Kardiologia Polska 2014; 72, 4: 389; DOI: 10.5603/KP.2014.0078 ISSN 0022–9032

STUDIUM PRZYPADKU / *CLINICAL VIGNETTE*

Acute anterior myocardial infarction due to stent thrombosis after bee stings

Ostry zawał ściany przedniej serca spowodowany zakrzepicą w stencie po użądleniu przez pszczoły

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A 60-year-old man was admitted to the emergency department with chest pain and pruritus. He was diabetic and two months previously his left anterior descending artery had been stented. He was exposed to multiple bee stings 4 h prior to presentation. On physical examination, vital signs were stable. A 12-lead electrocardiogram showed anterior ST-segment elevation. With the aim of primary percutaneous coronary intervention, he was transferred to the catheterisation laboratory. Selective coronary angiography demonstrated that the left anterior descending artery stent was totally occluded with thrombus (Fig. 1A). Non-critical lesions were visualised in other coronary arteries. The lesion was passed with wire easily and appropriate distal flow was achieved after balloon inflation (Fig. 1B). Follow-up period in the coronary care unit was uneventful, and he was discharged without any complication. A diagnosis of Kounis syndrome (KS) secondary to bee sting was made. In contrast to current literature, this is the first stent thrombosis case described after hymenoptera envenomation. KS is defined as a group of acute coronary syndromes that manifests as unstable vasospastic or nonvasospastic angina, and even as acute myocardial infarction (MI) triggered by the release of inflammatory mediators following an allergic insult. It was first described in 1991 as coronary vasospasm and MI secondary to allergic reactions, and is also known as allergic MI. Several etiologic factors have been reported including drugs, bees, ants, jellyfish and poison ivy. The most frequent symptoms are retrosternal chest pain, dyspnoea, palpitations, weakness, nausea, urticaria, itchiness, sweating, and hypotonia. Two types of this syndrome have been defined. In type 1, the coronary arteries are normal and coronary vasospasm secondary to hypersensitivity reaction is the responsible mechanism. The clinical presentation is usually angina, but if the response is prolonged, progression to myocardial necrosis and infarction may occur. The basic hypothesis is that type 1 KS is a manifestation of endothelial dysfunction. In type 2, there is underlying coronary artery disease and hypersensitivity reaction leads to plaque erosion and coronary occlusion. The inflammatory response is essentially mast cell driven, which are the key elements of allergic reactions. Mast cells are located between cardiomyocytes and around coronary arteries in healthy people. As a result of mast cell activation, several vasoconstricting and collagen degrading compounds including histamine, platelet activating factor, and neutral proteases release. The latter activate metalloproteinases in atheromatous plaques and play an important role in their erosion and rupture. Prognosis of patients with type 1 is better than type 2 cases. Prognosis depends on the magnitude of the allergic reaction, patient's sensitivity, comorbidities, allergen concentration, and the allergen's portal of entrance to the body.

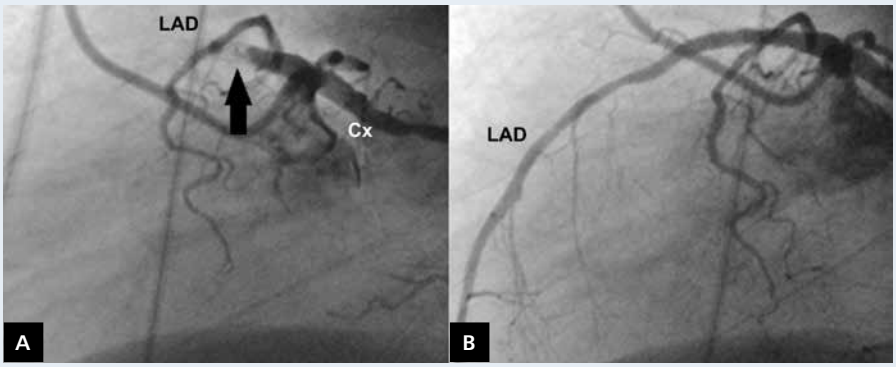

Figure 1. Lateral view of left coronary system; **A**. Black arrow shows thrombus in left anterior descending artery (LAD) stent; Cx — circumflex artery; **B**. Lateral view after balloon inflation; TIMI-3 coronary flow was achieved

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Conflict of interest: *none declared*