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Relations Between Umbilical Troponin T Levels And Fetal Distress.

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Abstract:

Background: one of the most important neonatal morbidity during labor is Perinatal asphyxia. Hypoxia causes release troponin from cardiac muscles. Fetal distress during labor may be detected by monitoring the fetal heart rate. Elevated levels of troponin T in cord blood may be associated with intrauterine hypoxia.
Aim: Relations between umbilical troponin T levels and fetal distress
Methods: Cord blood samples were collected from 80 neonates and analyzed. Data on birth weight, sex, APGAR scores, and mode of delivery were recorded.
Results: a total of 80 samples were collected, 40 samples from infants with fetal distress and 40 samples from infants without fetal distress. The gestational age of these infants ranged from 38 to 40 weeks and birth weight ranged from 2.5 to 4 kg. There was no relation between umbilical troponin T levels and mode of delivery. Fetuses with distress had significantly higher cord troponin T levels than control group (26/42 versus 50/46 µg /ml respectively; $p < 0.01$).
Conclusions: Troponin T levels in the cord blood are unaffected by mode of delivery. Infants with distress had significantly higher cord cardiac troponin T levels, suggesting that troponin T may be a useful marker for early detection of hypoxia in neonates.

Keywords: umbilical cord; troponin T, fetal distress

Introduction:

Despite significant advances in obstetrics and neonatology, asphyxia remains one of the main causes of perinatal mortality and morbidity.[1]

Cardiac troponin has a major role in screening and diagnosis of myocardial ischemia in adults and children. Their introduction has redefined the diagnosis of myocardial infarction in adults and provided valuable prognostic information. Cardiac troponin is released into circulation in response to ischemic cardiac injury. In the pediatric population, troponin shows a good correlation with the extent of myocardial damage following cardiac surgery and cardio toxic medication, and can be used as predictors of subsequent cardiac recovery and mortality. They may serve as a useful adjunct in the assessment of the magnitude of myocardial injury in respiratory distress syndrome and asphyxia.[2]

Several studies have investigated biochemical tissue specific injury markers in severe asphyxia in cord blood. Studies on the concentration of cardiac troponin in healthy term neonates, hypoxemic neonates, and small for gestational age infants have been published in the last few years.[1] Troponin T seems to be a useful marker of myocardial injury in birth asphyxia and cardiac troponin T is measurable in fetal life in pregnancies with signs of fetal chronic hypoxia.[3]

On the other hand fetal distress during labor may be detected by monitoring the fetal heart rate.[4, 5] Elevated levels of Cardiac troponin T in cord blood may be associated with intrauterine hypoxia.[1] Current fetal monitoring may be insufficient to capture prenatal stresses that result in neonatal myocardial injury.[6] On the other hand origin umbilical troponin is neonatal not maternal So it can be used for assessment fetal hypoxia.[7]

The present study was designed to analyze the association between cardiac troponin T concentrations in umbilical cord with perinatal fetal distress.

Method and Material:

In this cross sectional study troponin level measured in umbilical cord after birth in 80 samples.

Inclusion criteria for mothers were singleton pregnancies, absence of infection, fever, gestational age between 37-40 weeks. Inclusion criteria for neonates were birth weight between 2/5-4 kg. Neonates with congenital anomalies or cardiac diseases or IUGR were excluded from the study

Newborns were divided in two groups: 40 Newborns with fetal distress and 40 consecutive Newborns without fetal distress during labor

During labor fetal cardiac monitoring was done. Criteria for fetal distress include fetal cardiac rate less than 110, fetal cardiac rate more than 160, late deceleration, variable deceleration and amniotic fluid with thick meconium. Late deceleration, an intermittent or persistent drop in the baseline fetal heart rate of <100 beats per minute unrelated to uterine contractions

SAMPLE COLLECTION AND ANALYSIS: After birth, a 10 cm segment of the umbilical cord was double-clamped and approximately 1 ml of arterial blood was obtained for PH and blood gas. A second 5 ml sample of umbilical blood was col-

lected for troponin measurement with chemiluminescence method.

The first blood sample was immediately sent to laboratory and analyzed within 10 min of collection.

Sample obtained from the umbilical remnant was collected for troponin T over a three months period. The local university research ethics committee approved this study without the requirement for parental consent. A cord blood gas analysis is performed on all neonates Shahid Akbarabadi Women's Hospital.

SUBJECTS: We compared birth weight, sex, APGAR scores and mode of delivery, troponin level, and cord acid/base status between case and control groups. APGAR equal or less than 7 is viewed as low APGAR. We intended to establish a relation between troponin level and fetal distress.

STATISTICAL ANALYSIS: In two groups, the relation between cord cardiac troponin T levels and various variables was investigated using q2, independent sample T test. Statistical analysis was performed using SPSS 16.

Results:

A total of 80 cord blood cardiac troponin T samples were analyzed; 40 of them were collected from infants who were categorized as having fetal distress including 18 cases of fetal tachycardia, 20 cases of thick meconium passage in amniotic fluid, 22 cases of late deceleration . 48 neonates were female and 32 neonates were male. There was no significant difference in cardiac troponin T levels between male and female.

Table 1 shows the patient characteristics and cardiac troponin T levels for infants.

Table 1 Distributions of variables between two groups

	Fetal distress	No fetal distress
Total number of infants	40	40
Maternal age	30, 60(7.47)	28, 55 (6.04)
Birth weight (kg)	3.19 (89/356)	3.18 (30/8)
Caesarean section	31(75%)	22 (55%)
APGAR at 5 minutes= or < 7	19	3
Arterial cord pH	7.16 (SD=0/5)	7.28 (SD=0/5)
Cardiac troponin T (µg /ml)	46/05 (SD=11/18)	26/42 (SD=4/61)

There was significant relation between cardiac troponin T level and fetal distress. (P value<0/01) (table 1) .

There was significant relation between cardiac troponin T level and umbilical PH. (p = 0.002) (table 1).

There was no significant relation between cardiac troponin T level and birth weight (p value = 0.08). Table 1

There was no significant difference in cardiac troponin T levels when vaginal

delivery was compared with caesarean section. (P value =0.11) table 2.

Table 2- comparison troponin levels between normal vaginal delivery and caesarian section

Type of delivery	number	Mean	SD	Minimum	Maximum
NVD	27	22/25	9/67	17/80	59/60
C/S	53	35/38	13/83	17/10	67/60

There was significant relation between cardiac troponin T level and low APGAR score (p = 0.002) table 3.

Table 3- comparison troponin levels between neonates with normal and low APGAR

	number	Mean	SD	Minimum	Maximum
Neonates with Low APGAR	22	43/29	10/53	22/90	67/60
Neonates with Normal APGAR	58	33/29	12/83	17/10	67/20

Discussion:

Infants with fetal distress had cardiac troponin T levels that were significantly higher than those of without fetal distress. This result is consistent with Zaramella,Clark , turker and Rossely studies'.[3, 8,9,10]

Zaramella showed Troponin I was higher in the asphyxiated or depressed group than in controls .Troponin I is a useful shortterm index of birth asphyxia or perinatal respiratory depression. [3]

Clark measured troponin level in umbilical cord of 242 healthy neonates. Troponin level in 27 neonates with respiratory distress was higher than healthy neonates.[8]

Turker showed that troponin level in umbilical cord together with CK and CK-MB elevated in hypoxic infants compared to normal infants. Therefore troponin level may be an indicator for perinatal hypoxia in neonates [9]. Roselly showed that cardiac troponin T is a marker of subclinical cardiomyocyte injury even in the presence of cardiac function recovery. He showed troponin of umbilical cord in chronic hypoxia was elevated.[10]

In our study infants with fetal distress had lower PH levels that were significantly different from those of without fetal distress. (P value = 0.002) .this is consistent with Morrison' study who showed neonates who had lower PH, had higher troponin level.[11]

In our study there was no significant relation between cardiac troponin T level and birth weight, mode of delivery. This result was seen in Morisson ,Trevisanuto and Turker , studies'.[7, 9,11]

In our study there was significant relation between cardiac troponin T levels and APGAR score. In low APGAR group cardiac troponin T level was higher relation to normal APGAR group. This is consistent with Costa and Gua studies'. Costa showed that asphyxiated babies have significantly highercardiac troponin T lev-

els when compared to control healthy newborn infants. [2] Gua showed Serum umbilical troponin was negatively correlated to 1 min and 5 min APGAR scores.(11)

Our result not consistent with Trevisanuto and Lipshultz study. Trevisanuto studied normal neonates with normal APGAR. In Lipshultz study there were three neonates with low APGAR and samples were few.

In summary we conclude that umbilical troponin T increased in fetal distress. Troponin T measurements may be useful in early identification of infants with asphyxia.

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