Journal of Cardiology Cases xxx (2018) xxx–xxx

Contents lists available at ScienceDirect

Journal of Cardiology Cases

journal homepage: www.elsevier.com/locate/jccase

Case Report

A case of conservative management for left ventricular giant pseudoaneurysm without ST segment changes

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ARTICLE INFO

Article history:
Received 27 September 2017
Received in revised form 18 December 2017
Accepted 8 January 2018

Keywords:
Left ventricular pseudoaneurysm
Myocardial infarction
Transthoracic echocardiography
ST segment changes
Anticoagulation

ABSTRACT

Left ventricular (LV) rupture after myocardial infarction (MI) occasionally results in formation of LV pseudoaneurysm (LVPA) which is prone to rupture because of its thin wall. However, cases of LVPA without ST changes including segment elevation in electrocardiogram (ECG) are rare. In this case, we describe a patient who had relatively mild symptoms and giant LVPA with no specific ECG changes following MI with a confirmed diagnosis via transthoracic echocardiography. Although surgical treatment options are often recommended, conservative therapy was adopted, following which the patient had been well-medicated using antihypertensive drugs and anticoagulants.

<**Leaning objectives:** Left ventricular pseudoaneurysm (LVPA) is usually accompanied by ST segment changes on electrocardiogram (ECG) due to myocardial damage. However, we should take into account a LVPA without ECG specific changes, so echocardiography is better to be considered for an identification. Although many LVPA patients undergo surgery because of risk for rupture, some cases with stable hemodynamic status can have long-term survival with conservative therapy such as anti-hypertension and coagulation.>

Introduction

Left ventricular psedoaneurysm (LVPA) is one of the most serious complications that can occur after myocardial infarction (MI), which occurs subsequently following left ventricular (LV) rupture. Typically, LVPA involves ST changes such as ST elevation that can be observed on an electrocardiogram (ECG) and generally requires surgical treatment emergently due to the high potential rate of re-rupture compared with cases of LV true aneurysm. Here, we report a case of a woman with giant LVPA without ST changes, who had been well medicated for 12 months as part of a conservative management regimen.

Case report

An 85-year-old female was transferred to our institution emergently due to chest pain. Upon arrival, she had clear consciousness, and her blood pressure and saturation were 147/83 mmHg and 92%, respectively, with a heart rate of 105 bpm with sinus rhythm. Upon physical examination, no impressive findings were noted.

We could not find clear ST segment elevation or specific changes on ECG compared with an earlier one sourced from her patient medical records (Fig. 1). Chest X-ray (XP; Fig. 2A) and computed tomography (CT; Fig. 2B), however, showed an abnormal mass in

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https://doi.org/10.1016/j.jccase.2018.01.006
1878-5409/

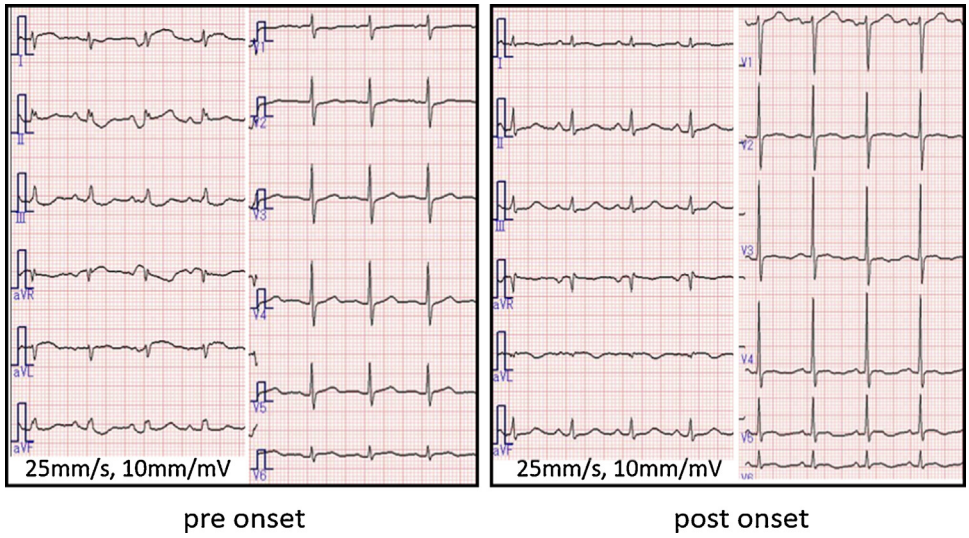

Fig. 1. Electrocardiogram demonstrates no clear changes in ST segment (right) compared with pre-onset (left).

her left lower sternal area, which was not present a year before at the time of regular examination. Laboratory testing indicated that fibrin/fibrinogen degradation product (FDP), D-dimer, brain natriuretic peptide (BNP), and troponin-I (TnI) were all significantly increased (FDP: 104 μg/mL, D-dimer: 29 μg/mL, BNP: 958 pg/mL, TnI: 137 pg/mL, respectively), but creatinine kinase (CK) was unchanged (CK: 43 U/L). At this time, we examined the patient for acute pulmonary embolism, which resulted in enlarged pulmonary dilatation as an abnormal signal on chest XP and CT. In addition, she had physical distress on admission day, so we did not examine positively further evaluation such as transthoracic echocardiography (TTE), coronary angiography, and enhanced CT.

The next day, we performed TTE and noted a giant (7–8 cm) abnormal space (Fig. 3 and Video 1 in Supplementary material) with LV communication, which had inflow synchronizing a cardiac cycle, from LV to space in systole and from space to LV in diastole (Video 2 in Supplementary material). The ejection fraction estimated by modified Simpson's method was about 50%; in addition, she had mild mitral regurgitation, but not tricuspid regurgitation. We diagnosed the space as LVPA after LV rupture due to MI, with the observation that LV had a relatively narrow neck (less than 40% of the maximal aneurysm diameter) and a thin wall containing epicardium and fibrosis tissue. Based on the patient's TTE findings, surgery was recommended immediately; however, both she and her family rejected our proposal. Hence, we considered the optimal non-surgical medical treatment such as management of blood pressure (received enalapril maleate 2.5 mg and amlodipine besilate 2.5 mg per day, respectively) and anticoagulation (received edoxaban tosilate hydrate 30 mg per day).

At 12 months follow-up, no significant changes were detected on TTE or chest CT, including LVPA size and thrombus formation, and no complications, such as thromboembolism, were demonstrated during 12 months.

Discussion

The incidence of mechanical complications following LV rupture is 6% in cases of acute MI, which can be stratified into 60% blowout type and 40% oozing type [1]. LVPA is a form of oozing type LV rupture, encapsulated by epicardium containing coagulation and fibrous tissue. Mortality rate in instances of blowout rupture is about 100%, but is only 50% in cases of oozing type [1]. Other critical complications of acute MI include ventricular septal rupture (VSR), which accounts for 5% of all deaths following acute MI; however, the frequency of these complications has decreased since the introduction of primary percutaneous coronary intervention [2].

LV aneurysm is classified as either LVA or LVPA. LVA occurs following MI, and has an incidence of up to 30%–35% in patients with Q-wave MI [3]. However, its incidence has been decreasing in recent years due to medical progression. About 70%–85% of instances of LVA originate in the anterior or apical walls due to total occlusion of the left anterior descending coronary artery [4]. The LVA wall is histologically characterized by muscle necrosis and intense inflammatory reaction [4]. There are serious complications that can follow LVA formation, including heart failure, ventricular arrhythmias, and thromboembolism [5]. Importantly, LVA may enlarge; however, unlike LVPA, a mature LVA rarely ruptures because of the dense fibrosis in its wall [6]. The natural history of patients with LVA is controversial; a study containing 40 patients not treated surgically showed that the 5- and 10-year survival rates were 47% and 18%, respectively [7].

While LVPAs that form following cardiac rupture contain adherent pericardium or scar tissue, unlike an LVA, LVPA has no endocardium or myocardium. Thus, the diagnosis needs to be established early because LVPA is more prone to rupture [6]. The primary cause of LVPA was found to be related to MI in 55% of a cohort of 253 patients, an occurrence that was twice as common as anterior infarction, with the cause of onset relating particularly to the inferior wall [8]. In addition, the second most common cause of LVPA was cardiac surgical procedure, such as a mitral valve replacement, which was responsible for 33% of cases; trauma accounted for 7% of cases [8]. In some reports, the most frequent symptoms associated with LVPA were chest pain and dyspnea [8]. However, the distribution of presenting manifestations was different; for example, the Mayo Clinic reported that 48% patients with LVPA were asymptomatic [9]. In terms of the diagnosis, most patients with LVPA had ECG changes, such as ST elevation and nonspecific changes; however, unchanging ST segment was also possible [8]. The performance of TTE is reasonable as a first step; however, a definitive diagnosis is made in only 26% of patients with LVPA using this technique. Therefore, there is still a need to use

Please cite this article in press as: Ousaka D, et al. A case of conservative management for left ventricular giant pseudoaneurysm without ST segment changes. J Cardiol Cases (2018), https://doi.org/10.1016/j.jccase.2018.01.006