representing approximately 14–20% of cases. The vast majority are intradural lesions, extradural lesions accounting for only 1–2%[4]. Extradural meningiomas affect the cranial vault in 68% of cases, such lesions being referred to as primary intraosseous meningiomas (PIMs), which mainly affect the frontal and parietal bones, as well as the region of the orbit[5–7]. Other common locations for extradural involvement are the subcutaneous tissue, paranasal sinuses, and parapharyngeal spaces, as well as, in rare cases, the lungs and adrenal glands[5,6]. Unlike typical intradural meningiomas, which primarily affect females between the ages of 50 and 69 years and usually have a benign course, PIMs can affect either gender, have a peak incidence in the second decade of life, and are more likely to evolve to malignant degeneration[6].

On CT, most PIMs (65%) present as expansile, osteoblastic bone lesions, with or without cortical destruction[6]. On MRI, they commonly hypointense in T1- and T2-weighted sequences, typically without significant contrast enhancement, as in the case reported here[5]. However, in rarer cases, if a PIM presents as an osteolytic lesion on CT, an MRI scan can show a hypointense signal in T1-weighted sequences and a hyperintense signal in T2-weighted sequences, as well as contrast enhancement[6,7]. Although PIMs do not present the dural tail sign that is often found in intradural meningiomas, there can be contrast uptake in the dura mater subjacent to the tumor due to venous stasis or to tumor invasion, as demonstrated in our case[7]. There are inherent differences between CT and MRI, the former allowing better delineation of bone involvement, whereas the latter provides a better assessment of the soft-tissue involvement and extradural extent of the lesion[6].

The differential diagnosis of osteoblastic PIM includes typical intradural meningioma with reactive hyperostosis, in which the meningeal component of the lesion is the most obvious. Other diagnoses that should be considered are metastases, plasmacytoma, fibrous dysplasia, osteoma, osteosarcoma, and Paget's disease[6].

In most cases of PIM, the treatment is total surgical resection, with subsequent cranial reconstruction. If the resection is partial, there should be radiological follow-up; if the disease has recurred or if the residual lesion has progressed, the next surgical procedure can be accompanied by adjuvant radiotherapy[6].

In conclusion, although rare, PIMs should be considered in the differential diagnosis of bone lesions, especially when the lesions are osteoblastic and located in the cranial vault.

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Hemobilia in a patient with arteriobiliary fistula after liver contusion

Dear Editor,

We report the case of a 25-year-old male patient with a history of blunt abdominal trauma (from a motorcycle accident), who presented with abdominal pain. Full abdominal computed tomography (CT) with intravenous contrast administration revealed that the patient had grade 2 liver contusion in the right lobe. Because the patient was hemodynamically stable, we opted for conservative treatment. However, he evolved to hemodynamic instability. An exploratory laparotomy revealed a mosaic pattern of liver injury, which was treated with hepatorrhaphy. On the eighth day after surgery, the patient was in a stable, lucid state and was discharged. However, he returned 17 days later with abdominal pain after a large meal, together with voluminous hematemesis and hypovolemic shock. We then performed abdominal CT angiography (Figure 1), which revealed a pseudoaneurysm in the right hepatic artery, in close proximity to the liver contusion. There was also spontaneously hyperdense content within the gallbladder, suggesting arteriobiliary fistula. Upper gastrointestinal endoscopy showed blood clots and active bleeding in the papilla of Vater, and arteriography (performed at a different facility) confirmed the existence of pseudoaneurysm in the right hepatic artery in the subbranch of liver segment V, with contrast extravasation suggestive of rupture. Therefore, embolization was carried out.

Hemobilia is an uncommon condition and is one of the differential diagnoses of upper gastrointestinal hemorrhage[1]. There are many causes of hemobilia, such as iatrogenic and accidental traumas, as well as gallstones, inflammation, vascular malformations, and tumors[2]. The clinical manifestations of hemobilia are determined by the quantity and velocity of the hemorrhage within the biliary tract. Its symptoms are jaundice, right hypochondrium pain, and gastrointestinal hemorrhage (ranging from chronic bleeding, resulting in anemia, to massive bleeding with hypotension), and it can develop several months after a trauma[4,5].

The improvement of radiological techniques has been fundamental in the diagnosis and treatment of hemobilia, especially in cases of traumatic pseudoaneurysms[3]. In patients with upper gastrointestinal hemorrhage, upper gastrointestinal endoscopy is the examination of choice, because it can identify blood clots in the ampulla of Vater and rule out other causes of bleeding. Ultrasound is a rapid, noninvasive method that is useful and effective in the detection of hemobilia, potentially revealing blood clots or echogenic intraluminal material in the biliary tree or gallbladder. However, contrast-enhanced CT (in the arterial phase) can detect pseudoaneurysms, obstruction of

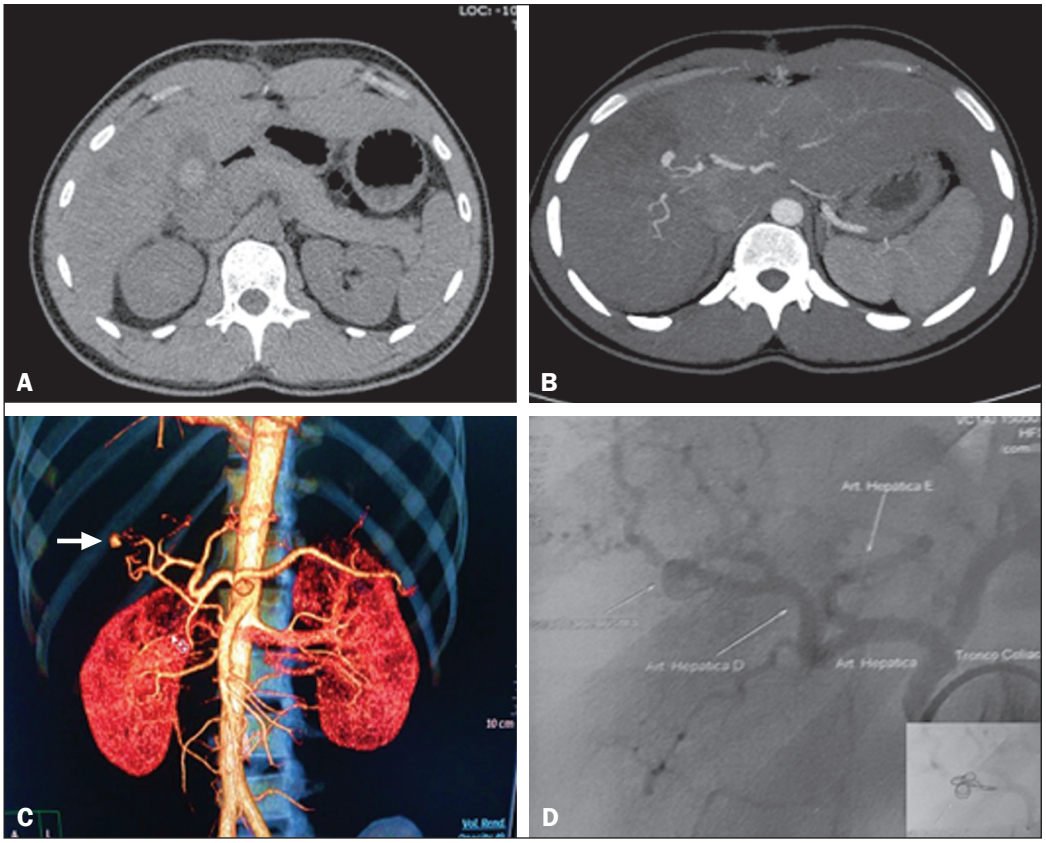

Figure 1. A: Noncontrast axial CT showing spontaneously hyperdense (possibly hematic) material in the interior of the gallbladder. **B:** Axial CT after intravenous contrast administration, with MIP, showing arterial lesion due to local liver contusion, with a probable communication between the artery and the biliary duct. **C:** Volumetric reconstruction (arterial phase) showing a pseudoaneurysm/contrast extravasation in a branch of the right hepatic artery (arrow). **D:** Arteriography with embolization and total exclusion of the pseudoaneurysm and of the arterial rupture, the final result being angiographic success.

the common biliary duct, and intrahepatic cavities that may require surgical debridement[3].

For cases of severe gastrointestinal bleeding in which there is a risk of death, the diagnostic procedure of choice is hepatic angiography, because it allows selective embolization of the appropriate vascular branches, preserving maximum liver parenchyma function. Transcatheter arterial embolization is used as an isolated form of treatment or as a way to keep the patient hemodynamically stable for definitive surgery, minimizing morbidity and mortality[6,7].

Currently, embolization of the hepatic artery is the gold standard treatment, due to its 80–100% success rate in controlling hemorrhage and its low rates of morbidity and mortality[2,8]. However, there are reports of fatal hepatic necrosis and the formation of intrahepatic abscess following embolization[8]. Technical failures may occur in cases of anomalous origin of the hepatic artery, previous surgery, vascular tortuosity, or previous ligation of the proximal vessel. In the case presented here, the patient underwent selective transcatheter embolization and remained in outpatient follow-up, with no abdominal pain and with resolution of the hemobilia.

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