IMAGE OF THE ISSUE

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Extensive Hepatic Infarction due to Polycythemia Vera

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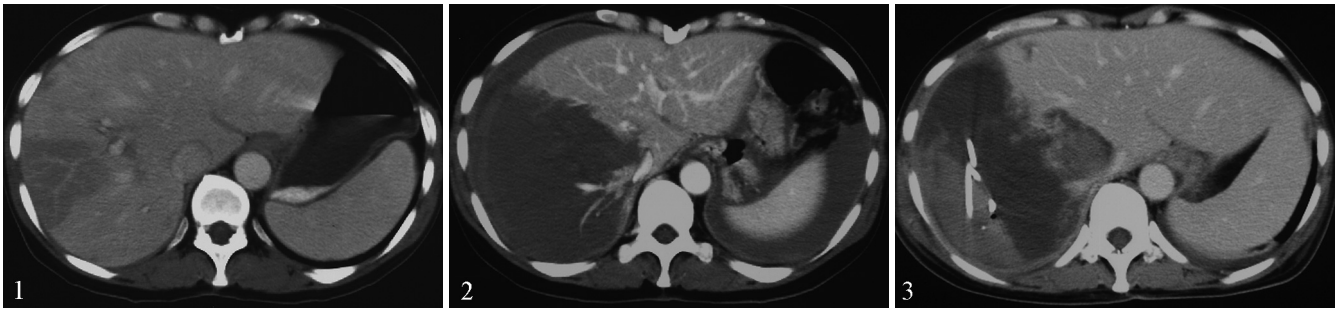

A 45-year-old woman emergently presented with abdominal pain and nausea. Blood tests revealed pancytosis with leukocytes of 27,660/μL, erythrocytes of 742 $\times 10^4$/μL, hemoglobin of 20.3 g/dL, hematocrit of 60.1%, and thrombocytes of 75.7$\times 10^4$/μL, as well as liver damage with aspartate aminotransferase of 344 U/L, alanine aminotransferase of 720 U/L, and total bilirubin of 2.4 mg/dL. Abdominal contrast-enhanced computed tomography (CECT) showed a wedge-shaped low-density area with internal portal veins and obstruction of the right hepatic vein in the right lobe of the liver (Fig. 1). She was hospitalized and went into shock a few hours later The second CECT revealed that the right branch of the portal vein and part of the inferior vena cava were not contrasted, while the right lobe was an extensively low-density area; therefore, hepatic infarction was diagnosed. Pleural effusion and ascites were also observed (Fig. 2). Systemic anticoagulation was immediately initiated for thrombophilia. However, liver damage worsened, resulting in hepatic failure. Plasma exchange was performed five times, which gradually attenuated liver damage. She was examined at the Hematology Department and diagnosed with polycythemia vera (PV). Hydroxycarbamide was initiated. Continuous fever developed from day 20 and was attributed to an abscess caused by hepatic necrotic materials; therefore, abscess drainage was performed, and the tube was continuously washed thereafter (Fig. 3). Fever subsequently resolved, the abscess cavity decreased, and the drainage tube was removed. The patient was discharged without subjective symptoms on day 135.

Polycythemia vera is a BCR-ABL1-negative myeloproliferative neoplasm characterized by activating mutations in JAK2, which clinically presents with erythrocytosis and has an increased risk of both thromboembolic events and progression to myelofibrosis and acute myeloid leukemia [1, 2]. Splanchnic vein thrombosis is a rare manifestation of venous thromboembolism in one or more abdominal vessels and is strongly associated with PV [3]. The management of splanchnic vein thromboses in PV requires a multidisciplinary approach involving gastroenterologists and hematologists [4].

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Conflicts of interest: None to declare.

REFERENCES

1. Bewersdorf JP, How J, Masarova L, et al. Moving towards disease modification in polycythemia vera. Blood 2023;142:1859-1870. doi:10.1182/blood.2023021503
2. Tefferi A, Barbui T. Polycythemia vera: 2024 update on diagnosis, risk-stratification, and management. Am J Hematol 2023;98:1465-1487. doi:10.1002/ajh.27002
3. Tremblay D, Winters A, Beckman JD, et al. Splanchnic vein thrombosis associated with myeloproliferative neoplasms. Thromb Res 2022;218:8-16. doi:10.1016/j.thromres.2022.08.003.
4. Finazzi G, De Stefano V, Barbui T. Splanchnic vein thrombosis in myeloproliferative neoplasms: treatment algorithm 2018. Blood Cancer J 2018;8:64. doi:10.1038/s41408-018-0100-9