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Spontaneous coronary artery dissection and takotsubo syndrome: An often overlooked association; review

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ABSTRACT

Spontaneous coronary artery dissection (SCAD) and takotsubo syndrome (TS) are two cardiovascular syndromes with predilection for women. Both conditions may be preceded by an emotional stress or, for the affected individual, an unusual severe physical exercise. "Restitution ad integrum" occurs in most cases suffering from SCAD or TS with complete angiographic resolution of the dissected vessel and left ventricular dysfunction respectively. Recently, many cases, which were initially diagnosed as TS because of typical left ventricular ballooning pattern showed to have SCAD, have been reported; these cases were deemed to be "SCAD misdiagnosed as TS". The left ventricular wall motion abnormality has been attributed to the ischemia caused by SCAD-affected coronary vessel especially in the left anterior descending artery (LAD) with "wrap-around course". However, the left ventricular ballooning pattern have occurred in patients with SCAD in non-long-wrap-around LAD and SCAD in other coronary branches where coronary ischemia on its own cannot explain the left ventricular ballooning. In this review, sufficient data supporting the evidence for the possibility of coexistence of SCAD and TS is provided. Misdiagnosis of the association of the two conditions may result in mismanagement of the patient with undesirable consequences. Furthermore, the causal links between SCAD and TS is discussed.

Contents

1. Introduction

Spontaneous coronary artery dissection (SCAD) and takotsubo syndrome (TS) are two cardiovascular diseases with a special predilection for women [1,2]. SCAD is defined as a non-atherosclerotic spontaneous separation of the coronary artery wall due to either intimal rupture initiating a tear with intramural hematoma formation or spontaneous bleeding into the coronary arterial wall also with intramural hematoma compressing the true coronary artery lumen resulting in acute coronary stenosis or occlusion (Fig. 1A) [1]. The most frequent presentation of SCAD is clinical signs and symptoms of acute coronary syndrome (ACS) [1]. Takotsubo syndrome also presents with signs and symptoms indistinguishable from ACS [2,3]. The left ventricular wall motion abnormality (LVWMA) in TS has a characteristic circumferential pattern resulting in a conspicuous regional ballooning of the left ventricle (Fig. 1B) [2]. During the last few years, some investigators reported on the occurrence of left ventricular ballooning resembling that of TS in patients with SCAD [4,5]. There has been disagreement on the possibility of simultaneous presentation of SCAD and TS in the same patient [6,7]. In this review, substantial evidence for the possibility of simultaneous occurrence of SCAD and TS is provided. Misdiagnosis of this association

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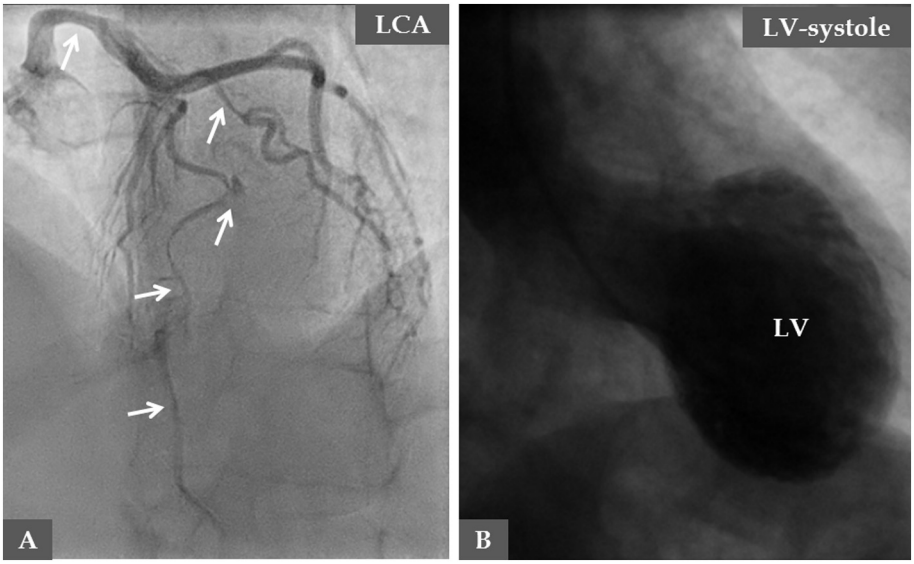

Fig. 1. Multiple sponataneous coronary artery dissection (SCAD) in LMS, LAD and diagonal branch (white arrows) of the left coronary artery (LCA) triggered by an emotional trigger factor (A) in one patient. Mid-apical pattern of takotsubo syndrome (TS) also triggered by an emotional trigger factor (B) in another patient. LAD, left anterior descending artery; LMS, left main stem; LV, left ventricle.

may result in mismanagement of the patient with serious consequences. Furthermore, the causal link between SCAD and TS is discussed.

2. "Spontaneous coronary artery dissection misdiagnosed as takotsubo syndrome"

During the last few years, four publications reported on 12 patients in whom SCAD was either "misdiagnosed as TS" [5,8] or TS was excluded or deemed to be misleading when the SCAD diagnosis was confirmed [9,10]. Chou et al. [5] described 9 cases of SCAD, 4 involving left anterior descending artery (LAD) and 5 involving other coronary side branches (diagonal, marginal or postero-lateral arteries), which according to the authors were "misdiagnosed" as TS. The authors have stated that the LVWMA in these 9 patients corresponded to the territory supplied by the dissected coronary arteries. Furthermore, the investigators have argued against TS because troponin elevation deemed to be much higher than that usually seen in TS [7]. Bakhit and Bin Abdulhak [8] reported on the case of a 30-year-old woman presented 4 days postpartum with a clinical picture of ACS. The initial coronary angiography was interpreted as "non-obstructive coronary artery disease". Left ventriculography showed mid-apical ballooning and the case was initially deemed to be TS. Five days after discharge, the patient returned with a similar presentation as the initial one. New coronary angiography showed signs of "coronary dissection in mid to distal LAD along with apical akinesis". The case was also deemed to be SCAD 'misdiagnosed as TS'.

In clinical practice, SCAD may be missed specially in patients with angiographically invisible type II and type III lesions according to Saw classification of SCAD [11]. SCAD may also be misdiagnosed as a coronary spasm [12]. Type IIB SCAD may be interpreted as a normal tapering vessel variant. Type III SCAD may be misdiagnosed as an atherosclerotic coronary artery lesion [13]. However, to misdiagnose SCAD as TS raises some justifiable questions because the pathology of the two diseases affects two different structures of the heart, the coronary arteries in SCAD and the myocardium with peculiar pattern in TS. Careful analysis of the information on the 9 patients reported by Chou et al. revealed that all the 9 patients had apical LVWMA although 5 patients had non-LAD-SCAD. Case 4 had apical LVWMA and the SCAD involved a relatively small postero-lateral branch, which did not supply the apical region. Case 5 had LVWMA in the anterolateral, apical and inferior regions and the SCAD was in a peripheral segment of the diagonal branch which absolutely did not supply the inferior region. Cases 7, 8 and even 9 had SCAD in the LAD where the apical segments of LAD are visible; a long wrap-around LAD cannot be seen to explain the inferior LVWMA. The higher troponin elevation in these patients does not argue against TS because the troponin elevation is partly explained by the associated myocardial infarction caused by the SCAD. Consequently, in my opinion the SCAD in the 9 patients have been missed but TS was not a misdiagnosis and the patients had most probably SCAD and TS concurrently. The mid-apical ballooning especially in the inferior region in the case described by Bakhit and Bin Abdulhak [8] extends beyond the supply region of the LAD and this case has also been commented [14]. It should be acknowledged that the authors of the above-mentioned two publications and this author disagree on the presence of an association between SCAD and TS [6,7,14]. However, similar thoughts on the existence of the association of TS and SCAD have been presented recently by other investigators [15].

3. "Spontaneous coronary artery dissection excluded takotsubo syndrome"

Ruggiero et al. [9] reported on the case of a 39-year-old woman with extensive SCAD of her mid and distal segments of LAD. Left ventriculography in that patient showed signs of mid-apical ballooning consistent with TS. The authors have "ruled out the diagnosis of TS when the LAD-SCAD diagnosis was confirmed". Probably, this conclusion was based on the existing diagnostic criteria for TS where ACS is an important exclusion's criterion for TS [16]. However, the left ventriculography in that case have shown clearly a large mid-apical ballooning where the LVWMA in the case extends beyond the LAD territory as seen from LAD anatomy in the case presentation. Information on cardiac magnetic resonance (CMR) imaging and the reversibility of LVWMA is lacking in that presentation. The most acceptable explanation of LVWMA in that case is post-ischemic myocardial stunning (PIMS). PIMS is a form of TS triggered by an acute ischemic insult [4] and was actually the starting point of TS [17,18]. Consequently, the case described by Ruggiero et al. suffered both SCAD and TS simultaneously. Recently, Macaya et al. [10] reported on a 58-year-old female patient who presented with chest pain after an emotional stress. Echocardiography and left ventriculgraphy revealed LVWMA with an apical ballooning pattern. The authors suggested the diagnosis of TS relying on the history of chest pain triggered by death of a relative and the finding of left ventricular apical ballooning. In addition, there was a moderate stenosis with

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