intraventricular gangliogliomas can originate in the lateral ventricles, in the third ventricle, and fourth ventricles—some even originating in the choroid plexus—and should always be included in the differential diagnosis of intraventricular lesions.

The case presented here was one of an intraventricular ganglioglioma apparently originating in the third ventricle, extending to the lateral ventricles and the fourth ventricle, the histopathological diagnosis being WHO grade I ganglioglioma with signs of CSF dissemination during subsequent examinations. In conclusion, a diagnosis of ganglioglioma should be considered in the presence of intraventricular lesions. In addition, imaging of the neuroaxis is recommended, regardless of the histopathological grade of the lesion, because CSF dissemination has been reported in the monitoring of other low-grade tumors, including gangliogliomas[12,13].

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http://dx.doi.org/10.1590/0100-3984.2016.0222

Malformation of the brainstem accompanied by cortical dysplasia

Dear Editor,

We present the case of a 20-year-old woman referred for investigation of epilepsy. A magnetic resonance imaging (MRI) study (Figure 1) showed bilateral areas of focal cortical dysplasia (FCD) along the perisylvian cortex, together with a brainstem malformation characterized by a ventral cleft at the pons-medulla junction. Diffusion tensor imaging (DTI) revealed the absence of transverse pontine fibers and of the medial lemniscus.

Midbrain-hindbrain (MBHB) malformations include a large group of posterior fossa malformations, with different mechanisms and genetic components involved. The clinical findings are nonspecific, varying from hypotonia to seizures and lack of developmental progress[1]. A recent classification of MBHB malformations proposed by Barkovich et al.[2] is based mainly on embryology and genetics[3]. According to that classification system, the ventral cleft seen in our case suggests a regional (group III) developmental defect. Predominantly brainstem malformations may be better evaluated in MRI with three-dimensional, heavily T2-weighted, steady-state sequences, which allow adequate visualization of the cranial nerve in the basal cisterns. DTI of the brainstem may also be helpful and shows promise for further delineating axonal path disorders of the brainstem in the absence of obvious structural defects[1]. Although MBHB malformations can occur in isolation, many of them are accompanied by other malformations, particularly supratentorial malformations, which tend to have a significant effect on the prognosis of these patients. Severe hypoplasia of the pons and medulla with a dorsal cleft and absence of the fascial colliculus can occur in a recently described syndrome—horizontal gaze palsy with progressive scoliosis—which is a rare autosomal recessive disease, characterized by congenital absence of conjugate horizontal eye movements,

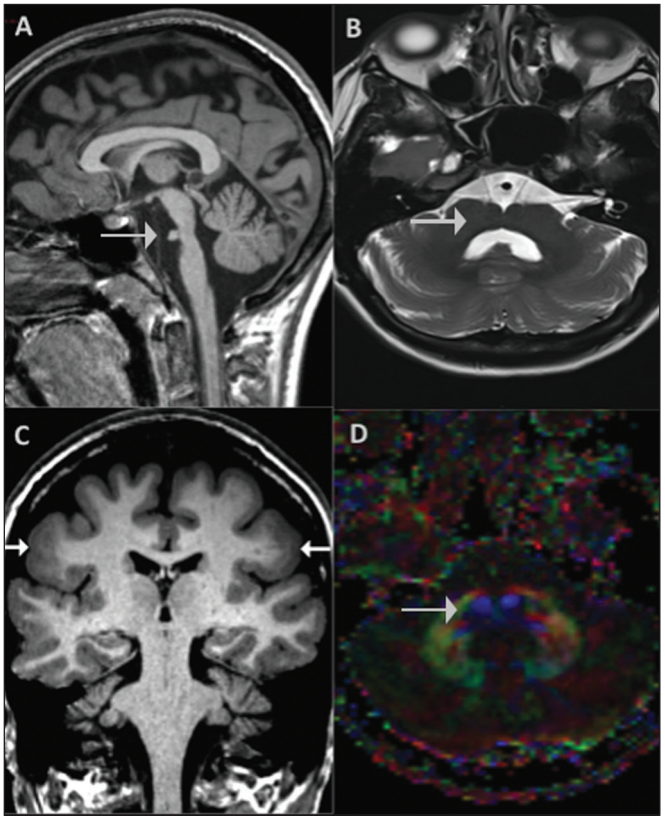

Figure 1. A: Sagittal T1-weighted image depicting a short pons (arrow). **B:** Axial T2-weighted image at the pons-medulla junction showing a ventral cleft (arrow). **C:** Coronal T1-weighted image showing cortical dysplasia (arrows) with a thickened cortex. **D:** Axial fractional anisotropy color map showing the absence of transverse pontine fibers and of the medial lemniscus (arrow).

preservation of vertical gaze, preservation of convergence, and progressive scoliosis, that develops in pediatric patients. The progressive scoliosis is probably secondary to neurological deficits that impair proprioceptive inputs[4].

Our patient had FCD, which is a major cause of epilepsy in children and adults[3]. FCD type II, also known as FCD with the transmantle sign or Taylor-type dysplasia, is classified as a category I malformation of cortical development (MCD), because it involves abnormal neuronal proliferation. The other MCD categories include abnormalities in neuronal migration (category II—e.g., periventricular nodular heterotopia) and abnormal late migration/cortical organization (category III—e.g., FCD type I and polymicrogyria)[5].

In a study of 220 patients with MCD and epilepsy, Kuchukhidze et al.[5] analyzed the combination of MBHB malformations and FCD. The authors identified MBHB malformations in 17% of the patients and found that the malformations were more commonly linked to late migration/cortical organization disorders; only one patient was found to have FCD type II. The cases of MBHB malformations were associated with more extensive MCD lesions, as well as with a poor clinical profile (earlier age at seizure onset, neurologic deficits, learning disability, and developmental delay), although no differences were found in the response to antiepileptic treatment. Nearly 25% of the patients with MBHB malformations had FCD type I, which was not detected in MRI studies and was identified only through pathologic examination of a surgical specimen.

Studies of MBHB malformations have improved with advances in neuroimaging, molecular biology, and molecular genetics, thus increasing understanding of developmental disorders related to such malformations. Functional MRI techniques can also contribute to a better description and understanding of these diseases.

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http://dx.doi.org/10.1590/0100-3984.2016.0216

Pediatric ovarian torsion: a diagnostic challenge

Dear Editor,

A 12-year-old female presented with a 6-h history of acute severe lower abdominal pain in the hypogastrium and left iliac fossa, together with episodes of vomiting. Physical examination revealed a soft abdomen with severe tenderness in the hypogastrium and left iliac fossa. Blood test results were normal. Ultrasound revealed and an enlarged (~ 52 mL) echogenic left ovary (Figure 1A) with free fluid surrounding the ovary and in the pelvic cavity. No cystic or solid lesion was identified within the enlarged ovary. Color Doppler (Figure 1B) revealed no vascularity in the enlarged ovary. The right ovary was normal in size (~ 9 mL). The patient underwent urgent laparoscopy, which revealed an enlarged, congested left ovary (Figure 1C), and left oophorectomy was performed. Histopathology confirmed the diagnosis of ovarian torsion.

Ovarian torsion is the fifth leading gynecological condition requiring emergency surgery[1]. Delayed diagnosis can lead to unsalvageable ovaries and complications like peritonitis. The dilemma in the diagnosis is due to the relative rarity of the condition (incidence, ~ 2–3%), especially in children, as well as to the nonspecificity of the symptoms and the other varied etiologies that take precedence over ovarian torsion in children[2].

Ovarian torsion is defined as the twisting of the ovary on its pedicle, leading to vascular obstruction. Pathophysiologically, the venous outflow is obstructed, resulting in congestion and hemorrhagic infarcts, which in turn result in arterial impairment[3]. It is more common in women of reproductive age, including pregnant women, probably due to the higher incidence of physiological and

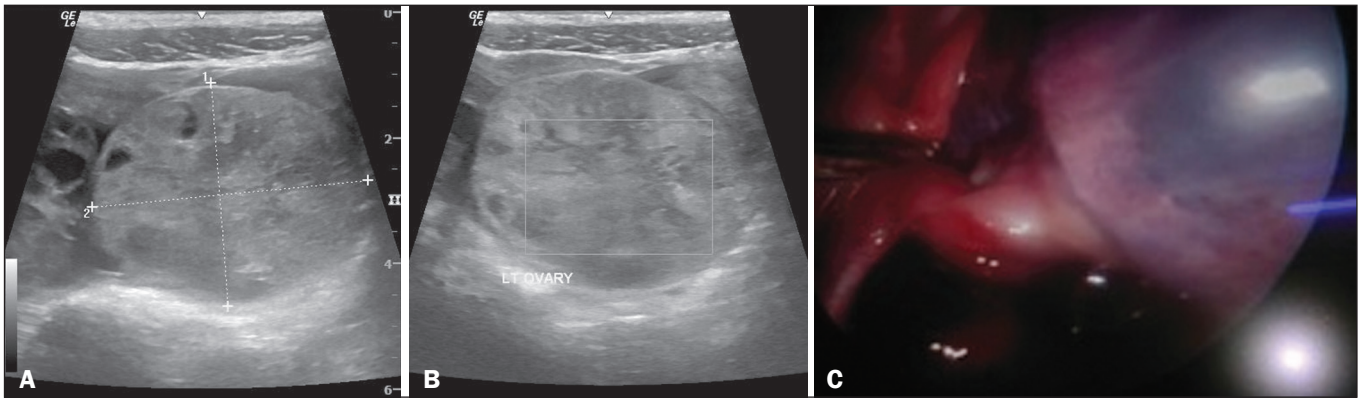

Figure 1. A: Ultrasound showing enlarged echogenic left ovary. **B:** No vascularity seen in the left ovary on color Doppler. **C:** Laparoscopic appearance of the left ovary.