Spontaneous conus branch occlusion mimicking anterior ST-segment elevation myocardial infarction

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A 64-year-old man was admitted due to out--of-hospital cardiac arrest. No bystander cardiopulmonary resuscitation was provided with at least 10 to 15 minutes of downtime prior to arrival of paramedics. The initial rhythm was ventricular fibrillation. Spontaneous circulation was restored after 20 minutes of cardiopulmonary resuscitation. A post-arrest electrocardiogram demonstrated ST-segment elevation in leads $V_1 – V_2$ (FIGURE 1A). The patient was transferred to the hospital and underwent coronary angiography, which revealed the acutely occluded conus branch of the nondominant right coronary artery (RCA) (FIGURE 1B; Supplementary material, Video S1). The conus branch had slow filling to the point of occlusion with a meniscus. Furthermore, a nonculprit lesion in the mid portion of the RCA and several nonculprit lesions in the left coronary artery were detected (Supplementary material, Figure S1).

After engagement of the RCA with a 6F IM Mach1 guide catheter, an occlusion in the infarct-related artery was crossed with a Prowater coronary guidewire (FIGURE 1C). Despite balloon angioplasty with a 1.5/15 mm Sprinter Legend balloon dilation catheter (FIGURE 1C), we could not restore the flow in the conus branch (FIGURE 1D). Given the risk-benefit ratio, no more attempts were made to open the infarct-related artery. As a result of prolonged downtime, the patient had poor neurological outcome and died of diffuse ischemic brain injury 3 days later.

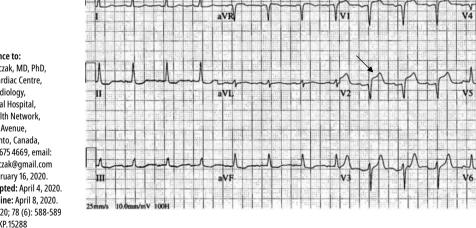


FIGURE 1 A – an electrocardiogram demonstrating ST-segment elevation in leads $V_1 - V_2$ (arrows)

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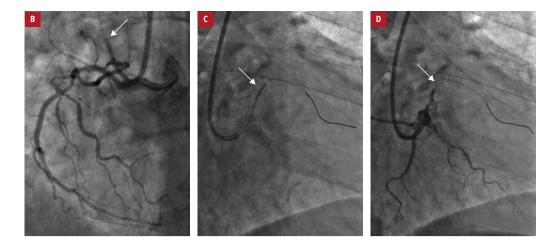


FIGURE 1 B – right coronary angiography showing an acutely occluded conus branch (arrow); **C** – balloon angioplasty of the conus branch (arrow); **D** – no restoration of blood flow through the infarct-related coronary artery (arrow)

The conus branch supplies the right ventricular outflow tract, which is a potential origin of ventricular arrhythmias. Therefore, despite the small caliber of the vessel, conus branch occlusion may result in fatal arrhythmic events. While several reports of an iatrogenic conus branch occlusion have been published, a spontaneous occlusion of the conus branch causing ST-segment elevation myocardial infarction (STEMI) is exceptionally rare. To the best of our knowledge, this is the third report of STEMI due to a noniatrogenic conus branch occlusion.

In some cases of STEMI, the culprit lesion or artery may be unclear. A careful analysis of the angiogram is essential to ensure that all myocardial territories are accounted for and no branches are missing. Occlusion of a branch at its ostium may be difficult to appreciate. Right ventricular branches, septal branches, and the conus artery are not typical targets for a coronary intervention, and thus have the potential to be unappreciated, yet they all can cause STEMI. In addition, the conus branch may arise separately from the right aortic sinus and may be undetected on selective right coronary angiography. Optical coherence tomography may be used in suspected culprit lesions to assess for the presence of an intraluminal thrombus and plaque rupture or erosion. Cardiac magnetic resonance imaging may be of use to localize infarction,5 bearing in mind that time to reperfusion is important in STEMI. In our case, careful assessment of the angiogram allowed us to identify acute conus branch occlusion.

Our case highlights that acute conus branch occlusion can mimic anterior STEMI due to the left anterior descending artery lesion and should be considered in cases with ST-segment elevation in anterior leads.

SUPPLEMENTARY MATERIAL

Supplementary material is available at www.mp.pl/kardiologiapolska.

ARTICLE INFORMATION

CONFLICT OF INTEREST None declared.

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