

Kardiologia Polska

The Official Peer-reviewed Journal of the Polish Cardiac Society since 1957

Online first

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ISSN 0022-9032 e-ISSN 1897-4279

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Article type: Clinical vignette **Received:** September 28, 2024

Accepted: November 7, 2024

Early publication date: November 25, 2024

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Drug refractory arrhythmic vasospastic angina successfully treated with bilateral cardiac

sympathetic denervation

Short title: Arrhythmic vasospastic angina treated with thoracic sympathectomy

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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 angina class III according to Canadian

Cardiovascular Society scale and episodes of ST segment elevation with symptomatic non-

sustained ventricular tachycardia (nsVT) recorded in 24-hours 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 left circumflex artery) and index of microcirculatory resistance (12 U for left anterior descending artery and 19 U for left circumflex artery). During a provocative test with acetylcholine significant left coronary artery spasm (>90% diameter), chest pain, ischemic changes, and nsVT on 12-lead ECG monitoring were observed (Figure 1B-C). Pharmacological treatment of vasospastic angina (VSA) based on the maximum tolerated doses of calcium channel blocker (diltiazem) and long-acting nitrate was administered, however persistent significant angina and symptoms of nsVT were reported by the patient. One month later, after signing an informed consent, the patient underwent bilateral cardiac sympathetic denervation (BCSD) by video-assisted thoracoscopic surgery with removal of sympathetic ganglia and fibers including Kuntz nerve from Th1 to Th4-Th5 region (Figure 1D). Immediately after procedure, the patient became asymptomatic with regard to angina and ventricular arrhythmia. The patient experienced no side effects from BCSD, but suffered from an intense, perioperative pain, which required the use of painkillers and lasted for up to two months. Two months after BCSD, a control electrophysiology study was performed, which revealed no inducibility of nsVT as well as ST changes by isoproterenol infusion and hyperventilation test. Nine months later, the patient underwent a scheduled invasive VSA reassessment. He had no clinical symptoms, nor ventricular arrhythmias on several 24-hours ECG monitoring. During the provocative test with acetylcholine, no significant epicardial artery spasm, no chest pain, no ischemic changes and no nsVT in ECG monitoring were registered (Figure 1E–F).

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

Supplementary material

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

Article information

Conflict of interest: None declared.

Funding: None.

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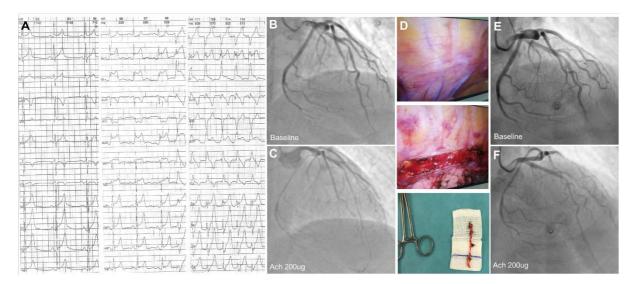


Figure 1. Vasospastic angina treated with bilateral cardiac sympathetic denervation (BCSD). **A.** Initial 24-hours electrocardiogram (ECG). **B.** Left coronary artery (LCA) angiography. **C.** Positive result of provocative acetylcholine test in LCA (critical spasm, chest pain, ischemic changes in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