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Spontaneous chronic subdural haematoma due to hypoplastic rostral superior sagittal sinus

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SUMMARY

The superior sagittal sinus (SSS) is a midline structure of the superficial cerebral venous system that drains the anterior cerebral hemispheres. Hypoplasia of the rostral SSS is a known variant, although associated complications are rare. A woman in her 30s presented for evaluation of a symptomatic left-sided acoustic neuroma and was found to have an incidental chronic subdural haematoma (SDH) over the left frontoparietal convexity without trauma or precipitating event. The SDH expanded on serial imaging and the patient eventually underwent left-sided frontoparietal craniotomy for haematoma evacuation. Haematological evaluation was benign, but angiography revealed absence of the anterior half of the SSS. We report the first case of spontaneous SDH in the setting of hypoplastic rostral SSS.

BACKGROUND

The superior sagittal sinus (SSS) is a midline structure of the superficial cerebral venous system that courses along the falx cerebri, draining the anterior cerebral hemispheres and terminating at the confluence of sinuses.[1] SSS anatomy is critical in diagnosing and managing dural venous thrombosis, arteriovenous fistulas and meningiomas, and creating burr holes in close proximity to the midline. Developmental alterations leading to anatomical variations of the cerebral venous system are frequent and most commonly involve the transverse sinus and sigmoid sinus.[2] However, variations of the SSS are relatively rare.[3–5] We report the case of a patient with hypoplastic rostral SSS and spontaneous subdural haematoma (SDH).

CASE PRESENTATION

A previously healthy woman in her 30s presented with 3 months of left-sided tinnitus, sensorineural hearing loss and disequilibrium. There was no trauma or precipitating event. She was otherwise asymptomatic and without focal deficits other than unilateral diminished hearing.

INVESTIGATIONS

MRI of the internal auditory canal revealed a 1 mm left-sided acoustic neuroma (figure 1) and an incidental 8 mm chronic SDH over the left frontoparietal convexity (figure 2A–D). The patient elected to follow the SDH with serial imaging. She remained clinically stable at 3-month follow-up, but repeat MRI showed the haematoma had enlarged to 13 mm with increased mass effect. Baseline chemistries and blood counts were unremarkable. Haematological evaluation revealed no coagulation abnormalities or results consistent with any known bleeding diathesis. CT angiogram demonstrated non-opacification of the anterior half of the SSS (figure 3).

TREATMENT

The patient was thus offered left-sided frontoparietal craniotomy for haematoma evacuation. The craniotomy was 3.5 cm in diameter and 5.9 cm lateral to the midline. The intraoperative findings were consistent with chronic SDH under moderate pressure, which was evacuated uneventfully.

OUTCOME AND FOLLOW-UP

The patient recovered well postoperatively and returned to her baseline level of activity. Follow-up diagnostic cerebral angiography performed 4 years after craniotomy showed no evidence of an underlying arteriovenous fistula, but did reveal absence of the anterior half of the SSS (figure 4A–C). This was thought to be developmental rather than acquired given the large cortical venous collaterals, skull remodelling over the sinus and absence of hypercoagulable disorder. The acoustic neuroma was managed expectantly with annual MRIs showing minimal growth, and vertigo was managed medically. At last follow-up 7 years after initial presentation, the patient was neurologically stable, without recurrence of the haematoma.

DISCUSSION

Spontaneous SDH is very uncommon in young patients.[6] Among 193 previously reviewed cases of acute spontaneous SDH, only 22 patients were younger than age 40.[7] It remains unknown whether the incidence is changed with hypoplastic SSS. Majority of the reported cases of SDH in younger patients occur insidiously, and a comprehensive evaluation of the most common aetiologies, including arterial, idiopathic, coagulopathic, neoplastic and spontaneous intracranial hypotension, is critical for effective management.

The SSS is an unpaired venous structure that originates at the junction of the frontal and ethmoid bones, directly posterior to the foramen caecum in proximity to the crista galli. It courses posteriorly along the cranial vault, just inferior to the sagittal suture and superior to the falx cerebri within two dural leaves, and terminates in the confluence of sinuses.[1] The SSS drains large regions of the superior cerebral hemispheres and also receives blood from diploic, meningeal and emissary veins from the scalp.[2] Developmentally, the SSS originates from a sagittal plexus of vessels that combine to

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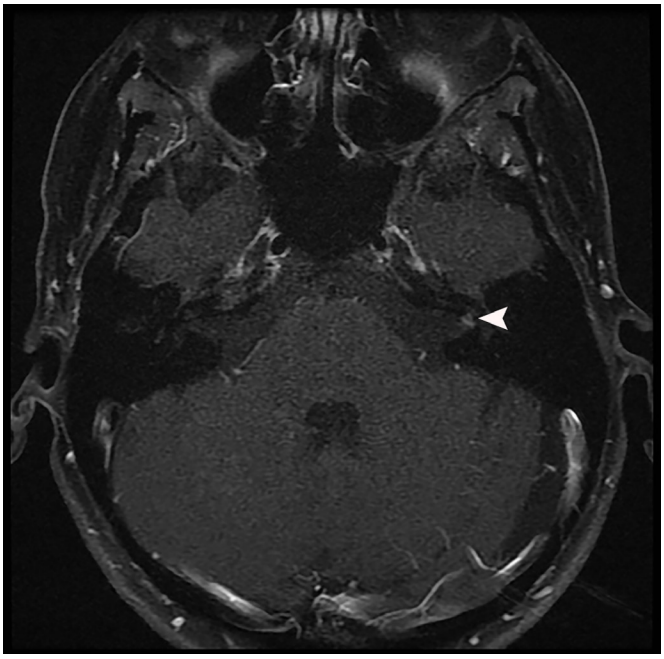

Figure 1 Axial T1-weighted, contrast-enhanced MRI showed a 1 mm homogenously enhancing lesion in the left internal auditory canal, consistent with a small vestibular schwannoma (arrow).

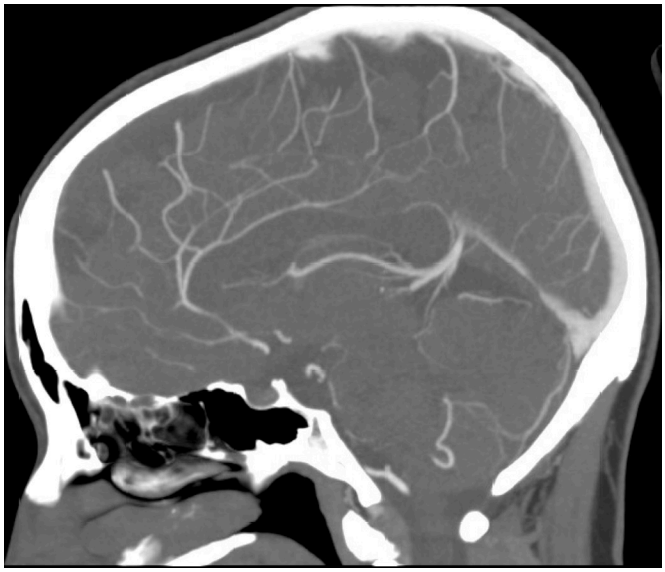

Figure 3 Sagittal CT angiogram of the head performed prior to craniotomy demonstrates non-opacification of the anterior half of the superior sagittal sinus.

form a single channel. A longitudinal venous channel becomes the anterior SSS, while elongation during displacement of the tentorium cerebelli inferiorly and the cerebral hemispheres posteriorly forms the posterior SSS.[8] Alterations in embryological development can cause anatomical variations. Rostral SSS hypoplasia occurs in 1.8%–6% of people,[4 5 9] although absence of more than the rostral one-third is significantly rarer.[5] Rostral SSS variants include completely developed, duplicated, complete or bilateral hypoplasia, and unilateral hypoplasia.[4] Developmental failure or delay in the formation of the rostral sagittal plexus can be associated with compensatory prominent bilateral superior frontal veins that follow a parasagittal course to drain into the origin of the SSS.[3]

It is important to distinguish between rostral SSS hypoplasia and thrombosis, which can be excluded based on absent clinical suspicion and lack of a filling defect or occlusion suggestive of intraluminal thrombosis.[4] Furthermore, haemorrhage associated with sinus thrombosis is more likely be parenchymal than subdural.[10] Complications in the setting of determined anatomical variants are rare but should be considered in atypical presentations. Our patient had compensated venous drainage with enlarged tentorial sinus, frontal hemispheric veins and parasagittal large venous channels. Although a causal relationship between hypoplastic SSS and SDH is difficult to prove, no other underlying abnormality was present. There is one published report of a subacute SDH in a patient with complete agenesis of the SSS and falx cerebri,[8] although our case differs in that the SDH was chronic and purely incidental, and both the SSS and falx were present. SSS variants will be identified more frequently with increasing use of neuroimaging. There are published reports of chronic SDH associated with coughing, constipation, sneezing or high altitude in older individuals, as well as minor trauma that patients do not remember. While these serve as potential alternative explanations for chronic SDH, the age and health of our patient make them less likely. Larger cohort studies are needed to establish associated risks and guide patient counselling. Standard neurosurgical management can be

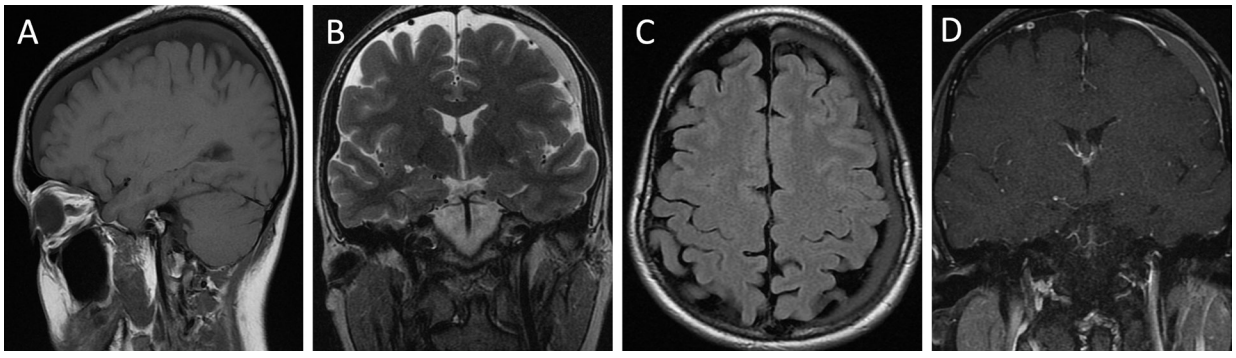

Figure 2 (A) Sagittal T1-weighted, (B) coronal T2-weighted and (C) axial T2 fluid-attenuated inversion recovery (FLAIR) MRIs show a chronic subdural haematoma (SDH) over the left frontoparietal convexity. The SDH is isointense to cerebrospinal fluid (CSF) on T2-weighted imaging and slightly hyperintense compared with CSF on T1 and FLAIR sequences. The coronal T2-weighted image displays absence of the anterior superior sagittal sinus (SSS). (D) A coronal T1-weighted, contrast-enhanced MRI in the anterior hypoplastic section demonstrates absence of the SSS and no evidence of abnormal signal, unlike a sinus thrombosis which would likely demonstrate increased signal.

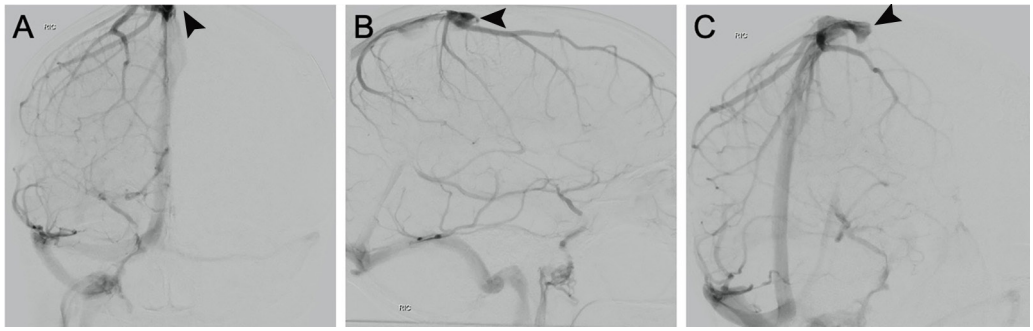

Figure 4 Venous-phase (A) posteroanterior, (B) lateral and (C) oblique digital subtraction angiograms of the anterior circulation performed by injection of the right internal carotid artery. The rostral half of the superior sagittal sinus (SSS) is absent (arrow). Large compensatory cortical venous collaterals drain into the SSS at its origin in the parietal region.

used for associated complications such as SDH, in addition to coagulopathy workup, vascular studies and serial imaging.

Learning points

- The superior sagittal sinus (SSS) is a midline structure of the superficial cerebral venous system that drains the anterior cerebral hemispheres.
- Hypoplasia of the rostral SSS is a known variant, although associated complications are rare.
- We report a rare case of spontaneous subdural haematoma in the setting of hypoplastic rostral SSS.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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