

of conjugate horizontal eye movements, 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.

REFERENCES

1. Doherty D, Millen KJ, Barkovich AJ. Midbrain and hindbrain malformations: advances in clinical diagnosis, imaging, and genetics. *Lancet Neurol.* 2013;12:381–93.
2. Barkovich AJ, Millen KJ, Dobyns WB. A developmental and genetic classification for midbrain-hindbrain malformations. *Brain.* 2009;132(Pt 12):3199–230.
3. Bertholdo D, Carvalho Neto A, Castillo M. Posterior fossa malformation associated with cerebral anomalies: genetic and imaging features. *Top Magn Reson Imaging.* 2011;22:295–302.
4. Rossi A, Catala M, Biancheri R, et al. MR imaging of brain-stem hypoplasia in horizontal gaze palsy with progressive scoliosis. *AJNR Am J Neuroradiol.* 2004;25:1046–8.
5. Kuchukhidze G, Koppelstaetter F, Unterberger I, et al. Midbrain-hindbrain malformations in patients with malformations of cortical development and epilepsy: a series of 220 patients. *Epilepsy Res.* 2013;106:181–90.

Sergio Eiji Ono¹, Débora Brighente Bertholdo¹, Gustavo Rengel dos Santos², Arnolfo de Carvalho Neto²

1. Clínica Diagnóstico Avançado por Imagem – DAPI, Curitiba, PR, Brazil. 2. Hospital de Clínicas da Universidade Federal do Paraná (UFPR), Curitiba, PR, Brazil. Mailing address: Dr. Sergio Eiji Ono. Clínica Diagnóstico Avançado por Imagem – DAPI, Rua Brigadeiro Franco, 122, Mercês. Curitiba, PR, Brazil, 80430-210. E-mail: ono.sergio@gmail.com.

<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 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.

rectomy 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

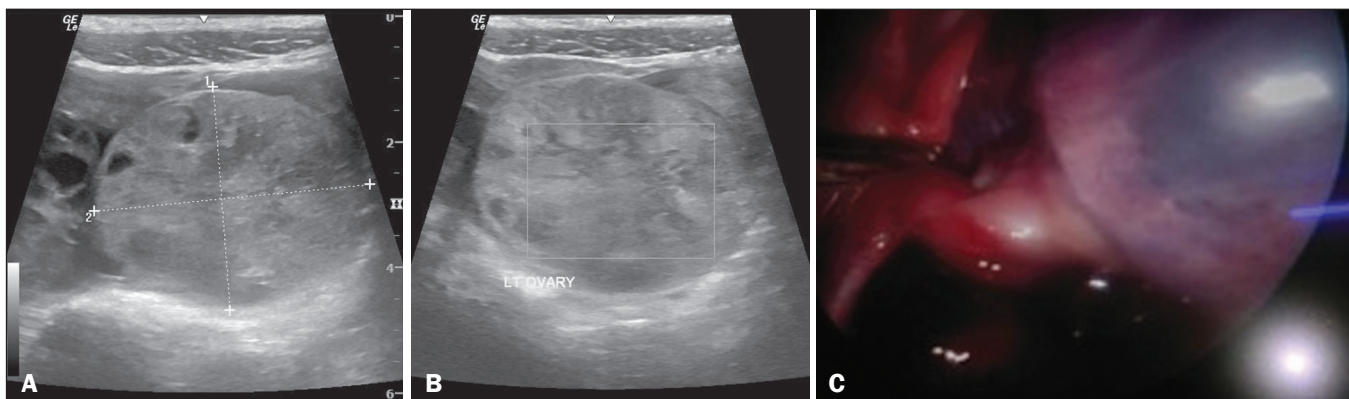


Figure 1. A: Ultrasound showing enlarged echogenic left ovary. B: No vascularity seen in the left ovary on color Doppler. C: Laparoscopic appearance of the left ovary.

of physiological and pathological masses in that age group^(4,5). It is relatively rare in the pediatric population. The symptoms are nonspecific and, due to the rarity of this condition in pediatric patients, etiologies such as appendicitis, diverticulitis, and renal colic are more likely explanations for the clinical symptoms than is ovarian torsion. Ultrasound is the first-line imaging modality in any case of acute abdomen.

One study showed that ultrasound has a positive predictive value of 87.5% and a specificity of 93.3% for the diagnosis of ovarian masses, as well as having other advantages such as low cost, easy accessibility, and no radiation⁽⁶⁾. On gray-scale imaging, the affected ovary appears enlarged, increasing to up to 28 times its original size⁽⁷⁾. The diagnostic criteria for enlarged ovaries include an ovarian diameter of > 4 cm or volume > 20 mL in women of reproductive age and > 10 mL in postmenopausal women^(8,9). Cystic or solid masses can also be identified on ultrasound. Cysts can show wall thickening. Free fluid can be seen in the pelvic cavity. The twisted vascular pedicle is typically seen as an echogenic round or beaked mass with multiple concentric, hypoechoic, target-like stripes. It can also appear as an ellipsoid or tubular mass with internal heterogeneous echoes, depending on the plane of orientation. Although color Doppler typically shows the absence of arterial flow, the presence of arterial flow does not rule out the possibility of torsion, because the arteries are affected at a later stage and there can be arterial supply from the uterine arteries as well. The twisted vascular pedicle can give rise to the whirlpool sign on color Doppler.

REFERENCES

1. Pedrosa I, Zeikus EA, Levine D. MR imaging of acute right lower quadrant pain in pregnant and nonpregnant patients. *Radiographics*. 2007;27:721–43; discussion 743–53.
2. Hibbard LT. Adnexal torsion. *Am J Obstet Gynecol*. 1985;152:456–61.
3. Albayram F, Hamper UM. Ovarian and adnexal torsion: spectrum of sonographic findings with pathologic correlation. *J Ultrasound Med*. 2001;20:1083–9.
4. Chiou SY, Lev-Toaff AS, Masuda E, et al. New clinical and imaging observations by sonography, computed tomography, and magnetic resonance imaging. *J Ultrasound Med*. 2007;26:1289–301.
5. Gorkemli H, Camus M, Clasen K. Adnexal torsion after gonadotrophin ovulation induction for IVF or ICSI and its conservative treatment. *Arch Gynecol Obstet*. 2002;267:4–6.
6. Graif M, Itzhak Y. Sonographic evaluation of ovarian torsion in childhood and adolescence. *AJR Am J Roentgenol*. 1988;150:647–9.
7. Graif M, Shalev J, Strauss S, et al. Torsion of the ovary: sonographic features. *AJR Am J Roentgenol*. 1984;143:1331–4.
8. Chang HC, Bhatt S, Dogra VS. Pearls and pitfalls in diagnosis of ovarian torsion. *Radiographics*. 2008;28:1355–68.
9. Pavlik EJ, DePriest PD, Gallion HH, et al. Ovarian volume related to age. *Gynecol Oncol*. 2000;77:410–2.

Chiyayath Gopalan Muralidharan¹, Shyam Krishna¹, Tony Jose²

1. Command Hospital (SC) Pune, Maharashtra, India. 2. Armed Forces Medical College, Pune, Maharashtra, India. Mailing address: Chiyayath Gopalan Muralidharan, MD, Department of Radiodiagnosis, Command Hospital (SC) Pune, Maharashtra-411040, India. E-mail: murali.cg@rediffmail.com.

<http://dx.doi.org/10.1590/0100-3984.2016.0227>

Asymptomatic apical aneurysm of the left ventricle with intracavitary thrombus: a diagnosis missed by echocardiography

Dear Editor,

We report the case of a 63-year-old male, with a history of acute myocardial infarction (AMI) and angioplasty 10 years prior, who was asymptomatic at presentation. He stated that he had not undergone routine clinical follow-up and was therefore submitted to echocardiography for functional evaluation. Moderate dilation and dysfunction of the left ventricle (LV) were detected, although with limitation in the evaluation of the apex, without information on the presence of an aneurysm or thrombus. Coronary computed tomography angiography (CCTA) was performed in order to identify in-stent restenosis, and the images showed apparent subocclusion distal to the stent in the anterior descending artery (Figure 1A) and a large aneurysm with parietal thinning in the anterior/anteroseptal medial segments, septal/

anterior apical segments, and apex of the LV. It was not possible to detect significant systolic ballooning, because there was a large thrombus lining the intracavitary portion and that was confused with normal wall thickness of the LV. The thrombus had an organized appearance, albeit without signs of calcification, and was markedly hypodense, with a fixed aspect and no contrast enhancement, which had likely made it difficult to identify in the initial (echocardiographic) assessment (Figures 1B and 1C).

Ventricular aneurysm is a serious complication of transmural myocardial infarction (occurring in 5–38% of cases), being the most common mechanical complication, typically evolving to physical limitations and having a negative impact on quality of life^(1–4). It is defined as myocardial ventricular wall thinning and dilation, with distinct margins, leading to akinesia or dyskinesia of one or more myocardial segments during ventricular contraction^(1,2–5). It typically affects the anteroapical region of the LV, because the blood supply of the anterior wall is highly

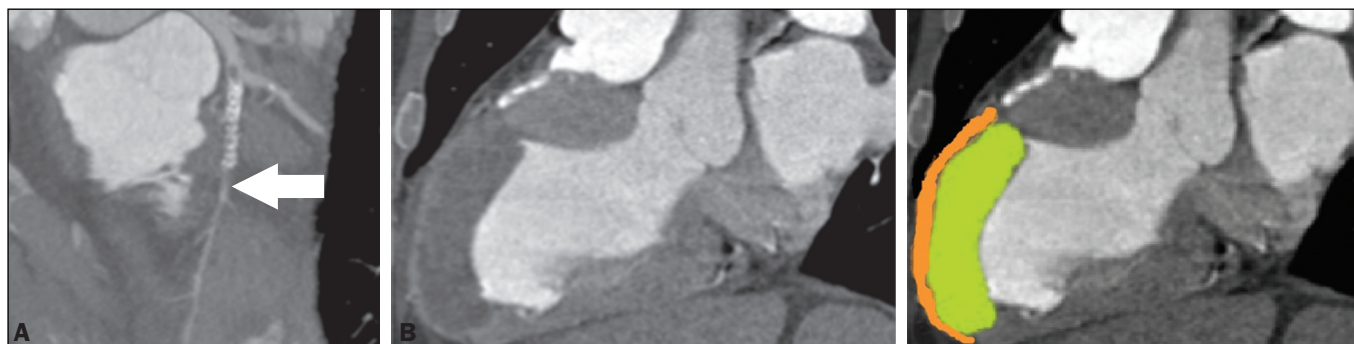


Figure 1. A: CCTA with a reconstruction curve showing probable subocclusion downstream of the stent (arrow). B,C: Cardiac computed tomography of the heart in the longitudinal axial plane, in a pseudo-two-chamber view, showing the region of the LV aneurysm with marked thinning of the medioapical anterior wall (2 mm thick – orange) and normal thickness in the anterior basal segment. Note the large thrombus simulating normal wall thickness of the LV (green).