Right atrial thrombosis and pulmonary embolism after atrial septal defect repair

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Abstract

A 3-year-old boy underwent surgical closure of a large ostium secundum atrial septal defect. This was complicated with extensive right atrial thrombus formation and pulmonary thromboembolism immediately following surgery. He was managed with emergency surgical thromboembolectomy and anticoagulation. However, new thrombus was formed again immediately. This prompted us to add thrombolysis to his treatment, but with no effect. He died on the fifth postoperative day. A postmortem study confirmed extensive thromboembolism.

Keywords: CHD • Septal defects • Pulmonary embolism • Coagulants surgery • Complications • Thrombosis

INTRODUCTION

Right atrial thrombus formation is a recognized complication of percutaneous device closure of atrial septal defects (ASDs) [1]. However, this is rare following direct surgical closure, with very few reports in the literature [2-5]. In those few reported cases, the thrombosis had occurred on the suture line of the ASD closure, but none had occurred immediately following surgery (11 days to 1 year following surgery). We present a case of hyperacute extensive right atrial thrombosis and diffuse bilateral pulmonary thromboembolism following surgical ASD closure.

CASE REPORT

A 3-year-old boy, weighing 13 kg, with an isolated ostium secundum ASD and normal pulmonary arterial pressure, underwent surgical closure of this defect. His preoperative coagulation profile was normal. The procedure was performed in a routine fashion with general anesthesia, median sternotomy, normothermic cardiopulmonary bypass (25 min), and cold blood cardioplegia (cross-clamp time: 10 min). The anesthesia was conventional with fentanyl, sevoflurane, midazolam, and cisatracurium. Cardiopulmonary bypass was established after the patient was anticoagulated with 55 mg of heparin, which was reversed at the end of the procedure with 45 mg of protamine. Our bypass protocol included three boluses of tranexamic acid (10 mg kg⁻¹) at the beginning of the procedure, during bypass, and after the administration of protamine. The procedure was uneventful. The defect was closed directly, without any tension in the suture line, with a 5/0 polypropylene suture. However, the patient suddenly became severely hypotensive, bradycardic, and hypoxemic while on the way to the intensive care unit. He required aggressive resuscitation with high doses of three inotropic agents (dopamine, dobutamine, and adrenaline) and ventilation at high pressures with 100% oxygen. Urgent echocardiography showed large amounts of thrombus in the right atrium and signs of pulmonary hypertension suggesting pulmonary thromboembolism (Fig. 1). He was taken back to the operating room urgently where his chest was reopened and he was put on cardiopulmonary bypass again. Large amounts of thrombus were removed from the right atrium. Pulmonary embolectomy was attempted but no thrombus could be retrieved, suggesting that the thrombus had broken down and embolized to small pulmonary arterial branches. The patient was weaned off cardiopulmonary bypass on the same high doses of inotropic support, without any significant impact on oxygen saturations. It was decided not to administer protamine and to start an infusion of heparin (1 mg kg⁻¹ min⁻¹). Echocardiography showed new thrombus formation in the right atrium and persistence of the signs of pulmonary hypertension. There was also a small residual ASD at the inferior end of the atrial septum with a right-to-left shunt. These new thrombi were impinging on the opening of the superior and inferior venae cavae. This prompted us to proceed to thrombolysis with recombinant thromboplastin activator (r-TPA) at a rate of 0.1 μg kg⁻¹ h⁻¹, increasing it gradually to 0.4 μg kg⁻¹ h⁻¹. The patient did not improve over the course of the following 24 h and developed upper body edema despite the combination of intravenous heparin and r-TPA. This strong thrombotic tendency discouraged us from using ECMO, although no clot was detected within the oxygenator of the
ASD suture line had dehisced. There were no other defects. Structurally normal and free of thrombus. The inferior end of the atrium also contained a large thrombus. Both venae cavae were bilateral peripheral pulmonary thromboembolism. The left immediacy, and its resistance to all antithrombotic and three reasons, namely the extent and size of the thrombus, its fth postoperative day. A postmortem study showed a near total filling of the right atrium with thrombus and diffuse bilateral peripheral pulmonary thromboembolism. The left atrium also contained a large thrombus. Both venae cavae were structurally normal and free of thrombus. The inferior end of the ASD suture line had dehisced. There were no other defects.

**DISCUSSION**

Right atrial thrombus formation following ASD closure is not new, nor is its association with pulmonary thromboembolism. It is also known that when pulmonary thromboembolism complicates right atrial thrombosis, the risk of mortality increases significantly [6]. However, the case presented here is unusual for three reasons, namely the extent and size of the thrombus, its immediacy, and its resistance to all antithrombotic and thrombolytic measures. This aggressive complication escapes our understanding, especially in the presence of completely normal detailed coagulation and hematological studies. Consequently, we can only hypothesize as to its etiology. We can think of two possible explanations.

One possibility is that the combination of the presence of a foreign object (the suture material), the surgical act, and the hemodynamic conditions of the right atrium may create a strongly prothrombotic environment within this chamber [2]. It is conceivable that this complication may not be as rare as one might think, but that many mild cases escape diagnosis. Indeed, we have diagnosed one such mild case with no clinical repercussion in our unit, on postoperative echocardiography. But the present case is the first severe one with a fatal outcome.

The second theoretical possibility is the use of tranexamic acid as part of our standard hemostatic protocol. This antifibrinolytic agent may have created conceivably a prothrombotic state akin to that which used to be seen with aprotinin [7]. However, tranexamic acid is generally regarded as a safe drug in this respect [7, 8] and has never been reported to cause such an exaggerated thromboembolic response. Nevertheless, the mechanism of action of this agent is such that the creation of a prothrombotic state may not be ruled out, as has been previously mentioned [8].

In view of the theoretical nature of the above explanations, it is difficult to make any specific recommendation with certainty. However, since other publications have also reported thrombus formation in the right atrium following ASD closure, it may be that the right atrium is indeed hemodynamically prone to this complication. Therefore, it may be argued not to use any procoagulant agent when operating on this chamber. It may even be argued to put such patients on aspirin, or even anticoagulate them, for a few months following surgery, as is done following catheter-based device closure of ASDs.

**Conflict of interest:** none declared.

**REFERENCES**


