Journal of Cardiology Cases 15 (2017) 14–17

Contents lists available at ScienceDirect

Journal of Cardiology Cases

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

Case Report

A case of transient apical hypertrophy associated with coronary vasospasm

Toru Awaya (MD)*, Masao Moroi (MD, PhD, FJCC), Takahito Takagi (MD), Go Hashimoto (MD), Tadashi Araki (MD, PhD), Hidehiko Hara (MD, PhD), Makoto Suzuki (MD, PhD, FJCC), Masato Nakamura (MD, PhD, FJCC), Kaoru Sugi (MD, PhD, FJCC)

Division of Cardiovascular Medicine, Toho University Ohashi Medical Center, Tokyo, Japan

ARTICLE INFO

Article history:
Received 29 March 2016
Received in revised form 5 September 2016
Accepted 14 September 2016

Keywords:
Coronary vasospasm
Apical hypertrophy
Myocardial edema

ABSTRACT

A 49-year-old woman was admitted to our hospital because of repeated loss of consciousness. On arrival, she was in cardiopulmonary arrest associated with arrhythmia of Torsades de pointes, and recovered from it after cardiopulmonary resuscitation and defibrillation. The administration of a β-blocker and amiodarone was initiated to prevent ventricular tachycardia. On day 2, coronary angiography revealed non-obstructive coronary artery, and left ventriculography (LVG) exhibited hypokinesis in the anterior and apical wall. On day 20, an acetylcholine provocation test revealed a multivessel vasospasm, and LVG showed "spade-shaped left ventricle" in end-diastole because of apical hypertrophy. Transthoracic echocardiography (TTE) also showed apical wall thickness. Subsequent apical wall thickness gradually decreased and returned to normal on day 51 as observed on the TTE. Thus, we report a case of transient apical hypertrophy associated with coronary vasospasm, which was demonstrated by both the TTE and LVG.

<**Learning objective:** A case of transient apical hypertrophy associated with coronary vasospasm was reported with serial left ventriculography and possible mechanisms.>

Introduction

Few clinical cases have reported transient apical hypertrophy following coronary vasospasm. Furthermore our case is rare, because there are two different abnormal motions on the left ventriculography (LVG) on day 2 and on day 21. LVG exhibited hypokinesis in the anterior and apical wall on day 2, and showed "spade-shaped left ventricle" in end-diastole because of apical hypertrophy on day 21.

Case report

A 49-year-old woman with unremarkable medical history was admitted to our hospital because of repeated loss of consciousness. On arrival, she was in cardiopulmonary arrest (CPA) with arrhythmia of Torsades de pointes (TdP). Immediately after admission, she received cardiopulmonary resuscitation and defibrillation. She also received general anesthesia for controlling repeated ventricular tachycardia (VT). A subsequent electrocardiogram (ECG) revealed negative T waves in the I, II, aVF, aVL, and V2–V6 leads, as well as frequent premature ventricular contractions (Fig. 1). The transthoracic echocardiogram (TTE) showed hypokinesis in the anterior and apical wall. Further, coronary angiography revealed non-obstructive coronary artery on day 2 (Fig. 2A). LVG revealed hypokinesis in the anterior and apical wall (Fig. 2C). We did not perform an acetylcholine (Ach) provocation test then owing to the risk of VT storm. The administration of a β-blocker and amiodarone was initiated to prevent VT. The TTE showed an improved left ventricular asynergy on day 3, but subsequent apical wall thickness mildly increased on day 11. Laboratory data showed a maximal level of creatine kinase (CK) (819 IU/L), but CK-MB was not elevated on day 4. These findings suggest the effect of cardiopulmonary resuscitation and defibrillation. An Ach provocation test on day 20 revealed a multivessel vasospasm [right coronary artery (RCA), #4 AV 99% stenosis; left coronary artery (LCA), #7 99% stenosis, and #11 90%

* Corresponding author at: Division of Cardiovascular Medicine, Toho University Ohashi Medical Center, 2-17-6, Ohashi Meguro-ku, Tokyo 153-8515, Japan. Fax: +81 3 3468 1269.
E-mail address: toru0228@gmail.com (T. Awaya).

http://dx.doi.org/10.1016/j.jccase.2016.09.003
1878-5409/

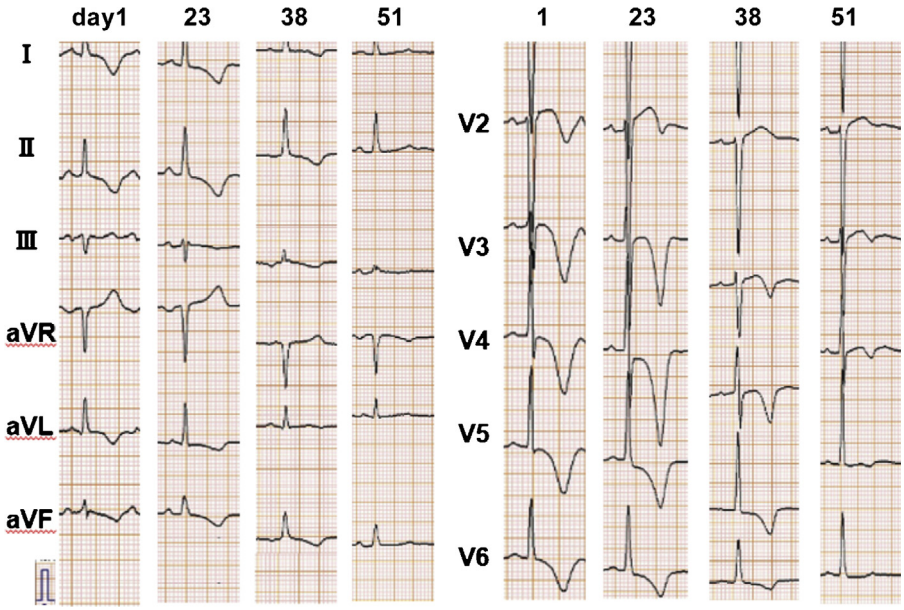

Fig. 1. Electrocardiogram (ECG) progress over the course of the patient's treatment. ECG showed a negative T wave in the I, II, aVF, aVL, and V2–V6 leads on admission. ECG exhibited extremely deep giant T waves on day 23. ECG revealed flat T waves on day 51.

stenosis; Fig. 2B]. In addition, a 50 μg of Ach was injected into the RCA and 100 μg into the LCA. The LVG revealed "spade-shaped left ventricle" in end-diastole because of apical hypertrophy (Fig. 2D).

TTE also revealed apical wall thickness. After the diagnosis of coronary vasospasm, we initiated a calcium channel blocker. On day 23, the ECG revealed extremely deep giant T waves (Fig. 1), and the TTE revealed a maximum wall thickness of 20 mm.

On day 29, the cardiac magnetic resonance imaging revealed late gadolinium enhancement and an increased signal on the T2-weighted images in the apex region (Fig. 3). From these observations, it can be inferred that myocardial injury due to coronary vasospasm causes myocardial necrosis and edema. Subsequent apical wall thickness gradually decreased on day 51, the ECG showed a flat T wave (Fig. 1), and the TTE displayed normal apical wall thickness (12 mm). Apical hypertrophic cardiomyopathy does not show an increased signal on the T2-weighted images, and does not change ECG and TTE in a short period. In this point of view, we diagnosed this case as transient apical hypertrophy.

In this case, multivessel vasospasm and CPA had occurred, so we decided to insert an implantable cardioverter-defibrillator (ICD) on day 32, considering the high risk of Tdp recurrence. Approximately 2 years later, ICD discharge was recorded against VT, so we treated VT with catheter ablation procedure.

Discussion

A case of transient apical hypertrophy associated with coronary vasospasm is rare. Few clinical cases have reported transient apical hypertrophy following takotsubo cardiomyopathy and acute myocarditis in addition to coronary vasospasm [1–4].

In these reports, the cases exhibited that transient left ventricular wall motion abnormalities improved 2 days or at least within 10 days from the onset. In contrast, apical wall thickness gradually increased, and apical hypertrophy appeared over a few weeks to months. Subsequently, the apical wall thickness gradually decreased and returned to normal thickness over a few months to a year [1–4]. In this case, the left ventricular wall motion abnormalities improved on day 3, and apical hypertrophy appeared on day 20, and returned to normal on day 51. For the first three days, wall motion abnormalities are considered to have been caused by myocardial stunning with reversible myocardial damage associated with coronary vasospasm [5]. Myocardial stunning is a reduction in the transient contractility that occurs after brief myocardial ischemia without myocardial necrosis. In experiments involving dogs, reperfusion after 15 min, minor coronary occlusion caused myocardial stunning (reversible damage) and restored normal wall motion after about 1 week. Reperfusion after coronary occlusion of $\geq$20 min resulted in myocardial necrosis (irreversible damage) and wall thinning. Coronary occlusions of $<$5 min were associated with only transient wall motion abnormalities [6,7].

In this case, motion abnormalities of the left ventricular wall improved on day 3, but apical hypertrophy appeared on day 20. The mechanisms involved in the transient apical hypertrophy observed in this case remain majorly unknown. We hypothesize that the borderline zone of 15–20 min of coronary occlusion can cause myocardial edema (apical hypertrophy). However, if the coronary occlusion is longer than 20 min, the myocardial wall becomes thin because of necrosis. In contrast, if the duration of coronary occlusion is shorter than 5 min, myocardial edema is rarely evident. Therefore, it is considered that all cases of coronary vasospasm do not always exhibit transient apical hypertrophy. The appearance of apical hypertrophy may depend on the degree of myocardial injury and the differences in the inflammatory responses.

We did not diagnose this case as takotsubo cardiomyopathy but as transient apical hypertrophy associated with coronary vasospasm, because the guidelines for diagnosing takotsubo cardiomyopathy showed spasm of a coronary artery in exclusion criteria [8]. Tsuchihashi et al. reported that coronary spasm was provoked in 10 of 48 (21%) patients with takotsubo cardiomyopathy without significant angiographic stenosis. Furthermore multivessel vasospasm is a rare case in 5 of 48 (10%) patients [9]. Therefore, we did not diagnose this case as takotsubo cardiomyopathy but as transient apical hypertrophy associated with coronary vasospasm.

Although ICD implantation was performed in this case, this strategy for patients exhibiting TdP related with vasospastic angina is controversial (Class IIb). Takagi et al. [10] reported that 14 of the 35 patients with vasospastic angina who survived out of hospital