A huge intra-abdominal hematoma with an unusual etiology

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QUESTION
A 51-year-old man with persistent abdominal pain for half a month consulted at our emergency department. The patient stated that the pain was dull, persistent, and located at the right lower abdomen. Physical examination revealed uncontrolled hypertension, with a blood pressure of up to 227/115 mmHg. A huge palpable mass was detected at the right lower quadrant, with local tenderness, but without peritoneal signs. Biochemistry examinations showed leukocytosis with a white blood cell count of 11020/uL and elevated C-reactive protein level of 73.10 mg/L. Abdominal computed tomography (CT) scans (Brilliance 64; Philips) revealed a huge hematoma of approximately 15×11×8 cm at the mesentery (Figure 1).

Angiography showed no hypervascular lesions, active contrast extravasation, aneurysms, or arteriovenous malformations. However, an indentation with severe stenosis was noted in the proximal celiac trunk, and the inferior pancreaticoduodenal artery (PDA) was dilated, possibly as a compensatory supply to the celiac territory via the gastroduodenal artery (Figure 2).

What is the patient’s most probable diagnosis?
The indentation at proximal celiac trunk with severe stenosis was best presented on parasagittal reformation CT (Figure 3). This finding was compatible with external compression by median arcuate ligament of diaphragm. In accordance with imaging findings, a mesenteric hematoma secondary to median arcuate ligament syndrome (MALS)-induced PDA aneurysm rupture was diagnosed. Considering the patient’s relatively stable vital signs and the lack of evidence of active bleeding, we initially achieved strict blood pressure control with amlodipine and valsartan. We did not perform surgery; instead, we employed conservative treatment with fasting, intravenous fluid, and bed rest.

A subsequent abdominal CT scan performed a week later revealed no signs of hematoma progression. The patient’s symptoms improved after he received the therapy, and he was discharged after 10 days of hospitalization. Four months later in the out-patient department follow-up, the CT scans showed shrinkage of the hematoma. The size of the hematoma decreased to 10×8×7 cm (Figure 4). Severe stenosis at the proximal celiac artery with a relatively enlarged superior mesenteric artery still existed (Figure 5). Thirteen months later, the CT scan revealed further shrinkage of hematoma with size approximately 4.4×4×3.7 cm (Figure 6). The patient recovered well without complications.

The causes of PDA aneurysms include atherosclerosis, trauma, pancreatitis, and MALS (1). MALS is a rare disorder. Narrowing of the median arcuate ligament compresses the celiac trunk and induces a series of symptoms. In rare cases, the stenosis or occlusion of the celiac artery can lead to a compensatory increase in the blood flow through the collateral arteries from the superior mesenteric artery (1-3). This increased blood flow creates turbulence and exerts a shearing force on the intima (4). This mechanism and the increased intraluminal pressure finally induce endovascular damage and aneurysm formation (1-4).

This syndrome was first discovered by Sutton and Lawton in 1973 (2), and it accounts for only 2% of all splanchnic aneurysms (2,4). Aneurysm ruptures are associated with approximately 10%-50% mortality rate (2,3). To date, no standard treatment guidelines for PDA aneurysm rupture have been established because of the limited number of cases (3). Recently, endovascular repair, including coil embolization and endovascular stent...
placement, has become the major treatment modality for PDA aneurysm rupture (1,3). Compared with open surgery, endovascular interventions are less invasive and have fewer complications, such as bowel resection, infection, and sepsis (3).

The necessity of median arcuate ligament division and celiac artery revascularization remains controversial. Decompression of the celiac trunk can prevent the formation of aneurysm (5), and celiac trunk revascularization is crucial for the blood supply to the liver (5). However, recent studies have challenged this viewpoint. The theory is that well-established collateral blood flow can provide adequate blood supply to the liver, with relatively lower intraluminal pressure (3). Hence, recurrent aneurysm formation is less likely, and division of the median arcuate ligament is not necessary (3).

Almost all authors disagree with the initial conservative treatment. All PDA aneurysms are suggested to be treated because the risk of aneurysm rupture is not related to the size (1-4). However, in our case, angiography detected neither a lesion with extravasation nor an aneurysm; therefore, the precise location for endovascular intervention was unclear. Our diagnosis is a ruptured and clogged PDA aneurysm. In this way, blood pressure control with conservative treatment seemed feasible in such a special situation. Intra-abdominal hematomas should be resected because of their mass effect; however, endovascular embolization or stent placement warrants further discussion.

Pancreaticoduodenal artery aneurysm secondary to MALS is extremely rare, but potentially life-threatening. Aneurysm rupture can lead to hemorrhagic shock and a high likelihood of mortality. Endovascular intervention is indicated for diagnosis and emergency treatment. Conservative treatment is not often suggested; however, it might be successful in specific situations in which precise location for endovascular intervention is unknown.

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REFERENCES


