

Figure 2. Graph of hemoglobin and reticulocyte values during parvovirus B19 infection. °indicate packed red cell transfusions; *immunoglobulin cycles. Antibody positivity to parvovirus B19 is reported underneath.

18 months the anemia gradually resolved, liver and spleen returned to normal size, and PCR for B19 became negative in the bone marrow. B19 specific IgG, positive during IVIG therapy, became negative after the treatment. Thirty months after the end of IVIG therapy the child's Hb is stable at about 120 g/L, and he is completely well.

Successful treatment of severe B19-induced intrauterine anemia after 4 months of transfusions was reported in 1994.7 Three other infants with anemia due to transplacental B19 infection were treated with IVIGs:2 two remained transfusion-dependent and one died after one course of IVIGs. B19 DNA was detected in their bone marrow but not in their serum. Despite persistent anemia, PCR analysis of the bone marrow became negative after IVIG therapy. Successful IVIG treatment of neurologic symptoms and anemia attributed to B19 infection was reported in a three-month-old hypogammaglobulinemic infant.8

Our patient was successfully treated with 12 cycles of IVIGs, with disappearance of the virus from the bone marrow. The immaturity of the fetal immune system, and the brief half-life of fetal IgM7 could explain the lack of B19 specific IgM. The brief thrombocytopenia could be attributed to reversible damage to megakaryocytes.9

To our knowledge this is the first reported case of proven B19-induced intrauterine anemia, in an otherwise normal child, which was successfully treated by IVIGs. If necessary, preparations of IVIGs with high titer of natural B19 specific antibodies could be tried.

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Key words

Neonatal anemia, parvovirus B19, intravenous immunoglobulins, fetal hydrops.

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Intramural hematoma of stomach after splenectomy for idiopathic thrombocytopenic purpura

We cared for a patient who developed a massive intramural hematoma of the stomach after splenectomy for refractory idiopathic thrombocytopenic purpura (ITP). This is the first report on this complication after splenectomy for ITP.

A 74-year old man with ITP underwent splenectomy following administration of high-dose immunoglobulin. His clinical course after the operation was uneventful, but the response was transient and his platelet count returned to the pre-operation levels within a week of splenectomy. On day 20 after the operation, when his platelet count was 15,000/µL, he suddenly complained of a severe chest pain radiating to the back and his blood pressure dropped

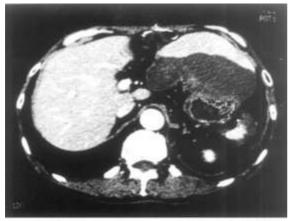




Figure 1. Upper panel: the abdominal computed tomographic scan with enhancement showed a large high density area in the gastric wall, which was considered a hematoma.

Lower panel: immediate digital subtraction angiography revealed two bleeding sites of the branches of the left gastroepiploic artery, which were successfully embolized with tornado coils and spongels.

below 50 mmHg. Because of his history of angina pectoris, we suspected cardiogenic shock due to ischemic heart disease, but this tentative diagnosis was made unlikely by the finding of an unremarkable electrocardiogram and reduced hemoglobin levels (6.4 g/dL). After intravenous administration of normal saline and a blood transfusion, the patients' vital signs became stable. Computed tomography of the abdomen revealed an intramural hematoma of the stomach and extravasation into the abdominal cavity (Figure 1, upper panel). Digital subtraction angiography of the abdomen showed active bleeding from two branches of the left gastroepiploic artery (Figure 1, lower panel). We embolized the left gastroepiploic artery with tornado coils and spongels, successfully achieving hemostasis of the bleeding sites.

Splenectomy is the second line therapy for ITP and indicated for patients who do not respond to the conventional corticosteroid treatment.¹ The risk of fatal perioperative bleeding is low in splenectomy for

ITP and the incidence of any complications is below 1.0%.² There have been only 15 reported deaths associated with splenectomy for ITP and most of them had other bleeding tendencies besides ITP.³ However, it is to be noted that our patient had no risk factors for hemorrhage except for thrombocytopenia.

Abdominal angiography revealed two active bleeding sites in the gastroepiploic artery. Although hemorrhage from the gastroepiploic artery is a wellknown complication of pancreatic pseudocyst,4 it is rarely involved in splenectomy and there is only one case report on gastroepiploic involvement after this operation.⁵ Considering the time interval between splenectomy and hemorrhage, suture insufficiency seemed unlikely and the anatomic location of the spleen and the left gastroepiploic artery suggested the association between the operation and hemorrhage. We suspected that false aneurysms might have been created by either the surgical procedure or pancreatic juice leakage after splenectomy and that they had ruptured 20 days after the operation. False aneurysms are frequently generated in the splenic and gastroepiploic arteries by blunt trauma or splenic laceration, 6,7 and are easily ruptured, sometimes leading to fatal hemorrhage.6

In conclusion, hemorrhage from the gastroepiploic and the splenic arteries can develop after splenectomy for ITP. Severe chest pain may occur due to the rapidly growing hematoma in the gastric serosa. Because symptoms are similar to those produced by ischemic heart disease, it is difficult to make an early diagnosis. We should recognize that splenectomy has a potential risk of massive hemorrhage from these arteries.

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Key words

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Leukemoid reaction preceding the diagnosis of colorectal carcinoma by four years

Sir.

We report the case of a patient in whom neutrophilic leukocytosis was discovered incidentally. He had no signs suggesting infection or neoplasia, and the results of the hematologic study were consistent with chronic neutrophilic leukemia. He remained asymptomatic for four years, with his leukocyte counts ranging from 12.9 to $40\times10^9/L$, until he developed intestinal obstruction that lead to the discovery of a colorectal carcinoma. The diagnosis of leukemoid reaction was finally established after surgical resection of the tumor was followed by normalization of the leukocyte count.

Chronic neutrophilic leukemia (CNL) is an exceedingly infrequent myeloproliferative disorder in which most of the circulating myeloid cells are mature granulocytes. ^{1,2} Differential diagnoses of CNL include, among others, leukemoid reaction to malignant neoplasias. In clinical practice, tumors producing leukemoid reaction characteristically have an aggressive clinical course and, because of this, both diagnoses are usually established either simultaneously or closely in time. We report the case of a patient with neutrophilic leukocytosis that preceded by four years the diagnosis of colorectal carcinoma.

A 78-year old man was referred to our center in November 1994 due to the incidental discovery of neutrophilic leukocytosis a few months earlier. The patient was asymptomatic, did not report visible blood loss in the stools or alterations in his bowel habits, and his physical examination was normal. Main laboratory data were: Hb 15.4 g/dL, WBC count 16.7×10°/L (neutrophils 84%, lymphocytes 10%, monocytes 6%), platelet count 106×10°/L, ESR 6 mm/h, serum LDH level 470 IU/L (normal value: 250-450 IU/L), and uric acid 8.4 mg/dL. Serum cobalamin level and vitamin B12 binding capacity were increased, and the leukocyte alkaline phos-

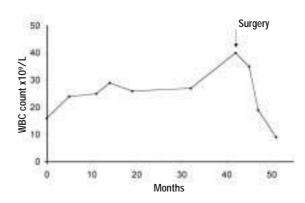


Figure 1. Evolution of the patient's leukocyte counts from first observation to latest follow-up.

phatase (LAP) score was also high. A bone marrow aspirate displayed increased cellularity with myeloid hyperplasia, and similar findings were seen in the marrow biopsy, with no fibrosis. Bone marrow cytogenetic study was normal and rearrangement of the bcr/abl gene was not observed. Since there were no signs suggesting infection or neoplasia, a tentative diagnosis of chronic neutrophilic leukemia (CNL) was established but, due to the moderate leukocytosis, no treatment was instituted. During the ensuing three and a half years, the patient remained asymptomatic, with his leukocyte counts ranging from 12.9 $\times 10^9$ /L to 40×10^9 /L. Most of the leukocytes were mature neutrophils. In June 1998 he suddenly developed intestinal obstruction, which lead to a diagnosis of colorectal carcinoma. Surgical resection of the carcinoma was followed by rapid normalization of the WBC counts, which have remained normal throughout the subsequent follow-up (Figure 1).

Persistent leukocytosis in the absence of infection in patients with malignant neoplasias is known as leukemoid reaction. The exact mechanisms underlying this phenomenon are not well established. Recent reports on solid tumors associated with neutrophilic leukocytosis suggest that cytokine production by the tumor increases granulocyte production, therefore resulting in the leukemoid reaction. Indeed, the production of granulocyte colony-stimulating factor (G-CSF), granulocyte-macrophage-CSF, interleukin-1, and/or interleukin-6 by bladder, 3,4 lung⁵ and tongue⁶ carcinomas has been described in association with leukemoid reactions. However, the fact that no detectable cytokines can be found in other patients indicates that neutrophilic leukocytosis may be induced by mechanisms other than the above described.⁷ From the clinical point of view, the presence of a leukemoid reaction in the setting of a malignant tumor usually reflects an aggressive clinical