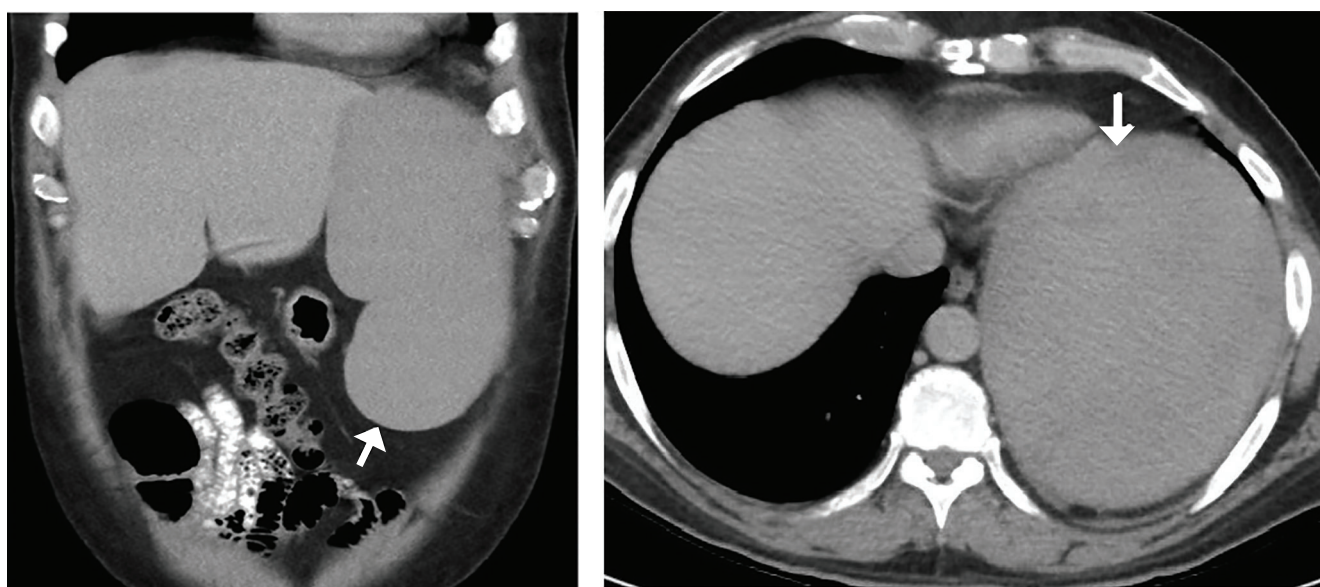


## THE CLINICAL PICTURE

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# Atraumatic splenic rupture in acute myeloid leukemia



**Figure 1.** Coronal (left) and transverse (right) views on initial computed tomography of the abdomen without contrast showed massive splenomegaly (white arrow).

**A** 50-YEAR-OLD MAN with acute myeloid leukemia (AML) with a complex karyotype was admitted to the hospital with several days of dull, left-sided abdominal pain. His most recent bone marrow biopsy showed 30% blasts, and immunophenotyping was suggestive of persistent AML (CD13+, CD34+, CD117+, CD33+, CD7+, MPO–). He was on treatment with venetoclax and cytarabine after induction therapy had failed.

On admission, his heart rate was 101 beats per minute and his blood pressure was 122/85 mm Hg. Abdominal examination revealed mild distention, hepatomegaly, and previously known massive splenomegaly, with the splenic tip extending to the umbilicus, and mild tenderness.

Results of laboratory testing revealed persistent

pancytopenia:

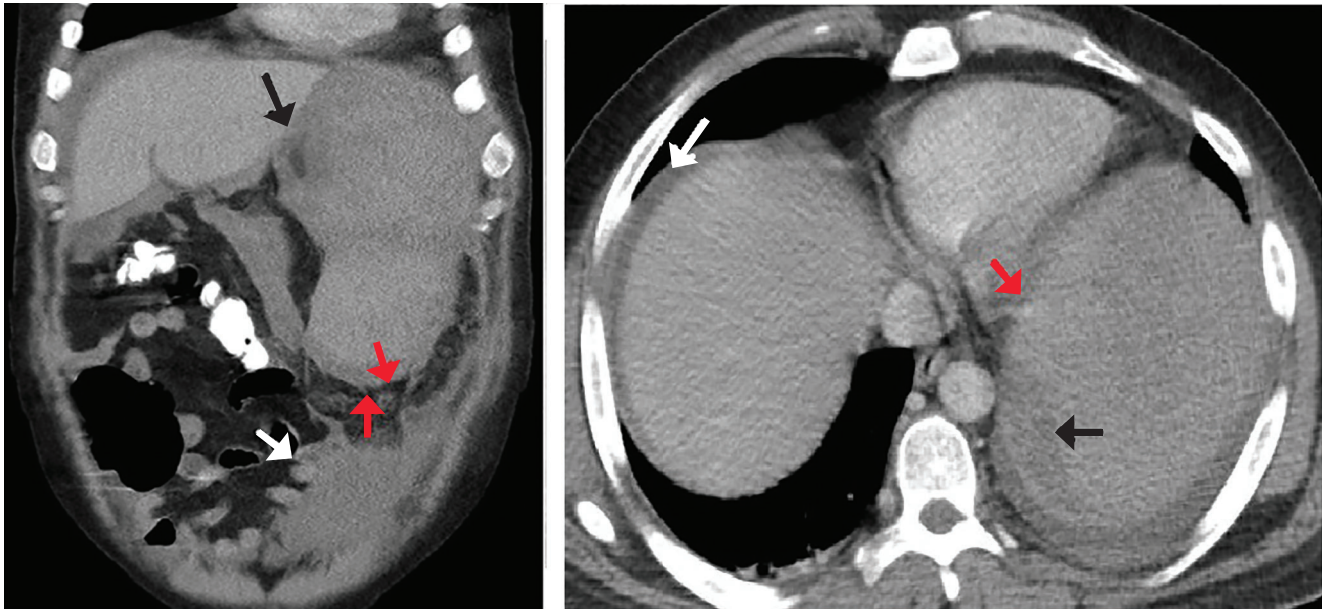
- Hemoglobin level 6.8 g/dL (reference range 13.0–17.0)
- Total white blood cell count  $0.8 \times 10^9/L$  (4.5–11.0)
- Platelet count  $8 \times 10^9/L$  (150–400).

Computed tomography (CT) of the abdomen (**Figure 1**) showed splenomegaly (the spleen measured 26 cm, unchanged from before), but without evidence of infarct or other acute abnormality.

The next day, he developed severe, acute-onset left-sided abdominal pain. A check of vital signs showed worsening sinus tachycardia at 132 beats per minute and a drop in blood pressure to 90/56 mm Hg. He had worsening diffuse abdominal tenderness with sluggish bowel sounds. His hemoglobin concentration was 6.4 g/dL and platelet count  $12 \times 10^9/L$ .

Urgent CT of the abdomen with contrast (**Figure 2**) showed heterogeneous splenic enhancement sug-

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**Figure 2.** On repeat computed tomography with contrast, coronal (left) and transverse (right) views showed irregular splenic margins (red arrows), intraparenchymal hemorrhages (black arrows), and hemoperitoneum (white arrows).

gestive of intrasplenic hemorrhage, irregularity of the margins suggestive of rupture, and moderate hemoperitoneum.

He received supportive transfusions of blood products. Surgical exploration was deemed risky, given his overall condition and severe thrombocytopenia. Splenic angiography showed no evidence of pseudoaneurysm or focal contrast extravasation. He underwent empiric embolization of the midsplenic artery, after which his hemodynamic status stabilized. He died 4 weeks later of acute respiratory failure from pneumonia.

**■ SPLENIC RUPTURE IN AML**

Atraumatic splenic rupture is rare but potentially life-threatening, especially if the diagnosis is delayed. Conditions that can cause splenomegaly and predispose to rupture include infection (infectious mononucleosis,

malaria), malignant hematologic disorders (leukemia, lymphoma), other neoplasms, and amyloidosis.<sup>1</sup>

The literature includes a few reports of splenic rupture in patients with AML.<sup>2-4</sup> The proposed mechanisms include bleeding from infarction sites or tumor foci, dysregulated hemostasis, and leukostasis.

The classic presentation of splenic rupture is acute-onset left-sided abdominal pain associated with hypotension and decreasing hemoglobin levels.

CT of the abdomen is confirmatory, and resuscitation with crystalloids and blood products is a vital initial step in management. Choice of treatment depends on the patient’s surgical risk and hemodynamic status; options include conservative medical management, splenic artery embolization, and exploratory laparotomy.

In patients with AML and splenomegaly presenting with acute abdominal pain, clinicians need to be aware of this potential hematologic emergency. ■

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