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SURGICAL OUTCOME OF CONGENITAL HEART DEFECTS WITH PULMONARY HYPERTENSION IN INFANTS

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Background: *Congenital heart diseases with large left-to-right shunt often have signs of pulmonary artery hypertension. It is an important determinant of morbidity and mortality in patients without educate surgical treatment especially in infants.*

Methods: ***N****inety patients with congenital cardiac septal defects and pulmonary arterial hypertension had operation to close their septal defects. All the patients have been checked by Chest X-ray, EchoCG, ECG, selectively performed the cardiac catheterization and lung biopsy. Before and after surgery the PA pressure was compared to systemic by needle puncture measurement.*

Results: *Twenty patients died in the hospital after operation and there were no later deaths in follow-up. Hemodynamic changes after operation included a significant decrease in pulmonary artery pressure (mean pulmonary artery pressure, 28.3± 2.4 mm Hg versus 58.45± 1.69 mm Hg before repair). The follow-up period was from 3 months to 4 years (mean 1.3 ±0.6 years).*

Conclusion: *Studies from developed countries have shown that in term infants, young age is not a risk factor for adverse postoperative outcome after surgical closure of septal left-to-right defects. The data presented in this study shows that operations to close cardiac septal defects in the presence of severe pulmonary hypertension are effective, but must be done during first 6 month.*

Key words
bypass, pulmonary hypertension, infants

Congenital heart defects (CHD) with left-to-right shunt carry high mortality in infancy due to development of pulmonary arterial hypertension. The natural mortality during the first year of life is about 8-11%. There are some reasons of high mortality: development of pulmonary arterial hypertension with congestive right heart failure, pulmonary hypertensive crisis, difficult for curing pneumonia,

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hypotrophy and septic conditions. In 30- 50% of this condition complicated with severe pulmonary arterial hypertension, which has a progressive character [1]. Pulmonary artery pressure is hyperkinetic in early phase but may eventually become a fixed elevation associated with fixed increase of pulmonary vascular resistance [2].

CHD with pulmonary hypertension requires urgent surgical treatment, which is caused by development of irreversible changes of in-lungs and critical condition of patients [3, 4]. This need requires surgical intervention in the first year of life.

During the past years many world leading hospitals have been excellently performing surgical interventions in infants. This report documents the experience and results of septal defects repair in a group of patients with pulmonary hypertension in infancy.

Material

90 patients with congenital cardiac defects and pulmonary hypertension had been operated in three institutions during the period from January 2, 2000, to May, 2008. There were 69 patients with isolated ventricular septal defects, 2 patients with ventricular septal defects with interrupted aortic arch, 2 cases of ventricular septal defect with coarctation of aorta, 4 patients with complete endocardial cushion defects, 5 patients with total anomalous pulmonary venous return, 3 patients with double outlet of right ventricle, 2 patients with truncus arteriosus and 1 patient with transposition of great arteries. The age ranged from 4 days to 36 months (average, 12.29 ± 1.63 months). All the patients had complains on often colds, lack of physical growing, strengthless, heart beating. All patients had hypotrophy about 30%. A systolic murmur was audible in 28 patients. The murmur was only of grade I in 8 patients and it was absent in 6 patients. The second heart sound was increased at the left base in all the patients.

Methods

All the patients had been examined on ECG, Chest X-ray, Echocardiography. Some patients were selectively chosen for cardiac catheterization before surgery and lung biopsy during surgery. Anesthesia was induced by fentanyl and intermittent positive pressure ventilation with isoflurane. Ketamine was used for premedication. Pressure in pulmonary artery was measured by needle puncture before repair of the septal defect and at completion of operation before closure of the wound. Systemic blood pressure and central venous pressure were monitored in the radial artery and femoral vein. We used hollow fiber oxygenators, the bypass performed on continuous and intermittent flow, with conventional and modified

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ultrafiltration for all the patients. For myocardial protection we used cristalloid cardioplegia (Stanford University formula) for the first time 20ml/kg and for additional infusions blood cardioplegia.

Results

During the preoperative examination we found that:

The electrocardiogram showed mostly right axis deviation. There were right ventricular hypertrophy in 35 patients and 7 showed biventricular hypertrophy. Chest roentgenogram showed dilation of main pulmonary artery in all the patients. The mean cardiothoracic ratio number is 0.64 ± 0.10. The pressure gradient through the intracardiac or extracardiac defect was present in all the patients by echocardiography and ranged from 45 to 5 mmHg (mean, 18 ± 8 mmHg). Cardiac catheterization was performed in 4 patients. We found that 3 patients had bidirectional shunt, and 1 patient with left to right shunt. The pulmonary artery pressure was relatively close to systemic arterial pressure. The angiography shows the same picture as on echo. Biopsy of the lung was performed in 10 consecutive patients. Pulmonary vascular disease was graded by Heath - Edwards' classification [4]. There were 4 patients in grade III, and 6 had grade IV pulmonary vascular disease. The results of biopsy were different than the results of echo and tensiometry. This means that the lung biopsy could present the real PH picture in infants. Pulmonary artery pressure to systemic pressure value before surgery was 85± 1.87% and 50.73± 2.11% after correction by intraoperative tensiometry. The defect was repaired via right atrium in most cases, but in 5 cases through right ventricle incision. We used Dacron, Teflon, Double Velour, Gore-Tex and glutaldehide treated pericardial patches. For the suture technique in most cases we used continuous suture, less interrupted sutures and in some cases combine method. We didn't find any differences and complication of two bypass techniques. The mean bypass time was 94.67 ±3.63 minutes and cardiac arrest time was 60.68 ±2.87 minutes. All the patients were checked intraoperative by TEE after correction for the prevention of air embolization and for checking of correction and cardiac performance after bypass weaning. The early postoperative period was complicated, because the most of the patients required urgent surgery or indicated for surgery by vital signs. After the surgery all the patients transferred to HICU and managed postoperatively. All the patients received dopamine, epinephrine, dobutamine, milrinone in sub- or maximal therapeutic dosage, in all cases partial parenteral nutritions were infused on the second postoperative day. The dynamics of pulmonary hypertension was estimated with echocardiography by the tricuspid and pulmonary regurgitation method before and during follow-up after operation.

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Sedation and oxygen supplement were considered to be important to these patients in postoperative care. The patients had been ventilated for 109± 8.68 hours and extubated after complete recovering of vital signs. The inotrops has been infused during 145.66± 8.14 hours and stopped after complete cardiac performance improving. The average postoperative stay in HICU was 12.04± 2.39days. The patients transferred to the general ward for continuous follow up of PH regress and improving patients' condition. The mean in hospital stay after operation was 16.25± 2.20 days.

Twenty patients died in the hospital after operation. The most common reason of mortality was heart failure, residual PH, respiratory failure. None of survived patients required reoperation. 6 patients were under the 6 months and other in age more than 1 year.

The follow-up period was from 3 months to 4 years (mean 1.3 ± 0.6 years). Symptoms of dyspnea on exertion were relieved, and physical growing improved. Hemodynamic changes after operation included a significant decrease in pulmonary artery pressure (mean pulmonary artery pressure, 28 ± 2.4 mm Hg versus 58.45± 1.69 mm Hg before repair). Arterial oxygen saturation increased significantly after operation (mean, 98% ± 1% versus 90 ± 4% before operation). The follow-up echocardiography showed the residual shunt in 6 patients which doesn't significantly changes hemodynamic and need reoperation.

Comments

The surgical treatment of congenital heart defects with pulmonary hypertension is controversial. Perioperative mortality to close septal defects is high (22.7% to 50%) [5-8]. Acute congestive heart failure, residual pulmonary hypertensive crisis, acute respiratory failure are the principal causes of perioperative deaths. Many authors reported that operations to close septal defects were carried out cautiously when the ratio of pulmonary to system flow was less than 1.5, and the arterial oxygen saturation is near to or less than 90% [2,9,10].

Pulmonary arterial pressure usually falls down in the first 3 months of age. If the congenital heart defect with left-to-right shunt present the pulmonary hypertension could progressively increase. Pulmonary artery pressure and pulmonary vascular resistance has always significantly elevated in patients with Eisenmenger's syndrome. Operations to correct congenital cardiac defects require the use of cardiopulmonary bypass, infusion of protamine, and other factors that could cause release of vasoactive substances such as thromboxane A2 and catecholamines, resulting in pulmonary vasoconstriction and acute pulmonary

hypertension [11-14]. Pulmonary hypertensive crisis can be associated with acute congestive heart failure and death after operation in some of these patients.

The data presented in this study show that operations to close cardiac septal defects in the presence of severe pulmonary hypertension are effective. Even if our 22.2 % overall mortality rate is much higher than the rates reported recently from the leading centers (0% to 5%) [13, 16]. In our point of view the surgical treatment of this category of patients must be done before 6 months, but it depends on the level of cardiology service developing.

Studies from developed countries [13,16] have shown that in term infants, young age is not a risk factor for adverse postoperative outcome after surgical closure of septal left-to-right defects. Also in this study, patients did not have significant impacts on mortality rates. However, younger patients had longer postoperative recovery periods (duration of mechanical ventilation, ICU stay, and hospital stay). Their slower recovery may have resulted, in part, from longer durations of CPB.

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