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Title page

Should include the title of the paper which should be concised but informative, name of the author(s) with highest academic degree(s) and institutional affiliation, name of the departments to which the work should be attributed, disclaimers, if any; name and address of author responsible for correspondence about the manuscript.

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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 detail to allow other workers 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.

Results

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 in the heading. Be sure that each table is cited in the text. Figure should be professionally drawn and photographed.

Tables

Type each table double spaced on a separate sheet. Do not submit tables as photographs. Number of tables consecutively in the order of first citation in the text and supply a brief title for each. Give each column a short or

abbreviated heading. Place explanatory matter in footnote, not in the heading. If you collect data from another published or unpublished source obtain permission and acknowledge fully. The use of too many tables in relation to the length of text may produce difficulties in the layout of pages.

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

i) the contributions that need Acknowledging but do not justify authorship, such as general support by a departmental chair
ii) Acknowledgements of technical help
iii) Acknowledgements of financial and material support, which should specify the nature of the support.

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Should be given in the text using Vancouver system. They should be numbered consecutively in the order in which they first appear in the text using superscript. If a reference is cited more than once the same number should be used each time. References cited in only in tables or figure legends should be numbered in accordance with the sequence from the last number used in the text and in the order of mentioned individual tables and figures in the text. At the end of the paper, on a page(s) separate from the text, references should be listed in numerical order. References to materials available on websites should include the full internet address and the date of the version cited.

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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Editorial

Carcinoma in deeply located abdominal organs often presents in advanced stage as an indirect death notice.

With increasing life expectancy in Bangladesh malignant disease of different organs are also raising in magnitude which will be a major burden to health care providers in near future. Carcinoma pancreas, gallbladder, kidneys, adrenals, liver & colon often remain asymptomatic until they become advanced. Fulminating presentation in advanced stage of the disease put the physician, patient and near relatives in a dilemma as regards cost benefit of treating such diseases. There are instances that people sell their properties to mitigate the treatment cost at home and abroad. Frequently it happens that dependents has to bear the loss of early demise of their only earning member and their last belongings.

Mrs. Alta begum, 35 years admitted in female surgery ward with acute pain at right hypo chondriac region, ultra-sonography report showed gallstone with gall bladder mass with probable hepatic metastasis. CT guided FNAC confirmed the diagnosis of metastatic adeno-carcinoma.

Mrs. Shazu Begum, aged 55 years admitted in surgery unit with cholelithiasis with a suspicious soft tissue mass in gall bladder, laparotomy revealed a small soft tissue mass in gall bladder having porta-hepatis metastasis histopathology report of which was carcinoma gall bladder.

Akhtery Begum aged 43 years had a cholecystectomy four month back, omental biopsy showed a metastasis, she was on chemotherapy for last three and a half months. Now she developed severe fixed abdominal pain with vomiting probably due to retroperitoneal involvement of shelve of malignant tissues and also involvement of gut.

Mr. Ajmat Ullah aged 56 years had laparoscopic cholecystectomy for gall stone disease, gall bladder histopathology reported as carcinoma involving muscle layer. Mr. Ullah is moving from surgeon to surgeon, and oncologist to oncologist for opinion regarding further surgery, and or chemotherapy. All the above scenarios are presentation of GB cancer.

Although gall bladder carcinoma is a relatively uncommon malignant tumor. It is the 5th most common tumor of gastrointestinal tract and accounts for 3% of all the gastrointestinal tumors. Chronic inflammation, gallstone disease, porcelain gall bladder, gall bladder polyp, chronic salmonella infection, congenital biliary cysts, and abnormal pancreaticobiliary duct junction are well-known predisposing factors of GB cancers. It may be associated with gallstones in 70% of cases and the risk of malignant metaplasia correlates with the length of time gallstones have been present. The tumor is twice as common in women as in men, as one would expect from the association with gallstones.

Patient may be asymptomatic at the time of diagnosis. The most common presenting complaint is of right upper quadrant pain or cholecystitis. Other cases present with anorexia, nausea, weight loss and obstructive jaundice with or without cholangitis due to secondary involvement of the common bile duct. Patient may present with advanced malignant disease like palpable gall bladder mass, hard nodular liver, ascites & gastric outlet obstruction etc.

The preoperative diagnosis is made with ultrasonography and confirmed by a CT scan or MRCP. Contemplated ERCP is a better option for evaluation and insertion of a biliary stent. Staging laparoscopy usually performed to detect the peritoneal or liver metastasis just prior to laparotomy which can help in avoiding unnecessary laparotomy in

about 33% cases. Despite the advances of medical imaging, most of the gallbladder carcinoma is incidentally detected intra-operatively or on histopathological examination after cholecystectomy.

Choice of treatment primarily depends on tumor stage. If a localized carcinoma is recognized at laparotomy, cholecystectomy should be performed along with en block wedge resection of an adjacent 2 cm of normal liver tissue and dissection of the regional lymphnodes. Patients can have the disease diagnosed following histopathological examination of the gall bladder removed for presumed benign disease. In these cases the need for further surgery is determined by the stage of disease. For early stage, disease confined to the mucosa or muscle of the gallbladder no further treatment is indicated. However, for transmural disease, a radical en-bloc resection of the gallbladder fossa and surrounding liver tissue along with the regional lymph nodes should be performed. If the initial procedure is performed laparoscopically, the laparoscopic port sites should be excised. Port site metastasis is a marker for a greater likely hood of subsequent peritoneal disease. A second look should always be offered in selected cases as 5-year survival improves by completion of radical cholecystectomy. For majority of patients, a non-operative approach to palliation is the best in the form of biliary stenting by endoscopic or transhepatic means, gastro jejunostomy or palliative cholecystectomy. In situations where curative resection is not possible, radiotherapy with or without chemotherapy has been tried but with only little impact on survival. Poor general condition and presence of jaundice may also suggest inoperable disease. However, with improved surgical technique and postoperative management, in case of stage II and stage III disease mortality has reduced to 5 percent and at the same time an improved survival of 35 to 42% has been achieved with radical resection.

The massage from the above discussion, in gall bladder malignancy the only hope of cure is early detection, early adequate surgery, and if anyone having the above mentioned predisposing factors get those treated early as once the disease is advanced the cure rate is negligible. Main stay of treatment is surgery and role of chemotherapy and radiotherapy is not yet satisfactory.

Professor Abu Ahmed Ashraf Ali

Original Article

Characteristics of mothers associated with immunization of their children in a selected urban slum.

Shireen Ayesha Siddiqua[1], Momena Khatun[2], Abeeda Tasnim Reza[3], Md.Tanvir Rahman[4],

Abstract

Objective : To determine the knowledge, attitude and practice of mother of a slum area in relation to immunization of their children.

Methods : This cross sectional descriptive type of study was conducted in selected slum area of Dhaka city. One hundred five mothers were interviewed purposively.

Results : It is revealed that 75.23% mothers were informed regarding immunization from the health workers. Only 12 (11.44%) mothers had correct knowledge and 74.23% had partial knowledge regarding the name of the six EPI target diseases. The study also showed that 45 (42.85%) mothers had correct knowledge and 50.48% had partial knowledge about the six EPI vaccines. Regarding practice i.e., immunization status 84.8% were vaccinated fully, 15. 2% were not vaccinated mainly due to negligence (75%) of the parents; the commonest reason for negligence was that they were preoccupied in the livelihood-generation activities.

Conclusion : The knowledge and attitude regarding immunization is quiet good but practice of vaccination what was proposed to be achieved under the immunization programme was lagging. This emphasizes the imperative need for urgent intervention to address the issues of both dropout and lack of access, which are mainly responsible for partial immunization and non-immunization respectively.

Key Word : Immunization, Knowledge, Attitude, Practice.

Introduction

Immunity is predetermining factor for growth and survival of all the children. The specific type of immunity can be artificially produced by administration of different types of vaccines. Immunization has been one of the most significant, cost-effective and stimulatory public health interventions. Bangladesh, along with the whole world, stands committed to the welfare of children, as reflected in the theme of 'World Health Day, 2005' viz., 'Make every mother and child count.[1]

The most important indicators mentioned in the Millennium Development Goals (MDGs) are the under-5 mortality rate (U5MR), infant mortality rate (IMR) and proportion of 1-year-old children immunized against measles (P1MV).

About one-quarter or 25% of under-5 mortality is due to vaccine-preventable diseases.[2] Though history of immunization is long one, it is only in last two or three decades that significant efforts have been made forwards the universal protection.[3] The World Health Organization (WHO) launched the Expanded Program on Immunization (EPI) in 1974 globally with focus on prevention of the six childhood vaccine-preventable diseases by the year 2000. This was endorsed by the Government of Bangladesh in 1979.[4] EPI is the only program of health sector which is working throughout the country. This program has achieved tremendous success in the last few years, but yet to reach its target due to some shortcomings both in the delivery system and among the service receivers.[5]

To ensure the success for any immunization campaign it is imperative to obtain the understanding and co-operation of the people who are directly involved in the programme.[6]

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To become successful in making available immunization services within easy reach to all children, it is most necessary to recognize the attitude and knowledge of our people towards immunization.[7] Low acceptance level of immunization service is commonly observed in certain group of people who have sometimes been characterized by low socio-economic status, low education level of mothers of children.[8]

In the last few years, population of our country grew rapidly, but urban population grew more rapidly. Most of this growth is due to migration, leading to mushrooming of slums. With the rapid growth of big cities, an impending threat of outbreak of vaccine-preventable diseases always exists due to the high population density, continuous influx of a new pool of infective agents with the immigrating population and poor coverage of primary immunization in the urban slums.[7,9] In view of this, it is necessary to understand the dynamics of utilization of immunization services by the community.[10,11] The current study seeks to determine the various factors related to knowledge, attitude and practice of mothers on immunization and prevention of these diseases by immunization and identify risk groups and reasons for under-immunization.

METHODOLOGY

This descriptive, cross sectional study was conducted in Malibagh Bazar slum of Dhaka city which is hugely populated and centrally located in Dhaka city from 1st March 2006 to 31st May 2006. A total of 105 mothers of children under-5 years of age were purposively interviewed by using pre-tested Questionnaire after getting a mother fulfilling selection criterion for inclusion in the study. Finally the data were analyzed, interpreted and tabulated by using computer. Knowledge regarding name of six EPI target diseases and number of vaccine was categorized as follows :

Name of six EPI target diseases :

Correct : Knows all the name of EPI target diseases.

Partially correct : Knows some of the name of EPI target diseases.

Incorrect : Does not know the name of EPI target diseases.

Number of EPI vaccines :

Correct : Knows all the numbers of vaccines.

Partially correct : Knows some of the numbers of vaccines.

Incorrect : Does not know the numbers of vaccines.

RESULTS

Table No. 1 : Distribution of respondents by age and educational qualification.

Age (in years)	Frequency	Percent
15-20	07	6.67
20-25	32	30.48
25-30	35	33.33
30-35	23	21.90
35 or above	08	7.82
Total	105	100.00
Educational qualification		
Illiterate	62	59.00
Primary (i-v)	28	26.70
Secondary (vi-x)	15	14.30
Total	105	100.00

Majority of the respondents 67(63.81%) belong to the age group of 20-30 years, most of the respondents 62(59%) were illiterate.

Figure No. 1 : Distribution of respondents by occupation.

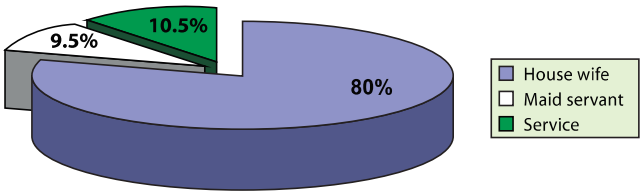

Table No. 2 : Distribution of the respondents by monthly income.

Income (in Taka)	Frequency	Percent
2000-3000/-	57	54.30
3001-4000/-	38	36.19
4001-5000/-	06	5.17
5001-6000/-	04	3.80
Total	105	100.00

Majority of the respondents i.e., 95(90.49%) were earning 2000-4000/- monthly.

Table No. 3 : Distribution of the respondents according to the number of children.

No. of children	Frequency	Percent
1	24	22.86
2	41	39.05
3	28	26.66
>3	12	11.43
Total	105	100.00

Among the respondents 41 (39.05%) have 2 children, 28(26.66%) have 3 children, 24 (22.86%) respondents have 1 children and 12 (11.43%) have 4 or more children.

Table No. 4 : Distribution of the respondents according to the source of information regarding Immunization.

Source of information	Frequency	Percent
Neighbour	22	20.95
Health worker	79	75.23
Physician	02	1.19
Radio / TV	02	1.91
Total	105	100.00

Majority of the respondents 79(75.23%) knew about immunization from health worker, 22(20.23%) from neighbour, 2(1.91%) from physician and remaining 2(1.91%) from Radio/TV.

Table No. 5 : Distribution of the respondents according to the knowledge regarding the name of six EPI target diseases and number of vaccines.

Knowledge About six EPI diseases	Frequency	Percent
Correct	12	11.44
Partially correct	78	74.23
Incorrect	15	14.33
Total	105	100.00
Knowledge about no. of EPI vaccines		
Correct	45	42.85
Partially correct	53	50.48
Incorrect	07	6.67
Total	105	100.00

Majority of the respondents 78 (74.23%) had partially correct knowledge about the name of the six EPI target diseases, only 12(11.44%) had correct knowledge about six EPI target diseases. 53(50.48%) of respondents had partially correct knowledge about the number of EPI vaccines, 45(42.85%) had correct knowledge and remaining 7(6.67%) had incorrect knowledge.

Table No. 6 : Distribution of the respondents according to the Immunization status at the children in the family.

Immunization status	Frequency	Percent
Immunized	89	84.80
Not immunized	16	15.20
Total	105	100.00

Among the respondents 89(84.8%) had immunized all of their children and remaining 16(15.2%) didn't immunize all of their children.

Table No. 7 : Distribution of the respondents according to the cause of not being vaccinated.

Causes of not being vaccinated	Frequency	Percent
Bad communication	01	6.25
Not available	03	18.75
Negligence	12	75.00
Total	16	100.00

Among the respondents 12(75%) didn't vaccinated their children due to negligence, 3(18.75 %) due to unavailability and 1(6.25%) due to bad communication

Table No. 8 : Relationship between education of mother and knowledge about six EPI target diseases & number of EPI vaccines.

Education of mother	Knowledge about 6 EPI target diseases			Total	Percent
	Correct	Partially correct	Incorrect		
Illiterate	00	50	12	62	59.05
Primary (i – v)	07	18	03	28	26.67
Secondary (vi-x)	05	10	00	15	14.28
Total	12	78	15	105	100.00
P<0.05					
	Number of EPI vaccines			Total	Percent
Illiterate	00	03	05	08	7.62
Primary (i – v)	10	08	02	20	19.05
Secondary (vi-x)	35	42	00	77	73.33
Total	45	53	07	105	100.00
P<0.01					

Statistically significant relationship between Education of mother and knowledge about EPI diseases and vaccines.

Table No. 10 : Relationship of economic-status and vaccination failure

Income (in Taka)	Causes of not being vaccinated			Total	Percent
	Bad communication	Not available	Negligence		
2000-3000/-	01	02	09	12	66.66
3001-4000/-	00	01	03	04	33.34
Total	01	03	12	16	100.00
P<0.01					

Table-10 Shows the statistically significant relationship between economic status and vaccination failure.

DISCUSSION

Our study revealed that a significant improvement in the percentage of complete immunization has occurred, in the urban slums of Dhaka, as a result of sustained efforts by the government; but the achievements lag far behind the national goal, even after more than 20 years of formal introduction of the EPI. 100% of the mothers knew from different sources that there are some diseases which are preventable by vaccination at poorer age. They also heard about immunization. It showed that, the health worker's of different government agencies and NGOs are playing a key role in the promotion of health care system.

The study showed that, the respondents had varying degrees of knowledge about immunization. Some of the mothers could answer correctly the name of the six EPI target diseases, while 42.85% of the mother could answer correctly the number of vaccines in EPI. In contrast to these findings, another study done by Khanam K. and Salahuddin AKM showed that most of the parents had knowledge about six EPI target diseases and number of vaccines.[12] In contrast to these findings another study showed that, most of the parents had poor knowledge about six EPI target diseases and number of vaccines.[13,14,15]

This study finding suggests that, the majority (84.8%) of the mothers immunized their children completely. The rest (15.2%) of the respondents failed to immunize their children mostly because of negligence (75%), unavailability (18.75%) and bad communication (6.25%) at their previous residence. According to the respondents, the commonest reason for negligence as they were preoccupied in the livelihood-generation activities. This reflects the un-met needs of the community, which require organization of outreach services on fixed date and timing with prior information to the locality. Similar study done by Malini Kar et al.[16] showed that unavailability of both the parents (17.2%) had gone either to village/native place during the scheduled date of vaccine (14.7%), carelessness (11.7%), apprehensiveness due to sickness of the child or an elder sibling as a result of vaccination (11.7%) and lack of knowledge (10.4%). Solving these would require proper education and constant motivation through an encouraging and persuasive interpersonal approach, regular reminders and removal of misconceptions prevailing among people and improving the quality of the services at the health facility, along with proper training of the health provider to seize the missed opportunities.[17,18]

CONCLUSION

The knowledge and attitude of the mothers regarding immunization were satisfactory but practicing of these two still lacking in significant proportion. Immunization is one of the most powerful and cost effective weapons of preventing disease. The goal of achieving universal immunization, especially in the disadvantaged, vulnerable urban slum population with poor health infrastructure, needs a coordinated effort and a multi-prolonged strategy to deal with both lack of access and dropout. The measures to be taken would include reaching out people effectively to generate demand for the services through interpersonal communication, which can be translated into a change in behaviour and then maintaining the demand consistently.

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Original article

Preventive role of Zingiber officinale (ginger) juice against hyperlipidemia in alloxan induced diabetic rats

Selima Sultana[1], Syed Ashrafuzzaman[2], Md. Ismail Khan[3]

Abstract

Objectives : This experimental animal study was undertaken to investigate the preventive role of ginger juice against hyperlipidemia in alloxan induced diabetic rats.

Methods : Male wistar rats, (130-150) gm wt. fed on standard diet and water ad libitum, were divided into 3 groups (n=6) in each group: Group-I, non-diabetic control, Group-II diabetic control & Group-III, normal rats pretreated with ginger before they were made diabetics. Diabetes was induced by inj. Alloxan 150mg/kg body wt. i.p (Group-II , on 2nd day & Group-III, on the 9th day).

Results : Rats having blood glucose level of more than 7mmol/L on day 5 (72 hrs after alloxaninj) were considered diabetic & selected for experiment. Rats of Group-III received Zingiber officinale (ginger) juice (4ml/kg. body wt, orally) for 7 days (day 2-day 8) through ryles tube before alloxan induction & 3 days after the induction. On day 12 animals were sacrificed under light ether anaesthesia, blood was collected by cardiac puncture and serum separated for estimation of lipids. Pretreatment with Zingiber officinale (ginger) juice significantly ($p< 0.01$) reduced alloxan induced hyperglycemia & hyperlipidemia.

Conclusion : Hyperlipidemia, a metabolic derangement, contributing to atherosclerosis in diabetes mellitus. Zingiber officinale (Ginger) is one of the most widely used spices and is reputed to have medicinal properties against diabetes mellitus & it has also hypocholesterolemic effects. This study suggest that pretreatment with Zingiber officinale (ginger) prevents the development of hyperlipidemia in alloxan induced diabetic rats.

Key words : Hyperlipidemia, Zingiber officinale (ginger), diabetic rats.

Introduction

Hyperlipidemia is a major risk factor for atherosclerosis in diabetes mellitus. Diabetic individuals have 2-4 folds increased risk of clinical atherosclerotic disease[1]. Atherosclerosis increases the risk of heart disease, stroke and other vascular diseases. The use of herbal remedies has increased many folds from 1990 onwards.

These herbal remedies are apparently effective, produce minimal or no side effects and are of relative low costs as compared to oral synthetic hypoglycemic agents. In recent years, ginger has become a subject of interest because of its beneficial effects on human health.

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Zingiber officinale commonly known as ginger is consumed worldwide as spice and is known to have wide variety of medicinal properties. It was reported that ginger has medicinal properties against digestive disorders, rheumatism & diabetes.[2] Akhani et al. reported that ginger pretreatment inhibited streptozotocin hyperglycemia & hypoinsulinaemia.[3] Sharma et al, 1996, have showed the hypolipidemic effect of ginger. In diabetic rats, the impaired utilization of carbohydrate leads to accelerated lipolysis resulted in hyperlipidemia.[4] Another study has reported that an ethanolic extract of ginger prevent hypercholesterolemia and development of atherosclerosis in cholesterol fed rabbits.[5] It was concluded that the hypo-cholesteromic effect of ginger could have possibly resulted from the inhibition of cellular cholesterol biosynthesis after the consumption of the extract.[6] Furthermore, Neess et al. reported that the reduction of cellular cholesterol biosynthesis is associated with increased activity of the LDL receptor, which in turn leads to enhanced removal of LDL from plasma, resulting in reduced plasma cholesterol concentration.[7]

The present study was undertaken to investigate the

preventive role of fresh ginger juice against hyperlipidemia in pretreated rats followed by the induction of diabetes.

MATERIALS AND METHODS

The experimental animal study was done in the department of Pharmacology & Therapeutics, Dhaka Medical College and Hospital in collaboration with the Department of Pathology of Ibrahim Medical College, Dhaka, during January 2009 to December 2009.

Plant materials and preparation of juice

The fresh juice of Zingiber officinale (ginger) was obtained from local market. 1 kg of fresh rhizome were crushed, and then squeezed in muslin cloth to obtain the juice using the method of Akhani et al.[3] Sodium benzoate (0.5%) was added as preservative. The juice was stored in the refrigerator at 2-8°c in a well-closed glass container.

Animals

Male wistar rats weighing between 130-150gm were housed in polycarbonate cage sat a regulated temperature (22±2°c) & humidity (55%) controlled room with a 12h light/12h dark cycle for 12 days and were fed on standard rat pellet diet and allowed to drink water and libitum.

Induction of diabetes in rats

After 24 hours fasting, rats of (group II & III) were injected alloxan 150 mg/ kg b.w. i.p on Day 2 and Day 9 of the study respectively.

Fasting blood glucose levels were estimated on day1(before inj. alloxan), on day 5 (72 hrs after inj. alloxan) and day 12 of the experimental study. Blood glucose was estimated by placing a test strip in the glucometer (ACCU-CHEK, Roche diagnostic GmbH). A drop of blood was collected by aseptically cutting the tail at the tip (0.1cm) with sharp sterile blade and then applying the drop of blood to the test area of the strip. Rats with blood glucose of more than 7mmol/L on day 5 (i.e. 72 hrs after inj. alloxan) were considered diabetic & selected for experiment.

Experimental design

Rats were divided into 3 groups (n=6, in each group). Group 1 : Normal (non diabetic) control, Group II : Diabetic control & Group III : Normal rats pretreated with ginger before they were made diabetics, rats of this group received Z. officinale (ginger) at a dose of 4ml/kg body weight (as per Akhani et al[3]) for 10 days (day 2- day 11) orally through ryles tube. On the 12th day of the study, animals were sacrificed under light ether anaesthesia, whole blood was collected by cardiac puncture and then serum was separated for estimation of lipids.

Statistical Analysis

The results are presented as mean ± SD. Unpaired 't' test was performed and p value <0.05 was considered as statistically significant.

RESULTS

Diabetic control is compared with normal. Treated group is compared with diabetic control.

A. Effects of Z. officinale (ginger) juice on blood glucose level in normal, diabetic & pretreated rats.

i. The mean ± SD of blood glucose (mmol/L), in normal non- diabetic rats (Group-I) on day 1 and day 12 of the study were 5.40±0.76 and 5.45±0.76 respectively, while in diabetic control rats (Group-II) were 5.57±0.12 and 8.52±0.68 respectively. The difference between two groups (Group-I vs. Group-II) were statistically significant ($p<0.001$) suggesting that inj. alloxan significantly increased the blood glucose level. The mean ± SD of blood glucose (mmol/L), of diabetic control rats (Group-II) and of pretreated rats (Group III, normal rats pretreated with ginger for 7 days before inj. alloxan and 3 days after induction) on day 12 of the study were 8.52±0.68 and 7.50±0.42 respectively. The difference between two groups (II & III) were statistically significant ($p<0.011$), suggesting that pretreatment with ginger juice before inj. alloxan produced significant decrease in blood glucose level when compared with diabetic control. The results are shown in Table-1.

Table - 1 : Effects of Zingiber officinale (ginger) on blood glucose in non-diabetic normal control (Group-I), & diabetic control rats (Group-II)

Fasting blood glucose (mmol/L)	Group-I (n=6)	Group-II (n=6)	p value
At 1st day	5.40±0.76	5.57±0.12	0.619ns
At 12th day	5.45±0.76	8.52±0.68	.001***
Fasting blood glucose (mmol/L)	Group-II (n=6)	Group-III (n=6)	p value
At 12th day	8.52±0.68	7.50±0.42	.011**

Data were expressed as Mean ± SD

**= significant at 0.01

***=significant at .001

ns= not significant

B. Effects of Zingiber officinale (ginger) juice on lipid profile in normal, diabetic & pretreated rats.

i. Effects of Zingiber officinale (ginger) juice on lipid profile in normal (non-diabetic) & diabetic control rats

Lipid profile (mean ± SD) of total cholesterol,

HDL-cholesterol, LDL-cholesterol and triglyceride G, all in mg/dl, (estimated on day 12 of the study) of normal (non diabetic) control rats (Group-I) were 85.67±977, 43.67±5.61, 22.83±4.83 and 51.67±4.67 respectively. While those of diabetic control rats (Group-II) were 111.00±5.87, 37.17±6.01, 55.50±3.94 and 75.00±3.58 respectively. The difference in lipid profile (increase in total cholesterol, LDL- cholesterol & triglyceride) in two groups (Group-I vs. Group-II) were statistically significant (p<.001 for Total cholesterol, LDL-cholesterol & Triglyceride), but HDL-cholesterol level statistically without change when compared to normal group.

ii. Effects of Zingiber officinale (ginger) on lipid profile in diabetic control (Group-II) & Pretreated rats before they made diabetics (Group-III).

Lipid profile (mean ± SD) of total cholesterol, HDL-cholesterol, LDL- cholesterol and TG, all in mg/dl (estimated on day 12 of the study) were 111.00±5.87, 37.17±6.01, 55.50±3.94 and 75.00±3.58 respectively, while those of pretreated rats, Group-III, (ginger juice 4ml/kg body weight for 7 days from 2nd day & 3 days after induction& inj. alloxan 150 mg/kg body weight given on the 9th day), were102.33±6.09, 55.17±6.27, 12.33±3.27 and 70.67±4.50 respectively. The differences in lipid profile (decrease in total cholesterol, LDL-cholesterol, and increased in HDL- cholesterol) in two groups (Gr.-II & III) were statistically significant (p<0.031 for total cholesterol, and for LDL- cholesterol p<.001, for HDL- cholesterol p<.001 &) when compared with diabetic control rats, while plasma TG statistically (p<0.095) did not change. The results are shown in table-2.

Table 2 : Effects of Zingiber officinale (ginger) juice on lipid profile in normal control (Group-I), diabetic control (Group-II) & pretreated rats (Group-III).

Lipid profile	Group-I	Group-II (n=6)	p value (n=6)
Total cholesterol (mg/dl)	85.67±9.77	111.00±5.87	0.001***
HDL (mg/dl)	43.67±5.61	37.17±6.01	081 ns
LDL (mg/dl)	22.83±4.83	55.50±3.94	0.001***
TG (mg/dl)	51.67±4.76	75.00±3.58	0.001***
Lipid profile	Group-II (n=6)	Group III (n=6)	p value
Total chol(mg/dl)	111..00±5.87	102.33±6.09	0.031*
HDL (mg/dl)	37.17±6.01	55.17±6.27	0.001***
LDL (mg/dl)	55.50±3.94	12.33±3.27	0.001***
TG (mg/dl)	75.00±3.58	70.67±4.50	0.095ns

All estimation were done on day 12 of the study
Data were expressed as Mean ± SD
*= significant at 0.05
***= significant at 0.001
ns= not significant

Discussion

The present study was undertaken to investigate the preventive role of Zingiber officinale (ginger) juice against hyperlipidemia in alloxan induced diabetic rats. Injection of alloxan (150 mg/kg body weight, intravenous) produced marked hyperglycemia and hyperlipidemia (increased total cholesterol, LDL-cholesterol & TG and decreased HDL- cholesterol). Treatment with Zingiber officinale (ginger) juice (4ml/kg body weight, per oral) for 7 days from 2nd day to normal rats before they were made diabetics & 3 days after the induction, produced significant blood glucose and lipid lowering (decreased total cholesterol, LDL- cholesterol & TG & increased HDL-cholesterol) effects. Thus, suggesting preventive role of Zingiber officinale (ginger juice) against hyperlipidemia in alloxan induced diabetic rats. The results are in agreement with those of previous studies 3-5, who showed similar lipid lowering effects in pretreatment with Zingiber officinale (ginger) in different experimental animal models.

Conclusion

The present study demonstrated the preventive role of Zingiber officinale against hyperlipidemia in alloxan induced diabetic rats. Further studies are suggested for investigating possible mechanism(s) of action. However further investigations may be required to find out the specific role of different lipid profile in large number of experimental animals in different settings.

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Original Article

Renal Bone Disease : Biochemical Marker in CKD

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Abstract

Objectives : The Renal bone disease is one of the important complication of chronic kidney disease. This study was undertaken to identify some biochemical markers like parathyroid hormone (PTH) and alkaline phosphatase (ALP) which can help to predict the renal bone disease before doing bone biopsy.

Methods : To understand the parameters 120 subjects were studied, ages ranging from 20 to 60 years. Among them 100 (male = 70, and female = 30) were chronic kidney disease patients stage III, IV & V as experimental group and 20 were healthy control group - A (male = 12, female = 08). The study subjects both in control and the experimental group were well matched by age and body weight. The chronic kidney disease patients were divided into three groups on the basis of creatinine clearence: group-B1 (CKD- III, n=34), group-B2 (CKD –IV, n=36) and group-B3 (CKD - stage V, n= 30). The study parameters were serum alkaline phosphatase & serum PTH.

Results : The Mean (±SD) of serum alkaline phosphatase were 49.40± 58.48, 97.54± 58.48, 173.44± 122.11 and 341.83 ± 159.08 in group A, B1, B2 and B3 respectively. The mean values of alkaline phosphatase in experimental group were compared with control group and the differences were found statistically highly significant. The mean values of serum alkaline phosphatase also showed highly significant differences in group B2 than B1 ($p < 0.001$) and group B3 than B1 ($p < 0.000$). The Mean (±SD) values of serum PTH were 40.60±10.44, 78.10±19.99, 105.89±37.22l, and 220.10±127.18 in group A, B1, B2 and B3 respectively. The mean serum PTH were lower in group A than group B1 ($p<0.004$), B2 ($p< 0.0001$) and B3 ($P<0.0001$) which were statistically highly significant. Serum PTH was compared within the groups of the experimental subjects, in group B2 was higher than group B1 ($p< 0.002$), group B3 was higher than B1 ($p < 0.000$) and group B3 also higher than group B2 ($p < 0.0001$). The differences were statistically highly significant. Serum PTH level was higher in all experimental groups than the control group ($p< 0.0001$***).

Conclusion : The Renal bone disease is one of the important complication of chronic kidney disease and the gold standard of diagnosing renal bone disease is bone biopsy. But assessment of these biochemical markers parathyroid hormone (PTH) and alkaline phosphatase (ALP) which can help to predict the renal bone disease before doing bone biopsy.

Key Word : Renal Bone Disease, Biochemical Marker, CKD (chronic kidney disease)

Introduction

The management of chronic kidney disease & mineral bone disorder (CKD-MBD) is central to the care of patients with kidney disease. Key to these efforts is the availability of clinically accessible biomarkers that can help distinguish between a wide variety of bone and mineral disturbances related to kidney failure.

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Two such markers, parathyroid hormone (PTH) and alkaline phosphatase, are well-established in current guidelines[7] for managing CKD-MBD are familiar to most clinical practitioners.

PTH has been a mainstay in the evaluation of bone and mineral metabolism in CKD patients for more than three decades. The long-term consequences associated with persistently elevated PTH levels in CKD have been well-described and include high-turnover bone disease, anemia, cardiovascular disease (CVD) and mortality.[1]

As a result, both the Kidney Disease Outcomes Quality Initiative (KDOQI) and Kidney Disease Improving Global Outcomes (KDIGO) guidelines recommend that PTH levels should be regularly monitored beginning in stage 3 CKD (i.e., estimated glomerular filtration rate [eGFR] < 60 ml/min/1.73m^2), and that elevated levels should be treated with a combination of dietary phosphorus restriction and supplement of vitamin D and/or calcimimetics.[1,2]

Alkaline phosphatase is an enzyme that removes phosphate from proteins and nucleotides and can be detected in a variety of tissues throughout the body.[3] Because the highest concentrations of the enzyme are found in the liver and bone, an elevated total alkaline phosphatase level is most often indicative of bone pathology such as high-turnover bone disease.

The measurement of alkaline phosphatase has been advocated as an adjunct test for non-invasively assessing bone turnover in CKD patients, particularly in clinical scenarios in which elevated PTH levels may be challenging to interpret.[2] These recommendations were based upon studies that showed that elevated alkaline phosphatase levels have predictive value in diagnosing high-turnover bone disease in both pre-dialysis and end-stage kidney disease population[2]. The more recent KDIGO guidelines recommend that the measurement of alkaline phosphatase levels should commence in stage 3 CKD, and that in patients with stage 4-5 CKD, alkaline phosphatase should be measured at least every 12 months, and more frequently when monitoring response to therapy.[2,4,5]

In addition to its potential utility in assessing bone health, recent evidence suggests that alkaline phosphatase level have value for predicting CVD outcomes.[4] Large prospective studies showed that elevated alkaline phosphatase levels were independently associated with increased risks of renal bone disease and CVD- related hospitalization and mortality in patients across the spectrum of kidney function.[6-7]

Materials & Methods

A total number of one hundred twenty (120) subjects of age ranging from 20 – 60 years, consisted of hundred (100) chronic renal failure patients (Group B) and twenty (20) apparently healthy subjects as a control (Group A). All the experimental subjects (Group B) was again subdivided into three groups on the basis of creatinine clearance rate :

Group B1 = Stage -III (30 – 59 ml / min / 1.73 m2 bsa),

Group B2 = Stage – IV (15 – 29 ml /min / 1.73 m2 bsa),

Group B3 = Stage - V (<15 ml / min / 1.73m2 bsa).

PTH was measured by chemi luminescent immuno assay, phosphate by colorimetric method in both control and experimental group.

Data were expressed as Mean ± SD. Comparison between two groups were done by using unpaired student's "t" test using SPSS version -10.

Results

Serum alkaline phosphatase:

The results are shown in table I.

The Mean (±SD) of serum alkaline phosphatase were 49.40± 58.48, 97.54± 58.48, 173.44± 122.11 and 341.83 ± 159.08 in group A, B1, B2 and B3 respectively.

The mean values of alkaline phosphatase in experimental group were compared with that of control group: A vs. B1 ($p < 0.007$), A vs. B2 ($p < 0.0001$) and A vs. B3 ($p < 0.0001$) and found statistically highly significant difference.

The mean values of serum alkaline phosphatase also showed highly significant differences in group B2 than B1 ($p < 0.001$) and group B3 than B1 ($p< 0.0001$), but there were no significant difference between B2 and B3 ($p < 0.157$).

Table – I

Mean (± SD) Serum alkaline phosphatase in different study groups (n =120)

Groups	n	Mean ± SD
A	20	49.54 ± 58.48
B1	34	97.46± 38.48
B2	36	173.44± 120.11
B3	30	341.83± 159.08

Statistical analysis

Groups	t	df	p value
A vs B1	3.632	52	0.007***
A vs B2	4.515	54	0.0001***
A vs B3	8.181	48	0.0001***
B1 vs B2	3.285	68	0.001***
B1 vs B3	8.345	62	0.0001***
B2 vs B3	4.863	64	0.157ns

The Results were expressed as Mean ± SD . Unpaired student 't' test was performed to compare between groups. The test of significance was done and p values <0.05 was accepted as significance level.

A = Apparently healthy control group
B1 = Stage III CKD
B2 = Stage IV CKD
B3 = Stage V CKD
CKD = Chronic kidney Disease
n = Number of subjects
ns = Not significant
df = degree of freedom
p*** = highly significant

Table –II

Mean (± SD) Parathyroid hormone in different study groups (n =120)

Groups	n	Mean ± SD
A	20	40.60 ±10.44
B1	34	79.11± 19.99
B2	36	105.89± 37.22
B3	30	220.10± 127.18

Statistical analysis

Groups	t	df	p value
A vs B1	7.974	52	0.004***
A vs B2	7.651	54	0.001***
A vs B3	6.276	48	0.0001***
B1 vs B2	3.719	68	0.002***
B1 vs B3	9.382	62	0.0001***
B2 vs B3	5.137	64	0.0001***

The Results were expressed as Mean ± SD. Unpaired student 't' test was performed to compare between groups. The test of significance was done and p values <0.05 was accepted as significance level.

A = Apparently healthy control group
B1 = Stage III CKD
B2 = Stage IV CKD
B3 = Stage V CKD
n = Number of subjects
ns = Not significant
df = degree of freedom
p*** = Highly significant

Serum PTH

The results are shown in table II

The Mean (±SD) values of serum PTH were 40.60±10.44, 78.10±19.99, 105.89±37.22 and 220.10±127.18 in group A, B1, B2 and B3 respectively.

The mean serum PTH were lower in group A than group B1 (p<0.004), B2 (p< 0.0001) and B3 (P<0.0001) which were statistically highly significant.

Serum PTH was compared between the groups of the experimental subjects. The values in group B2 was higher than group B1 (p< 0.002), group B3 was higher than B1 (p < 0.0001) and group B3 also higher than group B2 (p < 0.000). The differences were statistically highly significant. Serum PTH were negatively correlated with, serum inorganic phosphate (r = 0.362, P<0.000) (Fig: XIII) and serum alkaline phosphatase (r = 0.558, P <0.0001) (Fig. XIV). All these findings were statistically significant.

Discussion

Abnormal skeletal structure and function are relatively common findings in patients with CKD. This is especially in patients requiring dialysis.[8] Extraskeletal "soft tissue" calcification is often a feature of CKD-MBD with some evidence of reciprocity between skeletal and soft tissue calcium content.[9,10] This important interplay between skeleton, vessels, and outcome was recognized by the Kidney Disease Improving Global Outcomes (KDIGO) initiative in its CKD-MBD position paper of 2006.[10] The diagnosis of the skeletal component of the CKD-MBD triad is biopsy-based histomorphometric analysis of bone biopsy specimens which is a painful and invasive procedure, is now much less commonly performed in clinical practice.

The objective of this study was to estimate the early marker of renal bone disease as parathyroid hormone and alakaline phosphatase. It was found that in all the experimental group mean serum PTH was significantly higher than the control group A than group B1 (p<0.004), B2 (p< 0.0001) and B3 (P<0.0001) which were statistically highly significant. Kovesdy et. al studied and find that hyperparathyroidism, due to progressive phosphate retention and lack of vitamin D activity, is the major promotor of the development of osteitis fibrosa.[11]

This study also simulate with the findings that near universally sustained elevation of PTH concentrations is seen by the time of dialysis therapy begins that at the time of CKD stage -V and PTH is much more reflective of bone remodeling.[3] Although it has high sensitivity for detecting hyper-parathyroid renal bone disease.[11] The Serum PTH among the three stages of the experimental subjects.

The values in group B2 was higher than group B1 (p< 0.002), group B3 was higher than B1 (p < 0.0001) and group B3 also higher than group B2 (p < 0.0001). It was found that in advance stages of CKD the serum PTH level progressively and significantly increases. It is believed that the disease and treatment paradigm shift from the "high turnover"/high PTH osteitis fibrosa lesions predominating in the 1960s to 1980s has great significance for the bone abnormalities and must be able to detect using current biomarkers.

Tonelli et. al studied and find out that ALP is an important marker of high-turnover bone disease and, as such, it is associated with serum PTH, which itself has been linked to increased mortality.[2,11] This study also shows the same elevated level of ALP which supports our findings. Rogidor et al showed that even the lower ALP could also be indicative of low-turnover bone disease.[7–9] Eknoyan et al suggest to measure this readily available and inexpensive biomarker to singled out as an individual therapeutic target of CKD-MBD.[12]

Conclusion

From this study it can be concluded that parathyroid hormone and alkaline phosphatase are the important biochemical markers of renal bone disease and they started rising at the early stages of chronic kidney disease. It also be concluded that as the chronic kidney disease progresses the biochemical markers also gradually increases. There are so many complications of chronic kidney disease and important one is the renal bone disease.

So our findings suggest that these markers should be taken in consideration in making diagnosis and management of the patients of chronic kidney disease.

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Original Article

Breast feeding pattern among the employed mothers

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Abstract

Objectives : To assess the pattern of breast feeding among the employed mothers.

Materials & Methods : This cross-sectional study was carried out in Government and Non-Government organizations of Dhaka Metropolitan City. One hundred and ten employed mothers having one infant were selected and with minimum education graduation using pre-tested questionnaire.

Place & Period of study : The study was carried out from April to June 2009 in some Government and private organizations in Dhaka City.

Results : Mean age of the mother and infant were 30.22 years & 9-12 months. Majority 22 (57.8%) used artificial milk as pre-lacteal feeding just after birth. Only 4 (3.6%) never breast fed their infants. Mean duration of breast feeding was 6.4 months, majority 60 (54.54%) started complementary feeding at the age of six months. Among them 51 (46.36%) gave breast feed exclusively. During working hours a significant number of mothers (61.11%) gave breast milk in the form of Expressed Breast Milk (EBM). Maximum respondent 95 (86.4%) did not get any support for breast feed their infant in the work place. They faced problem for breast feeding due to inadequate maternity leave 50 (47.2%) and 29 (26.35%) due to lack of child care facilities.

Conclusion : This study concluded that an intensive program to be launched for the protection of breast feeding of the infants among the employed mothers.

Key words : working mother, infant feeding, prelacteal feeding, exclusive breast feeding

Introduction

Breast feeding is the best and most natural ways of feeding an infant. It is sufficient to provide all the necessary nutrients until about the first four to six months of life and supplies a major part of energy, protein and vitamin A during the weaning period.[1]

Exclusive breast feeding up to 6 months of age and weaning practices thereafter with appropriate energy dense food can ensure satisfactory growth and development of the children.[2] It was obvious that breast feeding pattern greatly varies between rural and urban mothers of both developed and developing countries.[3] Exclusive breast feeding is declining in developing countries like Bangladesh is found not only among urban women but also in rural mothers where the change in practice will affect many infants and young children.

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A high percentage of mothers return to work during their infants first year of life. Work environments are little considerate for women's particular needs both as workers and parents.[4] Many obstacles such as inflexible work schedules, no empathetic supervisions and absence of privacy often keep mothers from effectively draining breast milk while at work. In the face of hardship it has to be taken full advantage of one important resource, human milk.[5] However, although Bangladesh is a country with a high prevalence of Breast feeding, this recommendation is not widely followed. In a study done in rural area of Bangladesh, 85% of the children at one month of age and 30% at 6 months were breast fed predominantly.[6] Infant health and outcome of childhood can determine a countries potential for socioeconomic development. Ill health in childhood is a drain in the national economy.

Finally, taking into consideration the mentioned facts the ultimate goal of this research work is to determine the breast feeding pattern of employed women with a view to provide information for researchers and policy makers regarding infant breast feeding practices in Bangladesh, particularly among the employed women in details.

Methods And Materials

It was a descriptive cross-sectional study. Study sites were Government and private organizations in Dhaka City. Study populations were the employed women with minimum qualification of graduation having at least one living child of one year of age or below. Total 110 respondents were interviewed purposively using a semi-structure questionnaire.

Result :

Table 1 : Distribution of the respondents by socio-demographic characteristics

Characteristics	Group	No.	Percentage
Mean age	30.22 years ± S.D		
	3.05		
Education	Graduation	63	57.3
	Above graduation	47	42.7
Occupation	Government	36	32.7
	Non government	74	67.3
Income family	< 15,000 taka	7	15.5
	15,000-30000	49	44.59
	> 30,000	44	40
Mean taka	29440.91 + SD 11912047		
Type of family	Nuclear	77	70
	Joint	33	30
Number of children	One child	64	58.2
	Two child	36	32.7
	Three child	10	9.1
(Age of the last child) Mean age 9.12 months + SD 2.09			

Table-1 shows, majority of the respondents were 63 (57.3%) graduates & 74 (67.3%) were in Non-government services and 49 (44.59%) were in the income group between 15000/--30000/- Taka per month. Nuclear families were 77 (70%). Most of the respondents had one child (58.2%)

Table 2 : Type of pre-lacteal feeding after birth

Type of feeding	Frequency	Percentage
Colostrum	72	65.5
Water	10	26.3
Glucose	3	7.8
Sugar water	2	5.2
Artificial milk	22	57.8
Honey	1	2.6
Total	**38**	

Table-2 shows, 72 (65.5%) did not give anything other than breast milk just after the birth.

Figure 1 : Distribution of the respondents by breast feeding

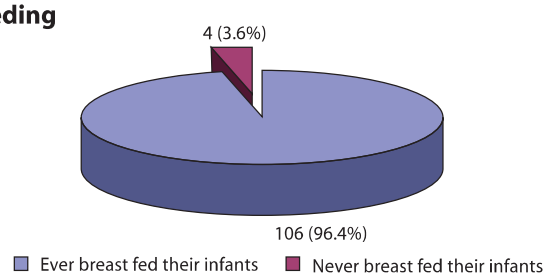

Fig-1 Among all respondent 106 (96.4%) ever breast fed their infants 4 (3.6%) never breast fed their infants.

Table 3 : Distribution of respondents by Exclusive breast feeding by duration

Duration in month	Frequency	Percentage
4 months	29	56.86
5 to 6 months	19	37.25
>6 months	3	5.88
Total	**51**	

Table - 3 shows only 19 (37.25%) mother gave exclusive breast feeding for 5 to 6 months The mean duration of exclusive breast feeding was 3.8 months.

Figure 2 : Duration of breast feeding

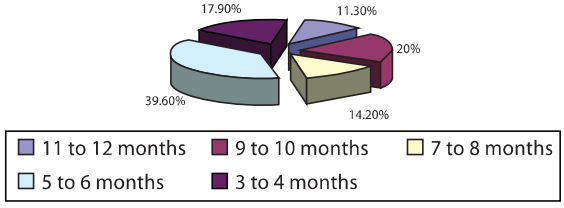

Fig - 2 showed that 39.60% respondents breast fed their infants in the duration of 5 to 6 months and 11.3% continued up to eleven to twelve months age of the infants. Mean duration of total breast fed was 6.4 months.

Table 4 : Reason for not breast fed exclusively n=55

Reason	Frequency	Percentage
Work out side	40	72.72
Insufficient milk	10	18.18
Sucking difficulties	5	9.09
Total	**55**	

Table - 4 shows reasons for not giving exclusive breast feeding.

Table 5 : Distribution by time of supplementary feeding

Time in month	Frequency	Percentage
5 months	42	38.18
6 months	60	54.54
>6 months	8	7.27
Total	**110**	

Table - 5 shows that 60 (54.54%) respondents initiated supplementary feeding at 5 -6 months of age and 8 (7.27%) initiated more than 6 months of age.

Table 6 : Pattern of breast feeding during working hours n=36

Pattern of breast feeding	Frequency	Percentage
Gave expressed breast milk	22	61.11
Went home to breast fed	08	22.22
Carried their babies to the working place	06	16.6
Total	**36**	

Table 7 : Problem faced by respondents for breast feeding

Type of problem	Frequency	Percentage
Inadequate maternal leave	50	47.2
Lack of child care facility	29	26.35
Lack of nursing break at work place	14	13.20
Lack of privacy at work place	13	12.26
Total	**106**	**100%**

Table -7 shows that the majority 50 (47.2%) faced problem to breast fed their infants due to inadequate maternal leave.

Figure 3 : Distribution of respondents by getting support from working place for breast feeding.

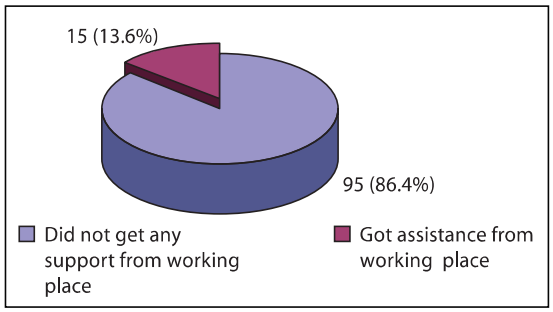

Fig - 3 shows only 15 (13.6%) got assistance from their work place for caring their infants.

Discussion

Majority of the respondents had initiated breastfeeding their infants after birth but unfortunately the number of incidence decreased as the age of the infants increased. In this study, regarding breastfeeding, only few mother continued up to 5 to 6 months The study finding had similarity to the survey report done in Dhaka City[7] where 100% of the mothers had breastfed their babies at birth but the incidence significantly declined at 6 months in all socio economic status and further declined to 14% and 45% at ten months in urban affluent and urban middle class respectively.

Regarding exclusive breastfeeding only small number of respondents practices exclusive breastfeeding up 5 to 6 months. These findings are also similarity with other studies conducted in Bangladesh.[8] The National nutritional survey in Mexico in 1999 found that only 25.4% mother has exclusive breastfed their infants <4 months and 20.3% of mothers fed their infants >6 months of age.[9] It was observed that more than fifty percent of the respondents gave breast milk to their infants during working hours in the form of (EBM) Expressed Breast Milk, one fifth mothers went home to fed and only a few carried their babies to the working place. A large number of respondents did not provide breast milk during working hours.[10]

Among the respondents those who were not giving exclusive breast milk or discontinued earlier were due to working outside and very few due to sucking difficulties, insufficient breast milk, and similar findings were also found in a study,[11] where exclusive breast feeding irrespective of the baby's age was practiced by only 19.5% of women. The reasons for introducing artificial feeding were maternal cause in 49%, child cause in 36.5% and social cause in 13.8%.

Regarding pre-lacteal feeding water, glucose or sugar water, honey and artificial milk were used. In a study done by UNICEF among 760 Bangladeshi mothers in 1989, revealed that 100% mothers gave pre-lacteal food to their newborn consisted of honey, sugar water, formula milk etc.[9]

The present study had shown that majority of the respondents initiated complementary feeding at the age of 6 months of the infants, a study done in South Africa, found that solid food was introduced early at two to three months and mixed family diet at 7 to 9 months. The conclusion of that study was inadequate nutrition knowledge and adherence to cultural practices led to poor quality feeding practices.[11] Another study had found that educational level had a direct relationship with the introduction of semi-solid before 6 months of age.[12] The mean duration of exclusive breast feeding was 3.8 months. It was observed that (86.4%) of the mothers did not get any support or assistant from their work place and only (13.6%) of the respondents got support for infant feeding from their work place.

The percentage of exclusive breast feeding irrespective of months was high among those who got assistance from their offices and it was also found statistically significant ($p< 05$).

Majority of the respondents did not fed exclusive breast feeding to their infants due to inadequate maternal leave and inadequate child care facility in the work places.

Data from FDA's infant feeding practice study showed that among 712 surveyed women, 54% of the women who returned to full time work within 3 months after giving birth had stopped breast feeding.[13]

In a study done among the employed women at Dhaka it was found that 20% of the women were aware of the benefits of exclusive breast feeding and continues exclusive breastfeeding till the 1st month of the employment but gradually the percentage decreases in the 2nd month and so on.[10]

Conclusion

The employed mothers did not feed breast milk during working hours. Only a few got supports for breast feeding in their working place. Children are valuable asset and future nation. For successful breast feeding among employed mothers, adequate maternal leave, set-up day care centre in work place, are immediate need to follow the WHO recommendation about successful breast feeding and building a healthy nation in Bangladesh.

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Review article

Hypothyroidism and cardiovascular diseases : A review

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Abstract

Increased or decreased function of thyroid hormone on the heart and vascular system causes cardiovascular derangements. Hypothyroidism is associated with impaired left ventricular (LV) diastolic function and subtle systolic dysfunction and an enhanced risk for atherosclerosis and myocardial infarction. Objective of this review was to make an update of knowledge about hypothyroidism and cardiovascular diseases. A systematic literature search of published articles relating to hypothyroidism and coronary heart disease (CHD) was conducted. Abstract, full-text, experimental studies and review articles that discussed thyroid function and its association with the development of coronary heart disease were included. The literature survey found that overt and subclinical hypothyroidism have profound effects on cardiac risk factors like pro-atherogenic lipids, C-reactive protein, homocystine and insulin resistance. These changes lead to the development of atherosclerosis, ischemic heart disease and impaired left ventricular function.

Keywords : hypothyroidism, cardiovascular disease

Introduction

Thyroid hormone has physiological effects on the cardiovascular system.[1] Many symptoms and signs recognized in patients with overt hyperthyroidism and hypothyroidism are due to the increased or reduced action of thyroid hormones on the heart and the vascular system, respectively.

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Thyroid hormone abnormalities also cause some hemodynamic derangements. In recent decades, it has emerged that subclinical thyroid dysfunction may affect the cardiovascular system, which may increase cardiovascular risk. It is becoming increasingly apparent that acute and chronic cardiovascular disease may alter thyroid hormone metabolism and contribute to cardiovascular impairment.[2] This article will provide a review of the effects of thyroid hormone in the development of coronary heart disease.

Rationale of the review

Ischaemic heart disease or atherosclerotic coronary artery disease has become global health problem of 21st century because of its high prevalence and concomitant increase in risk of morbidity and premature death. Thyroid dysfunction, not only overt thyroid hormone abnormality but even subclinical abnormality of thyroid hormone, is also a strong indicator of risk for atherosclerosis and MI. Many investigators have suggested that abnormal level of thyroid hormone may represent a cardiac risk factor. Therefore the present review was undertaken to find out the effect of hypothyroidism in development of cardiovascular diseases. So, the study will provide us the up-to-date knowledge about hypothyroidism and cardiovascular diseases. The information obtained from this review may help physician in taking decision in clinical practice.

Methods :

A systematic literature search of published articles on the association between thyroid dysfunction and coronary heart disease (CHD) was conducted. Abstract, full-text, experimental studies and review articles that discussed thyroid function and its association with the development of coronary heart disease were included.

Discussion :

Physiological aspect of thyroid hormones :

The mature thyroid gland contains numerous follicles composed of thyroid follicular cells that surround secreted colloid, a proteinaceous fluid that contains large amounts of thyroglobulin, the protein precursor of thyroid hormones.[3] The thyroid hormones are regulated by hypothalamus-anterior pituitary-thyroid gland axis through a negative feedback mechanism. Hypothalamus secrets thyrotropin releasing hormone (TRH) which stimulates thyrotrope cells of the anterior pituitary gland to produce thyroid stimulating hormone (TSH). TSH stimulates thyroid hormone synthesis and secretion. Thyroid hormones feedback negatively to inhibit TRH and TSH production. So the serum level of TSH is a sensitive and specific marker of thyroid function.[3] Under the stimulation of TSH, the thyroid cells trap iodide to join with tyrosine molecules of thyroglobulin to make mostly T_4 and some T_3 which are stored in follicular colloid within the gland. They are then released together, or some T_4 is further deiodinated to T_3 before release. This step is also under the influence of TSH.[4] T_4 is secreted from the thyroid gland in at least 20-fold excess over T_3. Both hormones circulate bound to plasma proteins. Only the free hormone is biologically available to tissues. About 80% of T_4 is metabolized by deiodination, 35% to T_3 and 45% to reverse T_3 (rT_3). The remainder is inactivated mostly by glucuronidation in the liver and secretion into bile, or to a lesser extent by sulfonation and deiodination in the liver or kidney.[5]

Triiodothyronine (T_3), the biologically active thyroid hormone, enters into the cardiomyocyte through specific transport proteins located within the cell membrane.[6] Once in the cardiomyocyte, T_3 enters the nucleus, binds to thyroid hormones receptors (TRs) and interacts with accessory transcription factors.[1,3,7] This complex binds with specific transcriptional activators (nuclear receptor α-1) or repressors (nuclear receptor α-2) depending on the nature of the regulatory elements in the target gene that, in turn, by acting as cis- or trans-regulators, modify the rate of transcription of specific target genes.[1,3,7] These specific target genes encode both structural and functional proteins.[1,8] Among various proteins expressed by transcription, the most-extensively characterized proteins are myosin heavy chains and the sarcoplasmic reticulum protein involved in the regulation of intracellular calcium handling, namely, calcium activated ATPase and its inhibitory cofactor, phospholamban.[1,8,-11]

The acute effects of thyroid dysfunction on the cardiovascular system are more readily detectable, however, the evidence on the long term effects of thyroid dysfunction on the heart and on the cardiovascular outcomes is less clear. For example, a 20-yr follow-up study of the original Whickham Survey[12] found no association between initial hypothyroidism, raised serum TSH levels, or antithyroid antibodies and the development of coronary artery disease.[12] However, the more recent Rotterdam Study[13] concluded that patients with subclinical hypothyroidism have a significantly increased prevalence of aortic atherosclerosis and myocardial infarctions.[13]

Hypothyroidism :

In the present review 'Euthyroidism' was defined as a normal TSH concentration (0.45-4.50 mU/L), 'Subclinical hypothyroidism' was defined as a TSH concentration of more than 4.50 mU/L and less than 20 mU/L with a normal FT_4 concentration and 'Overt hypothyroidism' was defined as a TSH level of 20 mU/L or more or a TSH concentration of more than 4.50 mU/L with an FT_4 concentration level below normal (<0.7 ng/dL).[14]

Hypothyroidism is a common endocrine disorder resulting from deficiency of thyroid hormone. It is often the primary process in which the thyroid gland produces insufficient amounts of thyroid hormone. It can also be secondary, i.e., lack of thyroid hormone secretion due to the failure of either adequate thyrotropin (TSH) secretion from the pituitary gland or thyrotropin-releasing hormone (TRH) from the hypothalamus (secondary or tertiary hypothyroidism) found that the prevalence of hypothyroidism, diagnosed by history and blood analysis, was 2%.[12,15] The mean age of diagnosis was 57 years, and the disease was ten-fold more common in women than in men. The disease is particularly prevalent in women older than 40 years of age. Hypothyroidism is prevalent in debilitated geriatric patients of both sexes.[15] Subclinical hypothyroidism is common in the adult population, especially among women above 60 years of age.[16,17] Up to two thirds of patients have serum TSH between 5–10 mU/L and thyroid autoantibodies.[16,17] Almost half of these individuals may progress to overt thyroid failure.[12,18]

Hypothyroidism and the cardiovascular system :

The clinical presentation of overt hypothyroidism is not obvious and most patients have few symptoms and signs.[19] Bradycardia and systemic hypertension, with narrow pulse pressure and slightly increased mean arterial pressure, and some degree of exercise impairment

are the most-common findings in patients with overt hypothyroidism.[19-21]

Hypothyroidism and arrhythmia

Many patients with overt hypothyroidism have abnormal standard ECG, including QT interval lengthening and flattening or inversion of the T wave, which reflects the prolonged cardiac action potential.[19,22,23] In addition, overt hypothyroid patients are more prone to develop ventricular arrhythmias, particularly in the presence of an underlying ischemic heart disease, due to increased electrical dispersion in the myocardium[19,22] In general, resting heart rate and blood pressure are normal in SCH subjects.[24]

Hypothyroidism and dyslipidaemia

Elevated levels of total cholesterol, LDL cholesterol, and apolipoprotein B are well documented features of overt hypothyroidism.[25]

Early studies in humans with hypothyroidism demonstrated a prolonged half-life of LDL cholesterol because of decreased catabolism, an effect that was reversible with T_4 therapy.[26] Studies have also shown that hypothyroidism causes qualitative changes in circulating lipoproteins that increase their atherogenicity. Two studies have shown that LDL is more susceptible to oxidation in patients with hypothyroidism, with normalisation after restoration of the euthyroid state.[27,28] In patients with subclinical hypothyroidism, the serum concentrations of total cholesterol, non-HDL-C, remnant-like particle cholesterol, and Apo B were significantly decreased, whereas no significant changes in the serum concentrations of low-density lipoprotein cholesterol, HDL-C, triglycerides, apolipoprotein A-I, and Lp(a) were observed. Additional potentially atherogenic effects of hypothyroidism on lipid metabolism include a reversible reduction in clearance of chylomicron remnants reduced activity of cholesteryl ester transfer protein, which is involved in reverse cholesterol transport pathway and decreased activity of hepatic lipase and lipoprotein lipase.[29,30-32] Some, but not all, cross-sectional studies have demonstrated that serum levels of total cholesterol and LDL cholesterol are higher in patients with SCH than in euthyroid controls. Danese et al[33] in their meta-analysis of the effect of therapy for subclinical hypothyroidism on serum lipid levels demonstrated a mean reduction in the total cholesterol level of 0.2 mmol/L and in the LDL cholesterol level of 0.26 mmol/L.[33]

Hypothyroidism and homocysteine

Several studies have demonstrated elevated homocysteine levels in hypothyroidism, with improvement after T_4 replacement.[28,34-38] This is likely to be caused by impaired renal homocysteine clearance, although an effect of thyroid hormone on enzymes involved in folate metabolism has also been proposed.[28,38,39] The magnitude of decline in homocysteine levels after T_4 treatment is sufficient to lower cardiovascular risk, with a decrease of 2-5 µmol/L when hypothyroid patients were treated with T4 to a level suppressing the serum TSH concentration.[37,39] One study of patients with spontaneous hypothyroidism showed a decrease of 4.6 µmol/L on restoring the euthyroid state.[28] In contrast, there are now considerable data showing that subclinical hypothyroidism is not associated with hyperhomocysteinaemia. Three case-control studies have reported no difference in homocysteine levels between individuals with subclinical hypothyroidism and euthyroid controls. Furthermore, Christ-Crain et al[36] found no significant change in homocysteine levels after treatment of subclinical hypothyroidism.[36,40,41]

Hypothyroidism and C-reactive protein (CRP),

C-reactive protein (CRP), another cardiovascular risk factor, has also been studied in relation to hypothyroidism. Christ-Crain et al.[36] measured CRP in 61 overtly hypothyroid and 63 subclinically hypothyroid patients and compared them with 40 euthyroid control subjects. CRP levels were significantly higher in both hypothyroid groups, compared with controls. However, CRP levels did not decrease with T_4 treatment of the subclinically hypothyroid patients.

Hypothyroidism and insulin resistance

Bakker et al.[42] postulated that relatively lower thyroid hormone levels might amplify the increased cardiovascular risk associated with insulin resistance.[42] Their study confirmed that insulin resistant subjects with high normal TSH levels had higher LDL cholesterol concentrations, whereas among insulin-sensitive individuals, TSH concentration was not associated with any difference in LDL level.

Hypothyroidism and atherosclerosis

An autopsy finding of diffuse atherosclerosis in a 58-yr old woman was published to William Ord's classical description of the syndrome of myxoedema.[43] Vanhaelst et al.[44] found a greater prevalence and severity of coronary atherosclerosis in the hypothyroid patients.[44] Steinberg[45] in 1968 found that women with myxoedema had more severe coronary artery disease on autopsy than did age matched women without myxoedema.[45] The association between hypothyroidism and atherosclerosis has also been shown in living patients. A study of patients undergoing coronary angiography demonstrated that those who had inadequate therapy for hypothyroidism were more likely to have angiographic progression of coronary artery disease than those with adequate replacement.[46] In a hospital-based study, men and women

with a TSH level of 4.0 mU/L or greater had higher prevalence of coronary artery disease than age matched controls (48% vs. 38% for men and 37% vs. 20% for women), although this was statistically significant only for women.[47] Conflicting data exist regarding the effect of hypothyroidism on coagulation.

Both increased and decreased platelet adhesiveness have been reported in hypothyroidism.[48,49] The degree of hypothyroidism may determine its ultimate effects on coagulation parameters.[50] These suggest a greater risk for thrombosis, which could precipitate myocardial infarction, in moderate hypothyroidism, and a bleeding tendency in severe hypothyroidism.[51] Whether SCH is an independent risk factor for cardiovascular disease is controversial.[14] Recently, a strong association between SCH and atherosclerotic cardiovascular disease, independent of the traditional risk factors (i.e., hypercholesterolemia, hypertension, smoking, diabetes mellitus), was noted in a large cross-sectional survey of postmenopausal women (the Rotterdam Study).[13]

Hypothyroidism and hypertension

The prevalence of systemic hypertension is nearly three-fold higher in patients with overt hyperthyroidism than in euthyroid subjects.[52,53] Two factors contribute to systemic hypertension in overt hypothyroidism. The first, and certainly the most-widely recognized, is the remarkable increase in peripheral vascular resistance.[19] The second, and more recently documented, is the increase in arterial stiffness, which likely results from myxedema of the arterial wall.[54,55] In general, systemic hypertension associated with overt hypothyroidism is poorly controlled by conventional treatments, whereas it promptly improves with achievement of euthyroidism.[54] This finding would encourage the routine assessment of thyroid function in all patients with preexisting systemic hypertension that becomes resistant to pharmacological treatment.[56] Significant hypofunctional abnormalities in the parasympathetic nervous system and an increased prevalence of systemic hypertension have been reported in patients with SCH.[21]

Hypothyroidism and LV function

The most-consistent cardiac abnormality recognized in patients with overt hypothyroidism is impairment of LV diastolic function, which is characterized by slowed myocardial relaxation and impaired early ventricular filling.[57,58] LV systolic function usually is only marginally subnormal, as demonstrated by slightly reduced values of ejection fraction and stroke volume filling.[57,58] On the one hand, the reduced cardiac preload, in combination with bradycardia and slightly depressed myocardial contractility, accounts for a subnormal cardiac output in overt hypothyroidism.[57,58] On the other hand, the lower cardiac performance and the abnormalities in peripheral and proximal vascular function may contribute to the poor exercise tolerance in overt hypothyroidism.[20]

Conclusion

The present review revealed that though there is controversy, overt and subclinical hypothyroidism have profound effects on non-traditional cardiac risk factors like pro-atherogenic lipids, C-reactive protein, homocystine and insulin resistance. These changes, along with traditional risk factors, lead to the development of atherosclerosis, ischemic heart disease and impaired left ventricular function.

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Review Article

Virulence and Laboratory Diagnosis of Enteropathogenic Esch. coli

Shimu Saha[1]

Abstract

Enteropathogenic Escherichia coli (EPEC) is a major cause of infantile diarrhoea among children in developing countries. The determination of virulence factors are needed to identify the EPEC strains. Timely identification of EPEC strains remains an urgent need if care and treatment of paediatric diarrhoeal patients are to be given early enough effectively. This review article summarizes the role of virulence factor to produce infantile diarrhoea and also attempts to provide some diagnostic method for rapid and reliable detection of EPEC is required for successful microbiological surveillance and for treatment of EPEC mediated diarrhoeal disease.

Key Word : Enteropathogenic Esch. coli (EPEC), infantile diarrhoea, virulence factors

Introduction

Diarrhoeal illness is a major public health problem worldwide, with over 2 million deaths occurring each year, particularly among infants younger than 5 years. Enteropathogenic Esch. coli (EPEC) is one of the most common causes of infantile diarrhoea.[1]

EPEC is a virulent strain of Esch. coli that causes disease in human being. It was the first strain of Esch. coli to be associated with diarrhoea, isolated from an outbreak in a pediatric nursery in London (UK) by John Bray and colleagues in 1945. The term 'enteropathogenic Esch. coli was coined in a publication by Neter et al in 1955 to indicate Esch. coli linked to childhood diarrhoea. EPEC targets our small intestine following ingestion of contaminated food or drink and causes severe watery diarrhoea, particularly among infants in developing nations. EPEC is one of the most prevalent causes of diarrhoea in infants, particularly those under 2 years old, and is responsible for up to 30% of diarrhoeal cases in developing countries.[2]

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Virulence mechanism of EPEC

When ingested, EPEC strains can colonise the intestine and cause attaching and effacing (A/E) lesions characterized by localized destruction of brush border microvilli, intimate attachment of the bacterium to the cell membrane and formation of an underlying pedestal-like structure of polymerized actin in the host cell[3] (Fig-1). The A/E lesion begins with the presence of adhesin, bundle-forming pilus (BFP). EPEC strains carry genes that encode the bundle forming pilus (BFP), a very important virulence factor that aids in the first step of host cell attachment. The BFP also enables the bacteria to form dense 'microcolonies', which are three dimensional clusters of bacteria that are often used as a diagnostic feature to identify typical EPEC strains. The reason for microcolony formation is not entirely clear. The Donnenberg Lab has extensively studied the BFP and revealed it to be a highly dynamic multi-protein structure. It has one of the most powerful molecular motors known in the living world.[4]

Following initial attachment, EPEC uses a sophisticated molecular machine called a type three secretion system (T3SS) to physically inject effector proteins directly into the cells of its host. EPEC injects over 20 known effector proteins into its host cells. Unlike invasive pathogens such as Salmonella and Shigella, EPEC does not enter host cells but usually remains extracellular and therefore relies on its effector proteins to do most of the work during infection. The collective activities of the effector proteins lead to diarrhoea, which in the case of EPEC are probably a combination of destruction of intestinal microvilli (fig-2),

intestinal tight junctions and an inhibition of water reabsorption.[5]

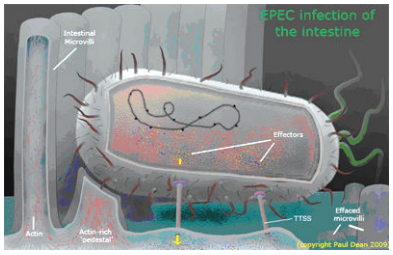

Fig-1 : EPEC infections in intestine.

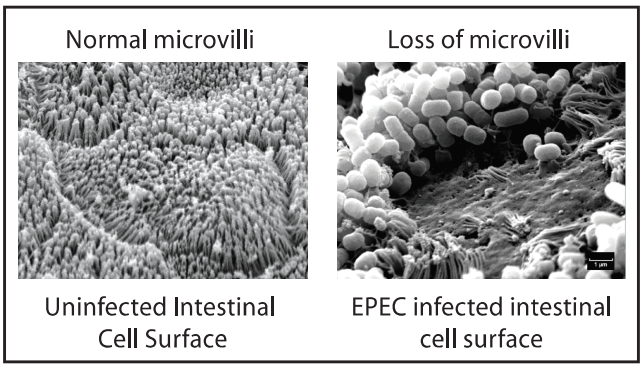

Fig-2: Intestinal microvillus.

Mechanisms of EPEC-Mediated Diarrhoea (Fig-3)

In volunteer studies, the incubation period between EPEC ingestion and the onset of diarrhea is less than 4 hours, suggesting that a more active secretory response may be involved. Interestingly, reports have shown that EPEC can actively alter ion transport, causing a rapid but transient increase in short circuit current in intestinal epithelial cell monolayers mounted in Ussing chambers, with chloride ion secretion. It should be noted that not all studies have supported a role for chloride ion secretion. Finally, other host factors beyond those present in epithelial cell cultures may also contribute to diarrhoea. There is substantial recruitment of neutrophils and other PMNs to the site of in vivo infection. The inflammatory response may be attributable to bacterial triggered signals from infected cells because EPEC activates both NF-kB and interleukin-8 expression in tissue culture cells . These signals are associated with transmigration of PMNs through epithelial cell monolayer. Increased paracellular permeability and stimulation of chloride secretion could be a consequence of this EPEC-induced PMN infiltration.[6] EPEC strains are said to be "moderately-invasive" meaning they are not as invasive as Shigella, and unlike ETEC (Entero toxigenic E coli) or EAggEC (entero aggregative E. Coli), they cause an inflammatory response. The diarrhoea and other symptoms of EPEC infections probably are caused by bacterial invasion of host cells and interference with normal cellular signal transduction, rather than by production of toxins.[1]

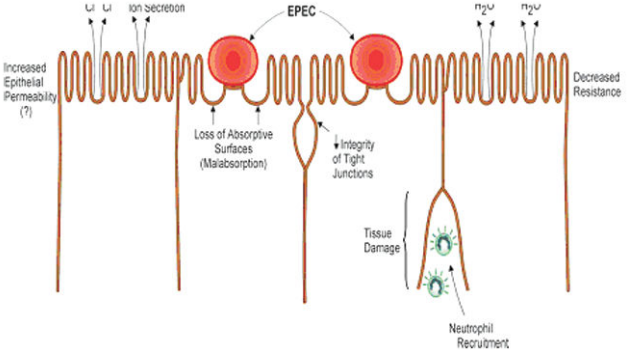

Fig-3 : Putative mechanisms underlying EPEC induced diarrhea include increased epithelial permeability and alterations in Cl- and HCO3- ion secretion. Contributing structural changes include loss of absorptive surfaces, reduced tight junction integrity, and tissue damage.

Three-stage model of EPEC pathogenesis

Multiple steps are involved in producing the characteristic A/E histopathology. In1992, Donnenberg and Kaper proposed a three stage model of EPEC pathogenesis consisting of

(i) localized adherence,
(ii) signal transduction, and
(iii) intimate adherence.

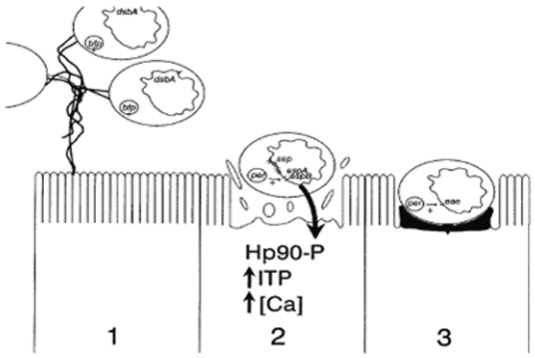

Fig-4 : Three-stage model of EPEC pathogenesis:

(i) Localized adherence (LA) (Fig-5)

Baldini et al10 showed that the ability of EPEC strain (O127:H6) to adhere in a localized pattern was dependent on the presence of a 60-MDa plasmid. Loss of this plasmid led to loss of the LA phenotype. Interactions between EPEC and host cells entail several distinct steps and have classically been viewed as a three-stage process. The first stage in EPEC pathogenesis involves the initial adherence of the bacterium to the host's intestinal epithelium. In this stage, EPEC form dense microcolonies on the surface of tissue culture cells in a pattern known as localized adherence. The bacterium is thought to initially attach to the host cell through a plasmid-encoded bundle forming pilus (BFP), called EAF plasmid. EPEC strains, which do not carry the EAF plasmid; that possessed the gene for intimin

and do not therefore express bfpA. As expected, this strain does not show LA on cultured cells. EPEC have been classified according to whether they do (class I) or do not (class II) possess an EAF plasmid, although they are more commonly termed typical and atypical EPEC, respectively.[9] As defined above, typical EPEC strains would possess both bfpA gene and eae gene and the EAFplasmid and atypical EPEC strain would possess the eae gene only without the EAF plasmid.[5]

(ii) Signal transduction

Adherence of EPEC to epithelial cells induces a variety of signal transduction pathways in the eukaryotic cell. The bacterial genes responsible for this signal transduction activity are encoded on a 35-kb pathogenicity island called the locus of enterocyte effacement (LEE), which encodes a type-III secretion system, multiple secreted proteins, and a bacterial adhesin called intimin.[7]

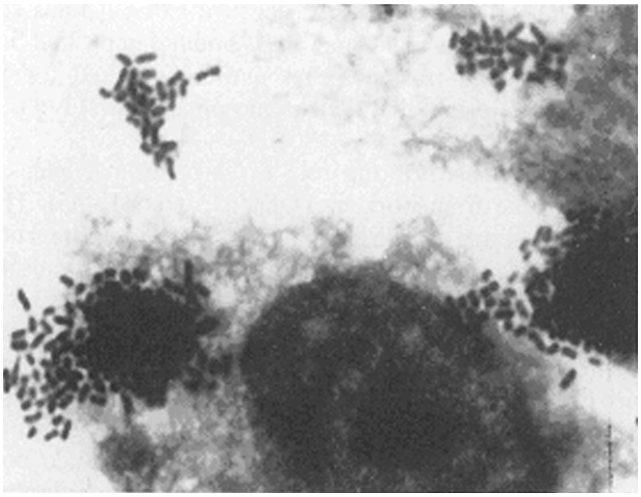

Fig-5 : Light micrograph of Giemsa-stained HeLa cells showing the localized adherence of EPEC

(iii) Intimate adherence

Intimate adherence of EPEC to epithelial cells is mediated by a 94- to 97-kDa outer membrane protein called intimin. The gene encoding intimin (eae, for Esch. coli attaching and effacing) was first reported.[8] The eae gene is present in all EPEC, EHEC, strains; the role of intimin in human disease was demonstrated by studies in volunteers, who ingested an isogenic eae.

EAF plasmids

The BFP is encoded on plasmids which range in size from 60 to 70 MDa, called the EAF plasmids. These plasmids share extensive homology among various EPEC strains.[9]

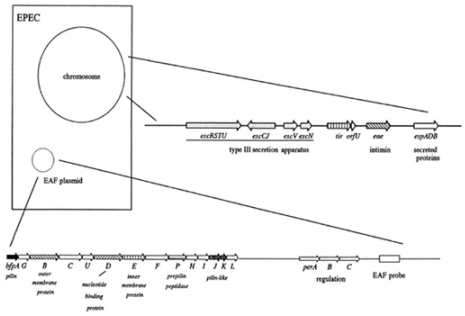

Fig-6 : Genes involved in EPEC pathogenesis. Genes involved in the pathogenesis of EPEC-induced diarrhea are presented in schematic fashion. Chromosomal virulence genes are clustered within the LEE (for locus of enterocyte effacement), which encodes a type III secretory apparatus as well as intimin and a cluster of secreted effector proteins. The EAF plasmid encodes the BFP as well as a cluster of genes required for normal expression of BFP and intimin.[5,7]

Epidemiology Of Epec

Age distribution

The most notable feature of the epidemiology of disease due to EPEC is the striking age distribution seen in persons infected with this pathogen. EPEC infection is primarily a disease of infants younger than 2 years. Numerous case-control studies have shown a strong correlation of isolation of EPEC from infants with diarrhea compared to healthy infants.[10] The correlation is strongest with infants younger than 6 months. In children older than 2 years, EPEC can be isolated from healthy and sick individuals, but a statistically significant correlation with disease is usually not found. However, several outbreaks of diarrhea due to EPEC have been reported in healthy adults[11] presumably due to ingestion of a large inoculum from a common source. Sporadic disease has also been seen in some adults with compromising factors (diabetics, those with achlorhydria, the elderly).[12].

Transmission and reservoirs

As with other diarrheagenic Esch. coli strains, transmission of EPEC is feco-oral. Contaminated hands, contaminated weaning foods/formula or contaminated fomites are serving as vehicles. Unless strict decontamination procedures are followed, admission of an infected infant to a pediatric ward can result in contamination of crib linen, toys, tabletops, hand towels, scales, carriages, rubber nipples, etc. The reservoir of EPEC infection is thought to be symptomatic or asymptomatic children and asymptomatic adult carriers, including mothers and persons who handle infant.[10]

Microbiological diagnosis of EPEC Infection

Isolation and identification

Although assays to identify all categories of diarrhoeagenic Esch. coli are available, in many situations it is not necessary to implicate a specific Esch. coli

pathogen in a particular patient. Culturing stools for most categories of diarrheagenic Esch. coli should be performed in cases of persistent diarrhoea, especially in travelers, children and the immune-compromised, as well as in outbreak situations.

Esch. coli can be isolated from the stool and sent to a qualified reference laboratory for definitive identification.

Serotyping

Diagnosis of EPEC infection needs serogrouping. But serogroupings of Esch. coli used for differentiating diarrhoeagenic Esch. coli are costly, time-consuming and poorly correlates with presence of virulence factors.[13] Determination of virulence factors is thus essential for diagnosis of EPEC strains. Moreover, these antisera are not easily available and serogrouping by 'O' antigens are not sufficient to identify EPEC strains. Because a single organism can contain different 'O' antigens and it can cross react with other organisms.[14]

EPEC strains, as with other diarrhoeagenic Esch. coli strains, are defined on the basis of virulence properties, there are two approaches to the detection of EPEC in the laboratory: phenotypic and genotypic.

(i) Phenotypic test

i) Cell cultures
ii) Fluorescence microscopy,
iii) The FAS test
iv) ELISA

An excellent phenotypic marker for the presence of the EAF plasmid is localized adherence on HEp-2 or HeLa cells. An ELISA for the detection of EAF-positive EPEC, based on an antiserum raised against an EAF plasmid.[15] The FAS test is highly specific for EPEC.[15]

(ii) Genotypic tests

i) DNA probes and
ii) Gene detection method by PCR - bfpA and eae gene.

These methods have been developed and evaluated for the three major characteristics of EPEC: A/E, EAF plasmid, and lack of Shiga toxin. A PCR method has been developed for the detection of the bfpA genes of EPEC that showed no amplification of DNA from any other bacterial enteropathogens and was 100% specific for EPEC strains which exhibited the characteristic LA phenotype.[16]

Plasmid DNA detection

Many important bacterial genes are not part of the main chromosome but are on separate circles of DNA called plasmid. Many virulence gene of diarrhoeagenic Esch. coli are plasmid encoded, such as bfpA gene of EPEC is encoded on plasmids which ranges in size from 60-70 MDa, called EAF plasmid.

Therapy of EPEC infection

The mainstay of EPEC infection therapy is similar to therapy of other diarrhoeal diseases in that fluid replacement and dehydration control are of prime importance (Boedeker, 1988). A controlled trial in Ethiopia has shown a beneficial response to antibiotics, which brought about complete resolution of diarrhoea in 76% of cases within 3 days. Other studies have documented a beneficial response to antibiotics particularly in cases of prolonged EPEC infection.[17] In one report of three infants, 7 to 8 months old, with a 3- to 4-week history of diarrhea that had become life-threatening, a 5-days course of antibiotics resulted in resolution of the diarrhoea.[18] In some areas of the world, antibiotic resistance among EPEC is very common[19] and susceptibility testing must be performed before the institution of therapy. Enteral feeding with infant formulas may result in worsening of diarrhea, even after EPEC eradication with antibiotics, as the damaged mucosa may require time to fully recover its functions of digestion, absorption, and defense. Nutritional support through intravenous alimentation may be needed to allow this recovery of the damaged mucosa to take place.[20] Breast-feeding has a protective effect and should be continued during the illness. At present, there is no vaccine available to prevent EPEC infection. Now that knowledge of the virulence attributes of these organisms are known, it may be possible to design vaccines to control this infection.

Discussion

EPEC virulence is highly complex, and there is much yet to be learned concerning the mechanisms that take place during infection. Identification of (EPEC) is difficult for most clinical laboratories, due to lack of distinct phenotypic differences with non pathogenic Esch. coli strains which are present in stool as normal flora.[21] But the diagnosis and characterization of EPEC is important, as it is one of the important causes of infantile diarrhoea which needs antimicrobial treatment.[22] Reported EPEC from Bangladesh were 5.85% (2008)[23], 6.4% were by (2010)[7] and 8.2% (2012).[24] Traditionally, diarrhoeagenic Esch. coli strains from stools are identified with the help of microscopic examination, serotyping and conventional culture methods.[23] Polyvalent and monovalent antisera for serogroupings of diarrhoeagenic Esch. coli are very expensive in comparison to the costs needed for gene detection by PCR method. The determination of virulence factors are needed to identify the EPEC strains.[25] The main virulence characteristics of EPEC strains depend on bfpA gene, 60-70 MDa plasmid DNA and characterization of HeLa cell adherence assay. Plasmid DNA and HeLa cell adherence assay are cumbersome and time consuming. Though the diagnosis of EPEC infections based on tissue culture assay is the best method, but the facility of this

investigations are not easily available. On the other hand diagnosis of EPEC strains by gene detection method gives more accurate results and it is less time consuming than other methods.[26] Therefore gene detection by PCR can be an effective method for diagnosis of EPEC infection.

Conclusion

EPEC pathogenesis is a complex mechanism involving a variety of different structures subject to complex environmental and genetic regulation. A great deal has been learned in the last 15 years concerning the virulence of EPEC. In particular, the use of genetic techniques has significantly improved our understanding of EPEC pathogenesis. Continuation of this work will lead to a better understanding of EPEC and how these organisms cause disease, which is hoped will result in better control and eventually eradication of this infection.

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Case Report

Post-cholecystectomy syndrome (PCS)

Abu Ahmed Ashraf Ali[1], Mahamud Riyad[2], Sadia Armin Khan[3]

Abstract

Post-cholecystectomy syndrome (PCS) is a common manifestation in patients with cholecystectomy. Patient may present with upper abdominal pain, vomiting, gastrointestinal disorders, jaundice, and dyspepsia. Choledocholithiasis, biliary dyskinesia, and dilation of cystic duct remnants are common causes of these symptoms. The symptoms can recur after a symptom-free period following cholecystectomy or they can persist after surgery. Ultrasonography & computed tomography (CT) are the two most common investigations along with magnetic resonance imaging scan have a high sensitivity in detecting the causes of PCS. We report a case of a 17-year-old girl who came to the General Surgery Department with recurrent episodes of abdominal pain following cholecystectomy. The MRCP showed a dilated cystic structure arising from common bile duct resembling a gall bladder whereas X-ray abdomen confirmed presence of clip of previous laparoscopic cholecystectomy.

Keywords : Biliary dyskinesia, choledocholithiasis, cystic duct remnant, post-cholecystectomy syndrome.

Introduction

Cholecystectomy is the most common method of choice for treating symptomatic gall stones. It can either be performed laparoscopically or by open cholecystectomy though laparoscopic cholecystectomy is the gold standard. Continuation of symptoms that patient experienced before cholecystectomy, or new symptoms like epigastric pain, vomiting, dyspepsia mimicking cholecystitis after cholecystectomy is called Post Cholecystectomy Syndrome (PCS).

About 10 to 15% of the patients with cholecystectomy experience PCS. The main symptoms of PCS include upper abdominal pain and dyspepsia. Ninety percent of the time, the etiology of PCS is identifiable, whereas recognized common causes are long cystic duct, choledocholithiasis, stone at cystic duct remnants & biliary dyskinesia.

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Case Report

17 years old girl presented to general surgery out-patient department with recurrent right upper abdominal pain for five years, which was colicky in nature, was radiating to back and right shoulder, associated with vomiting and was aggravating after fatty food. Patient was admitted several times at different hospitals in last five years with the same complaints. Few occasions she was diagnosed as acute pancreatitis and treated conservatively. She was not anorexic. Her bowel and bladder habit was normal. She didn't loss weight in this time interval. She was neither diabetic, nor asthmatic and didn't suffer from jaundice. Patient had laparoscopic cholecystectomy 7 years back at her age of ten for cholelithiasis.

On examination, she was healthy with average body build. She was neither anemic, nor icteric. Dehydration, edema & clubbing were absent. Her pulse was 80 bpm, BP 110/80 mm of Hg. Examination of abdomen showed healed scars of previous laparoscopic cholecystectomy. Right upper abdomen was slightly tender on deep palpation but Murphy's sign was negative.

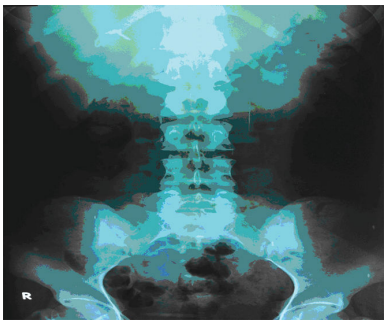
All laboratory results were within normal limits :

CBC - TC 7000, Neutrophil 56%, Hb 15gm/dl;

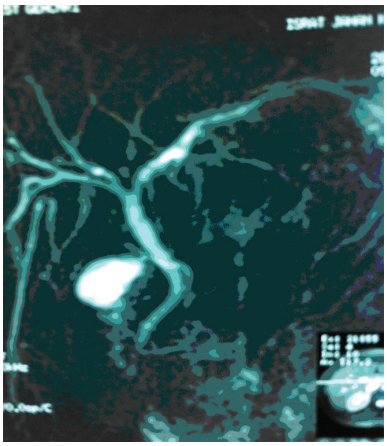
LFT - S. bilirubin 8gm/dl, SGPT 35 IU/L, Alkaline phosphatase 135 IU/L; Serum amylase 40 U/L; Serum lipase 68 U/L.

On imaging-

USG couldn't identify any pathology but reported as absent gall bladder.



X-ray abdomen showed three clips of previous laparoscopic surgery.



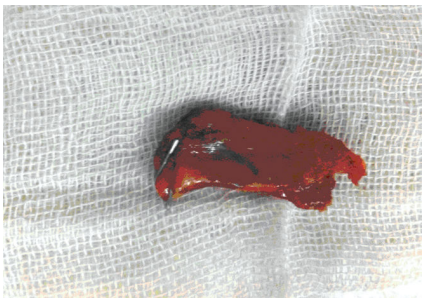
MRCP revealed a gall bladder like structure which was connected to common bile duct with a narrow duct.

Patient underwent laparotomy & findings were-

1) Adhesions around gall bladder fossa

2) Long cystic duct with dilation of terminal part, two clip attached on it.

Cystic duct was tied and the remnant excised as conventional open cholecystectomy. Post-operative recovery was smooth and uneventful in last 6 months of follow-up.



Discussion

Laparoscopic cholecystectomy is an established operation for symptomatic gall stone disease. However about 5% of patients may experience episodes of upper abdominal pain similar to those that they had prior to cholecystectomy.[1] These symptoms may be due to biliary stricture, retained / recurrent biliary calculi, stenosis or dyskinesia of sphincter of Oddi, cystic neuroma, remnant gall bladder / cystic duct stump calculi etc. and are together grouped as post cholecystectomy syndrome.

Long cystic duct remnant defined as residual duct greater than 1 cm in length, may cause post-cholecystectomy syndrome.[2] MRCP emerges as the optimal method for evaluating the biliary tree in these cases.[2,3] It has now been suggested that it is safe and feasible to remove the gall bladder or gall bladder remnants in such patients laparoscopically.[4,5]

Conclusion

Proper dissection and identification of gallbladder - cystic duct junction is paramount for complete removal of the GB and to prevent recurrent symptoms. If USG failed to diagnose the condition, MRCP is the best options available. Patients with recurrent symptoms and proven stones should be re-operated and laparoscopic surgery is no more a contra-indication for these revision surgeries.

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Case Report

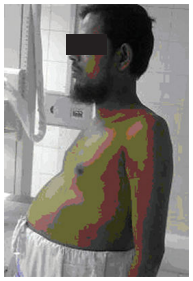
Radiological diagnosis of constrictive pericarditis - a rare disease in Bangladesh

A. H. Abedur Reza[1], Abeeda Tasnim Reza[2], Md. Tanvir Rahman[3]

Abstract

A 35-years old man reported in radiology & imaging department of National Heart Foundation Hospital & Research Institute with palpitation, gradual dyspnoea on exertion and generalized body swelling. The patient had a history of tuberculosis & treated with anti-tubercular drugs for 06 months, one & half years back. His X-ray chest, ECG, Ultrasonogram, Echocardiogram, CT scan of chest & Cardiac CT angiogram were performed and the case was diagnosed as Constrictive pericarditis. A postero-anterior & lateral chest X-ray demonstrated severe, dense calcification of the pericardium. Constrictive pericarditis is a rare disease in Bangladesh. The objective of this report is to discuss the etiology, the patho-physiological features, clinical findings & diagnostic tools of this condition.

Keywords : Constrictive pericarditis, right heart failure & pericardium.



Introduction

Constrictive pericarditis (CP) is a reduction in the elasticity, or stiffening, of the pericardium, a sack-like covering that surrounds the heart, resulting in impaired filling of the heart with blood. Constrictive pericarditis, designated as concretio cordis dates back more than 300 years. The symptoms of Constrictive pericarditis may include exercise intolerance, liver failure, dyspnoea, and renal failure, which appear insidiously and may be misleading.[1,2] In many cases, constrictive pericarditis is late sequelae of an inflammatory condition of the pericardium. The inflammatory condition is usually an infection that involves the pericardium, but it may also occur after a heart attack or after heart surgery.[3]

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Almost half the cases of constrictive pericarditis in the developing world are idiopathic in origin. In regions where tuberculosis is common like Bangladesh, it is the cause in a large portion of cases.[4]

Patients may present with increasing weight gain, cardiac cirrhosis, and massive ascites.[5-7] But the diagnosis is rarely considered by the referring physician. Although rare but it has to be kept in mind as one of the differentials and investigations should be done accordingly.

Patients respond dramatically to a complete surgical pericardiectomy when it is performed early in the disease process; therefore, it is important to consider CP when making the diagnosis. Anatomic imaging findings, such as calcifications (see the images below) and thickening of the pericardium may be present, but the most reliable and most important findings are related to the filling pattern of the heart. Radiological investigations specially x-rays & CT angiogram are playing important roles in the diagnosis of constrictive pericarditis.[8]

Rare forms of constrictive pericarditis: In addition to classical chronic (rigid shell, calcific) & sub-acute (elastic) forms, new presentations such as effusive-constrictive, localized, transient, occult & constrictive pericarditis with normal pericardial thickness, have been described.[9]

Pathophysiology

In the classical form of constrictive pericarditis, the rigid, heavily fibrosed or even calcified pericardium causes

restriction of the myocardium, preventing adequate ventricular filling after an initial expansion.[2, 5-11] Due to decreased pericardial compliance & increased venous pressure, the early diastolic filling occurs very rapidly and stops when the intracardiac volume & pressure reach their maximum limits. As myocardium is not affected, relaxation of the left ventricle is usually normal. Elevated diastolic pressure in all four chambers is due to the rigid pericardium. Consequently, the atrial waves on the jugular veins show a prominent & deep diastolic Y descent (Friedreich sign - diastolic collapse of cervical veins due to adherent pericardium). The hallmarks of constrictive pericarditis are the hemodynamic changes observed during respiration. Increase in the right heart volume, causing higher pressure on the inter-ventricular septum. Subsequently, the septum moves to the left & the left-side volume decreases. Kussmaul's sign is positive (raised JVP on inspiration). Pulse is usually low volume with tachycardia. Pulsus paradoxus may be present - disappearance of peripheral pulse during inspiration. This interesting phenomenon is explained by impaired filling of the left ventricle with an enhanced decrease in the systolic pressure during inspiration caused either by fibrosed & calcified pericardium or by the accumulation of pericardial fluid. It has been found that constricted pericardium are not able to increase the flow velocity in either the vena cava or the pulmonary artery during inspiration, a finding that may be the mechanism for the appearance of pulsus paradoxus.[12] Pooling of blood in the pulmonary bed & filling competition of the ventricles in the presence of a fixed pericardial sac may serve as an additional cause for appearance of pulsus paradoxus. These dynamic changes throughout the respiratory cycle can be used to differentiate constrictive pericarditis from restrictive cardiomyopathy.

Case Report

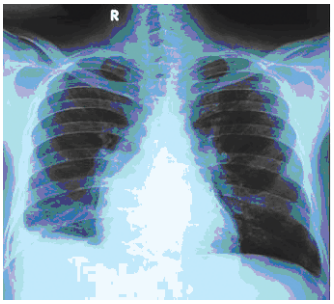
A 35-years old man reported in radiology & imaging department of National Heart Foundation Hospital & Research Institute with Palpitation, gradual dyspnoea on exertion and generalized swelling of the body. The patient had a history of tuberculosis & treated with anti-tubular drugs for 06 months, one & half years back.

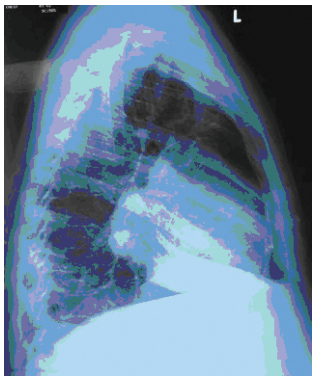
His X-ray chest, ECG, Ultrasonogram, Echocardiogram, CT scan of chest & Cardiac CT angiogram were performed. A postero-anterior & lateral chest X-ray demonstrated severe, dense calcification of the pericardium. ECG findings showed ischemia, atrial premature beats with evidence of atrial fibrillation. Ultrasonogram of abdomen showed hepatomegaly with evidence of hepatic venous congestion with ascites. Findings of echocardiogram were consistent with constrictive pericarditis. The pericardium was severely & diffusely calcified on CT scan (as seen also in 3D view).

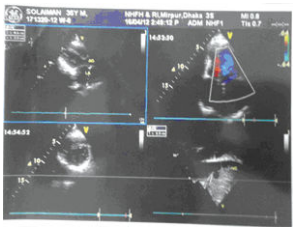
Investigations

(a) Chest X-ray :- Both the P/A & lateral views demonstrate severe, dense, calcification of the pericardium. Calcifications are mostly seen at the left heart border, anterior, inferior as well as diaphragmatic surfaces. Cardiac silhouette is not enlarged. There is also evidence of small pleural effusion on the right side.

(b) Electrocardiography : - This patient shows characteristic ECG changes for constrictive pericarditis. There is presence of tachycardia with low QRS voltage, ischemia, atrial extra systole with fibrillation.



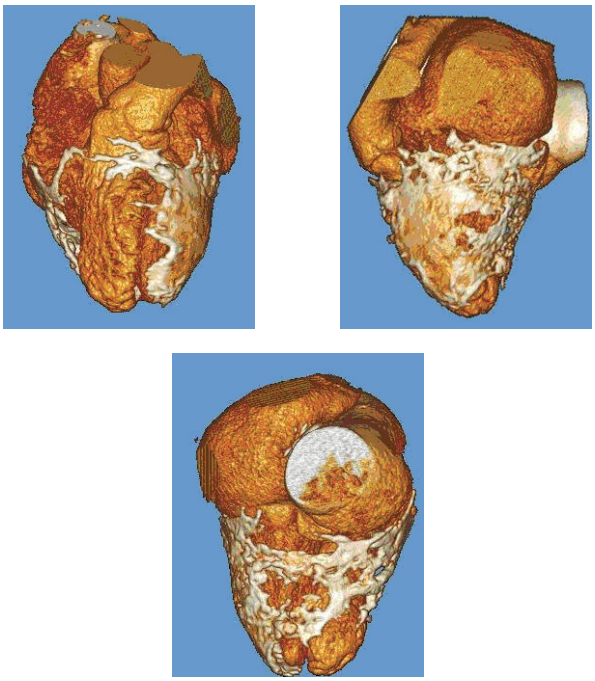




(c) Echocardiography : - In two dimensional Echocardiogram, parietal & visceral pericardium are adherent particularly at the left lateral & inferior wall of the heart with speckled calcification causing poor echo-window. There is an unusual motion of the

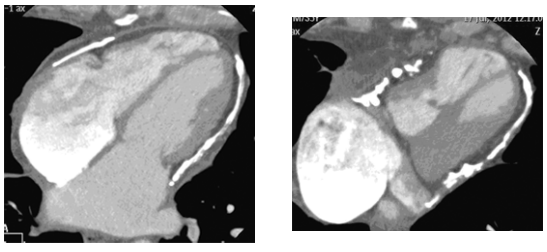
interventricular septum, designated as septal bounce, accompanied by an inspiratory septal shift to the left. The presence of a dilated, non-collapsing inferior vena cava is suggestive of constrictive pericarditis. On the other hand, a normal sized collapsing inferior vena cava at inspiration almost rules out heart constriction.

The hallmark of constrictive pericarditis on Doppler echocardiography is the respiratory flow variations through the heart valves, expressed as a decreased flow through the left side valves during inspiration & its increase with expiration, while on the right side valves the reverse phenomenon occur.



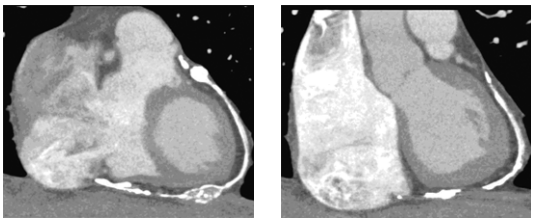
(d) CT angiogram : - This method allows direct visualization of the pericardium as well as heart. Pericardium is thickened (04 mm) with evidence of hyper-dense areas at the left lateral, anterior and inferior as well as diaphragmatic surfaces.

3D view shows calcified layer of the pericardium which looks like a cemented layer causing constriction of the heart.

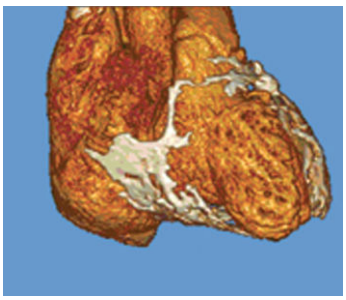


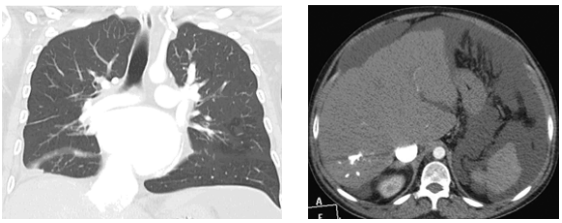
CT scan axial view showing the calcified pericardium

Four chambered heart is seen in axial views. There is evidence of increased concentration of the dye seen in the right side of the heart. Right atrium is enlarged in size. Both the ventricles are appearing tubular with evidence of pressure effects from outside (due to calcification) causing diastolic ventricular dysfunction secondary to constrictive pericarditis.



CT scan coronal views showing the calcified pericardium





Others

There is evidence of small pleural effusion on the right side. There is also huge ascites in the abdomen.

Discussion

The diagnosis of constrictive pericarditis is difficult because of its common clinical signs with many other possible diagnoses and the rarity of this condition. Constrictive pericarditis should be considered in patients with signs of right heart failure.[38]

Patients with constrictive pericarditis frequently present with symptoms of heart failure, such as dyspnoea, orthopnoea, and fatigability, and occasionally may present with liver enlargement and ascites. The causes of constrictive pericarditis have changed over time; at present, the most frequent causes are cardiac surgery and

radiation therapy.[21] Other causes include infection (viral or tuberculous), connective-tissue disease, uremia, neoplasm, or idiopathic condition.[22] Clinically, it is difficult to differentiate between constrictive pericarditis and restrictive cardiomyopathy. These two entities are characterized by similar clinical manifestations and similar findings at cardiac catheterization and echocardiography. In both conditions, ventricular filling is restricted, leading to an increase in diastolic pressure in all four cardiac chambers and to equalization of atrial and ventricular pressure. It is important, however, to distinguish between constrictive pericarditis and restrictive cardiomyopathy, because patients with constrictive pericarditis might benefit from pericardial stripping, whereas those with restrictive disease would not.

Traditionally, increased pericardial thickness has been considered a specific diagnostic feature of constrictive pericarditis, but these days there is also a subset of patients with hemodynamic signs of constrictive pericarditis and normal thickness of the pericardium.[39] Several criteria on invasive pressure tracings have been examined for the diagnosis of constrictive pericarditis. Among these, the change in the ventricular pressure curves during the respiratory cycle (reflecting ventricular interdependence) is unique to patients with constrictive pericarditis.[40]

Imaging is essential in arriving at the correct diagnosis.[41-43] Many imaging modalities offer potential choices for making the correct diagnosis. Plain radiography is helpful, but notoriously insensitive for demonstrating pericardial calcifications. Other plain radiographic evidence of constrictive pericarditis may be seen including cardiac enlargement, left atrial enlargement, and pulmonary vascular congestion.[44]

Echocardiographic findings indicate wall motion abnormalities and cardiac morphologic features consistent with constrictive pericarditis, and may demonstrate pericardial calcifications and thickening as well. Although transthoracic echocardiography is routinely performed for the evaluation of myocardial function in patients with symptoms of constrictive or restrictive physiologic change, it is not highly accurate in the depiction of pericardial thickening.[4] Transesophageal imaging allows better visualization of the pericardium, and respiration-correlated Doppler techniques are particularly useful in the diagnosis of constrictive pericarditis.[23] However, the transesophageal approach is limited by a narrow field of view and is relatively invasive.

The diagnosis of constrictive pericarditis is greatly aided by the excellent depiction of the pericardium at CT and MR imaging. Normal pericardial thickness is less than 2 mm.[8,9] Pericardial thickness of 4 mm or more indicates abnormal thickening and, when it is accompanied by clinical findings of heart failure, is highly suggestive of constrictive pericarditis.

MR imaging has a reported accuracy of 93% for differentiation between constrictive pericarditis and restrictive cardiomyopathy on the basis of depiction of thickened pericardium (4 mm).[24] Pericardial thickening may be limited to the right side of the heart or to an even smaller area, such as the right atrioventricular groove[25]. But MRI has difficulty in demonstrating calcification. Other findings seen on MRI and present in this case include septal bounce, enlarged atria, and small cone-shaped ventricles.[45,46]

An additional advantage of CT is its high sensitivity in depicting pericardial calcification, which is also associated with constrictive pericarditis. CT scan exquisitely demonstrate pericardial calcifications, but gated imaging is necessary to prove constriction, and the temporal resolution of CT is inferior to other dynamic modalities for imaging the heart, including MRI and echocardiography.[46,47] It is important to remember, however, that neither pericardial thickening nor calcification is diagnostic of constrictive pericarditis unless the patient also has symptoms of physiologic constriction or restriction.

At both CT and MR imaging, the central cardiovascular structures may show a characteristic morphology in constrictive pericarditis. The right ventricle tends to have a reduced volume and a narrow tubular configuration. In some patients, a sigmoid-shaped ventricular septum or prominent leftward convexity in the septum can be observed.[25] In the setting of dense calcifications or in the presence of normal pericardial thickness, however, evaluation of the pericardium can be difficult.[42,49,50] Systemic venous dilatation (particularly in the inferior vena cava), hepatomegaly, and ascites also are frequently seen.

No single approach should be used to diagnose all cases of constrictive pericarditis. The diagnostic approach taken should be individualized for each patient. In some patients, the diagnosis may be made on the basis of the history, physical examination, and chest radiograph. In other patients, echocardiography, visualization of the pericardium, and cardiac catheterization all may be

required. The most important diagnostic tool is the clinical suspicion of constrictive pericarditis in a patient with signs and symptoms of right sided heart failure that are disproportionate to pulmonary or left sided heart disease. Understanding the pathophysiology of this disease and using non-invasive and invasive techniques are helpful in diagnosis, particularly in the patient who has myocardial and pericardial disease. The differential diagnosis is right atrial myxoma, tricuspid valve dysfunction & restrictive cardiomyopathy, nephrotic syndrome, obstruction of the superior vena cava, hepatic diseases & abdominal malignancies.

In the chronic stage of tuberculous constrictive pericarditis, pericardial decortication with wide resection of both the visceral and the parietal pericardium, remains the definitive treatment.[51] However, there are no clear-cut determinants for surgical intervention when managed at the early stage. Yang et al. once reported that decision largely depended on the clinical symptoms of cardiac temponade, progression of heart failure, and constriction that lead to jugular vein engorgement.[52] They highlighted the importance of pursuing early pericardiectomy, rather than pericardiocentesis and window placement, to achieve sustained relief of symptoms in patients with advanced stage disease.

Conclusion

It is very important that the sooner the diagnosis is established the better the outcome is. Constrictive pericarditis is caused by fibrosis and calcification of the pericardium, processes that inhibit diastolic filling of the heart. This condition has posed a diagnostic dilemma since it was first recognized clinically. Because surgical intervention can provide complete relief of symptoms in many patients, accurate diagnosis of this disorder is important.[3] By the use of diagnostic tools such as X-ray Chest, Echocardiography, CT scan for heart visualization are important & may save patient's anguish & complication. With the advent of CT Angiogram the diagnosis of the constrictive pericarditis have become very easy, especially 3D views.

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Case Report

Alleged sexual assault

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Abstract

Child sexual abuse includes any activity with a child, before the age of legal consent or puberty, that is for the sexual gratification of an adult or a significantly older child. Sexual mistreatment of child by family members (Incest) and nonrelatives known to the child is the most common type of child sexual abuse. Intra-familiar sexual abuse is difficult to document & manage, because the child must be protected from additional abuse and coercion not to reveal or to deny the abuse, while attempts are made to preserve the family unit. The alleged victim child was a minor girl about 2 years of age. She was sexually abused by non-familial person. She was first admitted for treatment at Dhaka Medical College & then for medico-legal examination at Sir Salimullah Medical College.

Key words : Alleged, sexual assault

Introduction

Child sexual abuse has been defined by the American Academy of Pediatrics as the engaging of a child in sexual activities that the child cannot comprehend, for which the child is developmentally unprepared and cannot give informed, and violate the social taboos. In general, children cannot give consent to any sexual activity, but the legal age of consent may vary by state. Sexual activities involving a child may include activities intended for sexual stimulation, such as those involved in contact sexual abuse (eg, touching the child's genitalia or the child touching an adult's genitalia), penetrating injury (eg, penile, digital, and object insertion into the vagina, mouth, or anus), and non-penetrating injury (eg, fondling, sexual kissing).

The American Psychiatric Association states that "children cannot consent to sexual activity with adults," and condemns any such action by an adult: An adult who engages in sexual activity with a child is performing a criminal and immoral act which never can be considered normal or socially acceptable behavior.

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Case History

Profile of the victim
Name : .....
Age : 22 Months
Father's Name : .....

Address

- Present : House no- ..., Side road no: ... Shahid Nagar, Lalbag, Dhaka.
- Permanent: Chitulia, Kalipur, Shariatpur.
  - Lalbag Police Station Case No: 10, Date: 07-11-2010.
  - Nari o Shishu Nirhjaton Domon Ain (2003)-Shongshodhoni -9(1)/30
  - Memo no- SSMC: 140/2010, Date-08-11- 2010.

History of the case
Admission history at Dhaka Medical College Hospital
Hospital information

1. Name : .....
2. Age : 22 Months
3. Reg. no : 49067/127, PSU-1
4. Bed no : 6B, W- 35A
5. Date of admission : 05-11-2010.
6. Time : 10:45 am

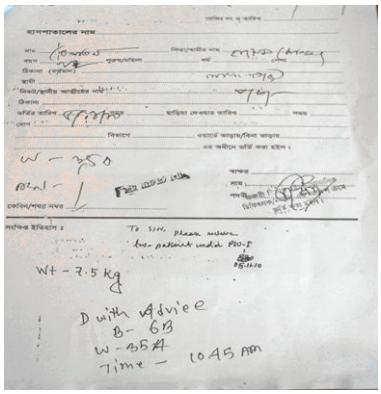


Figure : Admission certificate

C/C-

- Per vaginal bleeding for 2 ½ hours
- H/O trauma / accident 2 ½ hours back

History of present illness

According to the statement of the patient's attendance (mother), the patient was alright 2 ½ hours back. Then she developed P/V bleeding, possibly following a trauma or accident while playing 2 ½ hours back.

On general examination

Pulse :125/min, BP : 90/60 mm of Hg, Temp : 37°C

Multiple bruise all over the body.

On local examination

- Evidence of localized injury to genitalia
- Bleeding coming from posterior fourchette,
- Vulva edematous,
- Linear abrasion at both labia minora about 1 ½ cm and abrasion at posterior fourchette about 1 cm.

Treatment history

- Local management : (05-11-2010)
- Gauge pack was given in situ.
- Pressure bandage was given.
- Catheterization was done.

Drug management

- Inf. Baby saline.
- Inj. Ceftrioxone (500mg) i/v daily.
- Inj. Traxyl (250mg) ½ amp. i/v 8hourly
- Transfused 1/3 unit of Blood.
- Caprolysin soaked gauge pack in situ.

Details on discharge certificate

- The patient was discharged with advice on 07-11-2010 at 10:40 am.
- The patient was admitted with complaints of per vaginal bleeding for 2½ hours prior to admission following unexplained insult to vaginal structure. On examination patient was found to be anaemic and pulse 125/min and having multiple bruise over face and body. Local examination revealed evidence of localized injury around vaginal orifice. Bleeding coming from posterior fourchette and edematous vulva. Linear abrasion at both labia minora about 1 ½ cm and abrasion at posterior fourchette about 1cm.

Vaginal pack with pressure bandage given.

- Follow up- Bleeding controlled.

Treatment on discharge

- Syrp. Cef-3 (200mg/1ml)-½ TSF 2 times daily.
- Syrp. V. Plex. ½ TSF daily.

Advice on discharge

- Take the medicine regularly.
- If any difficulty arise report to OPD at PSU on Saturday .
- For follow up treatment report to OCC.

Medico-legal history at Department of Forensic Medicine, SSMC : (Date-08-11-2010)

According to the statement of the victim's mother, she (herself) has been doing household works in different houses of her locality. She (victim's mother) has seven children, of which the victim baby girl is the youngest of all about two years old. While she used to go for works, she generally keeps her child (victim) with another girl aged about 10/12 years, who lives near to her house. On 05-11-2010 about 10:30 am that young girl took away the victim with her to another place and returned home in the evening on the same day.

After their return, the mother of the victim observed that profuse blood was coming through the vagina of the victim. The victim's mother asked the young girl about the reason of bleeding through vagina. The girl explained that the bleeding was occurred due to sudden slipped and fall down on the ground, while the victim was playing.

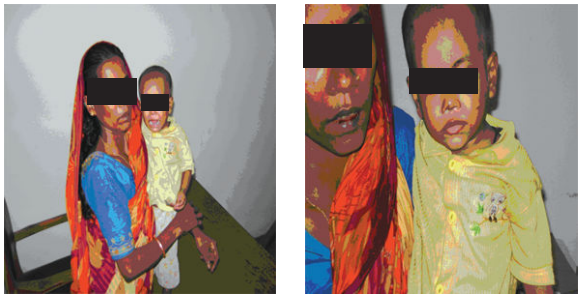


Figure : The alleged victim

On general examination
Multiple scratch mark were found over the face, back of the forearm & thigh.

On local examination
The baby was very much apathetic.

A small linear scratch mark was present in the posterior fourchette,

Another linear scratch mark found in labia minora. No vulval edema or bleeding was found.

** Vaginal swab was taken for detection of spermatozoa- Report of vaginal swab revealed that no spermatozoa are seen. (F-95)

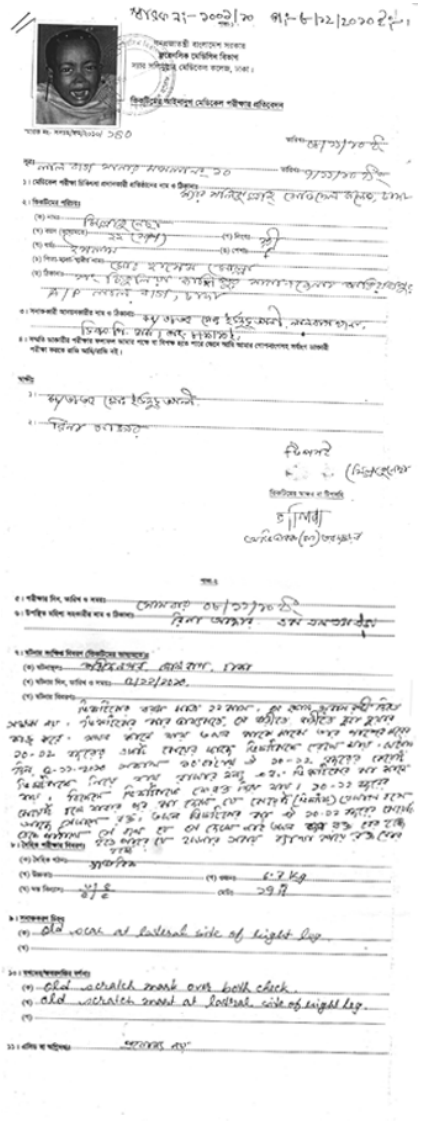
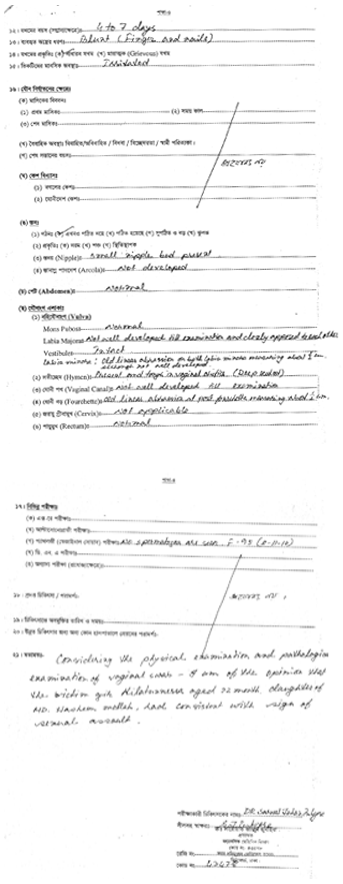


Figure : Medico-legal report at SSMC



Scene visit (Paedriatic surgery unit-1 of Dhaka Medical College hospital):

Statement of Assistant Registrar of PSU-1 of DMCH
The assistant registrar (CA) stated that when he received the baby girl, she was very much anaemic, her pulse was so rapid it seemed that the patient was going to shock at any moment. Meanwhile patient's mother refused to hospitalize her in casualty/ Gynae & Obs ward. To save the life of the baby and for beneficent, he started conservative treatment with I/V saline & antibiotics, managed her local bleeding by caprolysin soaked gauge and pressure bandage. Bleeding was controlled.

Statement of Associate Professor of PSU-1 of DMCH
The Associate Professor of PSU-1 stated that after admission in her ward she examined the baby thoroughly by herself and found the bleeding was stopped. They did not handle anything for local injury and transfused 1/3 unit of fresh blood.

Opinion regarding medico-legal report
(Forensic Medicine Department of SSMC)
Considering physical examination and pathological examination of vaginal swab, my opinion "victim girl named ..... aged 22 months, daughter of Md. ..... is consistent with sign of sexual assault.

Risk factors of child sexual abuse

Parent abused as a child: Most perpetrators are not strangers but are known to the child (e.g., stepfathers, uncles, mother's paramour). Female perpetrators are reported less often. Parents who have been abused do not always abuse their own children, but the risk for continued familial abuse is present.

- Multiple caretakers for the child
- Caretaker or parent who has multiple sexual partners
- Drug and/or alcohol abuse
- Stress associated with poverty
- Social isolation and family secrecy
- Child with poor self-esteem or other vulnerable state
- Other family members (e.g., siblings, cousins) abused Gang member associations.
- Causal factors.

Causal factors of child sex offenders are not known conclusively. The experience of sexual abuse as a child was previously thought to be a strong risk factor, but research does not show a causal relationship, as the vast majority of sexually abused children do not grow up to be adult offenders, nor do the majority of adult offenders report childhood sexual abuse. The US Government Accountability Office concluded, "the existence of a cycle of sexual abuse was not established." Prior to 1996, there was greater belief in the theory of a "cycle of violence," because most of the research done was retrospective—abusers were asked if they had experienced past abuse. Even the majority of studies found that most adult sex offenders said they had not been sexually abused during childhood, but studies varied in terms of their estimates of the percentage of such offenders who had been abused, from 0 to 79 percent. More recent prospective longitudinal research—studying children with documented cases of sexual abuse over time to determine what percentage become adult offenders—has demonstrated that the cycle of violence theory is not an adequate explanation for why people molest children.

Offenses may be facilitated by cognitive distortions of the offender, such as minimization of the abuse, victim blaming, and excuses.

Types of child sexual assault

Child sexual abuse includes a variety of sexual offenses, including:

Sexual assault – a term defining offenses in which an adult touches a minor for the purpose of sexual gratification; for example, rape (including sodomy), and sexual penetration with an object. Most U.S. states include, in their definitions of sexual assault, any penetrative contact of a minor's body, however slight, if the contact is performed for the purpose of sexual gratification.

Sexual exploitation – a term defining offenses in which an adult victimizes a minor for advancement, sexual gratification, or profit; for example, prostituting a child, and creating or trafficking in child pornography.

sexual grooming - defines the social conduct of a potential child sex offender who seeks to make a minor more accepting of their advances, for example in an online chat room.

Effects of child sexual assault

Psychological effects:

Fear. The offender may swear the child to secrecy and say that if they tell something bad will happen. Sexual abuse is usually accompanied by coercion, bribery or threats. The child is afraid to tell because of what the consequences might be e.g., punishment, blame, abandonment or not being believed.

Helplessness/powerlessness. Children in this situation often feel that they have no control over their own lives or even over their own bodies. They feel that they have no choices available to them.

Guilt and Shame. The child knows something is wrong and blames him or herself not others. The offender will often encourage the child to feel that the abuse is his or her fault and sometimes s/he will feel that s/he is a "bad" person.

Responsibility. The offender often makes the child feel responsible for keeping the abuse a secret. Sometimes the child also feels responsible for keeping the family together and the burden of this responsibility interferes with experiencing a normal childhood.

Isolation. Incest victims feel different from other children. They must usually be secretive. This even isolates them from non-offending parents and brothers and sisters.

Betrayal. Children feel betrayed because they are dependent upon adults for nurturing and protection and the offender is someone who they should be able to love and trust. They may also feel betrayed by a non-offending parent who they feel has failed to protect them.

Anger. Not surprisingly this is one of the strongest feelings which many children have about their sexual assault. Children may feel anger against the perpetrator and also against others who they feel failed to protect them.

Sadness. Children may feel grief due to a sense of loss, especially if the perpetrator was loved and trusted by the child.

Flashbacks. These can be like nightmares which happen while the child is awake. They are a re-experience of the

sexual assault and the child may experience all the feelings gained which they felt at that time.

In The Long Term The Child May Also Experience A Number Of Effects As An Adult. These May Include:

1. Depression, anxiety, trouble sleeping.
2. Low self esteem.
3. "Damaged goods" syndrome. i.e. negative body image due to self-blame. This may be intensified if physical pain was experienced during the abusive incidents.
4. Dissociation from feeling.
5. Social isolation.
6. Relationship problems such as an inability to trust, poor social skills or reluctance to disclose details about themselves.
7. Self destructive behaviour such as substance abuse or suicide attempts.
8. Sexual difficulties such as fear of sex or intimacy, indiscriminate multiple sex partners or difficulty in reaching orgasm.
9. Parenting problems such as fear of being a bad parent, or fear of abusing the child or being overprotective.
10. An underlying sense of guilt, anger or loss.
11. "Flashbacks" and/or panic attacks.

Discussion

The baby girl was first admitted for treatment at Dhaka Medical College Hospital on 05-11-2010 with the complaints trauma/ accident in the genitalia (No definite cause was mentioned). After 3 days of treatment the baby girl came to Forensic Medicine Department of SSMC as Medico-legal case. There were differences between hospital information & police inquiry.

Possible causes

1. False charge/ history.
2. False diagnosis/ negligence of the hospital.
3. Evil intension of the relatives of victim.
4. Lack of knowledge.
5. Delinquency of police inquiry.

It was made difficult to solve and to draw opinion as medico-legal case. Lack of knowledge or ignorance in Forensic Medicine lead to this situation. It is required to develop basic skill or knowledge about Forensic Medicine to all categories of doctors to overcome it.

Finally, Forensic Medicine Department of SSMC set the opinion on the basis of physical and pathological examinations of the victim.

Conclusion

Sexual assault is one of the first growing violent crime in Bangladesh. It includes rape, physical and mental torture, molestation, sexual harassment (Eve teasing) and prostitution of girl. The real magnitude of the crime is much higher as a large proportion goes unreported. Alleged victims of rape happen to be of different age groups but children, adolescents, and young women constitute the main target group. Major portion of alleged victims are refused to report sexual assault to law enforcing agency because of social stigma, prejudice with regard to the chances of marriage, being considered promiscuous and responsible for incidence, attendant humiliation and shame, embarrassment caused by appearance and cross examination in the court proceedings, publicity in the press, risk of losing love from family and respect of friends and society and that of her husband, if married. The major problem is that most of the victims brought for medico-legal examination after 5-7days after the incidence even more. Rapidly healing injuries can be missed in case with delayed examination, there may be chance of false allegation. Similarly value of pathological examination followed an alleged incident has limited value because most of the cases high vaginal swab for detection of spermatozoa found negative.

Of course special privileges are granted to the rape victim under the prevailing laws and rulings. Since there is no uniformity in the interpretation of the law, she has to bear the risk of a negative verdict. The loopholes in the laws and procedures are cleverly manipulated to save the criminals; in most cases, they manage to come out unscratched. In spite of various amendments to the laws, the decisions of various courts show that either therapists are still being let off on grounds of benefit of the doubt or awarded minimum punishment considering on various grounds.

Finally, we can say the legal dictum that rape or any allegation of sexual assault is-

"Easy to make,
Hard to prove and
Harder to disprove."

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গর্ভকালীন সময়ে
২৪ ঘন্টা আপনার পাশে আমরা

২৪ ঘন্টা এ্যাম্বুলেন্স সেবা

ঢাকা মহানগরীর মধ্যে মাত্র **২৬০** টাকা

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

আদ্-দ্বীন মেডিকেল কলেজ হাসপাতাল
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