Septal rupture with right ventricular wall dissecting haematoma communicating with left ventricle after inferior myocardial infarction

Luisa De Gennaro1,2, Natale Daniele Brunetti2*, Giovanni Ramunni1, Francesco Buquicchio1, Francesco Corriero1, Elisabetta De Tommasi1, Rossella Troccoli1, Antonio Grimaldi3, Matteo Di Biase2, and Filippo Boscia1

1Cardiology Department, ‘San Giacomo’ Hospital, Monopoli, Bari, Italy; 2Cardiology Department, University of Foggia, Foggia, Italy; and 3Department of Cardiac Surgery, San Raffaele University Hospital, Milan, Italy

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We report the case of an 86-year-old man referred for abdominal pain and ECG signs of inferior myocardial infarction. Transthoracic, transoesophageal and contrast echocardiographs showed a septal intra-mural haematoma, dissecting the right ventricle wall and partially obliterating the right ventricle lumen. A patent communication with left ventricle with extensive wall thrombosis was present at Doppler examination within dissecting haematoma. Although the patient refused any surgical treatment, a 3-month follow-up was uneventful.

Keywords Ventricular septal rupture • Right ventricular wall dissecting haematoma • Myocardial infarction • Ventricular thrombosis

Background

Ventricular septal rupture (VSR) is an uncommon complication after myocardial infarction (MI).1 In case of inferior MI, VSR usually involves basal posterior septum, with a variable degree of right ventricular wall extension.2 VSR is usually associated with higher mortality rates, even after surgical repair.3 Few cases of septal rupture with right ventricular wall dissection after MI have been reported;4,5 in these cases, the surgical correction is usually needed.

Case report

We report the case of an 86-year-old man, referred for abdominal pain that had begun 24 h earlier. A couple of weeks earlier, he had had a syncope, followed by dyspnoea. He was hypertensive, diabetic, and had been treated with aspirin, nifedipine, and oral antidiabetics; he had no history of ischaemic heart disease.

Systolic blood pressure at admission was 120/80 mmHg, and physical examination was unremarkable; no murmur was appreciable at auscultation. Resting ECG showed sinus tachycardia (110 bpm), Q waves, and ST elevation in inferior leads and ST depression in anterior and lateral leads. Chest radiograph showed no sign of pulmonary congestion, a mildly enlarged cardiac transverse diameter, and mild peripheral ‘pruning’. Cholesterol and creatinine levels were normal at admission, whereas troponin-I was 19 ng/mL (normal values 0.10 ng/mL) and N-terminal pro-brain natriuretic peptide was 9648 pg/mL (normal values 450 pg/mL).

At the first transthoracic echocardiography (TTE) examination, a severe inferior and right ventricular akinesis with signs of ‘apparently’ right ventricular thrombosis was found. Left ventricular ejection fraction was assessed as ~45% (Figure 1 and see Supplementary data online Video 1). Doppler examination showed a septal tear with left to right shunt (Figure 2).

Therefore, we hypothesized a ventricular septal defect with a dissection tract that originated on the left side of the middle infero-posterior septal segment, dissecting the right ventricular wall and partially obliterating right ventricular lumen. No sign of pericardial effusion was present.

However, clinical and haemodynamic conditions of the patient were stable, without any apparent signs of significant right ventricular failure (such as hypotension, peripheral oedema, jugular venous distension).
Transoesophageal echocardiography (TEE) confirmed the presence of a patent septal tear (Figure 3). A contrast echocardiography examination was then performed; however, the iv contrast medium (agitated saline) was unable to reach the haematoma lumen. This confirmed that the area with thrombosis was a dissecting haematoma chamber and that there was no patent communication between the left and right ventricles (no sign of contrast dilution) (Figure 4).

We therefore postulated the presence of a septal rupture with right ventricular wall dissecting haematoma after inferior MI; the haematoma lumen was characterized by wall thrombosis with patent communication with left ventricular chamber (Figure 5, see Supplementary data online Videos 2–3). The dissecting haematoma extended neither to the right ventricular inflow tract nor to the outflow tract, which were both patent.

The patient remained in stable clinical and haemodynamic conditions during the entire hospitalization, except for a transient rise in transaminases levels (peak glutamyl oxaloacetic transaminase 416 IU/L, glutamyl pyruvic transaminase 1182 IU/L). He refused...
any surgical correction and did not experience any in-hospital adverse event. Peak troponin-I level was 27.08 ng/mL. A 3-month follow-up was uneventful.

**Discussion**

To our knowledge, this is one of the first cases reporting septal rupture with right ventricular wall dissecting haematoma after inferior MI, without patent communication between left and right ventricles, not treated with surgery and still in stable clinical conditions. Few cases of VSRs with right ventricular wall dissection after MI have been reported; such cases usually required surgical correction.

In case of suspected VSR, TTE and/or TEE are paramount for the early diagnosis and treatment. A careful echocardiographic recognition of septum and right ventricular wall is mandatory in order to localize the septal tear and dissecting the lumen extension. For this purpose, the use of colour flow Doppler mapping and contrast echocardiography might be extremely helpful. The use of sub-costal views visualizing the right ventricular free wall might allow one to detect the left ventricular entry site.

In a recently published review, the overall mortality in the group with septal haematoma reached 78%. In five of the six cases with dissecting haematoma, not immediately lethal and not treated with surgery, echocardiographic evolution included spontaneous re-absorption of dissecting haematoma during the first year post-infarction.

It was proposed that the mechanism of ventricular rupture was the lack of support from infarcted tissue, with alterations in
small vessels wall, leading to haemorrhage and formation of a blood-containing mass that dissected, being forced by the intracavitary pressure. Some authors believe that VSR complicating inferior MI may lead to a variety of clinical presentations, ranging from wall haematoma, with or without communication into the ventricular cavity, to right ventricular wall dissection or free wall rupture.6,8

In our case, a watchful wait approach was preferred by the patient. No adverse events occurred in the 3-month follow-up.

**Conflict of interest:** none declared.

**References**


