Ischemic stroke due to occlusion of the artery of Percheron

Sir,

A 60-year-old female presented with acute onset altered sensorium, vertical gaze ophthalmoparesis, side-to-side, and up-and-down 2–4 Hz head tremors that were aggravated on sitting or standing and disappeared on sleeping along with mild memory disturbances. An urgent magnetic resonance imaging (MRI) of the brain demonstrated symmetric bilateral restricted diffusion in paramedian thalami and rostral midbrain on diffusion-weighted MRI [Figures 1 and 2] showing the “V” sign which was consistent with acute infarcts and compatible with occlusion of the artery of Percheron. Magnetic resonance angiography (MRA) showed patent posterior circulation including the tip of the basilar artery and both posterior cerebral arteries.

The thalami and midbrain have a complex blood supply with multiple feeding arteries. The medial parts of the thalami are supplied by the perforating thalamic arteries (also named as paramedian arteries), which arise from the posterior circulation. Percheron delineated four normal variations of the neurovascular anatomy of the thalami and midbrain. In variation II-b, the bilateral perforating thalamic arteries originate from one central artery known as the artery of Percheron, which arises from the P1 segment of one posterior cerebral artery. This artery is a single trunk that provides bilateral arterial supply to the paramedian thalami and the rostral midbrain. Occlusion of this artery leads to bilateral thalamic and mesencephalic infarctions. These infarcts are typically defined by a triad of altered mental status, vertical gaze palsy, and memory deterioration. However, the clinical diagnosis is difficult in majority of the cases because the complex arterial anatomy causes large clinical variability. Our patient presented with all the three typical features of this stroke syndrome along with head tremors. The “V” sign on MRI appears as a well-defined pattern of V-shaped hyperintensity on axial fluid-attenuated inversion recovery and/or diffusion-weighted images along the pial surface of the midbrain adjoining the interpeduncular fossa. Since artery of Percheron is too small to be visualized by computed tomography angiography or MRA, the angiographic studies are frequently normal in these cases.

To conclude, the artery of Percheron is an uncommon entity which originates from the first segment of the posterior cerebral artery and gives rise to bilateral medial thalamic and midbrain perforators. Our patient had artery of Percheron infarct along head tremors which has not been reported before. The etiology of head tremors might be due to red nucleus involvement.

Figure 1: Diffusion-weighted image (a) and corresponding apparent diffusion coefficient map (b) showing V-shaped area of diffusion restriction in midbrain consistent with acute infarcts

Figure 2: Fluid-attenuated inversion recovery-weighted image showing symmetrical hyperintense signal in bilateral paramedian thalami which were also showing diffusion restriction and hence consistent with acute infarction
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Conflicts of interest
There are no conflicts of interest.

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A rare aspect of Crohn's disease: Pulmonary involvement in a child
Sir,
I read with interest the case report by Ongun et al. on the pulmonary involvement in a Turkish child with Crohn's disease (CD). The authors mentioned that "the patient's history of bronchiolitis episodes in the past suspecting of a subclinical pulmonary involvement, the aggravation of respiratory symptoms after colonic surgery which is accepted as a triggering factor, a sudden decline in C‑reactive protein, and dramatic clinical improvement after the initiation of prednisolone appointed us the higher possibility of CD‑related pulmonary involvement." The authors did well in studying deep tracheal aspirates that were evaluated for Mycobacterium tuberculosis species and viral respiratory organisms to rule out infliximab‑related infections (all were negative).

However, they could not differentiate the cause of the inflammatory pulmonary disease by histopathological examination due to the parental disapproval for bronchoscopy. Importantly, the authors did not consider another granulomatous pulmonary lesion in pediatric population that has received ample attention worldwide. I presume that pulmonary sarcoidosis ought to be scrutinized in the case in question by the suitable diagnostic workup. My assumption is based on the following four points.

First, it is obvious that sarcoidosis, a chronic multisystem inflammatory granulomatous disorder of unknown origin, is a rare disease in children and pulmonary involvement is even much rare. Due to the nonspecific clinical features and the lack of a specific test, recognizing sarcoidosis can be difficult in the pediatric population.

Despite that, pediatric pulmonary sarcoidosis is increasingly reported in the literature. In Turkey, the available data pointed out that children with sarcoidosis were noticed to have more frequent pulmonary parenchymal involvement than in adults.

Second, sarcoidosis and concomitant gastrointestinal CD have been reported in patients, as well as the coexistence of CD and sarcoidosis in siblings. Common susceptibility loci have been identified in CD and sarcoidosis.

Third, the studied patient's clinical condition and pulmonary functions improved dramatically after initiation of steroid therapy. This further supports the need to consider concomitant sarcoidosis as steroid represents the main‑stay in the treatment of CD and sarcoidosis.

Fourth, due to the lack of a specific test to precisely diagnose sarcoidosis, a biopsy specimen remains the