Article published online: 2022-09-20

LETTER TO EDITOR

Revealing the hidden: Delight of susceptibility weighted imaging for neurosurgeons!

Sir,

We encountered an interesting and quite an eye-opener "imaging wonder" helping in imageological diagnosis in a patient. A 38-year-old male patient presented with sudden onset severe headache with weakness of the left upper limb for 5 days. Rest of the neurological examination was normal. Computed tomography scan revealed left posterior frontal parenchymal bleed [Figure 1]. Magnetic resonance imaging (MRI) of brain confirmed the bleed being hypointense in the center surrounded by peripheral hyperintensity on T1- and T2-weighted imaging [Figure 2] along with blooming on gradient echo (GRE). Rest of the brain was normal. After explaining the options of close observation versus biopsy, patient was taken up for excisional biopsy. Right frontoparietal craniotomy was done and the lesion was excised along with surrounding clot via trans-sulcal approach. The lesion had a thick capsule with a clear gliotic plane all around. To our surprise, the histopathology was inconclusive. Patient had an uneventful recovery from the weakness and was under regular follow-up for 2 months when he presented again with sudden onset of severe headache, right hemiparesis, aphasia and altered sensorium. This time the imaging revealed a 4 cm × 4 cm "*mirror bleed*" in the left temporoparietal area with a significant mass effect [Figure 3]. Patient underwent emergency left fronto temporoparietal craniotomy and excision of lesion along with clot evacuation was performed. Histopathological examination confirmed it as cavernous hemangioma. We also performed susceptibility weighted imaging (SWI) on our patient after the second surgery [Figure 4] and as many as eight new lesions were detected, which were missed in routine MRI and could have bled in future. Screening of his parents, siblings and children with SWI was negative for familial cavernomas, although genetic analysis could not be performed.

Cavernous malformations or cavernomas constitute 5-13% of all central nervous system malformations, which may be single/multiple, familial/sporadic and congenital/ denovo in origin. They are dynamic lesions that grow and produce symptoms by repeated episodes of hemorrhage manifesting as micro or macro-bleeds. Prospective annual risk of hemorrhage per lesion per year is around 2.4% (ranging from 1.6% to 3.1%) and hence making the pre-operative identification of these lesions is essential and imperative.[1] MRI forms the quintessential diagnostic investigation of choice as it can pick up lesions depending on the presence and state of hemorrhage within. T2-weighted GRE can better identify the hemosiderin laden tissue than conventional spin echo and fast spin echo T2-weighted sequences.[2] Zabramski *et al*. have classified the lesions into four types based on imaging findings and have predicted the risk of hemorrhage in them.[3] However as traditionally called angiographically occult, sometimes they may remain completely undiagnosed on routine MRI/GRE sequences making patients highly susceptible for re-bleeds. Micro-bleeds may still remain occult in the absence of considerable mass effect or seizures, but macro-bleeds like in our patient could be life-threatening and therefore neurosurgeons would

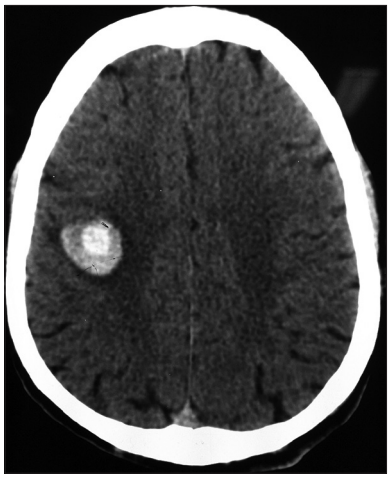

Figure 1: Computed tomography scan brain plain showing hyperdense, well-defined lesion in the right posterior frontal region

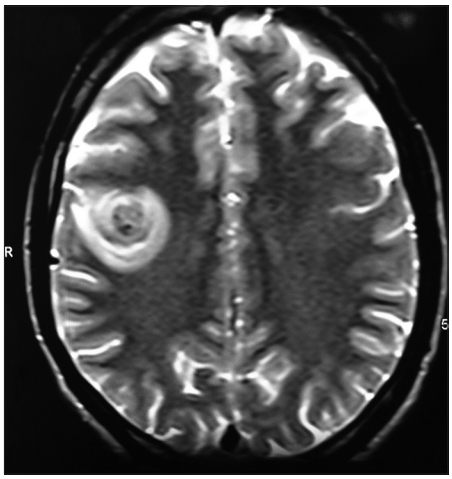

Figure 2: Magnetic resonance imaging brain T2-weighted image axial section showing the well-defined lesion with hypointense center and with peripheral hyperintensity in the right posterior frontal region

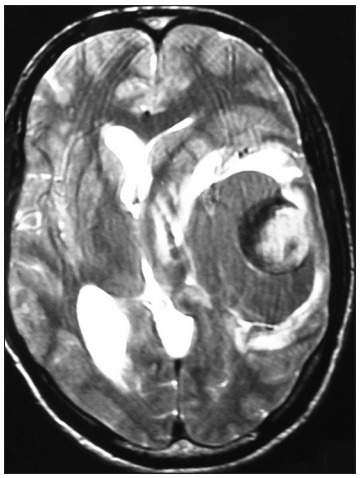

Figure 3: Magnetic resonance imaging brain T2-weighted axial image showing a large lesion with varying intensities in the left posterior frontal region with mass effect and midline shift. The biopsy of the lesion was confirmed as cavernoma

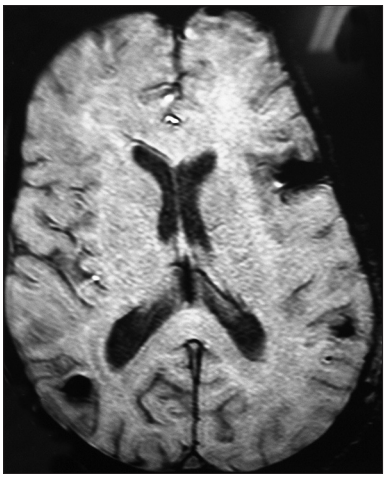

Figure 4: Magnetic resonance imaging brain susceptibility weighted imaging axial sections showing numerous other lesions on either side

like to have a diagnostic modality even more sensitive than the routine GRE sequence. SWI is one such modality, which has emerged in the neurovascular panorama off-late. SWI is assembled from both magnitude and phase images from a high-resolution, 3D velocity compensated GRE sequence.[4] SWI exploits different relaxation rates between venous and arterial blood as well as the phase changes caused by the susceptibility differences between the oxygenated and deoxygenated hemoglobin. Therefore, this technique is exquisitely sensitive to small differences and enhances the signal intensity loss in the venous circulation. SWI has been found useful in ischemic stroke,[5] spontaneous intracerebral bleeds, cerebral amyloid angiopathy, trauma[6] and sepsis.[7] Mittal *et al*. have also described about the role of SWI in detecting penumbra similar to diffusion-perfusion imaging performed routinely for cerebral stroke.[8] Furthermore, SWI has been found more sensitive than gradient-recalled echo T2* in detecting size, number, volume and distribution of hemorrhagic lesions.[4]

In the present case, T2 GRE sequence could not identify another lesion and we thought that we were dealing with a solitary cavernoma. This highlights the importance of getting SWI as a routine in detecting multiple occult lesions if any, even in sporadic and solitary cavernomas because it will help neurosurgeons prognosticate the disease in a better way and will also guide them in planning any possible surgical intervention for progressively growing and non-eloquent lesions in the future if required.

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Quick Response Code:	Website: www.asianjns.org
	DOI: 10.4103/1793-5482.145326