AN EARLY DIAGNOSIS OF FOETAL DISTRESS BY ESTIMATING THE MATERNAL BLOOD GAS LEVELS DURING INTRAPARTUM PERIOD

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Background: The purpose of this study was to find out the affect of maternal asphyxia influencing the foetus by comparison of the maternal arterial blood gas (ABGs) and acid base levels of normal full term females with the ABGs and acid base values of term females showing any subjective signs of foetal asphyxia during intrapartum period. **Methods:** In this case control study maternal ABGs and acid base levels of 40 normal term pregnant ladies were compared with 40 other term pregnant females with subjective signs of foetal asphyxia (i.e., foetal heart rate >160 bpm or <120 bpm, meconium staining liquor during intrapartum period or Apgar score <5 at one minute after delivery to confirm the foetal distress. Lactic acid and *p*H were also estimated to assess the acid base balance. **Result:** There was considerable difference of lactic acid (LA) and Pco_2 levels which were significantly increased ($p<0.025$) in asphyxiated mothers (AM) as compared with control mothers (CM). No significant difference was observed regarding pH values, bicarbonate ions (HCO_3^-), base excess (BE), carbon dioxide content (CO_2ct) and percentage saturation of oxygen ($\%O_2SAT$) levels in both CM and AM groups. The levels of haemoglobin (Hb), packed cell volume (PCV), partial pressure of oxygen (PO_2) and oxygen content (O_2ct) in AM group were found significantly lower ($p<0.0001$) as compared to CM group. **Conclusion:** We conclude that routine antepartum blood gases estimation will cover all the cases of maternal asphyxia and also mothers with signs of subjective foetal asphyxia like meconium staining, decrease foetal kick counts etc, this will be an additive successful diagnostic and therapeutic approach adapted to confirm the severity of the dangerous perinatal conditions which are responsible for high perinatal mortality rate. Greater awareness may eventually leads to the early diagnosis of the problem on authentic grounds, can also tell us the severity of asphyxia and later on helpful in proper management for a better outcome.

Keywords: Maternal asphyxia, Intrapartal asphyxia, Lactic acidemia.

INTRODUCTION

Intrapartum foetal asphyxia has long been recognized as a major contributor of neonatal morbidity and mortality and is one of the single leading cause of neurological deficits in full term newborns.[1] Recent studies in term newborns have reported an association between acidemia and newborn complications including the complications of CNS, CVS, respiratory system and kidneys. due to intrapartum asphyxia because of metabolic acidosis at delivery.[2]

Umbilical arterial blood gas analysis is becoming the fastest objective method to assess foetal well-being at birth. It is the gold standard assessment of uteroplacental function and foetal oxygenation/acid-base status at birth and it excludes the diagnosis of birth asphyxia in approximately 80% of depressed newborns at term.[3]

Maternal and foetal acid base balance is also important to understand foetal physiology and homeostasis. Acid base values in the foetal and mother blood can be used to assess the adequacy of foetal oxygenation and there by foetal well being *in utero*.[4] Severe hypoxemia during labour may interfere with maternal foetal respiratory exchange leading to anaerobic glycolysis and accumulation of lactic acid.[5]

The resulting acidemia due to lactic acid accumulation at delivery has been associated with complications in term newborns. Low *et al* in 1995 reported an association between intrapartum asphyxia with metabolic acidosis at delivery and an increase incidence of complication in term new born.[6]

Maternal problems related with reduced O_2 supply to the foetus include cyanotic heart disease, severe infection, and hyperventilation with hypocapnia, severe anaemia or placental hypo perfusion cause by hypotension, hypertension or uterine tetany. Impaired placental exchange e.g. placental separation or severe infarction can also be one of the factors. Foetal causes include anaemia or umbilical cord compression with decrease umbilical blood flow.[4]

The measurement of acid base status and blood gases can provide important information regarding clinical status of both mother and foetus

and can play a major role in decision concerning patient care and management.

SUBJECTS AND METHODS

This was a case control study carried out on 80 pregnant full term females (gestational period range:36-42weeks)of which 40 had subjective and objective sign of foetal asphyxia (referred to as asphyxiated mothers AM) and 40 were normal pregnant ladies with no sign of foetal asphyxia (referred to as control mothers CM) chosen.

The AM were the forty selected consecutively patients reporting to the labour room of JPMC Karachi, the included patient with full term pregnancy and sign of foetal asphyxia history of increase or decrease in foetal kicks count, leaking of meconium staining liquor or/with foetal heart rate (FHR) <120 or >160 beats per minutes (bpm), the control were the full term pregnant ladies in labour without any of the above signs or symptoms of foetal distress.

The participants were excluded if they had past history of any heart disease, hypertension, diabetes mellitus, renal or respiratory diseases, hepatic or GIT disorder or any infective or inflammatory diseases. Screening with blood count, fasting blood sugar, urine DR and LFT was also performed, and cases with any abnormal value were excluded.

The study included the qualitative estimation of blood gas analysis, i.e., partial pressure of CO_2 (Pco_2), partial pressure of O_2 (Po_2), O_2 content (O_2ct), CO_2 content (CO_2ct), percent saturation of O_2 ($\%SATO_2$), bicarbonate HCO_3^- and base excess (BE). Lactic acid (LA) and *p*H were estimated to determine the acid base balance and haemoglobin (Hb) and haematocrit (PCV) were also estimated. Blood gas analysis was carried out by the blood gas and electrolyte analyzer. Nova state profile-5, NOVA biochemical, USA, provided in the laboratory of NICVD (National Institute of Cardiovascular Disease) Karachi. The blood gas and electrolyte analyzer primarily provided the values of *p*H, Pco_2, Po_2 and HCO_3^- while the values of CO_2ct, O_2ct and $\%O_2SAT$ were calculated by formula, haematocrit values (packed cell volume PCV) were estimated by microhaematocrit method on microhaematocrit machine and haemoglobin was estimated by cynamethaemoglobin method as recommended by National Research Council of America and College of American Pathologist. For lactic acid estimation 2 ml arterial blood taken in centrifuge tube containing sodium fluoride/EDTA in a ratio of 2:1 and lactic acid was estimated by enzymatic colorimetric method which is the method of choice.

The data was entered in Microsoft Excel and analysis was done on SPSS version 11.0. The results are given in the text and table as Mean±SD for quantitative variables like pCO_2, pO_2, O_2ct, CO_2ct, LA, HCO_3, pH, Hb and PCV, while for qualitative variable like FHR, meconium staining, foetal kicks count values are given as number and percent. Statistical analysis was performed by analysis of variance with significance defined at $p \leq 0.05$.

RESULTS

In this study 40 full term pregnant ladies with asphyxia during labour were compared with 40 full term matched healthy pregnant ladies in labour

Table-1 shows the difference between qualitative variable of the two groups in which 27 (67%) AM showed the abnormal FHR where as 15 (37%) and 17 (42%) AM showed meconium stained liquor and abnormal foetal kick counts respectively .It reveals that these qualitative variables do not have 100% accuracy in early diagnosis of foetal distress.

In Table-2 acid base values are compared among the asphyxiated mothers and control mothers included in the study. Hb and PCV was found to be significantly lower in AM as compared to control while LA and *p*H was statistically significant. While BE, and HCO_3^- did not differ significantly among the two groups.

Levels of Pco_2, Po_2, O_2ct, CO_2ct and $\%O_2SAT$ were analyzed among the asphyxiated mothers in relation to control are given in Table-3.

Among the subjects asphyxiated mother had significantly lower Po_2 and O_2ct, and significantly higher levels of Pco2. The values of CO_2ct, $\%O_2SAT$ in AM were non significant as compared to control.

Table-1: Percentage of qualitative variable among asphyxiated mothers

Variables	Asphyxiated mothers n= 40	Percentage with relation to abnormal values.
FHR<120 or >160 bpm	27	67.5 %
Meconium staining	15	37.5 %
Foetal kick counts <5 or >10/hour	17	42.5 %

*A healthy foetus should move five to 10 times within one hour.[17]

Table-2: Comparison of acid base, haemoglobin (Hb) and haematocrit (PCV) values in between asphyxiated mother (AM) and control mother (CM) groups

Parameters (units)	AM (n=40)	CM (n=40)	*p*-value
LA (mmol/l)	1.676±0.3069	0.993±0.46	<0.02
*p*H	7.442±0.0693	7.414±0.0078	<0.03
HCO_3^- (mmol/l)	20.107±0.657	21.067±0.883	NS
BE (mmol/l)	-4.097±0.391	-4.66±0.4845	NS
Hb (g/dl)	7.363±0.1794	10.59±0.2116	<0.001•
PCV (%)	22.753±0.603	33.207±0.937	<0.001•

All values are given as Mean±SD, •Significant *p*-values, NS= non significant

Table-3: Comparison of mean values of blood gases in between asphyxiated mother (AM) and control mother (CM) groups.

Parameters (units)	AM	CM	*p*-value
Pco_2 (mm Hg)	37.632±2.174	31.643±0.828	<0.01
Po_2 (mm Hg)	95.653±1.631	111.08±3.859	<0.0001•
CO_2ct (mmol/l)	21.667±0.654	21.663±0.79	NS
O_2ct (ml/dl)	9.62±0.251	13.73±0.286	<0.0001•
$\%O_2$SAT	94.257±0.695	94.367±0.4855	NS

All values are given as Mean±SD, •significant *p*-values, NS= non significant

DISCUSSION

The aim of this study was to compare and confirm the alterations of blood gases and acid base levels of asphyxiated pregnant ladies with control mothers during intrapartum period, as mother continuously contributes blood gases through placental exchange the to foetus. An abnormal foetal assessment test is thus a valuable predictor of antepartum foetal asphyxia.

The increased frequency of moderate to severe foetal asphyxia in pregnancy, that is delivered preterm, implies a greater likelihood of long-term morbidity or death[9].If maternal blood is analyzed for ABGs it can be a better predictor for foetal asphyxia especially when it is secondary to maternal conditions.

In our study we did not found any significant difference in $\%O_2$SAT between the two groups. But the Po_2 and O_2ct were significantly lower in AM group as compared to CM group, so this gives a clue that maternal hypoxia is one of the contributing factor for foetal distress.

Haemoglobinemia in pregnancy has been validated as risk factor for maternal mortality and stillbirths.[10] Risk of preterm birth increases in women with low haemoglobin level in first and second trimester.[11]

Anaemic pregnant women who have haemoglobin levels of <11.0 g/dl deliver low birth weight babies with a high mortality rate and with a significant difference in Apgar scores of the new born as compared to non-anaemic pregnant women who have Hb levels >11.0 gm/dl.[12]

Mothers with nutritional or iron deficiency anaemia tend to deliver prematurely with low birth weight babies and a high mortality rate or stillbirths, as compared to non-anaemic mothers.[13]

Our values of Hb concentration and PCV are in agreement with the above studies because the females who had shown the subjective signs of foetal asphyxia as confirmed by ABGs were mostly anaemic. A low Hb and PCV level appears to be favouring foetal distress and lactic acidemia.

pH reference range for adult females is stated by Adams and Hahn, 1988 as 7.35–7.45 while we found 7.414±0.078 in control mothers but this value remains in limits in mothers of asphyxiated baby (AM), i.e., 7.442±0.0693.

Respiratory blood gases and pH values may show variation from normal depending upon exact etiological factors. Maternal, foetal and placental disease may alter the umbilical blood gas measurement. Acidosis may present despite a vigorous neonate with a normal Apgar score.[14]

An increase in Pco_2 indicates that there is respiratory component to acidosis, as in primary respiratory acidosis; there may be decrease in Pco_2 which is a respiratory compensation for metabolic acidosis.[15] Partial pressure of carbon dioxide in arterial blood reflects the adequacy of alveolar ventilation. Arterial Pco_2 is therefore taken to be the same as alveolar Pco_2, normally 40mmHg (range 36–44).[16]

Mothers normally hyperventilate during pregnancy, apparently because of direct stimulatory effect of progesterone on the respiratory centre. This activity lowers the Pco_2 of her blood and cause respiratory alkalosis.[15]

In our result Pco_2 mean value of asphyxiated mother was (37.632) significantly increase than CM group which was appear to be 31.64 mm Hg. This level was lower than range provided by Adams and Hahn,[16] but according to Blechner,[15] this might be due to maternal hyperventilation during pregnancy.

Great care was carried out during selection of subjects so that any underlying metabolic disease of mother and congenital abnormality of foetus must be ruled out which may affect the result. There is significant correlation between hypoxemia and hyperlacticacidemia supporting the concept of reduced oxidative metabolism of lactate. Hyperlacticacidemia is an early biochemical sign of oxygen deficit.[16]

Our study agrees with the above statement because there was considerable hypoxemia and hyperlacticacidemia found in AM group as compared with CM. The value of Pco_2, Po_2 and O_2ct are also quite significant when compared in AM with CM groups and this matches the statement of Westgate *et al*,[17] who stated in 1994 that 40% of the cases with a low arterial *p*H ($p<7.05$) had a respiratory acidosis during labour. During pregnancy and labour the woman hyperventilates and decreases her plasma HCO_3^- but keeps her plasma *p*H constant. She also has a relative anaemia and hypoproteinemia. In this second buffer base is depleted, creating a base deficit of 2–3 mEq/L. This can be expressed as base excess of -2 to -3 mEq/L.[4] In our finding control mothers show mean value of -4.66±0.4895 mmol/l as our pregnant females also have lower level of Hb and PCV than reference ranges, and BE depends on Hb concentration with Pco_2 and HCO_3^- levels.

CONCLUSION

We conclude that routine antepartum blood gases estimation will cover all the cases of maternal asphyxia and in those mothers which are with the sign of subjective foetal asphyxia like meconium staining, decrease foetal kick counts etc, this will be an additive successful diagnostic and therapeutic approach adapted to confirm the severity of the dangerous perinatal conditions which are responsible for high perinatal mortality rate. Greater awareness may eventually leads to the early diagnosis of the problem on authentic grounds, can also tell us the severity of asphyxia and later on helpful in proper management for a better outcome.

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