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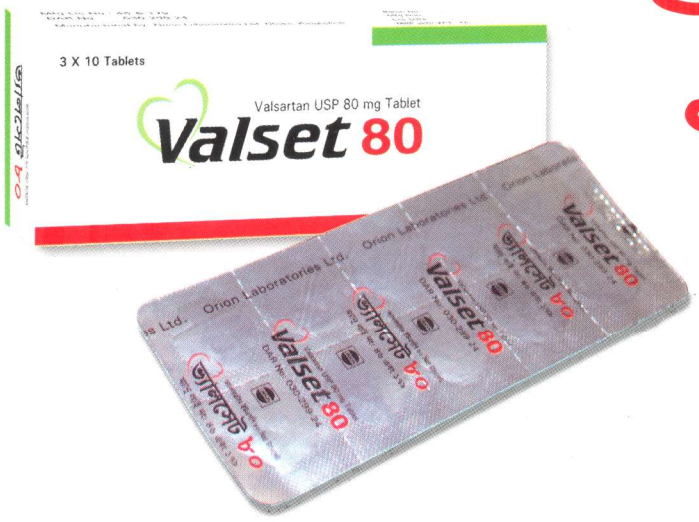

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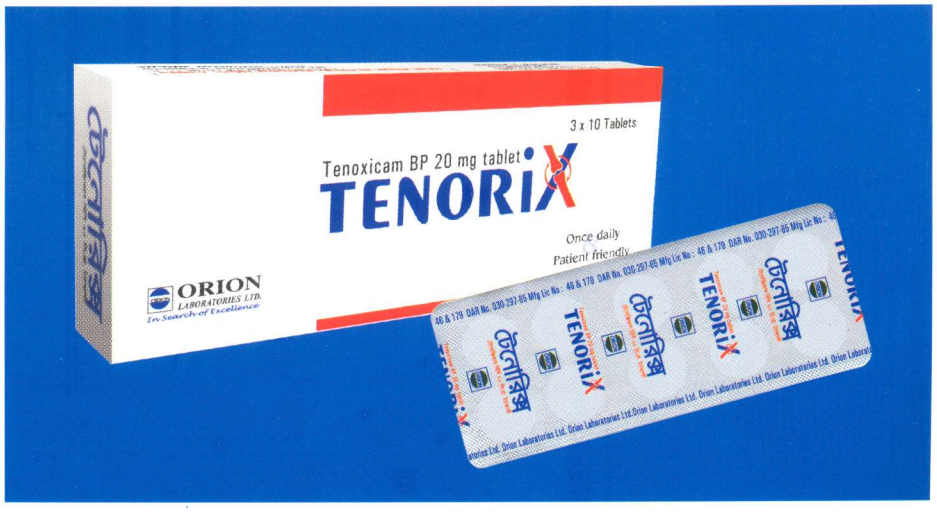

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"The ORION" one of the leading Medical journals of the country which reaches its `11 years landmark' in September, 2009. "The ORION", not only meets the quantity but also satisfies the quality of the publication by peer review, reference input, text index, latest medical and scientific news and information which is more user friendly to the authors, readers, members and subscribers. "The ORION" today is one of the mostly accepted journals in Bangladesh with highest number of publication. This issue (Volume 32, Issue 3) comprises of 04 original articles and 05 review articles.

Editorial (P-674) of this issue "Treatment of enteric fever" denotes the overall condition of enteric fever in Asia including Bangladesh and points out the treatment options in MDR, NART and DCS resistant *S.* Typhi strains and effectiveness of different treatment strategies.

The first original article (P-675) "Echocardiographic evaluation of left ventricular function after taking amlodipine for hypertension and chronic stable angina on 507 rural human subjects" describes the efficacy of amlodipine on left ventricular function on hypertension & IHD and suggests adding low dose amlodipine with a view to improving LV function.

Second original article (P-679) "Role of hyperhomocysteinaemia and folate deficiency in the development of preeclampsia and gestational hypertension" analyzes the relationship of homocysteine with PE and GH and concludes that elevated homocysteine is an important biochemical marker of endothelial damage though with other markers, the causal relationship between homocysteine and the disorders has not yet been established.

Next original article (P-682) "Role of intra uterine balloon catheter in controlling massive PPH: Experience in Rajshahi Medical College Hospital" suggests uterine tamponade using fluid filled condom as the first step for controlling PPH after exploration of the uterus prior to performing laparotomy, because it is quick, safe and effective method & result can be known within minutes.

Last original article of this issue (P-684) "Determination of MIC and MBC of selected tetracycline capsule commercially available in Bangladesh" reveals tetracycline should not be the choice of antibiotic for *pseudomonas spp.* and *E. coli* induced infection due to high MIC level, but *Shigella spp.* found sensitive to tetracycline in this study.

First review article (P-686) "Peripartum cardiomyopathy" reviews peripartum cardiomyopathy as a rare but life threatening form of heart failure affecting women in the last month of pregnancy or the first five months of postpartum without any significant previous heart disease. It concludes, subsequent pregnancy should be discouraged specially those who fail to regain normal left ventricular function.

Second review article (P-690) "Post-streprococcal reactive arthritis : A review" states PSRA is a poorly understood clinical syndrome in which oligo and polyarthritis coccurs following a group A streptococcal pharyngitis. This article also presents the clinical profile of PSRA and comparison with acute rheumatic fever, provides approach to its diagnosis and management.

Third review article (P-692) "Computer vision syndrome" briefs about the new emerging medical and visual problem, characterized by visual symptoms which result from interaction with a computer display or its environment.

Next review article (P-694) "New ideas and concepts- Rice bran : A nutrient-dense mill-waste for human nutrition" highlights on a very new concept of using rice bran for human nutrition which has been used as low level animal and poultry feed. Rice bran is widespread and affordable- this makes a powerful value proposition for bolstering scientific, humanitarian and business opportunities for its supply and use in promoting human health.

Last review article (P-702) "To achieve international standard in the field of interventional cardiology: What should we do" states our curriculum should be developed to find out international standard interventional cardiologist as well as to reduce the cardiac burden of our country.

Further opinion and suggestions are highly encouraged for development of "The ORION". The journal is freely available at www.orion-group.net/journals for contributing towards the advancement of public health and medical research. For reproducing multiple copies of any of articles published in "The ORION", please email:orionjournal@yahoo.com/ msdorion@yahoo.com/ journal@orio-group.net & mention the article title, author's name, volume, page number, year of publication and most important the purpose for reproducing.

May the Almighty bless all in the spirit of good health.

DR. KAZI RAFIQUL ALAM
Chief Editor
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Treatment of enteric fever

Rahman M[1]

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Enteric fever (typhoid and paratyphoid), caused by *Salmonella enterica* serovar Typhi (*S.* Typhi) or serovar Paratyphi A, B, C, represents a major burden of disease in the communities, due to lack of pure water and adequate sanitation. More than 27 million cases of enteric fever occur worldwide in each year, with 216000 deaths.[1] The incidence is highest (100cases/100,000 population per year) in South-central and Southeast Asia, medium in the rest of Asia, Africa, Latin America and some parts of Oceania and low in the rest of the world. It is more common among young children and adolescents (2-15 years) and in urban than rural areas. *S.* Typhi represents the commonest cause of bacteraemia in this age group, and annual typhoid rates (confirmed by blood culture) in recent studies from Bangladesh, India, Pakistan and Indonesia range from 149 to as high as 573 cases per 100000 children.[2] In developed countries, returning travellers or those visiting friends and relatives in their family's country of origin are at risk.[1,2,3] In endemic areas, most patients are treated with oral antibiotics as outpatients, but only those who are severely diseased need hospital admission. Relapse may complicate the illness, and faecal carriage in 1-5% of patients can become chronic and lead to further transmission in the community.

Typhoid fever is the most common febrile illnesses encountered by the physicians in Bangladesh. Proper and timely administration of appropriate antimicrobial therapy prevents complications of enteric fever and reduces cast fatality rate to <1%. The choice of empirical antimicrobial therapy depends on the susceptibility of *S.* Typhi and *S.* Paratyphi strains present in the area of residence or travel. Until 1987, two to three weeks of Chloramphenicol or Amoxicillin or Trimethoprim-sulphamethoxazole was the treatment of choice for enteric fever.[4] Diagnosis and treatment lately became a challenge due to emergence of MDR *S.* Typhi (resistant to Amoxicillin, Chloramphenicol and Trimethoprim-sulphmethoxazole) in 1990 in Bangladesh as an expansion of MDR *S.* Typhi strains from China, Pakistan, India and Nepal.[5,6] In 1989 all *S.* Typhi isolates were susceptible to all first-line antibiotics in Bangladesh. MDR strains emerged in 1990 in Bangladesh and increased to 44% in 1994 from 8% in 1990 and declined to 22% in 1996 and increased again to 42% in 2002[6] & 61% in June, 2009. Fluoroquinolones (Ciprofloxacin and Ofloxacin) became the treatment of choice for typhoid fever with 98% cure rate and <2% faecal carriage, and were simultaneously sold widely over the counter to treat fever of various aetiologies in developing countries including Bangladesh. The extensive pressure of quinolones leads to the selection of single point mutations in the *gyr A* gene of *S.* typhi, causing resistance to first-generation 4-quinolones Nalidixic acid (NART strains, MIC $\geq$32 µg/ml) and decreased Ciprofloxacin or Ofloxacin susceptibility [(DCS strains, MIC = 0.125-1µg/ml) (but formally these isolates are still within the Clinical Laboratory Standard Institute (CLSI) breakpoints for susceptibility] in 8% of *S.* Typhi strains in 2000 in Bangladesh that increased to 71% in 2002[5] & 90% in June, 2009. This resulted in a poor clinical response (prolonged recovery time, therapeutic and microbiologic failures, more relapses and prolonged faecal bacterial shedding period) to treatment with the Ofloxacin or Ciprofloxacin even with increased doses (1-2 g/day). With increase use of Fluoroquinolones such as Ciprofloxacin, Naldixic acid-resistant (MIC $\geq$ 32 µg/ml) *S.* Typhi (NART) isolates with decreased Ciprofloxacin susceptibility (MIC = 0.125-1µg/ml) appeared, have reached high levels in Central, South, and South East Asia.[2,3,6,7] Under this circumstances, the World Health Organisation recommends the Fluoroquinolones or Cefixime for the treatment of MDR typhoid fever and Azithromycin, the third-generation cephalosporins, or a 10-14 day course of high-dose older generation Fluoroquinolones (e.g. Ofloxacin or Ciprofloxacin) for the treatment of Nalidixic acid resistant typhoid fever.[7] Recently, nearly 4% of *S.* Typhi isolates developed complete Ciprofolaxin or Ofloxacin resistance (MIC $\geq$ 4 µg/ml) in Bangladesh due to additional mutations in *gyrA* and *parC* genes which has also been reported from India, Nepal and Kuwait making these drugs less optimum for empirical therapy in spite of recommendation by WHO. Ciprofloxacin is no more a drug for empirical therapy of enteric fever in almost all the countries of the world unless complete Ciprofloxacin susceptibility is proved by very low MIC values (MIC <0.125 µg/ml). Patients infected with NART strains should be treated with Gatifloxacin, Azithromycin or Ceftriaxone. High doses of ciprofloxacin are usually associated with delayed resolution time, complications, treatment failures and prolonged faecal carriage. The laboratory detection of DCS strains is problematic because they are still classified as susceptible by CLSI guidelines. Isolates with DCS are usually resistant to the first generation quinolone, Nalidixic acid, and this is a useful, but not 100% reliable, surrogate laboratory marker for resistance. In some areas of Asia such as India, Nepal, Bangladesh and Kuwait, S. Typhi isolates that are fully resistant to ciprofloxacin (MIC $\geq$ 4 µg/ml) have emerged at the same time as the proportion of MDR infections has remained unchanged or increased or declined. The emergence of fully Ciprofloxacin-resistant *S.* Typhi strains is a worrying development that further limits treatment options. Ceftriaxone, Cefotaxime and oral Cefixime are effective for the treatment of MDR, NART and Ciprofloxacin-resistant *S.* Typhi strains. It is likely that there will be more reports of fully resistant strains in the future from regions of the world where typhoid fever is endemic. Treatment of chronic carrier with antibiotics for 4-6 weeks is effective and surgery is required in failure cases. Dexamethasone (2 days course, 3mg/kg first dose followed by 1mg/kg 6 hourly for 8 doses) with Chloramphenicol reduced mortality rate in patients with severe disease with shock and obtundation.

For enteric fever acquired in Bangladesh and many other countries of Asia, more than 90% of isolates have DCS so Ciprofloxacin and Ofloxacin should be avoided. Azithromycin, Gatifloxacin, or Ceftriaxone can be used as empirical therapy depending on the severity of illnesses. Resistance to Ceftriaxone and Azithromycin is rarely reported, but isolates that are fully resistant to Ciprofloxacin are now being detected and Ceftriaxone is effective for these infections. Combinations of drugs are being used, but evidence for the effectiveness of this strategy is inadequate.

High quality & adequately powered multicentre clinical trials are needed to compare treatment options for enteric fever. Trials should include children, ambulatory patients and severe cases and be completed quickly to encounter rapidly changing resistance patterns. MDR *S.* Paratyphi A is emerging rapidly in India and other Asian countries to add a new health problem and one should keep it in mind when treating enteric fever.[2] Finally, prevention of enteric fever should not be forgotten, nor should the potential use of vaccination in areas where the disease burden is high and drug resistance is common.

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Echocardiographic evaluation of left ventricular function after taking amlodipine for hypertension and chronic stable angina on 507 rural human subjects

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Abstract

This is a prospective, community based, single blind, monocentric clinical study performed in a community based healthcare centre, Chatkhil, Noakhali for the period from June 23, 2006 to September 21, 2008. Patients of hypertension & IHD (chronic stable angina) were selected for study. Age limit 20-80 years, both genders were included; sample size 507. The prime objective is to study the efficacy of amlodipine on left ventricular function on hypertension & ischaemic heart disease. Patients were evaluated clinically & then investigated by X-Ray, ECG, Echocardiography. Amlodipine therapies (5 mg daily) was given with or without other medications followed by echo-evaluation of LV function for 9-12 months LV-EF & LV mass/BSA were measured very accurately. Among 507, male 355 (70.01%) & female 152 (29.98%). 50-59 years age group was affected much (210 cases, 41.42%). Second affected age group is 60-69 years (107 cases, 21.10%). Service holders & businessmen were affected much (135 cases- 26.62%, 134 cases- 26.42%). Normal LV-EF was observed in 95 (18.73%) & 114 cases (23.26%) before & after drug therapy. Besides normal LV mass/BSA was observed in 50 (9.86%) & 67 (13.67%) cases respectively before & after treatment. So it is concluded that amlodipine, a third generation CCB effectively control BP, helps in regression of LV hypertrophy & thus improves LV function (LV-EF).

Key words

Amlodipine, LV function, echocardiography.

Introduction

Calcium (Ca^{++}) is required for contraction of cardiac & smooth muscle, also responsible for propagation of cardiac impulse. Calcium Channel Blockers (CCBs) block the influx of calcium into cells. This relaxes the muscles in the walls of arteries resulting in dilatation.[1] This lowers the blood pressure and improves the blood supply to the heart muscle. All of these effects allow the heart to work with reduced blood supply together with relief of anginal pain.[2]

Calcium Channel Blockers (CCBs) may be divided into benzothiazepine (diltiazem); phenylalkylamine (verapamil); and dihydropyridines (first generation: Nifedipine, nicardipine, felodipine, nisoldipine; second generation: isradipine, nimodipine; third generation: amlodipine, lacidipine etc).

Amlodipine is a third generation CCB with long half-life. It has interaction with specific high affinity binding sites in the calcium channel complex. It maintains therapeutic efficacy throughout 24 hours. It has less negative inotrophic and chronotrophic action having lack of clinically, relevant increase in cardiac or peripheral sympathetic activity. It has higher lipophilicity; reflex tachycardia is minimal, relatively safe in heart failure.[3]

The L-type calcium channel is the dominant type in cardiac & smooth muscle. The calcium channel blockers act from the inner side of the membrane and bind more effectively to channels in depolarizing membranes.

In the cardiac myocyte, Ca^{++} binds to troponin and reduces inhibitory effects of troponin on contraction, favouring muscle contraction. CCBs reduce transmembrane movement of Ca^{++} reduce the amount reaching intracellular sites and therefore reduce vascular smooth muscle tone.

Amlodipine has got minimal or no effect on AV conduction.[4]

CCBs have direct negative inotrophic effects and showed some benefits on hemodynamic parameters alone or in combination with ACE inhibitors. Amlodipine has got potentially beneficial effects on hypertension and coronary artery disease specially stable angina. But amlodipine showed minimal beneficial effects in patients with heart failure which was observed on large, randomized, placebo-controlled trials.[5]

Aims and objectives

The objectives were-

i. To study the efficacy of amlodipine on left ventricular function in hypertension.
ii. To study the efficacy of amlodipine on left ventricular function in ischaemic heart disease (stable angina).
iii. To evaluate any side effects of amlodipine used for HTN & stable angina.
iv. Echocardiographic evaluation of LV functions after amlodipine therapy in HTN & IHD.
v. Finally to provide some new information on amlodipine therapy in this regard.

Materials and methods

It is a prospective, community based, single blind, mono-centric, clinical study performed in a Community Health Care Centre, Chatkhil, Noakhali from 23.06.2006 to 21.09.2008.

A larger geographical area of Noakhali district i.e. Chatkhil, Sonaimuri, Begumgonj and part of Lakhipur and Comilla districts were fairly covered in this study.

All patients attending the OPD were screened. Patients of hypertension & IHD (stable angina) were selected for study. Age limit was 20-80 years; no gender variation; associated heart failure was not a contraindication for inclusion. Consent was taken from all patients or relatives prior entry to study.

After clinical case selection, patients were investigated by X-ray, ECG, Echo, blood glucose and lipid profile. Then amlodipine therapy (5 mg daily) was given with other

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medications followed by echocardiographic evaluation of LV function every 2-3 months interval.

For 2D & M-mode echocardiography we used ALOKA SSD-1100 equipment. Left ventricular function was evaluated by the following parameters: wall thickness, chamber dimension, wall motion abnormality, ejection fraction, stroke index, LV mass/BSA etc.

Some LV parameters

A. Stroke Volume (SV)

SV	Vold- Vols
Vold	End diastolic volume in ml
Vols	End systolic volume in ml
LVEDV	90-140 ml
LVESV	27-85 ml
SV	50-100 ml

B. Stroke Index in ml/m^2 (SI)

S.I.	SV/BSA
SV	Stroke volume in ml
BSA	Body surface area in m^2
Example	SV- 75 ml; BSA- 1.5 m^2

So, S.I. = 75/1.5 = 50 ml/m^2

C. Cardiac Index in L/min/m^2 (CI)

CI	CO/BSA
CO	Cardiac output in L/min
BSA	Body surface area in m^2
Example	CO- 5 L/min; BSA- 1.5 m^2

So, C.I. = 5/1.5 = 3.3 L/min/m^2

D. LV mass- cube (gm) = 1.04 $[(IVS+LVID+PW)^3-(LVID)^3]$ (measurement in cm)

Example	IVS- 1.0 cm; LVID- 4.5 cm
	PW- 1.0 cm)

So, LV mass- cube = 190.84 gm

E. LV mass = 0.80 (LV mass cube) + 0.60 (ASE convention)
American Society of Echocardiography
Example, LV mass cube 190.84 gm
So, LV mass = 153.27 gm

F. LV mass (Penn Convention) = (LV mass cube) - 14

G. LV mass/BSA : 125-150 gm/m^2

H. Ejection fraction in percent (EF)

EF	100 (Vold-Vols) / Vold
Vold	End diastolic volume in cm^3
Vols	End systolic volume in cm^3

Results

Various data obtained from the study were presented below using various tables, figures and graphs.

Table 1: Gender distribution of patients (n=507)

Gender	Number	Percentage
Male	355	70.01
Female	152	29.98

Table 2: Age distribution of patients

Age in years	Number	Percentage
20-29	05	0.98
30-39	45	8.87
40-49	97	19.13
50-59	210	41.42
60-69	107	21.10
70-79	43	8.48

Table 3: Occupation of patients (n=507)

Occupation	Number	Percentage
Farmer	02	0.39
Day labourer	31	6.11
Service holder	135	26.62
Teachers	96	18.93
Fishermen	37	7.29
Beggars	01	0.19
House-wives	59	11.63
Businessmen	134	26.42
Others	12	2.36

Table 4: Disease profile in study subjects (n=507)

Diseas profile	Number	Percentage
HTN	249	49.11
IHD (Stable angina)	162	31.95
HTN + IHD (Co-exist)	96	18.93

Table 5: Gradings of hypertension (n=345)

Gradings	Number	Percentage
Mild	84	24.34
Moderate	157	45.50
Severe	104	30.14

Table 6: Echo-evaluation of LV-EF before treatment (n=507)

LV	Number	Percentage
Normal (60-75%)	95	18.73
Mild dysfunction (50-59%)	102	20.11
Moderate dysfunction (40-49%)	109	21.49
Severe dysfunction (30-39%)	114	22.48
Very severe dysfunction (<30%)	87	17.15

Table 7: Echo-evaluation of LV-EF after treatment (n=490)

LVEF	Number	Percentage
Normal	114	23.26
Mild dysfunction	110	22.44
Moderate dysfunction	90	18.36
Severe dysfunction	99	20.20
Very severe dysfunction	77	15.71
Drop out	17	3.35

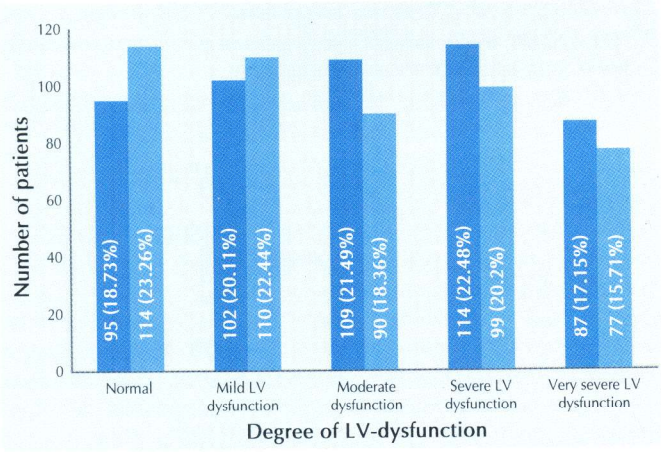

Figure 1: Bar diagram showing LV-EF before & after treatment

Table 8: Follow-up Echo-evaluation of LV mass/BSA before treatment (n=507)

LV mass/BSA (g/m^2)	Number	Percentage
Normal (120-129)	50	9.86
Grade-I hypertrophy (130-139)	107	21.10
Grade-II hypertrophy (140-149)	193	38.06
Grade-III hypertrophy (>150)	157	30.96

Suggested cut-off point for LVH is 125g/BSA

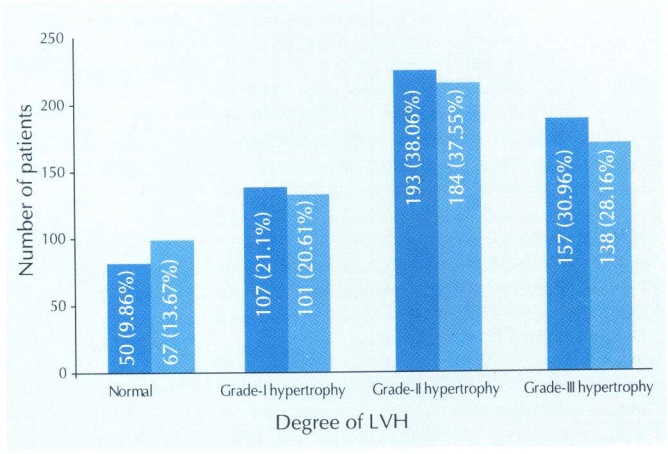

Figure 2: Bar diagram showing LV mass/BSA before & after treatment

Table 9: Echo-evaluation of LV mass/BSA after treatment (n=490)

LV mass/BSA (g/m^2)	Number	Percentage
Normal	67	13.67
Grade-I hypertrophy	101	20.61
Grade-II hypertrophy	184	37.55
Grade-III hypertrophy	138	28.16
Drop out	17	3.35

Discussion

For hypertensive patients, one should select the appropriate medication to control hypertension. Hypertension is one of the most prevelent risk factor for cardiovascular disease, affecting as many as 800 million people world wide.

Amlodipine effectively reduces the cardiovascular risk factors related to hypertension.

Table 1 shows the gender distribution of patients (n=507); male- 355 (70.01%), female- 152 (29.98%). We have observed prevelence of hypertension and coronary artery disease much more in males rather than females; which was supported by Scandanavian research group in 2007.[6]

Table 2 shows age distribution of patients. Maximum affected age group in 50-59 years (210 cases, 41.42%); next common affected group in 60-69 years (107 cases, 21.10%). Malhotra and Co-workers[7] also found the similar incidence in European Community; 50-59 years, 43.42% & 60-69 years age group 23.25% respectively.[7]

Various occupation of patients was showed in table 3. Maximum incidence was observed in service holders (135 cases, 26.62%) & businessmen (134 cases, 26.42%). Other researchers also observed the same phenomena in white, as well as black races.[8]

Table 4 narrated the disease profile in study subjects (n=507) i.e. only hypertension- 249 cases (49.11%), only IHD (stable angina)- 162 cases (31.95%) & associated HTN plus IHD- 96 cases (18.93%).

Gradings of hypertension were done on 345 cases:

a) Mild- 84 cases (24.34%)

b) Moderate- 157 cases (45.50%)

c) Severe- 104 cases (30.14%)

In our community population prevelence of moderate hypertension is higher, which was supported by Kamango, Picarno & other Co-workers in 2006.[9]

Left ventricular function of the study-subjects suffering from hypertension & chronic stable angina (n=507) was evaluated by echocardiography. Though various methods of calculation are available, simplest formula of calculating LV-EF through POMBO method was adopted.

Table 6 & 7 showed the comparative study of echo-evaluation of LV-EF before & after amlodipine treatment. Before treatment

normal LV-EF was observed on 95 cases (18.73%). After treatment it has become 114 cases (23.26%). This rise of 19 cases (4.51%) is due to control of BP resulting from amlodipine therapy. Before treatment severe dysfunction was observed in 114 cases (22.48%); but after treatment it has come down to 99 cases (20.20%). This reduction (2.28%) of LV systolic dysfunction is due to drug therapy. It was observed by Panthorian and Co-workers in 2007 i.e. 3.31%.[10]

Table 8 & 9 showed the comparative study of LV mass/BSA in study subjects just before & after amlodipine therapy. Suggested cut off point for LVH is 125g/BSA.

Table 8 showed different gradings of LV hypertrophy where grade-I: LVH- 107 cases (21.10%), grade-II: LVH- 193 cases (38.06%), grade-III: LVH- 157 cases (30.96%).

Despite drop out of 17 cases (3.35%), the scenario has been changed after control of BP with amlodipine with or without other medications i.e. grade-I: LVH- 101 cases (20.61%), grade-II: LVH- 184 cases (37.55%), grade-III: LVH- 138 cases (28.16%).

Reduction of LV mass/BSA index was observed in the study groups- Quinkibay and Co-worker in 2006,[11] Ducketty & Pathania in 2007[12] and also in LV mass/BSA trial in Scandanavian population in 2008.[13]

Conclusion

Amlodipine is a third generation calcium channel blocker with favourable pharmacokinetic profile. For its much higher affinity for vascular calcium channels, it is particularly useful in treating hypertension. Due to its intrinsic natriuretic effect, it is proven effective for mild, moderate & severe hypertension. It provides 24 hours angina protection including morning hours. It is also effective in variant angina. From various studies it was proved that amlodipine is efficacious in improving left ventricular function in hypertensive and ischaemic heart. So adjunct to other usual medications we shall suggest to add low dose CCB i.e. amlodipine with a view to improve left ventricular function, unless contraindicated; though it invites more research work worldwide.

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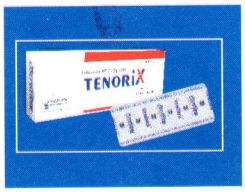
Launching of New Products

Tenorix

Tenoxicam BP 20 mg tablet

Once daily & patient friendly NSAID

Orion Laboratories Ltd. launched Tenorix tablet, each film coated tablet contains Tenoxicam BP 20 mg. Tenorix is a non-steroidal anti-inflammatory drug (NSAID) with anti-inflammatory, analgesic, antipyretic properties and it also inhibits platelet aggregation. Tenorix inhibits prostaglandin biosynthesis and used in the treatment of inflammatory and degenerative disorders of the musculoskeletal system. Tenorix has the superiority over most of the widely used NSAIDs, such as- Tenorix provides convenient once daily dosing, requires no dose adjustment, ensures GI friendly treatment option, helps to protect cartilage damage, provides antioxidant properties and offers convenient treatment cost. Tenorix is indicated for the symptomatic treatment of rheumatoid arthritis, osteoarthritis, ankylosing spondylitis, acute gout, tendonitis, bursitis, periarthritis of the shoulders or hips, post-operative pain and primary dysmenorrhea. The usual recommended dose of Tenorix for all indications (except primary dysmenorrhea, post-operative pain and acute gout) is 20 mg once daily. For primary dysmenorrhea the recommended dose is 20 to 40 mg once daily, for post-operative pain 40 mg once daily and for acute attacks of gout 40 mg once daily for two days followed by 20 mg once daily for a further five days. Tenorix is presented in a box containing 3 X 10 tablets in blister pack. MRP of Tenorix is Tk. 8.00/tablet.



Ortac IM/IV injection

Ranitidine BP 50 mg/ 2 ml

Shows rapid & immediate action in hyperacidic condition

Orion Laboratories Ltd. launched Ortac injection; each 2 ml injection contains 55.80 mg Ranitidine Hydrochloride BP (Equivalent to 50 mg Ranitidine BP). Ranitidine is a competitive, reversible inhibitor of the action of histamine at the histamine H2-receptors, including receptors on gastric cells. Ortac injection is indicated in gastric and duodenal ulcer, prophylaxis and treatment of NSAID-induced ulcer, gastro-esophageal reflux disease (GERD), Zollinger Ellison syndrome, acid related dyspepsia, heartburn. In some hospitalized patients with pathological hypersecretory conditions or intractable duodenal ulcers or in patients unable to take oral medication, Ortac injection may be administered parenterally according to the following recommendations: IM injection: 50 mg (2 ml) every 6 to 8 hours (no dilution is required). IV injection: 50 mg (2 ml) every 6 to 8 hours. Dilute Ranitidine 50 mg (2 ml) injection in compatible IV solution to a total volume of 20 ml and inject over a period of not less than 5 minutes. Parenteral administration may continue until oral feeding is commenced and if there is still a risk, oral ranitidine may then commence. Ortac injection is presented in a box containing blister pack of 2X5's ampoules. MRP of Ortac injection is Tk. 6.00/ ampoule.

Role of hyperhomocysteinaemia and folate deficiency in the development of preeclampsia and gestational hypertension

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Abstract

Elevated homocysteine is an important biochemical marker of endothelial damage, which is known to be associated with preeclampsia (PE) and gestational hypertension (GH); however, as with other markers, the causal relationship between homocysteine and the disorders has not yet been established. The aim of the present study was to explore the causal relationship of homocysteine with PE and GH using a model, which analyses the plasma level of the molecules in early pregnancy. It was also explored whether folate, a known precursor in the homocysteine metabolism, play role in the postulated abnormalities of homocysteine in PE and GH. Hypertension was diagnosed by the criteria of American College of Obstetrics and Gynecology (ACOG). Among 226 pregnant women who were followed until the end of pregnancies, 7 developed preeclampsia and 17 developed GH. A group of 69 randomly selected normotensive subjects were taken as control. Subjects were investigated for their plasma levels of homocysteine and folate. All the study groups age and gestational week matched. Plasma homocysteine (mean±SD) in controls was 5.80+1.80, in PE: 5.5±0.85 (p=0.675) and in GH 5.82±1.24 (p=0.215). No significant differences in plasma homocysteine value in early pregnancy were found between the groups who developed PE or GH and who remained normotensive throughout the pregnancy. The odds ratio (OR) of subsequent PE with a value greater than 6.3µmol/l was 0.44, CI (0.11, 1.81, NS). The folate levels also did not differ between any 2 of the 3 groups. A significantly higher level of urinary protein-creatinine ratio was found in both PE and GH groups as compared to the control. Plasma homocysteine and folate may not have a predictive significance in the development of PE and GH. Early measurement of urinary protein-creatinine ratio in pregnancy may have a role in the prediction of PE and GH at the later stages of pregnancy.

Introduction

Hypertension, a common disorder in pregnancy, constitutes a major risk factor for morbidity & mortality for both mother and child all over the world. Hypertension complicates about 5-10% of pregnancies and includes gestational hypertension, chronic hypertension, PE, Eclampsia as per definitions from the National High Blood Pressure Education Program. Gestational hypertension may be an early sign of either PE or chronic hypertension.[1] Preecelampsia is hypertension; associated with protienuria occurring after 20th weeks of gestation, which occurs most frequently near term (Mabie). Mortality from hypertensive disorder is much higher in the developing countries which is about 70-100 per 100,000 live births.[2] The incidence of PE in the developed countries has been reduced to 0.02% to 0.05% of all deliveries with a fatality of 2%.[3] PE and GH are highly important public health problems. Available epidemiological evidences support the view that, PE is a disease of multiple theories. Among them genetics immunologic, circulatory, uterine vascular changes and endothelial dysfunction is important. Current hypothesis for the pathogenesis of PE states immunological disturbances includes abnormal placentation resulting in decreased placental perfusion and release of various circulatory factors from placenta to both maternal and fetal circulation. These may lead to endothelial cell injury and vascular pathology with changes in vasomotor tone and coagulation. Studies show the vascular endothelial damage and dysfunction is present in uteroplacental bed in PE.[4] PE is a systemic disorder that occurs in the presence of placenta. Since delivery of the placenta abates the problem. Thy placenta is thought to be the key to its pathogenesis.[5] Homocysteine is a sulphydryl containing amino acid derived from the metabolic demethylation of dietary methionine, which is abundant in animal protein. It is present in plasma in four forms.[6] The term total plasma (or serum) homocysteine (tHcy) refers to the combined pool of all four forms of homocysteine. An abnormal tHcy is defined by an arbitrary cut off in the distribution of concentrations found the normal population in much the same way as hypertension and hypercholesterolaemia have been defined.[7] Several factors increase plasma homocysteine level like genetic defects in homocysteine metabolism, nutritional deficiencies in vitamin cofactors, increasing age, male sex, menopause and several diseases.[8] Homocysteine levels decrease significantly during pregnancy and are lowest in the second trimester. The fall in homocysteine parallel the decrease in albumin with pregnancy progression. This finding is not expected because 70% to 80% of homocysteine is albumin bound.[6] Another mechanism responsible for the reduction in homocysteine levels during pregnancy is utilization by the fetus. There is a decreasing plasma homocysteine concentration gradient from the maternal via a umbilical vein to umbilical artery, obtained at delivery, suggesting incorporation of homocysteine in the fetal metabolic cycle.[9] The most common enzyme defect associated with moderately raised tHcy is a point mutation in the coding region of the gene for Methylenetetrahydrofolate reductase (MTHFR), which is associated with a thermo labile MTHFR variant that has about half normal activity.[10]

Patients and methods

The study was carried out for a period of one year in the OPD of Gynaecology and Obstetrics department, Bangabandhu Sheikh Mujib Medical University (BSMMU). The prospective study was taking pregnant women of early trimester upto delivery. The study population comprised of pregnant women from urban or semi urban or rural areas who attended the antenatal check up of the study place. Single on pregnancies of early trimester with no associated medical complaining were included for research purpose. A total number of 330 patients were taken for the study. Finally, after fulfilling all criteria, 281 patients were enrolled for final assessment and during follow

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up, 226 patients were continued. A questionnaire was developed to obtained relevant information of demographic and socio-economic data. Anthropometric data included weight, height and body mass index and blood pressure were measured. Subjects were requested to fast over night (12 hours) and not to smoke or take any kind of medicine on the previous day. Blood samples were collected and relevant investigations were done with plasma to exclude diabetes mellitus, renal disease and hyperlipidaemia. The plasma samples were analyzed for fasting glucose, TG, total cholesterol, HDL, LDK, creatinine, folate and homocysteine. Plasma homocysteine concentrations were measured by florescence polarization immunoassay (FPIA) technology and Plasma folate level estimation by competitive immunoassay technique using IMMULITE analyzer. Data are expressed as mean±SD for parametric values and median for nonparametric values. The relationship between homocysteine and other variables was examined using Spearmen's nonparametric coefficient correlation analysis.

Results

Table 1: Proportion of PE and GH cases

Groups	Number of cases	Percentages (%)
GH	17	7.52
PE	7	3.09
Control	202	89.38
Total	226	100.00

In Table 1, total 281 pregnant women were included in the study. Among them 65 patients were lost in the follow up, 7 pregnant women developed preeclampsia and 17 developed GH among the remaining 226 patients. Thus the proportion of PE was 3.09% and GH 7.52%.

Table 2: Homocysteine status of the study subjects

Groups	Plasma homocysteine (µmol/l)
Control (n=69)	5.50±0.85
PE (n=7)	5.80±1.80
GH (n=17)	5.82±1.24

In Table 2, the plasma homocysteine, mean±SD of the study groups were 5.50±0.85 as control, 5.80±1.80 as PE and 5.82±1.24 as GH. There was no significant difference in Plasma homocysteine levels between different groups.

Table 3: Plasma folate status of the study subjects

Groups	Plasma folate (n mol/l)
Control (n=69)	10.92±4.35
PE (n=7)	13.42±5.58
GH (n=17)	10.04±5.21

In Table 3, the plasma folate, mean±SD of the study groups were 10.92±4.35 as control, 13.42±5.58 as PE and 10.04±5.21 as GH. There was no significant difference in folate (n mol/l) levels between different groups.

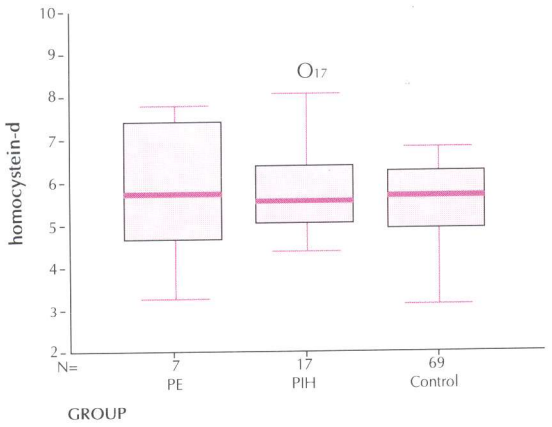

Figure 1: Box plot of homocysteine related to case and control pregnancies

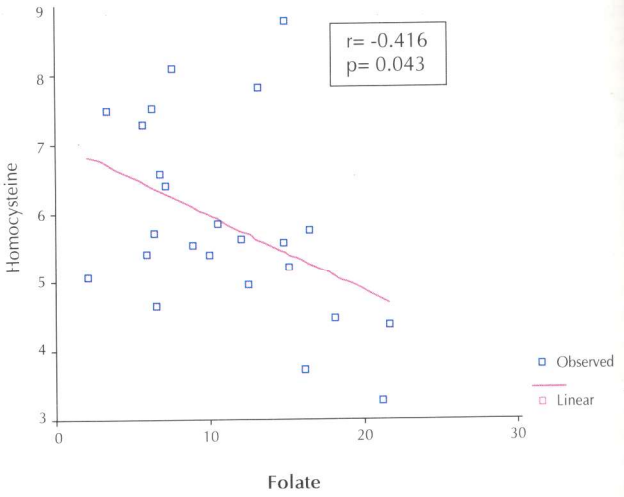

Figure 2: Relationship between folate and homocysteine in the PE and GH subjects

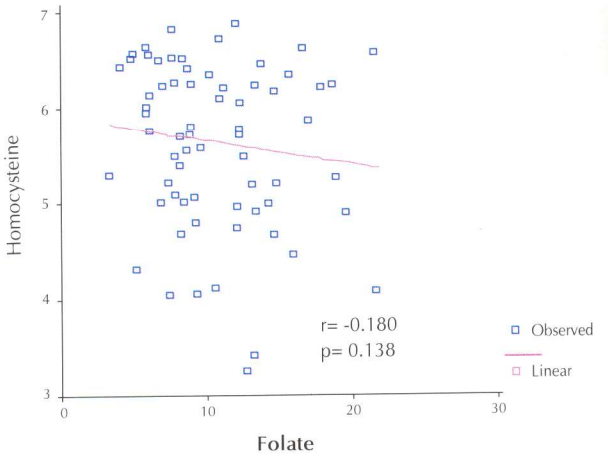

Figure 3: Relationship between folate and homocysteine in the control subjects

Figure 1 showed homocysteine related to case and control study. Figure 2 and 3 showed that relationship between Folate and Homocysteine in the PE and GH subjects and relationship between Folate and Homocysteine in the control subjects respectively.

Discussion

Preeclampsia is a leading cause of maternal and perinatal morbidity and mortality. In the present study the proportion of the PE cases were 3.09%, which is similar to worldwide incidence of preeclampsia. The proportions of GH cases were 7.52%, which is similar to incidence found by Gilford et al.[1] Elevated concentrations of homocysteine have been associated with increased risk of vascular disease.[11] In the present study, plasma tHcy level ranges from 3.80-7.20 (µmol/l) with a mean value of 5.50 (µmol/l) in early pregnancy in uncomplicated pregnancy. This finding is consistent with previous findings by Danna et al.[12] The present study is not able to demonstrate any difference in plasma homocysteine levels at early pregnancy between women who later developed PE or GH and those who remained normotensive. The plasma homocysteine, mean±SD of the study groups were 5.50±0.85 as control, 5.80±1.80 as PE and 5.82±1.24 as GH. Cotter et al carried out a prospective study taking 54,000 patients attending their 1st antenatal visit, mean±SD plasma homocysteine value of cases and controls were 9.8±3.3µmol/l and 8.4±1.9 µmol/l respectively with a p value of <0.0001.[13] In the present study plasma folate ranges between 2.22-19.62 n mol/l with a mean±SD in 10.92±4.35 as control, 13.42±5.58 as PE and 10.04±5.21 as GH. There may be two reasons for the observed increase of folate concentration in the present study, one may be dietary intake; another may be folate supplementation during pregnancy. There was no correlation between homocysteine and folate in PE, GH and control group. However, when PE and GH group were combined, there was significant negative correlation between homocysteine and folate as expected.

Conclusion

Preeclampsia and gestational hypertension are two hypertensive disorders of pregnancy with involvement of both placental and maternal circulation. Elevated homocysteine is an important biochemical marker of endothelial damage, which is known to be associated with PE and GH; however, as with other markers, the causal relationship between homocysteine and the disorders has not yet been established.

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MSD News

Round Table Meeting (RTM)

Mymensingh

Phulpur UHC, Mymensingh: Phulpur UHC arranged a RTM on 19th May 2009 on "Gastric acid related disorders" at the conference room. Dr. H. A. Mahmudunnessa, UH & FPO chaired the meeting and Dr. Raisul Huda Bhuyan, RMO was the chief guest.

Madan UHC, Netrokona: A RTM was arranged by Madan UHC on 20th April 2009 on "Common skin problems in outpatient department" at the seminar room. Dr. Sirajul Haque Talukder, UH & FPO chaired the meeting and Dr. Md. Abdul Kuddus, RMO was the chief guest.

Trishal UHC, Mymensingh: On 21st April 2009 a RTM was arranged by Trishal UHC on "Role of ceftriaxone to treat various infections" at the conference room. Dr. Sayed Habibullah, UH & FPO chaired the meeting and Dr. Abul Hossain (Sadhin) was present as the chief guest.

Rajshahi

Medicine Unit III, RMCH: Medicine Unit III of Rajshahi Medical College Hospital, arranged a RTM on "Community acquired Pneumonia and its management & Pain management" at the Nanking Chinese Restaurant at Rajshahi on 19th July 2009. Dr. Md. Azizul Haque Associate professor was the chairperson.

Dhaka

HCDP Bashabo: On 11th June 2009 HCDP Bashabo arranged a RTM on "Spasticity management" at China Park Chinese Restaurant, Khilgaon. Dr. Amal Chandar Singha, Centre Director was present as the chairperson.

Medicine Unit 1, DNMCH: On 21st July Medicine unit 1 of Dhaka National Medical College Hospital arranged another RTM on "Haemolytic anaemia and pneumothorax" at the Professors room. Dr. A K M Morshed chaired the meeting.

Kushtia

Horinakundu UHC, Jhenaidah: A RTM was arranged by Horinakundu UHC on "Gastric acid related disorders" on 21st June 2009 at the seminar room. Dr. Moslem Uddin, UH & FPO was present as the chairperson.

Jibon Nagar UHC, Chuadanga: Jibon Nagar UHC arranged a RTM on 22nd June 2009 on "Gastric acid related disorders" at the conference room. Dr. Md. Rafiqul Islam, UH & FPO chaired the meeting.

Chawmohani

Chagalnaia UHC, Feni: A RTM was arranged by Chagalnaia UHC, Feni on 22nd June 2009 on "Gastric Acid Related Disorders" at THA's room. Dr. Al-Haj Khirul Islam, THA was adorned the seat of Chairperson.

Feni Sadar Hospital: On 23rd June 2009 Feni Sadar Hospital, Feni arranged a RTM on "Short review of common skin diseases & use of Topical drugs" at the Conference Room of Feni Sadar Hospital. Dr. Sarwar Jahan, RMO was adorned the seat of Chairperson.

Barisal

Sadar Hospital, Barisal: A RTM was arranged by Barisal Sadar Hospital on 26th June 2009 on "Pain Management" at Director's room. Dr. Harun-ur-Rashid, ENT Consultant was adorned the seat of Chairperson.

Medicine Unit 1, SBMCH: On 26th June 2009 SBMCH (MU-1), arranged a RTM on "Pain Management" at Indoor conference room. Dr. Saidul Bashar, Asst. Reg. of MU-1 was the Chairperson.

Role of intrauterine balloon catheter in controlling massive PPH : Experience in Rajshahi Medical College Hospital

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Abstract

Postpartum hemorrhage (PPH) remains a significant complication of child birth world wide. The most common cause of PPH is uterine atony. Recently, uterine tamponade using intrauterine condom appears to be an effective tools in the management of uncontrolled PPH. Objectives of our study was to see the effectiveness of large volume fluid filled condom catheter in the management of primary PPH. *Methods:* A condom was inserted in the uterus by means of a size 16 rubber catheter and inflated with 250 to 300 ml normal saline until the bleeding was controlled. The condom was kept in situ for 24 to 48 hours. *Results:* Out of 53 cases, PPH was controlled in 52 cases. One patient died as the patient was eclamptic & developed disseminated intravascular coagulation (DIC). No patient required surgical intervention. *Conclusion:* Fluid filled intrauterine condom is an effective method in the management of primary PPH when usual measures & drugs fail to control PPH.

Keywords

Postpartum haemorrhage, Balloon tamponade, Condom, Rubber catheter.

Introduction

Obstetric hemorrhage is a significant contributor to maternal morbidity and mortality & accounts for half of the maternal death world wide.[1] In a review of maternal mortality, PPH accounted for 28% of all deaths in 11 population based studies from eight developing countries.[2] Guideline for the management of PPH involves a stepwise approach which includes oxytocin, ergometrine, misoprostol, prostaglandin F2α. If these attempt prove to be unsuccessful and the woman is not already having caesarean section, a laparotomy is considered. During this time, various surgical interventions may be used, like internal Iliac artery ligation, B-Lynch suture, peripartum hysterectomy etc. Sterile gauze was invariably used for uterine packing but problems encountered in achieving a good packing were concealed bleeding, uterine trauma and infection.[3]

Currently, the intrauterine balloon is believed to act by exerting inward to outward pressure that is greater than the systemic arterial pressure to prevent continual bleeding.[4] Many of these balloons have previously been used to control haemorrhage at the other anatomical sites, including the urinary bladder and oesophagus, as well as to control PPH from vaginal laceration.[5,6]

Condoms (latex) or plastic sheaths are used mainly as contraceptive devices and barriers against STDs. Recently obstetricians have given condoms a new image. A condom inflated with isotonic solution can be used to create the tamponade.[7] In Bangaldesh, the method was first used in Dhaka Medical Collage Hospital to manage intractable PPH due to uterine atonicity or placenta accreta. This study was done in light of two other studies that reported on the use of Sengstaken Blakemore tube and the Rush urologic hydrostatic balloon catheter for controlling PPH.[8,9]

Materials & methods

This prospective, observational study was done in the department of Obs & Gynae, Rajshahi Medical College Hospital, Rajshahi from August 2007 to September 2008. Total 53 patients who developed massive PPH & not controlled by usual measures & drugs were included in this study.

Description of the technique

- Patient was placed in dorsal / lithotomy position.
- A Foley catheter was inserted in the urinary bladder for urine indwelling drainage.
- Cervix was exposed by sponge holding forceps.
- A condom was inserted in the uterus by means of a size 16 rubber catheter & inflated with 250 to 300 ml of normal saline until the bleeding was controlled.
- The proximal end of the catheter was folded & tied with thread so that the saline solution could not escape.
- A roller gauze was packed in the vagina to keep the inflated condom in place.
- Condom was kept for 24 to 48 hours depending on the severity of blood loss.
- Maintenance of contraction was ensured by continuous infusion of oxytocin.
- Prophylactic antibiotic was administered in every case.

Results

Condom tamponades were used in a total 53 patients having massive PPH. Out of 53 patients, bleeding was controlled in 52 patients within 24 to 48 hours. One patient died as the patient was eclamptic & developed DIC. No patient required surgical intervention.

Table: Out come of use of condom temponade in PPH control (n=53)

Characteristics		No.	Percentage
Type of PPH			
	Primary	53	100 %
	Secondary	0	
Causes of PPH			
	Atonicity	51	96.23 %
	Placenta previa & morbid adhesion	2	3.77 %
Mode of delivery			
	Spontaneous vaginal	47	88.68 %
	Instrumental	2	3.77 %
	LUCS	4	7.55 %
Duration of retention of condom catheter			
	24 hours	32	60.38 %
	24-48 hours	21	39.62 %
Outcome			
	PPH controlled	52	98.11 %
	PPH uncontrolled	1	1.89 %

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Discussion

To arrest bleeding, balloon tamponade procedure has been accepted in medicine over 50 years, with the Sengstaken-Blakemore tube having an established place in the management of bleeding oesophageal varices & balloon temponade following prostatectomy & massive bladder hemorrhage.[10,11] Various studies showed that balloon temponade is not only effective in controlling PPH from atonic uterus but also equally effective in creating temponade when the uterus is well contracted & there is ongoing hemorrhage from the placental bed or vaginal laceration.[12] Once in- situ, not only bleeding will be halted, but also any consumptive coagulopathy can be reversed.[13,14] As these devices exert uniform pressure over the open sinuses of the uterus, bleeding controlled immediately.[15] However, although they are very effective for controlling PPH, are very expensive & not available in our country. On the other hand, condoms & plain rubber catheters are cheap & easily available in every part of the world. They can be easily & safely applied by the primary health workers before patient referral. They often eliminate the need for surgical intervention without compromising maternal lives. Therefore, balloon catheters should be readily available on all labour wards & should be part of all protocols in the management of PPH.

Conclusion

We would suggest uterine tamponade using fluid filled condom as the first step for controlling PPH after exploration of the uterus prior to performing laparotomy. It is quick, safe & effective method & results can be known within minutes.

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Launching of New Products

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Original Article

Determination of MIC and MBC of selected tetracycline capsule commercially available in Bangladesh

Kowser MM[1], Hoque MM[2], Fatema N[3]

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Abstract

Background: Usually we consider that the tetracycline capsules in Bangladesh maintain standard MIC and MBC. But how much is this assumption is true? this will be evaluated through this research work. *Objective:* This is a cross sectional study (January-2006 to Decmber-2006) to determine the minimal inhibitory concentration (MIC) and minimal bactericidal concentration (MBC) of tetracycline capsule commercially available in Bangladesh. *Method:* The collected samples were analyzed according to BP specifications. The MIC was determined by broth dilution method. MBC were determined by the drop plate method from the tubes, where apparently no visible growth found. *Results:* The MIC value of tetracycline capsule against *Bacillus pumillus, Pseudomonus* spp. *Staphylococcus aureus, Shigella* spp. and *E coli* were found 1.0, 8.0, 0.5, 1.0 and > 64.0 µg/ml (micro gram per milliliter) respectively. The MBC value of tetracycline capsule against *Bacillus pumillus, Pseudomonus* spp. *Staphylococcus aureus, Shigella* spp. and *E coli* were found 2.0, 16.0, 1.0, 2.0 and > 64.0 µg/ml respectively. Conclusion: MIC and MBC values higher than that of the peak serum concentration must have chance of therapeutic failure and development of antibiotic tolerance and resistance to the bacteria.

Key words

Minimal inhibitory concentration (MIC), Minimal bactericidal concentration (MBC), Colony forming unit (C.F.U).

Introduction

To evaluate the efficiency of antibiotic there are two factors[1], which influence potential utility of a antibiotic in a specific clinical situation. The first is the measure of potency of the antibiotic for the pathogen in question MIC and MBC. The second is relationship between the concentration time profile and potency of the antibiotic. This research work will play an important role to determine the MIC and MBC of selected tetracycline capsule in Bangladesh.

Materials and methods

1. Collection of sample: The tetracycline capsule collected from the retail seller and standard sample collected from the pharmaceutical company in the Dhaka city.

2. Collection of organisms: Pseudomonus spp. *Staphylococcus aureus, Shigella* spp. and *E coli* collected from the patient sample of Dhaka Medical College Hospital and *Bacillus pumillus* from the Microbiology Department of University of Dhaka.

Reagents

1. Sterile water
2. Hydrochloric acid
3. Potassium phosphate buffer (0.5M)

Media

1. Mueller Hinton Broth (MHB)
2. Mueller Hinton Agar (MHA)
3. S.S Agar
4. Mannitol Salt Agar (MSA)
5. Cetrimite Agar (CA)
6. Blood Agar (BA)

Instruments and apparatus

1. Sterile 5 ml screw cap test tubes
2. 250 ml conical flask
3. 250 ml measuring cylinder
4. Inoculating loop
5. 1 ml and 0.1 ml micro pipette
6. 10 ml glass pipette
7. Beaker
8. Marker
9. Bunsen burner
10. Small and large (7"x 7") petriplate
11. Voltex mixture machine (FISONS-11777)
12. Shaking or rotator machine (FISONS-200)
13. Electrical digital balance (AJ 150 L)
14. Spatula
15. pH meter (HANNA)
16. Laminar air flow (C-901)
17. Incubator adjuster at 37°C. (SLI-600)
18. Spectrophtometer (Spectronic-20)
19. Freeze (MDF-U20806)
20. Micropepette (GILSON)
21. Autoclave (HA-240M)

Preparation of tetracycline solution[2]

128 mg equivalent 330x128÷ 250=168.96 mg tetracycline hydrochloride (G-tetracycline) capsule was dissolved in 1000 ml sterile water and rotated at the rate of 75 rpm for 60 minute at room temperature.

MIC and MBC determination procedure

Culture: Overnight Mueller Hinton broth cultures of *Staphylococcus aureus, E. coli, Bacillus pumillus, Shigella spp.* and *Pseudomonas spp.* at 37°C were prepaered. The culture was adjusted to obtain turbidity comparable to that of the turbidity of MC, Farland 0.5 standard and then further diluted 1:200 in Mueller Hinton broth. The inoculums thus prepared expected to obtain 10^5 to10^6 C.F.U/ml.

Procedure[3]

1. An appropriate amount of tetracycline capsule was dissolved in respective solvent to prepare an antibiotic solution containing 128 µg/ml (0.128 g drug plus 1000 ml respective solvent).

2. Two fold dilutions of the antibiotic solution in Mueller Hinton broth were prepared and described below:

(a) Ten sterile tubes were placed in a rack and were labeled each 1 through 8 and first one labeled as A.C (antibiotic control) and last one was labeled as G.C (growth control).

(b) 1 ml of Mueller Hinton broth was added in each test tube.

(c) 1 ml of antibiotic solution was added to test tube no 1 and A.C.

(d) With a sterile micropipette and tips, after adequate mixture 1 ml was transferred from tube no 1 to tube no 2.

(e) After a through mixing, 1 ml was transferred with a separate micro pipette from tube no 2 to tube no 3.

(f) This procedure was repeated through the next-to-next upto the tube no 8. Except tube no G.C. (using fresh pipette for each dilution). From tube no 8, 1 ml was removed and

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discarded. The last tube (tube G.C) received no antimicrobial agent and was served as a growth control. First A.C labeled test tube was served as a antibiotic control.

3. Each tube was inoculated (including the growth control except antibiotic control) with 1 ml of the culture of respective organism. The final concentration of antimicrobial agent in this test tube was half of the initial dilution series because of the addition of an equal concentration of inoculums in Mueller Hinton broth.

4. The tubes were incubated at 37°C for 24 hours.

5. The tubes were examined for growth and were determined the MIC of tested antibiotics, which is bacteriostatic for the test organism. The tubes were examined for visible growth (cloudy) and was recorded growth as (+) and no growth as (-).

6. For determination of MBC, the concentration which was bactericidal, was then found by sub cultured the contents of selective tubes into a series of Mueller Hinton broth, which did not contain any antibiotic and started from last two non-visible tube to the 1st two visible tube (direction tube no 1 to tube no 8). Then was inoculated into Mueller Hinton agar containing Petri plate by 0.1 sterile micropipette and separate 0.1 ml sterile tips in drop method.

7. The plates were incubated at 37°C for 24 hours.

Tube No.	A.C	1	2	3	4	5	6	7	8	G.C
i) Mueller Hinton broth 1ml	1	1	1	1	1	1	1	1	1	1
ii) Antibiotic solution 1 ml	1	1	1	1	1	1	1	1	1	0
iii) Initial antibiotic concentration µg/ml	128	128	64	32	16	8	4	2	1	0
iv) Bacterial suspension 1 ml	0	1	1	1	1	1	1	1	1	0
v) Final Volume 2 ml	2	2	2	2	2	2	2	2	2	2
vi) Final antibiotic concentration µg/ml	64	64	32	16	8	4	2	1	0.5	0

A.C = Antibiotic Control, G.C = Growth Control

Results

Table 1: MIC & MBC values of antibiotics tested against five organisms

Antibiotics tested	Test organisms MIC / MBC in µg/ml				
	Bacillus pumillus	*Pseudomonus* spp.	*Staphylococcus aureus*	*Shigella* spp.	*E. coli* spp.
Tetracycline	1.0 /2.0	8.0 / 16.0	0.5 / 1.0	1.0 / 2.0	> 64.0
Cephradine	1.0 /2.0	8.0 / 16.0	0.5 / 1.0	4.0 / 8.0	> 64.0
Cefixime	1.0 /2.0	> 64.0	1.0 / 2.0	2.0 / 4.0	> 64.0
Azithromycin	1.0 / 2.0	> 64.0	2.0 / 4.0	> 64.0	> 64.0
Ciprofloxacin	4.0 / 8.0	2.0 / 4.0	8.0 / 16.0	1.0 / 2.0	> 64.0

Photograph represents that the MIC of tetracycline against *Bacillus pumillus* and *staphylococcus aureus* was found 1µg/ml & 0.5 µg/ml respectively

Table 1 showed that MIC values of tetracycline against *Bacillus pumillus*, *Pseudomonus* spp. *Staphylococcus aureus*, *Shigella* spp. and *E coli* were found 1.0, 8.0, 0.5, 1.0 and > 64.0 µg /ml, respectively, and the MBC values of tetracycline against *Bacillus pumillus*, *Pseudomonus* spp. *Staphylococcus aureus*, *Shigella* spp. and *E coli* were found 2.0, 16.0, 1.0, 2.0 and > 64.0 µg /ml, respectively.

Discussion

Oral doses of 500 mg every 6 hours of tetracycline hydrochloride produce peak blood level of 4-6 µg /ml[4]. Intravenous injection of tetracycline give some what higher levels only temporary. Table 1 showed that MIC values of tetracycline against *Bacillus pumillus*, *Pseudomonus* spp. *Staphylococcus aureus*, *Shigella* spp. and *E coli* were found 1.0, 8.0, 0.5, 1.0 and > 64.0 µg /ml, respectively. The MIC level of tetracycline for *Pseudomonas* spp. (8.0 µg /ml) and E. coli. (>64.0 µg /ml) were higher than the peak blood serum level (4-6 µg /ml). so, Tetracycline should not be the choice of antibiotic for these *Pseudomonas* spp. and *E. coli* induced infection due to high MIC level. Table 1 showed MBC values of tetracycline against *Bacillus pumillus*, *Pseudomonus* spp. *Staphylococcus aureus*, *Shigella* spp. and *E coli* were found 2.0, 16.0, 1.0, 2.0 and > 64.0 µg /ml, respectively. Rashed et al. reported that 80% *Shegilla* spp. were resistant to tetracycline[5]. The present study showed that Shegilla spp. was found sensitive to tetracycline. Maximum medical representative try to motivate the doctors to prescribe new and costly antibiotics. Some doctors think that more expensive and newer antibiotics will be more effective to treat infection.

Conclusion

The MIC and MBC values of selected tetracycline evaluated in this study. Because of shortage of fund, the study was performed only one type of sample antibiotic. No significant research work is available regarding the MIC & MBC of tetracycline in Bangladesh. For this reason it was difficult to obtain required amount of information for conducting this study.

Recommendation

To provide standard potent antibiotics in Bangladesh, suggestion and recommendations are as follows:

1. A complete study on biopotency of all antibiotics are essential.
2. A modern antibiotics testing laboratory have to be established.
3. Regular monitoring of the quality of antibiotics are essential.
4. Awareness must be created by using mass media about use and misuse of antibiotics.

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Peripartum cardiomyopathy

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Introduction

Peripartum cardiomyopathy is a rare but life threatening form of heart failure affecting women in the last month of pregnancy or the first five months of postpartum without any previous significant heart disease.[1,2] A relationship between pregnancy and dilated cardiomyopathy was first noted in 1870, when Virchow and Park found autopsy evidence of myocardial degeneration of a patient died during puerperium.[3] Gouley et al[4] in 1937 described the clinical and pathological feature of several pregnant patients and autopsy of four of them, who died, revealed enlarged heart with fibrosis and focal necrosis distinctly atypical from those of other causes of myocardial failure - leading to proposal that heart failure was related to pregnancy and puerperium either directly or indirectly for which most physician now refer to this condition as peripartum cardiomyopathy.[3]

Diagnostic criteria

In 1971 Demakis et al[5] established the initial criteria for peripartum cardiomyopathy (PPCM), and in 1977 participants in a National heart, Lung and Blood Institute (NHLBI) workshop[6] agreed on a standard definition, which included the following specific criteria:

1. Onset of cardiac failure with no specific cause in the last month of pregnancy or within five months after delivery.
2. Absence of heart disease before the last month of pregnancy.
3. Absence of any specific cause for the heart failure.
4. Presence of specific echocardiographic criteria which include a left ventricular systolic dysfunction with a left ventricular ejection fraction of less than 45 percent, fractional shortening of less than 30 percent on an M-mode echocardiographic scan, or both, and a left ventricular end diastolic dimension of more than 2.7 cm per meter square of body surface area.

Epidemiology

Incidence of peripartum cardiomyopathy has a ratio of 1:300 to 1500 live births.[5,6,7] In United States the prevalence is estimated of 1 case per 1300 - 1500 live births. Whereas it is 1 case per 6000 live birth in Japan, 1 in 1000 in South Africa and 1 in 350 in Haiti. Overall incidence is higher in Africa.[8]

Risk factors

Peripartum cardiomyopathy is associated with several risk factors such as older maternal age, greater parity, black race and multiple gestations.[5] It is not clear whether race is an independent risk factor, but probably a greater incidence of hypertension in this group may influence this observation.[1]

Pathogenesis

The pathogenesis of peripartum cardiomyopathy is controversial. Early evidence has suggested nutritional deficiency may have a role because of higher incidence of PPCM in malnourished women but recent studies don't support this view.[7,9,10] Although the cause of PPCM still remains unclear, several authors have suggested possible etiologies.

Endomyocardial biopsies have confirmed the presence of myocarditis in some patients diagnosed with PPCM[11] and immunosuppressive therapy with prednisolone and azothiaprine has resulted in clinical improvement in these patients.[12] The cause of myocarditis in these patient is unclear but viruses has been suggested as one of the causes[13,14,15] but toxins, drugs and autoimmunity are also possible etiology. Virus eg. Coxakie virus antibody has been identified and it may be possible that virus triggers autoimmune reaction in the myocardium.[13] Another possibility is suppressed immune response may allow unchecked viral proliferation and increased likelihood of myocarditis. Studies have shown increased susceptibility to viral myocarditis due to coxakie and echovirus during pregnancy.[6]

Another possible etiology is that there is a maternal immunological response to fetal antigen. This is based on fact that the fetal cell may enter maternal circulation and not rejected due to suppressed immune state of pregnancy. If the fetal cell deposit in cardiac tissue during pregnancy, they may be recognised nonself after delivery when immune competence regained and hence autoimmune response may be triggered.[6,16]

Other investigators have suggested that PPCM may be due to autoimmune process associated with high titre of autoantibody against particular cardiac tissue protein.[17] It is postulated that after delivery the fast degeneration of uterus results in fragmentation of tropocollagen by collagenolytic enzymes which release actin, myosin and their metabolites. Antibodies are formed against actin and cross react with myocardium.[18] Hemodynamic stress has also been considered as a possible cause of PPCM. During pregnancy there are some alterations in hemodynamic setup[19] with subsequent transient hypertrophy of heart.[20] There is reversible decline in LV systolic function that persists up to early postpartum but returns to normal thereafter. It is possible that PPCM is due to exaggeration of this decrease in systolic function.[21] Other possible etiological factors include prolonged tocolytic therapy[17,22] proinflammatory cytokines (TNF; IL1, 1L6),[23] excessive salt ingestion[1,10,24]; abnormalities of 'relaxin', an ovarian hormone with inotropic and chronotropic properties may cause excessive relaxation of the cardiac skeleton.

Clinical presentation

The clinical presentation of PPCM may include the following symptoms: shortness of breath, dyspnea on exertion, paroxysmal nocturnal dyspnea, cough, haemoptysis, palpitation, chest discomfort, pedal oedema, abdominal pain and non specific fatigue.[25] Physical examination may include oedema, rales, third and fourth heart sounds, JVP distension, new murmur.[6,17] NYHA (New York Heart Association) classification of heart failure is not relevant, because it lists the occurring signs and symptoms of normal pregnancy that may be similar to those of PPCM women.

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This classification may not therefore accurately reflect the severity of underlying cardiac dysfunction.[26] Unless one has a high clinical suspicion of PPCM, this disease may go unrecognised since many women have symptoms in the third trimester of pregnancy that include dyspnoea, pedal oedema and fatigue.[6]

Differential diagnosis

The clinician must be very careful to exclude other causes of heart disease before making the diagnosis of PPCM. During pregnancy there are many physiologic changes that can mimic heart failure. In 1st trimester there is increased blood volume with increased JVP, pedal oedema is often noted, dyspnoea and fatigue are also common symptoms.[27] Besides, these physiological changes may unmask subclinical or compensated heart disease for first time. Diagnosis of PPCM should be seriously considered in all patients with persistent or worsening heart failure in last month of pregnancy or in early puerperium. When diagnosis of PPCM is considered, nearly every other cause of left ventricular dysfunction must be excluded such as myocardial infarction, sepsis, severe pre-eclampsia, pulmonary embolism, idiopathic dilated cardiomyopathy, valvular disease and pulmonary vasculitis (SLE, scleroderma, rheumatoid disease).[28] Idiopathic dilated cardiomyopathy has clinical characteristic similar to PPCM but the onset is not restricted to the peripartum and can occur in the second trimester.[29]

Diagnostic methods

Electrocardiogram (ECG), chest radiogram, M-Mode and two-dimensional doppler echocardiographic studies should be routinely performed. ECG may be normal, but it usually demonstrates sinus tachycardia or atrial fibrillation; there may be normal or low voltage or left ventricular hypertrophy or non specific ST-T waves changes. QRS complex may show intraventricular conduction defect; bundle branch blocks are occasionally present.[1,30] Chest X-ray should be performed with abdominal shielding to exclude pulmonary pathology. Chest X-rays are not specific; it may show cardiomegaly with small bilateral pleural effusion, pulmonary venous congestion and bibasal infiltrates.[1,9,31] Echocardiography is very important to exclude other causes of heart failure such as mitral valve disease, left atrial myxoma and pericardial disease.[32] The echocardiogram usually shows a dilated left ventricle with marked impairment of overall systolic performance[1,33] as previously described under diagnostic criteria.[34]

Haemodynamic studies are not usually performed but may show an elevated right heart and left heart filling pressure with diminished cardiac output; left ventriculography demonstrates a global reduction in the left ventricular systolic performance; coronary angiograms are generally normal.[34] The endomyocardial biopsy should be considered to confirm the diagnosis if the nature of PPCM remains unclear.[18] To rule out infection as the cause of the cardiomyopathy serum samples should be tested for bacterial and viral culture and specially for Coxakie B virus titre.

Treatment

The treatment for peripartum cardiomyopathy is similar to that for other non-ischemic dilated cardiomyopathy; however, consideration must also be given for the fetus.

- Non pharmacological therapy
  Low sodium diet (<4gm/day); fluid restriction (<2L/day); modest daily exercise (i.e. walking)
- Oral pharmaceutical therapy
  A. *Prepartum:* Amlodipine; Hydralazine/Nitrate; Digoxin; Diuretic; β-Blocker: e.g. Cervidelol (α-blocker effect).
  B. *Post partum:* Angiotensin converting enzyme inhibitor or angiotensin II receptor blocker; Digoxin; Diuretic; Amlodipin; Hydralazine/ Nitrate; β-Blockers.

Intravenous pharmaceutical therapy for patients with severe symptoms (unresponsive to above oral therapy) Dobutamine; Dopamine; Milrinone; Nitroprusside. (Adapted from Brown et al[25]). In general, the goal is to reduce the amount of volume returning to the heart (preload reduction), decrease the resistance against which the heart must pump (after load reduction) and increase the contractile force of the heart (inotrophy).

Non pharmacological regimen as shown above is very important particularly in women with symptoms and signs of heart failure. Once heart failure symptoms have been controlled, modest exercise e.g. walking, cycling has been proven to improve survival. Bed rest is not recommended because it predisposes pregnant women to develop deep venous thrombosis with subsequent pulmonary embolism. Diuretics are indicated when sodium restriction alone is therapeutically unsuccessful.[1,35] Diuretic and nitrate reduce the preload. In pregnancy diuretics must be used with caution as to avoid dehydration.

Angiotensin-converting enzyme inhibitor or angiotensin II receptor blockers are effective in reducing the after load and should be considered a mainstay of treatment for PPCM after delivery.[36,37] But unfortunately they are contraindicated[36] during pregnancy for severe adverse neonatal renal effects.[38] Neonatal deaths[38] have been reported after ACE inhibitor therapy during pregnancy. ACE inhibitors are excreted into breast milk[39,40] and breast-feeding should be discouraged in patients who require ACE inhibitor therapy. Therefore hydralazine in combination with nitroglycerin or amlodipine[41,42] is the first choice of treatment for PPCM. Hydralazine is safe in pregnancy for both mother and fetus.[41] Among all the calcium channel blockers amlodipine has been found to improve survival of non-ischemic cardiomyopathy patient.[21]

Oral inotropic therapy is provided by digoxin[43,44] and also useful in case of atrial fibrilliation. Digoxin is generally considered safe as oral inotropic therapy in pregnancy and breast feeding.[16,19] β-Blockers have been shown to improve mortality in patients with dilated cardiomyopathy by reducing deleterious effects of excessive stimulation of sympathetic nervous system and delays progression of myocardial dysfunction.[45,46] β-Blockers with vasoactive property (α-blocker effect) eg. carvedilol have also shown improvement by reducing afterload.[47] Thus during pregnancy β-Blockers may improve haemodynamic functions by reducing heart rate, reducing catecholamine toxicity, up regulating myocardial β-adrenergic receptor and improving ventricular diastolic function and rate of the survival.[48,49] But long term use of β-Blocker is associated with low birth weight babies and neonatal apnea, hypotension, bradycardia and hypoglycemia and so often avoided in prepartum PPCM patients.[39]

Patients with PPCM are predisposed to thromboembolism due to hypercoagulable state of pregnancy and stasis of blood due to left ventricular dysfunction. Patients with severe left ventricular dysfunction benefit from anticoagulation. But since coumarin is contraindicated as it crosses the placenta and has

teratogenic effect, heparin should be used during antepartum. Whereas either heparin or coumarin can be used safely in postpartum since neither drug is secreted in breast milk.[22]

In severely symptomatic patients or in those treated for acute illness, intravenous preload and afterload reducing agents (e.g. nitroprusside, nitroglycerin) or inotropic agents (eg dobutamine, dopamin, milrinone) should be considered. In particular, risk of nitroprusside therapy should be evaluated because thiocyanate and cyanide may accumulate in the fetus.[39] Immunosuppressive therapy (e.g. prednisolone or azothiaprin) can be considered for pregnant women with myocarditis demonstrated by cardiac biopsy and for those who didn't improve after anti failure treatment.[50] A recent retrospective study suggest that patients with PPCM treated with immunosuppressive drugs showed greater improvement in ejection fraction than in patients treated conventionally.[51]

Patients who fail medical management may be considered for heart transplantation. Studies indicate that in patients with PPCM survival after transplant is comparable to age matched women requiring heart transplant for other reason, but showed a marginally higher rate of biopsy proven early rejection requiring more aggressive cytotylic therapy.[52]

Prognosis

Overall the maternal mortality rates for PPCM in the United States are estimated to be between 18 to 56 percent in the first three months of development of symptoms.[5, 53]

The prognosis is related to normalisation of left ventricular size and function within six months after delivery.[6] Furthermore, the left ventricular size and severity of dysfunction at the time of presentation are important determinants of outcome.[10] During long term follow-up, patients with PPCM, whose hearts return to normal within six to twelve months tend to have a better survival rate at five years compared with patients with cardiomyopathy of another etiology i.e. dilated cardiomyopathy.[54] The incidence of resolution is unclear. An early series reported that 50% of patients experienced resolution.[26] In another study 93% of patients had progressive or persistent cardiomyopathy. But these patients might had co-morbid disorders and no assessment of left ventricular function before pregnancy was available.[17] There is also a trend for a poorer prognosis in women who are black, multiparous and more than 30 years old.[24] Currently, the recommendation for future pregnancies after PPCM varies and there are few reports that describe the outcomes of patients in subsequent pregnancies.

In one study[55] it was found that subsequent pregnancy did not necessarily mean a deterioration of cardiac function. In contrast Heiberger et al[56] found high chance of relapse of symptoms even though they recovered from PPCM; while Sulton et al[57] in a small study reported that patients who recovered from PPCM didn't relapse in subsequent pregnancy. But on balance patients without resolution of the cardiomyopathy are at significant risk of death or exacerbation of the disease[1] and should be advised to avoid pregnancy. Even in a more recent study patients who had a return to normal LV function after PPCM still demonstrated impaired contractile reserve during a dobutamine challenge test.[58] Therefore these patients become pregnant should be cared for in collaboration with a high-risk obstetric centre. Furthermore, cardiomyopathy can also affect the fetus. In the study by Witlin el al[17] there was no fetal death but there was increased incidence of premature and low-birth weight infants.

Recommendations

It has been recommended that patient with PPCM have an echocardiogram repeated six months after the diagnosis and avoid becoming pregnant until ventricular ejection fraction (EF) has increased to >50% if they are to have a subsequent pregnancy at all.[55,59] The persistence of cardiac dysfunction six months after diagnosis typically suggests an irreversible heart condition and is felt by some authors to be an absolute contraindication to subsequent pregnancy.[55]

Conclusion

Peripartum Cardiomyopathy is an unusual form of dilated cardiomyopathy that is often fatal and strikes in the prime of a young women's life. There is much variation in the course of disease and there is also a lack of good data regarding the etiology of peripartum cardiomyopathy. Although evidence suggests peripartum cardiomyopathy may be the result of a form of myocarditis, there is controversy whether this disease is related to a viral illness or an autoimmune disease of pregnancy. When a pregnant patient has symptoms of heart failure, it is important to pursue the etiology because some may have ischemic or non-ischemic cardiomyopathy or valvular heart disease. In general, treatments for PPCM are similar to those of other cardiomyopathy. Use of immunosuppressive drugs, though logical in the light of immunological reaction, is still controversial and need further trial. Those who don't improve with conventional therapy should be referred to cardiac transplant centre.

The prognosis of PPCM seems to depend on regression of the cardiac changes within the first 6 to 12 months. No studies have confirmed improved long term prognosis with conventional treatment but as is the case with many uncommon disease it is extremely difficult to draw any conclusion about treatment outcome. Subsequent pregnancy should be discouraged, specially those who fail to regain normal left ventricular function.

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Post-streptococcal reactive arthritis : A review

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Abstract

Post-streptococcal reactive arthritis (PSRA) is a poorly understood clinical syndrome in which oligo or polyarthritis occurs following a group A streptococcal pharyngitis. There is lack of universally accepted guideline for diagnosis and management of these patients. But with the resurgence of acute rheumatic fever (ARF) and PSRA over the past decade,[1] it has become increasingly important to understand the definition, presentation and treatment options for these two diseases. This review article presents the clinical profile of PSRA and compare it with ARF so that both can be differentiated from each other, provides an approach to diagnosis and management, including the controversies regarding the management.

Introduction

Acute Rheumatic fever (ARF) is a common and serious public health problem in developing countries and remain a great challenge for both developed and developing countries.[2-7] The original jones criteria for diagnosis of ARF, first introduced in1944, have been modified four times and updated revised criteria have been published in 1992.[5] Arthritis that follows Group A β Hemolytic Streptococcus (GABHS) infection in patients whose illness otherwise does not meet the Jones criteria for diagnosis of ARF, were first described by Crea and Mortimer in 1959[8] who described eighteen patients with non-suppurative arthritis and a history of scarlet fever. Eighty-nine percent (16/18) of these patients did not had carditis or any other major manifestations of Jones criteria for ARF. No aditional reports were published until 1982 when Goldsmith and Long described twelve patients with the same arthritis and they were the first authors who designated this syndrome as Post streptococcal Reactive Arthritis (PSRA).[9] Barash and her group were able to analyze a large number of pediatric patients with ARF and PSRA and identify four criteria that could aid in discriminating between these two entities.

Methodology

This literature review on PSRA is based on articles obtained by internet search. The key words utilized in the strategy include post-streptococcal reactive arthritis, rheumatic heart disease, prophylactic treatment and group A streptococcus. The majority of the articles utilized included case reports and review articles on the topic of ARF and PSRA.

Definition and presentation

PSRA is a clinical syndrome which lacks clear diagnostic criteria and treatment recommendations. The term PSRA refers to a reactive arthritis characterized by a pharyngeal streptococcal infection, a symptom free interval of approximately ten days followed by aseptic inflammation of one or more joints.[10] The arthritis may present like acute septic arthritis with a sudden onset of fever, severe joint pain. The pain is often out of proportion to the degree of physical findings.[11] There is no specific pattern to the joint involvement; it may be monoarticular or polyarticular, migratory, additive or chronic. The arthritis is usually non-destructive and self-limiting, but the symptoms can last for months. The response to aspirin may be less dramatic than with ARF. There may not be clinical evidence of a preceding streptococcal infection and the throat culture may be negative.[10] Table 1 lists the similarities and differences between PSRA and ARF.[12]

Table 1: Comparision between ARF & PSRA

	ARF	PSRA
Epidemiology		
Age	5-15	3-15
Sex ratio (male: female)	1:1	1:1
Chorea	0-30%	0
Erythema marginatum	0-13%	0
Carditis	30-90%	Rare cases reported
Nodules	0-8%	0
Arthritis		
Flitting	+++	-
Persistent		+++
Large joints	+++	+++
Small joints	±	+++
Salicylate response	+++	Inconsistent
Erosive	-	-
Preceding streptococci infection	Requirement for diagnosis	
Approximate interval between infection and disease in days	21	10
Association with B-cell alloantigen D8/17	+++	+++

PSRA refers to patients with a Group A Beta hemolytic streptococcal (GABHS) infection who do not fulfill the modified Jones criteria for the diagnosis of ARF.[13] In other words, PSRA refers to reactive arthritis without cardiac or central nervous system involvement occurring after a streptococcal infection.[10] Nonetheless, there have been several reports of pediatric patients who have subsequently developed carditis.[8,13-15]

Etiology

Reactive arthritis refers to acute, non purulent arthritis complicating an infection occurring elsewhere in the body. In the recent years the term reactive arthritis, has been used primarily to refer to spondyloarthritis following enteric or urogenital infections and occurring predominately in individual with the HLA B 27.[16] The term implies that there is infection in other site of body and arthritis occurs due to immunologic cross reactivity and no organism is detected from joint aspiration. The exact etiology for PSRA remains unclear. To date, there has been no association between PSRA and HLA-B27 in pediatric patients.[17]

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Differential diagnosis

- ARF
- Juvenile rheumatoid arthritis (JRA)
- Spondyloarthropathies
- Viral arthritis
- Septic arthritis
- Reactive arthritis

Investigations

1. Complete blood count with ESR and CRP.
2. A throat culture for GABHS, although this may be negative at the time of presentation.
3. Blood cultures to rule out sepsis.
4. Chlamydial swabs (if patient is sexually active) to rule out chlamydia as a cause of reactive arthritis.
5. Stool culture.
6. Synovial fluid culture, cell count and differential.
7. ASO titre, anti-DNAse B.
8. An electrocardiogram.

Blood work usually indicates an increased white blood cell (WBC) count with elevated levels of ESR and CRP. The synovial fluid WBC count can be as high as >100 x 10^9/L (often in the "Inflammatory Range", normal value = 5.0-15 x 10^9/L) and the synovial and blood cultures are usually negative.

Treatment and complications

Once diagnosis of PSRA is made, antimicrobial therapy should be given to eradicate the GABHS. The long term development of rheumatic heart disease following PSRA has not been estabilished. So, decision regarding long term antibiotic prophylaxis is still controversial. As overt or silent mitral valvular insufficiency may accompany PSRA and patients with PSRA experience recurrences, some researchers recommend that all individuals with PSRA should be treated with long term penicillin antibiotic prophylaxis (as recommended for patients with ARF).[8,13,17,19,20] Others feel that prophylaxis may be considered on a short term basis but discontinued if there is no evidence of carditis or chorea after further evaluation.[12] Arthritis is treated symptomatically by NSAIDS.

Conclusion

It is still unclear whether PSRA represents an early or mild form of ARF or whether it is an entirely separate disease entity. Initial reports in the 1980's suggested that PSRA is a distinct entity from ARF.[16] The long term risk of RHD following PSRA has not been determined, although there have been several case reports of pediatric patients who have developed carditis with subsequent recurrences of PSRA.[13,15,22] We are still unsure how often these patients will develop later cardiac manifestations. Long-term studies are needed to delineate the current epidemiology of streptococcus related diseases, to establish the relationship between PSRA and RHD and to determine the long-term risk of carditis in patients with PSRA. Further genetic and longitudinal studies are needed to determine whether it is useful to distinguish between rheumatic fever and PSRA.[23] Finally, researchers need to assess the efficacy and appropriate duration of antibiotic prophylaxis of individuals with PSRA.[19]

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Computer vision syndrome

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Definition

According to the American Optometry Association, Computer Vision Syndrome (CVS) is the complex of eye and vision problems related to near work which are experienced during or related to computer use. CVS is characterized by visual symptoms which result from interaction with a computer display or its environment. In Most cases, symptoms occur because the visual demands of the task exceed the visual abilities of the individual to comfortably perform the task. If these symptoms occur without significant usage of computer they cannot be termed as CVS.

Introduction

The revolutionary technological advancement have made an impact in almost every aspect of our lives. Our daily tasks, office works, medical facilities, accounting, designing, database management and experimental works have all been greatly facilitated by this rapid pace of development in computer technology. The personal computer (P.C) thus become a single device which has made a great revolution in all aspect of our lives. Approximately 100 million people in the world are using computers now. In USA 71% of children work with computer in school. In India about 20 million PCs are in use. In our country the use of computer is increasing day by day. The cost of diagnosis and treatment of CVS in USA alone exceeds 2 billion annually which indicates a reflection of importance to think about this new emerging medical and visual problem.

Pathophysiology

CVS is contributed by several factors:

1. Decreased blinking reflex: Studies have shown that the normal blink rate in human eyes is 16-20 blinks/min. For persons working on the computer it is decreased to 6-8 blinks/minute. This leads to dry eyes.
2. Prolonged near focusing efforts: Puts strain on ciliary muscles of the eye and leads to a feeling of tiredness in the eyes. This can be a setting for early presbyopia.
3. Repeated head posture change/fixation in a wrong posture: Puts strain on the neck muscles and cervical spine.

Symptoms

Eyestrain (non-specific ocular discomfort), fatigues, headache, blurred near vision, blurred distant vision, dry or irritated eyes, neck pain and/or backaches, diplopia (double vision), difficulty in re-focusing the eyes.

Visual/ocular signs

Accommodative disorders, early presbyopia, binocular vision dysfunctions, refractive errors: hyperopia, astigmatism, myopia, dry eyes, conjunctival congestion (redness).

CVS associated musculo-skeletal disorders, Cumulative Trauma Disorder (CTD) or Repetitive Strain Injury (RSI): Carpal Tunnel Syndrome, bursitis, neck tension syndrome (muscle strain), tendon disorders- De quervain's disease, tenosynovitis- trigger finger.

Environmental factors of computer workstations

Contrast and resolution of the display, viewing distances and angles, adjustability of workstation, room lighting, sustained viewing.

Relationship of CVS to RSI or CTD (Musculoskeletal disorders)

Symptoms are work related and associated with repetitive activity. Problems are related to disorders of muscles, tendons, bones, or nerves. Problems occur or are aggravated by repeated movements. A lengthy period of time is required for the problems to develop and for the individual to recover.

Diagnostic tests

Tear film Break-up time (TF-BUT): Examined under slit-lamp with a red free light. Fluorescein dye is used. Normal->10 seconds.

Schirmer Test-1: Special graduated paper strip is used. Normal value- >10mm in 5 minutes.

Rose Bengal Staining: To detect corneal and conjunctival epithelial defect in dry eyes. A positive result is highly significant for CVS.

Management

Proper History

Symptoms, duration, aggravating and relieving factors. Nature of work, computer exposure time. Existing workstation setting, furniture, lighting, etc. Per-existing ocular or musculoskeletal diseases. Examinations. Refractive status-near, intermediate and distant. Ocular motility, Versions and Vergence. Thorough neuromuscular work-up.

Differential diagnosis

1. Cervical spondylitis
2. Migraine
3. Anxiety Neurosis
4. Dry Eye Syndrome

Investigations

1. X-ray of cervical and lumbo-sacral spine for exclusion of pre-existing spinal disease
2. TF-BUT, Schirmer-1
3. Rose Bengal test

Treatment

A. Counseling regarding
   1. Awareness building
   2. Work environmental modification

B. Computer eyewear

C. Tear substitute.

A. Counseling: 10 steps for relieving computer eye strain:

1. Get a computerized eye exam before start using computer. Repeat once a year.
2. Use proper lighting. Eliminate exterior light by closing

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drapes, shades or blinds. Reduce interior lighting by using lower intensity bulbs and tubes.

3. Minimize glare: To install an anti-glare screen on your monitor. Paint bright white walls a darker color with a matte finish.
4. Upgrade your display. Use LCD monitor instead of a CRT monitor.
5. Adjust the brightness and contrast of your computer screen.
   - The brightness of the screen should be the same as the work environment. Contrast between screen background and on- screen characters should be high.
   - The text size and color should be optimized for the most comfort.
6. Blink more often.
   - Every 20 minutes, blink 10 times by closing your eyes as if falling asleep (very slowly).
7. Exercise your eyes: Follow 20-20-20 rule i.e after every 20 minutes, look at 20 feet distance for 20 seconds.
8. Take frequent breaks- two 15-minute breaks -four additional five-minute "mini-breaks" throughout the work day (6-8 hrs).
9. Modify your workstation.
   - Proper posture during computer work. Ergonomic furniture. Position computer screen 20 to 24 inches from your eyes. The center of your screen should be about 10 to 15 degrees below your eyes. Top of the screen tilted back slightly (10-20 degree) away from the operator.
10. Consider computer eyewear and avoid contact lens use during computer work.

B. Computer eyewear

Customized eyeglasses specific for use during work on a computer screen. Anti-reflective coating in the lenses should be used. Presbyopia- Single vision lenses. Intermediate/near bifocals. Special multifocal lenses. Computer Eyewear should also be considered if a person have CVS related symptoms in: Latent hypermetropia, Low astigmatism, Heterophoria, Convergence insufficiency, Disorders of accommodation before 40. Person may not require spectacle correction for general vision needs.

C. Tear Substitute

Considered if 1. Symptoms occur/aggravated during computer work. 2. TF-BUT- <10 seconds. 3. Schimer-1- < 10 mm in 5 minutes. 4. Rose Bengal Test- Positive. Commercially available Tear Substitutes- Sodium Carboxymethyle Cellulose. Povidon Solution, Dextran 70 solution 2%, Hydroxypropyl Methyle Cellulose (Hypromellose) 0.5% etc.

Children and computer vision syndrome

The average American child now spends 1 to 3 hours per day on the computer. 90% of school age children have computer access at home or in school. 54 million children in the United States alone use a computer at home or in school. 25% to 30% of computer user children need corrective eyewear. A study in Singapore found that in 3 years the percentage of 7 to 9 years old with myopia had doubled, to 34%.

- Twenty years ago, most children played outside, and their distant vision was more important.
- Today it is a "Near Point World". In Bangladesh also, Specially, in upper middle and upper class, children spend on an average 1-3 hrs in front of a computer/video display terminal.
- Children have a limited degree of self awareness. Children are very adaptable. Obviously the size of the children are smaller than adults.

Five tips for preventing computer vision syndrome in children

1. Before starting school, every child should have a comprehensive eye exam, including near point (computer and reading) and distance testing.
2. Workstations should be arranged to suit a child-not an adult.
3. The recommended distance between the monitor and the eye for children is 18-28 inches.
4. Any behavior that indicates potential problems: Parents and teachers should be aware of eye redness. Frequent rubbing of the eyes. Head turns and other unusual postures, or complaints of blurriness or eye fatigue, avoidance of the computer.
5. Most importantly, have your child's eyes examined by a computer vision specialist.

Conclusion

In this computer era there is no scope to avoid this modern technology rather its use is expanding everyday in the perspective of digital Bangladesh along with global digitalization. If we can make awareness among the computer users regarding its proper uses in respect of optimum amount of work time, proper setup & work positions, related health & eye problems which may occur and some training and tips to the users to solve the common problems can be provided, at least 50% of the computer vision syndrome & closely related problems will be minimized. Rest of the problems which may be more specific & individualized need close co-operation between computer users and expert, trained ophthalmologist so that maximum work can be done with comfort with or without minimum discomfort. Here another point should be kept in the mind of the guardians of the children who are very much habituated to work on a video display terminal for hours together for their any behavioral change, ocular complaints, headache, even visual defects which may be ignored by the children easily for their more adaptiveness. But the parents should be very consious to identify their problems and should take proper measure by consulting with the computer vision syndrome specialists for the prevention of permanent disabilities.

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New ideas and concepts
Rice bran : A nutrient-dense mill-waste for human nutrition

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Introduction

Rice is the staple food for seventy percent of world population supplying as much as half of its energy needs (Chang TT,19 76). In Asian countries including China, India, Indonesia, Bangladesh, Vietnum, Japan, Thailand, Myanmar and Pakistan, rice is produced and eaten as the predominant source of energy.

Common rice, scientifically called Oryza sativital has more than 8,000 named species that are grown all over the world. Rice is sometimes classified as either short grain, medium grain or long grain. Short grain rice has the highest starch content and is stickiest when cooked as it contains more amylopectin; while long grain is lighter and tends to remain separate when cooked as it contains more amylose. The characteristics of medium grain rice fall between the other two types.

Rice is also classified according to the degree of milling; brown rice is the whole grain with only its inedible outer hull removed keeping its nutrient-rich bran layer intact (Marshall, 1994). In contrast, white rice is both milled and polished, which removes the bran and germ contents along with all the nutrients that reside within these important layers.

White rice is the most popular form for human consumption although brown rice is eaten in some communities.

History

Rice is an ancient crop. It is believed that rice cultivation first began in China around 6,000 years ago, but this period could be as long as 9,000 years according to recent archaeological investigations based on discoveries of primitive rice seeds and farm tools (Plant Rice 2001, Marshall, 1994). Historically, rice is thought to be a staple grain in Asia which was introduced to Greece by the Arab conquest of Asian land and to India by Alexander the Great.

During the Moorist conquent of Spain in the 8th century rice appeared in the Spanish markets while the crusaders brought it to France. In 17th century rice was introduced to South America by the Spanish settlers in this continent. A significant proportion of world production of rice comes from Asian countries where it plays an important role in the food culture of the people. Thailand, Vietnam, India and Pakistan are the three largest exporters of rice worldwide.

Rice bran

Rice bran constitutes the brown covering of the grain beneath the outer husk. It constitutes 8% of the weight of the whole grain and contains most of the nutrients (65%). During milling process rice bran containing nutrients is completely removed. Around 60 million metric tons of rice bran is produced worldwide each year and almost all of it is either thrown away or used as low level animal and poultry feed. The cost of rice bran is 0.2 cent per pound in the international market and 0.1 cent (Tk. 5-7) in Bangladesh. Rice bran is a very rich source of nutrients containing vitamins, minerals, oils, wax, trace elements, antioxidants, phytosterols, and phytochemicals. It is also an energy-dense (373 cal/1 cup or 118g), high protein (15.8 g/1cup), high fiber (99%), low sodium, low sugar (1.1g) cereal containing zinc, iron, folic acid and other nutrients but no cholesterol. It is a good source of manganese, magnesium, vitamins B_1, B_2, B_6, and minerals i.e., potassium, calcium, phosphorus, and pantothenic acid. Recent information indicate that its high nutrient contents are hard to ignore.

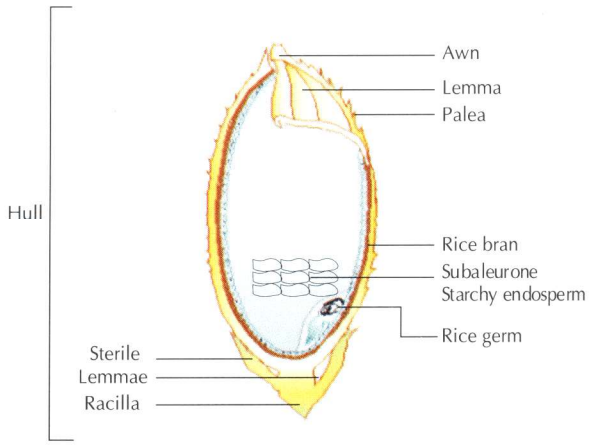

Figure: Structure of mature rice grain

Brown rice

Brown rice is un-milled or partially milled rice with removal of the outermost layer of the rice grain (husk) which becomes rancid more quickly. Parboiling destroys partially or completely the lipase activity of the rice bran thus providing longer shelf life of brown rice and helps preventing development of rancidity. Parboiled brown rice was traditionally and historically been consumed in most part of Bangladesh. With the advancement of industrialization, milled white rice has taken over the place of parboiled brown rice in this setting in past few decades.

The limitations of using brown rice with intact bran are related to its unpalatability due to high fibre content and short shelf life resulting from rapid hydrolysis of its fat content by the grain enzyme lipase (Champagne, 1994). Within 1 hour of separating the bran from the grain during milling the material turns rancid liberating toxic free fatty acids. These shortcomings have now been overcome by reducing the fibre content and destroying the lipolytic activity using an advanced stabilizing technology; the resulting material thus obtained is called "stabilized" rice bran which has a good taste, readily soluble with a longer shelf life of one year.

Rice bran in its crude or stabilized form is a potential dietary source which can be used as a nutritional supplement to

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replace and prevent nutrient deficiencies in malnourished children. However, no studies have yet been conducted to evaluate its role from the nutritional and metabolic point of view. Nevertheless, stabilized rice bran has been marketed as a popular brand of nutraceuticals for the management of diabetes, hypertension, dislipidemia, and arthritis (Anderson, 2000; Cade JE, 2007; Ensminger, 1983; Erkkila, 2005).

Nutritional deficiencies and rice bran

Nutritional deficiency among children is widespread in the developing countries especially in Asia. Of the malnourished children, about 70% live in Asia accounting for the highest concentration of malnourished children in the world. Fifty percent of deaths among these children are attributed to malnutrition which severely affects growth, cognition, motor and social development of young children. A large proportion of hidden burden of malnutrition among children are due to single or multiple micronutrient deficiencies. Factors influencing micronutrient deficiency includes poor body stores at birth, poor dietary intake, and losses from the body during illnesses. Protein energy malnutrition (PEM) along with micronutrient deficiency among young children and pregnant women of developing countries is the most important risk factor for disability and death. Micronutrient supplementation, food fortification, complementary food at home, and several other strategies have been implemented to minimize micronutrient deficiencies in children in many countries including Bangladesh; these interventions have been found to be efficacious and effective although malnutrition remains quite high in these countries.

Nutrient loss during milling

Brown rice is an excellent source of energy, vitamins, and minerals (Fortin, 1996). However, the process of milling and polishing that converts brown rice into white rice destroys 67% of the vitamin B_3, 80% of the vitamin B_1, 90% of the vitamin B_6, half of the manganese, half of the phosphorus, 60% of the iron, and all of the dietary fiber and essential fatty acids. By law in the United States, fully milled and polished white rice must be "enriched" with vitamins B_1, B_3 and iron. But the form of these nutrients when added back into the processed rice is not the same as in the original unprocessed grain, and at least 11 lost nutrients are not replaced in any form even with rice "enrichment".

Table 1: The nutrient contents of stabilized rice bran produced in US

Macronutrients	%
Protein	12-16%
Fat	18-23%
Total carbohydrates	45-55%
Total dietary fibre	23-35%
Soluble fibre	2-6%
Ash	7-10%
Moisture	4-8%
Free fatty acids	< 4%
Calories/100 g	33.50

Micronutrients (g/100g)	
Vitamin A; Carotenoids (mcg/100g)	
Beta carotene	37.00
Alpha carotene	0.40
Lycopene	2.30
Lutein	63.8
Zeaxanthin	18.40
Precryptoxanthin	7.40

Other micronutrients (mg/100g)	
Folic acid (ug/100g)	
Biotin (ug/100g)	14.10
Choline	104.80
Inositol	1496.00
Gama oryzanol	245.15

Physical	
Colour	Tan
Appearance	Powder
Bulk density (g/cc)	0.47
Mesh	78%

Microbiological	
Total plate count	10,000 cfu/g
Total plate count	10,000 cfu/g
E. coli	< 10 cfu/g
Yeast/mold, max	100 cfu/g

Minerals (mg/100g)	
Sodium	8.0
Potassium	1573.00
Calcium	40.00
Magnesium	727.00
Phosphorus	1591.00
Manganese	25.60
Iron	7.70
Copper	0.27
Zinc	5.50
Chromium (ppm)	< 1.0
Total sugars (g/100g)	8.0

Phytosteroids (mg/100g)	
Beta sitosterol	167.67
Stigmasterol	62.64
Campesterol	96.23
Brassicasterol	14.61

Vitamin B complex (mg/100g)	
Vitamin B_1	2.70
Vitamin B_2	0.28
Vitamin B_3	46.90
Vitamin B_5	3.98
Vitamin B_6	3.17
Vitamin B_{12}	(ug/100g)<0.5
Vitamin C	(mg/100g) <0.5

Vitamin E Complex (mg/100g)	
Tocopherol (T)	12.00
Tocopherols (T3)	13.60

Locally produced rice bran may have similar nutritional values to those of commercially produced rice bran as commercially produced rice bran does not fortify rice bran with any nutritional ingredients.

Health benefits of rice bran and brown rice

The difference between brown rice and white rice is not just color. Brown rice is obtained when only the outermost layer, the hull, is removed from the whole grain (paddy rice) during milling. This process is the least damaging to the nutritional value of the rice and avoids the unnecessary loss of nutrients that occurs with further processing. Further milling of brown rice to remove the bran coat and most of the germ layer will produce white rice which is devoid of the bran-based nutrients. Further milling of rice called 'polishing' will result in more whiter rice we buy from the market. During fine polishing the thin aleurone layer containing important nutrients and oil is removed from the grain. The fat layer is removed to extend the shelf life of the grain since this fat is liable to be decomposed by the grain enzyme lipase to produce free fatty acids and leading to the development of rancidity. Thus the final white rice is simply a refined starch that is largely bereft of its original nutrients. Health benefits derived from consumption of rice containing its bran layer have been described in a number of studies (Erkkila, 2005; Ensminger, 1983).

Prevention of heart diseases

Plant lignans are one type of phytonutrient abundantly present in whole grain rice, these are transformed by the resident microflora in the colon into mammalian lignans, such as enterolactone that is known to protect against breast cancers as well as heart disease (Liu, 2004; Lie, 2003). Lignans are present in fruits (nuts, seeds, berries) and vegetables, and beverages such as coffee, tea and wine. Higher levels of protective enterolactone were found in postmenopausal Scandinavian women eating whole grain meals with cabbage and other leafy vegetables.

Risk of type 2 diabetes

Regular consumption of brown rice and other whole grains are known to reduce the risks of developing adult type diabetes (McKown, 2004; Van Dam, 2006). Risk of type 2 diabetes was reduced by 31% in black women eating whole grains compared to those eating the least of these foods. According to the FDA, a food containing at least 51% whole grains by

weight is likely to display a health benefit linking lower risk of heart disease and certain cancers. In a 8 years study involving 41,186 black women participants, an inverse association between magnesium, calcium and major food sources relating to type 2 diabetes was documented (Van Dam RM, Hu FB, Diabetes Care). Similar observations were made in white population earlier. Whole grain rice is a rich source of magnesium, a mineral acting as a co-factor for regulating many enzymatic reactions regulating glucose and insulin metabolism.

Risk of metabolic syndrome

Refined grains and the foods made from them (e.g., white breads, cookies, pastries, pasta and rice) are now being linked not only to weight gain but to increased risk of insulin resistance (the precursor of type 2 diabetes) and the metabolic syndrome (a strong predictor of both type 2 diabetes and cardiovascular disease), while eating more wholegrain foods is being shown to protect against all these ills.

Common features of the metabolic syndrome include visceral obesity, low levels of protective HDL cholesterol, high triglycerides, and high blood pressure. In one of the most recent studies, which appeared in diabetes care, researchers who analyzed data on over 2,800 participants in the Framingham Offspring study, found that the prevalence of both insulin resistance and the metabolic syndrome was significantly lower among those eating the most cereal fiber from whole grains compared to those eating the least one (McKown, 2004). Prevalence of the metabolic syndrome was 38% lower among those with the highest intake of fiber from whole grains. Conversely, study subjects whose diets had the highest glycemic index and glycemic load, both of which are typically low in whole foods and high in processed refined foods, were 141% more likely to have the metabolic syndrome compared to those whose diets had the lowest glycemic index and glycemic load. The researchers concluded, "Given that both a high cereal fiber content and lower glycemic index are attributes of wholegrain foods, recommendation to increase wholegrain intake may reduce the risk of developing the metabolic syndrome."

It is observed that a way of eating that relies on the healthiest foods from all the food groups-the whole foods that contain the healthiest fats, carbohydrates and proteins-is the most effective, intelligent, and most enjoyable way to not only lower the risk of developing the metabolic syndrome, but to stay slim, vital and attractive throughout a long and healthy life.

Effects on trace elements

Magnesium, another nutrient for which brown rice is a good source, has been shown in studies to be helpful for reducing the severity of asthma, lowering high blood pressure, reducing the frequency of migraine headaches, and reducing the risk of heart attack and stroke (Jensen, 2004). Magnesium helps regulate nerve and muscle tone by balancing the action of calcium. In many nerve cells, magnesium serves as nature's own calcium channel blocker, preventing calcium from rushing into the nerve cell and activating the nerve. By blocking calcium's entry, magnesium keeps our nerves relaxed. If our diet is deficient in magnesium, calcium can gain free entry, and nerve cells can become over activated, sending too many messages and causing excessive contraction. Insufficient magnesium can thus contribute to high blood pressure, muscle spasms (including spasms of the heart muscle or the spasms of the airways symptomatic of asthma), and migraine headaches, as well as muscle cramps, tension, soreness and fatigue (Tabak, 2005).

Magnesium, as well as calcium, is necessary for healthy bones. About two-thirds of the magnesium in the human body is found in our bones. Some helps give bones their physical structure, while the rest is found on the surface of the bone where it is stored for the body to draw upon as needed. Brown rice can help keep those storage sites replenished and ready to meet the body's demands. A cup of brown rice can provide 21.0% of the daily value for magnesium.

In addition to niacin it supplies, brown rice may also help raise blood levels of nitric oxide, a small molecule known to improve blood vessel dilation and to inhibit oxidative (free radical) damage of cholesterol and the adhesion of white cells to the vascular wall (two important steps in the development of atherosclerotic plaques). A study published in the British Journal of Nutrition suggests that diets high in rice protein can help protect against atherosclerosis by increasing blood levels of nitric oxide (Ni W, 2003).

In this study, when researchers gave rice bred to mice to be apoliprotein-E deficient a purified diet containing either casein, the principal protein in dairy products, rice protein or soya protein, the mice given casein developed the largest atherosclerotic lesions. In humans as well as animals, apolipoprotein E plays an important role in cholesterol transport, so a deficiency of this protein increases risk for the development of atherosclerosis. Mice given rice or soya protein fared much better. In trying to understand why, the researchers evaluated blood levels of nitric oxide. Mice fed either rice or soya protein diets were found to have increased blood levels of L-arginine (the amino acid that the body uses to produce nitric oxide) and nitric oxide metabolites when compared to those given casein-based feed. However, the L-arginine content of the rice and soya diets was not high enough to explain the amount of protective benefit they conferred, so the researchers concluded that these foods must also contain other cardioprotective compounds.

Manganese-energy production plus antioxidant protection: Just one cup of brown rice can provide with 88.0% of the daily requirements for manganese. This trace mineral helps produce energy from protein and carbohydrates and is involved in the synthesis of fatty acids, which are important for a healthy nervous system, and in the production of cholesterol, which is used by the body to produce sex hormones. Manganese is also a critical component of a very important antioxidant enzyme called superoxide dismutase.

Superoxide dismutase (SOD) is found inside the body's mitochondria (the oxygen-based energy factories inside most of our cells) where it provides protection against damage from the free radicals produced during energy production.

Body weight reduction

A study published in the American Journal of Clinical Nutrition underscores the importance of choosing whole grains such as brown rice rather than refined grain, i.e., white rice, to maintain a healthy body weight. In this study, weight gain was inversely associated with the intake of high-fiber, whole-grain foods but positively related to the intake of refined-grain foods in 74,000 female nurses aged 38-63 years over a 12 year period (Anderson, 2000). Not only did women who consumed more whole grains consistently weigh less but they were also

49% less likely to gain weight compared to those eating foods made from refined grains.

Effects of fiber and selenium

Brown rice is a concentrated source of the fiber needed to minimize the amount of time, cancer-causing substances spend in contact with colon cells, and being a very good source of selenium, a trace mineral that has been shown to substantially reduce the risk of colon cancer (Most, 2005; Ni W, 2003).

In addition to supplying 14.0% of the daily value for fiber, a cup of cooked brown rice provides 27.3% of the DV for selenium, an important benefit since many do not get enough selenium in their diets. Selenium is an essential component of several major metabolic pathways, including thyroid hormone metabolism, antioxidant defense systems and immune function. Accumulated evidence from prospective studies, intervention trials and studies on animal models of cancer has suggested a strong inverse correlation between selenium intake and cancer incidence. Several mechanisms have been suggested to explain the cancer-preventive activities of selenium. Selenium has been shown to induce DNA repair and synthesis in damaged cells, to inhibit the proliferation of cancer cells, and to induce their apoptosis, the self-destructive sequence the body uses to eliminate worn out or abnormal cells (Liu, 2004).

In addition, selenium is incorporated at the active site of many proteins, including glutathione peroxidase, which is particularly important for cancer protection. One of the body's most powerful antioxidant enzymes, glutathione peroxidase is used in the liver to detoxify a wide range of potentially harmful molecules. When levels of glutathione peroxidase are too low, these toxic molecules are not disarmed and wreak havoc on any cells with which they come in contact, damaging their cellular DNA and promoting the development of cancer cells.

Not only does selenium play a critical role in cancer prevention as a cofactor of glutathione peroxidase, selenium also works with vitamin E in numerous other vital antioxidant systems throughout the body. These powerful antioxidant actions make selenium helpful in the prevention not only of cancer, but also of heart disease, and for decreasing the symptoms of asthma and the pain and inflammation of rheumatoid arthritis.

Cholesterol lowering effects

The oil in whole brown rice lowers cholesterol. In one study from Louisiana State University rice bran and rice bran oil were found to reduce cholesterol levels in volunteers with moderately elevated cholesterol levels (Most, 2005).

The study, published in the American Journal of Clinical Nutrition was divided into two parts. First, 26 subjects ate a diet including 13-22g of dietary fiber each day for three weeks, after which 13 switched to a diet that added defatted rice bran to double their fiber intake for five weeks. In the second part of the study, a randomized crossover trial, 14 subjects ate a diet with rice bran oil for 10 weeks. While the diet including only defatted rice bran did not lower cholesterol, the one containing rice bran oil lowered LDL cholesterol by 7%. Since all the diets contained similar fatty acids, the researchers concluded that the reduction in cholesterol seen in those receiving rice bran oil must have been due to other constituents such as the unsaponifiable compounds found in rice bran oil. The scientists suggest that the unsaponifiables present in rice bran oil could become important functional foods for cardiovascular health.

But why extract just one beneficial compound from brown rice when you can reap all the cardioprotective benefits supplied by the matrix of nutrients naturally present in this delicious whole food? In addition to unsaponifiables, this whole grain also supplies hefty doses of heart-healthy fiber, magnesium, and B vitamins.

Cardiovascular benefits for postmenopausal women

Eating a serving of whole grains, such as brown rice, at least 6 times each week is an especially good idea for postmenopausal women with high cholesterol, high blood pressure or other signs of cardiovascular disease (CVD) (Ysai, 2004). A 3-year prospective study of over 200 postmenopausal women with CVD, published in the American Heart Journal, shows that those eating at least 6 servings of whole grains each week experienced both: slowed progression of atherosclerosis and less progression in stenosis, the narrowing of the diameter of arterial passageways. The women's intake of fiber from fruits, vegetables and refined grains was not associated with a lessening in CVD progression.

Phytonutrients effects better than vegetables and fruits

Research reported at the American Institute for Cancer Research (AICR) International Conference on Food, Nutrition and Cancer, by Rui Hai Liu, M.D., PhD, and his colleagues at Cornell University shows that whole grains, such as rice, contain many powerful phytonutrients whose activity has gone unrecognized because research methods have overlooked them (Liu, 2003). Despite the fact that for years researchers have been measuring the antioxidant power of a wide array of phytonutrients, they have typically measured only the "free" forms of these substances, which dissolve quickly and are immediately absorbed into the bloodstream. They have not looked at the "bound" forms, which are attached to the walls of plant cells and must be released by intestinal bacteria during digestion before they can be absorbed.

Phenolics, powerful antioxidants that work in multiple ways to prevent disease, are one major class of phytonutrients that have been widely studied. Included in this broad category are such compounds as quercetin, curcumin, ellagic acid, catechins, and many others that appear frequently in the health news. When Dr. Liu and his colleagues measured the relative amounts of phenolics, and whether they were present in bound or free form, in common fruits and vegetables like apples, red grapes, broccoli and spinach, they found that phenolics in the "free" form averaged 76% of the total number of phenolics in these foods. In whole grains, however, "free" phenolics accounted for less than 1% of the total, while the remaining 99% were in "bound" form. In his presentation, Dr. Liu explained that because researchers have examined whole grains with the same process used to measure antioxidants in vegetables and fruits-looking for their content of "free" phenolics"-the amount and activity of antioxidants in whole grains has been vastly underestimated.

Despite the differences in fruits', vegetables' and whole grains' content of "free" and "bound" phenolics, the total antioxidant activity in all three types of whole foods is similar, according to Dr. Liu's research. His team measured the antioxidant activity of various foods, assigning each a rating based on a formula (micromoles of vitamin C equivalent per gram). Broccoli and spinach measured 80 and 81, respectively; apple and banana measured 98 and 65; and of the whole grains tested, corn measured 181, whole wheat 77, oats 75, and brown rice 56.

Dr. Liu's findings may help explain why studies have shown that populations eating diets high in fiber-rich whole grains consistently have lower risk for colon cancer, yet short-term clinical trials that have focused on fiber alone in lowering colon cancer risk, often to the point of giving subjects isolated fiber supplements, yield inconsistent results. The explanation is most likely that these studies have not taken into account the interactive effects of all the nutrients in whole grains-not just their fiber, but also their many phytonutrients. As far as whole grains are concerned, Dr. Liu believes that the key to their powerful cancer-fighting potential is precisely their wholeness. A grain of whole wheat consists of three parts-its endosperm (starch), bran and germ. When wheat-or any whole grain-is refined, its bran and germ are removed.

Although these two parts make up only 15-17% of the grain's weight, they contain 83% of its phenolics. Dr. Liu says his recent findings on the antioxidant content of whole grains reinforce the message that a variety of foods should be eaten for good health. "Different plant foods have different phytochemicals," he said. "These substances go to different organs, tissues and cells, where they perform different functions. What our body needs to ward off disease is this synergistic effect - this teamwork - that is produced by eating a wide variety of plant foods, including whole grains."

Lignans protect against heart disease

One type of phytonutrient especially abundant in whole grains including brown rice are plant lignans, which are converted by friendly flora in our intestines into mammalian lignans, including one called enterolactone that is thought to protect against breast and other hormone-dependent cancers as well as heart disease. In addition to whole grains, nuts, seeds and berries are rich sources of plant lignans, and vegetables, fruits, and beverages such as coffee, tea and wine also contain some. When blood levels of enterolactone were measured in over 850 postmenopausal women in a Danish study published in the Journal of Nutrition, women eating the most whole grains were found to have significantly higher blood levels of this protective lignan (Johnsen, 2004). Women who ate more cabbage and leafy vegetables also had higher enterolactone levels.

Effects of fiber against breast cancer

Diet rich in fiber from whole grains, such as brown rice, and fruit offered significant protection against breast cancer for pre-menopausal women in 35,972 participants in the UK (Cade JE, Burley VJ, et al., International Journal of Epidemiology, 2007). Pre-menopausal women eating the most fiber (>30 grams daily) more than halved their risk of developing breast cancer, enjoying a 52% lower risk of breast cancer compared to women whose diets supplied the least fiber (<20 grams/day). Fiber supplied by whole grains offered the most protection. Pre-menopausal women eating the most whole grain fiber (at least 13 g/day) had a 41% reduced risk of breast cancer, compared to those with the lowest whole grain fiber intake (4 g or less per day).

Fiber from fruit was also protective. Pre-menopausal women whose diets supplied the most fiber from fruit (at least 6 g/day) had a 29% reduced risk of breast cancer, compared to those with the lowest fruit fiber intake (2 g or less per day).

Table 2: Shows the fibre contents of different grains

Food fiber content in grams		
Oatmeal	1 cup:	3.98
Whole wheat bread	1 slice:	2
Whole wheat spaghetti	1 cup	6: 3
Brown rice	1 cup:	3.5
Barley	1 cup:	13.6
Buckwheat	1 cup:	4.54
Rye	1/3 cup:	8.22
Corn	1 cup:	4.6
Apple with skin	1 medium:	5.0
Banana	1 medium:	4.0
Blueberries	1 cup:	3.92
Orange	1 large:	4.42
Pear	1 large:	5.02
Prunes	1/4 cup:	3.02
Strawberries	1 cup:	3.82
Raspberries	1 cup:	8.36

Table 3: Full-fat, stabilized rice bran should meet the following specifications for human food quality (by AOAC Standards)

Fat	Min. 16%
Protein	Min. 13%
Total dietary fibre	Min. 20%
Crude fibre	Max. 9%
Ash (parboiled rice bran)	Max. 10%
Moisture	Max. 12%
FFA (in crude fat extract)	Max. 4%
Silica (SiO_2)	Max. 0.1%
$CaCO_3$	Max. 2%

Depending on preliminary success, longterm epidemiologic studies need to be conducted to examine the nutritional impact of rice bran and its products in malnourished children in Bangladesh.

Effects on gallstone prevention

Preventive effects of insoluble fiber, such as brown rice, on development of gallstones were reported in a study published in the American Journal of Gastroenterology.

Studying the overall fiber intake and types of fiber consumed over a 16 year period by over 69,000 women in the Nurses Health Study, researchers found that those consuming the most fiber overall (both soluble and insoluble) had a 13% lower risk of developing gallstones compared to women consuming the fewest fiber-rich foods (Tsai, 2004). Those eating the most foods rich in insoluble fiber gained even more protection against gallstones: a 17% lower risk compared to women eating the least. And the protection was dose-related; a 5-gram increase in insoluble fiber intake dropped the risk by 10%. How do foods rich in insoluble fiber help prevent gallstones? Researchers think insoluble fiber not only speeds intestinal transit time (how quickly food moves through the intestines), but reduces the secretion of bile acids (excessive amounts contribute to gallstone formation), increases insulin sensitivity and lowers triglycerides (blood fats). Abundant not just in brown rice but all whole grains, insoluble fiber is also found in nuts and the edible skin of fruits and vegetables including tomatoes, cucumbers, many squash, apples, berries, and pears. In addition, beans provide insoluble as well as soluble fiber.

Protection against childhood asthma

According to the American Lung Association, almost 20 million Americans suffer from asthma, which is reported to be

responsible for over 14 million lost school days in children, and an annual economic cost of more than $16.1 billion. Increasing consumption of whole grains and fish could reduce the risk of childhood asthma by about 50%, suggests the International study on Allergy and Asthma in childhood (Tabak C, Wijga AH, Thorax).

The researchers, from the Dutch National Institute of Public Health and the Environment, Utrecht University, University Medical Center Groningen, used food frequency questionnaires completed by the parents of 598 Dutch children aged 8-13 years (Tabak, 2005). They assessed the children's consumption of a range of foods including fish, fruits, vegetables, dairy and whole grain products.

While no association between asthma and intake of fruits, vegetables, and dairy products was found (a result at odds with other studies that have supported a link between antioxidant intake, particularly vitamins C and E, and asthma), the children's intake of both whole grains and fish was significantly linked to incidence of wheezing and current asthma. In children with a low intake of fish and whole grains, the prevalence of wheezing was almost 20%, but was only 4.2% in children with a high intake of both foods. Low intake of fish and whole grains also correlated with a much higher incidence of current asthma (16.7%), compared to only a 2.8% incidence of current asthma among children with a high intake of both foods. After adjusting results for possible confounding factors, such as the educational level of the mother, and total energy intake, high intakes of whole grains and fish were found to be associated with a 54% and 66% reduction in the probability of being asthmatic, respectively.

The probability of having asthma with bronchial hyperresponsiveness (BHR), defined as having an increased sensitivity to factors that cause narrowing of the airways, was reduced by 72 and 88% when children had a high-intake of whole grains and fish, respectively. Lead researcher, Cora Tabak commented, "The rise in the prevalence of asthma in western societies may be related to changed dietary habits." The Standard American Diet is sorely deficient in the numerous anti-inflammatory compounds found in fish and whole grains, notably, the omega-3 fats supplied by cold water fish and the magnesium and vitamin E provided by whole grains. One caution: wheat may need to be avoided as it is a common food allergen associated with asthma.

Meta-analysis explains whole grains' health benefits

In many studies, eating whole grains, such as brown rice, has been linked to protection against atherosclerosis, ischemic stroke, diabetes, insulin resistance, obesity and premature death. A new study and accompanying editorial, published in the American Journal of Clinical Nutrition explains the likely reasons behind these findings and recommends at least 3 servings of whole grains should be eaten daily (Jensen MK, 2004). Whole grains are excellent sources of fiber. In this meta-analysis of 7 studies including more than 150,000 persons, those whose diets provided the highest dietary fiber intake had a 29% lower risk of cardiovascular disease compared to those with the lowest fiber intake. But it's not just fiber's ability to serve as a bulking agent that is responsible for its beneficial effects as a component of whole grains. Wheat bran, for example, which constitutes 15% of most whole-grain wheat kernels but is virtually non-existent in refined wheat flour, is rich in minerals, antioxidants, lignans, and other phytonutrients-as well as in fiber.

In addition to the matrix of nutrients in their dietary fibers, the whole-grain arsenal includes a wide variety of additional nutrients and phytochemicals that reduce the risk of cardiovascular disease. Compounds in whole grains that have cholesterol-lowering effects include polyunsaturated fatty acids, oligosaccharides, plant sterols and stanols and saponins. Whole grains are also important dietary sources of water-soluble, fat-soluble and insoluble antioxidants. The long list of cereal antioxidants includes vitamin E, tocotrieonols, selenium, phenolic acids and phytic acid. These multifunctional antioxidants come in immediate-release to slow-release forms and thus are available throughout the gastrointestinal tract over a long period after being consumed. The high antioxidant capacity of wheat bran, for example, is 20-fold that of refined wheat flour (endosperm).

Although the role of antioxidant supplements in protecting against cardiovascular disease has been questioned, prospective population studies consistently suggest that when consumed in whole foods, antioxidants are associated with significant protection against cardiovascular disease. Because free radical damage to cholesterol appears to contribute significantly to the development of atherosclerosis, the broad range of antioxidant activities from the phytonutrients abundant in whole grains is thought to play a strong role in their cardio-protective effects. Like soybeans, whole grains are good sources of phytoestrogens, plant compounds that may affect blood cholesterol levels, blood vessel elasticity, bone metabolism and many other cellular metabolic processes. Whole grains are rich sources of lignans that are converted by the human gut to enterolactone and enterodiole. In studies of Finnish men, blood levels of enterolactone have been found to have an inverse relation not just to cardiovascular-related death, but to all causes of death, which suggests that the plant lignans in whole grains may play an important role in their protective effects.

Lower insulin levels may also contribute to the protective effects of whole grains. In many persons, the risks of atherosclerotic cardiovascular disease, diabetes and obesity are linked to insulin resistance. Higher intakes of whole grains are associated with increased sensitivity to insulin in population studies and clinical trials. Why? Because whole grains improve insulin sensitivity by lowering the glycemic index of the diet while increasing its content of fiber, magnesium, and vitamin E.

Bangladesh data

Rice production in Bangladesh has remarkably increased in recent years, mostly due to cultivation of high yielding variety of rice supplemented with irrigation, fertilization, and pest control. Current production of paddy is 40 million metric tons (MT) in Bangladesh which yields around 27 MT of white, polished rice along with 3 MT of crude rice bran. Most of the bran are not well utilized although some are used in making low quality poultry feed or fed to the cattle unprocessed. It is the cheapest of all grain products in Bangladesh, selling at 5-6 taka per kilogram. There is a great potential of agro-industrial development using rice bran and bran-based products. However, there is little interest and almost no activity in this potentially rewarding areas. The difficulties of using rice bran as industrial raw material is related to its short shelf life (due to fat decomposition) and the logistic problems of collecting rice bran from a large number of mills scattered all over the country. These problems can be overcome by appropriately designed technical and logistic operational procedures.

If the total amount of rice bran is used for extracting rice oil there would be 0.5 MT of edible oil produced annually to meet almost 50% of national demand in Bangladesh (12,000 MT crude oil, mostly imported palm oil and soybean oil). It is to be noted that only 150,000-200,000 MT of edible oil is produced locally in Bangladesh which meets only 10%-20% of national demand.

Table 4: Annual rice production in Bangladesh

Rice Variety	2006-07 Metric Tons x 000	2007-08 Metric Tons x 000	2008-09 Metric Tons x 000
Aus Rice			
Local	516	408	447
HYV	996	1099	1448
Total Aus	1512	1507	1895
Aman Rice			
Local	2467	1660	
HYV	7867	7715	NA
Total Aman	10481	9662	
Boro Rice			
Local	256	226	
HYV	14709	13984	NA
Hybrid	-	3552	
Total Boro	14965	17762	
Total Rice	**27318**	**28931**	**NA**

Source: Bangladesh Bureau of Statistics, Govt. of Bangladesh

Table 5 : Proximate composition of rice bran from parboiled rice (minicade variety) in Bangladesh

Sample	Particle size	Moisture %	Tot Ash %	Protein %	Fat %	Fibre %
1st polish	40 mesh	8.2	9.5	12.8	23.1	8.9
1st polish	60 mesh	10.0	10.3	13.2	23.6	8.6
1st polish	80 mesh	8.2	10.5	13.1	24.2	8.8
2nd polish	40 mesh	7.7	10.0	13.1	26.3	8.9
2nd polish	60 mesh	6.6	10.6	12.6	26.7	--
2nd polish	80 mesh	6.2	10.4	13.3	26.5	9.49
3rd polish*	Total	6.9	9.8	12.8	27.3	6.2
Silky polish*	Total	8.5	7.3	13.8	22.5	2.8

* These samples are too fatty to pass through sieve and could not be meshed.

Assayed by methods of AOAC (Association of Official Agricultural Chemists, USA

Source: Dr. Umme Ara, BCSIR Laboratory, Dhaka, Bangladesh

Storage conditions

Since brown rice contains an oil-rich germ, it is more susceptible to becoming rancid due to enzymatic conversion of its oil contents by the enzyme lipase than the white rice and therefore should be stored in the refrigerator. Stored in an airtight container, brown rice will keep fresh for about six months.

While white rice varieties should also be stored in an airtight container, they can be kept in a cool, dry place rather than the refrigerator. Stored properly, they will keep fresh for about one year. The storage of cooked rice is controversial. Most organizations commend 4-7 days of storage in the refrigerator at most. From all of the available evidence, however, and to err on the safe side, we believe it's best to cook only the amount of rice one can consume during the day it is cooked, or at most, the following day. Several potential toxins can be produced in rice under certain conditions involving time, temperature, presence of moisture, bacterial spores, or fungi. It appears that some fungi can turn one of the amino acids (tryptophan) in rice into alpha-picolinic acid, and that this substance, when excessive, can cause hypersensitivity reactions to rice in some persons. Another mycotoxin (fungus-triggered toxin) called T-2 can also be produced in rice by the fungus fusarium. About 300 mycotoxins are commonly found in many grains, not only rice, when these grains are allowed to become moldy. All of the research we've see on these potential toxins involves cultivation and harvesting of rice at the agricultural level rather than cooking and storage of rice at home.

Stability of rice bran and brown rice in Bangladesh

One of the important problem of storing brown rice or rice bran was associated with their rapid deterioration due to hydrolysis of bran oil by the enzyme lipase forming free fatty acids (FFA). Oxidation of rice bran fat also occurs after milling and prolonged storage, specially in moist conditions. Most rice millers in Bangladesh is unable to store their bran products and brown rice more than 18 months because of development of hydrolytic rancidity. With technological advancement the problem of fat decomposition has been largely overcome by destroying lipase activity either by application of heat or chemical treatment.

Fortunately, for Bangladesh these problems can be of limited concern cause long term storage is not needed for commercial marketing; almost all of the season's harvested is usually consumed before the next crop of the season comes to the market (usually within 3 months). Moreover, almost all rice grain consumed by the people of Bangladesh are parboiled, i.e., boiled before milling. This process effectively destroys most of the lipase contents of the grain thus extending its self-life during storage.

Rationale and expected benefits

Rice bran is widespread and affordable - this makes a powerful value proposition for bolstering scientific, humanitarian, and business opportunities for its supply and use in promoting human health. Therefore, with proper treatment of rice bran for human consumption, significant reduction in prevailing malnutrition can be accomplished through the use this simple, low-cost, culture-friendly local products.

Recommendations and conclusion

Need for Basic Research: Rice bran available in Bangladesh need to be characterized in the laboratory determining its macronutrient contents and physicochemical properties by proximate analysis. Attempts should be made to develop a useful and simple method of stabilizing rice from the damaging effects of hydrolytic rancidity; simple heating or boiling could be an important tool to accomplish this goal. Effects of cooking, storage, palatability, and digestibility of rice bran and its products also need to be determined by conducting appropriate studies.

Product formulation by local industries: Rice bran itself or a modification of it can be used to formulate confectionery products (cookies/biscuits/cakes) for feeding malnourished children through school feeding programmes in Bangladesh. Different preparations can be developed using different percentages of stabilized rice bran to establish a definite, acceptable, and useful recipe/formulation for this purpose by conducting trial feeding studies in children.

These products should be acceptable to and consumed by the local population depending on their food habit and preferences. These could be manufactured locally and marketed through the government, NGO or international donor agencies for supplying to the school feeding programmes. Leptogenic studies also need to be conducted.

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MSD News

Scientific Seminar (SS)

ICH & Shishu Shasthya Foundation Hospital, Dhaka: On 23rd July 2009 a SS was arranged by Institute of Child Health & Shishu Shasthya Foundation Hospital, Mirpur-2, Dhaka on "Swine Influenza" at the seminar room. Professor (Dr.) Md. F. H. Nazir, Professor of Paediatric Medicine chaired the session. Professor Monimul Haque and Professor (Dr.) A.F.M. Salim were the discussants. Dr. Md. Fazlul Haque, Associate Professor of Paediatric Medicine was the rapporteur and Dr. A. K. M. Shamsuzzaman, Senior Consultant, Paediatric Medicine was the special guest of the seminar.

BMA, Faridpur: A SS was arranged by Bangladesh Medical Association, Faridpur branch on 28th April 2009 at BMA Bhavan, Jhiltuli on "Management of Lymphoma". Dr. A.K.M. Hamidur Rahman, Associate Professor, Radiotherapy Department of Faridpur Medical College chaired the meeting and Dr. Md. Mostafizur Rahmna Shamim was the special guest. Dr. Radheshyam Saha, Assistant Professor, Neuromedicine, FMC; Professor Dr. Syed Golam Kibria, Professor & Head, Department of Pathology, FMC and Dr. Sunil Kumar Sikder, Assistant Professor, Radiotherapy Department, FMC were the keynote speakers. Dr. J. C. Saha, Scientific Secretary, was the rapporteur.

BMA, Gopalgonj: Bangladesh Medical Association of Gopalgonj branch arranged a SS on "Role of trimetazidine in ischemic heart disease" on 15th July 2009 at the seminar room of Sadar Hospital. Dr. Abid Hossain Sheikh, President, BMA, chaired the seminar and Dr. Nurun Nabi, Civil Surgeon was present as the chief guest. Dr. Chowdhury Shafiqul Alam, General Secretary of BMA, was the special guest and Dr. Proshanto Kumar Sarnokar was the keynote speaker.

Department of ENT, DMCH: A SS was arranged by Department of ENT on "Cochlear implant surgery" on 20th June 2009 at the seminar room. Professor Dr. Mohammad Abdullah, Professor, ENT, Dhaka Medical College and Project Director, National Institute of ENT was the keynote speaker.

Department of Medicine, CoMCH: Department of Medicine of Comilla Medical College Hospital arranged a SS on 14th July 2009 at Cpsicum Chinese Restaurant, Comilla on "Pain management" Dr. Md. Sahabuddin chaired the meeting.

Rest of MSD news at page- 681

Review Article

To achieve international standard in the field of interventional cardiology : What should we do

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The ORION Medical Journal 2009 Sep;32(3):702-703

Abstract

Bangladesh is highly dense populated area. We have many cardiac patients, deal with them with care to reduce cardiac burden in Bangladesh. We have been doing a lot of work in different centres, arranging training and educating cardiologists also. There are enough cath labs in our country to perform interventional procedures. We may build up computer scoring systems to evaluate and to categorize the interventional cardiologists. We already have been crossed knowledge acquisition phase and now we are entering into percutaneous coronary intervention development phase. Government already plays a tremendous role to highlight and to patronize the development of interventional cardiology in Bangladesh. In this article, we are trying to highlight the course and curriculum of interventional cardiology, to provide best service from cardiologists to the patients and way to achieve international standard. Thus it reflects authors view.

Key word

Interventional cardiology, international standard.

Introduction

We know that for many years, in different centres we have been doing a lot of work for the purpose of training, educating and developing skill of cardiologists. But question is- Are we now in international standard? What interventional cardiologists should to do from now to achieve international standard? For this reason, we need firstly more systematic training in general cardiology and especially for interventional cardiology. Secondly, interventional cardiologists should offer therapeutic procedures to patients with appropriate clinical indications. Its aim is to be treating `patients, not lesions'. Thirdly, we should have reliable long term follow up data to show that such procedures truly improve the survival rates or at least improving the quality of life. Fourthly, innovation and improvement of the devices and drugs which must be consistent with international research established clinical practice and quality manufacturing products; And with the exponential growth in the number of centres and of interventional procedures are performed in Bangladesh, it should be monitor regularly and the necessary steps would be taken to ensure the best and safest practice to the patients for receiving such therapies.

Finally, we should to share our knowledge with other cardiologists here and abroad, should be capable to accept modern technology and also should have urge to train new generation (cardiologists). In case of training, we may upgrade their knowledge in cardiology irrespective to their academic background and carrier. We may build up computerized scoring system to evaluate and categorize the interventional cardiologists.

We should keep respect for each others performance and should to create an unique forum to protects our colleagues.

Pyramidal approach of education

Medical advancement follows a pyramidal approach. The base of the pyramid is the knowledge and skill acquisition phase. The middle level is the update knowledge phase, where knowledge is obtained through original research. Finally the top level is the knowledge exchange phase, where ones knowledge is shared with others. It is through written (scientific publications) or oral communications.

Course and curriculum

How one becomes an interventional cardiologist ? As there is no fixed curriculum or structural course, so opinion may defers from person to person. But from my point of view, I reflect my opinion here is based on developed country.

After graduation (MBBS) and completion of internship training, graduate doctors (candidates) are must be assessed by both clinically and theoretically to be a specialist physician in medicine, through a structural examination process without any biasness which would be specified by the Government approved system or by Board, off course with scoring system also. By the above way, we may find out professionally efficient doctors. Then according to the successful score of exam, they would be entitled to enter into the subspecialty course and training such as cardiology. These courses must be uniform and would be formulated to eliminate in different categories for subspecialty. This course curriculum or core training programme would also cover all the skills required for the practice of that subspecialty such as well as clinical training followed by training in non-invasive and invasive procedures. In the course, a minimum number of procedures must be recorded in a logbook and would be supervised by an accredited supervisor. After the successful completion of all steps of training and requirements, then the candidate would be issued a certificate of specialist cardiology by the Government which is off course approved by the tertiary education authority.

With this specialist cardiology certificate, the accreditation board of the health ministry that might be BMDC will then license the specialist in cardiology and issue him a registration number for practice as a cardiology specialist and would be charged as an independent cardiologist. After post graduation as a cardiology specialist, further four years fellowship training would to be chosen for super specialty such as interventional cardiology or electrophysiology or echocardiography. This fellowship training must to be undertaken at home or abroad which would be approved by government body and provide certificate as an interventional cardiologist before one commences his private practise.

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Without having this further experience, it is unlikely to a specialist that he might be a successful to obtain a consultant position in a tertiary level hospital. So it is undoubtful now that such rigorous training and certification are really necessary to ensure a minimal clinical standard to treat and care the patient as a specialist at an outpatient clinic or at a private or public hospital.

Knowledge acquisition

Now days there are well established and evidenced guidelines to interventional cardiologists to recommend interventional therapeutic procedures to their patients with appropriate clinical indications. But it is essential to form a board to review the latest guidelines and modify it in our perspective. The latest referral guidelines are published in the journal of the American College of Cardiology in January 2009. In comparison of long term outcome of percutaneous coronary intervention with conventional medical therapy or coronary artery bypass graft, surgical revascularization must be taken into account when selecting the best therapeutic approach for each individual patient. This is especially pertinent now when the main coronary disease is being treated by coronary stent. The concern of late stent thrombosis associated with the use of drug eluting stent is still unresolved. Though interventional procedure is developed in delay in Bangladesh, now it may turnout into a blessing. It will be in the interest of patients and service providers to have accurate longitudinal percutaneous coronary intervention outcome data for quality assurances. After all percutaneous coronary intervention is the most commonest medical procedure performed now a days in the world.

In many countries, comprehensive data collection and audit of longitudinal procedural and clinical outcomes are compulsory and Bangladesh should follow this.

Optical coherence tomography, intravascular ultrasound, virtual histology intravascular ultrasound, rotaablator, distal protection devices, percutaneous valve repair or replacement, congenital percutaneous closure, primary percutaneous coronary intervention, use of intraaortic balloon pump, carotid and peripheral stenting are need to be more familial for our interventional cardiologist.

Bangladesh is highly dense populated area. We have, so many cardiac patients, to deal with them with care to reduce cardiac burden in Bangladesh.

There are enough cath labs in our country to perform interventional procedure. Therefore, knowledge acquisition phase now has crossed and we are entering now into percutaneous coronary intervention development phase. We have also the scope for new knowledge phase. We have a large population, so we should be able to conduct large nation in multi centre through randomized clinical trials. Our current percutaneous coronary intervention practices, conducted trials to contribute new knowledge to the advancement of percutaneous coronary intervention excellence.

Conclusion

In conclusion day by day, we should to develop our evidence based guidelines to help interventional cardiologists to recommend interventional therapeutic procedures with appropriate clinical indications to their patients. We have a large population which should be able to conduct large nation in multi centre through randomized clinical trials to contribute new knowledge to the advancement of percutaneous coronary intervention excellence. We must develop our course and curriculum to find out and to build up international standard interventional cardiologist. Thus we can reduce and can be successful to reduce the cardiac burden in Bangladesh.

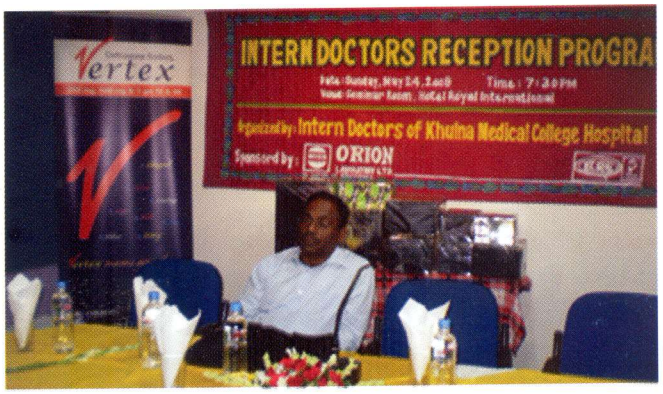
MSD News

Medical Services Department (MSD) of ORION Laboratories Ltd. successfully arranged significant number of Round Table Meetings, Scientific Seminars, Internee Doctors Reception Programs, Health Camp in different venues of all over Bangladesh during April 2009 to July 2009.

Intern Doctors Reception Program (IDRP)

Sher-E-Bangla Medical College Hospital, Barisal: An IDRP was arranged by Intern Doctors Association (IDA) of Sher-E-Bangla Medical College Hospital, Barisal on 22nd June 2009 at Dr. Pronab Gallery (No. 1 Gallery), SBMCH. Dr. S.M. Ramiz Ahmed, President IDA, SBMCH was the chairperson of the occasion and Dr. Dhirendranath Sarker, Director, was present as the chief guest. Dr. Md. Kamrul Hasan Selim, Assistant Director, SBMCH and Dr. Md. Estiak Hossain, Medical Officer, Model Family Clinic were the special guests of the program. There was an attractive raffle draw on that program.

Khulna Medical College Hospital: Intern Doctors of K-17 batch of Khulna Medical College Hospital arranged an IDRP on 24th May 2009 at Hotel Royal International, Khulna. Dr. Golam Hossain, Hostel Superintendent was present as the chairperson of the occasion. An attractive raffle draw was held there.



Scientific Seminar (SS)

Gynae Unit II, RMCH: Gynae Unit II of Rajshahi Medical College, arranged a SS on "Myomatous Polyp" and "UTI in pregnancy" at the Nanking Chinese Restaurant on 17th July 2009. Dr. Hasina Akther Assistant Professor was the chairperson.

Rest of MSD news at page- 701

pep Corner

The history of ORS and the inclusion of zinc supplementation for the treatment of diarrhea

Over two million children die as a result of diarrhea and dehydration every year. In the majority of cases, diarrhea is preventable through exclusive breastfeeding, improved hygiene and sanitation, and access to clean water, yet it is still one of the leading causes of death among children under five. A new Oral Rehydration Solution (ORS) formula and the introduction of zinc supplementation offer much improved outcomes for the treatment of childhood diarrhea. The introduction of zinc supplementation in diarrhea treatment provides for a high impact child survival intervention.

In the early 1980s, the introduction of ORS lead to significant and continuing decreases in the rate of diarrhea mortality that lasted for more than 20 years. Until recently, ORS, increased fluids, and continued feeding have been the only recommended treatments for episodes of noncomplicated diarrhea. Even though the accepted ORS formula was proven effective, researchers continued to work on developing an improved formula that would allow for more hydration while decreasing the amount of stool output. As a result, an ORS formula with lower glucose and sodium concentrations was developed, and it has proven to be more effective by decreasing the need for intravenous therapy, decreasing stool output, and decreasing the rate of vomiting. This new formula is recommended as one part of an improved diarrhea therapy and treatment. Zinc is another part.

Zinc is an essential micronutrient for human growth, development, and maintenance of the immune system. The first cases of zinc deficiency were recognized in the 1960s in adolescent boys in Egypt who suffered from growth retardation. A recent assessment by the International Zinc Consultative Group estimated that 20% of the world's population is at risk of inadequate zinc intake. In addition, high levels of zinc are lost in the stools during diarrhea episodes.

For more than 20 years, researchers have assessed the benefit of zinc supplementation during diarrhea episodes. A meta-analysis of eight trials of acute and persistent diarrhea found a 15% reduction in the duration of acute diarrhea and a 24% reduction in the duration of persistent diarrhea among children receiving zinc supplementation when compared to children who received a placebo. These studies also revealed that children receiving zinc supplementation experienced a decrease in the severity of their diarrhea episodes. Children who received 10-14 days of zinc supplementation also showed greater resistance to episodes of diarrhea and other infectious diseases for the 2-3 month period following treatment. Eleven additional trials have confirmed these results, supporting the inclusion of 10-14 days of 10-20 mg of zinc supplementation as another element in the recommended diarrhea therapy.

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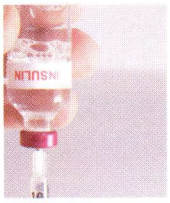
Medi News

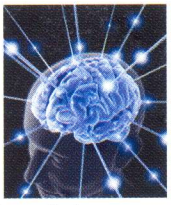
Elevated insulin levels linked to breast cancer



Postmenopausal women with elevated insulin levels may be at higher risk of developing breast cancer, a new study says. Researchers at Albert Einstein College of Medicine of Yeshiva University in New York City found a strong association between elevated insulin levels in the blood and increased risk of breast cancer. Their findings were published online in the International Journal of Cancer. "Up to now, only a few studies have directly investigated whether insulin levels are associated with breast cancer risk," said Geoffrey Kabat, the lead author and senior epidemiologist in the department of epidemiology and population health at Einstein. "Our study involved analyzing repeated measurements of insulin taken over several years -- which provides a more accurate picture of the possible association between insulin levels and breast cancer risk." Kabat's team examined data on 5,450 women who took part in the Women's Health Initiative, a large study that looked at how various factors influence women's health. The researchers found that women with insulin levels in the highest third were twice as likely to develop breast cancer as women in the bottom third. The team also discovered that the link between elevated insulin levels and breast cancer was stronger for thin women than for obese women, who tend to have higher insulin levels. "This finding is potentially important because it indicates that, in postmenopausal women, insulin may be a risk factor for breast cancer that is independent of obesity," Kabat said in the news release. The study is ongoing, but Kabat recommended that postmenopausal women try to keep insulin at normal levels through weight loss, regular exercise and other methods.

HealthDay

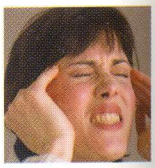
Lower IQ linked to higher risk of heart deaths



Intelligence appears to be one reason why poor people are more likely to die of cardiovascular disease, Scottish researchers say. They analyzed data on 4,289 former US soldiers, and found that IQ accounted for more than 20 percent of the difference in heart disease and stroke deaths between people of high and low socioeconomic status. This was in addition to well-established cardiovascular disease risk factors such as obesity and smoking. The study appears in the July 15 issue of the European Heart Journal. "We already know that socioeconomically disadvantaged people have worse health and tend to die earlier from conditions such as heart disease, cancer and accidents," study leader Dr. David Batty, an epidemiologist at the Medical Research Council's Social and Public Health Sciences Unit, University of Glasgow, said in a news release. "Environmental exposures and health-related behaviors, such as smoking and diet and physical activity, can explain some of this difference, but not all of it. This raises the possibility that, as yet, unmeasured psychological factors need to be considered. One of these is intelligence or cognitive function, commonly referred to as IQ. This measures a person's ability to reason and problem-solve. IQ is strongly related to socioeconomic status," Batty explained. He said the findings suggest that "public health messages on things like diet, exercise and smoking could be simplified. At present, the messages can be quite complicated, even contradictory, and they lack clarity. For instance, we often read about how some types of alcohol are good for you while others, or even the same ones, are not. These messages can be difficult to interpret, even by knowledgeable people." Furthermore, he recommends broad efforts to reduce socioeconomic inequalities. "Initiatives aimed at raising living standards and education of the most disadvantaged families with children could potentially make a difference to those children's health and well-being later in life," Batty said.

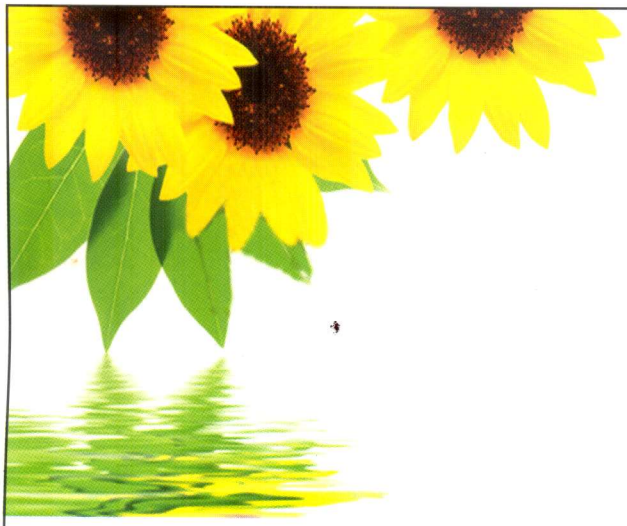
European Society of Cardiology

Breast cancer risk lower in migraine sufferers



For women there may be one good thing about having migraines: a reduced risk of breast cancer. In a study of more than 9,000 people, Dr. Christopher I. Li of Fred Hutchinson Cancer Research Center in Seattle and his colleagues found that those with a history of migraines were 26% less likely to develop breast cancer. The findings back up an earlier study, also by Li and his team, which included about 2,000 women and found a 33% lower breast cancer risk among women with migraines. Low estrogen levels appear to increase the severity and frequency of migraines in women, the researchers note in their report, while increased levels of the hormone are known to boost breast cancer risk, so it's "biologically plausible" that migraine sufferers would be less prone to breast cancer. In the current study, Li and his team compared 4,568 women with breast cancer, ranging in age from 34 to 64, to 4,678 healthy controls. They accounted for the effects of migraine triggers such as alcohol, smoking or hormone use, which hadn't been done in the previous study. The researchers found a 26% lower risk of breast cancer in the women with migraines, which didn't change when they took migraine triggers or whether or not a woman was menopausal into account. Similarly, use of prescription drugs for migraine did not change the risk. Migraine patients' greater use of non-steroidal anti-inflammatory drugs (NSAIDS), painkillers including ibuprofen and naproxen, could explain some, but probably not all, of their lower breast cancer risk, Li and his colleagues say. (A recent analysis of several studies linked NSAID use to 12% lower breast cancer risk.) "Further work is needed to resolve what accounts for this relationship," the authors conclude.

Reuters Health



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