ti-HBs(-), anti-HBc IgM(+), anti-HBc IgG(+), Anti-HBe(+), and polymerase chain reaction (PCR) HBV DNA >110000000 IU/mL. Serological tests for hepatitis B were consistent with reverse seroconversion from anti-HBs to HBsAg. Lamivudine treatment was started at 300 mg per day. INR progressively elevated, and hypoalbuminemia and ascites developed. The patient's consciousness worsened and she died of liver failure 21 days after the treatment was started.

Hepatitis B virus (HBV) is a DNA virus that can be cleared at rates of more than 95% in acute infection in adults. However, it can cause chronic infection in about 5% of adult patients. Immunosuppressive and chemotherapeutic agents can stimulate the replication of the virus. It is well known that reactivation of HBV in subjects receiving cytotoxic treatment for hematological malignancies occurs in 21-53% of chronic HBsAg carriers and in an unknown number of HBsAg-negative subjects harboring occult HBV infection (1). Immune reconstitution within the weeks and months following recovery from chemotherapy may be associated with a flare of hepatitis B manifested by hepatocellular injury (2). This status can lead to severe hepatitis and fatal liver dysfunction. In order to avoid this potentially fatal complication, it is important to ensure that all patients at risk of chronic HBV infection are screened before commencing immunosuppressive treatment. HBV-negative patients should be immunized. In the patients with prior HBV infection, even with anti-HBs(+), anti-HBc IgG(+) and PCR HBV DNA(-), anti-viral agents should be started before or at least at the same time as chemotherapy. Nucleoside analogues are more acceptable when compared with other agents in these patient groups. Most studies have suggested lamivudine for hepatitis B reactivation (3, 4).

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Spontaneous intraabdominal hematomas associated with arterial aneurysms in polycythemia vera

Polisitemia veralı olguda arteryal anevrizma ile ilişkili spontan intraabdominal hematom

To the Editor,

A 73-year-old male was diagnosed with polycythemia vera five years earlier and treated with periodic phlebotomies. He also suffered from chronic obstructive pulmonary disease for three years. In

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March 2005, he admitted to the emergency department with abdominal distension and dyspnea. Total blood count revealed a hemoglobin concentration of 19.3 g/dl, hematocrit 59.9 g/dl, white blood cell (WBC) count 12.5 x 10^9/L, and platelet count 650 x 10^9/L. A therapeutic phlebotomy was performed. At that time, an abdominal ultrasonography (US) was unremarkable except for hepatosplenomegaly and simple renal cysts.

He recently readmitted to the hospital with severe abdominal pain. Physical examination revealed splenomegaly and a palpable mass at the right upper quadrant. Laboratory tests revealed a hematocrit level of 60.2 g/dl, hemoglobin 19.5 g/dl, red blood cell (RBC) count 8.19 x 10^9/L, WBC count 13.3 x 10^9/L, platelet count 704 x 10^9/L, prothrombin time 20.1 sec, partial thromboplastin time 58.4 sec, and international normalized ratio (INR) 1.63. Abdominal US examination revealed 8x8 cm and 4.5x4 cm sized collocated hematomas in the subhepatic area. Doppler US showed a 2x2 cm pseudoaneurysm adjacent to the large hematoma, and a 1.5x1 cm pseudoaneurysm within the smaller hematoma (Figure 1). Multidetector computerized tomography (MDCT) with CT arteriography was performed and confirmed the hematoma formations and the pseudoaneurysms (Figure 2). CT angiography demonstrated the origin of both pseudoaneurysms from a branch of the gastroduodenal artery. In addition, CT angiography showed a 0.5 cm in diameter pseudoaneurysm formation at a branch of the right hepatic artery. No history of trauma or interventional procedure was present in this case. The medical and respiratory condition of the patient did not allow any surgical or interventional radiological procedures; thus, clinical and radiological follow-up was decided.

Patients with myeloproliferative disorders such as polycythemia vera have a paradoxical predisposition to bleeding and thrombotic complications that are major causes of morbidity and mortality. Both thrombosis and hemorrhage are frequent complications especially in the elderly, occurring in 33-50% of patients (1). Hemorrhage may be spontaneous or iatrogenic in these cases. Spontaneous bleeding manifestations are often associated with extreme thrombocytosis that may lead to acquired von Willebrand's disease (2). Although the precise mechanisms have not yet been clarified, acquired von Willebrand's disease, platelet dysfunction, consumption coagulopathy, and side effects of antiplatelet therapy have been implicated in the spontaneous hemorrhagic complications of myeloproliferative disorders (1-7). Iatrogenic hemorrhages have been reported usually after bone marrow biopsy.

The association of arterial pseudoaneurysm formation with von Willebrand's disease has been highlighted in a few case reports. The coagulation disorder was considered to be the predisposing factor in the pathogenesis of the pseudoaneurysm formation (8). In our case, the hematoma formation was due to the spontaneous pseudoaneurysm formation of the branch of the gastroduodenal artery, and an accompanying small intrahepatic arterial pseudoaneurysm. No history of trauma, or surgical, endoscopic, or interventional procedure was present in our case.

When a patient with myeloproliferative disorder complains of a sudden onset of abdominal pain, the possibility of visceral organ infarction due to thrombosis or a hemorrhagic event should be sus-

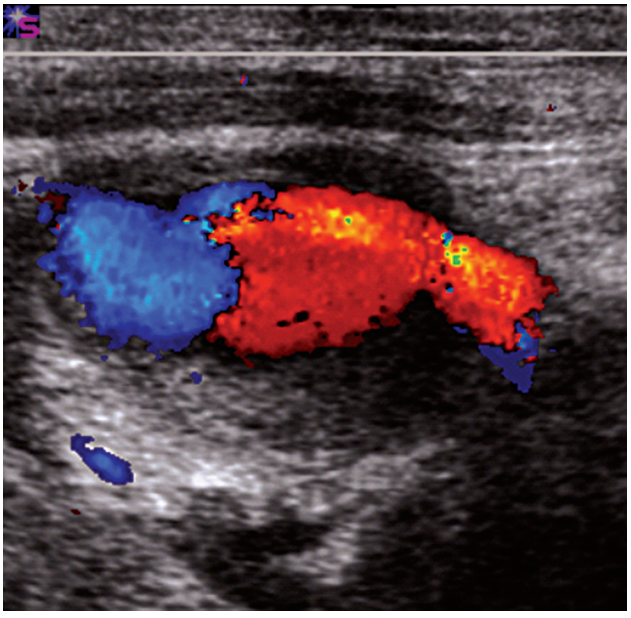

Figure 1. Color Doppler US shows color encoding that indicates pseudoaneurysm formation within the hematoma.

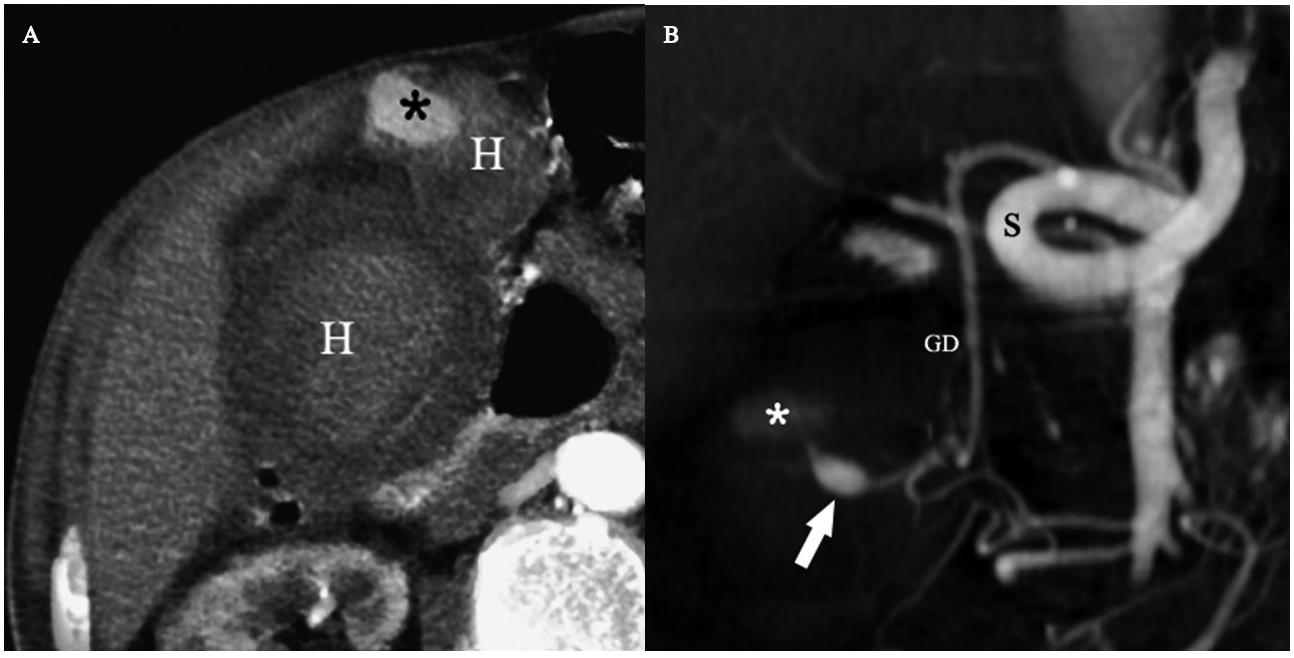

Figure 2. **(A)** Axial MDCT scan shows hematoma formations (H) and pseudoaneurysmal contrast filling (asterisk) in the small hematoma. **(B)** Coronal maximum intensity projection (MIP) image clearly shows both pseudoaneurysm formations (arrow and asterisk) originating from a branch of the gastroduodenal artery. (S: Splenic artery; GD: Gastroduodenal artery).

pected. An abdominal US examination is usually the initial radiological modality of choice in acute onset abdominal pain. US may easily identify a solid organ hematoma formation as well as free intraperitoneal hemorrhage. The presence of free intraperitoneal fluid should raise the suspicion of an intraabdominal hemorrhagic event, especially in the presence of fluid-fluid levels and echogenic clot within the free fluid. Abdominal US examination integrated with Doppler US may provide essential information regarding the cause of the acute abdominal pain. Doppler US may identify thrombotic events within the portal venous system. On the other hand, Doppler US may not definitely show an active bleeding site. In our case, the intraperitoneal hemorrhage was due to pseudoaneurysm formation of the gastroduodenal artery. The origin of the pseudoaneurysms can not always be exactly identified on Doppler US. Free intraperitoneal hemorrhage identified on US in a hemodynamically stable patient should be evaluated further by CT.

The ability to detect intraabdominal vascular pathologies and the active bleeding sites has been increased with the use of contrast-enhanced MDCT. A number of new applications for MDCT have been developed recently, one of which is abdominal CT angiography. Conventional diagnostic angiographic procedures remain the gold standard for the diagnosis of hemorrhagic events; however, contrast-enhanced MDCT may replace the diagnostic part of angiography. Angiographic procedures are now generally being preserved for problem solving or directional therapy, such as embolization. On CT, fresh hemorrhagic collections show soft tissue attenuation values. Fresh hemorrhage may be better detected on contrast-enhanced images as a hypoattenuating rim around the liver and spleen. Clots have relatively high CT attenuation values. Intraabdominal fluid-fluid levels may also be identified on CT. In our case, we identified two nonhomogeneous hyperattenuating fresh hematomas and enhancing pseudoaneurysms adjacent to the hematomas.

In conclusion, the association between spontaneous pseudoaneurysm formations in a patient with polycythemia vera has not been reported before. A presumptive association between coagulopathy and pseudoaneurysm formation was established in certain reports. Thus, our case enhances this association.

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