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Picture Archiving and Communication System in Modern Health Care Facilities

Dr. Moffazal Sharif, Assistant Professor, Department of Radiology and Imaging, Khwaja Yunus Ali Medical College, Sirajganj

PACS (picture archiving and communication system) is a medical imaging technology which provides economical storage and convenient access to images from multiple modalities like X-ray plain film, computed tomography (CT) and magnetic resonance imaging (MRI). The principles of PACS were first discussed at meetings of radiologists in 1982.[1] Dr Harold Glass, a medical physicist working in London in the early 1990s secured UK Government funding and managed the project over many years which transformed Hammersmith Hospital in London as the first filmless hospital in the United Kingdom. Dr Glass died a few months after the project came live but is credited with being one of the pioneers of PACS. The first large-scale PACS installation was in 1982 at the University of Kansas, Kansas City. This first installation became more of a teaching experience of what not to do rather than what to do in a PACS installation. As electronic images and reports are transmitted digitally via PACS, this eliminates the need to manually file, retrieve, or transport film jackets. The universal format for PACS image storage and transfer is DICOM (Digital Imaging and Communications in Medicine). A PACS consists of four major components, the imaging modalities such as X-ray , CT and MRI, a secured network for the transmission of patient information, workstations for interpreting and reviewing images and archives for the storage and retrieval of images and reports. Combined with available and emerging web technology, PACS has the ability to deliver timely and efficient access to images, interpretations, and related data.[2-4] PACS replaces hard-copy based means of managing medical images, such as film archives. With the decreasing price of digital storage, PACS provide a growing cost and space advantage over film archives in addition to the instant access to prior images at the same institution. Digital copies are referred to as Soft-copy. It expands on the possibilities of conventional systems by providing capabilities of off-site viewing and reporting (distance education, telediagnosis). It enables practitioners in different physical locations to access the same information simultaneously for teleradiology. PACS provides the electronic platform for radiology images interfacing with other medical automation systems such as Hospital Information System (HIS), Electronic Medical Record (EMR), Practice Management Software, and Radiology Information System (RIS). It is also used by radiology personnel to manage the workflow of patient examination.[5] A full PACS provides a single point of access for images and their associated data. That is, it should support all digital modalities, in all departments, throughout the enterprise. However, until PACS penetration is complete, individual islands of digital imaging not yet connected to a central

PACS may exist. These may take the form of a localized, modality-specific network of modalities, workstations and storage (a so-called "mini-PACS"), or may consist of a small cluster of modalities directly connected to reading workstations without long term storage or management. Such systems are also often not connected to the departmental information system. Historically, Ultrasound, Nuclear Medicine and Cardiology Cath Labs are often departments that adopt such an approach. In the US PACS are classified as Medical Devices, and hence if for sale are regulated by the USFDA. In general they are subject to Class 2 controls and hence require a 510 (k), though individual PACS components may be subject to less stringent general controls.[2,4] The Society for Imaging Informatics in Medicine (SIIM) is the world wide professional and trade organization that provides an annual meeting and a peer-reviewed journal to promote research and education about PACS and related digital topics.

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The Following are typical main headings:

i. Introduction
ii. Materials and Methods
iii. Results
iv. Discussion and Conclusion

Introduction

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

Materials and 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.

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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

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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.4th ed. Dhaka, Bangladesh: RHM Publishers, 2004: pp. 156-160.
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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].

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Kidney Screening and Estimation of Glomerular Filtration Rate by CG, MDRD and CKD-EPI Equations in Healthy Adults of Dhaka

*Md. Shariful Haque,[1] Md. Azizul Hoque,[2] Harun Ur Rashid,[3] Muhammad Rafiqul Alam,[4] Md. Shahidul Islam[5]

Received : August 20, 2016 Accepted : September 24, 2016

Abstract

Introduction: *Kidney disease screening should be done to detect kidney disease early. Blood pressure measurement and simple tests like urine examination, blood sugar, creatinine measurement and GFR estimation (eGFR) by creatinine based equations can detect early renal impairment. Among different GFR estimation equations, CG and MDRD are most widely used. The newer CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration) equation by Levey ASin 2009 has claimed superiority in terms of improved precision, accuracy and less bias. This equation has been used in many countries including India and found consistently improved performance. To our knowledge it was the first epidemiologic study applying CKD-EPI equation in Bangladesh.*

Methods: *We have conducted a population based cross-sectional observational study involving these 3 equations among 498 healthy adult volunteers in urban area of Dhaka from May 2010 to December 2010.*

Results: *Mean age of adult population (n-410) was 36.81±12.17. Mean creatinine 89.125± 13.82 µmol/L.CKD-EPI equation yielded highest eGFR. Estimated GFR with CG equation (CG-CCr), BSA adjusted/corrected CG (CG-GFR) equation, MDRD and CKD-EPI equation was 83.13±18.69 ml/min, 86.71±17.60ml/min/1.73^2, 84.80±17.67ml/min/1.73^2, and 89.92 ± 18.69 ml/min/1.73^2 respectively. Groups were significantly different from one another in multivariate analysis of variance (ANOVA) ($p<0.05$). Estimated GFR by CKD EPI equation is similar with measured GFR by DTPA renogram among healthy volunteers in a study conducted in CMH, Dhaka. Despite higher creatinine (92.04±12.75 vs 82.48±13.91 µmol/L in female), males have had higher GFR by all GFR estimation equations. Population having GFR less than 60 ml/min was 10.7% in CG-CCR, and 6.8% and 5.9% by MDRD and CKD-EPI equations respectively.*

Conclusion: *Despite a number of limitations such as purposive sampling, small sample size and single based institution, this study was meant to estimate GFR by different equations. Till now CKD-EPI equations is the best method to estimate GFR.*

Key words*: eGFR, MDRD, CKD-EPI*

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Introduction

Evidence from the Western countries is emerging that migrant populations of South Asian origin have a higher risk for chronic kidney disease (CKD) than the native whites.[1–3] Though widely used, creatinine is not a robust marker of kidney damage at early stage. An individual must lose 50% of their kidney function before the serum creatinine will begin to rise.[4] GFR is usually accepted as the best overall index of kidney function. Normal GFR varies according to age, sex, and body size; in young adults it is approximately 120-130 mL/min/1.73 m^2 and declines with age. Normal range of glomerular filtration rate (GFR) is significantly lower in Indian population compared to western population. Apparent low GFR among healthy Indians are physiological and is not are flection of any chronic subclinical subtle renal impairment.[5] Low nephron mass due to genetic predisposition also postulated. Moreover anthropometric measures like body surface area is different in south Asian population. GFR <60 ml/min/1.73 m^2 for at least three months, with or without kidney damage is a criterion for CKD. Decline in GFR precedes creatinine rise, that's why different authorities [The National Kidney Disease Education Program (NKDEP) of the National Institute of Diabetes,Digestive and Kidney Diseases (NIDDK), National Kidney Foundation (NKF) and American Society of Nephrology (ASN)] recommend estimating GFR from serum creatinine based GFR prediction equations.[6] Until now there are no fewer than 46 GFR prediction equations of which CG and MDRD is most widely used.[7] The newer CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration) equation by Levey AS published in 2009 has claimed superiority in terms of improved precision, accuracy and less bias.CG equation over estimate GFR when GFR is actually low, while MDRD under estimate GFR in healthy adult by about 30% and is not applicable below 18 years of age. CKD-EPI equation yield higher GFR, consequently lowers CKD prevalence and applied to all age and race. In NHANES, (National Health and Nutrition Evaluation Survey, USA) the median estimated GFR was 94.5 ml/min/1.73 m^2 vs. 85.0 ml/min/1.73 m^2, and the prevalence of chronic kidney disease was 11.5% versus 13.1% in CKD-EPI and MDRD equations.[8]

Materials and Methods

This population based cross sectional observational study was done from May 2010 to December 2010 by non-random purposive sampling among healthy volunteers. A total of 498 (Four hundred and ninety eight) adult respondents (18 and above) having no diabetes, hypertension or known acute or chronic illness were included. Study population was selected on two screening programme among morning exercisers in Ramna park, Shahbag, Dhaka. Fresh urine was tested for glucose, blood, protein, nitrite and leucocyte esterase with dipstix using 10 para 'Uric10 CF' reagent strips. About 3 ml of blood was centrifuged at the screening venue and serum was sent for measurement of serum glucose (random) and creatinine at Kidney Research Laboratory, Department of Nephrology, Bangabondhu Sheikh Mujib Medical University, Shahbag, Dhaka.

Serum creatinine was measured by alkaline picrate (Jaffe) kinetic method (without deproteination). A random blood sugar of ≥7.8mmol/L and s.creatinine of >120µmol/L was excluded. Estimated GFR was calculated by different GFR prediction equations namely Cockcroft-Gault (CG) equation, MDRD (Modification of Diet in Renal Disease) study equation and CKD-EPI equation.

GFR estimating equations:

I. Cockcroft-Gault Equation[9]
(140-Age) ×wt. (kg)

GFR CG equation (CG-CCR) = × (0.85 if female) ml/min 72×S.Cr (mg/dl)

After Body surface area (BSA)adjustment (CG-GFR) = (CG-CCR) ml/min ×1.73m^2÷ BSA

II. 4 variable MDRD Study Equation (Levey AS, 2000) [10, 11] eGFR (ml/min/ 1.73m^2 BSA) = 186 × (Scr)$^{-1.154}$ × (Age)x$^{-0.203}$ × (0.742 if female).

III. The 2009 CKD-EPI Equation[8] GFR = *a* × (serum creatinine/*b*) *c* × (0.993) age

The variable *a* takes on the following values on the basis of race and sex:Black (Women = 144, Men = 141); White/other (Women = 166, Men = 163).The variable *b* takes on the following values on the basis of sex:Women = 0.7, Men = 0.9.The variable *c* takes on the following values on the basis of sex and creatinine measurement: Women: Serum creatinine ≤ 0.7 mg/dL = -0.329, Serum creatinine> 0.7 mg/dL = -1.209.Men: Serum creatinine ≤ 0.9 mg/dL = -0.411, Serum creatinine> 0.9 mg/dL = -1.209.

Data was compiled and analyzed using statistical software SPSS-14. P=0.05 was considered as level of significance.

498 adult persons (18 years and above)were enrolled in study. Among them 352 were male and 146 were female. After screening, 88 cases (17.67%) were regarded “not healthy” and excluded from the study due to one or more of hypertension (17), hyperglycaemia (66) or abnormal urinary findings (45). (Table-1).410 (82.33%) eligible respondents were finally enrolled for GFR estimation by CG, MDRD and CKD-EPI equation

Table I: Abnormal results detected in screening of asymptomatic adults. Many have overlaps of abnormalities

Gender		HTN	High blood sugar	Raised creatinine	Abnormal Urine (dipstix test)			
					Alb	Glucose	Nitrite	Blood
M	67	11	55	6	10	17	10	5
F	21	6	11	3	-	-	1	2
n*	88	17	66	9	10	17	11	7

* Multiple responses were elicited.

Results

Among 410 eligible adult respondents 285 (69.50%) were males and 125 (30.50%) were females. Mean age of male and female was almost similar (36.74±12.61 vs 36.98±11.16 respectively). Age range was 18-83. Mean creatinine was higher in males (92.04±12.75 vs. 82.48±13.91). Mean creatinine was 89.125± 13.82 in study population. Mean SBP was 118.34 ±12.66 and mean DBP was 76.27±7.37 mm-Hg. Mean RBS was 5.50±1.03 mmol/L (Table II).

Table II: Demographic and baseline characteristics of the study population

Characteristics	Male (n-285)	Female (n-125)	Total (n-410)
Age	36.74±12.61	36.98±11.16	36.81±12.17
Height	166.38±7.01	157.37± 7.84	163.64 ± 8.37
Weight	62.83±11.42	57.71±10.45	61.273 ± 11.37
BSA(m^2)	1.70±0.15	1.57±0.15	1.657 ± 0.162
Creatinine(µmol/L)	92.04±12.75	82.48±13.91	89.125± 13.82
SBP (mm-Hg)	119.09±12.47	116.64±12.96	118.34±12.66
DBP (mm-Hg)	76.37±7.34	76.04±7.46	76.27±7.37
RBS (mmol/L)	5.46±.99	5.58±1.13	5.50±1.03

Mean eGFR by CG equation i.e. CG-CCr (ml/min) was83.13±18.69. After adjusted with BSA i.e. CG-GFR was 86.71±17.60 (ml/min/ 1.73^2). MDRD-GFR was 84.80±17.67 ml/min/ $1.73^2$and CKD-EPI revealed highest GFR 89.92±18.69 ml/min/1.73m^2 (Table III).

Table III: GFR in different equations

Characteristics	Male (n-285)	Female (n-125)	Total (n-410)
CG-CCR (ml/min)	85.72±17.35	77.22±20.31	83.13±18.69
CGGFR(ml/min/1.73^2)	87.59±16.90	84.69±19.0	86.71±17.60
MDRD-GFR	89.0±17.33	75.22±14.34	84.80±17.67
CKD-EPI	93.47±18.31	81.83±17.02	89.92±18.69

Respondents were divided into 6 age groups. Majority of respondents (40.98%) were among 18-30 years age. This age group had lowest creatinine (85.77±12.23 µmol/L) and highest eGFR by all equations (Table IV).

Table IV: Serum Creatinine and eGFR by different equations in different age groups

Age group	Serum Creatinine (µmol/L)	CG-CCR (ml/min)	CG-GFR (ml/min/1.73m^2)	MDRD (ml/ min /1.73m^2)	CK-DEPI (ml/ min/1.73m^2)
18-30	85.77±12.23	87.24±16.09	95.06±17.78	94.61±17.12	101.02±16.34
31-40	87.07±13.40	87.85±20.30	90.03±16.09	84.35±14.50	90.68±15.28
41-50	92.54±13.21	79.82±17.05	79.22±13.60	75.59±12.63	79.59±13.36
51-60	97.97±14.51	69.28±12.04	69.14±08.70	70.08±11.11	71.63±11.43
61-70	96.34±14.51	65.05±16.71	63.83±12.32	70.89±12.54	70.06±12.70
>70	115.50±10.61	35.00±18.38	36.0±12.73	49.50±16.26	46.50±16.26
Total	**85.77±12.23**	**83.13±12.69**	**86.71±17.70**	**84.80±17.67**	**89.92±18.69**

Respondents were divided into 6 groups as follows (Figure1).

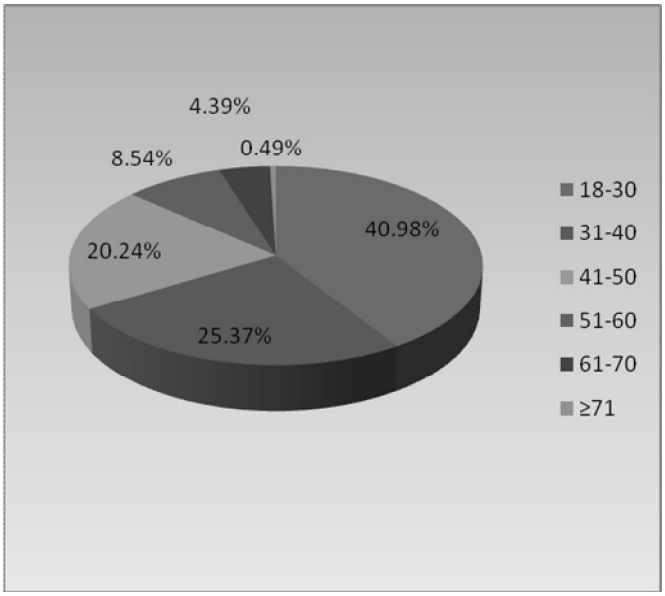


Figure 1: Pie diagram showing distribution of adult population in different age groups

Group 1: (18-30) years: 122 males and 46 females .Total 168(40.98%).
Group 2: (31-40) years: 64 males and 40 females. Total 104 (25.37%).
Group 3: (41-50) years: 56 males and 27 females. Total 83 (20.24).
Group 4: (51-60) years: 27 males and 08 females. Total 35 (8.54%).
Group 5: (61-70) years: 15 males and 03 females. Total 18 (4.39%).
Group 6: (71 and above): 01 male and 01 female. Total 02 (0.49%).

326 (79.51%) were having creatinine between 50-100 µmol/L. This group had higher GFR by all equations. While 84 (20.49%) have had creatinine>101 µmol/L (Table V).

Table V: Estimated GFR at creatinine 50-100 µmol/L groups and ≥101 µmol/L

Creatinine	CG-CCr ml/min	CG-GFR(BSA corrected) ml/min/1.73m^2	MDRD ml/min/1.73m^2	CKD-EPI ml/min/1.73m^2
50-100 µmol/L n=326	86.5±17.9	91.0±16.04	89.39±16.2	95.06±16.58
≥101 µmol/L n=84	70.11±15.86	70.02±12.79	67.0±10.35	70.0±11.86
Total n= 410	**83.13±12.69**	**86.71±17.70**	**84.80±17.67**	**89.92±18.69**

279 (68%) respondents were having BSA below 1.73m^2, and 131(32%) having ≥1.73m^2 BSA. In low BSA group highest GFR yielded in CKD-EPI formula (92.65±18.94ml/min/1.73m^2).In high BSA group highest yield was in CG-CCr without BSA adjustment (Table-VI).

Table VI: Level of creatinine and eGFR at Body Surface Area (BSA) below and above 1.73m^2

BSA	Creatinine	CG-CCR	CG-GFR	MDRD	CKD-EPI
<1.73m2	86.89±13.05	79.07±17.22	86.87±17.91	87.05±18.32	92.65±18.94*
≥1.73m^2	93.89±14.25	91.79±18.81	86.35±16.95	80.0±15.16	84.12±16.80**

*n=279 (68%), ** n=131(32%)

In low BSA group highest yield was in CKD-EPI formula (92.65±18.94ml/min/ 1.73m^2).

In high BSA group highest yield was in CG-CCr (actual CCr) without BSA adjustment.

Population was again divided into 2 groups on the basis of eGFR (below 60ml/min and above 60 ml/min). Lowest eGFRbelow 60ml/ minyielded with CG-CCr (10.7%), while BSA adjusted CG-GFR and CKD-EPI revealed almost similar result (5.6% and 5.9% respectively) (Table-VII, VIII).

Table VII: Healthy male and female population having GFR below or above 60 ml/min (despite without markers of kidney damage) by different GFR estimating equations

eGFR (ml/min/1.73m^2)	CG-CCr	CG-GFR	MDRD	CKD-EPI
<60	44(10.7%)	23 (5.6%)	28(6.8%)	24(5.9%)
≥60	366(89.3%)	387(94.4%)	382(93.2%)	386(94.1%)
Total	**410(100%)**	**410(100%)**	**410(100%)**	**410(100%)**

Table VIII: Healthy male and female population having GFR below or above 60 ml/min by different GFR estimating equations

Gender	CG-CCr		CG-GFR (Corrected CCR)		MDRD		CKD-EPI		
	<60	**≥60**	**60**	**≥60**	**60**	**≥60**	**60**	**≥60**	
M	16	269	12	273	9	276	9	276	285
F	28	297	11	114	19	106	15	110	125
Total	**44(10.7%)**	**366(89.3%)**	**23 (5.6%)**	**38 (94.4%)**	**28 (6.8%)**	**382 (93.2)**	**24 (5.9%)**	**386 (94.1%)**	**410**

We observed that creatinine tended to rise and all eGFR tended to fall with age. Though this rise or fall was not strictly linear (Figure 2).

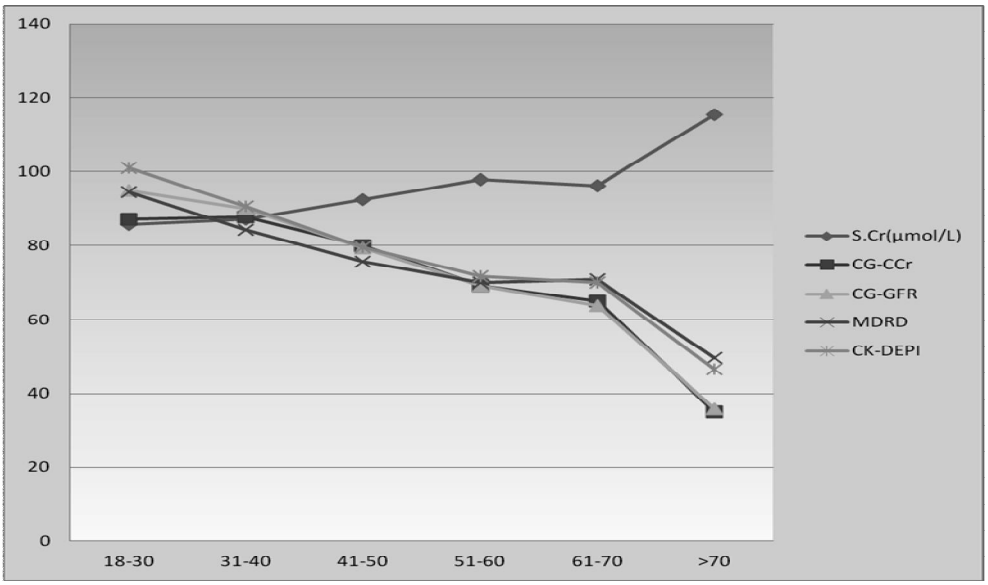


Figure 2: Trend of Creatinine and eGFR by GFR estimating equations in relation to creatinine in different age groups

Estimated GFR by 4 equations were significantly different by multivariate analysis of variance (ANOVA) ($p<.005$) where age, sex and creatinine are fixed factors.

Discussion

Total 88 cases (67 male and 21 female) have high BP, blood sugar, raised creatinine and abnormalities in urine. It is alarming, even in healthy fit exerciser 17.67% have had risk factors or kidney damage without their knowledge. In this study we have analyzed attributes of rest 410 adult persons of urban Dhaka majority of which were male 285 (69.50%). Females were 125 (30.50%). Mean age was 36.81 years. For male it was 36.74 and for female 36.98 years. Majority of respondents were between 18-30 years age group. 168 (40.98%) of which 122 male and 46 female were among this group.Mean crteatinine in study

population was 89.125± 13.82 µmol/L. In male creatinine was 92.04±12.75 µmol/L and in female- 82.48±13.91 µmol/L.

After estimating GFR in different equations eGFR was 83.13±18.69 ml/min in CG equation (CG-CCr). After correcting with BSA eGFR (CG-GFR) was 86.71±17.60 ml/min/1.73m^2. GFR by MDRD formula was 84.80±17.67 ml/min/1.73m^2.The new CKD-EPI formula reveals even higher GFR estimate. In population, mean GFR with CKD-EPI was 89.92±18.69 ml/min/1.73m^2. In male CKD-EPI eGFR was 93.47±18.31 ml/min/1.73m^2 and in female 81.83±17.02 ml/min/1.73m^2. We have conducted multivariate ANOVA to see difference of means among groups. Fixed factors (independent variables) were age, sex and creatinine (p<0.05) in all equations, groups were significantly different from one another. Post analysis couldn't be done.

Estimated GFR in female is lower than that of males in all equations of our study. Females have 9.95% low CG-CCr, 3.3% low corrected CG-CCr/CG-GFR, 15.48% low GFR in MDRD and 12.45% low GFR by CKD-EPI equation than males' estimated GFR.

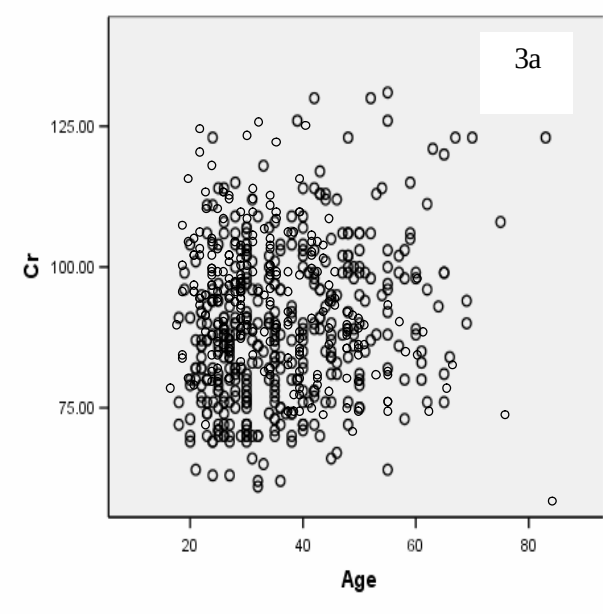
Population was divided into 6 age groups. Least creatinine value and highest eGFR was achieved at lowest age group, i.e, Group 1 (18-30 years) and vice versa. CKD-EPI and MDRD equation yielded highest GFR exception Group 3 (41-50 years) where unadjusted CG equation has the highest yield probably because of higher BSA 1.74m^2.

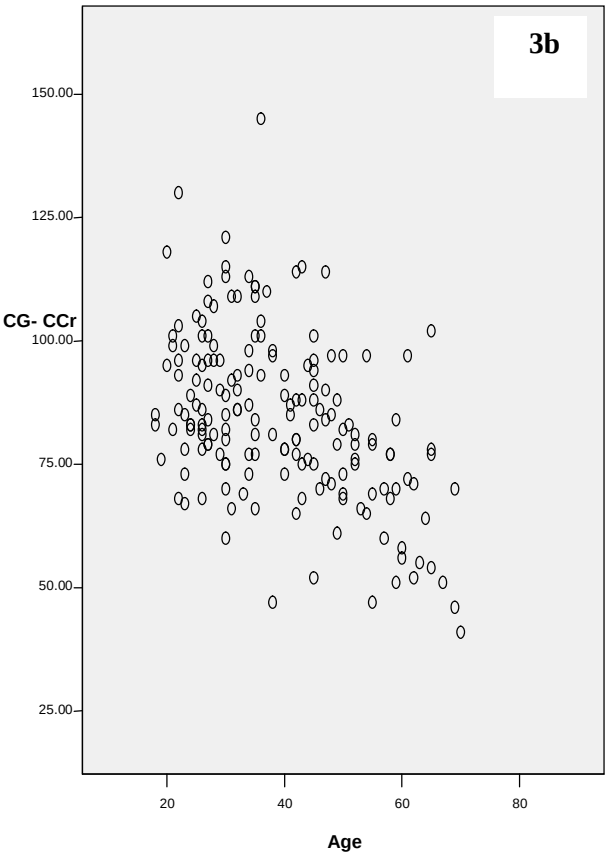
Estimated GFR varied as s. creatinine varies in population. In 50-100 µmol/L groups highest yield was by CKD-EPI equation: 95.06±16.58 ml/min/1.73m^2 followed by corrected CG GFR 91±16.04 ml/min/1.73m^2 and CG-CCr 86.49 ± 17.90 ml/min.

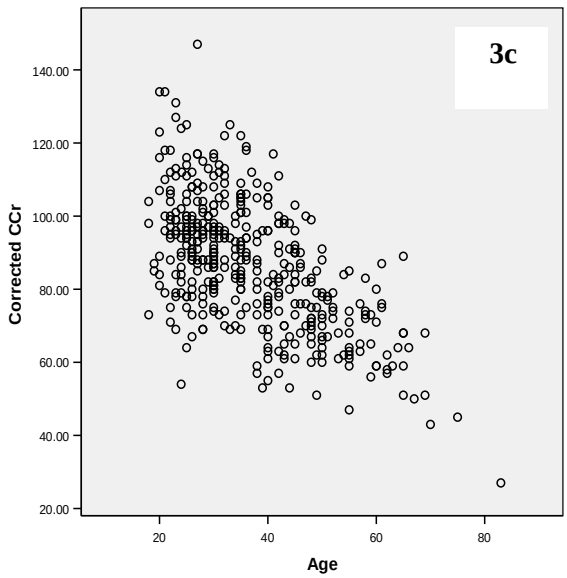
In ≥101 µmol/L creatinine group: CG-CCr and CG-GFR and CKD-EPI equation yielded almost equal mean GFR 70 ml/min. It seems that CG equation has overestimated eGFR in higher creatinine values. It is already known that CKD-EPI produces higher eGFR values in the high GFR range(>60 ml/min/1.73 m^2), and lower eGFR values in the lowest range.[8] In contrast C-G equation overestimate renal function at higher creatinine (lower GFR range).[12] In low BSA group highest yield was 92.65±18.94 ml/min/ 1.73m^2 in CKD-EPI formula. In high BSA group highest yield was by unadjusted CG-CCr. It seems CG equations has been influenced by BSA. MDRD and CKD-EPI equations are already adjusted for BSA, hence not influenced by BSA.

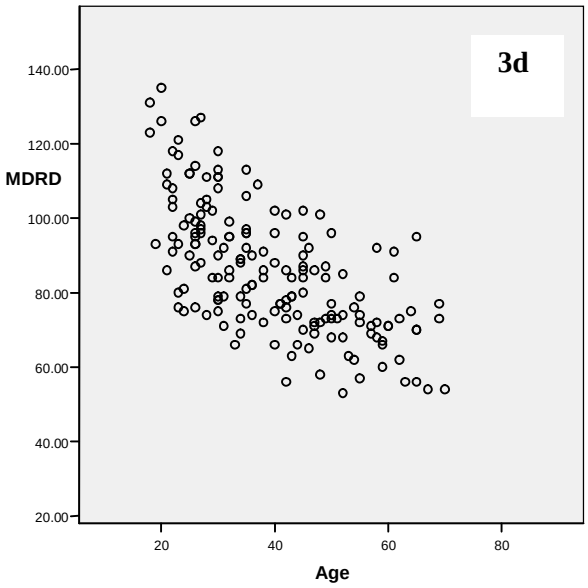
We have documented 10.7% of population having GFR <60 ml/min (i.e. CKD stage 3 range) by C-G equation without surface area correction. After surface area correction (CG-GFR/Corrected CCR) this value falls to 5.6%. By new CKD-EPI equation only 5.6% and with MDRD equation 6.8% of population was found to have GFR <60 ml/min/1.73m^2.

We have plotted scatter diagram for creatinine and all estimated GFR in relation to age to observe correlation. We found creatinine does not have any correlation with age. It signifies, it is not the norm that creatinine will increase or decrease with advanced age. However, we found moderate negative correlation with all form of GFR measured in different equations. This finding matches our understanding that GFR decrease with age (Figure 3).









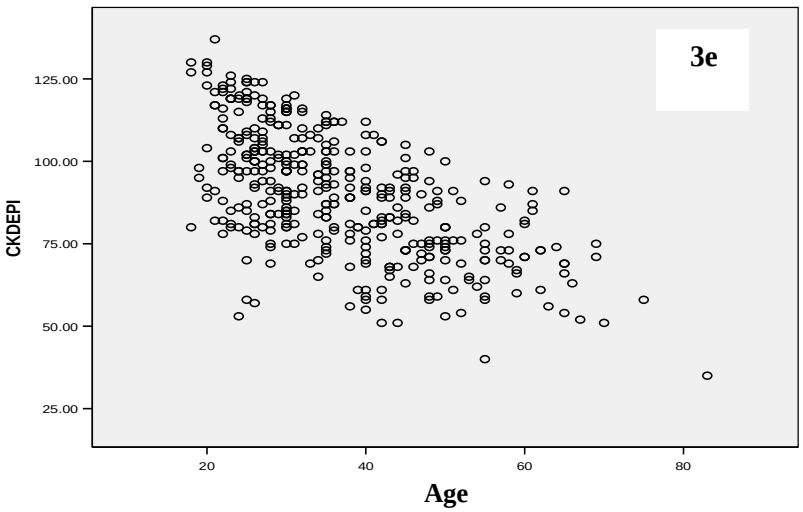


Figure 3: Scatter diagram showing correlation of creatinine and GFR with age

3a. No correlation is observed for creatinine, but moderate negative correlation is noted with age and all GFR prediction equations. **3b, 3c, 3d, 3e.** Moderate negative correlation is noted with age and all GER prediction equations.

Since creatinine does not linearly increase with age as we see in scatter diagram, we can say that somewhat linear decrease in GFR is not due to rise of creatinine; particularly age and other factors and exponentials inherent in the equations might have operating.

On histogram, distribution of creatinine was normal in male but somewhat right skewed in females (Figure 4).

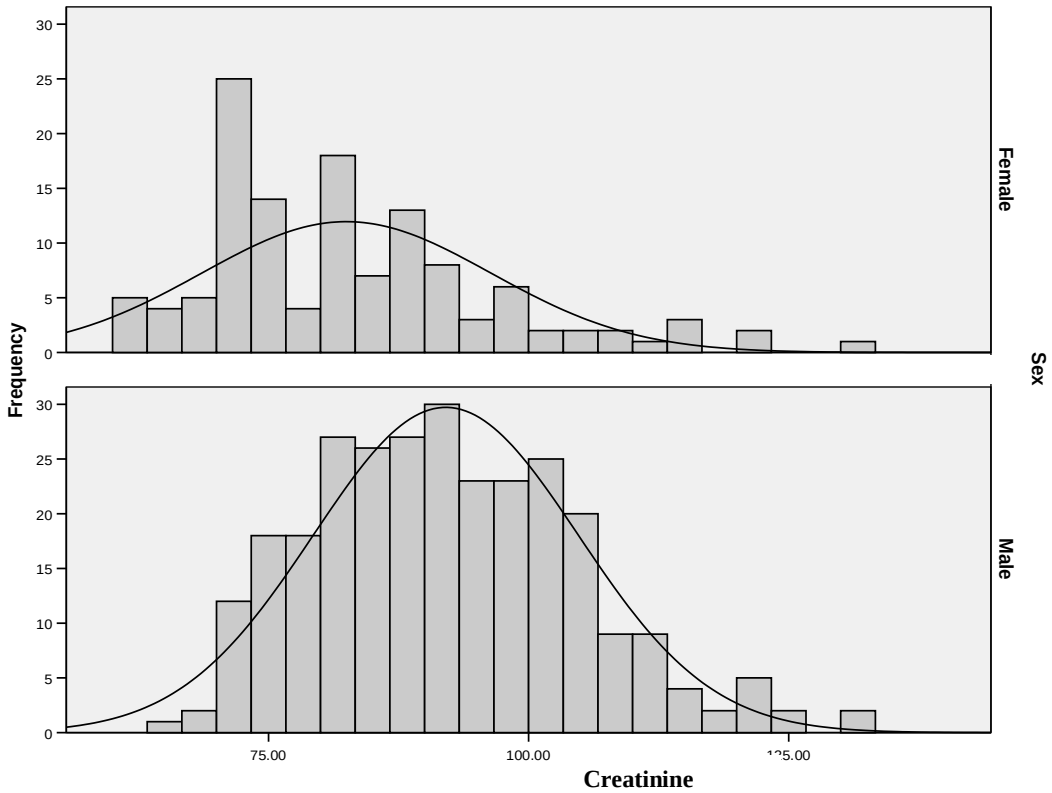


Figure 4: Histogram showing gender variations in creatinine level

Distribution is normal in male but somewhat right skewed in females.

We have compared our study with the study of Kabir E, Rahman M[13] conducted at rural village of Chakulia, Savar, Dhaka involving CG and MDRD equations only. Despite higher mean creatinine (89.125 vs 86.44 µmol/L in Chakulia) we have yielded higher mean GFR in all prediction equations. Probably this resulted from lower mean age (36.81±12.17 vs 41.37±14.85 in Chakulia), male dominance (69.50% male vs56.8% female in Chakulia) and anthropometric variables like BSA (1.657 vs 1.55 for Chakulia) in our study.

In a study, among 100 potential kidney donors mean GFR measured by DTPA, renogram was 89.05±10.96 ml/min/1.73m^2, MDRD GFR 88.81±10.47 ml/min/ 1.73m^2, CG-CCr 90.80± z13.43 ml/min, and CG-GFR was 93.55±11.23 ml/min/1.73m^2 done in Combined military hospital, Dhaka.[14] In that study, MDRD equation was not significantly different from measured GFR (p=0.671) so proved valid for healthy Bangladeshi adult. While CG CCR and CGGFR was significantly different (p<0.05 and <0.001) respectively from measured GFR by DTPA, so was proved not valid for healthy Bangladeshi adult. Our CKD EPI eGFR 89.92±18.69 is similar to measured GFR of CMH study.

Conclusion

Kidney screening test should be done to detect kidney disease early, and is cost effective. This study was meant to estimate GFR by different equations, not to see the prevalence of CKD. It requires repeated assessment of same populations at least after 3 months and document their eGFR to label as CKD. Large scale multicenter study involving newer CKD-EPI equations can be useful to see prevalence of CKD and status of renal function across the country. Moderate negative correlation is noted with age and all GFR prediction equations.

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Contribution of the Authors

First author was the principal researcher. Third and fourth were guide and co-guide respectively. Others helped in data collection and analysis.

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Association of Ovarian Tumour with Sociodemographic Background in a Tertiary Level Hospital

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Abstract

Intrduction: *Ovarian cancer is a common cause of death in women due to malignancy. Aim of this study was to observe the probable association of some predisposing factors with ovarian tumours.*

Methods: *This study was conducted at the Department of Obstetrics and Gynaecology of Rangpur Medical College Hospital, Rangpur from July 2012 to June 2014. Ovarian tumour cases diagnosed by history, clinical examination and ultrasonography were included in this study by purposive sampling technique; recurrent cases were excluded. Findings were expressed as 'Percentage involved'. In addition, χ^2 test, student's't' test and Odd's ratio were also used for statistical analysis.*

Results: *Total cases were 31–benign 24 and malignant 7. Peak age incidence of benign cases was about 35 years and of malignant cases was above 60 years. Past history of Pelvic Inflammatory Disease (PID) or endometriosis, or family history of ovarian tumour was negative among the cases. Mean parity of benign cases was 2.416 and of malignants was 1.857 ($p>0.10$). Among the oral pill users and non-users, Odd's ratio for benign vs malignant cases was 1.128 ($p>0.10$). Mean CA-125 level and ESR were higher in malignant than the benign (both p value<0.001). Histological types were serous cystadenoma, dermoid cyst, mucinous cystadenoma, poorly differentiated adenocarcinoma, serous cyst adeno-carcinoma and immature teratoma.*

Conclusion: *This small sample size is not at all suitable to draw any inference. Yet, considering peak incidence of benign cases around 35 years and of malignant cases was above 60 years of age; and, Odd's ratio goes in favour of protective role of oral pill against ovarian malignancy. Lack of awareness of our patients might have played some role for our non-conclusive findings regarding past medical or family history.*

Key words: *Ovarian tumour, Age, Parity, Oral contraceptive*

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Introduction

Ovarian cancer is a common gynaecological malignancy and one of the most common causes of death in women with malignancy; life time risk of ovarian cancer has been demonstrated to be 1.7% in general population.[1] The incidence of ovarian cancer is 7% out of total female cancers.[2] As ovary is

complex in its embryology and histology and has the potential to develop malignancy, ovarian neoplasm exhibits a wide variation in structure and biological behavior. The ovaries, after the uterus, are the second common site for development of gynaecological malignancy and the prognosis remains poor.[3] Malignant ovarian tumours are leading cause of death from gynaecological cancer.[4] But early stage of this disease is associated with poorly defined or vague symptoms, which often are not severe enough to prompt a woman to seek medical attention.[1] Excluding those which have an endocrine function, ovarian tumours are amazingly quite and rarely give rise to symptoms other than those induced mechanically by the size of the mass. This is why they are really dangerous and the malignant ones are often inoperable by the time they are diagnosed–commonly in stage III and IV.[5]

Benign tumours may occur at any point of life but they are most common during child bearing age with the peak incidence being between 25 and 34 years of age.[6] Borderline malignant ovarian tumours occur most frequently in 30 to 50 years whereas invasive carcinomas are seen more frequently between 50 to 70 years; and, germ cell tumours generally occur prior to puberty or in early adult life. An ovarian tumour in adolescent and postmenopausal women is more often malignant than benign. Most of the germ cell tumours occur in young girls.[5]

Early age at menarche and late age at menopause increase the risk of ovarian cancer whereas pregnancy and lactation reduce the risk.[7] Prolonged lactation is associated with lower risk of ovarian cancer.[8] Pelvic inflammatory disease (PID), especially those 35 years and younger were more likely to have developed ovarian cancer than control during 3 years of follow up.[9] Endometriosis is also known to be associated with endometroid müllarian adenocarcinoma.[4]

One strong risk factor of ovarian cancer is family history of the disease. Approximately 10-15% of ovarian cancers are attributed to genetic causes. In breast-ovarian cancer syndrome, majority of patients have mutation in BRCA 1 or BRCA 2 gene. Lynch II syndrome (hereditary non-polyposis colorectal cancer) also has 12% risk of developing ovarian cancer along with risk of developing colon, endometrial, breast cancers.[1]

One study with 7,308 cases and 32,717 controls demonstrated that the longer a women had used oral contraceptive pills the greater the reduction in ovarian cancer risk (p<0.0001). This reduction in risk persisted for more than 30 years after oral contraceptive pills use had ceased, with gradual attenuation of risk reduction over those 30 years.[10] Nulliparity and infertility were found to be associated with an increased risk and multiparity with a reduced risk of benign epithelial ovarian neoplasm. Infertility and PID were associated with increased risks of functional and dermoid cysts.[6] A Finnish cohort study including 87,929 cases also demonstrated lower risk of ovarian cancer in grand multipara, no matter, how many children and at which ages they had delivered.[11]

Evaluating all these into consideration, this study was conducted to observe the association of some predisposing factors with ovarian tumours found at Rangpur Medical College Hospital, Rangpur.

Materials and Methods

This cross sectional descriptive study was conducted at the Department of Obstetrics and Gynaecology of Rangpur Medical College Hospital, Rangpur from July 2012 to June 2014. Cases of the study were enrolled from the same Department and Institution.

The total number of cases (n) included in this study was 31. Sample was collected from in-patients' department by purposive sampling technique. Patients with ovarian tumours diagnosed by history, clinical examination and ultrasonography were included in this study. Previously diagnosed and treated ovarian tumours (recurrent case) were excluded.

The findings were expressed as 'percentage involved'. In addition, χ^2 test, student's '*t*' test and Odds ratio were also used and $p<0.05$ was considered as level of significance.

Results

Among all the cases (n=31) benign cases were 24 (77.41%) and malignant cases were 7 (22.59%) in number. Age range was 16 to 65 years, benign cases were 16 to 50 years of age with a pick incidence of 35 years and malignant cases were 24 to 65 years of age with a peak incidence above 60 years. No significant past medical history as PID or endometriosis that might have association with ovarian tumour was found from all the cases. As well as, family history of ovarian tumour was also negative.

Parity of all the cases ranged from 0 to 6, of which, among benign cases, it was 0 to 6 with mean (±SD) being 2.416 (±1.639) and among malignant cases, it was 1 to 6 with mean (±SD) being 1.857 (±1.864); the difference between the two groups was insignificant (p>0.10). (Table I).

Table I: Parity among benign and malignant cases of the study population (n-31)

Type of tumour	Parity (mean ± SD)	*p* value
Benign (n_1-24)	2.416 ± 1.639	>0.10
Malignant (n_2-7)	1.857 ± 1.864	

Student's '*t*' test demonstrates insignificant difference between the two groups.

Among all the cases, 9 (29%) gave history of oral contraceptive pills use only, 5 (16%) gave history of oral contraceptive pills and others (depot progesterone injections or IUCD), 6 (19%) gave history of depot progesterone injections or Intrauterine Contraceptive Device (IUCD) and 11 (36%) gave no history of the use of any of the above. (Table II).

Table II: Shows the use of different contraceptive methods in this study group (n-31)

Types of Contraceptive Used	Number of cases (n-31)	Subtypes of Contraceptive Used	Number of cases (n-31)
Users of Oral contraceptives	14 (45%)	Oral contraceptive pills only	9 (29%)
		Oral contraceptive pills & others (depot progesterone injections or IUCD)	5 (16%)
Non-users of Oral contraceptives	17 (55%)	Others (depot progesterone injections or IUCD)	6 (19%)
		No contraceptive (None of the above)	11 (36%)

Table III: Distribution of benign and malignant cases among the users and non-users of oral contraceptive pills (n-31)

Category	Malignant (n_2-7)	Benign (n_1-24)	*p* value
Oral pill users (14 cases)	3 (21%)	11 (79%)	>0.10
Oral pill non-users (17 cases)	4 (24%)	13 (76%)	

Odds ratio for this distribution is 1.128 which goes in favour of the comment that non-users of oral pill are more prone to develop malignant ovarian tumours than the users.

Among the oral pill users (14 cases), 11 (79%) were benign and 3 (21%) were malignant and among the non-users (17 cases), 13 (76%) were benign and 4 (24 %) were malignant and, the difference was insignificant (p>0.10). (Table III).

Mean (±SD) of CA 125 level in benign group was 25.97 (±15.98) and in malignant group was 7234.82 (±1120.22) (p<0.001); mean (±SD) of ESR in benign group was 24.50 (±13.83) and in malignant group was 46.57 (±14.51) (p<0.001); and, mean (±SD) of Hb% in benign group was 10.79 (±1.01) and in malignant group was 9.75 (±1.72) (p<0.10). (Table IV).

Table IV: Serum CA 125 level, erythrocyte sedimentation rate (ESR) and haemoglobin (Hb%) of benign and malignant cases are as follows

Parameters	Benign cases	Malignant cases	*p* value
Serum CA 125 level (Unit/Litre) Mean ± SD	25.97 ± 15.98	7234.82 ± 1120.22	<0.001
ESR (mm at 1st hour) Mean ± SD	24.50 ± 13.83	46.57 ± 14.51	<0.001
Hb% (gm/dl) Mean ± SD	10.79 ± 1.01	9.75 ± 1.72	> 0.10

Student's '*t*' test was used to demonstrate the level of significance.

The different types of tumours found in this study show that among all the cases (n-31), 12 (38.71%) were serous cystadenoma, 7 (22.58%) dermoid cyst, 5 cases (16.13%) mucinous cystadenoma, 4 (12.90%) poorly differentiated adenocarcinoma, 2 (6.45%) serous cyst adeno-carcinoma and 1 (3.23%) was immature teratoma (Figure 1).

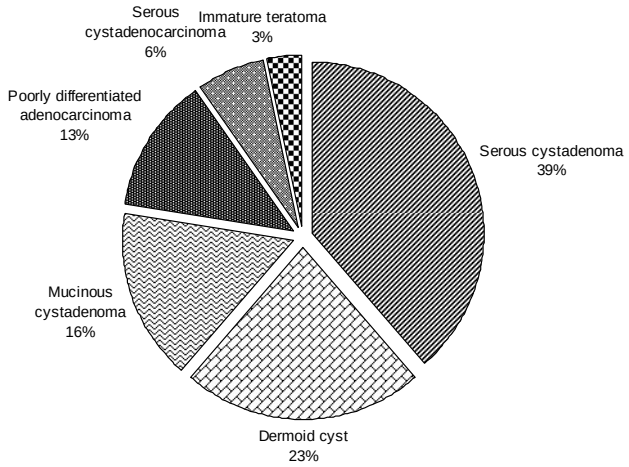

Figure 1: Different histological types of tumours found in this study (n-31)

Discussion

Among the 31 cases included in the present study, 24 (77.41%) were benign and 7 (22.59%) were malignant. In a study including 1,066 ovarian tumour patients, Timmerman et al.[12] found 800 (75%) cases had benign tumours and 266 (25%) had malignant tumours. Another study with 110 cases also found 80 (72%) benign and 30 (28%) malignant cases.[13] Both results are comparable to this study.

Here, the peak incidence of benign tumours was around the age of 35 years and the peak incidence of malignant tumour was above 60 years. Bukhari et al. found that the incidence of benign tumour is more in 20-40 years of age and for malignant tumour it is above 50 years of age.[14] Another study, demonstrated the maximum incidence of benign tumour around 40 years and maximum incidence of malignant tumour above 50 years.[15] Both are, more or less, comparable to this study.

In this study, some nullipara, as well as some grand multipara were found to develop ovarian neoplasm. Mean parity was higher in benign group than in malignant group, but the difference was insignificant (p>0.10). As there was no non-neoplastic control group in this study, it is not possible to demonstrate the protective role of parity in the development of ovarian neoplasm. On the other hand, contraceptive prevalence rate in our community has also been increased remarkably and so, scope of comparing grand multipara with the others has been reduced. In one study, multiparity was found to be associated with a significant reduction in risk of ovarian cancer (Odds ratio = 0.6 for 3, and 0.5 for 4 births).[16]

Again, in this study, it was found that 14 (45%) cases used oral contraceptive pills, 6 (19%) cases used anything other than oral pills and 11 (36%) cases used none as contraceptive. Among the non-users and users of oral pills, Odds ratio was 1.128 for malignant and benign ovarian tumours which goes in favour of the comment that “non-users of oral pill are more prone to develop malignant ovarian tumours than the users”; of course the difference was insignificant for ‘χ^2’ (p>0.10). To find out the protective role of oral contraceptive in the development of ovarian neoplasm, a large case control or a cohort study is required.

In the malignant group, CA-125 level (p<0.001) and ESR (p<0.001) were significantly higher

and Haemoglobin (p>0.10) was insignificantly lower. Terzic et al., involving 112 malignant and 544 benign cases, found mean CA-125 level to be 937.13 Units/Litre in the malignant group and 59.54 U/L in benign group (p<0.000); regarding mean ESR level, their result was 40.16 mm at 1st hour in the malignant group and 19.88 mm at 1st hour in benign group (p<0.000)[17].

The most common histologic type was serous cystadenoma 12 (38.71%). The next was dermoid cyst 7 (22.58%). And, then were mucinous cystadenoma 5 (16.13%), poorly differentiated adenocarcinoma 4 (12.90%), serous cyst adenocarcinoma 2 (6.45%) and immature teratoma 1 (3.23%).

Comparable to this study, done by Mondal et al., involving 957 cases over a period of 10 years, found serous cystadenoma (29.9%) as the most common histological type; followed by, were mature teratoma (15.9%) and mucinous cystadenoma (11.1%). Surface epithelial tumours (60.9%) were the major proportion of malignant ovarian tumours. Serous cystadeno-carcinoma was the predominant malignant tumour (11.3%). Bilateral malignant serous tumours were 49.5%.[18] The incidences found by Danish et al. are as follows: serous cystadenoma (32%), dermoid cyst (22%), mucinous cystadenoma (17%), poorly differentiated tumours (5%), serous cystadenocarcinoma (4%), mucinous cystadenocarcinoma (3%)etc.[19] Which is also more or less comparable to the histologic types of this study.

After all, this small sample size is not at all suitable to draw any inference. Yet, it may be assumed that lack of awareness of our patients probably has contributed to some of our non-conclusive findings regarding past history as PID or endometriosis, or early menarche, or late menopause, or family history of ovarian tumour.

Limitations of this study include

1. Sample size '31' is too small to predict anything definitely.
2. Present study does not represent the whole scenario of our community. Because there are many private hospitals and district hospitals as well, which are also giving services to our population and their scenario are not similar to this hospital.

Contribution of the Authors

First author was the main researcher. Others helped in data collection and statistical analysis.

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Identification and Prevalence of Mixed Infection of Bacteria and Fungus on Toe Webs of the Diabetic Patients in BIRDEM Hospital, Dhaka

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Abstract

Introduction: *A study was conducted in Bangladesh Institute of Research Rehabilitation in Diabetes, Endocrine and Metabolic Disorders (BIRDEM), Dhaka during March 2015 to December 2015, where mixed infection of bacteria and fungus on toe web of diabetic patients was observed.*

Methods: *It was a descriptive type of cross sectional study conducted among the diabetic patients in the department of Dermatology (Outpatient department) of Bangladesh Institute of Research And Rehabilitation in Diabetes, Endocrine and Metabolic Disorders (BIRDEM). The study was undertaken during March 2015 to December 2015.*

Results: *Observation under light microscope revealed that, Candida was the main fungus identified in the culture process of fungi which was oval shaped, violet colored and singly occurred in the culture media whereas Staphylococcus was the main bacteria identified in the culture process of bacteria which was cocci shaped, violet colored and occurred in cluster. In the present study it was observed that about 80% of the patients were above the age of 40 and only 20% were below the age of 40. Among the infected patients 65% were female and 35% were male. So it revealed that the women suffer more in this type of infection because the housewives use more water during household works. The percentage of infected housewives was 58.33%. The site of lesion was foot (53.33%), hand (31.67%) and both hand and foot (15%). Seasonal variation was another factor found in the investigation. The infection occur 13.33% during summer, 15% during winter and 71.67% during monsoon. The types of footwear used by the infected patients was sandal (45%), shoes (21.17%) and barefooted (33.33%).*

Conclusion*: It revealed that during the monsoon season the patients comes close contact with water more than the other seasons and different types of footwear also affect the infection.*

Key words*: Staphylococcus, Candida, Diabetes Mellitus*

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Introduction

The skin is the largest organ of the human body covering the entire surface of the body. It is subject to a wide range of medical conditions and infections ranging from simple manifestations to complicated ones like skin cancer.[17] Skin and venereal diseases cause a large part of illness. About 30% of people in Bangladesh suffer from it in their life time. However, fungal and bacterial infections are very common in the healthy people. Bangladesh is one of the poorest countries of the world with the highest density of population. Tropical region with 20-37^{0}c and humidity stimulates the development of fungal infection though the disease may occur in any climate. About 80% of population live in the rural areas, where poverty, literacy, ignorance, high family members, disease and disasters are the constant companion of them.

Skin and venereal diseases are a public health problem in developing countries.[12] The relation between the skin and venereal diseases of the diabetic patients of different age group and socio-demographic characteristics is very complicated. The socio-demographic aspects are very important to know because in different societies and social groups explain the causes of illness, the type of treatment they believe and to whom they turn if they go get ill.[10] Though it occurs in all class of society but people living in insanitary and poor housing conditions suffer more from the disease, poverty striken people with poor hygienic habits and unclean clothing are the usual victims of these diseases.

There are different kinds of fungal infections commonly affecting the skin of the diabetic patients. A yeast-like fungus called "*Candida albicans*" is responsible for many of the fungal infections causing skin problems in people with diabetes.[14] Akhter[3] worked in the Dermatology Department, BIRDEM hospital, Dhaka, to determine the prevalence of fungal infection and its causal factors. This study was done to assess the socio-demographic conditions, magnitude of skin and venereal disease and to find out the preventive knowledge regarding diseases problem attending in BIRDEM hospital, Dhaka. Fungus also can occur in between the toes and fingers.[13] This fungus creates itchy, bright red rashes, often surrounded by tiny blisters and scales. These infections most often occur in warm, moist folds of the skin.[15]

A variety of fungi may exacerbate intertrigo, including yeasts, molds, and dermatophytes. There are comparatively few species that are pathogenic to animals, especially mammals. According to[9] there are approximate a little 1.5 million described species of fungi.[5] Studied on noncandidal fungal infection of the mouth. Candida is the fungus most commonly associated with intertrigo. The inflammation may begin as a dermatophyte infection, which can damage the stratum corneum and encourage the proliferation of other, usually antibiotic-resistant bacteria. Dermatophytes commonly complicate interdigital intertrigo.[11] Gram-positive and gram-negative bacteria also can worsen the effects of interdigital intertrigo.[4] However, gram-negative and gram-positive infections occasionally occur simultaneously in interdigital areas. Gram-positive infections usually are caused *by S. aureus.* Dermatophytes and bacterial infections often occur together in interdigital areas. Yeasts also are commonly found at the site of interdigital intertrigo.[8] Sometimes seborrheic dermatitis is located in the folds. Whether Malassezia-complicated intertrigo is a distinct entity or a type of seborrheic dermatitis remains unclear.[6] Cutaneous erythrasma may

complicate intertrigo of interweb areas, intergluteal and crural folds, axillae, or inframammary regions.[16]

Toe web intertrigo usually is associated with a burning sensation between the toes, often with maceration. Toe web intertrigo may be simple, mild, and asymptomatic, but it also can be seen as intense erythema and desquamation, which sometimes is erosive, malodorous, and macerated.[18] Patients also may have profuse or purulent discharge and be unable to ambulate. In severe examples, patients may have a purulent discharge with edema and intense erythema of tissues surrounding the infected area. Patients with severe toe web intertrigo who are overweight or who have diabetes are at a higher risk for cellulitis. Patients with advanced gram-negative infections may have green discoloration at the infection site. Erythematous desquamating infection may be more chronic than the acute form and may present with a painful, exudative, macerating inflammation that causes functional disability of the feet.

Materials and Methods

It was a descriptive type of crosssectional study conducted among the diabetic patients in the department of Dermatology (Outpatient department) of Bangladesh Institute of Research And Rehabilitation in Diabetes, Endocrine and Metabolic Disorders. The study was undertaken during March 2015 to December 2015. The population of the study was the diabetic patient of all ages with different occupation during the data collection period. Among all the patients with skin disease only the toe web infected patients were selected. A total of 60 diabetic patients with infections were selected purposively. A structured pre-tested questionnaire was used for data collection by face-to-face interview.

Results

The following results are obtained after collecting and analyzing the data which are showed and discussed in Table I, II and III.

Table I: Observation of scrub of the diabetic patients in cultured media

Characteristics observed in potato dextrose agar media (culture media of fungus)	Characteristics observed in nutrient agar media (culture media of bacteria)
Shape- oval, color- violet, singly scattered yeast	Shape-cocci and rod shape, color-violet, occurred in cluster

Observation under light microscope revealed that, *Candida* was the main fungus identified in the culture process of fungi which contained the characteristics of oval shaped, violet colored and singly occurred in the culture media whereas *Staphylococcus aureus* was the main bacteria identified in the culture process of bacteria which contained the characteristics of cocci shaped, violet colored and occurred in cluster (Table I).

Table II: The socio demographic characteristics of the patients was as follows

Variable	Number of patients		Percentage (%)
Age	Above 40 yrs	48	80
	Below 40 yrs	12	20
Sex	Male	21	35
	Female	39	65
Educational status	Illiterate	22	36.67
	Primary level	17	28.83
	Secondary level	13	21.17
	Graduate	8	13.33
Occupation	Service	10	16.67
	Business	15	25
	Housewife	35	58.33
Monthly income	High	6	10
	Moderate	30	50
	Low	24	40
Residence	Urban	36	60
	Rural	24	40

In the present study it was observed that about 80% of the patients were above the age of 40 and only 20% were below the age of 40. Among the infected patients, 65% were female and 35% were male. The percentage of infected housewives was 58.33%. The highest percentage of diseases occurred among the illiterate group (36.67%) with moderate monthly income (50%). The residence of the infected patients were urban (60%) and rural (40%) (Table- II).

Table III: Diseases relation with different types of variables

Site of lesion	Hand	19	31.67
	Foot	32	53.33
	Both	9	15
Seasonal variation	Summer	8	13.33
	Winter	9	15
	Monsoon	43	71.67
Types of footwear use	Sandal	27	45
	Shoes	13	21.17
	Barefoot	20	33.33

The site of lesion was foot (53.33%), hand (31.67%) and both hand and foot (15%). Seasonal variation was another factor found in the investigation.

The infection occurred 13.33% during summer, 15% during winter and 71.67% during monsoon. The types of footwear used by the infected patients were sandal (45%), shoes (21.17%) and some 20 (33.33%) were barefooted. (Table III).

Discussion

The present study provides a description profile of socio-demographic characteristics of patients attending to skin OPD. As the study was conducted in a department of dermatology of a diabetic hospital, so the diabetic patients were preferred. A total of 60 diagnosed toe web infected patients were taken purposively as a sample size. This study was conducted for the first time in our country.

In the present study, it was found that toe web or inter digital infection occurs highly above the age of 40 years (80%), as the disease is related with diabetics whereas[10] reported that recurrence of several skin disease was high (55.06%) below the age of <10 yeas probably due to higher proportion of respondents in that age group, Moreover incomplete treatment and lack of knowledge regarding prevention of disease were also a contributing factor.

According to the present study, among the infected patients 65% were female and 35% were male. So it was revealed that the women suffer more in this type of infection because the housewives use more water during household works. The percentage of infected housewives was 58.33%. This study can be compared with the study where a total 160 dermatophyte-infected patients were studied[7] in skin and VD department of Mymensingh Medical College Hospitals. It was found that 76.9% were male and 3.5% were female and 76.25% were above 15 years and 23.25% were below 15 year since January 1997. Trichophytone mentagophytes (56.36%) were the highest incidence of skin infection in this study.

Another study was carried out by some specialist doctors at Comilla Medical College Hospital from 14th December 2000 to 10th February 2001 to shed some light on pattern of dermatology diseases.[1] Male patients were found to be visiting the inpatient departments more than females. The majority of the patients were within the age group of 10-20 and 20-30 years. Ninety four separate categories of skin disease were clinically diagnosed including some rare ones. Scabies, fungal infection, atopic dermatitis, and psychosexual diseases were however the more common ones.

Seasonal variation was another factor found in the investigation. The infections were found 13.33% during summer, 15% during winter and 71.67% during monsoon, during the monsoon season the patients comes close contact with water more than the other seasons and different types of footwear also affect the infection, it can be compared with the study whereas[2] reported that greater number of patients with scabies sought medical help during winter (43.4%), pyoderma in summer (38.1%), ringworm in monsoon (50%).

On the basis of treatment seeking pattern, it was observed that a reasonable number of patients obtain treatment from outside before came to this hospital. It indicates either irregular treatment or wrong treatment due to wrong diagnosis that worsened the diseases. The present study was conducted on a small number of patients so it can't reflect the exact situation and socio-demographic condition of the skin diseases in the whole population of our country. As skin diseases cause less mortality, patients though literate and solvent come to the specialist lately. Beside this chronic cause and most of the diseases, make patient reluctant about the long durable treatment and advice for the diseases.

Contribution of the Authors

First author was the principal researcher and data analyzer. Second one was the co-researcher, third one acted as designer of this research work. Last one worked as data collector.

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Serum Lipid Concentration and Prevalence of Dyslipidemia in Patients with Coronary Heart Disease in Tertiary Hospitals of Bangladesh

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Abstract

Introduction: *Coronary heart diseases (CHDs) are the most common form of heart disease and most important cause of premature death in developed countries. It was estimated that CHDs will become the major cause of death in all regions of the world by 2020. There were several modifiable risk factors for development of CHDs. Among them dyslipidemia was an important modifiable risk factor. Lipid abnormalities, including high levels of total cholesterol, high levels of low-density lipoprotein cholesterol (LDL-C), elevated triglycerides and low levels of high-density lipoprotein cholesterol (HDL-C), are associated with an increased risk of CHDs, thereby serving as contributors to this process.*

Methods: *The study was conducted in department of Biochemistry of BSMMU over a period of one year extending from July 2006 to June 2007. This cross- sectional study was done among 300 diagnosed patients of CHD of both sexes. Dyslipidemia was diagnosed by estimation of fasting blood lipid profile.*

Results: *The study revealed a higher rate of dyslipidemia (27.7 %) among the study subjects.*

Conclusion: *It can be concluded that the prevalence of dyslipidemia (an important modifiable risk factor) was relatively higher among the patient of CHD.*

Key words: *Risk factors, Dyslipidemia, Lipid profile, Coronary heart disease*

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Introduction

Coronary heart disease is a disease due to narrowing of the small blood vessels that supply blood and oxygen to the heart.[1] It has two principal forms, angina and myocardial infarction (MI). Both occurs because the arteries carrying blood to the heart muscle become narrowed or blocked, usually by a deposit of lipid substances, a process known as atherosclerosis. Angina is a severe pain in the chest brought on by exertion and is relieved by rest. Myocardial infarction (MI) is due to obstruction of coronary arteries either as a result of atherosclerosis or by a blood clot. Part of heart muscle is deprived of oxygen and dies.[2] At

different times, the heart has a varying need for blood flow and the oxygen it carries. The heart receives their blood flow through its own set of blood vessels called the coronary arteries. With the relatively decreased blood flow and oxygen, the heart muscle produces chemicals that produce pain and other symptoms of angina.[3] Myocardial infarction (MI) is the irreversible necrosis of heart muscle secondary to prolong ischemia. The appearance of cardiac enzymes in the circulation generally indicates myocardial necrosis.[4]

There are several risk factors for the development of coronary heart disease (CHD). Dyslipidemia is recognized as a prominent risk factor for CHD.[5] The link between CHD and lipid has been firmly established first by epidemiologic studies and more recently by long term outcomes trials that demonstrated that lowering low density lipoprotein cholesterol levels significantly reduced the risk of major coronary events. Genetically determined and metabolically induced disturbances in lipid metabolism, as manifested in several types of dyslipidemia, have been shown to be causally related to the development of CHD. A reduction in serum total cholesterol (TC) levels has been shown to reduce mortality in patients with CHD and to decrease the need for revascularization.[6]

National Cholesterol Education Programme (NCEP), USA guidelines were used for definition of dyslipidemia as follows: (i) Hypercholesterolemia–serum cholesterol levels ≥200 mg/dl (≥5.2 mmol/l); (ii) Hypertriglyceri-demia–serum triglyceride levels ≥150 mg/dl (≥1.7 mmol/l); (iii) Low HDL cholesterol–HDL cholesterol levels <40 mg/dl (<1.04 mmol/l); (iv) High LDL cholesterol–LDL cholesterol levels ≥130 mg/dl (≥3.4 mmol/l) calculated using the Friedewald equation.[7] The present study was designed to evaluate the prevalence of dyslipidemia in CHD patients on context of our country.

Materials and Methods

A cross sectional study was conducted in the department of Biochemistry, BSMMU from July 2006 to June 2007. For this study, 300 diagnosed patients (209 male and 91 female) of coronary heart disease were selected from department of cardiology of BSMMU, NICVD and Enam medical college, Savar, Dhaka. Consents were taken from all study subjects preserving their rights, privileges and freedom. Fasting blood samples were collected after 14 hours fasting. Lipids were measured by using cholesterol oxidase para aminoantipyrine, lipase/glycerol kinase (LIP/GK), enzymatic reaction respectively and low density lipoprotein (LDL) cholesterol was calculated by Freidwald formula. Dyslipidemia was considered when anyone of the NCEP lipid profile criteria was satisfied in a case. All data were recorded systemically in a data collection form. Statistical analyses were performed by using SPSS for windows version 12.

Results

Among the 300 patients selected, 209 were male and 91 were female. Mean ± SD age of the patients was 51.56 ± 24.04 with the range of 25-74 years (Table I).

Table I: Sex distribution of study subjects

Sex	Number	Percentage
Male	209	73.49%
Female	91	26.51%

The study subjects were divided on the basis of presence or absence of dyslipidemia. Among them 83 (27.7%) had dyslipidemia whereas 217 (72.3%) had no dyslipidemia (Table II).

Table II: Incidence of dyslipidemia in study subjects

Status of dyslipidemia	Prevalence	Percentage
Present	83	27.7 (%)
Absent	217	72.3(%)

The mean ± SD of serum total cholesterol of CHD patients with hypercholesterolemia and CHD patients without hypercholesterolemia were 206.38 ±4.44 mg/dl and 151.31± 17.97 mg/dl respectively, t value is 51.52 and p value is 0.0001. The difference between mean ± SD is statically significant indicated hypercholesterolemia increase the risk of CHD (Table III).

Table III: Comparison of serum total cholesterol between CHD patients with hypercholesterolemia and CHD patients without hypercholesterolemia

Variable	Means ± SD mg/dl	*t* value	*p* value
CHD patient without hypercholesterolemia	151.31 ± 17.97	51.52	0.0001
CHD patient with hypercholesterolemia	206.38 ± 4.44		

The mean ± SD of serum LDL cholesterol of CHD patients with high LDL cholesterol and CHD patients with normal LDL cholesterol were 171.31 ± 17.14 mg/dl and 97.25 ±18.18mg/dl respectively, t value is 51.34 and p value being 0.0001. The difference between mean ± SD is statically significant. That means high LDL cholesterol increase the risk of CHD (Table IV).

Table - IV: Comparison of serum LDL cholesterol between CHD patients with high LDL cholesterol and CHD patients with normal LDL cholesterol

Variable	Means ± SD mg/dl	*t* value	*p* value
CHD patient with LDL ≥130 mg/dl	171.31 ± 17.14	51.34	0.0001
CHD patient with LDL <130 mg/dl	97.25 ±18.18		

The mean ± SD of serum HDL cholesterol of CHD patients with low HDL cholesterol and CHD patients with normal HDL cholesterol were 34.91 ±3.56 mg/dl and 45.91 ±4.66 mg/dl respectively, t value is 32.48 and p value being 0.0001. The difference between mean ± SD is statically significant. That means low HDL cholesterol increases the risk of CHD.

Table V: Comparison of serum HDL cholesterol between CHD patients with low HDL cholesterol and CHD patients with normal HDL cholesterol

Variable	Means ± SD mg/dl	*t* value	*p* value
CHD patient with HDL ≥40 mg/dl	45.91 ± 4.66	32.48	0.0001
CHD patient with HDL <40 mg/dl	34.91 ± 3.56		

The mean ± SD of serum triglyceride of CHD patients with between CHD patients with hypertriglyceridemia and CHD patients without hypertriglyceridemia were 156.38 ± 4.44 mg/dl and 108.78 ± 15.93 mg/dl respectively, t value is 49.85 and p value being 0.001. The difference between mean ± SD is statically significant. That means hypertriglyceridemia increase the risk of CHD.

Table VI: Comparison of serum triglyceride between CHD patients with and without hypertriglyceridemia

Variable	Means ± SD mg/dl	*t* value	*p* value
CHD patient without hypertriglyceridemia	108.78 ± 15.93	49.85	0.001
CHD patient with hypertriglyceridemia	156.38 ± 4.44		

Discussion

In our study we measured fasting blood lipid profile. We found age range of the study subjects (300 CHD patients) 25-74 years, among them 209 were male and 91 were female. This study showed that the total cholesterol in CHD patients with hypercholesterolemia (206.38 ± 4.44 mg/dl) significantly differs from the total cholesterol in CHD patients without hypercho-lesterolemia (151.31 ± 17.97 mg/dl) which is statistically significant. We observed that total LDL in CHD patients with high LDL cholesterol (171.31 ± 17.14 mg/dl) significantly differs from total LDL in CHD patients with normal LDL cholesterol (97.25 ± 18.18 mg/dl) which is statistically significant. We also observed that total HDL cholesterol in CHD patients with low HDL cholesterol (34.91 ± 3.56 mg/dl) significantly differs from HDL in CHD patients with normal HDL cholesterol (45.91 ± 4.66 mg/dl) which is statistically significant. In addition serum triglyceride of CHD patients with hypertriglyceridemia (156.38 ± 4.44 mg/dl) significantly differs from serum triglyceride in CHD patients without hypertriglyceridemia (108.78 ± 15.93 mg/dl) which is statistically significant. In our study, 27.7% of the CHD patients had dyslipidemia. Above findings revealed that the incidence of dyslipidemia in CHD patients was 27.7% and differences between mean of abnormal and normal level of parameter of lipid profile (serum total cholesterol level, serum LDL level, serum HDL level and serum triglyceride level) are

statistically significant. Similar type of findings were also observed by Gupta et al,[8] in which dyslipidemia represent 28% of the CHD patients in the city of Rajasthan, India. But different findings was also observed by Namita et al,[9] in which dyslipidemia account for 41.3% of the CHD patients in India.

We found in our study the incidence of dyslipidemia in CHD patient different from the findings of other studies in different places probably due to low socio-economic condition, lack of education and variation of sample number selection. So, further study on larger sample size should be carried out in future.

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Contribution of the Authors

First author designed and conducted the study and wrote the manuscript. Second and third authors critically reviewed the manuscript. Fourth author helped in data collection and statistical analysis. Last one was the supervisor of this study.

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A Study on Chronic Backache at a Primary Health care Centre of Bangladesh

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Abstract

Introduction: *This study was carried out on chronic back ache cases to have a look into the presenting age, predisposing factors or causes and level of nerve root involvement.*

Methods: *Cases were collected from October 2010 to March 2011 by purposive sampling technique. Patients of either sex with age range of 15 - 65 years were included. Findings of the cases were recorded with a predesigned data sheet. Results were expressed as actual number as well as percentage of total involved.*

Results: *Total 112 cases were included in this study - 44% were male and 56% were female. Age range was 15 - 65 years; maximum age incidence was observed around 45 years. Regarding predisposing factors, heavy weight lifting or pulling was 21%, maintaining bending posture for long time was 25% and fall from height, injury or RTA was15%; non-spinal as gynaecological and renal cases were also seen. Maximum number of level of involvement was L_5-S_1 (54%) and next was L_4-L_5 (37%).*

Conclusion: *In conclusion, we may say, if we can develop public awareness regarding the use of a heavy waist-belt/lumbosacral corset (which can reduce the pressure or torsion in the waist) during occupational or personal activities; it may reduce huge amount of man-power loss due to back ache in those persons who are at risk.*

Key words*: Backache, Weight lifting, Prolapsed lumbar intervertebral disc*

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Introduction

Chronic low back pain is a common cause of long term disability in middle age in many countries.[1] This sort of pain is sometimes resistant to treatment and patients are often referred for multidisciplinary consultation.[2]

The lifetime prevalence of low back pain has been reported at between 60 to 80 %. By contrast, the lifetime prevalence of true sciatica is between 2 to 4%. It is generally accepted that 90% of acute low back pain episodes settle, allowing return to work within 6 weeks. However, some 5-7% of the population aged between 45 to 64 years will report back problems as a chronic sickness. Up to 70% of acute episodes of sciatica resolve within 3 months.[3]

The usual symptoms of back disorders are pain, stiffness, and deformity in the back and pain, paresthesia or weakness in the lower limbs. The mode of onset may be sudden, perhaps after a lifting strain or may be gradually without any antecedent event as in case of excess body weight. The symptoms may be constant, or there may be periods of remission. It may be related to some particular posture. Vertebral Tuberculosis or secondary deposits are associated with spine unrelated symptoms too. Pain, either sharp and localized or chronic and diffuse, is the commonest presenting symptom. Backache is usually felt low down and or either side of the midline, often extending into the upper part of the buttock and even into the lower limbs. Back pain made worse by rest would suggest pain arising from the facet joints. Pain made worse by activity probably comes from any of the soft tissue supports of the spine (muscles or ligaments) including the annulus of the intervertebral disc.[4]

Sciatica is the term originally used to describe intense pain radiating from the buttock into the thigh and calf more or less following the distribution of sciatic nerve and therefore suggestive of nerve root compression or irritation. Kellgren 1977, in a classic experiment, showed that almost any structure in a spinal segment can, if irritated sufficiently, give rise to referred pain radiating into the lower limbs. Unfortunately, with the passes of time, many clinicians have taken to describe all types of pain extending from the lumbar region into the lower limbs as sciatica. This is at best confusing an at worst a preparation for misdiagnosis. True sciatica, most commonly due to a prolapsed intervertebral disc pressing on a nerve root, is characteristically more instance than referred to back pain, is aggravated by coughing and straining and is often accompanied by symptoms of root pressure such as numbness and paraesthesia, is especially in the foot.[4]

Other causes of backache- tumours of the spinal column, TB spine, osteoarthritis, spondylolisthesis, prolapsed intervertebral disc, ankylosing spondylitis, vascular occlusion, intrapelvic mass, Arthritis of the hip, tumours of the ilium or sacrum etc.[5]

Non spinal causes of pain must also be considered–respiratory (mesothelioma), vascular (abdominal aortic aneurysm), renal (pyelonephritis), gastrointestinal (peptic ulcer, pancreatitis) and urogenital (testicular, ovarian or prostatic carcinoma).[3] In female, genital prolapse, chronic cervicitis (PID), pedunculated sub-endometrial uterine polyps or complications of gynaecological surgery may also produce chronic backache.[6]

Back pain is a common reason for patient visit to primary care clinics. Despite a large differential diagnosis, the precise aetiology is rarely identified, although musculo-ligamentous processes are usually suspected. Episodes of acute, non-specific low back pain are usually self limiting and so many patients treat themselves without contacting their primary care clinicians. When patients visit clinicians, they require proper evaluation. The history and physical examination usually provide clue to the potentially serious causes of low back pain as well as identify patients at risk of prolonged morbidity.[7]

Taking this matter into consideration, this study was done to have a look into present age, causes and level of spinal involvement of the patients with backache – a knowledge that may help us in further planning for management of backache cases in a primary health centre (ie, Upozilla Health complex) of our country.

Materials and Methods

This cross sectional descriptive type of study was conducted at Sariakandi Upozilla Health Complex, Bogra. Cases of this study were collected from October 2010 to March 2011 randomly and conveniently by purposive sampling technique from the patients came to the Out Patients Department (OPD) of that health complex. Patients with persistent or intermittent back pain, lasting for more than 6 weeks, of either sex with age range of 15 to 65 years were included in this study. To maintain proper randomness only the first case of a day with the complaint of backache was included in sample and was examined thoroughly and recorded in data sheet. For time and manpower constrain, i.e., for maintenance of proper OPD service side by side, rest of the OPD patients with backache were excluded. History was taken, patients were examined properly and necessary investigations were done and then findings were recorded with the help of a predesigned data sheet.

To determine the level of involvement following examinations were done: (i) Posture, kyphosis, scoliosis, muscular spasm, (ii) Gait, +ve heel walking, toe walking, (iii) Straight Leg Raising test (>70^0 was considered normal), (iv) Reverse SLR (femoral nerve stress test), (v) Well leg raising test (Cross sciatic tension test), (vi)- Lasegue's test, (vii) Knee and ankle jerks, and (viii) Extensor halucis longus tendon power.

To determine the cause of backache, cases with clinical examination positive for spondylosis were investigated for X-ray spine ± CT scan or MRI as needed. Cases with clinical examination negative for spondylosis were investigated for ultrasonography of abdomen, CBC, urine R/E, serum creatinine, PSA or chest X-ray etc. as per requirement. In addition, cases seemed to be non-orthopaedic were referred to respective physician for proper evaluation. Findings were expressed as actual number as well as percentage of total involved.

Results

A total of 112 cases were included in this study. Out of them 49 (44%) were male and 63 (56%) were female with a male to female ratio is 1:1.28.

Age range was 15 to 65 years with a mean (± SD) and 45.9 (±9.71) years. Mean (± SD) age of the male patients was 46.5 (± 9.47) and of female patients was 45.5 (± 9.96) years. The difference of age between male and female was insignificant (t = 0.543, p > 0.10). (Table I).

Table I: Mean age of male and female cases

Sex	Age mean (±SD)	*t* value & *p* value
Male (n-49)	46.5 (± 9.47)	t = 0.543
Female (n-63)	45.5 (± 9.96)	p = 0.10

Among all the cases, 2 cases were in the age group of 15 - <25 years, 10 cases in 25 - <35 years, 42 cases in 35 - <45 years, 35 cases in 45 - <55 years & 23 cases were in 55 - <65 years. Male to female proportion in different age groups are demonstrated in Figure-1 below:

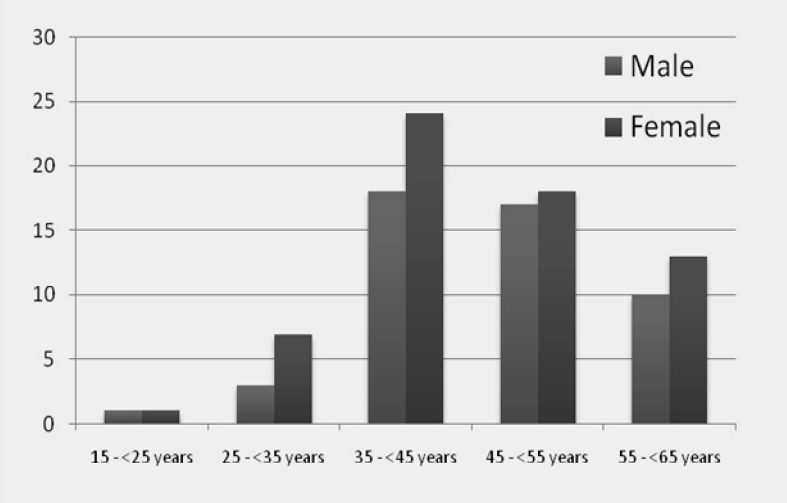

Figure 1: Distribution of cases in different age group among male and female

As per clinical examinations and investigation findings, the major predisposing factors of back pain was shown in (Table II).

Table II: Predisposing factors of backache among all the cases

Predisposing factors /or cause of backache	Number of cases		
	Male (n_1-49)	Female (n_2-63)	Total (n-112)
1. Heavy weight lifting or pulling	14 (29%)	10 (16%)	24 (21%)
2. Maintaining bending posture for long time	8 (16%)	17 (27%)	25 (22%)
3. Trauma (Fall from height, injury or RTA)	8 (16%)	7 (11%)	15 (13%)
4. Excessive traveling	2 (4%)	1 (2%)	3 (3%)
5. Excess body weight	3 (6%)	8(13%)	11 (10%)
6. Spina Bifida / deformity	2 (4%)	2(3%)	4 (4%)
7. TB spine	0	1 (2%)	1 (1%)
8. Osteoarthritis	5 (10%)	7 (11%)	12 (11%)
9. Inflammatory arthritis	2 (4%)	1 (2%)	3 (3%)
10. Malignancy / secondary deposits	2 (4%)	2 (3%)	4 (4%)
11. Gynaecology	--	7 (11%)	7 (6%)
12. Kidney	1 (2%)	0	1 (1%)
13. COPD	2 (4%)	0	2 (2%)

Out of total 112 cases 98 were confirmed by X-ray, CT scan and / or MRI.

The highest level of spinal involvement was at the level L5 - S1 (Table III).

Table III: Shows the level of involvement of all the cases

Spinal nerve root involved	Number of cases (Total-98)
T_{12}-L_1	1 (1%)
L_2-L_3	2 (2%)
L_3-L_4	5 (5%)
L_4-L_5	36 (37%)
L_5-S_1	53 (54%)

Discussion

In this study number of female cases were higher than male. Shakoor et al. also found similarly higher female number than male.[8] In a different study, males were found more in number than the females.[9] Mean age of all cases was 45.9 years with male patients mean age was 46.5 years and of female patients mean was 45.5 years and the difference was insignificant; maximum incidence was also observed around the age of 45 years. Shakoor et al. found mean age to be 42.2 years with the maximum incidence was around 42 years; which is also, more or less, similar to our study.[8] The predisposing factors of backache found in this study represents, more or less, to the causes of back ache mentioned earlier.[3-6]

In this study, highest frequency of spinal nerve root involvement was observed in L_5-S_1 segment (54%) and next to that was L_4-L_5 segment (37%); and, these two segments together makes 91%. In a study Wheeler et al. mentioned that over 90 percent are L_5 and S_1 radiculopathies and most sciatica is attributable to radiculopathy at the L_5 or S_1 level from a disc disorder.[10] It is to mention that in western countries, back pain is the most common cause of sickness related work absence and in UK 7% of adults consult their General Physicians each year with back pain.[11]

In our study, we found high number of cases related to heavy weight lifting or pulling and maintaining bending posture for long time in their occupational or personal lives. So, in conclusion, we may say, if we can develop public awareness regarding the use of heavy waist-belt/lumbosacral corset (which can reduce the pressure or torsion in the waist) during their occupational or personal activities, it will reduce huge amount of man-power loss due to back ache.

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Acral Acanthosis Nigricans: its update and management

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Abstract

Acanthosis nigricans is a skin condition that causes thick, velvety and darkened skin areas (due to increased thickness of epidermis). It commonly affects the skin of the armpits, the groin region, head and neck (back of the neck), and anal/genital region. Acral (indicating peripheral body parts) acanthosis nigricans is one among the seven types of acanthosis nigricans. It is different from the other types of acanthosis nigricans in that the lesions are present on the skin overlying the ankles, knee, fingers and toes. Acral acanthosis nigricans is usually diagnosed by a thorough clinical history and physical examination. Even though, it is a benign condition, dermatologist consultation and testing is necessary to rule out other causes of the condition. There is no definitive treatment for acral acanthosis nigricans. However, certain treatment modalities may be used for cosmetic reasons. The prognosis is typically good with no known major complications being noted.

Key words: *Acanthosis nigricans, Acral acanthosis nigricans, Obesity, Insulin resistance*

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Introduction

The disease acanthosis nigricans (AN) is characterized by the presence of a thick macule with a brown velvet-like surface.[1] Lesions commonly occur in the axillary region, neck, inguinal region, anticubital fossa, and popliteal fossa. Schwartz has categorized the entity into following eight types: benign, malignant, associated with obesity, syndrome, unilateral, drug induced, mixed and acral.[2,3] Acral-type acanthosis nigricans, also called as acral acanthotic anomaly is a dermatitis characterized by velvety, papilomatous, brownish black typically affected to the dorsum of the hand and foot. It is apparently_more common in persons with a dark complexion and affects acral areas without prominent affection of axilla and other flexures.[2] Acral AN usually

affects individual who are usually healthy and have no associated diseases/conditions unlike other forms of that is seen in association with an underlying condition. We accumulated several previous articles regarding this disease through internet web search, but in fact, we have a few literatures regarding this disease. In this review, we discuss with an aim to evaluate the pathogenesis of disease and its clinical implications and management.

Prevalence

A high prevalence has been observed recently due to the rising prevalence of obesity and diabetes. The prevalence varies from 7% to 74%, according to age, race, frequency of type, degree of obesity and concomitant endocrinopathy.

Clinical features

Individuals of any age group can be affected by Acral AN, but it is most commonly seen in adult population. Both males and females of all races and with ethnicities can be affected. However, it more commonly affects individuals of darker skin tones. Thus, people of African origin can be affected more than other populations. Other factors may play a role with respect to other types of AN, such as insulin resistance, cancer, medication etc. Clinically, it most commonly presents as hyperpigmented velvety, poorly-defined skin lesions as shown in clinical photograph (Figure 1). The lesions are limited to the top portion of the feet, elbows, knuckles, and knees (upper and lower extremities). The hyperpigmentation can be either brown or black. The affected individuals are usually healthy. Acanthosis nigricans is occasionally pruritic.[4]

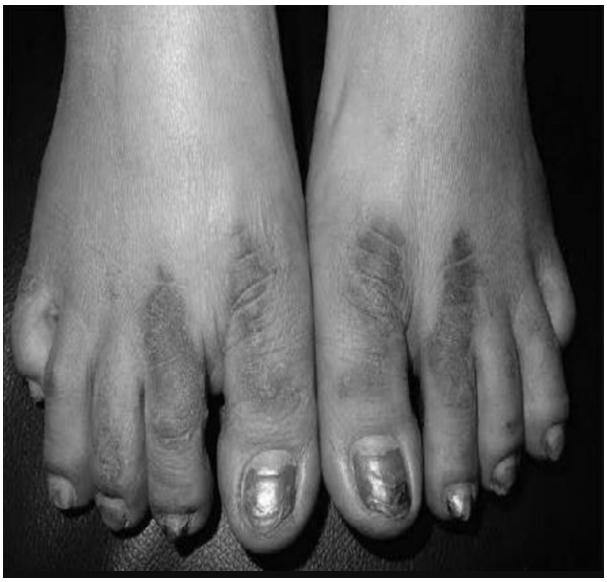

Figure 1: Bilateral hyperpigmented, hyperkeratotic skin lesions

Histopathology

Histopathology, it reveals a thickened stratum corneum with minimal involvement of the dermis except for thickened and elongated dermal projections. The thickening of stratum spinosum (acanthosis) is variable and typically mild.[5,6] The dark color of AN is likely due to hyperkeratosis rather than to a mild increase in melanin pigmentation.[2] Infiltration of lymphocytes, plasma cells or neutrophils may be present.

Association of other diseases

Acanthosis nigricans is linked to a variety of syndromes. Most are associated with insulin resistance or fibroblast growth factor receptor (FGFR). AN may also appear as an adverse effect of several medication such as glucocorticoids, niacin, insulin, oral contraceptives and protease inhibitors.[7-12] Recently, it has been reported that acral AN is associated with dermatofibrosarcoma protube-rans, non-Hodgkin's lymphoma.[13, 14]

Pathogenesis

The pathogenesis of acral type of AN is similar to acanthosis nigricans. Insulin has been demonstrated to cross dermoepidermal junction (DEJ) to reach keratinocytes. Elevated insulin concentrations result in direct and indirect activation of IGF-1 receptors on keratinocytes and fibroblasts, leading to proliferation (Figure 2). Other mediators may also contribute, including other tyrosine kinase receptors such as EGFR epidermal growth factor receptor and FGFR (fibroblast growth factor receptor). Acanthosis nigricans is commonly associated with disorders with insulin resistance, including obesity, type 2 diabetes, and the polycystic ovary syndrome.[15] At high concentrations, however, insulin can exert more potent growth-promoting effects through binding to insulin like growth factor 1 receptors (IGF-1Rs). A number of observations suggest that insulin-dependent activation of IGF-1Rs can promote cellular proliferation and facilitate the development of AN. The severity of AN in obesity correlates positively with the fasting insulin concentration.[16, 17] Thus, insulin may promote AN through direct activation of the IGF-1 signaling pathway. The true pathogenesis of AN, however, is likely to be more complex. Hyperinsulinemia may also facilitate the development of AN indirectly by increasing the levels of free IGF-1 in the circulation.

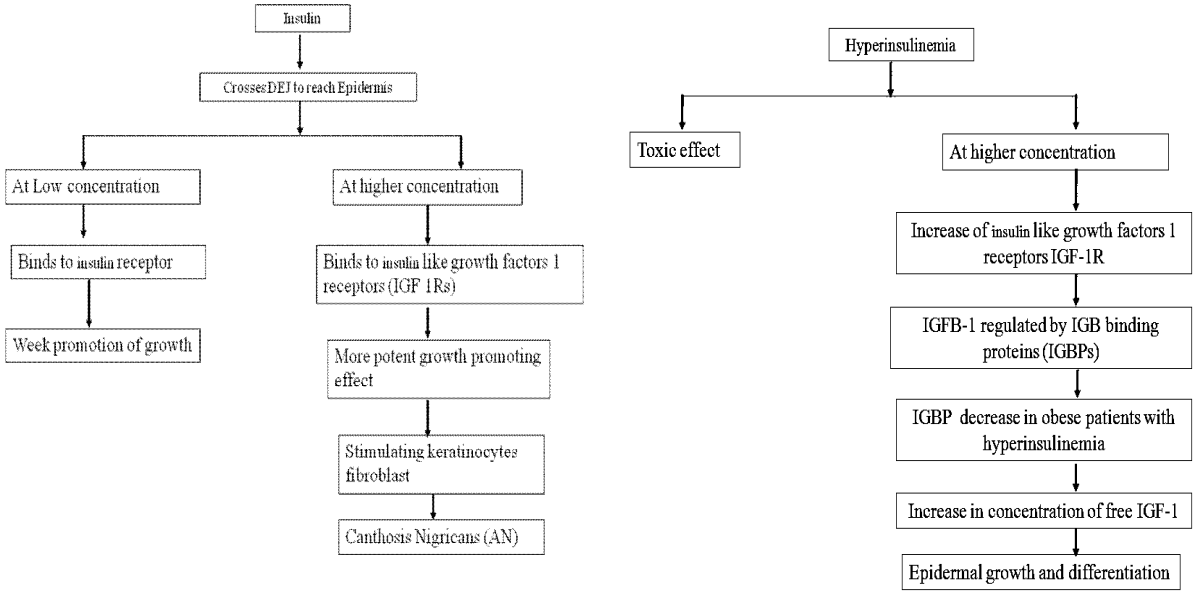

Figure 2: The flow chart showing the pathogenesis and role of IGF on signal pathways on keratinocytes and melanocytes

Diagnosis

Diagnosis is mostly clinical and can be confirmed by histopathologic observations. The lesion may demonstrate a benign epidermis with hyperkeratosis, papillomatosis, thickening of epidermis (acanthosis), increased number of melanocytes (melanocytic hyperplasia) and lymphocytic inflammation. Interestingly, there are only fewer acanthosis with no hyperpigmentation that do not fit the histologic terminology. The hyperpigmented appearance is actually due to hyperkeratosis. Microscopically, all the seven types of share similar features.

Investigations

Diagnosis is based on clinical with histopathology examination needed for confirmation. Histological findings are similar in all forms of AN. Spectroscopic and colorimetric measurements combined with chemometric analysis methods also provide sensitive and specific diagnosis of AN.[17]

Evaluation and Management

In majority of cases, improvement of the skin lesions is the primary concern of patients presenting to dermatologists. Treatment of its underlying condition often results in improvement of AN. Therapeutic approach involves treatment of underlying disease or tumor, cessation/avoidance of the inciting agent in drug-induced AN, use of topical/oral agents and cosmetic surgery.[18] Weight loss and exercise have shown to increase insulin sensitivity and reduce insulin levels causing improvement in obesity associated AN. Correction of hyperinsulinemia reduces hyperkeratotic lesions.

Topical Treatment

Retinoids

It is epidermopoietic and causes a reduction of the stratum corneum replacement time. It corrects hyperkeratosis and causes near complete reversion to normal state. Lahiri and Malakar in their study have reported that intermittent tretinoin application is needed to maintain improved status.[19]

Ammonium lactate and tretinoin

Lactic acid is an alpha-hydroxy acid that works as a peeling agent and also via release of desmogleins, indicating disintegration of desmosomes. Retinoids affect cell growth, differentiation, morphogenesis and alter cell cohesiveness. Though the exact mechanism of action of the two agents is unknown, synergistic interaction is thought to play a role.[20]

Peels

Trichloroacetic acid (TCA) is safe, easily available, cheap, and easy to prepare. It causes precipitation of proteins of epidermal cells leads to necrosis and destruction of epidermis, followed by inflammation followed by activation of wound repair mechanisms. This leads to re-epithelialization with replacement of smoother skin. The previous study have reported improvement of AN in six female patients after TCA peeling.[21] The advantages of TCA are that it is a stable product, safe and effective therapeutic modality for AN in comparison to other topical treatments. Topical tretinoin needs frequent application for long duration and improves hyperkeratosis, but not hyperpigmentation. Topical salicylic acid, podophyllin, urea, and calcipotriol need frequent application, while TCA peel is done in two to three sessions. Dermabrasion or alexandrite laser are expensive and may lead to post inflammatory hyperpigmentation.

Calcipotriol

A beneficial agent that inhibits keratinocyte proliferation and promotes differentiation by increasing intracellular calcium levels and cyclic GMP levels in keratinocytes. It is safe, well-tolerated, alternative beneficial treatment when an etiological treatment is not possible or necessary.[22]

Miscellaneous treatment

Other therapies (case reports) are included likely fish oil, 20% podophyllin in alcohol , and surgical

excision, Urea, salicylic acid and triple combination depigmenting cream (tretinoin 0.05%, hydroquinone 4%, fluocinolone aceto-nide 0.01%) with sunscreens are other options.[20-25]

Oral Treatment

Oral retinoids

Oral retinoids (isotretinoin, acitretin) can be effective agent causing improvement, it requires large doses and extended courses, and relapses are described.[26] The mechanism of action is probably normalization of epithelial growth and differentiation.[27] Acitretin has been rarely reported for AN treatment and has showed good success in cases with syndromic and benign AN. Oral isotretinoin has been used successfully to treat extensive AN.[28]

Metformin and rosiglitazone

Oral anti diabetic agents (Metformin and rosiglitazone) are useful in reduction in fasting insulin levels with rosiglitazone when compared to metformin and modest improvement of skin texture with both.[29] Metformin reduces glucose production by increasing peripheral insulin responsiveness, reduces hyperinsulinemia, body weight and fat mass and improves insulin sensitivity.[30, 31]

Cosmetic treatment

For cosmetic purpose, long-pulsed alexandrite laser, which was designed to target melanin in hair, could improve this condition. They hypothesized that thermal heating of epidermis and dermis results in tissue remodeling and pigment reduction. They reported 95% clearance after seven sessions and concluded that long pulsed alexandrite laser can effectively and safely treat AN. [20, 23, 32]

Prognosis (Outcomes/Resolutions)

Thus, the prognosis of AN is excellent with adequate (skin) treatment. However, the overall prognosis depends upon the underlying cause of the condition. The prognosis of acral AN is excellent with no associated major complication being noted.

Conclusion

Though mainly a disease of cosmetic concern, AN can be pointer to underlying metabolic syndrome or malignancy. A thorough investigation and treatment is therefore mandatory to prevent long term consequences.

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CASE REPORT NORTH BENGAL MEDICAL COLLEGE JOURNAL [2017], VOL., 3, No. 2 : 49-52

Delayed Recovery from General Anaesthesia after Adequate Reversal

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Abstract

Delayed recovery from anaesthesia is a distressing as well as awkward situation for an anaesthetist and also for the surgeons. Everybody wants their patient regain consciousness immediately after operation. We present a case of 35 years old lady who regained her consciousness, 24 hours after operation. All her investigations reports were almost normal and she was given anaesthetic fitness for Sub total Thyroidectomy operation. In the operation theatre, she was a bit worried and anxious looking. After a very good and successful operation she was given as usual reversal but she did not respond. After 24 hours, she started to follow commands from anaesthetist i.e opening eyes, protruding tongue etc. During these times she was treated accordingly with, Inj. 10% dextrose, Inj. Frusemide, Inj Hydrocortisone, whole blood transfusion etc.

Key words: *Delayed recovery, CNS depression, Hypoglycaemia, Drug overdose*

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Introduction

It would be normal to anticipate the return of consciousness within 10 minutes of the end of an operation. Failure to recover consciousness 60 minutes after the end of an anaesthesia requires investigation, unless an obvious preoperative cause is apparent.[1,2] In spite of apparent return of awareness, failure of memory and confusion may persist for sometime postoperatively and do not necessarily require treatment.[3,4] It is painful, alarming, as well as distressing situation for the anaesthetist and also for the surgeon which should be dealt carefully and patiently.[4,5]

Case Report

Khushi Begum, aged-35 yrs of age, post office Kazipur district Sirajganj was admitted in surgical ward of North Bengal Medical College Hospital. Thyroid enlargement was diagnosed and she was prepared for thyroidectomy operation and was sent to anaesthesiology department for anaesthetic fitness. All of her

investigations were found quite normal. She was given anaesthetic fitness. On the day of operation, she became a bit abnormal due to anxiety and fear. Her B.P was slightly elevated. She was given pre-medication with Inj.-Pethidine 75 mg and Inj-Atropine 0.6 mg intramuscularly. Induction of anaesthesia was given with Thiopentone and intubation was carried out by using Suxamethonium (at 1.5 mg per kg dose). Anaesthesia was maintained with Inj. Vecuronium, Halothane, Nitrous-Oxide and Oxygen. The surgeon was very competent and he took 1 hr. and 30 minutes to complete the operation. Intra venous Pethidine- 50 mg was given in between operation. At the end of operation all anaesthetic agents were cut-off and 100% oxygen were given. After that the patient was reversed with Inj. Neostigmine 2.5 mg and Inj. Atropine-1.2 mg. It was noticed that 5 minutes after reversal, there was no respiratory effort, intermittent positive pressure ventilation was continuing with 100% oxygen. After 15- 20 minutes breathing effort returned gradually. After 30 minutes, the patient started breathing quietly rhythmically, but consciousness did not return. All sorts of stimulation were given to her but there was no sign of returning consciousness. The patient was kept under close monitoring, and the pathologist was requested to repeat all the investigations again, including serum electrolytes, blood glucose level and blood gas analysis. Meanwhile, all supportive treatments were going on. On the next day at 11.00 pm, the patients' reflexes were returning (after 24 hours) back and the patient started to follow commands, i.e. opening the eyes, protruding the tongue, catching the fingers tightly. Within 28 hours, the patient became quite normal.

Discussion

The speed with which recovery of the response to pain and to command occurs depends upon the pre operative status of the patient's central nervous system. It may also affected by surgery, intraoperative events and the pharmacokinetics of the drugs administered during anaesthesia.[4] Full recovery of consciousness may vary according to the age of the patient, generally being longer in elderly and by pharmacokinetic effect of abnormal liver function, renal blood flow and protein binding of drug administered. It is not unusual for some degree of mental impairment to be present for 24 hrs. after major surgery.[6] It is difficult to assess the contribution of the anaesthesia, surgery and postoperative medication to this phenomenon, which is occasionally found after major surgery. Any condition reducing cerebral metabolism in the postoperative period is likely to cause delay in awakening from the anaesthesia. A fall in cerebral metabolism may be due to hypoxia, hypothermia, or reduced metabolic substrates in the blood.[7,9] Low cerebral perfusion pressure, caused by systemic hypotension, an obstruction to venous outflow from the brain or from increased intracranial pressure, will compromise cerebral function. Often a combination of factors is involved. Intraoperative cerebral hypoxia, due either to a fall in perfusion or to a critically lowered oxygen content of the blood, will affect postoperative recovery. Drug induced cerebral depression; including a failure to lower the plasma concentration of anaesthetic agents to sub hypnotic levels as a result of either overdose or pharmacokinetic factors is often a cause of modest delays in patients regaining full consciousness. Pathological or pharmacological condition that interferes with synaptic

transmission and neurotransmitter release causes drowsiness and prolonged unconsciousness. Hypothermia below 32^0C usually impairs consciousness. A failure of glucose to reach the cellular enzymes will cause drowsiness and unconsciousness. Respiratory and metabolic acidosis either associated with CO_2 retention or with a low bicarbonate will disturb the function of the pH-sensitive intracellular enzymes and may cause unconsciousness.[7,8] Adrenocortical failure may present as hypotension and unconsciousness.[9] During surgery prolonged retraction of cerebral tissue may have the same effect. Cerebral edema from water intoxication is usually a slowly progressive condition but if associated with inappropriate antidiuretic hormone secretion, it may complicate recovery from anaesthesia.[9] Hypotension and hypoxia occurring during an operation would alert one to this possible cause of cerebral damage. The commonest cause of a slow return of consciousness postoperatively is drug overdose or pharmacokinetic failure to lower plasma drug concentration often complicated by CO_2 retention. The administration of a dose of narcotic shortly before the end of the operation should alert to this as a cause of slow return of consciousness. In the presence of poor renal blood flow, narcotic metabolites may cause respiratory depression. If volatile anaesthetics have been administered in a concentration greater than two minimum alveolar concentrations they may well cause slow arousal.[10] In this case the patient regained consciousness delayed might be due to over anxiousness, fear and tension before operation. Nothing abnormality was detected in relevant investigation report.

Conclusion

The patient should be assessed carefully before operation, as it is a very awkward, distressing and unusual situation. If any doubt all the investigations should be repeated. Fluid and electrolyte imbalance should be corrected. Oxygen, carbon dioxide and glucose level should be normal and the blood pressure should be maintained at a mean of 70-90 mmHg. However a focal cerebral lesion such as intracranial tumors or localized haemorrhage should be looked for. Opiate analgesics should be used slowly and very carefully. Naloxone, an antidote for opiates should always be ready at hand. Blood grouping and cross matching should be done promptly if blood transfusion is required. Overdose of any drugs must be avoided.

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Canavan Disease- a rare Leukodystrophy

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Abstract

Canavan disease, also called Canavan–van Bogaert–Bertrand disease, is an autosomal recessive leukodystrophy that causes progressive damage to nerve cells in the brain and caused by a deficiency of the enzyme aminoacylase 2. A 22 years old male patient was presented in Department of Radiology and Imaging, Khwaja Yunus Ali Medical College and Hospital, Sirajganj with the complains of progressive weakness of all four limbs and gait disturbance since birth. On CT scan, it was revealed that diffuse hypointensity along white matter, subcortical arcuate fibers sparing thalamus and basal ganglia with thinning of cortex without any significant contrast enhancement. From the imaging findings and clinical presentations, the patient was diagnosed as a case of leukodystrophy and according to distribution of white matter ischemia, ultimately the patient was diagnosed as a case of Canavan disease. Although this is very rare disease, radiologist should be aware of this type of leukodystrophy so that patient is diagnosed properly and get adequate management.

Key words: *Canavan disease, Leukodystrophy, MRI*

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Introduction

Canavan disease, is an inherited genetic abnormality due to lack of an essential enzyme aminoacylase 2, causes destruction of myelin in the brain, thereby preventing the proper transmission of nerve signal.[1-3] Symptom appears in children between 3 to 6 month of age including developmental delay, significant motor slowness, enlargement of head (macrocephaly), loss of muscle tone (hypotonic), poor head control and severe feeding problem.[4] As the disease progresses seizures, shrinkage of the nerve of the eye (optic atrophy) and developed often blindness, as do heart burn (gastro-intestinal reflux) and deterioration of the ability to swallow. Canavan disease is inherited as an autosomal recessive condition with both parents silently carrying a single Canavan gene and each of their children

running about 25 % risk of receiving both genes and having the disease. Canavan disease is more prevalent among individuals of eastern European Jewish back-ground, then in others.[1,2,5] Canavan disease is caused by a defective ASPA gene which is responsible for the production of enzyme aspartoacylase. Decrease aspartoacylase activity prevents the normal break down of N-acetylaspartate, where in the accumulation of N-acetylaspartate or lack of its further metabolism interferes with growth of the myelin sheath of the nerve fibers of the brain. The myelin sheath is the fatty coverings that surround nerve cells and acts as insulator which allows for efficient transmission of nerve impulse.[5-7] On imaging, non-contrast CT scan shows diffuse low density throughout the cerebral white matter with normal sized ventricles. T1 wieghted scans in infantile Canavan Disease demonstrate homogeneous diffuse, symmetric low signal intensities throughout the white matter. T2 weighted images show near total high signal intensity in the supratentorial white matter. The subcortical arcuate fibres are predominantly involved. Relative sparing of the internal capsules is seen in few a cases. The cortex may appear thin (Figure 1).[6-8]

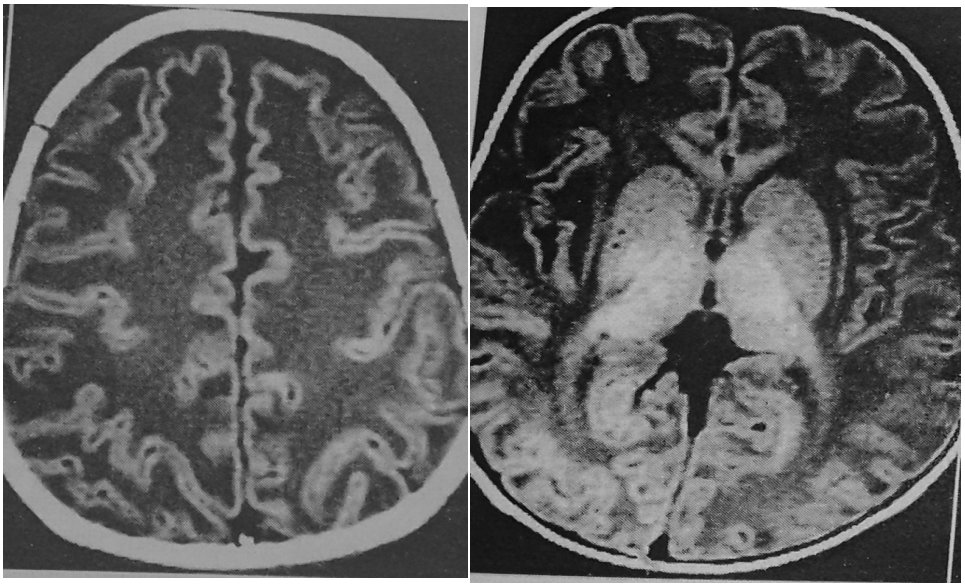

Figure 1: Non-contrast CT scan shows diffuse hypointensity along white matter, subcortical arcuate fibres sparing thalamus, basal ganglia with thinning of cortex

No definite treatment is present for Canavan disease. In addition, there is experimental trial of gene therapy, published in 2002, involving using a healthy gene to take over for the defective one that causes caravan disease. In human trials the results which were published in 2012. This method appeared to improve the life of the patient without long-term adverse effects during 5 years follow up. Death usually occurs before age 10 years but some children with milder forms of the disease survive up to their teen and 3rd decade.[3,5,7]

Case Report

A 22- years old male patient was presented with the complaints of progressive aesthenia of all

four limbs and gait disturbance since birth. On examination, muscle tone was decreased, GCS was 15/15, IQ was below average level (below 80). CT scan images demonstrated that diffuse hypointensity along white matter, subcortical arcuate fibre sparing thalamus, basal ganglia with thinning of cortex and normal sized ventricles without any significant contrast enhancement (Figure 2).

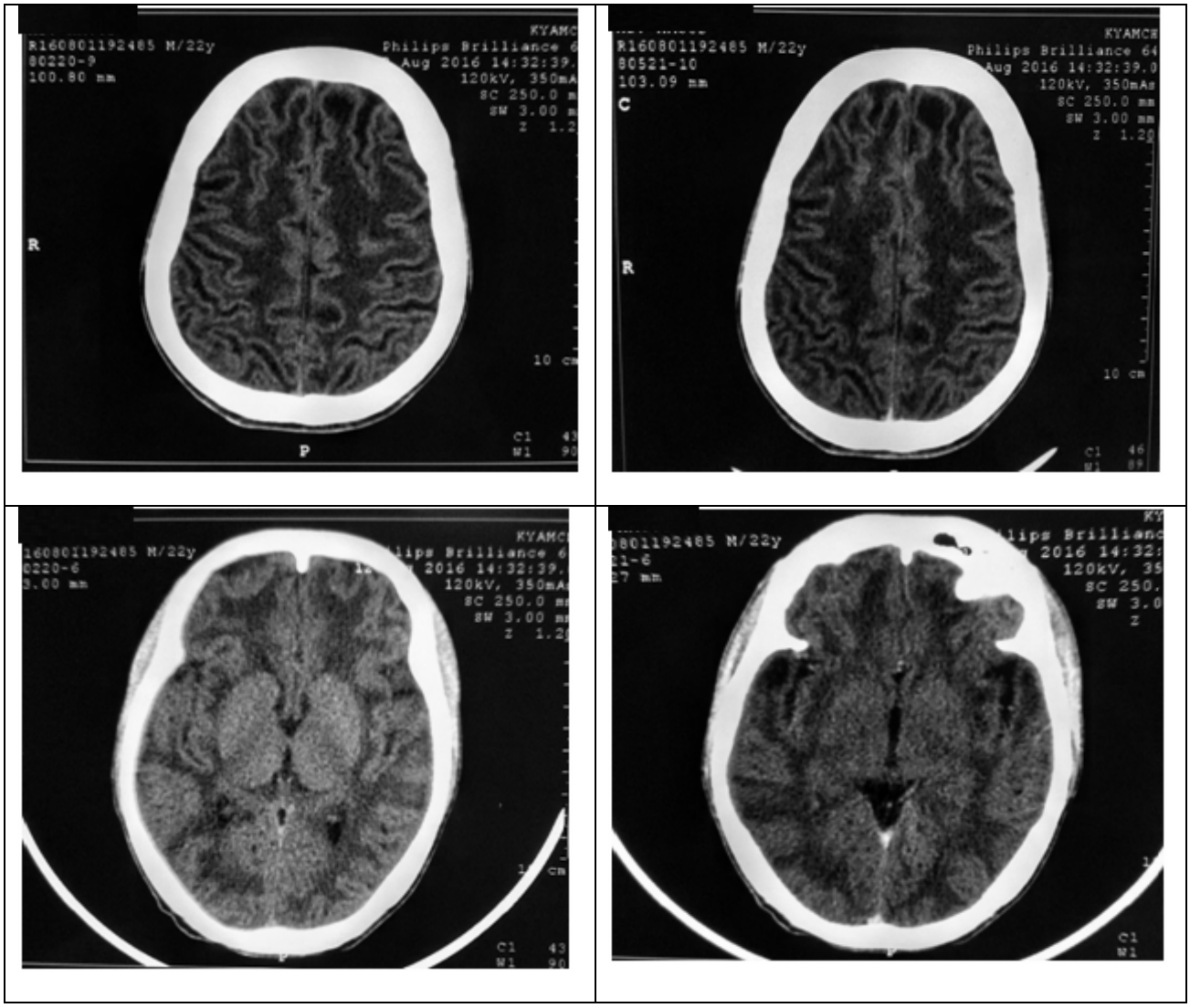

Figure 2: Non contrast and contrast CT of brain showing diffuse hypointensity along white matter, subcortical arcuate fibre sparing thalamus, basal ganglia with thinning of cortex and normal sized ventricles

MRI was advised but patient was unable to do it due to financial constrain. Considering the clinical presentations and imaging findings, the patient was diagnosed as a case of leukodystrophy and according to distribution of white matter ischemia; finally the patient was diagnosed as a case of Canavan disease.

Discussion

Canavan disease is a rare inherited disorder that damages the ability of neurons in the brain to send and receive messages. This disease is one of a group of genetic disorders called leukodystrophies.[1] Leukodystrophies disrupt the growth or maintenance of the myelin sheath, which is the covering that protects nerves and promotes the efficient transmission of nerve impulses. Neonatal/infantile Canavan disease is the most common and most severe form of the condition. Affected infants appear normal for the first few months of life, but by age 3 to 5 months, problems with development become noticeable. The mild/juvenile form of Canavan disease is less common. Affected individuals have mildly delayed development of speech and motor skills starting in childhood. These delays may be so mild and nonspecific that they are never recognized as being caused by Canavan disease.[3,4,6] Our had mild/juvenile form of Canavan disease as he crossed his infancy, had asthenia, hypotonia of all four limbs and gait disturbance since birth which was progressive. All the leukodystrophyies appear on imaging as diffuse hypodensity along white matter at CT scan and hyperintensity at T2 weighted MR imaging.[6] In present case of leukodystrophy, there was diffuse hypointensity along white matter, subcortical arcuate fiber sparing thalamus, basal ganglia with thinning of cortex. Previous researchers[5,6] observed that subcortical arcuate fibers are not affected in other leukodystrophy, where as in current case subcortical arcuate fibers were involved which favored Canavan Disease on imaging. Previous MR imaging[6] revealed thinning of cortex with normal sized ventricles in Canavan Disease. Similar findings were seen in our present case on imaging.

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