Microvascular spasm: between *experientia* and *evidentia*

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We thank our colleagues for their letter "No reflow or slow flow?" [1], referring to our Image in Intervention titled "Coronary slow flow and microvascular spasm as underrecognized cause of chest pain" [2].

First, we acknowledge that continuous thermodilution has less variability on repeated measurements than bolus thermodilution during the assessment of coronary microvascular function, as it quantifies absolute coronary flow and microvascular resistance independently of epicardial stenoses and myocardial mass [3]. We congratulate the authors on their interesting case demonstrating intact vasodilatory capacity but raised coronary resistance on continuous thermodilution in a middle-aged postmenopausal woman. Nevertheless, when presenting their case, the authors underlined the need for tailored therapy, but did not provide any information regarding the indication for angiography, initiated pharmacotherapy and patient response to treatment. In contrast, anginal symptoms of our previously described patient were clearly alleviated at the 2-month follow-up visit after the initiation of a calcium channel blocker, which is the recommended therapy in coronary microvascular spasm [4]. Hence, despite the limitations of bolus thermodilution-derived indices, the clinical course of the patient seems to confirm the diagnosis.

Second, the authors underline the differences between the acute and chronic coronary slow flow phenomenon (CSFP) and imply that the response to 2 µg of acetylcholine observed during the provocation test in our patient could have been due to a "no reflow" phenomenon from inadvertent air embolism rather than a "slow flow" phenomenon. Although we did not notice any macroscopic air embolization on angiography, this scenario cannot be excluded and a rechallenge with the same or an escalated dose of acetylcholine would indeed clarify the situation. The air-related "no-reflow" typically lasts more than 5 min and does not improve with i.c. nitroglycerine, but rather requires the administration of sodium nitroprusside or other microcirculatory agents. In the presented case, the coronary flow improved immediately after administering 200 mg of nitroglycerin, which speaks against the "no-reflow" theory. Furthermore, considering the clear improvement in patient symptom burden, repeated testing does not seem clinically justified for patient well-being.

Third, the authors imply that the patient had exertional and not rest angina, which is not typical for coronary microvascular spasm. The hitherto largest (n = 686), international, prospective cohort study by the Coronary Vasomotor Disorders International Study (COVADIS) Group showed that microvascular angina patients, in whom coronary microvascular spasm was the most frequent etiology (42%), present both with rest (36%) and effort angina (14%), a combination of both (18%), shortness of breath (18%) and other symptoms such as fatigue (19%). The reason for this is that mechanisms and etiologies of microvascular angina (MVA) are multifactorial, heterogeneous and overlapping, including reduced coronary flow reserve, microvascular spasm, increased microvascular resistance, and/or CSFP [5]. Hence, it is challenging to establish the etiology of MVA based solely on our clinical experience regarding anginal symptoms. Therefore, objective testing to find evidence of MVA was advocated in 2019 by the European Society of Cardiology [3]. Our patient presented with both evidence of microvascular spasm and CSFP in the acetylcholine provocation test, accompanied by chest pain and ST-segment depression in inferior leads, which resolved following intracoronary nitroglycerin administration, fulfilling the COVADIS diagnosis criteria for MVA [5].

To summarize, we thank the colleagues for the possibility of discussing our patient and for sharing their own experience with continuous thermodilution, which we are about to implement in daily clinical practice. MVA remains a complex and underappreciated diagnostic and therapeutic challenge from a scientific and clinical viewpoint.

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We hope this discussion will be of added value to other colleagues investigating MVA pathophysiology and developing novel therapeutic strategies to improve clinical outcomes and the quality of life of our patients with MVA.

Conflict of interest

The authors declare no conflict of interest.

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