A 55-year-old man was admitted for the rapid onset of severe pain, pallor, and paresthesia in the left foot. Three weeks earlier, he suddenly had developed bilateral lower-extremity claudication, which limited his ambulation to 30 feet. A diagnosis of paroxysmal nocturnal hemoglobinuria had been made in 1961 and later was confirmed by flow cytometry. Therapy had included corticosteroids with varying incremental dosing that was complicated by type II diabetes mellitus. Warfarin was added to his medical regimen in 1996 after a right hemispheric stroke and right renal infarction. For 6 months before admission, he had noted hematuria and fatigue with laboratory evidence of worsening hemolysis, requiring augmentation of his corticosteroid dose. The prothrombin time international normalized ratio at the time of admission was 2.7.

An aortogram performed soon after admission revealed a normal aortic contour without plaque or aneurysm (Figure 1, left). Multiple filling defects were noted in the abdominal aorta. The left popliteal artery was occluded (Figure 2), as was the right tibioperoneal trunk, with the right foot perfused by the anterior tibial artery only. CT (Figure 1, right) revealed thrombus extending from the aortic arch to the infrarenal aorta with multiple infarcts of the spleen, liver, and kidneys.

Figure 1. Subtracted aortogram (left) depicts multiple intraluminal low-attenuation filling defects (black arrows) consistent with thrombi. CT of the chest and abdomen (right) confirm intraluminal low-attenuation filling defects extending from the thoracic through the abdominal aorta (thick white arrows). Splenic infarction (thin white arrow) is readily appreciated.
Figure 2. Subtracted angiographic runoff of the left lower extremity reveals acute thromboembolic occlusion of the popliteal artery (arrows) with reconstitution of the anterior and posterior tibial arteries by collateral vessels.
Aortic Thrombosis as a Complication of Paroxysmal Nocturnal Hemoglobinuria
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