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Case Report

De Novo Left Atrial Appendage Thrombus Formation During Weaning from Cardiopulmonary Bypass in Redo Bentall Surgery

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Abstract

Left atrial appendage (LAA) thrombus formation during cardiac surgery is rare but poten-tially catastrophic because of the risk of systemic embolization and perioperative stroke. We report a 61-year-old man with hypertension, diabetes, heart failure, and severe coro-nary artery disease who underwent redo Bentall operation for paravalvular leakage after a prior aortic valve replacement. He was in sinus rhythm, and pre-bypass transesophageal echocardiography (TEE) showed no LAA thrombus. Heparin achieved an activated clot-ting time of 511 s; cardiopulmonary bypass (CPB) and cross-clamp times were 342 and 312 min under moderate hypothermia. During the weaning from CPB, TEE incidentally detected a new LAA thrombus. Partial LAA ligation and thrombus removal were per-formed; residual mural thrombus remained, but no embolism or neurologic deficit oc-curred. Follow-up transthoracic echocardiography showed no residual thrombus by postoperative day 7 and 20. Routine coagulation tests and a limited thrombophilia evalu-ation were unremarkable, while the initially suspected diagnosis of Behçet disease was not supported by established classification criteria. Prolonged CPB with transient low-flow conditions and pre-existing left atrial enlargement may have promoted local blood stasis and contributed to thrombus formation. This case highlights that de novo LAA thrombus can develop during CPB weaning despite adequate systemic hepariniza-tion and apparently adequate activated clotting time (ACT), even in a patient without atrial fibrillation or mitral stenosis. Systematic TEE reassessment during CPB weaning may facilitate early recognition and prompt management of this rare complication.

Keywords: Behçet disease; cardiopulmonary bypass; intracardiac thrombus; left atrial appendage; redo Bentall operation; transesophageal echocardiography; tranexamic acid

Abbreviations: ACT: activated clotting time; aPTT: activated partial thromboplastin time; AVR: aortic valve replacement; CPB: cardiopulmonary bypass; ICA: internal carotid artery; ICBD: International Criteria for Behçet's Disease; ISG: International Study Group (for Behçet's disease); LAA: left atrial appendage; LVEF: left ventricular ejection fraction; POD: postoperative day; TEE: transesophageal echocardiography; TTE: transthoracic echocardiography; TXA: tranexamic acid

Introduction

The left atrial appendage (LAA) is a major site of intracardiac thrombus formation, particularly in patients with atrial fibrillation or impaired atrial mechanical function [1]. Left atrial thrombus in patients in sinus rhythm is uncommon [2], and its de novo formation during cardiac surgery is clinically important because embolization can result in ischemic stroke or systemic arterial occlusion [1,3]. Cardiac surgery involving cardio-pulmonary bypass (CPB) induces complex and dynamic changes in coagulation, platelet function, fibrinolysis, endothelial function, and intracardiac blood flow [4]. Although sys-temic heparinization is routinely used during CPB [5], adequate prolongation of the acti-vated clotting time (ACT) does not necessarily exclude localized intracardiac thrombus formation [3,6].

We report a case of de novo LAA thrombus formation detected by sequential in-traoperative transesophageal echocardiography (TEE) in a patient undergoing redo Bentall surgery. The thrombus developed during weaning from CPB despite preoperative sinus rhythm and the absence of LAA thrombus on pre-bypass imaging. Potential con-tributing factors relevant to this case are discussed below.

Detailed Case Description

A 61-year-old man was admitted for redo cardiac surgery. He had undergone aortic valve replacement (AVR) for severe aortic regurgitation on 20 November 2024. Approximately 1 year later, follow-up echocardiography demonstrated newly developed paravalvular leakage.

The patient was 170 cm tall and weighed 69 kg. His medical history included hyper-tension, diabetes mellitus, heart failure, and severe coronary artery disease. He had no history of atrial fibrillation or other arrhythmias. He had reported recurrent oral ulceration and facial skin lesions for approximately 1 year before AVR, for which Behçet disease was considered as a differential diagnosis. Both findings resolved after the previous surgery; the facial lesions were diagnosed as adult acne on dermatologic consultation, and oral ulceration subsequently recurred once. There was no genital ulceration, erythema nodosum, pseudofolliculitis, ocular involvement, or pathergy reaction. The patient there-fore did not fulfill the 1990 International Study Group (ISG) criteria and scored only two points (recurrent oral aphthosis) on the International Criteria for Behçet's Disease (ICBD), below the ≥4-point threshold [7,8]. However, he was receiving empirical prednisolone 60 mg/day for suspected Behçet disease.

Preoperative platelet count (196 × 10^3/μL), prothrombin time-international normal-ized ratio (1.03), and activated partial thromboplastin time (aPTT) (32.1 s) were within normal ranges. A limited thrombophilia evaluation showed unremarkable results, with antithrombin III 83%, protein C activity/antigen 112%/104.5%, and protein S activi-ty/antigen 66%/91.1%, respectively. The collagen/epinephrine closure time was prolonged at >300 s. Antiphospholipid antibodies, lupus anticoagulant, and hereditary thrombo-philia mutations were not assessed.

Preoperative electrocardiography showed sinus bradycardia (54 bpm). Transthoracic echocardiography (TTE) demonstrated a left ventricular ejection fraction (LVEF) 55–60%, left atrial enlargement, and aneurysmal change of the aortic annulus with more than moderate paravalvular leakage; pseudoaneurysm or periannular abscess was considered in the differential diagnosis. Coronary computed tomography angiography confirmed se-vere coronary artery disease (Coronary Artery Disease Reporting and Data System 4b) with a paravalvular defect (<1 cm) at the right coronary sinus of Valsalva and abnormal aortic root–left ventricular outflow tract communication.

Brain MRI/MRA demonstrated chronic left internal carotid artery (ICA) occlusion with cross-filling. Computed tomography perfusion showed impaired cerebrovascular reserve, indicating an increased risk of perioperative cerebral ischemia.

Standard multimodal monitoring, including cerebral oximetry and TEE, was per-formed during general anesthesia. Pre-bypass TEE showed no thrombus within the LAA. Tranexamic acid (TXA) 1000 mg was administered shortly after induction of anesthesia. Once preparation for cannulation was complete, heparin 210 mg (3 mg/kg) was adminis-tered, increasing the ACT from 110 to 511 s. CPB was maintained for 342 min under mod-erate hypothermia, with a minimum nasopharyngeal/bladder temperature of 26.8 °C. The aortic cross-clamp time was 312 min. During weaning from CPB, TEE revealed a new echogenic mass within the LAA that had not been present on pre-bypass imaging, con-sistent with thrombus (Figure 1). Emergent partial LAA ligation and surgical throm-bectomy were performed. Most of the thrombotic material was removed, although residual mural thrombus remained (Figure 2). After reversal of heparin with protamine 210 mg, the ACT

decreased to 121 s. The patient remained hemodynamically stable with dobutamine and norepinephrine support. Gross hematuria was managed with fresh frozen plasma, apheresis platelets, and cryoprecipitate.

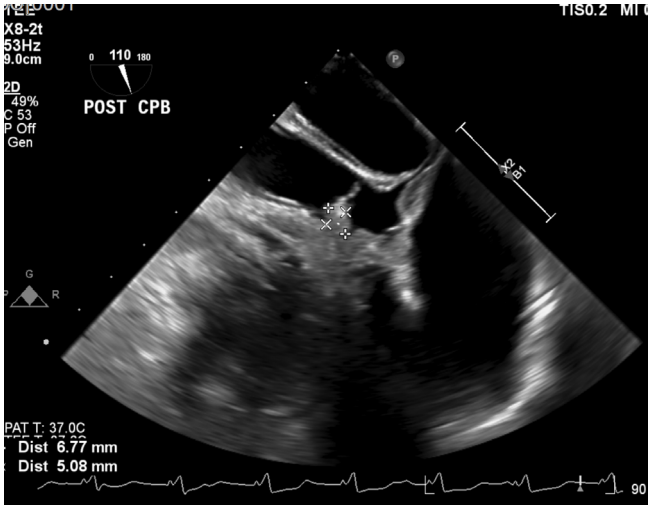

Figure 1: Intraoperative transesophageal echocardiography (TEE) demonstrating a newly formed thrombus in the left atrial appendage (LAA) during weaning from cardiopulmonary bypass (CPB). The thrombus was not present on pre-bypass imaging.

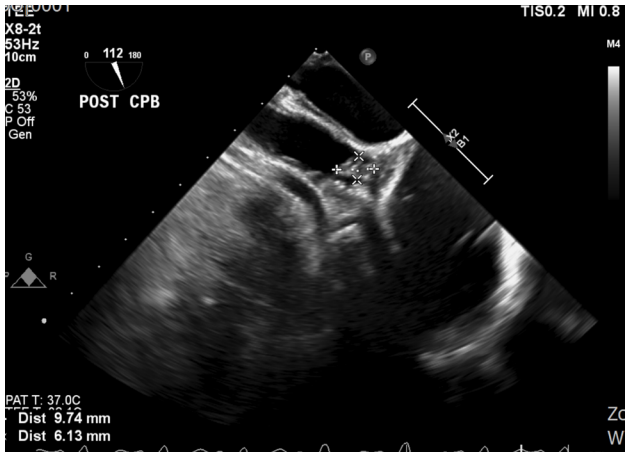

Figure 2: Residual mural thrombus within the left atrial appendage (LAA) after removal of the primary thrombus.

Preoperative blood culture and intraoperative tissue cultures were negative. On gross surgical inspection, the prosthetic aortic valve showed dehiscence (7–8 mm) between the non-coronary and right coronary cusps, the sinus of Valsalva appeared markedly in-flamed, and pannus-like tissue was noted in the subvalvular area. Histopathologic ex-amination of the dissected aortic wall was consistent with aortic dissection, and the aortic valve showed myxoid degeneration and foreign body granuloma with metallic material. The surgically removed LAA specimen yielded no pathologic diagnosis. Accordingly, the diagnosis of LAA thrombus was based on the sequential TEE findings and direct surgical observation.

The patient was extubated on postoperative day (POD) 1. Aspirin was initiated on POD 1 and warfarin on POD 2. Follow-up TTE on POD 7 showed no intracardiac throm-bus. The patient was discharged on POD 13 without major complications, and outpatient TTE on POD 20 again showed no intracardiac thrombus. Despite the intraoperative LAA thrombus and substantial baseline cerebrovascular risk, no clinically apparent neurologic deficit or systemic embolic event occurred. A summary of the perioperative timeline is presented in (Table 1).

Time / POD	Event
Preoperative days	Hypertension, diabetes mellitus, AVR due to severe aortic regurgitation and heart failure [2 years ago], severe coronary artery disease ECG: sinus bradycardia (54 bpm) TTE: no intracardiac thrombus, moderate paravalvular leakage Rule out Behçet disease (ISG-negative, ICBD two points)
Pre-CPB	TEE: absence of LAA thrombus Baseline ACT 110 s, TXA 1000 mg → heparin 210 mg (3 mg/kg) → ACT 511 s CPB (minimum temp. 26.8 °C) and aortic cross-clamp redo Bentall operation
CPB weaning	De novo LAA thrombus on TEE (Figure 1) partial ligation and extraction residual mural thrombus (Figure 2)
Post-CPB	CPB discontinued (total CPB time 342 min, cross-clamp 312 min) Protamine 210 mg; ACT 121 s
POD 1–2	Extubation aspirin (POD 1) and warfarin (POD 2) initiated ICU → ward
POD 7	TTE: No intracardiac thrombus
POD 13	Discharged without major complication (no embolic or neurologic event)
POD 20	Outpatient TTE: No intracardiac thrombus
Abbreviations: ACT, activated clotting time; AVR, aortic valve replacement; CPB, cardiopulmonary bypass; ECG, electrocardiography; ICBD, International Criteria for Behçet's Disease; ISG, International Study Group; LAA, left atrial appendage; POD, postoperative day; TEE, transesophageal echocardiography; TTE, transthoracic echocardiography; TXA, tranexamic acid	

Table 1: Condensed perioperative timeline.

Discussion

This case demonstrates de novo LAA thrombus formation detected by TEE during weaning from CPB in a patient with sinus rhythm and no LAA thrombus on pre-bypass imaging. The event occurred during redo Bentall surgery, a technically demanding pro-cedure that in this case involved prolonged CPB and aortic cross-clamp times.

Acute intracardiac thrombosis during or after CPB is rare, but similar events have been reported despite full heparinization and apparently adequate ACT [3,6,9]. In a sys-tematic review by Williams et al., prolonged CPB, impaired cardiac function, vascular in-jury, and hemostatic interventions were reported among cases of acute intracardiac thrombosis or pulmonary thromboembolism associated with CPB [9]. Of particular rele-vance, Kim et al. described de novo LAA thrombus formation during CPB despite ade-quate ACT in a patient with chronic atrial fibrillation and mitral stenosis, both strong pre-existing substrates for LAA thrombosis [3]. Another report described a mobile left atri-al thrombus that developed during CPB weaning in a patient with pre-existing atrial fi-brillation undergoing combined valve repair and a maze procedure [6]. In contrast to these reports, our patient had neither atrial fibrillation nor mitral stenosis. However, the pres-ence of prosthetic valve dehiscence and paravalvular leak indicates that structural valvu-lar pathology, rather than the complete absence of predisposing factors, may have con-tributed to thrombus formation.

Intracardiac thrombosis can be conceptualized within Virchow's triad: blood stasis, endothelial injury, and hypercoagulability [10]. In the present case, stasis appears to have been the most important mechanism. Evidence supporting a marked systemic hyperco-agulable state was limited. Routine preoperative coagulation tests and the limited throm-bophilia panel were unremarkable. Adequate heparinization was retained during CPB with increased the ACT to 511 s. ACT is a global measure of whole-blood coagulation; however, it does not directly assess local intracardiac flow, platelet activation, or fibrino-lytic activity; therefore, an adequate ACT cannot exclude localized thrombogenesis [3,6]. The absence of testing for antiphospholipid antibodies and hereditary thrombophilia should also be acknowledged. Histopathologic examination argued against an inflam-matory cause of endothelial injury. No vasculitis was described in the pathology report of the submitted LAA specimen, making overt inflammatory vascular injury such as that associated with Behçet disease unlikely, although the sensitivity of histopathology may have been reduced by empirical prednisolone therapy and limited representation of the appendage wall. Although mechanical or functional injury related to cannulation, ische-mia-reperfusion, or surgical manipulation may also occur and is difficult to identify by routine histopathology, the pathologic findings do not clearly demonstrate the occurrence of thrombus due to endothelial injury. In contrast, several conditions favored blood stasis. CPB duration exceeded 3 h in 37.5% of previously reported cases of post-CPB intracardiac thrombosis [9], and the CPB duration in our patient (342 min) also exceeded this threshold. Prolonged left-heart decompression followed by reperfusion and cardiac refilling during weaning may have produced transient regions of low intracardiac flow. In addition, the complex trabeculated anatomy of the LAA [1] and pre-existing left atrial enlargement may have further promoted local stasis. Taken together, these findings suggest that blood stasis may be an important contributor to de novo thrombus formation in this patient, although other mechanisms, including mechanical endothelial injury and coagulation-related fac-tors, cannot be excluded.

Additional, less certain contributors that could be considered in this patient include Behçet disease and TXA administration. Behçet disease is a systemic vasculitis associated with vascular thrombosis and, less commonly, intracardiac thrombosis, which has his-torically been reported predominantly in the right-sided cardiac chambers [11–14]. How-ever, the patient did not meet either the ISG or ICBD classification criteria, and no vascu-litis was described in the pathology report. Behçet disease was therefore considered in the differential diagnosis but could not be established as an explanation for the thrombus formation. TXA, meanwhile, inhibits fibrinolysis by interfering with plasminogen binding and activation [15], and could theoretically contribute to thrombus formation or persis-tence. However, a large randomized controlled trial involving 4,631 patients undergoing coronary artery surgery found no increase in death or thrombotic complications with TXA use [16]. In the present case, TXA was administered before thrombus formation, but this temporal associ-ation alone is insufficient to establish causality.

Sequential intraoperative TEE is a major strength of this case. Pre-bypass TEE showed no LAA thrombus, whereas repeat examination during CPB weaning revealed a newly developed echogenic mass. This temporal change supports de novo thrombus for-mation during surgery rather than incidental detection of a pre-existing lesion. Compre-hensive intraoperative TEE is recommended as a systematic diagnostic tool in cardiac surgery [17], and the present case suggests that reassessing intracardiac structures, in-cluding the LAA, may be clinically valuable, particularly after prolonged CPB or in the presence of additional thrombotic risk factors. Early recognition of the thrombus can allow immediate surgical intervention.

This case report has several limitations. First, as a single case, it cannot establish causality, and the findings have limited generalizability. Second, follow-up was limited to 20 days,

precluding assessment of long-term thrombus recurrence or clinical outcomes. Third, the thrombophilia evaluation was incomplete because antiphospholipid antibodies, lupus anticoagulant, and hereditary thrombophilia were not tested. Fourth, the diagnosis of LAA thrombus was based on sequential TEE findings and intraoperative observation rather than histopathologic confirmation, and the submitted LAA specimen was nondi-agnostic. Pre-bypass TEE may also have missed a small pre-existing thrombus. Fifth, the assessment for Behçet disease reflected the clinical findings and classification criteria at a single time point, and prior corticosteroid therapy may have masked clinical and histo-pathologic features. Finally, several potential contributors, including prolonged CPB, TXA exposure, hypothermia, and a possible underlying inflammatory predisposition, were present simultaneously, so their relative contributions to thrombus formation could not be clearly distinguished.

Further studies, including larger case series and mechanistic investigations of in-traoperative thrombogenesis during cardiopulmonary bypass, are warranted to clarify the relative contributions of these factors and to establish evidence-based strategies for pre-vention and early detection.

Conclusions

De novo LAA thrombus can develop during weaning from CPB despite systemic heparinization and an apparently adequate ACT, even in patients who remain in sinus rhythm and lack conventional predisposing factors such as atrial fibrillation or mitral stenosis. In this case, prolonged CPB, transient low-flow conditions during cardiac reloading, and pre-existing left atrial enlargement may have contributed to local blood stasis. These findings support systematic TEE reassessment of the LAA before separation from CPB to enable early detection and timely management of this rare but potentially devastating complication.

Supplementary Materials: Not applicable.

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