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[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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http://dx.doi.org/10.1590/0100-3984.2014.0057

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).

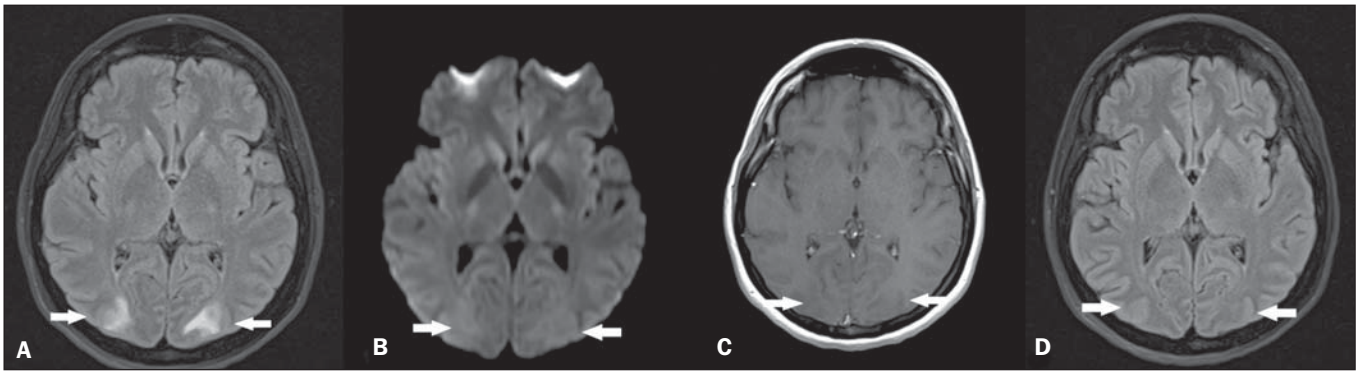

Figure 1. A: Axial MRI FLAIR sequence demonstrating hyperintensity in the occipital lobes white substance bilaterally and symmetrically (arrows). **B:** Axial diffusion-weighted MRI does not demonstrate any alterations (arrows). **C:** Contrast-enhanced T1-weighted sequence revealing absence of gadolinium-enhanced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

The Brazilian radiological literature has recently highlighted the relevant role played by MRI in the improvement of the diagnosis of central nervous system conditions[1–5].

PRES is a clinical-radiological entity of varied etiology, generally occurring in the setting of severe arterial hypertension. In some cases, however, it may be associated with immunosuppressive therapy, and is rarely described in the literature after the use of immunoglobulin[6–12]. Its physiopathogenesis is characterized by the presence of endothelial lesion and dysfunction of cerebral autoregulation mechanisms, leading to hypoperfusion and vasogenic edema[7–12]. The clinical manifestations present acute/subacute onset characterized by headache, decreased level of consciousness, visual alterations, tonic-clonic seizures and focal neurological signs. The symptoms are progressive. Complete regression is achieved provided the syndrome is appropriately treated; otherwise irreversible damages may occur[6–11].

MRI findings are quite suggestive and characterized by hyperintense areas on T2-weighted and FLAIR sequences, in general affecting the white substance bilaterally and symmetrically, with predilection for the parieto-occipital region. It may also affect the frontal lobes, internal and external capsules, cerebellum and encephalic trunk[7–9]. At early stages of the condition, diffusion MRI does not demonstrate any abnormalities, but inappropriate management may result in irreversible damages presented as diffusion restriction corresponding to cytotoxic edema.

Recent studies by means of retrospective analysis, utilizing MRI and laboratory data, have demonstrated the association between PRES and albumin serum levels. There are evidences that significantly decreased albumin serum levels lead to a higher risk to develop vasogenic-type edema[12]. This is due to the fact that, in conditions with endothelial damages caused by inflammatory processes, the decrease in the colloidosmotic pressure, directly related to the albumin levels, may facilitate the development of vasogenic edema. Thus, the early administration of human serum albumin might prevent ischemic damages and reduce possible sequelae[12].

Finally, despite being rare after administration of immunoglobulin, PRES should be considered in cases where typical MRI findings are present. One should not wait until the onset of a hypertensive episode to take such a diagnostic possibility into consideration.

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http://dx.doi.org/10.1590/0100-3984.2015.0129

Pulmonary paracoccidioidomycosis showing reversed halo sign with nodular/coarse contour

Paracoccidioidomicose pulmonar exibindo sinal do halo invertido com margens nodulares/rugosas

Dear Editor,

A 63-year-old man, living and working in urban area since his childhood, and smoking for 30 years. In 2012 he underwent investigation for chronic cough. At that same time, he reported gingival lesion. For a long time, he had the habit of weekly visiting rural areas for leisure and amateur fishing. The patient denied history of fever, weight loss or comorbidities. Blood counts since 2009 without any abnormalities.

Chest computed tomography (CT) in December 30, 2013 showed focal pulmonary ground glass opacities predominantly in the middle fields, some of them completely or partially surrounded by a thin and coarse consolidation ring representing the "reversed halo sign". Other findings include some areas with subtle interlobular septa thickening (Figures 1A, 1B and 1C).

The gingival lesion, characterized by granular, erythematous ulceration with fine blood-red dots, compatible with a "mulberry-like" appearance, was biopsied.

Biopsy result: eosinophilic epithelial cells of the squamous and spinous layers, giant, "foreign-body" type cells containing isolated and clustered spherical fungi with double and birefringent membranes, in association with inflammatory cells. The cytological diagnosis confirmed the presence of *Paracoccidioides brasiliensis* (Figure 1D).

On February 2, 2015, post-itraconazol therapy chest CT demonstrated rare areas of hypoattenuation associated with fibrocicatricial septal thickening.

Paracoccidioidomycosis is the most common endemic systemic mycosis in the Latin America, caused by infection by inhalation of the dimorphic fungus *Paracoccidioides brasiliensis*[1–7], a pathogen that is found only in Colombia, Argentina, Venezuela and principally subtropical regions in Brazil[1,3,4,6]. The incidence is high in men, rural workers[1–7] aged between 30 and 60 years[1,2,6].