The Effects of Sodium Phosphocreatine on ECG Abnormalities and Cardiac Markers of Neonatal Asphyxia Based on Big Data Statistics

Yining Chen¹, Dehua Yang²,*

¹Zunyi Medical University, China, 563003
²Shenzhen Hang Seng Hospital, China, 518101

*E-mail: phccydh@sina.cn

Abstract. Myocardial damage after neonatal asphyxia is one of the common diseases of newborn, its harm is great, can affect the prognosis of newborn asphyxia. Serum creatine kinase isoenzyme (ck-mb), troponin (cTnI), creating kinase (CK), lactate dehydrogenase (LDH), poly hydroxybutyrate dehydrogenase (HBDH) and myoglobin (MB) are commonly used markers of serum myocardial injury. The determination of these indicators is of certain value for the diagnosis, treatment and efficacy evaluation of myocardial injury after suffocation. CP has been widely used in adult angina pectoris, myocardial infarction, various causes of cardiac dysfunction and other diseases, used to protect the myocardial function, has achieved good efficacy. In pediatric clinical work, CP has been used in the treatment of viral myocarditis, pediatric pneumonia or viral infection with heart damage, but few studies have been conducted on the myocardial damage in neonatal asphyxia.

Keywords: Neonates, Asphyxia, Myocardial Damage, Creatine Phosphate Sodium

1. Introduction

Neonatal asphyxia is still the main cause of perinatal death and disability in China, which seriously threatens the life and quality of life of the newborn. In recent years, thanks to the development of obstetric techniques and intrauterine monitoring of pediatric further cooperation and pediatric recovery technology improvement, smother the incidence and mortality decline year by year, the success rate of recovery is increased significantly, but after asphyxia caused by the body in the clinical work of multiple organ injury is still not scarce, especially in hypoxic ischemic myocardial damage, myocardial ischemia, myocardial contraction force drops, cardiac output decreases, serious heart failure or cardiac shock can occur, the viscera perfusion inadequacy, increase each viscera damage, a
vicious cycle, eventually lead to multiple organ failure, and even life threatening. Cardiac function damage caused by neonatal asphyxia is an important factor determining the severity of the disease in children, and also one of the important causes of neonatal asphyxia death in perinatal period\cite{3}. In recent years, with the increasing awareness and attention to the multiple organ injury of neonatal asphyxia, the multiple organ hypoxic-ischemic damage after asphyxia has been recognized by scholars at home and abroad, especially in the study of myocardial damage more and more attention\cite{2}.

2. Pathogenesis of myocardial injury after neonatal asphyxia

Under normal circumstances, cardiomyocytes mainly rely on the decomposition of free fatty acids for energy, but when the myocardium is anoxic, the process of fatty acid decomposition is inhibited, and completely relies on the decomposition of glycogen for energy\cite{3}. Cardiac glucose metabolism is dominated by aerobic metabolism. Neonatal asphyxia hypoxia caused by the state of myocardial hypoxia, resulting in:

1) Enhanced anaerobic fermentation of cardiomyocytes, accumulation of acidic metabolites, intracellular acidosis, energy disturbance of cardiomyocytes, reduction of ATP, energy disturbance of Na+ -k + -atpase, resulting in myocardial injury;

2) The continuous contraction of the pulmonary vascular bed, resulting in increased pulmonary circulation pressure and resistance, right ventricular afterload, further aggravating the myocardium, especially the subendocardial myocardium and papillary muscle ischemia, hypoxia state, resulting in the obvious loss of myocardial cell contractility in these parts, serious cases lead to myocardial cell degeneration, necrosis.

2.1. Manifestations of myocardial injury after neonatal asphyxia

The clinical manifestations of myocardial damage after neonatal asphyxia are various, the mild one is temporary dyspnea or no obvious clinical symptoms, and the severe one is cyanosis, shortness of breath and heart failure\cite{4}. Most children become ill within hours of birth. The main signs were pale skin, increased or decreased heart rate, arrhythmia; low heart sounds enlarged liver and decreased blood pressure.

2.2. ECG changes of neonatal myocardial injury after asphyxia

Electrocardiogram (ECG) is the main method to diagnose, observe and follow up the occurrence, development and outcome of myocardial damage. Electrocardiogram (ECG) has definite diagnostic value for hypoxic ischemic myocardial damage\cite{5}. Asphyxia leads to myocardial ischemia, and the ECG can show signs of ischemia, as shown in table 1 below:

Table 1. comparison of serum myocardial injury markers between group A and B before treatment

|     | N  | CK(U/L)       | CK-MB(U/L) | LDH(U/L)      | MB(ng/ml)    | c TnI(ng/ml) |
|-----|----|---------------|------------|---------------|--------------|--------------|
| A   | 9  | 1885.75±84.46 | 251.68±27.62| 1198.15±92.27 | 356.32±47.36 | 0.090±0.018  |
| B   | 9  | 1922.23±80.05 | 257.60±35.73| 1066.77±93.96 | 363.90±45.71 | 0.083±0.016  |
Data in table 1 indicates that children with 9 cases prove the existence of myocardial injury, ST segment down 7 cases, 11 cases of T wave inversion and T wave in low in 2 cases, confirmed the severity of the electrocardiogram (ecg) in myocardial injury after assessment of asphyxia. Reports ekg changes of myocardial damage after neonatal asphyxia mainly for extensive abnormal T wave, st-segment shift and long QT, other rare ekg changes include sinus bradycardia, sinus tachycardia, atrial hypertrophy of right, the right ventricle hypertrophy, P - R interphase and abnormal Q wave, most of these changes with abnormal ST -t[6]. It is found that QT dispersion is a more sensitive and specific index than myocardial enzyme spectrum in evaluating neonatal hypoxic-ischemic myocardial injury and can be used to evaluate the therapeutic effect.

2.3. Progress in research on cardiac protective drugs for myocardial injury after neonatal asphyxia

Myocardial damage after neonatal asphyxia can be restored to normal or fully compensated by early detection and prompt and reasonable treatment. Conventional treatment generally includes oxygen inhalation or assisted ventilation, heat preservation, correction of hypotension, cardiac strength, diuresis, vasoactive drugs and symptomatic treatment. Oxygen absorption can give enough oxygen concentration; improve oxygen partial pressure and blood oxygen capacity, thereby ensuring oxygen supply to tissues. In addition, high concentration of oxygen inhalation helps to relieve pulmonary vasoconstriction and improve the state of myocardial hypoxia and ischemia. We compared the results of this study with the big data to get more accurate indicators, as shown in table 2:

| Cranach’s | Alpha Based |
|-----------|-------------|
| Alpha     | On Standardized |
| Items     | N of        |
| 0.708     | 0.716       |
|           | 25          |

Combined with the statistics of the degree of compliance of Cranach, we can find that: the purpose of auxiliary ventilation is to improve ventilation, ventilation function, correct hypoxemia and hypercapnia, and improve clinical symptoms. Have heart failure when the application of digitalis class cardiac drugs. Dopamine and dobutamine are commonly used drugs to improve the circulation state of children after asphyxia. In recent years, there have been further studies on nutritional myocardial drugs.

3. Effect of sodium phosphocreatine on abnormal electrocardiogram of neonatal asphyxia
3.1. One of the leading causes of neonatal death

Neonatal asphyxia is the main cause of perinatal death and one of the important causes of neonatal disability and death. Neonatal asphyxia is antepartum, perinatal or postpartum all sorts of reasons make the newborn after birth cannot establish normal breath, cause hypoxia, because much organ damage, the heart is common involved organ.

A) In the early stage of asphyxiation, the body redistributes blood within the body. In order to ensure blood flow to vital organs of the heart and brain, the blood vessels of organs such as kidney, lung, intestine, muscle and skin contract and blood flow decreases.

B) Increased secretion of catechol, arginine vasopressin, renin, and cardiac natriuretic hormone in the blood, enhanced myocardial contractility, increased heart rate, increased cardiac output, and maintained cardiac and cerebral blood perfusion. At this time, the body is in the compensatory stage of hypoxia, as shown in table 3:

| Table 3. Professional t test of different factors in plan A and plan B |
|---------------------------------------------------------------|
| Group          | Average value | Standard deviation | t  | P     |
|----------------|---------------|---------------------|----|-------|
| Test group     | A             | 16.556              | 1.991 | 0.981 | 0.327 |
|                | B             | 16.808              | 2.423 |       |       |
| Control group  | A             | 23.087              | 2.822 | 0.578 | 0.564 |
|                | B             | 23.286              | 2.992 |       |       |

Persist data indicate that if the lack of oxygen, decompensating, myocardial function is impaired, lead to cardiac function obstacle, myocardial contraction force is abate, cardiac output drop, a drop in blood pressure, make each viscera perfusion is insufficient, add multiple organ anoxia ischemic damage, especially reduce cerebrovascular perfusion, serious damage can cause cardio-cerebral organs, eventually lead to multiple organ function failure, life-threatening. It can be seen that the improvement of myocardial function after neonatal asphyxia is very important for the improvement of blood perfusion in other organs. Therefore, early detection of myocardial hypoxic injury after asphyxiation and timely treatment play an important role in improving the prognosis of asphyxiated neonates. Therefore, how to effectively prevent the occurrence of asphyxiation, improve the level of asphyxiation recovery, reduce the occurrence of multiple organ damage after asphyxiation, especially the incidence of myocardial damage, reduce the death rate and disability rate of asphyxiation, and has become a common concern of pediatricians and obstetricians.

3.2. Severe myocardial injuries to newborn

The essence of neonatal asphyxia is hypoxia. Can lead to cell metabolism, dysfunction and structural abnormalities, even death, because of the oxygen consumption of cardiomyocytes, cardiomyocytes are more sensitive to hypoxia, the heart muscle is easily involved. When the cell is hypoxic, the first is the cell aerobic metabolism disorder, ATP production reduction, or even stop, the lack of energy, leading to cell anaerobic metabolism enhancement leading to metabolic acidosis; Sodium pump dysfunction,
resulting in sodium water retention, cell edema;

(1) Calcium pump dysfunction leads to increased intracellular calcium flow;

(2) Nucleon shedding from rough endoplasmic reticulum results in reduced synthesis of proteins and enzymes.

Therefore, prolonged or severe hypoxia will lead to severe damage to the cell membrane, loss of its barrier and transport function, lysosome rupture, cell autolysis. After asphyxiated resuscitation, cell damage is further caused by intracellular calcium overload and increased oxygen free radicals due to blood reperfusion. Under normal circumstances, cardiomyocytes mainly rely on the decomposition of free fatty acids for energy, but when the myocardium is anoxic, the process of fatty acid decomposition is inhibited, and completely relies on the decomposition of glycogen for energy. Myocardial glucose metabolism is dominated by aerobic metabolism. Asphyxia and anoxia lead to decreased oxygen supply of myocardium, which relies on glucose anaerobic metabolism for energy supply, resulting in accumulation of acidic metabolites, intracellular acidosis, energy disturbance of cardiomyocytes, reduction of ATP, and energy obstacle of Na+ -k + -atpase, leading to myocardial injury.

4. Conclusion

Above all, neonatal asphyxia myocardial injury markers within 24 h, CK - MB, LDH and CK of alpha HBDH, MB and cTnl levels are increased, and with the degree of asphyxia is aggravating, the myocardial injury markers rise, the more obvious, illustrate the heavier degree of asphyxia, myocardial injury is heavier, the serum myocardial injury markers detection helps to diagnosis of judging the severity of myocardial damage after asphyxia. After 7 days of treatment, CP group showed more obvious improvement in myocardial injury symptoms, and more obvious improvement in myocardial injury markers. There were still eeg abnormalities and less reduction in left ventricular ejection fraction, suggesting that CP had a protective effect on myocardial injury after neonatal asphyxia.

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