Dear Editor,

Hemocholecyst describes the hemorrhage of gallbladder and has been related to gallbladder pseudoaneurysms. The exact mechanism for the development of a pseudoaneurysm remains unclear (1); however, it has been reported secondary to inflammatory conditions, malignancy, and biliary tract trauma. Interestingly, 6%-17% of gallbladders removed for cholecystitis contain no gall stones, and its etiological mechanisms have been postulated for acalculous cholecystitis (AC) (2). We describe a rare case of a hemocholecyst caused by ruptured aneurysms in relation to AC, which mimicked gallbladder cancer (GC). The association between pseudoaneurysms and AC and the primary etiology were inconclusive.

An 81-year-old male with tarry stool for a day reported to have direct bleeding from the ampulla of Vater had endoscopic confirmation of hemobilia. For the next 24 hours, significant Murphy’s sign was observed. Ultrasonography showed distended thick-walled gallbladder with protruding fundic lesions. Abdominal computed tomography revealed a distended, wide, double-border gallbladder with intracystic extravasation, whereas the biliary tract appeared normal (Figure 1). Hemocholecyst owing to ruptured aneurysms in relation to AC and GC could not be differentiated. Open cholecystectomy was performed, in combination with en bloc wedge-liver resection. Because a protruding gallbladder mass was identified at the liver bed region, GC could not be excluded. There was extensive mural thickening of gallbladder with adhesions involving the transverse mesocolon, omentum, and duodenum (Figure 2). The gallbladder contained several pseudoaneurysms in the fundus (Figure 3). AC was confirmed using histological studies, and no GC was observed by hematoxylin and eosin staining. This study was conducted with the approval of the institutional review board of Show Chwan Memorial Hospital, Taiwan (Approval No: 1050409). Informed consent was obtained from the patient who participated in this study.

Biliary colic, jaundice, and gastrointestinal bleeding are the classical triad of hemobilia originally reported by Quincke (3). However, the triad was only reported in 38% of cases (4). Our patient showed initiating symptoms with gastrointestinal bleeding, and laboratory examination revealed jaundice.

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In our patient, there was neither any obvious sign of biliary obstruction, nor an evidence of gallstone. AC should be responsible for the cause of hemocholecyst. It is reported that clot formation is affected by both the rate of bleeding and bile flow. In spite of bile being in fibrinolytic nature, the formation of “pure” clots without gallstone debris is highly observed in cases with minor bleeding. Blood loss into the duodenum may be slow and intermittent; therefore, this makes pure clots likely to be stable rather than to be dissolved, and therefore, more likely to cause cystic duct obstruction and further worsening cholecystitis (4).

Perioperative differential diagnosis of GC from hemocholecyst caused by ruptured aneurysms in relation to AC can be challenging when the inflammatory involvement of surrounding tissues is implicated. Because GC may coexist, as in our patient, treatment of radical resection is reasonable when we cannot exclude malignancy completely. In addition, Maeda et al. (5) reported a “two-step” strategy consisting of embolization of the cystic artery followed by elective cholecystectomy. Consequently, AC is a serious complication for hemocholecyst, and the history of diseases is crucial for the diagnosis.

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REFERENCES