

MR evaluation of cerebral Arachnoid Granulations in venous sinus using 3D T2 CUBE and 3D contrast-enhanced BRAVO sequence

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PURPOSE: To study the imaging pattern, location, prevalence, and multiplicity of arachnoid granulations in cerebral venous sinuses.

METHODS: We retrospectively reviewed 100 contrast-enhanced Brain MR studies, investigating the venous sinuses for discrete filling defects. After reviewing the imaging findings, we tried relating with clinical symptoms.

RESULTS: MR images show these entities as largely hypointense with cerebrospinal fluid in T1, hyperintense with cerebrospinal fluid in T2 sequences, isointense on FLAIR, hypointense on DWI and seen as filling defect on BRAVO. Septations were seen as linear variations of signal intensity within the granulations. Altered MR signal intensity was occasionally noted, when calcifications were present. The granulations appear as filling defects at MR angiography. Due to elliptical shape on oblique MR angiographic images, they could be mistaken for thrombus. No clinical significance could be given to the existence of any of these arachnoid granulations. They occur in 0.3 to 1 of 100 adults in the population.

CONCLUSION: On thin cross sectional imaging, arachnoid granulations in the venous sinuses are observed incidentally and are usually of no clinical significance. However, differentiation with intra sinus thrombus and tumor should be made.

Keywords: Anatomy, Arachnoid Granulations, Dural venous sinuses, Thrombosis.

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I. Introduction

Extensions of the arachnoid membrane into the dural venous sinuses, are called as Arachnoid Granulations (AG) and they drain the cerebrospinal fluid (CSF) from the subarachnoid space into the venous system^[1]. They are part of normal anatomy, and can be frequently demonstrated on imaging. AG contains adjacent blood vessels, which invaginate into the granulations^[2]. Most common location found for arachnoid granulations include superior sagittal sinus and transverse sinus. Commonly adjacent superficial draining cortical vein is seen abutting AG^[3]. Their incidence is seen to increase with age. Incidentally giant arachnoid granulation (>10mm) are mistaken for other pathologies, as they appear as sharp osteolytic lucency on X-ray and CT, or venous sinus filling defect^[4]. They should be differentiated from recanalized venous sinus thrombi, deep venous thrombosis, isolated cortical venous thrombosis, intracranial hypertension (idiopathic) parenchymal changes, hemorrhage^[5]. Less common conditions include intrasinus septa, fenestrations asymmetric superior sagittal sinus bifurcation^[6] and duplications^[7]. Previous imaging reports (as opposed to cadaver studies) have rarely found these structures in the superior sagittal sinus or straight sinus. This may be because of poor visibility of these two sinuses on conventional axial MR images and CT scans, limits of resolution on MR images, or volume averaging of the superior sagittal sinus with the cranial vertex. High resolution 3D contrast-enhanced imaging has been used as a means to depict intracranial dural sinus thrombosis^[8]. This pulse sequence,

which uses a 1.25-mm section thickness and 3D data set, would also be expected to precisely depict normal structures within dural sinuses and distinguish normal structures from occlusive disease. The purpose of this study was to describe the appearance, distribution, of arachnoid granulations and septa within patent dural sinuses by using a 3D contrast-enhanced BRAVO and 3D T2 weighted T2 CUBE pulse sequence^[7,8].

II. Materials and Method

This retrospective descriptive study was carried out in Department of Radiodiagnosis, Dr. Ram Manohar Lohiya Institute of Medical Sciences, Lucknow. The study included patient who had had normal appearance of the dural venous sinuses on conventional MR images and contrast-enhanced 3D BRAVO images, except for well-defined, focal filling defects consistent with arachnoid granulations. 3D contrast-enhanced BRAVO and 3D T2 CUBE imaging sequence, which was part of the clinical protocol at our institution.

Patients with findings consistent with dural sinus thrombosis, traumatic or surgical dural sinus disruption, mass lesions adjacent to or involving the dural sinuses, or those whose imaging studies were showing MR artifacts were excluded from this analysis.

3D contrast-enhanced BRAVO and 3D T2 CUBE images were retrospectively reviewed and findings were determined by the consensus of two experienced radiologists.

MR imaging studies were performed on a 3-T system (GE SIGNA 3.0T HDXT -32 CHANNEL) using 12 channel head coil.

An IV infusion of 0.1 to 0.15 mmol/kg of contrast medium was manually administered at a rate of 1 to 2 mL/s and then T1-weighted MR and contrast-enhanced MPRAGE imaging sequences were performed.

The order of the contrast enhanced MPRAGE imaging sequence and the contrast-enhanced T1-weighted MR imaging sequence varied among individual patients, although both of these sequences were performed within a period of 10 minutes after contrast material infusion. The contrast-enhanced BRAVO imaging sequence was begun within a period ranging from 10 seconds to 4 minutes after infusion of contrast material. All sequences were oriented in the axial plane. T2 weighted and Contrast-enhanced BRAVO imaging was performed through the entire head in thin sections, and source images were reviewed in the axial plane. Axial images were reconstructed in the sagittal plane by using the multiplanar reconstruction program operating on the MR imaging console. Overlapping sagittal images with a section thickness of 1.3 mm and an intersection distance of 1.2 mm were obtained in each case to better depict the superior sagittal sinus and straight sinus. In a few selected cases, coronal or orthogonal reconstructions were also performed.

Focal areas with well-defined margins showing non flow signal projecting into sinus lumen were recorded at superior sagittal sinus, middle to lateral transverse sinus, transverse sinus–sigmoid sinus junction, sigmoid sinuses and Torcular Herophili. They are most commonly seen at the junction between the middle and lateral thirds of the transverse sinuses near the entry sites of the superficial veins^[8]. Aforementioned differentials and artefacts due to sluggish or turbulent flow were differentiated by; visibility on all pulse and imaging planes sequences with maintained definite intra sinus position and circumferential surrounding of contrast.

III. Results

Focal, well-defined areas of non-flow signal protruding into the sinus lumen, producing defects in the contrast column, were identified in 32 (32%) of the 100 randomly selected contrast-enhanced MR examinations. AG appeared as hypointense relative to the brain on T1-weighted image (WI), hyperintense relative to the brain on T2-WI, showing complete suppression on fluid-attenuated inversion recovery sequences (FLAIR), associated lower signal, most likely representing collagenous connective tissue maintained venous flow around the lesion was observed (Figs 1,2). Incidentally hypoplastic sinus or veins were documented in vicinity of arachnoid granulations, helping in its identification. Transverse sinuses showed majority of the defects (90%) , particularly within the middle and lateral portions of the sinus (Figs 3). Left transverse sinus was slightly more affected than right. Torcular Herophili (dural venous sinus confluence) showed around eight defects, two were seen within the straight sinus, and two were in the sigmoid sinus. In one case, a large defect was present within the distal superior sagittal sinus (Fig 3). Twelve were located in the middle to lateral transverse sinus, and one was located in the superior sagittal sinus.

Of the 13 defects seen in the transverse sinus, 11 (85%) were directly adjacent to vein entry sites. The foci exhibited isointense to hypointense signal relative to brain parenchyma on T1-weighted images (12 were hypointense, one was isointense), and hyperintense signal on T2-weighted images (Figs 4,5). FLAIR images showed mixed signal intensities. Three were hypointense, six were hyperintense, two were isointense, and three were hyperintense peripherally, with a hypointense center. No contrast enhancement was identified in any case. T2-weighted images demonstrated focal clearly, which were further confirmed on all pulse

sequences and imaging planes. The mean size of the foci ranged between 2-5mm. The arachnoid granulations appeared as focal impressions into the sinus lumen, with surrounding flow signal on MR venograms (Figs 2, 3). 15 cases showed multiple intraluminal filling defects. No significant difference in

gender distribution was noted. Patients in whom filling defects were identified were older than those who had no filling defects. The mean age of the group with defects was 40-60 years. Transverse sinus was the most common site of filling defects (13AGs), followed by superior sagittal sinus (10AGs). Commonly encountered shapes of AGs were round or oval, with sharp outlines and homogeneous internal structure; of these 81% were associated with cortical vein. Some protuberances were spherical to linear in shape, with varying smooth to irregular contour. Histologically the larger arachnoid granulations are mixture of variably dense fibrous connective tissue containing numerous fibroblasts, scattered arachnoid cell nests, and an irregular network of small vessels and delicate endothelium-lined spaces, most prominent in the basal regions. Smaller granulations were composed of irregular, loose fibrous connective tissue core and a more peripheral, dense, hyalinized connective tissue layer.

IV. Discussion

Tufts of arachnoid villi invaginating into the dural sinuses are called Arachnoid granulations, through which cerebrospinal fluid (CSF) enters the venous system^[1]. Occasional hypertrophy of arachnoid villi in response to increasing CSF volume and pressure forms macroscopic lobulated AG. Randomly selected contrast-enhanced MR examinations of the brain revealed focal filling defects in the dural sinuses in 32% of cases, using volumetric 3D sequences^[4,5]. These defects are well circumscribed, T1 hypointense, T2 hyperintense, with near complete suppression on FLAIR images, and persistent filling defect on post contrast sequences.

The focal distribution of filling defects, well-defined morphology, and circumferential contrast around the defects helped in differentiating AG from intrasinus septa, venous sinus duplications^[8], and partial volume averaging of adjacent brain or dura, or normal variations in sinus contrast density^[9]. Imaging variability of AG most likely represents the variable amounts of connective tissue and cerebrospinal fluid within the granulation and partial volume effects from the adjacent contrast-filled dural sinus.

Arachnoid granulations can be easily distinguished from thrombosis. Usually entire segment of sinus or multiple sinuses are involved in thrombosis, with extension into cortical veins. Arachnoid granulations produce focal, well defined filling defects or signal foci. The intensity and signal of arachnoid granulations are also different from those seen with thrombosis. Acute thrombosis usually appears hyper dense on CT, and variable signal intensity (usually T1 hyperintensity) on MR images (depending on the age of

the thrombus and the pulse sequence)^[4,5]. Arachnoid granulations are never hyper dense on CT scan or T1-hyperintense in our cases. Abnormal flow with either partial or complete cut off, is usually seen distal to a thrombosed segment of sinus. Normal sinus contrast opacification on CT scans, flow void on MR images, and intrasinus signal on MR venograms are seen both proximally and distally to arachnoid granulations. Unusual signal within the dural sinuses on MR images due to slow or turbulent flow. However, these signals are rarely focal and not seen in post contrast images in the same location. Arachnoid granulations adjacent to venous entry sites represent perivascular protrusions of the leptomeninges at regions where the dura mater opens to allow the passage of veins into the dural sinus. Arachnoid granulations are also picked up due to its characteristic location. Even the largest filling defects don't show secondary signs of thrombosis or venous hypertension (collateral veins, dural enhancement, brain swelling).

Arachnoid granulations are normally occurring focal protuberances of the leptomeninges into the dural venous sinus lumen^[10, 11]. As compared to microscopic arachnoid villi, Arachnoid granulations are larger, granulations exhibit more extensive collagenous deposition and hyalinization. They most commonly protrude directly into the sinus lumen, and are found within transverse sinus, followed by superior and inferior sagittal sinus, torcula, straight sinus and sigmoid sinus in decreasing frequency; and they have also been described in relation to draining veins^[12]. This relationship of adjacent veins has been described grossly, microscopically, and ultrastructurally. Increase in number and conspicuity of Arachnoid granulations is seen with age^[13], a finding supported by this imaging study.

The neck of AG penetrates an aperture in the dura and expands to form the core of the granulation. This core is surrounded by a thin cupola of fibrous tissue continuous with the dura. This covering may be intact or fade out near the apex of larger granulations. The core is separated from the dural cupola by the subdural space, which diminishes toward the granulation apex^[12,13]. The core is composed of loose fibrous connective tissue forming a trabeculated network with wide interstices and endothelium-lined channels. Our study also demonstrated the same, arachnoid granulations assume a more lobular, complex morphology and become more hyalinised as they increase in size^[13,14].

AG is theoretically supposed to play a major role in resorption of cerebrospinal fluid and act as a potential cerebrospinal fluid volume buffer, although the exact mechanism is not well understood^[14]. There was mild left-sided distribution based on our observations on imaging studies. Cure' et al, reviewed normal dural sinus anatomy on CT and MR studies, and described two cases of large filling defects within the superior sagittal sinus and transverse sinus, and appeared as focal filling defects on MR venograms and standard angiograms^[15,16].

Similar analysis was attempted by Mamourian and Towfighi, who described a large arachnoid granulation within the superior sagittal sinus on MR images, MR venograms, and CT scans.

AG was focal, isointense to hypointense on T1-weighted images, hyperintense on T2-weighted images, and showed minimal heterogeneous contrast enhancement.

Even the largest granulations did not show any post contrast enhancement. Limited autopsy study of 10 cases was also performed by them. We observed higher prevalence of arachnoid granulations as we identified much smaller granulations, on multiplanar reconstruction of volumetric sequences and post contrast sequence. We documented optimal identification of arachnoid granulations on T2-weighted images or on fat suppressed contrast-enhanced T1-weighted images. The underestimation of the true prevalence on imaging examinations is most likely due to partial volume averaging effects on thick section images^[17].

Despite arachnoid granulations were most commonly mentioned within the superior sagittal sinus in previous anatomic studies^[17,18], we observed majority of the filling defects seen on MR examinations in our study within the transverse sinuses. Arachnoid granulations in superior sagittal sinus along anterior aspect protrude into lacunae laterals and not into the sinus lumen and, produce calvarial impressions, lateral to midline in this region and not present as focal intrasinus filling defects^[18,19]. Volumetric sequences in MRI allowed imaging in three planes, and no proximal superior sagittal sinus intraluminal foci were identified. Theoretically arachnoid granulations show psammomatous calcification; however it wasn't picked up on gradient images.

Even on literature search or our case analysis, we couldn't find arachnoid granulations (within the dural sinus) have been solely responsible for a patient's symptoms. However, large granulations could produce relative luminal compromise and lead to a pressure gradient or disturbed flow. The disturbed flow could in turn lead to venous hypertension (if the superior sagittal sinus or dominant transverse sinus were involved) or to thrombosis (if flow were hemodynamic compromise leading to hypercoagulable states)^[19,20].

V. Conclusions

Focal filling defects within the dural venous sinuses, consistent with arachnoid granulations, are seen on 32% of contrast-enhanced MR examinations in our study. They are typically located within the transverse sinuses, adjacent to venous entrance sites; followed by sagittal sinus, adjacent to torcula, in straight sinus and sigmoid sinus. They can be confused with thrombosis and intrasinus tumor, and should be differentiated by their characteristic location, well-defined morphology, density, and signal characteristics. In the majority of cases, the identification of AGs can be facilitated by their characteristic appearances: rounded or oval shaped, well-defined outlines and homogenous intensity. The presence of an adjacent cortical vein can be considered as an additional supportive element.

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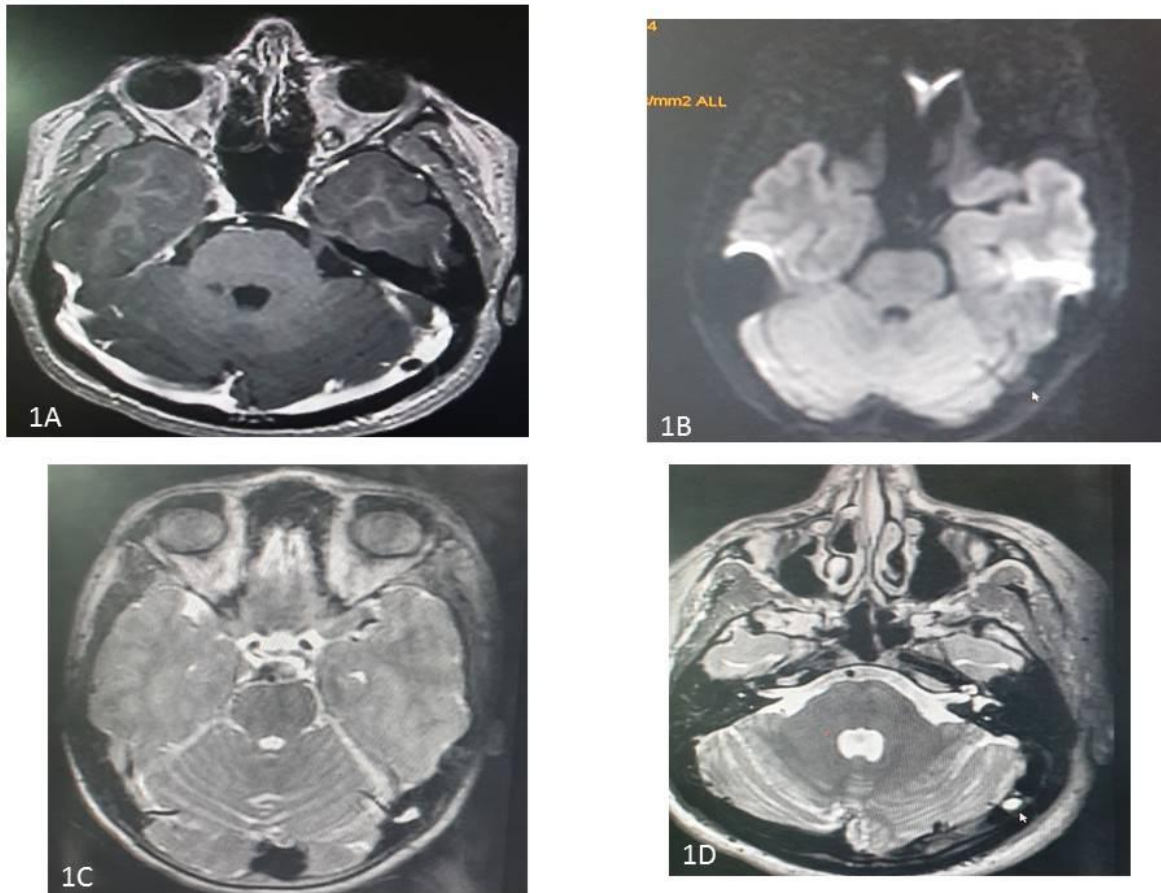


Fig1. (A) Axial reconstruction of post contrast fat suppressed T1 image shows intraluminal filling defect in left Transverse Sinus. (B) DWI image shows no significant restriction. (C&D) Axial T2 images confirm location, with normal adjacent brain parenchyma.

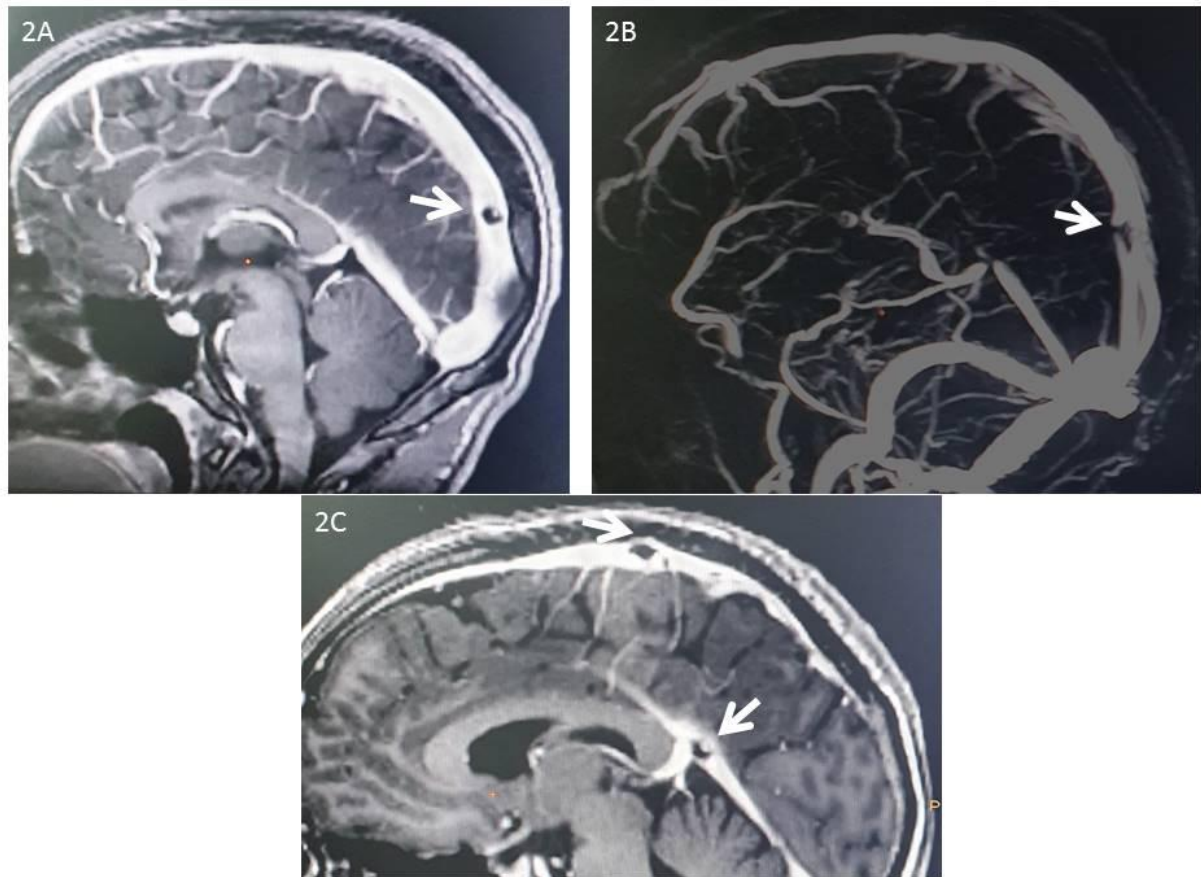


Fig 2. (A) Post contrast fat suppressed T1 weighted volumetric sequence in 65 year old man shows intraluminal filling defect in posterior aspect of superior sagittal sinus. However adjacent contrast opacification is maintained. Additional note is made of adjacent draining veins in close proximity. **(B)** Venographic image in same patient shows indentation . **(C)** BRAVO sequence in another patient shows Arachnoid Granulations in proximal part of straight sinus and middle aspect of superior sagittal sinus.

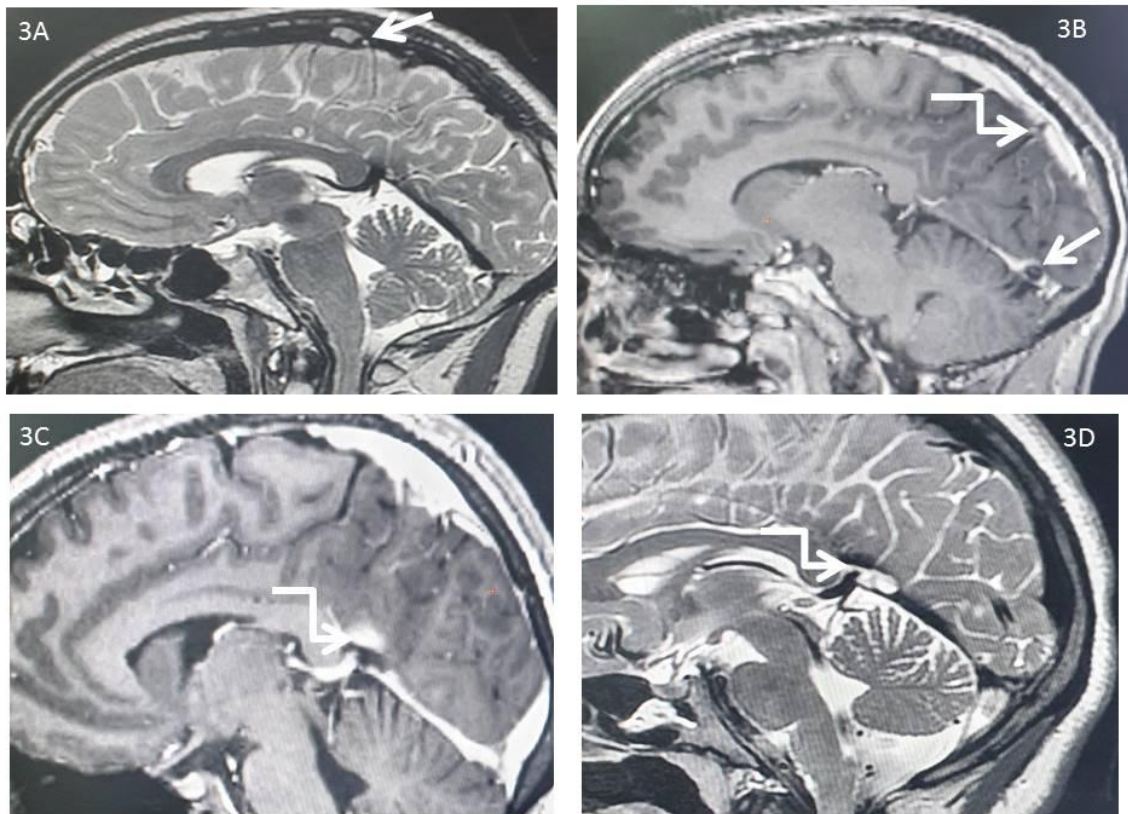


Fig 3.(A) Sagittal reconstruction of Volumetric T2 sequence shows Arachnoid Granulation(arrow) in middle part of Superior Sagittal Sinus.(B) Fat Saturated post contrast T1 image shows Arachnoid Granulations in posterior part of Superior Sagittal Sinus(elbow arrow connector) and in straight sinus, adjacent to Torcula(arrow). (C & D) Sagittal BRAVO and T2 images show lobulated arachnoid granulation with intraluminal projection in proximal part of straight sinus, just adjacent to confluence of internal cerebral veins and inferior sagittal sinus.

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