Journal of Clinical and Basic Cardiology

An Independent International Scientific Journal

Journal of Clinical and Basic Cardiology 2001; 4 (1), 83-84

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Indexed in
Chemical Abstracts
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Pulmonary Infiltrates Following Coronary Artery Stenting and Abciximab Therapy in a 64 Year Old Woman

J. Auer, R. Berent, C. Punzengruber, B. Eber

A 64 year old woman with no history of lung disease presented with unstable angina. Coronary angiography revealed single vessel disease with severe stenosis of the left anterior descending coronary artery (LAD). Therapy with abciximab was started during coronary artery stenting because of intraprocedural acute stent thrombosis. The final result of the procedure was excellent with no residual stenosis. Ten hours after stent implantation the patient developed shortness of breath, cough and chills. The platelet count fell from normal to a minimum of 9 G/l. Chest radiograph revealed a patchy infiltrate of the right lung. Diuretic drugs failed to be efficacious to improve symptoms. High dose corticosteroids and oxygen supply were initiated immediately. Within a few hours pulmonary symptoms as well as infiltrates on chest radiogram resolved without sequelae. Within the same time period platelet count increased and was documented to be within normal range two days later.

Thrombocytopenia has been demonstrated with abciximab use and is likely related to immunologic mechanisms and the production of antimurine antibodies. These immunologic mechanisms could cause derangement of alveolar capillary permeability and result in acute lung injury and pulmonary oedema. To our knowledge, this is the first report of simultaneous occurrence of pulmonary lesions and abciximab-induced thrombocytopenia that are with high probability linked by immunologic mechanisms. Acute lung injury should be considered as a rare adverse event with abciximab use. *J Clin Basic Cardiol 2001; 4: 83–84.*

Key words: abciximab, acute lung injury, thrombocytopenia, immunology, stent

A 64 year old woman with no history of lung disease (baseline chest radiograph is shown in Figure 1), who had a 20–30 pack year smoking history, was admitted to hospital because of unstable angina. The patient had no history of coronary artery disease and reported chest pain at rest and with minor exercise during the last week before admission.

On physical examination she was overweight (body mass index 31 kg/m²), the pulse was regular with a rate of 66 beats per minute. Blood pressure measurement revealed 135/80 mmHg. There was no heart murmur and no signs of congestive heart failure.

Laboratory examination showed a white blood cell count of 9.2 G/l, total cholesterol was 234 mg/dl with LDL cholesterol of 146 mg/dl. Creatine-phosphokinase (CPK) and troponin I were normal. Red blood cell count, platelet count, LDH, GOT, GPT, gamma-GT, alkaline phosphatase, creatinine, blood urea nitrogen, CRP, total protein, triglycerides and electrolytes were within normal range.

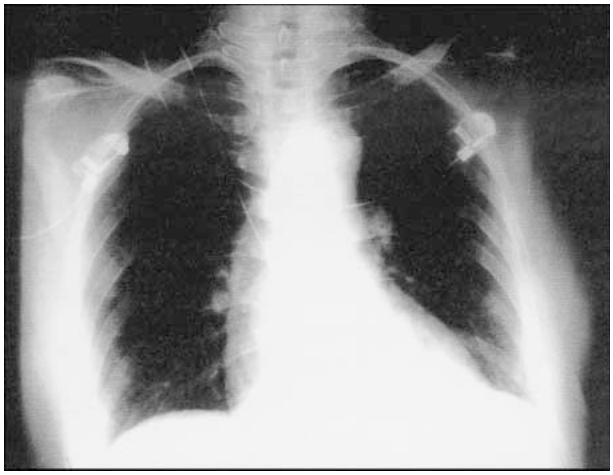

Figure 1. Posterior-anterior chest radiograph at admission revealed abnormalities

Electrocardiography revealed regular sinus rhythm with no remarkable abnormalities on repolarisation.

Echocardiography showed preserved left ventricular systolic function and normal heart valves. Chest radiograph performed on admission revealed no abnormalities (shown in Figure 1).

Coronary angiography demonstrated single vessel disease with severe stenosis of the left anterior descending coronary artery (LAD).

Clinical Course and Further Treatment

Therapy with abciximab (bolus and continuous infusion) was started after coronary stenting because of intraprocedural acute stent thrombosis. The patient received 10,000 IU of unfractionated heparin during coronary procedure, further medication consisted of isosorbid-mononitrate, metoprolol, and acetylsalicytic acid.

The final result of the procedure was excellent with no residual stenosis. TIMI 3 flow had been achieved within about 20 minutes after first balloon insufflation. 10 hours after the procedure the patient developed shortness of breath, cough and chills. The platelet count fell from normal (187 G/l) to a minimum of 9 G/l, red cell count and other laboratory data were within normal range on repeated laboratory examinations. Now, a posterior-anterior chest radiograph (Figure 2) revealed a patchy infiltrate of the right lung. Furosemid (80 mg intravenously) failed to be efficacious to improve symptoms. High dose corticosteroids (250 mg hydrocortisone) and oxygen supply were started. Within a few hours pulmonary symptoms as well as infiltrates on chest radiograph resolved without sequelae. Within the same time period platelet count increased and was documented to be within normal range two days later.

Received October 10th, 2000; accepted November 6th, 2000.
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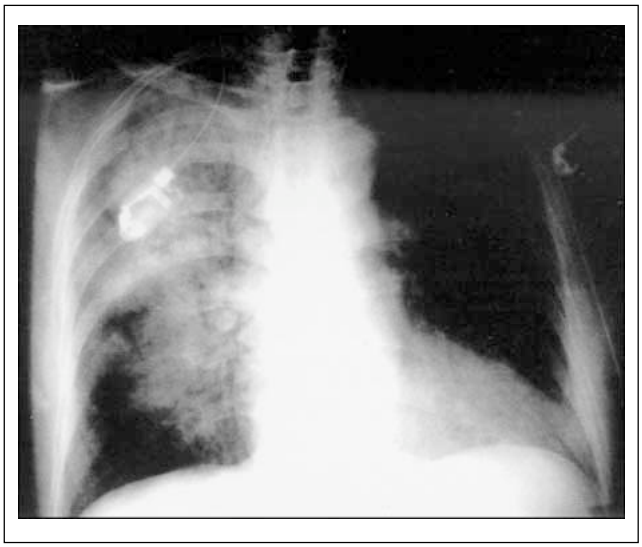

Figure 2. Posterior-anterior chest radiograph ten hours after stenting and abciximab bolus showed a patchy infiltrate of the right lung

Discussion

Abciximab represents a potent antiplatelet agent directed against platelet glycoprotein (GP) IIb/IIIa receptor that has been shown to prevent ischaemic complications of percutaneous transluminal coronary angioplasty (PTCA) [1–3]. However, its use is associated with an increased incidence of haemorrhagic complications and the need for blood transfusion. Thrombocytopenia has been observed in a few patients treated with abciximab bolus and infusion and is now considered as a well known adverse event [4–6].

In contrast, association of severe abciximab-induced thrombocytopenia and extensive pulmonary lesions is infrequent. Most reports describe alveolar haemorrhage as a platelet glycoprotein IIb/IIIa receptor-associated adverse reaction with pulmonary involvement [7–8]. Severe pulmonary haemorrhage is regularly associated with haemoptysis and longer lasting infiltrates on chest radiographs and therefore can be ruled out in the reported case clinically with high probability. The patient refused bronchoscopy.

Abciximab is a humanized chimeric Fab-antibody fragment of 7E3, a murine antibody directed against the integrin glycoprotein IIb/IIIa receptor located on platelets. The production of antimurine antibodies has been demonstrated with abciximab use [9]. Thrombocytopenia caused by abciximab is likely related to immunologic mechanisms. These immunologic mechanisms could cause derangement of alveolar capillary permeability and result in acute lung injury and pulmonary oedema [10–11].

We believe in a causal relationship for several reasons:

First, because of the close temporal association between starting abciximab and appearance of pulmonary infiltrates and symptoms as well as development of severe thrombocytopenia.

Second, because of the clinical time course with early clearance of the pulmonary infiltrates, which makes alveolar haemorrhage unlikely.

Third, because of recovery during treatment with corticosteroids. This therapeutic success with an anti-inflammatory drug additionally supports the hypothesis of underlying immunologic mechanisms.

To our knowledge, this is the first report of the simultaneous occurrence of pulmonary lesions and abciximab-induced thrombocytopenia that are, linked with high probability by immunologic mechanisms.

In conclusion, pulmonary lesions and abciximab-induced thrombocytopenia can be linked by the underlying haemorrhagic disorder or by immunologic mechanisms. Platelet count should be evaluated periodically during and after drug administration. Acute lung injury has to be considered as a rare adverse event with abciximab use.

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