The patient is a 12-year-old Hispanic boy with hemophilia A (factor VIII deficiency) and high Bethesda unit factor VIII inhibitor who presented to a clinic after 4 days of abdominal pain and clear, nonbilious vomiting. Viral gastroenteritis had been diagnosed, and the boy was given rabeprazole and promethazine, which failed to resolve symptoms. The patient was hospitalized when he became unable to tolerate oral fluids; his urinary output decreased. He denied fever, diarrhea, hematemesis, hematochezia, and melena.

On admission, the patient was afebrile, appeared mildly dehydrated, and complained of severe abdominal pain. An abdominal examination revealed guarding and tenderness in the left upper quadrant. His white blood cell count was $17 \times 10^3/\mu L$; hemoglobin level was 10.7 g/dL. Serum electrolyte, transaminase, amylase, and lipase levels were normal. A CT scan of the abdomen revealed an 8.5 x 9-cm intramural gastric hematoma.

William H. Black, Jr, MD, John Pohl, MD, and Lawrence Frankel, MD, of Scott and White Hospital, Texas A&M University, Temple, Tex, write that hemophilia A is an X-linked recessive bleeding disorder caused by the deficiency of factor VIII. It affects about 1 in 5000 males worldwide; there appears to be no ethnic preference.

Hematomas of various mucosal structures are well-known complications in patients with hemophilia. The most common sites of involvement are the joints and muscles. Bleeding into these areas is secondary to trauma sustained during vigorous activity. Other less commonly involved sites include the oral mucosa, genitourinary tract, GI tract, and CNS. Recurrent, forceful vomiting can result in a gastric mucosal tear and subsequent bleeding into the gastric wall. Although hematomas of the GI tract occur most commonly in the duodenum, they rarely occur in the stomach, as in this patient. Patients with factor VIII inhibitors are generally categorized as either low responders or high responders (as measured by the Bethesda unit antibody titer). Low responders typically are treated with higher doses of factor VIII, while high responders require aggressive support, including continuous factor VIII infusion or recombinant factor VIIa infusion. The latter interacts at the tissue injury site by activating factor X and bypassing the factor VIII-dependent step of coagulation.

This patient was given intravenous fluids, and a nasogastric tube was inserted. He also received multiple factor VIIa infusions, and serial hemoglobin levels were measured. The nasogastric tube was removed on the second hospital day, and a nasojejunral tube was inserted under fluoroscopic guidance to bypass the obstruction and to provide adequate nutrition as the hematoma resolved. Eventually, the patient was able to tolerate oral fluids; he had no further sequelae. An abdominal CT scan obtained several weeks later showed resolution of the hematoma.
References:


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