Karyotypic Study Of Chronic And Blastic Phase Chronic Myeloid Leukemia Patients of Gujarat State.

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Abstract:
For present work, 27 clinically diagnosed Chronic Myeloid Leukemia patients were selected, who attended the Out Patient Department of Gujarat Cancer and Research Institute, Ahmedabad. In all these cases relevant history, clinical findings, haematological data and other investigations were noted & bone-marrow samples were obtained for further study, which was done at Genetics Laboratory, B.J. Medical College, Ahmedabad. Samples were cultured, harvested, slides were prepared & photographs were obtained using photomicroscope and Karyotypes were prepared by using conventional cut and paste technique. Cytogenetic evaluation was done to detect the presence of Philadelphia chromosome and/or other chromosomal abnormalities. Out of 27 patients studied, 22 cases were having mild to moderate & remaining 5 cases were having huge splenomegaly. The blood picture showed, 9 were anaemic; 11 having total leukocytic count more than 1 lakh/mm$^3$; 8 cases were thrombocytopenic. 25 cases were in chronic and 2 cases were in blastic phase. Cytogenetic evaluation by Karyotypes revealed 13 Ph’ positive cases; 4 Ph’ negative; 3 mosaic & remaining 7 cases came out inconclusive. All relevant parameters including clinical, hematological and cytogenetic were evaluated, analyzed and compared with other similar studies.

Key words: CML, Karyotypic study, chronic and blastic phase CML, Ph’ chromosome

INTRODUCTION:
Chronic myeloid leukemia (CML) is a clonal myeloproliferative disorder of a pluripotent haemopoietic stem cell involving granulopoiesis, erythropoiesis and megakaryopoiesis. It is primarily a disease of adults, runs a biphasic or triphasic course with an initial chronic phase that usually lasts for 3-4 years and ends in a blastic phase; sometimes preceded by a short accelerated phase when the patient becomes refractory to treatment.

In the initial chronic phase, marked by a greatly increased pool of committed myeloid progenitor cells, terminal differentiation of cells is maintained; resulting in profoundly elevated counts of circulating mature granulocytes. The chronic phase can be managed by a variety of well-tolerated regimens. Patients respond well to therapy and generally have a nearly normal quality of life.

The cytogenetic hallmark of CML found in at least 90-95 % of patients is the presence of an abnormal chromosome, the Philadelphia Chromosome (Ph’); so named after its place of discovery. It was the first karyotypic marker consistently present in a human neoplasm, described by Nowell and Hungerford. The Ph’ chromosome which represents a shortened chromosome 22, results from a translocation between chromosome 9 and 22, i.e. t(9;22) (q34;q11). Molecular cytogenetic studies have shown that in this translocation, the c-abl proto-
oncogene normally present on chromosome 9 (9q34) is translocated to the breakpoint cluster region (bcr) on chromosome 22 (22q11) resulting in the formation of bcr/abl chimeric gene, which transcribes for hybrid 8.5 Kb chimeric mRNA which in turn produces an abnormal fusion protein (P210) with elevated tyrosine kinase activity.

Unfortunately, after several weeks to many years, a state of myeloproliferative acceleration develops in which the myeloid cells progressively lose their capacity for terminal differentiation as well as thrombocytosis & basophilia occurs. Additional clonal cytogenetic abnormalities often appear. These changes in about 75% of the cases, herald the terminal, blast-crisis stage, during which immature blast cells rapidly proliferate, the prognosis becomes poor and the patient inevitably dies, within a period of 6 months. Progression of CML to blast crisis is preceded or accompanied by additional chromosomal abnormalities like +8, +Ph', i(17q), loss of Y chromosome and less frequently +19.

**AIMS:**
- To assess and correlate the cytogenetic findings, in patients diagnosed morphologically & clinically as CML.
- On the basis of cytogenetic investigations, treatment modality as well as selection of the drug can be made.
- Subsequent reports can help to find out progress/regression of the disease.
- Clonal evolution of the metaphase, which can be a side effect of the drug used in the treatment of CML, particularly, imatinib mesylate and alpha-interferon, can be judged.
- If previously positive Ph' case becomes Ph' (-negative) along with improvement of clinical signs and symptoms, favourable prognosis can be predicted.
- Data can be useful in selecting a stable patient for bone-marrow transplantation, a curative treatment of CML.
- With a base-line investigation and follow-up cytogenetic findings, new drug trials can be possible. During which; Efficacy, dosage and adverse reactions in the form of evolution of additional cytogenetic abnormalities can also be detected.

**MATERIAL AND METHOD:**

This study includes observations of 27 cases, diagnosed clinically as CML including chronic and blastic phase, who attended outpatient department of Gujarat Cancer and Research Institute, Civil hospital campus, Ahmedabad. Patient’s personal data, relevant clinical history, vital statistics and in brief clinical assessments were noted. Findings of peripheral blood smear, bone marrow smear and other relevant investigations were also noted.

Bone marrow samples were obtained from GCRI, Ahmedabad and further processing of karyotyping was done at Genetics Laboratory, Anatomy department, B. J. Medical College, Ahmedabad. The process of karyotyping involves culture setting on the same day followed by harvesting and preparation of slides on the next day. The slides were scanned for good quality metaphases; readings were taken from each slide and noted. After 7 days, G-Banding procedure was carried out using freshly prepared
Trypsin solution and Giemsa stain. The procedure protocols were followed with the guidelines of Rooney DE, Czepulkowski Bh (1992) Human Cytogenetics- A practical approach, 2nd Edition. (5)

About 25 metaphase plates were observed in each case and finally, photographs were obtained using a photomicroscope. The chromosomal findings were described according to the International System of Human Cytogenetic Nomenclature and finally, Karyotypes were prepared using conventional cut and paste technique.

**OBSERVATIONS:**

<table>
<thead>
<tr>
<th>Table: I : Spleen size of CML patients studied</th>
</tr>
</thead>
<tbody>
<tr>
<td>Splen</td>
</tr>
<tr>
<td>Number of patients</td>
</tr>
</tbody>
</table>

**Table: II: Findings of blood investigations in the study group**

<table>
<thead>
<tr>
<th>PARAMETERS</th>
<th>VALUES</th>
<th>No of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb ( Haemoglobin )</td>
<td>&lt; 9.0 gm/dl</td>
<td>09</td>
</tr>
<tr>
<td></td>
<td>&gt; 9.0 gm/dl</td>
<td>18</td>
</tr>
<tr>
<td>Total Leukocyte count</td>
<td>&lt; 1.0 lakhs/mm$^3$</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>&gt; or = 1.0 lakhs/mm$^3$</td>
<td>11</td>
</tr>
<tr>
<td>Total Platelet Count</td>
<td>&lt; 2.0 lakhs/mm$^3$</td>
<td>08</td>
</tr>
<tr>
<td></td>
<td>2.0 – 5.0 lakhs/mm$^3$</td>
<td>14</td>
</tr>
<tr>
<td></td>
<td>&gt; 5.0 lakhs/mm$^3$</td>
<td>05</td>
</tr>
<tr>
<td>Blast cells</td>
<td>&lt; or = 5 %</td>
<td>22</td>
</tr>
<tr>
<td></td>
<td>Between 5 – 30 %</td>
<td>03</td>
</tr>
<tr>
<td></td>
<td>&gt; 30 %</td>
<td>02</td>
</tr>
</tbody>
</table>

**DISCUSSION:**

As far as the hematological studies are concerned, according to John Athens total leukocyte count in chronic phase usually remains > 1 lakhs/mm$^3$. (1)

According to the Kamada N. and co-workers, sequence of appearance of abnormalities in CML are as follows: Ph' chromosome $\rightarrow$ Basophilia and thrombocytosis $\rightarrow$ immature granulocytes (more than 5 %) $\rightarrow$ increase in serum Vitamin B$_{12}$ $\rightarrow$ splenomegaly $\rightarrow$ subjective symptoms (weight loss, anorexia, low grade fever, cough etc.). Increase in total leukocyte count also occurs along with the above mentioned changes.

According to E.Z.EZDINILI and co-workers (2) who studied 46 patients of CML (34 Ph' positive and 12 Ph' negative) have shown significantly higher number of leukocyte count and platelet counts among Ph' positive patients. Thrombocytopenia was fairly common in the Ph' negative group. 3 out of 24 Ph' positive cases were having impalpable spleen, 16 were having palpable spleen extending less than 10 cm below the costal margin and 15 had huge splenomegaly, extending more than 10 cm below the costal margin.
As shown in Tables I & II of observations: in present study, 10 out of 25 chronic phase CML cases were having total leukocyte count > 1 lakhs/mm3 and 6 out of 14 Ph' positive cases were having total leukocyte count > 1 lakhs/mm3. Out of 2 cases of blastic phase 1 case had total leukocyte count > 1 lakhs/mm3 and 2 out of 4 Ph' negative cases were having total platelet count < 2 lakhs/mm3 showing thrombocytopenia. 6 out of 13 Ph' positive cases were having just palpable spleen(+), 5 cases of Ph' positive were having moderately palpable spleen (+++) and 1 case of Ph' positive was having hugely palpable spleen (+++). Spleen size correlates reasonably well with the magnitude of the total leukocyte count.

Tura and Kantarajan(5) have given negative prognostic factors which are: age (> 60 years; spleen (10 cm below the costal margin, blasts (> 5% in marrow, platelets (7 lakhs/mm3 or any of the characteristics of the accelerated disease are associated with poor prognosis and a threefold higher hazard rate, or risk of death per unit of time, in the first year. From cytogenetic standpoint, the most extensively documented evidences for sequential genetic changes during tumor evolution come from studies of chronic myelogenous leukemia. In nearly every typical case, the leukemic cells in the early indolent phase of this disorder have only a single cytogenetic abnormality, the t(9;22) translocation that produces the Ph’ chromosome.

Presumably, the altered gene product plays an important role in the early expansion of the neoplastic clone within the myeloid lineage. When CML progresses to its accelerated terminal phase, the neoplastic population is overgrown by one or more sub clones having additional karyotypic changes.(4) The Philadelphia chromosome is the cytogenetic hallmark of chronic myeloid leukemia and is observed in more than 90% of CML cases. At diagnosis, in 5 to 10% of CML patients the Ph’ chromosome is derived from variant translocations other than the standard t(9 : 22).

Table: IV

<table>
<thead>
<tr>
<th>STUDY</th>
<th>No of patients studied</th>
<th>Ph’ positive</th>
<th>Ph’ negative</th>
<th>Patients having Ph’ chromosome (%)</th>
<th>Other cytogenetic findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>EZDINILI E.Z.et al (1959-1967)</td>
<td>61</td>
<td>43</td>
<td>18</td>
<td>70.49</td>
<td>+C,+8,-17, +7/F(in 5 cases)</td>
</tr>
<tr>
<td>Rowley J. D. (1973)</td>
<td>9</td>
<td>9</td>
<td>-</td>
<td>100</td>
<td>+C,+8,-17, +7/F(in 5 cases)</td>
</tr>
<tr>
<td>Walid Al Achkar (2001)</td>
<td>43</td>
<td>22</td>
<td>18</td>
<td>51.16</td>
<td>+19, t(4:22); +9</td>
</tr>
<tr>
<td>Rachal Thomas Jacob et al (2002)</td>
<td>525</td>
<td>453</td>
<td>72</td>
<td>86.28</td>
<td>Double Ph’ chromosome, +8,+19</td>
</tr>
<tr>
<td>Present Study*</td>
<td>27</td>
<td>13**</td>
<td>4</td>
<td>48.14</td>
<td>Mosaicism observed in 3 cases</td>
</tr>
</tbody>
</table>
Similarly other associated chromosomal abnormalities are also present in these remaining 5 to 10 % of the cases. Some of the concerned studies are discussed herewith. Table IV ( *Table : III in observation. ** Image : I )

All authors agree that patients with CML who have the Ph’ chromosome have a better prognosis than those who don’t. Mosaicism has been considered indicative of a good prognosis in most, but not all, series.

Cytogenetic investigations are mandatory in cases diagnosed morphologically as CML especially, to know the Ph’ status. Once the Ph’ status is known these studies will help in selection of cases for more effective treatment protocols. Use of newer drug regimens in patients who are Ph’ positive in the early stages of the disease can help to increase median survival period.(4)

However, in those cases that are Ph negative by conventional cytogenetics, Ph' negativity doesn't exclude bcr-abl translocation at molecular level, which requires to be confirmed by more specific molecular genetic studies like Fluorescence In Situ Hybridization(FISH).

In CML, cytogenetic investigations whether they are in the form of conventional studies as in case of the present study or upgraded in the form of FISH (Fluorescence In Situ Hybridization) or CGH (Comparative Genomic Hybridization), remain keystone at every stage of the disease; starting from initial diagnosis to final treatment and for predicting prognosis of the patients.

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Cardiovascular Responses With Valsalva Maneuver During Activities Of Daily Livings In Healthy Adults

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Abstract:
Purpose of Study: To measure CV responses in SLF, and SQ with & without VM, Study design: Cross sectional Observational study, Materials: assessment form, 36 SF Health Questionnaire, Modified Sphygmomanometer, BP apparatus, Nike-HRM-TRIAX, Methodology: 335 (M=146) subjects participated to perform SLF and SQ position with & without VM and SBP, DBP and HR were recorded. Data Analysis: SPSS -10.1, LOS was set at 0.05 or CI 95 %. Result: Study has shown significant increases in SBP in SQ and increase HR in SLF position with and without VM. Discussion: the impact of Heart Rate Variability and baroreflex sensitivity in CV system plays vital role in maintaining hemodynamic status while performing valsalva like activities. Conclusion: SQ has significant impact on SBP and DBP as compared to SLF position with and without VM as well as SLF position has a significant impact on HR as compared to SQ with VM, however these need to be taken into consideration while planning life style modification for high risk population

Key words: Valsalva Maneuver (VM), Sitting Lean Forward (SLF) position, Squatting (SQ) Position, Cardiovascular (CV) responses

INTRODUCTION:
Activities of daily livings (ADL) involve several stressful physical events such as lifting weight, pushing objective ground level activities, defecation, urination, etc... These stressful events are not recognized among healthy people, but it may be life threatening for peoples with compromised CV system.8, 10

It has been observed that more than 1/3 population, who dies following the cerebrovascular accident, dies in and around the toilet, in the morning. 7, 12, 15 Urination and defecation has integrated sequences of breath holding strategies which involves isometric muscular contraction to increases interathoracic and abdominal pressure.

It may be more stressful in case of constipation, obesity, pregnancy etc... Generating high intraabdominal pressure and frequent releasing of “breath hold” has great impact on hemodynamic status. Moreover, the positions of performing defection such as SLF and SQ have significant impact on CV system. Clinically, breath holding with forced expiratory effort against closed glottis is known as a Valsalva Maneuver (VM). It is also used as a test of functional integrity of the autonomic nervous system.1, 2 Hemodynamic changes describe into 4 phases when VM performed at 40-mmHg pressures for 20 seconds with open glottis.

CV responses in stress-provoked defection positions with VM among healthy adults will provide the associate evidence to understand the impact on compromised CV disorders.

CV responses on ADLs have been of common interest for many researchers. ADLs with maneuver like
valsalva may throw further light on integrity of autonomic and CV system, which may be useful or detrimental to normal and patient population. 2, 5, 10

The various researchers have shown interest in positional impact of defecation on CV status. It was observed that when normal subjects squat, the arterial mean pressure and pulse pressure increases. This increase is greater after few seconds due to which mean pressure falls. This fall in pressure eventually stabilizes at higher level than that observed in sitting position. 5, 1 The studies has concluded that CV stability during straining with VM is higher in the lean forward as compared to lean backward position. 9

The VM is a useful technique to assess the CV changes. 6 Some studies concluded that CV changes could be obtained as a result of a 15-sec VM, performed at 40-mmHg airway pressures. 7, 8 Some of the researchers have suggested 20 seconds of VM at 40-mmHg pressure. 12

**Figure I, Four phase valsalva response**

The hemodynamic changes associated with the VM were described by invasive techniques. 7, 6, 10. These changes are classically divided into four phases. (Fig-I) **Phase-I** is beginning of the strain to a transient rise in mean arterial BP (MABP), as the increase in intrathoracic pressure to constrain the arterial tree. **Phase-II** has further divided into Phase IIa and IIb. During phase IIa, the atrial filling pressure falls so MABP decreases. In phase IIb there is increased sympathetic activation, causing a rise in peripheral vascular resistance and HR, which leads to a small increase in MABP. **Phase-III** is associated with release of the strain and a sudden fall in MABP due to the influence of release of the intrathoracic pressure on the arterial tree. Finally, **phase-IV** sees an immediate “overshoot” in MABP because of the persistence of increased sympathetic tone and systemic vascular resistance. A reflex bradycardia then results due to stimulation of arterial baroreceptors, and both MABP and HR return to baseline values. 11, 13 It has been concluded with VM at severe degree, results in decrease of HR by 9.2 beats/min. 9

This study was designed to evaluate CV responses in two stress provoking defecation positions of ADLs, SLF and SQ positions. However, comparing the CV responses with and without VM in these stress-provoked positions can provide prospective guidelines for high-risk population with CV disorders.

**METHODOLOGY**

The Cross sectional study has sample of 235 healthy adults, (M=143) with the mean age = 26.6 year with prior informed consent and ethical clearance. Subjects were screened with 36-SF health questionnaire to consider health status. 17 Subjects were excluded with poor physical efforts to perform VM, failed to complete the procedure, and to recover to basal parameters in given time.

**Method:**

All subjects satisfying the criteria for the study were made to achieve
relaxed sitting (RS) position on the chair with feet supported on floor (hips and knees 90 degree flexion) for 3 min., prior to the test procedures, parameters (SBP, DBP, and HR) were measured in the RS position.

The subject blows air at 40-mmHg pressures for 25 seconds into a mouthpiece, which was attached to a modified sphygmomanometer to measure airway pressure. In this study, the glottis remains open to communicate for measurement of pressures from the thorax into the sphygmomanometer. A small needle was routinely placed into the rubber tube to provide a small air leak. This prevents the subject from closing their glottis and from developing the necessary pressures with the cheek muscles. (Fig-II)

SBP and DBP were recorded by sphygmomanometer from brachial artery at elbow. HR was recorded by Nike - strap at Xiphoid-ternal level. This strap made to sense the left ventricular apex beats and watch to receive the impulses. Considering the basal parameters in RS position in chair, subjects were allowed SLF with forearms supported on thigh for a minute and parameters were recorded.

Resting for a minute in SLF position, VM was performed and only change in HR (max) was recorded during 25 seconds. Immediate post VM release changes in parameters were recorded in SLF position. After the recording of post valsalva release response, subjects were allowed to resume RS position and parameters were recorded at the end of 3rd minute. (Fig – III) On the second day, subjects were asked to perform the similar procedure with SQ and parameters recorded.

Data analysis

Statistical analysis was performed with SPSS for repeated Measures ANOVA for comparison of the measured variables (SBP, DBP, and HR) within and between the two positions defecation. The LOS was set at < 0.05 or 95 % confident interval (CI).

RESULT

Graph I. Graphical representation suggesting of SBP responses in RS position with SLF and SQ positions

<table>
<thead>
<tr>
<th>Position</th>
<th>SBP 117</th>
<th>SBP 117</th>
<th>SBP 120.6</th>
<th>SBP 120.6</th>
<th>SBP 116.9</th>
</tr>
</thead>
<tbody>
<tr>
<td>SLF</td>
<td>117</td>
<td>117</td>
<td>116.7</td>
<td>116.7</td>
<td>116.7</td>
</tr>
<tr>
<td>SQ</td>
<td>117</td>
<td>120.6</td>
<td>123.9</td>
<td>116.8</td>
<td>116.8</td>
</tr>
</tbody>
</table>
Graph II. Graphical representation suggesting of DBP responses in RS position with SLF and SQ positions

![Graph II](image)

Graph III. Graphical representation suggesting of HR responses in RS position with SLF and SQ positions

![Graph III](image)

**DISCUSSION**

**SLF vs. SQ positions without VM:**

This study has shown significant difference in variables of SQ and SLF positions as compared to RS position. SBP and DBP significantly increases in SQ compared to SLF position. In SQ position, intraabdominal and so interathoracic pressures are responsible for increasing the SBP. HR shows significant response in SQ compared to SLF.

**SLF vs. SQ positions with VM:**

A high significant raise has shown in HR by both the positions with VM. But, increase in HR was more significant in SLF than SQ position. Significant increase in HR was higher in SLF position, because in VM at the onset of strain, contraction of thoracic cage compresses the lung and causes the large raise in interathoracic pressure; this compresses the vessels within the chest. Moreover, compression of thoracic vena cava compromise venous return to the heart, resulting in a large falls in cardiac output. This leads to secondary fall in aortic pressure as well as aortic pressure falls, the baroreceptor reflex increase the HR. 5, 9

**SLF vs. SQ post VM release:**

When the subject relieves VM, and begins with normal breathing again – deep inspiration follows. When compression of vena cava removed, venous return suddenly increases causing a rapid raise in cardiac output several seconds latter, which leads to overshoot of arterial pressure as the systemic vascular resistance increases due to sympathetic activation that occurred with VM. However, peripheral circulation to lower limbs is compromised in SQ compared to SLF position may be responsible to increase the BP in the upper half of the body, which was recorded from upper limbs by the sphygmomanometer.

HR reflexively decreases in response to the transient elevation in arterial pressure. Fall in HR post VM was seen in SLF position and in squatting position, this anatomical spacing of blood volume in abdominal cavity and lower limbs decreases the venous returns to the heart, a direct effect of kinking the femoral veins. So,
venous return is decreased in SQ when compared to SLF position. This might have caused a comparative decrease in cardiac output in squatting position, which was responsible for controlling the large variation in HR in SQ as compared to SLF position.\(^{13,14}\)

**CONCLUSION**

This study has considered the impact of CV system in SLF and SQ positions with and without VM. It is shown that SQ has significant impact on SBP and DBP as compared to SLF position with and without VM. SLF position has a significant impact on HR as compared to SQ with VM. It can be concluded that SLF and SQ with and without VM influence changes in cardio-vascular status. These need to be taken into consideration while planning life-style modifications for high risk population with compromised CV status.

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Academic Performance Of School Children With Their Intelligence Quotient

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Abstract:
Present study was carried out to correlate academic performance of Indian school children with their intelligence quotient (as measured by Porteus Maze Tests). These tests have been tried and tested successfully in previous studies, don’t require sophisticated equipment and are easier to administer than traditional tests like Wechsler Tests. Contrary to popular belief, no statistically significant correlation was found in this study between intelligence and academic grades. Thus intelligence is not a prerequisite to succeed in examinations and therefore in life.

Key words: Intelligence Quotient (IQ), Porteus Maze Tests, Intelligence, Academic performance Academic grades

INTRODUCTION:
Intelligence is the most valuable wealth of humans. Intelligence is assessed and not measured because in all its meaning and application, it is not a thing; it is only an idea, an abstraction. Thurnstone¹ (1946), Griffith² (1933) and Piaget³ (1983) have all come up with their definitions of intelligence but perhaps the pioneer in this field is David Wechsler. He defined intelligence as the aggregate or global capacity of an individual to act purposefully, to think rationally and so to deal effectively with his environment. It helps an individual to consciously adjust his thinking to new requirements. Thus, it is a general mental adaptability to new problems and conditions of life. Intelligent mind and efficient hands work in coordination with each other.

Majority of people are average, a few very bright and a few very dull. Intelligence also varies in the same individual from situation to situation. As child grows in age, so does the intelligence. Mental development occurs from the concrete to the conceptual, formal and symbolic. While heredity determines the level of intelligence, it is the environment that either slows down or expedites its development. Thus, the main purpose of education is to develop child’s intellect in a well-designed environment. Vertical growth of intelligence ceases at 16-20 years of age, but accumulation of knowledge and acquisition of skills continue throughout the life span of an individual. Therefore the so-called “tapping intellectual resources” means an effective advance in the function of the brain⁴.

Intelligence includes many basic factors such as attention, imagination, observation, thinking, judgment and mental perception through sensory
organs and memory. Porteus Maze Tests (used in the present study for assessing intelligence) while being simple, inexpensive and easy to administer, encompass all these aspects of intelligence.

It is popular belief that intelligent people always do well in life. Present study was carried out to correlate academic performance of Indian school children with their intelligence quotient (as measured by Porteus Maze Tests).

MATERIALS AND METHODS:
The study was conducted on 320 randomly selected students (studying in standards 1st to 8th) of a Government School in India half of whom were of either sex.

The area and school were chosen keeping in mind the composition of Indian society with due consideration to various parameters (like socio-economic status) so as to get an unbiased representative sample. The selected children were subjected to general clinical medical examination to rule out any major mental or physical illness or disability.

Informed consent was taken from the principal and parents after explaining them the aim and nature of the study and their wards’ role in it.

Percentage of marks secured by each participating student during the preceding academic year was noted as a measure of his/her academic ability. Children were graded according to their academic performance as follows:

Marks
✓ > 75%
✓ 60% - 75%
✓ 45% - 60%
✓ < 45%

Intelligence Quotient (IQ) of the participants was assessed using The Porteus Maze Tests for various ‘mental’ ages. These paper-pencil tests consist of successive puzzle charts of increasing levels of difficulty. The age inscribed on the toughest test chart which a subject was able to solve successfully was taken as his mental age. Then his IQ was calculated as:

IQ = (Mental age) / (Chronological age) × 100

Children were categorized according to their IQ levels as follows:

<table>
<thead>
<tr>
<th>IQ</th>
<th>Category</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 115</td>
<td>A</td>
</tr>
<tr>
<td>100 - 115</td>
<td>B</td>
</tr>
<tr>
<td>85 - 100</td>
<td>C</td>
</tr>
<tr>
<td>&lt; 85</td>
<td>D</td>
</tr>
</tbody>
</table>

Observations were recorded after taking due care to reduce instrument and observer errors to a minimum. Correlation academic performance with Intelligence Quotient was done using Chi-Square Test.

RESULTS AND DISCUSSION:

<table>
<thead>
<tr>
<th>Table I</th>
<th>Academic Performance</th>
<th>Subtotal</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>21</td>
<td>69</td>
</tr>
<tr>
<td>B</td>
<td>21</td>
<td>91</td>
</tr>
<tr>
<td>C</td>
<td>15</td>
<td>82</td>
</tr>
<tr>
<td>D</td>
<td>13</td>
<td>78</td>
</tr>
<tr>
<td>Subtotal</td>
<td>70</td>
<td>320</td>
</tr>
</tbody>
</table>

Our study found no statistically significant correlation between intelligence quotient and academic performance in the participating students. This finding is revolutionary because it challenges the layman’s notion that intelligence is the sole determinant of academic performance.
So what then is the mantra for success? Previous studies have concluded that an unhealthy child could have a dismal academic record even if his/her genes predict a high IQ for him/her\(^7\). On the other hand, with average intelligence, a person can still excel in studies provided he/she maintains adequate fitness\(^10\). Thus, success at work (which determines success in life) depends largely on fitness and health and not merely on one’s intelligence. Your genes could give you the edge but only after a healthy mind, body and soul have put in their best efforts\(^11\).

There is a lesson to be learnt here for parents and teachers. Your children could be god-gifted but if they are not nurtured in a proper environment, they may not realize their true potential. Physical health, mental peace, social security and spiritual well-being are perhaps more important than natural talent for success in life.

**CONCLUSION:**

Intelligence has no relation whatsoever with academic performance of school children. Children with average IQ can fare well in studies. Conversely, ‘above average’ children may not get the grades expected of them.

**BIBLIOGRAPHY:**

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2. Griffith JH, The psychology of human behaviour, London, George Allen, 1933
5. Mahajan, Gupta: Textbook of PSM (2\(^{nd}\) ed. 1988-89): Social environment (Ch. 11): Pg 135

---

*Graph I*

Chart showing Academic Grades obtained by school children having varying Intelligence

<table>
<thead>
<tr>
<th>Intelligence Quotient Category</th>
<th>No. of students</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>10</td>
</tr>
<tr>
<td>B</td>
<td>18</td>
</tr>
<tr>
<td>C</td>
<td>30</td>
</tr>
<tr>
<td>D</td>
<td>20</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Grade A</th>
<th>Grade B</th>
<th>Grade C</th>
<th>Grade D</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>20</td>
<td>30</td>
<td>24</td>
</tr>
<tr>
<td>18</td>
<td>23</td>
<td>22</td>
<td>22</td>
</tr>
<tr>
<td>30</td>
<td>25</td>
<td>15</td>
<td>19</td>
</tr>
<tr>
<td>20</td>
<td>22</td>
<td>15</td>
<td>13</td>
</tr>
</tbody>
</table>

[Graph showing Academic Grades obtained by school children having varying Intelligence]


A Study Of Nutrient Foramina Of The Metacarpal Bones

Dr. P. S. Shrimankar*, Dr. D. J. Trivedi*, Dr. V.B. Kariya**

* Associate professor, ** Assistant professor, Department of Anatomy, GMERS Medical College, Sola, Ahmedabad, Gujarat

Abstract:
1500 metacarpals, 300 each of 1st, 2nd, 3rd, 4th and 5th metacarpals of unknown age and sex and 200 metacarpals from 20 articulated skeletons for bilateral study where examined for the length of the bone, number, position, direction and distances from both the ends of nutrient foramina. It was observed that almost all the metacarpals has one nutrient foramen in the middle third of their shaft except in 1st metacarpal in which it was in distal third. It was observed that frequency of number of nutrient foramina differ in different metacarpals. In 1st and 2nd metacarpals foramina were situated mostly on the medial surface and in other metacarpals mostly on the lateral surface. There was a good deal of bilateral symmetry and invariably all foramina were directed away from the growing ends of the bone.

Key words: metacarpal, nutrient foramen, nutrient vessel, short long bone

INTRODUCTION:
During routine teaching of osteology, mention is always made of the nutrient foramen on the shaft of long bones. There is a jingle regarding direction of the nutrient canal “they seek the elbow and flee from the knee”! The nutrient artery is a principal source of blood supply to a long bone and being source of blood supply to a bone it plays an important role in the healing process or union after a fracture. The short long bones have only one epiphysis. The head forms the epiphysis in medial four metacarpals whereas in 1st metacarpal, base form its epiphysis. The nutrient artery enters the bone at right angle to the shaft and as the bone grows in length it becomes more oblique in direction towards the non growing end.

The human hand is often involved in machinery and road side accidents. Nowadays it is possible to reconstruct it by plastic surgeons. Therefore it is very important for a plastic surgeon to know all information regarding nutrient foramina to avoid possibility of necrosis if an injury cut off the nutrient vessel in the growing bone or he could avoid limited area of cortex containing the nutrient foramen during open reduction. The 2nd metacarpal as a vascularized bone graft may be harvested entirely in serious injuries with destruction of index finger. An artery located on dorsoulnar side and connecting the head of the 1st metacarpal with the dorsal arcade of proximal nail fold is useful for making a dorsoulnar flap of thumb that can be raised on its artery with a distal pedicle. This can be used for distal skin loss coverage of thumb and for reconstruction of finger pulp.

Because of lack of detailed information regarding nutrient foramina and with the view of its usefulness in academic, surgical as well as in medico-legal practice following study was done to measure number, position in relation to length and circumference, proximity from the ends, direction and bilateral symmetry of the nutrient foramina.
MATERIAL AND METHOD:

In the present study, 1500 metacarpal bones (300 of each 1st, 2nd, 3rd, 4th, and 5th metacarpals) of unknown age and sex from the collection in Department of Anatomy from various medical colleges of Gujarat were studied. For bilateral study, 20 articulated skeletons were examined. The bones were dry, macerated and cleaned.

The number and position of nutrient foramina were identified; a stiff wire was passed through the foramen to see the direction of the canal when in doubt. The measurements were taken (in cms.) with the help of vernier calipers. The whole length of the bone was taken from head of the metacarpal to the base. The distances of
nutrient foramen from both the ends and situation of the foramen on the circumference of the shaft were noted down. The foraminal index was calculated by the following formula:

\[ FI = \frac{DB}{L} \times 100 \]

(DB = distance of foramen from base, L = Total length of the bone)

In the study of bilateral symmetry, Complete symmetry means – Number and position of the foramina were equal in both the sides, Partial symmetry means – Number of the foramina remains same but their position varied on both the sides and No symmetry means number of the foramina unequal on both the sides.

**OBSERVATIONS (Table I to V):**

**TABLE- I**

NUMBER OF THE NUTRIENT FORAMINA

<table>
<thead>
<tr>
<th>Number of metacarpal bone</th>
<th>No. of bones</th>
<th>No. of bones (%)</th>
<th>Total no. of foramina</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Right</td>
<td>Left</td>
<td>Absent foramen</td>
</tr>
<tr>
<td>1&lt;sup&gt;st&lt;/sup&gt;</td>
<td>165</td>
<td>135</td>
<td>26 (8.7)</td>
</tr>
<tr>
<td>2&lt;sup&gt;nd&lt;/sup&gt;</td>
<td>173</td>
<td>127</td>
<td>10 (3.3)</td>
</tr>
<tr>
<td>3&lt;sup&gt;rd&lt;/sup&gt;</td>
<td>147</td>
<td>153</td>
<td>08 (2.7)</td>
</tr>
<tr>
<td>4&lt;sup&gt;th&lt;/sup&gt;</td>
<td>136</td>
<td>164</td>
<td>07 (2.3)</td>
</tr>
<tr>
<td>5&lt;sup&gt;th&lt;/sup&gt;</td>
<td>162</td>
<td>138</td>
<td>07 (2.3)</td>
</tr>
</tbody>
</table>

**TABLE – II**

POSTION OF NUTRIENT FORAMINA IN RELATION TO CIRCUMFERENCE OF THE BONE

<table>
<thead>
<tr>
<th>Number of metacarpal bone</th>
<th>No. of bones</th>
<th>No. of bones (%)</th>
<th>Total no. of foramina</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of bones</td>
<td>Medial surface</td>
<td>Lateral surface</td>
</tr>
<tr>
<td>1&lt;sup&gt;st&lt;/sup&gt;</td>
<td>300</td>
<td>263 (86.2)</td>
<td>27 (8.9)</td>
</tr>
<tr>
<td>2&lt;sup&gt;nd&lt;/sup&gt;</td>
<td>300</td>
<td>223 (69.3)</td>
<td>96 (29.8)</td>
</tr>
<tr>
<td>3&lt;sup&gt;rd&lt;/sup&gt;</td>
<td>300</td>
<td>54 (17.9)</td>
<td>248 (82.1)</td>
</tr>
<tr>
<td>4&lt;sup&gt;th&lt;/sup&gt;</td>
<td>300</td>
<td>49 (15.8)</td>
<td>259 (83.5)</td>
</tr>
<tr>
<td>5&lt;sup&gt;th&lt;/sup&gt;</td>
<td>300</td>
<td>32 (9.6)</td>
<td>290 (87.4)</td>
</tr>
</tbody>
</table>
## TABLE – III
LENGTH OF METACARPALS, DISTANCES OF FORAMINA FROM HEAD AND BASE OF THE METACARPALS

<table>
<thead>
<tr>
<th>Number of metacarpal bone</th>
<th>Mean length (cm)</th>
<th>Mean DH (cm)</th>
<th>Mean DB (cm)</th>
<th>Mean FI</th>
</tr>
</thead>
<tbody>
<tr>
<td>1&lt;sup&gt;st&lt;/sup&gt;</td>
<td>4.24</td>
<td>1.82</td>
<td>2.56</td>
<td>59.29</td>
</tr>
<tr>
<td>2&lt;sup&gt;nd&lt;/sup&gt;</td>
<td>6.54</td>
<td>3.22</td>
<td>3.26</td>
<td>47.18</td>
</tr>
<tr>
<td>3&lt;sup&gt;rd&lt;/sup&gt;</td>
<td>6.37</td>
<td>3.52</td>
<td>2.79</td>
<td>41.25</td>
</tr>
<tr>
<td>4&lt;sup&gt;th&lt;/sup&gt;</td>
<td>5.33</td>
<td>2.88</td>
<td>2.49</td>
<td>43.87</td>
</tr>
<tr>
<td>5&lt;sup&gt;th&lt;/sup&gt;</td>
<td>4.90</td>
<td>2.60</td>
<td>2.38</td>
<td>47.48</td>
</tr>
</tbody>
</table>

(DH = distance from the head, DB = distance from the base FI = foraminal index. The index around 60 = foramen on distal third, around 50 = middle third and below 40 = proximal third)

## TABLE – IV
BILATERAL SYMMETRY OF FORAMINA

<table>
<thead>
<tr>
<th>Number of metacarpal bone</th>
<th>Partial symmetry (%)</th>
<th>Complete symmetry (%)</th>
<th>No symmetry (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1&lt;sup&gt;st&lt;/sup&gt;</td>
<td>15</td>
<td>80</td>
<td>05</td>
</tr>
<tr>
<td>2&lt;sup&gt;nd&lt;/sup&gt;</td>
<td>20</td>
<td>60</td>
<td>20</td>
</tr>
<tr>
<td>3&lt;sup&gt;rd&lt;/sup&gt;</td>
<td>25</td>
<td>70</td>
<td>05</td>
</tr>
<tr>
<td>4&lt;sup&gt;th&lt;/sup&gt;</td>
<td>10</td>
<td>90</td>
<td>00</td>
</tr>
<tr>
<td>5&lt;sup&gt;th&lt;/sup&gt;</td>
<td>15</td>
<td>75</td>
<td>10</td>
</tr>
</tbody>
</table>

## TABLE – V
DIRECTION OF THE NUTRIENT CANAL

<table>
<thead>
<tr>
<th>Number of metacarpal bone</th>
<th>Total no. of foramina</th>
<th>Direction of nutrient canal</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Towards head</td>
</tr>
<tr>
<td>1&lt;sup&gt;st&lt;/sup&gt;</td>
<td>305</td>
<td>305</td>
</tr>
<tr>
<td>2&lt;sup&gt;nd&lt;/sup&gt;</td>
<td>322</td>
<td>00</td>
</tr>
<tr>
<td>3&lt;sup&gt;rd&lt;/sup&gt;</td>
<td>302</td>
<td>00</td>
</tr>
<tr>
<td>4&lt;sup&gt;th&lt;/sup&gt;</td>
<td>310</td>
<td>00</td>
</tr>
<tr>
<td>5&lt;sup&gt;th&lt;/sup&gt;</td>
<td>332</td>
<td>00</td>
</tr>
</tbody>
</table>
DISCUSSION:

The human hand is often involved in machinery and road side accidents. Nowadays it is possible to reconstruct it by plastic surgeons so the knowledge of exact position, number and direction of the nutrient foramina in metacarpal bones is mandatory for them.

Singh (1959) observed that absence of the nutrient foramina were common in 1st metacarpals and double foramina frequently seen in 2nd and 5th metacarpals. Patake & Mysorekar (1977) found that absence of foramina & double foramina both were common in 1st metacarpals. In present study absence of the foramina is most frequently noted in 1st metacarpals and double foramina in 1st, 2nd and 5th metacarpals.

Wood jones (1946) observed that the nutrient foramina of the 1st, 2nd and usually that of the 3rd and 4th were located on their lateral surfaces whereas that of the 5th on the medial surface. As per observations of Singh (1959), Patake & Mysorekar (1976) nutrient foramina of the 1st and 2nd metacarpals were located on medial surface of the bone and that of the 3rd, 4th and 5th metacarpals were on the lateral surface. Present study supports the findings of Singh, Patake & Mysorekar.

In present study, Foraminal index suggests that the nutrient foramina in case of 1st metacarpals were situated on distal 1/3rd of the bone and in rest of the metacarpals in middle 1/3rd of the bone. The datas regarding FI matches more or less with that of study of Patake & Mysorkar (1977).

Patake & Mysorkar (1977) studied that 41% of 1st and 2nd metacarpals, 25% of 3rd and 4th metacarpals and 8.3% of 5th metacarpals showed no symmetry at all. In present study, 5 % of the 1st and 3rd metacarpals, 10 % of the 5th metacarpals and 20 % of the 2nd metacarpals showed no symmetry.

As per observations made by previous workers and also the present study states that without any exception nutrient foramina were directed away from the growing end.

CONCLUSION:

From observations and discussions the following general conclusions can be derived for all the metacarpals studied:

The position of the nutrient foramina on the shaft of the bone (in relation to the length) is variable; however, it is observed in this series that nutrient foramen of the 1st metacarpals is in distal third, whereas in other metacarpals it is in the middle third.

The position of the nutrient foramen on the shaft of the bone (in relation to circumference) is also not constant; however, it is observed that in this series nutrient foramina of the 1st and 2nd metacarpals were on the medial surface whereas that of the 3rd, 4th and 5th metacarpals were on the lateral surface. It is proved by FI that nutrient foramina of 1st metacarpals were on the distal third whereas in other metacarpals they were in the middle third of the metacarpals.

The similar bones having the same or nearly same lengths possess nutrient foramen at variable distances. So, also the similar bones possessing the same or nearly same distance of the foramen from any one of the ends may not have the same length. Hence the derivation of length of a metacarpal from the position of the nutrient foramen is likely to be fallacious.

The numbers of foramina do not seem to have any significant relation to the length of the bone, the smaller bone may
have double foramina and the longer one may have no foramina at all.

Direction of the nutrient canal is always away from the growing end and position of the nutrient foramen is nearer to the growing end.

**BIBLIOGRAPHY:**


6. Su x y; cao q; he k l shanghai institute of hematology, rui-jin hospital, shanghai, 200025.zhonghua yi xue za zhi , china jan 1999, volume, issue, pages: 79; (1); p34-7 chromosomal abnormalities in 38 cml cases of various phases.

Study of Bacteriological Pattern Of Suspected Cases Of Meningitis

Dr Rajesh Soni
Assistant Professor, Department of Microbiology, K. J. Mehata Institute of Medical Studies., Bhavnagar

Abstract:
Meningitis is an inflammatory condition of the membranes that cover the brain and spinal cord. Present study was carried out to know The pattern of bacteriology of suspected cases of meningitis and to find the rate of susceptibility of various age groups. This work is a bacteriological, cytological and biochemical study of cerebrospinal fluid collected from patients clinically suspected of meningitis, and admitted in Civil hospital, Ahmedabad during the period of 5th April 1999 to 11th May 2000. Higher incidences (34 %) of bacteriological positive cases were found among the patients below one year of age. Most commonly isolated organisms was S.aureus (54.1 %) followed by E.coli and klebsiella. Study shows mortality rate of 29.16 %

Key words: Meningitis, Bacteriological Study, E. Coli

INTRODUCTION: Meningitis is an inflammatory condition of the membranes that cover the brain and spinal cord is called meningitis. It occurs as either primary disease or secondary to disease of other parts of the body. Meningitis is caused by bacteria, viruses, fungi and parasites. Most common organisms are Streptococcus pneumoniae, Neisseria meningitides, Haemophilus influenzae, Staphylococcus aureus. Meningitis is a severe acute medical emergency which requires prompt diagnosis and immediate treatment for better outcome of the patient. The incidence of meningitis greatly varies with age. The incidence is much higher in age less than 1 year and after 60 yrs. It is because of many predisposing factors like Respiratory infection, Head trauma, CNS malformation, Hemoglobinopathies, Immunodeficiency status.

Group B Streptococci H. influenzae and Enterobacteriae are most common organisms in neonates. From 3 months to 5 years of life H. influenzae is more common. Meningococci and Pneumococci can cause meningitis at any age of life. Meningitis by Listerial organisms is seen in infants and immuno-deficient adults. Approximately 25 % of newborns with septicemia have associated bacterial meningitis. And the incidence as well as the fatality rate is much higher in premature infants than in full term births. Premature infants are mostly affected because of poor development of blood brain barrier. Diagnosis of meningitis requires strict aseptic collection of cerebrospinal fluid and isolated of causative organisms from the sample without any delay, so that immediate treatment can be started. This work was carried out to know The pattern of bacteriology of suspected cases of meningitis and to find out the rate of susceptibility of various age groups.

MATERIAL AND METHOD: This work is a bacteriological, cytological and biochemical study of cerebrospinal fluid collected from patients clinically suspected of meningitis, and admitted in Civil hospital, Ahmedabad during the period of 5th April
1999 to 11\textsuperscript{th} May 2000 under standard Performa and laboratory diagnosis was done by standard culture procedure\textsuperscript{6} and the isolated organisms were identified by various sets of biochemical reactions. Antibiotic susceptibility testing was done on the isolate organisms by direct disc diffusion method on various commonly used antibiotics.

**RESULT:**
127 samples were collected from clinically suspected cases of meningitis, showed following results.

**TABLE NO. 1**  
Positive cases of CSF culture

<table>
<thead>
<tr>
<th>Total samples</th>
<th>Positive cultures</th>
<th>Negative cultures</th>
</tr>
</thead>
<tbody>
<tr>
<td>127</td>
<td>24 (18.9 %)</td>
<td>103 (81.1 %)</td>
</tr>
</tbody>
</table>

Table no. 1 shows out of 127 total samples out of which 18.9 % samples were positive and 81.1 % samples were negative for bacterial growth.

**TABLE NO.2**  
Distribution of organisms in positive CSF culture according to Gram stain

<table>
<thead>
<tr>
<th>Total positive cultures</th>
<th>Gram positive bacteria</th>
<th>Gram negative bacteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>24</td>
<td>13 (54.1 %)</td>
<td>11 (45.9 %)</td>
</tr>
</tbody>
</table>

Table no. 2 shows higher isolation rate of Gram positive organisms (54.1%) as compared to Gram negative organisms (45.9%)

**TABLE NO.3**  
Organism isolated from CSF

<table>
<thead>
<tr>
<th>Name of organisms</th>
<th>b. of strains Isolated.</th>
<th>Percentages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Staph. Aureus</td>
<td>13</td>
<td>54.1 %</td>
</tr>
<tr>
<td>E.Coli</td>
<td>04</td>
<td>16.6 %</td>
</tr>
<tr>
<td>Klebsiella</td>
<td>03</td>
<td>12.5 %</td>
</tr>
<tr>
<td>Pseudomonas</td>
<td>03</td>
<td>12.5 %</td>
</tr>
<tr>
<td>meningococci</td>
<td>01</td>
<td>04.2 %</td>
</tr>
</tbody>
</table>

Out of 24 bacteriologically positive cases, 54.1 % cases show presence of coagulase positive S.aureus. Gram negative bacilli were recorded in the rest of cases.

**TABLE NO. 4**  
Association of color of CSF with positive cases

<table>
<thead>
<tr>
<th>Color</th>
<th>Total cases</th>
<th>Culture positive</th>
<th>Percentages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clear</td>
<td>88</td>
<td>08</td>
<td>9.1</td>
</tr>
<tr>
<td>Red</td>
<td>14</td>
<td>01</td>
<td>7.1</td>
</tr>
<tr>
<td>Turbid</td>
<td>23</td>
<td>14</td>
<td>60.8</td>
</tr>
<tr>
<td>Yellow</td>
<td>02</td>
<td>01</td>
<td>50.0</td>
</tr>
</tbody>
</table>

Table no. 4 shows that turbidity of CSF samples increases isolation rate, while clear CSF samples have less isolation rate.

**TABLE NO.5**  
Correlation of the protein values in CSF with culture positive cases

<table>
<thead>
<tr>
<th>Protein</th>
<th>Total cases</th>
<th>Culture positives</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upto 50 mgm %</td>
<td>32</td>
<td>01</td>
<td>03.1</td>
</tr>
<tr>
<td>50 – 200 mgm %</td>
<td>91</td>
<td>20</td>
<td>21.9</td>
</tr>
<tr>
<td>200 – 400 mgm %</td>
<td>02</td>
<td>01</td>
<td>50.0</td>
</tr>
<tr>
<td>Above 400 mgm %</td>
<td>02</td>
<td>02</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Table no. 5 shows that protein level of CSF is increased and most of culture positive CSF showed protein level between 50 – 200 mgm%.

**TABLE NO. 6**  
Correlation of the sugar values in CSF with culture positive cases

<table>
<thead>
<tr>
<th>Sugar</th>
<th>Total cases</th>
<th>Culture positives</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Below 10 mgm %</td>
<td>12</td>
<td>05</td>
<td>41.6</td>
</tr>
<tr>
<td>10 – 30 mgm %</td>
<td>61</td>
<td>16</td>
<td>26.6</td>
</tr>
<tr>
<td>30 – 50 mgm %</td>
<td>51</td>
<td>02</td>
<td>03.9</td>
</tr>
<tr>
<td>Above 50 mgm %</td>
<td>03</td>
<td>01</td>
<td>33.3</td>
</tr>
</tbody>
</table>

Table no. 6 shows that sugar level of CSF is decreased, and most of culture positive CSF showed sugar level between 10 – 30 mg%.
**TABLE NO. 7**
Relation of Pandy’s test with culture positive cases

<table>
<thead>
<tr>
<th>Pandy’s test</th>
<th>Positive cultures</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>35 (positive)</td>
<td>18</td>
<td>51.4</td>
</tr>
<tr>
<td>92 (negative)</td>
<td>06</td>
<td>06.5</td>
</tr>
</tbody>
</table>

Table no. 7 shows that bacterial isolation rate is higher in positive Pandy’s test.

**TABLE NO. 8**
Showing sensitivity of Gram positive bacteria to various antibiotics

<table>
<thead>
<tr>
<th>Gram positive bacteria n = 13</th>
<th>AS</th>
<th>TE</th>
<th>CS</th>
<th>RF</th>
<th>M</th>
<th>OF</th>
<th>BA</th>
<th>PR</th>
<th>CF</th>
<th>CP</th>
<th>PF</th>
<th>GM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resistant</td>
<td>2</td>
<td>4</td>
<td>9</td>
<td>8</td>
<td>7</td>
<td>7</td>
<td>10</td>
<td>4</td>
<td>8</td>
<td>3</td>
<td>5</td>
<td>9</td>
</tr>
<tr>
<td>Sensitive</td>
<td>11</td>
<td>9</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>6</td>
<td>3</td>
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Table no. 8 shows that majority of Gram positive cocci were sensitive to Ampicillin / Sulbactam (84.6%) followed by Ciprofloxacin (76.9%) and Cephalexin (69.2%). But the same organisms were resistant to Gentamicin, Roxithromicin and Cloxacillin.

**TABLE NO. 9**
Showing sensitivity of Gram negative bacteria to various antibiotics

<table>
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<tr>
<th>Gram negative bacteria n = 11</th>
<th>Q</th>
<th>P</th>
<th>X</th>
<th>C1</th>
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<td>Resistant</td>
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<td>10</td>
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<tr>
<td>Sensitive</td>
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Table no. 9 shows that Gram negative bacteria were more resistant to Chlormphenicol, Cephalexin and Ampicillin, whereas they were more sensitive to Gentamicin, Norfloxacin and Pefloxacin.

**DISCUSSION:**
Meningitis is an inflammatory process involving the coverings of the central nervous system.

Meningitis occurs either primarily to secondary to a disease in other parts of the body. In newborn infants prematurely, birth trauma, maternal infection, abnormal perinatal histories, surgical & invasive procedures at birth time etc. are most liable factors for the early onset of symptoms of bacterial meningitis, prenatal infection may make newborn more susceptible to the disease. The portal of entry of infection in newborn may be lungs, umbilicus or conjunctiva, which may be infected during passage through birth canal and from these site organisms gain entrance to meninges via blood stream.

Several workers\(^7,8,9,10\) have observed that due to associated disease in other parts of body i.e. respiratory infection, ear infection, cerebral abscess, skull fracture, infection adjacent to meninges and septicemia, young children become susceptible to meningitis.

Patients of early infant ages are highly susceptible due to incomplete development of their immune system and poor capacity to transfer certain antibodies across the placenta. As placenta can transfer antibodies against H. influenzae organisms,
incidence of it is low in early infancy i.e. up to 3 months of age.

Simultaneously, there is increased incidence of H. influenzae meningitis in children with immunoglobulin deficiencies and functional or anatomical spleen defects. The incidence of bacterial meningitis was found to be 8.9% in present study which is almost comparable to several other previous findings.

The present study is also consistent with the finding that all age groups can be affected. In present study 7 patients were above 30 yrs of age one of which was above 65 yrs also. The incidence is higher in children below 3 yrs with Staph. Aureus being the predominant infection organism followed be Gram negative bacilli.

In present study in one patient smear was positive for meningococci but culture negative, whereas H. influenzae & S. Pneumoniae were not isolated from any of CSF samples even though they are very much common organisms.

In present study 12 cases were with in 1 – 5 years of age group out of which Staph aureus were isolated in 7 cases and E.coli in 1 case. But case history of these cases do not indicate any traumatic lesions, septicemia, bacteremia or ventriculo – peritoneal shunt surgery, hence the roll of these organisms in meningitis can not be established.

The failure to isolate these organisms may be due to the technical problems like (1) not using sterile containers in some cases (2) delay in transporting the samples (3) not using transport media which will preserve the viability of delicate organisms (4) contaminated inoculating media (5) high temperature of the incubator which does not permit the growth of delicate organisms\textsuperscript{11,12}.

However the incidence of meningococcal meningitis appears to be low. Haemophilus influenzae predominantly affects children up to 3 years of age. S. pneumoniae can affect patients of all age\textsuperscript{13,14}.

In present study 31 cases were microscopically suggestive of pyogenic meningitis by the presence of polymorphonuclear leucocytes but out of these 11 were culture negative. Recovery of microorganisms from clinically labeled meningitis was comparatively lower then the appearance of bacteria by direct smear examination, probably because of the presence of more exacting bacteria present in sample that did not grow upon conventional media of isolation. Dead or static bacteria due to effect of antimicrobial agent or immunoglobulin covering over the surface of bacteria fail to grow in culture.

However such bacteria can be demonstrated by Gram stain examination which is the simple most and accurate method and also an essential step for diagnosis of bacterial etiology of meningitis.

The Gram stain examination is highly valuable in diagnosis during antibacterial treatment and even prior the treatment has started. Therefore, it can be used as preliminary screening examination for the early management of infected patients and in such case treatment can be started according to Gram stain results.

However, the traditional method of isolation and identification of microorganisms from CSF specimen should not be neglected due to its own importance in tracing out the epidemiology of disease and to know the exact antibacterial susceptibility of causative organisms.

From the present study it is also apparent that the clinical picture of meningitis many a times is confusing and the clinical diagnosis may be contradictory to bacteriological, cytological and biochemical studies.

Biochemical and cytological examination are an additional help in the diagnosis of meningitis. In the bacterial
meningitis cell response if higher with predominance of polymorphonuclear leucocytes, increased protein levels and decreased sugar level. While in viral meningitis cell response is mainly lymphocytes with normal or raised protein and slightly or moderate decreased sugar level.

The lower level of sugar is associated with bad prognosis of patient. In present study 73.22 % cases labeled as bacterial meningitis had increased protein value above 50 mgm%.

In present study staphylococcus aureus was isolated in 13 species out of 24 culture positive cases. E.coli isolation rate was 16.6% and for klebsiella was 16.6 %. Isolation rate for pseudomonas was 12.5 % but H.influenzae was not isolated in present study.

An overall 19 % cases were found culturally positive, the low percentage of positive culture cases was also noted in other studies. It might be due to prior antibiotic treatment as the most probable factor responsible for culture negative cases. In the present study 90 – 95 % GPC was found to the sensitive to many antibiotics like ampicillin – sulbactum, ciprofloxacin, and cephelexin, but 70 – 80 % GPCwere found to be resistant to gentamicin, roxythromicin and cloxacinil also. But the study indicates that most of Gram negative bacteria were resistant to most of antibiotics. However the emergence of resistant strains of some Gram negative bacteria encountered in the study suggests that blind treatment with conventional antibiotics should not be started.

In present study the mortality in bacteriologically proven meningitis is 29.16 % which is comparable with other studies.

CONCLUSION:
It can be concluded from the present study that in 40 to 60% cases, biochemical and cytological response correlated well with bacterial meningitis. Higher incidences (34 %) of bacteriological positive cases were found among the patients below one year of age. Most commonly isolated organisms was S.aureus (54.1 %) followed by E.coli and klebsiella. From the organisms isolated, Gram positive cocci were sensitive to ciprofloxacin, ampicillin – sulbactem and cephelexin. Whereas Gram negative organisms were sensitive to gentamicin, norfloxacin and pefloxacin.with overall mortality rate of 29.16%.

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Brief Case History and Literature Review:

Hutchinson Gilford Progeria Syndrome

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Key words: Hutchinson-Gilford Progeria Syndrome (HGPS), Progeria

INTRODUCTION:

Hutchinson-Gilford Progeria Syndrome (Progeria or HGPS) is an extremely rare and potentially fatal genetic condition with an incidence of 1 in 8 million births worldwide. HGPS is characterized by premature ageing with death from heart attack or stroke at the age of 13-14 with average life expectancy of 13 years in age range of 7-27 years old. Each child with progeria may represent a new sporadic dominant mutation. Males outnumber females with a 1.5:1 ratio, and racial susceptibility strongly favors Caucasians who represent 97% of patients. The name of this syndrome has been derived from Greek word geras which means “prematurely old” and this disease was first described by Jonathan Hutchinson in 1886 and named by Hastings Gilford in 1904.

CASE REPORT:

A 7 year old male child presented to us with coarsening and generalized laxity of skin with failure to thrive for past 3 to 4 years. Perinatal history was uneventful, he was apparently normal till the age of 2 years after that his parents started to notice the changes in him. No family history suggestive of similar complaints could be elicited. The main concern of the parents was his old man like appearance.

GENETIC DEFECTS:

HGPS falls into category of group of disorders called laminopathies in which nuclear lamin is affected and causes the clinical manifestations. The gene defect causing HGPS has recently been identified as a single base mutation in the gene LMNA, coding for the nuclear protein Lamin A, which is the structural scaffolding that holds the nucleus of a cell together. Mutations in LMNA have been identified in 885 of cases and mechanism is unknown in remaining 12% of cases. The inheritance pattern in HGPS is autosomal dominant with all subjects having the disease as a result of denovo mutation, the most common being the mutation of p. G608G. Researchers now believe that the defective Lamin A protein makes the nucleus unstable. That cellular instability appears to lead to the process of premature aging in Progeria.
A 7 year old male child born of non-consanguineous marriage was presented to us with chief complains of old man like appearance, generalized laxity of skin, old man like appearance and not gaining proper weight and height.

Image 2 - Showing generalized lax skin more evident on face, anterior axilla, upper chest & over both the knee joint.
**CLINICAL FEATURES:**

Children with progeria usually have a normal appearance in early infancy. At approximately 9 to 24 months of age, affected children begin to experience profound growth delays, resulting in short stature and low weight. They also develop a distinctive facial appearance characterized by a disproportionately small face in comparison to the head; an underdeveloped jaw (micrognathia); malformation and crowding of the teeth; abnormally prominent eyes; a small nose and a subtle blueness around the mouth.

In addition, by the second year of life, the scalp hair, eyebrows, and eyelashes are lost (alopecia), and the scalp hair may be replaced by small, downy, white or blond hairs and gives a look of bird like faces. Additional characteristic features include premature generalized atherosclerosis, cardiovascular disease and stroke, hip dislocations, unusually prominent veins of the scalp, loss of the layer of fat beneath the skin (subcutaneous adipose tissue), defects of the nails, joint stiffness, skeletal defects, and/or other abnormalities, alopecia, horseman stance, pyriform thorax, thin legs with prominent joints, short stature. Individuals with Hutchinson-Gilford Progeria Syndrome are prone to develop insulin resistant diabetes and premature widespread thickening and loss of elasticity of artery walls (arteriosclerosis), which result in life-threatening complications during childhood, adolescence or early adulthood.

**DIAGNOSTIC FINDINGS**

The diagnostics methods in HGPS mainly comprise of clinical (abnormal lipid level, increased excretion of hyalurinoic acid in urine and abnormal blood count), histological (abdominal skin biopsies with abnormal nuclear morphologies), radiological (abnormal brain, thorax, long bones and phalanges x rays showing acro osteolysis, hypoplastic facial bones & sinuses, open cranial sutures & frontanelles, wormian bones & coxa valga) and genetical (screening for mutation in gene LMNA).

**TREATMENT:**

There is no effective cure by any treatment modalities for this HGPS. Children with HGPS usually die at the age of 14. Symptomatic treatments include regular diets, routine immunizations, aspirin, multivitamins, surgical procedure and physical and psychological therapies. A new promising therapy in future will includes treatment with Fernesyl transferase inhibitors which have shown to reverse the abnormalities in cells expressing progerin.

**BIBLIOGRAPHY:**


Brief Case History and Literature Review:

Superficial Angiomyxoma of the thigh

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Key words: Superficial angiomyxoma, Myxoid stroma

INTRODUCTION:

Superficial angiomyxoma is an under-recognized lesion which encompasses the majority of lesions referred to in earlier literature as "cutaneous myxoma". Most of the cases present as cutaneous papules or polypoid lesions and have a predilection for the head and neck region and the trunk.

It is a rare benign neoplasm characterized by a conglomerate of multiple, moderately to sparsely cellular angiomyxoid nodules with scattered small blood vessels. We present here a case of superficial angiomyxoma in a 22 year-old male and discuss the morphological features, both macroscopic and microscopic, which are helpful for distinguishing this tumor from other soft tissue tumors, especially cutaneous focal mucinosis, cutaneous myxoid cyst and aggressive angiomyxoma. We also discuss the disease entity `superficial angiomyxoma'.

CASE REPORT:

A 22 year old male came with complaints of a painless, gradually increasing swelling on the posterior aspect of right thigh since 5 years. Physical examination showed a soft subcutaneous swelling measuring 3x2 cm. The mass was completely removed. On gross examination, it was well circumscribed, partly encapsulated and soft in consistency. Cut surface was gray-white with myxoid change(Fig1).Histopathological examination of hematoxylin-eosin-stained sections at lower magnification revealed a well encapsulated tumour with a myxoid matrix(Fig2). At higher magnification, the tumor consisted of small thin-walled blood vessels and benign spindle-shaped or stellate tumor cells embedded in a fibromyxoid matrix(Fig3,4).Focal perivascular lymphocytes were also noted.

Based on these clinical and histologic findings, we diagnosed this condition as superficial angiomyxoma.

DISCUSSION:

Superficial angiomyxoma was first described as a cutaneous myxoma of Carney’s complex by Carney et al. in 19861. Carney’s complex is an autosomal dominant syndrome characterized by myxomas of the heart, skin and breast; pigmentation of the mucous membrane; and endocrine disorders such as Cushing’s syndrome and acromegaly.

In 1988, Allen et al. reported cutaneous tumors that were histologically similar to cutaneous myxomas of Carney’s complex without evidence of the complex2. He reported 30 superficial angiomyxomas in 28 patients aged 4 to 78 years. Most of the tumors were soft, measuring 0.5 to 9 cm, on the head, neck, trunk, or lower extremity. Microscopically, they were located between the dermis and the subcutaneous tissue and consisted of
Fig 1—Gross photograph showing a well circumscribed, partly encapsulated tumor, with a gelatinous cut surface.

Fig 2—Photomicrograph showing a well encapsulated tumour with a myxoid matrix (H&E, 40x).

Fig 3—Photomicrograph showing small thin-walled blood vessels and benign spindle-shaped or stellate tumor cells embedded in fibromyxoid matrix (H&E, 200x).

Fig 4—Photomicrograph showing benign spindle-shaped or stellate tumor cells (H&E, 400x).
mucoid material and small blood vessels.

The word `superficial' was used in order to distinguish superficial angiomyxoma which is a benign myxomatous neoplasm characterized by moderately to sparsely cellular angiomyxoid nodules with scattered small vessels from aggressive angiomyxoma, which is an uncommon, locally aggressive but non-metastasizing soft tissue neoplasm occurring most commonly in the female genital region. There is a need to differentiate superficial angiomyxoma from other benign cutaneous myxomatous lesions --cutaneous focal mucinosis and cutaneous myxoid cysts.

Macroscopically, cutaneous focal mucinosis and cutaneous myxoid cysts are small, ill-defined nodular lesions less than 2 cm in size. Aggressive angiomyxomas are usually infiltrative. In contrast, superficial angiomyxomas are generally larger than cutaneous focal mucinoses and cutaneous myxoid cysts, and are not infiltrative but well circumscribed. The present tumor is a capsulated myxomatous nodule, a feature never seen in aggressive angiomyxomas.

Microscopically, all the above-mentioned myxomatous tumors are composed of spindle-shaped, stellate and plump oval cells in a myxomatous stroma and show different quantities and patterns of vessel elements. Cutaneous mucinosis and cutaneous myxoid cysts contain clefts and mucin pools, but few vessels. In contrast, superficial angiomyxomas show a scattered distribution of small to medium sized thin-walled blood vessels, however large caliber vessels seen in aggressive angiomyxomas are absent. Occasional findings seen in superficial angiomyxoma are perivascular hyalinization, perivascular lymphocytes, fibrin thrombi, interstitial haemorrhage with blood lakes, and hemosiderophages. One unique feature in contrast to other cutaneous myxoid tumours is the presence of neutrophils, eosinophils, lymphocytes and mast cells in the stroma.

Also secondary changes including perivascular hyalinization, medial hypertrophy, a prominence of mast cells and focal areas of hypercellularity with associated nuclear hyperchromasia and pleomorphism are commonly seen in aggressive angiomyxomas.

In 1999, Calonje et al. reported clinicopathologic and immunohistochemical features of superficial angiomyxoma as an independent disease entity in 39 patients. The tumor was more common in men. The age of onset and size ranged from 0 to 82 years and 1 to 5 cm respectively. Immunohistochemically, tumor cells were positive for vimentin but negative for CD34, smooth muscle actin, HHF-35, S-100 protein, desmin, cytokeratin, and glial fibrillary acidic protein.

However, Fetsch et al. described superficial angiomyxomas to be sometimes positive for desmin and smooth muscle actin. Our patient did not have Carney’s complex because the subcutaneous tumor was not accompanied by non-cutaneous myxomas, pigmentation of the skin or mucous membrane, or endocrine disorders.

Based on the clinical and histopathological findings of the tumor, we have diagnosed this condition as superficial angiomyxoma. Thus these tumors can be defined as bland, superficial myxoid lesions with prominent thin walled vessels and are alternately called as cutaneous myxomas.

**DIAGNOSTIC FINDINGS:**

- Predominantly involves dermis and subcutis
  - May involve skeletal muscle on face
  - Multilobulated, poorly circumscribed
- Myxoid stroma
Superficial angiomyxoma is a rare benign tumour of adolescents and adults, with a predilection for the maxilla and mandible. It can arise in the genital region, trunk, lower and upper extremities. In the head and neck region the tumour can be seen on the head, lower eyelid, preauricular region, pinna and the external auditory canal. Multiple myxomas occurring in the external ear or breast are almost pathognomonic of Carney's syndrome. It is a benign lesion but local recurrence is common after incomplete excision.

It is positive only for vimentin. Since immunohistochemical features have not been established, some researchers mention that immunohistochemical studies are not helpful for the differential diagnosis of myxoid tumors. More reports and further analyses will be needed to characterize superficial angiomyxoma clinically and immunohistochemically.

There is a propensity for local recurrence if cutaneous myxomas are incompletely excised. However, no metastasis has been reported.

**BIBLIOGRAPHY:**


Brief Case History and Literature Review:

**Blount Disease in 3 year old boy from India.**

*Dr. Mehul M. Gosai*, Dhaval Solanki **Dr. Hareshwaree B. Hariyani***, Dr. Payal H. Purohit”,

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**Key words:** Blount disease, Idiopathic tibia vara,

**INTRODUCTION:**

Idiopathic tibia vara (Blount disease) is an acquired form of tibial deformity found mainly in children who are Afro-Caribbean and it is also associated with obesity, short stature and early walking. There does not appear to be an obvious genetic factor. It is uncommon1. The incidence of Blount disease in the India is unknown. The estimated prevalence of infantile Blount disease in the population of young children with significant bow legs in the United States is 0.007 or less than 1%. The exact frequency in persons of all ethnicities is unknown and most likely is less than 1%. 2,3,4,5 Blount disease or idiopathic tibia vara is a developmental deformity of the proximal tibial medial physis that produces a varus deformity of the knee6. It has been classified into three types depending on the age at onset: infantile (1-3 yr), juvenile (4-10 yr) and adolescent (11 yr or older).

The juvenile and adolescent forms are commonly combined as late onset tibia vara7. Although the etiology of Blount disease may be multifactorial, the strong association with childhood obesity suggests a mechanical basis due to an abnormal compression on the medial aspect of the proximal tibial physis. A comprehensive analysis of multiplanar deformities in the lower extremity reveals tibial varus, procurvatum and internal torsion along with limb shortening. Additionally, distal femoral varus is commonly noted in the late-onset form. The diagnosis of both forms of Blount disease is based on history, physical examination, and the most important tool is a radiographs of the affected knee. When a patient has early-onset disease, a realignment tibial osteotomy before the age of four years decreases the risk of recurrent deformity8. so early diagnosis is very much important for a better long term prognosis. Gradual correction with distraction osteogenesis is an effective means of achieving an accurate multiplanar correction, especially in patients with late-onset disease9. In child less than 3 yr old and langenskiold stage < 3, a bracing is effective and can prevent progression of the disease in 50% of these children. A maximum trial of 1 year of orthotic management is recommended. If complete correction is not obtained after 1 yr or if progression occurs during this time, then a corrective osteotomy may be indicated7. Many different types of osteotomies have been described in the literature including opening and closing wedge, spike, dome, and oblique osteotomies. 10,11,12 Complications associated with the treatment
of Blount disease include loss of alignment, vascular impairment, pathologic fractures, wound infection and malalignment. In general, the later the onset, there is a greater chances of progression of the deformity, the increased likelihood of articular changes. There were few case reports of infantile Blount disease other than Afro-Caribbean ancestry. So we want to share this case with Pediatricians of India.

A 3 year old child, a case of Blount Disease presented with typical left lower limb deformity and Radiological Images of our case : X – Rays :

Left upper tibial physis is showing medial slopping of the epiphysis, widening of the physis and fragmentation of the metaphysis with significant lateral thrust
3D- Reconstructive Ct-Scan of this pt. of Blount Disease s/o classical diagnostic findings
CASE REPORT:

Three year old Hindu (devipujak caste) boy has presented with deformity/bending of left leg and difficulty in walking since last one year. History was not suggestive of fever and trauma. Before one year child had suffered from measles. No similar complain noted in any family members. One female sib succumbed due to sepsis at six mmonth of age. Mother has completed DOTS therapy category I / antituberculous treatment before four month back. Boy was product of second degree consengous marriage. Child was fullterm hospital delivered and vaccinated for oral polio (OPV) and BCG. Child’s development was normal. child was able to walk at the 1yr of age. Child was on exclusive breast feeding up to sixth month and then shifted on staple diet. On examination vitals were normal. Teeth were 20 in number. Physical examination shows that the left lower leg was angulated inward. A non tender bony protuberance was palpable along the medial aspect of the proximal tibia. Pain was not evident. On observing the standing child from behind, the bowing was centered below the knee without involvement of the femur. Left leg tibia vera was noted but a wrist widening and rib beading was not found. On walking, a lateral thrust of the knee or sudden lateral knee movement with weight bearing was noticeable. Alkaline phosphatase(ALP) level was high 800 IU/L. Anteroposterior radiograph of both knees reported asymmetric, sharp angulation involving left tibia. Left metaphyseal-diaphyseal angle was more than 30 degree (>20 is the diagnostic). Tibia-Femoral angle was 150 and metaphyseal-epiphyseal angle 12.5. All these angles are very important and can be considered at the time of surgical correction in a case of Blount Disease. There were a medial slopping of the epiphysis, widening of the physis and fragmentation of the metaphysis with significant lateral thrust were noted on x-ray film of the affected knee. No abnormal findings were shown in the chest X-ray film and wrist X-ray film. Anthropometry examination was normal except leg length discrepancy. left leg was 4 cm shorter than right leg. Weight for height was more than 90 %. Height for age was 93 %. Body mass index (BMI) 15.5 and mid parental height (MPH) were 166.5 cm. Vitamin D 6 lac unit was administered as an empirical treatment in past, although there were no generalized radiological findings.

After three months of this empirical treatment, again child came to us with a progressive deformity in left leg and complaining of yellow color discharge from right ear for 5 days. Alkaline phosphatase(ALP) level was high 822 IU/L. S.c calcium was normal (1.21 mg /dl) and Serum phosphorous level was normal (7.2 mg/ dl). Renal function tests were also normal. Amoxicillin 75 mg/kg/day for 7 days was administered for otitis media treatment. Orthopedic surgeon’s expert opinion was taken for further management. We have also planned to vaccinate this child on follow up.

DISCUSSION:

Idiopathic tibia vara (Blount disease) is an acquired form of tibial deformity found mainly in children who are from Afro-Caribbean origin and it is also associated with obesity, short statur and early walking. The infantile type of Blount disease demonstrates a female predominance, whereas the late-onset types demonstrate a male predominance. However, we reported Blount disease in non obese male.

Sevastikoglou and Eriksson reported 4 persons with tibia vara in the same family, of whom 2 were identical twins.4 Schoenecker et al also found a positive family history in 14 of 33 patients.5 But, in our case we have not found any positive family history. Boy was a product of second
degree consengous marriage. However, no direct proof of a genetic relationship has been discovered.

The clinical presentation of the different types of tibia vara varies according to the age of onset. In infantile tibia vara, children generally start to walk early usually when aged 9-10 months. In our case child has started to walk at around 1 yr of age. At the onset of the disease, differentiating between early infantile Blount disease and marked physiologic bowlegs is difficult. This physiological deformity usually resolves spontaneously by the time the child is aged 2 years. Our case was presented at 3 yr of age. In contrast to physiologic genu varum, infantile Blount disease can progress to severe deformity.

Blount disease is associated with a prominent metaphyseal beak, internal tibial torsion, and leg-length discrepancy as noted in our case. We have founded unilateral involvement. However, it is bilateral in approximately 80% of cases.

The differential diagnosis of Blount disease includes physiologic bowing, congenital bowing, rickets, Ollier disease, trauma, osteomyelitis, and metaphyseal chondrodysplasia. The diagnosis of Blount disease is based on history, physical examination, and, most important the radiographs of the knee as reported in our case. Mild or healing rickets with residual bowing may be difficult to differentiate from stage 2 infantile tibia vara. However, rickets affects the skeleton in a generalized and symmetric fashion with loss of the zone of provisional calcification in the physis. In addition, the typical biochemical abnormalities of rickets helps to differentiate these both conditions.

CONCLUSION:
Always consider differential diagnosis of Blount disease in more than 2 yr old child present with tibia vara although Blount disease is rare in India. When a patient has early-onset disease, a realignment tibial osteotomy before the age of four years decreases the risk of recurrent deformity. So early diagnosis is important for a better long term prognosis.

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