STUDIUM PRZYPADKU / *CLINICAL VIGNETTE*

Delayed therapy for Wellens' syndrome resulted in acute myocardial infarction

Zawał serca w wyniku opóźnionego rozpoznania i leczenia zespołu Wellensa

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A 45 year-old man was admitted to the Internal Medicine Clinic with complaints of postprandial epigastric pain and concomitant sweating. He had no cardiovascular disease or risk factor except smoking for 30 years (1.5 packs/day). His family history was negative for premature coronary artery disease (CAD). Physical examination was unremarkable. Biochemistry panel including lipid profile and cardiac panel was within normal reference limits. Electrocardiography (ECG) revealed sinus rhythm (SR, 75 bpm), and symmetrical and deeply inverted T waves in leads V_2 to V_6 (Fig. 1A). He was diagnosed with gastritis, and proton pump inhibitor (PPI) (lansoprazol 30 mg/day) was initiated. Also coronary computerised tomography angiography (CTA) was performed to rule out CAD. After 48 hours, the patient developed sudden onset epigastric pain which was radiating to the retrosternal area just after a football match. He was admitted to the emergency department and ECG on admission showed SR of 75 bpm, sharpened T waves and ST segment elevation in leads V_1–V_5 indicative of anterior wall myocardial infarction (MI) (Fig. 1B). He was immediately transferred to the cath lab. Coronary angiography revealed totally occluded left anterior descending artery (LAD) (Fig. 2A). Balloon angioplasty and bare metal stent implantation were performed with successful angiographic and clinical results (Fig. 2B). The patient was hospitalised in the coronary care unit and the remainder of his hospital stay was uneventful. Retrospective analysis of the coronary CTA resulted in critical stenosis in the proximal segment of the LAD (Fig. 3). So, the patient was diagnosed with Wellens' syndrome (WS) which had progressed with exercise and resulted in acute MI. Among the several ECG changes indicative of acute coronary syndromes, all physicians must be familiar with the characteristics of the preinfarction stage of CAD known as WS. WS consists of T-wave changes in the precordial leads. These changes are associated with significant stenosis in the proximal segment of LAD. Patients with WS are at high risk for the development of an extensive MI of the anterior wall, as in our case. Due to this high risk, patients require immediate coronary angiography and revascularisation strategies. Two main patterns of T-wave abnormalities are indicative of WS. In 75% of patients, the T waves in the precordial leads ($V_{1–4}$) are symmetrical and deeply inverted. In our case, coronary CTA confirmed the diagnosis of WS. But the patient developed acute MI with exercise before the CTA result. In conclusion, patients with WS have an increased risk for anterior MI. Exercise stress tests should be avoided, and early invasive evaluation should be planned for such patients. Recognition of WS in patients with angina and equivalents by physicians is therefore important.

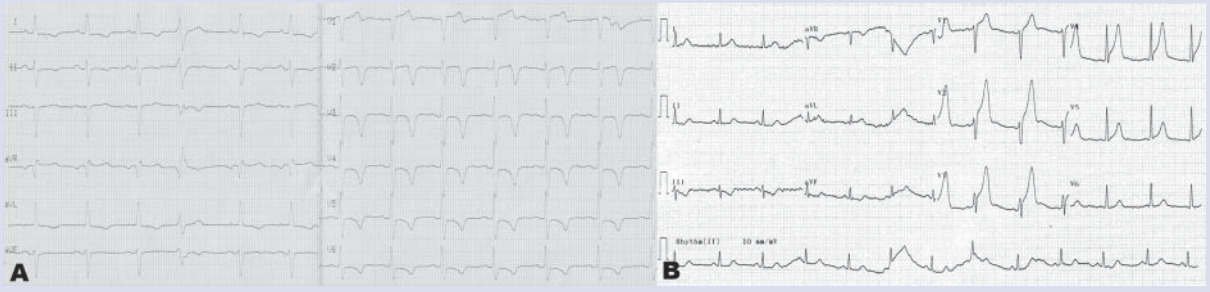

Figure 1. A. Initial ECG showing sinus rhythm with symmetrical and deeply inverted T waves in leads V_2 to V_6; **B.** ECG in emergency room showing sinus rhythm with sharpened T waves and ST segment elevation in leads V_1–V_5

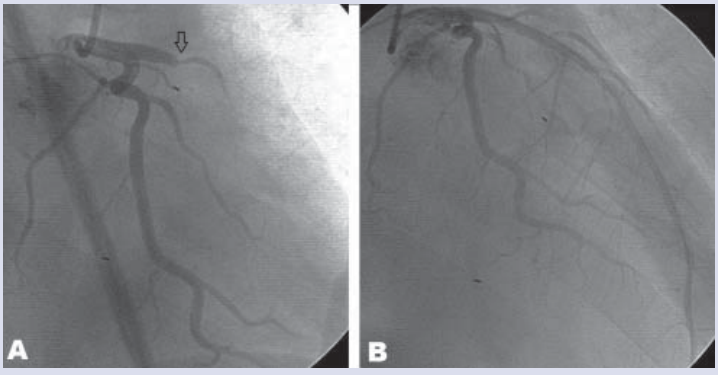

Figure 2. A. Coronary angiogram showing totally occluded LAD artery; **B.** After balloon angioplasty and stenting, TIMI 3 flow with successful angiographic result in LAD artery

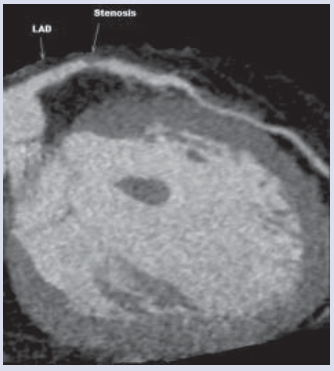

Figure 3. Coronary CTA revealed critical stenosis in proximal segment of the LAD artery

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Conflict of interest: *none declared*