Avoiding anticoagulation drugs on the post-operative atrial fibrillation made successful conservative treatment for left atrial dissection: A case report

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Abstract

Abstract Introduction: Left atrial dissection is a rare complication of cardiac surgery, most commonly associated with mitral valve surgery. Herein, we report on successful conservative treatment of left atrial dissection by avoiding anticoagulation. Case Report: A 64-year-old man developed left atrial dissection due to retrograde cardioplegia cannulation during operation for acute type A aortic dissection. As there was no connection between the left atrial dissection cavity and the left atrium on enhanced computed tomography, we did not administer anticoagulants to prevent expansion of the left atrial dissection cavity. However, the patient developed atrial fibrillation, which was successfully managed by beta-blocker and amiodarone administration. Follow-up imaging showed gradual left atrial dissection reduction, and the patient was started on anticoagulation therapy. Conclusion: We were able to resolve left atrial dissection by preventing the use of anticoagulation therapy in the acute stage by managing the atrial fibrillation with antiarrhythmic drugs.

Introduction

Left atrial dissection (LAD) is a rare complication of cardiac surgery. Its clinical presentation and course vary widely, from asymptomatic to severe, the latter being fatal due to hemodynamic collapse. Herein, we report we report on successful conservative treatment by avoiding anticoagulation in a case of LAD related to retrograde cardioplegia cannulation during operation for acute type A aortic dissection (ATAAD).

Case report

Akita University's Ethics Committee does not need to approve anonymized observational research. The patient provided informed consent for publication of his data.

A 64-year-old man was transferred to our hospital after being diagnosed with ATAAD. His vital signs were stable under administration of nicardipine and the echocardiography findings were unremarkable. We performed emergency total arch repair with frozen elephant trunk. Cardiopulmonary bypass (CPB) was established with bicaval drainage and left subclavian artery perfusion. We always perform antegrade infusion for cardioplegia from the ascending aorta and retrograde infusion from the coronary sinus, inserting the cannula from the right atrial appendage. We always use a self-inflating retrograde cannula (RC014T® 14Fr, Edwards Lifesciences Corp, One Edwards Way, Irvine, USA). The cannula insertion for retrograde cardioplegia was smooth. Upon infusion, the injection pressure rose to about 70 mm Hg, but it was momentarily; hence, we continued the infusion after the pressure dropped to approximately 30 mmHg, and cardiac arrest was induced. At CPB weaning, transesophageal echocardiography (TEE) showed a multivesicular space on the back of the left atrium that had no significant fistula with the left atrium (Fig. 1A,B). As the patient's vital signs were not affected, he was transferred to the intensive care unit after the operation. Follow-up TEE

again showed a space on the back of the left atrium, which at that time obstructed the left ventricle inflow. Enhanced computed tomography (CT) revealed a non-enhanced space on the back of the left atrium (Fig. 1C). We established the diagnosis of LAD. Because there was no connection between the LAD cavity and the left atrium on CT, we did not administer anticoagulant drugs to prevent expansion of the LAD cavity. However, the patient developed atrial fibrillation (AF) and his hemodynamics deteriorated. We considered that the LAD stimulated the left atrium and triggered AF. Therefore, we administered a beta-blocker (under intubation, landiolol hydrochloride was administered intravenously at 0–5 μg/kg/min, while after extubation, bisoprolol fumarate was given orally at a dose of 1.25–7.5 mg; the dose at discharge was 1.25 mg) and amiodarone, which successfully managed the AF, and the patient's hemodynamics improved.

The patient was extubated on postoperative day (POD) 5. Amiodarone administration was discontinued on POD 29, and he was started on anticoagulation therapy with a direct oral anticoagulant. We used warfarin potassium, with the dose adjusted so that the INR was approximately 2. There was residual paroxysmal AF rhythm, but the patient was hemodynamically stable. Follow-up enhanced CT performed on POD 37 showed LAD reduction (Fig. 2). The patient was discharged on POD 40.

Discussion

LAD is a rare complication of cardiac surgery, associated with mitral valve surgery in 0.16-0.84% of cases.[1,2] Tsukui et al.[1] reported a case of LAD related to retrograde cardioplegia cannulation, but they performed surgical treatment for the LAD. Other causes of LAD include insertion of left ventricular vent tube, aortic dissection, and others. In our case, the most likely cause was retrograde cardioplegia cannulation. The patient was diagnosed with ATAAD, but enhanced CT before operation did not show LAD. Furthermore, there was no resistance or any problems at insertion of the left ventricular vent tube.

The hemodynamic effects of LAD vary widely, from asymptomatic to hemodynamic collapse.[2,3] TEE, CT, magnetic resonance imaging, and catheter study are helpful for LAD detection and diagnosis. In our case, enhanced CT was useful for LAD diagnosis.

Regarding LAD treatment, according to the literature, reversal of anticoagulation could prevent LAD expansion. In our case, the follow-up CT demonstrated LAD resolution. However, in severe cases, surgical intervention should be considered. There are currently two types of surgical treatment: entry close and internal drainage.[3] In the available reviews,[2,3,4] the postoperative mortality rate was 9.8-12.7% and the overall mortality rate was 11.2-13.8%. In our case, conservative therapy was fortunately successful, but if worsening developed, we would have considered an internal drainage procedure.

Enhanced CT enabled us to diagnose LAD, which we considered was related to the retrograde cardioplegia cannulation. The coronary sinus is a low-pressure system; it might prevent inflow to the LAD cavity and LAD expansion. In addition, the LAD cavity had no connection with the left atrium, which indicated the possibility of preventing LAD expansion.

In a recent review by Cereda et al,[5] the authors recommended conservative care in cases of stable LAD. In our case, we avoided anticoagulation drug administration by managing the AF with antiarrhythmic drugs. Thus, we selected conservative treatment.

Conclusion

It is important to correctly diagnose LAD and detect any connection with another cavity. In cases where no connection is detected, conservative treatment can be considered. In addition, preventing the use of anticoagulation drugs might help to resolute LAD; in cases of postoperative AF, it is useful to administer antiarrhythmic drugs.

Figure legends

Fig. 1

A: Transesophageal echocardiography showed a multivesicular space on the back of the left atrium that had no significant fistula with the left atrium.

B: A schema of the transesophageal echocardiography findings. The grayed area was false lumen (FL) of the left atrium. The true lumen of the left atrium (asterisk) was compressed by the FL.

C, D: Enhanced computed tomography (C: axial view, D: sagittal view.) immediately after surgery showed a non-enhanced space on the back of the left atrium (arrow heads), which was not connected with the left atrium.

Fig. 2

Enhanced computed tomography on postoperative day 37 showed resolution of the left atrial dissection cavity (arrows).

Author contributions

Writing: KK

Review: TK, HY

Illustration: YI

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