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CLINICAL REPORT

Abrupt formation and spontaneous resolution of a right atrial thrombus detected by intraoperative transesophageal echocardiography during replacement of an abdominal aortic aneurysm

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Abstract Intraoperative formation of a thrombus in the right atrium and its management has occasionally been reported. However, spontaneous resolution of right atrial thrombi, without any event, is rare. We report a case of abrupt right atrial thrombus formation and spontaneous resolution, with no events, detected by transesophageal echocardiography during the replacement of an abdominal aortic aneurysm.

Keywords Thrombus · Transesophageal echocardiography · Abdominal aortic aneurysm

Introduction

Intraoperative formation of a thrombus in the right atrium is rare and may go unrecognized. However, it can be associated with severe consequences, including pulmonary embolism and death. Rapid diagnosis of a right atrial thrombus is an important factor for prognosis and can be confirmed by transesophageal echocardiography (TEE) during an operation. Here we describe a case of abrupt right atrial thrombus formation and spontaneous resolution, without any events, detected by intraoperative TEE during replacement of an abdominal aortic aneurysm (AAA).

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Case report

A 69-year-old man was admitted for replacement of an AAA. The aneurysm extended from just distal to the infrarenal artery to the bilateral common iliac artery and its maximum diameter was 89 mm. He had undergone axillo-femoral bypass to prevent growth of the AAA 1 year earlier and also had a past medical history of percutaneous transluminal coronary angioplasty (PTCA), coronary artery bypass grafting surgery (CABG), and transient ischemic attack.

Recent medications were isosorbide dinitrate 40 mg bid, digoxin 0.125 mg qd, rosuvastatin 10 mg qd, felodipine 30 mg qd, and warfarin 2 mg qd. He stopped warfarin on hospitalization for the operation.

Preoperative evaluations were conducted. Electrocardiography showed atrial flutter. Transthoracic echocardiography showed no systolic functional abnormality (left ventricular ejection fraction was 59%, although there was akinesia on the posterolateral wall). Coagulation tests were within normal ranges (prothrombin time, 14.2 s; activated partial thromboplastin time, 40.8 s; fibrinogen, 204 mg/dL; platelet count, 95000/μL; D-dimer, 1.2 μg/dL).

After preparing routine invasive (invasive arterial blood pressure) and noninvasive (electrocardiography, pulse oximetry, and bispectral index) patient monitoring, cerebral oximetry (INVOS® Cerebral/Somatic Oximeter, Somanetics, Troy, USA) was attached to the patient's forehead to monitor cerebral perfusion upon his arrival at the operating room. Activated clotting time (ACT) was measured and was 167 s. Anesthesia was induced with caution, to prevent abrupt blood pressure changes, and maintained using target-controlled infusion of propofol (target concentration, 1.2–1.3 μg/ml) and remifentanil (target concentration, 10–20 ng/ml). Muscle relaxation was

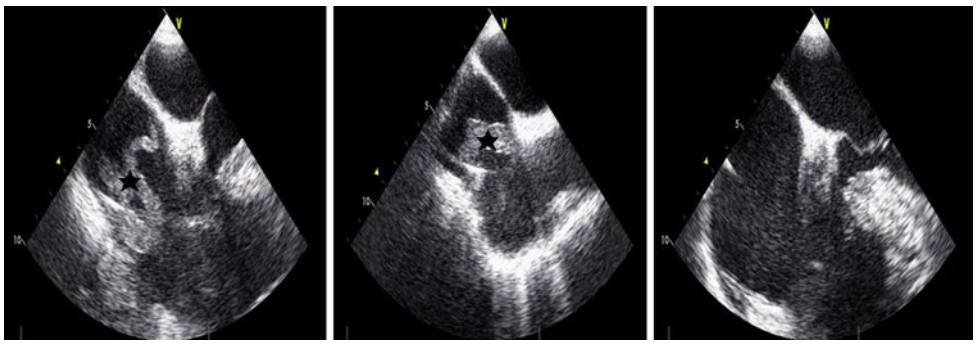

Fig. 1 Transesophageal echocardiography (mid-esophageal four-chamber and bicaval view) showing a right atrial thrombus (*filled star*) from abrupt formation (*left* and *middle*) to spontaneous resolution (*right*)

achieved by administration of a bolus of rocuronium under the guidance of peripheral monitoring of neuromuscular transmission. A pulmonary artery catheter (Swan-Ganz CCOmbo CCO/SvO_2®, Edwards Lifesciences, Irvine, USA) was placed through a 9F percutaneous introducer sheath (Advanced Venous Access High-Flow Device, Edwards Lifesciences, Irvine, USA) at right internal jugular vein without any complications.

A TEE probe (6T probe, GE Healthcare, USA) was inserted. TEE (Vivid 7, GE Healthcare) showed severe atherosclerotic changes in the thoracic aorta and akinesia in the posterolateral wall of the left ventricle, without systolic functional abnormality and any apparent valvular abnormality. There was no thrombus in the cardiac chamber. Atrial septal defect or patent foramen ovale was not observed.

Aortic cross clamping was done without systemic heparinization. Replacement of the AAA between the infrarenal artery and the bilateral common iliac artery level was performed at normothermia. No specific change was observed by TEE during the procedure. The aortic cross clamp time was 67 min. Two units of packed red blood cells were transfused. After replacement of the AAA, TEE detected a 3 × 4-cm, non-mobile, echogenic shadow, considered to be a thrombus, anchored in the right atrium (Fig. 1). Although proper treatment for the right atrial thrombus was needed, this was delayed until bleeding control was achieved around the AAA. At that time, ACT was 197 s. During bleeding control, the right atrial thrombus had no specific changes. There were no abnormal findings on right heart and pulmonary trunk. Left atrial appendage had no thrombus with flow velocity of above 0.4 m/s on pulsed Doppler. Regional wall motion was good except for akinesia of the posterolateral wall, as it did preoperatively and intraoperatively. After 30 min, TEE was performed to decide on the optimum treatment for the thrombus (anticoagulants vs. surgical removal). The shadow spontaneously disappeared (Fig. 1). There were no specific changes on the monitor. Changes in electrocardiography or capnography (end-tidal carbon dioxide pressure: 36 → 37 mmHg) were not recorded. No hemodynamic change, such as a decrease in systemic blood pressure (103/54 → 105/49 mmHg) or an increase in pulmonary artery blood pressure (21/13 → 21/12 mmHg), was seen. The volume was infused in the range 30 to 50 ml/h through a rapid infusion system (Belmont Instrument Fluid Management System (FMS) 2000™, Belmont Instrument, Billerica, USA) during bleeding control around the AAA.

On completion of the operation, the patient was transferred to the intensive care unit (ICU). Postoperative coagulation tests showed prothrombin time of 19.0 s, activated partial thromboplastin time of 44.3 s, fibrinogen of 114 mg/dL, platelet count of 51000/μL, and D-dimer of 10.7 μg/dL.

On the first postoperative day in the ICU the patient was able to open his eyes and obey commands to raise his legs. After 5 days, the patient made an uneventful recovery and was transferred to a general ward.

Discussion

Classically, Virchow's triad of risk factors for the development of thrombi includes hypercoagulability, blood stasis, and the presence of an abnormal endothelial surface [1]. The patient stopped taking warfarin. Preoperative and intraoperative electrocardiography showed atrial flutter (the risk factor of thromboembolism in atrial flutter is controversial but atrial flutter with history of previous atrial fibrillation history was a risk factor of thromboembolism [2, 3]), and TEE showed severe atherosclerotic changes in the thoracic aorta. Overall, the patient had a high risk of thromboembolism, although preoperative coagulation tests were within normal ranges.

The use of heparin was avoided because of bleeding control around the AAA and ACT of 195 s, despite the detection of the right atrial thrombus by TEE. Heparin may have increased bleeding, disturbed bleeding control, and required more transfusion, although not using it may have

facilitated thrombus formation. The possibility of increased bleeding with heparin and, perhaps more importantly, worries about toxicity and the direct anticoagulant effects of protamine sulfate, have led many surgeons to avoid heparin altogether [4]. Additionally, heparin is not necessary to prevent distal thrombosis during aorta cross clamping [5]. However, Thompson and colleagues [5] reported that heparin did not increase blood loss or the need for blood transfusion during replacement of an AAA and was an important prophylactic agent against peri-operative myocardial infarction in relation to replacement of an AAA. The patient had a medical history of axillo-femoral bypass, PTCA, and CABG. Thus, heparin should have been injected before aortic cross clamping to prevent thromboembolism and peri-operative myocardial infarction. Actually, careful consideration is needed to decide whether bleeding or pulmonary thromboembolism is more important. However, bleeding can be completely resolved with equipment and blood products whereas pulmonary thromboembolism cannot be completely resolved without rapid diagnosis and proper treatment.

Right atrial thrombi occur in two types (types A and B), according to morphology, etiology, pathophysiology, and prognosis. Type A appears to be long, thin, and mobile (worm-like or snake-like). It has a high incidence in deep-vein thrombosis and is almost always associated with pulmonary embolism. Its prognosis is poor. Type B is a less mobile, non-specific thrombus with morphology similar to that of a thrombus in the left heart and predominantly represents localized intracardiac thrombosis. It is not commonly associated with deep-vein thrombosis or pulmonary embolism [6]. The thrombus in this case was not long, thin, and mobile; it was apparently a type B thrombus. Thus, it might be expected to resolve spontaneously without any serious complication, such as pulmonary embolism. Perhaps, the thrombus broke into smaller pieces and moved on through the vascular tree, not influencing hemodynamics. If a 3 × 4-cm echogenic mass in right atrium flowed away, it would disturb pulmonary blood flow and result in hemodynamic change. However, there was no specific hemodynamic change in this case. Rose and colleagues reported that 16 out of 177 patients who were identified as having a right heart thromboembolism received no therapy [7]. Although the cases could not be checked, the most likely explanation is that peripheral clots stopped temporarily in the right atrium and then either broke up or moved into the pulmonary circulation, as may have occurred in this case. A ventilation/perfusion scan might be helpful postoperatively, but was not performed. D-dimer for suspecting pulmonary embolism was elevated (cut-off value in our hospital: 0.4 μg/dL) in this case but the possibility of false positive had to be considered [8–10].

Abrupt right atrial thrombus formation and spontaneous resolution, without any event, may go unrecognized. Thus, the actual incidence of right atrial thrombus may be higher than reported. More studies are needed to determine how and when a right atrial thrombus can spontaneously resolve, without events.

Treatments for a right atrial thrombus are anticoagulation with heparin, administration of thrombolytic agents, or surgical removal [11]. The most effective therapy remains unknown. This is a key issue, because the presence of a right atrial thrombus, complicating a pulmonary thromboembolism, seems to carry a poor prognosis [11, 12]. The mortality rate for right heart thromboembolism is 45% and all of these deaths occur within the first 24 h, underscoring the need to rapidly diagnose and treat right heart thromboembolism [13]. Therefore, rapid and proper treatment should be performed when right atrial thrombus is detected although the type of thrombus, preexisting pulmonary thromboembolism, cardiopulmonary reserve, comorbid illnesses, etc., should be considered. The treatment should not be delayed from only morphological diagnosis. In our case the outcome was satisfactory but anticoagulation should have been performed when right atrial thrombus, without regard of the type, was detected.

A retrospective review of the incidence of right atrial thrombi in more than 60,000 patient echocardiograms identified only 20 patients with the diagnosis during a 7-year period [14, 15]. However, nearly 100% of the detected cases were associated with the presence of proven pulmonary embolisms. Many cases go unrecognized. Schwartzbard and colleagues [15] reported that TEE had a significant effect on the diagnosis and management of patients with right atrial thrombi. Although the application of intraoperative TEE continues to expand, it is not routine for the replacement of an AAA. However, intraoperative TEE can contribute to altering patient management during replacement of an AAA, as in this case. TEE has a key-role of decision making. When an unusual cardiac mass is seen on TEE, normal anatomical variants must be distinguished from pathological entities. Normal structure, such as crista terminalis, eustachian valve, thebesian valve, chiari network, right atrial appendage, pectinate muscles and so on, can be misunderstood as a right atrial thrombus [16]. Although the whole TEE examination has to follow guidelines [17], mid-esophageal (ME) right ventricle inflow-outflow, ME ascending aortic short or long axis, ME aortic valve long axis, and upper-esophageal aortic arch short axis views were important for evaluation of pulmonary thromboembolism when a right atrial thrombus was detected. Left atrial appendage, through ME two-chamber view, and left ventricle was evaluated to rule out left heart thrombus because of atrial flutter and past medical history of PTCA and CABG in our case. Measuring

flow velocity with pulsed Doppler approximately 1 cm from the entry of left atrial appendage was helpful to identify blood stasis. Values less than 0.4 m/s are associated with an increased of thrombus formation [16]. Actually, we almost continuously monitored TEE for evaluation of a right atrial thrombus and early detection of pulmonary thromboembolism but did not observe resolution of a right atrial thrombus. Spontaneous echogenic contrast, key-image, representing gradual breaking down of a right atrial thrombus, might be helpful to discriminate between its abrupt disappearance and its gradual breaking down, and to count the time from its appearance to its disappearance [18], but unfortunately could not be obtained. The size of the cardiac chamber was in the normal range and not changed before and after abrupt formation and spontaneous resolution of the right atrial thrombus. There were no abnormal findings of the pulmonary trunk. Secondary signs of pulmonary artery obstruction (right ventricular dysfunction, tricuspid regurgitation, and leftward bowing of interatrial septum) [19] were not observed after spontaneous resolution of a right atrial thrombus.

In conclusion, we have reported a rare case that showed abrupt right atrial thrombus formation and spontaneous resolution, without any event, detected by intraoperative TEE during replacement of an AAA. We discuss the use of heparin in patients at high risk of right atrial thrombus formation and the utility of intraoperative TEE during replacement of an AAA.

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