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Transient Remission of Immune Thrombocytopenia during Acute Pancreatitis

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Secondary or 'reactive' thrombocytosis is not an uncommon occurrence in acute and chronic inflammatory disease [1–3]. The pathogenesis of this response is obscure and seems to be due to transient platelet overproduction rather than prolonged survival [4].

Case Report

Secondary thrombocytosis has not been recognized in acute pancreatitis. Recently, a patient under our care with idiopathic autoimmune thrombocytopenia refractory to therapy developed relative thrombocytosis following episodes of acute pancreatitis.

The patient was a 69-year-old woman. In May 1980 she was admitted to another hospital because of idiopathic autoimmune thrombocytopenia and Coombs'-positive hemolytic anemia (Evans' syndrome). She was started on prednisone 40 mg/day, folic acid 5 mg/day, and three 1-mg injections of vincristine. Following this therapy, her anemia improved, but she remained severely thrombocytopenic ($17 \times 10^9/l$) and experienced recurrent episodes of gastrointestinal bleeding. A splenectomy was performed with no improvement of the thrombocytopenia. She was discharged on prednisone 25 mg on alternate days and subsequently was admitted to our hospital for reevaluation. Physical examination revealed an obese woman with echymoses on the upper limbs. The hemoglo-

bin was 10.5 g/dl, reticulocytes 2.8%, the direct Coombs' test was positive +4 (anti IgG), platelets were $8-35 \times 10^9/l$, antinuclear factor and LE preparations were negative. An upper gastrointestinal series revealed cholelithiasis with a cholecysto-duodenal fistula.

The patient was started on intravenous hydro-cortisone 300 mg/day and cyclophosphamide 75 mg/day. After 1 month of treatment her hemoglobin rose to 13 g/dl, without any significant increase of the platelet count. At this time, she developed severe upper abdominal pain and tenderness with radiation to the interscapular area. The diastase level in the serum was found to be 1,050 IU (normal 70–300) and 7,700 IU in the urine (normal 150–2,300), the serum lipase was 3.6 U (normal 1.2). Plain films and ultrasonography of the abdomen excluded an acute abdomen due to retroperitoneal hemorrhage or a perforated viscus. Acute pancreatitis was diagnosed and treatment was begun with nasogastric drainage, intravenous fluids and antibiotics. Parallel to the development of the pancreatitis we noted a significant rise of the platelet count to $150 \times 10^9/l$ which receded to $8 \times 10^9/l$ with the resolution of the acute abdominal distress. 6 days later, there was a recurrent episode of pancreatitis with a similar response of the platelet count which reached $60 \times 10^9/l$. After her recuperation from the two episodes of pancreatitis, it

was decided not to attempt further therapy and she was discharged in a good general condition without any bleeding tendency, but with a low platelet count ($20 \times 10^9/l$).

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Discussion

It is unlikely that the observed rise of the platelet count was due to increased thrombopoiesis because of preexisting bone marrow megakaryocyte hyperplasia secondary to the prolonged thrombopoietic stimulus in a patient with immune thrombocytopenia [4].

We speculate that in this patient there may have been an interaction between activated pancreatic factors released during pancreatitis and the antiplatelet antibody or the RES system, resulting in a marked but transient rise of the platelet count.

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