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Should be in second page and should usually be not more than 150 words in unstructured abstracts or 250 words for structured abstract (original article).

The structured abstract should have the following sections

(i) Objectives

(ii) Materials and methods

(iii) Place and period of work

(iv) Results

(v) Conclusion.

Below the abstract author should provide 3-10 key words.

Text

Should be presented in the form of –

Introduction:

The purpose of the article the rationale for the study or observation should be summarized, only strict pertinent references to be given and data or conclusion from the work being reported not to be included.

Material and methods

In this section selection of the observational or experimental subject (patient or laboratory animals, including control) should be described clearly. The age, sex and other characteristics of the subjects should be identified. Identify the methods, apparatus, and procedure in sufficient detail to allow other worker to reproduce the result. Give references to establish methods, including statistical methods. Precisely identify all drugs and chemicals used, including generic name, dose and route of administration. Author submitting review manuscripts are advised to include a section describing the methods used for locating, selecting, extracting and synthesizing data.

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In result section, when data are summarized, specify the statistical methods used to analyze them. Results to be presented in a logical sequence in the text, table and illustration Tables should be numbered consecutively in the order of their first citation in the text, and supply a brief title for each.

Place explanatory matter in footnotes, not the heading. Be sure that each table is cited in the text. Figure should be professionally drawn and photographed.

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Discussion

Should emphasize the new and important aspect of the study and the conclusions that follow from them. Relate the observations to other relevant studies.

Conclusion

Should be linked with the goals of the study Recommendation when appropriate, may be included.

Acknowledgements

May go to the text, one or more statements may specify

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Editorial action

Manuscripts are examined by editorial board and are sent to reviewers. All discussions to accept, review or refuse will be made by the editors. Rejected manuscript will not be returned to the authors. Proof correction by the author will be appreciated.

No reprint will be provided.

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An official organ of Ad-din Women's Medical College

Editorial

Urbanization and health

— Bangladesh perspective

"Urbanization and Health": The 21st century's challenge is related to many of the social and health hazards.

Although the degree of urbanization in Bangladesh today is relatively low. Which is more or less 30%? Bangladesh has one of the fastest rates of urban population Growth in the word. The capital city of Bangladesh, Dhaka, with around 15 million people, is already now one of the ten largest cities in the word. Its Slum population has been doubled in the last decade. From 1.5 to 3.5 million people. According to some statistics, more or less 40% of the total urban population in Bangladesh today lives below national poverty line, and the proportion had increased over the past few years. Within 30 years, 100 million people are expected to live in the cities of Bangladesh.

The slums of the major cities in Bangladesh grew by more than 13% a year between 1996 and 2005. Particularly the situation of children in these slums can be quite dire: They are frequently left alone in the care of older siblings while their parents work. They often do not attend school. Poor urban children also suffer from high levels of malnutrition and severe stunting than poor children also suffer from higher levels of malnutrition and severe stunting than poor children in the rural areas. Urbanization is undoubtedly a sign of development. However, unplanned and uncontrolled urban growth becomes a hindrance to sustainable development and multiplies different environmental hazards to the health of its citizens.

In Bangladesh, most migrants arrive at big cities – mostly Dhaka and Chittagong, because the city corporation and pourashavas (secondary cities) at present are able to offer few of the amenities associated with urban life.

Thus, in order to promote balanced urban growth in Bangladesh and to ease the pressure on the resources and infrastructure of the big cities, it is absolutely essential to develop the potential of the pourashavas to make them dynamic and livable cities, Where people can make a living: educate their children: find decent housing with running water, electricity and sewerage system and get high quality medical care.

Because of rapid urbanization and recent climate changes, several health and social hazards occur. They are as follows:

- Changes in geographic ranges and incidence of vector- borne diseases e.g. Malaria, Dengue, Kala-a-zar, etc.
- Changed incidence of diarrheal and other infectious disease, e.g. Typhoid. Paratyphoid. Hepatitis, Helminthic infections, Measles.etc.
- Malnutrition and Hunger, and consequent impairment of child growth and development.
- Psychological disorders, civil strife.
- Acute and chronic respiratory disorders.
- Tuberculosis, HIV/ AIDS and sexully transmitted diseases.
- Increased Violence.
- Road traffic and other accidents.
- Autism and neuro-development disorders.

To mitigate and prevent the health related hazards/problems due to urbanization. The health and other related social sectors can help by:

- Assessing the needs and demands of the peoples problem, how to organize the provision of care for

those problems considering the various aspects of social structure of Bangladesh.

- Conducting research to help understand the extent of the problem with a view to identify risk factors and develop evidence-based programs.
- Promoting healthy behavior and lifestyles through Life Skills Education using both formal and non-formal education channels.
- Creating a very precise supportive policy environment, technical and financial commitment of donors and UN-bodies, active engagement of professional bodies and academic institution.
- Stressing the need for more partnerships among the countries and regions.
- Encouraging the policy makers and health professionals to be prioritizing distinctly the health problems, especially Mother and Child.
- Achieving gender equality and empower all Women and girls.
- Ensuring availability and sustainable management of water and sanitation for all health in terms of adopting public healthy strategy and allocation of funds and other resources coping up with urbanization and climate change.
- Building active public private partnership.
- Developing and publicizing hotline emergency telephone services for summoning medical support on site.
- Developing policy guidelines in order to mobilize internal resources through intersectoral partnerships that include the private sector.
- Identifying epidemic-prone areas and developing sustainable operational plans for epidemic preparedness and control.
- Preparing an information system that is sensitive. Reliable and timely for monitoring program and serving as an early warning system.
- Strengthening local capacities along with continuous reassessment of the local situation.
- To secure people's right to ecologically healthy environment.
- To control water pollution, at the directive of the Department of Environment related industries set up Effluent Treatment Plant (ETP) for the treatment of their waste water.
- Home-based screening of autism and neuro developmental disorders in children, their early detection and to take immediate curative, preventive measures (e.g. household- based training for mothers) and their rehabilitation processes.

Dr. Md. Anwar Hossain Munshi
MBBS (DU), MPH (DU)

Original Article

Association of insulinotropic hormone with glycemic status in impaired glucose tolerance

Dr. Md.Ashraf-uz-zaman[1], Dr. Nasreen Sultana Lovely[2], Prof. Bilquis Ara Begum[3]

Abstract

Objective : Impaired glucose tolerance means that blood glucose is raised beyond normal levels. The major objective of the present work was to explore the association of GIP with dysglycemia or abnormal insulinemic status.

Methodology : Serum glucose was measured by glucose-oxidase method, serum lipid profile by enzymatic method, and serum C-peptide and serum glucose dependent insulinotropic hormone (GIP) were measured by ELISA method. Insulin secretory capacity (HOMA%B) and insulin sensitivity (HOMA%S) were calculated from fasting serum glucose and fasting serum insulin by homeostasis model assessment. The analytic observational study was conducted under a case subjects (n=51) and Controls (n=47). IGT was diagnosed following the WHO study group criteria.

Results : Mean (±SD) age (yrs) of the Control subjects and IGT subjects were 40±6 and 41±5 respectively (p=0.502). Mean (+SD) BMI of the Control and IGT subjects were 24.0±2.9 and 24.0±3.5 respectively (p=0.955). WHR of IGT subjects were statistically higher than the Control subjects, [Mean (+SD), 0.92±0.08 vs 0.88±0.05, (p=0.02)]. Median (Range) value of fasting serum glucose (FSG) of Control and IGT subjects were 5.2 (4.1-6.0) and 5.3 (4.4-6.0) mmol/l respectively. Fasting C-peptide value of IGT subjects was not significantly different from the value of the Controls. Serum (Range) value of fasting serum C-peptide of Control and IGT subjects were 0.64 (0.18-1.62) and 0.68 (0.21-1.39); (p=0.310). A significant positive correlation was found between fasting C-peptide and fasting GIP (p=0.019) in the control subjects. In multiple regression analysis a significant positive association was found between fasting C-peptide and fasting GIP (p<0.01). A significant negative association was found between fasting GIP and insulin sensitivity (HOMA%S) (p=0.05). No association was found between fasting glucose and fasting GIP both in simple correlation and multiple regression.

Conclusion : GIP does not have any association with insulin secretion in IGT subjects, but it has an association with insulin resistance.

Key Words : insulinotropic hormone, glycemic status, impaired glucose tolerance

Introduction

Diabetes mellitus (DM) is a heterogeneous group of metabolic disorders characterized by chronic hyperglycemia with disturbances of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, insulin action or both. When fully expressed, diabetes is characterized by fasting hyperglycemia, but the disease can also be recognized during less overt stages, most usually by the presence of glucose intolerance.

All forms of diabetes can pass through a stage of IFG and/or IGT. These categories are a part of the natural history of diabetes and not a type of diabetes. That is why they are not included in the classification of diabetes mellitus by ADA.

The pathophysiology of T2DM is a field of rigorous investigations. T2DM is well characterized by defects in insulin secretion, insulin action or both[1]. The progressive deterioration of pancreatic insulin secretion has been implicated as the proximate cause of the progressive increase in plasma glucose level.[2] Thus decrease in insulin secretion is a major contributor to the development of the overt T2DM state.

Two GI peptide hormones (the incretins) GLP-1 and GIP — were found to exert major glucoregulatory actions[3] within minutes of nutrient ingestion, GLP-1 is secreted from intestinal L cells in the distal ileum and colon, while GIP is released by intestinal K cells in the duodenum and jejunum.[3] GLP-1 and GIP trigger their insulinotropic actions by binding beta-cell receptors[3,4]. GLP-1 receptors are

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expressed on pancreatic glucagon-containing alpha and delta cells as well as on beta cells, whereas GIP receptors are expressed primarily on beta cells.[4] GLP-1 receptors are also expressed in the central nervous system (CNS), peripheral nervous system, lung, heart, and GI tract, while GIP receptors are expressed in adipose tissue and the CNS. GLP-1 inhibits glucose-dependent glucagon secretion from alpha cells.[3] In healthy individuals, fasting glucose is managed by tonic insulin/glucagon secretion, but excursions of postprandial glucose (PPG) are controlled by insulin and the incretin hormones.[5]

Methodology

The study was conducted in the Department of Biochemistry, General Medical Hospital, Dhaka during the period of July 2014 to June 2015. It was an observational analytic study with a case-control design. A group of 98 adult subjects with age ranging from 30-55 years and voluntarily agreed to include in this study were included and they were recruited from the Hospital. Of them impaired glucose tolerance (IGT) 51 were taken as study subjects and 47 as control. Patients with serious co-morbid diseases (severe infection, stroke, myocardial infarction, major surgery, mal-absorption etc) were excluded.

Fasting blood was collected between 8.00-9.00 am. Venous blood (05 ml) was taken with the subject sitting comfortably in a chair. After 30 minutes blood samples were centrifuged for 10 minutes at 3000 rpm to obtain serum. Separated serum was adequate and preserved in a freezer (-27° C) for future biochemical analysis. Fasting serum glucose, triglyceride, total cholesterol, HDL cholesterol, creatinine and SGPT were measured in the same day. Homeostasis Model Assessment (HOMA) is a simple widely used method which derives separate indices of B cell secretion (HOMA-B) and insulin sensitivity (HOMA-S) from the serum glucose and serum insulin concentrations under basal conditions by using HOMA CIGMA software.[6] Data were expressed as mean ±SD and/or median (range) where appropriate. Comparison between two groups was done using Students't' test (paired and unpaired), Mann-Whitney 'U' test and Wilcoxon 'Z' test. Bivariatte correlation analysis was done by using Spearman's Correlation analysis. Data were managed and statistical analyses were performed using Statistical Package for Social Science (SPSS) for Windows version 18. p value <0.05 was taken as level of significance.

Results

Table-1 : Clinical and anthropometric measurements of the study subjects

Variables	Control (n=47)	IGT (n=51)	t/p value
Age (yrs)	40+6	41+5	0.673/0.502
BMI (kg/m2)	24.0+2.9	24.0+3.5	0.570/0.955
WHR	0.88+0.05	0 .92+0.08	2.340/0.02
S Creatinine (mg/dl)	1.03±0.12	1.06±0.24	0.43/0.597
SGPT (U/L)	29.5±21.5	27.4±15.3	0.568/0.571

Table-2 : Glycemic and Insulinemic status of the subjects

Variables	Control (n=47)	IGT (n=51)	z/p value
FSG (mmol/l)	5.2 (4.1-6.0)	5.3 (4.4-6.0)	0.502 /0.615
PSG (mmol/ l)	6.3 (4.8-7.8)	9.3 (7.9-11.0)	**8.502/0.001**
FCPEP (pmol/l)	0.64 (0.18-1.62)	0.68 (0.21-1.39)	1.000/0.310
HOMA%B	117 (58.4-461)	117 (39.4-455)	0.109/0.914
HOMA%S	71 (27-247)	65 (22-222)	**1.890/0.050**

Table-3 : Lipidemic status of the study subjects

Variables	Control (n=47)	IGT (n=51)	t/p value
TG (mg/dl)	159±101	186±116	0.229/0.819
T Chol (mg/dl)	190±36	190±50	0.004/0.997
HDL-C (mg/dl)	36.3±6.6	38.2±5.7	1.56/0.121
LDL-C (mg/dl)	122±31	114±44	0.951/0.344

Table-4 : Absolute levels of glucose dependent insulinotropic polypeptide (GIP) of the study subjects

Variables	Control (n=47)	IGT (n=51)	z/p value
F GIP (ng/ml)	49.62 (6.10-278.0)	74.21 (10.0-189.9)	3.30/0.001
P GIP (ng/ml)	267 (37.76-700.36)	254.95 (130.9-616.3)	0.175/0.861
PGIP:FGIP	5.14 (0.96-19.85)	3.47 (0.98-22.0)	4.469/0.001
FGIP (0 min)	49.6 (6.10-278)	74.21 (10.0-190)	
PGIP (120 min)	267 (37.76-700)	254.95 (131-616)	
z/p value	**5.958/0.001**	**6.205/0.001**	

Discussion

Type 2 Diabetes Mellitus is a major health problem all over the world and its rise in epidemic proportion warrants urgent measures for its prevention. This, in turn, presupposes evidence based knowledge on the predisposing factors of the disorder in individual population. IGT is thought to be an intermediate stage in the natural history of diabetes and intervention at this stage has been proven to be a highly cost-effective measure in combating diabetes. Data on the pathophysiology of IGT in Bangladeshi population is relatively scarce and the present study is the first one exploring its association with GIP. Analysis of the anthropometric data in the present study shows that the IGT subjects do not have generalized obesity as evident by no difference in BMI between control and IGT groups. IGT, however, is associated with central obesity as evident by the higher WHR in these subjects compared to control. The finding is consistent with the observations in a previous study conducted on the same population.[7,8] Central obesity is known to be more specifically related to the increased secretion of adipocytokines (like resistin and adiponectin) and inflammatory markers (like hs CRP) which, in turn, are associated with insulin resistance. Thus, consistent finding of central obesity in the IGT population can be a central issue in designing preventing campaigns for reducing abdominal fat through lifestyle and dietary modifications.

Although prediabetic stages (IFG, IGT and IFG-IGT) are getting increased importance in the prevention of T2DM, their pathophysiology is still not fully understood. For example, it is still not fully decided whether isolated IFG, isolated IGT and combined IFG-IGT should be treated as etiologically separated disorders[9] Data from the Bangladeshi population indicate that IFG has primarily insulin secretory defect, IGT has primarily insulin resistance, and combined IFG-IGT has both the defects[7,8] There are similar heterogenecity in the Bangladeshi diabetic population with different kinds of abnormalities being predominant in different patient groups[10-12]. There is no insulin secretory abnormality in the present IGT group, but there is significant insulin resistance in IGT (p=0.05) compared to Control (Table 2). Insulin resistance in IGT has been reported by many authors in other populations[13].

The major objective of the present work was to explore the association of GIP with dysglycemia or abnormal insulinemic status. GIP was found to be raised in T2DM as well as in IGT[14] but some authors reported reduced GIP in IGT cases[15,16] In the present study the fasting level of GIP in IGT cases were significantly higher than Control and both the groups showed acute rise in the GIP values in response to oral glucose. However, the difference between the two groups were totally lost at the postprandial stage because there was a proportionately higher rise (about 5 times) in the Control compared to a blunted response (about 4 times) in the IGT group. A ratio analysis also reveals that relative GIP values are already higher at the fasting stage compared to the Control; however, the scenario becomes reverse at the postprandial state with the corresponding ratio being significantly less in IGT compared to Control. The significance of the higher GIP values in the fasting state is currently unknown as focus have been mostly given to the GIP values after nutrient intake. This issue, however, now needs to be investigated further.

GIP is known to have a considerable insulinotropic effect and, along with GLP -1, it is thought to be a major contributor of the 50-60% additional rise of insulin secretion through incretin effect in response to oral diet. The effect of oral glucose on GIP release in the Control group could be observed by about 5 fold rise of GIP in postprandial state, but it was about 4 times in case of IGT subjects. It would have been interesting to see how it affected serum C-peptide levels, but the postprandial C-peptide value was not available for analysis. The fasting C-peptide level however was not different between the Control and IGT groups although the GIP was higher in IGT cases. Consequently, the FC-peptide to GIP ratio is significantly lower in IGT compared to Control ($p< 0.05$). It is thus evident that the insulinotropic effect of GIP seems to be already diminished at the fasting stage. The data is in conformity with the observations of decreased activity of GIP in both prediabetic and diabetic subjects.[13,14] In support of the finding in univariate analysis, multiple regression showed a significant association of glucose and C-peptide with GIP. The present data also showed a significant negative association of insulin sensitivity (HOMA%S) with GIP. The mechanism of insulin resistance from deficient GIP action is still uncertain & this needs to be studied further.

Conclusion

IGT subjects have insulin resistance but their pancreatic β cell function seems to be still uncompromised. GIP secretion in IGT subjects is probably upregulated at the fasting state and it has a blunted response to oral glucose in this disorder. The incretin effect of GIP is diminished in

impaired glucose tolerance. GIP does not have any association with insulin secretion in IGT subjects, but it has an association with insulin resistance.

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Original article

Association between index finger length (2D) with height, weight and BMI in adult female population of Bangladesh

Karim Rezwan Hasan[1], Shamim Ara[2], Rubaba Tajreen[3]

Abstract:

Objective : Digital lengths of human hand vary from person to person according to age, sex, races, occupation or even environmental influences. It has been found that the digital lengths and their ratios are not the same in different sexes or even both hands. Specially, index to ring digit lengths and their ratios which already have been proved to represent sexual dimorphism may differ in both hands of an individual as well as shown positive correlations with other morphological traits like height, weight and BMI. In this study, this variation of the index finger (2D) length has been analyzed and compared with height, weight and BMI of adult male Bangladeshis.

Materials & Methods : A Cross sectional analytical study was conducted in the department of Anatomy, Dhaka Medical College, Dhaka, from July 2012 to June 2013. The study was performed on 100 female MBBS students (20-25 years of age) of Dhaka Medical College, Dhaka. With the help of digital vernier caliper measurements of index finger length (2D) was recorded. Height and weight was measured by the stadiometer and weighing scale respectively. BMI was calculated from height and weight. Pearson's correlation analysis was done to find out the correlations between index finger length with height, weight and BMI.

Results : Significant correlation has been found between the lengths of index finger (2D) with height and weight ($P<0.01$) but there was no significant correlation with index finger length with BMI ($P>0.05$ ns)

Conclusion : Study over the variations of digital lengths and their correlations with other body morphological traits have great medicolegal importance to determine age, sex and race of an individual.

Keywords : Right finger length (R2D), Left index finger length (L2D), BMI (Body Mass Index)

Introduction

On the basis of Anatomy, any measurements of body parts can change with the alterations in size of the organs involved or general body size and this concept was defined concisely by Levinton[1].Throughout the following decades, one such study has been a marked increase in interest, that is measurements of digital length and its sexual variations. The index finger located between thumb and middle finger is the second digit (2D) which is usually the most dexterous and sensitive fingers of a human hand[2]. Researchers claimed that the relativelengths of digits are set before birth[3] and interestingly in human hands, the relative lengths of the index finger differs between male and female[4]. In the study of Manning[4], it is seen that smaller index fingers in women have been associated with higher levels of physical aggression throughout their life[5]. Women with less smaller index finger are reported as being more masculine and dominant in nature and tend to perform better in a number of physical activities[6]. In human, number of physical and behavioral traits depends on index finger length (2D) in both sexes which were statistically proven. For example, females with longer index finger are more fertile and have high life time reproductive success. Also, they are more aggressive and assertive in nature and have high musical and sports aptitudes[7]. Again, female with excessive smaller index finger often has some attributes like left-handedness, good visuo-spatial ability[8], fast running speed[9] but they may also experience some severe health related problems like autism, Asperger's syndrome, Hepatitis-B related hepatocellular carcinoma, urolithiasis and rheumatoid arthritis but female having longer index finger often has

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higher risk of early heart disease[8]. In general, the average height of male is more than female[10, 11]. During puberty, male deposit adipose and muscle tissue around the upper body whilst females deposit adipose tissue around the thighs and buttocks produces a typical male body shape (android) and female body (gynoid) respectively. So, the main reason for the stability of sexual dimorphism of height, weight, body mass index (BMI), is the sex-hormone profile of an individual. The length of index (2D) is determined by intrauterine sex hormones at prenatal event of life. Also, other physical traits like height, weight and BMI which are largely determined at puberty are mainly influenced by adult sex hormone profile. So, the index (2D) digit lengths could have some relationships with height, weight and BMI among adult.

Materials & Methods:

The study was performed on one hundred (100) female medical students of Dhaka Medical College, Dhaka age ranging from 20-25 years. With the help of a digital vernier caliper the index (2D) lengths were recorded in millimeters.

Table-1 : Measurements of different variables

Variables	Female (n=100)	
	Range	mean ± SD
R2D (cm)	5.952 - 7.813	6.710 ± 0.369
L2D (cm)	5.765 - 7.912	6.720 ± 0.362
Height (cm)	147.30-167.00	155.45 ± 4.40
Weight (kg)	41.00 - 75.00	54.59 ± 7.80
BMI (kg/m^2)	17.08 - 30.73	22.48 ± 2.89

Length was measured by measuring the crease-tip (c-t) length where "c" is the midpoint of proximal crease at the base and "t" is extreme end (tip) of the finger that is furthest from the crease. The distance between these two points indicates the length of index (2D) finger9. Measurements were taken three times independently and the maximum length was taken for analysis. Height of the subject was taken by stadiometer. According to the standard procedure the subject was stand bare footed. The subject was standing in erect posture so that weight would be evenly distributed between both feet on a stadiometer. The position of the head was in the Frankfurt plane (the upper border of the external acoustic meatus and the infraorbital margin lies on the same horizontal line). The subject was looking straight ahead, shoulder was relaxed, and arms were at sides. Measurement was taken bare footed and weight was measured by the weighing scale in Kilogram (kg) while the subject stand on the scale facing forward with both feet placed on the scale and weight evenly distributed between the feet[12]. Body mass index was calculated by dividing the body weight in kg by the square of the height in meters. (Fig-1: a, b, c, d, e)

Results : Results are shown in Tables and Figures.

Table-2 : Correlations of hand variables with height weight and BMI in female

Variables	Correlation coefficient (r)	Significance of correlation (P –value)
Height with R2D	+0.540	P < 0.01**
Height with L2D	+0.513	P < 0.01**
Weight with R2D	+0.366	P < 0.01**
Weight with L2D	+0.314	P < 0.01**
BMI with R2D	+0.148	P > 0.05 ns
BMI with L2D	+0.096	P > 0.05 ns

Pearson's correlation-coefficient (r) test ns = not significant, ** = significant at P<0.01

Discussion:

In present study, height and weight positively correlated with the length of right index (R2D) finger and left index (L2D) finger that significant (P< 0.01) but BMI didn't show any significant correlation with neither of the index finger lengths (P > 0.05 ns). Similar kind of study has been done by Fink B., Neave N. and Manning J.T. (2006, pp.711-14) in the University of Vienna (Austria) and in the Northumbria University (United Kingdom)[13]; Dongen S.V (2009, Vol.3, pp.01-06) in the Antwerp University, Belgium[14]; B. Danborno, S.S. Adebisi, A.B. Adelaiye and S.A. Ojo (2009, Vol.3, No.3) in Ahmadu Bello University, Zaria, Nigeria[15] and Ibegbu A.O. et al. (2012, pp.79-84) in Ebira ethnic extraction of local Govt. area in Nigeria[16]. All of these studies revealed similar kind of result where finger length was strongly correlated with height but not with body weight or BMI.

Fig-1 : (a) measurement of height, (b) Measurement of weight (c) Identification of crease and tip of index figure. (d) Measurement of the breath of the crease of the index finger to mark the midpoint of the crease and (e) measurement of length (c-t) of the index figure (2D)

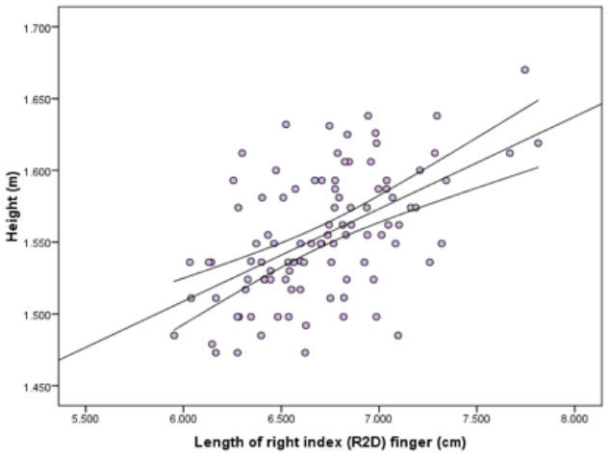

Fig-2 : Correlation of right index finger (R2D) with height in female

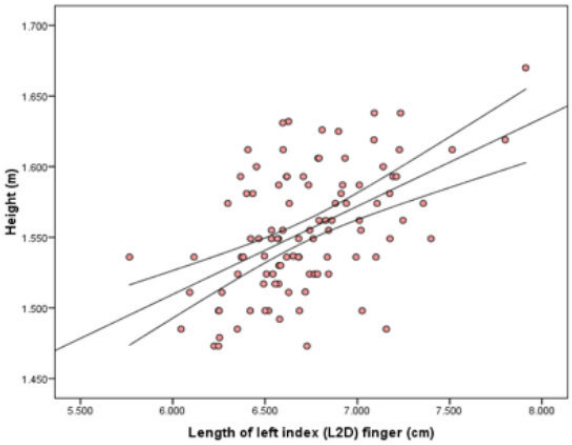

Fig-3 : Correlation of left index finger (L2D) with height in female

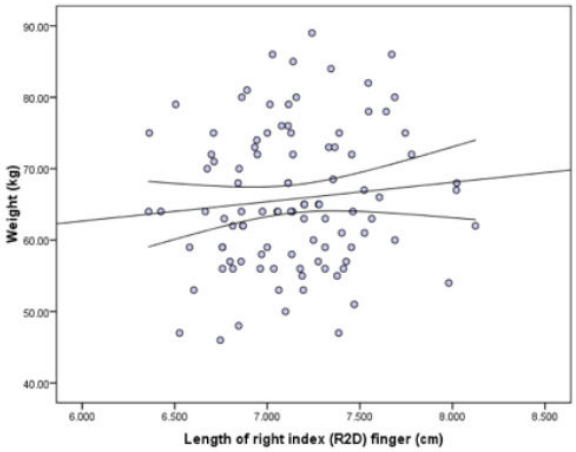

Fig-4 : Correlation of right index finger (R2D) with weight in female

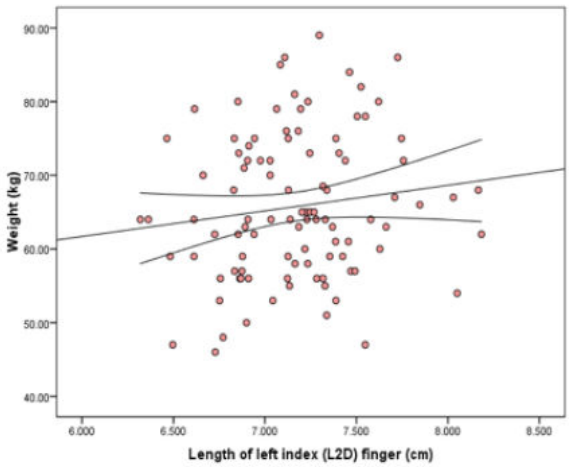

Fig-5 : Correlation of left index finger (L2D) with weight in female

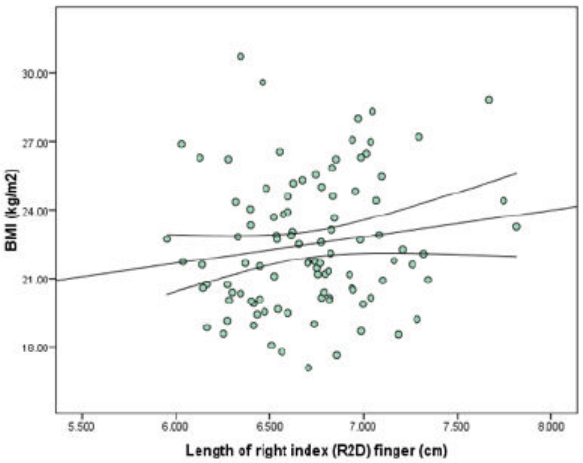

Fig-6 : Correlation of right index finger (R2D) with BMI in female

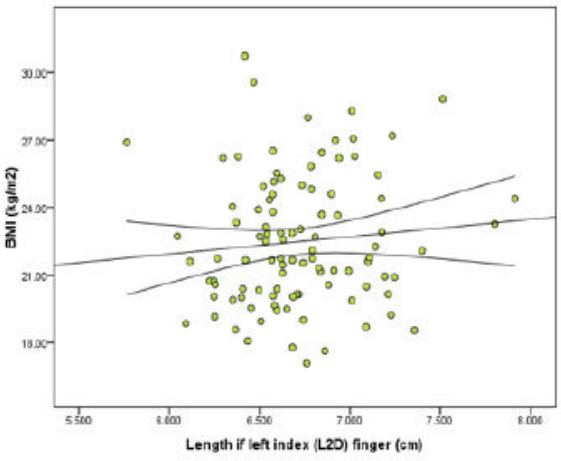

Fig-7 : Correlation of leftt index finger (L2D) with BMI in female

Conclusion:

Study over the variations of digital lengths have great medicolegal importance to determine age, sex and race of an individual. Doing studies on digital lengths and their correlations with other physiological traits can reveal so many mysterious characters of human hand and body morphometry that indicates general sexual characters and hormonal status of adult population of Bangladesh.

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Original article

Evaluation of serum copper, magnesium and glycated haemoglobin in type 2 diabetes mellitus

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Abstract

Objectives: Type 2 diabetes mellitus (DM) is associated with the alteration of trace elements like copper, zinc, magnesium and manganese which may be a contributing factor in the progression of DM and its complications1. Objective of the present study was to estimate serum copper, magnesium, zinc, manganese and glycated haemoglobin in patients with type 2 DM and compare it with controls (non diabetic healthy subjects).

Materials and methods : This cross sectional analytic study was conducted in 200 subjects, out of which 100 were type 2 diabetes mellitus patients as cases (52 males, 48 females) and 100 were non diabetic healthy subjects as control (40 males, 60 females). Serum copper, magnesium, zinc, manganese, glycated haemoglobin were measured by using the auto analyzer Beckman Coulter DXC 600. Serum copper, magnesium, zinc, manganese were measured by modified spectrophotometric micromethod using Guanidine hydrochloride and Bathocuprine disulphonate disodium salt (BCDS).

Results and observations : The mean age of type-2 diabetic patients were (48±10.44) years versus (42±9.37) years of non-diabetic control subjects. The type-2 diabetic patients were generally heavier (BMI 25.04±3.29) than the control subjects(BMI 25±2.71) where p<.05. The BMI, waist circumference, hip circumference and fasting blood glucose were significantly higher in the type-2 diabetic population when compared to the non-diabetic control population of the study. Type-2 diabetic patients had higher levels of fasting blood glucose, glycated hemoglobin and lipoproteins than non-diabetic control patients. The (Mean±SD) of plasma glucose in type-2 diabetic patients and controls were (7.65± .25) m.mol/l and (4.99±.95)m.mol/l respectively, (p<.001) and the mean HBA1C of type-2DM patients and controls were (8.41±1.04) mg/dl and (5.87± .98) mg/l respectively (p<.001). The (Mean±SD) of Zn, Cu and Mg for type-2 diabetic patients and controls were (.941±.246) mg/l, (.771±.483) mg /l, (14±3.613) mg/l and (1.21±.105) mg/l , (1.142 ± .239)mg/l, (18 ± 1.72)mg/l respectively where p<.001 and For Mn was (.091±.049) mg/l and (0.106±0.030) mg/l respectively (p<o.o1). The TAG, Cholesterol, LDL-C and HDL-C for type-2 diabetic patients and controls were (226±124.16)mg/l,(192±42.11)mg/l ,(113±34.52)mg/l, (37±5.49)mg/l and (138±89.23)mg/l, (165±33.06)mg/l, (95±30.05), (41±9.003)mg/l respectively and p<.001. A significant correlations were found between serum Zn and TAG of type-2 DM(r = 0.209) and HDL-C of type 2 DM(r=.199) where p<.05 and non significant relationships were found in between Zn and lipid profile (TAG, Cholesterol, LDL-C, and HDL-C) of control group. A significant correlation found between serum magnesium and TAG of control where p<.01 and non significant correlations were found in serum Mg and Total cholesterol, HDL-C, LDL-C of both type-2 diabetic and control. There were significant correlations in between serum Cu and Mn and TAG of control where p<.05 and non significant correlations were found in between Cu and Mn and other component of lipid profile of both type -2 DM and control.

Conclusion : Patients with type 2 DM had altered metabolism of copper, zinc, magnesium , manganese and this may be related to the increased level of glycated haemoglobin. Impaired metabolism of these trace elements may have a contributory role in the progression of DM and its complications.

Keywords : Copper, Hypomagnesemia, Glycated haemoglobin, type 2 diabetes mellitus, Oxidative Stress

Introduction

Type-2 DM is an endocrinological disease associated with hyperglycaemia characterised by both insulin resistance and defective insulin secretion[1]. A relationship between DM and minerals is frequently reported. Alteration in the metabolism of trace elements like copper, magnesium is associated with DM[2]. Trace elements are accepted as essential for optimum health, because of their diverse metabolic characteristic and functions[3]. Trace elements participate in production of reactive oxygen species (ROS), which contribute to oxidative stress. Oxidative stress contributes to the pathogenesis of many diseases including DM. Previous studies have shown that copper causes oxidative stress[1-5]. Copper acts as a pro-oxidant and may participate in metal catalyzed formation of free radicals[2]. The increased production of free radicals is likely to be associated with development of type 2 DM. Magnesium is an essential element involved in glucose homeostasis & a cofactor for various enzymes in carbohydrate metabolism. It also involves at multiple levels in insulin secretion, binding and activity. Reduced level of magnesium has been documented in type-2 impact on glucose homeostasis and insulin sensitivity in type 2 DM patients[6]. Hypomagnesaemia may also have some effect in the development of diabetic complications with other risk factors[7].

Zinc is an essential trace metal that is directly involved in the synthesis, storage, secretion, and conformational

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integrity of insulin monomers and that Zn assembles to a dimeric form for storage and secretion as crystalline insulin8,9. Lower levels of Zn may affect the ability of pancreatic islet cells responsible for the production and secretion of insulin, in type-2 diabetes[10]. Epidemiological studies have reported decreased plasma and intracellular Zn concentrations in conjunction with increased urinary Zn excretion in diabetic patients. In subjects with type 2 DM with low Zn intake, the risk of coronary heart disease increases by a factor of two to four times and is a major cause of mortality among diabetic patients[11,12].

Material and methods

The study was approved by the Ethical Committee; a written informed consent was obtained from all participants in this study. A total of 100 patients (aged 30-70 years) with type-2 DM recruited from BIRDEM's Medicine and Endocrinology departments. The diagnosis of type-2 DM was confirmed by biochemical investigations as per WHO criteria Patients were excluded when diagnosed with type 1 DM, acute complications such as severe infection, major operations, trauma, GI disorders, severe cardiovascular/ respiratory diseases, pregnant and breast feeding women. Patients taking supplements such as antioxidants, vitamins, minerals were also excluded. Age and sex matched 100 controls were recruited after clinical and biochemical evaluation. The baseline demographic data and family history were obtained. Three ml of venous blood sample was collected for estimation of blood glucose, HbA1c, magnesium, manganese, zinc and copper. Serum magnesium was measured by a timed end point calmagite method[8] and serum copper, manganese; zinc was measured by modified spectrophotometric micro-method using guanidine hydrochloride and HbA1c concentration was measured using HPLC reference method[10]. All the above mentioned parameters were measured using the autoanalyzer Beckman Coulter DXC 600. Statistical analysis of data was performed using the SPSS (Version 15.0). For the comparison of values between the groups, students't' test was used, represented by 'p' value. Statistical significance was considered at a 'p' value of < 0.05. For the correlation, Pearson's correlation coefficient was used.

Results

One hundred (100) patient with type-2 DM as cases (52 males, 48 females) and 100 healthy subjects as control (40 males, 60 females) comprised the study group. The mean age of type-2 diabetic patients was (48±10.44) years versus (42±9.37) years of non-diabetic control subjects. The type-2 diabetic patients were generally heavier (BMI 25.04±3.29) than the control subjects (BMI 25±2.71) where p<.05(Table 1). Table-1 also shows The BMI, waist circumference, hip circumference and fasting blood glucose were significantly higher in the type-2 diabetic population when compared to the non-diabetic control population of the study. It has been established that type-2 diabetic patients had higher levels of fasting blood glucose, glycated hemoglobin and lipoproteins than non-diabetic control patients (table-2). Table-2 showed The (Mean ± SD) of plasma glucose in type-2 diabetic patients and controls were (7.65± .25) m.mol/l and (4.99±.95)m.mol/l respectively, (p<.001) and the mean HBA1C of type-2DM patients and controls were (8.41±1.04) mg/dl and (5.87±.98) mg/l respectively (p<.001). The (Mean±SD) of Zn, Cu and Mg for type-2 diabetic patients and controls were (.941±.246) mg/l, (.771± .483) mg/l,(14±3.613)mg/l and (1.21±.105) mg/l, (1.142 ± .239)mg/l, (18 ± 1.72) mg/l respectively p<.001 and For Mn were (.091±.049) mg/l and (0.106±0.030) mg/l respectively (p<o.o1). The TAG, Cholesterol, LDL-C and HDL-C for type-2 diabetic patients and controls (226±124.16) mg/l, (192±42.11) mg/l, (113±34.52)mg/l, (37 ±5.49) mg/l and (138±89.23) mg/l, (165±33.06) mg/l, (95± 30.05), (41±9.003) mg/l respectively and p<.001. A significant correlations were found between serum Zn and TAG of type-2 DM (r = 0.209) and HDL-C of type 2 DM(r=.199) where p<.05 and non significant relationships were found in between zn and lipid profile (TAG, Cholesterol, HDL-C, and HDL-C) of control group (table-3,4). There was a significant correlation was found between serum magnesium and TAG of control where p<.01 and non significant correlations were found in serum Mg and Total cholesterol, HDL-C, LDL-C of both type-2 diabetic and control (table-3). Significant correlations found in between serum Cu and Mn and TAG of control where p<.05 and non significant correlations were found in between Cu and Mn and other component of lipid profile of both type -2 DM and control (table 3, 4).

Table1 : Descriptive physical characteristics of diabetic patients and controls

Characteristics	Type-2 DM (case)	Non-Diabetic Controls	P Value
Age in years	48±10.44	42±9.37	<.001
Height in (C.M)	158±11.77	163±7.7	<.001
Weight(in K.g)	62±9.004	65±7.7	<.01
B.M.I(Ht/m2)	25.04±3.29	25±2.71	<.05
Hip (C.M)	101±7.94	96±8.26	<.001
Waist (C.M)	94±8.52	86±8.22	<.001
W:H	.924±.033	.881±.04	<.001

Table 2 : Descriptive chemical characteristics of diabetic patients and controls

Biochemical characteristics	Type-2 DM Mean±SE	Non-Diabetic controls mean±SE	P value
TAG	226±124.16	138±89.23	<.001
Cholesterol	192±42.11	165±33.06	<.001
LDL-C	113±34.52	95±30.05	<.001
HDL-C	37±5.49	41±9.003	<.001
HBA1C	8.41±1.62	5±.330	<.001
Zn	.941±.246	1.21±.105	<.001
Cu	.771±.483	1.142±.239	<.001
Mg	14±3.613	18±1.72	<.001
Mn	.091±.049	.106±.030	<.01

Table 3 : correlation of serum Mg, Zn, Cu, Mn concentration with lipid profile parameters in type-2 DM

Lipid profile	Mg of type-2DM (r value)	Zn in Typ e-2 DM (r value)	Cu of type-2DM (r value)	Mn of type-2DM (r value)
TAG P value	r =-.023 ns	r = .209 <.05	r=.049 ns	r=.090 ns
Cholesterol P value	r =-.130 ns	r = .148 ns	r=.086 ns	r=.167 ns
HDL-C P value	r =-.151 ns	r = .199 <.05	r= .051 ns	r=-.148 ns
LDL-C P value	r =-.076^{ns} ns	r =.004 ns	r=.129 ns	r=-.082 ns

Table 4 : correlation of serum Mg, Zn, Mn, Cu concentration with lipid profile parameters in controls

Lipid profile	Serum Mn	Serum Cu	Serum Mg	Serum Zn (r value)
TAG P value	r=.191 p<.05	r=.232 p<.05	r =.260 p<.01	r =.026 ns
Cholesterol P value	r=.228 ns	r=.011 ns	r =.182 ns	r = .087 ns
HDL-C P value	r =.120 ns	r =.060 ns	r =-.180 ns	r =.038 ns
LDL-C P value	r=.045 ns	r=-.129 ns	r =.117 ns	r =.132 ns

Table 5 : The effect of level of HBA1C in trace elements level at diabetic and Non diabetic patients

Parameters	HBA1C		T test
	Diabetic	Non Diabetic	
Zn	5.819±.052	.045±.003	Ns
Cu	2.035±.149	2.492±.138	Ns
Mg	.297±2.004	.405±.892	Ns
Mn	.287±.004	.512±.017	Ns

Discussion

Type 2 DM is a major global health problem that affects 200 million individual worldwide[7]. It is characterized by insulin resistance in peripheral tissues and an insulin secretory defect of beta cells of the pancreas[11]. The relationship of DM with minerals has been reported1-5. Among these minerals copper and magnesium are of particular interest. In the present study we obtained a significant increase in serum copper level in patients having type 2 DM as compared to controls. Zargar HA et al showed that copper levels were significantly elevated in NIDDM patients than in non diabetic subjects4. In a study done by Schlienger et al, elevated levels of copper were found in patients with IDDM and NIDDM[12]. Sarkar A et al, also found out a significant increase in serum level of copper in type 2 DM as compared to controls1. It is well known that copper plays a vital role in oxidative stress[1, 2]. Copper in its free form is a potent cytotoxic element because of its redox chemistry. It readily participates in Fenton and Heiber Weiss reactions to generate reactive oxygen species[13,14]. A high level of copper enhances the toxic effect of metal dependent free radicals. Moreover the increase in copper levels in patients with type-2 DM might also be attributed to hyperglycaemia, which stimulates glycation and causes release of copper ions from copper binding sites of proteins. The release of copper ions into blood further accelerates the oxidative stress 15. The other finding of this study was a significant decrease in serum magnesium level in type 2 DM as compared to controls. Similar such decrease in serum magnesium level in diabetics patients as compared to controls has been reported by some authors[2-4,16]. Magnesium is a cofactor for several enzymes involved in carbohydrate metabolism[17]. Magnesium is important for the effectiveness of insulin. It is involved at multiple levels in insulin secretion, binding and its activity. A reduction of magnesium in the cells strengthens insulin resistance[18,19]. Magnesium deficiency decreases insulin sensitivity via alteration of the insulin receptor associated tyrosine kinase in type 2 DM patients[17]. Hypomagnesaemia can increase the platelet reactivity, increase vascular and adrenal responses to angiotensin II enhanced thromboxane A2 release and lead to organ damage from free radicals[20,21]. Magnesium itself has been reported to possess antioxidant properties by scavenging oxygen radicals probably by affecting the rate of spontaneous dismutation of superoxide anions. Increased free radical formation and reduction in antioxidant potential contributes to the development of oxidative stress in type 2 DM4. The cause of hypomagnesemia may be attributed

to osmotic renal loss from glycosuria, and also decrease in net tubular reabsorption of magnesium[22]. The present study showed a significant ($p<0.001$) rise in HbA1c level in cases as compared to controls, which is similar to the findings of other studies[2,5,22]. The patients with DM who had altered metabolism of copper and magnesium were probably related to increase in HbA1c. Zinc plays a key role in the synthesis, storage and secretion of insulin and it accounts for the conformational integrity of insulin in its hexameric crystalline form. The addition of zinc to the insulin structure will increase the insulin's ability to bind to its receptor. A decrease in zinc affects the ability of the islet cells to produce and secrete insulin, which could compound the problems of Type 2 diabetics in particular. In addition to the findings that zinc levels are often low in diabetics, it is also felt that zinc (in concert with other micronutrients) may participate as an integral component of antioxidant enzymes. Many of the complication of diabetes may relate to an increase in intracellular oxidant and free radicals associated with decrease in intracellular zinc and zinc dependent antioxidant enzyme[23]. Although this study shows a decrease in zinc with the age yet this was not significant and this consistent with results obtained by many studies, which showed that the elderly are at particular risk of zinc deficiency due to their low energy intake and poor dietary zinc consumption[22].

Conclusion

The present findings demonstrate the imbalance in levels of serum copper and serum magnesium among the patients of type 2 DM in comparison to controls. These changes may play an important role in the pathogenesis of type 2 DM by the involvement of these elements in the oxidative stress. Moreover increased levels of copper and decreased level of magnesium are associated with increased values of HbA1c. This suggests that the impaired metabolism of these minerals may have a contributory role in the progression of DM and later development of complications.

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Original article

Perception and awareness regarding Nipah virus infection among rural people in a selected village of Bangladesh

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Abstract

Objectives : Nipah virus disease is a newly discovered disease of swine and humans associated with a new paramyxo virus given the name Nipah virus. The present study makes an attempt to assess the perception and awareness regarding Nipah virus infection among selected villages of Bangladesh.

Methods : This rural based cross sectional study was conducted among 146 villagers of Kurigram Upazilla, in Rangpur from January to June, 2011.

Results : Out of 146 respondents 75(51.4%) were female and 71(48.6%) were males and their mean age were 29.38(±7.401) years with a range of 18-25years. Among them 44 (30.1%) completed primary level of education and 56 (38.4%) were housewives. Majority 143(97.9%) were Muslim. Their mean monthly family income was Tk.4154 (±2181.5). According to the respondent's knowledge, more than fifty percent had no perception that Nipah virus infection is a communicable disease. Only 40 (27.4%) knew the cause of Nipah virus infection and 68(46.6%) had no perception about the spread of the infection and 75(53.4%) had no perception regarding treatment. Regarding knowledge of prevention of Nipah virus infection, 55 (37.7%) taken fruit (including eaten by bat) through washing, 01 (0.7%) take TT vaccination, 08 (5.5%) taken fruit (excluding eaten by bat) through washing, do not take the fruit eaten by bat, 7 (4.8%) don't take rotten food, 3 (2.1%) do not take fruit and majority 72 (49.3%) do not know. The effect of age, sex, occupation, education, monthly income on awareness, showed that education and monthly income of the respondents had significant influence on Nipah virus infection perception and knowledge.

Conclusion : Useful media to educate the rural people regarding Nipah virus infection Government and Non-Government approach is strongly suggested for providing perception and awareness to control the spread of Nipah virus infection among the rural people in Bangladesh.

Key Words : Nipah virus, Perception, Awareness.

Introduction

Nipah virus is a recently identified paramyxovirus that is closely related to Hendra virus. The first recognized outbreaks of Nipah virus illness in humans occurred in Malaysia and Singapore from September 1998 through June 1999; 283 persons, mostly pig farm and abattoir workers were infected through contact with sick pigs. A case-fatality rate of 40% was observed in Malaysia and Singapore; patients presented primarily with CNS symptoms. A second outbreak of Nipah virus infection, with a case-fatality rate of 68%, occurred from January through February 2001 in Siliguri, India, a town close to the northern border of Bangladesh. Patients affected by this outbreak presented with both encephalitis and respiratory symptoms.[1]

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Nipah virus was first recognized in a large human outbreak that affected 283 persons and caused 109 deaths in Malaysia in 1999. The outbreak was preceded by a large Nipah outbreak among pigs. Contact with sick pigs was the primary risk factor for human Nipah virus infection. The porcine outbreak, in turn, was thought to be caused by transmission of Nipah virus from fruit bats to pigs. Antibodies against Nipah virus were identified in the 2 native Pteropus species, and the virus was subsequently isolated from pooled urine samples from a P. hypomelanus colony on Tioman Island, Malaysia. The most likely initiating event was that a fruit bat that was shedding Nipah virus in its saliva dropped a piece of partially eaten fruit into a pig sty, and 1 or more of the pigs became infected.2 Genetic characterization of the Nipah virus strains isolated from pigs in the Malaysia outbreak identified 2 strains, 1 of which was identified in humans, and 1 of which may have given rise to the other through genetic drift. These findings suggest that as few as 1 or 2 instances of spillover of Nipah virus from bats to pigs occurred.[2]

Genetic data demonstrate that the isolates from Malaysia

(NiV-M) and Bangladesh (NiV-B) represent two distinct Nipah virus strains. Nipah virus outbreaks have case fatality rates of up to 100% and there are no approved vaccines or treatments and these viruses have been categorized as a biosafety level 4 (BSL4) agents. Nipah virus differs from other paramyxo viruses in its ability to infect a wide range of mammals including bats, dogs, horses, pigs, and cats. Wild life surveillance at the time of the first outbreaks, along with several subsequent studies, has identified fruit bats of the family Pteropodidae as the natural reservoir of Nipah virus.[3]

Table : 1 Socio-demographic characteristics of the respondents (n=146)

Characteristics Age Group	Frequency	Percent
18-25	50	34.2
25-30	44	30.1
31-40	42	28.8
41-50	10	6.8
Mean ±SD	29.38±7.401 years	
Sex		
Male	71	48.6
Female	75	51.4
Education		
Primary	44	30.1
Secondary	33	22.6
SSC	30	2.50
HSC	15	10.3
Bachelor	14	9.6
Illiterate	10	6.8
Occupation		
Businessman	26	17.8
Service holder	48	32.9
Day laborer	01	0.7
Farmers	07	4.8
Students	08	5.5
housewife	56	38.4
Religion		
Muslim	143	97.9
Hindu	3	2.1
Income		
5000-15000	89	61.0
16000-25000	41	28.1
26000-35000	12	8.2
36000-45000	2	1.4
46000-55000	2	1.4
Mean±SD	1.54±815	
Family Member		
2-4	90	61.6
5-7	50	34.2
8-10	4	2.7
11-15	2	1.4
Mean ±SD	4.52±1.828	

Table-2 : Distribution of the respondents regarding spread of Nipah virus infection (n=146)

Spread of Nipah virus infection	Frequency	Percent
Juice of Date	7	4.8
Date is eaten by Bat	6	4.1
Contaminate by Man	1	.7
Juice of Date, Date is eaten by Bat	44	30.1
Juice of Date, Contaminate by Man	14	9.6
Juice of Date, Date is eaten	3	2.1
by Bat, Contaminate by Man		
Jujube/Kul Boroi	3	2.1
Do not Know	68	46.6
Total	146	100.0

Table-3 : Distribution of the respondents regarding prevention of Nipah virus infection (n=146)

Prevention of Nipah virus infection	Frequency	Percent
Taken fruit (including eaten by bat) by washing	55	37.7
Take TT vaccination	01	0.7
Taken fruit (excluding eaten by bat) by washing	08	5.5
Don't take rotten food	07	4.8
Don't know	72	49.3
Don't take fruit	03	2.1
Total	146	100.0

Table 4 : Distributions of the respondents regarding treatment of Nipah virus infection. (n=146)

Treatment of Nipah virus infection	Frequency	Percent
Yes	71	46.6
No	75	53.4

Materials and Methods

This descriptive cross sectional study was conducted in the village Noahgram of Kurigram district under Rangpur division from January to June 2011. 146 respondants, 18 years and above were included in the study. Convenient sampling technique was used to select the sample for data collection. Verbal informed consent was taken from the respondents. Data were collected using a semi structured questionnaire. The analysis was carried out with the help of SPSS version 17.

Results: Out of 146 respondents, 50 (34.2%) were in the age group18-24years. The mean age of the respondents were 29.38 years (SD= ±7.401). About 75(51.4%) respondents were female and most of them were house wives 71(48.6%) were males. Most 143(97.9%) of the respondents were Muslims. Majority 89(61.0%) of the respondents monthly income were

5000-15000Tk., Majority 90(61.6%), of respondents had 2-4 family members.

Discussion

The study reveals that majority of the respondents perception regarding Nipah virus infection is poor. About 80% of the people live in rural community of village. And they are more or less vulnerable to Nipah virus infection because most of them are illiterate. They do not know the source, mode of transmission, sign symptoms and effect of Nipah virus infection.

In this study the respondents were 146, of them 50 (34.2%) were from the age group of 18-25 years and 44 (30.1%) were from the age group of 25-30years. The mean age of the respondents was 29.38 years and SD = (±) 7.401. Majority of the respondent were female (51.4%) and rest of them were male (48.6%). Female respondents were more than male respondents in the study because the males were busy in the field during the time of data collection. As per BBS, 2002[4], majority (80%) of the people of Bangladesh are Muslim. Current study also found almost the same picture.

Among 146 of the respondents 64 (43.8%) were know that Nipah virus is a communicable disease and 82 (56.2%) do not know which correlates in a Malaysian study where a limited respondents known about nipah virus diseases.6 Out of 146 respondents, regarding spread of Nipah virus infection 13(9.6%) were juice of date, date if eaten by bat and contaminate by man, 44(30.1%) by juice of date, date is eaten by bat, 14 (9.6%) juice of date contaminated by man, 3 (2.1%) juice of date, date is eaten by bat, contaminate by man, 3 (2.1%) jujube/ kul boroi and 68 (46.6%) do not know. An epidemiological investigation in Bangladesh conducted by Rahman M and Karim R7 have identified three pathways of transmission of Nipah virus infection from bats to people. The most frequently implicated route is ingestion of fresh date palm sap. In a review of the 142 Nipah case patients identified in Bangladesh from 2001 through 2011, 75 (68%) developed illness after close contact with another Nipah patient. A second route of transmission for Nipah virus infection from bats to people in Bangladesh is via domestic animals. Some people may come into direct contact with Nipah virus infection infected by bat secretions. Several Bangladeshi Nipah outbreaks resulted from person-to person transmission.

Regarding prevention of Nipah virus infection were 55 (37.7%) taken fruit by washing, 01 (0.7%) take TT vacctination,0 8 (5.5 %) taken fruit by washing, don't take the fruit eaten by bat, 7 (4.8%) don't take rotten food, 3 (2.1%) don't take fruit and 72 (49.3%) do not know about the prevention of Nipah virus infection. A study conducted in Bangladesh[7] that the prevention of Nipah virus infection is better considered in two ways – one is by limiting exposure of people to contaminated fresh date palm juice, and the other is by reducing exposure of caregivers to respiratory secretions and saliva from ill patients. For the former, it presents a dilemma - date palm sap collection provides critical income to low income collectors and is a seasonal national delicacy enjoyed by millions every year. So most effort needs to be directed at strategies to prevent the bats from gaining access to the collecting pots, and various methods are being tried, especially the use of bamboo screens or nets. For the latter, the social and cultural actions need to be addressed in a way that are consistent with the low-income setting and which help family members and other caregivers to be aware of the risks and how best to avoid or reduce them while maintaining care giving activities.

Conclusion

The study provided information about the knowledge regarding Nipah virus infection among the rural people of Bangladesh, which will help health policy makers and planners to formulate a plan and a health education program.

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Original article

In vivo Animal Models for evaluation of Antiinflammatory Activity

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Abstract

Objectives : A prospective study was carried out to find out the anti-inflammatory effects of the ethanolic extract of ground seeds of Nigella sativa in inflammed rats. Place & Period of study: Department of Pharmacology and Therapeutics, Dhaka Medical College (DMC), during the period from January to December 2008.

Methodology : The effect was compared among sixty Long Evan Norwegian rats with reference standard aspirin and hydrocortisone. Acute inflammation was induced by Carrageenan injection at the sub-plantar surface of the hind paw of rat. Ethanol extract of ground seed of Nigella sativa, aspirin, hydrocortisone and normal saline (as control) were administered to evaluate anti-inflammatory effects.

Results : The anti-inflammatory effects (acute) measured by 'inhibited oedema formation' was 43.79% by Nigella sativa, 40.52% by aspirin, 47.71% by hydrocortisone. Again chronic inflammation was induced by implantation of a sterile cotton pellet in rat's groin region for 14 days and treated with Nigella sativa extract, aspirin, hydrocortisone and normal saline. The anti-inflammatory effects (chronic) were measured by weighing cotton pellet to evaluate 'inhibited granuloma formation' and were 41.42%, 27.67% and 38.58% respectively. Moreover, Nigella sativa extract was administered in two different doses (250mg/kg and 500 mg/kg body weight) and significant anti-inflammatory effect was observed by the higher dose.

Conclusion : The study was basically pharmacological one and both the modern drugs and herbal products were used to influence the biological system. It was evident that the biological systems have certain limitations, like individual variations, interference in the response with the system, variability in methods and other factors, which might have interfered with primary findings.

Keywords : In vivo, Animal Model, Antiinflammatory Activity

Introduction

The occurrence of inflammatory disorder is seen worldwide with no racial predilection. However the poor and developing countries are lacking proper management of inflammatory diseases. As a result the prevalence of inflammatory conditions are considerably high in developing countries including Bangladesh. The anti-inflammatory drugs which are currently available are a heterogeneous group of compounds, often chemically unrelated, which nevertheless share certain unwanted effects. The most common is a propensity to induce ulceration.

Therefore, the present trend is to evolve more acceptable agents which will be devoid of potential adverse effect. Use of herbal medicine throughout the world is increasing. Plants still remaining the primary source of supply of many important drugs used in modern medicine.

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The Nigella sativa Linn (Family: Ranuculaceae) is a common spice of south East Asia. Nigella sativa (locally called Kalajira) has been in use in Bangladesh, India and many Middle Eastern communities as natural remedy of many acute conditions for two thousand years[1]. Various research works stated that Thymoquinone-an active component of Nigella sativa is a potent inhibitor of PGs, histamine,5HT, leucotrienes and polymorpho nuclear leucocytes. Plants still remain the primary source of supply of many important drugs used in modern medicine. Therefore, studies are still going on in search of more potent, less toxic, cheaper and easily available anti-inflammatory agents. To treat inflammatory conditions medications are used, but medications can have side effects. However herbal medications usually are devoid of such problems. Nigella sativa Linn (Family: Ranuculaceae) is a common spice of south East Asia especially in Bangladesh and locally called Kalajira. The plant enjoys vast folklore uses as traditional medicine. The Nigella sativa that is kalajira is a healing for all diseases except death1. Thymoquinone(TQ),the main active constituent of the volatile oil extracted from Nigella sativa's seeds,has been reported to have an anti-inflammatory effect on

inflammation.TQ showed a significant effect in inhibiting IL-4,IL-5,IL-13 and IL-1,TNF-alpha.The analgesic and anti-inflammatory effects of Nigella sativa seed polyphenols was evaluated by[1].

Considering its medicinal value and availability in Bangladesh, the study was done to evaluate the anti-inflammatory effect of the ground seed of Nigella sativa in rat model and acute and chronic inflammation were compared with both steroidal and non steroidal anti-inflammatory agents. Objective of the study was to induce acute and chronic inflammation by 1% Carrageenan injection and by subcutaneous implantation of cotton pellet to compare the effects of ethanol extract of Nigella sativa with Aspirin and Hydrocortisone[2].

Materials and method

This study was carried out at the Department of Pharmacology and Therapeutics, Dhaka Medical College (DMC), Dhaka, during the period from Jan 2008 to December 2008.

Materials

The following materials were used to see the anti-inflammatory effect of Nigella sativa linn in experimentally induced inflammation in rats.

1. Animals

The experiments were carried out on 60 Long Evan Norwegian rats. They were collected from the ICDDRB, Dhaka. The rats were of either sex, weighing between 150-200g.

Living condition of the animals

The rats were kept in the animal house of the Department of Pharmacology and Therapeutics, DMC. Rats of different batches of different groups were kept in different plastic cages They were allowed to feed on standard laboratory diet and to drink water ad libitum. The animals were maintained at room temperature under condition of natural light and dark schedule.

The animals were kept in proper hygienic condition under the supervision of animal caretaker and they were inspected almost daily throughout the experiment period. The room was well-ventilated. The wood dust and the excreta were removed every alternative day.

2. Drugs and Chemicals:

a) Plant material :

The Plant Nigella sativa is an annual plant with terminal, grayish-blue flowers reaching between 30 cm and 60 cm in height. The toothed seed pod contains the distinctive tiny (1mm to 2mm long) black seeds[3] that are plant parts seed for medicinal purposes.

The ground seed of Nigella sativa was collected from National Herborium and was taxonomically identified by Department of Botany, University of Dhaka,where a voucher specimen has been deposited.

After collection, the seeds were sun-dried and finally ground to coarse powder. The powdered plant material was extracted with distilled ethanol at room temperature for 10 days. The filtrate was concentrated in vacuum (50°C) yielding the crude ethanol extract. The crude extract obtained as such was kept and dried in refrigerator at 4°C for three days. The extract was diluted with normal saline prior to any pharmacological use.

b) Chemical reagents :

i) Acetyl salicylic acid (aspirin):300mg tablet manufactured by Reckitt and colman, Bangladesh Ltd.Equivalent amount of aspirin from the tablet after making powder was taken and a suspension was prepared for administration to the animals. Carrageenin,hydrocortisone injection collected from local market,ethanol(BDH,India),diethyl ether (BDH, India)

ii) Hydrocortisone: Hydrocortisone was available in the vial as powder form and fresh solution was prepared in distilled water.

iii) Carrageenin: Carrageenin was collected from the Department of Pharmacy, University of Dhaka, Bangladesh 1% Carrageenin solution/suspension in normal saline was prepared. It is white to yellowish or brown, coarse or fine, practically odorless powder[8]. Chondrus crispus is the main source of Carrageenin, material with similar composition and physical properties has been isolated from other seaweeds[9].

After collection of crude ethanol extract was formed and kept in 40C in refrigerator. The extract was oil in nature. Tween-80 (suspending agent) and water was added with oil to make a suspension.This suspension was arbitrarily devided into two doses-low dose (250mg/kg)and high dose(500mg/kg)for dose dependent response. Long Evans Norweigian rats, collected from, BSMMU (3-4 months old, 200-250gm of weight), had free access to food and water ad libitum. Acute inflammation was induced by Carrageenan a chemical agent. The antiinflammatory effect was compared with reference

standard drugs aspirin and hydrocortisone.

The animals were kept in the laboratory environment for seven days and fasted overnight and weighed before the experiment. The animals were randomly divided into different groups consisted of six rats in each group. Group I: Received 0.6ml normal saline administered orally and served as control.

Group-II: Received ethanol extract of Nigella sativa 250mg/kg (0.6ml) body weight administered orally. Group-III: Received ethanol extract of Nigella sativa 500mg/kg(0.6ml) body weight administered orally. Group-IV: Received aspirin 100mg/kg body weight administered orally. Group-V: Received hydrocortisone 2mg/kg body weight administered subcutaneously. After one hour of drug administration, 0.1 ml of 1% Carrageenan in sterile saline solution was injected into the sub-plantar surface of the right hind paw for the production of acute inflammation. Paw volumes were measured by volume displacement method using plethysmometer[2] after 1hour of carrageenan injection.

For chronic inflammation the rats were divided into five groups (n=6), fasted overnight and allowed free access to water. The rats were administered with vehicle, standard drug and test drugs. One hour after the first dosing, the rats were anesthetized with ether3 and 50mg of the sterile cotton pellet was inserted one in each axilla and groin of rats by making small incisions were sutured by sterile catgut. Group-I: received subcutaneous incision. The 0.6ml normal saline administered orally for 14 days and served as control.Group-II: received ethanol extract of Nigella sativa 250mg/kg body weight administered orally for 14 days.Group-III: received ethanol extract of Nigella sativa 500mg/kg body weight administered orally for 14 days.Group-IV: received aspirin 100mg/kg body weight administered orally for 14 days.Group-V: received hydrocortisone 2mg/kg body weight administered subcutaneously for 14 days.The animals were sacrificed by excess anesthesia on the 14th day and cotton pellets were removed surgically. Pellets were separated from extraneous tissue and dried at 600C unit weight become constant. The net dry weight i.e. after subtracting the initial weight of the cotton pellet was determined. The average weight of the pellet of the control group as well as of the test groups was calculated. The percent change of the granuloma weight relatively with vehicle control is determined and statistically evaluated. The percentage inhibition increases in the weight of the cotton pellet is calculated.

Results

1. Acute inflammation :

The mean initial (0 hour) paw volume of group-I, II, III, IV and V were 117.25±1.28, 121.05±3.32, 133.69±2.48, 128.63±5.16, 131.59±4.63 respectively. Simultaneously the mean paw volume after one hour of carrageenan injection pretreated with test drugs were 193.75±2.14, 175.55±2.10, 176.69±1.17, 174.13±1.68, 171.59±1.23 respectively (Table – 1) All units were expressed in cub.mm. The percentage inhibition of oedema formation in group - II, III, IV and V were 28.75%, 43.79%, 40.52%, 47.71% respectively (Table - I) in comparison to control. From the result it was found that a significant anti-inflammatory effect was exhibited by the ethanolic extract of N. sativa at 500mg/kg body weight with 43.79% inhibition.

Table-I : Effects of administration of ethanol extract of Nigella sativa, aspirin and hydrocortisone on Carrageenan-induced paw oedema after 1 hour of Carrageenan injection(mean±se):

Groups	Initial (0hr) Paw volume	Paw volume after 1 hour of Carrageenan injection	Increased paw volume (cu.mm.)	Inhibition of oedema formation
Group I	117.25±1.28	193.75±2.14	76.50±2.11	--
Group II	121.05±3.32	175.55±2.1	54.50±1.24*	28.75%
Group III	133.69±2.48	176.69±1.17	43.00±4.65*	43.79%
Group IV	128.63±5.16	174.13±1.68	45.50±3.82*	40.52%
Group V	131.59±4.63	171.59±1.23	40.00±2.11*	47.71%

* P<0.05 in a test of significance difference from control.

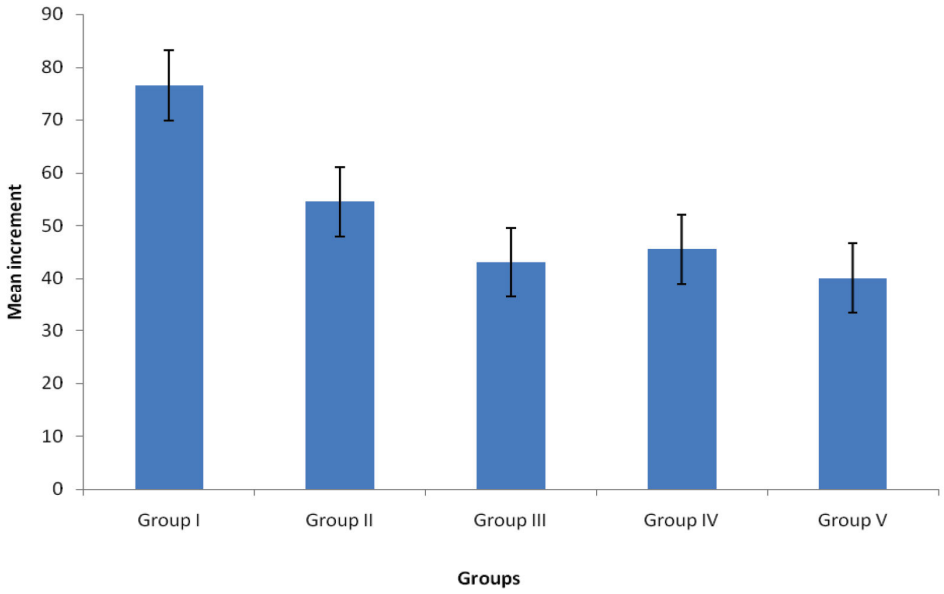

Fig 1: Mean increment of anteroposterior volume of rats paw after 1 hour of Carrageenin injection *** P<0.05 in a test of significance difference from control.

Group-I : Received 0.6ml normal saline administered orally and served as control.

Group-II : Received Ethanol extract of Nigella sativa 250mg/kg body weight administered orally.

Group-III : Received Ethanol extract of Nigella sativa 500mg/kg body weight administered orally.

Group-IV : Received Aspirin 100mg/kg body weight administered orally.

Group-V : Received Hydrocortisone 2mg/kg body weight administered subcutaneously

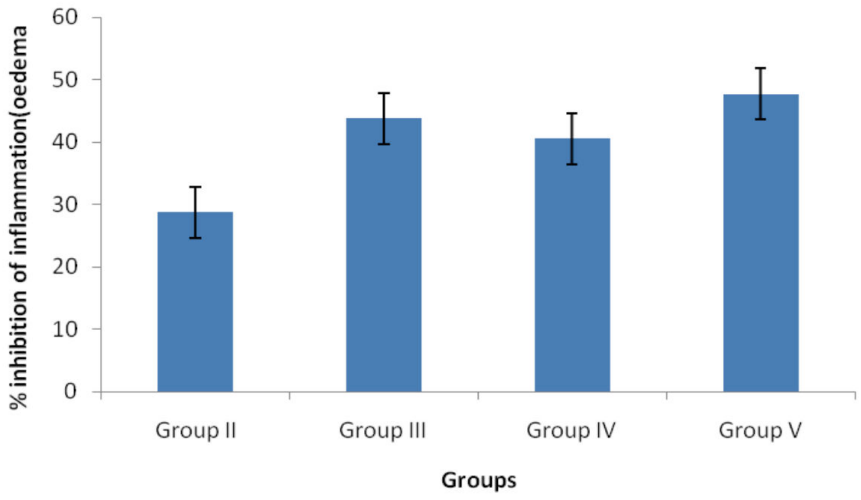

Fig 2 : Percentage of inhibition of Carrageenin induced inflammation by different doses of ethanol extract of Nigella sativa, Aspirin and Hydrocortisone in comparison to control.

Group-II : Received Ethanol extract of Nigella sativa 250mg/kg body weight administered orally.

Group-III : Received Ethanol extract of Nigella sativa 500mg/kg body weight administered orally.

Group-IV : Received Aspirin 100mg/kg body weight administered orally.

Group-V : Received Hydrocortisone 2mg/kg body weight administered subcutaneously

2. Chronic inflammation :

At the end of the chronic anti-inflammatory study after 14 days the pellets were removed from the site of insertion sacrificing the animals[4]. The final weight of the cotton pellets were determined. The weights were 207.83±0.69mg, 177.63±5.31mg, 142.45±5.58mg, 164.16±15.86mg, 146.93±7.12mg for group - I, II, III, IV and V respectively. The increment in the weight of cotton pellet in ethanol extract of N. sativa 250mg/kg body weight, ethanol extract of N. sativa 500mg/kg body weight, aspirin and hydrocortisone treated groups were 127.63±5.31, 92.45±5.58, 114.16±15.86, 96.93±7.12mg respectively (Table - II). Where as, the increment the pellet for the control group was 157.83±8.69 mg. The percentage of inhibition of granuloma formation were 19.13, 41.42, 27.67, 38.58 as compared to the control for ethanol extract of N. sativa 250mg/kg, ethanol extract of N. sativa 500mg/kg, aspirin 100mg/kg and hydrocortisone 2mg/kg body weight respectively (Table - II). In this chronic study an anti-inflammatory effect was observed at 500mg/kg body of the ethanolic extract of N. sativa.

Table - II : Effects of extracts of Nigella sativa, Aspirin and Hydrocortisone on cotton pellet induced granuloma in rat.

Groups	Initial weight of cotton pellet	Final weight of cotton pellet	Increase of weight of cotton pellet (mg)	Inhibition of granuloma formation %
Group I	50±0.22	207.83±0.69	157.83±8.69	--
Group II	50±0.22	177.63±5.31	127.63±5.31*	19.13
Group III	50±0.22	142.45±5.58	92.45±5.58**	41.42
Group IV	50±0.22	164.16±15.86	114.16±15.86**	27.67
Group V	50±0.22	146.93±7.12	96.93±7.12**	38.58

* P<0.05= significant difference from control.

** P<0.001=highly significant difference from control

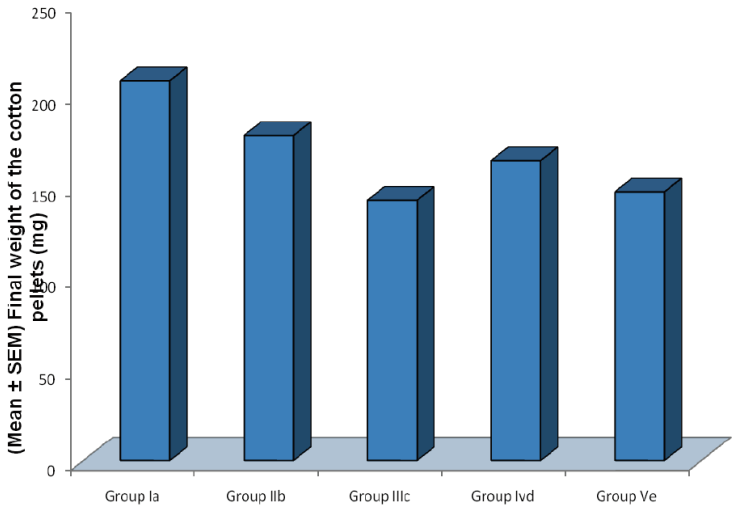

Fig 3 : Comparative final weight of cotton pellet

Group-Ia : Received 0.6ml normal saline administered orally for 14 days and served as control.

Group-IIb : Received Ethanol extract of Nigella sativa 250mg/kg body weight administered orally for 14 days.

Group-IIIc : Received Ethanol extract of Nigella sativa 500mg/kg body weight administered orally for 14 days.

Group-IVd : Received Aspirin 100mg/kg body weight administered orally for 14 days.

Group-Ve : Received Hydrocortisone 2mg/kg body weight administered subcutaneously for 14 days.

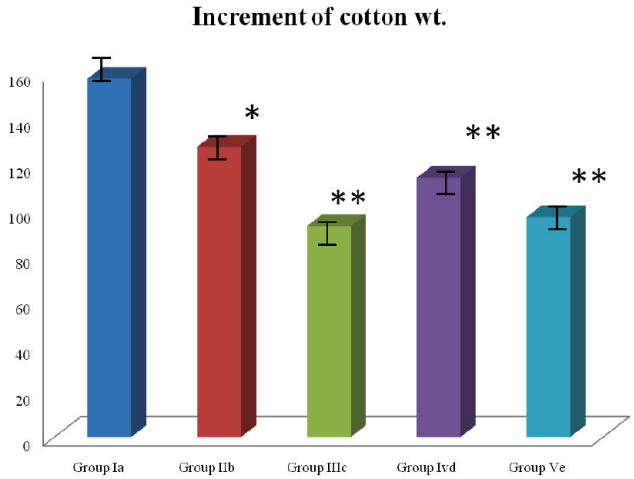

Fig-4 : Comparative increment of weight of cotton pellet
* P<0.05 in a test of significance difference from control
** P<0.001 in a test of significance difference from control

Group-Ia : Received 0.6ml normal saline administered orally for 14 days and served as control.
Group-IIb : Received Ethanol extract of Nigella sativa 250mg/kg body weight administered orally for 14 days.
Group-IIIc : Received Ethanol extract of Nigella sativa 500mg/kg body weight administered orally for 14 days.
Group-IVd : Received Aspirin 100mg/kg body weight administered orally for 14 days.
Group-Ve : Received Hydrocortisone 2mg/kg body weight administered subcutaneously for 14 days.

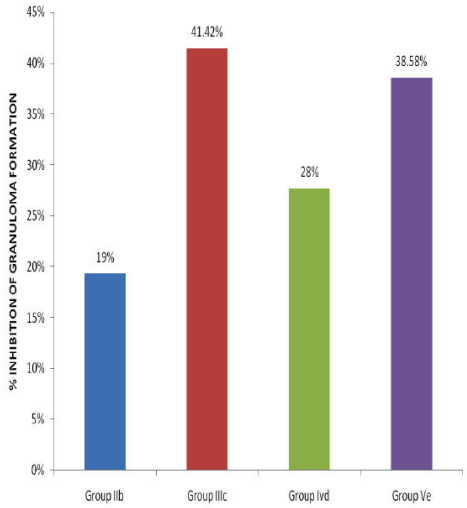

Fig 5 : Percentage of inhibition of granuloma formation by different doses of ethanol extract of Nigella sativa, aspirin and hydrocortisone in comparison to control.

Group-Ia : Received 0.6ml normal saline administered orally for 14 days and served as control.
Group-IIb : Received Ethanol extract of Nigella sativa 250mg/kg body weight administered orally for 14 days.
Group-IIIc : Received Ethanol extract of Nigella sativa 500mg/kg body weight administered orally for 14 days.
Group-IVd : Received Aspirin 100mg/kg body weight administered orally for 14 days.
Group-Ve : Received Hydrocortisone 2mg/kg body weight administered subcutaneously for 14 day.

Statistical Analysis

All the results have been expressed as mean plus/minus standard error of mean (mean ± SEM). Significance of difference between groups was assessed by using ANOVA Test.

Discussion

The above mentioned models have given a broad spectrum for the evaluation of the anti-inflammatory activity. In different models, the inflammation was produced by different inducers by releasing anti-inflammatory mediators. Each having different mechanism of action for producing inflammation either by increasing the vascular permeability, the infiltrations of leukocytes from the blood into the tissue or granuloma formation and tissue repair. To give a scientific validation to the plant N. sativa, an attempt was made to study the anti-inflammatory activity of the ethanolic extract of its seeds[5]. From the result it was observed that the effect of at dose of 500mg/kg body weight was better than that of non-steroidal reference standard aspirin, and little bit less that the steroidal reference standard hydrocortisone.

In this study, Nigella sativa may inhibit release of histamine and serotonin (5-HT) and the formation of TNF-∝ IL-1ß, and IL-6 and enhanced the production of IL-10, thus resulting in an overall attenuation of the pro-inflammatory / anti-inflammatory mediators and cytokine ratio in Carrageenan-injected paws, which may contribute to its anti-inflammatory effect [6] .

Nigella sativa reduces the vascular component of inflammation and impair the release or formation of inflammatory mediators such as PGs, histamine, leucotrines etc. responsible for increasing vascular permeability and inflammation. It may also inhibit the amoeboid activity of the reticuloendotelial cells and polymorphonuclear leucocytes resulting a reduction in the cellular exudates [7].

Treatment with Nigella sativa extract at doses of 250mg/kg body weight orally daily for 14 days produced significant anti-inflammatory effect and at a doses of 500mg/kg body weight orally daily for 14 days produced significant anti-inflammatory effect and the percentage of inhibition of granuloma formation were 19.30% and 41.42% respectively. This was also in a dose dependent manner. Following administration of aspirin and hydrocortisone for 14 days showed also anti-inflammatory effect and the percentage of inhibition of granuloma formation were 27.67% and 38.58% respectively.

In the cotton pellet granuloma model, inflammation and granuloma develops during the period of several days. This model is an indication for the proliferative phase of inflammation. Inflammation involves proliferation of macrophages, neutrophils and fibroblasts, which are basic sources of granuloma formation. Hence, the decrease in the weight of granuloma indicates that the proliferative phase was effectively suppressed by the ethanol extract of Nigella sativa.[8]

In this study, crude ethanol extracts of ground seed of Nigella sativa steroidal and non-steroidal anti-inflammatory drugs daily for 14 days reduced weight of granulation tissue. The reduction was statistically significant in comparison to control group which was observed at the higher does (500mg/kg body weight). But the reductions of weight of granulation tissue in case of steroidal and non-steroidal anti-inflammatory drugs were highly significant in comparison to ethanol extract of ground seed of Nigella sativa.

The study was basically pharmacological one and both the modern drugs and herbal products were used to influence the biological system. It was evident that the biological systems have certain limitations, like individual variations, interference in the response with the system, variability in methods and other factors, which might have interfered with primary findings. However, the results obtained in this experiment may not represent the exact effect. Despite all these limitations, interpretation of the results obtained in this study was made carefully and cautiously.

The study provides an initial step in demonstrating the anti-inflammatory effect of ethanol extract of ground seed of Nigella sativa. The obtained data support the basis for future use of Nigella sativa in traditional system of medicine. Thus, it could be a new agent in reducing morbidity and mortality resulting from inflammatory disease condition. The findings presented here provide a baseline for future studies designed to quantify the effects of ethanol extract of ground seed of Nigella sativa. The experimental results suggest that the possible mechanism of anti-inflammatory activity of polyamines may be due to their impairment of the release or formation of inflammatory mediators such as histamine, 5-HT, PGs, and lysosomal membrane stabilization as supported by present experimental findings. Studies on polyamines may be helpful in developing a new approach for better understanding of the inflammatory process and the generation of new anti-inflammatory drugs.[9]

Further investigations are warranted to reconfirm and identify the anti-inflammatory active principles and elucidate their mechanism of action. Toxicological studies should also be under taken before any clinical use. The experimental results suggest that the possible mechanism of anti-inflammatory activity of polyamines may be due to their impairment of the release or formation of inflammatory mediators such as histamine, 5-HT, PGs, and lysosomal membrane stabilization as supported by present experimental findings. Studies on polyamines may be helpful in developing a new approach for better understanding of the inflammatory process and the generation of new anti-inflammatory drugs.

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Review article

Probiotics: A new hope for Arsenic management

Nafisa Rashid[1], Nasreen Akter Maya[2], Munira Afrin[3], Tarek Golam Mostafa[4], Nadia Farah[5]

Abstract : Nowadays drinking water contamination by arsenic is a major health problem of the world. The chronic low dose (>50µg/L) exposure to arsenic through contaminated tube well water may cause the development of arsenicosis. The present knowledge about the management of arsenicosis is far from satisfactory. Gut bacteria have important and specific metabolic, trophic and protective functions A study conducted by Upreti et al17, indicated that arsenic resistant probiotic Lactobacilli would be useful in the prophylactic interventions of arsenic related gastro-intestinal toxicity. probiotics are `Live microorganisms which when administered in adequate amounts confer a health benefit on the host'. Probiotic bacteria are known to be promoters of the host body's defense mechanism by stabilizing the local microflora, triggering a humoral immune response and constructing a barrier against immunological disorder. Here in this study, it could be assumed that there was multiplication of probiotics bacteria in the digestive tract, therefore an increase of good bacteria in the gut and thus increased stool arsenic excretion

Key word : Probiotics, arsenic contamination, management

Introduction

Arsenic is the 20th most abundant element in the earth's crust and is widely distributed throughout the nature as a result of weathering dissolution, fire, volcanic activity and anthropogenic input.[1] The majority of the human are chronically exposed to low levels of arsenic, principally through ingestion of food and water and to some extent due to inhalation of arsenic contaminated air.[2] Now a days drinking water contamination by arsenic is a major health problem of the world. In our country, the situation is deteriorating day by day as the new cases of arsenic poisoning are still being reported in different parts of the country.[3] Out of 64 districts, 61 districts have arsenic concentration in ground water above the maximal permissible limit of 0.05 mg/L[6]. Official reports (Director General of Health Services, Government of Bangladesh) show that more than 60,000 people are suffering from arsenicosis in Bangladesh[4]. About 35-77 million Bangladeshis have already been chronically exposed to increased concentrations of arsenic through drinking water[8] and food [9,10].

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Effect of arsenic on health

The chronic low dose (>50µg/L) exposure to arsenic through contaminated tube well water may cause the development of arsenicosis.[5] Already more than 40,000 people developed signs and symptoms of chronic arsenic poisoning. Skin manifestations mainly melanosis and leukomelanosis are considered to be the earliest dermatological findings but hyperkeratotic lesions of the skin (keratosis) are the most distinctive skin features of long-term arsenic exposure[7]. A large number of populations in Bangladesh are suffering from melanosis, leuco-melanosis, keratosis, hyperkeratosis, dorsum, non-pitting oedema, gangrene. These manifestations of chronic arsenicosis develop slowly usually 6 months to 2 years[14]. In the study by Fierz (1965), the minimal latency period from exposure to development of arsenical keratoses was 2.5 years, and the average latency period for skin cancer was 14 years. The present knowledge about the management of arsenicosis is far from satisfactory. Though several studies are making progress on this issue but there are no sufficient evidences to confirm or to reject their effectiveness. Some of them cause side effects like GIT upset, dermatomyositis, auto-immune disease and also recurrence of the sign-symptoms after withdrawal of drugs.

Gut flora

In our body, there are 10 trillion cells but bacterial count is approximately 100 trillion. That is ten times than the total body cell. Micro-oganisms start colonization of the gastro intestinal tract soon after birth and this process continues throughout the life. The human gastrointestinal tract can

be described as a complex microbial ecosystem. The intestinal habitate of an individual contains 300-500 different species of bacteria[1,3]. Gut bacteria have important and specific metabolic, trophic and protective functions5. The constant interaction between the host and its microbial guests can infer important health benefits like regulation of colonic pH, production of short chain fatty acid and salvage of energy from non digestible food components[14]. Available literature is scanty regarding the interaction between inorganic arsenic and gut microflora[8].

Role of gut flora on arsenic metabolism

Studies carried out suggest that bacteria present in the gastro-intestinal tract may play a role in arsenic detoxification (Rowland and Davies, 1981). They showed an in vitro metabolism of inorganic arsenic by gastro–intestinal microflora of rat. They suggested that in presence of caecal contents, small amounts of methylarsonic acid and dimethylarsenic acid were formed. The metabolism was inhibited by adding antibiotics, thus indicating that bacteria were involved. Gut bacteria have important and specific metabolic, trophic and protective functions5. The constant interaction between the host and its microbial guests can infer important health benefits[16]. Available literature is scanty regarding the interaction of inorganic arsenic with gut microflora[9].

The gastrointestinal (GI) tract presents itself as the first organ susceptible to attack by ingested xenobiotics consequently; concentrations that must be endured by this tissue are often many times higher than those endured by other tissues[10]. An important study was carried out to compare the effects of arsenic toxicity on intestinal epithelial cells and resident gut flora18. The bacteria selected for this study from stool culture were E. coli, Pseudomonas sp., Lactobacilli sp and Staphylococcus sp. Intestinal epithelial cells were treated with different concentration of arsenic (0-10 ppm). Here, dehydrogenase activity (DHA) and esterase activity (EA) of intestinal bacteria and epithelial cells were measured. The growth profile of gut bacteria and intestinal cell viability revealed an arsenite concentration dependant inhibition following 24 hours in vitro exposure to arsenic. This study suggests that arsenic is toxic to resident gut flora in high concentration (2-10 ppm). There was significant concentration dependent inhibition of DHA and EA (40%-72%) as compared to controls, with arsenite exposure to 5 ppm and 10 ppm, respectively[11]. In 2009, Choudhury et al, 3 carried out an in vivo study on rats to see the influence of arsenic on aerobic gut flora. The study shows a significant inhibition of gut flora after 2 weeks administration of arsenic (1 mg/L) with a decrease in stool arsenic level and increase in liver arsenic level. However, this inhibitory effect of arsenic on gut flora was not observed in presence of vitamin E (1 mg/day) or selenium (0.4 µg/day). Rats that received only arsenic, a decrease in stool and an increase in liver arsenic level was might be due to inhibition of gut bacterial count resulting in decreased bacterial detoxification of arsenic by gut bacteria and its increased deposition in liver[3]. Microbes have been shown to reduce a wide range of toxic metals through detoxification and elimination[11]. Bacteria have been reported to produce MMA (V) or DMA (V)[2] which are non toxic and finally TMAO in gaseous form whose volatilization lowers arsenic concentration that may contribute to global cycling of arsenic[12]. So, more stress is being given on bacterial detoxification of arsenic which is considered to be non-toxic.

Concept of probiotics

Probiotics is a Greek word in which `Pro' means for and `bios' means life. So, probiotics means `for life'. According to WHO (2002), probiotics are `Live microorganisms which when administered in adequate amounts confer a health benefit on the host'. The idea of using microbes to promote good health or to prevent disease is not new. In the early 1900, Russian immunologist Elie Metchnikoff, reported the favorable effects of soured milk in human and suggested that consumption of live microbes (possibly LAB) in such fermented milk may help improve the balance of the gut Microflora. Since then, microbial probiotics have gained an increased interest and their use is now widely accepted. Several microorganisms,under the name of "probiotics" have been proposed and used in a wide range of clinical trials, ranging from diarrheal disease to cancer prevention.

Role of probiotics

The composition of the gastro-intestinal flora varies between the individuals and also within the same individual during life. It contains both pathogenic and non-pathogenic bacteria that exist in a complex symbiosis. Various factors such as diet, climate, aging, medication (particularly antibiotic consumption), illness, stress and life style can upset this balance leading to diarrhea, mucosal inflammation, or other serious illnesses. Maintenance of an optimal gut flora balance requires that 'friendly or non-pathogenic' bacteria, such as the Gram

positive lactobacilli and bifidobacteria (>85%of total bacteria), form a barrier against pathogenic bacteria9. Probiotics are possibly the most natural and safe means of maintaining this balance

Before birth, the digestive tract of the fetus is sterile, but within few hours of birth, the baby acquires a complex collection of microorganisms which populate in the mouth and then eventually the full length of the tract are colonized. The development of specific microorganisms is influenced by the exposure to certain factors such as maternal microbiota, environmental contact, mode of delivery and the infant's diet. Through normal vaginal birth, an infant is exposed to the mother's vaginal and fecal flora, which results in the colonization of Lactobacillus, Bifidobacterium etc[15]. Studies show that less than 7 years of age the symptoms of arsenicosis are rare[3,12]. So, there may be a relation between Lactobacillus and arsenicosis. They also suggested that a bacteria, before being selected as probiotics, should be non-pathogenic, non-toxigenic, should retain viability during storage and use, should have the capacity to survive and metabolise in the gut and finally should have documented health effects. Bifidobacterium and Lactobacilli exert some health promoting properties. A study conducted by Upreti et al17, indicated that arsenic resistant probiotic Lactobacilli would be useful in the prophylactic interventions of arsenic related gastro-intestinal toxicity. Another study conducted by Rashid et al., (2012) showed that the colony count of E. coli in arsenic exposed controls was reduced and in arsenicosis patients it was severely reduced when compared to healthy volunteers. There was significant decrease in stool arsenic level and increase in nail arsenic level of both arsenic exposed controls and arsenicosis patients. After 12 weeks supplementation of probiotics, the E. coli count and stool arsenic level both were significantly increased in arsenic exposed controls and arsenicosis patients in comparison to healthy volunteers. On the other hand, nail arsenic level decreased in the above mentioned two groups in relation to healthy volunteers. Bifidobacterium is one of the most important strictly anaerobic bacterium which acounts for 25% of the total anaerobic counts[14]. So, Lactobacilli may be helpful in arsenicosis patients. Here, in this study, the probiotics containing Lactobacilli bulgaricus and Bifidobacterium along with fructo-oligosaccaride as prebiotics were chosen as they are commercially available, cheap and easily tolerated by patients. There is now strong evidence for their use in treating and preventing some human diseases.

Conclusion:

Manipulation of the human intestinal flora offers potentially to improve health through a variety of mechanisms[14]. Probiotic bacteria are known to be promoters of the host body's defense mechanism by stabilizing the local microflora, triggering a humoral immune response and constructing a barrier against immunological disorder. Here in this study, it could be assumed that there was multiplication of probiotics bacteria in the digestive tract, therefore an increase of good bacteria in the gut and thus increased stool arsenic excretion. Increased bacterial multiplication is associated with increased bacterial activity. In addition increased bacterial contain high amount of glutahione which efficiently reduces toxic substances. Moreover application of lactic acid bacteria in removing toxic metals from water has been studied. They have also been reported to remove mycotoxins and cyanotoxins from food and water respectively. Moreover probiotics are safe, natural, inexpensive and have no known negative long term effects.

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Case Report

Non-Hodgkin Lymphoma presenting as breast lump

Sadia Armin Khan[1], Abu Ahmed Ashraf Ali[2]

Abstract : Primary tumors of the breast arise mainly from the ductal system. However, tumors arising from the connective tissue had also been seen in clinical practice. Primary lymphoma of the breast is a rare diagnosis with an incidence of only 0.5%1. We report a case of primary Non Hodgkin Lymphoma of the breast initially misdiagnosed as case of chronic granulomatous mastitis on FNAC.

Key word : Lymphoma, FNAC.

Introduction

Primary lymphomas of the breast are uncommon with incidence only 0.12 – 0.5 %[1]. But they are potentially curable neoplasms. The pathogenesis of breast lymphomas is still unknown. The clinical stage, histological type of lymphoma, and patient's age seem to be important for the prognosis of primary lymphoma of the breast. Diagnosis in most of the cases is revealed by routine FNAC performed for breast lumps, but sometimes it is inconclusive or false reporting as our case was misdiagnosed as chronic granulomatous mastitis.

Case Report

30 years old lactating female presented to us with the complaint of painless lump in the right breast for one year. She denied associated pain, fever, chills, or skin changes. Both axilla and left breast were normal. Her medical history was unremarkable, and review of symptoms was negative for night sweats, weight loss, or fever. On examination patient was conscious, oriented and afebrile. The physical examination was notable for a large lobulated mass, firm in consistency seen in the upper outer quadrant of right breast extending towards right axilla. Left breast was normal. The rest of the physical examination findings were normal.

Investigations

Ultrasonogram of both breasts and axilla revealed there was a cystic area with regular margin contains low level echoes seen within the outer quadrant of right breast, about (3.6x2.8x2.9). Peripheral vascularity is seen-suggestive of galactocele. FNAC showed Chronic granulomatous mastitis. Surgical treatment Wide local excision was performed. Histopathology examination showed non Hodgkin lymphoma of the breast. Immuno-histochemistry was not performed. Metastatic workup revealed no metastasis. The patient was offered post operative chemotherapy in form of CHOP regimen. Presently the patient is under chemotherapy follow up.

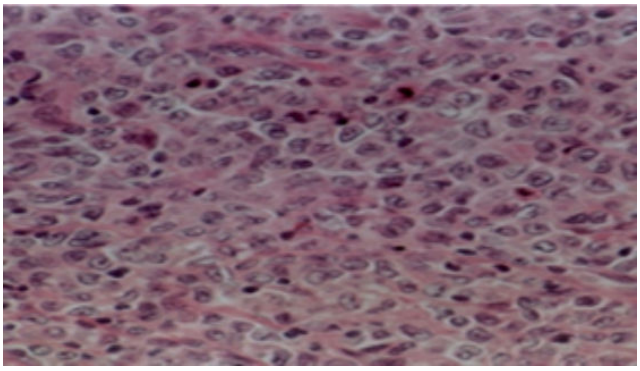

Fig : Microphotograph showing Non-Hodgkin Lymphoma

Discussion

Non Hodgkin Lymphoma involving the breast either as a primary site or as a site of recurrence from lymphoma previously diagnosed elsewhere is rare. Several series have reported varying incidences of primary and secondary cases. Primary NHL of the breast is a rare disease, representing only 0.04%-0.50% of malignant breast neoplasms[2], 1.7% of all extranodal NHL and 0.7% of all NHL[3]. Primary non Hodgkin lymphoma of the breast should fulfill following criteria[4].

(a) adequate pathological evaluation

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(b) both mammary and lymphomatous infiltrate in close association

(c) exclusion of either systemic lymphoma or previous extramammary lymphoma.

It is very difficult to explain whether primary disease was in breast or in axillary lymph nodes as in our case. However ipsilateral presence has been acceptable[4]. The clinical presentation and radiological features of breast lymphoma and carcinoma are similar. Both presents as painless enlarging breast lump. On mammogram, lymphomas may lack the irregular borders of infiltrating carcinoma and more than half exhibit no calcification. However, there is considerable overlap in these features, and pathology remains gold standard to differentiate these two malignancies. Despite the clinical and radiographic similarities, the treatment options differ. For this reason, it is important to correctly differentiate lymphoma from other breast malignancies. Fine needle aspiration (FNA) cytology is a commonly used procedure in the evaluation of these lesions. Although its sensitivity is 90%, diagnostic pitfalls exist in the use of FNA to diagnose lympho-proliferative disorders. Confirmatory core needle biopsy is recommended by most authors for suspected primary lesions. The histological differential diagnosis of lymphoma includes reactive lymphoid infiltrate, medullary carcinoma, amelanotic melanoma, lobular carcinoma, and poorly differentiated ductal carcinoma[5].

The treatment of PNHBL (Primary Non Hodgkin's Breast Lymphoma) is similar to that used for other lymphomas and depends on the histological type. Most Clinicians agree that multimodality treatment is necessary[6-8]. However, recent studies have shown that aggressive B-cell lymphomas should always be treated with chemotherapy alone or in combination with radiotherapy[6,9-11]. The most effective combination reported in the literature is radiotherapy and 3 to 10 cycles of treatment with CHOP[6,9]. Only studies with relatively small cohorts of patients have been reported in the literature. For aggressive tumors, the literature recommends CHOP type chemotherapy and mastectomy with lymph node resection, if needed, for management of PNHBL. We were able to achieve an excellent response in our patient. Survival rate of primary breast lymphoma is better as compare to both lobular cases and systemic lymphoma with secondary involvement of breast. Anticancer drugs are main treatment rather than surgery so it is very important to accurately diagnose primary lymphoma of breast.

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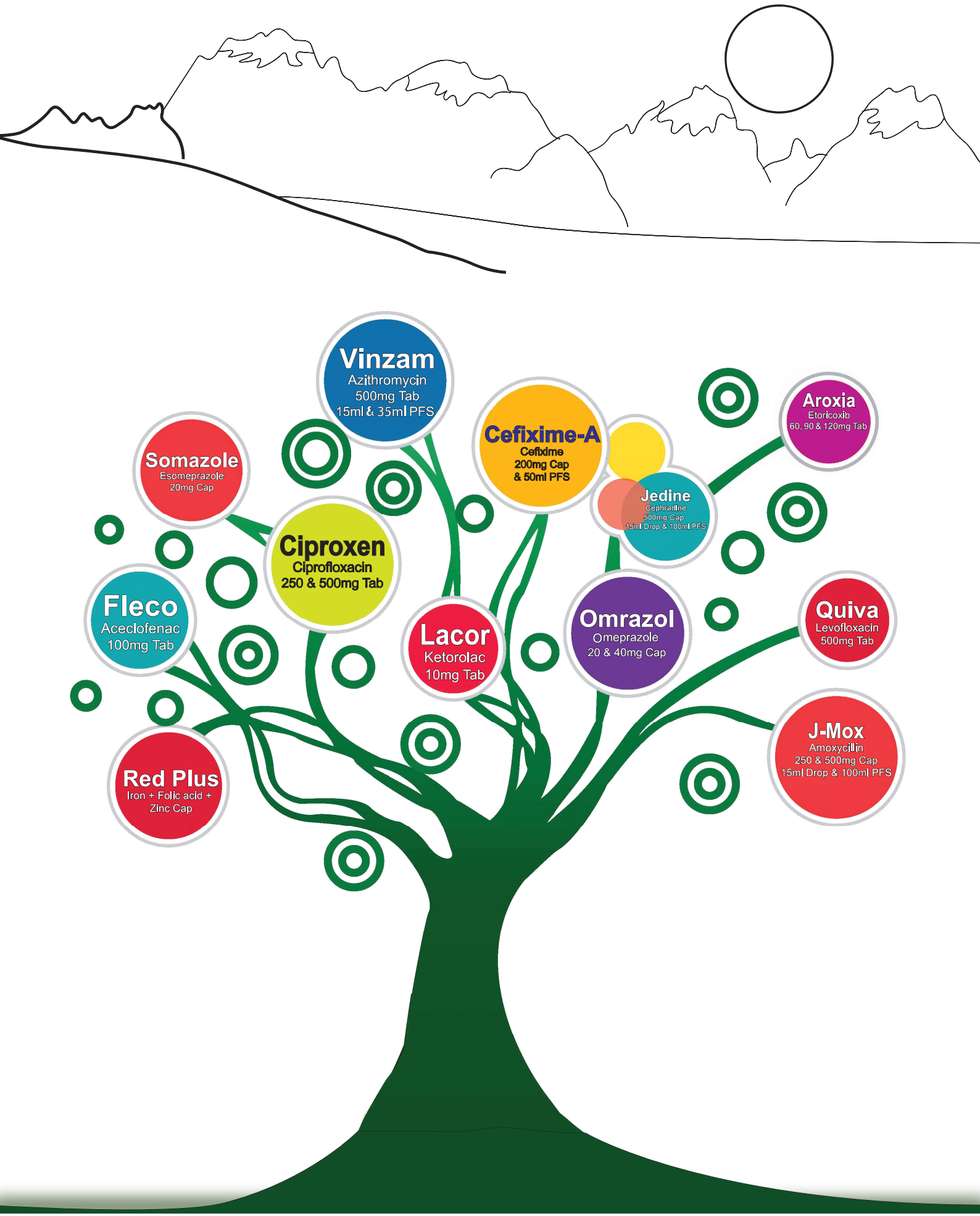

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ঢাকা মহানগরীর মধ্যে মাত্র **২৬০** টাকা

■ ফোন করুন **১০৬১০, ০১৭১৩-৪৮৮৪১১, ০১৭১৩-৪৮৮৪১২**

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