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Giant pedunculated hemangioma of the liver

Hemangioma hepático gigante pedunculado

Dear Editor,

A previously healthy, 28-year-old woman presenting a palpable mass in the right hypochondrium for 3 years, evolving with local discomfort over the last 20 days. Ultrasonography (US) demonstrated an expansile mass best characterized by computed tomography (CT) and magnetic resonance imaging (MRI) which showed a well defined solid mass in continuity with the liver by a thin pedicle originating from the segment V and caudally extending towards the pelvis, measuring $18.0 \times 9.4 \times 5.2$ cm, with features and pattern of enhancement suggestive of hemangioma (Figures 1A and 1B). Surgical resection was the treatment of choice because of the patient's symptoms and the risks of torsion. The anatomopathological analysis confirmed the diagnosis (Figure 1C).

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Gabriela Maria Ribeiro e Ribeiro¹, Marcelo Ricardo Canuto Natal¹, Eduardo Felipe Silva¹, Sabrina Cardoso Freitas¹, Waldete Cabral Moraes¹, Fernanda Cunha Maciel¹

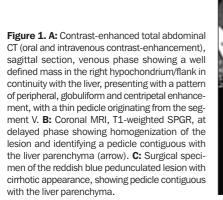
1. Hospital de Base do Distrito Federal (HBDF), Brasília, DF, Brazil. Mailing Address: Dra. Gabriela Maria Ribeiro e Ribeiro. Rua Gomes de Carvalho, 1005, ap. 3110, Vila Olímpia. São Paulo, SP, Brazil, 04547-004. E-mail: gabiribeiroeribeiro@yahoo.com.br.

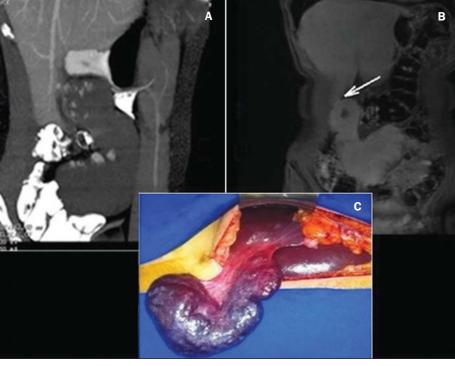
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Hemangioma is the most common benign liver tumor⁽¹⁻⁸⁾, with a prevalence of 0.4–20% in necropsies^(1,5-8). In most cases, hemangiomas are small, asymptomatic and incidentally found at imaging studies^(1,2,5,6).

In spite of the lack of consensus about the dimensions to define a giant hemangioma, ranging from 4 to 10 cm according to the literature, it is known that the exophytic presentation, particularly those pedunculated, are very rare $^{(1-3,5,6)}$. The first case was reported by Ellis et al. in 1985; and up to 2013, only 24 cases were described in the literature $^{(1,4)}$.

In almost 50% of cases, pedunculated hemangiomas are symptomatic at the diagnosis⁽¹⁾ and, likely any giant lesion, may determine compression of the intrahepatic biliary ducts, vascular structures or adjacent organs, manifesting with pain, early satiety, hemorrhage, jaundice, nausea and vomiting^(1,2,5,6,8). Main complications include torsion due to a long and mobile pedicle,





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infarction^(5,6), spontaneous or traumatic rupture, congestive heart failure, and Kasabach-Merritt syndrome^(2,6,7).

A correct diagnosis of the pedunculated lesion may be difficult, despite the typical radiological presentation, because of the limitation in define the origin of the mass, since a thin pedicle may be almost undetectable at images^(1,4,5).

The most used modalities of imaging in diagnosis include US, CT and MRI^(1-4,6,8). At US, the image is typically hyperechoic, homogeneous, with well defined margins; and, in cases of giant lesions, central heterogeneity may be present⁽⁸⁾. At CT, with a certain frequency, giant hemangiomas do not present with the typical pattern of hypoattenuating lesion with centripetal enhancement and homogenization at delayed sections, due to the presence of avascular areas of necrosis, fibrosis or hemorrhage^(3,8). MRI is the most sensitive and specific (> 90%) diagnostic method^(4,6). The lesions are well defined, homogeneous, with low signal intensity at T1-weighted sequences, and high signal intensity at T2-weighted sequences.

Biopsy is not recommended in such cases, due to the risk of hemorrhage $^{\rm (6)}.$

There are reports in the literature describing pedunculated hemangiomas as gastric, adrenal tumor^(1,4), retroperitoneal mass⁽¹⁾, other pedunculated liver tumors such as hepatocellular carcinoma, mesenchymal hamartoma, focal nodular hyperplasia or adenoma⁽⁴⁾.

Surgical treatment is reserved for cases of giant or symptomatic lesions, uncertain diagnosis, lesions with complications^(1,2,4-7), and for cases of pedunculated hemangiomas due to their tendency to torsion^(5,6).

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Paula de Castro Menezes Candido¹, Izabela Machado Flores Pereira¹, Breno Assunção Matos¹, Mario Henrique Giordano Fontes¹, Teófilo Eduardo de Abreu Pires¹, Petrônio Rabelo Costa¹

1. Hospital Felício Rocho, Belo Horizonte, MG, Brazil. Mailing Address: Dra. Paula de Castro Menezes Candido. Hospital Felício Rocho – Setor de Radiología. Avenida do Contorno, 9530, Barro Preto. Belo Horizonte, MG, Brazil, 30110-934. E-mail: paulacmcandido@yahoo.com.br.

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Posterior reversible encephalopathy syndrome following immunoglobulin therapy in a patient with Miller-Fisher syndrome

Síndrome encefálica reversível posterior em paciente com síndrome de Miller-Fisher pós-tratamento com imunoglobulina

Dear Editor,

A 54-year-old female patient presenting with ophthalmoparesis, ataxia and areflexia for one week. The patient denied fever, muscle weakness, and did not report any previous comorbidity. At physical examination, she was normotensive, oriented, with bilateral flexor cutaneous-plantar reflex and preserved superficial/deep sensitivity. Human immunodeficiency virus, Epstein-Barr virus, cytomegalovirus, HTLV-1 and VDRL serologies were negative. Considering such findings, the hypothesis of Miller-Fisher syndrome was raised, and liquor cerebrospinalis analysis demonstrated hyperproteinorachia, confirming the diagnosis.

Within 24–48 hours after immunoglobulin therapy initiation, the patient presented with intense headache followed by tonic-clonic seizures and later decreased level of consciousness, with no association with hypertensive peaks. Magnetic resonance imaging (MRI) (Figure 1A,B,C) showed sparse hyperintense areas in the white substance, bilaterally on T2-weighted and FLAIR sequences, predominantly in the parieto-occipital regions, without diffusion restriction and without gadolinium enhancement, demonstrating an imaging pattern suggestive of posterior reversible encephalopathy syndrome (PRES). After the therapy suspension and adoption of support measures, the patient progressed satisfactorily, with no sequelae and reversion of the MRI findings (Figure 1D).

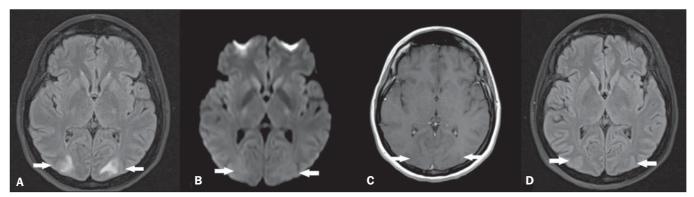


Figure 1. A: Axial MRI FLAIR sequence demonstrating hyperintensity in the occipital lobes white substance bilaterally and symmetrically (arrows). B: Axial diffusionweighted MRI does not demonstrate any alterations (arrows). C: Contrast-enhanced T1-weighted sequence revealing absence of gadolinium-enhanced areas (arrows). D: Axial FLAIR sequence acquired after four weeks demonstrating resolution of the alterations in the occipital lobes white substance (arrows).