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Left main coronary artery thrombosis in a 19 year-old patient

Zakrzepica pnia lewej tętnicy wieńcowej u 19-letniego pacjenta

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Abstract

Left main coronary artery occlusion is a very rare entity that often results in death. It usually manifests as acute myocardial infarction (MI) with cardiogenic shock and fatal arrhythmias. Here, we report the case of a 19 year-old patient who presented with acute anterior MI secondary to left main coronary artery thrombosis. There were no classical risk factors for coronary heart disease in the anamnesis of the patient. Leukaemia was regarded to be the most probable predisposing condition in the patient.

Key words: young, myocardial infarction, bypass grafting, leukaemia

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INTRODUCTION

Left main coronary artery (LMCA) occlusion is a very rare entity often resulting in death. It usually manifests as acute myocardial infarction (MI) with cardiogenic shock and fatal arrhythmias [1]. We report the case of a 19 year-old patient who presented with acute anterior MI secondary to LMCA thrombosis.

CASE REPORT

A 19 year-old man free of classical risk factors was referred to our hospital complaining of chest pain of three days' duration and progressive dyspnea over the previous 24 hours. Electrocardiography showed ST segment elevation in anterior and lateral leads with a right bundle branch block (Fig. 1). Blood pressure was 60/40 mm Hg, heart rate was 150 bpm. On physical examination, there was a bilateral diffuse crackle which extended to the upper segments of the lung. Oxygen saturation was 85%. The patient was classified clinically as Killip 4 and hospitalised with a diagnosis of cardiogenic shock and anterior MI. Emergency echocardiography ruled out mechanical complications, and showed severe left ventricular dysfunction with akinesis of anterior, septal, lateral and apical segments. Immediate coronary angiography revealed a normal right coronary artery and total thrombotic occlusion of the LMCA. There was no anterograde flow in the left anterior descending (LAD) artery, and weak distal TIMI 0–1 flow in the circumflex artery (CX) (Fig. 2). Although angioplasty was chosen as the treatment, a 0.014 inch guidewire could not be advanced to the LAD osteum. The patient underwent emergency coronary bypass grafting. Massive thrombi were obtained throughout all parts of the LAD and the CX during anastomosis of coronary grafts. Two vein grafts were anastomosed to the LAD and the obtuse margin branch of the CX. Post-operatively, an intra-aortic balloon pump was inserted and the patient was treated with inotropic agents including levosimendan. Approximately 3 L of fluid were aspirated from the lung. Laboratory tests showed a mar-

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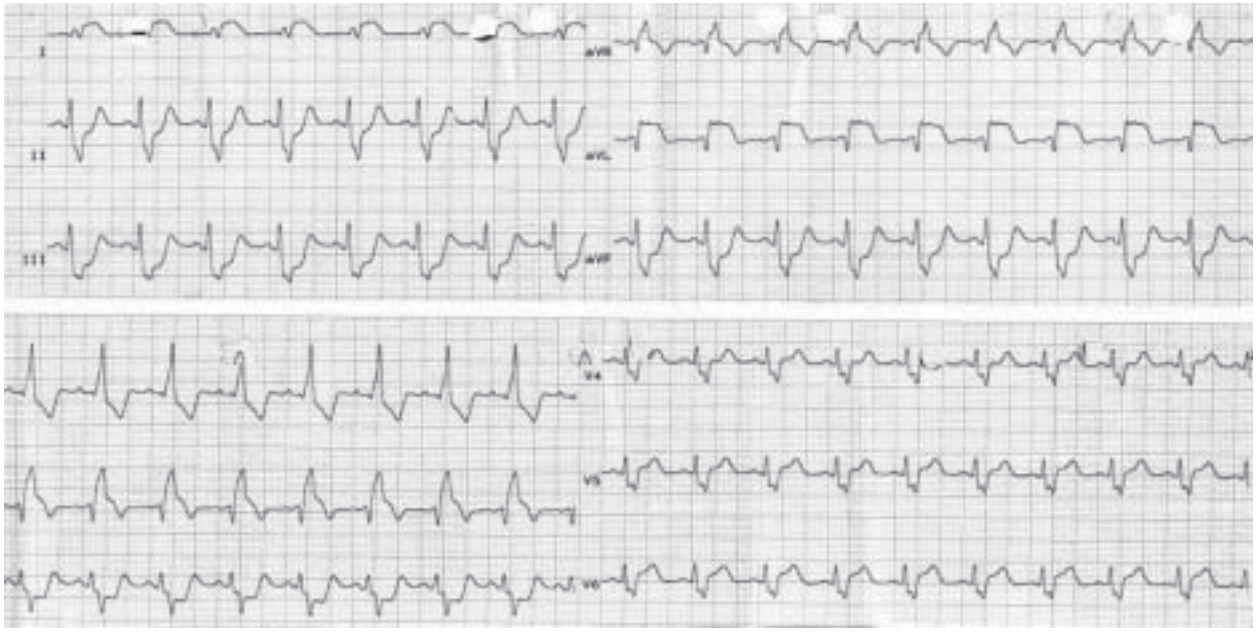

Figure 1. ST segment elevation in anterior and lateral leads with a right bundle branch block

ked elevation of cardiac markers (CK-MB > 400 ng/mL, troponin I > 80 ng/mL and troponin T > 2). In spite of intensive medical treatment, the patient died six hours after the operation due to left ventricular systolic dysfunction.

DISCUSSION

Left main coronary artery occlusion is seen rarely. Even with appropriate treatment, the mortality rate is high [2]. There are as yet no clear-cut guidelines describing the best approach to managing such patients [3]. Intracoronary thrombolytic infusion, angioplasty and stenting and coronary bypass grafting are the usual treatments [4]. Left main coronary artery occlusion is extremely rare before the age of 20. The only previously reported case was aged 14 [5] He was treated with primary stenting, but in the following course, the patient underwent cardiac transplantation due to left ventricular dysfunction.

If we focus on the medical history of our case: he had described anaemia history for a year. But no further investigation had been carried out. Complete blood count showed mild anaemia with haemoglobin of 10.9 g/dL. Haematological disorders with abnormal platelet dysfunction can lead to MI in the young. In our case, platelet count was 263/nL, aPTT was 35 s and prothrombin time was 20.8 s. Although there was no evidence for platelet dysfunction in our case, white blood cell count was found to be 42.2/nL, with granulocyte predominance.

Our case was most probably one of leukaemia, which can be a major triggering factor for LMCA thrombosis. A tendency to hypercoagulability is commonly seen in leukaemic patients. Besides thrombophilia and mass effect of leukocytes, decreased levels of proteins C and S, decreased antithrombin 3 activity and increased levels of homocysteine have beeen reported in leukaemic patients. All these conditions contribute to the formation of a hypercoagulable state in leukaemia [6–8]. There have been reported cases of MI associa-

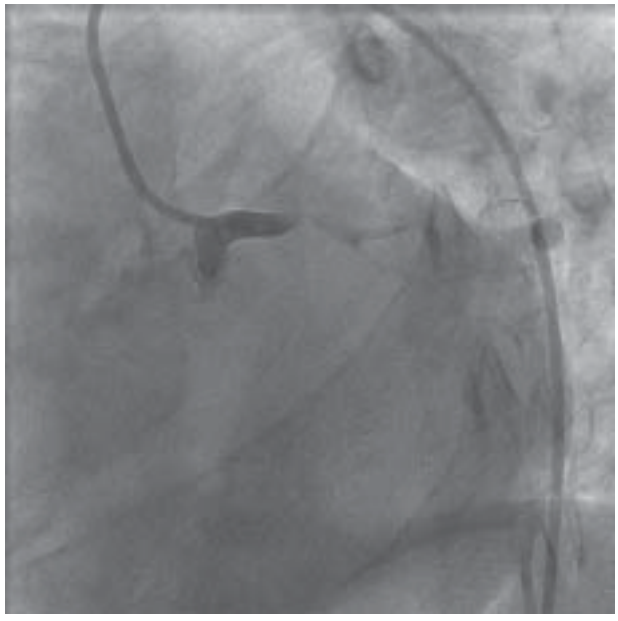

Figure 2. Left anterior oblique caudal projection (spider view) showing total occlusion of left main coronary artery and image of thrombus

ted especially with myeloid type leukaemia [9–11]. But all reported cases have been in adults. There has been no reported case of LMCA thrombosis associated with leukaemia in a young person.

Because of the early death of the patient, further pathological investigation for an exact diagnosis of leukaemia could not be carried out. However, anamnesis, the clinical aspects of the patient, and laboratory findings all support a probable diagnosis of leukaemia.

CONCLUSIONS

Left main coronary artery thrombosis and associated MI before the age of 20 is extremely rare. Haematological disorders, such as leukaemia, thrombocytemia, sickle cell anaemia and polycytemia can present with MI in young people, and should be considered in a differential diagnosis.

Conflict of interest: *none declared*

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