Editorial
113
—— DR. H. R. NAGRALE

Original Article
115
Low birth weight and some material risk factors. A multivariate analysis.
—— DR. B. D. NAIK, DR. A. P. KULKARNI

Original Article
119
Low plasma ascorbic acid in acute ischaemic stroke
—— DR. PRASHANT P. JOSHI, DR. A. S. GAWANDE, DR. S. N. UGHADE, DR. R. G. SALKAR.

Original Article
126
Autoinfection In SSI
—— DR. SUNIL P. GILANI, DR. NEETA JANGALE, DR. ABHAY CHOWDHARY, DR. G. B. DAVER.

Original Article
132
Study of genito-ulcerative lesions with special reference to Haemophilus Ducreyi
—— DR. R. D. KULKARNI, DR. PRATIBHA DALAL, DR. NEELMA HIRANI, DR. ARUNA GOHIL

........Contd
Original Article 137

Cytokine interplay in HIV / AIDS infection
— DR. S. KOTHARI, DR. V. AGNIHOTRI, DR. A. WADEGAONKAR, DR. R. DESHMUKH

Original Article 145

Comparison of surgically induced post-operative astigmatism following phacoemulsification by temporal clear corneal incision versus superior scleral tunnel incision.
— DR. A. H. MADAN, DR. DILIP KUMRE

Original Article 149

Clinical profile of sickle cell trait in tertiary care hospital - central India
— DR. DPITY JAIN, DR. ANJU MEHROTRA

Case Report 154

Microfilaria in bone marrow
— DR. A. G. VALAND, DR. SHAHEEN NAJMI, DR. SUSHMA RAMRAJE, DR. K. G. GHORPADE

Case Report 157

Foreign bodies in both main bronchi – A unique experience.
— DR. SAMIR V. JOSHI

History 159

Government Medical College, Aurangabad
— DR. A. B. SOLEPURE.

Guidelines for Authors 162
The idea of bringing out, the journal of Director of Medical Education and Research was conceived two years back. It was aptly named as, “Milestone”. The purpose was not only to promote or boost research activities at midlevel faculty in various medical institutions of repute under the Directorate and do the capacity building exercise, but more importantly to provide them a platform for publishing their own research work, realising the fact that it is equally important to get the work published. This provides incentive to researchers to do more reproducible research.

After couple of issues were published, there was a period of dormancy and lull, because of varied reasons. It was discontinued fully realising the facts that, this was important activity and also that only publication of the journal may not be enough to promote research activities, to explore real research potentials, to do extensive research and capacity building exercises at institutional level. Under the dynamic leadership and guidance of our Hon. Principal Secretary, Mr. G. S. Gill the activity was revived, reexplored and expanded.

As a result, “The Medical research council of Maharashtra” was established with twofold activities:

1) To promote medical research at the midlevel faculty in various medical institutions of repute under the Directorate by implementing the “Star Research Project Scheme”. Under this scheme proposals are invited from faculties and after critical review by a committee if found worth, are funded. The council also monitors the project till it’s completion.

2) The regular and sustained publication of “Milestone” is the second important activity under the council. The journal will provide opportunity to all projects under the “Star research scheme” to get published apart from publishing review articles, original articles, critical reviews etc.
It is great honour to be the Member Secretary of this prestigious council. I am thankful to our Hon. Principal Secretary, Mr. G. S. Gill for giving me this responsibility and reposing faith in me. Because of his constant inspiration, the council is moving in right direction, several projects have already been funded under “Star research scheme” and several are in pipeline. More and more articles are being submitted for publication in, “Milestone”.

I am also thankful to our Director, Dr. W. B. Tayade for his valuable suggestions and support. “Milestone” is a medium of spreading medical knowledge from and to every faculty in medical field in every corner of Maharashtra, specially in areas lacking infrastructure and access. I am trying my level best to keep progressing in this direction. I am getting lot of feedback and guidance from my colleagues and friends.

Dr. A. G. Valand, Associate Professor in pathology has been of great help to me, without his support, it would not have been possible for me to publish the journal on time. He is ably shouldering responsibility with me looking after different aspects involved in publication.

I welcome original articles, special articles, reviews, short communications, and case reports for publication. Guest editorials will also be invited for this prestigious journal.

Once again, I thank one and all who are helping me for publication of this journal which is informative, thought provoking and will guide the research activity.
ABSTRACT

Total 1252 newborns were studied. Of these, 394 were low birth weight. The incidence of Low birth weight was found to be 31.94%. The mean birth weight was 2.60 with S.D. 0.50 kg. The Simple Linear Regression shows that maternal age, weight, haemoglobin (gm%), income, birth interval, Tab FS received were significantly associated with birth weight. Birth order, ANC visits, and T.T. doses received were found to have no significant association with low birth weight.

KEY WORDS:

Low birth weight, multivariate analysis, risk factors for low birth weight

INTRODUCTION

Birth weight is a critical determinant of survival, growth and development of baby and also a valuable indicator of maternal health, nutrition and quality of life. Worldwide, out of 139 million live births about 23 million infants had low birth weight i.e. birth weight below 2500 gms. In India the prevalence of low birth weight is about 26%. One of the factor of infant mortality is low birth weight. The maternal factors play crucial role in the birth weight of baby. The mortality of low birth weight can be reduced if the maternal risk factors are detected early and managed by simple techniques. This study has made an attempt to identify the maternal factors that have a significant association with low birth weight with the help of Linear Regression Model.

OBJECTIVES:

1. To know the prevalence of low birth weight.
2. To identify the maternal risk factors associated with low birth weight.

MATERIAL & METHODS:

Design: Hospital based observational study.
Setting: Shri Guru Govind Singh Memorial Hospital, Govt. Medical College, Nanded, for a period of one year from 1st January 1999 to 31st December 1999.

*Lecturer, Dept. of P.S.M., Govt. Medical College, Nanded.
**Professor and Head, Govt. Medical College, Nanded.
Participants: All mothers giving birth to singleton live baby are included in the study.

METHODOLOGY:
The birth weight of new born was measured preferably within the first hour of life with conventional beam balance machine having accuracy of 100gm. Low birth weight is defined as birth weight less than 2500 gm i.e. up to and including 2499 gm. Mother’s weight and height was taken as per guidelines given by Jelliffe. The other information was collected by interview of the mother with pre-designed and pre-tested pro-forma and review of records like ANC cards. Main Outcome Measure: Birth Weight.


RESULTS:
A total of 1483 eligible mothers delivered during the study period. Of these, 129 left the hospital against medical advice and 21 absconded. Total 1252 (93.9%) mothers could be studied. Of the total 1252 deliveries 394 were low birth weight. The incidence of low birth weight was found to be 31.94% with mean birth weight was 2.60 ± 0.50 kg. The maternal factors-Age, Weight, Height, Haemoglobin (gm%), Income, Birth interval, Birth order, ANC visits, T.T.doses, Tab. FS received were studied in relation to birth weight by simple multiple linear regression analysis.

Birth weight is considered as a dependent variable and other maternal factors as independent variable. Two steps were involved, in first step (Table No.1) birth weight is tested with each variable individually and “r” (correlation coefficient), regression coefficient (b) with 95% confidence intervals and y-intercept for each factor were calculated. In the second step (Table No.2) simple multiple linear regression was applied.

From the Table No.1, it was seen that the following maternal factors, had positive, & significant association with birth weight.

1. Maternal weight, 2. Height, 3. Haemoglobin (gm%), 4. Income, 5. Birth interval, 6. Tab. FS received. Negative and significant correlation is with the age of mother. The interesting finding in table No 2 is that the factors birth order, ANC visits, and T.T. doses when tested individually (Table 1), were significantly associated with birth weight but at second step (Table 2), these factors are not found to be significantly associated to birth weight, indicating that these factors are dependent on the other factors (confounding factors).

CONCLUSION:
Of the total 1252 deliveries 394 i.e. 31.46% were low birth weight. There is considerable variation in the prevalence of LBW in India. The disparity has ranged from a prevalence of 10% to 59%. There is wide inter-regional, socio-economic and urban verses rural difference in the prevalence of LBW have been recorded. The prevalence of LBW was 30.3% as reported by Deshmukh, 32.2% by Mohammad Zafar and 29% by Hirve which is comparable to our study. The simple multiple linear regression shows that maternal age, weight, haemoglobin (gm%), income, birth interval, Tab FS received have significant association with birth weight even if association of other factors is taken into account. Birth order, ANC visits and T.T. doses received have significant association with low birth weight.
when tested individually, but have no such association if other factors are taken into account. Various hospital based studies with multivariate analysis show that the maternal risk factors are associated with low birth weight. Our findings are consistent with these studies.

**ACKNOWLEDGEMENTS:**

We thank Dr. S.B. Rathod, Dean Govt. Medical College, Nanded for allowing us to carry out the study. We are also thankful to Dr. A.R.Mahale, Professor and Head, Dept. of Obst. & Gyn. to carry out the study.

### Table 1. Co-relation of various risk factors with LBW.

<table>
<thead>
<tr>
<th>SR.</th>
<th>Risk Factors</th>
<th>Correlation coefficient (r)</th>
<th>Value of b Lower</th>
<th>95% CI Upper</th>
<th>Y Lower</th>
<th>95% CI Upper</th>
<th>Intercept</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Age (yr)</td>
<td>-0.11</td>
<td>-0.0142</td>
<td>-0.0214</td>
<td>-0.0069</td>
<td>2.9362</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Weight (kg)</td>
<td>0.63</td>
<td>0.1019</td>
<td>0.094</td>
<td>0.1088</td>
<td>-1.9389</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Height (cm)</td>
<td>0.43</td>
<td>0.0434</td>
<td>0.0383</td>
<td>0.048</td>
<td>-3.76</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Haemoglobin (gm%)</td>
<td>0.53</td>
<td>0.2646</td>
<td>0.2409</td>
<td>0.2882</td>
<td>0.1809</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Income (Rs)</td>
<td>0.23</td>
<td>0.0001</td>
<td>0.0009</td>
<td>0.001</td>
<td>2.3717</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Birth interval</td>
<td>0.21</td>
<td>0.0891</td>
<td>0.0655</td>
<td>0.1126</td>
<td>2.4757</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Birth order</td>
<td>-0.15</td>
<td>-0.0621</td>
<td>-0.0844</td>
<td>-0.0398</td>
<td>2.7335</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>ANC visits</td>
<td>0.34</td>
<td>0.1147</td>
<td>0.0972</td>
<td>0.1323</td>
<td>2.2856</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>T.T.doses</td>
<td>0.15</td>
<td>0.1475</td>
<td>0.0947</td>
<td>0.2004</td>
<td>2.3719</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>Tab FS received</td>
<td>0.36</td>
<td>0.1219</td>
<td>0.1041</td>
<td>0.1396</td>
<td>2.3938</td>
<td></td>
</tr>
</tbody>
</table>

### Table 2. Multiple Linear regression analysis of risk factors of LBW.

<table>
<thead>
<tr>
<th>SR. No.</th>
<th>Risk Factors</th>
<th>Mean</th>
<th>B coefficient</th>
<th>95% CI Lower</th>
<th>95% CI Upper</th>
<th>Standard error</th>
<th>Partial F-test</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Age (yr)</td>
<td>23.9633</td>
<td>-0.02167</td>
<td>-0.0298</td>
<td>-0.0135</td>
<td>0.0041</td>
<td>27.05</td>
</tr>
<tr>
<td>2</td>
<td>Weight (kg)</td>
<td>44.4888</td>
<td>0.0675</td>
<td>0.0591</td>
<td>0.0758</td>
<td>0.0042</td>
<td>252.98</td>
</tr>
<tr>
<td>3</td>
<td>Height (cm)</td>
<td>146.4065</td>
<td>0.0076</td>
<td>0.0028</td>
<td>0.0123</td>
<td>0.0024</td>
<td>9.84</td>
</tr>
<tr>
<td>4</td>
<td>Haemoglobin (gm%)</td>
<td>9.1269</td>
<td>0.1118</td>
<td>0.0886</td>
<td>0.1350</td>
<td>0.0118</td>
<td>89.14</td>
</tr>
<tr>
<td>5</td>
<td>Income (Rs)</td>
<td>1757.96</td>
<td>0.00003</td>
<td>0.000007</td>
<td>0.00005</td>
<td>0.00001</td>
<td>6.71</td>
</tr>
<tr>
<td>6</td>
<td>Birth interval</td>
<td>1.3491</td>
<td>0.0830</td>
<td>0.0620</td>
<td>0.1039</td>
<td>0.0106</td>
<td>60.40</td>
</tr>
<tr>
<td>7</td>
<td>Birth order</td>
<td>2.2133</td>
<td>0.0001</td>
<td>-0.0259</td>
<td>0.02625</td>
<td>0.0133</td>
<td>0.0001</td>
</tr>
<tr>
<td>8</td>
<td>ANC visits</td>
<td>2.7029</td>
<td>-0.0166</td>
<td>-0.0397</td>
<td>0.0063</td>
<td>0.0117</td>
<td>2.01</td>
</tr>
<tr>
<td>9</td>
<td>T.T. doses</td>
<td>1.5176</td>
<td>0.0139</td>
<td>-0.0323</td>
<td>0.0603</td>
<td>0.0236</td>
<td>0.34</td>
</tr>
<tr>
<td>10</td>
<td>Tab FS received</td>
<td>1.6573</td>
<td>0.0495</td>
<td>0.0285</td>
<td>0.0705</td>
<td>0.0107</td>
<td>21.33</td>
</tr>
</tbody>
</table>

Y-Intercept -2.2494

---

**MILESTONE**

*Journal of DMER* 117
REFERENCES


ABSTRACT

Objective: To test the hypothesis that plasma ascorbic acid (AA), a common antioxidant, is significantly lower in cases of acute ischemic stroke as compared with age and sex group matched controls, in multivariate analysis. Role of low plasma AA in pathogenesis of acute ischemic stroke is controversial and data from India is scarce. Search for new, modifiable risk factors is crucial since the conventional risk factors cannot fully explain the emerging epidemic of cardiovascular diseases (stroke and myocardial infarction).

Design: Case-Control study

Setting: Medicine wards, Government Medical College, Nagpur, India.

Participants: One hundred and four consecutive, incident, prospective cases of first acute ischemic stroke, with 104 age-sex group matched healthy controls.

Main Outcome Measures: Acute ischemic stroke cases, (CT scan proved).

Study factors: Plasma AA (measured by calorimetric method), Conventional risk factors: Hypertension, diabetes mellitus, serum cholesterol, obesity, alcohol intake, smoking (measured by an interviewer-administered questionnaire).

Results: Mean plasma AA was significantly lower in cases than in controls (23.63 ± 6.43 mmol/L Vs 27.54 ± 3.6 m01/L, p<0.00001). Sixty-five (62.5%) cases had low plasma AA (<23mmol/L) as compared with 9 controls (Odds Ratio=17.5, 95%CI=6.7-45, p<0.0001). After controlling for all the conventional risk factors in multiple logistic regression analysis, low plasma AA was significantly associated with acute ischemic stroke (adjusted Odds Ratio=18, 95%CI=7.6-43.1 p<0.0001).

Conclusion: Low plasma AA is a strong, independent risk factor for acute ischemic
stroke. Antioxidant deficiency probably causes acute ischemic stroke. AA supplementation is recommended to prevent acute ischemic stroke, until the results of randomized trials are available.

BACKGROUND:

India is experiencing an epidemiological health transition characterized by rapid decline in nutritional and parasitic diseases (pre-transitional diseases) with an alarming rise in cardiovascular diseases, mainly coronary heart disease, and stroke (post-transitional diseases)\(^1\)\(^2\). The second half of the twentieth century witnessed a dramatic rise in cardiovascular diseases, mainly myocardial infarction and stroke. In India, they account for 15-20% of total deaths and are the leading cause of adult mortality and morbidity. This epidemic in India is accelerating and advancing rapidly and it is predicted by the World Bank that cardiovascular disease (CVD) mortality will double from 1985 to 2015\(^3\).

The present conventional risk factors are not able to fully explain this epidemic in India. Hence the search for new, modifiable, preventable risk factors continues. Among them antioxidant deficiency, homocysteine, selenium, lipoprotein(a), fibrinogen have received considerable attention. Antioxidant deficiency leads to unchecked production of free radicals, which possess an unpaired electron causing oxidative stress and have been implicated in the pathogenesis of atherosclerosis and cancers.

The role of antioxidants in the pathogenesis of CVD like stroke has generated considerable interest in recent times. Ascorbic acid is the most important and most common antioxidant. It not only limits oxidative damage but also reverses endothelial dysfunction, increases RBC glutathione and normalizes homocysteine\(^4\)\(^5\) and hence deficiency of ascorbic acid may be an important risk factor for acute ischemic stroke. There have been a plethora of articles studying the relationship of Vitamin C with acute ischemic stroke with contradictory results. Simon JA (1998)\(^6\) analyzed data from 6624 US men and women enrolled in the second National Health and Nutrition Examination Survey and reported that 0.05mg% decrease in serum AA resulted in 11 % reduction in stroke and coronary heart disease prevalence, while Ascherio (1999)\(^7\) showed that there was no relationship of serum ascorbic acid with stroke. More importantly, there have been no Indian studies, where nutritional deficiency of ascorbic acid may be common.

Amidst this controversy and lack of data from India, we performed this study with the aim to study the relationship of plasma Ascorbic Acid (AA) with acute ischemic stroke in univariate and multivariate context and we hypothesized that the plasma AA is significantly lower in cases of acute ischemic stroke as compared with age and sex matched controls after controlling for conventional risk factors.

**Study design:** Case Control Study. This was a hospital based, age group-sex- socio-economic status matched study.

**Setting:** Medicine Wards, Govt. Medical College, Nagpur

**Selection of Cases:** All consecutive cases of acute ischemic stroke admitted to Medicine wards. The diagnosis of acute ischemic stroke was based on clinical examination and
confirmed by CT scan. Only CT scan proved cases were included. We reviewed Medicine ward admissions daily for acute ischemic stroke cases and if eligible, consent was obtained. A detailed questionnaire was administered to enquire about dietary habits, prior vitamin intake, alcohol intake and smoking. Physical measurements were made. Height was measured without footwear in meters, and weight was measured in kilograms and body mass index was calculated as weight in meters, height in meters-square. Waist was measured with a non-stretchable tape in light clothing as the smallest diameter between the costal margin and the iliac crest. Hip was measured at the level of greater trochanter—generally the greatest diameter at the buttocks and Waist by Hip ratio was calculated.

Selection of controls: Age group (within five year range), sex and socio-economic status matched healthy controls, without any past or present evidence of stroke or transient ischemic attack (TIA), were selected randomly from subjects attending the outpatient department for minor complaints or relatives or attendants of these patients or from relatives or attendants of non cardiac patients admitted in non cardiac wards.

Exclusion Criteria: 1) Patients with known liver, thyroid, renal disease or malignancy. 2) Pregnancy. 3) Past or present evidence of stroke or Transient Ischemic Attack (TIA). 4) Refusal to give consent.

Statistical Analysis: Statistical analysis was performed using STAT AA version 5.0 (1997) software on a personal computer. Student ‘t’ test was used to compare continuous variables and chi-square test was used to compare categorical variables. Unadjusted Odds Ratios with 95% confidence intervals and exact ‘p’ values were computed. Multiple logistic regression analysis was also performed with acute ischemic stroke as the independent dichotomous outcome variable and conventional risk factors and plasma ascorbic acid as dependent predictor variables and adjusted Odds Ratios and 95% confidence intervals along with ‘p’ were calculated in multivariate analysis.

RESULTS:

104 cases and an equal number of controls were studied. There were 72 (69.2%) males and 32 (30.3%) females. The mean age in cases of stroke was 62.9 years ± 9.6 (range: 28-84 years), which was similar to mean age in controls (62.7 years). The mean plasma ascorbic acid was significantly lower in cases of acute ischemic stroke as compared with controls (Table 1).

Table 1: Comparison of mean plasma ascorbic acid in cases of stroke and controls

<table>
<thead>
<tr>
<th></th>
<th>Cases (n = 104)</th>
<th>Controls (n = 104)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean plasma ascorbic acid in μmol/L (± SD)</td>
<td>22.63 (± 6.43)</td>
<td>27.54 (± 3.58)</td>
<td>t = 6.80 p = 0.0001</td>
</tr>
</tbody>
</table>

Table 2: Comparison of low plasma ascorbic acid in cases of stroke and controls

<table>
<thead>
<tr>
<th>Plasma ascorbic acid</th>
<th>Cases n = 104</th>
<th>Controls n = 104</th>
<th>Total % n = 208</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low &lt;= 23 μmol/L</td>
<td>65 (62.5)</td>
<td>9 (8.65)</td>
<td>74 (35.58)</td>
<td>p&lt;0.0001 Chi Sq.65.78</td>
</tr>
<tr>
<td>Normal &gt; 23 μmol/L</td>
<td>39 (37.5)</td>
<td>95 (91.35)</td>
<td>134 (64.42)</td>
<td>Or 17.5 95% CI 6.77-45.39</td>
</tr>
</tbody>
</table>

The frequency of low plasma ascorbic acid defined as plasma ascorbic acid <=23 μmol/L.
was significantly lower in cases as compared with controls (Table 2)

### TABLE 3: Comparison of conventional risk factors for ischaemic stroke (Confounding variables) in cases and controls (Univariate Analysis)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Cases (%) n=104</th>
<th>Controls (%) n=104</th>
<th>Total (%) n=208</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean age</strong> (± SD) in yrs.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>62.92 (±9.63)</td>
<td>62.70 (±9.30)</td>
<td>62.81 (±9.44)</td>
<td>t=0.1676</td>
</tr>
<tr>
<td></td>
<td>28-84</td>
<td>30-80</td>
<td>28-84</td>
<td>p=0.8671</td>
</tr>
<tr>
<td><strong>Hypertension</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>+</td>
<td>34 (32.69)</td>
<td>7 (6.73)</td>
<td>41 (19.71)</td>
<td>Chi.sq.=22.15; OR = 6.73</td>
</tr>
<tr>
<td></td>
<td>(± SD)</td>
<td>(± SD)</td>
<td>(± SD)</td>
<td>95% CI 2.87-5.71</td>
</tr>
<tr>
<td></td>
<td>98-275</td>
<td>97 (93.27)</td>
<td>167 (80.29)</td>
<td><strong>p&lt;0.001</strong></td>
</tr>
<tr>
<td></td>
<td>70 (67.31)</td>
<td>97 (93.27)</td>
<td>167 (80.29)</td>
<td></td>
</tr>
<tr>
<td>-</td>
<td>94 (90.38)</td>
<td>101 (97.12)</td>
<td>195 (93.75)</td>
<td></td>
</tr>
<tr>
<td><strong>Diabetes Mellitus</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>+</td>
<td>10 (9.62)</td>
<td>3 (2.88)</td>
<td>13 (6.25)</td>
<td>Chi.sq.=4.02; OR=3.58</td>
</tr>
<tr>
<td></td>
<td>(± SD)</td>
<td>(± SD)</td>
<td>(± SD)</td>
<td>95% CI 1.02-12.40</td>
</tr>
<tr>
<td></td>
<td>94 (90.38)</td>
<td>101 (97.12)</td>
<td>195 (93.75)</td>
<td><strong>p=0.045</strong></td>
</tr>
<tr>
<td>-</td>
<td>94 (90.38)</td>
<td>101 (97.12)</td>
<td>195 (93.75)</td>
<td></td>
</tr>
<tr>
<td><strong>Mean serum cholesterol</strong> (± SD) in mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>161.97 (± 47.71)</td>
<td>150.11 (± 39.98)</td>
<td>156.04 (± 44.31)</td>
<td>t=1.94</td>
</tr>
<tr>
<td></td>
<td>98-275</td>
<td>102-270</td>
<td>98-275</td>
<td><strong>p=0.05</strong></td>
</tr>
<tr>
<td></td>
<td>16 (15.46)</td>
<td>14 (13.46)</td>
<td>26 (12.51)</td>
<td></td>
</tr>
<tr>
<td><strong>Total S. Cholesterol &lt; 200 mg%</strong></td>
<td>62 (59.64)</td>
<td>70 (66.35)</td>
<td>132 (63.57)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(± SD)</td>
<td>(± SD)</td>
<td>(± SD)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>98-275</td>
<td>102-270</td>
<td>98-275</td>
<td></td>
</tr>
<tr>
<td></td>
<td>32 (30.77)</td>
<td>14 (13.46)</td>
<td>46 (22.12)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(± 13.46)</td>
<td>(± 13.46)</td>
<td>(± 13.46)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>98-275</td>
<td>102-270</td>
<td>98-275</td>
<td></td>
</tr>
<tr>
<td><strong>Mean BMI</strong> (± SD)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>23.21 (± 3.73)</td>
<td>23.64 (± 3.59)</td>
<td>23.42 (± 3.66)</td>
<td>t=0.84</td>
</tr>
<tr>
<td></td>
<td>17.02-30.2</td>
<td>18.04-30.78</td>
<td>17.02-30.78</td>
<td><strong>p=0.39</strong></td>
</tr>
<tr>
<td></td>
<td>23 (22.12)</td>
<td>27 (25.53)</td>
<td>50 (24.11)</td>
<td></td>
</tr>
<tr>
<td><strong>BMI</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 25</td>
<td>39 (37.50)</td>
<td>30 (28.85)</td>
<td>69</td>
<td>Chi.sq.=1.76; OR=1.48</td>
</tr>
<tr>
<td></td>
<td>(± SD)</td>
<td>(± 13.46)</td>
<td>(± 13.46)</td>
<td>95% CI 0.83-2.63</td>
</tr>
<tr>
<td></td>
<td>98-275</td>
<td>102-270</td>
<td>98-275</td>
<td><strong>p=0.1850</strong></td>
</tr>
<tr>
<td>&lt; 25</td>
<td>65 (62.50)</td>
<td>74 (71.15)</td>
<td>139</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(± SD)</td>
<td>(± 13.46)</td>
<td>(± 13.46)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>98-275</td>
<td>102-270</td>
<td>98-275</td>
<td></td>
</tr>
<tr>
<td><strong>Mean W:H</strong> (± SD)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>0.856 (± 0.044)</td>
<td>0.844 (± 0.024)</td>
<td>0.850 (± 0.036)</td>
<td>t=2.23</td>
</tr>
<tr>
<td></td>
<td>0.8-0.96</td>
<td>0.8-0.92</td>
<td>0.8-0.96</td>
<td><strong>p=0.026</strong></td>
</tr>
<tr>
<td></td>
<td>(± SD)</td>
<td>(± 0.024)</td>
<td>(± 0.036)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>98-275</td>
<td>102-270</td>
<td>98-275</td>
<td></td>
</tr>
<tr>
<td>Abnormal</td>
<td>20 (19.23)</td>
<td>16 (15.38)</td>
<td>36 (17.31)</td>
<td>Chi.sq.=0.54; OR=1.309</td>
</tr>
<tr>
<td></td>
<td>(± SD)</td>
<td>(± 13.46)</td>
<td>(± 13.46)</td>
<td>95% CI 0.64-2.67</td>
</tr>
<tr>
<td></td>
<td>98-275</td>
<td>102-270</td>
<td>98-275</td>
<td><strong>p=0.4635</strong></td>
</tr>
<tr>
<td>Normal</td>
<td>84 (80.77)</td>
<td>88 (84.62)</td>
<td>172 (82.69)</td>
<td></td>
</tr>
<tr>
<td><strong>Smoking</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>+</td>
<td>42 (40.38)</td>
<td>28 (26.92)</td>
<td>70 (33.65)</td>
<td>Chi.sq.=4.22; OR=1.83</td>
</tr>
<tr>
<td></td>
<td>(± SD)</td>
<td>(± 13.46)</td>
<td>(± 13.46)</td>
<td>95% CI 1.02-3.28</td>
</tr>
<tr>
<td></td>
<td>98-275</td>
<td>102-270</td>
<td>98-275</td>
<td><strong>p=0.0399</strong></td>
</tr>
<tr>
<td>-</td>
<td>62 (59.62)</td>
<td>76 (73.08)</td>
<td>138 (66.35)</td>
<td></td>
</tr>
<tr>
<td><strong>Alcohol intake</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>+</td>
<td>20 (19.23)</td>
<td>13 (12.50)</td>
<td>33 (15.87)</td>
<td>Chi.sq.=1.76; OR=1.66</td>
</tr>
<tr>
<td></td>
<td>(± SD)</td>
<td>(± 13.46)</td>
<td>(± 13.46)</td>
<td>95% CI 0.788-3.51</td>
</tr>
<tr>
<td></td>
<td>98-275</td>
<td>102-270</td>
<td>98-275</td>
<td><strong>p=0.1840</strong></td>
</tr>
<tr>
<td>-</td>
<td>84 (80.77)</td>
<td>91 (87.50)</td>
<td>175 (84.13)</td>
<td></td>
</tr>
<tr>
<td><strong>F/Hofstroke</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>+</td>
<td>10 (9.62)</td>
<td>6 (5.77)</td>
<td>16 (7.69)</td>
<td>Chi.sq.=1.08; OR=1.73</td>
</tr>
<tr>
<td></td>
<td>(± SD)</td>
<td>(± 6.77)</td>
<td>(± 13.46)</td>
<td>95% CI 0.629-4.75</td>
</tr>
<tr>
<td></td>
<td>98-275</td>
<td>102-270</td>
<td>98-275</td>
<td><strong>p=0.2980</strong></td>
</tr>
<tr>
<td>-</td>
<td>94 (90.38)</td>
<td>98 (94.23)</td>
<td>192 (92.31)</td>
<td></td>
</tr>
</tbody>
</table>

HT= Hypertension, DM = Diabetes Mellitus, BMI = Body Mass Index, W:H = Waist : Hip, F/H = Family History
In univariate analysis, hypertension, diabetes mellitus, mean serum cholesterol, mean Waist: Hip Ratio and cigarette smoking were significantly associated with acute ischemic stroke (Table 3). In multiple logistic regression analysis with acute ischemic stroke as dichotomous outcome variable, low plasma ascorbic acid was found to be significantly associated with ischemic stroke after controlling for all the confounding variables in multivariate analysis. In addition to low plasma AA, hypertension and hypercholesterolemia were found to be independently associated with ischemic stroke in multivariate analysis. (Table 4)

**TABLE 4: Multiple Logistic Regression showing association of various risk factors with ischaemic stroke (Plasma AA, Sr. Cholesterol, BMI and W:H ratio were taken as categorical variables)**

<table>
<thead>
<tr>
<th>Risk Factors</th>
<th>Adjusted odds ratio</th>
<th>95% CI</th>
<th>‘Z’ value</th>
<th>‘p’ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low plasma ascorbic acid (&lt; 23 mmol/L)</td>
<td>18.06</td>
<td>7.58 - 43.11</td>
<td>6.519</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Hypertension</td>
<td>4.93</td>
<td>1.66 - 14.69</td>
<td>2.87</td>
<td>0.004</td>
</tr>
<tr>
<td>Diabetes Mellitus</td>
<td>3.35</td>
<td>0.62 - 17.93</td>
<td>1.41</td>
<td>0.156</td>
</tr>
<tr>
<td>Hypercholesterolemia (³ 200 mg/dl)</td>
<td>2.87</td>
<td>1.16-7.10</td>
<td>2.29</td>
<td>0.022</td>
</tr>
<tr>
<td>Body Mass Index (³ 25)</td>
<td>1.19</td>
<td>0.53 - 2.67</td>
<td>0.43</td>
<td>0.666</td>
</tr>
<tr>
<td>Abnormal W : H ratio in male ³ 0.95, in female ³ 0.85</td>
<td>1.91</td>
<td>0.70 - 5.16</td>
<td>1.275</td>
<td>0.202</td>
</tr>
<tr>
<td>Smoking</td>
<td>2.12</td>
<td>0.90 - 4.95</td>
<td>1.74</td>
<td>0.082</td>
</tr>
<tr>
<td>Alcohol</td>
<td>0.92</td>
<td>0.30 - 2.79</td>
<td>0.131</td>
<td>0.896</td>
</tr>
<tr>
<td>Family History of stroke</td>
<td>3.67</td>
<td>0.98 - 13.62</td>
<td>1.94</td>
<td>1.944</td>
</tr>
</tbody>
</table>

We also analyzed the association of low plasma ascorbic acid with the conventional risk factors and found plasma ascorbic acid to be significantly lower in elderly, males, hypertensives, diabetics, smokers, alcohol intake and those with hypercholesterolemia.

**Discussion:**

We found plasma AA to be significantly lower in cases of acute ischemic stroke as compared to controls, independent of the conventional risk factors. Plasma AA was also found to be lower in those with conventional risk factors like advancing age, male sex, smoking, high blood pressure and diabetes mellitus.

L-Ascorbic Acid (Vitamin C) is considered to be the most important and most common dietary antioxidant. It forms the first line of defense against oxidative damage. Oxidative modification of LDL, a process called lipid peroxidation, is an important step in the pathogenesis of atherosclerosis, which can be modified by use of antioxidants. Ascorbic acid
is not synthesized in the human body. Hence plasma levels are more conclusive indicator of the antioxidant status than the calculated dietary intake, since actual status in vivo is the outcome of the bioavailability of antioxidant (depending on the dietary supply, intestinal absorption, hepatic excretion and metabolic regulations)\textsuperscript{11}.

Several studies have indicated a low level in plasma and low dietary intake of vitamin C with high blood pressure\textsuperscript{13} and smoking is inversely associated with serum vitamin C levels in adults\textsuperscript{14}. Oxidative stress has been proposed to play an important role in diabetes mellitus\textsuperscript{12}.

Increased intake of fruits and vegetables result in increase of plasma concentrations of vitamin C, alfa carotene and beta carotene, which protects against cardiovascular diseases and stroke\textsuperscript{15} by reducing the susceptibility of low density lipoprotein to oxidation\textsuperscript{16}. Studies from North India have shown that cardioprotective diet containing plenty of fresh fruits and vegetables reduces mortality after myocardial infarction\textsuperscript{17}. Guava fruit has been shown to decrease blood pressure while garlic decreases blood cholesterol and increases blood fibrinolytic activity\textsuperscript{18}. The Framingham study has shown that regular intake of fruits and vegetables of three or more servings per day was associated with 22% reduction in incidence of stroke in men\textsuperscript{19}. Antioxidant supplemented drink can reduce lipid peroxidation and susceptibility of LDL to oxidation in smokers and may ameliorate the oxidative stress of cigarette smoke\textsuperscript{20}.

The implications of this study are profound and the applications are widespread. Plasma AA is a strong, independent risk factor for stroke, and more importantly it is modifiable. Antioxidant status is probably important in the causation of stroke. Widespread consumption of fresh fruits and vegetable, which are rich in ascorbic acid, or vitamin C supplementation in pill form, may prevent stroke. However, this hypothesis needs to be tested by a large randomized controlled trial in Indian context, where dietary intake of ascorbic acid is presumably low.

REFERENCES


4. Frie B. Ascorbic acid protects lipids in human blood plasma and low density lipoprotein against oxidative damage. Am J Clin Nutr 1991; 54 (III), 35S (III) 8S.


ABSTRACT

Purpose: To study the role of autoinfection in surgical site infection.

Materials and methods: In the period from May 2001 to July 2002, 190 patients admitted for surgery (clean and clean-contaminated elective cases) were assessed preoperatively, intraoperatively and postoperatively. Normal microbial flora was studied within 24 to 48 hours of admission and patients were followed up to 30 days postoperatively to look for development of surgical site infection. Infected wounds were studied bacteriologically and clinically.

Results: The overall infection rate was 8.95%. Surgical site infection rate was 3.03% in clean surgeries and 22.41% in clean-contaminated surgeries. Autoinfection developed in two cases, one due to Staphylococcus aureus and other due to Escherichia coli.

Key words: Autoinfection, Surgical site infection, Clean and Clean-contaminated surgeries, Staphylococcus aureus, Escherichia coli.

INTRODUCTION

Surgical site infections are the third most commonly reported nosocomial infection and they account for approximately a quarter of all nosocomial infections.1-3 Surgical site infections usually arise as a result of intraoperative seeding of exogenous bacteria or as a consequence of dissemination of endogenous bacteria to the operative site. Exogenous bacteria are generally transmitted, as a result of direct or indirect contact, from fomites or operating room personnel. The latter include heavy droplets of bacteria-laden particles transmitted through the air for a short distance.4 Though many workers have studied the role of exogenous source in surgical site infection; there are very few reports on the role of endogenous organisms as a source of SSI.5-8 Hence this study was undertaken to know the role of autoinfection in surgical site infection.

MATERIALS AND METHODS

The present study was conducted on patients admitted for surgery in general surgery...
units of Grant Medical College and Sir J. J. Group of Hospitals, Mumbai. A total of 190 clean and clean-contaminated elective surgeries in two general surgery units from May 2001 to July 2002 were included in the study.

The patients were assessed preoperatively, intraoperatively and postoperatively. Normal microbial flora of all patients was studied within a period of 24 to 48 hours of the admission to the general surgical ward. The sites included nose, throat, axilla, skin (the site of operation), perineum, and rectum.

Each patient was followed up from the time of admission till discharge from the hospital and also for 30 days postoperatively. Surgical wound was inspected at the time of first dressing and weekly thereafter for 30 days. Wound infection was diagnosed if any one of the following criteria were fulfilled:

- serous or non-purulent discharge from the wound.
- pus discharge from the wound.
- serous or non-purulent discharge from the wound with signs of inflammation (oedema, redness, warmth, raised local temperature, fever >38°C, tenderness, induration).
- wound deliberately opened up by the surgeon due to localized collection (serous/purulent).

[Note: Stitch abscesses were excluded from this study.]

Swabs obtained from infected wounds were processed aerobically and anaerobically & isolates were identified by standard laboratory methods. Staphylococcus aureus strains were sent for phage typing to National Reference Centre for Staphylococcal Phage Typing, Department of Microbiology, Maulana Azad Medical College, New Delhi-110002. Data were evaluated by Chi square (X²) statistical test. P £ 0.05 was considered to be significant.

**RESULTS AND OBSERVATIONS**

Out of total 190 patients, 132 (69.47%) were clean surgeries and 58 (30.53%) were clean-contaminated surgeries (table-1, 2).

Of the total 190 patients included in this study, 17 developed surgical site infection with the overall infection rate of 8.95%. Surgical site infection rate was 3.03% in clean surgeries and 22.41% in clean-contaminated surgeries (table-1).

Out of 190 patients studied, 56 (29.47%) were nasal carriers of Staphylococcus aureus. Coagulase-negative staphylococcus was the commonest isolate from all the sites except throat and rectum where Streptococcus viridans and Escherichia coli were the commonest isolates respectively (table-3). Amongst the gram-negative bacilli, Escherichia coli was the commonest normal flora organism and was isolated mostly from rectum and perineum. Gram-negative bacilli were more frequently isolated from perineum and rectum as compared to other sites (table-3).

The frequencies with which various organisms were isolated from normal flora sites in patients who developed surgical site infection have been depicted in table-4.

Of the total 190 operated patients, 17 developed surgical site infection according to the criteria used. Three of 17 infected wounds were culture negative. From the remaining 14 infected wounds, total 16 isolates were recovered (table-5). Of these, Staphylococcus
Staphylococcus aureus was isolated from six infected wounds (37.5%) while gram-negative bacilli were isolated from 62.5% of infected cases.

In one patient who underwent mastectomy, strain of Staphylococcus aureus isolated from the infected wound postoperatively and the strain isolated from the axilla during normal flora surveillance preoperatively, were having same antibiogram and belonged to the same phage type (type 80). In remaining five patients with Staphylococcus aureus wound infection, Staphylococcus aureus was not found to be carried at any of the normal flora sites during preoperative surveillance.

In another patient who underwent ileal resection anastomosis for stricture of the terminal ileum, Escherichia coli was isolated from the patient’s infected wound postoperatively, and also from the same patient’s perineum and rectum preoperatively as a normal flora. Due to non-availability of sero-typing and colicin-typing facility for Escherichia coli typing, these strains were studied on basis of antibiogram. All these three strains had similar antibiograms.

In rest of the patients, normal flora isolated did not match the wound isolate.

DISCUSSION

Source of postoperative wound infection may be exogenous or endogenous, but it is difficult to determine the actual source, which led to the development of particular infection. National Research Council report14 indicates that 2/3rd of postoperative infections of all types in general surgery are due to endogenous source. It is known that the development of SSI involving Staphylococcus aureus is definitely associated with preoperative nares carriage of the organism in surgical patients.1 On the basis of phage typing; Davidson et al6 reported auto infection in four of the 60 staphylococcal skin carriers. Ashok Kumar et al (1985),5 reported autoinfection of surgical wound due to Staphylococcus aureus skin carriage. In the present study, out of the 17 patients who developed surgical site infection, possibility of autoinfection could be considered in only two patients. One was a case of mastectomy for carcinoma of the breast where Staphylococcus aureus was isolated from the infected wound and the patient was an axillary carrier of Staphylococcus aureus as detected during preoperative normal flora surveillance. Another was a case of ileal resection anastomosis for stricture of the terminal ileum where the wound isolate was Escherichia coli and Escherichia coli was isolated from the patient’s perineum and rectum preoperatively.

In both the cases cited above, circumstantial evidence indicates endogenous contamination of the wound leading to autoinfection. Staphylococcus aureus wound infection due to endogenous source has been reported by many workers. On the contrary, very few studies have reported endogenous acquisition of gram negative enteric bacilli with rectum, perineum, groin and urethra acting as sources.5,8

Table-1: Surgical site infection rate by wound classification.

<table>
<thead>
<tr>
<th>Wound Class</th>
<th>Number of Patients</th>
<th>Number Infected</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clean</td>
<td>132</td>
<td>4</td>
<td>3.03</td>
</tr>
<tr>
<td>Clean-contaminated</td>
<td>58</td>
<td>13</td>
<td>22.41</td>
</tr>
<tr>
<td>Total</td>
<td>190</td>
<td>17</td>
<td>8.95</td>
</tr>
</tbody>
</table>
Table-2: Infection rate in various specific operations.

<table>
<thead>
<tr>
<th>Operation</th>
<th>Number Performed</th>
<th>Number Infected (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hydrocele</td>
<td>27</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Inguinal Hernia</td>
<td>57</td>
<td>1 (1.75)</td>
</tr>
<tr>
<td>Other Hernias</td>
<td>13</td>
<td>2 (15.38)</td>
</tr>
<tr>
<td>Appendix*</td>
<td>10</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Hepato-biliary*</td>
<td>21</td>
<td>3 (14.29)</td>
</tr>
<tr>
<td>Breast (Malignancy)</td>
<td>4</td>
<td>1 (25)</td>
</tr>
<tr>
<td>Breast (Fibroadenoma)</td>
<td>9</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Lungs and Thoracic Cavity*</td>
<td>9</td>
<td>4 (44.44)</td>
</tr>
<tr>
<td>Thyroid and Parathyroid</td>
<td>7</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Upper urinary*</td>
<td>8</td>
<td>1 (12.5)</td>
</tr>
<tr>
<td>Lower urinary and Genital*</td>
<td>8</td>
<td>2 (25)</td>
</tr>
<tr>
<td>Oesophageal, Gastric and Small Bowel*</td>
<td>6</td>
<td>1 (16.67)</td>
</tr>
<tr>
<td>Large Bowel*</td>
<td>2</td>
<td>2 (100)</td>
</tr>
<tr>
<td>Others</td>
<td>9</td>
<td>0 (0)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>190</strong></td>
<td><strong>17 (8.95)</strong></td>
</tr>
</tbody>
</table>

*Clean-contaminated surgeries

Table-3: Normal flora organisms from the entire study population.

<table>
<thead>
<tr>
<th>Organisms Recovered</th>
<th>Nose</th>
<th>Throat</th>
<th>Axilla</th>
<th>Skin</th>
<th>Perineum</th>
<th>Rectum</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNS(122)</td>
<td>CNS(53)</td>
<td>CNS(167)</td>
<td>CNS(153)</td>
<td>CNS(138)</td>
<td>CNS(97)</td>
<td></td>
</tr>
<tr>
<td>S. aureus(56)</td>
<td>S. aureus(19)</td>
<td>S. aureus(11)</td>
<td>S. aureus(4)</td>
<td>S. aureus(13)</td>
<td>S. aureus(11)</td>
<td></td>
</tr>
<tr>
<td>S. viridans(27)</td>
<td>S. viridans(107)</td>
<td>S. viridans(12)</td>
<td>S. viridans(2)</td>
<td>S. viridans(14)</td>
<td>S. viridans(9)</td>
<td></td>
</tr>
<tr>
<td>β-haemolytic</td>
<td>β-haemolytic</td>
<td>β-haemolytic</td>
<td>β-haemolytic</td>
<td>β-haemolytic</td>
<td>β-haemolytic</td>
<td></td>
</tr>
<tr>
<td>streptococci(5)</td>
<td>streptococci(78)</td>
<td>streptococci(3)</td>
<td>K. pneumoniae(3)</td>
<td>streptococci(2)</td>
<td>streptococci(3)</td>
<td></td>
</tr>
<tr>
<td>M. catarrhalis(3)</td>
<td>M. catarrhalis(28)</td>
<td>E. coli(1)</td>
<td>K. oxytoca(1)</td>
<td>Enterococci(11)</td>
<td>Enterococci(20)</td>
<td></td>
</tr>
<tr>
<td>K. pneumoniae(3)</td>
<td>K. pneumoniae(2)</td>
<td>K. pneumoniae(1)</td>
<td>P. mirabilis(1)</td>
<td>E. coli(75)</td>
<td>E. coli(127)</td>
<td></td>
</tr>
<tr>
<td>P. mirabilis(1)</td>
<td>NFGNB(1)</td>
<td>Acinetobacter</td>
<td>Acinetobacter</td>
<td>K. pneumoniae(26)</td>
<td>K. pneumoniae(32)</td>
<td></td>
</tr>
<tr>
<td>NFGNB(1)</td>
<td>Candida species(3)</td>
<td>species(1)</td>
<td>species(1)</td>
<td>K. oxytoca(5)</td>
<td>K. oxytoca(4)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>NFGNB(3)</td>
<td>NFGNB(2)</td>
<td>P. mirabilis(9)</td>
<td>P. mirabilis(13)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Candida species(10)</td>
<td>Candida species(2)</td>
<td>C. diversus(2)</td>
<td>Acinetobacter species(2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>NFGNB(8)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

CNS: Coagulase-negative staphylococci
NFGNB: Non-fermenter gram-negative bacilli,
Table-4: Normal flora and infected wound organisms recovered from patients who developed SSI.

<table>
<thead>
<tr>
<th>Organisms Recovered</th>
<th>Nose</th>
<th>Throat</th>
<th>Axilla</th>
<th>Skin</th>
<th>Perineum</th>
<th>Rectum</th>
<th>Wound</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNS(12)</td>
<td>CNS(4)</td>
<td>CNS(15)</td>
<td>CNS(15)</td>
<td>CNS(13)</td>
<td>CNS(8)</td>
<td>CNS(6)*</td>
<td>CNS(4)</td>
</tr>
<tr>
<td>S. aureus(4)</td>
<td>S. aureus(2)</td>
<td>S. aureus(2)*</td>
<td>CNS(15)</td>
<td>CNS(13)</td>
<td>Enterococcus(2)</td>
<td>E. coli(2)*</td>
<td>CNS(4)</td>
</tr>
<tr>
<td>S. viridans(2)</td>
<td>S. viridans(9)</td>
<td>S. viridans(1)</td>
<td>CNS(15)</td>
<td>CNS(13)</td>
<td>K. pneumoniae(2)</td>
<td>K. pneumoniae(2)</td>
<td>CNS(4)</td>
</tr>
<tr>
<td>b-haemolytic</td>
<td>Acinetobacter species(1)</td>
<td>Acinetobacter species(1)</td>
<td>CNS(15)</td>
<td>CNS(13)</td>
<td>K. pneumoniae(5)</td>
<td>K. pneumoniae(8)</td>
<td>CNS(4)</td>
</tr>
<tr>
<td>streptococci(6)</td>
<td>C. diversus(1)</td>
<td>NFGNB(1)</td>
<td>CNS(15)</td>
<td>CNS(13)</td>
<td>NFGNB(1)</td>
<td>NFGNB(1)</td>
<td>CNS(4)</td>
</tr>
<tr>
<td>M. catarrhalis(2)</td>
<td>CNS(12)</td>
<td>CNS(4)</td>
<td>CNS(15)</td>
<td>CNS(13)</td>
<td>CNS(8)</td>
<td>CNS(6)*</td>
<td>CNS(4)</td>
</tr>
<tr>
<td>CNS: Coagulase-negative staphylococci.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NFGNB: Non-fermenter gram-negative bacilli.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* In one patient who underwent mastectomy for breast cancer, strain of Staphylococcus aureus isolated from the infected wound was similar to strain carried in axilla preoperatively.

@ In one patient who underwent ileal resection anastomosis for stricture of the terminal ileum, strain of Escherichia coli isolated from the infected wound was similar to strain carried in perineum and rectum preoperatively.

Table-5: Frequency of various pathogens causing surgical site infection.

<table>
<thead>
<tr>
<th>Organisms isolated</th>
<th>Number (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Staphylococcus aureus</td>
<td>4+2* (37.5)</td>
</tr>
<tr>
<td>Pseudomonas aeruginosa</td>
<td>4 (25)</td>
</tr>
<tr>
<td>Escherichia coli</td>
<td>2 (12.5)</td>
</tr>
<tr>
<td>Klebsiella pneumoniae</td>
<td>1 (6.25)</td>
</tr>
<tr>
<td>Citrobacter diversus</td>
<td>1 (6.25)</td>
</tr>
<tr>
<td>Non-fermenter gram-negative bacilli</td>
<td>1 (6.25)</td>
</tr>
<tr>
<td>Acinetobacter species*</td>
<td>1 (6.25)</td>
</tr>
<tr>
<td>Total</td>
<td>16 (100)</td>
</tr>
</tbody>
</table>

*In two cases, Staphylococcus aureus was isolated along with other organisms, one with Escherichia coli and another with Acinetobacter species.

REFERENCES


ABSTRACT

Genital ulceration is a common presentation in patients attending S. T. D. clinic. Chancroid is a common differential diagnosis of genital ulcer in tropical countries. The diagnosis is often clinical and laboratory assistance is sought sparingly. Syndromic approach to the treatment is popular and also recommended. Strong association between genital ulceration and HIV sero-positivity has been well established; thus requiring rapid healing of ulcer to reduce transmission of HIV. Advanced non-culture diagnostic tests like PCR, use of DNA probes, immunofluorescence using monoclonal antibodies etc. are out of reach in our country. An attempt has, therefore, been made to establish techniques for definitive diagnosis of Chancroid and to understand the in vitro resistance pattern of the *Haemophilus ducreyi* in Mumbai. Seventy two patients with genital ulceration are studied. Culture for *H. ducreyi*, DGI for *Treponema pallidum*, wet mount for Candida and *T vaginalis*, Tzank cells or Donovan bodies by Giemsa stain were the tests attempted. Presumptive diagnosis of Chancroid was made on Gram stain in 38 cases (52.8%) and 22 samples (30.6%) yielded *H. ducreyi* on culture. Erythromycin was found to be the best antibiotic to which 21 of the 22 isolates were susceptible (95.5%). Twenty samples (27.8%) were found to be reactive for HIV I &/or II.

INTRODUCTION

Genital ulceration is a common presentation in patients attending S. T. D. clinic.
The diagnosis is often clinical and laboratory assistance is sought sparingly, when the patients do not respond to treatment. Clinical diagnosis is often syndromic rather than being precise. Also, the therapeutic regimens employed to treat various etiologies of genital ulceration show marked diversity (1) in spite of availability of national guidelines on STD syndromic management (2). Strong association between genital ulceration and HIV seropositivity has been well established; thus requiring rapid healing of ulcer to reduce transmission of HIV (3).

Chancroid is one of the common differential diagnosis of genital ulcer in tropical countries (4). The diagnosis is often made by exclusion and Gram staining even in tertiary care centers. Advanced non-culture diagnostic tests like PCR, use of DNA probes, immunofluorescence using monoclonal antibodies etc. are out of reach in our country. An attempt has, therefore, been made to establish techniques for definitive diagnosis of Chancroid and to understand the in vitro resistance pattern of the Haemophilus ducreyi in Mumbai.

MATERIAL AND METHODS

A total of 72 patients presenting with genital ulceration were included in the present study. Only one of them was a female and the rest were males belonging to low socioeconomic group. All the male patients gave the history of exposure to commercial sex workers (CSWs). The age range was between 17 to 51 years. This study was undertaken from January 1995 to October 1995.

The ulcers were cleaned with sterile gauze pieces soaked in sterile saline. Exudate from the margin of the ulcer was collected by sterile cotton swabs soaked in normal saline. The swab was rolled in only one direction to collect the material. Three swabs were collected from each patient for Gram, Giemsa and Fontana staining and for culture on MH-Blood agar with supplements (5). Effort was made to express exudates from these ulcers for DGI examination. A wet mount examination was also performed.

Blood was collected from each patient for VDRL, TPHA and HIV-ELISA. The sera reactive for HIV were confirmed by second ELISA.

The colony morphology and colony smears were studied carefully. The identification and antibiotic sensitivity was done according to WHO guidelines (5).

OBSERVATIONS

Out of the 72 patients included in this study one patient was below 20 years of age. The age group between 21 to 30 years included 29 patients (40.3 %) followed by and 31.9 % in 31 to 40 age group. In all 52 (72.2 %) cases in our study belonged to sexually active age group between 21 to 40 years of age. (Table I)

1. All the samples were examined by DGI for T pallidum of which two were positive.

2. Wet mount examination of these samples did not reveal Candida or T. vaginalis.
None of the samples were positive for Tzank cells or Donovan bodies by Giemsa stain.

Six ulcers revealed *T. pallidum* like organisms in smears on Fontana staining.

Presumptive diagnosis of Chancroid was made on Gram stain in 38 cases (52.8%).

Exudates from all the cases were studied by Gram stain for *Haemophilus ducreyi*. Out of the 72 samples 38 samples revealed characteristic Gram negative, intra or extra cellular, pleomorphic coccobacilli, arranged in clumps or in whorls. At places organisms were placed along mucus threads. However the "school of red fish" and "rail road pattern" was not observed. Smears showed a large number of pus cells in the background.

All the samples were cultured on MHBA for isolation of *H. ducreyi*. 22 samples out of 72 (30.6 %) yielded *H. ducreyi*. The colonies were observable by naked eye at the end of 48 hrs. In the beginning they were very small. The colonies enlarged on further incubation to reach the size of 2 mm, at the end of five days. In the initial phase the colonies could be moved intact on the surface of the medium and it was difficult to pick up the colony for further processing. On further incubation the colonies became softer and opaque.

Antibiotic sensitivity was done by Kirby Bauer’s disk diffusion method. Following antibiotics were used viz., Ceftriaxone (110 mg), Ciprofloxacin (10 mg), Erythromycin (15 mg), Penicillin (10 IU) and Tetracycline (30 mg). Erythromycin was found to be the best antibiotic to which 21 of the 22 isolates were susceptible (95.5 %). Ceftriaxone was found to be the second best antibiotic in the present study. Twenty strains (90.9 %) were susceptible to it. Tetracycline to which only 31.8 per cent strains were sensitive (7 out of 22) was the least effective antibiotic (Table II).

All the sera drawn were tested for HIV I & II antibody using two different ELISA kits namely Detect HIV followed by Immunocomb. Twenty samples (27.8 %) were found to be reactive. The details of these HIV reactive 20 patients are given in table III.

Culture or serology for HSV -2 could not be done.

**DISCUSSION AND CONCLUSIONS:**

*Haemophilus ducreyi* is one of the commonest pathogen known to cause genital ulcerations in the tropical countries. In tertiary care units it is necessary to confirm clinical diagnosis with appropriate laboratory support. It has been a frequent observation that clinical presentation of sexually transmitted diseases and their laboratory diagnosis show considerable discrepancy (6). Other ulcerative STDs are known to mimic Chancroid closely (5,7). Laboratory diagnosis of Chancroid is particularly problematic because of fastidious nature of the pathogen (8).

Out of the 72 patients evaluated, 38 showed presence of organisms suggestive of *H. ducreyi* on Gram staining. The organisms were usually present in clumps or whorls or arranged along the mucus threads. The typical
“school of red fish” appearance or the classical “railroad” arrangement was never found in direct smears.

There is paucity of literature on isolation and antibiotic sensitivity pattern of \textit{H. ducreyi} from India thus making it difficult to compare or understand the prevalence rate of this infection. In the present study \textit{H. ducreyi} were isolated in 22 cases out of the 72 patients (30.6%). However the incidence reported by many workers ranges from 61 % to 71 % (9,10,11). The low isolation rate in the present study may be attributed to indiscriminate use of antibiotics and use of single isolation medium for \textit{H. ducreyi}. To establish true prevalence of this infection it would be worthwhile to determine antibody prevalence to \textit{H. ducreyi} among patients attending STD clinic (12).

\textit{H. ducreyi} has been found to show resistance to variety of antibiotics. Strains carrying transferable plasmids are also known (3). It is very important to have a correct and periodic estimation of resistance pattern of \textit{H ducreyi} isolates in any geographical area to choose a preferred antibiotic. (Because bacteria are known to dishonor the WHO recommendations). This would provide an appropriate direction to the therapeutic approach in a patient of genito-ulcerative disease. Erythromycin with 95.5% sensitivity was found to be the best antibiotic followed by ceftriaxone (90.9% sensitivity) in our study. Tetracycline showing only 31.8% sensitivity was the least effective antibiotic for strains belonging to this area.

All the 72 sera tested for HIV I & II antibody, 20 samples (27.8%) were reactive by two different ELISA tests. HIV sero-reactivity in ulcerative genital lesions in our area is very high. Immediate and proper curative treatment of genital ulcers to prevent HIV transmission is essential. The high prevalence of HIV reactivity in genital ulcerative diseases may be attributed to collection of inflammatory cells at the site of ulcer. Some of the cells in this collection carry CD4 antigen on their surface, which are ‘Welcome gates’ for the HIV (3).

Sexually Transmitted Diseases are observed more frequently in sexually active age group of 21 to 40 years all over the world. Present study showed a similar pattern giving a percentage of 72.22 patients falling in this age range.

Thus in conclusion, implementation of techniques for quick microbiological diagnosis of genito-ulcerative lesions is imperative. Periodic review of resistance pattern of \textit{H. ducreyi} must be taken for a specified geographical area. Young and sexually active age group should be the target for preventive and curative therapy with most appropriate antibiotic.

\begin{table}[h]
\centering
\begin{tabular}{|c|c|c|}
\hline
\textbf{AGE} & \textbf{NUMBER} & \textbf{PERCENTAGE} \\
\hline
<20 & 01 & 1.4 \\
21-30 & 29 & 40.3 \\
31-40 & 23 & 31.9 \\
41-50 & 12 & 16.9 \\
51 & above & 07 & 09.7 \\
\hline
\end{tabular}
\caption{Age wise distribution of cases.}
\end{table}
TABLE-II. ANTIBIOTIC SENSITIVITY PATTERN OF THE ISOLATES BY DISC DIFFUSION METHOD

<table>
<thead>
<tr>
<th>ANTI BIOTIC</th>
<th>POTENCY</th>
<th>NO. SENSITIVE</th>
<th>PERCENTAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>CEFTRIAXONE</td>
<td>10 mcg</td>
<td>20</td>
<td>90.9</td>
</tr>
<tr>
<td>CIPROFLOXACIN</td>
<td>10 mcg</td>
<td>18</td>
<td>81.8</td>
</tr>
<tr>
<td>ERYTHROMYCIN</td>
<td>15 mcg</td>
<td>21</td>
<td>95.5</td>
</tr>
<tr>
<td>PENICILLIN</td>
<td>10 units</td>
<td>12</td>
<td>54.5</td>
</tr>
<tr>
<td>TETRACYCLINE</td>
<td>30 mcg</td>
<td>07</td>
<td>31.8</td>
</tr>
</tbody>
</table>

n = 72

TABLE-III PREVALENCE OF HIV ANTIBODIES

<table>
<thead>
<tr>
<th>HIV / ELISA reactive</th>
<th>Total sera tested</th>
<th>72</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIV + Chancroid</td>
<td>20</td>
<td>20 (27.80%)</td>
</tr>
<tr>
<td>HIV + Syphilis</td>
<td>...</td>
<td>4</td>
</tr>
<tr>
<td>HIV + Chancroid + Syphilis</td>
<td>...</td>
<td>3</td>
</tr>
<tr>
<td>HIV + Undiagnosed ulcer</td>
<td>...</td>
<td>7</td>
</tr>
</tbody>
</table>

HSV-2 infection was not confirmed

REFERENCES

ABSTRACT

In India, an estimated 6 million people are presently infected with HIV. The degree of HIV disease progression in different cohorts has been observed to be varying in nature. The host factors involved in the development of AIDS include the cytokine responses, CD4 T cell counts, as well as the opportunistic infections the host suffers from. The time taken for all the antibody bands to become positive on a Western blot varies from a few weeks to several months after acute infection. Cellular immune responses during primary HIV infection include elevated levels of IFN-γ, TNF-α, Neopterin and β-2-microglobulin and decrease in the CD4+T cell levels. Cytokine induced immune dysregulation is a central component in progression to AIDS. A qualitative and quantitative depletion of B lymphocytes is also seen in HIV. Autoimmune responses have also been observed in HIV infection. The final outcome in HIV infections is the dominance of the detrimental counterpart of the chronic state of immune activation and severe deterioration of the immune system.

Key Words: HIV/AIDS, cytokines, CD4+T cells

Acquired immunodeficiency syndrome (AIDS) cases have been diagnosed in most regions of the world. The spread of HIV infection to some of the most populous areas of the world threatens future development in these countries. As per the updated reports of the UNAIDS/WHO Working Group on Global HIV/AIDS and STD Surveillance, the estimated number of individuals living with HIV/AIDS is 40 million. Of the 37.2 million infected adults, women constitute about 17.6 million. In addition, 2.7 million children less than 15 years are infected with HIV/AIDS. About 14,000 new HIV infections occur in a day. More than 95%
of those infected are from the developing world. Seventy six percent of the world's infections are in Africa, followed by Asia with about 20% and Latin America with 4%. The annual death rate due to AIDS is about 3 million, of which adults constitute 2.4 million and children below 15 years constitute 5.8 lakhs. Among adults, 1.1 million are women. In India, an estimated 6 million people are presently infected with HIV. This is the highest number of HIV infected persons in any single country. [1] It is estimated that 3.5% of the antenatal clinic attendees in Maharashtra are infected with HIV. This can be an understimation of figures, as many pregnant women in India do not attend antenatal clinics.

The degree of HIV disease progression in different cohorts has been observed to be varying in nature. The "typical progressors" develop the full-blown AIDS in ten years, while the "rapid progressors" develop AIDS within three to five years. The "non-progressors" are apparently healthy for two or more decades.

The factors responsible for the wide variation observed in the rate of disease progression in HIV-infected patients are multivariate. Factors like nutritional status, personal hygiene and environmental cleanliness play an important role in the progression of the disease. The important viral factors are the strain and serotype of the virus, its virulence, drug sensitivity, as well as the viral load in patients. These not only determine the adaptability of the virus in the host system but also the progression period. The host factors involved in development of AIDS include the cytokine responses, CD4 T cell counts, as well as the opportunistic infections the host suffers from. The immune system defects that the host develops and autoantibodies, which are caused by the viral infection, play an important role in the progression of the disease.

During viral infection, a number of specific immune effector mechanisms, together with non-specific defense mechanisms, are called into play to eliminate an infecting virus. Simultaneously, the virus acts to subvert one or more of these mechanisms in order to prolong its own survival.

Both humoral and cellular responses constitute a very important feature of immunity against HIV. The immunoglobulin M class of antibodies is usually against the HIV gag, core or envelope proteins. The IgM is usually transient and is followed by a long-lived IgG isotype response. Studies in acute seroconversion indicate that the IgG antibodies to the gag (p24) and envelope (gp160, gp 120, gp41) proteins appear first. These are followed in days or weeks by antibodies to HIV viral enzymes. Antibodies to regulatory proteins, although not primary, also appear early in the seroconversion period. The time taken for all the antibody bands to become positive on a Western blot varies from a few weeks to several months after acute infection. Although the development of a full IgG immunologic response to all HIV proteins may take several weeks, the ELISA is usually positive within the first three months. In rare cases, the patients may not manifest seroconversion or may have prolonged period of seroconversion. It is
unclear why this kind of humoral immunologic escape is possible.

Cellular immune responses during primary HIV infection may include significant CD4+ lymphopenia and concomitant CD8 lymphocytosis. Increased levels of activated CD8 activated lymphocytes expressing CD45RA, CD88 or CD27 are seen. In addition, cytokines such as IFN-γ, TNF-α, Neopterin and β2-microglobulin are elevated. None of these immune responses occur uniformly or predictably enough to use a reliable diagnostic marker for HIV infection.

During HIV infection, the disease progression is seen to be dependent not only on the viral life cycle, but also the immune defects in the host. The CD4+ T lymphocytes present with both qualitative and quantitative defects. There is an impaired recognition of the soluble antigen. The production of lymphokines and expression of IL-12 receptors is decreased. All these lead to depletion in T helper cell function. At the same time, a T cell depletion that is secondary to direct cytotoxicity and also due to syncytia formation has been observed. The destruction of non-infected T cells near an infected cell, called the "innocent bystanders" is said to occur due to the soluble gp 120. Viral infection of the progenitor or stem cells is also observed.

A similar qualitative and quantitative depletion of B lymphocytes is also seen in HIV. HIV presents with an impaired antibody response to antigen, bacteria and vaccination (recall antigen), while there is a quantitative increase in IgG and IgA secretion. There is a concomitant increase in the number of circulating B cells as well as immune complexes, leading to a polyclonal activation.

The cells of the monocyte/macrophage lineage show an increased IL-2 receptor expression and a decreased antigen presentation and activation. There is an increased IL-1 secretion as well as chemotactic ligand-receptor expression. IL-2 production is however impaired in the natural killer cells, leading to a functional depletion.

Certain autoimmune responses also been observed in HIV infection, which presents with a chronic inflammatory state. Anti-CD4 production might promote the CD4 cell depletion and thus lead to immune dysfunction. The viral expression might be augmented by the production of an Anti-CD2. The antibody to the gp41 might mimic the antibody to the Major Histocompatibility (MHC) class II antigen, and bind to the Human Leukocyte Antigen (HLA) class II molecules. This would lead to a general cellular toxicity. The T cell cytotoxicity may be effected by the anti-histones. Antibodies to IFN-γ may modulate the host response to viral infection. Factors like the Rheumatoid factor and the anti-nuclear antibodies might mimic the cell defence mechanisms at a molecular level by the formation of immune complexes and generation of anti-idiotype.

Development of non-related antibodies to antigens of the collagen, cardiolipin, erythrocytes, cytoskeleton and various organelles are also observed. Their
immunomodulatory effects, are however, not well known.

The development of an effective immune response involves lymphoid cells, hematopoietic cells and inflammatory cells. Cytokines are a group of low molecular weight proteins, which act as messengers of the immune system. They regulate the complex network and interaction of the above mentioned cells. Cytokines are produced by widely distributed cells like lymphocytes, macrophages, platelets, and fibroblasts and act either locally near the producer cells (paracrine effect) or directly on the producer cells (autocrine effect). Some cytokines are known by their common name, e.g. Tumor necrosis factor, interferons, etc.

Better understanding of viral and immunological factors that relate to rapid or delayed progression of HIV infection has emerged as an urgent priority. The immune response to pathogens can be differentially regulated by two functionally distinct T lymphocyte compartments called Th1, which mainly induce Cell Mediated Immunity through the production of IFN-\(\gamma\), IL-2, and IL-12, and Th2, which primarily activate B lymphocytes and the generation of antibodies through the production of IL-4, IL-5, IL-6 and IL-10. Some of these cytokines have cross regulatory prepares i.e. the production of Type 1 and Type 2 cytokines.

Human T cells exhibit a less restricted cytokine profile than murine T cells. IL-2, IL-6, IL-10, and IL-13 tend to segregate less clearly among human CD4+ subsets than in mouse. Human IL-2 has a proliferative effect on both Th1 and Th2 cells, while IL-4 exerts its effect only on Th2 cells. Similarly, IL-10 has an inhibitory effect on both Th1 and Th2 cells, while IFN-\(\gamma\) has a selective inhibitory effect on the proliferation response of Th2 cells. Th2 clones that usually have no cytolytic potential induce IgM, IgG, IgA, IgE synthesis by autologous B cells in the presence of the specific antigen.

In contrast, Th1 clones of which majority are cytolytic, provided B cell help, for IgM, IgG, IgA (but not IgE) synthesis, at a low T:B cell ratio. At T:B cell ratios higher than 1:1, there was decline in B cell help, that appeared to be related to their lytic activity against autologous antigen presenting B cell targets. This may represent an important mechanism for the downregulation of antibody response in-vivo.

Human Th1 and Th2 cells exhibit different ability to activate monocytic cells. Th1 (not Th2) help tissue factor production and pro-coagulation activity by monocytes. Cell to cell contacts with activated T cells and Th1 cytokines, in particular IFN-\(\gamma\) are required for optimal tissue-factor synthesis, whereas Th2 derived IL-4, IL-10 and IL-13 are inhibitory.[2]

Despite the immune response towards it, the human immunodeficiency virus not only persists but also thrives inside the host system. HIV, a highly complex virus establishes itself in the host immune network and brings about a dysfunction in the immunoregulatory mechanism of the host. The loss of recovery of T helper cells' function can occur in HIV positive individuals and AIDS patients independently of

Cytokine induced immune dysregulation is a central component in progression to AIDS. This shift in type of immune response and consequent progression to AIDS may be on account of the fact that HIV infects Type-1 cells easily compared to Type-2 cells. The shift in immune response is associated with shift in cytokine profile also (Th1 to Th2) and progression to AIDS. This change in cytokine profile results in the inability of host macrophages to get rid of opportunistic pathogens resulting in opportunistic infections and neoplasm, which ultimately prove fatal.

In progression to AIDS, the prototypes of T helpers cells, viz. Th1 and Th2 play an important part. The Th1 type of CD4+ cells do not allow the virus to replicate in them and even showed the ability to inhibit HIV replication in other HIV infected cells. Thus they are protective in nature. The Th2 type of cytokine production allows higher virus replication. This in turn allows preferential killing of Th2 type CD4+ cells and further spreads the infection of HIV [12].

The existence of functionally polarized responses by CD4+T helper cell and CD8+T cytotoxic cell subsets depending on the cytokines they produce has been proved. [13] Th1 cells produce Interferon -gamma (IFN-\(\gamma\)), Interleukin-2 (IL-2), Tumor necrosis factor-beta (TNF-\(\beta\)) responsible for both humoral and cell mediated immune responses (antibody production, macrophage activation, antibody dependent cell cytotoxicity and delayed type hypersensitivity).

Th2 cells produce IL-4, IL-5, IL-6, IL-10, and IL-13 and provide optimal help for humoral immune responses. These include mucosal immunity through production of mast cells and eosinophil growth and differentiation, and facilitation of IgA synthesis. CD+ T cells with less differentiated cytokine profiles than Th1 or Th2 cells are designated as THO. They mediate intermediate effects depending on the ratio of lymphokines produced and nature of responding cells. Lymphokines produced by TH1 and TH2 cells exert mutual regulatory interactions. In particular, IL-4 and IFN-\(\gamma\), the principal products of the TH2 and TH1 cells respectively, oppose one another's action.

The fundamental abnormality in HIV infected individuals is decrease in CD4+T lymphocytes. The qualitative and quantitative abnormality in CD4+T helper cell population in an HIV infected individual has profound effects on immune system. A shift away from the dominant production of immune protective TH1 cytokines to a dominant TH2 cytokine profile is observed in the progression to AIDS. As HIV infection progresses along with loss of production of IL-2, there is concomitant increase in production of IL-4 in response to mitogen.
This suggests the possibility of shift towards TH2 cytokine profile as a part of progression of the diseases. TH1 cytokines and strong cellular immune responses elicited by them are important in protection against HIV infection and progression to AIDS. [11]

The loss of T helper cell function in asymptomatic individuals is seen even before the fall in the number of CD4+ cells. Progression of HIV infection from asymptomatic to symptomatic stage and then AIDS involves shift of Th1 type of responses (cell mediated immune responses) to Th2 responses (humoral immune responses). The cytokines that modulate this shift are the Th1 cytokines like IFN-\(\gamma\), IL-2, IL-12, IL-15, TNF-\(\alpha\), and Th2 cytokines like IL-4, IL-10, IL-12, IL-5, IL-3 and IL-6.

IL-2 aids in the growth of natural killer (NK) cells/Lymphokine activated killer (LAK) cells. It augments the induction and production of IFN-\(\gamma\) and also its tumoricidal activity. It induces the growth of T cells. IFN-\(\gamma\) induces the proliferation and differentiation of B cells. It induces the cytolytic activity in LAK cells and cytotoxic T lymphocyte (CTL) cells. It brings about the secretion of IgG, while the proliferation of Th2 type cells is inhibited. TNF-\(\alpha\) enhances the development of protective response to viral intracellular pathogens. It acts synergistically with IL-12 to induce IFN-\(\gamma\) in NK cells. It increases the HIV-1 expression in infected cells. It induces cachexia on enhanced expression. Though secreted by macrophages rather than Th1 type cells, it is included in this category since it shows cell-mediated response and presents a Th1 profile.

IL-4 induces the differentiation of CD4+ T helper cells into Th2 cells. It enhances the production of IgG1, slgM and IgE by B cells. It possibly counterbalances the overexpression of TNF-\(\alpha\) and IL-6. IL-6 is an inducer for the differentiation of the B cells and cytotoxic T cells. It also induces production and action of acute phase proteins. Inhibition of IL-6 may cause immunosuppression. IL-10 enhances the differentiation of a Th2 type of cell into a mast cell. It plays an important part in the immunoregulation since it inhibits the production of IFN-\(\gamma\) and IL-2 by cross regulation. [14].

Evidence suggests the cross regulation by both Th1 and Th2 cells. The final outcome in HIV infections is the dominance of the detrimental counterpart of the chronic state of immune activation and severe deterioration of the immune system. The parasitic infections that induce type 2 profiles, e.g. malaria, filaria, salmonellosis, tuberculosis contribute to the higher incidence of seroconversion and more rapid progression to AIDS. [11]. Thus, there is a possibility that a low TH1 or a dominant TH2 cytokine profile will lead to more susceptibility to infection following low doses of HIV. Other infections, such as helminthic infestations, certain mycobacterial diseases or sexually transmitted diseases (syphilis) could shift the balance from TH1 to TH2 cytokines. Such infections therefore, could render the host more susceptible to HIV infections and could be considered as cofactors which may contribute to the higher incidence of seroconversion and more rapid progression to AIDS in those areas of the world in which parasitic infestations that
induce TH2 profiles are endemic.

The CD4+ levels theoretically mark the status of the disease in an HIV positive individual, though they are not foolproof markers for the disease progression. Along with the clinical symptoms, cytokine levels and viral load, they form a decent basis for prognosis of the disease. The CD4+ count is above 500 cells/ml in the early stage of the disease, while in the later stage, it is less than 200 cells/ml[15]. The fact that cytokines function as either activators or suppressors of CD4+T cell activity, and are themselves produced by activated cells, led to investigation of these factors in the regulation of HIV expression.

It was observed that the defective production of IL-12 and IFN-\(\gamma\) by HIV infected macrophages and dendritic cells probably favours the preferential development of Th0/Th2 responses even in response to pathogens that usually stimulate IL-2 and/or IFN-\(\alpha\) production by antigen presenting cells (APCs) and therefore promote Th1 development [16]. Also, HIV infection of Th0/Th2 cells results in high HIV replication and HIV-induced cell death, at least in part, due to CD30/CD30L interactions, which promote induction of NF-\(\kappa\)B and activation of the viral Long terminal repeats (LTRs). The CD30 expression is high and persistent in activated Th2 cells and is enhanced by HIV infection [17]. Th0 and Th2 cells express high levels of Fas and therefore are subjected to undergo apoptosis by FasL positive Th1 cells and CD8+ cells.

This favours Fas upregulation such that in absence of a protective IL-12 and in presence of small amounts of IL-4 an IL-10 which favour apoptosis, the Th1 cells are rendered susceptible to apoptosis induced by other FasL positive Th1 cells and/or CD8+ cells.

The cytokine interplay thus has an important role in the induction and destruction of CD4+ cells. Though the regulatory effects of the cytokines is limited to in-vitro studies, the increased levels of several cytokines observed in HIV infected individuals suggest their possible effect in-vivo. Therefore the detection of the host signal for the production, regulation, and destruction of cytokines would be definitive indicator of the cytokine status of the HIV positive individual.

REFERENCES:


COMPARISON OF SURGICALLY INDUCED POSTOPERATIVE ASTIGMATISM FOLLOWING PHACOEMULSIFICATION BY TEMPORAL CLEAR CORNEAL INCISION VERSUS SUPERIOR SCLERAL TUNNEL INCISION.

Dr. A. H. Madan*
Dr. Dilip Kumre**

SUMMARY

The aim of this prospective study was to compare the surgically induced astigmatism in cataract extraction performed by phacoemulsification using suture less superiorly placed scleral tunnel incision and by suture less temporal clear corneal incision. There were 50 patients in each group Keratometry was done pre-operatively, at 1 week, 3 weeks and 8 weeks post-operatively. Analysis of the induced astigmatism was done by simple substraction technique. We found the degree of induced postoperative astigmatism was significantly high at the end of 1st week in the superior scleral tunnel group as compared to the other group. There was no such significant difference between the two groups at the 3rd and 8th week. The postoperative astigmatism remained <1D throughout the study. In our view temporal clear corneal incision is a better site for doing phacoemulsification especially in patients with filtering bleb.

INTRODUCTION

Cataract surgery is an art, phacoemulsification a step towards perfection.

Phacoemulsification, a technique invented and developed by Dr. Charles Kelman during the sixties, has evolved from being just a small incision technique for cataract extraction to being a suture less way of ending the procedure, thereby causing minimal distortion of the corneal curvature. A choice of incision helps in reducing the pre-existing toricity in the cornea. So, all the parameter (size, site, shape and axis of incision) that go into the creation of a reproducible leak proof and astigmatic neutral incision have assumed great importance today.

*Associate Professor, Ophthalmology Dept., GMC, Nagpur.
**Lecturer, Ophthalmology Dept., GMC., Nagpur.
Scleral tunnel incision and clear corneal incisions are commonly used incisions. For many years superior scleral incision has been the favourable approach for most of the surgeons. The need for simpler and less time consuming incision shifted the site of incision from sclera to the cornea. However initial experience of most of the surgeon with superior corneal incision showed a significantly higher incidence of induced corneal astigmatism. This major drawback was found to be overcome if the incision was shifted from superior to a temporal approach.

With this background the following study was designed to compare the post-operatively induced corneal astigmatism following superior scleral and temporal clear corneal incision.

**MATERIAL AND METHODS**

We preformed a prospective study of 100 cases of senile cataract managed surgically by phacoemulsification with a posterior chamber implant. 50 patients underwent phacoemulsification by superior scleral tunnel incision (group 1) and 50 patients underwent same procedure via a temporal clear corneal incision (group 2). Those patients with any other ocular pathology, those with grade 4 or more nuclear cataract, patients with preoperative corneal astigmatism of >2D and patients with intra and post-operative complications were excluded from the study. All cases included in the study were operated by the same surgeon. Pre and post-operative keratometric readings as well as visual acuity were taken by the same observer. In all the cases anesthesia was achieved by peribulbar block using 5 ml of 50:50 mixture of 2% lignocaine with 1:100000 adrenaline with 1 ampoule of hyaluronidase (150U) and 0.5% bupivacaine. In group 1 after taking superior nectus suture a scleral incision was performed by a superior fornix based conjunctival flap. Homeostasis was achieved with wet field bipolar cautery of the episcleral blood vessels. Two side port entries with a 15 degree blade were made at 10 and 2 o’clock positions. The anterior chamber was filled with viscolastic material and a continuous curvilinear capsulorrhexis was done by a cystitome. A 3.2 mm partial thickness straight scleral incision was made about 2 mm behind the limbus at 12 o’clock position. A tunnel was made with a crescent blade until 1 mm entry into the clear cornea was achieved. The anterior chamber was entered with a 3.2 mm angled keratome. Phacoemulsification was done using Alcon’s Universal 2 phaco-machine. Acrylic foldable IOL was inserted into the capsular bag. No sutures were required to close the section.

In the 2nd group a 3.2 mm linear partial thickness incision was made at the limbus at 3 or 9 o’clock meridian. A short 1 mm tunnel was created into the clear cornea. As in group 1 anterior chamber was entered with a 3.2 mm angled keratome and the further operative steps were identical. Again sutures were not used to close the section. All patients were put post-operatively on Dexamethasone 0.1% + Neomycin 0.3% Q1D, Fluribiprofen 0.03% Q1D and Homatropine 0.5% BD.

Keratometry was performed pre-operatively and at 1st, 3rd and 8th week post-operatively using a Baush and Lomb keratometer. Analysis of astigmatism was done
RESULTS

The mean preoperative astigmatism in group 1 was 0.18D and in group 2 it was 0.49D (figure 1) (chart 1).

The mean postoperative astigmatism at 1 week was 0.65 D and 0.55 D respectively (figure 2) (chart 1),

which shows that there was a small but statistically significant difference between the two groups in the 1st postoperative week. The mean postoperative astigmatism at the end of 3 weeks was 0.565D & 0.455D in group 1 and group 2 respectively (figure 3) (chart 1),

The postoperative astigmatism remained <1D throughout the study. Thus apart from the early postoperative period the two groups show comparable results.

46 patients in group 1 and 48 patients in group 2 attained a best corrected visual acuity of >6/12 at the end of 8 weeks (figure 5) (chart 1).
DISCUSSION

In our study we found that the degree of induced post operative astigmatism in the two groups was marginally higher in the superior scleral tunnel incision group as compared to temporal clear corneal incision group at the end of 1st week. There was no such significant difference between the two groups at 3rd and 8th week. Our study correlates well with that of Robert Gross and Kevin Miller1 who found similar results in 198 eyes and concluded that there was a significantly greater induced astigmatism in the 1st postoperative day in the scleral tunnel group than clear corneal group and the effect disappeared by the 6th post operative week.

The earlier higher post operative induced astigmatism in the scleral tunnel incision can probably be explained either by the episcleral cautery, by the edema that developed in the scleral and cornea tissue surrounding the scleral tunnel by the compressive effect of the eyelids or by a combination of factors. The temporal clear corneal incison being farthest from the visual axis theoretically is claimed to be more refractively stable.

CONCLUSION:

The temporal clear corneal incision is more astigmatically neutral than superior scleral incision. It offers the advantage of avoiding scleral cauterization and availability of virgin conjunctiva for future filtering surgery if required. The clear corneal tunnel is easier to make and the intra operative manipulations are also more comfortable in this type of incision.

REFERENCES:


INTRODUCTION

Sickle Sickle Cell trait is an autosomal dominant disorder in which, less than half of hemoglobin in each red cell is Hbs. The abundance of normal hemoglobin i.e. HbA in the cell prevent sickling under most physiological circumstances.

Sickle cell trait does not produce any abnormalities of blood counts, red cell life span and exceedingly a rare cause of mortality. (Barbedo MMR. 1974). Many physicians believe that sickle cell trait is a mild type of sickle cell disease. (kellon DB et al, 1974).

There are numerous case reports about many clinical conditions in sickle cell trait. Sears DA in 1978 concluded that there is an association of the trait with certain clinical features.

Most of the complications associated with sickle cell anemia have been described in individuals with sickle cell trait under unusual physiological conditions-sudden death in soldier undergoing strenuous activities, transient hematuria and hyposthenuria, splenic infarct in persons traveling in unpressurised air crafts, patient under effect of anesthesia succumbed post-operatively.

This study is an attempt to focus on the complaints with which children with sickle cell trait are seeking medical attention. It also emphasizes on the clinical features at the time of presentation.

MATERIAL AND METHODS:

Cases of sickle cell trait who were admitted in Government Medical College and Hospital, Nagpur with various complaints were enrolled in the study from July, 2002- June 2003. All information was entered in pre-designed proforma, which included clinical history and examination findings.
A sickling test and electrophoresis on cellulose agar medium at alkaline PH were done to confirm the diagnosis.

**OBSERVATIONS**

In present study total children with sickle cell trait were 41 from July 2002 to June 2003. The age distribution of 41 cases of sickle cell trait is shown in the table below.

<table>
<thead>
<tr>
<th>Age</th>
<th>Male</th>
<th>Female</th>
<th>Total cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 5 years</td>
<td>10</td>
<td>4</td>
<td>14 (34%)</td>
</tr>
<tr>
<td>5-10 years</td>
<td>3</td>
<td>5</td>
<td>7 (17%)</td>
</tr>
<tr>
<td>&gt; 10 years</td>
<td>13</td>
<td>6</td>
<td>20 (49%)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>26</strong></td>
<td><strong>15</strong></td>
<td><strong>41</strong></td>
</tr>
</tbody>
</table>

Most of the cases were above 10 years of age (49%), whereas 34% were less than 5 years, of age and rest (17%) were between 5-10 years of age. Mean age of the children in study group was 7.9 years.

63% of the children were boys and 37% were girls. Age of presentation was after 10 years of life in 41.46% of children whereas, 34% presented below 5 years of age.

The duration of hospital stay was less than 7 days in most of the children (48.7%), 7-14 days in 36.5% and >14 days in 14.6%.

Maximum duration of hospital stay was 43 days in a child with osteomyelitis of (Lt) leg and minimum duration was 2 days in child admitted for Blood transfusion.

Only 2 children were admitted for more than one occasion during the study period.

**Seasonal Variation:**

<table>
<thead>
<tr>
<th>Month</th>
<th>No. of admn.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jan-March</td>
<td>7 (17%)</td>
</tr>
<tr>
<td>April-June</td>
<td>12 (29%)</td>
</tr>
<tr>
<td>July-September</td>
<td>7 (17%)</td>
</tr>
<tr>
<td>Oct. Dec.</td>
<td>15 (36%)</td>
</tr>
</tbody>
</table>

In the, present study, through admission occurred throughout the year, but more admissions were seen in winter (i.e., October to December) (36%), followed by admissions in the period from April to June.

**Distribution of painful crisis during study period :**

<table>
<thead>
<tr>
<th>Months</th>
<th>No. of cases with painful crisis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jan-March</td>
<td>1 (0.05%)</td>
</tr>
<tr>
<td>April-June</td>
<td>5 (29%)</td>
</tr>
<tr>
<td>July-September</td>
<td>4 (23%)</td>
</tr>
<tr>
<td>Oct.- Dec.</td>
<td>7 (41%)</td>
</tr>
</tbody>
</table>

Incidence of VOC is more in extremes of temperature in our present study group i.e. 41% in winters & 29% in summer.

**Caste Distribution:**

<table>
<thead>
<tr>
<th>Caste</th>
<th>No. Of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mahar</td>
<td>27 (65%)</td>
</tr>
<tr>
<td>Gond</td>
<td>3 (7.3%)</td>
</tr>
<tr>
<td>Teli</td>
<td>2 (4.8%)</td>
</tr>
<tr>
<td>Kunbi</td>
<td>1 (2.4%)</td>
</tr>
<tr>
<td>Rajput</td>
<td>2 (4.8%)</td>
</tr>
<tr>
<td>Pradhan</td>
<td>1 (2.4%)</td>
</tr>
<tr>
<td>Kalhar</td>
<td>1 (2.4%)</td>
</tr>
<tr>
<td>Sutar</td>
<td>1 (2.4%)</td>
</tr>
<tr>
<td>Wardhan</td>
<td>1 (2.4%)</td>
</tr>
<tr>
<td>Sikh</td>
<td>1 (2.4%)</td>
</tr>
<tr>
<td>Muslim</td>
<td>1</td>
</tr>
</tbody>
</table>
The percentage of cases were more in mahar caste in present study group i.e. 65%, followed by Gond (7.3%), Teli & Rajput contributed (4.8%) each. Rest of castes contributed 2.4% each.

**Clinical Findings on Examination:**

<table>
<thead>
<tr>
<th>Clinical Features</th>
<th>No. of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pyrexia</td>
<td>28 (68%)</td>
</tr>
<tr>
<td>Pallor</td>
<td>35 (85%)</td>
</tr>
<tr>
<td>Jaundice</td>
<td>4 (10%)</td>
</tr>
<tr>
<td>Epistaxis</td>
<td>1 (2%)</td>
</tr>
<tr>
<td>Hepatomegaly</td>
<td>16 (39%)</td>
</tr>
<tr>
<td>Splenomegaly</td>
<td>24 (59%)</td>
</tr>
<tr>
<td>Osteomyelitis</td>
<td>1 (2%)</td>
</tr>
</tbody>
</table>

As shown in above table, most of the children had anemia. Pyrexia was another common findings. Spleen was palpable in 59% of the children with sickle cell trait. Hepatomegaly was seen in 39% of children. 10% had Jaundice Epistaxis and Osteomyelitis contributed 2% each. One of the child was admitted with hydrocephalus, which was sequelae of pyogenic meningitis. No children with splenic infarct, cholelithiasis and splenic abscess, were seen in our present study group.

**Cause of Admission:**

<table>
<thead>
<tr>
<th>Cause</th>
<th>No. Of cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vaso-occlusic Crisis</td>
<td>17</td>
<td>41.46%</td>
</tr>
<tr>
<td>Anemia</td>
<td>13</td>
<td>32%</td>
</tr>
<tr>
<td>Acute Febrile Illness</td>
<td>9</td>
<td>21.9%</td>
</tr>
<tr>
<td>Infection</td>
<td>7</td>
<td>17%</td>
</tr>
<tr>
<td>Hydrocephalus</td>
<td>1</td>
<td>2%</td>
</tr>
<tr>
<td>Aplastic Crisis</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Sequestration Crisis</td>
<td>0</td>
<td>0%</td>
</tr>
</tbody>
</table>

In present study group, most common presentation was vaso-occlusive crisis (VOC) i.e 41.46%, followed by anemia (32.0%) 21.9% cases presented with Acute Febrile illness and 7% had infections (i.e., 3 children with Acute Respiratory infection, 1 with osteomyelitis, 1 with encephalitis and 2 children, had enteric fever).

Mortality was 2.4% in study group. The cause of death is one child was encephalitis? Viral with raised intracranial pressure.

**Hemoglobin levels at the time of admission:**

<table>
<thead>
<tr>
<th>Hemoglobin levels</th>
<th>No of cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 5 gm%</td>
<td>4</td>
<td>9.7%</td>
</tr>
<tr>
<td>5-9 gm%</td>
<td>31</td>
<td>75.6%</td>
</tr>
<tr>
<td>&gt; 9 gm%</td>
<td>6</td>
<td>14.6%</td>
</tr>
</tbody>
</table>

In study group 9.7% cases had hemoglobin levels less than 5 gm% showing severe anemia, while 75.6% children had hemoglobin levels between 5 to 9 gm% showing mild to moderate anemia.

63% of cases required blood transfusion. Mean Hemoglobin level in study group was 7.6 gm% (Range- 3.2 gm% -12.6 gm%)

- Patients with > 9 gm% of Hb constituted only 14.6% of the total.

**DISCUSSION**

Sickle cell trait is generally perceived as an innocuous condition, a large number of clinicopathological features have been attributed to it often without adequate scientific basis. But it is also true that they can suffer from
any disease like a normal person. It is often difficult to decide which of them is caused by the haemoglobinopathy. Certain conditions splenic or pulmonary infarction, hematuria, aseptic bone necrosis, epistaxis, jaundice, anemia and bone joint or muscle pain etc., Which are commonly seen in sickle cell disease, also occur in sickle cell trait. Except a few specific conditions there has rarely been any systematic clinicopathological control study to decide the roll of sickle cell trait in this condition.

In present study, only children with sickle cell trait were included, hence this study has predilection for symptomatic sickle cell children. Symptomatic group is not taken for consideration. This study trait has selection bias.

The age distribution chart showed, 49% of children above 10 years of age and \35% below 5 years of age. It can be concluded that most of the children present late. It can also be proposed that children were brought, for some other ailment and was incidentally found to have ‘AS’ pattern. Age of presentation (or diagnosis) was more than 10 years of age in 41.46% of total, whereas 34% children presented below 5 years of age.

In study group, boys outnumbered girls. Boys contributed 63% of the total, whereas girls contributed 29%. Boys to Girls ratio was 1.7: 1 The possible reason being the ‘Gender Bias’ in our society, where boys are given more attention. Konotey, Ahulu in,1974\(^5\) stated that males were more prone to develop symptoms as a result of more’ exposure to known precipitating factors as compared to females.

Duration of Hospital stay is important predictor of morbidity. In children of our study’ group, it was less than 7 days in 48.7% and more than 14 days in 14.6% maximum duration of study was 43 days and minimum was 2 days.

In present study, only children with sickle cell trait were included, hence this study has predilection for symptomatic sickle cell children. Symptomatic group is not taken for consideration. This study trait has selection bias.

The age distribution chart showed, 49% of children above 10 years of age and \35% below 5 years of age. It can be concluded that most of the children present late. It can also be proposed that children were brought, for some other ailment and was incidentally found to have ‘AS’ pattern. Age of presentation (or diagnosis) was more than 10 years of age in 41.46% of total, whereas 34% children presented below 5 years of age.

In present study group, winter season and extremely hot season were associated with more number of admissions with painful crisis\(^2\). 41 % admissions were in months of October to December, Followed by 29% in April to June. Many of them followed up for a few, 1 months had no recurrence. Investigation revealed no specific disorder. A big Question arises whether they are due to sickling disorder. Absence of recurrence after the diagnosis raises the possibility of a psycologcal basis because many people in this area are well acquainted with pain attacks of sickle cell disease but attack even prior to detection of positive stickling test can not expain the situation on psycologolical basis. Thus, the possibility of such features in sickle cell trait needs further explorations.

In present study, 63% of children were transfused blood and rest were treated with Folic acid only.

Clinical presentation with which children with sickle cell trait were admitted -VOC (41.46%), anemia (32%), Acute febrile episodes (21.9%) and infections (17%). Only one child had hydrocephalus (as a squeal of Phylogenetic meningitis). Hence it can be concluded that commonest cause of morbidity in study group is Vaso-occlusive Crisis.

Clinical findings which were encountered on history & examinations are
pyrexia (68%), Followed by splenomegaly (59%), hepatomegaly (39%), 85% children had anemia and 2% contributed by epistaxis & osteomyelitis contributed 2% each.

Other clinical conditions noted among sickle cell trait need no expaination as they occurred has in person with normal hemoglobin.

CONCLUSION AND RECOMMONDATION

In the present study of clinical profile of children with sickle cell trait, it was observed that the ailments of sickle cell trait are of milder degree than that of sickle cell disease.

The children usually present late and boys outnumber the girls.

Anemia was found to be commonest clinical finding in our study group and its cause is unknown. More specific investigations should be done to rule out iron deficiency anemia. Other findings like hepatomegaly; epistaxis needs further study to implicate haemoglobinopathy in their pathogenesis.

Most common cause of morbidity in our study group was vaso-occlusive crisis. Occurrence of vaso-occlusive crisis was found to be more in extremes of temperature i.e in winter seasons & in extremely hot months between April to June.

BIBLIOGRAPHY

2. Ibrahim AS: Relationship between metereological change and occurrence of painful sickle cell crisis in Kuwait; Trans R SOC Trop med Hyg; 1980:74:159-61;
5. Konotey, Ahulu FID: The sickle cell disease; Arch Intern med.;1974:133:611-619
ABSTRACT

Filaria, a huge public health problem of the tropics and subtropics is commonly seen in countries like India, China, Indonesia, Africa and the Far East. Microfilariae have been found in many organs as well as in uncommon situations e.g. vagina, nipple secretion, ascitic fluid and bone marrow.

Key words – Microfilariae, Bone marrow, Wuchereria Bancrofti.

INTRODUCTION

Lymphatic filariasis is a major health problem in India. Though control measures are effective, human population explosion plays a large role in the disease showing an upward trend. However microfilariae are not so commonly found in fine needle aspiration cytology smears and body fluids. Small numbers of cases have been reported in bone marrow, pleural and pericardial fluid.

CASE REPORT

A 40 years old male was admitted in J. J. Hospital with a history of weakness since 15 days. On examination he was emaciated, malnourished and had marked pallor and mild oedema. There was no hepatosplenomegaly or lymphadenopathy.

The patient was investigated for anemia and showed counts of - Hb - 4.4gms%, TLC-2000/cumm and platelets-44,000/cumm. Peripheral blood smear showed severe hypochromia and mild anisopoikilocytosis. Platelets were reduced on smear.

The differential leucocyte count was polymorphs - 68% and lymphocytes-32%. There was no eosinophilia. Microfilaria was not detected in PBS.

Bone marrow aspirate examination revealed a diluted marrow with reduced M : E ratio. There was erythroid hyperplasia with megaloblastic and micronormoblastic maturation. The myeloid series was reduced,
but normal in maturation. Megakaryocytes were reduced, but normal in morphology. Also seen in the aspirate were sheathed microfilariae of Wuchereria Bancrofti, which is the common species in India. The bone marrow biopsy revealed a normocellular marrow with erythroid hyperplasia. There was no microfilaria seen. Midnight peripheral blood smears taken subsequently did not reveal microfilaria.

**DISCUSSION**

Lymphatic filariasis is caused by either Wuchereria Bancrofti or Brugia Malayi. Adult worms lie in lymphatic vessels, while their offspring, the microfilariae circulate in peripheral blood. Pacheco and Orihel demonstrated that non circulating microfilariae in microcirculation represent a major proportion of the total. Microfilariae in bone marrow may have migrated from the microcirculation. Drinker et al pointed out that microfilariae can pass through unbroken blood capillaries, traverse tissue and re enter lymphatics. Present case report brings out clearly that microfilariae of Wuchereria Bancrofti can lie in the bone marrow. The infection may be tolerated well and can exit without symptoms of microfilaria. Relation of filarial infection and anemia is not clear and anemia may be an incidental finding.

**REFERENCES**


Case Report

FOREIGN BODIES IN BOTH MAIN BRONCHI- A UNIQUE EXPERIENCE

Dr. Samir V. Joshi M.S. (ENT) **

ABSTRACT

An unusual case of foreign bodies in both the main bronchi of a 2 year old girl child is documented for its rarity in world literature.

INTRODUCTION

Foreign bodies in tracheobronchial tree occur mainly in children particularly those under 2 years of age(1). Carelessness in one form or the other accounts for most foreign bodies(2). Frequently it is seen that foreign body accidents take place when children are playing and eating simultaneously, when inedible substance like watermelon seeds, nails, tacks, pins are put in mouth.

The entrance of a foreign body into tracheobronchial tree produces severe spasmodic cough lasting for approximately 30 minutes. During this period the foreign body travels from one part of the tracheobronchial tree to the other and finally lodges itself into its destination and a latent period begins. The family often assumes that foreign body has been coughed out and may forget the incidence; physician may also make the same assumption. However careful evaluation of the chest may reveal the presence of a foreign body.

Case: A 2 year old girl child was brought to the casualty department with a history of foreign body inhalation. Detailed history revealed that elder brother poured a ghutaka pocket (betel nut sachet) in the mouth of younger sister, mimicking the adults. There was change of voice and respiratory distress since the time of event approximately 11 am in morning and the child was brought to the hospital at 2 p.m. On examination the child was restless, there was no cyanosis, air entry was reduced on both sides of chest,

** Associate Professor, Department of E.N.T.
Shri Vasantrao Naik Government Medical College, Yavatmal-1
Address for Correspondence:-
Dr. Samir V. Joshi,
Flat GA-1 Block-E, Bharti Builders, Darwaha road, Yavatmal 445001
Phone-07232- 239369. E- mail- drsamirjoshi@yahoo.co.in
intercostal recession, epigastric and suprasternal indrawing was present. X-rays were taken which did not reveal any significant finding. Believing the positive history of foreign body inhalation aided with respiratory findings we took the decision of bronchoscopy and immediately the patient was taken on the operating table. Bronchoscope used was storz paediatric rigid bronchoscope size 4 with optical forceps and Hopkins rod lens telescope.

Patient developed hypoxic convulsions on table. Anesthetists were keen on intubation but quick negotiation of the airway by bronchoscope was preferred. A foreign body was seen at distance of 1 cm from left main bronchus and was removed. Ventilation improved on left side but air entry on right side was still less. Bronchoscope was negotiated in right main bronchus and to the surprise of everyone there was another foreign body in right main bronchus. Both of them were small pieces of betel nut reportedly of a Ghutka.

Post operative recovery was rapid with no residual chest findings and patient was discharged on 4th day.

**Discussion:** Although foreign bodies in tracheobronchial tree are common in children they are generally seen in either of the bronchi or the trachea, more frequently in right main bronchus because of its wider dimensions and straighter angle with the trachea. Foreign bodies are usually single. Multiple foreign bodies are noted in the past but they are usually related to bad road side accidents. To the best of our knowledge such foreign bodies in both main bronchi have not been reported in world literature. Time factor proves to be very crucial in the management of such foreign bodies.

And last but not the least, prevention is always better than cure and hence physicians particularly clinicians attending children should advice the parents to observe certain precautions.

1. Do not allow children under 6 years of age to eat nuts (unsupervised)
2. Keep small objects out of the reach of children and
3. Children should not run, scuffle or laugh while eating.
4. Toys with movable small parts should not be given to children below 3 years of age

**REFERENCES**

The city of Aurangabad, the headquarter of Marathwada region occupies a place of pride on the tourist map of world. The tourist will find in and around Aurangabad an inexhaustible treasure house of art, antiquities in temples, mausoleums and caves. The Ajanta monuments could be considered as high water mark of Buddhist classical tradition of art because of its absolute precision and uncommon paintings. They keep the spectator spellbound by their splendid expressions, delicacy of colour scheme and fine modeling of graceful figures depicted in natural poses. The magnificent group of rock — hewn temples at Ellora representing three different faiths — Buddhism, Hinduism and Jainism, marks the final stage of culmination of cave temple architecture in western India. The invulnerable fort at Deogiri, later on called Daulatabad, which had resisted many onslaughts, is the greatest contribution to this region by Yadav Dynasty.

The mausoleum built in memory of Dilras Bano Begum, wife of Aurangzeb is the only significant example of Moghul architecture in the Deccan. The general architectural features such as its central structures with main dome, its four diagonal minarets, its subsidiary building on four sides and the garden layout reveals that the builders of Makbara were inspired by the plan of Taj Mahal.

It is situated on the banks of river Godavari. The Paithan dam (Nath Sagar) built across river Godavari and elaborately well laid Dnyaneshwar Garden resembling the Brindavan garden are major tourist attractions at Paithan.

The other attractions include the Aurangabad caves, Soneri Mahal, the gates of Aurangabad, Water Mil (Panchakki) and the tomb of great Moghul Emperor Aurangzeb.

In present times, Aurangabad has become the nucleus of education and industrialization. In such a historical city, Govt. Medical College was established in June 1956 to fulfill a long felt need of Marathwada region. The college started functioning in Nizam Bungalow and was affiliated to Osmania.
University initially. In the year 1958 it got affiliated to Dr. Babasaheb Ambedkar Marathwada University. Since 1998 it is affiliated to Maharashtra University of Health Sciences, Nashik.

The present college building was constructed in 1960 on elevated area. So it is also called as Ghati Hospital. The new college building was inaugurated on 20th June 1964 by Dr. Sushila Nayar, the then Central Health Minister.

Main building is having administration division, library and various non clinical and para clinical departments. Department of Preventive and Social Medicine is also situated in the same building. There are separate buildings for out patient departments, surgical and medical wards, casualty, radiotherapy, C.T. Scan unit, mortuary, nursing college and hostels. The number of sanctioned hospital beds is 1020. In the year 2003 about 3,32,087 patients attended the outpatient department with daily average of 921 while 49492 were indoor admissions with daily new admissions of 130.

The college was started with intake capacity of 40 medical students, which was increased to 100, later it has become 150 in the year 1998-99. Except few, all the postgraduate degree and diploma courses are available in the institution.

The institution is having facilities of intensive respiratory care unit, voluntary counseling and testing center (VCTC), burn unit, lithotripsy unit, cobalt unit. Cardio Vascular Thorasic Surgery unit and Medical Intensive Care units are ready and about to start in near future.

Government Medical College, Aurangabad acts as a referral center for Marathwada region, and other adjacent districts such as Buldhana, Dhule and Ahmednagar.

The institution is armed with Urban and Rural Health Centers, run by department of Preventive and Social Medicine. This department has identified goiter pocket in Ajantha region first time in India.

Urban Health Center was established on 16th October 1967. The building is donated by Late Shri Durgaprasad Gupta and hence the center is named after him. A part of building is given by Municipal Corporation Aurangabad, on rental basis.

The center is located in Shahaganj area. It is three kilometers away from Medical College and Hospital. The area covered by the center is about 1.5 k.m. around the center. All preventive, and curative services are available at this center as outdoor facility.

Rural Health Training Center is situated at Paithan, 50 k.m. away from Aurangabad. The Rural Health Training Center is having 30 bedded hospital. This center is highly appreciated by Medical Council of India in its report.

This college will be celebrating its golden jubilee in the year 2005-2006. With support from government, elected members of Parliament and State Legislatures as well as past and present students, staff and well-wishers it is
expected to rise to a new height in the unending pursuance of excellence in patient care. The college may emerge as a super speciality center in this region to serve the people in a great way.

G.M.C. Aurangabad (College Building)

G.M.C. Aurangabad (Causalty)
GUIDELINES FOR AUTHORS

Scope

*Milestone* a quarterly journal entertains original communications on all aspects of biomedical research contributing to the advancement of knowledge in Medical sciences. The scope of the Journal allows publication of papers on medical education at undergraduate and postgraduate levels in either medical or paramedical courses; innovations in techniques; epidemiologic investigations and interdisciplinary work in human diseases. Readers are encouraged to write comments on papers published in the Journal in the form of letter to the Editor. Short communications containing significant findings will be given priority. Those who wish to contribute review articles may write to the Editor expressing their intention. It is issued quarterly in January, April, July and October every calendar year. All papers are subjected to peer review by Editorial Board and experts in the field before acceptance for publication. All papers are accepted subject to editorial changes.

Submission of Manuscripts

Manuscripts should be submitted with the undertaking that they are not under consideration elsewhere and have not been reported earlier partly/ totally. It is necessary that all the authors give an undertaking (in the format given at the end) indicating their consent to be co-authors in the sequence indicated on the title page. Typescripts in triplicate should be sent to the EDITOR, *Milestone*, DMER, St. Georges Compound, Near CST, Mumbai: 400001.

Preparation of Manuscripts

Authors are advised to consult a recent issue of the Journal to get familiar with the format adopted in respect to various elements of a paper. Manuscripts should be presented in as concise form as possible, typewritten in double space on one side of good quality bond paper (21.0 x 29.7 cm). Pages should be numbered consecutively and the contents arranged in the following order: Title, Name(s), of the author(s) with highest academic qualification of each author, abbreviated title, manuscripts are to be submitted. Department(s) and Institution(s), Abstract, Key words, Introduction, Material & Methods, Results, Discussion, Acknowledgment, References. Abstract, Tables and legends for figures should be typed on separate sheet and not in continuation of the main text. Three copies of the manuscript are to be submitted. Due to the high cost of postage, it may not be possible for the Editor to return the original manuscripts to the authors if not accepted for publication.

The name and mailing address of the author to whom requests of reprints or correspondence should be directed must be indicated. Submission of e-mail address is encouraged.

Title

Title of the article should be short and yet sufficiently informative so as to be useful in indexing and information retrieval.

Abstract

The abstracts should be brief (about 200 words) and structured (for special and original articles) to contain purpose/ background, material and methods, results and conclusions of the paper. It should only highlight the principal findings and conclusions so that it can be used by abstracting services without modification. Conclusions and recommendations not found in the text of the article should not be inserted in the Abstract.

Key Words

Upto five key words may be given, which will be helpful for indexing purposes.

Introduction

Introduction should be brief and state precisely the scope of the paper. Review of the literature should be restricted to reasons for undertaking the present study and provide only the most essential background.

Material & Methods

The nomenclature, the source of material and equipment used, with the manufacturer's details in parenthesis, should be clearly mentioned, the procedures adopted should be explicitly stated to enable other workers to reproduce the results, if necessary. New methods may be described in sufficient detail and indicating their limitations. Established methods can be just mentioned with authentic reference and significant deviations, if any, given with reasons for adopting them. When reporting experiments on human subjects, it should be indicated whether the procedures followed were incoordinate with the ethical standards on human experimentation (as per the guidelines laid down by the Central Ethical Committee of the Indian Council of Medical Research).

When reporting experiments on animals, procedures adopted for the care and use of laboratory animals need to be mentioned. The drugs and chemicals used should be precisely identified, including generic name(s), dosage(s) and route(s) of administration.

The statistical analysis done and statistical significance of the findings when appropriate should be mentioned. Unless absolutely necessary for a clear
understanding of the article, detailed description of statistical treatment may be avoided. Articles based heavily on statistical considerations, however, need to give details particularly when new or uncommon methods are employed, others need to give only authentic references.

Results

Only such data that are essential for understanding the discussion and main conclusions emerging from the study should be included. The data should be arranged in unified and coherent sequence so that the report develops clearly and logically. Data presented in tables and figures should not be repeated in the text. Only important observations need to be emphasized or summarised. The same data should not be presented both in tabular and graphic forms. Interpretation of the data should be taken up only under the Discussion and not under Results.

Discussion

Long, rambling and involved discussion should be scrupulously avoided. The discussion should deal with the interpretation of results without repeating what already was presented under Results. It should relate new finding to the known ones and include logical deductions.

The conclusions can be linked with the goals of the study but unqualified statements and conclusions not completely supported by the data should be avoided. Claiming of priority on work that is ongoing should also be avoided. A hypothesis should, if warranted, clearly be labelled as such; recommendations may be included as part of the Discussion, only when considered absolutely necessary and received.

Case report

For description of uncommon infections. It should be divided into introduction, case history and discussion with not more than 10 references as possible. Illustrations and tables when included should be limited to one each. It should have an abstract (unstructured) limited to 100 words and key words limited to 3 words.

Brief Communications

Recommended for brief observations that do not warrant a full length paper. It may be divided into sections as for the full paper. It must not exceed 1000 words. References must be as few as possible and not more than 13. Illustrations and tables when included should be limited to one each. It should have an abstract and (unstructured) limited to 100 words and key words limited to 3 words.

Correspondence

Addressed to the editor, correspondence can be related to previously published articles or for presentation of preliminary results. It should be limited in length to 250 words. And should be continuous without headings. It may include 2-3 paragraphs, not more than 5 references and no tables and figures.

Acknowledgment

Acknowledgments should be brief and made for specific scientific and technical assistance only and for providing routine departmental facilities and encouragement or for help in the preparation of the manuscripts (including typing or secretarial assistance).

References

References will not be checked in the Editorial office. Responsibility for their accuracy and completeness lies with the author. The total number of references should normally be restricted to a maximum of 20 for an original research article, 10 for a case report, 13 for a brief communication and 5 for correspondence. References to literature cited in the text should be numbered consecutively and placed at the end of the manuscripts. The references in the text should be indicated above the line (superscript). As far as possible mentioning names of authors (s) under reference should be avoided in text.

Journals

The titles of the journals should be abbreviated according to the style used by the Index Medicus. The January issue of the Index Medicus may be consulted. In citing reference to research papers, names and initials of all the authors should be given, followed by: the title of the article, journal (italics), year (in circular brackets), volume number (bold), and first page and last page of the reference.


If a paper has been accepted for publication, the name and initials of all the authors and the journal should be given followed by the words “in press” within circular brackets. Parikh M and Singh N. Rapid diagnosis of neonatal bacteraemia Indian J Med Microbiol (1995) (in Press).

Books

When the book has only authors and no editor(s). 1) S.C. Parija A text book of Medical Parasitology: 1 st ed. (All India Publishers and Distributors, Madras) 1996.:30-34.


Proceedings of Symposia/confferences

Published proceedings of conferences/ symposia should be treated as books.

Monographs/reports

The name of the book should appear first followed by the other details.

O. Satyavati GV, Raina MV and Sharma M. (Eds) Medicinal plants of India (Indian council of Medical Research, New Delhi) 1976, vol I: 332.

Thesis (doctorate)


Unpublished data/personal communications

Unpublished abstracts and abstracts reference. personal communications should be indicated in the text itself and not numbered as (I) (Swami KS, unpublished work); (II) (Swami KS, personal communication): (III) (National Institute of Nutrition unpublished data).

On Line Journals


Illustrations:

Three sets of Illustrations (one set original and 2 copies) should be submitted, numbered consecutively in Arabic numerals. Line drawings should be made on good quality tracing paper or Bristol board. Letters, numbers and symbols should be clear in the figures and of sufficient size, so that when reduced, they could be accommodated in single column (8.5 cm) or double (17.0 x 21.0 cm ) column sizes. All the illustrations must be protected by thick card board packing against damages during transit. Photomicrographs should have internal scale markers regarding details of magnification to facilitate reduction in size. Symbols, arrows and letter used in the photomicrographs should contrast with the background.

All published material should be acknowledged and copyright material should be submitted along with the written permission of copyright holder. Colour illustrations will be accepted only at the author's expense.

Electronic manuscript

Upon acceptance for publication or at the time of revision when a manuscript is likely to be accepted for publication, the corresponding author will be requested to send an electronic file on floppy, in addition to the original manuscript. Disks that are IBM PC compatible (non macintosh) will be accepted. Floppy disks should be 3.5 inch, double sided and high density. Text and tables files should be in MS word for windows. The disk should be labelled with the manuscript number title of the article, author’s name, the file name and software used including version. The disk must contain exactly the same material as the revised manuscript including the tables, Legends and graphs. Graphs and diagrams must be sent in graphic format, preferably MS EXCEL, MS Power Point and Adobe Photoshop. Do not send graphs and diagrams in freehand. The disk should be sent in proper packaging to avoid damage and corruption of the information during transit.

Tables

Tables should be typed separately and numbered consecutively with Arabic numerals (1,2,3, etc..). They should bear brief title and column headings should also be short. Units of measurement should be abbreviated and placed below the headings. Statistical measurement variations such as SO and SE should be identified. Also table should not be submitted as photographs. Any other queries may be clarified from the Editor.

Undertaking

We, the undersigned, give an undertaking to the following effect with regard to our article entitled “…………………..” submitted for publication in the milestone Journal of Medical Research Council of Maharashtra.

1. The article mentioned above has not been published or submitted to or accepted for publication in any form, in any other journal.
2. We also vouchsafe that the authorship of this article will not be contested by anyone whose name(s) is/are not listed by us here.
3. We also agree to the authorship of this article in the following sequence:-

Authors Names (in sequence) Signature of Authors with date

1. ………………………………………
2. ………………………………………
3. ………………………………………

Important

1. All the authors are required to sign independently in this form in the sequence. 2. Each author should have generated at least part of the intellectual content of the paper.
3. Each author should be able to defend publicly in the scientific community, that intellectual content of the paper for which he/she can take responsibility.
4. No addition/deletion or any change in the sequence of the authorship will be permissible at a later stage, without valid reasons/ permission of the Editor.
5. If the authorship is contested at any stage, the article will not be processed for publication till the issue is resolved.
SUBSCRIBE NOW

The Medical Research Council of Maharashtra

MILESTONE
Ahead with changing medical times

Yes! I want to Subscribe ☐ Renew ☐

<table>
<thead>
<tr>
<th>Term</th>
<th>Subscription Offer for individual</th>
<th>Subscription Offer for institution</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Year (4 issues)</td>
<td>Rs. 300/-</td>
<td>Rs. 2,000/-</td>
</tr>
<tr>
<td>Life membership</td>
<td>Rs.2,500/-</td>
<td>Rs.19,000/-</td>
</tr>
</tbody>
</table>

Name: Dr./Mr./Ms. .................................................................................................................................

Qualifications ............................................ Speciality ..............................................................

Designation ................................................ Institution ...............................................................

Address for Correspondence ....................................................................................................................

..........................................................................................................................................................

Phone........................................... Fax.......................... E-mail.......................................................

Payment Details:

Cheque/D.D.No............................................ Dated............................................ For Rs..................................

Drawn on (Bank) : ....................................................................................................................................

Note: Payment should be made in the name of -

“The Medical Research Council of Maharashtra, Mumbai”.
* Please add Rs.30/- for cheques from outside Mumbai.
* Please allow 2 weeks for delivery of 1st issue.

Director, Medical Education & Research,
4th Floor, Dental College Building, St.George’s hospital Compound, CST, Mumbai:400001.
Tel: 022-2262 0361-65/ 2265 2257. Fax: 022 2262 0562/ 2265 2168
GRAM: MEDICATNSEARCH, E-Mail: milestone@dmer.org. Website: www.dmer.org

To,

________________________________________
________________________________________
________________________________________

Dear Sir/Madam,

“Milestone”, a quarterly journal of DMER is published regularly. It reaches in all Medical/Dental/Ayurved College and Hospitals all over State of Maharashtra and India.

Your patronage will help dissemination of knowledge about your product and services in addition to Medical/Health education.

The journal is being published in A4 size on a high quality glossy art paper in multicolor and in Black and White.

The commercial advertisement from Pharmaceuticals, Educational Organisations Medical equipment & medical Machinories etc. to advertise their products/services are welcome. However, advertisement from liquors, tobacco is not accepted. The rates of the advertisement per issue are as follows:

a. Back cover (4 Colour) .. Rs.10,000/-
b. Inside front cover (4 Colour) .. Rs. 7,000/-
c. Inside back cover (4 Colour) .. Rs. 5,000/-
d. Full page (4 Colour) .. Rs. 5,000/-
e. Full page (B/W) .. Rs. 3,000/-

The cheques or D.D. payable at Mumbai, should be in the name of “The Medical Research Council of Maharashtra”.

Thanking you.

Yours Sincerely,

( Dr.W.B.Tayade)
Chief Editor and Director,
Medical Education and Research, Mumbai.