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Case Report

Large arachnoid granulation protruding into the transverse sinus: A probable cause of intermittent otologic symptoms *

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ABSTRACT

A 69-year-old woman suffered attacks of hearing disturbance and vertigo for seven years. Her otologic and ophthalmological examinations did not show any significant findings. Cerebral magnetic resonance imaging revealed a cystic mass in the left cerebellar convexity. Computed tomography demonstrated a contrast defect of the distal left transverse sinus. Magnetic resonance imaging revealed a cyst protruding into the transverse sinus, and enlarging in the supine. Cerebral angiography demonstrated a congestive venous flow in the left transverse sinus, at the upstream of the cyst. At rest, the venous sinus pressure was 13 cm H2O at the upstream of the cyst and 8 cm H2O at the downstream. When the patient held a breath, the upstream pressure increased to 37 cm H2O, while the maximal downstream pressure was 22 cm H2O. A large AG protruding into the cranial dural sinus may cause intermittent venous congestion and associated otologic symptoms. Movements accompanied by a transient decrease in cardiac venous return and changes in head position can attribute to an enlargement of such AG.

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Introduction

Idiopathic intracranial hypertension (IIH) is an entity assumed to develop due to excessive amount of cerebrospinal fluid (CSF) in and around the brain. It frequently causes otologic symptoms such as vertigo and tinnitus, in addition to headache, nausea, and visual impairment [1]. There are large arachnoid granulations (AGs) occasionally identified to protrude into the lumen of the cranial dural sinuses [2–4]. These AGs have been documented to cause symptoms of IIH [5–8]. Recently, endovascular venous sinus stenting has been reported to be effective for the treatment of such AGs [9]. However, to the best of our knowledge, there has not been a study exploring an association between large arachnoid granulation protruding into the cranial dural sinus and flow dynamics of it.

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Case report

A 69-year-old woman presented with recurrent episodes of transient hearing disturbance and vertigo for 7 years. They consistently developed immediately after posture changes, not depending on specific head positions, and spontaneously resolved in 20 minutes. At the presentation, she did not show any focal neurological deficits. Her body mass index was 25.7 kg/m² with 7.5 kg of weight gain over the last 5 years. The otologic and ophthalmological examinations did not show any significant findings. Cerebral magnetic resonance imaging showed a round mass in the left cerebellar convexity. It appeared hypointensity on T1-and diffusionweighted sequences and inhomogeneously hyperintense on T2-weighted imaging. Post-contrast computed tomography (CT) scans demonstrated a localized contrast defect in the distal part of the left transverse sinus with engaged cortical veins in it. The constructed interference steady-state imaging revealed the cystic mass, occupying the lumen of the transverse sinus, and was consistent with an AG involving the herniated brain. Its maximal dimension changed from 10.9 \times 8.2 mm in the supine to 12.8 \times 10.2 mm in the prone (Fig. 1). Cerebral angiography showed congestive venous flow in the left transverse sinus, at the upstream of the AG. During the examination, venous sinus pressures were measured with a microcatheter (PhenomTM $27^{(R)}$, Medtronic plc, Watford, United Kingdom), at the upstream and downstream of the AG, deployed through the contralateral transverse and ipsilateral sigmoid sinus, respectively (Fig. 2). At rest in the supine, the farmer pressure was 13 cm H2O, while the latter was 8 cm H2O. When the patient held a breath for 10 seconds, the former pressure increased to 37 cm H2O, while the latter pressure was maximally 22 cm H2O. Furthermore, the patient underwent a lumbar cerebrospinal fluid (CSF) puncture in the left lateral position. The opening pressure was measured at 19 cm H2O and a CSF tap did not show any changes in her symptoms. Currently, an endovascular venous sinus stenting is being scheduled for the treatment of the patient.

Discussion

In this case, congestive venous flow was angiographically demonstrated in the transverse sinus, at the upstream of the AG obstruction site. Also, a pressure gradient was detected between the upstream and downstream of the obstruction

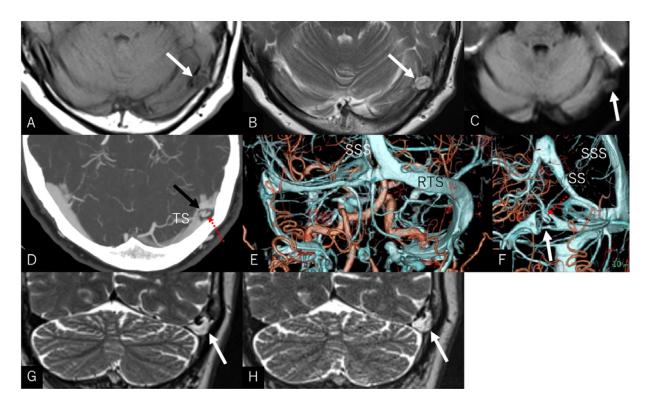


Fig 1 – (A–C) Axial T1- (A), T2- (B), and diffusion-weighted (C) magnetic resonance imaging shows a round mass in the left cerebellar convexity, appearing hypointensity on T1-and diffusion-weighted sequences and inhomogeneously hyperintense on T2-weighted imaging (arrow). (D–F) Postcontrast axial computed tomography (CT) scan (D) and 3-dimensional CT angiography, posterior (E) and left lateral (F) views, show a localized contrast defect in the distal part of the left transverse sinus (D and F, arrow) with engaged cortical veins in it (D and F, dashed arrow). (G, H) Coronal constructed interference steady-state imaging show that the cystic mass, occupying the lumen of the distal transverse sinus, is consistent with an arachnoid granulation involving the herniated brain (*arrow*). The maximal dimension of it is 10.9 x 8.2 mm in the supine (G), while 12.8 x 10.2 mm in the prone (H). RTS: right transverse sinus; SS: straight sinus; SSS: superior sagittal sinus; TS: transverse sinus.

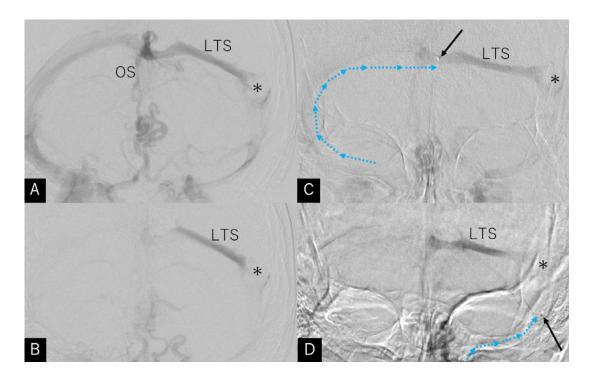


Fig. 2 – (A, B) Anteroposterior views of the left vertebral angiography, venophase (A), and later venophase (B), show congestive venous flow in the left transverse sinus (LTS), at the upstream of the arachnoid granulation involving herniated brain (asterisk). OS: occipital sinus. (C, D) Anteroposterior views of the left vertebral angiography, venophases, show measurements of the venous sinus pressure at the upstream (C) and downstream (D) of the arachnoid granulation (asterisk), through the contralateral transverse sinus and ipsilateral sigmoid sinus, respectively. Arrow: tip of the microcatheter; *Dashed arrows*: route of the microcatheter through the dural sinuses at measuring the pressure.

site. At rest, the gradient was 5 cm H2O, while increased to 15 cm H2O when the patient held a breath that retrogradely increased the intracranial pressure through a decrease in venous return to the right atrium. Furthermore, when changing the position of the patient's head from supine to prone, dimension of the offending AG enlarged. On the other hand, the patient's symptoms were intermittent and consistently developed immediately after posture changes. Therefore, we assumed that the symptoms were caused by venous congestion in the transverse sinus. It might be developed in association with the movements causing a transient decrease in cardiac venous return and changes of head position causing enlargement of the AG. The patient had gained 7.5 kg in the last 5 years that might partly attribute to the development of venous congestion. Our patient presented with intermittent hearing disturbance and vertigo, while common AG-associated otologic symptoms have been documented as pulsatile tinnitus and vertigo [5,6,9]. Benign paroxysmal positional vertigo is known to be highly prevalent in elderly people [10]. However, based on careful observations of the clinical picture and the above assumptions, we considered that the patient's otologic symptoms were caused by an AG protruding into the transverse sinus and benign paroxysmal positional vertigo could be excluded. The present patient did not show any changes in symptoms after a lumbar CSF tap. This might be mainly derived from the property of the AG involving herniated brain tissue, not a CSF-filled cyst, which did not result in a remarkable regression after the tap.

A large AG protruding into the cranial dural sinus may cause intermittent venous congestion and associated otologic symptoms. Movements accompanied by transient decreases in cardiac venous return and changes in head position affecting the dimension of such AG may be associated with the symptoms.

Ethical standards

We declare that all procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and the 1964 Declaration of Helsinki and its later amendments.

Author contributions

All the authors contributed equally to the study.

Patient consent

We obtained written patient consent to publish this case report.

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