the current health workforce is inadequate to deal with an increasing prevalence of NCDs which require a huge preventative health care. Rehabilitation specialists such as physiotherapists remain a largely untapped resource in Bangladesh. Participation in physical exercise can help prevent and reduce many chronic diseases, and physiotherapists can advise on the management and prevention of future health problems associated with NCDs. According to the WHO estimate 2011, the cost of preventative action would be US$11.4 billion per year across all low- and middle-income countries which equates to the annual cost of less than US$1 per person living in a low-income country.[9] Addressing NCDs should be the top priorities not only for health but for rapid economic development of the country through saving scarce resources.

Manual labor and the garments industry are noted for their poor work conditions. In a small-scale study, almost 62% of workers in a garments factory suffered some form of musculoskeletal problems.[10] According to the Global Burden of Disease 2015 study, the health problems, which resulted in the two highest causes for disability in Bangladesh, were back and neck pain and "other musculoskeletal problems".[11] Physiotherapy is effective in reducing both acute and chronic pain, limiting the risk of further disability and contributes to improved physical function, including return to work and recreational activity.[12-14] Rising motorization in South Asia has not been accompanied sufficiently by improvements in road safety strategies. According to a WHO report, 1.3 million deaths globally are due to road traffic accidents (RTAs), 90% of which occur in low- to middle income countries.[15] In addition to this, between 20 and 50 million people are estimated to have nonfatal injuries, with many of those who survive left with temporary or permanent disabilities. According to the Bangladesh Health and Injury Survey 2016, there are over 23,000 road traffic fatalities a year in

Bangladesh, equating to 64 deaths a day. Over 3.4 million people a year suffer nonfatal injuries as a result of RTAs with over 80,000 experiencing permanent disability.[16] Effective rehabilitation interventions is very essential for these victims.

In addition to the physical and emotional toll on those affected, disabilities can also incur a considerable economic loss to victims, their families, and the nation as a whole. Losses arise from the cost of treatment, reduced or lost wages, and for family members who need to take time off work to care for the disabled. A study published in April 2017 found that residents in Bangladesh faced serious difficulties with health-care financing; 1 in 10 households incurred financial catastrophe and 1 in 20 non-poor households became poor due to health-care costs.[17] Those who are poor are more likely to become disabled, and those who are disabled are more likely to become poor. Besides the direct benefits physiotherapy can have to the health and quality of life of a patient population, rehabilitation can also have a positive impact on the economy. A period of structured rehabilitation for injuries and musculoskeletal problems can reduce the degree of impairment, restoring function, improving recovery time, and return to work, thus reducing the financial burden.[18,19]

At present, the health system does not have the capacity to answer the needs of these patients, leaving them without a proper treatment, at risk of further complications and hamper their reintegration into society. Physiotherapy plays an integral role to promoting and improving health in a population. With this in mind, it is vital that the government addresses the paucity in a workforce that is skilled in addressing rehabilitation needs, thus improving quality of life and enabling those with disabilities to be able to contribute to the economy. The sustainable development Goals (SDGs) cannot be effectively achieved without addressing the unmet needs for rehabilitation services.[20]

Rehabilitation services in public and private health care poorly exist in Bangladesh. As a result, physiotherapy is not sufficiently included in health policies by the government and is both under-resourced and underfunded. The government recruits no qualified physiotherapists in the public health sector. Stroke, fractures, amputees, and spinal cord injuries are just a few of the many conditions that receive neither inpatient nor community rehabilitation through the public sector. Many of these patients are simply discharged home once medically fit without any follow-up or rehabilitation which could reduce their dependence and help integrate them into society. This is a significant oversight by a government that is committed to implementing the global 2030 SDGs. Universal health coverage is a prominent part of the SDGs and aspires to ensure that all people can use the promotive, preventative, curative, rehabilitative, and palliative health services they need, while also ensuring that the use of these services does not expose the user to financial hardship.[21]

There is a severe shortage of physiotherapists to serve the huge population of Bangladesh. Whereas approximately 54.7 thousand physiotherapists were registered in the United Kingdom in 2017 (a population of around 65 million), only an estimated 1.7 thousand physiotherapists (BSc graduates) exist in Bangladesh today (a population of around 160 million). Ironically, the employment opportunities for these physiotherapists are very limited in Bangladesh. Many graduates set up private practices and few find work in private hospitals, nongovernmental organizations (NGOs), or seek employment abroad.[22]

This drab situation can be largely attributed to several major issue: firstly, BSc physiotherapists are not formally recognized by the government. As a result, they are not employed to work clinically in the public health sector. Despite BSc qualifications being issued by government institutions, only diploma

physiotherapists (known as health or medical technologists) are employed to work in public hospitals at this time.[23] This work is carried out under the instruction of a physiatrist (a doctor trained in physical medicine), rather than a physiotherapist, and is largely restricted to electrotherapy in a musculoskeletal outpatient setting. Secondly, as like many developing countries in Bangladesh, physiotherapy is poorly understood. Most of the, common people, health care provider and even health planer and policy maker are not aware about the physiotherapy.

In order to reach SDGs goal by 2030, the country will have to improve the availability and the skill mix of its health workforce.[24] and acknowledging the specialty of physiotherapist in providing rehabilitation is an essential step.

References:

1. Physiotherapy. NHS-choices. Available at: http://www.nhs.uk/Conditions/Physiotherapy/Pages/Introduction.aspx ; last accessed December 18, 2018.
2. What Is Physical Therapy (physiotherapy)? What Does A Physical Therapist (physiotherapist) Do? MNT-Medical News Today. Available at: http://www.medicalnewstoday.com/articles/160645.php. last accessed December 18, 2018.
3. J. B. Allis, "Orienting the physical therapist to public health practice," Public Health Reports 1965; 80(11): 975–80.
4. World Health Organization. World Report on Disability: Summary, 2011. Available at: http://www.who.int/disabilities/world_report/2011/report. pdf; last accessed November 26, 2018.
5. World Health Organization. Global Disability Action Plan 2014–2021—Better Health for All People with Disability. (2015). Available at: http://apps.who.int/iris/bitstream/10665/199544/1/ 9789241509619_eng. pdf?ua=1; last accessed November 14, 2017.
6. Gupta N, Castillo-Laborde C, Landry MD. Health-related rehabilitation services: assessing the global supply of and need for human resources. BMC Health Serv Res (2011) 11:276. doi:10.1186/1472-6963-11-276
7. World Health Organisation. Noncommunicable Diseases (NCD) Country Profiles—Bangladesh. (2014). Available at: http://apps.who.int/ iris/bitstream/10665/128038/1/9789241507509_eng.pdf?ua=1; last accessed August 20, 2018.
8. World Health Organization. Non-Communicable Disease Risk Factor Survey Bangladesh. (2010). Available at: http://www.who.int/chp/steps/2010_STEPS_Report_Bangladesh.pdf;last accessed November 20, 2018).
9. World Health Organization. Scaling Up Action Against Noncommunicable Diseases: How Much Will It Cost? (2012). Available at: http://www.who.int/nmh/publications/cost_of_inaction.pdf; last accessed November 26, 2018.
10. Haq S, Shazzad N, Ahmed S, Al-Qadir AZ, Shahin S. AB1157 prevalence of musculoskeletal disorders among garment industry workers in Bangladesh. Ann Rheum Dis (2017) 76:1460.
11. Global Burden of Disease 2015 Disease and Injury Incidence and Prevalence Collaborators. Global, regional, and national incidence, prevalence, and years living with disability for 310 diseases and injuries, 1990–2015: a systematic analysis for the Global Burden of Disease study 2015. Lancet 2016 388:1545–602.
12. Norrefalk JR, Linder J, Ekholm J, Borg K. A 6-year follow-up study of 122 patients attending a multiprofessional rehabilitation programme for persistent musculoskeletal-related pain. Int J Rehabil Res 2007 30(1):9–18.

13. Korthals-de Bos IBC, Hoving JL, van Tulder MW, et al. Cost effectiveness of physiotherapy, manual therapy, and general practitioner care for neck pain: economic evaluation alongside a randomised controlled trial. BMJ 2003 326(7395):911.

14. Scottish Intercollegiate Guideline Network. Management of Chronic Pain (SIGN 136). (2013). Available at: http://www.sign.ac.uk/assets/sign136.pdf; last accessed October 03, 2018).

15. World Health Organisation. The Global Status Report on Road Safety. (2015). Available from: http://www.who.int/violence_injury_prevention/road_safety_status/2015/en/; last accessed November 26, 2018.

16. Rahman A, Chowdhury SM, Mashreky SR, Linnan M, Rahman AKMF. Bangladesh Health and Injury Survey 2016: Summary Report. NonCommunicable Disease Control Progarmme, Directorate General of Health Services (DGHS), Ministry of Health and Family Welfare (MOHFW), Government of the People's Republic of Bangladesh (www.dghs.gov.bd), Centre for Injury Prevention and Research, Bangladesh (CIPRB; www.ciprb. org) (2016).

17. Islam MR, Rahman MS, Islam Z, Nurs CZB, Sultana P, Rahman MM. Inequalities in financial risk protection in Bangladesh: an assessment of universal health coverage. Int J Equity Health 2017 16:59.

18. Bultmann U, Sherson D, Olsen J, Lynsbeck-Hansen C, Lund T, Kilsgaard J. Coordinated and tailored work rehabilitation: a randomised controlled trial with economic evaluation undertaken with workers on sick leave due to musculoskeletal disorders. J Occup Rehabil 2009; 19:81–93.

19. Turner-Stokes L, Williams H, Bill A, Bassett P, Sephton K. Cost-efficiency of specialist inpatient rehabilitation for working-age adults with complex neurological disabilities: a multicentre cohort analysis of a national clinical set. BMJ 2016; 6(2):e010238. doi:10.1136/bmjopen-2015-010238

20. World Health Organization. Rehabilitation 2030: A Call for Action. (2017). Available at: http://www.who.int/disabilities/care/rehab-2030/en/; last accessed November 15, 2018.

21. United Nations General Assembly. Transforming Our World: The 2030 Agenda for Sustainable Development. A/RES/70/1. (2015). Available at: http://www.un.org/ga/search/view_doc.asp ? symbol= A/RES/70/1&Lang=E; last accessed October 04, 2018.

22. Health and Care Professions Council. Physiotherapists—Number of Registrants. (2017). Available at: http://www.hcpc-uk.org/aboutregistration/professions/index.asp?id=11; last accessed October 03, 2018.

23. Ahmed SM, Alam BB, Anwar I, Begum T, Huque R, Khan JAM, et al. Bangladesh Health System Review. Health Systems in Transition 2015;5:3.

24. El-Saharty S, Sparkes SP, Barroy H, Ahsan KZ, Ahmed SM. The Path to Universal Health Coverage in Bangladesh: Bridging the Gap of Human Resources for Health. A World Bank Study. Washington, DC: World Bank Group (2015).

Neuropsychiatric Comorbidity in Children with Epilepsy

Md Zakirul Islam[a], Md Abdul Mannan[b], Muhammad Ataul Haqq[c], AKM Harunur Rashid[d], Md Kawser Hossain[e]

Abstract

Background: Children with epilepsy often associated with various neuropsychiatric comorbidities. Many of the comorbidities have a significant impact on the medical management and quality of life of these patients. **Objective:** The aim of the study was to determine the neuropsychiatric problem in children with epilepsy. **Methods:** This was a cross-sectional study conducted at the Epilepsy Clinic of the Department of Paediatrics, Sir Salimullah Medical College Mitford hospital, Dhaka. Children aged 5 to 15 years with active epilepsy attending at the clinic constituted the study population. A total of 175 active epileptic children were enrolled in the study. Data were collected by pretested semi-structured questionnaire through a diagnostic interview of the study children to characterize the neuropsychiatric comorbidities. The data were analysed using standard statistical analysis software IBM® SPSS® Statistics Version 20. The p value <0.05 was considered statistically significant. **Results:** Majority (56%) of the study children had associated comorbidities. Of them, intellectual disability was most common 36.6% followed by communication disorder 17.7%, emotional disorder and learning disorder 13.3%, attention deficit and hyperactive disorder 10%, psychotic disorder 6.6% and Autism 3.3%. **Conclusion:** Children with epilepsy need proper assessment for associated comorbidities. Treatment of these coexisting disorders is very important to achieve the best seizure control and overall quality of life for the patients and their families.

Key word: epilepsy, children, comorbidity, psychiatric disorder

[a]Associate Professor, Department of Paediatrics, Cumilla Medical College, Cumilla, Bangladesh.

[b]Assistant Professor, Department of Paediatrics, Sir Salimullah Medical College, Dhaka, Bangladesh.

[c]Associate Professor, Department of Paediatrics, OSD DGHS attached to Department of Paediatric Cardiology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh.

[d]Assistant Professor, Department of Paediatrics, Cumilla Medical College, Cumilla, Bangladesh.

[e]Assistant Professor, Department of Paediatrics, Cumilla Medical College, Cumilla, Bangladesh

Correspondence to : MZ Islam dr.zakir90@yahoo.com

Cite this as: BMCJ 2019; 5(1):5-10

Received : 5 November 2018
Accepted : 11 December 2018

Introduction

Epilepsy is the most common childhood neurologic disorder affecting 0.5 to 1.0% of children younger than 16 years[1]. Epilepsy in children is associated with variable comorbidities although frequency of such comorbidities is often difficult to determine. Some studies have shown that psychiatric disorders can emerge in children early in the course of the illness[2] even prior to the onset of seizure[3]. The common neurological comorbidities are: cognitive impairment, language problem and the common psychological comorbidities are autism spectrum disorders, attention deficit hyperactive disorder, mood disorders (anxiety and depression), psychosocial and rarely psychosis, conduct disorder etc.

Compare to general population, incidence of neurobehavioral disorders is higher in patient with epilepsy[4,5]. In children with epilepsy, various studies have reported a prevalence of mental health problems ranging from 16% to 77%, and a 3-fold to 9-fold risk compared with control[6]. In a recent study of children and adolescents aged 10 to 19 years who had epilepsy, the most common observed comorbid psychiatric disorder were attention deficit hyperactive disorder (ADHD) and anxiety, and 26.8% of those with a psychiatric

diagnosis demonstrate executive dysfunctions[7]. Other researcher has also revealed higher rate of depression and autism spectrum disorder in this population[8].

Management strategies of children with epilepsy should focus not only on the controlling seizures but also on early diagnosis and treatment of comorbid conditions. Often, in many circumstances the comorbid condition is often overlooked in children with epilepsy. As a result, there will be incomplete treatment that may lead to increase morbidity of the children with epilepsy. Identification of such morbidities should be an integral part of management of childhood epilepsy. In country, very few studies are done to find out the associated comorbidities in children with epilepsy. So, the study was designed to explore the common comorbid condition in children with epilepsy and thus to take necessary measures to overcome the problem.

Methods

The study was conducted at the Epilepsy Clinic of the Paediatrics Department, Sir Salimullah Medical College Mitford Hospital, Dhaka from July 2015 to June 2017. It was a cross sectional study involving children aged 5 to 15 years with active epilepsy. With the informed consent a total of 175 active epileptic children were enrolled in the study. Data were collected by detailed interview and physical examination of the children, interview of the mothers or attending attendants, from treatment records and interview of the concerned attending doctors if necessary using a semi structured questionnaire and data collection sheet. EEG was done for every patient and diagnosis of epilepsy was done by the pediatric neurologist by using International League Against Epilepsy Classification. Psychiatric assessment was done by using structured clinical interview using diagnostic and statistical manual/ non patient version (SCID-1/NP)[9] and diagnosis was confirmed according to DSM-IV[10] by a consultant psychiatrist. The data were coded, entered and analysed using standard statistical analysis software IBM® SPSS® Statistics Version 20. Descriptive analytical techniques involving frequency distribution, computation of percentage e etc were done. Chi-square test was applied to verify an association of sociodemographic and clinical characteristics with neuropsychiatric comorbidity status of the children with epilepsy.

Results

A total of 175 children with epilepsy, 136 (77.7%) children were aged between 5 to 10 years, and 125 (71.4%) were male. Majority (53.7%) of them belonged to lower socio economic status strata followed by middle strata (34.2%) and then upper strata (12.0%) (Table 1). Highest percentage (44.1%) of the children were in primary school, 33.1% did not attended at any school and the rest 22.8% of the children were in secondary school (Table 1).

Out of 175 study children, majority (56.0%) had associated neuropsychiatric comorbidities. Partial seizure was the most common seizure in 67.4% cases followed by absence seizure in 28.5% cases. In case of 153(87.4%) children, seizures were in controlled. Most (94.9%) of the cases had a family history of epilepsy. More than 42.0% of the children had a history of birth or developmental delay (Table 2)

Table 1: Sociodemographic characteristics of the children with epilepsy (n=175)

Characteristics	Number (N)	Percentage (%)
Age		
5-10 years	136	77.7
10-15 years	39	22.3
Sex		
Male	125	71.4
Female	50	28.6
Socio economic status		
Lower	94	53.7
Middle	60	34.2
Upper	21	12.0
Education		
Not attended class yet	58	33.1
Primary	77	44.0
Secondary	40	22.8

Table 2: Clinical characteristics of the children with epilepsy (n=175)

Characteristics	Number (N)	Percentage (%)
Child with comorbidity		
Present	98	56.0
Absent	77	44.0
Family history of epilepsy		
Yes	9	5.1
No	166	94.9
Birth or developmental delay		
Present	75	42.9
Absent	100	57.1
Seizure Type		
GTCS	07	04.0
Absence	50	28.5
Partial seizure	118	67.4
Seizure Status		
Active	22	12.6
Controlled	153	87.4

Table 3: Association of Sociodemographic and clinical characteristics of the children with psychiatric disorder. n= 175

Characteristics	Psychiatric disorder		P value
	Present (n=98) N(%)	Absent (n=77) N(%)	
Age			
5 to 10 years (n=136)	75(76.5)	61 (79.2)	0.671
10 to 15 years (n=39)	23 (23.5)	16 (20.8)	
Sex			
Male (n=125)	67 (68.4)	58 (75.3)	0.311
Female (n=50)	31 (31.6)	19 (24.7)	
Education			
Not attended class yet (n=58)	44 (44.9)	14 (18.2)	
Primary (n=77)	36 (36.7)	41 (53.2)	0.0009
Secondary (n=40)	18 (18.4)	22 (28.6)	
Socioeconomic status			
Lower (n=94)	51 (52.0)	43 (55.8)	
Middle (n=60)	35 (35.7)	25 (32.5)	0.878
Upper (n=21)	12(12.3)	09 (11.7)	
Family history of epilepsy			
Yes (n=9)	06 (6.1)	03 (3.9)	0.508
No (n=166)	92 (93.9)	74 (96.1)	
Birth or developmental delay			
Present (n=75)	54 (55.1)	21 (27.3)	0.0002
Absent (n=100)	44 (44.9)	56 (72.7)	
Seizure Type			
Generalized tonic clonic seizure (GTCS) (n=7)	05 (5.1)	02 (2.6)	
Absence (n=50)	29 (29.6)	21 (27.3)	0.635
Partial seizure (n=118)	64 (65.3)	54 (70.1)	
Seizure Status			
Active (n=22)	14 (14.3)	08 (10.4)	0.440
Controlled (n=153)	84 (85.7)	69 (89.6)	

More than three forth (75.9%) of the children, who did not attended at any school, had neuropsychiatric comorbidities. It was 46.8% and 45.0% in primary and secondary school attending children. The differences of comorbidities among the different educational groups was statistically significant (p=0.0009). The proportion of neuropsychiatric comorbidities in the children having a history of birth or developmental delay was significantly (p=0.0002) higher than those, who have not. Age, gender, economic status, family history of epilepsy, seizure type and seizure status were not significantly associated with neuropsychiatric comorbidities of the children with epilepsy (Table 3).

Table 4: Distribution of Neuropsychiatric disorders (n=98)

Disorders	Number N(%)	Percent N(%)
Intellectual deficit	35	35.7%
Communication disorder	18	18.4%
Learning disorder	14	14.2%
Emotional disorder	12	12.2%
ADHD	10	10.2%
Psychotic disorder	6	6.1%
Autism	3	3.0%

The most common neuropsychiatric disorder in the children with epilepsy was intellectual deficit (35.7%) followed by communication disorder (18.4%). The other comorbidities were found as learning disorder 14.2%, emotional disorder 12.2%, and attention deficit hyperactive disorder 10.2% (Table 4).

Discussion: Comorbidity not only adds up burden to illness but also increase uncontrolled epilepsy duration, leading to increased morbidity and disability affected life years. In this study, neuropsychiatric comorbidity was found 56% of the children with epilepsy. It is higher in comparison to others study done by Dharmadhikari et al.[11] and Davier et al.[12], which was 32.2% and 37% of children, respectively. But a recent meta-analysis of 46 studies in 2434 children with epilepsy measuring psychiatric comorbidity using CBCL (ref) has found comorbidity in range of 16% to 77%[13].

Comorbidity of psychiatric disorder in epilepsy was not uniform, but it varied with different types of epilepsy. In our study, most common seizure type in epilepsy associated with psychiatric comorbidity was partial seizure (65.3%). Generalized tonic clonic seizure (GTCS) was the most uncommon type of epilepsy to be associated with comorbidity in children with epilepsy. It is consistent with the other study findings[11,14,5]. In our study, there was high chance of developing comorbidity in children with no education and also in children with significant birth and development history(P <0.001). Dharmadhikari et al.[11] also found Similar findings in their study.

Intellectual disability, as comorbidity, was higher (35.7%) in children with epilepsy in our study but in Dharmadhikari et al.[11] it was 21.2%. Some other studies demonstrated significantly higher rate of intellectual comorbidity in children with epilepsy which was similar to our study[12, 16].

Social communications that involve the use of language in formulating and organizing thought. Epilepsy may cause temporary loss of function in one or more parts of brain. If these parts are involved with

comprehension, organization and speech process, communication difficulties results. Parkinson et al. found a high incidence of language disorder in children with focal epilepsy[17]. In our study, communication disorder was found to be 18.4% of children with epilepsy. It may be noted that in our study not only focal epilepsy but all types of epilepsy were included.

In Learning disorder there is interference of academic performance or daily activities that require reading, writing or mathematical skills in subjects with a normal intelligence quotient (IQ). Learning disorder is more common in children with epilepsy than normal population[18]. In our study we found learning disorder to be 14.2% of the children with epilepsy.
Depression in epilepsy is common finding in children with epilepsy, especially in partial seizure of temporal lobe origin. In our study, emotional disorder was found 12.2% in children with epilepsy which is consistence with other studies done in previously[11,19,20].

In many studies ADHD was found in 14% -38% of children with epilepsy [15, 2122,23] , but in our study , it was little bit lower than the lower limit of the rang. It may be due to strictly follow the criteria as per DSM-IV for the diagnosis of ADHD.

Psychosis as a comorbidity in children with epilepsy was found 6.6% in children in our study. It is very similar to the study done by Dharmadhikari et al.[11] where psychosis was found in 6.69% of children with epilepsy.

Management strategies should be focused not only on the controlling seizures but also on early diagnosis and treatment of comorbid conditions. Screening of neuropsychiatric comorbidities must be an integral part of the management of children with epilepsy. This is a small scale hospital based study. So it did not reflect the overall situation in the country. Further large study is needed to understand the overall situation in the country.

Reference

1. S.Sinnar, J.M.Pellock. Update on the epidemiology and prognosis of pediatric epilepsy. J Cild Neorol 2002; 17: 4-17.
2. Hoare P. Psychiatric disturbance in the families of epileptic children. Dev Med Child Neurol 1984; 26(1): 4-9.
3. Austin JK, Harezlak J, Dunn DW, Huster GA, Rose DF, Abrosius WT. Behavior Problems in children before first recognize seizures. Pediatrics 2001; 107: 115-22.
4. Hernandez-Frau PE. Psychiatric disorders associated with epilepsy. Available online at http://emedicine.medscape.com/article/1186336-overview; Last accessed November 18, 2018.
5. Dunn DW. Neuropsychiatric aspect of epilepsy in children. Epilepsy and behavior 2003; 4: 101-6.
6. Pliopys S, Duun DW, Cap;an R. 10 years research update review: Psychiatric Problems in children with epilepsy. J Am Acad Child Adolesc Psychiatry 2007; 46(11): 1389-1402
7. Alfstad KA, Torgersen H, Van Roy B, et al. Psychiatric comorbidity in children and youth with epilepsy: an association with executive dysfunction? Epilepsy behave 2016; 56: 88-94.

8. Bolton PF, Carcani-Rathwell I, Hutton J, Goode S, Howlin P, Rutter M. Epilepsy in autism: features and correlates. Br J Psychiatry 2011; 4: 289-94.
9. First MB, Spitzer RL, gibbon M, Williams JBW. (2002) Structured Clinical interview for DSM-IV-TR axis I disorders, research version, non-patient edition (SCID-I/NP) New York State Psychiatric Institute. [Google Scholar]
10. World Health Organization. The ICD-10 Classification of Mental and Behavioral Disorders: Clinical Description and Diagnostic Guidelines. Geneva: World Health Organization; 1992 [Google Scholar].
11. Dharmadhikari AS, Sinha VK. Psychiatric Comorbidity in Children with epilepsy: A cross sectional 5 years rural prevalence study. J Neurosci Rural Pract. 2017; 8 (2): 179-84.
12. Davies S, Heyman I, Goodman RA. Population survey of mental health problem in children with epilepsy. Dev Med Child Neurol 2003; 45: 292-5.
13. Rodenburg R, Meijer AM, Dekovic M, Alden Kamp AP. Psychopathy in children with epilepsy: A meta-analysis Epilepsia 2006; 47: 601-14.
14. Caplan R, Arbelle S, Magharious W, et al. Psychopathology in Pediatric Complex partial seizure and generalized epilepsy. Dev Med Child Neurol 1988; 40: 805-11.
15. Thome-Souza S, Kuczynski E, Assumpcao F, et al. which factors may play a pivotal role on determining the type of psychiatric disorder in children and adolescent with epilepsy? Epilepsy Behav 2004; 5: 988-94.
16. Stefenburg S, Gillberg C, Stiffenburg U. Psychiatric disorder in children and adolescents with mental retardation and active epilepsy. Arch Neurol 1996; 53: 904-12.
17. Parkinson G. High incidence of language disorder in children with focal epilepsy. Developmental medicine and child neurology 2002; 44 (8): 533-7.
18. Beghi M, Cornaggia CM, Frigeni B, Beghi E. Learning disorder in epilepsy. Epilepsia 2006; 47 suppl 12: 14-8.
19. Ettinger AB, Weisbrot DM, Nolan EE, et al. Symptom of depression and anxiety in pediatric epilepsy patient. Epilepsia 1998; 39: 595-9.
20. Caplan R, Siddarth P, Gurbani S, Hanson R, Sankar R, Shields WD. Depression and anxiety disorder in pediatric epilepsy. Epilepsia 2005; 46: 720-30.
21. Dunn DW, Austin JK, Haneczlak J, Ambrosius WT. ADHD and epilepsy in childhood. Dev Med Child Neurol 2003; 45: 50-4.
22. Semrud-Clikeman A, Wical B. Components of attention in children with complex partial seizure with and without ADHD. Epilepsia 2003; 44: 591-7.
23. Ott D, Siddartha P, Gurbani S, et al. Behavioral disorders in pediatrics epilepsy; Unmet psychiatric need. Epilepsia 2003; 44: 591-7.

ULTRASONOGRAPHIC STUDY OF SPLENIC LENGTH AMONG ADULT MALE BANGLADESHI PEOPLE

Md. Atiqur Rahman[a], Akhtari Afroze[b], Shahin Sharmin[c], Ashrafi Akter Zahan[d], Tarifat Alam[e], Md. Golam Maula[f]

Abstract

Background: Scanning of the viscera is carried out to know the normal dimensions, echo patterns and deviations from normal, leading to diagnosis or prediction of pathological conditions. **Objective:** The purpose of the study was to establish guidelines for normal splenic length at different ages among adult male by using a simple and reproducible ultrasonographic method. **Methods:** The study was performed on spleen during the period of July 2015 to June 2016 in the Department of Anatomy, Rajshahi Medical College, Rajshahi. Ultrasonographic measurement of the spleen was done on adult males in Radiology and Imaging Department of Rajshahi Medical College Hospital and other Medical Colleges in Rajshahi city with legal permissions from the authority. Seventy-five adult males, from 18 years to 60 years old, were selected for ultrasonography because of abdominal and/or pelvic problems unrelated to the spleen. The splenic length was measured by obtaining a coronal view that included the hilum during deep inspiration to minimize masking by lung. The greatest longitudinal distance between the most superomedial and the most inferolateral points through the hilum (splenic length) during deep inspiration was measured. **Results:** The mean ultrasonographic length of the spleen in 4 age groups 18-30 years, 31-40 years, 41-50 years and 51-60 years were 10.76±0.96 cm, 10.31±0.72 cm, 9.88±0.42 cm and 8.96±0.37 cm respectively. The overall mean ultrasonographic length of the spleen in adult male was 9.98±0.61 cm. **Conclusion:** The present study findings sugesst splenic length in adult males inversely correlates with age, In most of the adult males, it is <11 cm.

Key words: Ultrasonography, splenic length, adult spleen.

[a]Assistant Professor, Department of Anatomy, Ad-Din Akij Medical College, Khulna, Bangladesh.

[b]Professor, Department of Anatomy, Barind Medical College, Rajshahi, Bangladesh. .

[c] Assistant Professor, Department of Anatomy, Rajshahi Medical College, Rajshahi, Bangladesh. .

[d]Assistant Professor, Department of Anatomy, Gonoshasthaya Samaj Vittik Medical College, Dhaka, Bangladesh.

[e]Assistant Professor, Department of Pharmacology, Ad-Din Akij Medical College, Khulna, Bangladesh.

[f]Associate Professor, Department of Physiology, Barind Medical College, Rajshahi, Bangladesh.

Correspondence to : M A Rahman @yahoo.com

Cite this as: BMCJ 2 019;5(1):11-16

Received : 27 April 2018
Accepted : 18 August 2018

Introduction

The human spleen is an organ demanding constant attention from the anatomical, immunological and clinical point of view.[1] It is a large encapsulated mass of vascular and lymphoid tissue situated in the upper left posterior region of the abdominal cavity between the fundus of the stomach and the diaphragm.[2]

Ultrasonographically, the spleen is crescent in shape with smooth outer convexity. The inner margin may be indented or nodulous. The echogenicity is homogenous which is slightly more echogenic than healthy liver tissue and markedly hyperechoic compared to kidney tissue.[3]

The spleen is affected by several groups of diseases. Gross splenomegaly can be detected both clinically and ultrasonographically.[4] But the clinical examination is far from accurate to detect a small increase in spleen size. It must be two to three times enlarged before it is palpable.[5] The precise measurement of spleen by palpation is not reliable, as in cases a normal sized spleen is palpable and a non-palpable spleen may not be of normal sized.[6] So, the scanning of the viscera is carried out to know the normal dimensions, echo patterns and deviations from normal,

leading to diagnosis or prediction of pathological conditions.[7] The variation in the anthropometric features of various population, races and regions were an established fact.[8] So, the standard normal range of spleen size is prerequisite for correct interpretation. In the literature, normal spleen sizes in different age groups among adult male have been reported.

Splenomegaly is an important diagnostic clue to the existence of an underlying disorder. The spleen is affected by number of disorders e.g. Malaria, Kala-azar, Chronic myeloid leukaemia, Myelofibrosis, Lymphoma, Hereditary hemolytic anaemia, CLD with portal hypertension, Enteric fever, Subacute bacterial endocarditis (SBE), Viral infections, Collagen diseases (SLE).[9-11] So, the estimation of the splenic size in vivo is often important in the diagnosis, treatment and prognosis of these disorders. Surgical procedures such as splenic surgery, splenic repairing and surgery of other organs associated with spleen require knowledge about the spleen.[12] Racial differences in splenic length could result in incorrect interpretation of the spleen size. So, population specific normogram of spleen would provide more accurate standards. Visceral organs change with age and disease processes. So, it is utmost important to know the normal range of spleen length in adult male of Bangladesh to assess splenomegaly in disease conditions affecting the spleen. These measurements need to be correlated with age and sex.

Length of the spleen can be determined by several methods such as Simple palpation method, ultrasonography and radiography. Ultrasonography has been found to be both accurate and reliable for the calculation of splenic length with the advantages of lack of ionizing radiation, low cost, easier, safe, quick, portability of instrument, non-invasive, lack of risk of allergic reactions as compared to other diagnostic tools, such as simple x-ray, Radionuclide imaging, Angiography, Sulfur colloid and Scintigraphy, CT and MRI.[13-15]

Though ultrasonography is used routinely to evaluate visceral measurements in adults in Bangladesh, very few studies were found to measure spleen length by ultrasonography to co-relate with age distribution. So, the objectives of the present study were to measure the spleen length by ultrasonography, to find out the reference values of splenic length in different age groups and correlations with the age among adult male Bangladeshi people.

Methods

This was a cross-sectional study conducted in the Department of Anatomy in Rajshahi Medical College, Rajshahi from July 2015 to June 2016. A total 75 healthy males attending in the Radiology and Imaging Department of Rajshahi Medical College Hospital and other Medical Colleges in Rajshahi city were selected for this study. The inclusion criteria were: the individuals willing to participate; individuals suffering from diseases which did not affect the spleen directly or indirectly; individuals with demonstrated normal homogenous echopattern of the spleen without evidence of any focal splenic abnormality. The exclusion criteria were: the individuals not willing to participate; history of medical disorders affecting spleen, liver; history of splenic, hepatic or upper abdominal surgery; history of oncologic, haematologic or abdominal traumatic condition. The study variables were age, sex (male) and splenic length (in centemeters). The individuals were classified into following

age groups: 18-30 years, 31-40 years, 41-50 years and 51-60 years. Ethical Review Committee of Rajshahi Medical College, Rajshahi approved this study. After getting permission from the concerned authority, the informed consent from the respective individual was taken after fully explaining the purpose of the research work in their own language. Full confidentiality of the individual was maintained. The data was collected from in patients as well as out patients fulfilling the inclusion criteria and attending the Department of Radiology and Imaging. An elaborate history was taken for each individual regarding present & previous history of illness suggesting the splenic diseases and any abdominal surgery. Individuals were physically examined. During ultrasonography, the patient was placed on the bed in a supine position. The individual was made to take a deep breath and hold it for a few seconds to evaluate the spleen and its echogenicity and related structures in general to exclude any diseased conditions. Then the sonographic image of the spleen was taken and measured the length along the longitudinal coronal axis between the most superomedial and the most inferolateral points through the hilum. All the measurements and images were saved in the ultrasound machine. Statistical analysis was performed in computer with SPSS version 16.

Table-1: Grouping of the samples in relation to age of the respondents.

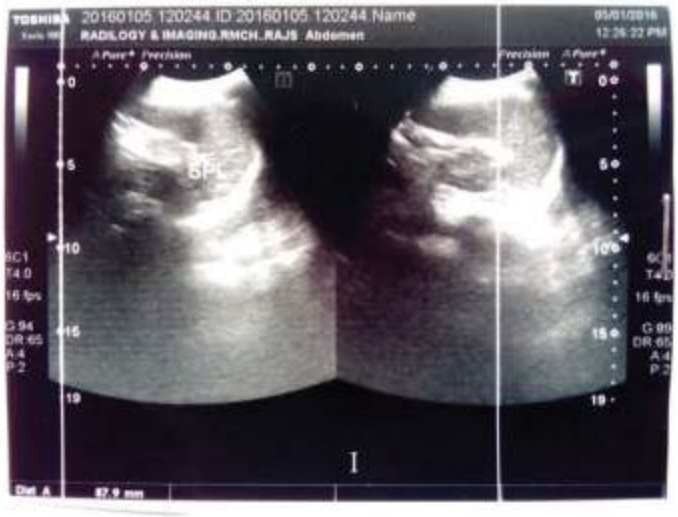
Age groups (in years)	Number of samples n (%)
18-30	22(29.4)
31-40	20(26.7)
41-50	18(24.0)
51-60	15(20.0)
Total	75 (100.0)

Results

Table-I Shows that total number of the sample was 75. Out of which, 22 (29.4%) were in 18-30 years, 20 (26.7%) were in 31-40 years, 18 (24.0%) were in 41-50 years and 15 (20.0%) we re in 51-60 years age group.

Table 2: Minimum and Maximum splenic length in different age groups.

Age groups (in years)	Length of spleen (cm)	
	Minimum	Maximum
18-30	8.9	11.9
31-40	9.1	11.5
41-50	9.3	10.6
51-60	8.5	9.8



The minimum and maximum length of the spleen in adult male varied from 8.53 cm to 11.98 cm. Most of the cases (82.67%), spleen length were found within the range of 8 cm to below 11 cm except in thirteen (17.33%) cases, the length were found above 11 cm. These thirteen cases were distributed among 18-30 years and 31-40 years age groups. It was observed that

with increased age the spleen length was decreased.

Table 3: The means and standard deviations of age and spleen length in all age groups.

Age groups (in years)	Age (years)	Length of spleen (cm)
18-30	23.54±3.78	10.76±0.96
31-40	34.95±3.15	10.31±0.72
41-50	45.33±3.16	9.88±0.42
51-60	55.00±3.38	8.96±0.37
Overall	39.71±3.37	9.98±0.63

From the above table, it was observed that spleen length was decreased in older age in males. Spleen length was the highest in age group 18-30 and then decreased with increase in age. The overall mean length of the spleen in adult male was 9.98±0.63 cm.

Table 4: Correlation of spleen length and age.

Variables	Length of spleen (cm)	
Age (years)	Pearson correlation	p value
	-.786	0.000

Correlation is significant at $p < 0.05$ level (2-tailed).

Correlation analysis showed that, spleen length was negatively correlated with the age in males. So, with increasing age spleen length was found to be decreasing which was significant ($p<0.05$).

Table 5: Comparison of spleen length among different age groups.

Ratio	F	Significant Value
Between the groups	19.525	.000
Within the groups		

Correlation is significant at $p < 0.05$ level.

Ultrasonographic length of the spleen was statistically significant ($p<0.05$) when compared between groups and within the groups (by ANOVA, Tukey HSD test).

Discussion

Diagnostic imaging techniques were superior to clinical examination in determining the sizes of the organs.[16,17] Ultraonography was one of the most common imaging methods, which was used in routine practice.[9]

The splenic length may give information about the diagnosis and progress of the gastrointestinal and hematological diseases.

Kaneko *et al.* (2002)[18] evaluated the splenic size in patients with sarcoidosis and thrombocytosis, the splenomegaly was present in 57% of the patients (using ultrasonography) but only clinically palpable in 8% of the cases. Therefore, the imaging has become essential for the accurate measurement of the splenic size.

The length of the spleen varies with age, with the individual and in the same individual under different conditions. In adult it is usually about 12 cm and diminished size is observed in older people.[2] The normal spleen size on ultrasonography in adults is about 10 to 12 cm in length and a spleen size of about 12 cm is considered as normal.[3,19] Thus splenic length more than 12 cm is considered as enlarged spleen.[3]

In the present study, the splenic length was decreased at a slow rate up to the age of 50 years and then rapid fall after the age of 50 years. The above findings were consistent with the findings of Loftus *et al.* (1998)[19], Arora *et al.* (2013)[5], Chakraboti *et al.* (2016)[20] and Yahuza *et al.* (2016)[21].

In this study, overall ultrasonographic length of the spleen in adult male was 9.98±0.63 cm (Table-3). According to

Serter *et al.* (2010)[22] and Ashgar *et al.* (2011)[23] the mean length of the spleen were 10.76±1.84 cm and 10.67±1.62 cm respectively. Serter *et al.* (2010)[23] performed his sonographic study of spleen on male military personnel in Turkey. Ashgar *et al.* (2011)[23] measured the length of spleen by CT scan on North Indian adult population. The variations of these results may be due to the variations in persons height, weight, body surface area, genetic factors, nutritional factors, environmental factors and the method of measurement.

In this study, it was observed that the ultrasonographic splenic length was less than 11 cm in most of the subjects which was similar to the findings of Serter *et al.* (2010)[22], Arora *et al.* (2013)[5] and Chakraboti *et al.* (2016).[21]

Conclusion

Spleen is frequently involved in various systemic and local diseases. Clinically spleen is an important organ as it becomes enlarged and reduced in many diseases. The present study was an attempt to determine the normal range of the spleen length which correlated variably with different age groups in adult males of Bangladesh. To get a comprehensive and conclusive data, we need to accommodate much larger sample size covering the population of the cross section of the state.

References

1. Poulin E, Thibault C. The anatomical basis for laparoscopic Splenectomy. Can J Surg 1993; 36(5): 484-8.
2. Standring S. ed. Gray's Anatomy: The anatomical basis of clinical practice. 40th edition. Edinburgh: Elsevier Churchill Livingstone 2008; 1191-5.
3. Benter T, Klühs L, Teichgräber U. Sonography of the spleen. J Ultrasound Med 2011; 30: 1281-93.
4. Al-Imam O, Suleiman A, Khuleifat S. Ultrasound assessment of normal splenic length and spleen-to-kidney ratio in children. Eastern Mediterranean Health Journal 2000; 6(2/3): 514-6.
5. Arora N, Sharma PK, Sahai A, Singh R. Sonographic measurement of the spleen: splenic length in adults and its correlation with different parameters. Journal of the Anatomical Society of India 2013; 62: 57-61.
6. Schindler G, Longin F, Helmschrott M. The individual limit of normal spleen size in routine x-ray film. Radiology 1976; 16(4): 166-71.
7. Marco P, Vincenzo M, Rosanna C, Ernsto S, Roberto M, Antonio S, et al. Measurement of spleen volume by ultrasound scanning in patients with thrombocytosis: a prospective study. Blood J 2002; 99(11): 4228-30.
8. Krestin GP, Brennan RP. Ultrasound diagnosis of the abdomen. Ther Umsch 1992; 49(6): 395-404.
9. Odorico ID, Spaulding KA, Pretorius DH, Lev-Toaff AS, Bailey TB, Nelson TR. Normal splenic volumes estimated using three-dimensional ultrasonography. J Ultrasound Med 1999; 18: 231–6.
10. Kumar V, Abbas AK, Fausto N, Aster JC, Eds. Robbins and Cotran pathologic basis of disease. 8th edition. New Delhi Saunders Elsevier 2010; 632-5.
11. Sagiroglu A, Acer N, Ertekin T, Kurtoglu E, Coskun A, Yildirim A, Zararsiz G. Estimation of spleen volume and surface area of the newborns' cadaveric spleen using stereological methods. Folia Morphol 2013; 73(2): 183-92.

12. Garden OJ. The spleen. In: NS, Williams CJK. Bulstrde PR. O'Connell eds. Bailey and Love's short practice of surgery. 25th edition. London: Hodder Arnold 2008; 1101-10.
13. Singh A, Ansari H, Das JK, Chandra N. Ultrasonographic Measurement Of Splenic Length In Relation With Height In Bihari Adult Population A Prospective Study. J Anat Soc India 2011; 60(2): 188-9.
14. Petzoldt R, Lutz H, Ehler R, Neidhardt B. Determination of spleen size by ultrasonic scanning. Med kiln 1976; 71(48): 2113-6.
15. Frank H, Deland. Normal spleen size. Radiology 1970; 97(3): 589-92.
16. Sapira JD, Wiliamson DL. How big is the normal liver? Arch Intern Med 1979; 139: 971-3.
17. Safak AA, Simsek E, Ahcebasi T. Sonographic assessment of the normal limits and percentile curves of liver, spleen, and kidney dimensions in healthy school-aged children. J Ultras. Med 2005; 24: 1359-64.
18. Kancko J, Sugawara Y, Matsui Y. Normal splenic volume in adults by computed tomography. Hepatogastroenterology 2002; 49: 1726-7.
19. Loftus WK, Chow LT, Metreweli C. Sonographic measurement of splenic length: correlation with measurement at autopsy. J Clin Ultrasound 1999; 27: 71-4.
20. Chakraboti S, Saha N, Debbarma B, Das S, Leishram D. Normal Spleen Length by Ultrasonography in Adults of Tripura. IOSR-Journal of Dental and Medical Sciences 2016; 15(1): 55-60.
21. Yahuza MA, Tabari AM, Isyaku K, et al. Sonographic measurement of spleen dimensions in healthy adults in North-Western Nigeria. Nigerian Journal of Basic and Clinical Sciences 2016; 13(1): 30-5.
22. Serter S, Ceylan C, Tuncyurek O, Orguc S, Pabuccu Y. Sonographic evaluation of spleen size and prevalence of accessory spleen in a healthy male Turkish population. Turk J Hematol 2010; 27: 25-8.
23. Asghar A, Naaz S, Agrawal D, Sharma PK. orphometric Study of Spleen in North Indian Adult Population: CT Scan Images Based Study. Journal of Clinical and Diagnostic Research 2011; 5(5): 974-7.

Number of parietal cell in the gastric gland of gastric mucosa (body) and its relationship with different age group

Md. Muazzem Hossain[a], Humaira Naushaba[b], Nasrin jahan shammi[c], Uttam kumar Paul[d]

Abstract

Background: The stomach is the most dilated part of the alimentary tract. It is concerned with a number of diseases like gastritis, peptic ulcer along with its complications like perforation, haemorrhage, gastric outlet obstruction and cancer. In many of this disorder, the treatment procedures include surgical interventions of stomach. A massive knowledge of the perfect histomorphology of stomach carries great importance in the medical and surgical treatment of various stomach-related diseases. The most conspicuous cells of the gastric mucosa are the parietal cells. These are the source of hydrochloric acid and intrinsic factor of gastric juice. **Objective:** To find out the number of parietal cell mass of the gastric gland in the gastric mucosa of the stomach and its association with age. **Methods:** This descriptive type of study was conducted in the department of Anatomy, Sir Salimullah Medical College (SSMC), Dhaka from July 2005 to June 2006. Sixty (60) human postmortem stomach were included in this study, age ranging from 0 to70 years. The samples were collected from apparently fresh unclaimed dead bodies within 24 to36 hours of death before showing any signs of decomposition from the morgue of Sir Salimullah Medical College (SSMC) and Dhaka Medical College (DMC), Dhaka.The collected samples were divided into three (3) different age groups: A= 0-14years, B=15-22 years and C=23 years and above . Unpaired "t" test was applied to test the differences of mean parietal cell among the different age groups **Results:** The mean (±SD) number of parietal cell in group A (0-14 years), B (15-22 years) and C (23 years and above) were 1355.76±52.84, 1420.29±48.32 and 1426.08±114.48 cells/mm^2 respectively. The mean numbers of parietal cell increased with age but the differences were not significant (P>0.05, >0.10 and>0.50). **Conclusion**: The number of parietal cells showed significant positive correlation with age but had no significant difference among` different age groups.

Key word: Parietal cell, stomach, gastric gland.

[a]Professor, Department of Anatomy, Kumudini Women's Medical Medical College, Mirzapur, Tangail, Banglsadesh.

[b]Professor, Department of Anatomy, Dhaka Medical College, Dhaka, Banglsadesh.

[c]Associate Professor, Department of Pharmacology and Therapeutics, Barind Medical College, Rajshahi, Bangladesh.

[d]Professor, Department of Anatomy, Sir Salimullah Medical College, Dhaka, Banglsadesh.

Correspondence to : MM Hossain

Cite this as: BMCJ 2 019;5(1):17-22
Received : 12 November 2018
Accepted : 18 December 2018

Introduction

The stomach (ventriculus or referably gaster) is the most dilated part of the alimentary tract that is continuous with the oesophagus proximally and the duodenum distally. Anatomically, the stomach is divided into cardia, fundus, body or corpus, and pylorus .[1]

The fundus and body have identical histological structure, so the stomach has only three histologically distinct regions.[2] The stomach consists of four layers. The inner mucosa contains different types of glands. Gastric glands comprise cardiac , body fundus (fundic glands) and pyloric glands.[1]

The parietal cell, one of the important cells of the gastric glands, secrete hydrochloric acid and intrinsic factor which is necessary for the reabsorption of vitamin B_{12}. Parietal

cells are scattered along the gastric gland and occupy much space due to their large size. In gastric mucosal tissue sections, the parietal cell comprises 12% of all epithelial cells in humans. [3]

The normal stomach contains approximately one billion parietal cells.[4]The quantity of acid produced by the stomach is related to the parietal cell mass, and peptic ulcer patients generally have a larger parietal cell mass than normal persons.[5] The number of parietal cells tend to increase with age making the elderly people more susceptible to gastric mucosa damage.[6] In human subjects, a significant decrease in parietal cell mass index in males over 60 years has been recorded.[7]A decrease in the large acid secretory areas have been found over the age of 60 years .[8]

Material and Method

The present study was performed on sixty (60) postmortem stomachs of Bangladeshi people of different age groups. The viscera were collected from apparently fresh unclaimed dead bodies autopsied in the morgue of the department of Forensic Medicine, Sir Salimullah Medical College (SSMC) and Dhaka Medical College (DMC), Dhaka after requisite legal formalities. The samples were collected within 24 to 36 hours of death before showing any signs of decomposition. Each specimen was tagged properly with an identification number. The age, sex and cause of death were noted down in a separate register book from the morgue's record book.

After bringing to the dissection room, the samples were washed thoroughly with running tap water. Fat and other unwanted tissue were removed from the stomach. Then the samples were kept in 10% formol saline solution for preservation and fixation. The collected samples were divided into 3 groups. Group A (0-14years), group B (15-22years) and group C (23 years and above).

Table 1: Age distribution of different study groups

Group	Age in years	Number of sample	Percentage %
A	0-14	7	11.7
B	15-22	17	28.3
C	23 and above	36	60.0

Procedure of histological study:

a. Preparation of histological slide: Eighteen relatively fresh samples (6 stomachs each from three age groups) were selected to determine the number of parietal cells/sqmm area of stomach mucosa. For histological study, tissues measuring approximately 1cmx1cmxwhole thickness were taken from anterior wall of the stomach.

By standard histological procedure, the tissue blocks were made and 6μm thick paraffin sections were prepared. The better sections were then taken on albuminized slides and dried in air. Six slides from the body of the stomach from each study group were prepared for the study. Thus, a total of 6x3=18 histological slides were made. Staining of the histological section was done by Hematoxylin and Eosin.

Parameter studied

The number of parietal cell/ sqmm area of stomach mucosa.

b. Procedure of measurement of the number of parietal cell:

The tissue sections on the slides were

divided into three equal parts by drawing transverse lines with fine marker pens on the cover slips at right angle to the long axis of the section.[9] From each division, one microscopic field from apparently maximal mucosal folding area was chosen keeping equal distance between the inner and outer border of the mucosa. Because of the small size of the parietal cells, the slide were viewed in high magnification (X 40 objectives). Thus, 18 fields were examined from 6 slides of body region from each study group. The counting was done within a counting circle specially devised to count the parietal cells.

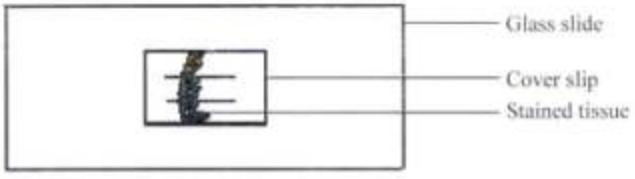


Figure 1: Three equal divisions of the tissue section.

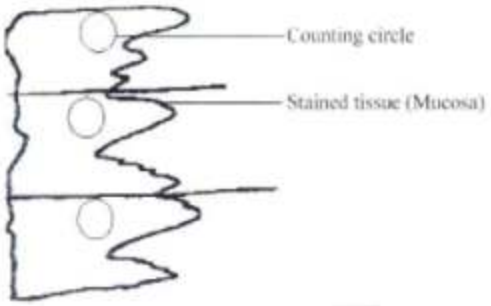


Figure 2: Showing the microscopic fields for counting parietal cell number.

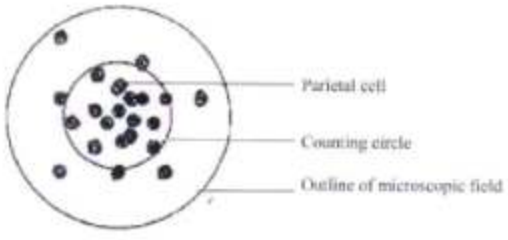


Figure 3: Counting circle

The circle was 5 mm in diameter printed on a transparent plastic sheet, which was cut to fit into eyepiece, thus a black circular outline was superimposed over the actual microscopic field. The intact parietal cells within the counting circle were counted. Average count was calculated from three different fields of each slide. Six average counts were available for the body from each age group. The count was then converted into numbers per square millimeter (mm^2) by a stage micrometer.

The inner diameter of the counting circle corresponded with 11divisions of stage micrometer under an X40 objective and an X10 eyepiece, that is,

11 divisions of stage micrometer=110μm

[Because, one smallest division of stage micrometer equal to 10 micrometer]

Therefore, radius of the circle=110÷2=55μm

Therefore, area of the circle

$=r^2$

$=3.1416 \times [55\mu m]^2$

$=3.1416 \times 55\mu m \times 55\mu m$

$=3.1416 \times 3025\ \mu m^2$

$=9503.34\ \mu m^2$

It is known that $1mm^2=1000 \times 1000\ \mu m^2=1000{,}000\ \mu m^2$

If it is thought that the number of cells counted within the circle is n, then 9503.34 μm^2 contains n number of cells

Therefore, 1 μm^2contains

n ÷ 9503.34 cells

Therefore,

1,000,000 μm^2 contains

[n ÷ 9503.34] 1,000,000 cells

= [n x 1,000,000] ÷ 9503.34 cells

= nx105.22 number of cells

Therefore, N=nx105.22

Where, N= Number of cells per mm² area of the microscopic field
n= Average number of cells counted within the circle (9503.34μm²)

Unpaired t test was applied to test the differences of mean parietal cell among the different age groups.

Results

The mean (±SD) number of parietal cell in group A (0-14 years), B (15-22 years) and C (23 years and above) were 1355.76±52.84, 1420.29±48.32 and 1426.08±114.48 cells/mm² respectively. The mean number of parietal cell was highest in group C (1426.08) and lowest in group A (1355.76). The number of parietal cell increase with age but the differences in the number of parietal cell of the body of the stomach in all age groups (A, B and C) did not reach up to significant level (P>0.05, >0.10 and >0.50).

Student's 't' test, ns = Not significant.
Group A : Age 0-14 years
Group B : Age 15-22 years
Group C : Age 23 years and above

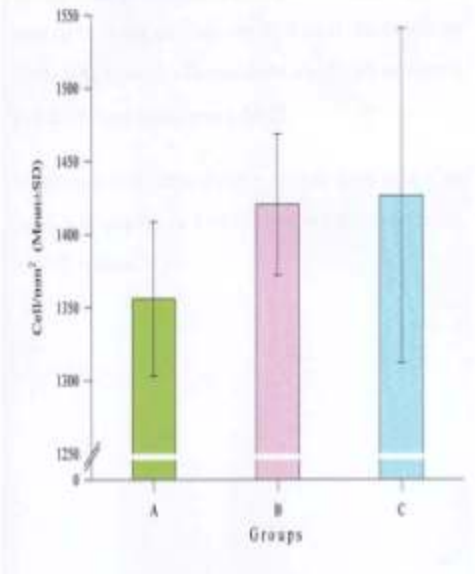


Figure 4: Parietal cell in the histological section of the body of the stomach in different study groups

Group A : Age 0-14 years
Group B : Age 15-22 years
Group C : Age 23 years and above

Table 2: Parietal cells in the histological section of the body of the stomach in different study groups

Group	n	Parietal cell/mm² Mean±SD
A	6	1355.76±52.84 (1297.36-1402.58)
B	6	1420.29±48.32 (1367.86-1473.08)
C	6	1426.08±114.48 (1262.64-1613.02)
		P value
A vs B		$>0.05^{ns}$
A vs C		$>0.10^{ns}$
B vs C		$>0.50^{ns}$

Note: Figures in parentheses indicate range. Statistical analyses were done by Unpaired

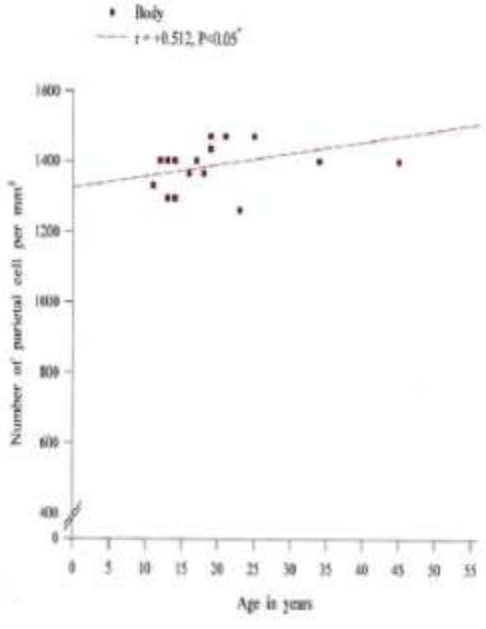


Figure 5: Relationship between age and number of parietal cell in body of the stomach

Discussion

The parietal cells are found in high abundance in the gastric gland and the capacity of the stomach to secret hydrochloric acid is almost linearly related to parietal cell numbers. The number of parietal cell/sq.mm area of microscopic field were counted in the body region of the stomach in the present study.

The mean (±SD) number of parietal cell per square millimeter was highest (1426.08±114.48) in group C and lowest (1355.76±52.84) in group A. The differences of values were not significant (P>0.05, >0.10 and >0.50) when group A was compared with group B and C. The findings were slightly higher than that of Berger.[10]

Age showed positive relationship with number of parietal cell in body (r = +0.512) region which is significant (P<0.05).The findings of this is in agreement with that of Farinnati.[6]

Parietal cells bear receptors for stimulators like Acetylcholine, gastrin, histamine which are reflecting a triumvirate of neural, paracrine and endocrine control.[11] However, parietal cells volume in gastric gland should be dermined appropriately and further studies are also required.

References

1. Borley NR. Stomach and abdominal oesophagus. In: Standring S, Ellis H, Healy JC, Johnson D, Williams A, Collins P, *et al.*, editors. Gray's anatomy: the anatomical basis of clinical practice. 39th ed. Edinburgh: Elsevier Churchill Livingstone, 2005.
2. Eroschenko VP, editor. di Fiore's atlas of histology with functional correlations. 8th ed. Baltimore: Williams and Wilkins, 1996.
3. Helander HF. Physiology and pharmacology of the parietal cell. Baillieres Clin Gastroenterol 1988; 2(3): 539-54.
4. Andreoli TE, Hoffman JF, Fanestil DD, Schultz S Geditors, Clinical Disorders of Membrane Transport Process SS 2nd ed. New York: springer-verlag, 2012.
5. Card WI, Marks IN, the relationship between the acid output of the stomach following 'maximal'histamine stimulation and the parietal cell mass. Clin Sci 1960; 19: 147-63.
6. Farinati F, Formentini S, Libera GD, *et al.* Changes in parietal and \ mucous cell mass in the gastric mucosa of normal subjects with lage: a morphometric study. Gerontology 1993; 39(3): 146-51.
7. Giacosa A, Cheli R. Correlations between structure and function of the stomach and age in normal subjects. Gastroenterol Clin Biol 1979;3:647- 50.

8. Khanna PB, Davies I, Faragher EB. Age-related changes in the stomach of the laboratory mouse: a quantitative morphological study. Age and Aging 1988; 17:257-64.

9. Naushaba H. Study of the effects of spirulina on experimentally induced hypercholesterolaemia and atherosclerosis in rabbits (thesis) University of Dhaka, 1996.

10. Berger EH. The distribution of parietal cells in the stomach: a histotopographic study. Amer J Ana 1933; 54:87-114.

11. Yao X, Forte JG. Cell biology of acid secretion by the parietal cell. Annu Rev Physiol 2002; 65: 103-31.

Juvenile Dermatomyositis in a 12 years old girl: A case report.

MB Uddin[a],MA Rahim[b], MH Tarafder[c], MY Ali[d], RK Nath[e], N Parveen[f], MR Ali[g], N Tasmi[h], A Islam[i]

Abstract

Juvenile Dermatomyositis (JDM) is a rare but potentially life threatening autoimmune disease of childhood. We report the case of a 12 years old female child with proximal muscle weakness of four limbs, heliotrope rash on the eyelids and Gottron papules was diagnosed to have JDM. Since it was early diagnosed and treated, the patient was recovered quickly.

Introduction

Juvenile Dermatomyositis (JDM) is a rare inflammatory myositis in children, distinguished by proximal muscle weakness and characteristics rash. Inflammatory cell infiltrates result in vascular inflammation is underlying pathology in this disorder. It has an incidence of 1.9 - 4 per million children and prevalence of 2.5 per 100,000.[1] Peak age of onset is between 4 and 10 yr.There is a second peak of dermatomyositis onset in late adulthood(45-64)yr.[2]

Etiology of JDM is multifactorial, based on genetic predisposition and an unknown environmental trigger. Human leukocyte antigen (HLA) alleles such as B8, DRB1*0301, DQA1*0501,and DQA-1*0301 are associated with increased susceptibility to JDM in selected populations. Common environmental triggers like enterovirus and group B streptococcus infection are likely to play an important role in the etiology.[2]

JDM has been associated with significant mortality and morbidity in developing countries.[3,4] Early diagnosis and treatment with adequate dose with corticosteroid and other immunosuppressive drugs have improved mortality and morbidity in children.

This paper reports the rare case of JDM with clinical features, laboratory findings and treatment with its response.

Case description

A 12 year old female child, admitted to paediatric department of Rajshahi Medical College hospital, born to a non-consanguineous parents belonging to lower socioeconomic with uneventful antenatal, natal and neonatal period and normal development, immunized as per the EPI schedule of Bangladesh, presented with proximal muscle weakness of all four limbs with generalized muscle pain since one and a half years and photosensitive rash over the face, trunk and upper limbs since one year. She also had low grade irregular fever for same duration.

Figure1: A 12 years girl with proximal weakness and rash .

[a]Professor, Department of Paediatrics, Rajshahi Medical College, Rajshahi, Bangladesh.

[b]Asisstant Professor, Department of Paediatrics, Barind Medical College, Rajshahi, Bangladesh.

[c]Registrar, Department of Paediatrics, Rajshahi Medical College, Rajshahi, Bangladesh

[d]MD student, Department of Paediatrics, Rajshahi Medical College, Rajshahi, Bangladesh

[e]Associate Professor, Department of Biochemistry, Barind Medical College, Rajshahi, Bangladesh.

[f]Assistant Professor, Departmentt of Paediatrics, Rajshahi Medical College, Rajshahi, Bangladesh.

[g]Registrar, Department of Paediatrics, Rajshahi Medical College, Rajshahi, Bangladesh.

[h]Assistant Registrar, Department of Paediatrics, Rajshahi Medical College, Rajshahi, Bangladesh.

[i]Registrar, Department of Paediatrics, Rajshahi Medical College, Rajshahi, Bangladesh.

Correspondence to : MB Uddin
drmbuddin@yahoo.com

Cite this as: BMCJ 2 018;4(2):23-27
Received : 8 August 2018
Accepted : 13 November 2018

There was no history of altered speech or difficulty in swallowing. She also deny history joint pain, oral ulceration and color change of fingers during cold exposure. She got treatment from homeopathic and kobiraz before admission.

On examination the girl was severely ill febrile and mildly pale. She had violacious rash over the both eyelid with periorbital edema (Heliotrope rash), Gottron's papules over metacarpophalangeal and proximal interphalangeal joints of both hands. There was no cutaneous ulceration and sign of arthritis. Muscle wasting was present over proximal muscle group and Power of proximal muscles (2/5) was less compared to distal muscles (4/5). In addition to this, child had a waddling gait and positive Gower sign.

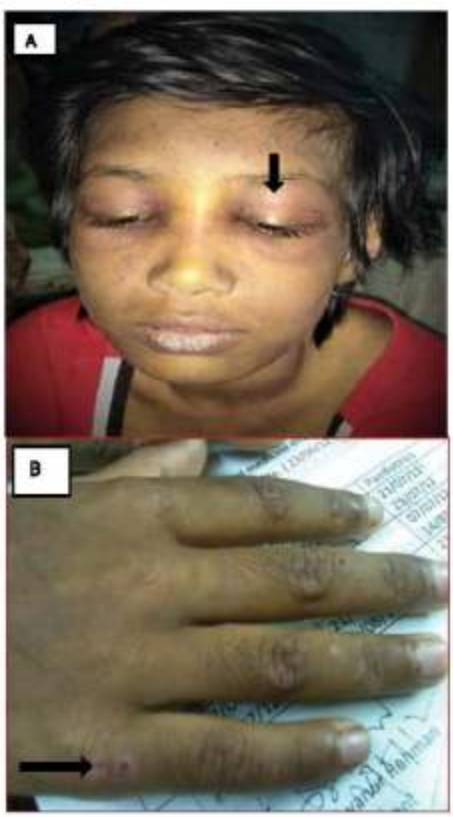


Figure2: A-Heliotrope Rash. B- Gottron's papules

Investigations included a complete haemogram, renal function tests, urine routine which were normal except mild normocytic normochromic anemia (Hb-10.5 gm/dl). ESR-60 mm in 1st hour, ANA positive but anti Ds DNA,Anti Jo-1, Anti Mi-2 were negative. Additional investigations included:
Creatine phosphokinase >13845U/L, alanine aminotransferase 176U/L, MRI of Muscle revels myositis and fascitis of proximal muscle . EMG showed features of myositis and NCS of crossed limbs were normal. These findings clinched the diagnosis of Dermatomyositis.

Figure 3: MRI of Right thigh muscle revels myositis and fasciitis of quadriceps muscle.

Differential diagnosis considered was collagen vascular disease like Systemic Lupus Erythematosus (SLE), but SLE was ruled out based on American College of Rheumatology 1997 revised criteria. Anti DS DNA was also negative.

Diagnosis of juvenile Dermatomyositis was confirmed since the diagnostic criteria included classic rash. Heliotrope rash of the eyelids, Gottron papules, plus 3 of the following:
1) Symmetric and proximal muscle weakness
2)Muscle enzyme elevation:(1 or more) Creatine kinase, Alanine transaminase,
3) Characteristics EMG findings

After proper counseling child was given prednisolone at 2 mg/kg/day. Weekly oral Methotrexate 1 mg/kg was also used as a steroid sparing agent. Folic acid was given with Methotrexate, started at a dose of 1 mg daily to reduce toxicity and side effects of folate inhibition.

Follow up after 4 weeks of treatment muscle weakness was reduced and skin rash were mostly disappeared . Muscle enzyme returned to normal level after 8 weeks of treatment. Then we started to tapering the dose of prednisolone. At present overall condition of the child is better with normal muscle power.

Discussion

JDM is an autoimmune connective tissue disease occurring in children less than 16 years old.[5] The etiology of JDM is multifactorial, based on genetic predisposition and an unknown environmental trigger. Human leukocyte antigen (HLA) alleles such as B8, DRB1*0301, DQA1*0501, and DQA1*0301 are associated with increased susceptibility to JDM in selected populations. Maternal microchimerism may play a part in the etiology of JDM by causing graft-versus-host disease or autoimmune phenomena.[2]

Children with JDM present with either rash, insidious onset of weakness, or both. Fevers, dysphagia or dysphonia, arthritis, muscle tenderness, and fatigue are also commonly reported at diagnosis. Rash develops as the first symptom in 50% of cases and appears concomitant with weakness only 25% of the time. Heliotrope rash of eyelids and Gottron's papules are two most important pathognomic findings of JDM present 66-83 % and 57-91% of patient respectively.[2]

Diagnosis of dermatomyositis requires the presence of characteristic rash as well as at least three signs of muscle inflammation and weakness.

Diagnostic criteria of JDM[6] developed in 1975 by Bohan A and Peter are as:[6]

Classic rash: Heliotrope rash of the eyelids
Gottron papules
Plus 3 of the following:
Weakness Symmetric Proximal
Muscle enzyme elevation (≥1):
Creatine kinase
Aspartate aminotransferase
Lactate dehydrogenase
Aldolase
Electromyographic changes:
Short, small polyphasic motor unit potentials,Fibrillations
Positive sharp waves
Insertional irritability
Bizarre, high-frequency, repetitive discharges
Muscle biopsy: Necrosis, Inflammation

There are three patterns of disease in JDM.[7,8]

Monocyclic course, in which there is one disease episode that responds to standard treatment without relapse (approximately one-third of patients),

Polycyclic course with multiple remissions and relapses (approximately 3 percent).

Chronic persistent course, sometimes with persistent complications (approximately two-thirds)

An increased severity of muscle histo pathologic features was associated with an increased risk of a chronic persistent course (as judged by the need for a longer course of treatment), whereas presence of anti-Mi-2 autoantibodies was, perhaps, associated with a decreased risk.[9] Chronic continuous or polycyclic disease is predictive of a poorer outcome.[10,11] These patients are at increased risk for persistent pain, calcinosis, and disability.

Most complications from this disease are related to prolonged and severe weakness, including muscle atrophy, to cutaneous

calcifications and scarring or atrophy, and to lipodystrophy.[2] Children with acute and severe weakness are at risk for aspiration pneumonia and respiratory failure.[2]Crampy abdominal pain and occult GI bleeding may indicate bowel wall vasculitis and lead to ischemia, GI bleeding and perforation.[2] Cardiac involvement is rare but includes arrhythmias.[9] Malignacy is common in adult onset dermatomyositis but usually not associated with JDM.[2]

Advances in the treatment have improved mortality and morbidity rates in children with JDM. Early treatment may limit JDM to a monocyclic pattern.[12]At 7 yr of follow-up, 75% of patients have little to no residual disability, but 25% continue to have chronic weakness and 40% have chronic rash.[2]

The reported mortality rate has declined from greater than 30 percent in the 1960s[13], before routine glucocorticoid therapy was administered, to less than 2 or 3 percent in the 2000s with the advent of early combination immunosuppressive therapy.[8,10,14,15] So increased awareness should be made for early diagnosis and treatment for better outcome and to prevent long term complications

References:

1. Meyer A, Meyer N, Schaeffer M, Gottenberg JE, Geny B, Sibilia J. Incidence and prevalence of inflammatory myopathies: A systematic review. Rheumatology (Oxford) 2015;54(1):50-63.
2. Rheumatic diseases of childhood, nelson textbook of paediatrics, 20th edition ,page 1181-1186.
3. Prasad S, Misra R, Agarwal V, Lawrence A, Aggarwal A. Juvenile dermatomyositis at a tertiary care hospital: Is there any change in the last decade? Int J Rheum Dis 2013;16(5):556-60.
4. Singh S, Suri D, Aulakh R, Gupta A, Rawat A, Kumar RM. Mortality in children with juvenile dermatomyositis: Two decades of experience from a single tertiary care centre in North India. Clin Rheumatol 2014;33(11): 1675-9.
5. Adelowo O, Nwankwo M, Olaosebikan H; Juvenile dermatomyositis in a Nigerian girl. BMJ Case Rep.2014.
6. Bohan A, Peter JB: Polymyositis and dermatomyositis (second of twoparts), N Engl J Med; 1975;292(8):403.
7. Huber AM, Lang B, LeBlanc CM, et al. Medium- and long-term functional outcomes in a multicenter cohort of children with juvenile dermatomyositis. Arthritis Rheum 2000; 43(3):541-9.
8. Stringer E, Feldman BM. Advances in the treatment of juvenile dermatomyositis. Curr Opin Rheumatol 2006; 18(5):503-6.
9. Deakin CT, Yasin SA, Simou S, et al. Muscle Biopsy Findings in Combination With Myositis-Specific Autoantibodies Aid Prediction of Outcomes in Juvenile Dermatomyositis. Arthritis Rheumatol 2016; 68(11): 2806-16.

10. Ravelli A, Trail L, Ferrari C, et al. Long-term outcome and prognostic factors of juvenile dermatomyositis: a multinational, multicenter study of 490 patients. Arthritis Care Res (Hoboken) 2010; 62(1):63-72.

11. Sanner H, Sjaastad I, Flatø B. Disease activity and prognostic factors in juvenile dermatomyositis: a long-term follow-up study applying the Paediatric Rheumatology International Trials Organization criteria for inactive disease and the myositis disease activity assessment tool. Rheumatology (Oxford) 2014; 53(9):1578-85.

12. Christen-Zaech S, Seshadri R, Sundberg J, et al. Persistent association of nailfold capillaroscopy changes and skin involvement over thirty-six months with duration of untreated disease in patients with juvenile dermatomyositis. Arthritis Rheum 2008; 58(2):571-6.

13. Bitnum s, Daeschner cw Jr, Travis lb, et al. Dermatomyositis. J Pediatr 1964; 64:101-31.

14. Huber A, Feldman BM. Long-term outcomes in juvenile dermatomyositis: how did we get here and where are we going? Curr Rheumatol Rep 2005; 7(6):441-6.

15. Sullivan DB, Cassidy JT, Petty RE, Burt A. Prognosis in childhood dermatomyositis. J Pediatr 1972; 80: 555-63.

Sublingual Salivary Stone
A Case Report

M. Manzurul Haque[a], Md. Ashraful Alom[b], Liza parvin[c], Dewan Md. Musfiq Newas[d], Md. Lutfor Rahman[e], Kh. Md. Adib Hasan[f]

[a]Professor, Department of surgery Barind Medical College, Rajshahi, Bangladesh.

[b]Registrar, Department of Surgery, Rajshahi Medical College, Rajshahi, Bangladesh.

[c]Medical Officer, Department of Surgery, Barind Medical College, Rajshahi, Bangladesh.

[d]Assistant Registrar, Department of Surgery, Barind Medical College, Rajshahi, Bangladesh.

[e]Assistant Registrar, Department of Surgery Barind Medical College . Rajshahi, Bangladesh.

[f]Medical Officer, Department of Surgery, Barind Medical College, Rajshahi, Bangladesh.

Correspondence to : M M Haque
drmanzur7@gmail.com
Cite this as:
BMCJ 2 019;5(1):28-30

Received : 15 October 2019
Accepted : 23 November 2019

Abstract:
The sublingual salivary glands are paired sets of major salivary glands. Sialolithiasis is a frequently occurring disease of the salivary glands. However sublingual salivary stone are not that common. Exact etiology of stone formation in the gland is unknown. Different hypothesis have been submitted about etiology of salivary gland calculi such as mechanical, inflammatory, chemical, neurogenic, infectious, foreign bodies etc. Bacterial infections also play an important role in calculi formation. A 44 years old male presented to us with a swelling in the floor of the mouth associated with pain for 2 years. During intra oral examination of the patient two firm to hard masses opposite the canine and premolar region of the floor of the mouth was demonstrated (Figure 1). Intraoral bimanual palpation revealed the presence of two hard formations over the floor of mouth. A dental radiograph (Figure 2) confirmed that the swelling was radio opaque structure in the floor of the mouth. There were two small sialoliths. We removed the sialoliths surgically under local anaesthesia. Post operative and follow up course was normal.

Introduction

Sialolithiasis is a common disease of the salivary glands with an incidence of 1.2 %[1]. Males are affected more frequently than the female patients.[1] Submandibular gland affected mostly with sialoliths (80%-95%) followed by Parotid gland (5%-20%), Sublingual gland and minor salivary glands are the least affected (1%-2%).[2] Most of the stone formed within the duct rather than in the gland.[3] Mucin rich alkaline saliva contributes to the formation of the sialoliths.[3] Sialoliths measuring >15 mm in any dimension or weighting >1 g are defined as 'giant sialolith'.[4] Diagnosis of sialolithiasis can be done by ultrasonography, radiography and in particular of sialo-magnetic resonance imaging.[5] In case of non radio opaque stone (40% of parotid and 20% of submandibular stones) sialography/sialoendoscopy may be required to locate them.[1] Sialolithiasisis characterized by pain and inflammation and in some occasions with an infection of the affected gland.[3]

Case report

Mr. E 44, years old male hailing from Rajshahi presented with the complaints of a swelling in the floor of the mouth for 2 years associated with pain. During intra oral examination of the patient, two firm to hard masses opposite the canine and premolar region of the floor of the mouth was noticed. The patient was unaware of the swelling until it was associated with pain. Intraoral bimanual palpation revealed the presence of two hard swellings approximately 6-8 mm in length, in the anterior aspect of the floor of mouth opposite the canine and premolar region (Figure 1). The entity was not adherent to the underlying structures. The oral mucosa was normal in texture. A dental radiograph (Figure 2) confirmed that the swelling was radio-opaque structure in the floor of the mouth. On the basis of clinical and radiological findings, a diagnosis of sublingual sialolithiasis was made. Two sublingual sialoliths were removed from the lesion under local anesthesia (Figure3). Post

operative period was uneventful. Follow up till six months was normal.

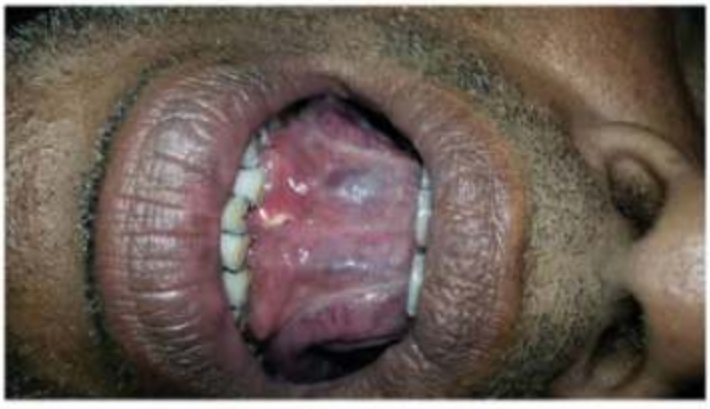


Figure: 1

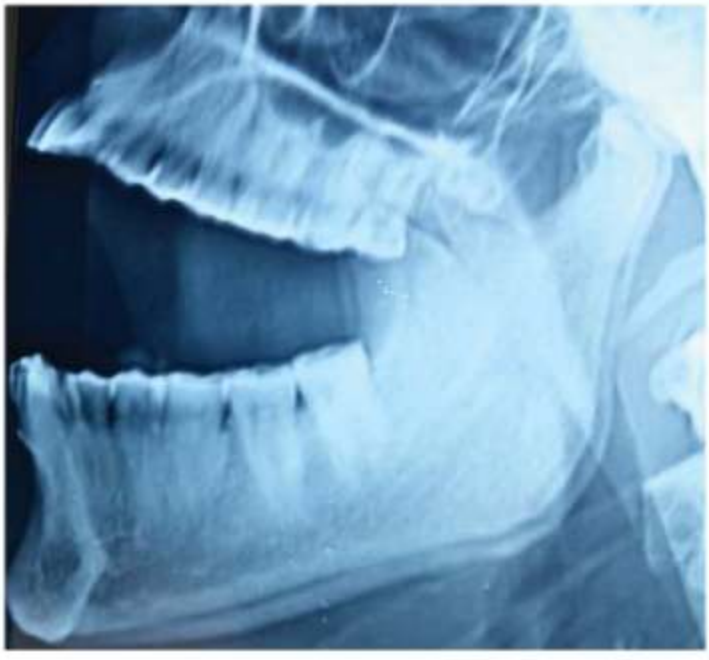


Figure: 2

Figure: 3

Discussion

Sialolithiasis is a frequently occurring disease of the salivary glands. This is presented with pain and inflammation and in certain cases, infection of the affected gland may also be present[3]. Patients usually present with the complaints of pain and swelling of the respective gland[6]. Patients with sialolithiasis present with a painful swelling (59%) painless swelling (29%) and only pain (12%)[7]. They complains of recurrent salivary colic and spasmodic pain upon eating.[7] Pain and swelling gets worse during salivary stimulated condition like meal, sight and hunger so called "mealtime syndrome".[8] Different hypotheses have been postulated about etiology of salivary gland calculi: mechanical, inflammatory, chemical, neurogenic, infectious, foreign bodies, etc. In calculi formation bacterial infections plays an important role.[6] Though the exact etiology is unknown, it is considered that the formation of sialolith is due to deposition of mineral salts around an initial nidus consisting of salivary mucin, bacteria or desquamated epithelial cells.[3] Predisposing factor of sialolith formation is stagnation of salivary flow, high alkalinity and increased calcium content[3]. Poor oral hygiene and delayed teeth may act as etiologic risk

factors.[6] Sialoliths may be single or multiple.[6] There are various methods available for management of sialoliths depending on the gland affected and location of the stone[1]. The treatment of choice for sialolithiasis is the surgical removal of the Sialolith by an intraoral approach.[9] We decided to remove the sublingual sialoliths surgically under local anesthesia. However the newer treatment modalities such as extracorporeal shock wave lithotripsy (ESWL) and more recently endoscopic intracorporeal shock wave lithotripsy (EISWL) are effective alternatives to conventional surgical removal[1].

References

1. Ali I, Gupta A. K, Natu S. S, A. K. Gupta; Unusually large sialolith of Wharton's duct ; Ann Maxillofac Surg 2012;2(1);70-3.

2. Bodner L, Gaint salivary gland calculi; diagnostic imaging and surgical management. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2002; 94(3): 320-3.

3. Arslan S, Vuralkan E, Cobanoglu B, Arslan A, Ural A; Giant sialolith of submandibular gland: report of a case: J Surg Case Rep 2015; 2015(4); rjv043.

4. Gupta A, Rattan D, and Gupta R; Gaint sialoliths of submandibular gland duct; Report of two cases with unusual shape; Contemp Clin Dent. 2013 ; 4(1): 78-80.

5. Andretta M, Tregnaghi A, Prosenikliev V, Staffieri A; Current opinions in sialolithiasis diagnosis and treatment; Acta Otorhinolaryngol Ital 2005 ; 25(3); 145-9.

6. Eyigor H, Osma U, Yilmaz M. D, Selcuk O. T; Multiple sialothiasis in sublingual gland causing dysphagia ; Am J Case Rep 2012;13; 44-6.

7. Honk KH, Yang Y; Sialolithiasis in the sublingual gland. J Laryngol Otol. 2003;117(11); 905-7.

8. Capaccio P, Torretta S, Ottavian F, Sambataro G, Pignataro L. Morden management of obstructive salivary disease, Acta Otorhinolaryngologica Italica 2007, 27(4): 161-72.

9. Jung J. H, Hung S. O, Noh K, Lee D. W; A large sialolith on the parenchyma of the submandibular gland: A case report. Exp Ther Med. 2014; 8(2): 525-6.

Barind Medical College Journal (BMCJ)

Information for Authors

General

1. Articles for publication, original articles, review articles, case reports and other communications from authors should be directed to Prof. Dr. Md. Manzurul Haque, **Editor-in-Chief at the following address:**
BMCJ Division
Barind Medical College
Choto Bongram, Namovodra, Boalia, Rajshahi-6207
Mobile: +8801711815827

2. Manuscripts submitted to ***Barind Medical College JOURNAL (BMCJ)*** should be submitted with the understanding that they have neither been published, nor are, under consideration for publication elsewhere, except in the form of an abstract. Prior abstract publication(s) should be described in the form of a footnote to the title. Articles should contain original data concerning the course (prognosis), cause (etiology), diagnosis, treatment, prevention, or economic analysis of a clinical disorder or an intervention to improve the quality of healthcare. Published manuscripts become the sole property of the Journal and will be copyrighted by Barind Medical College; By submitting a manuscript to the Journal, the author(s) agree(s) to each of these conditions.

3. All submissions should be accompanied by a forwarding letter listing signed by all authors with affiliations and addresses of the authors and identifying the author to receive correspondence and proofs. (A corresponding author's name, address, mobile no., and e-mail address must be specified in the letter).

4. All submissions to BMCJ are subject to peer review. The principal author may be asked for the names and email addresses of potential suggested reviewers familiar with the field. Please ensure preferred reviewers are not from your university or institution with whom you have collaborated. Anyone whom the author does not want to be considered may also be named as a non-preferred reviewer. Ultimately, the final selection of reviewers is at the discretion of the Editor(s) of BMCJ.

5. All authors should be responsible for a significant part of the manuscript. All authors should have taken part in writing, reviewing, and revising the intellectual and technical content of the manuscript. Any author whose name appears on an article assumes responsibility and accountability for the results. The editorial Board does not subscribe to the opinion and views expressed in the article.

6. It is incumbent upon the submitting author/agent **to ensure the accuracy and inclusion of all contributing authors' names and affiliations** upon original submission of the article. Once an article is accepted for publication, changes in authorship while the article is in production – including page proofs – are NOT permitted. Changes in authorship after publication are strictly prohibited.

7. Three paper (hard) copies of the manuscript and a soft copy on a CD-Rom in MS-Word (in Doc. Format) should be sent.

Manuscript Preparation

All manuscripts should be concise and written in English in a readily understandable style.

WORD and DATA LIMITS

Article Type	Maximum Word Limit*	Abstract Word Limit	References	Figures	Tables
Original	6,000	300 (structured)	100	5	5
Review	10,000	400	200	10	8
Case reports	4,000	250	15	2	2
Short communication	2,000	200	15	2	2
Editorials	1000	N/A	10	2	2
Letter to the Editor	500	N/A	5	2	1

**These limits relate to the text of the manuscript; word limits do NOT include the abstract, figure and/or table legends, acknowledgments, disclosures, or references.*

Requirements for text

Prepare all text, double spaced, in Microsoft Word. Do NOT supply a PDF of your manuscript. Provide the order of items as follows:

- Title page
- Abstract (structured)
- Text (*Introduction, Materials and Methods, Results, Discussion and Conclusion(s))*
- Acknowledgments
- Author Disclosure Statement(s)
- References
- Correspondence address
- Legends
- Tables
- Figures

Requirements for Title Page

The title page of your submission should be prepared in Microsoft Word and MUST be included as part of your main text document (not as a separate file) and should contain the following items:

- The complete title of the article
- **All** contributing authors' full names, complete affiliation(s), including department, institution, city, state, country.
- A brief running title of no more than 50 characters, including spaces
- The corresponding author's complete contact information including address, working fax number, and email address

Requirements for Abstract

- The abstract should be prepared in Microsoft Word
- Abstract should be no more than 300 words
- Abstract should be **structured**, stating the background, methods, results (including the sample size), and conclusions drawn from the study
- The use of the first person should be avoided
- Do not use proprietary or trade names in the title or abstract
- Clearly summarize the results and conclusions of the work
- References are not permitted in the abstract
- 3-5 key words which must being with small letter unless special necessity

Text

In general, the text should be organized under the headings: *Introduction, Materials and Methods, Results, Discussion, Conclusion(s), Acknowledgment(s) Author Disclosure Statements,* and *References*. Use only standard abbreviations, which can be found in the AMA's Manual for Authors & Editors or the Council of Biology Editors Style Manual. At first usage, spell out terms and give abbreviations in parentheses. Thereafter, use only abbreviations. It is not necessary to spell out standard units of measure, even at first usage. Use generic names for drugs if possible.

Acknowledgments

The author should acknowledge only those people and their institutions that have made significant contributions to the study.

Disclosure Statement

Immediately following the *Acknowledgments* section, include a section entitled, *Author Disclosure Statement*. In this portion of the article, authors must disclose any commercial associations that might create a conflict of interest in connection with submitted manuscripts. This statement should include appropriate information for EACH author, thereby representing that competing financial interests of all authors have been appropriately disclosed according to the policy of the Journal. It is important that all conflicts of interest, whether they are actual or potential, be disclosed. This information will remain confidential while the article is being reviewed and will not influence the editorial decision. Please see the Recommendations for the Conduct, Reporting, Editing and Publication of Scholarly Work in Medical Journals at www.icmje.org/recommendations/browse/roles-and-responsibilities/author-responsibilities-conflicts-of-interest.html for further guidance. If no conflicts exist, the authors must state "No competing financial interests exist."

References

References must be prepared double spaced and **numbered consecutively as they are cited in the text using superscript numbers.** Do not include reference numbers in parentheses or brackets. References appearing for the first time in tables and figures must be numbered in sequence with those cited in the text where the table or figure is mentioned. **Use journal abbreviations as provided by Medline.** List all authors when there are six or fewer. When there are more than six authors, list the first three, followed by et al. Personal communications or unpublished data are not used as references.

Sample references:

- ***Journal article with up to six authors:*** Preyde M, Hatton-Bauer J, unningham C, Panjwani D. Evaluation of an informational pamphlet on distress and erceptions of supportive care for men with prostate cancer. J Men's Health 2012;9:160–167.
- ***Journal article with more than six authors:*** Sandhu NP, Brid Mac Bride M, Dilaveri CA, et al. Male breast cancer. J Mens Health 2012;9:146–153.
- ***Book:*** Klabunde RE. Cardiovascular Physiology Concepts. Phildadelphia, PA: Lippincott Williams & Wilkins, 2011.
- ***Chapter in a book:*** Certo CM, De Turk WE, Cahalin LP. History of Cardiopulmonary Rehabilitation In: De Turk WE, Cahalin LP, editors. Cardiovascular and pulmonary physical therapy, second edition: an evidence-based approach. New York, NY: McGraw Hill-Medical, 2010. p. 3-15.
- ***Websites:*** *Please follow this structure for website references, including capitalization and punctuation:* List author/organization name (if available). Article title. List website address. Last accessed on (include last date the site was accessed.)
- ***Conference Proceedings****: Please follow this structure for Conference Proceeding references, including capitalization and punctuation:* List all Authors' (or) Editors' names (last name first, followed by first and middle initials). Conference title. Date of conference. Location of conference. City of publisher: Publisher; Year of publication. Complete number of pages in proceedings book.

Correspondence Address

Following the references, provide the name, postal mailing address, and valid email address of the corresponding author. If accepted, this information will be published and made available to the public.

Legends

Supply a single, separate page/file containing all figure legends. Provide a double-spaced legend for each supplied figure. Legends should be numbered consecutively. If applicable, provide explanations in the legend for any abbreviations, arrows, etc. in the figure. If a figure is being reprinted from a copyrighted publication, appropriate credit must be given in the legend.

Tables

Prepare all tables double spaced in one separate page for each in Microsoft Word. Be sure to provide a title for each table. Cite tables in sequence in the text. Explain abbreviations used in the body of the table in footnotes. If a table is being reprinted from a copyrighted publication, appropriate credit must be supplied in a footnote.

Figures

Cite figures consecutively in the manuscript within parentheses: [*Example:* These keratotic areas can be confused with condyloma (Fig. 2A).]

PREPARATION OF FIGURE FILES

- Figures should be numbered in the order cited in the text.
- Figures should not show the name of a patient or a manufacturer.
- Name figure files using only alphanumeric characters. Do not use symbols, dots, or dashes.
- File names should be formatted with first author's last name and the figure number. (Ex: SmithFig1)
- **Do not embed any figures or tables in the main text.**

• Publication of color figures is encouraged, but the cost for color printing must be subsidized by the author(s). Contact the Publisher for an estimate. Please consider these costs when preparing your manuscript for submission.

IMPORTANT

Patient Release Information

If applicable, it is incumbent upon the author(s) to obtain patient release statements of permission to reproduce any identifiable images of patients. The submitting author should provide written confirmation of this critical information. Acceptable forms of consent statements are emails or letters. The Journal does not provide a generic patient release form.

The written consent must contain specific information about the patient's name, age, and if pertinent, conservatorship – as well as stated permission – granting the Journal the rights to publish the photograph within its pages (which should include the name of the Journal and your article title).

INFORMED CONSENT, STUDY ETHICS APPROVAL, AND SUBJECT CONFIDENTIALITY

Patients and Study Participants

All manuscripts must comply with the privacy and confidentiality requirements outlined on the Recommendations for the Conduct, Reporting, Editing and Publication of Scholarly Work in Medical Journals website. For more information, visit www.icmje.org/recommendations/browse/roles-and-responsibilities/protection-of-research-participants.html

When articles include reports of studies on human subjects, state in the Methods section that an appropriate institutional review board or ethics committee approved the study. Authors who do not have formal ethics review committees should follow the principles of the Declaration of Helsinki. In the Methods section, state that informed consent was obtained from subjects (specify written or verbal).

The principal author must state that if animals were used experimentally, permission was obtained from the appropriate committee(s), and that the animals were treated humanely and the standards conformed to those of current ethical animal research practices.

In addition, text and photographs should not reveal any identifying information unless it is essential for scientific purposes (in which case, consent should be obtained). Masking the subjects' eyes in photographs is often insufficient to protect their identity.

ETHICAL CONSIDERATIONS IN THE CONDUCT AND REPORTING OF RESEARCH: PROTECTION OF HUMAN SUBJECTS AND ANIMALS IN RESEARCH

Protection of research participants

When reporting experiments on people, authors should indicate whether the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national), or if no formal ethics committee is available, with the Helsinki Declaration as revised in 2008. If doubt exists whether the research was conducted in accordance with the Helsinki Declaration, the authors must explain the rationale for their approach and demonstrate that the institutional review body explicitly approved the doubtful aspects of the study. When reporting experiments on animals, authors should indicate whether institutional and national standards for the care and use of laboratory animals were followed.

PUBLISHER

The Journal is published by **BMCJ Division, Barind Medical College, Choto Bongram, Namovodra, Boalia, Rajshahi-6207, Mobile: +8801711815827, E-mail: md.anayet_u@yahoo.com**

For speedy relief in Acid Peptic Diseases

acifix®

Rabeprazole 20 mg Tablet

Fixes acid FAST

Faster activation rate due to highest pKa (5.00)

12

times faster onset of action than conventional proton pump inhibitors

Ensures faster relief from Acid peptic diseases

Highest parietal cell concentration

13%

more potent than conventional proton pump inhibitors

Ensures powerful relief from acid peptic diseases

Longest methoxy-propoxy side chain

45%

additional mucus and mucin secretion

Provides more protection against acid and NSAIDs

Non-cytochromic metabolism

Less inter-patient variation in clinical efficacy

Ensures superior efficacy in all patients

Absorption and activation at high pH

Can be taken irrespective of meal

Ensures dosing compliance and adherence to therapy

** Full prescribing information is available upon request*

References: 1. Annual Review of Genomics and Human Genetics.Sept, 2001. Vol. 2: 9-39 2.www.aciphex.com 3. J. vet. Pharma col. Therap. 27, 455–466, 2004.

BEXIMCO PHARMA

BEXIMCO PHARMACEUTICALS LTD.
Dhaka, Bangladesh

Approved by the U.S. FDA
also Certified by

AGES

Health Canada

For more information visit: www.beximcopharma.com
PTG- 109714/02-17/55,000 HPL

The Newest technological approach

FIRST TIME IN BANGLADESH

MUPS

Multiple
Unit
Pelliet
System

Once daily

Nexum® MUPS 20

Esomeprazole 20 mg

Tablet

The only **Esomeprazole**
can be taken **with** or **without** food

Since 1958

SQUARE
PHARMACEUTICALS LTD.
BANGLADESH

www.squarepharma.com.bd