

Left Ventricular Free Wall Rupture During Dobutamine Stress Echocardiography

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Dobutamine stress echocardiography is associated with a very low rate of serious complications, lower than 0.5% (death, infarction or sustained ventricular tachycardia). We report the case of a 75 year-old female patient that suffered a fatal left ventricular free wall rupture during a dobutamine stress echocardiography after acute myocardial infarction.

Key words: *Stress echocardiography. Myocardial infarction.*

Rotura de la pared libre del ventrículo izquierdo durante prueba de estrés con dobutamina

La ecocardiografía de estrés con dobutamina se asocia a una baja tasa de complicaciones graves, menor del 0,5% (muerte, infarto o taquicardia ventricular sostenida). Presentamos el caso de una paciente de 75 años que sufrió una rotura de la pared libre del ventrículo izquierdo durante la realización de una prueba de estrés con dobutamina para estratificar el riesgo postinfarto.

Palabras clave: *Ecocardiografía de estrés. Infarto de miocardio.*

INTRODUCTION

Dobutamine stress echocardiography (DSE) is widely used to determine the level of post-infarct risk, particularly in patients who are unable to successfully perform a conclusive stress test. DSE is associated with a very low rate of serious complications (such as death, infarct, or sustained ventricular tachycardia) of less than 0.5%, according to most studies.^{1,2} We describe a case of cardiac rupture that occurred during DSE performed on a patient to determine the level of post-infarction risk.

CLINICAL CASE

The patient was a 75-year-old woman with a history of arterial hypertension, hypercholesterolemia, vasomotor rhinitis, and extrinsic asthma. She was

referred to the cardiac unit after presenting for hip surgery, because preoperative electrocardiogram showed an existing posteroinferior infarct. The patient reported a prolonged bout of retrosternal pain 10 days prior to admission. Physical examination on admission did not reveal any significant changes. Blood work revealed an elevated troponin level (4.87), with normal cardiac enzymes. Electrocardiogram revealed elevated Q-wave in the ST segment in DII, DIII, and an aVF, and a subendocardial lesion in the right precordials. The basal echocardiogram showed inferior akinesia with dyskinesia of the basal segment (Figure 1). Given the clinical and electrocardiographic data, along with the normal examination results with the exception of the troponin level, an inferior infarct of more than 10 days duration was considered. Forty-eight hours after admission, given the impossibility of performing a stress test due to physical limitations, a DES was requested. A 5-stage protocol was initiated, beginning with 5 µg/Kg/m. During the test, no signs of viability or ischemia were observed. Before completing the final stage of testing, the patient noted sudden precordial pain accompanied by the appearance of an ST segment in the inferior face and cardiorespiratory arrest by electromechanical failure. The

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ABBREVIATIONS

TIMI: Thrombolysis in myocardial infarction
DES: Dobutamine stress echocardiography

electrocardiogram showed severe pericardial hemorrhage with signs of tamponade (Figure 2); this was diagnosed as an acute rupture of the free wall of the left ventricle,³ and the patient died within a few

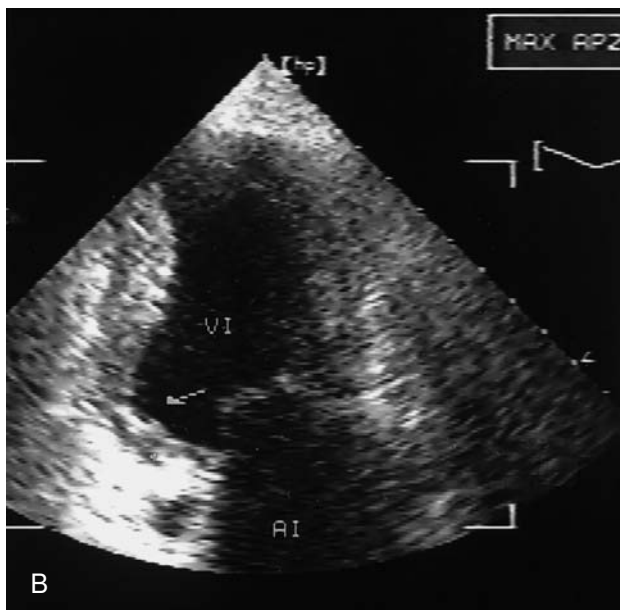
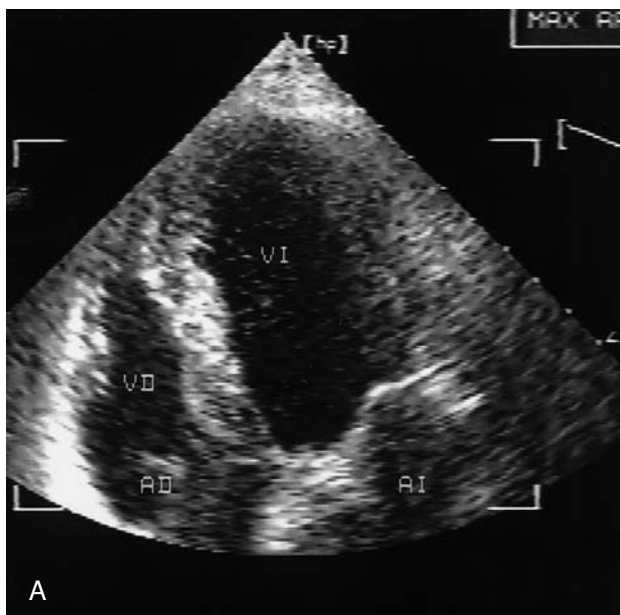


Fig. 1. A. Basal echocardiogram, apical 4-chamber view. B. Basal echocardiogram, 2-chamber apical view. LV indicates left ventricle; RV, right ventricle; RA, right auricle; LA, left auricle. Arrow, aneurysm of the basal segment of the inferior wall.

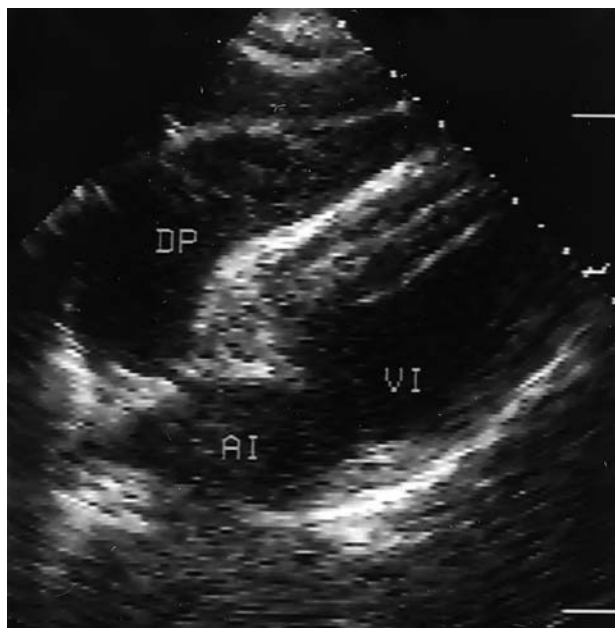


Fig. 2. Subcostal echocardiogram showing a severe pericardial hemorrhage with collapse of the right cavity. PH indicates pericardial hemorrhage; LV, left ventricle; LA, left atrium.

minutes.

DISCUSSION

DSE is associated with a very low rate of serious complications; the rate of death, myocardial infarction, or life-threatening ventricular arrhythmias is less than 0.5% according to most studies,^{1,2} and the mortality rate is 0% in most studies. Cardiac rupture is the second most common cause of nosocomial death in patients admitted for acute myocardial infarct, with an incidence of 2% to 4% and usually associated with factors such as advanced age, female sex, arterial hypertension, and in patients treated with primary angioplasty, the presence of TIMI 0 fluid at the beginning of the procedure.

There are discrepancies about the importance of the location of the infarct; the incidence is probably lower in patients treated with beta-blockers.³⁻⁵

Dobutamine administered through after a myocardial infarct can, in theory, trigger cardiac rupture. Very few cases are described with this complication during DES.^{6,7} This is probably due to the fact that the greatest incidence of rupture takes place 48 to 72 hours after the development of the infarct, with DES-caused stratification usually occurring at about 1 week.

We describe a case of cardiac rupture that occurred during DSE. The fact that there was no pericardial hemorrhage at the beginning of the test excluded the possibility that a complete rupture of the free wall

existed previously, although the existence of a previous ventricular wall lesion of the ventricular wall cannot be excluded.

The two cases published cardiac rupture during DSE^{5,6} occurred in patients with recent inferior infarct and dyskinesia of the basal segment. Although speculative, it is possible that the anatomy of the inferior basal infarct could indicate a muscle fiber tear between the area of the infarct and healthy muscle, secondary to the inotropic effect produced by the dobutamine. From our point of view, nevertheless, this complication has a very low incidence rate during DES and, on the other hand, DES has probably not been performed without accelerating and/or uncovering an existing incomplete rupture. Therefore, this complication should probably not change the management of post-infarct patients evaluated by DES.

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