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.

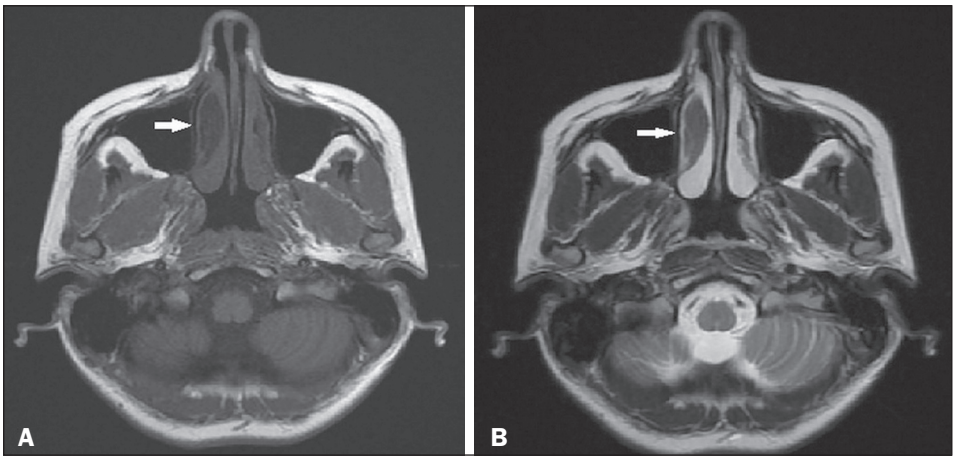

Figure 1. Axial T1- and T2-weighted MRI sequences (**A** and **B**, respectively), showing a hypointense lesion (arrow) consistent with FD.

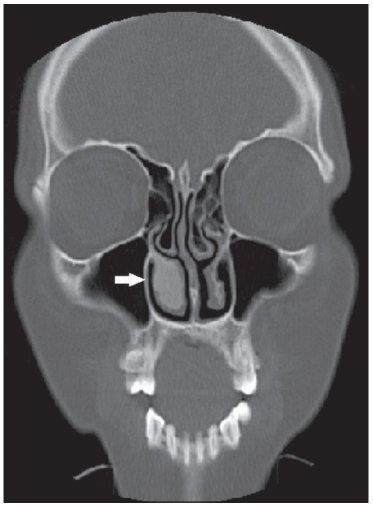

Figure 2. CT of the paranasal sinus showing a ground-glass opacity causing expansion of the right inferior turbinate (arrow).

Three different CT imaging patterns have been reported in FD[2,4]: ground-glass (the most common and characteristic); sclerotic; and lytic. MRI is used for diagnostic purposes or to show relationships with the adjacent anatomical structures. On T1- and T2-weighted images, the lesion is sharply marginated and hypointense. However, the diagnostic efficacy of MRI decreases when the signal is hyperintense on T2-weighted images in particular[3].

Lysis developing in the area of an FD lesion, calcified foci, a periosteal reaction contiguous to the lesion, a cortical defect, and a soft-tissue mass observed at radiology indicate malignant transformation. In monostotic FD, malignant transformation is more common in individuals with craniofacial involvement than in those with involvement of other bones. The most common malignant transformation of FD is to osteosarcoma[4]. In the case presented here, homogeneous ground-glass opacity, which is typical of FD, was seen on CT. The preliminary diagnosis of FD, based on the radiological findings, was confirmed by histopathology.

In patients with FD, small, asymptomatic lesions not causing any cosmetic deformity can be monitored. Patients in pain can be treated with bisphosphonates, vitamin B, or calcitonin[3]. Surgical treatment, if required, involves radical excision and reconstruction[3,5].

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Disseminated intramuscular cysticercosis diagnosed incidentally in a patient with joint pain

Dear Editor,

A 43-year-old male resident of a rural area presented with a two-month history of pain in his right leg, predominantly in the hip region, the pain having progressively increased in intensity. Physical examination showed mild pain during active and passive movement of the limb. Laboratory tests showed no significant changes, except for a slightly elevated erythrocyte sedimentation rate. A plain X-ray demonstrated multiple radiopaque rhizoid images, distributed throughout various muscle planes (Figures 1A and 1B). Computed tomography revealed numerous calcifications in the muscle groups of both legs (Figures 1C and 1D). The diagnostic hypothesis was muscular cysticercosis. Pathological analysis of a muscle tissue sample confirmed that diagnosis. The patient was started on a 30-day course of albendazole and prednisone, which resulted in improvement of all of the signs and symptoms.

Diseases caused by uncommon infectious agents have recently been described in the radiology literature of Brazil[1–4]. Taeniasis and cysticercosis are two distinct entities. Although cestodes of the same genus are responsible for both diseases, the stage of the cestode at infection differs between the two. Taeniasis is caused by the adult forms of *Taenia solium* or *Taenia saginata* infesting the small intestine of its definitive host, human beings. However, cysticercosis is caused by the larval form of tapeworms infesting the tissues of its intermediate hosts (pigs and cows). In human cysticercosis, humans are categorized as accidental intermediate hosts[5]. The eggs enter the human intestine by fecal–oral contamination, by autoinfection, or by ingestion of contaminated food or water[6]. Eggs develop into