Pancreatitis and the broken heart

Petra Grubić Rotkvić^{1,*},
Jozica Šikić^{1,2},
Jasna Čerkez Habek^{1,3},
Dean Strinić¹,
Zdravko Babić⁴,
Marin Pavlov⁴

¹University Hospital „Sveti Duh“, Sveti Duh 64, Zagreb, Croatia

²University of Zagreb School of Medicine, Zagreb, Croatia

³Croatian Catholic University, Zagreb, Croatia

⁴University Hospital Centre „Sestre milosrdnice“, Zagreb, Croatia

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***ADDRESS FOR CORRESPONDENCE:** Petra Grubić Rotkvić, Klinička bolnica Sveti Duh, Sveti Duh 64, HR-10000 Zagreb, Croatia. / Phone: +385-99-593-5192 / E-mail: petra.grubic84@gmail.com

ORCID: Petra Grubić Rotkvić, <https://orcid.org/0000-0002-2587-1932> • Jozica Šikić, <https://orcid.org/0000-0003-4488-0559> • Jasna Čerkez Habek, <https://orcid.org/0000-0003-3177-3797> • Dean Strinić, <https://orcid.org/0000-0001-6345-2037> • Zdravko Babić, <https://orcid.org/0000-0002-7060-8375> • Marin Pavlov, <https://orcid.org/0000-0003-3962-2774>

Background: Broken heart syndrome or Takotsubo syndrome (TTS) is considered a type of acute and usually reversible heart failure episode, often indistinguishable from acute coronary syndromes, characterized by the lack of significant obstructive coronary artery disease. The most characteristic wall motion pattern is apical ballooning. Reversible LV dysfunction affects more than one coronary territory and timelines of recovery is variable. It is believed that enhanced sympathetic stimulation induces transient myocardial stunning through a variety of mechanisms associated with emotional or physical stress. To the best of our knowledge, there is only one published case report of TTS resulting from the exacerbation of chronic pancreatitis¹.

FIGURE 1. ECG on admission showing ST elevation in leads V1-V3.

Case report: We report the case of a 69-year-old lady with four-year history of chronic pancreatitis who came to our emergency room with acute epigastric pain and vomiting associated with ST-elevation in precordial leads (**Figure 1**) and elevated troponin (troponin I 1496 ng/L). Moreover, serum amylase (315 U/L) and lipase (249 U/L) were increased. Echocardiography revealed a dilated and hypokinetic apex with reduced left ventricle ejection fraction (LVEF 40%). The abdominal ultrasound showed inhomogeneous pancreas with calcifications. Obstructive lesions of coronary arteries were absent on the angiogram and apical ballooning was demonstrated on left ventriculography (**Figure 2**). The findings were consistent with TTS and acute exacerbation of chronic pancreatitis. The patient was managed with intravenous crystalloids, analgesics, anti-emetics, beta blockers, ACE inhibitors. Over the next 2-3 days she was able to tolerate an oral diet. The ECG typi-

FIGURE 2. Left ventriculography demonstrating apical ballooning.

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cally evolved with T wave inversion and QT interval prolongation (**Figure 3**) without significant arrhythmia. Echocardiography at discharge showed normalization of LVEF (55%) with mildly hypokinetic apical segment of the interventricular septum

FIGURE 3. ECG on day 3 demonstrating T-wave inversions and QT prolongation.

Conclusion: Acute pancreatitis as well as the exacerbation of chronic pancreatitis are stressors that increase sympathetic stimulation of the heart and are associated with distributive shock that leads to transient myocardial ischemia and microvascular hypoperfusion^{1,2}. Our case highlights the rare association of exacerbation of chronic pancreatitis with TTS.

LITERATURE

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