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Valve replacement for valvular heart disease with giant left ventricle

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Abstract. – OBJECTIVE: To discuss early efficacy of the cardiac patient with giant left ventricle underwent valve replacement.

PATIENTS AND METHODS: Fifty-five patients, who are suffering cardiac valve disease with giant left ventricle, underwent valve replacement. Among them, sixteen patients underwent aortic valve replacement; thirty patients underwent mitral valve replacement; nine patients underwent double valve replacement. All of them use mechanical heart valve.

RESULTS: The number of early death after operation was five. Two patients died of malignant arrhythmia; two died of intractable low cardiac output syndrome; the last one's mechanical valve lost its ability to function after operation and died of respiration-circulation failure after an emergency operation. The death rate was 9%. The remaining 55 patients were cured and their cardiac function was significantly improved.

CONCLUSIONS: The definite effective myocardial protection and perfect and detailed preoperative treatment can reduce the possibility of operative complications and death rate of this kind of patients.

Key Words:
Giant left ventricle, Valve replacement, Valvular heart disease.

Introduction

From January 2004 to January 2014, there were 55 cases of the heart valve replacement in our hospital, based primarily on the examination of two-dimension ultrasound Doppler before operation[1]. That their left ventricular end diastolic diameter (LVDD) inspected is ≥ 70 mm.

Patients and Methods

General Information

Among the 55 patients, there were 31 males and 24 females, aged from 26 to 67. All the cases were caused by diseased rheumatic valvular, include 16 cases were aorta valve disease (AVD), 30 cases were mitral valve disease (MVD), and the remaining 9 cases were double valve disease (DVD). Six patients had accepted balloon dilatation 6 months to 15 years ago. Ten had accepted congenital heart disease (CHD) ventricular septal defect repair 10 to 20 years ago. Their disease duration ranged from 4.5 to 22 years. All the patients had the history of heart failure, of which 33 cases had the experience of repeated heart failures. On admission, the number of NYHA class II patients was 14, class III was 32, and class IV was 14. Fifteen patients' weight was more than 15%, lower than normal standard. X-ray film exams showed that all had pulmonary blood stasis and increased heart shadows of different degrees. The cardiothoracic ratio was 65-82%. The LVDD I evaluated by the echocardiogram was 70-89 mm and ejection fraction (EF) was 32-69%. Only ten cases were > 60%, examined by the heart color ultrasound with x-ray chest films, the pulmonary artery pressure (PAP) of 32 cases was > 40 mmHg. ECG examined that 22 cases were atrial fibrillation, fourteen cases of which were rapid atrial fibrillation. Twenty cases were sinus rhythm; eight cases were nodal tachycardia; nine cases were ventricular premature beat (VPB); six cases were class I atrio ventricular block; six cases were incomplete right bundle branch block (IRBBB); three cases were left anterior fascicular block; three cases were right posterior fascicular block; three cases were intra venticular conduction difference; 33 cases were left ventricular hypertrophy and 42 cases were myocardial damage. In twelve patients over 55 years old, no stenosis phantom was found through the examination of coronary artery disease before operation. The major complications of the patients in this group were six patients with diabetes mellitus, twelve

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patients with high blood pressure, three patients with cerebral infarction, and one patient with renal insufficiency.

Operation Methods

The operation methods included were intravenous anesthesia, trachea cannula, median sternotomy, mid-hypothermia extracorporeal circulation, oxygenator application mode, and hyper filtration. All cases were adopted with warm oxygenated blood cardioplegia solutions to lead to cardiac arrest and reperfused at warm blood terminals. A quantity of 4°C cold crystalloid solution was intermittently injected to maintain cardiac arrest to protect myocardium.

Eighteen patients with left atrium thrombus accepted valve replacement after the complete removal of blood dots. After mitral valve replacement, the patients with preservation of subvalvular apparatus were 48. Which means, two group of main chordae tendineae and a small part of valve tissue, which are near the junction of anterior mitral valve, were sutured to the two junctions near the posterior valve side, respectively, and the valve leaflet of anterior mitral valve with no chordae tendineae attached to it was removed, keeping the whole posterior valve and subvalvular apparatus. For the patients, whose valve was thickening, calcified, shortening, and fused, together with the papillary muscles contraction, the anterior mitral valve and its chordae tendineae and chordae tendineae of the papillary muscles were removed, keeping the whole posterior valve and subvalvular apparatus. 21 cases accepted the tricuspid valve annuloplasty. 3 cases added the operation of atria-septal defect repair and one patient added the operation of ventricular septal defect repair. The aortic cross-clamp time was 53-206 min and cardiopulmonary bypass time was 92-255 min. The number of auto-rebeat after operation was 19, 30 cases returned heart rebeat with the help of one or two times of electric defilbrillation. The sternum was closed after the epicardium temporary pacing electrode was placed. The artificial valves replaced in this group were all mechanical valves.

Results

Among the 60 cases, five cases died in the hospital. Two cases were resulted from multifocal ventricular tachycardia 9 days after the double valve replacement, and during the process of intravenous injection of lidocaine, the multifocal ventricular tachycardia turned into ventricular fibrillation. Though the heart rebeat after three times of electric defilbrillation, the repeated ventricular fibrillation led to death (check this sentence). Two died of intractable low cardiac output syndrome after double valve replacement. The last one's mechanical valve lost its ability to function in the next day after mitral valve replacement and the patient died of respiration-circulation failure after an emergency operation. The death rate was 8.8% and the remaining 55 patients were cured and their cardiac functions were significantly improved.

Discussion

The clinical features of giant left ventricular heart valve replacement were that the preoperative treatment was difficult, the operative death rate was high, and the long-term outcome was generally poor[2,3]. In order to increase the cure rate, medical experts from home and abroad devoted great efforts, such as getting to know the risk factors before operation, improving cardiac functions and the body conditions and enhancing myocardial protection during the operation[4]. When undergoing mitral valve replacement, the preservation of subvalvular apparatus and great attention to check and deal with tricuspid valve disease did some help. And shortening the anesthesia duration and operative time, preventing and treating early about the ventricular arrhythmias and low cardiac output syndrome played positive roles in increasing the cure rate. We can say that from the start of operation to the guidance after operation, we have summarized sound experiences, which have greatly improved the effect of surgical therapy.

Since 1997 we have adopted standard therapeutic schedule to treatment giant left ventricle heart valve replacement. Patients with LVDD ≥ 70 mm were all treated with preordained solutions. Before operation we focused on the improvement of cardiac functions[5]. Meanwhile, attention was paid to balance water and electrolyte, protect renal function and liver function. Oxygen inhalation through nasal tube was administered twice a day, half-an-hour each time. The whole group applied cardiac stimulant, diuretics, and energy mixture to adjust heart rate, controlling it under 100 times/min[6]. When focusing on mitral valve insufficiency or aortic valve diseases, we

added vasodilators, such as isosorbide dinitrate, to reduce the reflux by alleviating resistance of peripheral vascular. In patients without tachycardia, atrial fibrillation, and chronic heart failure syndromes, the milrinone was added for 3 days[7,8]. Nine cases were given albumin and a small amount of whole blood. Except for six cases of emergency patients, cardiac functions and the body conditions of the rest 54 patients were improved of different degrees by the preoperative preparation.

The definite effective myocardial protection during the operation is very important[9]. All cases adopted the integrative comprehensive myocardial protection measures, that is to say, using warm oxygenated blood cardioplegia solution to lead to cardiac arrest and were reperfused at warm blood terminals, and 4°C cold crystalloid solution was intermittently injected to maintain cardiac arrest[10]. No patient died of malignant arrhythmia or low cardiac output syndrome. By using warm blood to induce cardiac arrest, this situation happened in the state with aerobic metabolism, where it can significantly increase myocardial uptake of oxygen and avoid the trauma of cooling contracture and uneven distribution of cardioplegic solutions[11]. A quantity of 4°C cold crystalloid cardioplegia solution was instilled promptly for one time after cardiac arrest so as to leave the electrical and mechanical cardiac activities at relaxed stationary state[12]. Before opening ascending aorta, warm blood was instilled to maintain cardiac arrest in the aerobic condition for a period of time and it is called "the second-time cardiac arrest". It can decrease the demand of myocardium for oxygen, make the potential ischemic myocardium apply the uptaking oxygen to recover the intact issue. By repaying the oxygen debt during the period of ischemia, it will actively resuscitate the myocardium. The measure above can provide better myocardial protection, especially for those high-risk patients with cardiac insufficiency and low myocardial energy reservation[13,14].

The surgical procedure required quickness, accuracy, and coordination so as to shorten the time of operation and cardiopulmonary bypass as much as possible. The preservation of subvalvular apparatus in mitral valve replacement can decrease the incidence rate of low cardiac output syndrome, especially for the patients with giant heart1[15]. Subvalvular apparatus played an important role in adjusting diastolic dilatation and systolic strain of left ventricular wall[16]. Okita et al[17] found that preservation of subvalvular apparatus in mitral valve replacement can make the contractive activity of ventricular walls, such as left anterior base, anteriolateral, and left ventricular axis, function well. Sixteen cases in this group have the preservation of the subvalvular apparatus in mitral valve replacement. In the early review of heart after operation, color Doppler flow imaging (CDFI) showed[18] that left ventricular diastolic diameter (LVDD), left ventricular systolic dysfunction (LVSD), ejection fraction (EF), and stroke volume (SV) were greatly improved. Attention was paid to the treatment of tricuspid valve. It was reported that patients with minor functional tricuspid insufficiency had functional reflux or even continuous right-sided heart failure after the correction of left cardiac valve disease. One case in this group ignored tricuspid valve plasty and right-sided heart insufficiency occurred in the early post-operation.

Patients after operation were sent to ICU and closely monitored about arterial pressure, pulse rate, electrocardiogram, central venous pressure, percutaneous blood oxygen pressure, and pulmonary wedge pressure. Urine volume and pericardia drainage volume were recorded each hour. Intermittent inspection was done on hemoglobin concentration and plasma electrolytes concentration. Focusing on the supplementation of blood volume, positive inotropic drugs, such as dopamine and dobutamine, were added in a proper volume to maintain formal cardiac output and the level of hemoglobin, which stayed at or above 10 g[19]. The heart rate and blood pressure stayed at the normal range. Infusion quantity was controlled strictly, making sure that the output was bigger than the input. The crystal quantity in the transfusion was strictly controlled, proper colloids such as plasma and albumin were provided [20]. These increased plasma colloid osmotic pressure and remained good for the stability of the circulatory system. Generally, in the 6 h after operation, patients were in the stage of high resistance and low output. The increasing resistance in the surrounding blood vessels and augmenting afterload were usually the main factors that affected cardiac functions. When the blood volume was basically complemented, vasodilator drugs, sodium nitroprusside or phentolamine, were applied to lower the circulatory resistance and further improve the cardiac functions [21]. The pulmonary wedge pressure was kept at normal or slightly at low level. When patients come across with exact low cardiac output syndrome, epi-

nephrine should be added early or using intra-aortic balloon pump (IABP)[22].

The complications of ventricular arrhythmia and ventricular fibrillation after operation have directly affections on the operation mortality. In order to prevent and control arrhythmia, cedilanid should be applied 0.4-1.0 mg at the operative day and maintenance dose in the following days[23]. In the first 3 days after operation, the patients were given 0.5-1.0 mg lidocaine. Once the ventricular arrhythmia occurred, the patients were given lidocaine intravenous injection, 50 mg per time and then lidocaine intravenous drip were administered with the concentration of 2:1 or 4:1 until the heart rate returned back to normal. If the super ventricular tachyarrhythmia occurred, the patients would be given lidocaine promptly and 2-5 μg/kg amiodarone would be prepared in the meantime to be applied in accordance with the changes on Q-T interval strictly monitored by EKG[24]. At the same time, electric defibrillation was also prepared in case it turned into ventricular fibrillation during the process. Once there is fatal risk in patients, the prognosis is closely linked to the time of defibrillation, and the mechanical ventilation parameters are adjusted[25,26]. If it is the slow heartbeat, drug using is accompanied with temporary cardiac pacing to make the heart rate reach over 80 times per minute so as to avoid the incidence of low cardiac output syndrome, overfilling left ventricular and premature beat. The concentration of blood potassium is the sole factor that causes arrhythmia. After operation, every 500 ml urine output, the patient is added 1-2 g potassium[27]. The concentration of blood potassium is now checked and then made sure the blood potassium stays at a relatively high level in the normal range. The first 3 days after operation, 25% magnesium sulfate is given 5-10 ml per day by the routine methods and the electrolyte changes are checked regularly[28, 29].

Besides, it is worth noting that one case with nodal tachycardia in this group cannot be effectively and lastingly controlled both before and after operation. It is until 4 weeks after operation that this patient is definitely diagnosed with recessive hyperthyroidism and cured by the drug treatment.

Conflict of Interest

The Authors declare that they have no conflict of interests.

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