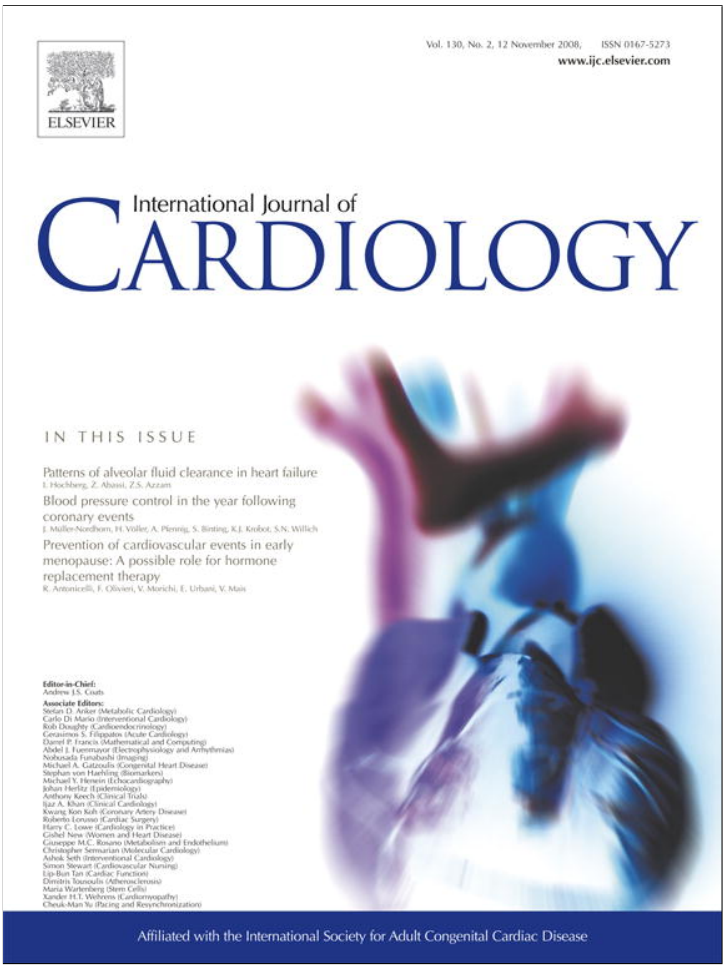

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Letter to the Editor

Apical ballooning syndrome and previous coronary artery disease: A novel relationship

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Abstract

Apical transient left ventricular diskynesia is a recently described entity able to imitate acute coronary syndrome. The presence of previous coronary artery disease (CAD) is an exclusion criterion for this diagnosis in several studies.

We report the case of a sixty-three year-old-caucasian man with previously known CAD, left anterior descending artery (LAD) stented-disease, presenting in the emergency room with angina and ST-segment elevation. A coronariography was urgently performed. No new coronary lesions could be demonstrated. LAD-placed stents were patent and showed no change in their angiographic appearance. Left ventriculogram demonstrated apical diskynesia (Takotsubo-like). Complete and rapid resolution of left ventricular dysfunction was echocardiographycally displayed seven days later. Months after, coronary lesions increased associated with new acute coronary syndromes and new revascularization procedures were required.

The present case supports the idea that CAD and apical transient diskynesia could coexist in the same patient, arising further questions about the pathophysiology, prognosis and management of the latter.

Keywords: Apical ballooning syndrome; Takotsubo cardiomyopathy; Coronary artery disease

1. Case report

Left ventricular transient diskynesia was first described in Japan in 1990 by Sato et al. [1] This syndrome, also called Takotsubo cardiomyopathy or apical ballooning, presents early features similar to acute coronary syndromes. The main characteristics are the presence of normal coronary arteries assessed by coronariography and the rapid and complete resolution of the segmental motion alterations. The estimated annual population incidence is low, calculated as 0.00006% [2]. Prognosis is excellent compared with classic myocardial infarction [3]. Although the majority of studies and case-series/reports exclude patients with previous CAD, this relationship has recently been recognized [4].

We present the case of a 63 year-old caucasian man. No other disease record was present but dislipaemia and a 2 year-history of coronary artery disease initiated with unstable angina on 2001. The coronariography showed a severe stenosis in the proximal left anterior descending artery (LAD) segment. A percutaneous coronary angioplasty with one bare-stent implantation was performed then, and again on February 2003 because of restenosis, with two new bare-stents implant and brachytherapy. Left ventriculogram showed normal systolic function without segmental motion abnormalities (Fig. 1A).

On April 2003, patient developed rest angina, accompanied by inferior 1 mm ST segment elevation and V2-3 ST-depression at admission. The episode lasted 6 h and only

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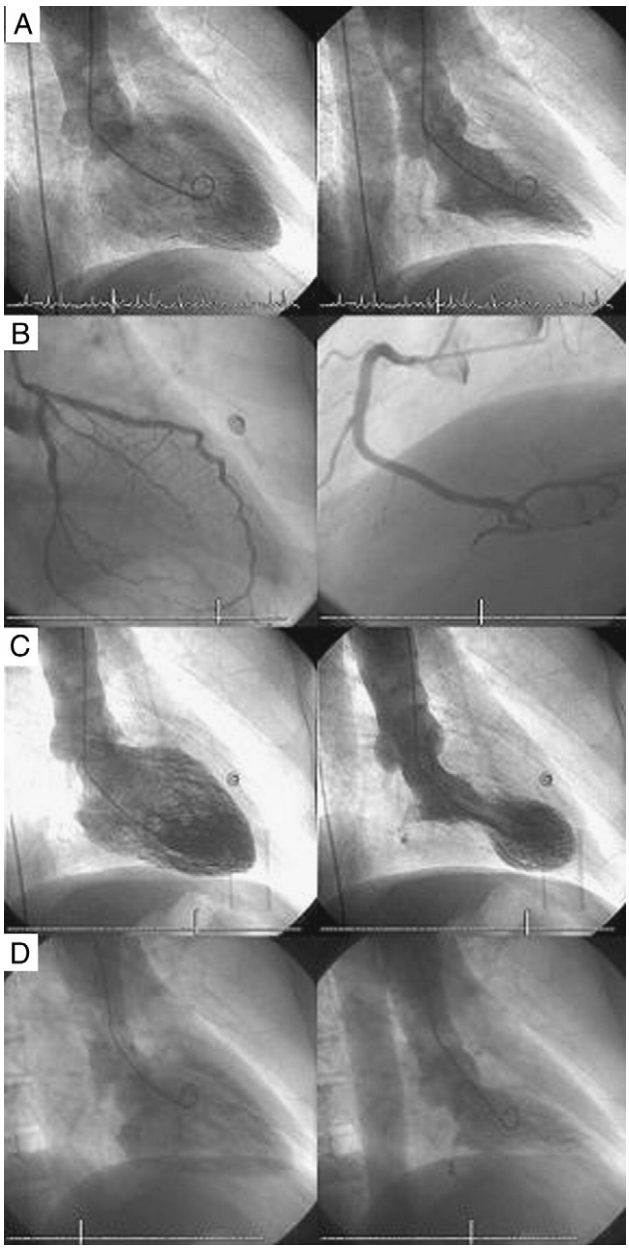

Fig. 1. A) Ventriculogram previous to the episode of transient apical diskynesia, diastole (left) and systole (right). B) Left coronary artery (left) and right coronary artery (right) during the episode of transient apical diskynesia. C) Ventriculogram during the episode of transient apical diskynesia diastole (left) and systole (right) with apical dyskinesia. D) Ventriculogram two years later: diastole (left) and systole (right).

relieved after medical treatment with intravenous nitrates and anticoagulation. He did not complain of fever, drowning nor other symptoms. Coronariography was performed showing no new coronary lesions with LAD-placed stents patent and normal TIMI 3 distal flow. Circumflex (CX) and right coronary (RCA) artery had no significant lesions either (Fig. 1B). Left ventriculogram showed apical diskynesia (Takotsubo-like) with global left ventricular ejection fraction preserved (Fig. 1C). Peak creatin kinase was 933 (CK normal < 170 U/L) and troponine I was 31.0 (normal < 0.2 ng/mL).

Outcome was uneventful and left ventricular segmental motion appeared fully normalized assessed by transthoracic echocardiogram seven days later. In hospital stay was 8 days and treatment at discharge included acetylsalicylic acid, clopidogrel, nifedipine, nitrates and simvastatine.

On January 2005, because of recurrent symptoms a repeated cardiac cathererization was performed displaying normal left ventricular ejection function without new segmental motion abnormalities (Fig. 1D). CAD progressed with LAD total occlusion and antero-lateral wall supplied by collaterals. Then, surgical revascularization was decided, with left internal mammary artery bypass to distal LAD. Currently, the patient presents a NYHA functional class I.

The transient myocardial dysfunction also known as apical ballooning or Takotsubo myocardiopathy represents an special form of myocardial stunning [5]. The pathophysiology remains unknown although several theories have been proposed: anatomic coronary alterations; left ventricular outflow tract obstruction; coronary microvascular dysfunction; vasospasm, evanescent intracoronary thrombus, myocarditis and cathecolamine excess (brain-stress related) [2–5]. Although the apical affectation remains the most frequent transient cardiomyopathy of this type, the recent publication of new variants of transient myocardial dyskinesia arises new questions about this syndrome [4].

In order to standardize diagnosis, various clinical criteria have been proposed. Mayo Clinic criteria [6] require transient hypokinesis, akinesis or dyskinesis of left ventricular segments, the regional wall-motion abnormalities extend beyond a single epicardial vascular distribution; ECG abnormalities or elevated cardiac troponin; absence of recent significant head trauma, pheochromocytoma, myocarditis or hypertrophic cardiomyopathy and the absence of obstructive coronary artery disease or angiographic evidence of acute plaque rupture.

Our aim in presenting this case was to remark that these two entities, CAD and apical ballooning syndrome, could coexist in the same person.

Of note, the transient-motion episode from our patient met the Mayo Clinic criteria, including transient left ventricular motion abnormalities, ST elevation, elevated cardiac necrosis markers and absence of obstructive coronary lesions.

Undoubtedly, the diagnosis of “apical ballooning” in the presence of CAD must be made with caution. But in the other hand, suffering from CAD probably does not protect against “apical ballooning”. Further investigation is needed to explain the link between these two apparently separated entities.

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