Spontaneous coronary artery dissection (SCAD) and takotsubo cardiomyopathy (TTC) are two non-atherosclerotic causes of myocardial infarction. They share several common features. Firstly, they have a predilection for the female gender and, in both, the exact mechanism has yet to be fully established. Both could be responsible for an acute coronary syndrome. Hence, we want to do further consideration based on pathophysiology, literature review, and cases presented affected by both entities up today.

Keywords: Spontaneous coronary artery disease (SCAD); takotsubo cardiomyopathy (TTS); coronary artery disease (CAD); acute coronary syndrome (ACS); interventional cardiology; coronary angiography; intravascular imaging

Submitted May 28, 2017. Accepted for publication Nov 02, 2017.

doi: 10.21037/jtd.2017.11.07

View this article at: http://dx.doi.org/10.21037/jtd.2017.11.07
ventricular motion abnormalities in post-ischemic myocardial stunning and TTC have similar clinical presentation and course, ECG, imaging and histopathologic findings (5).

Moreover, natural history of both SCAD and TTC is often represented by spontaneous healing.

Recently a chicken or egg question on the topic has been raised by several authors: is it possible that SCAD and TTC overlap? Two main hypothesis have been made: (I) SCAD could be the stressful event leading to TTC, as previously suggested for MIs in general (4); (II) The external torsion forces and mechanical solicitations associated with typical wall motion abnormalities in TTC could cause the dissection of intima especially in segments located at the border between basal hyperkinesia and mid-apical akenesis (6).

Coexistence of SCAD and TTC has been reported in five case reports and one retrospective case series until today, including a totality of 14 patients. Their features are listed in Table 1 and in Table S1 and analyzed below.

All patients were female with an average age of 51 years. Only one-third of them had cardiovascular risk factors (hypertension) whether none had remarkable past medical history. Eight cases (57.1%) followed stressful precipitating factors.

Twelve patients presented with elevation of troponin (the remaining two case reports do not refer to cardiac biomarkers).

ECG showed ST-elevation in 9 patients (64.3%), ST-depression or anterolateral T-wave inversion in 3 patients (21.4%) and ventricular tachycardia in 2 patients (14.3%). Evolution of ECG in case reports showed the appearance of widespread or anterior T-wave inversion and prolongation of QTc, stereotypical evolution of TTC that can also be found in myocardial stunning (Wellen’s pattern).

Echocardiographic findings and/or ventriculography were consistent with the typical takotsubo pattern of wall abnormalities, namely basal hyperkinesia and mid-apical akenesis of the mid-apical segments, with concurrent ballooning in 5 patients (35.7%).

Importantly, cardiac magnetic resonance was performed in three patients and late gadolinium enhancement was

---

Table 1: Clinical, instrumental and angiographic features of patients reported in literature

<table>
<thead>
<tr>
<th>Reference</th>
<th>Age (years)</th>
<th>Stressor</th>
<th>Clinical presentation</th>
<th>Location of ECG abnormalities</th>
<th>IRA</th>
<th>Segment</th>
<th>SCAD type</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chou, 2015 (7)</td>
<td>54</td>
<td>–</td>
<td>STEMI</td>
<td>Inferolateral RPL</td>
<td>–</td>
<td>–</td>
<td>2B</td>
</tr>
<tr>
<td>Chou, 2015 (7)</td>
<td>70</td>
<td>–</td>
<td>STEMI</td>
<td>Inferior OM1 D1</td>
<td>–</td>
<td>–</td>
<td>2A</td>
</tr>
<tr>
<td>Chou, 2015 (7)</td>
<td>45</td>
<td>Exercise</td>
<td>NSTEMI</td>
<td>Anterolateral LAD Distal</td>
<td>2A</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chou, 2015 (7)</td>
<td>42</td>
<td>Work</td>
<td>TVNS</td>
<td>–</td>
<td>LPL</td>
<td>Distal</td>
<td>2B</td>
</tr>
<tr>
<td>Chou, 2015 (7)</td>
<td>62</td>
<td>–</td>
<td>NSTEMI</td>
<td>Anterolateral D1</td>
<td>–</td>
<td>2A</td>
<td></td>
</tr>
<tr>
<td>Chou, 2015 (7)</td>
<td>59</td>
<td>Virus</td>
<td>STEMI</td>
<td>Lateral D1 Mid</td>
<td>2B</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chou, 2015 (7)</td>
<td>60</td>
<td>Mourning</td>
<td>NSTEMI</td>
<td>Anterolateral D3-LAD Distal</td>
<td>2B</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chou, 2015 (7)</td>
<td>50</td>
<td>Virus</td>
<td>TV</td>
<td>–</td>
<td>LAD</td>
<td>Distal</td>
<td>2B</td>
</tr>
<tr>
<td>Chou, 2015 (7)</td>
<td>50</td>
<td>–</td>
<td>STEMI</td>
<td>Anterolateral LAD Distal</td>
<td>–</td>
<td>–</td>
<td></td>
</tr>
<tr>
<td>Y-Hassan, 2013 (8)</td>
<td>39</td>
<td>–</td>
<td>STEMI</td>
<td>Anterolateral LAD Mid</td>
<td>2A</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Y-Hassan, 2016 (9)</td>
<td>61</td>
<td>–</td>
<td>STEMI</td>
<td>Anterolateral D1 Distal</td>
<td>2A</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bakhit, 2016 (10)</td>
<td>30</td>
<td>Post-partum</td>
<td>STEMI</td>
<td>Anterior LAD Mid-distal</td>
<td>2B</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ruggiero, 2016 (11)</td>
<td>39</td>
<td>Quarrel</td>
<td>STEMI</td>
<td>Septal LAD Mid-distal</td>
<td>2A</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Y-Hassan, 2017 (12)</td>
<td>54</td>
<td>Mourning</td>
<td>STEMI</td>
<td>Inferior OM1 Distal</td>
<td>2B</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

D1, first diagonal branch; D3, third diagonal branch; IRA, infarct related artery; LAD, left anterior descending artery; LPL, left posterolateral artery; NSTEMI, non-ST-elevation myocardial infarction; OM1, first obtuse marginal branch; RPL, right posterolateral artery; SCAD, spontaneous coronary artery dissection; STEMI, ST-elevation myocardial infarction; TV, ventricular tachycardia; TVNS, non-sustained ventricular tachycardia.
only present in the supply region of the dissected arteries, typically sparing mid and apical regions. This could be the proof of the simultaneous presence of TTC (with its typical absence of delayed enhancement) (2) and myocardial ischemia, confirmed by regional subendocardial delayed hyper enhancement, caused by a coronary dissection.

Coronary angiography was always performed. 13 out of 14 angiograms (one was not available in the Table S1 of the case series) showed Type 2 SCADs, the most common angiographic appearance (3) of coronary dissection. This diffuse and usually smooth narrowing frequently involves the mid to distal segments of coronary arteries and may need intravascular imaging for diagnostic certainty. Nevertheless, IVUS was performed in only two of this cases. Seven lesions were subtype B.

In 12 angiograms (85.7%) the culprit lesion was found in the left anterior descending artery or in one of its branches and in 4 patients, the mid segments were involved. In these patients, the mechanism proposed by Madias (6) (hypothesis 2) seems plausible.

In 13 patients (92.9%) a conservative strategy was chosen, in line with the evidence. In three of these patients, coronary angiography was repeated in the follow-up period and showed complete spontaneous healing. One patient was treated with the implant of a biodegradable vascular scaffold.

In conclusion, while it is virtually impossible to declare whichever occurs first, it appears likely that each of them may have provoked the other equally. The key to this dilemma could be represented by coronary angiography, and furthermore by intravascular imaging, especially optical coherence tomography (OCT), since (I) SCAD could be underestimated because of its misleading angiographic appearance; and (II) the prognostic value of coronary slow flow in patients with TTC has been previously described. These findings could be in fact associated with microvascular dysfunction as well as with an underlying dissection of the infarct related artery.

We believe that given the absence of international guidelines or expert consensus documents on the topic, a systematic approach for the early detection of SCAD is needed. Our group already proposed a scoring system in this respect (13). Expanding the role of angiography and, moreover, of intravascular imaging, currently underperformed and restricted to exclusion tools for the diagnosis of TTC, could increase the diagnostic certainty and improve prognosis in these two clinical conditions.

Acknowledgements
None.

Footnote
Conflicts of Interest: The authors have no conflicts of interest to declare.

References


Cite this article as: Buccheri D, Zambelli G. The link between spontaneous coronary artery dissection and takotsubo cardiomyopathy: analysis of the published cases. J Thorac Dis 2017;9(12):5489-5492. doi: 10.21037/jtd.2017.11.07