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## SHORT REPORT

# Reversible Myocardial Contraction Abnormality after Aortic Surgery in a non Cardiac Patient

**S. Haulon[1*], J. Gaudric[1], T. Le Tourneau[2], C. Decoene[3], P. Halna[1], L. Destrieux-Garnier[1] and M. Koussa[1]**

*Departments of [1]Vascular Surgery, [2]Cardiology, and [3]Anesthesiology, Hopital Cardiologique, CHRU, Lille, France*

### Introduction

Cardiac morbidity is the leading cause of death after peripheral vascular surgery because of the systemic nature of atherosclerotic vascular disease.[1] Before aortic surgery, preoperative cardiac evaluation is performed in every patient to identify occult cardiac disease that cold be unmasked by the stress of surgery.

We report a case of a young woman with a normal coronary preoperative assessment who underwent aortic surgery. Forty-eight hours after surgery she developed global left ventricular apical abnormalities identical to those caused by acute myocardial ischemia (ACI). "Stress cardiomyopathy" was considered to be the most likely diagnosis. This pathological condition has been reported to occur during the acute phase or aggravation of various systemic disorders which results in the release of catecholamines secondary to intense activation of the sympathetic nervous system. This syndrome has thus been described in association with pheochromocytoma or brain death, but rarely after vascular surgery in a patient with a normal coronary preoperative assessment.

### Case Report

An infrarenal aortic thrombosis was diagnosed in a 45-year old female, presenting with a history of significant bilateral lower extremity claudication and rest pain. She did not present with any symptoms of coronary or cardiac insufficiency. Her only cardiovascular risk factor was tobacco (50 packs/year). Prior to surgery, stress echocardiography was performed with a 40 gamma dobutamine pick and 1 mg of atropine. This examination revealed no wall motion abnormalities, no thoracic pain, and no ECG changes (Fig. 1). This examination was recorded and was reviewed later because of subsequent events.

An aorto bifemoral bypass was performed with a proximal end-to-side anastomosis between the infrarenal aorta and the bifurcated graft. A supraceliac (15 min) clamp was first applied to perform a juxta renal aortic endarterectomy. Thereafter, the clamp was repositioned below the renal arteries and the proximal suture performed. Total blood loss was estimated to be 500 ml, with 100 ml restored by the cell saver. Total anesthesia time was 3 h. No β-blockers or ACE inhibitor were administrated peri-operatively to our patient. Throughout the procedure and the early post operative period, the patient remained hemodynamically stable without the need for medicinal supports. She was extubated in the recovery room. Her urine output was calculated to be $>0.5$ cc/kg/h. The acid–base status always remained stable (pH was 7.3 at the end of the surgical procedure, and 7.4 24 h later).

The first 48 hours after surgery were uneventful. The patient then rapidly developed symptoms of dyspnea, crackles were present in both lung fields, and the systolic blood pressure dropped to 60 mmHg. A chest X-ray revealed bilateral hilar opacities, and ECG an infero-apical ST-segment depression associated to a 1 mm V1-V2 ST-segment elevation (Fig. 2). Troponin T remained in normal range ($<0.25$ ng/ml). Hemoglobin level was stable and there was no evidence of

*Corresponding author. S. Haulon, Chirurgie Vasculaire, Hopital Cardiologique, CHRU de Lille, 59037 Lille Cedex, France.

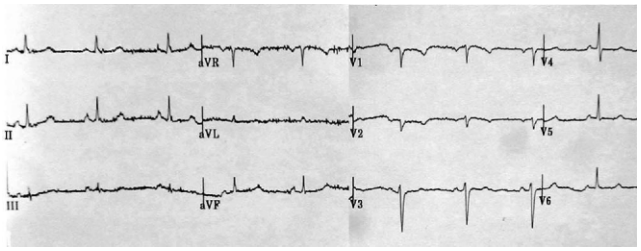


**Fig. 1.** Preoperative ECG.

hemorrhage. A trans thoracic echocardiogram (TTE) revealed a global wall motion abnormality with a mean left ventricular ejection fraction (LVEF) of 30%. Cardiac catheterization was performed and this revealed a 30 to 50% stenosis of the left anterior descending artery, and a <30% stenosis of the circumflex and the right coronary arteries, and confirmed the 30% mean LVEF. The treatment consisted of catecholamine support (dopamine/dobutamine).

Cathecholamines were progressively reduced and stopped after 7 days. A second TTE was then performed. Both the regional and global left ventricular systolic function had returned to the preoperative state, with a mean EF of 55%, as shown on a TTE performed on post operative day 9. Troponin T never increased during hospitalization.

The patient was discharged 14 days after surgery. After 6 months of follow-up, no further complication was diagnosed.

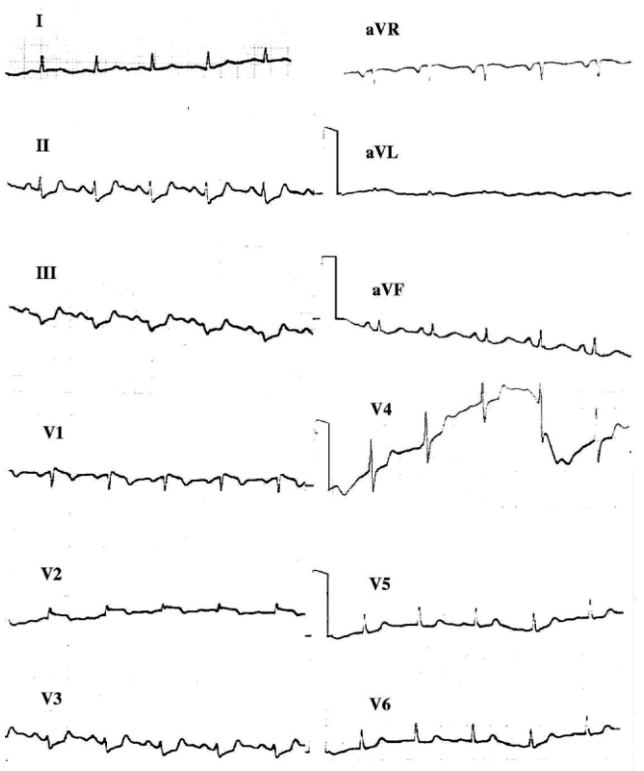


**Fig. 2.** Inferior ST-segment depression revealed by the post operative ECG.

## Discussion

This case describes an early transient postoperative cardiac failure secondary to global left ventricular wall motion abnormalities in a 45-year old woman. The clinical presentation parallels that of an ACI in this heavy smoker, but the preoperative stress echocardiography and the postoperative cardiac catheterization did not show any significant coronary lesions, and troponin T always remained with the normal range. This case was therefore different from the "stunned myocardium" described by Braunwald *et al.*, where transient cardiac failure is diagnosed after myocardial ischemia in patients with coronary lesions.[2] It was also different from myocardial ischemia in patients with normal coronary arteries as a result of disease of the coronary microcirculation (syndrome X) or of coronary vasospasm (Prinzmetal's angina); where transient cardiac failure is focal and troponin T is elevated.

Our diagnostic hypothesis of stress cardiomyopathy, or catecholamine induced cardiomyopathy, relies on the absence of any other etiology, and on the presence of recent surgery that induces intense activation of the sympathetic nervous system, by catecholamine receptor saturation or sympathetic innervation.

Data on endo or exogenous increases in catecholamines responsible for a decreased response in myocardial contraction have been reported.[3,4] Identical to AMI, catecholamine induced cardiomyopathy secondary to various acute systemic disorders exhibits segmental or global left ventricular apical wall motion abnormalities as well as the T wave and QTc interval ECG changes.[5] However, angiographic evidence of coronary lesions is absent. This particular cardiomyopathy was most frequently described in association with brain death[7,8] or pheochromocytoma.[9]

As far as brain death is concerned, the syndrome is frequently seen in young patients without cardiovascular risk factors. Postnecropsy studies did not provide any histological evidence of coronary atherosclerotic lesions responsible for heart failure. Myocardial dysfunction was induced by intense catecholamine release after brain death.

In the case of pheocromocytoma, the high catecholamine concentration was also responsible for myocardial dysfunction, and the absence of coronary

atherosclerotic lesions was confirmed by cardiac catheterization.

Non specific ECG changes have been reported in association with left myocardial dysfunction symptoms: inversion of the T wave, ST-segment elevation or depression, QTc lengthening. Echocardiography always diagnosed a decrease in left ventricular systolic function associated with global or regional wall motion abnormalities often involving the apex. The myocardial wall motion abnormality is global when catecholamine mediation is humoral due to the overload of the catecholamine receptors, and is regional when mediation is neurogenic due to the inhomogeneous overload of the cardiac sympathetic nervous network.

When histological studies were performed,[6–9] specific catecholamine lesions, referred to as a "contraction band", were identified. These bands include degeneration and focal necrosis of myocytes (myocyte eosinophilia) with secondary inflammatory infiltrates and fibrosis. These lesions were previously described in patients with pheochromocytoma and in experimental models of stressed dogs. The pathophysiological mechanism is a membrane permeability modification of the sarcoleme induced by noradrenaline increase, responsible for intracellular increase of calcium and cellular death. Free radicals induced by catecholamine oxidation are also responsible for direct cell toxicity.

There are no correlations between the intensity of myocardial dysfunction on echocardiography and the histological findings. The presence of histological lesions reflects neurogenic dysfunction, that do not cause echocardiographic abnormalities.

Acute myocardial ischemia is the most common cause of acute postoperative myocardial contraction abnormality after aortic surgery. However, in patients with no history of coronary artery disease, and with complete recovery of their sudden wall motion abnormality, the diagnostic of stress cardiomyopathy can be entertained, especially if cardiac enzymes are normal.

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