Highlights in this issue

Hypertension, Homocysteine & Insulin Resistance
CAPD Peritonitis
Chiari Malformation Type-1
Ascaris Induced Pancreatitis
Chilaaditi Syndrome
Snake Bite
Catamenial Hemoptyisis

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Sanjeeb Kakati

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## History of APICON Assam

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<th>YEAR</th>
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<td>Dr.Pramathesh Barua</td>
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<td>Dr.K.Neog</td>
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<td>Dr. G. N. Gogoi</td>
<td>Dr. Atul Ch. Saikia</td>
<td>Dr. Madhab Mishra</td>
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*Source: Souvenir, APICON (Assam) 2010*
Maiden issue –
Hypertension, Insulin Resistance and Homocysteine

Sanjeeb Kakati*

It was a long felt need to the physicians across Assam and North-East India to have a technical Journal to share their scientific view, knowledge and experience. The demographic structure, geographic location provided a unique status to our region amongst the globe. Many diseases have shown its existence confined to this region only. The diverse ethnicity appeals the scientific community to meet various research queries. Keeping in view of the above facts The Association of Physician of India, Assam Chapter has taken a bold step to publish its official journal the maiden issue of which is released in October 2011 in the 21st annual conference APICON, ASSAM 2011 held at Tinsukia. This remains as an important mile-stone in the history of API-Assam Chapter. To start with it will be Biannual, one in January and the other in the month of June. The future of the journal depends on the generous participation and scientific contribution of the members. Scientific contribution from non members will also be accepted if it contains materials of interest to the physicians at large. It will contain sections like— Original article, Review article, Case reports, Pictorial CME and correspondence etc. Guideline for submission of scientific materials is mentioned in subsequent pages.

The theme of this issue is based on Hypertension. Two original articles of epidemiological study are included here addressing two different metabolic association or consequence of hypertension observed in the population of this region. One is on Insulin Resistance and the other is Homocysteine level.

Cardiovascular diseases caused 2.3 million deaths in India in the year 1990 which is projected to be double by the year 2020. Hypertension is directly responsible for 57% of all stroke deaths and 24% of coronary heart disease. Diabetes mellitus is commonly associated with hypertension. Epidemiological data suggest this association independent of age and obesity. Evidence points toward Hyperinsulinism as the possible link between the two. A state of cellular resistance towards insulin action subdents the observed Hyperinsulinism. The reasons for the association of insulin resistance and essential hypertension can be postulated by the following mechanisms— Sodium retention, Sympathetic overactivity, Altered membrane ion transport and Smooth muscle proliferation in the resistance vessels. Aerobic exercise, calorie restriction improve insulin resistance which is an established fact. At the same time it is also postulated that blood pressure is lowered by these maneuvers in both normotensive and hypertensive individuals.

When insulin resistance is defined as an M-value at clamp of <4.4mg/kg per min based on calculation from a healthy control sample, about 25% of a sample of hypertensive subjects taking no antihypertensive medicine in absence of diabetes mellitus was found to be insulin resistant. The relation of blood pressure to both insulin action and circulating insulin levels is compatible with distinct influences on blood pressure by insulin resistance and compensatory hyperinsulinemia.
Homocysteine (Hcy) is a nonessential sulfur-containing amino acid and serves as a metabolic intermediary product. Hyperhomocysteinemia, in addition to the traditional risk factors like smoking, hypercholesterolemia and diabetes is also considered as one of the risk markers for cardiovascular diseases. High plasma homocysteine concentration may be an important determinant for coronary artery disease (CAD), hypertension and stroke in India.

Reference:

4. Ele Ferrannini; Andrea Natali; Brunella Capaldo; Insulin Resistance, Hyperinsulinemia, and Blood Pressure—Role of Age and Obesity. Hypertension, 1997; 30:1144-1149
Keywords: Homocysteine; tribal population; Mizoram; North East region

Introduction

Homocysteine (Hcy) is a nonessential sulfur-containing amino acid and serves as a metabolic intermediary product. Concentration of Hcy varies with age, sex, ethnicity as well as geographical location, genetic, dietary and lifestyle related factors. Hyperhomocysteinemia, in addition to the traditional risk factors like smoking, hypercholesterolemia and diabetes is also considered as one of the risk markers for cardiovascular diseases. Moderate hyperhomocysteinemia is associated with an increased risk for atherosclerosis in the coronary, cerebral, and peripheral vasculature. Study conducted for quantitative assessment of plasma homocysteine as a risk marker for vascular disease showed that a 5-μmol/L Hcy increment elevates coronary artery disease risk as much as the increase of cholesterol by 20mg/dl.

India, like other developing countries contributes a much larger share to the global cardiovascular disease (CVD) burden than the developed countries which cannot be fully explained by conventional risk factors.

Original Article

Distribution of Plasma Homocysteine Concentration and Risk of Hypertension in a Tribal Population from North East India

P. K. Borah *, H.C. Kalita**, D. Hazarika***, P. Shankarishan****, J. Mahanta*****

Abstract

Objective: Elevated plasma homocysteine (Hcy) concentration is an important biochemical marker for cardiovascular diseases. We conducted the present study in the tribal population of Mizoram to find out distribution of plasma Hcy concentration and its association with hypertension.

Methods: A total of 541 (Male: 247, Female: 294) Mizo subjects aged ≥18 years were selected randomly from Aizawl district, Mizoram. Socio-demographic profile, ECG and blood pressure were recorded for all subjects. Blood samples were collected and processed for estimation of plasma Hcy and other biochemical parameters including blood glucose, urea, serum creatinine, uric acid and lipid profile.

Results: The mean Hcy concentration (geometric mean) in the study population was 11.5 ± 7.0 μmol/L which was higher in males (12.5 ± 7.3 μmol/l) than that of females (10.7 ± 6.7 μmol/l). The concentration showed an upward trend with increase in age and blood pressure categories of JNC-VII classification. The study subjects were classified by quartile of Hcy and revealed an increase of systolic and diastolic blood pressure by 5.6 and 4.3 mmHg respectively and prevalence of hypertension from the lowest to highest quartiles of Hcy concentration.

Conclusion: Mean plasma Hcy concentration in the study population was towards higher side of the normal range. High plasma Hcy concentration is associated with higher systolic and diastolic blood pressure and hypertension.
alone. Indeed a high plasma homocysteine concentration may be an important determinant for coronary artery disease (CAD)\(^2-9\) hypertension\(^10\) and stroke\(^11\). The present study was undertaken in a subset of tribal population from a hill state of Northeastern region to have an idea about distribution and its association with hypertension.

**Materials and methods**

Study subjects of both sex and aged 18 years and above from urban localities of Aizawl district, Mizoram were selected randomly for the present study. The study subjects were recruited considering equal distribution of age and sex. Data on socio-demographic variables and dietary habits along with the record of smoking and alcohol consumption pattern was recorded using a pre designed and pre-structured proforma. Height and body weight of the study subjects were measured by trained personnel following standard methodology. Height was measured with an anthropometric rod. Weight was measured using platform balance. Body mass index (BMI) was expressed as weight (kg) divided by height (m) squared. Mercury column sphygmomanometer was used to record blood pressure using standardized technique. Three readings were taken and the average of three readings was taken for analysis. Hypertension was defined as per JNC-VI criteria i.e. systolic blood pressure (SBP) > 140 mmHg and/or diastolic blood pressure (DBP) >90 mmHg or on anti-hypertensive treatment\(^12\). Hcy concentration in relation to different levels of blood pressure categorized by JNC VII criteria was also analyzed.

Fasting venous blood samples were collected in EDTA vial from study subjects after obtaining informed consent. Plasma were separated immediately by centrifugation and transported to the main laboratory (RMRC, Dibrugarh). The samples were stored at –20°C till analysis. Estimation of homocysteine was done by EIA method using Axis Homocysteine EIA kit (Axis-Shield Diagnostics Ltd., United Kingdom). The method was essentially as per manufacturer’s instruction and based on reduction of protein bound homocysteine to free homocysteine, which is subsequently converted to S-adenosyl-L-homocysteine (SAH) by the enzyme SAH hydrolase. The following solid phase enzyme immunoassay is based on competition between SAH in the sample and immobilized SAH bound to the walls of the microtitre plate for binding sites on a monoclonal anti-SAH antibody. A calibration curve was constructed using six calibrators provided with the kit. With every assay, controls were run to maintain batch-to-batch quality control. Hyperhomocysteinemia was defined as homocysteine level >15 \(\mu\) mol/L.

Estimations of blood glucose, urea, serum creatinine, uric acid and lipid profile were carried out by enzymatic oxidation method using different commercial kits. The institutional ethical committee of Regional Medical Research Centre, Dibrugarh, Assam, approved the study.

**Statistical analysis:**

All analyses were performed using Statistical Package for Social Science (SPSS) software version 17. Because distribution of plasma Hcy concentration in the study population was skewed, the values were transformed logarithmically to normalize their distributions for statistical analysis and geometric mean was taken. Spearman’s correlation coefficients were calculated between dependent variables including systolic & diastolic blood pressure and plasma Hcy concentration and statistical significance was calculated at p value = 0.05.

**Results:**

The present study included a total of 541 study subjects (M=247, F=294) from urban areas of Mizoram. The base line characteristics of the study sample are displayed in Table-1. The mean age of the study population was 44.6 ± 16.8 years (M=46.8 ± 17.3, F=42.7 ±16.2) and mean BMI was 23.7 ± 4.5. The mean values of blood glucose, lipid profile, blood urea, serum creatinine and uric acid in the study population are shown in the Table -1. Mean homocysteine concentration was 11.5±7.0 \(\mu\)mol/L (M=12.5 ± 7.3, F=10.7 ± 6.7) and prevalence of hyperhomocysteinemia was 35.3%. Mean SBP and mean DBP of the study subjects were 124.3 ± 20.0 and 79.5 ± 11.6 mmHg respectively. Prevalence of hypertension in the study subjects were 27.0% and 7.0% respectively. 38.3% and 7.8% of the study subjects were smokers and alcohol users respectively (Table-2).
Table-1: Clinical characteristics of the study population (n=541)

<table>
<thead>
<tr>
<th>Clinical variables</th>
<th>Male (n=247)</th>
<th>Values¹</th>
<th>Female (n=294)</th>
<th>Overall (n=541)</th>
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<tbody>
<tr>
<td>Age (years)</td>
<td>46.8±17.3</td>
<td>42.7 ± 16.2</td>
<td>44.6 ± 16.8</td>
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<tr>
<td>BMI (Kg/m²)</td>
<td>24.0 ± 4.9</td>
<td>23.4 ± 4.1</td>
<td>23.7 ± 4.5</td>
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<tr>
<td>Waist hip ratio</td>
<td>0.9 ± 0.06</td>
<td>0.9 ± 0.07</td>
<td>0.87 ± 0.07</td>
<td></td>
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<tr>
<td>Blood glucose (mg/dl)</td>
<td>118.3 ± 59.0</td>
<td>105.8 ± 53.9</td>
<td>111.5 ± 66.8</td>
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<tr>
<td>Total cholesterol (mg/dl)</td>
<td>192.6 ± 54.4</td>
<td>199.4 ± 58.9</td>
<td>196.3 ± 56.9</td>
<td></td>
</tr>
<tr>
<td>LDL (mg/dl)</td>
<td>122.1 ± 50.3</td>
<td>133.3 ± 47.5</td>
<td>128.2 ± 49.1</td>
<td></td>
</tr>
<tr>
<td>HDL (mg/dl)</td>
<td>44.8 ±9.1</td>
<td>45.2 ± 7.4</td>
<td>45.1 ± 8.2</td>
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<tr>
<td>Triglycerides (mg/dl)</td>
<td>161.4 ± 73.4</td>
<td>167.2 ± 82.3</td>
<td>164.4 ± 77.4</td>
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<tr>
<td>Blood urea (mg/dl)</td>
<td>25.4±9.6</td>
<td>24.3 ± 8.9</td>
<td>24.8 ± 9.3</td>
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<tr>
<td>Creatinine (mg/dl)</td>
<td>1.0 ± 0.3</td>
<td>1.0 ± 0.6</td>
<td>1.0 ± 0.3</td>
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<tr>
<td>Serum uric acid (mg/dl)</td>
<td>6.0 ± 2.2</td>
<td>5.3 ± 2.1</td>
<td>5.3 ± 2.2</td>
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<tr>
<td>Homocysteine (µ mol/L)²</td>
<td>12.5 ± 7.3</td>
<td>10.7 ± 6.7</td>
<td>11.5 ± 7.0</td>
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<tr>
<td>Mean SBP (mmHg)</td>
<td>127.7 ± 19.5</td>
<td>121.4 ± 20.0</td>
<td>124.3 ± 20.0</td>
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<td>Mean DBP (mmHg)</td>
<td>82.1±11.7</td>
<td>77.3 ± 11.0</td>
<td>79.5 ± 11.6</td>
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¹Values depicted are mean ± SD
²Geometric mean

Table-2: Prevalence of different risk factors of cardiovascular disease in the study population

<table>
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<tr>
<th>Clinical variables</th>
<th>Values</th>
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<td>Male (%)</td>
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<tr>
<td>Smokers</td>
<td>59.1</td>
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<td>Non smoked tobacco users</td>
<td>45.7</td>
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<td>Alcohol users</td>
<td>17.0</td>
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<td>Obesity (BMI&gt;=30)</td>
<td>6.5</td>
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<td>Central obesity</td>
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<td>Cholesterol (&gt;=200 mg/dl)</td>
<td>30.4</td>
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<tr>
<td>Triglycerides (&gt;=200 mg/dl)</td>
<td>26.3</td>
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<tr>
<td>LDL (&gt;=100 mg/dl)</td>
<td>51.0</td>
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<td>HDL (&lt;40 mg/dl)</td>
<td>29.6</td>
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<td>Blood glucose (&gt;=110 mg/dl)</td>
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<td>Prevalence of Hypertension (%)¹</td>
<td>34.4</td>
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<td>Prevalence of CHD (%)²</td>
<td>7.3</td>
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<tr>
<td>Hyperhomocysteinemia (%)³</td>
<td>42.1</td>
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¹Hypertension is defined as per JNC-VI criteria i.e. SBP= 140 mmHg and/or DBP = 90 mmHg or on antihypertensive therapy
²WHO-Rose questionnaires positive angina ± ECG changes suggestive of Ischaemia or myocardial infarction
³Homocysteine concentration > 15 µ mol/L
Fig-1 Age and gender wise distribution of plasma Homocysteine concentration (geometric mean)

Fig-1 shows age group and gender wise distribution of Hcy concentration in the study population. In all age groups Hcy concentration in female subjects was lower than that of male subjects and the values show a gradual upward trend with increasing age except the age group 50-59 years. Relation between Hcy concentration and different levels of blood pressure (JNC-VII) show a graded rise in the concentration with increase in blood pressure (Table-3).

Table-3 Plasma homocysteine concentration in relation to blood pressure categorized by JNC-VII

<table>
<thead>
<tr>
<th>Category</th>
<th>Overall (Mean* ± SD)</th>
<th>Male (Mean* ± SD)</th>
<th>Female (Mean* ± SD)</th>
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<tr>
<td>Normal</td>
<td>9.8 ±6.4</td>
<td>11.6 ± 6.4</td>
<td>9.1 ±6.3</td>
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<td>Pre-hypertensive</td>
<td>11.7± 7.0</td>
<td>11.9± 7.2</td>
<td>11.5± 6.7</td>
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<td>Stage-I</td>
<td>12.7± 7.5</td>
<td>13.1± 8.3</td>
<td>12.1± 6.3</td>
</tr>
<tr>
<td>Stage-II</td>
<td>14.1± 7.1</td>
<td>14.7± 7.1</td>
<td>13.2± 7.3</td>
</tr>
</tbody>
</table>

* Geometric mean

Normal: SBP<120 and DBP <80 mmHg; Pre-hypertensive: SBP120-139 and/or DBP 80-89 mmHg; Stage-I: SBP140-159 and/or DBP 90-99 mmHg and Stage-II: SBP>160 and/or DBP >100 mmHg
The quartiles of homocysteine in the study subjects were estimated and the mean or percentage values of different clinical characteristics were analyzed for each quartiles of homocysteine. The average SBP and DBP measurement increased by 5.6 and 4.3 mmHg respectively from lowest to highest quartile of Hcy. Higher level of creatinine and percentage of male subjects, smokers, alcoholics and prevalence of hypertension were observed in the group with highest quartile than that of lowest quartile of Hcy (Table-4).

### Table-4 Mean values of different characteristics of the study subjects by quartiles of Hcy

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<td>I</td>
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<tr>
<td>Age (years)</td>
<td>42.8</td>
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<tr>
<td>Sex (% male)</td>
<td>39.7</td>
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<tr>
<td>BMI (Kg/Metre²)</td>
<td>23.5</td>
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<tr>
<td>Blood glucose (mg/dl)</td>
<td>116.5</td>
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<tr>
<td>Cholesterol (mg/dl)</td>
<td>198.1</td>
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<tr>
<td>Triglycerides (mg/dl)</td>
<td>165.5</td>
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<tr>
<td>LDL (mg/dl)</td>
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<tr>
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<td>Creatinine (mg/dl)</td>
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<tr>
<td>Urea (mg/dl)</td>
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</tr>
<tr>
<td>Uric acid (mg/dl)</td>
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<tr>
<td>Hcy (μ mol/L)*</td>
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</tr>
<tr>
<td>Smoking (%)</td>
<td>35.3</td>
</tr>
<tr>
<td>Non smoked tobacco (%)</td>
<td>66.0</td>
</tr>
<tr>
<td>Alcohol consumption (%)</td>
<td>7.1</td>
</tr>
<tr>
<td>CHD (%)</td>
<td>3.2</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>120.1</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>76.9</td>
</tr>
<tr>
<td>Hypertension (%)</td>
<td>16.7</td>
</tr>
</tbody>
</table>

* Geometric mean

Spearman’s correlation coefficients and statistical significance between plasma total Homocysteine level and other parameters including SBP and DBP was found out. The results revealed significant correlation with height (r=0.126, p=0.003), waist hip ratio (r=0.092, p=0.032), creatinine (r=0.139, p=0.001), uric acid (r=0.086, p=0.046), SBP (r=0.147, p=0.001) and DBP (r=0.160, p=0.000) (Table-5).
Table 5: Spearman’s correlation coefficients and statistical significance between plasma total Homocysteine level and other parameters

<table>
<thead>
<tr>
<th>Variables</th>
<th>Spearman’s correlation coefficients</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>0.080</td>
<td>0.063</td>
</tr>
<tr>
<td>Height</td>
<td>0.126**</td>
<td>0.003</td>
</tr>
<tr>
<td>Weight</td>
<td>0.074</td>
<td>0.086</td>
</tr>
<tr>
<td>BMI</td>
<td>-0.005</td>
<td>0.907</td>
</tr>
<tr>
<td>Waist hip ratio</td>
<td>0.092*</td>
<td>0.032</td>
</tr>
<tr>
<td>Glucose</td>
<td>-0.020</td>
<td>0.640</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>-0.015</td>
<td>0.726</td>
</tr>
<tr>
<td>LDL</td>
<td>-0.049</td>
<td>0.352</td>
</tr>
<tr>
<td>HDL</td>
<td>-0.044</td>
<td>0.305</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>0.000</td>
<td>0.997</td>
</tr>
<tr>
<td>Creatinine</td>
<td>0.139**</td>
<td>0.001</td>
</tr>
<tr>
<td>Blood urea</td>
<td>-0.036</td>
<td>0.401</td>
</tr>
<tr>
<td>Serum uric acid</td>
<td>0.086*</td>
<td>0.046</td>
</tr>
<tr>
<td>SBP</td>
<td>0.147**</td>
<td>0.001</td>
</tr>
<tr>
<td>DBP</td>
<td>0.160**</td>
<td>0.000</td>
</tr>
</tbody>
</table>

* Correlation is significant at the 0.05 level (2 tailed)
** Correlation is significant at the 0.01 level (2 tailed)

Discussion

The present study investigated for the first time the distribution of plasma concentration in a tribal population of Mizoram (n=541) inhabiting in an urban set-up. Our study population revealed mean Hcy concentration (11.5 ± 7.0 μmol/L) which was within normal limit. The observed value was found to be higher than a population based study carried out in Southern Iran (6.8 μmol/L) and Japan (6 μmol/L) and lower than a hospital based study conducted in Hyderabad and healthy south Indian subjects in Chennai. The study conducted in South Asians in Canada, Asian Indians living in the UK and Singapore had shown that Indians tend to have higher homocysteine levels as compared to other population. This was attributed to reduced intake of vitamin B12 by Indians and prolonged cooking of vegetables damaging folic acid, which had been observed in some Indian households in the UK.

A graded increase in both systolic and diastolic blood pressure from lowest to highest quartile of homocysteine indicates its association with blood pressure. It is proposed that hyperhomocysteinemia induces an elastolytic process in the arterial wall which may lead to the stiffening of the arterial wall and hypertension. There is a very sparse data on association of hypertension and homocysteine level. Sharabi Y. et al. studied homocysteine levels in hypertensive patients with a history of cardiac or cerebral atherothrombotic events. They concluded that hyperhomocysteinemia was not a feature of hypertension with atherothrombotic events. Mendis et al. in a Sri Lankan population and Bortolotto et al. in a study of measurement of arterial stiffness had positively correlated homocysteine and hypertension.

The present study had shown a higher prevalence...
of smokers with hyperhomocysteinemia. Many authors\textsuperscript{20,21} had also observed that cigarette smoking was positively associated with high-homocysteine concentration. In the Hordaland homocysteine study, Nygard et al.\textsuperscript{22} observed that compared to nonsmokers, smokers had a distinctly higher plasma Hcy levels that increased almost linearly with the number of cigarettes smoked daily.

Geographical variation in food, fruit, vegetable intakes and either low intake of folic acid\textsuperscript{23} or damage during cooking process\textsuperscript{24} might be responsible for obtaining homocysteine level towards higher side of the normal range. Studies\textsuperscript{25,26} show that supplementation of folic acid and vitamin B\textsubscript{12} in the general population can lower the total Homocysteine concentration in a general population which can be responsible for the present study population.

Estimation of vitamin B\textsubscript{6}, B\textsubscript{12} and folic acid could not be done due to the lack of infrastructure, which was a limitation of the present study. A more extensive study is required to come into a better conclusion with respect to distribution, determinants of hyperhomocysteinemia including genetic factor, association with coronary heart disease and hypertension. Moreover, from a public health viewpoint, it is important to identify modifiable factors that influence plasma total homocysteine levels for subsequent prevention of cardiovascular disease in a population.

**Acknowledgements**

The authors acknowledge financial support from Indian Council of Medical Research, New Delhi, India.

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Study Of Insulin Resistance in Non Diabetic Hypertensive Patients


Abstract

In the present study an impressive correlation of Insulin resistance was observed in non-diabetic hypertensives where age correlates better with fasting serum insulin (correlation coefficient 0.404) than in control population (correlation coefficient 0.2136). Systolic and diastolic blood pressure level correlate with fasting serum insulin in hypertensive patients. This correlation was absent in the controls. There was no statistically significant correlation found among age, BMI in both cases and control group.

KEY WORDS: Insulin Resistance (IR), Homeostasis model assessment (HOMA), Waist- Hip Ratio (WHR)

INTRODUCTION

Essential hypertension is associated with multiple metabolic abnormalities including hyperinsulinemia. The association of insulin resistance and hyperinsulinemia in hypertension has been extensively studied during the last decades. Indeed, insulin resistance and/or hyperinsulinemia have been suggested as being responsible for the increased arterial pressure in some patients with hypertension1. The hyperinsulinemia is attributed to the decreased metabolic clearance in addition to insulin resistance.

Hypertension is twice common in diabetics comparing non diabetics 2. High plasma insulin has been shown to be associated risk factor for coronary heart disease in non-diabetic subject in prospective population studies3.

Recently several reports are coming up to relate insulin resistance and Hyperinsulinemia to coronary atherosclerosis. Moreover insulin resistance in association with hyperinsulinemia accelerates hypertension, decreases HDL cholesterol level and increases triglyceride level. Based on these observations, insulin sensitivity is important in normotensive, non-diabetic and non-obese subjects.

The incidence of hypertension in the urban population of Upper Assam has been reported to be higher (28.9%) than that of the national average for hypertension (Kotokey RK et al, 2006) 4.

It is observed in various studies that insulin resistance is one of the major etiological factors in non-diabetic hypertensive patients. Though various studies have been conducted on the pathogenesis of hypertension in this part of Upper Assam but the association of insulin resistance and hypertension has not yet been studied in this population group.

Therefore it is thought to be appropriate to carry out a study to examine insulin resistance in non-diabetic hypertensive patients at Assam Medical College and Hospital, Dibrugarh which is a tertiary care centre and the premiere institute of Assam.
MATERIALS AND METHODS

The present study was carried out in 50 hypertensive non-diabetics admitted or attending Assam Medical College, Dibrugarh during the period of 1st July 2005 to 30th June 2006.

SELECTION OF CASES:

- **INCLUSION CRITERIA:**
  Hypertensive non-diabetic patients were selected as follows:
  1) All patients admitted in Medicine wards or attending the Out-Patient Medicine Department with a history of hypertension as per the JNC VIII criteria without having Diabetes mellitus
  2) All the hypertensive non diabetic patients evaluated for any associated disease or regular/irregular intake of any drug

- **EXCLUSION CRITERIA:**
  1. Subjects less than 18 years.
  2. Subjects with secondary hypertension due to endocrine disorders like thyroid dysfunction, chronic pancreatitis or renal diseases and diabetes mellitus.
  3. Subjects on medications known to affect glucose and/or insulin levels.
  4. Female patients who are pregnant or lactating.

SELECTION OF CONTROLS:

50 number; age and sex matched; healthy normotensive non-diabetic volunteers who did not have a history of diabetes mellitus were taken as controls. This was confirmed by a detailed questionnaire, blood sugar estimation etc.

Patients were divided into two groups randomly in cyclical manner:

- **GROUP A: CASES** : Non-Diabetic Essential Hypertensive Patients (disease group)
- **GROUP B: CONTROLS** : Healthy Volunteers

PLAN OF STUDY:

1) As per selection criteria hypertensive and non-hypertensive (as control) cases admitted or attending OPDs in Assam Medical College and Hospital, Dibrugarh, were examined thoroughly.

2) The demographic profile of all patients was recorded with special stress on age, sex, diet, occupation and socioeconomic status.

3) BMI and Waist-Hip ratio compared between cases and control

4) All participants underwent thorough physical examination with special emphasis on pulse, systolic and diastolic blood pressure along with other relevant parameters.

5) Relevant laboratory investigations were done as required.

6) Plasma glucose estimation by glucose-oxidase-peroxidase method.

7) Serum insulin level was estimated by radioimmunoassay RIA Kit (RAIK-I) at Regional RIA Centre, BARC, GOI at Assam Medical College and Hospital, Dibrugarh.

CALCULATION OF INSULIN RESISTANCE:

The degree of insulin resistance was calculated using homeostasis model assessment 162 (HOMA) where insulin resistance (IR) is calculated by the following formulae.

\[
IR = \frac{\text{fasting blood glucose (mmol/l)} \times \text{fasting insulin level (mU/ml)}}{22.5}
\]

ESTIMATION OF INSULIN:

The radio immunoassay method is based upon the competition of

Unlabelled insulin in the standard or samples and radio iodinated (1-125) insulin for the limited binding sites on a specific antibody.

At the end of incubation, the antibody bound and free insulin is separated by the second antibody-polyethylene glycol (PEG) aided separation method. Insulin concentration of samples is quantitated by measuring the radioactivity associated with the bound fraction of sample and standards.

RESULTS AND OBSERVATIONS:

The study was conducted in Assam Medical College and Hospital from 1st July 2005 to 30th June 2006. The data was analyzed by the student ’t’- test, Chi-square test and linear regression analysis and the results are shown with the necessary tabulation.
### TABLE - 1

**AGE DISTRIBUTION**

<table>
<thead>
<tr>
<th>Age in groups (in years)</th>
<th>Group-A (n=50)</th>
<th>Group-B (n=50)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>18-30</td>
<td>1</td>
<td>2.00</td>
</tr>
<tr>
<td>31-40</td>
<td>2</td>
<td>4.00</td>
</tr>
<tr>
<td>41-50</td>
<td>10</td>
<td>20.00</td>
</tr>
<tr>
<td>51-60</td>
<td>15</td>
<td>30.00</td>
</tr>
<tr>
<td>61-70</td>
<td>21</td>
<td>42.00</td>
</tr>
<tr>
<td>71-80</td>
<td>1</td>
<td>2.00</td>
</tr>
<tr>
<td>&gt;80</td>
<td>0</td>
<td>0.00</td>
</tr>
</tbody>
</table>

### TABLE - 2

**SEX DISTRIBUTION**

<table>
<thead>
<tr>
<th>Sex</th>
<th>Group-A (n=50)</th>
<th>Group-B (n=50)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>Male</td>
<td>28</td>
<td>56.00</td>
</tr>
<tr>
<td>Female</td>
<td>22</td>
<td>44.00</td>
</tr>
</tbody>
</table>

### TABLE - 3

**CLINICAL DATA FROM CASES (GROUP-A) AND CONTROLS (GROUP-B)**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Group-A (n=50)</th>
<th>Group-B (n=50)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic BP (mmHg)</td>
<td>148±7.6±25</td>
<td>121±4.9</td>
</tr>
<tr>
<td>Diastolic BP (mmHg)</td>
<td>85.82±8.±68</td>
<td>71±9</td>
</tr>
<tr>
<td>BMI (Kg/m²)</td>
<td>223±</td>
<td>21±3.3</td>
</tr>
<tr>
<td>Waist–hip ratio</td>
<td>0.82±0.07</td>
<td>0.83±0.07</td>
</tr>
</tbody>
</table>

### TABLE - 4

**BIOCHEMICAL CHANGES IN CASES (GROUP-A) AND CONTROLS (GROUP-B)**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Group-A (n=50)</th>
<th>Group-B (n=50)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FBS(mg%)</td>
<td>95±6.51</td>
<td>95.58±10.98</td>
<td>0.27</td>
</tr>
<tr>
<td>PPBS(mg%)</td>
<td>141±12.19</td>
<td>133.94±12.26</td>
<td>2.454</td>
</tr>
<tr>
<td>FSI (mIU/ml)</td>
<td>6.78±3.05</td>
<td>5.24±2.45</td>
<td>2.74</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>1.55±0.75</td>
<td>1.20±0.57</td>
<td>2.63</td>
</tr>
</tbody>
</table>
### TABLE - 5
**INSULIN RESISTANCE IN CASES (GROUP-A) AND CONTROLS (GROUP-B)**

<table>
<thead>
<tr>
<th>HOMA-IR</th>
<th>Group-A (n=50)</th>
<th>Group-B (n=50)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>&gt;75 percentile</td>
<td>30</td>
<td>60.00</td>
</tr>
<tr>
<td>&lt;75 percentile</td>
<td>20</td>
<td>40.00</td>
</tr>
</tbody>
</table>

### TABLE - 6
**INSULIN RESISTANCE IN MALES AND FEMALES IN CASES (GROUP-A) AND CONTROLS (GROUP-B)**

<table>
<thead>
<tr>
<th>Sex</th>
<th>Group-A (n=50)</th>
<th>Group-B (n=50)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>Male</td>
<td>22</td>
<td>73.30</td>
</tr>
<tr>
<td>Female</td>
<td>8</td>
<td>26.70</td>
</tr>
</tbody>
</table>

### TABLE - 7
**CORRELATION COEFFICIENTS OF PARAMETERS IN CASES (GROUP-A) AND CONTROLS (GROUP-B)**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Group-A (n=50)</th>
<th>Group-B (n=50)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Correlation coefficients</td>
<td>Correlation coefficients</td>
</tr>
<tr>
<td>Age and IR</td>
<td>0.1784</td>
<td>0.0530</td>
</tr>
<tr>
<td>Age and WHR</td>
<td>0.1480</td>
<td>0.1500</td>
</tr>
<tr>
<td>Systolic BP and IR</td>
<td>0.288</td>
<td>0.083</td>
</tr>
<tr>
<td>Diastolic BP and IR</td>
<td>0.281</td>
<td>0.126</td>
</tr>
<tr>
<td>Age and FSI</td>
<td>-0.404</td>
<td>0.2136</td>
</tr>
<tr>
<td>BMI and FSI</td>
<td>0.0115</td>
<td>0.169</td>
</tr>
<tr>
<td>WHR and FSI</td>
<td>0.2289</td>
<td>0.1890</td>
</tr>
<tr>
<td>FBG and FSI</td>
<td>0.133</td>
<td>0.0915</td>
</tr>
<tr>
<td>PPBG and FSI</td>
<td>0.127</td>
<td>0.121</td>
</tr>
<tr>
<td>IR and WHR</td>
<td>0.076</td>
<td>0.0247</td>
</tr>
<tr>
<td>IR and BMI</td>
<td>0.100</td>
<td>0.086</td>
</tr>
<tr>
<td>Systolic BP and FSI</td>
<td>0.275</td>
<td>0.111</td>
</tr>
<tr>
<td>Diastolic BP and FSI</td>
<td>0.115</td>
<td>0.118</td>
</tr>
</tbody>
</table>
DISCUSSION:

It is observed in our study that advancing age is a predominant factor contributing to insulin resistance. Out of 50 numbers of non-diabetic hypertensive patients, 15 (30%) individuals were in the age group of 51-60 years and 21 (42%) individuals were in the age group 61-70 years. Similarly in control group, out of 50 individuals, 15 (30%) were in the age group of 41-50 and 22 (44%) individuals were in the age group of 61-70 years. Due to ethical considerations 18 years and below were excluded from the study.

Recent studies show that Indian population have a higher prevalence of hypertension in both rural and urban areas. To determine the changing trend in hypertension both urban and rural populations aged 20-70 years have been evaluated in epidemiological studies. It is also reported that in elderly individuals, mean age 70 years, in Kerala hypertension was present in 51.8% cases. And similarly it is reported that in adults aged 40-55 years blood pressure levels were highest among Indian men as compared to those in other twenty developing countries.

NC Hazarika et al (2002), ICMR, Lahowal, Dibrugarh, Assam showed that among the tea-tribe community above the age of 30 years, 66.6% were hypertensives meaning 6 to 7 persons out of 10 above the age of 30 were hypertensive and even in Assamese community prevalence of hypertension is 35% above the age of 30 years. So that present study is in accordance with other previous studies of Assam.

NC Hazarika et al (2002), reported that a large number of men and women are unaware of their hypertension status. The age-adjusted prevalence of hypertension was also highest in North-East states of India.

Recent Indian hypertension prevalence study conducted by Gupta R et al (1996) reported that men and women were 29.5% and 33.5% respectively. Gupta R et al (2002) reported that men and women were 36.4% and 37.5% respectively in Jaipur. Gupta R et al (2004) also showed that men and women were 43.8% and 44.5% in Mumbai respectively.

Bhatnagar et al (1995) reported that mean blood pressure in immigrants of South-Asian as compared with Indian siblings was found 146 ± 23 in men and women 143 ± 28 versus 142 ± 23 respectively. There are many studies like Bhopal et al (1999), SHARE Study (2000) etc. found to be having similar results with that of the present study. It is also reported in Kanuchi et al (2003) found to be 148 ± 1 mm of Hg systolic and diastolic blood pressure 82 ± 9 mm of Hg respectively.

The mean body mass index in the cases was 22 ± 3 Kg/m² and that in control group was 21 ± 3.3 Kg/m² in present study. There was no statistical difference observed. The mean waist-hip ratio in the cases was 0.82±0.07 and that in the control group was 0.83±0.07. The differences between the values were not statistically significant. A waist-hip ratio of greater than 1.0 in men and 0.8 in women indicates abdominal obesity.

Saad et al (1991) showed in their study that the subjects were all young (30±7 SD) years and obese (BMI = 32.9 ± 9.3) Kg/m². The association between insulin sensitivity and blood pressure could be verified across a wide range of age and body mass. Demirbas B et al (2002) showed BMI and WHR 32.2 ± 3.7 and 0.93 ± 0.8 respectively which is compatible with our present study.

In the cases mean fasting blood glucose was 95±6.51 mg% and that in the control was 95.58 ± 10.98 mg%. In the cases the mean post-prandial blood glucose was 141.12 ± 12.19 mg% and in the control was 133.94±12.26 mg%. There was no statistical difference found in individuals with normal fasting and post-prandial blood glucose level selected for the study. In 1968, Polonsky KS demonstrated that individuals with impaired glucose tolerance have greater insulin response after a glucose challenge than do other frankly diabetic or non-diabetic subjects. The same general principal hold true for fasting plasma insulin level. In the study report of Heise Tim et al (1998), in patients with reduced insulin sensitivity to glucose uptake insulin resistance is present in the vasculature resulting in blunted response to insulin.

It is also observed by Demibas et al (2002), that FBG 95.9 ± 10.7 mg/dl in cases and controls 89.8 ±7.7 mg/dl respectively. It was found to be almost similar to our study. The mean fasting serum insulin in cases was 6.78 ± 3.05 mIU/ml and that in control was 5.24±2.45 mIU/ml. There was statistically significant difference found. Normotensive, non-diabetic hypertensive is inversely related to insulin sensitivity and directly related to fasting plasma insulin concentration in either sex or any age and...
regardless of body size. Pimenta et al (1995)\textsuperscript{18}, Haffner S et al (1992)\textsuperscript{19} found that subjects were young (30 ± 7) years and obese (BMI=32.9±9.3Kg/m\textsuperscript{2}). So the association between insulin sensitivity and blood pressure could not be verified between a wide range of age and body mass. Drugs like Phenytoin, Verapamil, Diazoxide, Pentamidine suppress insulin release. Glucocorticosteroids and Thiazides suppress insulin sensitivity. Therefore, individuals on these medications were excluded from the study.

Insulin resistance calculated by homeostasis model assessment (HOMA-IR) was assessed in the study group. The 75 percentile was taken as the cut-off point for HOMA-IR in our groups. The cut-off point was found to be 1.8148. By this definition 60% of the cases were insulin resistant. Out of them 73.3% were male and 26.6% were females. 24% of the control was insulin resistant and 83.3% were male and 16.6% were female. The mean HOMA-IR in the cases was 1.55 ± 0.75 and in the controls was 1.20 ± 0.57. Statistically significant difference could be found in the insulin resistance between these groups. Kanuchi et al(2003) recently reported statistically significant association of insulin resistance in non-diabetic, non-obese hypertensives. There has been recent interest in the insulin resistance which has been noted in the non diabetic population because of its possible link to hypertension and cardiovascular disease\textsuperscript{22}. It has been estimated that 25% of the population are having insulin resistance. The prevalence of insulin resistance in lean subjects with hypertension was only 16 %. Despite their lean body mass insulin resistance was found to be a characteristic feature of Asian Indians\textsuperscript{23}. Nevertheless, a critical point is that diabetes do not usually accompany insulin resistance. On the other hand there are substantial number of reports which show a relationship of blood pressure to both insulin sensitivity and circulating insulin level. It is compatible with distinct influences on blood pressure by insulin resistance and compensatory hyperinsulinemia\textsuperscript{22}.

Correlation coefficients between systolic blood pressure and insulin resistance in cases and controls were found to be 0.281 and 0.126 which is also statistically significant.

There was statistically no correlation found with insulin secretion and insulin resistance and waist-hip ratio of individuals irrespective of hypertension. Correlation efficiency was 0.0076. There is no statistically significant correlation found between BMI and insulin resistance in the same groups (correlation coefficient 0.100). This correlation did not exist in the control group also. Polonsky KS et al (1988)\textsuperscript{24} in their study showed that both basal and 24-hour insulin secretion rates strongly correlate with body mass index. Kisselbak AH et al (1982)\textsuperscript{25} demonstrated that the central pattern of fat distribution with its increased waist hip ratio is associated with more insulin resistance than peripheral patterns of distribution, in which fat is more plentiful in the buttocks and upper leg areas. Individually with central pattern of obesity are likely to have glucose intolerance, hypertension, hyperlipidemia, vascular disease; a constellation of features that has been coined ‘Syndrome X’ by Gerald Reaven. In a study by Snehalata C (2001)\textsuperscript{26} it was demonstrated that despite their lean body mass, insulin resistance was found to be a characteristic feature of Asian Indians. Our study showed that in non-diabetic patients, age correlate better with fasting serum insulin (correlation coefficient 0.404) than in control population (correlation coefficient 0.2136).

ACKNOWLEDGEMENT

We are thankful to Principal-cum-chief Superintendent Assam Medical College and Hospital Dibrugarh for kindly allowing us to publish this Hospital Record.

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CAPD Peritonitis and Management

*M. Sharma, **S. Kakaty, ***P. K. Doley

INTRODUCTION:
Peritonitis is a frequent complication of Continuous ambulatory peritoneal dialysis (CAPD). Sixty percent of all patients on CAPD will have at least one episode of peritonitis during the first year of this mode of dialysis. Most of the episodes of peritonitis are caused by touch contamination of the dialysis tubing or by extension of the catheter exit site or tunnel infection. Coagulase-negative and coagulase-positive Staphylococcus are the two most common organisms, accounting for 50% or more of all CAPD peritonitis. Other gram-positive and gram-negative bacteria and fungi account for the rest. Intraperitoneal antibiotic treatments are usually effective in eradicating the infection. The choice of antibiotics depends on organisms isolated from cultured dialysate. Fungal peritonitis and, occasionally, Pseudomonas peritonitis require removal of the catheter to eradicate the infection. Prompt identification and treatment of peritonitis are essential to ensure success of a CAPD program. Although with newer techniques, like Y-connector or ultraviolet light system, the rate of peritonitis has declined; however, it has still remained the major complication of the CAPD program. Various terminology for peritonitis has been given in table -1

Let us now discuss this topic in five subheadings as recommended by International Society for Peritoneal Dialysis (ISPD).

1. Reporting of peritonitis rate.
2. Exit-site and tunnel infections.
3. Initial presentation and management of peritonitis.
4. Subsequent management of peritonitis (organism specific).
5. Future research.

Prevention and reporting of PD related infections: Prevention of PD related infection should be utmost of importance to optimize outcome on PD. The PD team should search or monitor the cause of exit-site infection and peritonitis, every effort should be made to find out the etiologic organism and if necessary to take appropriate interventions. Infection rate should be monitored at least once in a year. The center’s peritonitis rate should be no more than 1 episode every 18 months (0.67/year at risk), although the rate achieved will depend to some extent on the patient population. However, overall rates as low as 1 episode every 41 – 52 months (0.29 – 0.23/year) have been reported, a goal that centers should strive to achieve. Regular bowel movement should be encouraged in each and every patient. The double –cuff catheter and a downward directed tunnel may decrease the risk of

| **Recurrent:** | An episode that occurs within 4 weeks of completion of therapy of prior episodes. but with different organism |
| **Relapsing:** | An episode that occurs within 4 weeks of completion of therapy of prior episodes but with same organism or one sterile episode |
| **Repeat:** | An episode that occurs more than 4 weeks of completion of therapy of prior episode but with different organism |
| **Refractory:** | Failure of the effluent to clear after 5 days of appropriate antibiotics. |
| **Catheter related peritonitis:** | Peritonitis in conjunction with an exit-site or tunnel infection with the same organism or one site sterile. |

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catheter related peritonitis. Exit-site infection prevention is the primary goal of exit-site care. Antibiotic protocols for preventing exit-site infection is given in table -2.

Table-2

<table>
<thead>
<tr>
<th>Antibiotic protocols for preventing exit-site infection.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Exit-site mupirocin:</td>
</tr>
<tr>
<td>A. Daily, after cleansing in all patients.</td>
</tr>
<tr>
<td>B. Daily after cleansing in carriers only.</td>
</tr>
<tr>
<td>C. In response to a positive exit-site culture for S aureus denoting carriage.</td>
</tr>
<tr>
<td>2. Intranasal mupirocin twice per day for 5-7 days:</td>
</tr>
<tr>
<td>A. Every month, once patients identified as a nasal carrier.</td>
</tr>
<tr>
<td>B. Only in response to positive nose culture.</td>
</tr>
<tr>
<td>3. Exit-site gentamycin cream daily in all patients after cleansing.</td>
</tr>
</tbody>
</table>

EXIT-SITE AND TUNNEL INFECTIONS

DEFINITIONS:

Purulent drainage from the exit site indicates the presence of infection. Erythema may or may not represent infection. An exit-site infection is defined by the presence of purulent drainage, with or without erythema of the skin at the catheter–epidermal interface. Pericatheter erythema without purulent drainage is sometimes an early indication of infection but can also be a simple skin reaction, particularly in a recently placed catheter or after trauma to the catheter. Clinical judgment is required to decide whether to initiate therapy or to follow carefully. A positive culture in the absence of an abnormal appearance is indicative of colonization rather than infection. Intensification of exit-site cleaning with antiseptics is advised. A tunnel infection may present as erythema, edema, or tenderness over the subcutaneous pathway but is often clinically occult, as shown by sonographic studies. A tunnel infection usually occurs in the presence of an exit-site infection but rarely occurs alone. Staphylococcus aureus and Pseudomonas aeruginosa exit-site infections are very often associated with concomitant tunnel infections and are the organisms that most often result in catheter infection-related peritonitis; aggressive management is always indicated for these organisms.

THERAPY FOR EXIT-SITE AND TUNNEL INFECTIONS:

The most serious and common exit-site pathogens are Staphylococcus aureus and Pseudomonas aeruginosa. As these organisms frequently lead to peritonitis, such infections must be treated aggressively. Oral antibiotic therapy is generally recommended, with the exception of methicillin-resistant S. aureus (MRSA). Oral Antibiotics used in Exit-Site and Tunnel Infection are Amoxicillin, Cephalaxin, Ciprofloxacin, Clarithromycin, Dicloxacillin, Erythromycin, Fluvoxacin (or cloxacillin), Fluconazole, Flucytosine, Isoniazid, Linezolid, Metronidazole, Moxifloxacin, Ofloxacin, Pyrazinamide, Rifampicin.

INITIAL PRESENTATION AND MANAGEMENT OF PERITONITIS:

CLINICAL PRESENTATION OF PERITONITIS

Peritoneal dialysis patients presenting with cloudy effluent should be presumed to have peritonitis. In peritonitis, abdominal tenderness is typically generalized and is often associated with a rebound. Localized pain or tenderness should raise the suspicion of an underlying surgical pathology such as acute appendicitis. The physical examination of the patient presenting with peritonitis should always include a careful inspection of the catheter exit site and tunnel. Any drainage from the exit site should be cultured along with the effluent. If the exit site grows the same organism as the effluent (with the exception of CoNS), then it is very likely that the origin of the peritonitis is the catheter. This is confirmed by obtaining effluent cell count, differential, and culture. It is important to initiate empiric antibiotic therapy for PD-associated peritonitis as soon as possible. Patients with peritonitis usually present with cloudy fluid and abdominal pain; however, peritonitis should always be included in the differential diagnosis of the PD patient with abdominal pain, even if the effluent is clear, as a small percentage of patients present in this fashion. Other causes, such as constipation, renal or biliary colic, peptic ulcer disease, pancreatitis, and acute intestinal perforation, should also be investigated in the PD patient with abdominal pain and clear fluid. Conversely, while patients with peritonitis most often have severe pain, some episodes are associated with mild or even no pain.
The abdomen should be drained and the effluent carefully inspected and sent for cell count with differential, Gram stain, and culture. An effluent cell count with white blood cells (WBC) more than 100/ml (after a dwell time of at least 2 hours), with at least 50% polymorphonuclear neutrophilic cells, indicates the presence of inflammation, with peritonitis being the most likely cause. To prevent delay in treatment, antibiotic therapy should be initiated as soon as cloudy effluent is seen, without waiting for confirmation of the cell count from the laboratory. Patients with cloudy effluent may benefit from the addition of heparin (500 units/L) to the dialysate to prevent occlusion of the catheter by fibrin. Heparin is also usually added in cases of hemoperitoneum.

EMPIRIC ANTIBIOTIC SELECTION

Empiric antibiotics must cover both gram-positive and gram-negative organisms. The Committee recommends center-specific selection of empiric therapy, dependent on the local history of sensitivities of organisms causing peritonitis. Gram-positive organisms may be covered by vancomycin or a cephalosporin, and gram-negative organisms by a third generation cephalosporin or aminoglycoside. Intrapitoneal administration of antibiotics is superior to IV dosing for treating peritonitis; intermittent and continuous dosing of antibiotics are equally efficacious. It is important that the protocol cover all serious pathogens that are likely to be present. For many programs, a first-generation cephalosporin, such as cefazolin or cephalothin, with a second drug for broader gram-negative coverage (including coverage for Pseudomonas) will prove suitable. This protocol has been shown to have results equivalent to vancomycin plus a second drug for gram negative organism. While an extended course of aminoglycoside therapy may increase the risk for both vestibular and ototoxicity, short-term use appears to be safe and inexpensive and provides good gram-negative coverage. There does not appear to be convincing evidence that short courses of aminoglycosides harm residual renal function. Repeated or prolonged courses (e.g., longer than 2 weeks) of aminoglycoside therapy are probably not advisable if an alternative approach is possible. If an aminoglycoside is used for the initial gram-negative coverage, intermittent dosing is strongly encouraged and prolonged courses of longer than 3 weeks should be avoided. A recent study showed that systemic vancomycin and ciprofloxacin administration might also be an effective first line antibiotic. Quinolones (oral levofloxacin 250 mg daily, or oral pefloxacin 400 mg daily) appear to be an acceptable alternative to aminoglycosides for gram-negative coverage and do reach adequate levels within the peritoneum, even with cycler PD. No role has been shown for routine peritoneal lavage or use of urokinase, although one or two rapid exchanges often help to relieve pain, and continuous peritoneal lavage (for 24 – 48 hours) is often used for patients with septic shock and grossly turbid PD effluent. In a recent randomized control trial of 88 patients, IP urokinase had no significant benefit as an adjunct therapy in the treatment of bacterial peritonitis resistant to initial antibiotic therapy. The following table-3, showing the antibiotics dosing schedule for CAPD patients.

SUBSEQUENT MANAGEMENT OF PERITONITIS

Once culture results and sensitivities are known, antibiotic therapy should be adjusted to narrow spectrum agents as appropriate. For patients with substantial residual renal function (e.g., residual glomerular filtration rate >5 mL/minute/1.73 m2), the dose of antibiotics that have renal excretion may need to be adjusted accordingly. Patients who are high transporters and those with high dialysate clearances may have a more rapid removal of some antibiotics. Adjustments in dosing for such patients are not yet known but the clinician should choose the side of higher dosing. Within 48 hours of initiating therapy, most patients with PD-related peritonitis will show considerable clinical improvement. The effluent should be visually inspected daily to determine if clearing is occurring. If there is no improvement after 48 hours, cell counts and repeat cultures should be done. Antibiotic removal techniques may be used by the laboratory on the effluent in an attempt to maximize culture yield.

FUTURE RESEARCH:

Pharmacokinetic data of many new antibiotics, administered either systemically or IP, are urgently needed. Further clinical trials in PD patients are required, particularly double-blinded randomized trials assessing different treatment strategies and powered to detect meaningful differences using appropriate numbers of patients, and with sufficient follow-up. Such studies require large and enough patient numbers to evaluate significant
### TABLE -3

**Intraperitoneal Antibiotic Dosing Recommendations for CAPD Patients**

<table>
<thead>
<tr>
<th></th>
<th>Aminoglycosides</th>
<th>Continuous (per exchange, once daily)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LD 25, MD 12</td>
<td>Amikacin 2 mg/kg</td>
</tr>
<tr>
<td></td>
<td>LD 8, MD</td>
<td>Gentamicin, netilmicin, or tobramycin 0.6 mg/kg</td>
</tr>
<tr>
<td></td>
<td>LD 500, MD 125</td>
<td>Cefamandole, cephalothin, or cephradine 15 mg/kg</td>
</tr>
<tr>
<td></td>
<td>LD 500, MD 125</td>
<td>Cefepime 1000 mg</td>
</tr>
<tr>
<td></td>
<td>Cefazidime</td>
<td>1000–1500 mg</td>
</tr>
<tr>
<td></td>
<td>Cefazidime</td>
<td>1000 mg</td>
</tr>
<tr>
<td></td>
<td>Cefazidime</td>
<td>1000 mg</td>
</tr>
<tr>
<td></td>
<td>Penicillins</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>Amoxicillin</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>Ampicillin, oxacillin, or nafcillin</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>Azlocillin</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>Penicillin G</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>Penicillin G</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>Penicillin G</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>Ciprofloxacin</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>Vancomycin</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>Teicoplanin</td>
<td>Oral 200–300 mg q.d.</td>
</tr>
<tr>
<td></td>
<td>Vancomycin</td>
<td>15 mg/kg</td>
</tr>
<tr>
<td></td>
<td>Trimethoprim/sulfamethoxazole</td>
<td>25 mg/L in alternate bagsb</td>
</tr>
<tr>
<td></td>
<td>Bactrim</td>
<td>Oral 960 mg b.i.d.</td>
</tr>
</tbody>
</table>
differences in outcomes and such studies may need to be multicenter in design. Outcomes to be examined should include not only resolution without catheter removal but also duration of peritoneal inflammation, relapse, and repeat peritonitis, as well as change in peritoneal solute transport. Investigations into the role of biofilm in repeat episodes are also needed. Many of the antibiotic stability data are old and need to be repeated in new PD solutions (e.g., glucose polymer and amino acid solutions). More information is needed on modifiable risk factors for peritonitis. The benefit of screening for S. aureus carriage, either after an episode of staphylococcal peritonitis or routinely in a PD unit, needs to be clarified. Conventional dialysis solutions inhibit peritoneal immune function, decreasing the ability of the patient to fight infection. More studies are needed on the newer dialysis solutions, which are more biocompatible and may possibly impact on peritonitis risk. The development of antibiotic resistance in PD patients requires further study. It is probably a matter of time before PD infections due to extended-spectrum beta-lactamase- and carbapenemase- producing gram-negative rods and multiresistant gram-positive bacteria will be diagnosed. Treatment protocols should always include simple and small-spectrum antibiotics but research is warranted on the dosage and pharmacokinetics of new antibiotics and antifungal agents so that we are better prepared when multi resistance is observed. Multicenter studies will probably be needed to enable recruitment of the number of patients required to answer most of these questions.

REFERENCES:

Table – 4
Indications for Catheter Removal for Peritoneal Dialysis-Related Infections
- Refractory peritonitis
- Relapsing peritonitis
- Refractory exit-site and tunnel infection
- Fungal peritonitis
- Catheter removal may also be considered for
  - Repeat peritonitis
  - Mycobacterial peritonitis
  - Multiple enteric organisms

ND = no data; q.d. = every day; NA = not applicable; IP = intraperitoneal; b.i.d. = 2 times per day; LD = loading dose in mg/L; MD = maintenance dose in mg/L.

For dosing of drugs with renal clearance in patients with residual renal function (defined as >100 mL/day urine output), dose should be empirically increased by 25%.


**Case Report**

**Chiari Malformation Type 1 Presenting with Sneeze Syncope**


**ABSTRACT:**
Arnold Chiari Type 1 Malformation refers to herniation of the cerebellar tonsils alone (5mm or greater) below the foramen magnum. Headache, autonomic dysfunction, syncope and pain can be a presenting complaint or can accompany the more classic presentations. Here we report a case presenting with less commonly encountered symptoms like sneeze syncope, lower limb weakness and spasticity, ataxia and predominant ocular symptoms without accompanying sensory involvement.

**KEYWORDS:** Chiari type 1 malformation, Cranio-vertebral Abnormalities, Basilar Invagination, Downbeat nystagmus, Sneeze syncope.

**INTRODUCTION:**
Arnold Chiari malformation is a condition characterized by caudal displacement of the cerebellar tonsils through the foramen magnum. Chiari's subsequent studies expanded the spectrum of malformations in a classification system consisting of types 1, 2, 3, and later 4. Chiari type 1 refers to herniation of the cerebellar tonsils alone, and radiologically as simple tonsillar herniation 5mm or greater below the foramen magnum; type 2 refers to herniation of both the cerebellum and lower brainstem; type 3 refers to a rare type of brainstem herniation in association with a cervical or occipitoencephalocele; type 4 involves extreme cerebellar hypoplasia and caudal displacement of the posterior fossa contents.

Two additional types of Chiari malformation have been described. Chiari type 0 is defined as syringohydromyelia with distortion of contents in posterior fossa but without cerebellar tonsillar herniation. Chiari type 1.5 has been characterized as caudal migration of the brainstem and cerebellar tonsils often associated with syringomyelia but without spina bifida.

Clinical presentations resulting from Chiari malformation vary along a spectrum of severity and depend on anatomic involvement. Many individuals with Chiari 1 malformation do not have symptoms and go undiagnosed until adolescence. Alternatively, because of brainstem involvement, type 2 malformations are typically diagnosed earlier in childhood. Therefore, some categorize type 1 malformations as “adult type” and type 2 malformations as “congenital type.”

Headache and neck pain are the most common presenting symptoms of Chiari 1 malformations. Chiari 1 malformation may present with seizures and developmental delay in children. Symptomatic Chiari malformation can present at any age. Children and adults present with similar symptoms and signs. Saez and colleagues grouped presentations into 6 categories: (1) foramen magnum compression, (2) central cord syndrome, (3) cerebellar dysfunction, (4) bulbar palsy, (5) paroxysmal intracranial hypertension, and (6) spasticity. In their study of 35 patients, Pillay and colleagues found the frequency of clinical presentations to be the following: headache and neck pain, 73%; sensory dysesthesias or numbness, 56%; gait problems, 43%; upper extremity weakness, 25%; cranial nerve dysfunction, 23%; blurred vision, 17%; and lower extremity weakness, 15%. Some authors have stressed disorders of oculomotor motility, particularly downbeat

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nystagmus, as being important in making the diagnosis of a symptomatic Chiari malformation. Indeed, a variety of eye movement disturbances has been identified as a feature of this condition, including convergence nystagmus, internuclear ophthalmoplegia, oscillopsia, horizontal nystagmus, and ophthalmoplegia. Although eye movement disturbances may be seen, other signs are usually more prominent on presentation as the above described classification systems attest. Syringomyelia, when associated with a Chiari I malformation, usually involves the cervical spinal cord, with accompanying symptoms or signs of upper extremity involvement. Cases involving thoracic syrinx can cause scoliosis without upper extremity findings. This includes sleep apnea in adults, dysphagia, progressive myelopathy, sensorineural hearing loss, and vertigo if accompanied by neck pain or weakness, as well as other otolaryngological symptoms, disturbances of respiratory drive. Headache, autonomic dysfunction, syncope, and pain can be a presenting complaint of Chiari malformation, or can accompany the more classic presentations noted above. These intermittent symptoms can be cough-induced.

The nature of head pain or headaches in Chiari malformation varies and can be exertional, with characteristics of a spinal headache, either migrainous or cough induced. Chiari I malformation has been implicated as a cause of such syndrome complexes as chronic fatigue onset, gradually progressive unsteadiness while walking for past two years and gradual painless visual disturbances in the form of diplopia and oscillopsia, for past six months. On historical evaluation the child’s friends were first to notice his unsteadiness while walking to school in the form of “drunken gait” for last two years which initially was evident only while walking through narrow passages and playing in school. Later over the next few months, he developed progressive difficulty in walking. For the last six months he requires support while walking but can stand up without support. During this period he developed progressive weakness of the upper limbs, but he could button unbouton himself and there was no difficulty in writing except for his visual problems.

The child also developed visual disturbances in the form of double vision, initially at far objects that progressively involved near objects too. There was associated movements of the visual objects in the form of oscillations of the objects in both side-to-side and up & down fashion. His near vision decreased progressively as evident from the fact that he can read only when he keeps his books very close to his eyes. His performance was average in his school till last six months. There is no history of recent or past trauma to head and neck. He did not have any bladder or bowel symptoms or any abnormal sensations or numbness in any part of the body during this period.

The child had an uneventful institutional birth, no feeding difficulties, achieved developmental milestones in time; but he had history of repeated falls while coughing and sneezing since his childhood. He developed a habit of sitting down while sneezing. He hesitates to walk through narrow passages or crowded places since early childhood. Further, the child complained of a persistent sense of

presented to our Institute in mid August of 2011 with insidious MRI Image showing Tonsillar herniation
warmth in the nape of the neck for which he used to apply coconut oil in the affected part three to four times to obtain symptomatic relief.

On physical examination, the child had an obvious bony protuberance in the sub-occipital region with a normal facial morphism and average neck length. He had no postural hypotension or other clinical evidence of dysautonomia. He did not have any restriction of neck movements, scoliosis or kyphosis or other bony obvious deformities. Neurological examination revealed a horizontal nystagmus in lateral gaze with fast component towards the right and a prominent down beat nystagmus. All four limbs showed spasticity, reduced power(4/5) and exaggerated deep tendon reflexes. There was bilateral ankle clonus and positive Babinski’s sign. Cerebellar signs like incoordination, ataxic gait and positive Romberg sign were noted.

Routine Examination of blood revealed a hemoglobin of 11.8 % with a total leukocyte count of 6500/cumm with 70% neutrophils, 25% lymphocytes, 5% monocytes and platelet count of 2.2 lakhs /cumm. MR imaging of CV junction revealed herniation of the Cerebellar tonsil in spinal canal for a distance of 1.7 cm below foramen magnum without syringohydromyelia with kinking in posterior aspect of cervical cord at C2 level. Welcher’s basilar angle measures 136 degree. The tip of the odontoid process is 7mm above McGregor line. Atlanto dental interspace is increased by 4cm. The Odontoid process transects the Wachenheim’s line suggestive of basilar invagination. The features suggestive of syndromic findings of Chiari type I malformation.

Discussion:
Chiari malformation is a relatively uncommon disorder with a poorly understood etio-pathogenesis and an wide array of clinical manifestations. Yet, certain clinical findings with a suggestive symptomatology can clinch a diagnosis of the disease. Our case is unique in the sense that it is a rare presentation of Chiari malformation with minimal sensory symptoms and no radiological evidence of syringomyelia.

Conclusions:
Ever since the initial post mortem description by Chiari in 1981 of this group of malformation that bears its name, it seems that there have always been more questions on this subject than answers10. So, in a young adult with progressive gait unsteadiness, and ocular symptoms with suspected Cranio-vertebral junction abnormalities, Chiari Type 1 malformation may be a strong suspect.

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A Case of Round Worm Pancreatitis

Anupam Dutta*

ABSTRACT:
Round worm (A. lumbricoides) infestation is very common in tropical and temperate regions. An important but rare cause of acute pancreatitis is obstruction of the main pancreatic duct following round worm infestation. Here I present my experience with a patient of acute pancreatitis due to obstruction of the main pancreatic duct by round worm.

Introduction:
In tropical and temperate regions round worm (Ascaris lumbricoides) infestation is a very common phenomenon. It is mostly asymptomatic but sometimes can lead to complications like biliary or intestinal obstruction. On very rare occasions obstruction of the main pancreatic duct due to its smaller lumen may also occur. This is a case report of round worm in the main pancreatic duct, which is not a common site, presenting as acute pancreatitis and recovering uneventfully with conservative management.

Case Report:
A 26-year-old female presented with acute abdominal pain from the morning which was sudden in onset, severe in intensity, colicky in nature, not associated with food, originating in the epigastric region and radiating to the back. Patient had two episodes of vomiting which was bitter in taste and did not include food material. She had no similar attack in the past. She had two children, one 7 and another 4 years old, both had uncomplicated home delivery. She had normal menstrual cycles. In the emergency department she was treated with intravenous proton pump inhibitor and oral antacids which did not provide relief to her. She got little relief with an injection of ondansetron and diclofenac but still complained of pain abdomen in the epigastric region. On physical examination, the patient was afebrile, had a pulse rate of 104/min and blood pressure (BP) of 96/60 mmHg, with epigastric tenderness without guarding or rigidity. The bowel sounds were normal. An intravenous line was setup and normal saline was given. A ryles tube was given with an intention of gastric lavage. A complete blood count demonstrated normal leukocyte count of 7200/mm³, normal liver enzymes with elevated serum amylase (1651 IU/l) and serum lipase (5422 IU/l). Ultrasonogram of the abdomen revealed a round worm in main pancreatic duct with normal ductal caliber[Figure 1]. The pancreas was visualized throughout and it appeared bulky with a hazy outline and had minimal peripancreatic collection. The
gallbladder and common bile duct lumen was normal. Round worms were seen in the small bowel. The patient was managed conservatively. She was kept nil orally. She was given 3.5 liters of intravenous fluids per day with mostly normal saline and ringers lactate. Analgesics and antispasmodics were given 12 hourly and on s.o.s basis. Proton pump blockers and prophylactic antibiotics (ceftriaxone 1 gram intravenously after negative skin test) were added. Albendazole syrup was also given via the ryles tube at bedtime. Her symptom started to resolve from the 2nd day of admission and she became completely free of pain on the 3rd day of hospital stay. She gave history of expulsion of worms the next day. Follow-up ultrasonogram done 2 days after relief of symptom showed a normal pancreatic size, outline, parenchyma echogenicity and worm free main pancreatic duct. Patient was discharged on 7th day of admission with pancreatic enzymes, proton pump inhibitors, diet restriction and a repeat dose of albendazole after 14 days. Follow-up ultrasonogram done after a month showed a normal biliopancreatic tree.

Discussion:

A. lumbricoides is the most common helminthic infection in humans and occurs when Ascaris eggs are ingested from infected soil, food, or water. Larvae emerge in the duodenum and then migrate to the caecum, where they access veins of the portal system and are transported to the liver. From the hepatic veins, larvae pass to the heart and lungs. After migrating up the tracheo-bronchial tree, the larvae are swallowed and the mature worms develop in the small intestine over several months, and eggs are passed in the faeces[1] to continue the life cycle. The worms can move freely in and out of the hepatopancreato-biliary tree and therefore can be easily missed. Stool studies may show Ascaris ova and dead worm; parasite detection by stool examination for ova may approach 100%. [3] The diagnosis of pancreatic ascariasis can be done with ultrasonogram, endoscopy/ endoscopic retrograde cholangiopancreatogram (ERCP), computed tomography (CT) or magnetic resonance cholangiopancreatogram (MRCP). Ultrasonography is a simple, noninvasive and highly accurate test reflecting the worm morphology which may be single or multiple, long, linear echogenic strips without acoustic shadowing in the biliary or pancreatic ducts (strip sign). [4] In transverse scanning, the hepatopancreato-biliary worms produce a bull's eye appearance. This has a sensitivity of 50–86% for worms in the biliary tree, but the sensitivity for detecting worms in the pancreatic duct is not known. Endoscopic imaging can demonstrate active worms within the duodenal lumen and ampulla. At ERCP, Ascaris worms can be identified as smooth, linear filling defects within the ducts. [1] ERCP images may also show the worms as parallel smooth filling defects or as curves or loops traversing the ducts. [5] Use of ERCP allows better identification of worms in the duodenum and in the pancreatic-biliary tree, while providing a safe, therapeutic option of removing the worms.

References:

An Incidental Presentation of Chilaiditi Syndrome with Acute Gastritis
Abdul Ahad*

ABSTRACT:
Chilaiditi’s sign, or interposition of the right side of the colon between the diaphragm and liver is a benign process. Although not an uncommon incidental finding in clinical settings, radiological appearance can be confused with more catastrophic conditions associated with pneumoperitoneum, which is usually an indication of bowel perforation. Reevaluation is essential when a pattern of clinical finding does not fit into a clinical diagnosis.

Keywords: chilaiditi syndrome, blunt trauma abdomen, pneumoperitoneum, incidental presentation with gastritis, hypokalemia.

INTRODUCTION:
Chilaiditi sign, or interposition of the right side of the colon between the diaphragm and liver is a benign process. Although not an uncommon incidental finding in clinical settings, radiological appearance can be confused with more catastrophic conditions associated with pneumoperitoneum, which is usually an indication of bowel perforation, as it happened in our case. Reevaluation is essential when a pattern of clinical finding does not fit into a clinical diagnosis.

CASE REPORT:
A 40 year old male adult presented with acute pain abdomen and 4-5 episodes of copious vomiting since previous night. Pain was diffuse, aggravated with deep breathing and movement. Vomiting was greenish-yellow in colour, regurgitant. The patient did not give history of black stool or vomitus. He is non alcoholic, non diabetic, non hypertensive. He had a history of a car accident on the previous night, where he had sustained mild injuries on the back and chest wall. He was seen by a local physician and discharged with antiseptic dressing and analgesics. He was conscious and well oriented. Physical examination revealed nothing abnormal in the vital signs or the cardiovascular and respiratory systems. The abdomen was mildly distended with moderate tenderness and guarding in the right upper quadrant. Bowel sounds were sluggish to absent. Liver was palpable measuring 2 cm below costal margin in the mid clavicular line. Rest of the examination was unremarkable. Complete blood counts showed haemoglobin of 13.9 gm/dl, a total leucocyte count of 11,400 cells/mcl., platelet count of 1.9 lac /mcl. His serum creatinine and serum transaminases were normal. The chest X-ray revealed an elevation of the right hemi diaphragm (Figure 1).

Patient was provisionally diagnosed as acute...
gastritis possibly induced by NSAIDS. He was treated conservatively with a PPI, anti emetics, iv fluids and empirical antibiotics. However in view of history of injuries sustained in a car accident, sluggish bowel sounds, distended bowels and persistent vomiting a suspicion of bowel obstruction and/or sealed perforation was kept in mind. Possibility of blunt trauma abdomen was considered. A subsequent CT scan of the abdomen showed large intestinal loops predominantly the transverse colon, which were dilated and gas filled suggestive of obstruction. Dilated hepatic flexure was seen compressing the right hepatic lobes, reaching up to subdiaphragmatic location (Figure 2). However he improved within a few hours and discharged with a diagnosis of NSAID induced gastritis, hypokalemia, and incidental finding of chilaiditi syndrome.

DISCUSSION:

Interposition of the right side of the colon between the diaphragm and liver is a benign process. The radiological appearance can be confused with more catastrophic conditions associated with pneumoperitoneum[1], which is usually an indication of bowel perforation, as it happened in our case. There were cases which presented as acute pain in the abdomen, needing corrective surgical procedure; or as mistaken renal colic, or as suspected subphrenic abscess, or as pneumoperitonium[2-6]. Presence of bowel sounds, absence of free fluid, tachypnea and tachycardia, were against it. Although the majority of cases may be merely radiological curiosities, patients may present with a variety of abdominal signs and symptoms. Usually this causes no symptoms, and this is called Chilaiditi's sign. Chilaiditi's sign is named after the Greek radiologist Demetrius Chilaiditi who first described it when he was working in Vienna in 1910.[7] The sign can be present permanently, or sporadically. The exact cause is not always known, but it may occur in patients with a long and mobile colon (dolichocolon), chronic lung disease such as emphysema, or liver problems such as cirrhosis and ascites. Absence or laxity of the ligament suspending the transverse colon or of the falciform ligament are also thought to contribute to the condition. It can also be associated with relative atrophy of the medial segment of the left lobe of the liver. When the gallbladder position is often anomalous as well - it is often located anterior to the liver, rather than posterior.

The diagnosis of Chilaiditi syndrome is made when pain occurs due to transposition of a loop of large intestine (usually transverse colon) in between the diaphragm and the liver, visible on plain abdominal X-ray or chest X-ray.[1] The occurrence (incidence) on abdominal or chest X-rays is around 0.1% but it can be up to 1% in series of older adults.[8]

References:
7. Synd/2326 at Who Named It? eDigestive system • Digestive disease • Gastroenterology (primarily K20–K93, 530–579)
Case Report

Snake Bite

M Teron +, SA Shinde+, M Sharma *, C Baruah*, SK Baruah**

ABSTRACT:
A 13 year old female presented with a history of cobra bite with features of ptosis and severe local reaction of the right upper limb which later developed local reaction and necrosis at the bitten area. She was treated with Polyvalent Anti Snake Venom and wound debridement later on. The second case, a 25 year old male presented with a history of snake bite without signs of systemic and local envenomation at presentation. On the third day he developed features of severe haematological abnormalities with acute renal failure. He was treated with Polyvalent Anti Snake venom, Steroids and haemodialysis. Both patients recovered in due course of time.

Introduction:
Poisonous Snake bite is a common occurrence in the North-East Region of the country with considerable morbidity and mortality. Therefore, it is imperative to identify the features of common poisonous snake bites and treat the patient promptly. The common poisonous snakes found in India are King Cobra, Common Cobra, Krait (Elapidae family), Russell’s Viper and Saw Scaled Viper (Viperidae family). (1)

Cobra bites mainly cause Neurotoxicity apart from its local envenoming effects. The vipers usually cause haematological abnormalities and recurrent coagulopathy associated with it is an area of concern. Russell’s viper bite may involve other systems like nervous system, muscles and endocrine system.

Case 1
A 13 years old female attended the Emergency Department with history of snake bite around 6 hours back at the anterior aspect of the right wrist. On presentation, no abnormalities were detected except Ptosis and fang marks over the right wrist. Her single breath count was 32. There was no history of breathing difficulty, dark brown discoloration of urine, nausea, vomiting, fatigue, local pain at the site of bite etc. There was no bleeding from any site. There was a tight tourniquet at the right forearm.

On investigation, 20 minute whole blood clotting test was normal. Other relevant investigations were found to be normal. The limb was immobilized. Immediately the tourniquet was removed and the limb was kept elevated. She was infused 15 vials of Polyvalent Anti Snake Venom. Other drugs used were 5 ampoules of Neostigmine, Atropine, Antibiotics, Anti-inflammatory agents, TT, IV fluids.

Ptosis disappeared after few hours but she developed intense pain and swelling of the right forearm, upper arm and wrist with necrosis around the bitten area which lasted for few days. Wound debridement was done later. The recovery was uneventful and she was discharged from the hospital after 8 days.

Case 2
A 25 year male presented in the Emergency department with a history of snake bite around 13 hours back. He presented with nausea, vomiting, pain and oedema of left leg. There was a tourniquet at mid thigh. On examination, no abnormalities were detected locally except fang marks. Systemic examination was normal.
There was no ptosis, fatigue and respiratory distress. There was no bleeding from any site. The urine output was normal.

All relevant investigations were within normal limits. Doppler study of lower limbs revealed normal patency of blood vessels. Tourniquet was removed and limb kept immobilized and elevated. He was treated conservatively and planned for discharge shortly.

However he complained of decrease in urine output and reddish discoloration of urine on the third day. He was found to have haemoglobinuria, with raised AST, ALT. Serum creatinine was 4.6 mg/dl, platelet count was 96,000/mm³. Urine was negative for Myoglobin and FDP.

Patient was immediately started on Polyvalent Anti Snake Venom and Methyl Prednisolone considering vasculotoxic snake bite. First haemodialysis was done on the 5th day. Subsequently, patient developed Anuria, Pulmonary Oedema and Hyperkalaemia. His Creatinine was raised upto 12.8 mg/dl within one week. He underwent Haemodialysis eight times within a span of two weeks.

He was discharged after one month with Serum Creatinine 2.9 mg/dl and urine output 2300 ml/24 hr. On subsequent follow up after 2 weeks all blood parameters became normal. Leg oedema resolved completely.

Discussion:

Snake venoms contain Toxins which are mostly peptides and proteins of low molecular weight; enzymes like proteinases, hydrolases, transaminases, hyaluronidase, cholinesterase, phospholipase, ATPase, ribonuclelease, deoxyribonuclease, and miscellaneous agents like agglutinins, proteolysins etc. However, snake venoms are classified into Neurotoxic (elapids), Haematotoxic (Vipers) and Myotoxic (sea snakes). (2) Clinical features can be divided into local, general and systemic manifestations. Local symptoms and signs includes fang marks, pain, bleeding, bruising, lymphangitis, lymph node enlargement, inflammation, blistering, infection, necrosis etc. General manifestations include nausea, vomiting, malaise, abdominal pain, weakness, drowsiness, prostration.

The Elapidae mainly causes Neurological manifestations. The viperside involves many systems causing Cardiovascular, Renal and Bleeding and Clotting disorders. Russell’s viper also causes Neurological manifestations, Skeletal muscle breakdown and Endocrine abnormality like Acute Pituitary/Adrenal insufficiency. Long term sequelae of Russell’s Viper bite includes Chronic Renal Failure due to bilateral cortical necrosis and Chronic panhypopituitarism or Diabetes Insipidus reported from Myanmar and South India.(3,4) 20 minute whole blood clotting test is very useful and informative bedside test requiring very little skill. In the South-east Asian region, incoagulable blood is diagnostic of a viper bite and rules out an elapid bite. Other investigations include CBC, peripheral blood film, aminotransferases, muscle enzymes, serum creatinine, BUN, serum potassium, ABG, oxygen saturation and urine examination for haemoglobin, myoglobin, protein and microscopy.(3,6) However, viper bites should be observed for recurrent coagulopathy as occult coagulopathy can recur up to 2 weeks after the bite.(7) Administration of Antivenom is the mainstay of management of poisonous snake bites. Apart from that, first aid, transport to hospital, other ancillary treatment, treatment of the bitten part, observation of the response to antivenom and rehabilitation forms the comprehensive management strategy. Polyspecific/polyvalent antivenoms are preferred in many countries because of the difficulty in identifying species responsible. ELISA kits are available in some areas like Australia to help in identifying specific species responsible. Antivenom is recommended if any one of the signs of Systemic envenoming like haemostatic abnormalities, neurotoxic signs, cardiovascular abnormalities, acute renal failure and supporting laboratory evidence of systemic envenoming is present. It is also recommended if any of the signs of Local envenoming like local swelling involving more than half of the bitten limb in absence of a tourniquet, swelling after bites on the digits, rapid extension of swelling and development of enlarged lymph nodes is present. Other ancillary treatment of neurotoxic manifestations include anticholinesterase, atropine. Assisted ventilation may be required in case of bulbar and respiratory paralysis. Dialysis is an effective supportive treatment in viper bites developing acute renal failure.(1,3,?)

In the first case, the patient presented with features
of neuromuscular toxicity but there was no sign of any local reaction at presentation. She developed severe local reactions including necrosis of the bitten area later. Similarly, in the second case, there was no features of both local and systemic envenoming at presentation. These patients of viper bites should be observed very closely for occult and recurrent coagulopathy. One should remain vigilant about delayed manifestations of poisonous snake bite.

References:
2. Pillay W, Modern Medical Toxicology, 3rd edition, pg-120
3. WHO/SEARO guidelines for management of snake bite in south eastern region
6. Paul V, Prativa S, JAPI vol 52, Jan 2004
7. Auerbach PS, Norris RL. Harrison’s Principles of Internal Medicine, 16th Ed. 2005, P 2593-95
Catamenial Hemoptysis Following Dilation and Curettage (D&C) Procedure: A Rare Clinical Entity

Sir,

Catamenial hemoptysis is a rare disorder that is characterized by hemoptysis occurring concomitant with menstruation in female patients and is considered as a subset of extra-pelvic endometriosis. It is estimated that about 2% of cases of extra-pelvic endometriosis involve the thorax. The diagnosis of catamenial hemoptysis is usually clinical (1). One female patient, 25 years old, newly married 8 months back, presented with 2 days h/o hemoptysis of frank blood of around 20 to 30 ml, 1 to 2 times per day. She had no exacerbating factor, no h/o chronic cough, fever, weight loss, loss of appetite, dyspnoea, palpitation, chest pain, gastro-intestinal complication or h/o easy bruising or bleeding. No h/o DM, Hypertension, and no h/o features suggestive of SLE. She denied smoking, use of alcohol or any drugs. Patient got admitted into hospital & was treated conservatively with systemic antibiotic and blood transfusion. Patient had normal menstrual cycle & was on 2nd day of menstruation. She had mild pallor and tachycardia. Systemic examination revealed no abnormality. Biochemical profile (CBC, KFT, LFT) and Clotting profile were normal except low hemoglobin (9.5 gm %) and mild increase in ESR (38 mm/AEHF). CXR showed no abnormality. Sputum for AFB not detected. Mantoux test- negative. She recovered over next 2 days and subsequently discharged with a course of antibiotic.

The patient was again admitted in hospital 2 months later with the complain of hemoptysis. By this time she was on ATT which was prescribed by a local physician one month back when she presented to him with the same complain. ATT was stopped and a CT scan thorax taken this time showed ground glass opacity in anterior segment of left upper lobe. There was past history of hemoptysis only during her menstruation period & in between the periods she used to remain symptom free. On repeated query she gave h/o dilation & curettage procedure which was undertaken for a missed abortion 4 months back. Hemoptysis resolved spontaneously on 5th day of her menstruation and she was discharged and advised to go for bronchoscopy at a higher centre & come for regular follow-up. But unfortunately the patient did not come back to our hospital again.

From the history of cyclical hemoptysis starting with the onset & resolving on the 4th or 5th day of menstruation with the background h/o D&C for missed abortion, the provisional diagnosis is catamenial hemoptysis probably due to ectopic pulmonary endometriosis. Pathogenesis of thoracic endometriosis remains uncertain. Pulmonary parenchymal endometriosis is thought to result from hematogenous dissemination of endometrial particles. Hemoptysis results from fluid shift during menstruation causing capillary rupture within the lesions. Previous uterine instrumentation is strongly associated & supports this hypothesis (3). Currently, there are only few cases reported in the literature. Jeon-Seon Ryu, Eun-Seuf Song, et.al from Inha University, Republic of Korea studied the natural history & therapeutic implications of patients with catamenial hemoptysis. In their observations they found that all 4 patients had a h/o undertaking one or two D&C before diagnosis (2). Yu.Z, Fleischman JK et.al of Mount Sinai services of Queens Hospital centre, Jamaika, NY 11432, USA published a case of presumptive endometriosis in a 32 years old woman with a history of an induced abortion who presented with catamenial hemoptysis occurring first on 3 days of menstruation over an 11 month period (4).

This patient’s being presented here to highlight that D&C is a risk factor for the development of catamenial hemoptysis which is a rare entity in clinical practice.

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REFERENCES:
Article Submission

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