Transient ballooning of the left ventricle: a case report

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Left ventricular insufficiency; Stunned myocardium.

A 67-year-old woman was admitted to the coronary care unit for chest pain with ECG modifications suggestive of acute myocardial infarction. The clinical course of the disease and the absence of abnormalities of the epicardial vessels with reversible asynergy of the apical segments of the left ventricle were suggestive of the syndrome of transient apical ballooning of the left ventricle. To my knowledge, this is the first case of the disease described in the Italian population.

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A novel syndrome with transient asynergy of the apical segments of the left ventricle has been recently described both in Japanese and in European patients[1-4]. This report concerns the case of a woman with features that fulfill the diagnostic criteria for the syndrome of transient apical ballooning. In this case, the diagnosis has been considered likely about 18 months following hospital discharge.

Case report

B.G., a 67-year-old woman, was admitted to the coronary care unit of the Lavagna hospital for chest pain lasting about 6 hours, triggered by a significant emotional stress. She had a previous history of hypertension treated with carvedilol, and a recent endoscopic diagnosis of hemorrhagic duodenitis.

Upon admission, chest pain was improving, there was no sign of heart failure and ECG showed ST-segment elevation in the D1- aVL and V_6 leads (Fig. 1). Faced with these clinical features and the history of gastrointestinal bleeding, the patient received no thrombolytic drug, but nitrates and low-dose enoxaparin were administered.

A few hours later, the patient developed significant hypotension, unrelieved neither by the interruption of nitrates nor by fluid infusion, and required dopamine administration. The echocardiogram revealed akinesia of the four apical segments of the left ventricle, with a mild reduction in the ejection fraction (45%) and with Doppler parameters of mitral flow and pulmonary vein flow suggestive of a pulmonary capillary wedge pressure < 16 mmHg. The peak creatine kinase level was 260 IU/l, with a troponin I peak of 16 ng/ml.

The following day the patient complained of dyspnea, the ECG showed negative T waves in the precordial leads, echocardiography showed a further reduction in the ejection fraction, and a rise in the pulmonary capillary wedge pressure was estimated by Doppler evaluation. Therefore, an urgent catheterization and coronary angiography was performed, in order to exclude extensive multivessel disease. Angiography showed the absence of coronary stenoses; in particular, the right artery was dominant and the left anterior descending passed beyond the apex, with a not unusually long course along the diaphragmatic surface of the left ventricle (Fig. 2). Ventriculography showed a severe impairment of the ejection fraction, with ballooning of the middle and apical segments of the left ventricle, and a preserved function of the basal segments (Fig. 3). The left ventricular end-diastolic pressure was 30 mmHg and no intraventricular gradient was observed.

The patient was then treated with dobutamine, digoxin, diuretics and low dose captopril. The clinical and echocardiographic parameters showed rapid amelioration. The serologic markers for viral myocarditis excluded recent viral infection. The patient was discharged on day 9 in stable clinical conditions with a diagnosis of acute myocardial infarction. One month later echocardiography showed a normal

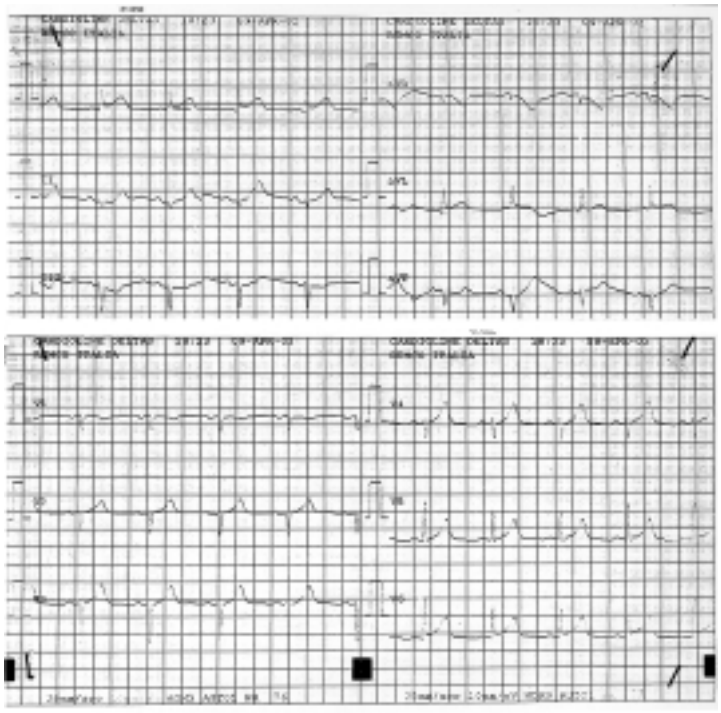

Figure 1. *ECG morphology upon admission.*

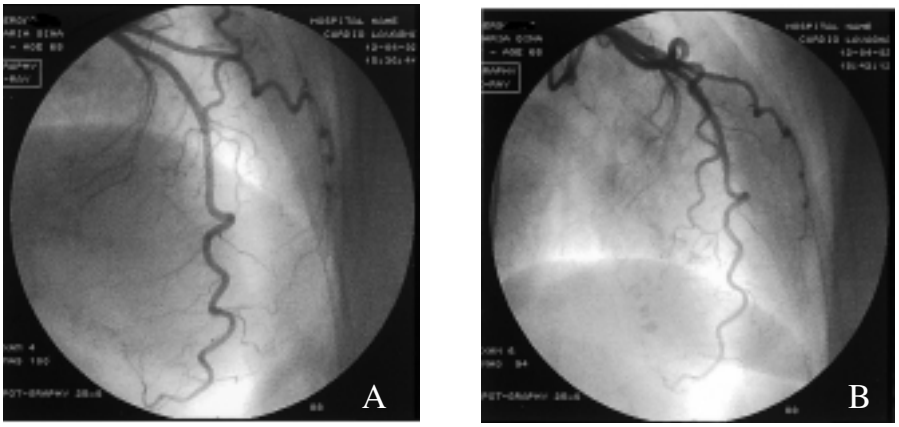

Figure 2. *Right anterior oblique projections of the left coronary artery, showing the course of the left anterior descending artery beyond the apex.* A: *right anterior oblique 10°, with 45° cranial angulation;* B: *right anterior oblique 30°, with 30° cranial angulation.*

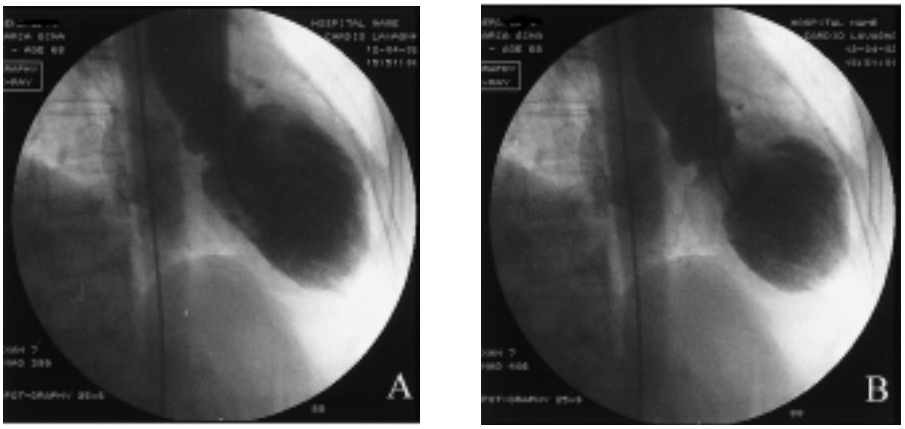

Figure 3. *Ventriculography images.* A: *end-diastole;* B: *end-systole. A large segment of the left ventricle is akinetic, with preserved function of the basal segments.*

ejection fraction; at a recent clinical control, 18 months later, she is fully asymptomatic with a normal ECG.

Discussion

The reported case has been an enigmatic one for the clinician. In fact, the patient was admitted with the typical features of an acute myocardial infarction, as diagnosed on the basis of chest pain, ST-segment elevation and evolving changes on ECG and a typical increase in the serum markers of necrosis; however, the severity of the clinical course was not in agreement with the relatively limited ECG abnormalities at presentation and the modest enzymatic raise. The first hypothesis was that of a limited necrosis with a large area of stunned myocardium, due to the presence of significant coronary obstruction. The absence of abnormalities of the epicardial vessels excluded this possibility; on the other hand, the hypothesis of coronary spasm was ruled out by the extension of the asynergy that overcame the limits of the territory of a single vessel. Besides, multiple spasms are quite unlikely. An alternative hypothesis, that is inflammatory disease, was not confirmed by the laboratory tests, even though owing to the fact that a biopsy was not performed, it could not be completely ruled out.

Tsuchihashi et al.[1] and, more recently, Abe et al.[2] have described, in the Japanese population, a syndrome that they named transient left ventricular apical ballooning; this syndrome has been subsequently described in a European series by Desmet et al.[3]. The diagnostic criteria proposed by Abe and Kondo[5] are classified as major and minor. The patient of this report met all the criteria: in fact, she had chest pain triggered by an emotional stress with ST-segment abnormalities mimicking acute myocardial infarction, she had a typical transient and reversible mid-apical asynergy with an ampulla-shaped left ventricle in systole and, finally, there was a significant discrepancy between the severity of the clinical and echocardiographic presentation and the enzymatic elevation. Furthermore, she had no cardiac events at an 18-month follow-up. Other causes of left ventricular transient asynergy, such as subarachnoid bleeding, a pheochromocytoma crisis or acute myocarditis, were excluded.

At present, the pathogenesis of the syndrome in still unclear; several hypothesis including catecholamine damage[2], microvessel dysfunction[6] and transient acute intraventricular gradient[7] have been proposed; in the present case, no intraventricular gradient was observed.

The present report is, to my knowledge, the first described in the Italian population; the diffusion of reports about this syndrome could help to better define its characteristics, possibly by the creation of a registry as proposed by other authors[5].

Acknowledgments

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