Drug refractory arrhythmic vasospastic angina successfully treated with bilateral cardiac sympathetic denervation

Piotr Szolc[1, 2], Sebastian Stec[4], Paweł Kleczyński[2, 3], Piotr Zamorski[5], Łukasz Niewiara[2, 3], Bartłomiej Guzik[2, 3], Michalina Jelonek[6], Jacek Legutko[2, 3]

[1]Department of Emergency Medicine, Faculty of Health Sciences, Jagiellonian University Medical College, Kraków, Poland
[2]Clinical Department of Interventional Cardiology, Saint John Paul II Hospital, Kraków, Poland
[3]Department of Interventional Cardiology, Faculty of Medicine, Institute of Cardiology, Jagiellonian University Medical College, Kraków, Poland
[4]Institute for Cardiovascular Science, CardioMedicum Medical Centre, CardioNeuroLab, Kraków, Poland
[5]Department of Thoracic Surgery, HolyFamily Hospital, New Medical Technologies, Rudna Mała, Poland
[6]Student Scientific Group of Modern Cardiac Therapy at the Department of Interventional Cardiology, Jagiellonian University Medical College, Kraków, Poland

Correspondence to:
Prof. Jacek Legutko, MD, PhD, FESC,
Department of Interventional Cardiology,
Faculty of Medicine,
Institute of Cardiology,
Jagiellonian University Medical College,
Prądnicka 80, 31–202 Kraków, Poland,
phone: +48 12 614 35 01,
e-mail: jacek.legutko@uj.edu.pl

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We present the case of a 53-year-old male with ischemia and non-obstructive coronary arteries. The patient was initially admitted to our department with class III angina according to the Canadian Cardiovascular Society scale and episodes of ST-segment elevation with symptomatic non-sustained ventricular tachycardia (nsVT) recorded on 24-hour electrocardiography (ECG) monitoring (Figure 1A). Transthoracic echocardiography revealed normal left ventricle ejection fraction and no significant valve pathology. Coronary angiography showed left coronary artery dominance with no significant lesions. Physiological assessment using a PressureWire X (Abbott Vascular, Santa Clara, CA, US) and Coroflow software (Coroventis, Uppsala, Sweden) revealed normal results for coronary flow reserve (3.5 for left anterior descending artery and 3.1 for the left circumflex artery) and index of microcirculatory resistance (12 U for the left anterior descending artery and 19 U for the left circumflex artery). In contrast, a provocation test with acetylcholine showed significant left coronary artery spasm (>90% diameter), chest pain, ischemic changes, and nsVT on 12-lead ECG monitoring (Figure 1B–C). Pharmacological treatment of vasospastic angina (VSA) based on the maximum tolerated doses of calcium channel blocker (diltiazem) and

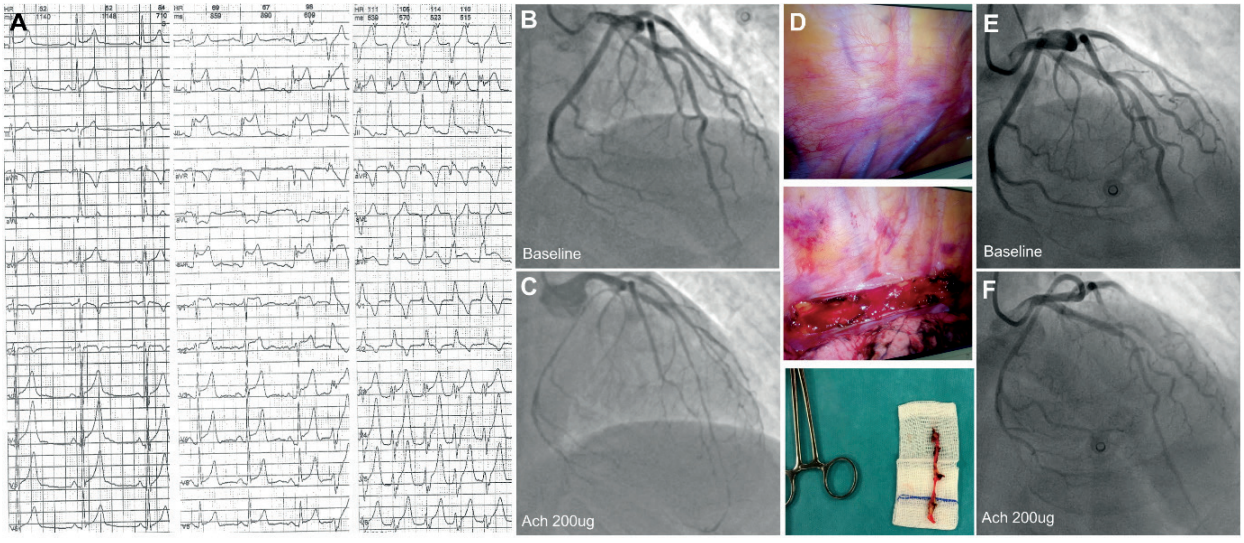

Figure 1. Vasospastic angina treated with bilateral cardiac sympathetic denervation (BCSD). **A.** Initial 24-hours electrocardiogram (ECG). **B.** Left coronary artery (LCA) angiography. **C.** Positive result of the provocation acetylcholine test in the LCA (critical spasm, chest pain, ischemic changes in ECG). **D.** BCSD performed in video-assisted thoracoscopic surgery. **E.** Re-assessment: LCA angiography. **F.** Re-assessment: negative result of the provocation acetylcholine test in the LCA (no significant spasms, chest pain, or ischemic changes on ECG)

long-acting nitrate was administered. However, the patient reported persistent, significant angina and symptoms of nsVT. One month later, after signing informed consent, the patient underwent bilateral cardiac sympathetic denervation (BCSD) in video-assisted thoracoscopic surgery with removal of sympathetic ganglia and fibers including the Kuntz nerve from Th1 to Th4–Th5 region (Figure 1D). Immediately after the procedure, the patient did not show any symptoms of angina or ventricular arrhythmia. The patient experienced no side effects from BCSD, but suffered from intense, perioperative pain, which required painkillers and lasted for up to two months. Two months after BCSD, a control electrophysiology study with an isoproterenol infusion and a hyperventilation test showed no inducibility of nsVT and ST changes. Nine months later, the patient underwent a scheduled invasive VSA reassessment. He had no clinical symptoms, nor ventricular arrhythmias in the several records of 24-hour ECG monitoring. During the provocation test with acetylcholine, no significant epicardial artery spasms, chest pain, or ischemic changes were registered. Likewise, there were no nsVT on ECG monitoring (Figure 1E–F).

Thise case illustrates a multidisciplinary approach to arrhythmic VSA with a second reassessment of the asymptomatic course by an invasive electrophysiology study and a provocation test with acetylcholine. VSA, especially arrhythmic VSA, is a recognized risk factor for myocardial infarction and sudden cardiac death [1, 2]. Conventional pharmacotherapy, based on calcium channel blockers and long-acting nitrates, is not always efficient [3, 4]. BCSD should be considered an effective treatment method in patients with refractory arrhythmogenic VSA. [5]. BCSD implementation and further invasive reassessment of treatment efficacy appear to be a promising management strategy for such patients and deserves implementation in clinical practice.

Supplementary material

Supplementary material is available at https://journals.viamedica.pl/polish_heart_journal.

Article information

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