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CASE REPORT

Fenestration of the superior sagittal sinus in a ring configuration

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ABSTRACT

The superior sagittal sinus is a major component of the cerebral superficial venous system. Knowledge of this structure, variations, and possible anomalies is of practical clinical importance for neurosurgeons, neurologists, and radiologists to rule out a number of pathologies. Developmental anomaly of the superior sagittal sinus may be falsely interpreted as sinus thrombosis or arachnoid granulations. A nine-year-old boy presented with acute severe headache and lower limb weakness, resulting in three days of immobility. Initial brain multislice computed tomography raised suspicion of sagittal sinus thrombosis. Subsequent magnetic resonance angiography identified a large arachnoid granulation (18×13×13 mm) in the posterior third of the superior sagittal sinus, displaying cerebrospinal fluid signal without contrast enhancement and containing a cortical venous vessel. Blood flow through the sinus remained normal, with no venous obstruction. Magnetic resonance angiography of the brain (three-dimensional time-of-flight mode) revealed fenestration of the upper sagittal sinus in the form of a ring; no signs of venous outflow disturbance were detected. Neurosurgical intervention was deemed unnecessary due to preserved venous outflow and absence of acute pathology. The patient was diagnosed with fenestration of the superior sagittal sinus (Q28.8), although the etiology of the acute symptoms remained undetermined. Follow-up magnetic resonance angiography at 12 months revealed no structural changes, and the patient remained asymptomatic. The ring-shaped fenestration of the superior sagittal sinus that we described may be misinterpreted as sagittal sinus thrombosis, creating the illusion of a "delta sign" (absence of blood flow in the venous sinus), which is considered pathognomonic for sinus thrombosis. This is the first case described in the literature in which the superior sagittal sinus is divided as a ring. The case demonstrates the importance of differential diagnosis of arachnoid granulations and congenital venous sinus anomalies in acute neurologic symptoms in children.

Keywords: Anomaly, arachnoid granulation, fenestration, superior sagittal sinus, thrombosis.

The cerebral venous system is divided into superficial and deep components. The superior sagittal sinus (SSS) serves as the primary element of the superficial cerebral venous system, and understanding this structure and its variations is of practical clinical importance to neurosurgeons, neurologists, and radiologists in the treatment of various diseases. A reliable anatomic view of the SSS is important for clinicians as it determines the treatment strategy of the patient.[1]

Knowledge of the possible anatomic variations of the dural venous sinuses is crucial to avoid erroneous neuroradiologic interpretation, either labeling them as pathology or not indicating their presence in cases of impending neurosurgical or endovascular intervention, which may lead to preventable complications.

While SSS duplication and fenestration are documented,[2,3] annular configurations remain

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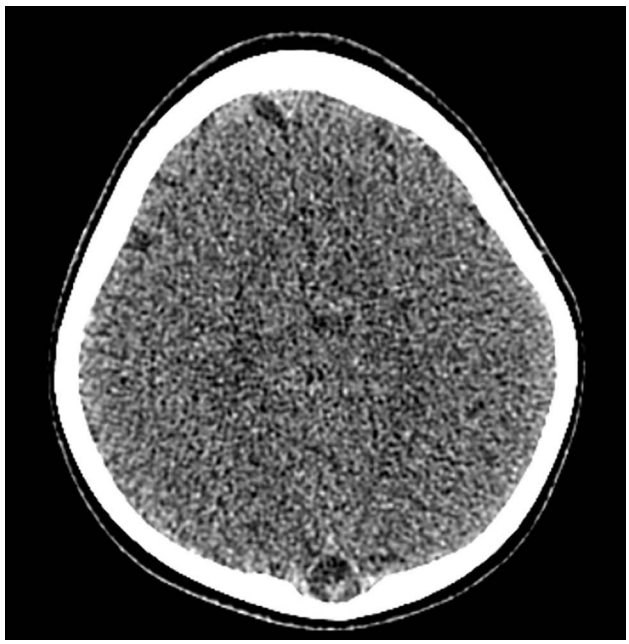

Figure 1. Suspected delta sign on the patient's axial multislice computed tomography.

unreported, posing unique diagnostic challenges. Herein, we reported a case of SSS fenestration in a ring configuration.

CASE REPORT

A nine-year-old male patient was admitted due to a sudden onset of severe headache and leg weakness. Brain multislice computed tomography (MSCT) scan was conducted on the same day, revealing suspected sagittal sinus thrombosis (Figure 1). Despite the passage of time, the leg weakness persisted, rendering the patient immobile for three consecutive days. Subsequent to the resolution of the headache and leg weakness, the patient was referred to the outpatient clinic of the Federal Center of Neurosurgery in Novosibirsk for further evaluation. Written informed consent was obtained from the parent of the patients.

The magnetic resonance angiography (MRA) revealed a large arachnoid granulation (AG) within the posterior third of the SSS, measuring 18×13×13 mm (Figures 2a-c). The granulation exhibited cerebrospinal fluid (CSF) signal content without evidence of contrast accumulation, accompanied by the presence of a cortical venous vessel within the lumen. Normal blood flow was observed through the sagittal sinus, with no indications of venous outflow obstruction. Additionally, three-dimensional (3D) time-of-flight (TOF) MRA identified fenestration of the SSS without venous outflow disruption. Magnetic resonance imaging (MRI) of the lumbar spine showed no abnormalities. Consequently, neurosurgical intervention was deemed unnecessary. After an acute condition was ruled out, the patient was diagnosed with Q28.8 fenestration of the SSS based on MRI. The cause of the weakness in the legs and headache could not be determined, and the patient had no prior history of this condition. The patient was advised to undergo monitoring by a neurologist, along with a follow-up MRA of the brain after 12 months for further assessment.

The control MRI at the 12-month follow-up revealed no changes (Figure 3). The patient had no further complaints.

DISCUSSION

The SSS is an unpaired venous structure that originates at the junction of the frontal and ethmoid bones, located immediately posterior to the foramen cecum in close proximity to the crista galli. Due to its considerable length, the SSS is the largest venous structure in the brain.

In addition to its main function of draining blood from the cerebral cortex, the SSS plays a key role in maintaining CSF homeostasis. The walls of the SSS contain the greatest number of arachnoid villi and granulations, which serve as one-way valves that facilitate the movement of CSF into the dural sinuses. These structures play an important role in directing CSF toward the SSS for reabsorption and elimination. This continuous circulation of CSF is essential for the regulation of cortical and ventricular volume. Any disruption of this complex process can lead to various neurological disorders.[1,4] The present case described a rare annular fenestration of the SSS, initially misdiagnosed as thrombosis due to its imaging resemblance to the "empty delta sign." This anatomic variant, distinct from previously reported duplications or bifurcations,[2,3,5] posed a diagnostic challenge by mimicking thrombotic occlusion on MSCT. The subsequent MRA clarified the absence of thrombosis, demonstrating preserved venous flow around the fenestration and a coexisting giant AG with characteristic CSF signal intensity.[6]

In the early stages of embryogenesis (before week eight), venous blood from the brain drains into the paired primary cerebral sinuses, which

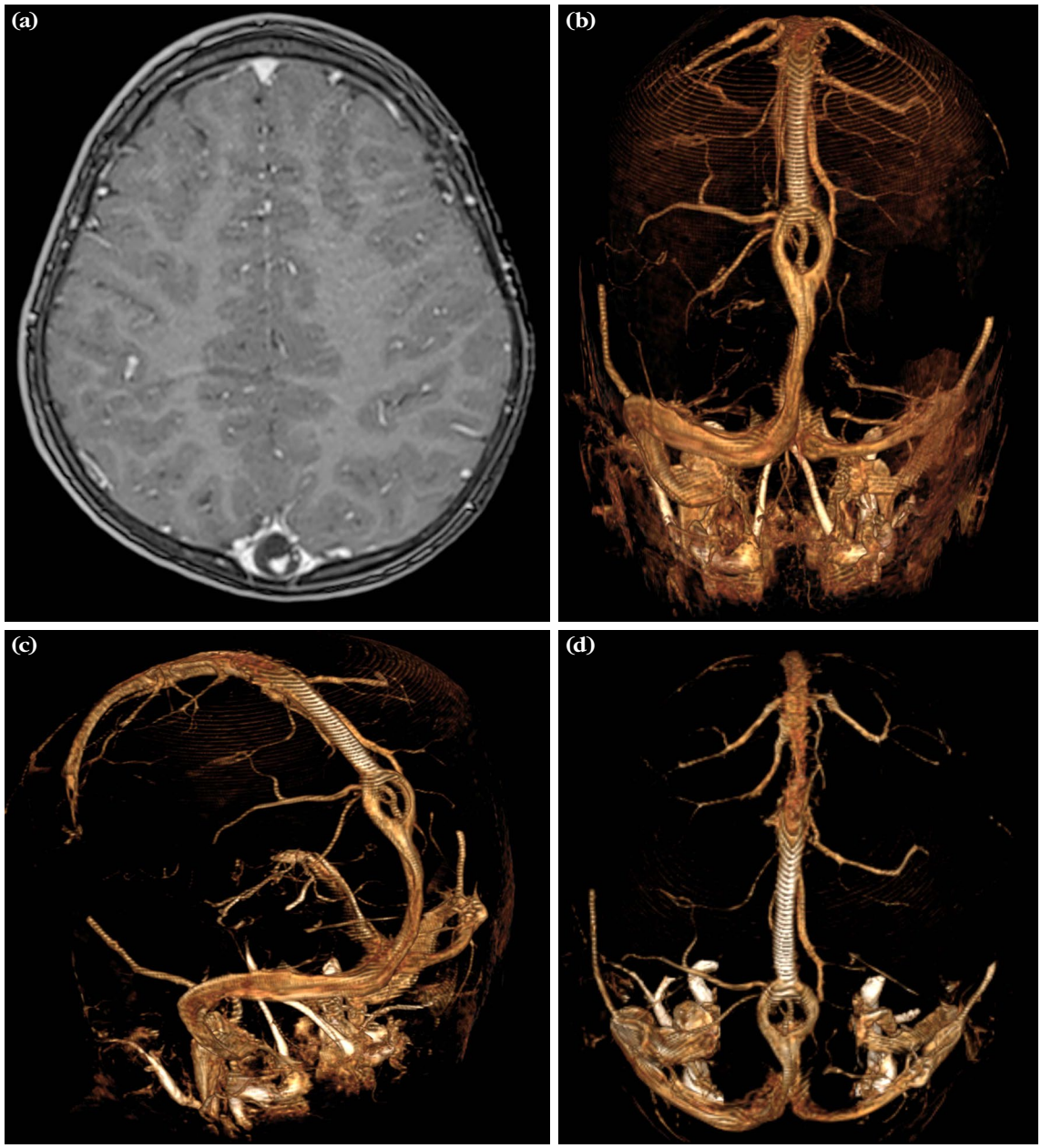

Figure 2. (a) Patient's axial magnetic resonance imaging demonstrating an abnormal sagittal sinus. Magnetic resonance images of the patient with an abnormal sagittal sinus on **(b)** back, **(c)** oblique, and **(d)** top views.

continue into the anterior cardinal veins. In the 5-mm embryo, blood outflow from the entire primary venous network of the head occurs in three plexuses that anastomose with each other. The anterior venous plexus collects blood from the eyes, cerebral hemispheres, and midbrain structures. The middle venous plexus receives blood from the hindbrain. The posterior plexus receives blood from the medulla oblongata. Blood from these plexuses flows into the primary cerebral sinus, which flows into the anterior cardinal vein.[7]

At the beginning of the eighth week, vascular plexuses continue to develop in the 16- to 21-mm embryo. The sagittal plexus begins to form between the growing cerebral hemispheres from the anterior plexus, and the tentorial plexus begins to form over the midbrain between the terminal medulla and the trunk.[7]

In a 40-mm long, nine-week-old embryo, the SSS forms from the sagittal venous plexus and is laid down as a paired formation located in the

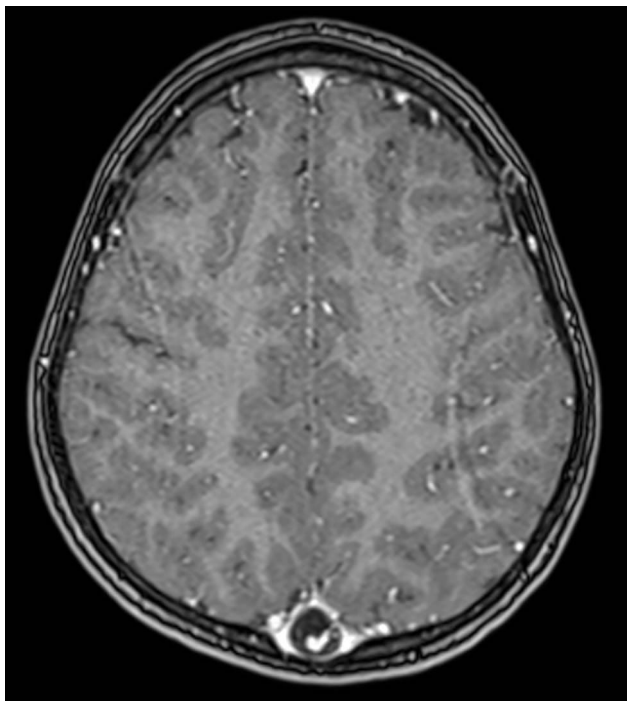

Figure 3. Magnetic resonance images of the patient with abnormal sagittal sinus (top view).

forebrain. Subsequently, a solitary sinus arises from the paired sagittal sinus, lying dorsally along the midline. It is this period that is critical for the onset of the anomaly embryology of sinuses.[7]

In a 60- to 80-mm long fetus (12 weeks), a rather distinct pattern of venous structure is revealed. Somewhat posterior to the SSS is the primary tentorial plexus (Figure 4), which forms a very variable and asymmetric sinus drainage.[8]

The fenestration likely arose during the 9th to 12th gestational weeks, when the SSS forms from the sagittal plexus.[7] While most SSS anomalies involve bifurcation[3,5] or duplication,[9] annular fenestration may represent incomplete fusion of embryonic venous channels. Unlike duplication, which involves two separate sinuses, fenestration represents a localized split within a single vessel (Figure 5).[9]

Due to the intricate network of connections that perform the essential function of draining the cerebral hemispheres and receiving blood from the diploid, meningeal, and emissary veins of the scalp, the SSS is prone to a number of complications and pathologic processes. First and foremost is thrombosis, a condition characterized by multiple symptoms such as headache, hemiparesis, sixth nerve palsy, papilledema, nausea, and seizures. Septic thrombosis of the SSS, although infrequent, is most often associated with bacterial meningitis or paranasal sinus infections.[1]

Thrombosis of the SSS is of great clinical importance, as the occlusion of the dural vein can lead to increased intracranial pressure and potentially irreversible consequences. Given its critical involvement in CSF circulation, occlusion of the vein usually disrupts the physiologic CSF drainage pathway, resulting in increased intracranial pressure. In addition, another factor contributing to increased intracranial pressure in such cases is the development of edema after venous occlusion.[10]

The primary goal in the management of patients with upper sagittal sinus thrombosis is to stabilize and prevent cerebral herniation. Although heparin remains the cornerstone of acute treatment, there is growing support for the limited use of mechanical thrombectomy. Diagnosis of this condition can be challenging, but in cases where strong clinical suspicion exists, computed tomography venography, magnetic resonance venography (MRV), or deliberate catheter arteriography focusing on the venous phase may serve as diagnostic modalities.[4]

Among the sagittal sinus anomalies described, duplication of the sagittal sinus is the least common. Only a few cases of duplicated SSS with or without associated parietal encephalocele were published.[3,9] Kokidko et al.[9] described a patient with a parietal cephalocele (encephalocele) that caused “splitting” of the SSS, thus forming two venous channels. Variations of the most proximal (i.e., drainage site) of the SSS are common. For example, this venous structure may drain directly into one or both transverse sinuses (TSs).[3] However, the distal SSS is rarely variable.

In this spectrum of variations, SSS bifurcation is a recognized variant. Previous attempts to determine its prevalence have yielded a range of estimates from 4 to 23.5% among the individuals studied.[2,10] Özen et al.[5] reported a variant bifurcation of the SSS, with the bifurcation occurring up to 53.3 mm above the torcula. Boukobza et al.[2] described an even rarer presentation: variant of bifurcation of the SSS in which each fork fell into one TS. This variant can mimic thrombosis or other venous pathologies, necessitating precise imaging differentiation to avoid unnecessary interventions. Unrecognized

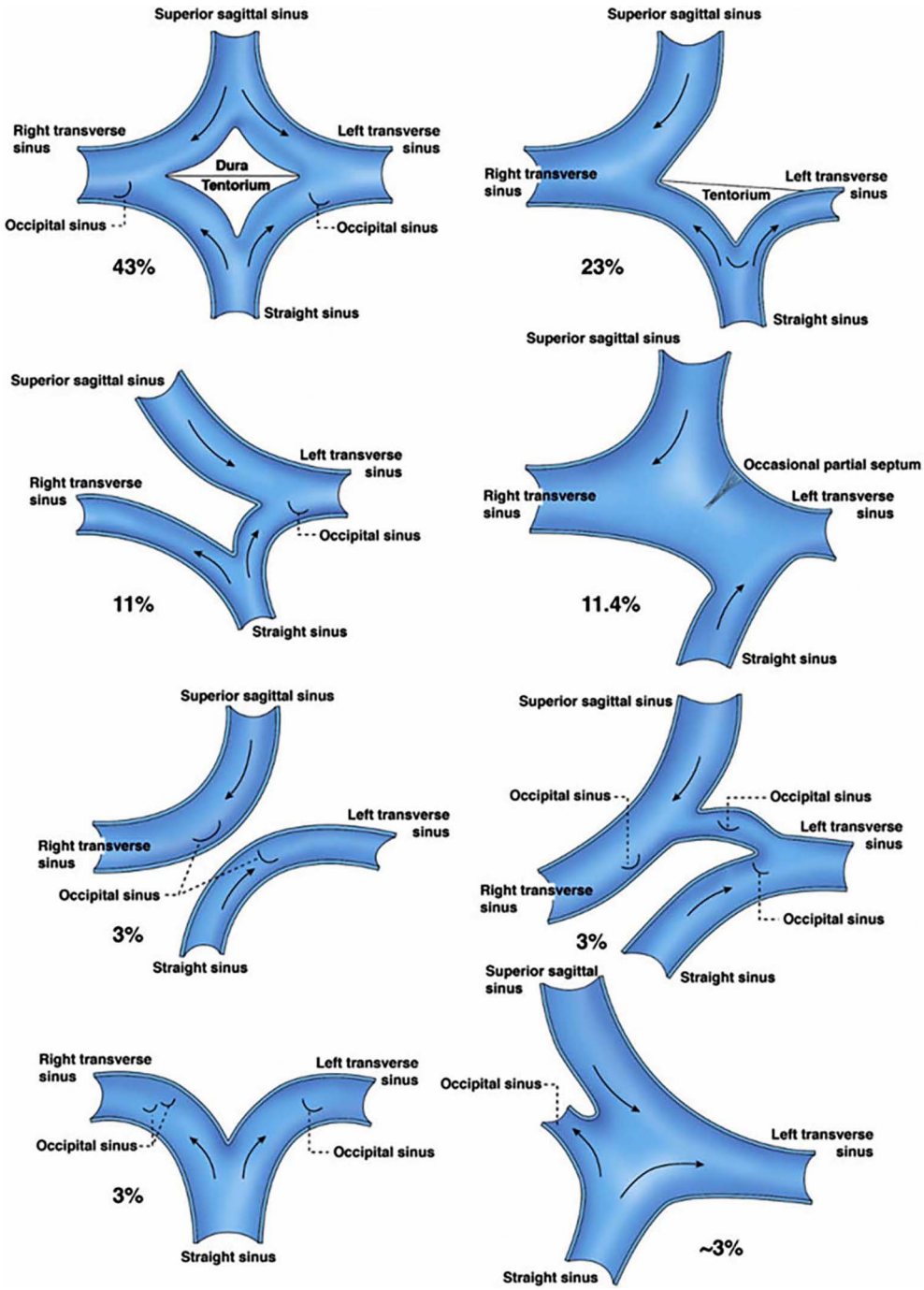

Figure 4. Variations of the torcular herophili, distilled from various sources after.[8]

bifurcation/fenestration may lead to accidental sinus ligation during craniotomy.[2,9]

Of particular clinical significance in our case, this anatomical variant posed a notable diagnostic challenge due to its potential to mimic venous sinus thrombosis on imaging, particularly through the illusory appearance of the "empty delta sign," a classic imaging hallmark of thrombosis.[6,10] Thrombosis classically presents with headache (88.8%), seizures (39.3%), or papilledema (28.3%).[10] Our patient's transient symptoms (isolated headache/weakness) and lack of thrombotic risk factors argued against this diagnosis.

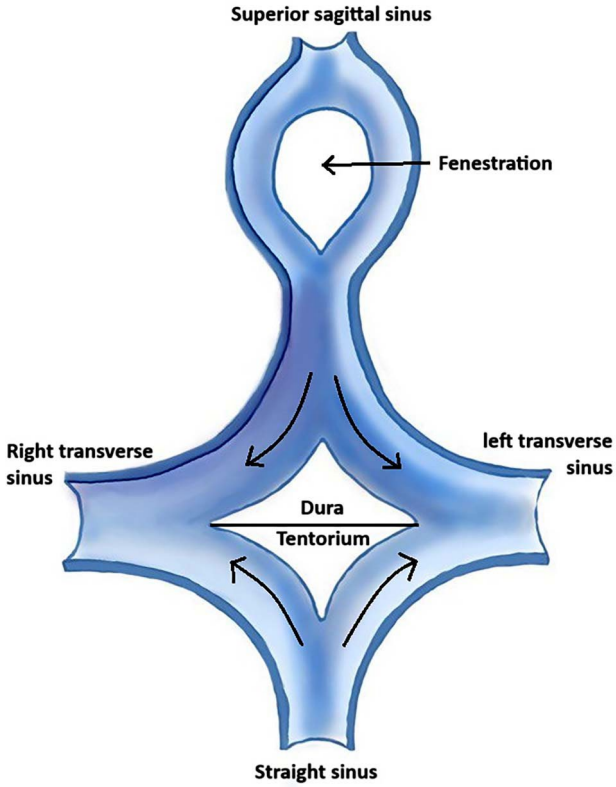

Figure 5. Schematic representation of the fenestration of the superior sagittal sinus in the form of a ring.

Ischemic disorders in cerebral artery basins can also be included in the differential series. Thus Shafer and Wolff[11] described in 1978 two patients with neurological deficit and focal abnormalities on static brain imaging. Upon examination, infarction in the posterior cerebral artery distribution zone was suspected. However, a posterior flow study revealed the cause of the abnormality as a fenestrated anomaly of the sagittal sinus, at which time the term "sagittal sinus fenestration" was first used. According to their findings, the hemodynamic alterations caused by SSS fenestration, particularly the resultant venous hypertension, may have induced perfusion deficits in vulnerable watershed areas between posterior cerebral artery and middle cerebral artery vascular territories, producing imaging characteristics indistinguishable from thromboembolic infarction.[11]

Rădoi et al.[12] documented a remarkable venous anatomical variant characterized by bilateral agenesis of the TSs coupled with fenestration of the SSS. This unusual vascular configuration deviates from standard neuroanatomical paradigms of cerebral venous drainage and presents significant diagnostic challenges, particularly in its differentiation from pathological entities such as dural sinus thrombosis or congenital venous malformations. The absence of TS necessitates compensatory venous drainage through hypertrophy of the occipital and marginal sinuses, illustrating the remarkable adaptive capacity of intracranial venous collateral pathways.

There are cases of other dural sinus anomalies in the literature that present a diagnostic problem. McComiskey and Glikstein[13] described a differential problem with giant AG and sinus thrombosis, with TS fenestration. The presence of a giant AG may be misdiagnosed as dural sinus thrombosis. Arachnoid granulations can be distinguished by their characteristic features: round or oval shape, well-defined edges, and uniform intensity. Thrombosis usually involves the entire sinus segment or several sinuses and may extend to the cortical veins, whereas AG produces focal, well-defined, nodular defects in the sinuses.[6]

The typical MRI finding for dural sinus thrombosis is a thrombus in the sinus, which appears iso- or hyperintense on T1-weighted MRI and hyperintense on T2-weighted MRI. The key MRI characteristic of giant AG is a nonintense lesion with central linear enhancement and surrounding blood flow on contrast-enhanced MRV.[6]

A thrombus located within the dural sinus may show contrast enhancement and result in occlusion of venous blood flow. The presence of focal central linear enhancement in the arachnoid nodule may indicate dilation of endothelium-lined venous sinuses into the granulation gap.[6] The traditional time-lapse MRV method, which does not require the use of paramagnetic contrast agent, has become a reliable and widely used method to evaluate the venous system. However, this method has limitations, including susceptibility to pulsation artifacts, in-plane saturation effects, and spin dephasing when laminar flow is disturbed.[4]

To our knowledge, this represents the first documented case of an annular fenestration anatomically partitioning the SSS. This novel variant poses significant diagnostic challenges by potentially mimicking the radiological appearance of sinus thrombosis through pseudopathological

flow voids, while simultaneously presenting surgical risks due to the reduced wall integrity of fenestrated segments that may predispose to iatrogenic tearing during dural manipulation. The potential for intraoperative misinterpretation of the fenestration as thrombus or vascular malformation further compounds these challenges, as it could prompt unnecessary surgical intervention.[2]

The annular configuration of the SSS fenestration in our case is unprecedented in the literature. Unlike thrombotic occlusion, which typically involves segmental flow disruption and hyperintense T1/T2 thrombus signals,[1,10] fenestration preserved normal flow dynamics, as confirmed by 3D-TOF MRI. The AG further complicated interpretation, as giant AGs (>15 mm) may mimic thrombosis but are distinguished by their round morphology, CSF-intensity core, and lack of contrast enhancement.[6,13] Notably, the patient's transient symptoms (headache and leg weakness) lacked classic thrombotic features (e.g., seizures and papilledema),[10] supporting a nonthrombotic etiology. The congenital anomaly of the SSS that we described was most likely an incidental finding and does not appear to be related to the patient's symptoms.

From a practical standpoint, this case highlights several critical considerations. First, SSS fenestration should be suspected when imaging reveals thrombosis-like defects, particularly pseudodelta signs, in patients lacking classic thrombotic symptoms (headache with seizures or papilledema) or risk factors. Second, 3D-TOF MRI must be prioritized as the first-line diagnostic tool to confirm preserved venous flow, while contrast-enhanced MRV serves as an adjunct for evaluating endothelial integrity when findings remain ambiguous. Importantly, reliance on noncontrast CT alone should be avoided due to its high false-positive rate for thrombosis. For asymptomatic cases similar to ours, no intervention is required beyond periodic monitoring when large AGs (>15 mm) are present. However, should surgical intervention be contemplated for other reasons, preoperative venography becomes mandatory to prevent iatrogenic injury to fenestrated segments. Clinicians can reassure patients and families that such anatomical variants are typically incidental findings; however, their recognition remains crucial to prevent unnecessary anticoagulation or invasive procedures. When evaluating potential SSS pathology, key discriminators include preserved flow in fenestrations versus flow void loss in thrombosis, isointense T1 signal versus thrombus hyperintensity, and peripheral rim enhancement versus the central linear pattern observed in AGs.

In conclusion, the precise etiology of the patient's acute neurological symptoms remained undetermined, as neither the identified SSS fenestration nor AG adequately accounted for the transient motor deficits. While speculative, potential mechanisms may include localized CSF dynamics alterations or venous hypertension-induced reversible neural dysfunction, warranting further hemodynamic investigations. This case highlights the diagnostic significance of annular SSS fenestration, a novel anatomical variant, which may radiologically simulate sinus thrombosis. The findings emphasize the critical need for advanced neuroimaging modalities, particularly 3D-TOF MRI, to reliably distinguish such congenital venous anomalies from true pathological states, thereby preventing inappropriate therapeutic interventions including unnecessary anticoagulation. This anatomical configuration demonstrated radiological features indistinguishable from the pathognomonic "empty delta sign" of dural sinus thrombosis, thereby establishing high-resolution MRV as an essential diagnostic modality for accurate characterization.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: Idea/concept: S.K.; Design, control/supervision, data collection and/or processing, analysis and/or interpretation, literature review, writing the article, critical review, references and fundings, materials: A.I.

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