Case Reports

Myocardial Infarction in a Young Man With Nephrotic Syndrome

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Summary

A 26-year-old man diagnosed with nephrotic syndrome (NS) 2 years previously presented with chest pain. An electrocardiogram (ECG) performed at a local hospital showed ST-elevation in chest leads. Cardiac troponin-I was significantly positive. Echocardiography revealed mild regional wall-motion abnormalities in the heart apex. Seven days later, angiography (CAG) revealed a thrombus in the left anterior descending branch (LAD). Tirofiban was injected into the LAD for thromboclasis. ECG after CAG showed the ST-segment was much lower than before. The diagnosis after CAG was ST-segment elevation myocardial infarction (MI) and thrombogenesis in the LAD. He continued to receive antiplatelet and anticoagulation medication and atorvastatin after CAG, and was discharged 3 days later. MI is very rare in young males, but the incidence of MI is 8 times higher than normal in patients with NS. For young patients with MI, clinicians should pay more attention to the history of previous diseases with high risk of thromboembolism and they should actively promote prevention and the treatment of renal disease patients to reduce the incidence of complications of thromboembolism. (Int Heart J 2017; 58: 275-278)

Key words: Acute coronary events, Hypercoagulation

Myocardial infarction (MI) is very rare in young males. Unlike older patients, in whom the rupture of an atherosclerotic plaque causing occlusion of the coronary artery is the main underlying pathology, the pathogenesis in younger patients can be varied, one of which is thromboembolism caused by nephrotic syndrome. We present here a case of MI in a young male.

Case Report

A 26-year-old man presented to the emergency department with chest pain lasting 17 hours, preceded by cough for 7 days. He was diagnosed with nephrotic syndrome (NS) 2 years previously at Xiangya Second Hospital, and was being treated with prednisone to which he responded for about 12 weeks without any subsequent follow-up. He refused a kidney pathological examination at that time. There was no history of diabetes mellitus, hypertension, hepatitis B, malignant tumor, allergic purpura, systemic lupus erythematosus, connective tissue disease, or smoking. Before being transferred to our hospital, he was diagnosed with acute anterior myocardial infarction and an electrocardiogram (ECG) (Figure 1) at a local hospital showed ST-elevation in the chest leads. At the time of presentation, his blood pressure was 128/70 mmHg, his pulse rate 105 beats per minute, and his body temperature 36.7°C. Heart, lung, and abdominal examinations were negative. An ECG showed ST-elevation in the chest leads (Figure 2). Cardiac troponin-I (cTn-I) and MB isoenzyme of creatine kinase (CK-MB) were significantly positive. The provisional diagnosis was ST-segment elevation myocardial infarction (STEMI), with possible severe myocarditis. He refused primary percutaneous coronary angiography (CAG) because his chest pain relieved after he arrived at our hospital. Bedside echocardiography revealed mild regional wall-motion abnormalities in the heart apex, and the left ventricular ejection fraction was 63%. A computed tomography (CT) scan of the chest showed little pleural effusion and cervical pleural adhesion. An ECG obtained after admission showed the ST-segment of chest leads was lower than before (Figure 3). The levels of cTn-I at admission and at 12, 24, and 48 hours after admission were 21.858, 40.714, 46.316, and 15.593 ng/mL, respectively (The 99th percentile upper reference limit in our laboratory was 0.100 ng/mL). The levels of CK-MB at the corresponding time points were 63.80, 90.68, 41.66, and 3.56 ng/mL, respectively (The reference values in our laboratory was 0.30-4.00 ng/mL). The urine test at the time of admission demonstrated proteinuria, for protein dipstick grading 3+ (about 300 mg/dL), with other parameters such as erythrocytes and leukocytes all negative, and 24-hour urinary protein excretion was 2.16 g. Cholesterol and low-density lipoprotein were 7.05 mmol/L and 4.49 mmol/L, respectively. Seven days after presentation, the CAG showed thrombi in the left anterior descending branch (LAD), and most thrombi were in distal LAD (Figure 4). Tirofiban was injected into the LAD for thrombosis during the CAG procedure. An ECG obtained after CAG showed the ST-segment...
Figure 1. ECG at local hospital showed ST-elevation in chest leads.

Figure 2. ECG at emergency department showed ST-elevation in chest leads.
of chest leads was much lower than before and a negative T wave (Figure 5). The diagnosis after CAG was STEMI, thrombogenesis in LAD, nephrotic syndrome, hyperlipidemia, infection of the upper respiratory tract, and pleurisy. The postoperative course was uneventful. The patient received antiplatelet therapy, anticoagulation, and atorvastatin (20 mg/day), and he was discharged 3 days later.

**Figure 3.** ECG after admission showed ST-segment of chest leads lower than before.

**Figure 4.** White arrow showing a small thrombus in proximal left anterior descending branch (LAD), and black arrow showing most thrombi in distal LAD.

**Figure 5.** ECG after CAG showing the ST-segment of chest leads much lower than before and negative T wave.
**Discussion**

Myocardial infarction (MI) is very rare in young males. Although thromboembolic complications are seen in 35% of patients with NS, arterial thromboses associated with NS are much less common, being observed in only 1.8-5%. When the disease affects the vital organs, the prognosis of the patient is seriously affected. Thromboses are extremely rarely observed. NS patients frequently have hyperlipidemia and hypercoagulability and the incidence of MI is 8 times higher than normal in patients with NS. Our case suggests NS as a possible cause of thrombotic occlusion of the coronary arteries, causing MI in this young man. It is interesting that the duration between the nephrotic syndrome findings and onset of myocardial infarction was 2 years in this case. The causes of myocardial infarction in this patient include: 1) Long-term continuation of abnormal blood lipids that can lead to atherosclerosis, including of the coronary arteries. 2) Blood coagulation factor changes, including decreases in factors IX and XI, increases in factors V, VIII, and X, increased fibrinogen and platelet levels, increased adhesion and aggregation of platelets, and decreased activity of antithrombin-III and antiplasmin, finally leading to a hypercoagulative state. 3) Long-term hormone therapy can promote and aggravate thrombosis. 4) The high level of inflammatory factors in patients with NS, leading to unstable atherosclerotic plaque rupture and thrombosis. 5) ST-segment elevation had continued even 7 days after onset of myocardial infarction with chest pain relief, and this phenomenon may be due to the thrombus slowly dissolving itself in hypercoagulable states. In most reported cases of thromboembolic events in NS, treatment with high-dose heparin with or without thrombolytic agents, has been found to be effective in resolution of thrombus. So for these patients, long-term anticoagulant therapy is particularly important.

In this case, the patient did not receive a good education, his family’s economic situation was poor, and he did not pay sufficient attention or adequately understand the disease. He and his family refused a kidney pathological examination 2 years prior, and the most likely etiology might be IgA nephropathy (IgAN). IgAN is the most common glomerulonephritis worldwide and in China. In addition, IgAN occurs more easily in young male adults. This 26-year-old patient with NS did not continue proper treatment, which ultimately led to the MI. For young patients with ECG findings of MI, clinicians should pay greater attention and not simply consider myocarditis, but also should consider MI and carefully investigate any previous medical history with a high risk of thromboembolism. Young patients can have acute coronary events in the absence of atherosclerotic disease. In such a setting, the possibility of underlying NS should be considered. We recommend that all young patients with acute coronary events without evidence of definitive atherosclerotic disease should have their urine tested for proteinuria. Furthermore, clinicians should actively promote the prevention and treatment of renal disease in order to reduce the incidence of the complications of thromboembolism.

**References**