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Media and Suicide

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Now-a-days, people are increasingly using new media technology (e.g., smartphones, tablets, laptops, and desktops for Internet access), with increasing frequency to text message, E-mail, blog, as well as access social networking websites (like Facebook, Twitter, WhatsApp, Instagram etc.) for business, entertainment, and to stay in touch with family and friends and simultaneously acquiring information about national and world events from other traditional sources of mass media (like television, newspaper, magazines, books, movies, radio etc.). Mass media have a strong effect on our expectations for family, parents, and children, creating standards for our way of life, affection, worship, and society. It also serve as socializing agents that aids in construction and perpetuation of perceptions and learned behaviors.¹ In simple words, we view reality in terms of our own experience that is influenced by primary groups (Family and Friends); secondary groups (School, religious institutions, and government), and mass media. While the influence of the primary group is waning and the secondary groups are time limited, the influence of mass media is increasing as it is a pervasive and permanent fixture of our lives. In addition, time spent with media decreases the amount of time available for pursuing other more healthy activities such as sports, physical activity, community service, cultural pursuits, and family time. Children who watch more television than their peers experience significant impairments in comprehending stories, a crucial skill in achieving

academic success.⁷

Two mass communication theories, cultivation theory, and social learning theory work in tandem to influence the construction and perpetuation of mental illness stigma. Cultivation theory proposes that those who spend more time "living" in the virtual world of television may perceive the "real world" as per the imagery, principles, and portrayals depicted on the small screen. People who spend a lot of time watching television are likely to assume a television worldview of mental illness.²

There is evidence to suggests that particular mode of reporting and portrayal of suicide in the mass media may result in increased rates of suicide in vulnerable people.⁴ By modifying their reporting and portrayal of suicide, the media can contribute to suicide prevention as was proved by the Vienna experiment where changes in media reporting resulted in a > 80% reduction in the number of subway-suicides and suicide-attempts.⁵ The protective media effects are termed the *Papageno effect*, as opposed to the harmful *Werther effect*.⁶ The consensus at present is that prominent display of media reports about suicide result in a significant increase in suicide attempts, especially among adolescents and young adults, within the media outlet's coverage area. Based on research experience, a number of guidelines on media reporting have been formulated.³ While reporting suicide, media have to avoid generalizations based on little evidence and shun catchy, sensational but inaccurate expressions such as

“epidemic of farmer suicide” or “suicide capital of the world”; etc. Before releasing the news, the journalist/editor should consider its effect on families and other survivors regarding both stigma and psychological suffering. Sensational reporting in explicit detail of suicides or self-harm especially when a celebrity is involved is probably the norm and may be legitimate news. However, it should actually be avoided or minimized to the extent possible. Detailed descriptions of the method used and how it was procured should be completely avoided.

In print media, the news should not appear on the front page with a banner or large font headline and should not mention suicide. The method should not be mentioned in the headline like “jumped from a building”. Mentioning the full name or other personal information of the deceased or attempter or printing his photo or location may pass a wrong signal to the vulnerable people that committing suicide can make them famous. Do not illustrate the suicide method or venue of suicide in graphic presentation. The reporting should pay special attention to this. The reason for suicide must not be oversimplified. Suicide never occurs due to a single factor or event, but is the result of a complex interaction of a number of factors and often there is a background of psychosocial problems. It should be emphasized that the overt cause was the precipitating event and not the only cause of the suicide. While publicizing the background factors that may have played a causative role is neither necessary nor desirable, they should be acknowledged. Any history of psychiatric disorders including drug abuse should be mentioned.

In the visual media, avoid presenting suicide cases as the headline TV news unless the reporting involves public interest; avoid repetitive, ongoing and excessive reporting of the events. The breathless excitement of the reporter should be tempered by the tragic event that he is reporting. Mourning the dead is appropriate. Glorification of the suicide victims as martyrs may encourage vulnerable persons to imitate the behavior to win public adulation. Highlighting the adverse consequences of deliberate self-harm (brain damage, paralysis, etc.) may deter future attempts.

Celebrity suicide

Undoubtedly, celebrity suicide has great news value; however, it can also influence the vulnerable and suicidal people. The reporting should be cautious, factual and mourning and not glorify, sensationalize or romanticize. While oversimplification of the causes of suicide should be avoided, a history of psychiatric disorder including alcohol or drug abuse should be clearly stated. Repetitive reporting of a celebrity suicide should be avoided since susceptible people may develop enhanced suicidal ideation on being inundated and overwhelmed with details about a specific suicide.^{8,9}

Interviewing surviving relatives and friends

In the immediate aftermath of suicide, the grieving relatives and friends may have fluctuating emotions, anger, and even suicidal thoughts. Out of humanitarian considerations, the media should avoid disturbing them for a sound bite. The relatives and friends are unlikely to reveal any earth-shattering news but may say things which they would regret later. Great restraint and sensitivity is called for if the relatives have to be interviewed. Publishing photographs of the deceased or the surviving relatives should be avoided as it may greatly hurt them.²

Conclusion: Mass media, due to its tremendous reach and constant exposure have the unique ability to alter perception and sway popular opinion of large number of people. In fact, the media shape our ideas and understanding of various issues and events. Persistent repetition along with reporting intricate details about various aspects of suicidal behaviour may help in some cases but can also increase rates of suicide in vulnerable people and does harm society. So, it is necessary to build up an awareness of these adverse consequences and sensitive reporting of issues relating suicide. This may help to reducing suicide rates.

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REFERENCES

1. Bandura A. Social cognitive theory of mass communication. In: Bryant J, Oliver MB, editors. *Media Effects: Advances in Theory and Research*. 2nd ed. Mahwah, NJ: Lawrence Erlbaum; 1992.
 2. Gerbner G, Gross L, Morgan M, Signorielli N, Shanahan J. Growing up with television: Cultivation processes. *LEA's Communication Series*. In: Bryant J, Zillmann D, editors. *Media Effects: Advances in Theory and Research*. Mahwah, NJ, US: Lawrence Erlbaum Associates Publishers; 2002. p. 43–67.
 3. Fernando SM. *Mental health and the Media*. Colombo: Voluntary Services Overseas; 2011.
 4. Williams K, Hawton K. *Suicidal Behavior and the Media*. Oxford: Oxford University Press; 2001.
 5. Etzersdorfer E, Sonneck G. Preventing suicide by influencing mass-media reporting. The Viennese experience 1980–1996. *Arch Suicide Res*. 1998; 4: 67–74.
 6. Niederkrotenthaler T, Stack S. *Media and Suicide: International Perspectives on Research, Theory, and Policy*. London: Routledge; 2017.
 7. Calvert SL. Media and early development. In: McCartney K, Phillips DA, editors. *Blackwell Handbook of Early Childhood Development*. Boston, MA: Blackwell; 2006. p. 843–879.
 8. Niederkrotenthaler T, Fu KW, Yip PS, Fong DY, Stack S, Cheng Q, et al. Changes in suicide rates following media reports on celebrity suicide: A meta-analysis. *J Epidemiol Community Health*. 2012; 66: 1037–1042.
 9. Niederkrotenthaler T, Voracek M, Herberth A, Till B, Strauss M, Etzersdorfer E, et al. Role of media reports in completed and prevented suicide: Werther vs. papageno effects. *Br J Psychiatry*. 2010; 197: 234–243.
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Prevalence of 25-hydroxy Vitamin D Deficiency: A Hospital Based Study among Healthy Bangladeshi Volunteers

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ABSTRACT

Introduction: Vitamin D insufficiency is currently recognized as a pandemic. The aim of this study was to determine 25-hydroxy vitamin D [(25(OH)D] concentrations in a hospital-based healthy Bangladeshi volunteer. **Methods:** This cross-sectional study was conducted in the Department of Rheumatology at Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh from July to September 2014. A total number of 100 adult healthy volunteers of both genders were recruited purposively in this study. The anthropometric parameters, dietary evaluation (24 hours food recall) to quantify calcium and protein intake and serum 25(OH)D level were measured. **Results:** Among the respondents, 73 were female and 27 were male. The age range of the participants was 21 to 39 years. The mean 25(OH)D (ng/ml) was 20.58 ± 4.35 . Only 3% of the participants had sufficient 25(OH)D ($>30\text{ng/ml}$); furthermore, 97% respondent had vitamin D deficiency (VDD) or insufficiency. Dietary calcium intake (mg/day) was 310.51 ± 188.02 . Protein intake was (gm/day) 52.55 ± 16.82 . **Conclusion:** 25(OH)D insufficiency and deficiency were found in a considerable number of healthy hospital-based populations.

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INTRODUCTION

Vitamin D insufficiency affects almost 50.0% of the population worldwide.¹ An estimated one billion people worldwide, across all ethnicities and age groups, have a vitamin D deficiency (VDD).¹⁻³ This pandemic of hypovitaminosis D can mainly be attributed to lifestyle and environmental factors that reduce exposure to sunlight, which is required for ultraviolet-B (UVB)-induced vitamin D production in the skin.

Vitamin D is critical for bone health. VDD causes impaired calcium absorption which can lead to rickets and osteomalacia.⁴⁻⁶ Additionally, VDD is associated with osteoporosis and an increased risk of fractures.^{7,8} Furthermore, accumulating observational evidence suggests that low vitamin D levels are associated with extra-skeletal sequel, including increased risks of cancer, cardiovascular disease, infection, and autoimmune diseases.¹ Emerging research supports the possible role of vitamin D against influenza, type-2 diabetes, and depression.⁹ Vitamin D deficiency has also been associated with female-specific health concern, including preeclampsia, breast cancer and post-menopausal syndrome.¹⁰⁻¹²

Serum 25(OH)D levels reflect body stores of vitamin D.¹ VDD has been historically defined and currently recommended by the Institute of Medicine (IOM, USA: <http://www.iom.edu/>) as a 25(OH)D <12ng/ml. Vitamin D insufficiency has been defined as a 25(OH)D between 12 and 20 ng/ml.¹⁰ Some of the older publications defined VDD as 25(OH)D <15 (or 20) ng/ml and insufficiency as 16-30 (or, 21-30) ng/ml.¹¹⁻¹⁵ Endocrine Society of America, National Osteoporosis Foundation (Arlington, VA. USA), International Osteoporosis Foundation (Switzerland), and American Geriatric Society suggest that a minimum 30 ng/ml is necessary in older adults to minimize the risk of falls and fracture.

VDD is common in Australia, the Middle East, India, Africa, and South America.^{16,17} A recent study conducted in Pakistan revealed that 90% of premenopausal female had 25(OH)D concentration < 20ng/ml,¹⁸ despite the fact that the country is located in the subtropical region with sunny climate. Another study at Lahore, Pakistan,

among healthy women of child bearing age concluded that illiteracy, decreased sun exposure (religious culture of putting on 'hijab' might have contributed) and lack of multivitamin were responsible for VDD.¹⁹

Prevalence of VDD among staffs in an academic institution in Costa Rica was high. Only 17% of the study population had 25(OH)D \geq 30 ng/dl.²⁰ Study conducted in Bangladesh²¹ among 189 young women of two socio-economic groups in rural and urban regions revealed that 67.0% and 50.0% from the low and high socioeconomic groups respectively had VDD or insufficiency.

Data from the National Health and Nutrition Examination Surveys (NHANES) in the US showed a decrement trend in mean 25(OH)D.²¹⁻²³ This prompted us to an exploratory investigation aimed to determine 25(OH)D in Bangladesh among a hospital based healthy Bangladeshi volunteers.

METHODS

This cross-sectional study was conducted in the Department of Rheumatology at BSMMU, Dhaka, Bangladesh from July to September 2014 for a period of three months. Apparently healthy young subjects aged more than 18 years of both genders were selected as study population. The subjects were 'hospital based', like, physicians/ students of department of rheumatology and some of their spouses, attendants of the patients, hospital staffs like staff nurses, laboratory technicians, ward boys, cleaners, security staffs. After obtaining the informed written consent of the study subjects, study questionnaire was served.

Pregnant and lactating women, those who had received drugs likely to affect vitamin D status in last 2 years (like corticosteroids, gonadotrophin-releasing hormone agonists, aromatase inhibitors, thyroxine, anticonvulsants, heparin), current tobacco user, those with medical disorders likely to affect bone mineral density and vitamin D status like hypogonadism, hyperthyroidism, hyperparathyroidism, Cushing's syndrome inflammatory bowel disease, rheumatoid arthritis, ankylosing spondylitis, chronic liver disease, chronic kidney disease, malabsorption were

excluded from the study. Subjects who were on calcium and vitamin D supplements for more than 3 months were also excluded from this study.

After selection of subjects, screening, non-fasting venous blood sample was collected for vitamin D assay. Anthropometric parameters including weight, standing height, BMI was obtained. Food recall interview was done by an experienced nutritionist. Study subjects stated about the food and beverages they consumed in the last 24 hours (24 hour food recall), from which daily calcium intake (mg/day) and elemental protein intake (gm/day) were determined. Any inconsistency was dealt with by re-interview. Vitamin D level was expressed as nanogram/ml (ng/ml). VDD was defined as serum 25 (OH) D <10.0 ng/ml and insufficiency as 25(OH) D between 10 and 29.9 ng/ml. Simple descriptive measures like percentage, mean and standard deviation of

different variables were used. Analysis was done using SPSS version 15.

RESULTS

A total number of 100 adult respondents were recruited for this study (female 73, male 27). The age range of the study population was 21 to 39 years. Baseline demographic characteristics of the study subjects and their daily intake of calcium and protein are depicted in Table I. Mean age of the study subject was 29.16 ± 5.32 years, male 30.63 ± 5.15 , and female 28.61 ± 5.32 . Mean height and weight of male (165.33 ± 5.85 cm, 64.74 ± 9.735 kg), were higher than those of female (152.23 ± 5.70 cm, 56.36 ± 10.61 kg). The BMI (Body mass index) of female (24.28 ± 4.13 kg/m²) was higher than that of male (23.68 ± 3.31 kg/m²).

Dietary intake of calcium (mg/day) for male was 346.25 ± 196.77 , for female was 297.29 ± 184.31 in the series. Protein intake (gm/day) for male was 65.34 ± 19.70 , for female was 47.83 ± 12.85 .

Table I: Demographic characteristics and daily intake of calcium and protein of the study subjects

Variables	Mean \pm SD		
	Female	Male	Total
Age (years)	28.61 ± 5.32	30.63 ± 5.15	29.16 ± 5.32
Height (cm)	152.23 ± 5.70	165.33 ± 5.85	155.88 ± 8.21
Weight (kg)	56.36 ± 10.61	64.74 ± 9.735	60.28 ± 10.36
BMI(kg/m ²)	24.28 ± 4.13	23.68 ± 3.31	24.01 ± 3.61
Calcium intake (mg/day)	297.29 ± 184.31	346.25 ± 196.77	310.51 ± 188.02
Protein intake (gm/day)	47.83 ± 12.85	65.34 ± 19.70	52.55 ± 16.82

The vitamin D level was estimated in all 100 study subjects (female 73, male 27). The mean of 25(OH)D (ng/ml) was 20.58 ± 4.35 (all study

population), 19.57 ± 3.53 (female), 23.30 ± 5.18 (male) (Table II).

Table II: Vitamin D Status of Study Subjects (n-100)

Gender	Age Group in years	Number	Total Number	25(OH) D (ng/ml) mean \pm SD
Female	21 to 30	49	73	19.57 ± 3.53
	31 to 39	24		
Male	21 to 30	14	27	23.30 ± 5.18
	31 to 39	13		
Total			100	20.58 ± 4.35

The prevalence of sufficiency, insufficiency, and deficiency of 25(OH)D in the study subjects were

3(3.0%), 96 (96.0%) and 1 (1.0%) respectively (Table III).

Table III: Distribution of Study Population according to vitamin D level (ng/ml)

Vitamin D range	Frequency	(%)
30-100 (sufficient)	3	3.0
11-29 (insufficient)	96	96.0
<10 (deficient)	1	1.0
Total	100	100.0

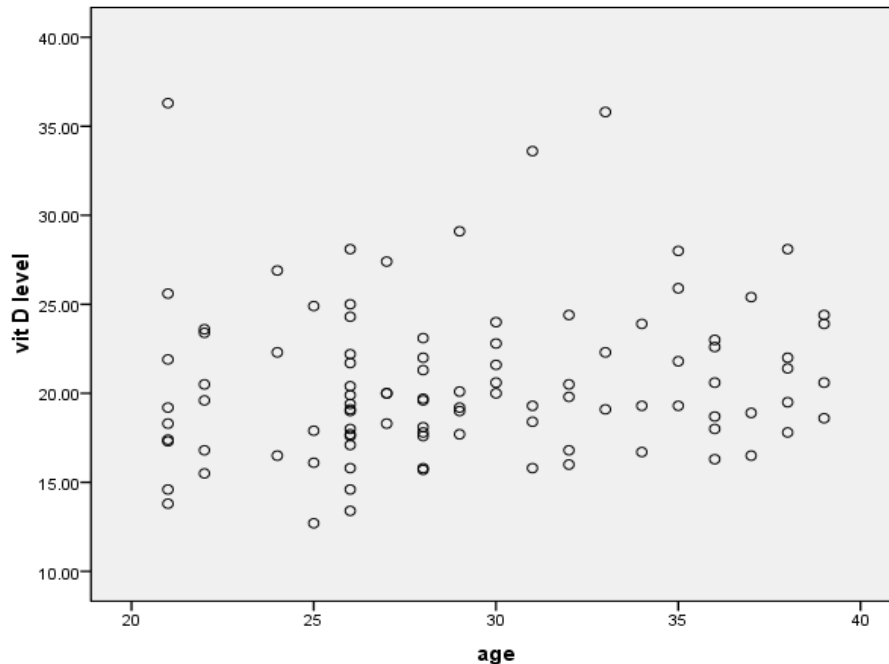


Figure 1: Vitamin D level plotted against age of the participants

DISCUSSION

This study evaluated the concentration of 25(OH)D in a small sample of an apparently healthy, hospital based young (age 21-39) population of both gender in Dhaka city in 2014 (July to September). The anthropometric parameters, dietary evaluation to quantify calcium and protein intake was also done.

The mean 25(OH)D (ng/ml) was 20.58 ± 4.35 (All study population), 19.57 ± 3.53 (female), and 23.30 ± 5.18 (male). This mean value is a bit lower than the level found in a similar study conducted in Costa Rica (23.9 ± 7.0) on a similar number of population.²⁰ Among the study population 96% had 25(OH)D in the insufficiency range. Only 3% of the study population had sufficient 25(OH)D. Value [25(OH)D] of the female subjects was found significantly lower than the male in our study.

Mean daily protein intake (gm/day) for male was 65.34 ± 19.70 , and for female was 47.83 ± 12.85 . Male meets more or less the recommended daily allowance (RDA, 1 mg/kg body weight) but female consume significantly less amount of protein. Significant difference of protein intake between male and female might have impact on the vitamin D level difference between male and female.

The number of study population (n-119) and study results (92% had vitamin D deficiency or insufficiency) conducted in Aga khan university hospital, Pakistan²⁴ is similar to this present study. Although that study was conducted on a group of ambulatory patient, from medicine and endocrine clinic. Vitamin D status among Bangladeshi women of reproductive age (study conducted in 2009 Pabna, Bangladesh) revealed that 119 women (80.96%) had vitamin D deficiency or insufficiency and 28(19.04%) had sufficient vitamin D.²⁵

Another study conducted in Bangladesh²¹ among 189 young women of two socio-economic groups in rural and urban regions (Dhaka and Nandail of Mymensingh respectively) revealed that 67.0% and 50.0% from the low and high socio-economic groups respectively had VDD or insufficiency.

This current study showed a poorer vitamin D status compared to both of those earlier Bangladeshi studies. Vitamin D level of 18 physicians and their spouses in rheumatology and medicine department revealed even a poorer status (mean vitamin D 19.50 ± 3.92 ng/ml). Daily calcium intake was observed low in the series (male 346.25 ± 196.77 mg/day, female 297.29 ± 184.31 mg/day), which is well below the recommended daily allowances (400 mg/day). It is lower in comparison to a study conducted in India.²⁶ Global decrement trend of the level of vitamin D, and the fact that most of the current study population spend more indoor hours during day time, and a lower (mean) intake of calcium and protein, than the recommended daily allowances, may explain the vitamin D status.

Sample size was small (n=100) and sampling was purposive. For resource constraint, serum level of calcium, inorganic phosphate, alkaline phosphates and serum albumin were not done. Further study may be carried out in the context of low vitamin D, in particular female subpopulation.

Vitamin D insufficiency and deficiency were found in healthy hospital based population in Dhaka, Bangladesh. Only 3% of the study population had sufficient vitamin D, 96% had their values (vitamin D) in the insufficiency range. From a public health approach, healthy solar ultraviolet radiation exposure along with physical activity and vitamin D supplementation could be recommended in office workers and homebound citizens with VDD. Comprehensive study may help to resolve many critical issues on vitamin D deficiency and insufficiency.

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REFERENCES

1. Holick MF. Vitamin D deficiency [Review]. *N Engl J Med*. 2007; 357: 266–281.
2. Gordon CM, DePeter KC, Feldman HA, Grace E, Emans SJ. Prevalence of vitamin D deficiency among healthy adolescents. *Arch Pediatr Adolesc Med*. 2004; 158: 531–537.
3. Lips P, Hosking D, Lippuner K, Norquist JM, Wehren L, Maalouf G, et al. The prevalence of vitamin D inadequacy amongst women with osteoporosis: An international epidemiological investigation. *J Intern Med*. 2006; 260: 245–254.
4. Kumar R. Vitamin D and calcium transport. *Kidney Int*. 1991; 40: 1177–1189.
5. Holick MF. Resurrection of vitamin D deficiency and rickets. *J Clin Invest*. 2006; 116: 2062–2072.
6. Bhan A, Rao AD, Rao DS. Osteomalacia as a result of vitamin D deficiency. *Endocrinol Metab Clin North Am*. 2010; 39: 321–331.
7. Dawson-Hughes B, Harris SS, Krall EA, Dallal GE. Effect of calcium and vitamin D supplementation on bone density in men and women 65 years of age or older. *N Engl J Med*. 1997; 337: 670–676.
8. Bell TD, Demay MB, Burnett-Bowie SM. The biology and pathology of vitamin D control in bone. *J Cell Biochem*. 2010; 111: 7–13.
9. Nair R, Maseeh A. Vitamin D: The “sunshine” vitamin. *J Pharmacol Pharmacother*. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3356951/> 2012; 3(2): 118–126.
10. Institute of Medicine (US) Committee to Review Dietary Reference Intakes for Vitamin D and Calcium; Ross AC, Taylor CL, Yaktine AL, et al., editors. *Dietary Reference Intakes for Calcium and Vitamin D*. Washington (DC): National Academies Press (US); 2011. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK56070/doi/10.17226/13050>.
11. Heaney RP. Functional indices of vitamin D status and ramifications of vitamin D deficiency. *Am J Clin Nutr*. 2004; 80(6): 1706S–1709S.

12. Malabanan A, Veronikis IE, Holick MF. Redefining vitamin D insufficiency. *Lancet*. 1998; 351: 805–806.
13. Heaney RP, Dowell MS, Hale CA, Bendich A. Calcium absorption varies within the reference range for serum 25-hydroxyvitamin D. *J Am Coll Nutr*. 2003; 22: 142–146.
14. Hansen KE, Jones AN, Lindstrom MJ, Davis LA, Engelke JA, Shafer MM. Vitamin D insufficiency: Disease or no disease? *J Bone Miner Res*. 2008; 23: 1052–1060.
15. Bischoff-Ferrari HA, Can U, Staehelin HB, Platz A, Henschkowski J, Michel BA, et al. Severe vitamin D deficiency in Swiss hip fracture patients. *Bone*. 2008; 42: 597–602.
16. Marwaha RK, Tandon N, Reddy DR, Aggarwal R, Singh R, Sawhney RC, et al. Vitamin D and bone mineral density status of healthy schoolchildren in northern India. *Am J Clin Nutr*. 2005; 82: 477–482.
17. Thacher TD, Fischer PR, Strand MA, Pettifor JM. Nutritional rickets around the world: Causes and future directions. *Ann Trop Paediatr*. 2006; 26: 1–16.
18. Khan AH, Naureen G, Iqbal R, Dar FJ. Assessing the effect of dietary calcium intake and 25 OHD status on bone turnover in women in Pakistan. *Arch Osteoporos*. 2013; 8(1–2):151. doi: 10.1007/s11657-013-0151-2. Epub 2013 Nov 6.
19. Junaid K, Rehman A, Jolliffe DA, Wood K, Martineau AR. High prevalence of vitamin D deficiency among women of child-bearing age in Lahore Pakistan, associating with lack of sun exposure and illiteracy. *BMC Women's Health*. 2015;15:83. Published online 2015 Oct 12. doi: 10.1186/s12905-015-0242-x
20. Gamboa GT, Soto GA, Jimenez-Montero JG. Prevalence of 25-hydroxyvitamin D deficiency in healthy personnel from an academic institution of an urban area in Costa Rica. *Research and Reports in Endocrine Disorders*. 2015; 5: 135–140.
21. Islam MZ, Lamberg CA, Karkkainen M, Outila T, Salamatullah Q, Shamim AA. Vitamin D deficiency: a concern in premenopausal Bangladeshi women of two socio-economic groups in rural and urban region. *Eur J Clin Nutr*. 2002; 56: 51–56.
22. Yetley EA. Assessing the vitamin D status of the US population. *Am J Clin Nutr*. 2008; 88: 558S–564S.
23. Looker AC, Pfeiffer CM, Lacher DA, Schleicher RL, Picciano MF, Yetley EA. Serum 25-hydroxyvitamin D status of the US population: 1988–1994 compared with 2000–2004. *Am J Clin Nutr*. 2008; 88: 1519–1527.
24. Lubna MZ, Aysha H, Naeemul H, Abdul J. Vitamin D Deficiency in Ambulatory patients: Journal of the Pakistan Medical Association. 2008;58(9): 482–484.
25. Micka A. Vitamin D Status among Bangladeshi Women of Reproductive Age. Master's Thesis, February 2014. University of Massachusetts Amherst.
26. Shivane VK, Sarathi, Anurag RI, Tushar B, Shashank RJ, Padmavathy SM, et al. Peak bone mineral density and its determinants in an Asian Indian Population. *J OCD*. 2012; 15(2): 152–158.

Bacteriological, Cytological and Biochemical Profile of Ascitic Fluid in Children with Chronic Liver Disease

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ABSTRACT

Introduction: Ascitic fluid bacterial infection is a fatal condition in children, especially with chronic liver disease (CLD). Culture negative neutrocytic ascites is the commonest type of infection in children with CLD. **Objective:** To observe bacteriological, cytological and biochemical profile of ascitic fluid in Children with CLD. **Methods:** Thirty five consecutive children with clinical features suggestive of CLD, aged between 2-14 years of both sexes with clinically detected ascites and admitted at the Department of Paediatric Gastroenterology and Nutrition, Bangabandhu Sheikh Mujib Medical University (BSMMU) from January 2013 to June 2013 were enrolled in this cross-sectional study. Children who had features of ascitic fluid infection like fever, abdominal pain with tenderness were categorized as symptomatic and children who had no features of ascitic fluid infection were categorized as asymptomatic children. Demographic, clinical, haematological, biochemical and ascitic fluid study were done for both symptomatic and asymptomatic children. Variants of ascitic fluid bacterial infection was identified by ascitic fluid polymorphonuclear leucocyte count and culture report. **Results:** Among 35 children, majority 17 (48.6%) were between 6-10 years of age group with male predominance. About 12 (34.3%) were symptomatic and among the symptomatic children 7 (58.4%) were infected and 5 (41.6%) were non-infected, 16 (45.7%) had culture negative neutrocytic ascites (CNNA) variant of ascitic fluid bacterial infection as evident by ascitic fluid polymorphonuclear leucocyte count of $\geq 250/\text{mm}^3$ and negative culture report. Mean ascitic fluid polymorphonuclear neutrophil count was $515 \pm 177.82/\text{HPF}$ among infected children and $85.47 \pm 70.60/\text{HPF}$ among non-infected children, which is statistically significant ($p=0.001$). **Conclusion:** Culture negative neutrocytic ascites (CNNA) variety of ascitic fluid infection was the only variety in this study.

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INTRODUCTION

Chronic liver disease (CLD) is not uncommon among paediatric population in Bangladesh. It was found that 4% of hospitalized children in the Department of General Paediatrics and Paediatric Gastroenterology and Nutrition of Bangabandhu Sheikh Mujib Medical University (BSMMU) were due to liver disease and among them 40% had CLD.¹ Infections in children with CLD are an important cause of morbidity and mortality. Mechanism of increased susceptibility to infection is unclear.² Common bacterial infections in children with CLD are spontaneous bacterial peritonitis (SBP), urinary tract infection (UTI), community acquired pneumonia, dermatologic infections and bacteremia.³ For the purpose of diagnosis and classification of ascitic fluid infection, culture of the ascitic fluid is essential.⁴ Ascitic fluid bacteriological culture is negative in approximately 40 % of adult patients with clinical manifestation suggestive of spontaneous bacterial peritonitis.⁵ Ascitic fluid infection is classified in to five categories (including three spontaneous categories) based on ascitic fluid culture results, polymorphonuclear leucocyte counts (PMN), and the presence or absence of a surgical source of infection.⁴ Among five variants, SBP and culture negative neutrocytic ascites (CNNA) are common ascitic fluid infections. Prompt detection of ascitic fluid infection is helpful to use appropriate antibiotics for treatment of ascitic fluid infection. Ascitic fluid bacterial infection of cirrhotic patients may be symptomatic in 30% of cases.⁶ Symptomatic ascitic fluid bacterial infection means patients who have fever, abdominal pain, abdominal tenderness either singly or in combination in patients of liver cirrhosis. Asymptomatic ascitic fluid bacterial infection means patients who have none of these symptoms and signs.⁷ Positive ascitic fluid culture for bacteria was considered essential to establish the diagnosis of Spontaneous Bacterial Peritonitis (SBP). However relying on ascitic fluid culture for diagnosis of SBP has the disadvantages of poor sensitivity and relatively long time before the results are known. To circumvent problem with culture, the ascitic fluid white blood cell (WBC) and Polymorph-

onuclear leucocyte (PMN) counts have become the standards for making a diagnosis of SBP.⁸ The aim of this study was to determine the variants of ascitic fluid bacterial infections in children with chronic liver disease.

METHODS

This present cross-sectional study was carried out enrolling 35 children, clinically suggestive of Chronic Liver Disease (CLD) with ascites in the Department of Paediatric Gastroenterology and Nutrition during the period of January, 2013 to June, 2013. Children who had features of ascitic fluid infection like fever, abdominal pain with tenderness were categorized as symptomatic and children without features of ascitic fluid infection were categorized as asymptomatic. Clinical and laboratory data of both symptomatic and asymptomatic children were recorded. Complete blood count, serum albumin, ALT, AST, Bilirubin, serum Creatinine, Prothombin time, HBsAg (ELISA), anti-HCV (ELISA), Anti LKM1 antibody (ELISA), Liver biopsy, Ultrasonography (USG) and endoscopic findings were recorded whenever necessary for selected children. Ascitic fluid was aspirated with all aseptic precautions. Thirty milliliter of ascitic fluid was collected with an 18 gauge sterile needle attached to a 50 cc disposable syringe. Ascitic fluid culture was done by conventional culture method using Tryptic Soya Broth. Ten milliliter ascitic fluid was used for culture. Five ml of ascitic fluid was sent for total WBC count, differential count and absolute neutrophil count (processed by auto analyzer and finally rechecked manually). Five milliliter ascitic fluid was sent for estimation of total protein and albumin. Demographic, clinical, haematological, biochemical and ascitic fluid study (cytology, Gram's and Ziehl Neelsen staining, total protein, albumin and culture) were done for both symptomatic and asymptomatic children. Variants of ascitic fluid bacterial infection were identified by ascitic fluid neutrophil count and culture report. Culture Negative Neutrocytic Ascites (CNNA) variety ascitic fluid infection was diagnosed by the presence of ascitic fluid polymorphonuclear leucocyte count of $\geq 250/\text{mm}^3$ and negative culture report.⁴ Presentation difference of both infected and non infected children were found on the basis of difference of

presenting symptoms, signs, haematological data and ascitic fluid total WBC count, neutrophil count, ascitic fluid albumin and total protein level.

RESULTS

It was observed that the age range of the children were from 2 years to 14 years and mean age was 7.39 ± 3.0 years. The highest 48.6% (17) incidence

of CLD was found in the age group of 6-10 years. A male predominance was observed in the study. Male were 54.3% (19) and female 45.7% (16). Out of 35 children, 12 (34.3%) were symptomatic i.e. they had features of ascitic fluid infection like fever, abdominal tenderness, and 23 (65.7%) asymptomatic (Table I).

Table I: Age and gender distribution of the studied subjects (n-35)

Age (in years)	Gender		Total (%)
	Male	Female	
2-5	07 (36.84%)	05 (31.25%)	12 (34.3%)
6-10	08 (42.1%)	09 (56.25%)	17 (48.6%)
11 and above	04 (21.05%)	02 (12.5%)	06 (17.1%)
Total	19 (54.3%)	16 (45.7%)	35 (100%)

Table II shows the difference of ascitic fluid infection in symptomatic and asymptomatic children. Out of a total 12 symptomatic children, 7 (58.4%) were infected and 5 (41.6%) were non-infected. Out of a total 23 asymptomatic children,

9 (39.1%) were infected and 14 (60.9%) were non-infected, but this difference is not statistically significant ($p > 0.05$). So, there is no association between ascitic fluid infection and clinical symptoms.

Table II: Ascitic fluid bacterial infection in symptomatic and asymptomatic children (n-35)

Group	Symptomatic (n-12) (34.3%)		Asymptomatic (n-23) (67.7%)		χ^2 -value	df	p value
	N	%	N	%			
Infected	7	58.4	9	39.1	1.15	1	0.279
Non-infected	5	41.6	14	60.9			
Total	12	100	23	100			

Table III shows range of ascitic fluid (AF) total WBC count of infected and non-infected children was 312-1200/mm³ and 44-312/mm³ respectively. Mean AF total WBC count of infected and non-infected children were 641.62 ± 231.99 /mm³ and 146.05 ± 115.6 /mm³ respectively. Range of ascitic fluid absolute neutrophil count

of infected and non infected children were 255-960/ mm³ and 10-243/ mm³ respectively and mean ascitic fluid absolute neutrophil count of infected and non-infected children were 515 ± 177.82 /mm³ and 85.47 ± 70.6 /mm³ respectively. Mean difference in cell count were statistically significant.

Table III: Cytological profile of ascitic fluid (n-35)

Group	Neutrophil count/mm ³ (Mean \pm SD) (Range/mm ³)	WBC count /mm ³ (Mean \pm SD) (Range/mm ³)
Infected group (n-16)	515 ± 177.82 (255-960)	641.62 ± 231.99 (312-1200)
Non infected (n-19)	85.47 ± 70.60 (10-243)	146.05 ± 115.60 (44-312)
p value (T-value, df)	0.001* (9.68, 33)	0.001* (3.84, 33)

*Significant

Biochemical profile (Table IV) revealed range of ascitic fluid total protein (AFTP) of infected and non- infected children was 0.7-2.8 gm/dl and 0.5-2.5 gm/dl respectively. Mean AFTP of infected 1.21±0.63 gm/dl and non infected 1.07±0.64 gm/dl, which was statistically not significant (p -

0.05). Range of ascitic albumin of infected and non infected children was 0.2-1.4 gm/dl and 0.2-0.8 gm/dl respectively. Mean ascitic fluid albumin of infected and non-infected children were 0.56±0.35 gm/dl and 0.43±0.19 gm/dl respectively which was also not significant ($p>0.05$).

Table IV: Biochemical profile of ascetic fluid (n-35)

Group	AFTP (gm/dl) (Mean ±SD)(Range)	AF Albumin (gm/dl) (Mean ±SD)(Range)
Infected group (n=16)	1.21±0.63 (0.7-2.8)	0.56±0.35 (0.2-1.4)
Non-infected (n=19)	1.07±0.64 (0.5-2.5)	0.43±0.19 (0.2-0.8)
p value (T-value, df)	0.51, (0.65, 33)	0.18 (1.35, 33)

Regarding ascitic fluid culture, among 35 studied children none had culture positive ascitic fluid bacterial infection, though 16 children with Culture Negative Neutrocytic (CNNA) variants had ascitic fluid bacterial infection evident by ascitic

fluid neutrophil count of ≥ 250 cells/mm³ and negative culture report. In the present study, 16 (45.7%) children had one type of ascitic fluid bacterial infection which is CNNA. Other children had no ascitic fluid bacterial infection (Table V).

Table V: Different variants of ascitic fluid bacterial infection in studied children (n-35)

Type of ascitic fluid bacterial infections	Number	%
Spontaneous Bacterial Peritonitis (SBP)	0	00
Culture-Negative Neutrocytic Ascites (CNNA)	16	45.7
Secondary Bacterial Peritonitis	0	00
Monomicrobial Non-neutrocytic (MNB) Bactericides	0	00
Polymicrobial bacterascites	0	00

DISCUSSION

In this study, most of the children were <10 years of age and the highest incidence of Chronic Liver Disease (CLD) with ascites was found in the age group of 6-10 years. The age range of the studied children was 2-14 years and the mean age was 7.39±3.0 years. In this study, male were 19 (54.3%) and female 16 (45.7%). This male predominance was also observed in another study done by Sarker⁹ and Hossen¹⁰ which was similar to the present study. Ascites polymorphonuclear cells increase with peritoneal infection or with other intra-abdominal inflammatory conditions such as diverticulitis, cholecystitis. In the present study, among the thirty five studied children, 16 children had ascitic fluid

neutrophil count of ≥ 250 cells/mm³ which was a diagnostic parameter of Culture Negative Neutrocytic Ascites (CNNA) type of ascitic fluid infection. In a study⁹ in adult patient with Chronic liver disease (CLD) with ascites showed that, mean ascitic fluid absolute neutrophil count was 704.50±480.44 /mm³ among infected and 56.89±45.26/mm³ among non infected patients. Hossen¹⁰ showed in another study in children that median ascitic fluid neutrophil count was 720/mm³, with a range of 360-3600/mm³ among infected children and median ascitic fluid neutrophil count was 30/mm³ with a range of 3-192/mm³ among non-infected children which is statistically significant ($p=0.001$). In the present study, mean ascitic fluid neutrophil count was 515±177.82/mm³ among infected children and

mean ascitic fluid neutrophil count among non-infected children was $85.47 \pm 70.60/\text{cmm}^3$ which was statistically significant ($p=0.001$). So, the findings of the present study in children were similar to findings of the other studies.

In sterile ascites, ascitic fluid white blood cell count is usually less than $100/\text{mm}^3$ with a predominance of mononuclear cells and a low number of polymorphonuclear cells.¹¹ In another study done by Hoseen¹⁰ median ascitic fluid WBC counts among infected children was $1200/\text{mm}^3$ with a range of $600-10,000/\text{mm}^3$ and among non-infected children it was $100/\text{mm}^3$ with a range of $20-600/\text{mm}^3$ which is statistically significant ($p=0.001$). In another study¹⁰ in adult patient with CLD with ascites showed that mean ascitic fluid WBC count among infected patients was $2560 \pm 1871.91/\text{mm}^3$ and mean ascitic fluid WBC count among non-infected patients was $181.85 \pm 105.30/\text{mm}^3$. In the present study, mean ascitic fluid WBC count among infected children was $641.62 \pm 231.99/\text{mm}^3$ and among non-infected children it was $146.05 \pm 115.60/\text{mm}^3$ which was also statistically significant ($p=0.001$).

Patients with ascitic fluid total protein $< 1\text{gm/dl}$ were the most prone to develop ascitic fluid infection and the opsonic activity of the ascitic fluid was proportional to the ascitic fluid protein concentration.¹² In a study done by Hossen,¹⁰ mean ascitic fluid total protein was $0.36 \pm 0.23\text{gm/dl}$ and $1.28 \pm 1.13\text{gm/dl}$ among infected and non-infected children respectively ($p=0.087$). Another study⁸ in 35 adult patient with CLD with ascites showed that mean ascitic fluid total protein was $1.53 \pm 0.61\text{ g/dl}$ among infected and $1.20 \pm 0.59\text{ g/dl}$ among non-infected patients. In this study, mean ascitic fluid total protein was $1.21 \pm 0.63\text{gm/dl}$ and $1.07 \pm 0.64\text{ gm/dl}$ among infected (CNNA) and non-infected children respectively. So, the mean value of ascitic fluid total protein of infected children of this present study is not similar with the previous study,⁹ may be due to variation of age of the study populations of these two studies, though the values of the both studies are not statistically significant ($p>0.05$). Hossen¹⁰ showed in another study in children that culture negative neutrocytic ascites (CNNA) is 16.67% In fact, the

sensitivity of culture in detecting bacterial growth in neutrocytic ascites (i.e., ascitic fluid with a PMN count greater than or equal to $250\text{ cells}/\text{mm}^3$) varies widely depending on the method of culture used. In published studies, the conventional method of culture has been found to detect bacterial growth in approximately 50% of neutrocytic sample.⁴ In a recent study⁹ in adult patient of CLD with ascites, showed that out of a total 35 patients ascitic fluid culture in conventional method showed no growth of organism, though in that study out of a total 35 patients 8 (22.8%) had PMN count of $\geq 250/\text{mm}^3$. In the present study in children, out of a total 35 children, ascitic fluid culture result was found negative in all children, though 16 (45.7%) children had neutrocytic ascites (PMN count $\geq 250/\text{mm}^3$). So, this study was almost similar to the study done in adult patient⁹ Gene probes are now commercially available for the detection of bacteraemia; hopefully, they will lead to rapid (30 minute) and accurate detection of organisms in ascitic fluid.¹⁴ In other study¹⁵ in children showed that *Streptococcus pneumoniae* was isolated in 9 of 12 children suffering from cirrhosis with ascites. Another study¹⁸ in adult patients showed that ascitic fluid culture by conventional methods was positive in 46% cases and all the culture positive cases had Gram-negative bacilli: *Escherich coli* being the most common microorganism. Ascitic fluid culture was found negative in the present study; the reason may be that the media used was not enriched enough or due to neutrophil mediated killing of bacteria.¹³ On the basis of our facility only aerobic culture was done, though anaerobes can cause ascitic fluid infection rarely (1%). Most episodes of CNNA are diagnosed using insensible cultured methods, where there are insufficient numbers of bacteria to reach the threshold of detectability.¹⁷ The conventional method of culture probably requires at least 100 organisms/ml.¹³ However, even when optimal culture methods are used, a small percentage of patients grow no bacteria in their neutrocytic ascitic fluid.¹⁸ In a study⁸ in adult patients showed that out of a total 35 patients of cirrhosis with ascites 8 (22.8%) had CNNA. In the present study, among 35 children, 12 (34.3%)

children were symptomatic, i.e. they had features of ascitic fluid bacterial infection like fever, abdominal pain or tenderness and 23 (65.7%) asymptomatic, i.e. they had no features of ascitic fluid bacterial infection like fever, abdominal pain or tenderness. Out of a total 12 symptomatic children, 7 (58.4%) were infected and 5 (41.6%) were non-infected. Out of a total 23 asymptomatic children, 9 (39.1%) were infected and 14 (60.9 %) were non-infected, but this difference is not statistically significant ($p>0.05$). The reason of presence of features of ascitic fluid infection in non infected children may be presence of infection other than ascitic fluid infection like UTI or pneumonia etc. The reason of absence of symptoms of infection among the infected children may be due to immunosuppression.

Limitations

The limitations of the present study were small sample size, only aerobic cultivation, and nutrient enriched media was used. Other causes of fever i.e. urinary tract infection were not excluded, so, some non-infected cases were found to be symptomatic. Many of the infected cases were asymptomatic due to immuno-suppression. This is a hospital based single centre study. Further studies with larger sample size are necessary to know the facts about the bacterial infection of ascitic fluid in children with chronic liver disease.

CONCLUSION

Culture negative neutrocytic ascites variety of ascitic fluid infection was the most common variety in this current study. Polymorphonuclear neutrophil (PMN) cell count was found significantly higher in this group of children. Many of the infected cases were asymptomatic. Clinical features of ascitic fluid infection are needed to differentiate the infected and non infected cases.

Conflicts of Interest: None declared.

REFERENCES

1. Karim ASMB, Akter S, Karim MA, Nazir NFH. A study of the clinical profile of chronic liver disease in children. *D S (Child) H J*.1999; 15: 16-25.

2. Taneja SK, Dhiman RK. Prevention and Management of bacterial infections in Cirrhosis. *Int J Hepatol*. 2011; 784540.
3. Cheruvattath R, Balan V. Infections in patients with end-stage liver disease. *J Clin Gastroenterol*. 2007; 41: 403-411.
4. Runyon BA. Ascites and Spontaneous Bacterial Peritonitis. In: Sleisenger MH, Feldman M, Friedman LS and Brandit L J. eds. *Sleisenger and Fordman's Gastrointestinal and Liver Disease, Pathophysiology/ Diagnosis/ Management*. 9th ed. Philadelphia: Saunders. 2010; p. 1935-1960.
5. Rimola A, Garcia-Tsao G, Navasa M. Diagnosis, treatment and prophylaxis of spontaneous bacterial peritonitis. A consensus document. *J Hepatol*. 2000; 32: 142-153.
6. Guarner C, Soriano G. Spontaneous Bacterial Peritonitis. *Hepatology*. 1997; 1: 203-217.
7. Evans LT, KIM WR, Oterucha JJ, Kamnath S. Spontaneous Bacterial Peritonitis in asymptomatic out patients with Cirrhotic Ascites. *Hepatology*. 2003; 37: 897-901.
8. Thomas, Boyer. Diagnosis and management of cirrhotic ascites. In: Zakim D, Boyer TD. (eds). *Hepatology. A text book of liver disease* 5thed. Philadelphia: Saunders.2003; p.639.
9. Sarker JA. Variants of ascitic fluid bacterial infection in patients of cirrhosis. MD Thesis. Department of Hepatology. BSMMU, Dhaka Bangladesh, 2008.
10. Hossen K, Rukunuzzaman M, Alam R, Benzamin M, Yasmin A, Thakur SB, et al. Study of Ascitic Fluid in Children with Chronic Liver Disease in Different Variants of Peritonitis at a Tertiary Care Hospital. *Bangladesh. Sch J App Med Sci*. 2019; 4: 410.
11. Garcia-Tsao G. Ascites', *Sherlok's Diseases of the Liver and Biliary System*, 12th ed., UK: Willey- Blackwell, Oxford. 2011; p. 210.
12. Runyon BA. Low-protein concentration ascitic fluid is predisposed to spontaneous bacterial peritonitis. *Gastroenterology*. 1986; 91: 1343-1346.

13. Runyon BA. Patients with deficient opsonic activity are predisposed to SBP. *Hepatology*. 1988; 8: 632-635.
14. Davis TE, Fuller DD. Direct identification of bacterial isolates in blood cultures using a DNA probe. *J Clin Microbiol*. 1991; 29(10): 2193-2196.
15. Larcher VF, Manolaki N, Vegnente A, Mowat AP. Spontaneous bacterial peritonitis in children with chronic liver disease: clinical features and etiologic factors. *J Pediatr*. 1985; 106: 907-912.
16. Khalaf AS, AL Myahi MH. Primary ascitic fluid infection in patients with chronic liver diseases. *Saudi J Gastroenterol*. 2001; 7: 62-70.
17. Runyon BA. Monomicrobial non-neutrocytic bacterascites: a variant of spontaneous bacterial peritonitis. *Hepatology*. 1990; 12: 710-715.
18. Runyon BA, Hofes JC. Culture negative neutrocytic ascites; a variant of spontaneous bacterial peritonitis. *Hepatology*. 1984; 4: 1209-1211.

Clinico-Cytological Pattern of Cervical Lymphadenopathy

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ABSTRACT

Introduction: Cervical Lymphadenopathy is one of the commonest causes of head and neck swellings with aetiology varying from benign to malignant conditions. In fact it is also essential to establish that the swelling is a lymph node or not. Fine needle aspiration cytology (FNAC) plays a vital role in solving these issues. FNAC has emerged as an easy, quick cost-effective, minimally invasive and safe technique, with high sensitivity and specificity in the evaluation of cervical lymphadenopathy. **Objective:** To determine the clinico-cytological pattern of various lymph node diseases present in cervical region. **Methods:** The two years cross sectional study was conducted in the Department of Pathology, North Bengal Medical College, Sirajganj, Bangladesh. Two hundred and ninety (290) neck swelling patients obtained during the period of study. Cytological reports were done according to standard guidelines and the diagnosis was classified and correlated with patient's age and sex to explore the pattern. **Results:** Of a total of 290 cases of FNAC performed on neck nodes, the most frequent cause of lymphadenopathy was found to be tuberculosis with 94 cases (32.41%), followed by reactive lymphoid hyperplasia (31.38%), acute suppurative lymphadenitis (16.55%), necrotizing lymphadenitis (11.03%), Metastatic carcinoma (6.55%), lymphoma (1.03%) and inconclusive (1.03%). In first decade, predominant cause of lymphadenopathy was reactive in nature; in second decade, tuberculosis starts predominating than the other causes. After 6th decade, metastatic lymph nodes overshadowed the tuberculosis and reactive lymphadenopathy. **Conclusion:** In this study, predominant cause of cervical lymphadenopathy was tuberculosis followed by reactive lymphadenitis. The relationship of malignant, tubercular and reactive lymphadenopathy with age deserves further study. Fine needle aspiration cytology is a useful first-line investigating tool for diagnosis of cervical lymphadenopathy.

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INTRODUCTION

Cervical lymphadenopathy is one of the commonest clinical problem presenting not only to head neck surgeons, but also to general surgeons and physicians. Aetiology may

vary from simple inflammation to malignancies and tuberculosis- sometimes it may be non-specific.¹ Diagnosing the cause of these enlarged lymph nodes has always been challenge for the doctors. Fine needle aspiration cytology (FNAC)

helps solving the cause of enlarged lymph nodes. It is a tool to obtain material from a swelling for cytological examination, performed as an outpatient procedure. It has potential benefits over the other diagnostic modalities that have increased the utility of FNAC in recent years. It is simple, cost-effective procedure that is minimally invasive with almost no complications. Results obtained by FNAC are quick as compared to histopathological diagnosis. FNAC of superficial lesions need no anesthesia eliminating the risk of complications associated with anesthesia. No scar is formed at the site of FNAC. Other diagnostic modalities like incision biopsy or excision biopsy leave a scar. In case of suspected malignancy, FNAC is the best choice as it does not cause spread of tumor through the skin tract. The sensitivity of FNAC for the diagnosis of lymphadenopathy averages 90% with a specificity of 95%.² FNAC has been recommended as of first line screening method in suspected metastatic malignancy.³ The high degree of accuracy, low costs and minimally disruptive nature of the procedure makes FNAC a highly desirable alternative to open biopsy for investigation of cervical lymphadenopathy.

In this study we have analyzed FNAC of neck lymph nodes and studied the clinical and cytological patterns of enlarged neck nodes and diagnostic utility of the procedure.

METHODS

The two years cross-sectional study was conducted in the Department of Pathology, North Bengal Medical College, Sirajganj, Bangladesh from 1st July, 2016 to 31st June, 2018. Two hundred and ninety (290) neck swelling patients enrolled during the period of study, registered from different regions of Sirajganj district. Neck swellings other than lymph nodes were excluded from the study. FNAC smears were stained with Papanicolaou staining (Pap). All the clinical details including age, sex, site, size, consistency and other relevant clinical investigations were recorded. Slides were reviewed and the cases with equivocal results or inadequate material were also excluded from the study. The slides were examined for cytomorphological details and diagnosis was reviewed. The patient was informed about the procedure and informed written consent was obtained from the patient before subjecting to FNAC. Data was analyzed using SPSS 17.

RESULTS

Total number of patients was 290, who underwent FNAC for enlarged cervical lymph nodes. Demographic profile is shown in Table I and Table II. Males constituted 38.62% (112 cases) of cases whereas females constituted 61.38% (178 cases) of cases with a male to female ratio of 1:1.59. The age ranged from 1 year to 100 years with a mean age of 31.27years.

Table I. Sex distribution of patients

Particulars	Number of patients	Percentage (%)
Male	112	38.62
Female	178	61.38
Total	290	100.00

Table II. Age distribution of patients

Age group (in years)	Number of patients	Percentage (%)
0-10	37	12.76
11-20	77	26.55
21-30	74	25.51
31-40	38	13.10
41-50	29	10.00
51-60	16	5.52
61-70	14	4.82
71 and more	5	1.72
Total	290	100.00

The most common overall diagnosis was tuberculous lymphadenitis (Figure 1), constituting 32.41% (94 cases) of the cases, whereas the second most common diagnosis was nonspecific reactive lymphadenitis (Figure 2) constituting 31.38% (91 cases), acute suppurative lymphadenitis constituted 16.55% (48 cases) and necrotizing lymphadenitis constituted 11.03% of cases (32 cases) (Table III). Of the malignant diagnosis,

metastatic tumours constituted 6.55% (19 cases) and lymphomas constituted 1.03% (3 cases). Among the metastatic tumours, squamous cell carcinomas constituted 3.45% (10 cases) and adenocarcinomas constituted 3.10% (9 cases). Among lymphomas, all cases are Non Hodgkin Lymphomas. Although overall inconclusive, atypical cells were seen in 1.03% of the cases.

Table III. Cytological pattern of cervical lymphadenopathy

Diagnosis	Number of cases	Percentage (%)
Tuberculosis	94	32.41
Reactive lymphadenitis	91	31.38
Acute/ suppurative lymphadenitis	48	16.55
Necrotizing lymphadenitis	32	11.03
Metastatic carcinoma	19	6.55
Lymphoma	3	1.03
Atypical lymphoid hyperplasia	3	1.03
Total	290	100.00

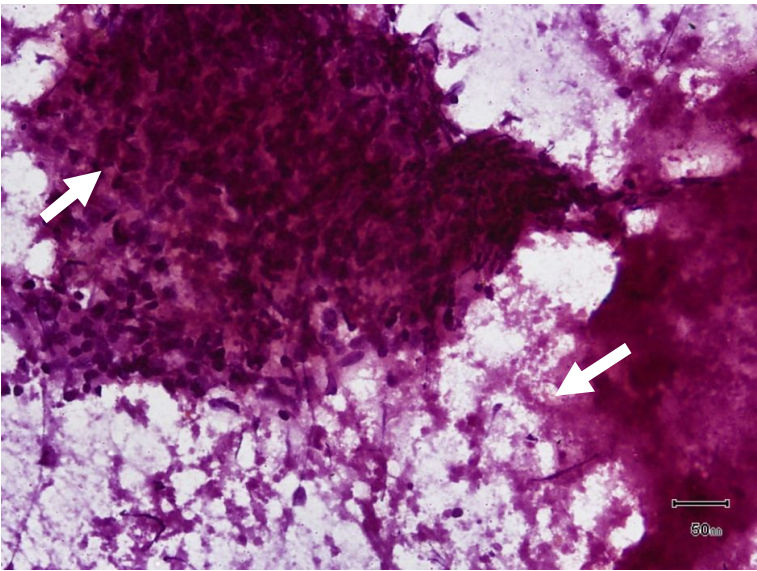


Figure1. Chronic Granulomatous Inflammation (Tuberculosis), featuring epithelioid histiocytes (arrows) with caseous necrosis (400x, Pap stain)

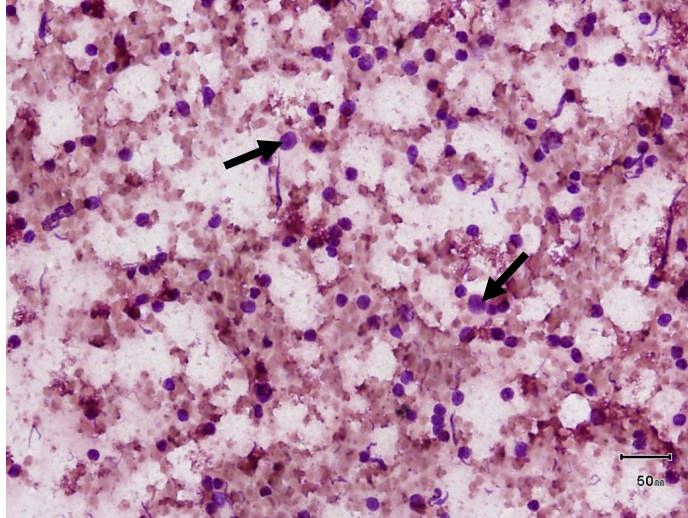


Figure 2: Non specific reactive Lymph node, photomicrograph showing polymorphous population of lymphocytes with predominance of mature lymphocytes (arrows) (400x, Pap stain)

The results were stratified into 10 years age interval from 0 to 70 years and then 71 years and more. In the first ten years, predominant cause of lymphadenopathy was reactive in nature accounting for 48.50% of the cases in this group. The next common cause was tuberculous lymphadenopathy (20.70%). The second decade was the age group when tuberculosis starts predominating than the other causes, approximately 63.50% the total cases in this group. Reactive lymph nodes being the second

common cause (23.30%). In patients after 10 years up to the age of 50 years, tuberculosis is over passing all other causes. After 50 years, metastatic lymph nodes overshadowed the tuberculosis and reactive lymphadenopathy. Reactive lymphadenopathy, was a smaller group in this age and was no longer seen in age groups after 60 years. The common metastatic tumors were squamous cell carcinoma. Cytological findings were recorded (Figures 1, 2 and 3).

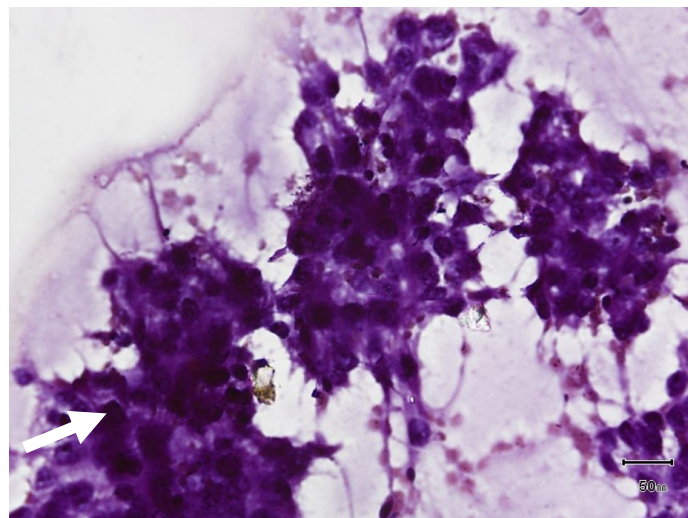


Figure 3. Metastatic Adenocarcinoma, featuring malignant epithelial cells (arrow) forming glandular pattern in a lymphoid background (400x, Pap stain)

DISCUSSION

Fine needle aspiration cytology (FNAC) has been important tool to make the diagnosis in cervical

lymphadenopathy. This study shows tuberculosis is the commonest cause (32.41%) of cervical lymphadenopathy. Many studies within India and

other developing countries similarly show tuberculosis as the commonest cause. Studies from Pakistan reported tuberculous lymphadenitis as the most common pathology. Two studies reported 36% and 52.7% tuberculosis in their study respectively.^{2,4} Kafi showed reactive lymphadenitis (50%) as the most common cause followed by tuberculosis (28.33%).⁵ A study from Saudi Arabia had reactive lymphadenitis as the most common cause followed by tuberculosis.⁶ Gupta et al. in 2003 showed 59% of granulomatouslymphadenopathy.⁷ Tuberculosis seen as the most common cause of granulomatous inflammation in South East Asia and in developing countries. Initial western studies did not report tuberculosis in their studies. But after the world wide increasing incidence of HIV infection, tuberculosis is being reported from western population as a significant cause of cervical lymphadenopathy.^{8,9} In our study, tuberculosis was more common in females (67%) as compared to the males (33%). This may be because of poor nutrition and overall health in developing countries.

In a study by Agarwal, the commonest cause of lymphadenopathy in pediatric age group was reactive hyperplasia (70.9%), while tuberculous lymphadenitis was the predominant cause in adolescents and middle aged patients (40.8%).¹⁰ In these studies, similar findings were observed when stratified into age groups. Metastatic carcinoma was the major cause of lymphadenopathy in patients above 60 years of age (66.3%). In this study 22 cases have been diagnosed as malignancy on FNAC in this study. Within this 19 metastatic malignancy, 9 cases are metastatic adenocarcinoma and 10 cases are metastatic squamous cell carcinoma.

CONCLUSION

Based on the finding in this study we found that FNAC is an extremely useful tool in the evaluation of palpable cervical lymph node. In majority of the cases it may excludes the need of a more invasive procedure and helps to initiate the appropriate treatment. FNAC is a reliable diagnostic tool in evaluation of lymphadenopathy for both screening and follow-up. Our experience suggests that FNAC combined with clinical correlation is useful as a first line investigation which can be performed in the outpatient department.

Conflict of Interest: None

REFERENCES

1. Batni G, Gaur S, Sinha ON, Agrawal SP, Srivasatva A. A Clinico-pathological study of cervical lymph nodes. *Indian J Otolaryngol Head Neck Surg.* 2016; 68(4): 508-510.
2. Ahmed T, Naeem M, Ahmad S, Samad A, Nasir A. Fine needle aspiration cytology and neck swellings in the surgical outpatient. *J Ayub Med Coll.* 2013; 20(3): 30-32.
3. Bhuiyan M, Fakir M, Hossain A, Huq A, Gupta S. Role of Fine needle aspiration cytology in the diagnosis of Cervical lymphadenopathy. *Bangladesh J Otorhinolaryngol.* 2008; 14(2): 63-65.
4. Fatima S, Arshad S, Ahmed Z, Hasan SH. Spectrum of Cytological Findings in Patients with Neck Lymphadenopathy - Experience in a Tertiary Care Hospital in Pakistan. *Asian Pacific J Cancer Prev.* 2011; 12: 1873-1875.
5. Kafi AH, Arif HB, Ruhul AH. Role of Fine needle aspiration cytology in the diagnosis of cervical lymphadenopathy. *Bangladesh J Med Sci.* 2012; 11(1): 25-27.
6. El Haq IA, Chiedozi LC, Al Reyees FA, Kollur SM. Fine needle aspiration cytology of head and neck masses. Seven years' experience in a secondary care hospital. *Acta Cytol.* 2003; 47: 387-392.
7. Gupta RK, Naran S, Lallu S, Fauk R. The diagnostic value of fine needle aspiration cytology (FNAC) in the assessment of palpable supraclavicular lymph nodes; a study of 218 cases. *Cytopathology.* 2003; 8: 511-514.
8. Schelkum PM, Grundy WG. Fine needle aspiration biopsy of head and neck lesions. *J Oral Maxillofac Surg.* 1991; 49: 262-267.
9. Cheng AT, Dorman B. Fine needle aspiration cytology: the Auckland experience. *Aust NZ J Surg.* 1992; 62: 368-372.
10. Agarwal D, Bansal P, Rani B, Sharma S, Chawla S, Bharat V, et al. Evaluation of etiology of lymphadenopathy in different age groups using fine needle aspiration cytology; a retrospective study. *Internet J Pathol.* 2009; Available at: <http://ispub.com/IJPA/10/2/7886>; Accessed on: 19.11.2019, 2019.

Hypothyroidism and Hyperprolactinemia among Sub-fertile Patients attending a clinic in Sirajganj

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ABSTRACT

Introduction: Thyroid dysfunctions are relatively common among women of reproductive age, and can affect fertility in various ways, resulting in anovulatory cycles, high prolactin levels, and sex hormone imbalances. On the other hand, excessive prolactin secretion causes reproductive dysfunction and sub-fertility by decreasing the pulsatile release of Gonadotrophin releasing hormone. So, measurement of TSH and PRL should be routinely done as a part of sub-fertility workup. **Methods:** This is cross-sectional analytic study done in a private clinic from September 2016 to August 2019. A total 359 patients from Sirajganj or neighbouring districts, having either primary or secondary sub-fertility, were enrolled in this study. As a part of sub-fertility workup, TSH and Prolactin level in serum of these patients were done by hormone analyzer. The results were analyzed by SPSS version 25. **Results:** Among 359 sub-fertile patients, maximum (44.0%) came from 15-25 years age groups. Mean age of the respondents was 23.23 ± 4.13 years. Out of them, 74.4% had primary sub-fertility and 25.6% had secondary sub-fertility. Majority (88.9%) of them were euthyroid (TSH 0.40-5mIU/L), rest (11.1%) were hypothyroid. Nobody in this study was hyperthyroid. On the contrary, majority (55.2%) of the patients were hyperprolactinemic, the rest had normal prolactin in their serum (1-20ng/L). Duration of marriage was 5.64 ± 3.61 years in primary and 8.05 ± 4.43 years in secondary sub-fertility. Out of 40 hypothyroid patients, 28(70%) had hyperprolactinemia, whereas, out of 319 euthyroid patients, 170(53.3%) had the same. Interestingly 28(7.8%) patients had both hypothyroidism and hyperprolactinemia. Mean TSH level was 2.75 ± 1.89 mIU/L in primary and 2.73 ± 2.10 mIU/L in secondary sub-fertility. Mean prolactin level was 29.25 ± 24.19 ng/L and 25.41 ± 22.80 ng/L in the respective variety of sub-fertility. A strong correlation between TSH and prolactin in the serum of sub-fertile patients was found in this study. **Conclusion:** Measurement of TSH and PRL should be done at early stage of infertility check up. This can avoid more costly tests or invasive procedures at the very beginning by giving medical treatment only.

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INTRODUCTION

Human sub-fertility is a complex problem, which has numerous consequences depending on the society and cultural background, gender, lifestyle, sexual history of the people it affects. Sub-fertility is a global public health concern, this is partly due to its complexity in aetiology as well as difficulty in preventing, diagnosing and treating it.¹ According to the International Committee for Monitoring Assisted Reproductive Technology (ICMART) and the World Health Organization (WHO), sub-fertility is a disease of the reproductive system defined by the failure to achieve a clinical pregnancy after 12 months or more of regular unprotected sexual intercourse.² The term primary sub-fertility is used for a couple who have never achieved a pregnancy despite cohabitation and regular sexual intercourse and secondary sub-fertility is used for a couple who had previously succeeded in achieving at least one pregnancy, even if it had ended in abortion. Worldwide around 8 to 12% of the couples experience some form of sub-fertility during their reproductive lives.³ This has led the problem of sub-fertility to be recognized as a public health issue.⁴ The cause of sub-fertility lies within the female in 45% of the couples, male factor sub-fertility in 30% and in the remaining 25% the cause is unexplained.⁵

The main causes of female sub-fertility include ovulatory disorders, pelvic inflammatory disease (PID), endometriosis, polycystic ovarian syndrome, and advanced age, environmental and occupational exposure to chemicals, congenital abnormalities and hormonal imbalance.⁶ Hypothyroidism is associated with a broad spectrum of reproductive disorders ranging from abnormal sexual development to menstrual irregularities and subfertility.⁷ Thyroid dysfunction reduces the chances of pregnancy and also adversely affects pregnancy outcome.⁸ Prevalence of hypothyroidism in the reproductive age group is 2–4% and has been shown to be the cause of sub-fertility and habitual abortion.^{9,10} Thyroid dysfunction can affect fertility in various

ways resulting in anovulatory cycles, luteal phase defect, high prolactin (PRL) levels, and sex hormone imbalances. Therefore, normal thyroid function is necessary for fertility, pregnancy, and to sustain a healthy pregnancy, even in the earliest days after conception. Thyroid evaluation should be done in any woman who wants to get pregnant with family history of thyroid problem or irregular menstrual cycle or had more than two miscarriages or is unable to conceive after 1 year of unprotected intercourse. Hypothyroidism can be easily detected by assessing TSH levels in the blood. Hyperprolactinemia, the presence of abnormally high levels of prolactin in the blood, is the most common endocrine disorder of the hypothalamic-pituitary axis with the prevalence ranging from 0.4% in an unselected normal adult population to as high as 9-17% in women with reproductive disorders.^{11,12}

Excessive prolactin secretion causes reproductive dysfunction and sub-fertility by decreasing the pulsatile release of Gonadotropin releasing hormone (GnRH). It impairs pituitary production of Follicle stimulating hormone (FSH) and Luteinizing hormone (LH) and causes other disorders like amenorrhea and galactorrhoea.^{13,14} It has been recommended that in the presence of raised PRL, the treatment should be first given to correct the hypothyroidism before evaluating other causes of raised PRL. Measurement of TSH and PRL is routinely done as a part of sub-fertility workup. As hypothalamic thyrotropin releasing hormone (TRH) increases the secretion of both TSH and prolactin, serum prolactin levels may be increased in cases of hypothyroidism.¹⁵ Hypothyroidism and hyperprolactinemia are found to be closely interrelated.¹⁶ This study intends to assess the status of thyroid and prolactin hormone levels and their relation in patients of sub-fertility.

METHODS

The study enrolled 359 female subjects who were suffering from primary and secondary sub-

fertility. The cases were selected in a private clinic over a period of three years. The inclusion criteria for the selection of cases were diagnosis of sub-fertility, age between 15-35 years and duration of marriage more than one year. The exclusion criteria that were adopted during case selection were male factor sub-fertility and amongst the female factors-tubal factor, any congenital anomaly of the urogenital tract, or any obvious organic lesion. Any history of thyroid disease or previous thyroid surgery or being on thyroid medications was also excluded from the study. The participants were enrolled after signing on informed consent. Two milliliters of fasting venous blood was obtained in the morning of day three of menstrual cycle for serum biochemical analysis. Serum was separated and stored for further analysis. All the hormones were estimated using an analyzer ELISA Microplate Reader Biogen-6500 (Made in Germany) and reagent Nova Tec^(R) was used for the purpose. The normal range of serum Prolactin and TSH were 1-20ng/ml and 0.4-5mmol/L, respectively. The following are the operational definition used in this study.

- I. Euthyroidism: TSH is within the normal range.
- II. Hypothyroidism if serum TSH is >5mIU/L.
- III. Hyperprolactinemia when serum Prolactin >20ng/ml.

Statistical analysis was done by using SPSS software, version 25. A *p*-value of <0.05 was considered statistically significant. Descriptive analysis, Chi-square test, unpaired student's *t* test and Pearson's correlation test were done for analysis of the results.

RESULTS

Among the total 359 enrolled women, highest no (158, 44%) of sub-fertility was found in 21-25 years age group. The mean age of the respondents was 23.23±4.13 years. Among them, 267(74.4%) patients were with primary sub-fertility and 92(25.6%) patients with secondary sub-fertility. Out of the sub-fertile patients, majority-(319, 88.9%) were euthyroid and 40(11.1%) were found to be hypothyroid. In contrast, a higher number of patients showed prolactin level more than normal. Out of 359 sub-fertile patients, 161 (44.8%) had normal prolactin and 198(55.2%) had hyperprolactinemia (Table I).

Table I: Distribution of the sub-fertile patients according to characteristic (n-359)

Variables	Frequency	Percentage
Age group (years)		
≤ 20(15-20)	117	32.6
21-25	158	44.0
26-30	67	18.7
31-35	17	4.7
Mean±SD (Range)	23.23±4.13(18-35) years	
Type of Sub-fertility		
Primary infertility	267	74.4
Secondary infertility	92	25.6
TSH status		
Hypothyroid (> 5.0 mIU/L)	40	11.1
Euthyroid (0.40- 5.0 mIU/L)	319	88.9
Prolactin status		
Normal (1-20ng/L)	161	44.8
Hyperprolactinemia (> 20 ng/L)	198	55.2

Mean duration of marriage in primary and secondary sub-fertility were 5.64±3.61years and 8.05±4.43 years respectively. Duration of marriage was significantly (*p*<0.001) associated with primary and secondary sub-fertility (Table II).

Table II: Duration of marriage and it's association between primary and secondary Sub-fertility (n-359)

Duration of Marriage (In years)	Sub-fertility		p-value
	Primary (n-267) No. (%)	Secondary (n-92) No. (%)	
2-4	124(46.4%)	17(18.5%)	
5-7	70(26.2%)	32(34.8%)	
8-10	46(17.2%)	24(26.1%)	
11-13	17(6.4%)	11(12.0%)	
>13	10(3.7%)	8(8.7%)	
Mean±SD	5.64±3.61	8.05±4.43	<0.001*

Unpaired student's t test, *Significant

Out of 40 hypothyroid patients 28(70%) had hyperprolactinemia and among 319 euthyroid patients 170(53.3%) had hyperprolactinemia. Hypothyroidism was significantly associated with hyperprolactinemia (Table III).

Table III: Distribution of sub-fertile patients according to hormonal status (n-359)

Prolactin	TSH (mIU/L)		p-value
	Hypothyroid(n-40) No.(%)	Euthyroid (n-319) No.(%)	
Normal	12(30.0%)	149(46.7%)	0.045*
Hyperprolactinemia	28(70.0%)	170(53.3%)	
Total	40 (100.0%)	319(100.0%)	

Chi-square test, *Significant

Chi square test showed that hypothyroidism was significantly associated with hyperprolactinemia ($p=0.045$). At same time, 28 patients had both hypothyroidism and hyperprolactinemia, whereas 331 patients had either of the two or none (Table IV).

Table IV: Frequency of both hypothyroid and hyperprolactinaemia of the study patients (n-359)

Both hypothyroid and hyperprolactinemia	Frequency	Percentage
Yes	28	7.8
No	331	92.2
Total	359	100.0

Of the 267 primary sub-fertile patients, mean TSH level was 2.75 ± 1.89 mmol/L and 92 secondary sub-fertile patients had mean TSH level of 2.73 ± 2.10 mmol/L (Table V).

Table V: TSH and prolactin status in sub-fertile patients

TSH status	Sub-fertility	
	Primary(n-267)	Secondary(n-92)
Hypothyroid(>5.0 mIU/L)	29(10.9%)	11(12.0%)
Euthyroid(0.40-5.0mIU/L)	238(89.1%)	81(88.0%)
Mean±SD	2.75±1.89	2.73±2.10
Prolactin status		
Hyperprolactinemia>20 ng/L	161(60.3%)	37(40.2%)
Normal(1-20 ng/L)	106(39.7%)	55(59.8%)
Mean±SD	29.25±24.19	25.41±22.80

On the contrary, the primary and secondary sub-fertile patients had mean prolactin level of 29.25 ± 24.19 ng/ml and 25.41 ± 22.80 ng/ml respectively. Pearson's correlation test was done between TSH and prolactin in two varieties of sub-fertility which showed highly significant relation (Table VI).

Table VI: Pearson's correlation of TSH with prolactin in primary and secondary sub-fertility (n-359)

Type of Infertility		TSH	
		r-value	p-value
Primary Sub fertility	Prolactin	+2.19	<0.001
Secondary Subfertility	Prolactin	+.285**	0.006

Scatter diagram in Figure 1 showed the trend of both hormones of increasing in both types of fertilities.

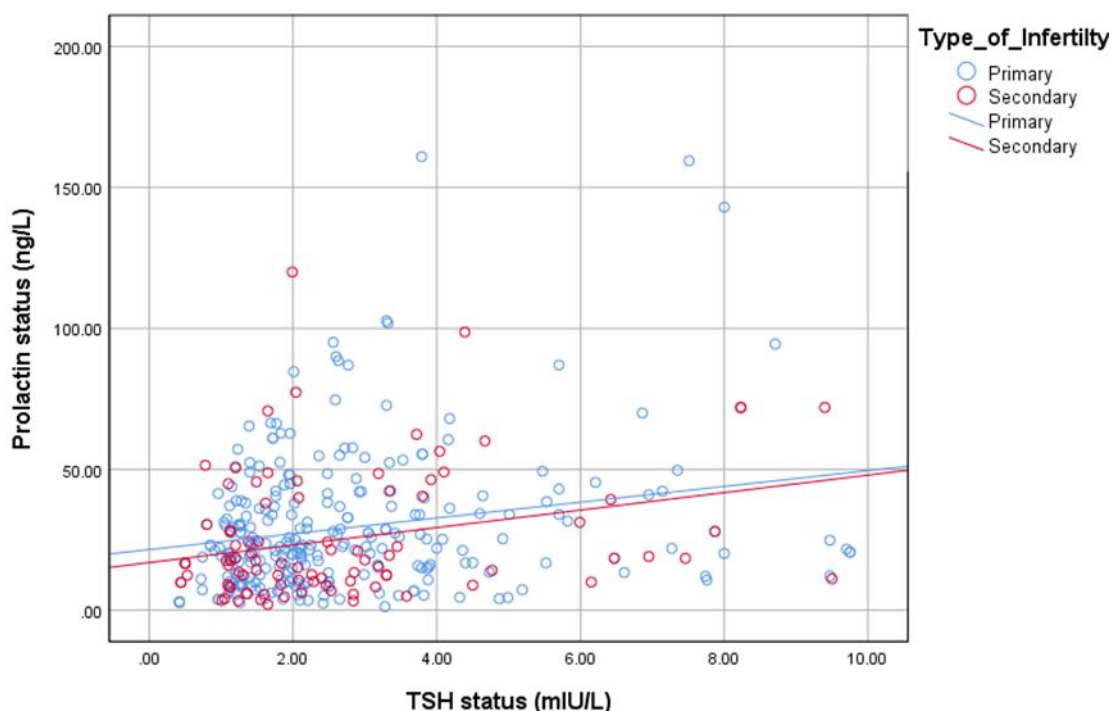


Figure 1: Scatter diagram showing the correlation between TSH and prolactin in primary and secondary sub-fertile patients

DISCUSSION

Thyroid hormones have profound effects on reproduction and pregnancy. The prevalence of hypothyroidism in women of reproductive age (20-40 years) varies between 2% to 4%.^{17,18} Thyroid dysfunction is a common cause of sub-fertility which can be easily managed by correcting the appropriate levels of thyroid hormones.^{19,20} In our study, 40 patients out of 359 had hypothyroidism (11.1%). Relatively higher rate of hypothyroidism was found due to

special referral pattern of sub-fertile patients to the clinic. Singh *et al* found 57% of women with hypothyroidism in their study.²¹ Elder *et al* in their cohort study found 20.5% sub-fertile women had associated subclinical hypothyroidism.²² In our study, majority of sub-fertile patients had hyperprolactinemia (55.2%). This higher propensity of hyperprolactinemia is in agreement with the findings of Kumkum *et al*²³ who had depicted a prevalence of 46% in their study. Another study found a higher prevalence

of hyperprolactinemia in primary sub-fertility (60.3%) than in secondary sub-fertility (40.2%).²⁴ It has been recommended that in the presence of raised TSH along with raised PRL levels, the treatment should be first to correct the hypothyroidism before evaluating further causes of hyperprolactinemia. Hormone therapy with thyroxine is the choice of treatment in established hypothyroidism. It normalizes the menstrual cycle, PRL levels and improves the fertility rate. As per our study, we observed 7.8% sub-fertile patients with hypothyroidism exhibiting hyperprolactinemia. In our study, the mean TSH level in primary and secondary sub-fertility was 2.75 ± 1.89 mIU/L and 2.73 ± 2.10 mIU/L respectively. A study showed that women who never achieved basal TSH < 2.5 mIU/L had lower conception rates.²⁵ Out of 359 patients prolactin level was found to be 29.25 ± 24.19 ngm/L and 24.41 ± 22.80 ngm/L in primary and secondary sub-fertility respectively. Choudhary and Goswami²⁶ observed hyperprolactinemia in 16.6% patients with hypothyroidism. A significant positive correlation between TSH and Prolactin levels was found in subjects enrolled in our study ($r = 0.219$, $p < 0.001$) in primary sub-fertility and $r = 0.285$ ($p = 0.006$) in secondary sub-fertility. This finding is also consistent with the findings of Goswami et al.²⁷ Similar findings were reported by Poppe and Velkeniers.²⁸ They observed that in hyperprolactinemic patients without any sign of pituitary dysfunction, there are normally reduced levels of thyroid hormones. Tasneem et al.²⁹ also observed in their study, that some of the women with high prolactin levels had subclinical hypothyroidism.

CONCLUSION

It is therefore, concluded that thyroid dysfunction with hyperprolactinemia may be a major contributor hormonal factor in sub-fertility. Measurement of TSH and PRL should be done at early stage of sub-fertility check up rather than straight away going for more costly tests or invasive procedures.

Conflicts of Interest: There is no conflict of interest.

REFERENCES

1. KL Wright. Defining infertility: what infertility means for clinicians and clients. Network. 2003; 23(2): 4–6.
2. Zegers-Hochschild F, Adamson GD, de Mouzon J, Ishihara O, Mansour R, Nygren K, et al. The International committee for monitoring assisted reproductive technology (ICMART) and the World health organization (WHO) revised glossary on ART terminology, 2009. Human Reproduction. 2009; 24(11): 2683-2687.
3. Cong J, Pingping L, Zheng L, Jichun. Infertility: a tabulation of available data on prevalence of primary and secondary infertility program on Maternal and Child Health and Family Planning, Division of Family Health. World Health Organization, Geneva; 1991. 2016.
4. Boivin J, Bunting L, Collins JA, Nygren KG. International estimates of infertility prevalence and treatment-seeking: potential need and demand for infertility medical care. Human Reproduction. 2007; 22(6): 1506-1512. <https://doi.org/10.1093/humrep/dem046>.
5. Blundell R. Causes of infertility. International Journal of Molecular Medicine and Advance Sciences. 2007; 3(1): 63-65.
6. Evers MC. The infertile couple. Am Fam Physician. 2002; 54(3): 1001–1010.
7. Thomas R, Reid RL. Thyroid disease and reproductive dysfunction: a review. Obstet and Gynecol. 1987; 70(5): 789-798.
8. Trokoudes, Krinos M, Skordi SN, Picolos, MK. "Infertility and thyroid disorders." Current Opinion in Obstetrics and Gynecology. 2006; 18(4): 446-451.
9. Lincoln R, Ke RW, Kutteh WH. Screening for hypothyroidism in infertile women. J Reprod Med. 1999; 44: 455–457.
10. Krassas GE. Thyroid disease and female reproduction. Fertil Steril. 2000; 74: 1063–1070.
11. Freeman ME, Kanyicska B, Lerant A, Nagy G. Prolactin: structure, function, and regulation of secretion. Physiol Rev. 2000; 80(4): 1523-1631.

12. Biller BMK, Luciano A, Crosignani PG, Molitch M, Olive D, Rebar R et al. Guidelines for the diagnosis and treatment of hyperprolactinemia. *Journal of Reproductive Medicine for the Obstetrician and Gynecologist*. 1999; 44(12): 1075-1084.
13. Cooke PS, RHolsberger D, JWitorsch R, WSylvester P, MMeredith J, ATreinen K, et al. <https://www.sciencedirect.com/science/article/pii/S0041008X03004721>!Thyroid hormone, glucocorticoids, and prolactin at the nexus of physiology, reproduction and toxicology. *Toxicology and Applied Pharmacology*. 2004; 194(3): 309-335.
14. Crosignani PG. Management of hyperprolactinemic infertility. *Middle East Fertility Society Journal*. 2012; 17(2): 63-69.
15. Krassas GE. Thyroid disease and female reproduction. *Fertility and Sterility*. 2000; 74(6): 1063-1070.
16. Affia T. The incidence of hyperprolactinemia and associated hypothyroidism: local experience from Lahore Centre for Nuclear Medicine, Mayo Hospital Lahore, 2 Government College for Boys Gulberg Lahore. *PJNM*. 2011; 1: 49-55.
17. Wang C, Crapo LM. The epidemiology of thyroid disease and implications for screening. *Endocrinol Metab Clin North Am*. 1997; 26(1): 189-218.
18. Bjoro T, Holmen J, Krüger O, Midthjell K, Hunstad K, Schreiner T, et al. Prevalence of thyroid disease, thyroid dysfunction and thyroid peroxidase antibodies in a large, unselected population. The Health Study of Nord-Trondelag (HUNT). *Eur J Endocrinol*. 2000; 143(5): 639-647.
19. Davis LB, Lathi RB, Dahan MH. The effect of infertility medication on thyroid function in hypothyroid women who conceive. *Thyroid*. 2007; 17: 773-777.
20. Dajan CM, Saravanan P, Bayly G. Whose normal thyroid function is better –yours or mine? *Lancet*. 2002; 360: 353-354.
21. Singh L, Agarwai CG, Chowdhary SR, Mehra P, Khare R. Thyroid profile in infertile women. *J Obstet Gynecol India*. 1990; 40: 248-253.
22. Eldar Geva J, ShohamM, Rosler A. Subclinical hypothyroidism in infertile women: the importance of continuous monitoring and the role of thyrotrophin releasing hormone stimulation test. *Gynecol Endocrinol*. 2007; 23: 332-337.
23. Kumkum A, Jasmine K, Shweta G, Pal Ajeshwar N. Hyperprolactinemia and its correlation with hypothyroidism in infertile women. *J Obstet Gynecol India*. 2006; 56(1): 68-71.
24. Akhter N, Hassan S. Sub-clinical hypothyroidism and hyperprolactinemia in infertile women: Bangladesh perspective after universal salt iodination. *Internet J Endocrinol*. 2008; 5(1): 1-6.
25. Raber W, Nowotny P, Vytiska-Binstorfer E, Vierhapper H. Thyroxine treatment modified in infertile women according to thyroxine-releasing hormone testing: 5 year follow-up of 283 women referred after exclusion of absolute causes of infertility. *Hum Reprod*. 2003; 18: 707-714.
26. Choudhury SD, Goswami A. Hyperprolactinemia and reproductive disorders--a profile from north east. *J Assoc Physicians India*. 1995; 3(9): 617-618.
27. Goswami B, Patel S, Chatterjee M, Koner BC, Saxena A. Correlation of prolactin and thyroid hormone concentration with menstrual patterns in infertile women. *J Reprod Infertil*. 2009; 10(3): 207-212.
28. Poppe K, Velkeniers B. Thyroid and infertility. *Verh K Academy Geneesk Belg*. 2002; 64(6): 389-399.
29. Tasneem A, Fatima I, Ali A, Mehmood N, Amin MK. The incidence of hyperprolactinaemia and associated hypothyroidism: local exsaperience from Lahore. *Pak J Nuclear Med*. 2011; 1: s49-55.

Study of Electrolytes and Blood Gas changes in Acute and Acute-on-Chronic Intestinal Obstruction

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ABSTRACT

Introduction: Intestinal obstruction is the significant mechanical impairment or complete arrest of the passage of contents through the intestine. It accounts for 20% of all acute surgical admissions. The study was conducted to find out the electrolytes pattern and arterial blood gas changes among different variety of intestinal obstruction. **Methods:** This cross-sectional observational study included 200 subjects in acute and acute-on-chronic intestinal obstruction in different general surgical wards in Dhaka Medical College Hospital, Dhaka during July to December, 2013. **Results:** The highest number of the patients with acute intestinal obstruction presented belongs to 31-40 years age group (35, 32%). Majority (64, 32%) of causes of intestinal obstruction were adhesion followed by intestinal TB (57, 28.5%). In acute intestinal obstruction, 76 (69.0%) patients had hyponatraemia and 80 (72.7%) had hypokalaemia during admission, while most of hyponatraemia 92 (83.6%) and hypokalaemia 89 (80.9%) were corrected after resuscitation. Majority of the subjects had alkalosis (70.9%) and decreased PaCO₂ (70.9%) before resuscitation. After resuscitation, patients with 80.9% alkalosis and 78.0% PaCO₂ returned to normal. In acute-on-chronic intestinal obstruction, 71 (78.9%) had hyponatraemia and 74 (82.2%) had hypokalaemia before resuscitation, while majority of hyponatraemia (70, 77.8%) and hypokalaemia (68, 75.5%) were corrected following resuscitation. Majority of the subjects had alkalosis (67, 74.4%) and decreased PaCO₂ (66, 73.3%) before resuscitation. Following resuscitation P^H, PaCO₂ of patients returned to normal by 75.5% and 73.3% respectively. **Conclusion:** Acute and acute-on-chronic obstruction patients had hyponatraemia and hypokalaemia with abnormalities in Arterial Blood Gas (ABG) on admission. Correction of electrolytes imbalance before surgery reduces mortality and morbidity.

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INTRODUCTION

Acute intestinal obstruction occurs when there is an interruption in the forward flow of intestinal contents. This interruption can occur at any point along the length of the gastrointestinal tract, and clinical symptoms often vary based on the level of obstruction. Acute-on-chronic intestinal obstruction may present with short history of abdominal distension against background of pain and constipation. It usually spread from large gut to involve small bowel. The condition is often treated conservatively over a period of 2–5 days with the patient's progress regularly monitored by an assigned physician. Surgical procedures are performed in life-threatening cases.^{1,2} The clinical presentation generally includes nausea and emesis, colicky abdominal pain and a failure to pass flatus or bowel movements. The classic physical examination findings of abdominal distension, tympani to percussion, and high-pitched bowel sounds suggest the diagnosis. Radiologic imaging can confirm the diagnosis and can also serve as useful adjunctive investigations when the diagnosis is less certain.³ Combined water and electrolyte depletion may occur from gastrointestinal losses due to vomiting and sequestration of large volume of fluid in intestine. Proximal to the point of obstruction, the intestinal tract dilates as it fills with intestinal secretions and swallowed air.⁴

Fluid loss from emesis, bowel oedema and loss of absorptive capacity leads to dehydration. Emesis leads to loss of gastric potassium, hydrogen and chloride ions, and significant dehydration stimulates renal proximal tubule reabsorption of bicarbonate and loss of chloride, perpetuating the metabolic alkalosis.⁵ In addition to derangements in fluid and electrolyte balance, intestinal stasis leads to overgrowth of intestinal flora, which may lead to the development of feculent emesis. Additionally, overgrowth of intestinal flora in the small bowel leads to bacterial translocation across the bowel wall.⁶ Ongoing dilation of the intestine increases luminal pressures. When luminal pressures exceed venous pressures, loss of venous drainage causes increasing edema and hyperemia of the bowel.

This may eventually lead to compromised arterial flow to the bowel, causing ischaemia, necrosis and perforation. A closed-loop obstruction may undergo this process rapidly. Intestinal volvulus, the prototypical closed-loop obstruction, causes torsion of arterial inflow and venous drainage, and is a surgical emergency.^{7,8}

More severe or prolonged under perfusion of kidney due to hypovolaemia, shock may lead to failure of compensatory mechanism and hence an acute decline in GFR. This may lead to formation of low volume of urine or anuria. Ischaemia and toxic insults to the kidney preferentially cause cell death of tubular epithelial cells, promoting decreased kidney function.^{9,10}

As the P^H decreases (<7.35), it implies acidosis, while if the P^H increases (>7.45) it implies alkalosis. In the context of arterial blood gases, the most common occurrence will be that of respiratory acidosis. Carbon dioxide is dissolved in the blood as carbonic acid, a weak acid; however, in large concentrations, it can affect the P^H drastically. Whenever there is poor pulmonary ventilation, the carbon dioxide levels in the blood are expected to rise. This leads to a rise of carbonic acid, leading to a decrease in P^H . The first buffer of P^H will be the plasma proteins, since these can accept some H^+ ions to try and maintain homeostasis. As carbon dioxide concentrations continue to increase ($PaCO_2 > 45$ mmHg), the condition is known as respiratory acidosis. The body tries to maintain homeostasis by increasing the respiratory rate. This allows much more carbon dioxide to escape the body through the lungs, thus increasing the P^H by having less carbonic acid. If a patient is in a critical setting and intubated, one must increase the number of breaths mechanically.

On the other hand, respiratory alkalosis ($PaCO_2 < 35$ mmHg) occurs when there is too little carbon dioxide in the blood. This may be due to hyperventilation or else excessive breaths given via a mechanical ventilator in a critical care setting. The action to be taken is to calm the patient and try to reduce the number of breaths being taken to normalize the P^H . The respiratory pathway tries to compensate for the change in P^H

in a matter of 2–4 hours. If this is not enough, the metabolic pathway takes place.

The kidney and the liver are two main organs responsible for the metabolic homeostasis of P^H . Bicarbonate is a base that helps to accept excess hydrogen ions whenever there is acidaemia. However, this mechanism is slower than the respiratory pathway and may take from a few hours to 3 days to take effect. In acidaemia, the bicarbonate levels rise, so that they can neutralize the excess acid, while the contrary happens when there is alkalaemia. Thus when an arterial blood gas test reveals, for example, elevated bicarbonate, the problem has been present for a couple of days, and metabolic compensation took place over a blood acidaemia problem.

In general, it is much easier to correct acute P^H derangements by adjusting respiration. Metabolic compensations take place at a much later stage. However, in a critical setting, a patient with a normal P^H , high CO_2 and high bicarbonate means that, although there is a high carbon dioxide level, there is metabolic compensation. As a result one must be careful as to not artificially adjust breaths to lower the carbon dioxide. In such case, lowering the carbon dioxide abruptly means that the bicarbonate will be in excess and will cause a metabolic alkalosis. In such a case, carbon dioxide levels should be slowly diminished.¹¹

METHODS

This was a cross-sectional observational study was carried out among 200 subjects suffering

from acute and acute-on-chronic intestinal obstruction to observe the electrolytes and blood gas changes. The study was carried out in different surgical wards of Dhaka Medical College, Dhaka during July – December' 2013. The study subjects were enrolled after fulfillment of the inclusion criteria. They were collected from the referred patients attending in out-patient department of surgery and also from in-patient department of the respective discipline. Blood was collected immediately after admission (before resuscitation) and after resuscitation of the patients those arrived in hospital with early or delayed onset of symptoms. Blood samples were collected from radial or femoral artery with aseptic and necessary precaution in the Intensive Care Unit (ICU). Then the findings of the different samples were compared among the acute and acute-on-chronic intestinal obstruction cases.

RESULTS

Among total 200 cases of intestinal obstruction, 110 were acute intestinal obstruction and 90 were acute-on-chronic intestinal obstruction. Age range of patients was 18-65 years. The highest number (35, 32%) of age group was 31-40 years in acute intestinal obstruction and that in acute-on-chronic obstruction was 41-50 years age groups (30, 33.3%). In acute obstruction, 70 (63.7%) were male and 40 (36.3%) were female and in acute-on-chronic obstruction group, 62 (68.8%) were male and 28 (31.2%) were female (Table I).

Table I: Demographic characteristics of patients (n=200)

Demographic variables		Acute intestinal obstruction (n= 110) Number (%)	Acute-on-chronic obstruction (n=90) Number (%)
Gender	Male	70 (63.7)	62 (68.8)
	Female	40 (36.3)	28 (31.2)
Age group in years	18- 30	25 (22.7)	10 (11.1)
	31-40	35 (32)	15 (16.7)
	41-50	20 (18.2)	30 (33.3)
	51-60	20 (18.2)	25 (27.8)
	>61	10 (9)	10 (11.1)

Common causes of intestinal obstruction in this study were adhesion 64 (32%), intestinal TB 57

(28.5%), malignancy 24 (12%), hernia 23 (11.5%), volvulus 18 (9%) (Table II).

Table II: Causes of Intestinal Obstruction

Causes of obstruction	n (%)
Adhesion	64 (32%)
Intestinal tuberculosis(TB)	57 (28.5%)
Malignancies	24 (12%)
Hernia	23 (11.5%)
Volvulus	18 (9%)
Worms	08 (4%)
Faecal impaction	06 (3%)
Total	200 (100%)

Most subjects with acute intestinal obstruction had hyponatraemia (69.0%), 72.7% hypokalaemia, 70.9% hypochloraemia and 67.3% hypocalcaemia before resuscitation while 83.6% hyponatraemia, 80.9% hypokalaemia, 81.8% hypochloraemia and 80% hypocalcaemia were corrected after resuscitation (Table III).

Table III: Electrolytes change in acute intestinal obstruction

Electrolytes	Number (%) of acute intestinal obstruction showing electrolytes changes					
	On admission (%)			After resuscitation (%)		
	Normal	Below normal	Above normal	Normal	Below normal	Above normal
Na ⁺	16 (14.5)	76 (69.0)	18 (16.4)	92 (83.6)	8 (7.3)	10 (9.0)
K ⁺	17 (15.4)	80 (72.7)	13 (11.8)	89 (80.9)	14 (12.7)	7 (6.4)
Cl ⁻	14 (12.7)	78 (70.9)	18 (16.5)	91 (81.8)	9 (8.2)	10 (9.0)
Ca ⁺⁺	18 (16.4)	74 (67.3)	18 (16.5)	88 (80.0)	13 (11.8)	9 (8.2)

Table IV shows that, majority of the subjects had alkalosis (70.9%) and decreased PaCO₂ (70.9%)

before resuscitation. After resuscitation, P^H (80.9%) and PaCO₂ (78%) returned to normal.

Table IV: Blood gas changes in acute intestinal obstruction

Blood gases	Number (%) of acute intestinal obstruction showing blood gas changes					
	On admission (%)			After resuscitation (%)		
	Normal	Below normal	Above normal	Normal	Below normal	Above normal
P ^H	18 (16.3)	14 (12.7)	78 (70.9)	89 (80.9)	14 (12.7)	7 (6.4)
PaCO ₂	18 (16.3)	78 (70.9)	14 (12.7)	87 (78.0)	15 (13.6)	9 (8.2)
PaO ₂	84(76.4)	26 (33.6)	00	94 (85.4)	14 (12.5)	2 (2.0)
HCO ₃	20 (18.1)	14 (12.7)	76 (69.9)	85 (77.3)	17 (15.4)	8 (7.3)

In acute-on-chronic intestinal obstruction, most subjects (78.9%) had hyponatraemia, 82.2% hypokalaemia, 77.8% hypochloraemia and 81.1% hypocalcaemia before resuscitation while 77.8%

hyponatraemia, 75.5% hypokalaemia, 80% hypochloraemia and 77.8% hypocalcaemia was corrected after resuscitation (Table V).

Table V: Electrolytes change in acute-on-chronic intestinal obstruction

Electrolytes	Number (%) of acute-on-chronic intestinal obstruction showing electrolytes changes					
	On admission (%)			After resuscitation (%)		
	Normal	Below normal	Above normal	Normal	Below normal	Above normal
Na ⁺	10 (11.1)	71 (78.9)	09 (10.0)	70 (77.8)	14 (15.5)	06 (6.7)
K ⁺	06 (6.7)	74 (82.2)	10 (11.1)	68 (75.5)	13 (14.4)	09 (10.0)
Cl ⁻	11 (12.2)	70 (77.8)	09 (10.0)	72 (80.0)	12 (13.3)	06 (6.7)
Ca ⁺⁺	07 (7.8)	73 (81.1)	10 (11.1)	70 (77.8)	14 (15.5)	06 (6.7)

Among these patients, majority of them had alkalosis (74.4%) and decreased PaCO₂ (73.3%) before resuscitation. After resuscitation, P^H (75.5%) and PaCO₂ (73.3%) returned to normal (Table VI).

Table VI: Blood gas changes in acute-on-chronic Intestinal Obstruction

Blood gases	Number (%) of acute-on-chronic intestinal obstruction showing blood gas changes					
	On admission (%)			After resuscitation (%)		
	Normal	Below normal	Above normal	Normal	Below normal	Above normal
P ^H	07 (7.8)	16 (17.8)	67 (74.4)	68 (75.5)	14 (15.5)	08 (8.9)
PaCO ₂	10 (11.1)	66 (73.3)	14 (15.5)	66 (73.3)	15 (16.7)	09 (10.0)
PaO ₂	64 (71.1)	26 (28.9)	00	81 (90.0)	09 (10.0)	00
HCO ₃ ⁻	10 (11.1)	16 (17.8)	64 (71.1)	70 (77.8)	14 (15.5)	06 (6.7)

DISCUSSION

Nowadays, acute and acute-on-chronic intestinal obstructions remain the most serious common cause for emergency laparotomy. Although the mortality rate continues to decrease with a better understanding of the pathophysiology, improvement of diagnostic techniques and greater stress on correction of fluid and electrolyte imbalance, most of these are limited to developed countries or the major centers in other countries.^{12,13} Previous study revealed that adhesions are the single most common cause for small bowel obstruction. Non adhesive aetiologies of bowel obstruction include incarcerated hernias, obstructive lesions (malignant and benign), and a number of infrequent causes for bowel obstruction such as

bezoars, inflammatory bowel disease, and volvulus.¹⁴ The causes of SBO in pediatric patients include intussusceptions, congenital atresia and stenosis.¹⁵ However, in present study, causes of intestinal obstruction were adhesion (32%), intestinal TB (28.5%), malignancy (12%), hernia (11.5%), volvulus (9%), worms (4%) and faecal impaction (3%).

Due to repeated vomiting in acute and acute-on-chronic obstruction, there is loss of sodium and potassium with gastric acid loss resulting in hyponatraemia, hypokalaemia and metabolic alkalosis.^{3,4,5} On admission, these patients usually found collapsed due to excessive fluid loss which is resulting electrolytes imbalance and altered arterial blood gas level.^{6,7,8}

In current study, most of the subjects with acute intestinal obstruction (69.1%) had hyponatraemia and 72.8% hypokalaemia before resuscitation. Another study shown that, serum sodium levels were ranged from 121 mEq/L to 133.3 mEq/L. Persistent hyponatremia with no relation to duration of obstruction and no changes in serum potassium level were noted.¹⁶

Any type of obstruction is first treated conservatively, followed by investigation to find out cause of obstruction than definitive management by laparotomy. Mortality and morbidity are dependent on the early recognition, correct diagnosis of obstruction and proper surgical intervention.^{17,18}

Hyponatraemia is a common electrolyte disorder among hospitalized patients and has been associated with increased mortality. The goal is to raise the serum sodium level by 1.5 to 2mEq/L/hour until symptoms subside or until the concentration has risen to a safer level-- usually greater than 118 to 120mEq/L, with the primary focus being to minimize the risk of seizures.¹⁹

CONCLUSION

In this study, it was seen that both acute and acute-on-chronic intestinal obstruction patients had hyponatraemia, hypokalaemia and abnormal ABG. The majority of the abnormalities of ABG were corrected in both conditions. But the correction of electrolytes imbalance (hyponatraemia and hypokalaemia) is relatively more in acute obstruction than in acute-on-chronic obstruction which influences the better surgical outcome in acute intestinal obstruction.

Conflicts of Interest: None

REFERENCES

1. Diaz JJ Jr, Bokhari F, Mowery NT, Acosta JA, Block EF, Bromberg WJ. Guidelines for management of small bowel obstruction. *J Trauma*. 2008; 64(6): 1651-1664.
2. Gill SS, Eggleston FC. Acute Intestinal Obstruction. *Arch Surg*. 1965; 91(4): 589-591.
3. Patrick G, Manish R. Evaluation and Management of Intestinal Obstruction. *Am Fam Physician*. 2011; 83(2): 159-165.
4. Wright HK, O'Brien JJ, Tilson MD. Water absorption in experimental closed segment obstruction of the ileum in man. *Am J Surg*. 1971; 121(1): 96-99.
5. Wangenstein OH. Understanding the bowel obstruction problem. *Am J Surg*. 1978; 135(2): 131-149.
6. Rana SV, Bhardwaj SB. Small intestinal bacterial overgrowth. *Scand J Gastroenterol*. 2008; 43(9): 1030-1037.
7. Gilbert R, Kinsey D, Mark D, Okusa D. Pathogenesis of Acute Kidney Injury: Foundation for Clinical Practice. *Am J Kidney Dis*. 2011; 58(2): 291-301.
8. Zerey M, Sechrist CW, Kercher KW, Sing RF, Matthews BD, Heniford BT. The laparoscopic management of small-bowel obstruction. *Am J Surg*. 2007; 194(6): 882-887.
9. Khaikin M, Schneidereit N, Cera S, Sands D, Efron J, Weiss EG. Laparoscopic vs. open surgery for acute adhesive small-bowel obstruction: patients' outcome and cost-effectiveness. *Surg Endosc*. 2007; 21(5): 742-746.
10. Di Saverio S, Coccolini F, Galati M, Smerieri N, Biffl WL, Ansaloni L, et al. Bologna guidelines for diagnosis and management of adhesive small bowel obstruction (ASBO): 2013 update of the evidence-based guidelines from the world society of emergency surgery ASBO working group. *World J Emerg Surg*. 2013; 8(1): 42.
11. Samuel TR, Vyshnavi R, Vanaja RA, Ragashree CM, Rajagopalan B. Graphical Analysis of Arterial Blood Gas Analysis Using Standard Base Excess. *Int. J. Pharm. Sci. Rev. Res*. 2017; 46(1): 223-228.
12. Cappell MS, Batke M. Mechanical obstruction of the small bowel and colon. *Med Clin North Am*. 2008; 92(3): 575-597.
13. Fevang BT, Fevang J, Stangeland L, Soreide O, Svanes K, Viste A. Complications and death after surgical treatment of small bowel obstruction: A 35-year institutional experience. *Ann Surg*. 2000; 231(4): 529-537.
14. Ten Broek RPG, Krielen P, Saverio SD, Coccolini F, Biffl WL, Ansaloni L, et al. Bologna guidelines for diagnosis and management of adhesive small bowel obstruction (ASBO): 2017 update of the evidence-based guidelines from the world society of emergency surgery ASBO working group. *World J Emerg Surg*. 2018; 13:24

15. Nakanwagi AM, Kijjambu SC, Peter Ongom (RIP), Luggya TS. Aetiology and Presentation of Intestinal Obstruction among Patients Presenting to a Tertiary Hospital in Uganda. *Int J Crit Care Emerg Med*. 2016; 2(2): 018
16. Kumara S, Kazmib SA. Electrolyte Imbalances in Acute Intestinal Obstruction in Adults. *WJPMR*. 2017; 3(1): 101-105.
17. Rex DK. Colonoscopy and acute colonic pseudo-obstruction. *Gastrointest Endosc Clin N Am*. 1997; 7: 499–508.
18. De Giorgio R, Barbara G, Stanghellini V. The pharmacologic treatment of acute colonic pseudo-obstruction. *Aliment Pharmacol Ther*. 2001; 15: 1717–1727.
19. Douglas I. Hyponatraemia: Why it matters, how it presents, how we can manage it. *CCJM*. 2006; 73(3): S4-S11.

Buerger's Disease among the Lower limb Ischaemic Patient in Rajshahi, Bangladesh

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ABSTRACT

Introduction: It is well established that, atherosclerosis is the major cause of chronic lower limb ischaemia in the western countries. Buerger's disease is common in South and Southeast Asia. In Bangladesh, no accurate data is available about the incidence of chronic lower limb ischaemia. Whether Buerger's disease is common or not and how the patients with chronic lower limb ischaemia present in Bangladesh are still not established. **Objectives:** To identify the cause and assess the severity of chronic lower limb ischaemia among the patients admitted in Rajshahi Medical College Hospital. **Methods:** This cross-sectional type of descriptive study was conducted in the Department of Surgery, Rajshahi Medical College Hospital, Rajshahi. A total of 70 cases were included by purposive sampling technique. The inclusion criteria for cases were the patient diagnosed as chronic lower limb ischaemia. Diabetic patients and limb ischaemia following trauma were excluded from the study. **Results:** Among the cases, 59 (84.29%) had Buerger's disease and 11 (15.71%) had atherosclerosis. **Conclusion:** Chronic lower limb ischaemia seemed to be a common problem in northern Bangladesh. Majority of the patients were likely to suffer from Buerger's disease.

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INTRODUCTION

As compared to coronary artery disease (CAD), peripheral arterial disease (PAD) has long been addressed as being negligible in number and importance; a view that had been reassessed in recent years. The PAD has been estimated to reduce quality of life in approximately 2 million symptomatic Americans¹ and millions more Americans without claudication are likely to suffer PAD associated impair-

ment.² In USA, there had been 413,000 discharges per year with chronic PAD, 88000 hospitalization involving lower extremity arteriography and 28000 discharges citing embolectomy or thrombectomy of lower limb arteries.³ In the UK, at least 1 in 20 people over the age of 55 have some degree of PAD.⁴ So, PAD is clearly a big burden in western society. Chronic limb ischaemia is manifested by intermittent claudication, rest pain, non-healing ulcers or

gangrene and may ultimately results in limb losses, atherosclerosis is the main cause. But from different studies, it is clear that there is geographical variation. Though thromboangitis obliterans (Buerger's disease) is rare in western countries, it is common in south and Southeast Asia, the Middle East, and eastern European countries.⁵ In young adults presenting to the Mayo clinic (1953-1981) with lower limb ischaemia, Buerger's disease was diagnosed in 24%.⁶

In western India, Buerger's disease is the predominant peripheral vascular disease. In the period of 1988-1991, RM Jindal et al treated 62 cases of Buerger's disease. Among them, all were male below 42 years of age. A total of 51 cases had peripheral gangrene with intractable pain, while the remaining 11 had non-healing ulcers with severe pain.⁷

Buerger's disease is not uncommon in Japan. Matushita et al studied 105 patients with Buerger's disease, admitted at Nagoya University Hospital between January 1985 and December 1996. Among them, 96% were men and presenting with either gangrene or ulcer in 64%. They mentioned that at their institute, the prevalence of Buerger's disease had been appearing to be decreasing but not significantly that for Atherosclerosis.⁸ Chronic lower limb ischaemia is not an uncommon disease in Bangladesh. In Mymensingh Medical College Hospital, a total of 450 patients underwent limb amputations during the period of 1982- 1987. Among them, 366 (81%) patients had been suffering from chronic limb ischaemia.⁹

In a study of 39 patients with Buerger's disease in Kumudini Hospital Tangail, Bangladesh, found that majority of the patients had ulceration or gangrene at presentation.¹⁰ High incidence of Buerger's disease in the northern part of Bangladesh found in a study at Rajshahi Medical college.¹¹

But still the data for chronic lower limb ischaemia, regarding their incidence, aetiology or clinical presentation, are not sufficient. So, this study was conducted to explore the incidence of Buerger's disease in the north-western part of Bangladesh, which may help to generate a base

line data about the lower limb ischaemic diseases.

METHODS

It was a cross-sectional type of descriptive study, carried out in the Department of Surgery, Rajshahi Medical College, Rajshahi, during the period of January, 2009 to December, 2010. Total 70 cases of chronic lower limb ischaemia were selected by purposive sampling. Patients with acute limb ischaemia and also with chronic limb ischaemia due to trauma and diabetes mellitus were excluded from the study. A face-to-face formal interview of the cases, regarding particulars of the patients's symptoms, history of trauma, co-morbidity and family history, were collected. Thorough clinical examination was carried out of each study participant to assess anaemia, and nutritional status. Examination of lower limbs including inspection and palpation about colour change, skin temperature, trophic changes, ulcer and gangrene were carried out. Vascular assessment was done by examination of peripheral arterial pulsation and Ankle-Brachial Pressure Index (ABPI) measurement. A hand held Doppler (summit Doppler lifedop 150 vascular Doppler, USA) was used for measuring ABPI.

Laboratory investigations included complete blood count, fasting blood sugar and post-parandial blood Sugar, lipid profile, chest X-ray, electrocardiogram, echocardiogram and duplex imaging of lower limb vessels. Biopsy of arteries likely to be involved, such as arteria dorsalis pedis, anterior tibial artery and popliteal artery, had been done in cases underwent amputation. A checklist was used to collect and record the data.

The quantitative data of the study were entered and analyzed by using SPSS (Statistical Package for the Social Science, IBM 2009) software programs. The descriptive analysis included frequency distribution, mean, median and standard deviation. Univariate analysis was done to describe the characteristics of the population. Statistical analysis was carried out by using Chi-square test and t-test. The findings of the study were presented with the help of frequency distribution table, charts, bar diagram etc. Ethical clearance was accepted by the Institutional Review Board of Rajshahi Medical College before starting the study.

RESULTS

In present study, majority 34 (48.6%) was included from age group 31-40 years, followed by 16 (22.9%) in the more than 50 years (Table I).

Table I: Age distribution of 70 cases

Age groups in years	Frequency	
	N	%
20-30	8	11.4
31-40	34	48.6
41-50	12	17.1
More than 50	16	22.9
Total	70	100

All the patients in the study were male. It was revealed that among 70 patients, 34 (48.6%) were farmers, 18 (25.7%) were day laborers, 10 (14.3%) were service holder and others in smaller numbers included businessmen, rickshaw puller (Table II).

Table II: Occupation of study cases

Occupation	Frequency	
	N	%
Farmer	34	48.6
Day labour	18	25.7
Businessman	6	8.6
Rickshaw puller	2	2.9
Service	10	14.3
Total	70	100

All the patients found current smokers. Among them, 30 (42.9%) patients used to smoke 10-20 pack years, 22 (31.4%) patients more than 20 pack years and 18 (25.7%) patients less than 10 pack years. Among 70 patients all had history of pain at presentation; out of them 58 (82.9%) with rest pain and another 12 (17.1%) with intermittent claudication. There was gangrene in different parts of affected limbs in 64 (91.4%) patients; mostly at great toe 52 (81.3%). In cases of 4 (6.3%) patients, gangrene found at dorsum of foot and 2 (3.1%) patients at different toes. Non-healing ulcers with punched out edge found in 14 (20%) patients; among them, 8 (57.1%) at dorsum

of foot and 4 (28.6%) at different toes. There were ischaemic changes in upper limb along with chronic lower limb ischaemia in 8 (11.5%) patients; findings were gangrene in different fingers in 2 patients, had amputee fingers in 6 patients and absent radial arterial pulse in 2 patients. Out of 70 patients, 66 (94.3%) had Ankle-Brachial Pressure Index (ABPI) lower than normal (<1.0) in right lower limbs; among them 34 (51.5%) had ABPI within 0.3-0.49, 24 (42.4%) within 0.5-0.8 and 4 (6.06%) less than 0.3. There were ABPI lower than normal in 58 cases (82.8%); among them 44 (75.8%) within 0.3-0.49 and 14 cases (24.14%) within 0.5-0.8.

Duplex imaging revealed that out of 70 patients, 60 (85.71%) had arteritis and 10 (14.29%) had atherosclerotic lesions. It was also shown in duplex imaging that 55 (78.9%) cases had right lower limbs involvement; among them 24 (43.6%) had popliteal and 28 (32%) had infrapopliteal involvement. In 60 (85.7%) cases, left lower limb involved on duplex imaging; among them 22 (37.7%) had infrapopliteal and 20 (33.3%) had popliteal arterial involvement (Table III).

Table III: Duplex imaging finding of 70 cases with chronic lower limb ischaemia

Cases	Frequency	
	N	%
Arteritis	60	85.7
Atherosclerotic lesion	10	14.3
Total	70	100

Dyslipidemia was found in 6 (8.6%) patients; among them, all had hypertriglyceridemia and 4 cases had elevated LDL and low HDL. Arterial biopsy was possible in those 11 patients who were treated by amputation. Among them, 9 (81.8%) had Buerger's disease and remaining 2 (18.2%) had atherosclerosis (Table IV).

Table IV: Histopathological findings of 11 cases with Chronic lower limb ischaemia

Histopathological diagnosis	Frequency	
	N	%
Buerger's disease	9	81.8
Atherosclerosis	2	18.2
Total	11	100

DISCUSSION

This study explores a high incidence of Buerger's disease in the Rajshahi region of Bangladesh. Out of 70 patients, 59(84.3%) had Buerger's disease and only 11(15.7%) had atherosclerosis. In the current study, Shionnoya's clinical criteria, along with duplex imaging of lower limb vessels made the diagnosis reliable. By considering occupation of majority of study patients 54 (77.2%), it is assumed that majority of the cases were from lower socioeconomic background. Jindal RM et al⁷ stated that invariably all patients affected by Buerger's disease come from the lowest socioeconomic strata of the Indian society and suffer from varying degree of malnutrition.

In this study, no patient was found without gangrene or non-healing ulcer. Most common site of gangrene (90.6%) was great toe including other toes. In addition, all of them complained either intractable rest pain (82.9%) or intermittent claudication (17.1%). Jindal RM et al⁷ studied 62 cases with Buerger's disease in western India, showing 51(82.3%) cases had peripheral gangrene with intractable pain and the remaining 11 (17.7%) had non-healing ulcers with severe pain. Vascular association of Bangalore mentioned that in their experience with about 80 patients with Buerger's disease have not seen a single patient without gangrene or ulcer. Similarly, Grove WJ et al¹⁰ in their study shown that majority of the patients presented with gangrene or ulceration in the Tangail region of Bangladesh.

Most of the study patients 42 (60%) were between 20-40 years. Mean age for Buerger's disease was 40.37 years and that for atherosclerosis was 57.27 years. A very few cases have been reported over 50 years. WJ Grove et al¹⁰ found three 03 (5%) such patients.¹⁰ In this study we found 6(8.9%) cases over 50 years.¹⁰ Atherosclerosis is a disease of old age.

Grove et al¹⁰ confirmed a high incidence of Buerger's disease in the Tangail region of Bangladesh. Rahman et al¹¹ in their study demonstrated a high incidence of Buerger's disease in the northern part of Bangladesh. They stated that smoking bidi might be an important factor for this. Besides smoking, lifestyle habits in

Bangladesh such as not wearing shoes, cultivating rice and jute in the fields of ankle deep mud and water, and the squatting posture of farmers while working in the fields have been reported as the possible aetiological or aggravating factors. Genetic susceptibility has also been suggested with smoking acting as trigger.

All but 2 of the Buerger's disease patients of the present study had palpable femoral artery. Majority of the patients (87.50%) had distal arteries like arteria dorsalis pedis, posterior tibialis and popliteal arteries involvement. Bunicardi et al¹² stated that, Buerger's disease mostly affects small and medium sized arteries, that is digital, plantar and leg arteries and the involvement is distal to proximal.

No female case found in this study might be due to the fact that only 11 patients of atherosclerosis were studied and the incidence of smoking is very low among female in this region.

Smoking found to be a very important risk factor for chronic lower limb ischaemia.¹⁰ All the study subjects were smoker. This study also revealed that the risk of atherosclerosis is related to the amount of smoking and in case of Buerger's disease, smoking history was always present but the amount might not be important. These results were similar to the findings by Hai et al.¹³

In the present study, out of 12 atherosclerotic patients 9 (75%) had comorbidities like ischaemic heart disease (IHD) or stroke. Similar facts were described by Hai et al.¹³

All the cases in this study were male. Buerger's disease is far more common in males than in females.¹⁵ Very few cases have been reported in female. Atherosclerosis is also more common in male than female.¹⁶

More than half (60%) of the present study subjects had bilateral involvement. There was upper limb involvement in 8 (11.5%) cases among the present study subject. All of them had been suffering from Buerger's disease. Mills et al¹⁴ stated that Buerger's disease may involve upper limb in addition to lower limb in up to 16% patients.

Trophic changes seemed to be very common in chronic lower limb ischaemia. In 64 (91.4%) cases of the present study patients found trophic

changes (thinning of skin, hair loss, nail brittling, minor ulcerations in toes, heel, malleoli and ball of foot).

Dyslipidemia, in the form of elevated cholesterol and decreased HDL levels was found to be strongly associated with atherosclerosis. All of the 6 patients with dyslipidemia in this study were atherosclerotic.

The present study had few limitations which were the sample size was relatively small, diagnosis made on clinical criteria with noninvasive investigations and arterial biopsy was possible only in 11 cases.

CONCLUSION

Chronic lower limb ischaemia was found a common problem in the Rajshahi region of Bangladesh showing high incidence of Buerger's disease.

Considering the above facts, it may be recommended that Buerger's disease should be considered first in a patient with lower limb ischaemia in the Rajshahi region of Bangladesh. Social awareness should be raised that smoking is the leading cause of limb ischaemia and gangrene. It is also assumed that further studies should be carried out with larger sample size.

Conflicts of interest: None

REFERENCES

1. Marcoux RM, Larrat EP, Taubman AH, Wilson J. Screening for peripheral arterial disease. J Am Pharm Assoc (Wash). 1996; NS36: 370-373.
2. Hirsch AT, Criqui MH, Treat Jacobson D, Regensteiner JG, Creager MA, Olin J, et al. Peripheral arterial disease detection, awareness and treatment in primary care. JAMA. 2001; 286(11):1317-1324.
3. Gallium RF. Peripheral arterial occlusive disease of the extremities in the United States: hospitalization and mortality. Am Heart J. 1990; 120(6 pt 1): 1414-1418.
4. Pairolero PC, Joycee JW, Skinner CR, Hollier LH, Cherry KJ Jr. Lower limb ischaemia in young adults: prognostic implications. J Vasc Surg. 1984; 1(3): 459-464.
5. Vink M. Symposium on Buerger's disease-19th congress of European Society of Cardiovascular Surgery, Warsaw. July 2, 1970 - Introduction. J Cardiovascular surgery. 1973; 14: 1-5.
6. Pairolero PC, Joyee JW, Skinner CR. Lower limb ischemia in adults. J Vasc. Surg. 1984; 1:459.
7. Jindal RM, Patel SM. Buerger's disease in Western India. J Cardiovasc Surg.1993; 21:326-327.
8. Matsushita M, Nishikimi N, Sakuria T, Naimura Y. Buerger's disease in Japan, Pubmed.1998; 124(3): 498-502.
9. Aftabuddin, M Islam, Jafar N, Haque MA. The status of lower limb amputation in Bangladesh: a 6-year review. Surgery Today. 1997; 27(2): 130-134.
10. Grove WJ, Stansby GP. Buerger's disease and cigarette smoking in Bangladesh. Ann R Coll Surg E. 1992; 74: 115-118.
11. Rahman M, Chowdhury AS, Fukui T, Hira K, Shimbo T. Association of Thrombo-angitis Obiterans with cigarettes, bidi smo-king in Bangladesh: a case control study. Int J Epidemiol. 2000; 29(2): 266-270.
12. Brunicardi FC, Ana D, Anderson K, Timothy Billiar R. Swart's principle of surgery. 8thed; UK: McGraw-Hill Professional. 1999; p.792-793.
13. Ahmed AH, RabindraShrivasta B. Textbook of Surgery by the Association of Surgeon of India (ASI). 2002; p.1332-1353.
14. Mills JA, Taylor LM, Porter JM. Buerger's disease in the modern era. Am J Surg. 1987; 154: 123-127.
15. Buerger's disease symptoms and causes. Available at: <https://www.mayoclinic.org/diseases-conditions/buergers-disease/symptoms-causes/syc-20350658>: Accessed on: 20.01.2020.
16. Atherosclerosis: A Man's Worst Enemy. Available at: <http://www.nhs.uk/Conditions/Atherosclerosis/Pages/Introduction.aspx> Accessed on: 20.01.2020

Cretinism presented as a Case of Craniopharyngioma

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ABSTRACT

In Congenital hypothyroidism (CH) there is inadequate thyroid hormone production in newborn infants. Short stature with a mentally challenged state is frequently attributed to CH. Here we report a case of craniopharyngioma in an adult with untreated congenital hypothyroidism. CT scan of the brain revealed a large sella and supra sellar mass compressing the optic chiasma. MRI features of brain was found consistent with sella and supra sellar complex mass with mass effect having solid and cystic components. The clinical diagnosis of congenital hypothyroidism was confirmed by elevated TSH, low FT4, skeletal survey and ultrasonography of thyroid gland. This was an unusual situation of a large craniopharyngioma detected in an adult with untreated congenital hypothyroidism.

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INTRODUCTION

In Congenital Hypothyroidism (CH), there is inadequate thyroid hormone production in newborn infants. It can occur because of an anatomic defect in the gland, an inborn error of thyroid metabolism, or iodine deficiency. Short stature with a mentally challenged state is frequently attributed to CH. The prevalence of cretinism in Bangladesh is 0.5% (hilly, 0.8%; flood-prone, 0.5%; and plains, 0.3%).¹ Nearly 69% of Bangladeshi population has biochemical iodine

deficiency. Most patients have thyroid ectopia, aplasia or hypoplasia and present with varied clinical manifestations with a 2:1 female to male preponderance.²⁻⁴ The pseudotumour generally regresses with adequate levothyroxine supplementation, but craniopharyngioma does not respond to such treatment.⁵ We report a rare case of a craniopharyngioma in a 25 years old man of short stature in a mentally challenged state with untreated CH. Thyroid function test, X ray, clinical history helps to confirm CH and

Computed Tomography (CT) and MRI of brain helps to know the presence of craniopharyngioma and the extent of the lesion.

Craniopharyngioma is a slow-growing, non-cancerous brain tumour that develops near the pituitary gland and the hypothalamus.^{1,6} This tumour most commonly affects children between 5 and 10 years of age; however, adults can sometimes be affected.¹ Although craniopharyngiomas are not cancerous, they may grow and press on nearby parts of the brain, causing symptoms including hormonal changes, vision changes, delayed growth, headaches, nausea and vomiting, loss of balance, hearing loss and changes in mood or behaviour.² The cause of these tumours is not well understood; however, researchers suspect that they begin to form during the early stages of embryo development in pregnancy and may result from metaplasia.^{2,4}

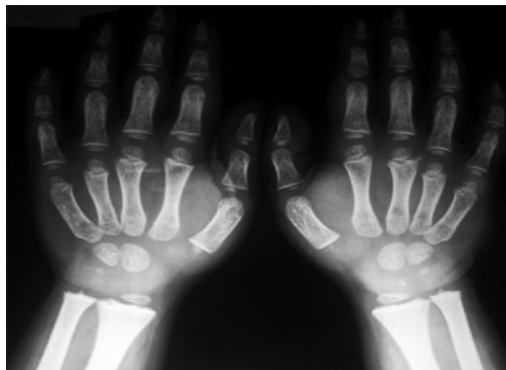


Figure 1: X ray of both hands showing unfused epiphyses and four carpal bones

Most of the problems with hormones and vision do not improve with treatment, and sometimes the surgery can make them even worse, because it may damage the brain structures neighbouring the tumour.⁶ Craniopharyngiomas tend to develop again, mostly in the first 3 years after surgery. Overall recurrence rates range from 0-17% after total removal of the tumour and from 25-63% after partial removal of the tumour with radiotherapy.⁴

The case

A 25-years-old man hailing from Demra, Narayanganj, Dhaka with clinical features of loss of vision for 5 months, failure to gain height from the age of 8 years, poor secondary sexual

These tumours are closely related to another cystic mass occasionally seen in the pituitary called Rathkes cleft cyst.⁵⁻⁸ Craniopharyngioma has two major pathologic variants like adamantinomatous and papillary. The adamantinomatous type is most common in children.^{9,10} Treatment for craniopharyngioma varies and may involve surgery to remove the tumour, radiation therapy, chemotherapy, biologic therapy and/or hormone therapy to replace various hormones no longer produced or secreted due to the tumour or its treatment.⁵ The prognosis for each patient depends on several factors, including the ability of the tumour to be completely removed and the presence of neurological problems or hormonal imbalances caused by the tumour prior to treatment, as well as caused by the treatment itself.



Figure 2: X ray both knee including hip joints showing unfused small epiphyses and metaphyseal irregularity

characteristics and mental retardation. He was born with delayed motor and mental milestones of non-consanguineous parents. He could not hear and cannot talk from childhood. On examination, he was disproportionately short statured with a height of 114 cm, weight of 28 kg, BMI of 21.5. He looked dysmorphic, with prominent temporal bones and hypertelorism. He also had proximal myopathy and was unable to walk. On biochemical evaluation, the patient's thyroid-stimulating hormonal (TSH) level was 100 $\mu\text{U/mL}$ (normal reference range 0.47–5.01) with a FT4 of 5.15 pmol/L (normal reference range 9.14–23.18). X ray of his both hands revealed a nonfused epiphysis with a bone age of four (04)

years (Figure 1), and both knee, hip showed epiphyseal and metaphyseal dysplasia (Figure 2). In view of his loss of vision, a CT of the brain was performed, which revealed a large sella and supra

sellar mass compressing the optic chiasma suggested craniopharyngioma (Figure 3).

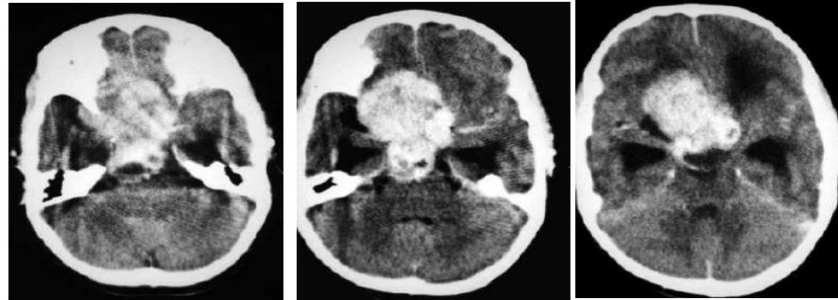


Figure 3: Axial post contrast images of CT scan shows heterogeneously enhancing sella and supra sellar mass

MRI features of brain were consistent with sella-supra sellar complex mass with mass effect

having solid and cystic components suggesting craniopharyngioma (Figure 4).

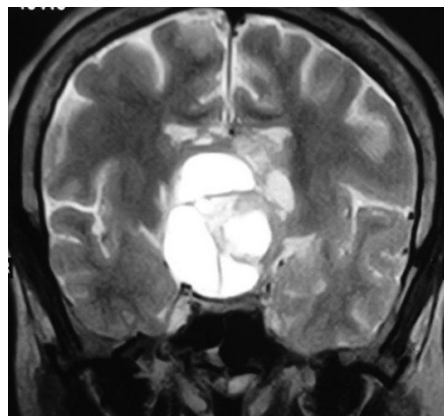


Figure 4: Sagittal and coronal images of MRI of brain shows sella and supra sellar complex mass having cystic and solid components

His hormonal profile revealed a serum cortisol of 67.12 nmol/L (normal reference range 101.2–690.0), serum prolactin 3983.59 mIU/L (normal reference range 945.0–375.0), free testosterone of 11.28 pg/mL (normal reference range 8062–54.69), luteinizing hormone 1.28mIU/mL and follicular stimulating hormone 4.76mIU/mL. USG of neck showed small sized in homogenous thyroid lobes. The patient's short stature and mentally challenged state led to the clinical suspicion of CH. The differential diagnoses considered were pituitary macroadenoma and pituitary pseudotumour caused by long-standing untreated primary hypothyroidism.

DISCUSSION

Congenital hypothyroidism (CH) is a condition of thyroid hormone deficiency present at birth. The prevalence of cretinism in Bangladesh is 0.5% (hilly, 0.8%; flood-prone, 0.5%; and plains, 0.3%) and nearly 69% of Bangladeshi population has biochemical iodine deficiency.¹ Common symptoms include decreased activity and increased sleep, feeding difficulty, constipation, and prolonged jaundice. On examination, common signs include myxoedematous facies, large fontanels, macroglossia, and distended abdomen with umbilical hernia, pseudohypertelorism and hypotonia. Neurologic examination findings include hypotonia with delayed reflexes.⁹ Affected patients with congenital

hypothyroidism could have sensorineural deafness.⁹ In our case, the patient had prior history of poor secondary sexual characteristics and mental retardation and was born with delayed motor and mental milestones. He had deafness and inability to talk since childhood. On examination, he was disproportionately short stature. He looked dysmorphic, with prominent temporal bones and hypertelorism. The diagnosis of congenital hypothyroidism should be and was confirmed by finding an elevated serum TSH and low T4 or free T4 level. In our case, patient had delayed milestone of development, mental retardation, myopathy and walking difficulty. Our hypothyroid patient came for CT and MRI evaluation with history of visual loss for five months. Previous case reports^{4,5,8} either revealed association of craniopharyngioma with congenital hypothyroidism only in younger children or showed craniopharyngioma in children causing hypothyroidism as well as other hormonal deficiencies related to pituitary gland. Children or adolescents with craniopharyngioma showed signs of deficiency of growth hormone, gonadotropin, ACTH as well as ADH or TSH dysfunction.¹⁰ But no previous reported case showed coexistence of craniopharyngioma in an adult person with prior history of untreated congenital hypothyroidism. Our case was an unusual situation of a large craniopharyngioma detected in an adult with untreated congenital hypothyroidism.

Conflict of interest: None

REFERENCES

1. Yusuf HK, Quazi S, Kahn MR, Mohiduzzaman M, Yousuf HKM, Rahman MM, et al. Iodine deficiency disorders in Bangladesh. *Indian J Pediatr.* 1996; 63(1): 105-110.
2. Craniopharyngioma. Medline Plus. Available at: <http://www.nlm.nih.gov/medlineplus/ency/article/000345.html>: Accessed on: 30.07.2018.
3. General Information about Childhood Craniopharyngioma. Available at: <http://www.cancer.gov/types/brain/patient/child-cranio-treatment-pdq>: Accessed on: 10.06.2018.
4. Garnett MR, Puget S, Grill J, Sainte-Rose C. Craniopharyngioma. *Orphanet.* 2007; Available at: http://www.orpha.net/consor/cgi-bin/OC_Exp.php?lng=EN&Expert=54595: Accessed on: 25.01.2019.
5. Bobustuc GC, Jallo GI, DeMonte F, Fuller GN, Groves MD, Hwang LS. Craniopharyngioma. *Medscape online.* Available at: <http://emedicine.medscape.com/article/1157758>: Accessed on: 27.10.2018.
6. General Information about Childhood Craniopharyngioma. National Cancer Institute (NCI). June 10, 2016; Available at: <http://www.cancer.gov/types/brain/patient/child-cranio-treatmentpdq>: Accessed on 10.01.2019.
7. Bobustuc GC, Jallo GI, DeMonte F, Fuller GN, Groves MD, Hwang LS. Craniopharyngioma. *Medscape.* Oct 27, 2014; Available at: <http://emedicine.medscape.com/article/1157758>: Accessed on: 12.01.2019.
8. Hsu EA, Miller JL, Perez FA, Roth CL. Oxytocin and Naltrexone Successfully Treat Hypothalamic Obesity in a Boy Post-Craniopharyngioma Resection. *J Clin Endocrinol Metab.* 2018; 103(2): 370-375.
9. Rastogi MV, LaFranchi SH. Congenital hypothyroidism. *Orphanet J Rare Dis.* 2010. 5: 17. Published 2010 Jun 10. doi: 10.1186/1750-1172-5-17.
10. Halac I, Zimmerman D. Endocrine manifestations of craniopharyngioma. *Childs Nerv Syst.* 2005; 21: 640–648.

Isolated Hydatid Cyst in lung in a 13 Years Old Female patient

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ABSTRACT

Hydatid disease is caused by Echinococcus granulosus; it's transmitted to human through sheep and cattle. People who lived in an endemic area should be suspected to have the disease. Pulmonary hydatid disease can be presented by respiratory manifestations as in our case. We report a case of a female child, 13 years old, who presented with shortness of breath and non-productive cough 2 months ago. The patient had history of fever with weight loss but no other constitutional symptoms or any medical illness. The patient had history of close contact with cattle in her house. On examination, the patient oriented and vitally stable. Both sides of the chest were moving equally with decreased air entry on the right side of the chest. The X-ray shows a large globular mass in right lower chest, while CT scan showed- side. The patient was treated medically. The patient is now receiving Tablet Albendazole 200 mg/BID/Orally for 3months.

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INTRODUCTION

Hydatid disease is a parasitic infestation caused by a tapeworm of the genus *Echinococcus*, which is the larval cystic stage. It's traveled to human through sheep and cattle and the definitive host is the dog. There are four known species of *Echinococcus*-three of them are medically relevant: "*Echinococcus granulosus*, causing cystic echinococcosis (CE); *Echinococcus multilocularis*, causing alveolar echinococcosis (AE); and *Echinococcus vogeli*".¹ Liver and lungs are the most common organs that get infected by echinococcosis disease.² Muscles, brain and kidneys may rarely get involved in the hydatid disease. Pulmonary manifestations include chronic cough either dry or productive,

dyspnoea, pleuritic chest pain and haemoptysis.³ The patient also may be presented by a complication of pulmonary hydatidosis including compression of bronchi or intrabronchial rupture as a result of late diagnosis.³ Hydatid disease rarely infects children, but more common in adult with an average age at diagnosis of 30-40 years.³ Patient with pulmonary hydatid cyst is usually presented by respiratory symptoms including; a dry or a productive cough, chest pain, hemoptysis, dyspnoea, fever or could be presented by the signs of complication as compression of bronchi or intrabronchial rupture.³ If the cyst is ruptured, the patient may develops allergic symptoms and anaphylaxis, transbronchial spread to other lobes, pleural

hydatidosis and pleural effusion.⁷ Therefore those patients may be misdiagnosed by other respiratory diseases as in our case; the patient was first misdiagnosed as having tuberculosis due to respiratory symptoms and cervical lymph node enlargement. The above mentioned data are in keeping with those of Mohsen et al.⁴ who reported a case of hydatid cyst in a 9-year-old child which was misdiagnosed as having plural effusion. Similarly, Fraz et al.³ found in their observational study that dry cough is the commonest symptom among patient with pulmonary hydatid cyst. Imaging studies should be used for the diagnosis and exclusion of another disease that enter in the differential diagnosis of pulmonary hydatidosis. Chest X-ray is a screening test and basic tool. Computerized tomography (CT) can rule out any pulmonary disease, nevertheless, the using of CT is not preferable in children to avoid radiation exposure.⁸ Bronchoscopy usually used as a diagnostic test as well as a therapeutic one for clearance of the obstructed bronchial passages.⁴ An indirect hemagglutination test and enzyme-linked immunosorbent assay can be performed first in acute cases but there are high false positive results due to cross reaction with other helminthic infestation, so we have to use Arc-5 antigen, which is considered the only specific serologic test for hydatidosis.³ Casoni intradermal test and complement fixation test (CFT) are antibody assay tests that have a good accuracy and remain positive even after death of parasite or surgical removal of the cyst. Half of patients with isolated pulmonary cysts lack detectable anti echinococcal antibodies. So the diagnosis can be confirmed by detection of protoscolices or hydatid membranes after percutaneous aspiration of the cyst, guided by ultrasonography and under antihelminthic coverage.² The disease may be misdiagnosed and treated as another respiratory disease.⁴ Transmission of the disease to humans by ingestion of eggs which presented in the contaminated water or food and can be transmitted when they contact with dogs.⁵ There are several domestic animals may be involved as

an intermediate host of echinococcosis transmission such as sheep, pig, goat, camel, deer, and cattle as in our cases.⁶ However, direct transmission from human to human doesn't happen.

The Case

A 13 years old Bangladeshi girl Miss Khadiza, presented to the OPD with complaint of shortness of breath and non-productive cough 2 months ago. The patient had history of fever with weight loss but no other constitutional symptoms or any medical illness.

On examination, the patient was well oriented and vitally stable. There was no lymphadenopathy, bony tenderness or any organomegaly. Both sides of the chest were moving equally with decreased air entry on the right side of the chest. Laboratory results were: CBC {HGB: 10 g/dl, ESR-85mm in 1st hour, WBC: 6500/ml, total circulating eosinophils: 585/cmm, Platelet: 350,000/ml}, Chemistry {Potassium: 4.8 mmol/l, Sodium: 140 mmol/l, Alanine aminotransferase (ALT): 28 U/l, Aspartate aminotransferase (AST): 25 U/l, Direct bilirubin: 1.2 µmol/l. Total bilirubin: 18 µmol/l and total protein: 65g/l} and coagulation profile {PT: 12 seconds, PTT: 29 seconds and INR:1.03}.

Chest X-ray revealed A sharply defined, round homogenous opacity involving right lower chest with almost clear surrounding lung parenchyma (Figure1) and high-resolution computed tomography (HRCT) of chest with contrast revealed a well defined cystic lesion measuring about 5.27×4.37 cm within posterior basal segment of lower lobe of right lung ,rest of the parenchyma appears normal. Post contrast scan revealed no abnormal enhancement.

The visualized parts of abdominal organs appear normal (Figure 2). No calcification of cyst wall noted.

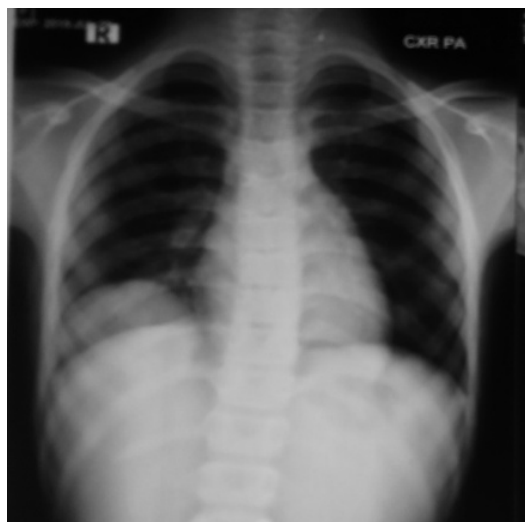


Figure 1: A sharply defined, round homogenous opacity involving right lower chest

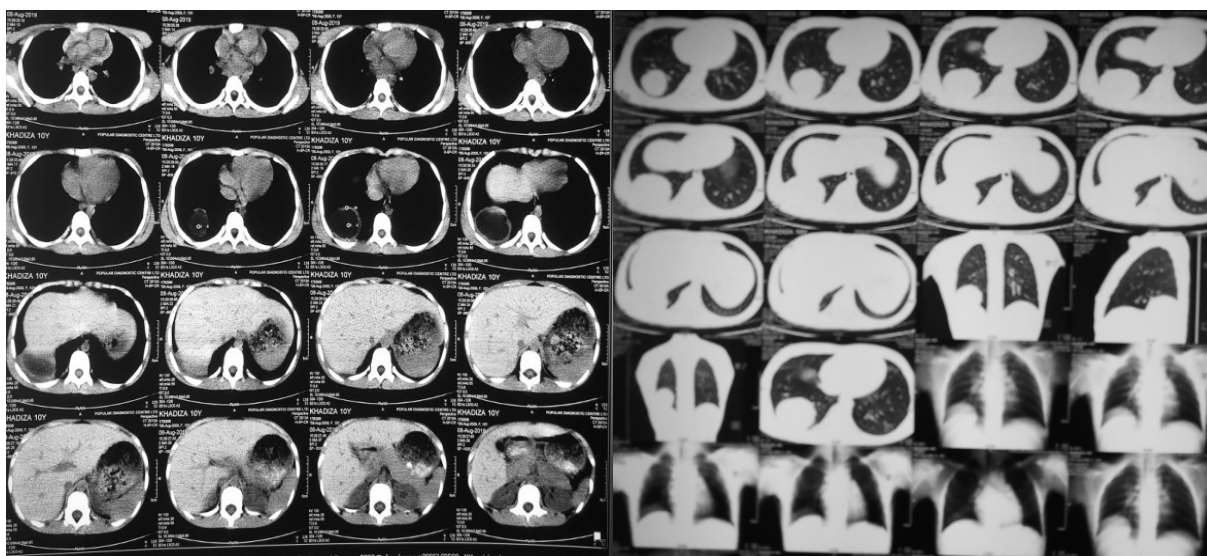


Figure 2: HRCT of Chest with contrast revealed a well defined cystic lesion measuring about 5.27×4.37 cm within posterior basal segment of lower lobe of right lung, rest of the parenchyma appears normal. Post contrast scan revealed no abnormal enhancement. The visualized parts of abdominal organs appear normal.

Patient got admitted into North Bengal Medical College Hospital with a provisional diagnosis of hydatid cyst. USG of abdomen reveals normal. The patient was discharged with albendazole 200 mg/BID/Orally for 3 months and referred to a cardiothoracic surgeon.

DISCUSSION

The prevalence of cystic echinococcosis disease is higher in an endemic area such as Middle east, the central part of Europe, Russia, the Central Asian Republics, China, Northern Japan,

Northwestern Canada, and Alaska.¹ Cysts in the lungs are usually solitary and mostly unilateral. Arinc et al.⁹ reported unilateral cysts in 82.9% of cases. Similarly, Ghoshal et al.¹⁰ reported unilateral single cysts in 81.13% cases. Lower lobe of the lungs is the most common site of pulmonary involvement, and there is a predilection for the posterior segments and the right lung, although Sadrizadeh et al. reported left lower lobe predominance.¹¹ About 60% of cases occur in the lower lobes. Bilateral

involvement occurs in 20% of cases, and multiple cysts in 30% of cases.¹² There are certain unique characteristics of the paediatric hydatid cyst. Unlike an adult, lung involvement is more common than liver in the children, with frequencies of 64% and 28%, respectively.^{12,13} Concomitant hepatic involvements is more common in adults than in children. Kanat et al¹⁴ in a retrospective study reviewed the medical records of 145 patients with hydatid disease hospitalized over the last 10 years. They found a concomitant hepatic cyst in 79% of adults as compared to 33% of children. Therefore, isolated pulmonary cysts are more common in children. The surgical intervention is a definitive treatment for Hydatid cyst. Resection of the cyst can be done with other surgical modalities; lobectomy, wedge resection, pericystectomy, and endocystectomy. It depends on cyst size,⁸ however, we should avoid any aspiration or puncture which can cause allergic reaction and anaphylactic shock.⁸ Albendazole used to avoid recurrent and spread of disease as in our case we used albendazole 200 mg/BID/Orally for 3 months. In conclusion, pulmonary hydatid disease can be presented by respiratory symptoms that may mimic another pulmonary disease, therefore; hydatid disease should be considered as one of differential diagnosis for any patient coming with respiratory symptoms and in those who are live in the endemic area.

Conflicts of Interest: None declared

REFERENCES

1. Wang K, Zhang X, Jin Z, Ma H, Teng Z. Modeling and analysis of the transmission of Echinococcosis with application to Xinjiang Uygur Autonomous Region of China. *J Heor Biol.* 2013; 333: 78-90.
2. Halezeroglu S, Celik M, Uysal A, Senol C, Keles M, Arman B. Giant hydatid cysts of the lung. *J Thoraco Cardiovasc.* 1997; 113: 712-717.
3. Fahim F, Salamah Al SM. Cystic echinococcosis in Central Saudi Arabia: a 5-year experience. *Turk J Gastroenterol.* 2007; 18: 22-27.
4. Sokouti M, Shokouhi B, Sokouti M, Sokouti B. Giant Pulmonary Hydatid Cyst and Trauma in a 9 Year-Old Child: A Case Report. *Open Respir Med J* 2015; 9: 67-69.
5. Madan K, Singh N (2012) Bronchoscopic diagnosis of pulmonary hydatid cyst. *Can Med Assoc J.* 184: E158.
6. Love S. Hydatids—The Basics, NSW DPI Primefact. 2008; 812, NSW.
7. Özdemir A, Bozdemir ŞE, Akbiyik D, Daar G, Korkut S. Anaphylaxis due to ruptured pulmonary hydatid cyst in a 13-year-old boy. *Asia Pac Allergy.* 2015; 5: 128-131.
8. Sinmaz E, Celiksöz A. A Giant pulmonary hydatid cyst treated without lobectomy. *Yonsei Med J.* 2009; 50: 856-858.
9. Arinc S, Kosif A, Ertugrul M, Arpag H, Alpay L, Unal O, et al. Evaluation of pulmonary hydatid cyst cases. *Int J Surg.* 2009; 7: 192-195.
10. Ghoshal AG, Sarkar S, Saha K, Sarkar U, Kundu S, Chatterjee S, et al. Hydatid lung disease: An analysis of five years cumulative data from Kolkata. *J Assoc Physicians India.* 2012; 60: 12-16.
11. Sadrizadeh A, Haghi SZ, Masuom SH, Bagheri R, Dalouee MN. Evaluation of the effect of pulmonary hydatid cyst location on the surgical technique approaches. *Lung India.* 2014; 31: 361-365.
12. Polat P, Kantarci M, Alper F, Suma S, Koruyucu MB, Okur A. Hydatid disease from head to toe. *Radiographics.* 2003; 23: 475-494.
13. Haliloglu M, Saatci I, Akhan O, Ozmen MN, Besim A. Spectrum of imaging findings in pediatric hydatid disease. *AJR Am J Roentgenol.* 1997; 169: 1627-1631.
14. Kanat F, Turk E, Aribas OK. Comparison of pulmonary hydatid cysts in children and adults. *ANZ J Surg.* 2004; 74: 885-889.

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- The author should sign a covering letter mentioning that final manuscript has been seen and approved by all authors. Irrelevant person or without any contribution should not be entitled as co-author. The cover should accompany a list and sequence of all authors with their contribution and signatures.

First title page with author information (1st page should not be numbered).

Title page must include:

- Full title of the article not exceeding 50 characters with a running title for use on the top of text pages.
- Authors' names, highest academic degrees, affiliations and complete address including name of the

departments in which they worked (not where is currently posted), email address & phone number of the corresponding author. The authors should reveal all possible conflicts of interest on this page.

Abstract page (First numbered page)

- Please make abstract page with title of the article and without authors name to make it anonymous for review.
- Prepare structured abstract (with all sections of the text) within 250 words.
- the abstract should cover Background and Purpose (description of rationale for study); Methods (brief description of methods); Results (presentation of significant results) and Conclusion (succinct statement of data interpretation) in a running manner and not under separate headings.
- Do not cite references in the abstract.
- Limit use of acronyms and abbreviations. Abbreviations must be defined at the first mention.
- Include 3-5 key words

The Text

The Following are typical main headings:

- i. Introduction**
- ii. Methods**
- iii. Results**
- iv. Discussion**
- v. Conclusion**

Introduction

Summarize the rationale for the study with pertinent references. The purpose (s) of the study should be clearly elicited.

Methods

Identify type of study and describe the study subjects and methods used with methods of statistical analysis. Cite reference (s) for standard study and statistical methods. Describe new or modified methods. Give proper description of the apparatus (with name and address of

manufacturer) used. ***Generic name of drug must be given. Manuscripts that describe studies on humans must indicate that the study was approved by an institutional Ethical Committee and that the subjects gave informed consent.***

Results

Present only important findings in logical sequence in the text, tables or illustrations with relevant statistics.

Discussion

Emphasize new and important results and the conclusions that follow including implications and limitations. Relate observations to other relevant studies.

Conclusion

Include brief findings and authors suggestions on basis of findings of study.

Acknowledgments

List all sources of funding for the research with contributions of individuals.

References

Accuracy of reference data is the author's responsibility. Verify all entries against original sources especially journal titles, inclusive page numbers, publication dates. All authors must be listed if six or less than six. Use et al, if more than six. Personal communications, unpublished observations, and submitted manuscripts must be cited in the text as "[Name(s)], unpublished data, 20xx)." Abstracts may be cited only if they are the sole source and must be identified in the references as "Abstract". "In press" citations must have been accepted for publication and add the name of the journal or book including publisher. Use Vancouver style, for example:

1. World Health Organization (WHO). WHO Recommendations: Low Birth Weight: preventing and managing the Global Epidemic. Geneva, Switzerland: WHO, 2000 (Technical Report Series no.894)

2. Rashid M. Food and Nutrition. In Rashid KM, Rahman M, Hyder S eds. Textbook of community Medicine and Public Health. 4thed. Dhaka, Bangladesh: RHM Publishers, 2004: p. 156-160.
3. Arefin S, Sharif M, Islam S. Prevalence of pre diabetes in a shoal population of Bangladesh. BMJ. 2009; 12: 155-163.
4. Jarrett RJ. Insulin and hypertension (Letter). Lancet. 1987; ii: 748-749.
5. Reglic LR, Maschan RA: Central obesity in Asian men. J Clin Endocrinol Metab 2001; 89: 113-118 [Abstract].
6. Hussain MN, Kamaruddin M. Nipah virus attack in South East Asia: challenges for Bangladesh. Prime Med Coll J. 2011; I (1): i-ii [Editorial].

Tables:

Each Table must be typed on a separate page. The table number should be followed by a Roman brief informative title. Provide explanatory matter in footnotes. For footnotes use symbol in this sequence; *, **, +, ++, etc.

Figures:

Line drawings, photomicrographs, colour prints and halftones should be camera ready, good quality prints. Submit only originals of laser prints, not photocopies. Original figures must be submitted indicating figure number, short figure title on top of figure lightly in pencil. Any abbreviations or symbols used in the figures must be defined in the figure or figure Legend.