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1. Typing should be on one side of the A4 size paper in English.
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4. All authors' names, posts, affiliation at the time they did their work and their highest academic degree(s), cell phone numbers and e-mail addresses should be included. The name of the corresponding author should be mentioned.
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Requirements

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• A cover letter addressed to the Editor-in-chief of the journal (Sample given at the end)

• A title page

In general, original article should be divided into following sections:

a. Title page
b. Abstract
c. Introduction
d. Methods
e. Results (Tables with titles and Figures with legends)
f. Discussion
g. Conclusion
h. Acknowledgement (if any)
i. References

Each of the sections is to start on a separate page. Page should be numbered consecutively beginning from the abstract.

Title

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• Providing a distinct description of the complete article
• Should include the names of all authors with their designation & their affiliations
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• Subtitles may be used to amplify the main title

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• Statement of the problem with a short discussion of its importance and significance
• Review of the literature(s) related to the problem with pertinent reference
• Objectives/hypothesis/benefits should be clearly stated

Methods

• Research type, place and time
• Description of research variables
• Discussion of research population
• Selection criteria (Exclusion criteria if any)
• Approval of the research involving human subjects by ethical review committee (IRB/ERC) and description of the ethical aspects in such research
• Description of procedure, methods, apparatus, drugs or chemical as applicable
• Statistical procedure should be described and supported by reference

Result

• Present results in a logical sequence in text, table and illustration with most important finding first
• Figure should be numbered as Fig. 1 and table in Roman numerical (Tab. I)
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2. Dr.----------
3. Dr.----------
4. Dr.----------

Editorial

Management of Pregnancy-induced Hypertension

Begum B

Pregnancy-induced hypertension (PIH) is one of the main reasons of maternal, fetal and neonatal morbidity and mortality. Hypertension signals an underlying pathology and may be a pre-existing condition or occurs for the first-time during pregnancy. PIH is defined as having a systolic blood pressure (SBP) > 140 mmHg and the diastolic blood pressure (DBP) being >90 mmHg [1]. PIH refers to one of four conditions including, pre-existing hypertension, gestational hypertension and preeclampsia, pre-existing hypertension and superimposed gestational hypertension with proteinuria and unclassified hypertension[2]. PIH is also known as hypertensive disorder of pregnancy (HDP). It is classified as mild (SBP 140-149 and DBP 90-99 mmHg), moderate (SBP 150-159 and DBP 100-109 mmHg) and severe (SBP>or=160 and DBP>or= 110mmHg). This is a clinically challenging group of pregnancy complications that contribute to maternal morbidity and mortality and ranks second only to hemorrhage as a direct cause of maternal death.

The health concerns and risks that woman with PIH could be exposed to include failure of organs, cerebrovascular events, disseminated intravascular coagulation and higher risks of abruptio placentae. Adding to the concern, women with PIH may have fetuses with a greater risk of prematurity, intrauterine death and intrauterine growth retardation. Risk of hypertension in pregnancy can be reduced with screening, strict management, fetal and maternal monitoring and intrapartum care with advices[3]. Preeclampsia is a serious multi-organ complication in pregnant women, defined by the new onset of hypertension and proteinuria after 20 weeks. Incidence of HDP is 10.3% i.e. 2.7 million cases annually. Incidence of eclampsia is 1.9% so around 0.5 million cases per year (NER) and 19% of maternal deaths are due to hypertension in pregnancy (WHO). Anti-platelet drugs have moderate benefits when used for prevention of PIH.

There are several available screening tests for HDP[4]. For placental perfusion and vascular resistance dysfunction, this includes the roll over test, doppler ultrasound, isometric exercise test, intravenous infusion of angiotensin I I, platelet angiotensin I I binding, platelet calcium response to arginine vasopressin renin and 24 hours ambulatory blood pressure monitoring. When it comes to feto-placental unit dysfunctions, this is inclusive of tests for alpha fetoprotein, human chorionic gonadotropin, estriol, activin A and corticotropin releasing hormone, among others.

Screening is important as it allows early detection and planning of appropriate monitoring strategies, favors early incorporation of appropriate interventions, and identifies most cases likely to be affected by the disease. Therefore, screening will allow more vigilant monitoring of pregnant women and prevent complications of HDP such as eclampsia and others. The current "gold standard" for pre-eclampsia diagnosis involves blood pressure measurement and determination of protein in urine. Due to its syndromic nature and varying clinical presentation of preeclampsia phenotypes, the specificity and reliability of these assessments to predict who will develop HDP or HELLP (H = Hemolysis, EL = Elevated Liver enzymes, LP = Low Platelets) syndrome is poor[5].
PIH treatment is dependent on gestational age, blood pressure levels, and presence of symptoms alongside associated risk factors. A non-drug management approach is recommendable if the range for blood pressure is 140-149 mmHg for SBP between 90-99mmHg for DBP. This recommended threshold for blood pressure varies between distinct health organizations when it

comes to drug management in pregnancy[6]. Regarding assessment of gestational hypertension, a full assessment should be carried out in a secondary care setting by a healthcare professional who is trained in the management of hypertensive disorders of pregnancy. In women with gestational hypertension, the following risk factors that require additional assessment and follow-up need to be accounted for including, nulliparity, aged 40 years and above, calculated body mass index being 35 kg/m^2 and above the interval between pregnancies being over 10 years, multi-fetal pregnancies, family or previous histories relating to pre-eclampsia or gestational hypertension, gestational age at presentation, preexisting kidney disease and preexisting vascular disease. Placental growth factor (PIGF)-based testing can also be offered to help rule out pre-eclampsia (for example, with gestational hypertension) between 20 weeks and up to 35 weeks of pregnancy. Labetalol can be considered to treat gestational hypertension, and nifedipine for women in whom labetalol is not suitable and methyldopa if labetalol or nifedipine are not suitable. The choice is based on careful consideration of side effect profiles, risk and the patient's preferences. Bed rest should not be offered all the time in hospital as a treatment for gestational hypertension[7].

Regarding timing of birth, planned early birth before 37 weeks should not be done in women with gestational hypertension whose blood pressure is lower than 160/110mmHg, unless there are other medical symptoms. When it comes to women with gestational hypertension with blood pressure less than 160/110 mmHg after duration of 37 weeks, birth timing alongside fetal and maternal indications relating to birth should be agreed between the woman and the senior obstetrician. If planned early birth is necessary, a course of antenatal corticosteroids and magnesium sulfate should be offered if indicated. Regarding postnatal investigation, monitoring and treatment, in women with gestational hypertension who have given birth, the blood pressure should be measured at the following intervals: daily for the first 2 days after birth, at least once between day 3 and day 5 after birth and as clinically indicated if antihypertensive treatment is changed after birth.

In women with gestational hypertension who have given birth, antihypertensive treatment should be continued if required. It should be advised to patients that the duration of their postnatal antihypertensive treatment will usually be similar to the duration of their antenatal treatment and antihypertensive treatment should be reduced if their blood pressure falls below 130/80 mmHg.

Women who have undergone treatment for gestational hypertension by taking methyldopa, within 2 days after birth, the dose should not be taken anymore and if necessary, an alternative treatment should be initiated. Antihypertensive treatment should be initiated for women with gestational hypertension who have not undergone antihypertensive treatment previously, in case their blood pressure is 150/100mmHg or higher. Women who have had gestational hypertension and who remain on antihypertensive treatment should be offered a medical review with their general physician or specialist 2 weeks after transfer to community care. All women who have had gestational hypertension should be offered a medical review with their GP or specialist 6-8 weeks after the birth.

This knowledge of the existence of pregnancy with induced hypertension subclasses will be capable of identifying all these groups early in

gestation and allow for the development and implementation of etiology-based treatments aimed at specific subclasses of this disorder. However, there is still no proper research on PIH in Bangladesh despite a 7-10% incidence, depending on seasonal changes, nutritional variation, and the patient's socioeconomic status.

In conclusion, personalized approach along with screening, assessment for management, proper birth planning, post-natal follow-up all link to improve short- and long-term health outcomes for both the mother and the child.

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References:

1. Braunthal S, BrateanuA. Hypertension in pregnancy: pathophysiology and treatment. SAGE Open Med. 2019;7:1-15.

2. Poon LC, Hyett JA. The International Federation of Gynecologyand Obstetrics (FIGO) initiative on pre-eclampsia: a pragmatic guide for first trimester screening and prevention. Int J Gynaecol Obstet. 2019; 145:1-33.

3. Granger JP, Alexander BT, Bennet WA, Khalil-RA. Pathophysiology of pregnancy induced hypertension. Am J Hypertens. 2001;14:178S-85S.

4. Konar H. DC Dutta's Textbook of Obstetrics. 8thed. India: Jaypee Brothers Medical Publishers (P) Ltd; 2020.

5. Royal College of Obstetricians and Gynecologists, Hypertension in Pregnancy, NICE Clinical Guideline 107.

6. World Health Organization, 2011 WHO Recommendations for prevention and treatment of pre-eclampsia and eclampsia.

7. Kintiraki E, Papakatsika S, Kotronis G, Goulis DG, Kotsis V. Pregnancy-Induced hypertension. Hormones. 2015; 14(2):211-23. doi: 10.14310/horm. 2002.1582. PMID: 26158653.

Original Article:

Effect of Ageing on the Concentration of FT3, FT4 and TSH Levels among Different Age Groups

***Kabir F[1], Ali MOI[2], Haque MA[3], Ghosh E[4], Rupa SN[5], Zaman MAU[6]**

Abstract :

Background: *The activities of the thyroid gland depend on the age. However, with advancing age the concentration of FT_3, FT_4 and TSH decrease in apparently healthy elderly persons.* ***Objective:****This cross-sectional study was designed to observe the influence of ageing on thyroid hormone levels.* ***Methods:****This descriptive study was carried out in the department of physiology in collaboration with institute of Nuclear medicine and Allied Sciences, Rajshahi. 120 apparently healthy persons in age group of 8-65 years were studied. Random blood sample was taken to measure the level of free T_3, free T_4, TSH by Radioimmunoassay(RIA) and Immunoradiometric assay(IRMA). Data were analyzed by ANOVA Test.* ***Result:****Significant drop of FT_3 level and non-significant drop of FT_4, TSH level with advancing age.* ***Conclusion:****This study suggested that ageing has effect on FT_3 level more than FT_4 and TSH level.*

Keywords: Free triiodothyronine (FT_3), Free thyroxine (FT_4), Thyroid Stimulating Hormones (TSH), Radioimmunoassay (RIA), Immunoradiometric assay (IRMA).

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Introduction

The thyroid gland synthesizes and releases two hormones thyroxine (T_4) and Triiodothyronine (T_3) and their concentrations are 93% and 7% respectively. Both T_4 and T_3 hormones are iodine containing amino acids. T3 is about four times as potent as T_4, but it is present in the blood in much smaller quantities and persists for shorter time than does T_4[1].

About 99.98% of T_4 in plasma is bound; the free T_4 level is only about 2 ng/dl. Free T_3 accounts for only about 0.5% of the total T_3. The free T_4 in the plasma are physiologically active causing the inhibition of the Thyroid stimulating hormone (TSH) secretion. The free T_4 in plasma is important in the metabolic control of human body and therefore free T_4 is believed to be a direct indicator of thyroid status in an individual. Free T_3 like free T_4 measurement also reflects the thyroid status of individual accurately[2].

The function of thyroid gland controlled by TSH. The secretion of this tropic hormone is in turn regulated in part by thyrotropin releasing hormone (TRH) from hypothalamus and is subjected to 'negative feedback control' by high circulating levels of thyroid hormones acting on the anterior pituitary and hypothalamus[1].

Ageing is a physiological process which is characterized by a progressive generalized impairment of many functions of the body resulting in the loss of adaptive responses to stress and a growing risk of age associated disease. However, after the age of 60 years a person is commonly known as old aged person, when it involves progressive loss of cells, reduced metabolic activities and decreased efficiency of many functions of different organs[3]. Ageing is not solely an intrinsic process, as it also occurs in the context of an individual's interaction with the environmental factors such as lack of exercise, poor diet, cigarette smoking and heavy alcohol consumption. Moreover, genetic factor also plays a role in causing physiological changes in ageing process[3].

The relation of the thyroid gland to the ageing

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process is of interest because of the importance of the organ in regulating the rates of various body functions. It was found that during a normal human life span, serum T_3 remains high during adolescence, then it remains stable until late middle age and ultimately decrease with their increasing age[4].

Rahman *et al*.[3]and Dambal *et al*.[5] found that FT_3 and FT_4 level were declined with increasing age but TSH levels raised as the age advanced. But Dika *et al*.[6] found no statistically significance difference of T_3, T_4 and TSH with increasing age.

Wllke *et al.*[7] found that free T_4 progressively decreased. But Peeters et al.[8] observed serum TSH and T_3 level decrease with age whereas serum free T_4 level usually remains unchanged.

In addition Kumari *et al*.[9] and Bremner *et al*.[10] found FT_4 level remain almost static throughout all decades of life and TSH level Increase with age.

So, the present study has been designed to find out the normal level of FT_3, FT_4 and TSH among different age.

Materials and Methods

This cross-sectional descriptive study was carried out in the department of Physiology in collaboration with institute of Nuclear medicine and Allied Sciencesbetween the period of January 2016 to December 2016. The protocol of this study was approved by Institutional Review Board (IRB) and Ethical Review Committee (ERC) of Rajshahi Medical College. Apparently 120 healthy persons aged 8-65 years were selected residing in Rajshahi City. Purposive sampling technique was applied to select each subject. Subject having history of thyroid disorder, chronic illness, renal failure, malignancy, cirrhosis of liver, diabetes mellitus, pregnancywas excluded from this study.

After proper counseling, the aim, objectives, benefit, risk and procedure of the study were explained in details to the subjects. After taking informed consent, complete history taking and physical examination were done and recorded in a preformed data sheet.After breakfast, 5ml of venous blood sample were drawn into the test tube (from the anticubital space of the forearm) by venipuncture after taking all aseptic precautions. After coagulation serum was separated by centrifugation at 3500 rpm for 2 minutes.Then serum was used for estimation of FT_3, FT_4 and TSH level by Radioimmunoassay (RIA) and Immunoradiometric assay (IRMA). The results of TSH were expressed in µIU/ml and FT_3 and FT_4 were expressed in fmol/ml. Collected data were analyzed by using SPSS (statistical package for social sciences) computer software programmed (version-20) and the tests of significance were calculated by using ANOVA test. P value at or below 0.05 was taken as level of significance.

Result

Table-I: Serum free triiodothyronine (FT_3) levels in different age groups (n=120).

Age group in years (including both male and female)	FT_3 fmol/ml (mean±SD)	p-value
Group 1 (8-17 yrs) Group 2 (18-40 yrs) Group 3 (41-65 yrs)	8.01±2.32 8.21±1.75 7.42±1.23	0.144 ^{ns}

Level of significance among different age group figured by ANOVA test

	FT_3 fmol/ml
(Group 1) Vs (Group 2)	0.907^{ns}
(Group 2) Vs (Group 3)	0.018^{ns}

ns=Not significant (p=>0.05).
s=Significant (p=<0.05)

The mean FT_3 levels is almost same in adolescent group and in people up to 40 years of age. After that slight decrease of FT_3 level is seen. FT_3 level showed significant variation between people from 18 to 40 years and people whose age is above 40 years shown in table I.

The test of significance was calculated using ANOVA test.

Table-II: Serum free thyroxine (FT_4) levels in different age groups (n=120).

Age group in years (including both male and female)	FT_4 fmol/ml (mean±SD)	p-value
Group 1 (8-17 yrs)	22.10±5.88	0.932 ^{ns}
Group 2 (18-40 yrs)	21.46±12.53	
Group 3 (41-65 yrs)	22.42±5.94	

Level of significance among different age group figured by ANOVA test

	FT_4 fmol/ml
(Group 1) Vs (Group 2)	0.170^{ns}
(Group 2) Vs (Group 3)	0.979^{ns}

ns=Not significant (p=>0.05).

FT_4 level does not differ significantly among different age group. Which was shown in table II.The test of significance was calculated using ANOVA test.

Table-III: Serum thyroid stimulating hormone (TSH) levels in different age groups (n=120).

Age group in years (including both male and female)	TSH µIU/ml (mean±SD)	p-value
Group 1 (8-17 yrs)	2.62±3.50	0.862 ^{ns}
Group 2 (18-40 yrs)	3.17±7.60	
Group 3 (41-65 yrs)	2.59±3.90	

Level of significance among different age group figured by ANOVA test

	TSH µIU/ml
(Group 1) Vs (Group 2)	0.346^{ns}
(Group 2) Vs (Group 3)	1.00^{ns}

ns=Not significant (p=>0.05).

TSH level does not differ significantly among different age group which was shown in table III.The test of significance was calculated using ANOVA test.

Discussion:

In this study, significant drop of FT_3 level and non-significant drop of FT_4, TSH level with advancing age. These findings are compatible with Ahmed *et al*.[11], Khan *et al*.[12], Suzuki *et al*.[13], Abbas *et al*.[14], Alom *et al*.[15] and Kumari *et al*.[9]. Ageing affect FT_3 level more than FT_4 level.[9] It occurs due to reduced secretion and concentration of FT_3 levels and increaseturnover rate of FT_3[11]. FT3 levels decrease with advancing age and slightly increased level of FT_4 in older age, due to primary retardation process for hormone metabolism within the cell which is associated with ageing process, increase degradation rate of thyroid hormone in old age[15]. Decline of hepatic 52-deiodinase activity with ageing that may reduce peripheral conversion of T_4 to T_3 which leads to higher level of FT_4 but lower level of FT_3[16].

On contrary, Dika *et al*.[6] showed non-significant influence of aging on FT_4, FT_3 and TSH level. It may be due to narrow age range of their sampling which failed to find out influence of aging process.However, there is increased level of FT_4, FT_3 and decrease level of TSH concentration in advanced age[3]. This discrepancy may be due to the increased requirement of these hormones for the various biochemical and physiological function of the body[12].

This study showed higher FT_4 and TSH concentration in (8-17) years age group in comparison to all other groups. Almost similar type of result observed by other previous studies[4]. This finding represents that marked changes occur in thyroid function during puberty as an adaptation to physical and sexual development. Adaptation of hypothalamo-pituitary-thyroid axis during puberty in response to increase energy expenditure may be the reason[5].

One of the strengths of this study is that we have included only healthy persons (both males and female) who are absolutely free from any diseas-

es. So, it was possible for us to detect the variation of FT3, FT4 and TSH level in different age.

Conclusion :

After analyzing the result of the study, it can be concluded that ageing has significant effect on the level of FT_3, without significant effect on the level of FT_4 and TSH.

Limitations :

- The sample size was small.
- Only healthy persons range from 8 to 65 years were included in the study.
- Infant, newborn baby, pregnant women were not included in this study.
- Seasonal variation was not considered.

Recommendation :

- A study with larger population should be conducted.
- Infant, newborn, pregnant women, BMI group should be included in the study for understanding their thyroid status.
- Comparative study between different variables should be done.

References

1. Guyton AC, Hall JE. Thyroid metabolic hormones, Textbook of Medical Physiology. 12th Ed.Singapore: Harcourt Publication, W.B. Saunders Company; 2011. 907-916.

2. Ganong WF. The Thyroid gland, Review of Medical Physiology. 24th edition.New Delhi:Tata McGraw Hill Education Private Limited;2012. 339-351.

3. Rahman S, Jahan N, SultanaN. Age related change in thyroid function.J Bangladesh Soc Physiol.2012; 7(2): 72-77.

4. Kaur G, Kalstra L, Sadhoo AK. Age and sex related changes in thyroid functions in normal healthy subjects of Jammu region.JK Science. 2007; 9(3): 132-136.

5. Dambal AA, Padaki S, Herur A, Kashinakunti SV. Manjula R, Gurupadappa K. Thyroid status in relation to age and gender-A cross sectional study.MRIMS Journal of Health Sciences. 2013; 1(2): 37-40.

6. Dika H, Kasolo J, Bimenya G. Thyroid hormones profile in students of Makerere College of Health Sciences in Kampala Uganda.Tanzania Journal of Health Research. 2010; 12(1): 1-7.

7. Wllke TJ. Influence of age and sex on the concentration of free thyroxin in serum and on the free thyroxin: total thyroxin ratio.Clin. Chem. 1983; 29(7): 1428-1430.

8. PeetersRP.Thyroid hormones and ageing. Hormones.2008; 7(1): 28-35.

9. Kumari T, Prasad A, Sinha KK, Bharti MLG, Satyam K. Age and sex specific thyroid hormone profile in euthyroid subjects.J Biochem Tech.2015; 6(3): 1008-1012.

10. Bremner AP, Feddema P, Leedman PJ, Brown SJ, Beilby. JP, Lim EM, Wilson SG, O'Leary PC, Walsh JP.Age-related changes in thyroid function: A longitudinal study of a community-based cohort.J Clin Endocrinol Metab.2012. 97(5): 1554-1562.

11. Ahmed Z, KhanMA, Haq Au, Attaullah S, Rehman JU. Effect of race, gender and age on thyroid and thyroid stimulating hormone levels in North West Frontier Province, Pakistan. J Ayub Med Coll Abbottabad. 2009; 21(3): 21-24.

12. KhanA, Akhter S, Siddiqui MM, Khan MMA, Nawab G. Effect of age, sex and seasons on the concentration of thyroid and thyroid stimulating hormones. The Sciences.2001; 1(4):224-227.

13. Suzuku S, Nishio SI, Takeda T, Komatsu M. Gender-specific regulation of response to thyroid

hormone in ageing.Thyroid Research.2016; 5(1): 56-59.

14. Abbas R, Abbas HG, Shahid A. Chand S, Nawaz. S. Reference intervals for free T3 and Free T4 in Pakistani euthyroid patients: effect of age and gender on thyroid function.Journal of the College of Physicians and Surgeons Pakistan.2014; 24(11): 806-809.

15. Alom MJ, Islam MR, Haque MM. Gender, age and race related thyroid hormones People of normal healthy subjects in Northern Area, Bangladesh.IJSR.2016; 5(10): 506-509.

16. Chaurasia P, Modi B, Mangukiya S, Jadav P, Shah R. Variation in thyroid hormones level among people of different age, gender and seasons, Piparia, Gujarat. National Journal of Medical Research.2011; 1(2): 57-59.

Original Article

Histological Features Of Alopecia Areata and its Associated Diseases

***Basak AK[1], Debnath J[2]**

Abstract :

Background: *Alopecia areata (AA) is an autoimmune disease that presents as nonscarring hair loss, although the exact pathogenesis of the disease remains to be clarified. AA can affect any hair-bearing area. It often presents as well demarcated patches of nonscarring alopecia on skin of overtly normal appearance. The presence of AA is associated with a higher frequency of other autoimmune diseases.* ***Objective:*** *To evaluate the histological features of alopecia areata and its associated diseases.* ***Materials and methods:*** *It was a cross sectional study carried out in the Department of Dermatology and Venereology, KYAMC, Enayetpur, Sirajgonj during the period of January 2020 to December 2020. Patients suffering from alopecia areata of scalp with or without involvement of other sites. Total 50 sample were included in this study. Data were collected by structured questionnaire. statistical analysis was performed with SPSS (Statistical package of social science) windows version 25.* ***Results:*** *This study found that alopecia areata is more common among the age group of 21-30 years. Among respondents 27(54%) were within this group. Mean age of this study participant was 28.42±7.85 years with a range of 18 to 50 years. Among them 29(58%) were male and 21(42%) were female. Majority (78%) were seen on the scalp and 22% were seen in face. Majority (92%) were duration of disease 1-3 years and only 8% were 3-5 years. Most of the patients, 39 (78%) were in the chronic stage. It was revealed that 16% respondent had autoimmune disease.* ***Conclusion:*** *This study shows most common associated skin disease in hypothyroidism among the systemic diseases and chronic stage. Early diagnosis and treatment of alopecia areata can reduce the burden of associated diseases.*

Key words: Biofilm, tissue culture plate method

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Introduction:

Alopecia areata (AA) is a non-scarring hair loss with an unpredictable course and a wide spectrum of manifestations. It affects both genders equally with a cumulative lifetime incidence of about two percent and no significant racial predominance[1-2]. It is an autoimmune disease with a variable, typically relapsing or remitting especially when hair loss is extensive. Alopecia areata is the second-most frequent non-scarring alopecia, after male and female pattern alopecia. Clinical patterns of hair loss in alopecia areata are usually very distinct. The most common pattern is a small annular or patchy bald lesion (patchy alopecia areata), usually on the scalp to total loss of scalp hair only (alopecia totalis), and total loss of all body hair[3].

The pathophysiology of alopecia areata remains unknown. The most widely accepted hypothesis is that alopecia areata is a T-cell mediated autoimmune condition that is most likely to occur in genetically predisposed individuals[4]. In the histological assessment of scalp biopsy specimens from alopecia areata, the diagnostic pathologic feature is peribulbar lymphocytic inflammation ("Swarm of bees") affecting anagen follicles or follicles in early catagen. The normal structural features of the anagen follicle and of the hair follicle cycle is needed for study of the abnormal changes involved in the histopathology of alopecia areata[5]. Alopecia areata is a multifactorial disease with autoimmune components, which although seen in genetically predisposed individuals, the real causes have yet to be determined and various factors should be considered. There are indications for a T-cell-mediated autoimmune process directed against an unknown autoantigen of the hair follicle. T lymphocytes that have been shown to be oligoclonal and autoreactive are predomi-

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nantly present in the peribulbous inflammatory infiltrate. Alopecia areata frequently occurs in association with other autoimmune disorders such as vitiligo, lichen planus, morphea, lichen sclerosis, pemphigus foliaceus, atopic dermatitis, Hashimoto's thyroiditis, hypothyroidism, endemic goiter, Addison's disease, pernicious anemia, lupus erythematosus, diabetes mellitus, Down's syndrome and others[6]. The purpose of this study was to evaluate the pathological changes of alopecia areata and it's associated with disease.

Materiais and Method

It was a cross sectional study carried out in the Department of Dermatology and Venereology, KYAMC, Enayetpur, Sirajgonj during the period of January 2020 to December 2020. Patients suffering from alopecia areata of scalp with or without involvement of other sites. Total 50 sample were included in this study. Data were collected by structured questionnaire. A careful history was taken from each patient regarding the presence of atopy or other autoimmune diseases. A family history of alopecia areata, atopy and autoimmune diseases were also recorded. Data were collected by structured questionnaire. Statistical analysis was performed with SPSS (Statistical package of social science) windows version 25.

Results

Table I: Demographic characteristics of the patients

Characteristics	Frequency	Percent
Age in years		
≤20	7	14.0
21-30	27	54.0
31-40	12	24.0
41-50	4	8.0
Mean±SD	28.42±7.85	
Sex		
Male	29	58.0
Female	21	42.0
Male female ratio	1.3:1	

Table II: Clinical presentation of the study subject

Clinical presentation	Frequency	Percentage (%)
None	7	14.0
Patch (1-2)	36	12.0
Patch (3-4)	9	18.0
Patch (5-6)	4	8.0

Table III: Distribution of Alopecia Aerata according to site

Site of hair loss	Frequency	Percent
Scalp	39	78.0
Face	11	22.0

Table IV: Distribution of duration disease in study subject (n=50)

Duration	Frequency	Percentage
1-2 years	46	92.0
3-5 years	4	8.0

Table V: Distribution of duration disease in study subject (n=50)

Stages of alopecia areata	Frequency	Percentage
Acute	4	8.0
Subacute	7	14.0
Chronic	39	78.0

Table VI: Associate with systemic disorders of the study subject (n=50)

Systemic diseases	Frequency	Percentage
Hypothyroidism	2	4.0
Diabetes	1	2.0
Anaemia	2	4.0
Bronchial Asthma	1	2.0
Leukonychia	1	2.0
Lichen Planus	1	2.0
None	42	84.0

Discussion

Alopecia areata (AA) is a clinically distinctive form of 'non-scarring' alopecia that is rarely biopsied and hence, many pathologists are unfamiliar with its interpretation. The role of a pathologist is vital when dealing with atypical presentations, such as patients progressing to scarring, use of topical medications that alter the picture and of late, in trying to provide prognostic information[7]. This present study demonstrated that the histological features of alopecia areata and its associated diseases. The present study findings were discussed and compared with previously published relevant studies.

This study found that alopecia areata is more common among the age group of 21-30 years. Among respondents 27(54%) were within this group. Mean age of this study participant was 28.42±7.85 years with a range of 18 to 50 years. These findings consistent with other studies[8,9].

In this study shows among them 29(58%) were male and 21(42%) were female. These findings consistent with Husain et al. they revealed that males (56.7%) were predominant than that of females (43.3%). This result also supported by Sharma et al[8]. They showed that 61.2% patients were male and 38.8% were female in their study.

This study shows majority (78%) were seen on the scalp and 22% were seen in face. Therefore, the findings of the study are in well agreement with the findings of the other research works[10]. Similar study Sharma et al. they reported eyebrows alopecia in 5.2% of their patients along with scalp alopecia. This result is not consistent with the present study[9].

This study shows majority (92%) were duration of disease 1-3 years and only 8% were 3-5 years. Similar study Chaitra et al. the duration of the lesions at the time of biopsy ranged from 3 months to 5 years[7].

Present study revealed that most of the patients, 39 (78%) were in the chronic stage. Therefore, the findings of the study are in well agreement with the findings of the other research works.10 Similar study Whiting11 they studied 50 patients among them 31 (62%) patients were in chronic stage of alopecia areata.

From the present study it was revealed that 16% respondent had autoimmune disease. Therefore, the findings of the study are in well agreement with the findings of the other research works[10]. Similar study Whiting[11]. found that in 20% to 30% of patients were associated with other autoimmune disease. Bronchial asthma was seen in 1.0 % cases which is lower to the prevalence reported by Gopal et al.12 (4.2%). Diabetes mellitus was seen in 1.0 % cases. Hypothyroidism was seen in 5% cases, while Gopal et

al[12]. reported 2.5 % cases with hypothyroidism. Lewinski and Broniarczyk-Dyla et al[13]. also confirmed the frequent coexistence of alopecia areata and thyroid abnormalities. Hypothyroidism, bronchial asthma and diabetes mellitus have a proposed autoimmune etiology. Muller and Winkelman[14] studies showed association of alopecia areata with atopy in 18% of children and 9% of adults. These associations trends are available that could indicate that alopecia areata could be an autoimmune disorder. But further studies are needed in this regard.

Conclusion

The present study concludes alopecia aerata is more prevalent in males with scalp and face are the most common areas involved. Most common associated skin disease and hypothyroidism among the systemic diseases. It can be recommended that transverse histologic section of the biopsy specimen may be taken to see the alterations of terminal and vellus hair ratio which is also helpful to differentiate the chronic stage from other stages of alopecia areata. Thus, this study recommends that there is a need for early diagnosis and treatment of alopecia areata which can reduce the burden of various dermatological and systemic diseases.

References:

1. Rajabi F, Drake LA, Senna MM, Rezaei N. Alopecia areata: a review of disease pathogenesis. Br J Dermatol. 2018;179(5):1033-1048.

2. Fricke ACV, Miteva M. Epidemiology and burden of alopecia areata: a systematic review. Clinical, cosmetic and investigational dermatology 2015;8: 397.

3. Pratt H, King LE, Messenger AG, Christiano AM, John P. Sundberg JP. Alopecia areata. Nat Rev Dis Primers. 2017;3:17011.

4. Ahmed Z, Banik RL. Histopathological changes in different stages of alopecia areata. Mymensingh Medical Journal 2010;19(1):100-5.

5. Ioffreda MD. Inflammatory diseases of hair follicles, sweat glands and cartilage', In: Elder DE, Elenitsas R, Johnson BL, editors. Lever's Histopathology of the skin. Philadelphia: Lippincott Williams Wilkins. 9th ed. 2005;480-485.

6. Thomas E.A, Kadyan R.S; Alopecia and autoimmunity : A clinical study; Indian Journal of Dermatology; 2008;53(2); 70-74.

7. Chaitra V, Rajalakshmi T, Kavdia R. Histopathologic Profile of Alopecia Areata in Indian Patients. Int J Trichology. 2010; 2(1): 14-17.

8. Husain MA, Alam MN, Rahim R, Joarder Y, Wahiduzzaman M, Ferdous M, Afroz F. Study on Clinical and Histopathological Changes of Alopecia Areata. Medicine Today 2018;30(1):30–33.

9. Sharma VK, Dawn G, Kumar D, 1996, ' Profile of alopecia areata in northern India', International Journal of Dermatology 1996;35(1): 22-27.

10. Kumar S, Bali S, Mittal3 C, Agarwal A. A Study Of Alopecia Areata And Its Associated Diseases At A Tertiary Care Hospital. IOSR Journal of Dental and Medical Sciences 2018;17:17-20.

11. Whiting DA. Histopathologic features of alopecia areata- A new look. Arch dermatol 2003;139:1555-1559.

12. Gopal MG, Praveen Kumar S, Sharath Kumar B.C, Ramesh M; A Clinico-Investigative study of Alopecia Areata with special reference to its Association With Various Systematic and Dermatological Disorders; Journal of Evolution of Medical and Dental Sciences 2013; 2(Issue 48): 9239 – 9249.

13. Lewinski A, Broniarczyk-Dyla G, Sewerynek E, Zerek-Melen G, Szkudlinski M. Abnormalities in structure and function of the thyroid gland in patients with alopecia areata. J Am AcadDermatol. 1990;23:768–9.

14. Muller HK, Winkelmam RK. Alopecia areata. Arch Dermatology 2013;88: 290-97.

Original Article

Study of Histo-morphological Patterns of Colonic Polyps

***Kundu R[1], Hassan A[2], Jannat H[3], Bosu SK[4], Jeba R[5]**

Abstract :

Background:*Colorectal carcinoma is the third most common cancer worldwide and the fourth most common cause of death.Among the key risk factors colonic adenoma is a factor closely linked to the development of colon cancer. Development of colorectal adenocarcinoma principally occurs via adenoma- carcinoma sequence of a multiple step process of tumor progression. This results from accumulation of genetic changes in the cells of intestinal mucosa.* ***Objectives:****To detect histomorphological subtypes of colonic polyps.****Methods:****This is a cross-sectional observational study conducted in the Department of Pathology, Dhaka Medical College, Dhaka from March 2017 to January 2019 with 54 colonic polyp patients attending in Dhaka Medical College Hospital, Dhaka. All obtainted samples are processed and selected for routine histopathological study . Pertinent demographic data including patient's age and diagnosis were collected from pathology requisition forms. Statistical analysis was carried out as required.* ***Result:****In this study, 29.6% hyperplastic polyps that was the most common type of colonic polyps and 22.5% adenomatous polyps. Most of them were male predominance and mean age was 34.03±19.85. Rectum was the most common site of colonic polyps about 48.1%.* ***Conclusion:*** *Colonic adenoma is a factor closely linked to the development of colon cancer. Thus, the management of adenomas has an important role in the prevention of colorectal cancer.*

Keywords: Colonic polyp, colorectal carcinoma,histopathological study.

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Introduction

Colorectal cancer is a major cause of morbidity and mortality throughout the world. It accounts for over 9% of all cancer incidence. It is the third most common cancer worldwide and the fourth most common cause of death.[1] Among the key risk factors colonic adenoma is a factor closely linked to the development of colon cancer. Thus, the management of adenomas has an important role in the prevention of colorectal cancer.[2]

Polyp is a grossly visible protrusion from a mucosal surface.[3] Polyps are most common in colorectal region but may occur in esophagus, stomach, small intestine etc.[4] Polyp may develop as a result of epithelial or stromal cell hyperplasia, inflammation, ectopia or neoplasia.[5] Colonic polyps can be classified as non-neoplastic and neoplastic polyp. The non-neoplastic polyps are classified as inflammatory, hamartomatous and hyperplastic polyp. The neoplastic polyps are adenomatous polyp.[5] Hyperplastic and adenomatous polyps are by far the most common polyp.[6]

Tubular adenomas account for more than 80 percent of colonic adenomas which are less likely to become malignant. Villous and tubulovillous adenomas account for 5 to 15 percent of adenomas. Their malignant potentiality is about 15-25% when their size is more than 2cm. [6]

It has been estimated that 15% of all adenomas measuring >1 cm will progress to carcinomas within10 years of their detection and overall chance of developing carcinoma in a polyp is estimated at 5%.[7] The appearance of adenomas and their progression to adenocarcinomas is the result of an accumulation of genetic changes in cells of the intestinal mucosa that have been inherited or acquired during life [2]. So, characteristics histological features of colonic polyps could be valuable parameters for selecting patients seemed to be most deserving of close surveillance in follow-up cancer prevention programs from the total adenoma population. [8]

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Materials and Methods

This study was conducted in Department of Pathology , Dhaka Medical College during the period from March 2017 to February 2019. After approval from the institutional ethics committee, 54 patients were successively assigned in this study who had colonic polyps of any age group patients. Patients with current or previous history of colorectal neoplasm were excluded from the study. All obtainted samples are processed and selected for routine histopathological study.After obtaining informed written consent from the patients, a descriptive cross-sectional study was carried out to detect histomorphological subtypes of colonic polyps.

The collection data were cleaned, edited and analyzed by using computer based SPSS (Statistical Package for Social Science) software Version 19.0 for windows. Data was classified into group, frequency observed and descriptive status (mean, median, mode, standard deviation) was calculated.

Results

This cross-sectional descriptive study included 54 patients who had colonic polyps.Total 63 cases should be included according to sample size but 9 cases could not be included due to time limitation. Finally 54 sample was enrolled in the study. These colonic polyps obtained by colonoscopically and surgically resected. Histopathological examinationwith hematoxylin & eosin stain. The results are as follows:

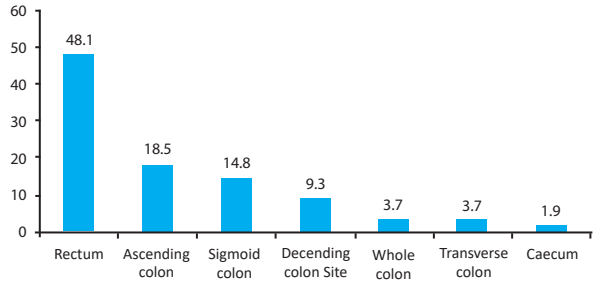

Figure 1: Site distribution of colonic polyps. (n=54)

Table-I: Distribution of the study patients according to age group and sex.(n=54)

Demographic parameters	Number of patients	Percentage
Age (years)		
< 20	15	28
20-30	9	16.9
31-40	8	14.9
41-50	12	22.2
51-60	7	13.1
> 60	3	5.7
Mean ± SD	34.03 ± 19.85	
Range (min-max)	3-75	
Sex		
Male	32	59.3
Female	22	40.7

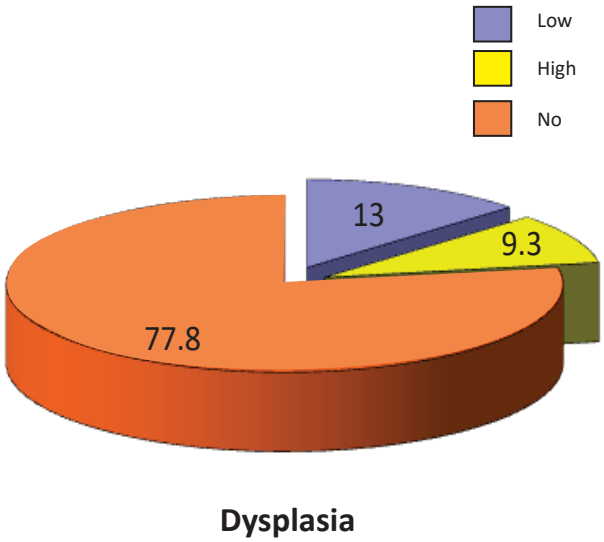

Figure 2: Dysplasia of colonic polyps . (n=54)

Discussion

The concept that colorectal cancers may arise from pre-existing adenomas is now widely accepted, based on epidemiological, clinical, postmortem, and molecular biological studies.[9] In this study the mean age of the patients was 34.03±19.85 with age ranges from 3-75 years. The highest number of cases were in the second decades (28%) . But different findings were stated in the study by 10Suheil. and Mahdi., 2015 in which most age group affect by colonic polyps include age group of 36-50 that have high percentage.

More than half 32(59.3%) of the patients were male and 22(40.7%) patients were female. This indicates male predominance of colonic polyps. A Study by[8] Nussrat et al., 2011 also showed gender distribution of colorectal adenoma cases were male predominance 28 (60%) compared with female 19 (40%).

Regarding the site distribution of this study patients, it was observed that about 48.1% polyps were in rectum which was disagreed with previous study by[2] Sousa et al., 2012 and Nussrat et al., 2011. They showed that the distal site of the colon was the predominant with 53.2%, 34% in the proximal site and 17% in the rectum.

Of all the 54 cases of colonic polyps in our study showed six different types of colonic polyps includes: hyperplastic polyps: 29.6%, juvenile polyps : 25.9%, adenomatous polyps: 22.5% and inflammatory polyps: 16.7%, mesenchymal polyps: 3.8% and peutz jeghurs polyps: 1.9%. In our study the commonest (29.6%) polyp was hyperplastic polyps.Another study of 50 colonic polyps performed by[10] Suheil. and Mahdi., 2015 showing four different types of polyps includes: adenomatous polyps 32%, inflammatory polyps 30% , hyperplastic polyps 20% and juvenile polyps 18%.

In this study, about 77.8% patients were without dysplasia; 13.0% patients were low grade dysplasia and 9.3% patients were high grade dysplasia. There were 57% cases with low grade dysplasia, and 43% cases with high grade dysplasia in a study by[8] Nussrat et al., 2011 which was similar to our study according to frequency of low grade dysplasia.

From this study it can be stated that colonic polyps were male predominance with average mean age presentation was 34.03±19.85 and age range 3-75 years. According site distribution 48.1% colonic polyps were in rectum. Most common type polyps were hyperplastic polyps (29.6%) and most were pedunculated (50%).

Conclusion

Colorectal cancer is the third most common cancer worldwide in both sexes. Colonic adenoma is a factor closely linked to the development of colorectal carcinoma. In this study 29.6% hyperplastic polyps that was the most common type of colonic polyps. Most of them were male predominance and mean age was 34.03±19.85. Rectum was the most common site of colonic polyps about 48.1%. This study also showed that 22.5% are adenomatous polyps and among them 9.3% patients were high grade dysplasia.

The concept that colorectal cancers may arise from pre-existing adenomas is now widely accepted. Finally expression of biological markers may be added to the future histopathological evaluation and play a role in planning the follow up of patients with colonic polyp.

References

1. Haggar FA, Boushey RP. Colorectal cancer epidemiology: incidence, mortality, survival and risk factors. Clinics in colon and rectal surgery 2009; 22(4):191.

2. Sousa WATD, Rodrigues LV, Silva Jr RGD and Vieira FL. Immunohistochemical evaluation of p53 and Ki-67 proteins in colorectal adenomas. Arquivos de gastroenterologia 2012 ; 49(1): 35-40.

3. Fenoglio Preiser CM and Hutter RV. Colorectal polyps: pathologic diagnosis and clinical significance. CA. A cancer journal for clinicians 1985; 35(6): 322-344.

4. Eshghi MJ, Fatemi R, Hashemy A, Aldulaimi D. and Khodadoostan M. A retrospective study of patients with colorectal polyps. Gastroenterology and Hepatology from bed to bench 2011; 4(1): 17.

5. Kumar V, Abbas AK and Aster JC. Robbins and Cortan pathologic basis of disease. 9th ed. Philadelphia: Saunders; 2015.

6. Odze RD and Goldblum JR. Surgical pathology of the GI tract, liver, biliary tract and pancreas: Elsevier Health Sciences; 2009 ; Part-1: 481-534.

7. Winawer SJ, Zauber AG, Fletcher RH et al. Guidelines for colonoscopy surveillance after polypectomy: a consensus update by the US Multi Society Task Force on Colorectal Cancer and the American Cancer Society. CA.A cancer journal for clinicians 2006; 56(3): 143-159.

8. Nussrat FL, Ali HH, Hussein HG, and Al-Ukashi RJ. Immunohistochemical expression of ki-67 and p53 in colorectal adenomas: A clinicopathological study. Oman medical journal 2011; 26(4): 229.

9. Atkin WS and Saunders BP. Surveillance guidelines after removal of colorectal adenomatous polyps. Gut 2002 ; v6-v951: (suppl 5).

10. Suheil SS and Mahdi LH. The Expression of P53 and ki67 in Colonic Polyps. Kufa Journal for Nursing Sciences 2015 ; 5(2).

Original Article

A Study of Height and Width of the Pedicles of Human Dry Fifth Lumbar Vertebrae

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Abstract:

Background: *Lumbar segment is the most mobile part of human spine and thus most vulnerable to wear and tear. Minute anatomic details of lumbar vertebra is essential to know for its repair procedures.* ***Objective:*** *This study was carried out with an attempt to construct data on horizontal & transverse diameters of the pedicles of 153 fully ossified dry human fifth lumbar vertebrae.* ***Method:*** *This was a cross sectional, analytic type of study which was carried out on 153 dry fifth lumbar vertebrae that are fully ossified, complete & morphologically normal bones. This study was performed in the Department of Anatomy, Sir Salimullah Medical College, Dhaka from January 2012 to December 2012.* ***Result:*** *This study observed that the mean ± SD of pedicle height was 10.4 ± 1.6 mm on left & 10.9 ± 1.8 mm on right side in male. The mean ± SD of the same variables was 9.4 ± 1.4 mm on left & 9.7 ± 1.5 mm on right side in female. The mean ± SD of pedicle width was 16 ±2 mm on left & 15.3 ± 2 mm on right side in male. The mean ± SD of the same variables was 13.5 ± 2.2 mm on left & 13.3 ± 2.5 mm on right side in female. All values were significantly higher in male than that of female.* ***Conclusion:*** *The size of pedicle of fifth lumbar vertebrae varies in accordance to ethnic as well as racial variations. Bangladeshi people have their own morphological variations of fifth lumbar vertebra in comparison to western citizens.*

Keywords: Lumbar vertebra, pedicle height, pedicle width

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Introduction

The vertebral column is the axial skeleton which acts as a firm support to the body, transfer the body weight to the legs, enclose & protects the spinal cord& cauda equina[1].Due to present lifestyle & with its speed, the incidence of assaults on the vertebral column is increasing[2].Since the lumbar segment is the mobile part of vertebral column, it is subject to instability following trauma, in particular that related to road traffic accidents, the use of heavy mechanical devices & adventure sports apart fromnumerous orthopedic disorders such as prolapsed intervertebral discs, spondylosis, kyphosis, scoliosis, ankylosing spondylitis & neoplastic metastases[3]. Therefore, it may require immobilization of the vertebral column for its activity to be regained[4]. With the help of screw, various devices like rods, plates, wires etc. can be applied to spine for immobilization or fixation[5].The fifth lumbar vertebra consists of a body in front and a neural arch behind.The pedicle connects the neural arch to the body.It is short thick, dorsal projections from the superior part of body at the junction of its lateral & dorsal surfaces[6]. Its upper margin forms the superior vertebral notch & lower margin forms the inferior vertebral notch & both contribute to corresponding intervertebral foramen containing spinal nerves[7]. As pedicle is the strongest part of the vertebra made of entirely cortical bone with a small core of cancellous bone, so it has become the preferred anchoring site for fixation[8]. Pedicle screws allow short segment & rigid fixation[9]. Transpedicular screw fixation of spine is becoming increasingly popular as it is more stable & versatile because it

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provides three dimensional fixations. In several studies, researchers demonstrated fusion rates of 90% or greater with pedicle screw fixation[10]. The success of this technique depends upon the ability of the screw to obtain & maintain purchase within the vertebral body. This is determined, among other factors, by the choice of screw for a particular pedicle size[11]. A screw that is larger than the pedicle may result in cortex perforation or even break the pedicle. Knowledge of the minimal dimensions is, therefore needed before inserting a screw into a pedicle[12]. The complications associated with oversized pedicle screw are dural tears, leakage of C.S.F & injuries to nerve roots7. Morphometric data on the dimensions of the pedicles are therefore useful in preoperative planning & in the designing of pedicle screws[11]. Being part of vertebral body, pedicle is subject to ethnic variations as reported in various studies. Hence, ethnic specific data on pedicle morphometry is necessary to avoid misplacement & inappropriate size of implants[13].

Objectives

The aim of the present study is to collect data on the different dimensions of the pedicles of adult dry fifth lumbar vertebrae in the Bangladeshi population to establish normative data & to find out whether they differ from those of other population.

Materials and Methods

Operational definition for the variables used in this study:

Pedicle height (PH):Minimum vertical distance between the upper and lower borders of the lateral surface of the pedicle7.

Figure 1: Measurement of pedicle height

Pedicle width (PW): Minimum transverse distance between the medial and lateral borders of the superior surface of the pedicle7

Figure 2: Measurement ofpedicle width

Result

Sex of the collected bones were determined by using discriminant function analysis formula[14]& other sex differentiating features of the fifth lumbar vertebra. Then grouping was done (Table 1). To evaluate the significance of the results obtained unpaired Student's 't' test were carried-out between male & female.

Table I: Grouping of the samples

Sex	No
Male	74
Female	79

Table II:Pedicle height at the left & right side of fifth lumbar vertebrae in male & female in mm

	Pedicle height		
Sex	**Left Mean + SD**	**Right Mean + SD**	**Comb. Total Mean + SD**
Male (n=74)	10.4 ± 1.6 (7.32 - 14.5)	10.9 ± 1.8 (7.42 - 15.9)	10.7± 1.7
Female (n=79)	9.4 ± 1.4 (5.41-12.21)	9.7 ± 1.5 (6.36 -15.34)	9.6 ±1.5
p value	<0.001***<0.001***		

Figure in parentheses indicate range. Comparison between sex was done by unpaired Student's 't' test, *** = significant, Comb. = Combined.

Table III:Pedicle width at the left & right side of fifth lumbarvertebrae in male & female in mm

	Pedicle width		
Sex	**Left Mean + SD**	**Right Mean + SD**	**Comb. Total Mean + SD**
Male (n=74)	16 ± 2 (10.82 -20.88)	15.3 ± 2 (9.9 - 20.38)	15.6± 2.0
Female (n=79)	13.5 ± 2.2 (7.51-17.23)	13.3 ± 2.5 (7.4 -18.59)	13.4 ±2.4
p value	<0.001***<0.001***		

Figure in parentheses indicate range. Comparison between sex was done by unpaired Student's 't' test, *** = significant, Comb. = Combined.

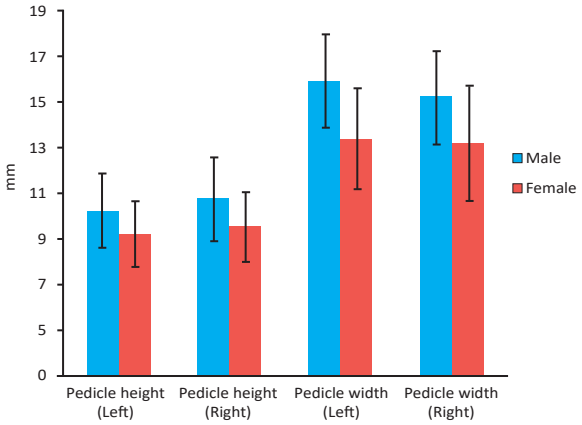

Figure 3: Bardiagram showing pedicle height and pediclewidth of 5th lumbar vertebrae of left and right side in male and female

Discussion:

This study was carried out with an aim to establish the normal ranges of diameters of the pedicle height & width of 153 adult human dry fifth lumbar vertebrae.These bones of unknown sex were subjected to discriminant function analysis for determination of sex & morphological measurements. The results of the current study were compared withthe results of different researchers of abroad.

The calculated results of the present study showed greater mean +SD of pedicle height in male than that of female in both left and right side which were statistically significant (p<0.001).The mean of both left and right sided values of the present study were significantly dissimilar (0.001) with that of Nojiri[15], Singe[17] and Jariyapong[16] conducting the study on the Japanese, Gujaratian and Thai population respectively. The observed differences might be due to different food habits and cultures which exhibit different patterns of stress on skeleton. The results of the present study was also in contrary with that of cadaveric study by Attar et al.[17]on Turkish (male p<0.001, female p= 0.080), MRI study by Karabekir et al.[18] on Turkish (p<0.001) and Radiological study by Amaza et al.[19] on Nigerian (p<0.001) &Amonoo-Kuofi[20] on Saudi people. Different measurement techniques might

be the cause of this dissimilarity.

In the present study, the mean + SD of pedicle width of both left & right side were greater in male than that of female which were statistically significant (p<0.001). Singe[17] and Jariyapong[16] found significant dissimilarity with that of the present study by conducting the study on the people of Gujarat (male p<0.01, female p<0.001) and Thailand (p<0.001). In cadaveric study on Turkish, Attar et al.[17]found significant dissimilarity (p<0.001) than that of the present study in case of male and similarity (p=1.000) in case of female. Amonoo-Kuofi[20] on Saudi &Amaza et al.[19] on Nigerian (p<0.001)by radiological study, Sugisaki et al.[9]by computed tomographic study on the people of Chicago and Karabekir et al.[18]by MRI study on Turkish (p<0.001) people found dissimilarity to that of the present study. Difference in the properties between dry and living bones might be the cause of this variation.

Conclusion

A comparison of the present study with the published data supports the view that there are ethnic as well as racial variations in the size of pedicle of fifth lumbar vertebrae. So, it is necessary to compile tables that are applicable to every population. This study showed that height & width of the lumbar pedicle were higher in males than in females & this can be explained in terms of the greater upper body weight of males. There are also significant differences in pedicular morphology in Bangladeshi population when compared with western people. This may be due to ethnic related morphologic differences as Bangladeshi have noticeably smaller body build than their western counterparts. The results of the present study provide useful information for safe surgery of posterior segmental screw fixation & for the development of new spinal implant system.Further progressive study with larger sample size with known age, sex, stature, ethnicity & comparative study between dry bone and living bone by radiological methods are recommended.

References

1. Snell RS. Clinical Anatomy by Regions, 8th edition. Philadelphia: Lippincott Willams and Wilkins, 2008; 851-898.

2. Chandanwale AS. Giripunje NB, Rajput Y, Laganathan B, Jadhao AB, Nawale SN. Morphometric study of lumbar pedicles in Indian population. Journal of Orthopaedics 2009; 6(4):9-17.

3. Nachemson AL. The lumbar spine: An orthopaedic Challenge. SPINE 1976; 1(1): 59-71.

4. Chawla K, Sharma M, Abhaya A, Kocchar S. Morphometry of the lumbar pedicle in North west India, 2011;15(3):155-161.

5. Wolf A, Shoham M, Michale S, Moshe R. Morphometric study of lumbar spine for operation-workspace specifications. SPINE, 2001; 26(22): 2472-2477.

6. Arora L, Dada R, Singh V. Morphometric study of lumbar pedicles in Delhi regions of Northern India. Indian Journal for the practicing doctor, 2006; 3(5):11-12.

7. Singel TC, Patel MM, Gohil DV. A study of width & height of lumbar pedicles in squrashtra region. J Ana Soc India 2004; 53(1):4-9.

8. Praleash, Pravhu LV, Vadgaonkar R, Pai MM, Ranade AV & Singh G. Morphometry of vertebral pedicles: A comprehensive anatomical study in the lumbar region. International Journal of Morphology 2007; 25(2):393-406.

9. Sugisaki K, Howard S, Orfas AAE, Rhim R, Anderson GBJ, Inoue N. In vivo three-dimensional morphometric analysis of the lumbar pedicle isthmus. SPINE, 2009;34(24):2599-2604.

10. Ajay SC, Nilesh BG, Yuvraj R, Bharath L, Amit BJ, Shasas Morphometric study of lumbar pedicles in Indian population. Journal of Orthopaedics 2009; 6(4):51-62.

11. Anonoo-Kuofi HS. Maximum and minimum

lumbar interpedicular distances in normal adult Nigerians. Journal of Anatomy 1982; 135(2):225-233.

12. Chaynes P, Sol JC, V?aysee JB, Lagarrigue J. Vertebral pedicle anatomy in relation to pedicle screw fixation: a cadaver study. Surg adio Anat 2000; 23:85-90.

13. Acharya S, Dorje T & Srivastav A. Lower dorsal & lumbar pedicle morphometry in Indian population. SPINE 2010; 35(10):378-384.

14. Allbright AS. Sexual Dimorphism in the vertebral column. Master of Arts. Knoxville:The University of Tennessee, 2007:29-124.

15. Nojr K, Matsumoto M, Chiba K & Toyama Y. Morphometric analysis of the thoracic and lumbar spine in Japanese on the use of pedicle screws.Surgical Radiological Anatomy 2005; 22(4):326-332.

16. Jariyapong P & Mahakkanukrauh P. Pedicle morphology of the lumbar vertebra in Thais. Srinagar ind Med J 2007; 22(4):326-332.

17. Attar A, Ugur HC, Ayson UZ, Tekdemir I, Egemen N & Gene Y. Lumbar pedicle: surgical anatomic evaluation & relationships. European Spine Journal 2001; 10:10-15.

18. Karabekir HS, Mas NG, Edizer M, Ertekin T, Yaziki C & Atamturk D. Lumbar vertebra morphometry and steriological assessment of intervertebral spac volumentry: Amorphological study. Annals of Anatomy 2011; 193: 231-236.

19. Amaza DS, Rufai AA, Jacks TW, Zirahei JV, Ahmed A & Humman LL. Estimation of sex using demarking points from diameters of lumbar pedicles in Adult Nigerians. British Journal of Medicine & Medical Research 2012; 2(4):553-567.

20. Amonoo-Kuofi HS. Age related variations in the horizontal and vertical diameters of the pedicles of the lumbar spine. Journal of Anatomy 1995; 186: 321-328.

Original Article

E. faecalis are common for biofilm production than E. faecium and tissue culture plate method is gold standard for biofilm detection in Enterococci.

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Abstract :

Objective: *The ability of Enterococci to produce biofilms is fundamental in causing endodontic and urinary tract infections, as well as endocarditis. The aim of the study were to investigate that E. faecalis are common for biofilm production than E. faecium and tissue culture plate method (TCP) is gold standard for biofilm production in Enterococci.* ***Materials &Method:*** *Biofilm of Enterococci was detected by tissue culture plate method, tube method and congo red agar method.* ***Results:*** *Biofilm formation were more in E. faecalis than E. faecium (P = 0.005) and the TCP method was considered the gold standard method for detection of biofilm in Enterococci(sensitivity and specificity 100%).* ***Conclusion:*** *The purpose of the study was to show biofilm producing Enterococci and the gold standard method of biofilm detection in Enterococci.*

Key words: Biofilm, tissue culture plate method

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Introduction:

Biofilm are defined as microbial derived sessile communities characterized by the cells that are irreversibly attached to a substratum or to each other. They are embedded in a matrix of extracellular polymeric substance they are produced, and exhibit an altered phenotype with respect to growth rate and gene transcription[1]. With a biofilm bacteria communicate with each other by production of chemotactic particles or pheromones, a phenomenon called quorum sensing[2]. Availability of key nutrients, chemotaxis towards surface, motility of bacteria, surface adhesins and presence of surfactants are some factors which influence biofilm formation[2]. According to a publication by the National Institutes of Health, more than 80% of all infections involve biofilms. Biofilm are associated with many medical conditions including indwelling medical devices, dental plaque, upper respiratory tract infection, peritonitis and urogenital infections[3]. Both gram positive and gram negative bacteria have the capability to form biofilms. Bacteria commonly involved include Enterococcus faecalis, Staphylococcus aureus, Staphylococcus epidermidis, Viridans streptococci, Escherichia coli, Klebsiella pneumoniae, Proteus mirabilis and Pseudomonas aeruginosa[4]. The two most common Enterococci species are Enterococcus faecalis and Enterococcus faecium, both are capable of producing biofilms, which consists of a population of cells attached irreversible on various biotic and abiotic surfaces, encased in a hydrated matrix of exopolymeric substances[5].

Materiais and Method

This cross-sectional study carried out at the department of microbiology, Dhaka Medical College from January 2015 to December 2015. Among 350 urine samples, 30 Enterococcus faecalis and 10 Enterococcus faecium were

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detected by culture and PCR method. Among the Enterococci, biofilm detection was done by tube method, congo red agar method and tissue culture plate method.

Tube method

This is a qualitative method for biofilm detection. A loopful of test organisms was inoculated in 10 ml of brain heart infusion broth (BHIB) with 1% glucose in test tubes. The tubes were incubated at 370C for 24 h. After incubation, tubes were decanted and washed with phosphate buffer saline (pH 7.2) and dried. Tubes were then stained with crystal violet (0.1%). Excess stain was washed with deionized water. Tubes were dried in inverted position. The scoring for tube method was done according to the results of the control strains. Biofilm formation was considered positive when a visible film lined the wall and the bottom of the tube. The amount of biofilm formed was scored as 1-weak/none, 2-moderate and 3-high/strong. The experiment was performed in triplicate and repeated three times.[6]

Congo Red Agar method

A simple qualitative method to detect biofilm production by using Congo Red Agar (CRA) media. CRA medium was prepared with brain heart infusion broth (Oxoid, UK) 37 g/L, sucrose 50 g/L, agar No. 1 (Oxoid, UK) 10 g/L and Congo Red indicator (Oxoid, UK) 8 g/L. First Congo Red stain was prepared as a concentrated aqueous solution and autoclaved (121ºC for 15 minutes) separately from the other medium constituents. Then it was added to the autoclaved brain heart infusion agar with sucrose at 550C. CRA plates were inoculated with test organisms and incubated at 370C for 24 h aerobically. Black colonies with a dry crystalline consistency indicated biofilm production. The experiment was performed in triplicate and repeated three times.

Tissue Culture Plate Method (TCP)

The microorganisms are grown in polystyrene tissue culture plates for 24 hours then after washing, fixed with sodium acetate (2%) and stained with crystal violet (0.1% w/v). Biofilm formation is detected by measuring optical density with ELISA reader[6].

The organisms were grown overnight in brain heart infusion broth (BHIB) with 0.25% glucose at 37°C. The culture was diluted 1:40 in TSB 0.25% glucose, and 200 µl of this cell suspension was used to inoculate sterile 96 well polystyrene microtiter plates. After 24h at 370C, wells were gently washed three times with 200 µl of phosphate buffered saline (PBS), dried in an inverted position, and stained with 0.1 % crystal violet for 15 min. The wells were rinsed again, and the crystal violet was solubilized in 200 µl of acetone (80:20, vol/vol). The optical density at 595nm (OD595) was determined using a microplate reader. Each assay was performed in triplicate and repeated three times[7].

Calculation of OD values

OD value was calculated by using the following method.The average OD values were calculated for all tested strains and negative controls, since all tests were performed in triplicate and repeated three times. Second, the cut off value (ODc) was established. It was defined as three standards (SD) above the mean OD of the control: ODc = average OD of negative controls + (3 X SD of negative control). Final OD value of a tested strain was expressed as average OD value of the strain reduced by ODc value (OD = average OD of a strain – ODc). ODc value was calculated for each microtiter plate separately. If a negative value is obtained, it should be present as zero, while any positive value indicates biofilm.

Results

Among 42 isolates, TCP, detected 28 (66.67%) biofilm producers. By TM, the number of biofilm

producers were 22 (52.38%) and non-biofilm producers were 20. Very different results were observed by the CRA method, with which only 11 (26.19%) were biofilm producers. Among 30 E. faecalis, 25 (83.33%) were biofilm producers and among 10 E. faecium, 3 (30%) were biofilm producers. Biofilm formation were more in E. faecalis than E. faecium (P = 0.005).

Table I: Screening of the isolates for biofilm by Tissue culture plate (TCP), Tube method (TM) and Congo Red Agar (CRA) method (N=42)..

Method	E. faecalis (N=30) n (%)	E. faecium (N=10) n (%)	Unidentified (N=2) n (%)	Total n (%)
TCP	25(83.33)	3 (30.00)	0(0.00)	28(66.67)
TM	19 (63.33)	3 (30.00)	0(0.00)	22(52.38)
CRA	10 (30.00)	1 (10.00)	0(0.00)	11(26.19)

N = Total number of Enterococci.
n = Number of biofilm formation.

A Significant difference was observed in biofilm formation among E. faecalis and E. faecium (P = 0.005).Statistical analysis of Tissue Culture plate, Tube method and Congo Red Agar method. The TCP method was considered the gold-standard for this study and compared with data from TM and CRA methods. Sensitivity and specificity of TM was 78.57% and 100% respectively. For CRA methods, sensitivity and specificity were 25% and 71.42% respectively.

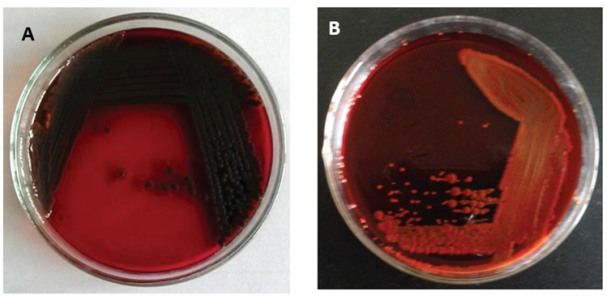
Table II: Statistical analysis of tissue culture plate, tube method and Congo Red Agar methods for biofilm detection of Enterococcus

Method	Sensitivity	Specificity	PPV	NPV	Accuracy
TCP	100%	100%	100%	100%	100%
TM	78.57%	100%	100%	100%	85.71%
CRA	25%	71.42%	63.63%	32.25%	40.47%

Discussion

In the present study, 83.33% E. faecalis were biofilm producers and 30% E. faecium were biofilm producers and these results are similar to the data reported by Baldassarriwhere 80% for E. faecalis and 48% for E. faecium isolated from infected patients which were able to form biofilm[8]. Seno reported that 100% E. faecalis isolates isolated from urinary tract infection were capable of producing biofilm[9]. In this study, E. faecalis produced biofilm more than E. faecium (P = 0.005). Other investigators have reported that E. faecalis (95%) isolates produce a biofilm more often than E.faecium (29%).

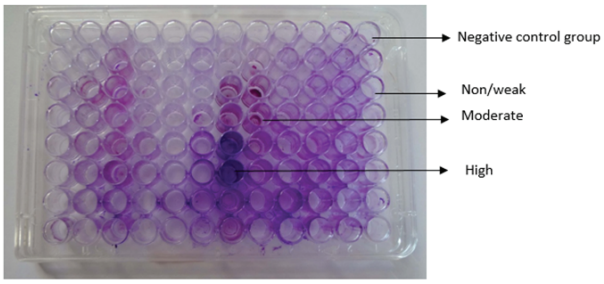
In the present study, the TCP method was considered the gold-standard and compared with data from TM and CRA methods. Hasan also considered the TCP method as a gold-standard for their study compared with TM and CRA methods. Parameters like sensitivity, specificity, positive predictive value, negative predictive value and accuracy were calculated[10]. In the present study, sensitivity, specificity, PPV, NPV and accuracy of TM was 78.57%, 100%, 100%, 70% and 85.71% respectively. Similar results were reported in other study where sensitivity, specificity, PPV, NPV and accuracy was 73%, 92.5%, 94%, 66% and 80% respectively[10]. In this study, for CRA method, sensitivity and specificity remained low which were 25% and 71.42% respectively. Hasan reported that sensitivity and specificity were 11% and 92% respectively for CRA method[10].



Enterococci in congo red agar media.

Left: (A) black crystalline colonies of biofilm positive strain.

Right: (B) pinkish-red colonies of biofilm negative strain.



Screening of biofilm producers of Enterococci by TCP method.

Conclusion

Results of the present study showed that the presence of biofilm is more common in E. faecalis than E. fauciumand among the three procedure of biofilm detection in Enterococci,tissue culture plate method is the gold standard method.

References:

1. Donlan RM, Costerton W. Biofilms: Survival mechanisms of clinically relevant Microorganisms. Clin microbial Rev, 2002; 15(2): 167-93.

2. Thomas D, Day F. Biofilm formation by plant associated bacteria. Annu Rev Microbiol, 2007; 61: 401-22.

3. Reid G. Biofilms in infectious disease and on medical devices. Int J Antimic Ag, 1999; 11: 223-6.

4. Donlan RM. Biofilms and device-associated infections. Emerg Infect Dis, 2001; 7(2): 277-81.

5. Mohamed JA, Huang DB. Biofilm formation by Enterococci. J Med Microbiol 2007; 56: 1581-8.

6. Christensen GD, Simpson WA, Younger JA, Baddour LM, Barrett FF, Melton DM, et al. Adherence of coagulase negative Staphylococci to plastic tissue cultures: a quantitative model for the adherence of Staphylococci to medical device. J Clin Microbiol, 1985; 22(6): 996-1006.

7. Toledo-Arana A, Valle J, Solano C, Arrizubieta MJ, Cucarella C, Lamata M, Amorena B, Leiva J, Penades JR and Lasa I. The Enterococcal Surface Protein, ESP, Is Involved in Enterococcus faecalis Biofilm Formation. Appl Environ Microbiol, 2001; 67(10): 4538-4545.

8. Baldassarri L, Cecchini R, Bertuccini L, Ammendolia MG, Losi F, Arciola CR, et al. Enterococcus spp. Produces slime and survives in rat peritoneal macrophages. Med Microbial Immunol, 2001; 190: 113-120.

9. Seno Y, Kariyama R, Mitsuhata R, Monden K & Kumon H. Clinical implications of biofilm formation by Enterococcus faecalis in the urinary tract. Acta Med Okayama, 2005; 59: 79-87.

10. Hassan A, Usman J, Kaleem F, Omair M, Khalid A, Iqbal M. Evaluation of different detection methods of biofilm formation in the clinical isolates. Braz J Infect Dis, 2011; 15 (4); 305-311.

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