Short Communication

"Venous congestion" as a cause of subcortical white matter T2 hypointensity on magnetic resonance images

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Abstract

Subcortical T2 hypointensity is an uncommon finding seen in very limited conditions such as multiple sclerosis, Sturge-Weber syndrome, and meningitis. Some of the conditions such as moyamoya disease, severe ischemic-anoxic insults, early cortical ischemia, and infarcts are of "arterial origin." We describe two conditions in which "venous congestion" plays a major role in T2 hypointensity — cerebral venous sinus thrombosis (CVST) and dural arteriovenous fistula (dAVF). The third case is a case of meningitis, showing T2 hypointensity as well, and can be explained by the "venous congestion" hypothesis. The same hypothesis can explain few of the other conditions causing subcortical T2 hypointensity.

Key Words

Cerebral venous sinus thrombosis (CVST), dural arteriovenous fistula (dAVF), magnetic resonance imaging (MRI), meningitis, venous congestion

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Introduction

Most cerebral pathologies cause T2 hyperintensity, while few conditions cause T2 hypointensity. In certain conditions such as nonketotic hyperglycinemia,[1] meningitis, encephalitis, leptomeningeal metastasis, the cause of subcortical hypointensity is not known. In some conditions such as moyamoya and infarcts, the T2 hypointensity is attributed to cerebral ischemia leading to accumulation of free radicals/ nonheme iron. Our case series adds further to the list of etiologies of subcortical T2 hypointensity. We hypothesize "venous congestion" as a cause of some of these findings as well.

Case Reports

Case 1

57-year-old male presented with one episode of a sudden transient loss of consciousness. Additionally, he gave a history of severe headache of a short duration and one episode of generalized tonic-clonic seizures. He underwent magnetic resonance imaging (MRI) that revealed right frontal sulcal subarachnoid hemorrhage (SAH) [Figure 1a-c], with associated subcortical white matter T2/ fluid attenuated inversion recovery (FLAIR) hypointensity. Gradient recalled echo (GRE) imaging showed thrombosed superficial cortical veins [Figure 1d] and GRE blooming within the sulci corresponding to SAH [Figure 1e]. Magnetic resonance (MR) venogram showed occlusion of the anterior 1/3rd of the superior sagittal sinus [Figure 1f]. His blood investigations revealed polycythemia. He was treated with anticoagulants and venesection, after which he became symptom-free.

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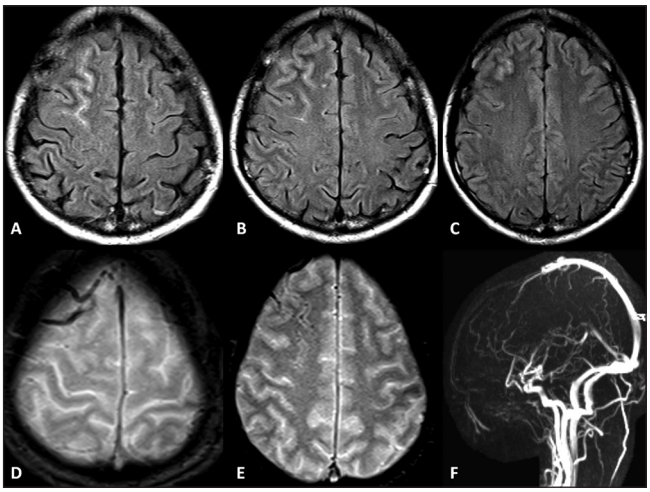

Figure 1: (a-c) Axial FLAIR MR image showing focal subcortical T2 hypointensity in the right frontal lobe, FLAIR hyperintensity in corresponding sulci (d and e) GRE images showing blooming in superficial cortical veins and within sulci (f) MR venogram showing occlusion of the anterior 1/3rd of the superficial sagittal sinus

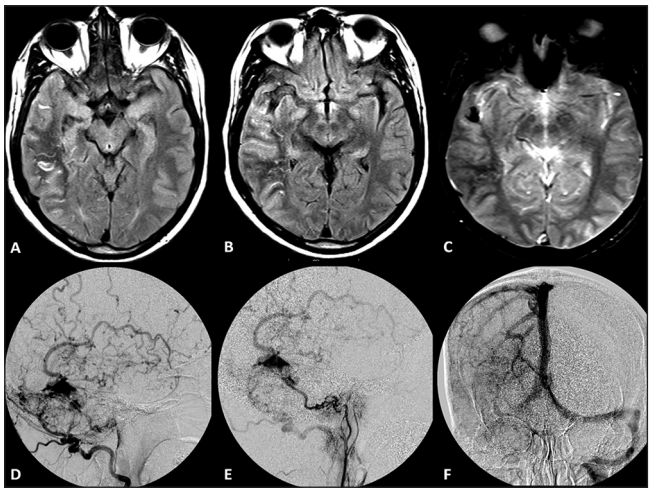

Figure 2: (a and b) Axial FLAIR MR images showing subcortical white matter T2 hypointensity in right parietotemporal lobe (c) Corresponding GRE image shows subtle blooming (d and e) Right ECA angiogram showing right transverse dAVF with venous reflux into superficial middle cerebral vein (f) Right proximal and distal transverse sinus occluded. Left transverse sinus patent

Case 2

40-year-old male gave a history of road traffic accident 2 years back, during which he sustained a head injury. He was managed conservatively at that time. After 15 days of accident, he had complaints of severe headache with vomiting, was diagnosed as right transverse sinus thrombosis, and was treated with anticoagulants for 6 months. He came to our hospital with a sudden onset of severe headache of a 10-day duration. He underwent repeat MRI that was suggestive of arteriovenous fistula with right transverse sinus occlusion. In addition, MRI showed T2/FLAIR hypointensity of right temporoparietal lobe white matter [Figure 2a and b]. The corresponding GRE blooming was seen in the GRE sequence. Few tortuous vessels were seen in the right temporoparietal lobe with slow flow within the sulci [Figure 2a and b]. He underwent digital subtraction angiography (DSA) that was suggestive of Cognard Type IIB dural arteriovenous fistula (dAVF) of right mid-transverse sinus [Figure 2d-f], fed by the right occipital artery, ascending pharyngeal artery, middle meningeal artery, and the posterior meningeal artery. Proximal and distal portions of the right transverse sinus were occluded. There was significant cortical venous reflux into the right superficial middle cerebral vein that drains the brain territory corresponding to the region of MRI T2 hypointensity. Left transverse sinus was patent. Computed tomography (CT) scan showed few small foci of acute bleeding in the right temporoparietal lobe. Endovascular embolization was suggested.

Case 3

A 22-year-old female presented with severe headache of 3-day duration and one episode of generalized tonic-clonic seizures. In view of clinical suspicion of meningitis, MRI was done that showed subcortical T2/FLAIR hypointensity in the right parietal lobe [Figure 3a-d]; in the corresponding region leptomeningeal contrast enhancement was seen [Figure 3e and f], suggestive of meningitis. Her cerebrospinal fluid (CSF) study showed 84 cells with predominant neutrophils, protein −48 mg/dL, and sugar −60 mg/dL [corresponding random blood sugar (RBS) −159 mg/dL]. CSF was clear. CSF gram staining was negative for bacteria; tuberculosis polymerase chain reaction (TB PCR) was negative. Possible diagnosis of viral encephalitis was made.

Discussion

Subcortical white matter T2/FLAIR hypointensity is an uncommon finding of diverse etiology. The previously reported causes are Sturge-Weber syndrome,[2] moyamoya disease,[3] multiple sclerosis,[4] meningitis,[5] viral encephalitis,[5] and leptomeningeal metastasis.[5] Deep white matter T2 hypointensity along with deep gray matter nuclei is seen in arterial infarcts.[6,7] Deep white matter T2 hypointensity is reported in young children with diffuse ischemic-anoxic cerebral injury as well.[8]

The few cases of T2 hypointensity secondary to ischemia reported previously are of arterial origin — Infarction, moyamoya disease, etc. However, we describe two cases, where "venous congestion" undoubtedly played a role in cerebral ischemia, causing subcortical white matter T2 hypointensity. Though previous observations by Hurst RW and Grossman RI[9] suggested the possible venous congestion as etiology of T2 hypointensity in spinal cord white matter, no cerebral cases have been reported till date. The underlying pathophysiology appears to be the same as arterial ischemia — accumulation of nonheme iron and/or free radicals in subcortical white matter.[10-15] In the first case, acute onset of superficial cerebral vein thrombosis might have predisposed to increased accumulation of free radicals. While in the second case, chronic venous congestion due to dAVF associated with occlusion of proximal and distal portion of the transverse sinus lead to the interruption of axonal transport of iron and accumulation of nonheme iron. Such accumulation of paramagnetic substances in venous hypertensive myelopathy[9] are confirmed in a few studies; similar pathomechanism might be playing a role in subcortical T2 hypointensity as well, though further studies needed to substantiate the statement. The third case

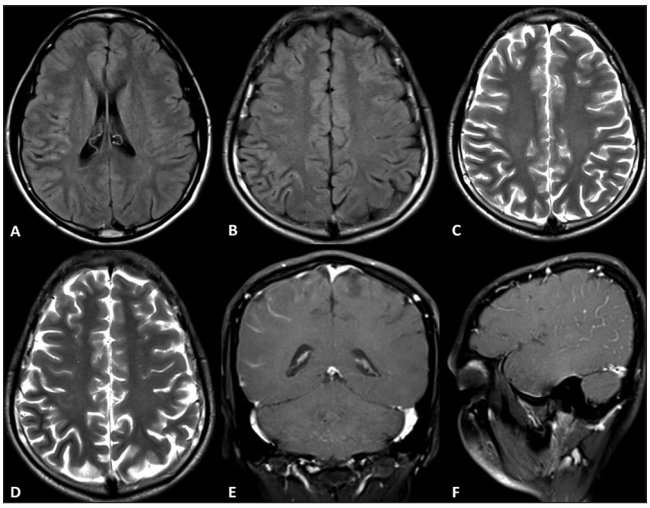

Figure 3: (a-d) Axial T2 and FLAIR MR images showing focal subcortical T2 hypointensity in the right parietal lobe, with corresponding sulcal FLAIR hyperintensity (e and f) Corresponding region leptomeningeal contrast enhancement seen

is a case of focal meningitis; contrast study showed abnormal leptomeningeal enhancement possibly contributing to venous congestion.

In addition, our findings hypothesize the possible cortical and subcortical venous congestion as an etiologic factor for T2 subcortical hypointensity in focal meningitis and encephalitis.[5] The "venous congestion" hypothesis can explain as well the reversal of T2 hypointensity after resolution of underlying etiology (meningitis/cerebral venous sinus thrombosis (CVST)/encephalitis/dAVF/leptomeningeal metastasis). The initial stage of T2 hypointensity could be a predisposing factor of later subcortical mineralization (as demonstrated by CT scans) in advanced cases of dAVF.[16] Possibly, similar pathomechanism might play a role in parenchymal mineralization of subcortical white matter in the vein of Galen malformation.[17] Additionally, in advanced cases of dAVF/vein of Galen malformation, dystrophic calcification might contribute to T2 hypointensity as well. Sturge-Weber syndrome is a disease due to primary venous dysplasia with resultant venous hypertension.[18] Again, "venous congestion" hypothesis can explain the excessive iron accumulation in subcortical white matter (as evidenced by T2 hypointensity/blooming in susceptibility weighted imaging).[19] Utility of perfusion studies in such cases may give more information about underlying pathophysiology, while histology/necropsy studies remains the gold standard for anatomical changes/mineral composition assessment.

However, the question raised by Lee *et al*.,[5] remains valid to date as to why subcortical low intensity is seen in only few patients of various pathology, while not seen in others with the same pathology. There is a possibility of genetic factors, duration of illness, severity of illness playing a major role in answering these questions.

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Conflicts of interest
There are no conflicts of interest.

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