Dynamic Contrast-enhanced Magnetic Resonance Imaging in Diagnosis of Cavernous Hemangioma of Cavernous Sinus

Sir,

Cavernous hemangiomas of the cavernous sinus (CHCS) comprise <1% of all parasellar masses and closely mimic tumors such as meningioma, pituitary macroadenoma, or schwannomas at this site.[1] Although magnetic resonance imaging (MRI) can help in diagnosis, addition of dynamic contrast-enhanced sequence to conventional MR sequences demonstrates the characteristic gradual “filling in” of the lesion and helps in preoperative diagnosis with a better accuracy, thus guiding appropriate surgical approach and reducing the associated perioperative morbidity.

A 43-year-old female presented with on and off headache and diminution of vision for 2 months. She had left-sided facial pain for 2 months. X-ray of the skull lateral view revealed widened sella. MRI showed T1 hypointense and T2 hyperintense lesion in the suprasellar and left parasellar location, encasing the left cavernous internal carotid artery (ICA) completely without stenosis. Dynamic postcontrast MRI was performed after giving a bolus dose of 10 ml of gadolinium contrast agent at a rate of 3 ml per second. Dynamic images were acquired in six phases (at TR/TE-560/10) which show progressive filling in of the lesion [Figure 1]. Digital subtraction angiography had also demonstrated tumor blush. Based on characteristic imaging findings, a diagnosis of CHCS was made. Surgery was not preferred in this patient as the lesion was completely encasing left cavernous ICA and in relation with right cavernous sinus. The patient underwent radiotherapy. Follow-up imaging 4 months after radiotherapy showed significant reduction in the size of the lesion [Figure 2]. The patient also improved symptomatically.

Although extra-axial cavernous hemangiomas have the same histologic features as intra-axial lesions, former have different clinical pictures, natural history, and radiologic findings. CHCS present with slowly progressive symptoms due to compression on adjacent cranial nerves and retro-orbital structures. Other symptoms include headache, proptosis, and hypopituitarism. They are frequently encountered in the fourth and fifth decades of life and have a female predominance and tendency to proliferate during pregnancy. Although highly vascular, CHCS do not manifest with acute spontaneous hemorrhage.[2]

Closest differential of CHCS includes suprasellar meningiomas, pituitary macroadenoma, and schwannoma. Meningiomas are usually isointense to gray matter on T1 and T2 sequences and show intense homogeneous postcontrast enhancement. A dural tail is frequently seen extending away from the edge of the tumor. Meningiomas narrow the lumen of adjacent ICA. Pituitary macroadenomas are heterogeneous lesions usually hypo- to iso-intense on T1 and mildly hyperintense on T2. Pituitary adenomas usually do not narrow the lumen of ICA. Schwannomas are isointense to hypointense masses on T1 images, mostly T2 hyperintense, and show heterogeneous contrast enhancement. A clue to the diagnosis is that they follow the expected course of the nerves from which they arise.[3]

CHCS grow as asymmetrical dumbbell-shaped masses occupying the sella, suprasellar, and parasellar region causing mass effect and encasement of neurovascular structure. Characteristic finding is encasement of vessels without occlusion. These lesions are isointense to hypointense on T1-weighted image (T1-WI) and markedly hyperintense on T2-WI. These highly vascular lesions show intense homogeneous postcontrast enhancement.[4] This intense enhancement is attributed to large amount of thin-walled vascular sinusoids with varying amount of intervening connective tissue according to the type of CHCS. Actually, in early phases, heterogeneous enhancement is noted, then it gradually become homogeneous as reported by Jinhu et al. in their sequential axial, coronal, and sagittal
Our dynamic contrast-enhanced sequence demonstrates the characteristic gradual filling in of the lesion on successive phases. Progressive contrast “filling in” in the parasellar and suprasellar lesion on dynamic contrast-enhanced MR images can suggest the preoperative diagnosis of CHCS and reduce operative morbidity as these lesions have high propensity to bleed during surgery. Alternative treatment modalities include gamma knife radiosurgery and radiotherapy.

Progressive contrast filling in a T2 brightly hyperintense and T1 hypointense parasellar mass lesion involving the cavernous sinus is highly suggestive of CHCS.

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There are no conflicts of interest.

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Case 1: A 42-year-old male presented with a history of gait unsteadiness of 2-day duration. He used to sway on to his left side while walking. Gait unsteadiness was associated with blurring of vision on looking near objects. There was no upper limb incoordination or speech difficulty. He did not have headache, vomiting, or sensory disturbances. He was hypertensive on medications with good glycemic control. He had marfanoid habitus. Systemic examination was not remarkable. Speech was unremarkable. His pulse rate was 86/min and blood pressure was 132/86 mmHg. He was conscious and oriented. He had paramedian midbrain. MR angiography of intracranial vessels showed thinning of the right posterior cerebral arteries showed thinning of the right posterior cerebral artery (PCA). Carotid and vertebral arteries Doppler were normal. Two-dimensional (2D) transthoracic echocardiography showed mild aortic regurgitation. Serum complete hemogram, renal, hepatic, and thyroid functions were normal. Glycated hemoglobin was 7.6%. Brain MRI and CT scans showed a hypointense lesion in the right anteromedial thalamus and right rostral ventral thalamus. MR angiography of intracranial arteries showed thinning of the right posterior cerebral artery. The resulting thalamo-subthalamic paramedian artery. The resulting paramedian thalamic infarction can also present as vertical gaze abnormalities that are reported due to damage to the descending fibers controlling the vertical gaze. Damage causes vertical gaze abnormalities as they interrupt the midbrain, are the brain stem centers for vertical eye movement.

Vertical vestibulo-ocular reflex was preserved. Convergence exercises were normal, but left knee-heel finger–nose coordination was normal, but left knee–heel plantar responses were flexor. Vertical conjugate supranuclear vertical gaze palsy due to infarct in the thalamo-mesencephalic junction and isolated medial paramedian thalamus. Hereby, we describe three patients who presented with unique vertical gaze disorder in the form of acute bilateral one-and-a-half syndrome, coexistence of vertical and horizontal one-and-a-half syndrome, contralesional monocular elevation syndrome, and vertical half-and-a-half syndrome. One-and-a-one Syndrome – Report of Three Cases

REFERENCES

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