Editorial

Assessment of Myocardial Function in Birth Asphyxia: Editorial

Patel U[1], Gedam DS[2]

[1]Dr Umesh Patel, Associate Professor in Pediatrics & [2]Dr D Sharad Gedam, Associate Professor in Pediatrics. Both are affiliated with L N Medical College, Bhopal, India & members of Editorial board, IJMRR.

Address for correspondence: Dr Umesh Patel, Email: drumeshpatel@gmail.com

Abstract

Perinatal Asphyxia is leading cause of Neonatal Mortality. Neurological manifestations are predominant in form of abnormal movement and seizures. It involves almost all body system but cardiac functions are not commonly studied.

Perinatal asphyxia is a common problem with the incidence varying from 0.5 – 2% of live births[1]. It is an important cause of admission to neonatal intensive care units (NICU) with multi organ dysfunction[2]. When an asphyxic event occurs, it leads to a series of physiological mechanisms in order to preserve the function of vital organs (especially brain and heart), whereas other organs such as the kidneys, gastrointestinal tract, and skin are affected to a varying degree based on the duration of the episode[3]. However, in spite of compensatory mechanisms, it may progress to hypoxic- ischemic encephalopathy (HIE) involving the brain and heart[4].

The incidence of clinical cardiac dysfunction in perinatal asphyxia varies from 24 – 31% [5,6]. Affection of the heart after asphyxia is usually part of a multi-organ involvement but isolated cardiac events also founded. In birth asphyxia myocardial injury can occur in both ventricles. The reduced myocardial performance and cardiac output following perinatal asphyxia may significantly complicate perinatal management and may contribute to increase end-organ damage and mortality. Even though this is generally a transient effect it can result in cardiogenic shock and death.

Apart from the clinical presentation, electrocardiography (ECG), echocardiogram, Doppler and determination of cardiac enzymes are useful tools to detect myocardial involvement in up to two third of affected infants. In contrast to adults, recognition of myocardial ischemia is far more difficult in neonates.

The various clinical features related to cardiac dysfunction are respiratory distress, congestive cardiac failure, hypotension, peripheral circulatory failure, cardiogenic shock and systolic murmur (usually pan-systolic because of mitral regurgitation and tricuspid regurgitation)[7] . Respiratory distress is the most important clinical feature of hypoxic heart damage. All neonates with clinical evidence of asphyxia should be evaluated for myocardial injury by clinical hemodynamic evaluation and the use of biomarkers for myocardial damage (e.g., troponin). If there is no clinical evidence of cardiovascular compromise and no elevation of biomarkers, echocardiography is unlikely to be useful. If there are clinical manifestations suggesting poor end-organ perfusion, comprehensive echocardiography may be helpful for identifying possible underlying structural or functional heart disease. If abnormalities are detected, standard targeted neonatal echocardiography (TNE) can be used to monitor functional recovery and the hemodynamic effects of treatment.

Strain and Strain-Rate by tissue Doppler are novel indices of myocardial function. Strain is the relative change in length of the myocardial wall and Strain-Rate is Strain per unit of time. The conventional index of myocardial function in neonates is the Fractional Shortening, the relative change in the diameter of the left ventricle. Birth asphyxia can lead to impaired myocardial function, not always detected by Fractional Shortening. In tissue doppler we can assess-

1. Peak Systolic Strain-Rate (The maximal rate of relative shortening of the ventricle wall during the ventricle systole, which is usually lower after birth asphyxia)
2. Early Diastolic Strain-Rate (The maximal rate of relative lengthening of the ventricle wall in the early phase of the ventricle diastole, which is usually lower after birth asphyxia)
3. Strain-Rate during Atrial Systole (The maximal rate of relative lengthening of the ventricle wall in the late phase of the ventricle diastole, which is usually lower after birth asphyxia).
4. Peak Systolic Strain (The maximal relative shortening of the ventricle wall during the ventricle Systole, which is usually lower after birth asphyxia)
5. Fractional Shortening (The relative shortening of the diameter of the left ventricle lumen during the ventricle systole)

The new myocardial function indices were more sensitive than the conventional index of myocardial function for assessing the impaired function in asphyxiated term neonates.

Biochemical markers like CK total level, CK MB and troponin I levels are elevated in birth asphyxia indicate heart muscle damage. These enzyme levels shows significant rise with increasing severity of HIE; indicating more myocardial ischemia in severe HIE. The measurement of cardiac troponin-I may have a role in the early identification of neonates with myocardial damage secondary to ischemia. These enzymatic indicators often associated with electrocardiographic changes. However, cardiac abnormalities often are under diagnosed and require a high index of suspicion[9].

In the ECG there may be 'T' wave inversion (indicate significant myocardial ischemia), 'T' wave flattening, transient AV block and arrhythmias[7]. Third degree AV block and ventricular arrhythmias is associated with increased risk of death in first few weeks of life. In some newborn there may be "fixed heart rate" frequently associated with low apgar score or "lack of sinus rhythm" shown to follow diminished uteroplacental perfusion.

In echocardiography there are a lot of changes occurs like ventricular hypokinesia, poor myocardial contractility, cardiomegaly secondary to AV valve insufficiency (eg- MR and TR), left to right shunting across a patent ductus arteriosus often occurs in the setting of respiratory distress syndrome[7].

Myocardial ischemia also identified by thallium uptake, which shows poor uptake in affected regions of the myocardium. Foci of myocardial necrosis have been demonstrated histologically, as have coronary artery intimal thickening and intravascular thrombi. The papillary musculature seems particularly susceptible to ischemic damage.

Myocardial dysfunction secondary to perinatal asphyxia is more frequent than thought, for which it will be useful to submit asphyxiated neonates to ECG monitoring and assay of cardiac enzyme markers complemented with clinical findings[9].

A significantly increased cardio-thoracic ratio in an asphyxiated infant should alert the radiologist and pediatrician to the possibility of such a disorder. The early detection and prompt treatment of condition will help in improving prognosis of these asphyxiated newborns.

Goel et al[10] demonstrated ECG & Echocardiographic changes in her article in this issue. She could have done some biochemical analysis with marker like CK total level, CK MB and troponin I levels. Their elevated level in birth asphyxia indicates heart muscle damage. More studies are needed to study effect of birth asphyxia in various organ functions.

References

1. Adcock LM, Papile LA: Perinatal asphyxia. In Manual of neonatal care. 6th edition. Edited by Cloherty JP, Eichenwald EC, Stark AR. New Delhi: Wolters Kluwer; 2008:518–523.

2. Shah P, Riphagen S, Beyene J, Perlman M: Multiorgan dysfunction in infants with post-asphyxial hypoxic-ischaemic encephalopathy. Arch Dis Child Fetal Neonatal Ed 2004, 89:F152–F155.

3. Pasternak JF: Hypoxic-ischemic brain damage in the term infant. Pediatr Clin North Am 1993, 40:1061–1071.

4. Tapia-Rombo CA, Carpio-Hernandez JC, Salazar-Acuna AH, AlvarezVazquez E,Mendoza-Zanella RM, Perez-Olea V, et al: Detection of transitory myocardial ischemia secondary to perinatal asphyxia. Arch Med Res 2000,31:377–383.

5. Dubin. Arrhythmias in the Newborn. *Neoreviews* 2000; 1:146-151.

6. Kothari DS, Skinner JR Neonatal tachycardias: an update.*Arch Dis Child - Fetal Neonatal* 2006; 9: 136-144.

7. P.S. Rajakumar, B. Vishnu Bhat, M.G. Sridhar1, J. Balachander2, B.C. Konar1, P. Narayanan and G. Chetan. Electrocardiographic and Echocardiographic Changes in Perinatal Asphyxia. Indian journal of pediatrics.2009;76(3):261-64.

8. Luc Mertens, Istvan Seri, Jan Marek, Romaine Arlettaz, Piers Barker, Patrick McNamara, Anita J. Moon-Grady, Patrick D. Coon, Shahab Noori, John Simpson,Wyman W. Lai. Targeted Neonatal Echocardiography in the Neonatal Intensive Care Unit: Practice Guidelines and Recommendations for Training. J Am Soc Echocardiogr. 2011;24:1057-78.

9. Jyoti Agrawal, Gauri S Shah, Prakash Poudel, Nirmal Baral, Ajay Agrawal, Om P Mishra. Electrocardiographic and enzymatic correlations with outcome in neonates with hypoxic-ischemic encephalopathy. Italian Journal of Pediatrics. 2012: 38:33.

10. Goel M, Gohiya Poorva, Yadav BS. Assessment of Myocardial Function in Birth Asphyxia. *Int J Med Res Rev* 2013;1(5):228-232

.....

How to cite this article?

Patel U, Gedam DS. Assessment of Myocardial Function in Birth Asphyxia: Editorial. *Int J Med Res Rev* 2013;1(5):220-221

.....