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Sticky platelet syndrome as a cause of acute myocardial infarction in a young soccer player

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Introduction

Acute myocardial infarction (AIM) is still a rare condition in young athletes, even though its incidence is slightly increasing. We present the case of a 25-year-old professional soccer player, who was initially resuscitated due to ventricular fibrillation, subsequently with a finding of massive thrombotic occlusion of left anterior descending artery. Sticky platelet syndrome (SPS) was considered as the most probable cause of this state. The pathophysiology of acute myocardial infarction in the presence of "normal" coronary arteries remains unclear, but can be explained on the basis of their thrombosis, embolization, spasm, or a combination of these processes. Coronary thrombosis can be seen in hypercoagulable conditions such as nephrotic syndrome, antiphospholipid syndrome, protein S or factor XII deficiency and, of course, SPS.

Patient presentation

The 25-year-old active football player was initially admitted to our workplace about an hour after he collapsed during the match and had to be resuscitated on the field itself. From the information of the emergency medical service crew, the ventricular fibrillation was evaluated as the primary cause of the collapse–the patient was initially given a total of 5 defibrillation shocks, and after nine minutes of basic and later advanced cardiopulmonary resuscitation, spontaneous circulation returned and consciousness was restored.

Initial work up

Based on the electrocardiographic examination with the finding of three millimeter ST segment elevations in leads I, aVL, V3-V5, and also contralateral ST depressions in leads II, III, aVF, a diagnosis of acute STEMI myocardial infarction (AMI) of the anterior wall was established. The patient was intubated and connected to artificial lung ventilation by the EMS doctor on the playing field. Values of blood pressure (155/60 mmHg) and heart rate (80/min.) were normal. During transport, 5000 units of unfractionated heparin were administered intravenously. Upon arrival at our workplace, the patient was referred to the intervention room for the purpose of performing an emergent coronary angiography examination, during which a single-vessel coronary involvement with the nature of a massive thrombotic occlusion of the proximal part of the left anterior descending artery (LAD) was documented, TIMI 2 (**Figure 1**). Thromboaspiration was performed ad hoc, 1x DES stent implanted, glycoprotein IIb/IIIa inhibitor (eptifibatide) administered to the periphery, and dual antiplatelet therapy (DAPT) (acetylsalicylic acid (ASA) + ticagrelor) to the probe.

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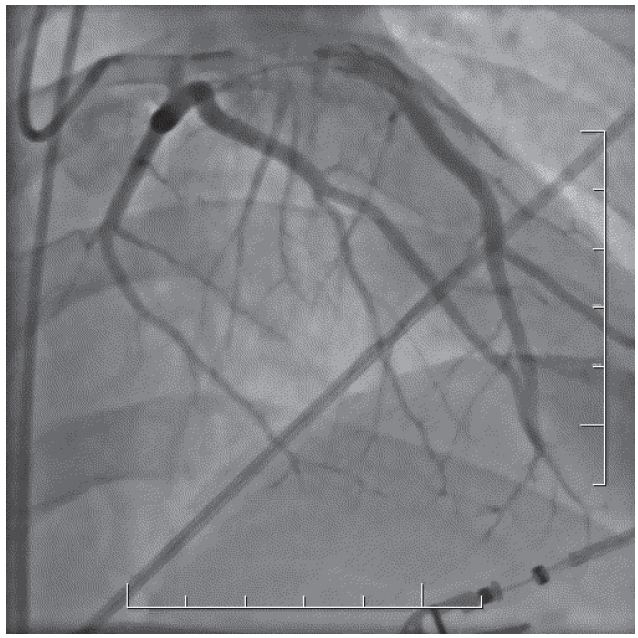

Figure 1 A massive thrombotic occlusion of the proximal part of the LAD

After the end of the procedure, a fully restored flow through the coronary artery was recorded (**Figure 2**). In the laboratory, elevation of cardiospecific enzymes dominated – hs Troponin I 95124.0 ng/l (0.0 – 60.4), CK 87.92 ukat/l (0.12 – 5.11) and CK-MB 277.41 ug/l (0.00 – 5.00), with other findings in the norm. Echocardiographically (immediately after the procedure) documented extensive disturbances of kinetics – akinesis of the tip, the apical third to half of the interventricular septum, thirds of the front and anterolateral wall, the best kinetics were in the area of the inferior wall and inferolaterally. Left ventricular ejection fraction (LVEF) was in the range of 35 to 40%, and skiagram of the chest without significant pathology.

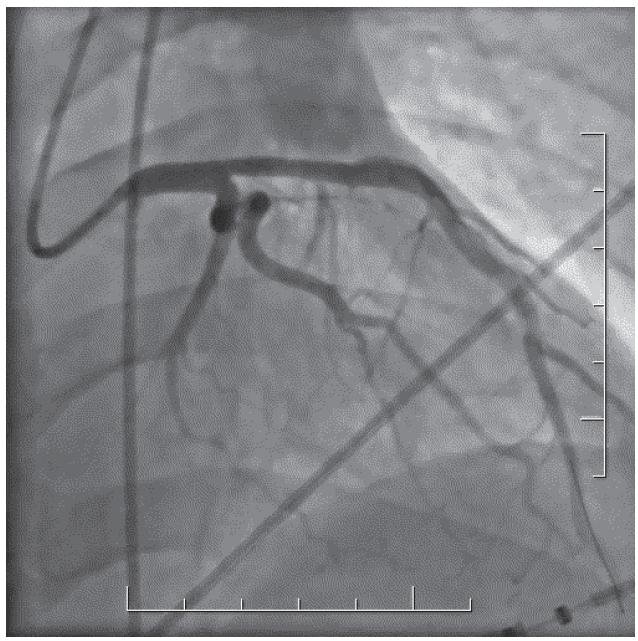

Figure 2 Final result of the PCI procedure

Diagnosis and management

Eight hours after the procedure, the patient was sedated, extubated, breathing spontaneously, with sufficient respiration, conscious, GCS 15, and without subjective complaints. Laboratory parameters remained satisfactory, and diuresis sufficient. By supplementing the family and personal anamnesis, we learnt from the patient that his mother was in the hematologist's dispensary for a thrombophilic condition, and he had not been examined from this point of view until then. Until then, without chronic medication, at that time he was vaccinated twice against COVID-19 disease with the Comirnaty vaccine. He avoids abuse, as well as steroids and other supplements. The rest of the prehospitalization course was without complications, the patient rehabilitated, subsequently in a stabilized condition, after ten days was released to the outpatient care of the relevant specialists. As part of chronic medication he was prescribed DAPT (ASA + prasugrel), then bisoprolol, atorvastatin, pantoprazole, and eplerenone.

Follow-up

Subsequently, at intervals of 6 and 12 months after discharge, the patient underwent a control complex cardiology examination with us, including an echocardiographic examination, where we recorded an increase in LVEF to the level of 50%. In the given period, the patient was still without subjective difficulties, without regular training load, and tolerated normal physical activity without difficulties. Due to a positive family history of thrombophilic status, the patient underwent a comprehensive examination by a hematologist after

completing 12 months of DAPT, which confirmed the diagnosis of sticky platelet syndrome. As part of chronic medication, the patient was left on ASA monotherapy (for both cardiology and hematological indications). A total of 18 months after the event in question, the patient gradually returned to training. He currently undergoes regular cardiological and hematological checks, is free of any subjective problems, and tolerates physical activity without restrictions.

Conclusions

SPS can be defined as a qualitative change in platelet function with autosomal dominant inheritance and familial occurrence, characterized by hyperaggregation of platelets with simultaneously reduced concentrations of adenosine diphosphate (ADP) and epinephrine (ADR) (type I) or only ADR (type II) or only ADP (type III). The most common is SPS II. type, the least frequent type III. Currently, no specific epidemiologic data on SPS are available. The most common clinical picture of the disease is arterial thrombosis. The diagnosis of SPS is based on the examination of platelet aggregation after its activation by ADP and ADR. Platelet aggregation is usually investigated turbidimetrically, where the change in light transmission in the examined sample is monitored depending on the percentage of aggregated platelets over time, using three subthreshold concentrations of each of the inducers. Currently, no specific recommendations for the treatment of SPS are available, but at the same time, it is based on the results of observations that ASA in low doses can normalize the nature of platelet aggregation. For this reason, it is recommended to start treatment at a dose of 80 to 100 mg per day. In patients who do not achieve an adequate response, it is recommended to increase the dose to 325 mg daily. As far as we know, this is the first documented case of a young athlete with an overcome AMI based on SPS. AMI is rare in adolescents and young adults. Patients can be divided into two groups: those with normal coronary angiographic findings and those with ischemic heart disease of various etiologies. The pathophysiology of AMI in the presence of "normal" coronary arteries remains unclear, but can be explained on the basis of their thrombosis, embolization, spasm, or a combination of these processes. Coronary thrombosis can be observed in hypercoagulable states such as nephrotic syndrome, antiphospholipid syndrome, protein S or factor XII deficiency and, of course, SPS.

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