ORIGINAL ARTICLE

Comparison of serum cardiac troponin- I and creatine kinase MB isoenzyme concentrations in asphyxiated neonates

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ABSTRACT

Objective

To assess the correlation of signs of myocardial damage to serum cardiac troponin I (cTnI) and creatine kinase MB isoenzyme (CK-MB) concentrations.

Material- Methods

Blood samples were collected from 25 asphyxiated term neonates and 25 controls at 12h of age by immunoassay. The asphyxiated neonates were followed up until discharge or death.

Results

Asphyxiated neonates had significantly higher concentrations of cTnI and CK-MB than controls (P<0.001). Serum cTnI concentrations were significantly higher in asphyxiated neonates who developed hypotension, heart failure or those with low ejection fraction (P<0.01). Serum cTnI concentrations were significantly higher in asphyxiated who died than those who survived (P<0.01). There was no significant difference in serum CK-MB mass concentrations between asphyxiated neonates with and without these complications.

Conclusions

Unlike CK-MB, serum cTnI concentrations were significantly higher in asphyxiated neonates who died or developed cardiac dysfunction.

Keywords: *Troponin I, perinatal mortality, asphyxia neonatorum, creatine kinase/MB form, cardiac failure.*

INTRODUCTION

Perinatal asphyxia, defined as an impairment of exchange of respiratory gases during the perinatal period, results in hypoxic and/or ischemic injuries to various organs of the newborn.[1,2] When myocardial perfusion is compromised, the papillary muscles and subendocardial areas of the neonatal heart are particularly vulnerable,[3] and myocardial ischemia and infarction are reported to be relatively common in asphyxiated neonates.[4] However, different clinical pictures are associated to neonatal myocardial ischemia.[5]

Electrocardiogram (ECG) is not so helpful in neonates as it is in adults.[6] Newborns do not manifest a classical pattern of ECG changes representative of asphyxiated myocardium. The pathological Q-waves in adults with myocardial infarction are almost non-existent in these neonates.[7] Echocardiography (EchoCG) changes suggestive of myocardial damage are present only when the damage is severe.[8] On the EchoCG ischemia is manifested by poor left ventricular function and mitral or tricuspid value incompetence.[9]

Troponin is an inhibitory protein complex located on the actin filament in all striated muscle and consists of three subunits T, C and I. The cardiac troponins T (cTnT) and I (cTnI) are currently used as biochemical markers for myocardial ischemic changes in adults, but their clinical significance in neonates is still questioned.[10]

Elevated cTnT levels have been detected in newborn and in different perinatal conditions such as placental insufficiency, birth asphyxia and respiratory distress syndrome.[11] An elevation in cord blood cTnI has been shown to be a predictor of severe perinatal hypoxia and no detectable concentrations in healthy term neonates.[12]

Serum Creatine Kinase MB isoenzyme (CK-MB) mass concentration is a biochemical marker of neonatal myocardial damage.[13] The main disadvantage of CK-MB is its lack of cardiac-specificity in children below 4 years of age. Gestation, sex, modes of delivery, and birth weight may affect CK-MB levels. [14]

Our aim was to investigate the usefuleness of cardiac troponin I (cTnI) and Creatine Kinase MB (CK-MB) in the diagnosis of myocardial injury due to birth hypoxia.

PATIENTS AND METHODS

This was a prospective observational study carried out in the labour room and neonatal intensive care unit (NICV) of Cairo University Hospital over a 6 month period from September 2007. This study was approved by the Scientific and Ethics Committee of Cairo University.

Twenty five term neonates (gestational age from 37 to <42) were eligible for the study. The inclusion criteria of perinatal asphyxia consisted of at least two of the following features: (i) evidence of fetal distress as indicated by thick meconium-stained liquor and/or abnormal cardiotocographic changes (sustained fetal bradycardia, late deceleration) (ii) Apgar score ≤ 3 at 1-min and ≤ 6 at 5 min (iii) an umblical

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