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CASE REPORT

Vasoplegic Syndrome after Cardiopulmonary Bypass for Paravalvular Leak of Mitral Bioprosthesis

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Vasoplegic syndrome (VS) is characterized by low systemic vascular resistance paired with relatively preserved cardiac output that may result in increased morbidity and mortality rate after open-heart surgery utilizing cardiopulmonary bypass. We report a case of a 61-year-old male who underwent redo open-heart surgery due to a paravalvular leak of the previous implanted mitral bioprosthesis and severe tricuspid regurgitation. The patient experienced the VS in the intensive care unit after the operation. He was resuscitated by vasopressors and recovered uneventfully.

Key words: Vasoplegic syndrome, cardiopulmonary bypass, paravalvular leak

INTRODUCTION

Vasoplegic syndrome (VS) is one of the causes related to postoperative hypotension that involving 9%–44% of patients undergoing cardiac surgery. [1] It may cause severe hypotension due to decreased systemic vascular resistance (SVR) despite a normal or increased cardiac index (CI) and increases the risk of morbidity and mortality following the application of cardiopulmonary bypass (CPB) for cardiac surgery.[1] Here, we describe a male patient with redo open-heart surgery for a paravalvular leak of the previously implanted mitral bioprosthesis and severe tricuspid regurgitation who experienced VS.

CASE REPORT

A 61-year-old male (height 170 cm and weight 67 kg) was referred to our hospital due to exacerbated dyspnea for 6 months. He had a history of severe mitral regurgitation and underwent mitral valve replacement with bioprosthesis about 6 years ago. He presented with intermittent shortness of breath for 4 years and the symptoms got worse in recent half a year (NYHA Fc III-IV). The physical examination revealed grade IV systolic murmurs. Echocardiography demonstrated moderate mitral regurgitation with moderate paravalvular leakage, moderate-to-severe tricuspid regurgitation with pulmonary hypertension with a pulmonary artery systolic pressure of about 89 mmHg. He was then referred to our hospital and was scheduled to undergo redo open-heart surgery for a paravalvular leak of mitral bioprosthesis and severe tricuspid regurgitation.

During the operation, a dehiscence tear about 1 cm between the A2 annulus and the sewing ring of the previously implanted bovine valve was noted [Figure 1], no valve abnormality was noted. Teflon pledgets sutures were used to repair the tear site, DeVega annuloplasty was done for the tricuspid valve. Intraoperative transesophageal echocardiography showed normal cardiac systolic and diastolic function after weaning the CPB. During the closure of the sternal wound, a normal range of mean arterial pressure (MAP) was noted under the administration with dopamine, epinephrine, and dobutamine at a rate of 9.64 μg/kg/min, 0.1 μg/kg/min, and 3.01 μg/kg/min after the surgery. The patient was then transferred to the intensive care unit (ICU) for subsequent postoperative care. However, hypotension and metabolic acidosis with extreme hyperlactatemia (pH: 7.252, pCO_2: 36 mmHg,

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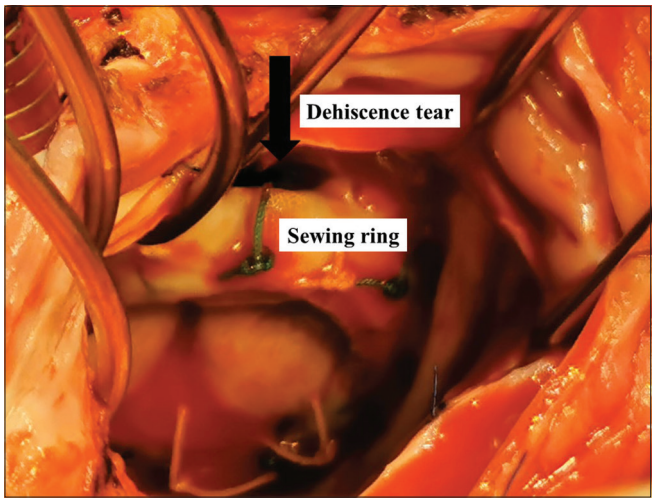

Figure 1: The operative photography revealed a dehiscence tear (black arrow) about 1 cm between the A2 annulus and the sewing ring of the previously implanted bovine valve

HCO_3^-:16 mmol/L, base deficit: 11.4 mmol/L and lactate level: 15.9 mmol/L) developed 3 h later after transferring to ICU. Meanwhile, no massive drainage from the mediastinal drainage tubes was recorded. Aggressive fluid resuscitation and intravenous sodium bicarbonate were infused to correct shock and acid-base imbalance. The lowest systolic blood pressure was 62 mmHg with a mean blood pressure ranging around 37–53 mmHg despite volume expansion. The CI was in the normal range at 4.3 L/min/m^2 and SVR of 648 dynes/s/cm^{-5}. VS was suspected, then vasopressor as norepinephrine was administered at a rate of 2.67 μg/kg/min. The blood pressure increased gradually and achieved the normal range. The vasopressors were then tapered and discontinued. No hypotension episode was noted during the following recovery course. The patient was transferred to an ordinary ward on postoperative day 7 and was discharged uneventfully. He is now in satisfactory functional status without significant mild mitral and tricuspid regurgitation from the follow-up echocardiography in the outpatient department.

DISCUSSION

VS, also known as vasoplegic shock or distributive shock, is a condition that characterized by significant hypotension due to vasodilation and low SVR in the presence of normal or supranormal cardiac output. Without immediate treatment, it might associate with increased postoperative morbidity and mortality after cardiovascular surgery.[1,2] From previously published investigations, the risk factors for post-CPB VS include the use of preoperative angiotensin-converting enzyme inhibitors or beta-blocker, low preoperative ejection fraction, procedure type (valve surgery, heart failure treatment), need for vasopressors before or during CPB, warmer core temperatures on CPB, relatively longer durations of aortic cross-clamping and CPB.[1,3]

The pathophysiology of VS after CPB is complex that may involve a myriad of interactions and cause the associated impaired vascular smooth muscle contracting resulting in vasoplegia: Derangements in receptor signaling, metabolic changes, the depletion of endogenous vasoactive hormones, and the alteration of the endothelial glycocalyx.[4] In clinical fields, other specific mechanisms of post-CPB vasoplegia are probably related to the pathologic response secondary to surgical trauma, ischemic-reperfusion injury, transfusion, and exposure of blood to the foreign surfaces CPB circuit that results in activation of a variety of cytokines and pro-inflammatory status.[1,2]

The first-line treatment of VS is vasopressors to restore vascular tone if a fluid challenge has been performed without improvement. Catecholamines exert their physiologic effects by modulation of adrenergic receptors. At present, there is no established first-line vasopressor for VS following CPB.[5-7] Norepinephrine, phenylephrine, epinephrine, and dopamine all have been used successfully to increase MAP. However, the concern about using high-dose vasopressors is potentially detrimental effects including peripheral vasoconstriction and end-organ injury.[5-7] There are several noncatecholamine vasopressors that can be considered as salvage treatment for catecholamine-resistant vasoplegia. Vasopressin is the first choice that induces vascular smooth muscle contraction by activating the vasopressin-1 receptor.[1] In addition, methylene blue is considered to have the ability to increase vascular smooth muscle tone and suggested a possible mortality benefit of the drug.[8] Hydroxocobalamin[9] and angiotensin II[10] could also be used if the goal of MAP is not achieved by infusing high doses of catecholamines. Ascorbic acid, thiamine, and corticosteroids may also lessen the VS through multiple mechanisms.[4]

In this presented case, no definite risk factors could be traced from the patient's record. We would like to stress that VS could be occurred in patients without definite risk factors except open-heart surgery utilizing CPB. Therefore, early recognition and timely management are the key issues to resuscitate this postoperative complication.

In addition, VS could be found in patients undergoing heart transplantation, Kao *et al*. reported that VS is not rare in post-heart transplant patient with an incidence of 24.5% without increased mortality.[11,12] Recent studies have supported the potential therapeutic roles of noncatecholamine agents when used in conjunction with norepinephrine, especially vasopressin and methylene blue. Early recognition and timely

treatment are the most effective approach in this group of patients without an increased hospital stay and mortality rates.

Ethical approval

This study proposal was approved by the Institutional Review Board of Tri-Service General Hospital. TSGHIRB No.: A202215048. Date of Approval: 2022/3/21.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published, and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

Dr. Chien-Sung Tsai and Dr. Chih-Yuan Lin, editorial board members at *Taiwan Journal of Medical Sciences*, had no roles in the peer review process of or decision to publish this article. The other authors declared no conflicts of interest in writing this paper.

REFERENCES

1. Omar S, Zedan A, Nugent K. Cardiac vasoplegia syndrome: Pathophysiology, risk factors and treatment. Am J Med Sci 2015;349:80-8.
2. Shaefi S, Mittel A, Klick J, Evans A, Ivascu NS, Gutsche J, *et al.* Vasoplegia after cardiovascular procedures-pathophysiology and targeted therapy. J Cardiothorac Vasc Anesth 2018;32:1013-22.
3. Levin MA, Lin HM, Castillo JG, Adams DH, Reich DL, Fischer GW. Early on-cardiopulmonary bypass hypotension and other factors associated with vasoplegic syndrome. Circulation 2009;120:1664-71.
4. Busse LW, Barker N, Petersen C. Vasoplegic syndrome following cardiothoracic surgery-review of pathophysiology and update of treatment options. Crit Care 2020;24:36.
5. Hajjar LA, Vincent JL, Barbosa Gomes Galas FR, Rhodes A, Landoni G, Osawa EA, *et al.* Vasopressin versus norepinephrine in patients with vasoplegic shock after cardiac surgery: The VANCS randomized controlled trial. Anesthesiology 2017;126:85-93.
6. Cheng Y, Pan T, Ge M, Chen T, Ye J, Lu L, *et al.* Evaluation of vasopressin for vasoplegic shock in patients with preoperative left ventricular dysfunction after cardiac surgery: A propensity-score analysis. Shock 2018;50:519-24.
7. Elgebaly AS, Sabry M. Infusion of low-dose vasopressin improves left ventricular function during separation from cardiopulmonary bypass: A double-blind randomized study. Ann Card Anaesth 2012;15:128-33.
8. Ozal E, Kuralay E, Yildirim V, Kilic S, Bolcal C, Kücükarslan N, *et al.* Preoperative methylene blue administration in patients at high risk for vasoplegic syndrome during cardiac surgery. Ann Thorac Surg 2005;79:1615-9.
9. Shapeton AD, Mahmood F, Ortoleva JP. Hydroxocobalamin for the treatment of vasoplegia: A review of current literature and considerations for use. J Cardiothorac Vasc Anesth 2019;33:894-901.
10. Chawla LS, Busse L, Brasha-Mitchell E, Davison D, Honiq J, Alotaibi Z, *et al.* Intravenous angiotensin II for the treatment of high-output shock (ATHOS trial): A pilot study. Crit Care 2014;18:534.
11. Kao CH, Lin CY, Lu CW, Tsai YT, Lee CY, Lin YC, *et al.* Vasoplegia syndrome is not associated with higher mortality rate after heart transplant. Acta Cardiol Sin 2012;28:129-36.
12. Hsu YC, Hsu CH, Huang GS, Lu CC, Wu ZF, Tsai YT, *et al.* Extreme hyperlactatemia after heart transplantation: one center's experience. Transplant Proc 2015;47:1945-8.