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Intraoperative hemodynamic deterioration caused by pre-existing mitral regurgitation in a patient with type A dissection: a word of caution

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ABSTRACT

Acute type A aortic dissection is a life-threating disease, and preoperative congestive heart failure caused by newly developed aortic regurgitation is known as a risk factor for increased hospital mortality. Herein, we report didactic case of hemodynamic deterioration during surgical treatment of acute type A aortic dissection, which was induced by pre-existed but undiagnosed severe mitral regurgitation. Not only surgeons but also all members of surgical team should bear in mind the possibility of pre-existed mitral valvular disease, which possibly induce congestive heart failure in patients undergoing surgery of acute type A aortic dissection even in emergent setting.

Keywords: Type A aortic dissection, mitral regurgitation, heart failure, perioperative management

INTRODUCTION

Acute type A aortic dissection (ATAAD) is a life-threatening condition, however, the results of open surgery continue to improve in the modern surgical era[1]. On the other hand, preoperative congestive heart failure (CHF) was known to as a risk for increased hospital mortality[2]. In most cases, the cause of preoperative CHF is newly developed aortic regurgitation (AR) associated with aortic root dissection[2]. Herein, we present a didactic case of hemodynamic deterioration during surgical treatment of ATAAD, which was induced by pre-existed but undiagnosed severe mitral regurgitation (MR).

CASE REPORT

A 61-year-old woman was transferred to our institution with the diagnosis of ATAAD at midnight. The patient had never been diagnosed with valvular heart disease and was not receiving any medication during routine outpatient visits to a neighboring hospital. The patient's heart rate was 92bpm (irregular), blood pressure was 118/69mmHg, respiratory rate was 14/min, and SAT was 98% (room air). In the emergency room, her back pain she had been previously experiencing had completely disappeared. Computed tomography (CT) demonstrated ascending aortic dissection with thrombosed false lumen (Fig. 1). There was no abnormal finding on electrocardiogram that might suggest a coronary arterial problem. Transthoracic echocardiography (TTE) was performed to exclude pericardial tamponade and AR, however, it was a poor study because the patient was obese with a height of 155cm, weight of 63 kg, and BMI of 26 m^2/kg. There was no left ventricular or atrial enlargement, and ejection flaction was 62% in sinus rhythm. The cardiologist on duty at night did not detect any pericardial effusion or significant

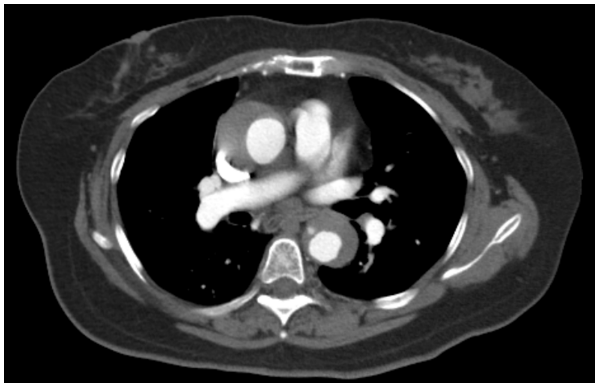

Figure 1. Preoperative computed tomography demonstrated ascending aortic dissection. The ascending aorta was 40 mm in diameter and false rumen was thrombosed completely.

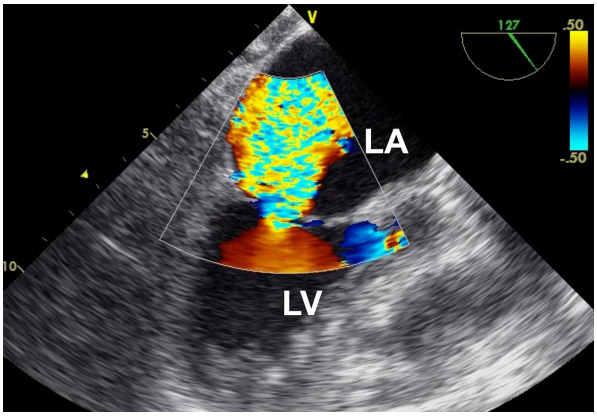

Figure 2. Intraoperative transesophageal echocardiographic image demonstrated massive mitral regurgitation during the weaning period of cardiopulmonary bypass.

valvular disease. Although emergent operation was planned, the patient suddenly complained of dyspnea with a small amount of bloody sputum during the waiting period in the intensive care unit (ICU). the patient received a continuous intravenous infusion of antihypertensive agents in sufficient volume to achieve an arterial blood pressure of ≤ 120mmHg, as well as an extracellular fluid load of approximately 1000ml. Immediately after endotracheal intubation, emergent operation was started. Cardiac tamponade due to rupture of the ascending aorta was suspected as the cause of the worsening respiratory condition, but a quick bedside TTE by a cardiovascular surgeon confirmed no cardiac effusion. During sternotomy, the anesthesiologist performed transesophageal echocardiography, which did not reveal any pericardial effusion too. Subsequent to hemi-arch replacement without any technical difficulty, weaning from cardiopulmonary bypass (CPB) was difficult because of severe MR (Fig. 2). A significant amount of tracheal bleeding and decreased oxygenation suggesting pulmonary hypertension due to left heart failure, and we decided that mitral valve intervention was necessary. The mitral valve was inspected through right-sided left atriotomy, and a torn chorda of the degenerated anterior mitral leaflet was revealed. No evidence of chordal rupture was observed. We performed mitral valve replacement (MVR) with a mechanical valve. Weaning from CPB for the second time was also difficult, even after the MVR, due to severe lung congestion and hemodynamic deterioration. Therefore, establishment of veno-arterial extracorporeal membrane oxygenation (ECMO) was necessary (Fig. 4). Despite intensive treatment, weaning off ECMO was difficult, and the patient died 8 days after surgery because of severe respiratory failure.

DISCUSSION

Despite disease severity, the results of open surgery for ATAAD are relatively favorable, and in the 2017 annual report of the Japanese Association for Thoracic Surgery, the surgical mortality rate in Japan was 11.2%[1]. The major causes of hospital mortality are known to be associated with preoperative CHF and organ malperfusion, such as acute myocardial infarction (AMI), cerebral infarction, and bowel necrosis[3]. CHF is observed in 6% of ATAAD patients, and most CHF cases are due to newly developed AR associated with aortic root dissection[2]. In such patients, precise recognition of proximal extension of the dissection and possible emergent operation is necessary to avoid CHF development. A simultaneous aortic valve procedure is sometimes required.

However, patients with ATAAD rarely have pre-existing MR[4], except in the case of connective

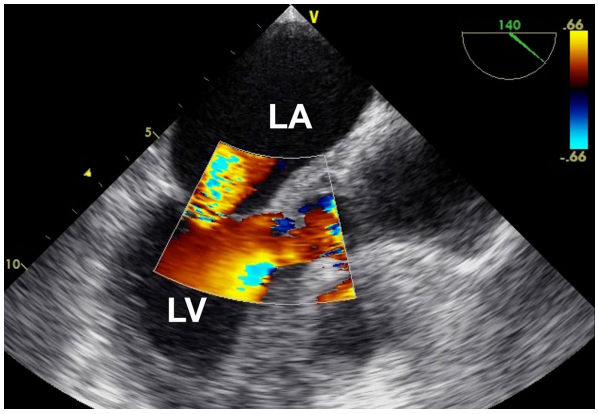

Figure 3. Transesophageal echocardiographic image during the sternotomy showed moderate-grade mitral regurgitation.

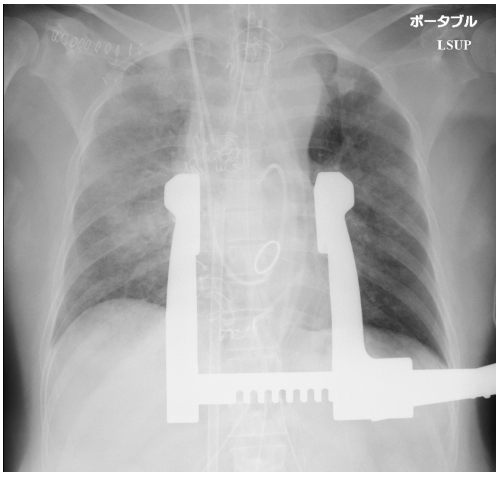

Figure 4. Chest x-ray on return to intensive care unit demonstrated that opacity in whole lung fields with right predominance.

tissue disorders[5]. In the present case, there were no intraoperative findings of chordal rupture in the mitral valve due to AMI; therefore, pre-existing and asymptomatic MR was highly suspected. The patient appeared to be asymptomatic prior to the onset of ATAAD. Considering these clinical findings, the pathophysiology of the intraoperative worsening of CHF might be influenced by ATAAD, such as life-threatening hypertension and volume overload subsequent to preoperative antihypertensive treatment. There have been no previous reports of ATAAD patients with worsening CHF due to MR. Aortic surgeons should consider the possibility of pre-existing MR, which can induce worsening of CHF in patients undergoing surgery for ATAAD.

In the present case, both the cardiologist and anesthesiologist could not detect the presence of MR at midnight prior to surgery. We retrospectively consulted the transesophageal echocardiographic (TEE) examination performed by the anesthesiologist. Moderate MR was clearly demonstrated by TEE during sternal entry (Fig. 3). Especially in emergent operations with deteriorated hemodynamics, not only cardiologists and anesthesiologists but also surgeons tend to pay attention to the presence of pericardial effusion and AR in preoperative patients with ATAAD. This leads to a potential risk of undiagnosed valvular diseases except in cases of aortic valve insufficiency.

According to the American Society of Anesthesiologists guidelines, TEE in ATAAD surgery is recommended as a class 1 diagnostic procedure[6]. TEE is reported to provide additional information in the management of ATAAD in 64% of patients, leading to a change of surgical strategy in 39% of patients[7]. In fact, intraoperative severe lung congestion may lead to irreversible respiratory failure. To avoid such a catastrophic event, the entire surgical team, consisting of surgeons, cardiologists, and anesthesiologists, should recognize precisely all valvular diseases and cardiac function even in an emergent setting.

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