Case Reports

Two Cases of Acute Myocardial Infarction Associated with Aplastic Anemia during Treatment with Anabolic Steroids

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SUMMARY

Thrombosis is a rare complication in patients with aplastic anemia because of the presence of coincidental thrombocytopenia. We have recently treated two cases, a 61-year-old male and a 59-year-old female, with acute myocardial infarction associated with aplastic anemia. Although their platelet counts were lower than normal in spite of treatment with anabolic steroids for aplastic anemia, the coronary angiographic findings strongly suggested coronary thrombosis in both cases. Anabolic steroids, which have been commonly used for the treatment of aplastic anemia, are a possible risk factor for coronary thrombosis because they have an accelerating effect on thrombus formation. We report two very rare but clinically important cases. (*Jpn Heart J* 35: 369–373, 1994)

Key words: Aplastic anemia Coronary thrombosis Myocardial infarction Anabolic steroid

APLASTIC anemia is characterized by bone marrow hypoplasia and peripheral pancytopenia; anabolic steroids are used widely for its treatment. Clinically, patients with this disease usually exhibit a bleeding tendency, as well as fatigability and palpitations. Thrombosis is rarely observed due to the very low platelet count in the peripheral blood. In fact, there are few reports of thrombosis in association with aplastic anemia, and in particular, as far as we know, only one case of acute myocardial infarction has been reported to date.¹⁾ Moreover, coronary thrombosis has never been reported in this setting.

We have recently treated 2 cases of acute myocardial infarction associated

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with aplastic anemia. In both cases coronary arteriograms strongly suggested the existence of coronary thrombosis. As far as we know, these are the first cases of acute myocardial infarction due to coronary thrombosis associated with aplastic anemia to be reported.

CASE REPORT

Case 1 was a 61-year-old male with no individual or family history of heart disease. He had no coronary risk factors, including diabetes or hypertension. His total cholesterol level was 162 mg/dL. Moreover he was not obese (163.6 cm in height, 55 kg in weight), and he had never smoked. In September 1989 he complained of gingival bleeding and visited a nearby clinic. Pancytopenia was noted (white blood cell 2,100/mm³, hemoglobin 8.7 g/dL, platelets 6,000/mm³). As a result of detailed examinations, aplastic anemia was diagnosed. At the time when the administration of metenolone enanthate (45 mg), an anabolic steroid, was initiated (October 13), the patient's platelet count was 3,000/mm³. During the early stages following the administration of anabolic steroid, the platelet count remained at a relatively constant level after an initial rise, ranging between 10,000–20,000/mm³, it then started to increase gradually and reached 30,000/mm³ on November 8, and 37,000/mm³ on November 22, 1989.

On the morning of December 29, epigastric pain occurred suddenly and the patient visited our hospital. The platelet count at the time of the patient's hospital visit was 40,000/mm³. Elevations of ST segments were observed in leads II, III and aVf on his electrocardiogram (ECG). Acute myocardial infarction of the inferior wall was diagnosed from the ECG and echocardiographic findings. The peak value of serum creatinine phosphokinase (CPK) was 4,794 IU/L on the evening of the second hospital day. Cardiac catheterization was performed on the 48th day. No atherosclerotic lesions were observed in either coronary artery, but multiple filling defects were revealed in segments 1–3 of the right coronary artery, and the distal ends of these regions were visualized with severe delays. These filling defects strongly suggested the presence of thrombi (Figure 1).

Case 2 was a 59-year-old female. She had no coronary risk factors except for steroid-induced glucose intolerance. Her total cholesterol level was 154 mg/dL, and her blood pressure was 110/60 mmHg. She was not obese (150 cm in height, 53 kg in weight). She was not a smoker and had no significant family history. In August 1977, she visited the outpatient clinic complaining of easy fatigability and palpitations. Severe anemia was observed and the patient was admitted. Aplastic anemia was diagnosed as a result of detailed examinations. Treatment with metenolone enanthate (100 mg) was initiated in September. Treatment was continued using oxymetholone (30 mg), another anabolic steroid, in 1982. During

Figure 1. Right coronary angiogram (case 1) showing multiple filling defects in segments 1-3. Distal region was visualized with severe delay. These findings indicate thrombus formation.

Figure 2. Right coronary angiogram (case 2) showing thrombotic defects at segment 2 and occlusion at the distal portion.

the courses of treatment the patient's platelet count ranged between 10,000–30,000/mm³. Myocardial infarction occurred in November 1990 and again in February 1991. On each occasion, the patient was hospitalized for treatment. At the time of the first myocardial infarction, her platelet count was 10,000/mm³. ST elevations were observed in leads II, III and aVf on her ECG, and acute myocardial infarction of the inferior wall was diagnosed. The peak value of serum CPK was 1,802 IU/l. She was re-admitted in February 1991 due to the occurrence of a second myocardial infarction. The platelet count at that time was

14,000/mm³. Relapse infarction of the inferior wall was diagnosed based on ECG findings. The peak value of serum CPK was 707 IU/l. Cardiac catheterization was performed on the 24th hospital day. Left ventriculography revealed akinetic motion of the posterobasal wall (segment 5). Coronary arteriography showed no sclerotic lesions in the left coronary artery. Thrombotic filling defects were observed at segment 2 of the right coronary artery, and a complete occlusion was seen beyond these lesions (Figure 2).

There were no coronary risk factors in either case except for glucose intolerance in case 2. Both patients were receiving treatment with anabolic steroids for aplastic anemia. The platelet counts in the peripheral blood at the onset of acute myocardial infarction were far less than normal in both cases.

DISCUSSION

Aplastic anemia, in itself, is not considered to be a risk factor for atherosclerosis or ischemic heart disease. Indeed, there are few reports of thrombosis or acute myocardial infarction associated with aplastic anemia. The use of anabolic steroids as a treatment for this disease might however make the situation different. Firstly, anabolic steroids reportedly increase the serum LDL-cholesterol level and decrease the HDL-cholesterol level,²⁾ and these effects accelerate arteriosclerosis. Secondly, anabolic steroids are reported to act on the arachidonic acid cascade in the vascular wall to accelerate thromboxane A₂ synthesis and suppress prostacyclin production; these effects result in acceleration of platelet aggregation.³⁾ Thirdly, juvenile platelets released in response to treatment with anabolic steroids have strong aggregation activity, and enhance the production of coagulation factors. In this manner, anabolic steroids may cause atherosclerosis and thrombosis. In fact, 3 cases of acute myocardial infarction in healthy athletes during anabolic steroid use have been reported thus far, with normal coronary angiographic findings in one,⁴⁾ arteriosclerosis in another,⁵⁾ and thrombi in the coronary artery in the third.⁶⁾ In our 2 cases, neither abnormalities in lipid metabolism nor atherosclerotic findings in the coronary arteries were seen. The multiple filling defects seen in the coronary angiograms strongly suggested that the cause of acute myocardial infarction in these patients was thrombi in the coronary arteries. Moreover, in case 1, the onset of acute myocardial infarction coincided with a rise in platelet count.

We conclude that patients with aplastic anemia receiving anabolic steroid, even if their platelet counts are low, are at risk of thrombosis, and acute myocardial infarction, particularly when the platelet count is on the rise.

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