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*Interact CardioVasc Thorac Surg* 2004;3:675-677

DOI: 10.1016/j.icvts.2004.07.012

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Case report - Valves

# Native aortic valve thrombosis: a rare cause of acute ischemia of the lower limb

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Received 4 May 2004; received in revised form 8 July 2004; accepted 12 July 2004,

## Abstract

Spontaneous native aortic valve thrombosis is an uncommon event occurring after heart valve disease, during cardiac catheterization, bacterial endocarditis, or as a hypercoagulable state as in antiphospholipid antibody syndrome. We report the case of a 55-year-old woman in whom thrombi developed on a native aortic valve with no predisposing cause. The thrombi were responsible for recurrent lower limb ischemia. Eighteen months after aortic valve replacement, the patient is doing well.

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**Keywords:** Aortic valve; Thrombosis; Lower limb ischemia

## 1. Introduction

Native aortic valve thrombosis is a rare disorder. Six adult cases have been published to date and they were always associated with heart valve disease [1–3], heart valve replacement [4], a hypercoagulable state [5] or an auto-immune disease [6]. We report a case of native aortic valve thrombosis without any valvular or biological factor in a patient presenting a history of systemic embolism.

## 2. Case report

A 55-year-old woman was admitted for acute ischemia of the left lower limb. Clinical examination revealed no heart murmur and iliac thrombosis was confirmed by the absence of left femoral pulse. Biological work-up revealed a leukocytosis at 12,000/mm<sup>3</sup>. No other hematological abnormalities were found. Coagulogram, chest X-rays and ECG were strictly normal. Heparin was started after

the diagnosis and thrombectomy, performed by a left femoral approach, allowed the extraction of a left iliac thrombus 9 cm long. A complete arteriogram ensuring the patency of iliac and distal arteries concluded the surgery. The symptoms disappeared in the immediate post-operative period. However, 4 h later, she suddenly presented signs of acute ischemia in the right lower limb. Arterial Doppler revealed a thrombus in the right popliteal artery. By an infrageniculate medial popliteal approach, a thrombectomy was performed and a whitish 2 cm thrombus was extracted. Due to the occurrence of multiple emboli, a transesophageal cardiac sonography was performed immediately after the second surgery. It demonstrated a 13-mm anechoic formation on the tricuspid aortic valve located at the junction of the left and right coronary arteries (Fig. 1) without evidence of stenosis or aortic insufficiency. The aortic valve was explored surgically immediately after echocardiography under extracorporeal circulation, hemodilution and general hypothermia at 34 °C. The aortic valve was tricuspid, not dystrophic, and at the left and right coronary cusp junctions, there was a continuous tumor over 1 cm long which could not be excised with conservation of the valve. The valve was replaced by an Ultracor 23 mechanical valve. The post-operative period was uneventful both in vascular and cardiac terms.

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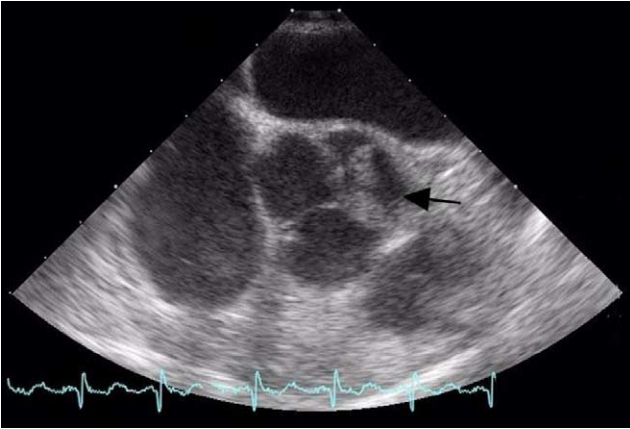


Fig. 1. Transesophageal echocardiography. Anechoic lesion at the right and left cusp coronary junctions.

Both valve thrombus and peripheral emboli pathological analysis (Fig. 2a–d) demonstrated a thrombus containing platelets, eosinophilic granulocytes and eosinophilic pigments. It was fibrinous with an extensive leukocytic infiltration, rich in granulocytes and in lytic enzymes of the matrix (metalloproteases 9). Numerous cavities were found but they were not stained with endothelial markers. The sigmoid valves presented no particular dystrophy, especially in their endothelium. Valve culture was negative

and there was no evidence of endocarditis. Search for auto-antibodies (anti-phospholipids, anti-cardiolipin, lupus anticoagulant, anti-nucleus, anti- $\beta$ 2GPI antibodies), tumoral and coagulopathy markers (prothrombin gene mutation, deficit in protein C-S, anti-thrombin III, resistance to activated protein C) remained negative. The patient is well 18 months later.

### 3. Discussion

The first report of native aortic valve thrombosis concerned a neonate after heart catheterization for coarctation [7]. The thrombosis resulted from trauma to the valve endothelium. Valve dystrophy such as aortic stenosis may induce endothelial lesions which, associated with an abnormal blood flow, may trigger the mechanism of thrombosis [1,2,8]. Without endothelial lesion, a coagulopathy (anti-phospholipid antibodies, protein S deficiency) may induce aortic valve thrombosis [4,6,9]. Circulating antibodies may have a particular affinity for valve endothelium favoring autoimmune complex formation [6]. In our patient, we did not find any factor favoring the occurrence of the valve thrombosis. The valve was tricuspid, not dystrophic, without functional abnormality, and the biological search for circulating antibodies,

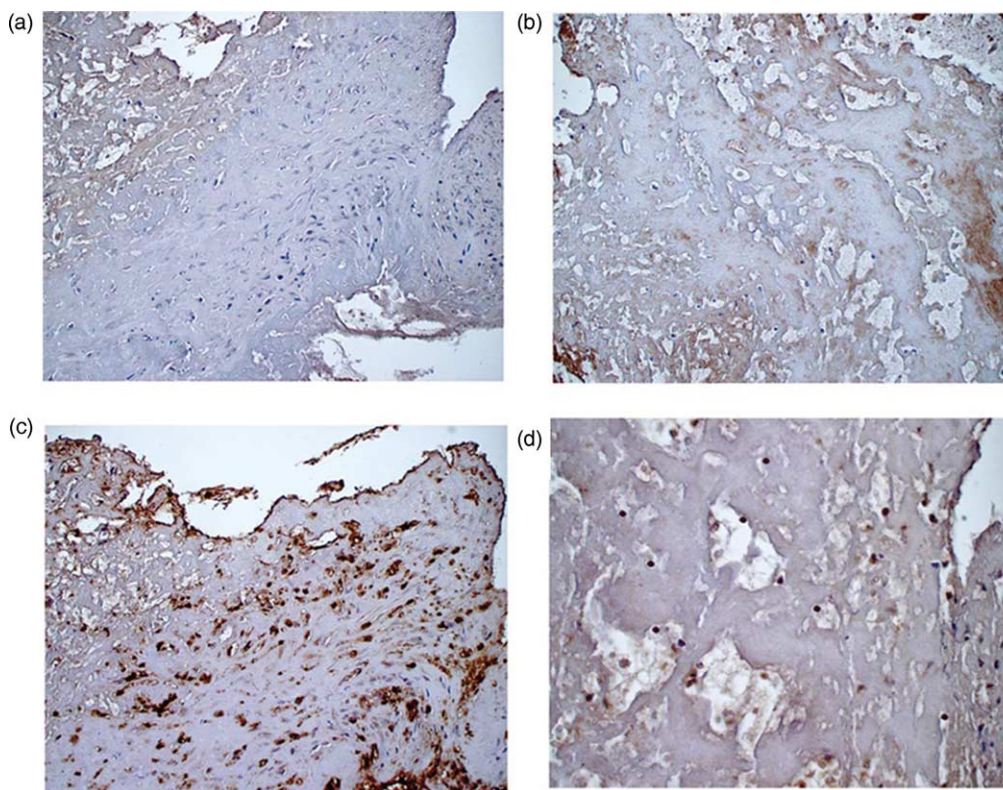


Fig. 2. (a) Amorphous thrombus (WEIGER-staining),  $\times 10$ . (b) Thrombus cavities without endothelial staining (von-Willebrand Factor,  $\times 20$ ). (c) Cavities surrounded by granulocytes (peroxidase-staining,  $\times 20$ ). (d) Granulocytes expressing metalloproteases 9, thus likely accounting for cavities,  $\times 40$ .

tumoral markers and coagulation disorders were negative. However, we cannot exclude such events occurring in the future and regular immunological follow-up is mandatory. Despite the patient's clinical history and histological and bacteriological analysis, a non-bacterial silent thrombotic endocarditis of long standing cannot be ruled out. Unfortunately, it was not possible to repair the valve (with direct suture or pericardium patch) and extemporaneous histological analysis would be helpful. However, the tumor could not be excised with valve conservation. The thrombus triggered a severe inflammatory reaction rich in metalloproteases, which induced the formation of numerous cavities. These cavities do not resemble those reported in a case of angiogenesis of a thrombus (spontaneous reperfusion phenomenon), owing to the absence of positivity to vWF staining. In contrast, the metalloproteases activity throws little light on the origin of our patient's thrombus. Systolic–diastolic movements and biological degradation probably favored its fragmentation, leading to embolic complications.

It would certainly have been possible to avoid the second ischemia if we had performed an echocardiographic examination just after the diagnosis. The natural history of this patient was such that echocardiography should have been performed as soon as possible, particularly since no predisposing factor was known.

In conclusion, this patient who had no risk factor, previous history of thrombosis or valve dystrophy, has an idiopathic native aortic valve thrombosis.

## Acknowledgements

We thank R. Cooke for editing the article.

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