厚生連医誌 第12巻 1号 48~50 2003

Original Article

An Additional Consideration for "Anesthesia for Tricuspid Valve Replacement after Heart Transplantation"

Kariwagun General Hospital, Department of Anesthesiology[1], 2nd Hospital of Tokyo Women's Medical College, Department of Internal Medicine[2], Department of Anesthesiology[3]

Kappei Matsumoto[1], Shin-ichi Nunoda[2], Toru Ichiseki, and Miwako Kawamata[3]

Objective, Study desing, and Results: We present our method of anesthesia for a case of progressively severe tricuspid regurgitation that ultimately necessitated TVR on cardiopulmonary bypass, might be in a period of cardiac sympathetic reinnervation post heart transplantation (HTX) and with congenital progressive muscular dystrophy. Anesthesia was maintained with a total of 20mg of midazolam, 12mg of pancuronium, 1.1mg of fentanyl, and 450mg of propofol. The operation was completed with no problems. Prior intravascular fluid administration and infusion of dopamine and dobutamine stabilized hemodynamics at the weaning of cardiopulmonary bypass and with a standard external VVI pacing enabled us to obtain stable hemodynamic changes during maintenance of anesthesia. The patient was weaned from mechanical ventilation on the first postoperative day (at 19h).

Conclusion: We have reported a patient with severe TR developed secondarily due to biopsy-induced damage after HTX. Our anesthesia method consisted of the administration of fentanyl, pancuronium, midazolam and propofol was suitable for the case, 65 month after HTX..

Key Words: Anesthetic management, Tricuspid valve replacement, Transplanted heart, Reinnervation

Background

We experienced intraoperative anesthetic management of tricuspid valve replacement (TVR) in a 41-year-old man who underwent orthotopic human heart transplantation (HTX). The world experience with anesthetic management for valve replacement of the transplantated heart is limited.

Case

A 41-year-old man (weight 52kg, height 150cm, body surface area 1.55m2, BMI 23.1) with severe heart failure due to dilated cardiomyopathy received orthotopic human heart transplantation in 1993 at the university of Utah, Salt Lake city in USA. The donor was a young girl with no history of heart disease. The postoperative course was uneventful. He was discharged on postoperative day 9, when his echocardiography revealed no tricuspid regurgitation. The patient's post operative course was stable with only two episodes of ISHLT (International Society for Heart and Lung Transplantation) grade 「rejection. However, his echocardiogram gradually had shown the progression of tricuspid regurgitation since the 3rd post-operative year. He had been asymptomatic, but he noticed easy fatigability and dyspnea on exertion at the 4th post-operative year (1997). At evaluation the five years post transplantation. echocardiogram demonstrated severe TR with right atrial and right ventricular dilatation and a torn chordae which seemed to be occurred by endomyocardial biopsy. On February 1, 1999, 65 month after HTX, he underwent replacement of the tricuspid valve. Preoperative pulmonary function data were VC 1.87ll, %VC 53.1%, FEV1.0% 73.0%, CTR75%. Premedication with scopolamine 0.4mg and pethidine Hcl 35mg intramuscularly 30 minutes preoperatively was done. In the operating room, the patient was monitored with a five-lead ECG, ST segment analysis, SpO2, ETCO2, invasive radial artery blood pressure, pharyngeal and urine bladder temperature were measured. And a pulmonary artery catheter and TEE was placed. The pulmonary artery pressure and calculated pulmonary resistance were 20/11mmHg (mean 15mmHg), and 2.1 Wood units, the cardiac index was 1.6, respectively. Anesthesia was induced with 5mg midazolam, 0.5mg fentanyl, and 0.1mg/kg pancuronium to facilitate tracheal intubation. Anesthesia was maintained with mixture of air and oxygen (50%/50%), 0.3-0.5mcs/ kg/min infusions of fentanyl, 15-40mcs/kg/min propofol and additional pancuronium to sustain paralysis as required. He received infusion of PGE1 0.5mcs/kg/min during the operation. A total of 3400ml of acetate Ringer's solution, 490ml of autoblood, 300000 KIU of aprotinin, and 1000mg of methylprednisolone was administrated during the anesthesia, which lasted for a total of 420 minutes. Anesthesia was maintained with a total of 20mg of midazolam, 12mg of pancuronium, 1.1mg of fentanyl, and 450mg of propofol. Prior intravascular fluid administration and infusion of dopamine and dobutamine stabilized hemodynamics at the weaning of car-

diopulmonary bypass and with a standard external VVI pacing enabled us to obtain stable hemodynamic changes during maintenance of anesthesia. During hypothermia to 28℃ and cold crystalloid cardioplegic arrest, a 31mm Carpentier-Edwards porcine valve was implanted with a 65 minutes aortic cross-clamp time. The heart came off bypass without difficulty. The patient was weaned from mechanical ventilation on the first postoperative day (at 19h) and there was no tricuspid regurgitation and no respiratory disturbance. The operation was completed with no problems. He was discharged on postoperative day 26. A postoperative echocardiogram revealed marked reduction in right ventricular and atrial dimensions.

Discussion

Tricuspid regurgitation is fairly common findings after cardiac transplantation[8-10]. Most of the time this tricuspid regurgitation is mild or moderate and does not result in clinically significant[2-4]. Although various factors are discussed, the etiology of tricuspid regurgitation is not entirely clear. But it is thought to be due to pulmonary hypertension, myocardial edema and poor lymphatic drainage, alteration in the geometry of tricuspid valve related to the technique of right atrial anastomosis, or to mismatch in size between the donor heart and the recipient pericardial cavity[5]. Recently, however ruptured chordae of tricuspid valve as a complication of endomyocardial biopsy has been reported [6,7]. We think that the same cause might be implicated in our case. CPB triggers a systemic inflammatory response, which can lead to end-organ injuries affecting postoperative morbidity. TR and CPB causes hemolysis and activates leucocytes and platelets, after bypass white cells have reduced opsonization, metabolism and phagocytosis. Myocardial stunning to some degree is inevitable during aortic cross clamping. Causative factors include complement activation, surgical trauma, and immune responses, contact of blood with the extra corporeal circuit, lung reperfusion injury, coagulation and cerebral dysfunction. Postoperative renal insufficiency and neurological problems are seen with CPB[5]. Pulmonary interstitial edema increases and atelectasis develops. The preparation of anesthetic and cardiovascular drugs for patients undergoing heart transplantation does not differ from that for other cardiac patients. Because immunosuppressed patients are predisposed to infection, efforts are made to organize and use equipment in as sterile a manner as possible. This includes the use of an "intubation set" that contains all the necessary items, previously sterilized, for laryngoscopy and intubation. Monitoring catheters must be inserted using sterile technique. After placement of monitors, induction of anesthesia follows the basic principles of cardiac anesthesia. Our induction consisted of the administration of fentanyl, pancuronium and midazolam. The response of the patient was observed in terms of anesthesia, ventilation, blood pressure and heart rate. If a drastic response occurs, it is usually within 30seconds to one minute after initial dosing. In our patient, the hemodynamics was stable during both induction and maintenance of anesthesia. Anesthesia was maintained with a mixture of air and oxygen, and infusion of fentanyl, propofol and additional pancuronium. In the maintenance of anesthesia, the most prevalent problem is vasodilatation in the face of a poorly contracting heart and a decreased blood volume. It must be realized that the patient's blood pressure can be safely allowed to drop as far as a mean arterial blood pressure of 70mmHg without concern that the patient will be harmed. This mean arterial pressure falls within the autoregulatory range of both the brain and the kidney and, presumably, of other organs[10].The overall aim is to maintain an adequate blood pressure, to maintain urinary output over the longer term, and to ascertain that arterial and venous pH give no evidence of intracellular acidemia. In our patient, the postoperative pulmonary arterial pressure and pulmonary capillary wedge pressure kept to near normal values post operatively. With this technique of anesthesia, we can prevent potential pulmonary and renal morbidity. His physical examination and preoperative laboratory data including cardiac function were unremarkable. And HTX results in cardiac denervation by surgical interruption of postganglionic sympathetic fibers and subsequent rapid depletion of catecholamines storage in nerve terminals[2]. Their data confirm the low likelihood of sympathetic reinnervation within 18 month after HTX. Once the reinnervation process is initiated, a continuous growth is observed even late after HTX, suggesting a progressive nature of reinnervation. Reinnervation, however, remained regionally heterogeneous, and a complete restoration was not found until 15 years after HTX. For our patient, we administrated 0.1mg/kg of intravenous pancuronium before induction, heart rate did change because there might be occurred the partial sympathetic reinnervation in the transplanted heart of 65 month after HTX. The anesthesia was completed with no trouble, pancuronium was effective for increasing the heart rate too before CPB. And so we did not need the prior intravascular fluid administration particularly for stabilizing the hemodynamics during the induction of anesthesia. Prior intravascular fluid administration and infusion of dopamine and dobutamine stabilized hemodynamics at the weaning of cardiopulmonary bypass and with a standard external VVI pacing enabled us to obtain stable hemodynamic changes during maintenance of anesthesia. The operation was completed with no problems. He was discharged on postoperative day 26. A postoperative echocardiogram revealed marked reduction in right ventricular and atrial dimensions. There were no any differences for anesthetic management between this case and other TVR cases of on pump.

Discussion

We have reported a patient with severe TR developed secondarily due to biopsy-induced damage after HTX. Our anesthesia method consisted of the administration of fentanyl, pancuronium, midazolam and propofol was suitable for the case, 65 month after HTX..

References

1. Ichiseki T, Matsumoto K, Kawamata M, et al. [Anesthesia for tricuspid valve replacement after heart transplantation]. Jpn J Anesthesiol 2001;50:902-3. (in Japanese with English abstract).
2. Butler J, Rocher GH, Westaby S. Inflammatory response to cardiopulmonary bypass. Ann Thorac Surg 1993;25:1580-1.
3. Utley JR. Pathophysiology of cardiopulmonary bypass: current issues. J Card Surg 1990;5:177-89.
4. Kikickan L, Baykara N, Toker K, Tekinalp H, Akbas H: Anesthetic management of heterotopic heart transplantation on beating heart. J Anesth 2000;14:109-112.
5. DeMarco T, Dae M, Yuen GM, Kumar S, Sudhir K, Keith F, Amidon TM, Rifkin C, Klinski C, Lau D. Iodine-123 metaiodobenzylguanidine scintigraphic assessment of the transplanted human heart: evidence for late reinnervation. J Am Coll Cardiol. 1995;25:927-31.
6. DiCarli MF, Tobes MC, Mangner T, Levine AB, Muzik O, Chakroborty P, Levine TB. Effects of cardiac sympathetic innervation on coronary blood flow. N Engl J Med 1997;336:1208-15.
7. Bengel FM, Ueberfuhr P, Ziegler SI, Nekolla S, Reichart B, Schwaiger M. Serial assessment of sympathetic reinnervation after orthotopic heart transplantation: A longitudinal study using PET And C-11 hydroxyephedrine. Circulation 1999;99: 1866-71.
8. Crumbley III AJ, Van Bakel AB. Tricuspid valve repair for biopsy-induced regurgitation after cardiac transplantation. Ann Thorac Surg 1994;58:1156-60.
9. Wijburg ER, Balk AHMM, Van Herwerden LA. Double valve repair in a transplanted heart. J Thorac Cardiovasc Surg 1998;115:250-1.
10. Winn HR, Dacay RG, Mayberg MR. Cerebral circulation. In: Textbook of physiology. Philadelphia (PA): WB Saunders; 1989;952-60.

和 文 抄 録

症例

「移植心の三尖弁置換術麻酔経験」への追加考察－再神経化心臓の麻酔下の反応について－

刈羽郡総合病院、麻酔科
松本克平

心臓移植施行症例数の増加に伴い心移植後の症例にたいする心血管系手術、または非心臓手術も増加してくる。前回、心移植後の三尖弁置換術の麻酔管理を報告したが、今回さらに、離脱神経化、免疫反応について詳細な考察を加えた。本症例は移植後６年を経過し、ほぼ移植心のReinnervationが完成されていると思われ、通常脱神経化された心臓には作用しないパンクロニウムの内因性交感神経刺激作用に充分反応した。免疫抑制剤の副作用である高血圧、心筋繊維化等による心不全の発症を予防して行えば、Reinnervationの完成した移植心には、フェンタニール、パンクロニウム、ミダゾラム、プロポフォールによる通常の心臓麻酔が安全に行えると思われた。

キーワード：移植心、麻酔管理、再神経化、三尖弁置換術