

Cardiac Troponin T Levels of Umbilical Cord in Neonates with Abnormal Fetal Heart Rate

Rafati, Shiva¹, MD; Rabi, Maryam^{*2}, MD; Borna, Hajie¹, MD

1. Department of Pediatrics, Faculty of Medicine, Shahed University, Tehran, Iran
2. Department of Gynecology, Mustafa Khomeini Hospital, Shahed University, Tehran, Iran

Received: Mar 05, 2012; Accepted: Jun 16, 2012; Available Online: Dec 24, 2012

Abstract

Objective: Most neonatal encephalopathic disorders appear to be caused by perinatal events. Persistent myocardial ischemia leads to cellular necrosis and release of troponin from cardiac muscles. Fetal distress during labor may be detected by monitoring the fetal heart rate. However little is known about the relationship, if any, that exists between fetal heart rate abnormalities and the fetal cardiac musculature and its function. The aim of this study was to investigate the relationship, if any, of umbilical cord serum levels of cardiac troponin T with fetal bradycardia or late deceleration.

Methods: In this cross sectional study, troponin T level in umbilical cord blood of 80 neonates are measured. There were 23 versus 57 fetuses with and without late deceleration or bradycardia.

Findings: Level of cardiac troponin T in umbilical blood of neonates with fetal bradycardia or late deceleration was elevated in comparison to neonates without bradycardia or late deceleration. There was no relation between umbilical troponin T level and mode of delivery.

Conclusion: Infants with fetal bradycardia or late deceleration during labor had significantly higher cord cardiac troponin T levels. If troponin level is normal, the probability of hypoxia will be very low.

Iranian Journal of Pediatrics, Volume 23 (Number 1), February 2013, Pages: 85-88

Key Words: Umbilical Cord; Cardiac Troponin T; Fetal bradycardia; Late Deceleration

Introduction

Hypoxic-ischemic encephalopathy is an important cause of permanent damage to central nervous system tissue that may result in neonatal death or cerebral palsy or developmental delay. Most neonatal encephalopathies appear to be due to perinatal events. Fetal distress during labor may be detected by monitoring the fetal heart rate^[1].

Cardiac troponin is released into circulation following ischemic cardiac injury. Cardiac troponin has a major role in screening and diagnosis of myocardial ischemic injury in

adults^[2].

Previous studies showed that elevated cardiac troponin T (cTnT) levels of umbilical cord blood may be associated with intrauterine hypoxia^[3]. Other studies showed that troponin I is a useful marker of myocardial injury in birth asphyxia^[4]. However little is known about the relationship, if any, that exists between fetal heart rate abnormalities and the fetal cardiac musculature and its function^[5].

The purpose of this study was to compare cTnT levels of umbilical cord blood postnatal in neonates with and without fetal bradycardia or

* Corresponding Author:

Address: Department of Gynecology, Mustafa Khomeini Hospital, Italia St., Faculty of Medicine, Shahed University, Tehran, Iran

E-mail: dr-rabi-maryam@shahed.ac.ir

© 2013 by Pediatrics Center of Excellence, Children's Medical Center, Tehran University of Medical Sciences, All rights reserved.

Table 1: Mean and SD of cTnT levels (pg/ml) of umbilical cord between neonates with and without fetal bradycardia or late deceleration

Fetal Heart Rate	Number	Mean cTnT (SD)	Minimum cTnT	Maximum cTnT
Fetal bradycardia or late deceleration	23	45.82 (11.59)	22.90	67.60
Without fetal bradycardia or late deceleration	57	32.12 (11.43)	17.10	59.60

cTnT: cardiac troponin T; pg: Picogram; SD: standard deviation

late deceleration during labor.

Subjects and Methods

Umbilical cord blood samples of 80 neonates whose mothers referred to hospital, during a three-month period were examined. The live born neonates of singleton pregnancies with intact membrane, and no infection or fever, having a birth weight between 2.5-4 kg were included. Premature neonates and those with IUGR, and/or congenital anomalies and cardiac diseases were excluded from the study.

Newborns were divided into two groups: group A consisted of 23 newborns that had late deceleration or bradycardia during labor; group B consisted of 57 newborns who had not late deceleration or bradycardia during labor.

Subjects in group A had an abnormal fetal heart rate recording during labor in the last hour prior to delivery as determined by the presence of any of the following: late deceleration or bradycardia, i.e., an intermittent or persistent drop in the baseline fetal heart rate of less than 100 beats per minute unrelated to uterine contractions^[6].

Those in group B had fetal heart rate recordings without late deceleration or bradycardia in the last hour prior to delivery.

After birth, a 10 cm segment of the umbilical cord was double-clamped and approximately 5 ml

of blood was obtained for cTnT measurement with chemiluminescence method (pg/ml).

Sample obtained from the umbilical cord remnant was collected for cTnT. The local university research ethics committee approved this study.

We compared cTnT levels according to fetal bradycardia or late deceleration, Apgar scores, mode of delivery. Apgar less than seven is viewed as low Apgar.

Statistical analysis was performed with chi square test for categorical variables and the two tailed t test for continuous variables. SPSS 16 was used for Statistical analysis. We considered differences significant at $P < 0.05$.

Findings

We compared cTnT level of umbilical cord blood between 32 male and 48 female neonates with and without fetal bradycardia or late deceleration and found statistically significant differences ($P=0.001$) (Table 1).

Comparing birth weight between the two groups, there was no significant difference (3.18 versus 3.19, $P=0.08$). There were 23 neonates with low Apgar scores (1 min < 7) and 47 neonates with normal Apgar scores. Here the difference in cTnT levels between the two groups was significant ($P=0.002$) (Table 2).

Table 2: comparison cTnT(pg/ml) levels of umbilical cord between neonates with normal and low Apgar scores

Apgar scores	Number	Mean of cTnT (SD)	Minimum cTnT	Maximum cTnT
Neonates with low Apgar	23	43.29 (10.53)	22.90	67.60
Neonates with normal Apgar	57	33.29 (12.83)	17.10	67.20

cTnT: Cardiac troponin T; pg: Picogram; SD: standard deviation

Table 3: Comparison of cTnT (pg/ml) levels of umbilical cord between neonates delivered by NVD and C/S

Type of delivery	Number	Mean cTnT (SD)	Minimum cTnT	Maximum cTnT
Normal vaginal delivery	27	22.25 (9.67)	17.80	59.60
Cesarean section	53	35.38 (13.83)	17.10	67.60

cTnT: Cardiac troponin T; pg: Picogram; SD: standard deviation

Fifty-three neonates were delivered by caesarian section and 27 neonates through vaginal delivery. We found no significant difference in cardiac troponin T levels when vaginal delivery was compared with cesarean section ($P=0.1$) (Table 3).

Discussion

The purpose of this study was to compare cTnT levels of umbilical cord blood postnatal in neonates with and without fetal bradycardia or late deceleration during labor.

The results indicated that troponin T levels increased in fetuses with fetal bradycardia or late deceleration. Fetal bradycardia or late deceleration in labor may have impact on cardiac myofibrillary function. Fleming et al demonstrated that troponin I did not correlate with abnormal fetal heart rate^[5]. Other studies had investigated troponin T or I levels in birth asphyxia. Our results are consistent with those of other studies^[4,7-9]. Zaramella et al showed that Troponin I was higher in the asphyxiated or depressed newborns^[4]. Zecca et al found that asphyxiated neonates had significantly higher cTnT levels when compared to control healthy newborn infants^[7]. Turker stated that troponin I level in umbilical cord blood as well as creatine kinase and CK-MB were elevated in hypoxic infants compared to normal infants. Therefore troponin I may be an indicator for perinatal hypoxia in neonates^[8]. Roseli showed that umbilical cord cTnT levels were elevated when there was absent or reversed end diastolic flow velocity in the umbilical artery in compromised fetuses with placental insufficiency^[9].

Our results are in contrast with those of Fleming et al who found no correlation between abnormal fetal heart rate and troponin I levels in umbilical cord blood. This difference might be

explained by the fact that in Fleming's study fetuses with pulse rates more than 160 were included in case group but in our survey only fetuses with pulse rates less than 100 or late deceleration were studied.

The study was also intended to examine the probable relation between cTnT levels and Apgar score. Umbilical cord blood cTnT level was negatively correlated to Apgar scores. The results were consistent with findings of other studies^[7,10]. Our results were in contrast with those of Trevisanuto et al^[11] and Lipshultz et al^[12]. This difference may be explained by the fact that in Lipshultz study there were only two neonates with low Apgar and sample size was limited. Trevisanuto and Lipshultz studied normal neonates with normal Apgar^[11,12].

We examined also the possibility of association of cTnT levels with mode of delivery. The results indicated that there was no significant relation between cardiac cTnT levels and mode of delivery. Our results were consistent with those of other studies^[2,12-14]. Yet our results were in contrast to those of Kocylowski et al who found higher cTnT in vaginal delivery versus cesarean section^[15].

Our study had one limitation. Because of ethical considerations in relation to blood sampling in the neonatal period it was difficult to acquire more blood samples for analysis.

We recommend further studies using a larger sample to investigate the relation between cTnT levels and fetal bradycardia and late deceleration.

Conclusion

Infants with fetal bradycardia or late deceleration during labor had significantly higher cord cardiac troponin T levels. On the other hand, with normal troponin level, the probability of hypoxia would be very low.

Acknowledgment

We kindly appreciate Shahed University for financial support. In particular, we thank Dr. Davati, Dr Jalali, Dr Yosefi, for their cooperation. We are also grateful to the Mustafa Khomeini and Shahid Akbarabadi Hospitals staffs.

Conflict of Interest: None

References

1. Stoll BJ, Chapman IA. The fetus. In: Kliegman, Behrman, Jenson (eds). Nelson textbook of Pediatrics. 19th ed. Philadelphia: Saunders. 2011; P: 569
2. Alexandre SM, Almedia D, Guinsburg R, et al. Cord blood cardiac troponin I, fetal Doppler velocimetry, and acid base status at birth. *Int J Gynecol Obstet* 2008; 100(2):136-40.
3. Clark SJ, Newland P, Yoxall CW, et al. Cardiac troponin T in cord blood. *Arch Disease Child Fetal Neonatal* 2001; 84(1):F34-7.
4. Zaramella P, Saraceni E, Federica Freato F, et al. Can tissue oxygenation index (TOI) and cotside neurophysiologic variables predict outcome in depressed/asphyxiated newborn infants? *Early Hum Dev* 2007; 83(8):483-9.
5. Fleming SM, O'Gorman T, O'Byrne L, et al. Cardiac Troponin I and N-terminal pro-brain natriuretic peptide in umbilical artery blood in relation to fetal heart rate abnormalities during labor, *Pediatr Cardiol* 2001; 22(5):393-6.
6. Cunningham FG, Leveno KJ, Bloom SL, et al. Prenatal diagnosis and fetal care. In: Williams Obstetrics. 23rd ed. New York, NY McGraw-Hill: 2010; Pp: 415-28.
7. Zecca E, Rosa G, Luca D, et al. Is serum troponin T a useful marker of myocardial damage in newborn infants with perinatal asphyxia? *Acta Paediatr* 2007;96(2):181-4.
8. Türker G, Babao lu K, Duman C, et al. The effect of blood gas and Apgar score on cord blood cardiac troponin. *J Maternal Fetal Neonatal Med* 2004; 16(5):315-9.
9. Nomura RM, Cabar FR, Costa VN, et al. Cardiac troponin T as biochemical marker of cardiac dysfunction and ductus venosus Doppler velocimetry. *Eur J Obstet Gynecol Reprod Biol* 2009;147(1):33-6.
10. Chao G, Ling Y, Jun W. Role of pH value of umbilical artery blood in neonatal asphyxia. *Zhongguo Dang Dai Er Ke Za Zhi* 2009; 11(7):521-4. [In Chinese]
11. Trevisanuto D, Pitton M, Altinier S, et al. Cardiac troponin I, cardiac troponin T and creatine kinase MB concentrations in umbilical cord blood of healthy term neonates. *Acta Paediatr* 2003; 92(12):1463-7.
12. Lipshultz SE, Simbre VC, Hart S, et al. Frequency of elevations in markers of cardiomyocyte damage in otherwise healthy newborns. *Am J Cardiol* 2008; 102(6):761-6.
13. Morrison J, Grimes H, Fleming S, et al. Fetal cardiac troponin I in relation to intrapartum events and umbilical artery pH. *Am J Perinatol* 2004; 21(3):147-52.
14. Bader D, Kugelman A, Lanir A, et al. Cardiac troponin I serum concentrations in newborns: A study and review of the literature. *Clin Chim Acta* 2006; 371(1-2):61-5.
15. Kocylowski R, Dubiel M, Gudmundsson S, et al. Biochemical tissue-specific injury markers of the heart and brain in postpartum cord blood. *Am J Obstet Gynecol* 2009; 200(3):273e1-e25.