

ECG changes in birth asphyxia and its correlation with Cardiac troponin-I

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Abstract

Introduction: Cardiac dysfunction is well known in perinatal asphyxia caused by transit myocardial ischemia. Sometimes cardiac dysfunction may be so severe that it can cause congestive cardiac failure and shock that leads to death of newborn. ECG and serum levels of cardiac enzymes can be used to demonstrate impaired myocardial function. **Material and Methods:** It was a case control study conducted in the department of paediatrics, Gandhi Medical College, Bhopal over a period of 12 months from January 2013 to December 2013. Forty asphyxiated full term neonates were taken as cases and 20 healthy full term neonates as controls. Forty neonates with asphyxia admitted to NICU with gestational age >37 completed weeks and with birth weight >2 kg taken as cases and twenty healthy full term neonates (37 completed wks) unasphyxiated weighing >2 kg at birth with clear liquor and 1 min Apgar score >7. **Results:** Myocardial dysfunction was present in 90% of the newborns with severe birth asphyxia and 40% of newborns with moderate birth asphyxia. T wave changes were seen in 80% neonates with severe asphyxia and 33% neonates with moderate asphyxia. In present study, cTn I levels in severely asphyxiated neonates were significantly higher than moderately asphyxiated neonates and control group neonates (4.6 ng/ml, range 2.1 – 7.8, and 1.8 ng/ml, range 0.2 – 4.8 ng/ml and 0.6ng/ml, range 0.2-1ng/ml respectively). **Conclusion:** We found a linear relationship between levels of cardiac troponin-I and birth asphyxia. Therefore cardiac troponin-I level may be useful in predicting the mortality and outcome in perinatal asphyxia.

Key words: Birth Asphyxia, Electrocardiography, Cardiac Troponin I, Neonates.

Introduction

In India, the incidence of perinatal asphyxia is as high as 8.4% (considering the definition of birth asphyxia as Apgar score of < 7 at 1 min) [1]. Perinatal asphyxia is one of the leading cause of neonatal mortality (28%) in our country and it is the most common and important cause of preventable cerebral injury occurring in the neonatal period [2]. Perinatal asphyxia affects almost all organ systems of body. In most of the cases multiple organs are involved but sometimes brain may be the only organ exhibiting dysfunction following asphyxia.

Perinatal asphyxia leading to hypoxic-ischemic encephalopathy (HIE) is a common problem causing multi organ dysfunction including myocardial involvement which can affect the outcome [3]. Sometimes cardiac dysfunction may be so severe that it can cause congestive cardiac failure and shock that leads to death newborn. ECG and serum levels of cardiac enzymes can be used to demonstrate impaired myocardial function.

Cardiac troponins T and I are the preferred markers for myocardial injury as they have the highest sensitivities and specificities for the diagnosis of acute myocardial infarction [4].

Cardiac troponin is a protein released from myocytes when irreversible myocardial damage occurs. It is highly specific to cardiac tissue and accurately diagnoses myocardial infarction with a history of ischaemic pain or ECG changes reflecting ischaemia. Cardiac troponin level is dependent on infarct size, thus providing an indicator for the prognosis following an infarction [5].

Studies of cardiac troponin I (cTnI) in newborns are very limited and less diagnostic because of wide range of serum cTnI I concentrations in newborns. cTnI concentrations > 1ng/ml in asphyxiated newborns may be considered significant.

ECG changes have been linked to the myocardial ischemia in newborns which include generalized T-wave inversion or flattening, ST segment elevation or depression, abnormal Q wave and bundle branch block.

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Material and Methods

It was a case control study conducted in the department of paediatrics, Gandhi Medical College, Bhopal over a period of 12 months from January 2013 to December 2013.

Sample size

Forty asphyxiated full term neonates were taken as cases and 20 healthy full term neonates as controls.

Study group

Forty neonates with asphyxia admitted to NICU with gestational age >37 completed weeks and with birth weight >2 kg.

Inclusion criteria

All cases referred from Sultania Zanana hospital with the diagnosis of HIE bases on clinical history at birth and Sarnat and Sarnat staging [6].

Exclusion criteria

1. Newborns with congestive heart disease
2. Newborns with congenital malformations
3. Metabolic disorders
4. Preterm newborn (>37wks)

Control group

Twenty healthy full term neonates (37 completed wks) unasphyxiated weighing >2 kg at birth with clear liquor and 1 min Apgar score>7.

Clinical examination: A detailed history was taken and complete physical, neurological and cardiovascular examination was done on admission and 48-72 hrs.

Electrocardiogram recording

ECG was recorded around 48-72 hours of age. during ECG recording calibration factor fixed at 1 standardization that indicate 1 mv equal to 10mm for the six limb leads and six precordial leads. Recording speed of paper was 25 mm/sec. The electrocardiogram was recorded using the ECG machine lead I, II, III, avr, avl and avf were recorded in direct sequence using neonatal section electrodes. ECG scores were calculated by using the scoring system developed by R Jedeikin [7].

Method of determining Cardiac troponin I

The SD bioline troponin I rapid test was used. It is rapid immune chromatographic assay for the qualitative detection of cTnI in human blood, serum and heparin plasma as an aid in the diagnosis of myocardial ischemia. Two ml of whole blood is collected in collection vial by venopuncture under aseptic precaution and then centrifuge to get plasma specimen by using dropper. Specimen is taken up to the fill line, then specimen is added into the sample well of the test device and result read at 15 minutes.

Results

The present study included 60 subjects of which 40 were cases of perinatal asphyxia of moderate to severe degree and 20 were healthy neonates without any evidence of asphyxia as controls. The asphyxiated cases were further divided into two groups on the basis of severity of hypoxic ischemic encephalopathy (HIE). Group A (HIE III) had 10 cases and group B (HIE II) had 30 cases, group C served as controls.

Table No. 1: Distribution of the newborns into various groups

Category	Degree of Asphyxia	No. of Neonates	Sex		Group
			Male	Female	
HIE staging III	Severe	10(25%)	7(70%)	3(30%)	A
HIE staging II	Moderate	30(75%)	21(70%)	9(30%)	B
Normal	Nil	20	13(65%)	7(35%)	C

Table No 2: Summary of clinical manifestation as evidence of myocardial dysfunction in birth asphyxia

Myocardial Dysfunction	Group A(n=10)	Group B(n=30)
Shock	2	4
Respiratory distress	4	7
Murmur	2	1
CCf	1	0
Total	9	12

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Myocardial dysfunction was present in nine (9) newborns with severe birth asphyxia and twelve (12) newborns with moderate birth asphyxia.

Table No 3: Summary of ECG changes in birth asphyxia

Group	Abnormal Q wave	Abnormal T wave	Abnormal ST segment	Prolonged QRS	Bundle branch block
Asphyxiated	0	18	6	1	1
Control	0	9	0	0	0

Table No 4: Jedeikins grading versus HIE cross tabulation

Jedekins grading	HIE grading		Total
	II	III	
I	3	0	3
II	7	4	11
III	1	4	5
IV	0	1	1
Normal	19	1	20
Total	30	10	40

Cardiac Troponin I (cTn I)

In present study, cTn I levels in severely asphyxiated neonates were significantly higher than moderately asphyxiated neonates and control group neonates (4.6 ng/ml, range 2.1 – 7.8, and 1.8 ng/ml, range 0.2 – 4.8 ng/ml and 0.6ng/ml, range 0.2-1ng/ml respectively, P value <0.005)

Discussion

In our study, CVS was the most common organ system to suffer from dysfunction (52.5%). Similarly in a study conducted by P.S. Rajkumar et al. in 2005, cardiovascular dysfunction was seen in 70% cases [8]. P. Shah et al. also studied 130 newborns with birth asphyxia and reported cardiovascular dysfunction in 62% cases [9].

Electrocardiographic Evaluation

In the present study no significant abnormality of heart rate and rhythm was observed in asphyxiated babies. T wave changes were seen in 80% neonates with severe asphyxia and 33% neonates with moderate asphyxia. Thus the overall incidence was 45% in asphyxiated babies.

Control group had grade I ECG changes in 45% cases. Mean axis was 128, which is within normal limit. Prolonged QRS duration was seen in one severely asphyxiated baby. The incidence of RBBB was 10% in severely asphyxiated babies. No one in the study group showed LBBB. Q wave was normal in all cases. Abnormal ST segment was seen in 50% neonates with severe asphyxia and 33% neonates with moderate asphyxia.

Thus the overall incidence was 15%, so according to Jedekins criteria grade I ECG changes were present in 3

(7.5%) cases, grade II in 11 (27.5%), grade III in 5 (12.5%) and grade IV in 1 (2.5%) cases.

I Barberi et al. reported grade II ECG changes in 100% cases with moderate asphyxia, grade II in 38.5%, grade III in 38.5% and grade IV in 23% cases with severe asphyxia. Control group had 41% cases with grade I changes that were similar to our present study [10]

Esra Kanik et al. also reported ECG changes in asphyxiated neonates where ECG changes increased with severity of asphyxia [11].

Cardiac Troponin I (cTn I)

In present study, cTn I levels in severely asphyxiated neonates were significantly higher than moderately asphyxiated neonates and control group neonates (4.6 ng/ml, range 2.1 – 7.8, and 1.8 ng/ml, range 0.2 – 4.8 ng/ml and 0.6ng/ml, range 0.2-1ng/ml respectively, P value <0.005). Gulcan turker et al. reported that infants with hypoxia had significantly higher cord blood cTnI levels than normal newborns and in non survivors cord blood cTn I levels were significantly higher than survivors (5.9 ng/ml, range 2.1 – 12.8, and 1.6 ng/ml, range 0.4 – 5.8 ng/ml respectively) [12].

Research Article

Esra Kanik et al. studied that serum cTn I level was significantly higher in newborns with birth asphyxia [11]. Danicle Trevisanoto et al. concluded that in asphyxiated neonates, cTn I concentrations were higher with respect to healthy neonates, suggesting the presence of myocardial damage in this group of high risk infants, cTn I did not correlate with traditional marker of asphyxia [13].

Conclusion

It has been well established that troponin-T is elevated in myocardial ischemia in perinatal asphyxia. Cardiac troponin-I is specific and sensitive in the diagnosis of cardiac dysfunction in perinatal asphyxia. Mean cardiac troponin-I level in cases with cardiac dysfunction was found significantly higher in cases with severe asphyxia. We found a linear relationship between levels of cardiac troponin-I and birth asphyxia. Therefore cardiac troponin-I level may be useful in predicting the mortality and outcome in perinatal asphyxia.

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