A cataclasm due to spasm

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Abstract We describe a 59-year-old patient presenting with ST-elevation acute coronary syndrome. Coronary angiography with intravascular ultrasound (IVUS) showed an important nonsignificant atherosclerotic lesion in the proximal left anterior descending artery (LAD) and presence of bridging in the mid-LAD. Our hypothesis was that focal spasm at this site was the cause of transmural ischaemia; therefore, treatment was given by performing a percutaneous coronary intervention (PCI) of the lesion. The patient remained symptom-free which confirmed our conclusion. The myocardial bridging had no clinical implications at this moment.

Keywords Coronary spasm · Myocardial bridging · Intravascular ultrasound

A 59-year-old negroid male presented to the emergency department with chest pain. His previous medical history consisted of heterozygous alpha thalassaemia type 2, reflux disease and lumbar hernia. A coronary angiogram 6 years ago had shown only slight vessel wall abnormalities. Cardiac risk factors were hypertension, a positive family history for coronary artery disease and nicotine abuse in the past. There was no history of cocaine abuse. During the last

5 months he had been complaining of sudden chest pain occurring at rest, which radiated to the left arm and only lasted for roughly 10 min. He had never had any exercise-induced symptoms. The pain also reacted swiftly to sublingual nitrate. An exercise test performed in the week before his admission showed no abnormalities. His current medication was acetylsalicylic acid, amlodipine, losartan, bisoprolol, proton pump inhibitor and ranitidine. Blood pressure was 129/79 mmHg with a pulse of 53 beats/min. Further physical examination revealed no abnormalities. The symptom-free electrocardiogram showed sinus rhythm and diffuse repolarisation abnormalities with T-wave inversion in leads II, III and V4 to V6. During chest discomfort, there was ST elevation in leads I, aVL, and V2 to V6 with reciprocal depression in leads III and aVF (Fig. 1). Laboratory tests showed no abnormalities and the cardiac enzymes remained normal. He was treated for an acute coronary syndrome with clopidogrel, fraxiparine and intravenous nitroglycerin. Coronary angiography showed 50 % stenosis of the proximal LAD (Fig. 2), myocardial bridging of the mid LAD, 90 % stenosis of the first and second diagonal branch and 70 % stenosis of margo obtuse and posterolateral branches. The culprit lesion appeared to be the proximal LAD as this could explain the electrographic changes. Due to the fact that this stenosis did not seem to be significant on visual assessment, an FFR measurement was performed. This showed a value of 0.96; therefore, the lesion was not considered to be haemodynamically important and PCI was not performed. Although the angiogram showed no evidence of coronary spasm the clinical evidence started to point in this direction. After discontinuing bisoprolol and under treatment with maximum doses of oral nitrate and calcium antagonist he had recurring short and sudden episodes of chest pain. The electrocardiogram again showed

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Fig. 1 Electrocardiogram without symptoms (a) and with chest pain showing ST elevation in the precordial and lateral leads (b)

transient elevation in the precordial and lateral leads. A new angiogram was performed with an additional IVUS to judge whether a PCI could treat his condition. This showed significant plaque (atheroma area 14.9 mm^2 , atheroma burden 72 %) in the proximal LAD with a lumen area stenosis of 62 %. Furthermore the bridging segment in the mid-LAD was visualised (Fig. 3). The short-lasting symptoms together with the anterolateral electrographic changes led us to

believe that spasm at the site of the proximal LAD plaque was the culprit lesion. Again there was no angiographic evidence of spasm and we chose not to medically induce this.

The proximal LAD was treated with PCI and placement of a drug-eluting stent (Xience prime 4.00/12 mm). He remained symptom-free after 4 months follow-up which further supports our hypothesis. The syndrome of angina

Fig. 2 Coronary angiography showing the LAD in diastole (**a**) and systole (**b**). Proximal lesion (*arrow*). Bridging segment (*asterisk*)

Fig. 3 Intravascular ultrasound of the proximal LAD showing a significant plaque (**a**), and distal LAD showing an echo lucent band typical for myocardial bridging (**b**)

pectoris with transmural ischaemia due to spasm is known as variant angina and was described by Prinzmetal in the 1950s. The majority of patients have underlying coronary artery disease and spasm occurs mostly at or near the site of a significant luminal obstruction. Less often there is mild or no obstruction. It also occurs in patients with normal coronary arteries, although small areas of occult atherosclerotic disease in these cases cannot be ruled out. Usually, medical treatment with nitrates or calcium antagonists is the treatment of choice in this condition and beta blocker therapy is discontinued. However, there are patients who are refractory to oral therapy similar to our case [1, 2]. Although the FFR measurement demonstrated no ischaemia in the absence of spasm, additional IVUS demonstrated a large plaque burden. It can be appreciated that when spasms appear this would result in a severe, significant stenosis. We were convinced that this had been the case, although we could not prove the mechanism of spasm by means of additional ergovinine or acetylcholine. These medications should be used with great caution as they can lead to severe complications such as myocardial infarction. The other challenging aspect in this patient was the accidental finding of accompanying bridging in the mid-LAD. Bridging has a typical

appearance with an echo lucent signal surrounding the coronary artery ('half-moon phenomenon') representing the intramyocardial course of the coronary artery. One should avoid confusing the visual aspect with a coronary dissection. The systolic compression can also be seen with IVUS [3–5]. Our case illustrates that additional IVUS can help in choosing the appropriate treatment strategy.

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