INTRODUCTION

The 26th Royal College of Obstetricians and Gynecologists Study Group on intrapartum fetal surveillance in 1993 recommended measurement of acid base balance of the umbilical cord arterial and venous blood after delivery as measure of the fetal response to labor.¹ The cord acid base assessment provides an objective measure of neonatal condition at delivery.¹⁻⁷ Acute fetal distress during labor remains one of the primary objectives of obstetrical surveillance. As there is a weak correlation between the biochemical parameters that provide more reliable diagnosis (i.e. lactic acid, pH and HCO₃⁻) in comparison to the subjective criteria for fetal acidaemia i.e. Apgar score (0-10), fetal heart rate 120-160 beats per minute (bpm) and meconium staining of liquor. Acute fetal distress induces asphyxia leading to hypoxia of most of the organs which causes increase in lactic acid level, along with alteration in pH and HCO₃⁻.³ Most of the parameters used for fetal monitoring have good sensitivity but low specificity.⁵,¹⁰ Now a days, umbilical cord vessel acid base analysis is increasingly employed as an end point in the study of antepartum and intrapartum care.¹¹ It is the most accurate method of quantifying the acid-base status of the fetus at the moment of delivery¹² and is an important adjunct for determining the extent and cause of fetal acidaemia at delivery.¹³ The cord acid base assessment provides an objective measure of neonatal conditions at delivery.¹¹ The only scientific, objective mean of diagnosis of fetal asphyxia at delivery is through umbilical blood acid base studies.¹² Umbilical cord blood sampling is regarded as the “gold standard” in the analysis of the biochemical state of the fetus. Umbilical cord blood acid base analysis has emerged in recent years as a method of assessing the newborn objectively because other predictors of outcome, including the Apgar score, are based on subjective criteria and they correlate poorly with neonatal outcome.¹³⁻¹⁴

SUBJECTS AND METHODS

This study was conducted at the Basic Medical Sciences Institute, Jinnah Postgraduate Medical Centre Karachi, Sindh - Pakistan. The control samples of cord blood were collected from 40 normal term neonates just after the delivery. Acidaemic group of 40 neonates included in the study were those who showed any subjective or objective signs of fetal distress. The newborns were chosen from the labor room of Gynecology and Obstetrics Department of JPMC, Karachi. The fetal acidotic subjects included in the study were 40 newborn infants with subjective or objective signs of
fetal distress i.e. fetal heart rate (FHR) < 120 (bpm) or FHR > 160 bpm, history of meconium staining of liquor or Apgar score below 7 at 1 minute and at 5 minutes intervals. Only those neonates were included in control group who were born as full term delivery of at least 36-40 weeks gestational period with no subjective signs of fetal distress i.e. Apgar score >7 in 1 minute, normal FHR (120-160 bpm) and no history of liquor staining with meconium. Those neonates were not included in study with any congenital abnormality, low birth weight or small for date or their mothers suffering from any cardiac disease, renal disease, diabetes mellitus, pregnancy induced hypertension, any hepatic or respiratory problem or any acute illness prior to the labour. Soon after delivery before the first breath of baby, 2 ml blood was taken from umbilical artery in centrifuge tube containing sodium floride/EDTA in ratio 2:1, used for lactic acid estimation by enzymatic colorimetric method which is method of choice. A corning-248 blood gas analyzer (Ciba Corning Diagnostics, Ltd.) was used to estimate the level of pH and HCO₃⁻. This machine is self calibrating and another 2 ml of umbilical blood sample collected in bottle containing 3 mg EDTA used for Hb estimation done by cyanemet hemoglobin method.

RESULTS

In this study, 40 full term newborns of age about 36-40 weeks with acidemia were compared with 40 full term age matched healthy newborns as controls during a period of 6 months. Table I presents the comparison between the two groups. There was marked difference between the values of control and acidic groups. The lactic acid was significantly higher in acidic group, with mean of 1.87±0.075 mmol/l and in the controls, mean was 0.92±0.035 mmol/l (p=<0.001) while pH and HCO₃⁻ levels were low in acidemic group with the mean of 7.13±0.009 (p=0.01), 18.43±0.498 (p=<0.001) respectively in comparison to control group in which the mean was 7.30±0.009, 22.70±0.526 respectively showing the significant importance with the application of chi-square test. Hemoglobin (Hb) concentration was low in acidotic group, which also may be the contributing factor in neonatal acidemia. Table II shows the comparison between acidemic neonates with controls for the presence of subjective criteria. Here the control neonates showed normal findings like Apgar score > 7, FHR in between 120-160 bpm and no history of meconium staining. While 23 of acidotic group had Apgar score < 7 (57.5%), 13 neonates with meconium staining (32.5%) and 17 (42.5%) showed abnormal FHR < 120 or >160 bpm.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control Neonates (n=40)</th>
<th>Acidemic Neonates (n=40)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lactic acid</td>
<td>0.92±0.035</td>
<td>1.87±0.075</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>pH</td>
<td>7.30±0.009</td>
<td>7.13±0.009</td>
<td>0.01</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>22.70±0.526</td>
<td>18.43±0.498</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hb (gm/dl)</td>
<td>14.94±0.214</td>
<td>14.01±0.213</td>
<td>0.01</td>
</tr>
</tbody>
</table>

Table II: COMPARISON OF SUBJECTIVE SIGNS IN ACIDOTIC NEONATES WITH CONTROL GROUPS

<table>
<thead>
<tr>
<th>Variable</th>
<th>Controls</th>
<th>Acidemic Group</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apgar Score</td>
<td>&gt;7**</td>
<td>23*</td>
<td>57%</td>
</tr>
<tr>
<td>Fetal Heart Rate</td>
<td>120-160 Bpm</td>
<td>17*</td>
<td>42.5%</td>
</tr>
<tr>
<td>Meconium Staining</td>
<td>-Ve</td>
<td>13*</td>
<td>32.5%</td>
</tr>
</tbody>
</table>

* Number of neonates showing abnormal subjective signs.
** Infants in good condition, combine score 8-10

DISCUSSION

This study revealed how little attention was paid to confirm fetal academia on authentic basis. Acidotic neonates with abnormal Apgar score were seen in 57.5%, meconium staining in 32.5% and abnormal fetal heart rate in 42.5% in this study population. Different investigators have reported different frequency rates in subjective criteria for fetal acidemia. Only 5% of newborns with meconium staining were acidotic at the time of delivery because the Apgar score does not reflect the fetal acid base status, it cannot be used as an index of academia. In one study, 37% neonates with Apgar score below 1 minute had normal pH and in another study, 73% acidotic babies had normal Apgar scores (Blackstone & Young, 1993). Yeoman, et al in 1985 reported that 53% had
acidemia with meconium staining while other authors have concluded that mostly meconium staining is associated with acidosis and neurologic depression. Blackstone & Young (1993) observed 36-60% FHR problems with acidosis. Meanwhile, Goldaber & Gilstrap had also observed that newborns who had abnormal FHR, 80% were not acidemic at birth while 21% had acidosis. This study was designed to determine and confirm the acid base disturbances of blood during intrapartum period in the fetus. These changes may be very harmful for the neonates if not recognized and treated properly. In our population, at present, there is very little orientation about these disturbances, especially in the newborns. No significant association was found between a pathological CTG recording, fetal Apgar score and acidemia. An increase in cesarean section rate in babies with pathological CTG stresses on the need for additional test to differentiate hypoxic from non-hypoxic fetuses thus avoiding unnecessary intervention. Several studies have reported normal data for vaginal delivery for acid base analysis. In this study, the mean values in control population for acid base are in complete agreement with the mean values given in the studies of above authors but the acidemic values are more marked. The probable reason may be that there is still no any proper orientation about fetal acidemia during intrapartum period and its management. Fetal metabolic acidosis is reflection of dysfunction of fetoplacental unit, which results in anaerobic glycolytic pathway and increase production of lactate. The lactic acid is buffered in the fetus by sodium bicarbonate and some of the excess lactic acid is also buffered by hemoglobin. This study is in complete agreement with the above study because fetal acidemia is associated with metabolic acidosis which results in increased lactic acid, decreased bicarbonate and pH values. The severity of metabolic acidosis usually reflects the degree of the fetal hypoxic insult. Our observations are in accordance with above mentioned research workers and values are highly significant (P<0.001) where decrease in bicarbonate levels, a high lactate level and fall in pH were observed. Regarding fetal anemia of newborn with massive fetomaternal hemorrhage, the fetal blood loss led to severe late decelerations in the antepartal CTG. The newborns showed a marked anemia (Hb 4.9 g%) and a rapidly progressive heart failure. Although, in this study, the fetuses were not grossly anemic and it was an incidental finding, but it appears that there may be some fetomaternal hemorrhage that is consequently influencing the CTG and other subjective parameters of fetal distress.

REFERENCES


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