CASE REPORT

Giant arachnoid granulation with a thrombosed dural arteriovenous fistula

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SUMMARY

Arachnoid granulations are common incidentally detected small dural lesions which are usually asymptomatic and follow cerebrospinal fluid density/signal intensity on CT/MRI. Such lesions reaching a size of more than 1 cm are termed as giant arachnoid granulations (GAGs) which have been previously reported to cause venous hypertension and headaches. We report a case of GAG involving the proximal half of the superior sagittal sinus in a 45-year-old male patient which was associated with left temporal thrombosed dural arteriovenous fistula (AVF) whose thrombosed draining veins were seen converging towards the site of GAG. The patient presented with three episodes of generalised tonic-clonic seizures and improved with conservative treatment. No reports of such association of GAG with AVF is available in the literature, and we believe it could have occurred due to venous hypertension induced by GAG.

BACKGROUND

Arachnoid granulations are small extensions of the arachnoid membranes into the dural sinuses which aid in the resorption of cerebrospinal fluid (CSF) into the venous system. They are usually subcentimetre in size and can occasionally grow more than a centimetre when they are termed as giant arachnoid granulation.¹ Though most remain asymptomatic, some large lesions can fill and obstruct the sinuses causing headache and symptoms of venous hypertension.^{2 3} The development of the dural arteriovenous fistula (AVF), on the other hand, is aetiologically ascribed to venous hypertension resulting from obstruction of venous outflow where the obstruction is commonly caused by dural sinus thrombosis.⁴ Hence, logically a large arachnoid granulation could also induce the formation of dural AVF, and such occurrence is not reported in the published literature so far.

CASE PRESENTATION

A 45-year-old man presented to the casualty with a history of three episodes of generalised tonic-clonic seizures followed by brief loss of consciousness. He had no history of vomiting, altered sensorium, weakness or focal deficits and was apparently well in the past with no such symptoms. He was non-diabetic and non-hypertensive with no significant medical or family history. On examination, his vitals were stable, Glasgow Coma Scale

was E4V5M6, pupils were equally reactive. Central nervous system examination revealed no deficits.

INVESTIGATIONS

An MRI of the brain was performed to look for intracranial abnormality. It revealed a large intrasinus cystic lesion in the anterior half of the superior sagittal sinus causing displacement and compression of the venous sinus suggestive of a giant arachnoid granulation ([figure 1](#)). A discrete linear enhancing intrasagittal vein was seen within the cyst. There was associated dural-based thrombosed vascular mass in the left temporal region with few thrombosed superficial draining veins emanating from the mass and converging towards the site of the arachnoid granulation ([figure 2](#)). We subsequently performed an MR venogram and angiogram of the brain. The venogram confirmed the displacement and stenosis of the superior sagittal sinus ([figure 3](#)), and there was no involvement of any draining veins through the vein of Labbe to the transverse sinus. The angiogram showed branches of the left temporal and occipital arteries (external carotid system) and the left middle cerebral artery (internal carotid system) reaching until the site of the thrombosed vascular mass suggestive of a thrombosed dural AVF ([figure 4](#)). Rereview of the brain MRI images revealed mild atrophy of the left insular cortex in the form of relative widening of its sulci with mild prominence of the adjacent sylvian fissure ([figure 5](#)) that could have occurred due to shunting of blood while the AVF was active. No infarct or any other abnormality was detected. Further, a digital subtraction angiography (DSA) of the brain was planned to check for any patent portion of the AVF, but the procedure was deferred as the patient felt better with the antiepileptic medication and did not consent for the semi-invasive procedure.

DIFFERENTIAL DIAGNOSIS

The imaging differentials for the giant arachnoid granulation that are relevant to the present case are an arachnoid cyst, dermoid and epidermoid cysts. The intrasinus location of the cyst with the presence of an intracystic vein ruled out arachnoid cyst; absence of fatty areas or calcification and no diffusion restriction on diffusion-weighted images ruled out dermoid and epidermoid cysts.

The imaging differential for the thrombosed AVF is cerebral cortical vein thrombosis (CVT). The thrombosed veins emanating from a single focus of

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Figure 1 Coronal T2 (A,B), axial T2 (C,D), sagittal precontrast T1 (E) and sagittal postcontrast T1 (F) images of the brain. The bold white arrows indicate the giant arachnoid granulation located within the anterior portion of the superior sagittal sinus which is causing displacement and stenosis of the sinus (indicated by white arrowheads in A, B and C images). The long thin white arrows in images (D) and (F) show an intracystic vein enhancing after administration of contrast.

dural-based vascular lesion with the presence of arterial branches from both external and internal carotid system reaching up to the mass (feeders) on MR angiogram and absence of associated infarct in the brain parenchyma ruled out CVT.

TREATMENT

The patient was started on antiepileptics with which he recovered and remained stable during the hospital course. He was discharged after 5 days of hospital stay.

OUTCOME AND FOLLOW-UP

The patient recovered fully and was advised to continue the antiepileptics. He is on regular follow-up every 4 weeks and is advised to immediately contact the hospital services if he has a recurrence of the symptoms.

DISCUSSION

The association between a giant arachnoid granulation and symptomatic venous hypertension is not new and has been objectively established by few investigators by measuring significant venous pressure gradients across the lesion. They also treated them by placing a venous stent across the site of obstruction achieving good post-therapeutic responses.^{5 6} Dural sinus thrombosis is another well-known cause of venous hypertension which triggers the formation of dural AVF by two hypothetical

Figure 2 Sequential caudocranial susceptibility-weighted images (SWI) of the brain showing a thrombosed dural-based vascular focus in the left temporal region (bold white arrows in the top row). Few thrombosed cortical veins (bold black arrows in middle and bottom rows) are emanating from the focus and running upwards to converge into the superior sagittal sinus at the site of partial obstruction by giant arachnoid granulation (black asterisks in the bottom row). Thrombosis is indicated by marked hypointensity on SWI.

mechanisms.⁴ The first involves opening of dormant physiological shunts between the meningeal arteries and dural veins, and the second mechanism involves stimulation of new vascular communication or fistula secondary to reduced perfusion inducing angiogenesis.⁷⁻¹⁰ As both the hypothetical mechanisms basically happen due to venous hypertension, it is plausible to associate a giant arachnoid granulation with dural

Figure 3 MR venogram images at different projections, confirming the stenosis of the superior sagittal sinus (white arrows) at the site of giant arachnoid granulation.

Figure 4 MR angiogram images in oblique and frontal projections showing the branches of the left temporal artery (bold white arrows), left occipital artery (black arrows) and left middle cerebral artery (thin white arrows) reaching until the thrombosed vascular focus in the temporal region (black asterisks) suggestive of arteriovenous fistula.

AVF. Also, the site of dural AVF formation can be at the site of venous obstruction or anywhere along the venous pathway that drains into the obstructed sinus and hence the latter scenario is pertinent in the present case.¹¹

Few more peculiar findings were observed in our case study. They are with regard to the unusual location of giant arachnoid granulation and the unusual type of dural AVF. While the usual locations of giant arachnoid granulation are in the mid and lateral portions of the transverse sinus followed by the posterior portion of the superior sagittal sinus, it was located in the anterior half of the superior sagittal sinus in our case.^{2 12} The usual type of dural AVF is 'sinus type' which is commonly located in the transverse sinus, cavernous sinus, along the tentorium cerebelli and the superior sagittal sinus while in our case it is 'vault dural AVF' which is a rare non-sinus type located over the left temporal convexity.^{13 14} This type is an aggressive form of AVF whose spontaneous thrombosis is even rarer in occurrence.^{14 15} Different authors have ascribed various associations or reasons for thrombosis which include the 'sparse type' of AVF, presence of an intracranial haematoma, thrombosis of draining vein, recanalisation of obstructed sinus, trauma-induced or contrast-induced thrombosis, slow-flow and intrinsic compression.^{15–22} Out of these, the association of 'sparse-type' of AVF with thrombosis appears to be a possible explanation in our case although it needs a DSA for confirmation.

Figure 5 Axial T2-weighted images of the brain showing relative atrophy of the left insular cortex in the form of relative widening of its sulci with mild prominence of the adjacent sylvian fissure (area under white bracket). The white arrow shows the thrombosed focus of arteriovenous fistula.

Patient's perspective

I never had any health-related problems in the past. Suddenly having fits (seizures) and getting admitted to hospital was a shocking experience for me. Doctors say I have a fluid sac in my head that is normally small in many people but has become big in me. They also told there is a tangle of blood vessels which has clotted on its own thankfully. However, they wanted to investigate more on those blood vessels by putting a tube through my leg artery but am very scared to get that test done. I am on fits tablets and alright now with no problem. I am told I may have to undergo that test in future if I get a similar problem but am completely hopeful that the vessel problem has fully rectified on its own.

Learning points

- ▶ Giant arachnoid granulation could lead to the formation of dural arteriovenous fistula (AVF) secondary to induced venous hypertension in the manner similar to dural sinus thrombosis causing AVF formation.
- ▶ The site of dural AVF formation can be at the site of venous obstruction or anywhere along the venous pathway that drains into the obstructed sinus.
- ▶ Vault dural AVF is a rare non-sinus type of lesion having an aggressive course. The usual type is sinus type that occurs in the walls of the dural sinuses.
- ▶ A sparse fistulous network has a strong association with spontaneous closure of dural AVF.

Contributors LHK was involved in conception, drafting the manuscript and in final approval of the version to be published. KR was involved in the interpretation of data, drafting and editing the manuscript and in final approval of the version to be published. SKK was involved in the acquisition, analysis of data, critical revisions of the manuscript for important intellectual content and in final approval of the version to be published. SL was involved in the design of the work, drafting the manuscript and in final approval of the version to be published. All authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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