oligodendroglioma, and astrocytoma with calcifications[3,12,13]. Due to the increasing number of cases described in recent years, CAPNON should be included in the differential diagnosis of calcified CNS lesions.

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Hepatic changes after treatment with oxaliplatin

Dear Editor,

A 54-year-old female with colon cancer was treated surgically and with a chemotherapy regimen (fluorouracil, leucovorin, and oxaliplatin). Pre-treatment imaging examinations had shown that her liver had a normal aspect, with no evidence of focal lesions. Follow-up (post-treatment) examinations showed mild splenomegaly and slightly lobulated liver contours. Some liver nodules also appeared, most of them characterized by an isointense signal in T1- and T2-weighted sequences with enhancement in the arterial phase and persistent uptake in the hepatobiliary phase after intravenous injection of a hepatobiliary-specific contrast agent, suggesting focal nodular hyperplasia (FNH)-like lesions (Figure 1).

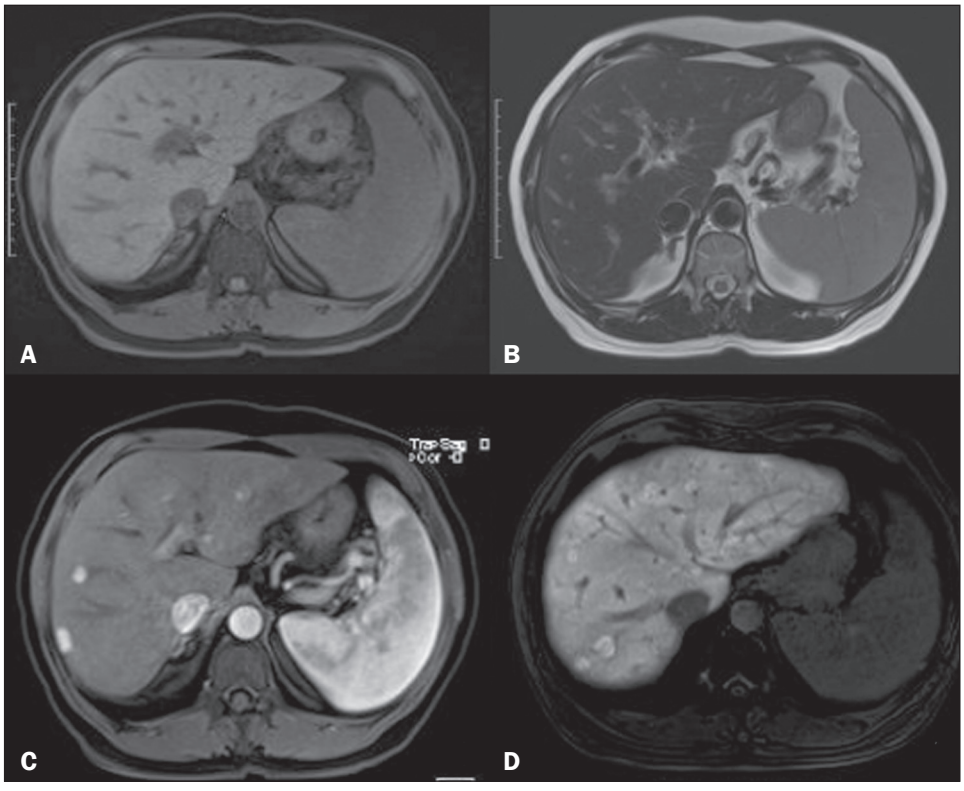

Figure 1. Magnetic resonance imaging. Unenhanced, fat-suppressed T1-weighted sequence (**A**), unenhanced T2-weighted sequence (**B**), and contrast-enhanced T1-weighted sequences in the arterial and hepatobiliary phases (**C** and **D**, respectively), showing mild splenomegaly and hepatic nodules with isointense signals on T1- and T2-weighted sequences, arterial enhancement, and persistent enhancement in the hepatobiliary phase, suggesting FNH-like lesions. Other areas containing nodules and showing contrast uptake in the hepatobiliary phase can be seen, also suggesting benignity.

Colorectal carcinoma is the third leading cancer worldwide, and hepatic metastasis occurs in 40–50% of cases. Various chemotherapy regimens are used in order to treat this type of neoplasia, several of them including oxaliplatin[1].

Various classes of chemotherapeutic agents have been associated with hepatic lesions such as steatosis, steatohepatitis, sinusoidal obstruction syndrome, acute hepatitis, and liver necrosis[2]. Oxaliplatin-based treatments are most often associated with sinusoidal obstruction syndrome and regenerative nodular hyperplasia[2,3].

Sinusoidal obstruction syndrome, previously known as hepatic veno-occlusive disease, is caused by deposits of fibrous material into the small branches of the hepatic veins, causing obstruction and sinusoidal dilatation, resulting in congestion, perisinusoidal fibrosis, and hepatocellular lesion. The condition can lead to hepatosplenomegaly and portal hypertension.

Regenerative nodular hyperplasia typically manifests as relatively small lesions, diagnosed in pathological studies, although its pathogenesis has yet to be well established. It is believed to be related to intrahepatic vascular disorders that promote areas of hypoperfusion (atrophic areas) adjacent to areas of hyperperfusion (regenerative areas) and are regarded by some authors as a final stage of vascular lesion induced by chemotherapeutic agents[3,4].

Macroscopic nodules with radiological and pathological characteristics identical to those of FNH-like lesions have been related to various hepatic conditions[5] such as cirrhosis[6], vascular changes such as Budd–Chiari syndrome[6], and, more recently, the use of oxaliplatin-based chemotherapy regimens[7,8], as in the case reported here.

Focal hepatic lesions that appear in imaging examinations during the follow-up of cancer patients oblige us to make a careful analysis because of the possibility of a secondary neoplasm. Metastases of colon carcinomas are usually poorly vascularized and show low uptake in the hepatobiliary phase.

Knowing the histological type of the primary neoplasm, as well as the radiological pattern of metastasis of chemotherapy-induced lesions, is essential for the correct diagnosis and appropriate clinical guidance in cases of hepatic changes after treatment with oxaliplatin.

Understanding the hepatic changes related to chemotherapy, especially the possibility that FNH-like hypervascular lesions will occur after treatment with oxaliplatin, can facilitate the diagnosis of chemotherapy-induced lesions and prevent unnecessary invasive procedures in patient follow-up.

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Monostotic fibrous dysplasia invading the inferior turbinate: computed tomography and magnetic resonance imaging findings

Dear Editor

A 25-year-old woman presented to our hospital with a headache and nasal congestion. Although she reported a one-year history of nasal congestion, she had no chronic diseases. On local examination, the inferior turbinate was enlarged and hard. Systemic cutaneous examination revealed no pathology. Magnetic resonance imaging (MRI), performed to investigate the headache, showed a 3 × 1.5 × 1.0 cm lesion in the right inferior turbinate (Figure 1). On T1- and T2-weighted images, the lesion was hypointense with well-defined margins. Computed tomography (CT) of the paranasal sinus showed that the turbinate was enlarged, and that the mass causing the expansion was homogeneous and hyperdense (Figure 2). No soft tissue invasion or bone destruction was seen on CT or MRI. The patient was treated with surgical excision and was discharged without complications.

Fibrous dysplasia (FD) is a benign skeletal disorder characterized by fibroblastic proliferation. Although factors such as genetics and trauma have been implicated, the etiology remains uncertain. Involvement of the facial bones is rare, and when present, the maxilla and mandible are commonly affected[1]. Inferior turbinate involvement has previously been reported in only a few cases.

In cases of FD with craniofacial involvement, the clinical findings include facial asymmetry, nasal obstruction, and pain. Involvement of the inferior turbinate has been reported in only a few cases. Karligkiotis et al.[2] reported the first such case in 2012, describing a 6-month history of persistent nasal obstruction in a 68-year-old woman who was subsequently treated with corticosteroids. The diagnosis is typically made on the basis of the radiological findings. The radiological findings vary depending on bone matrix development within the lesion and the amount of that matrix. It may assume the form of ground-glass opacity or a radiolucent lytic area[3]. The differential diagnosis includes Paget's disease and ossifying fibroma.