

İNSİDENTAL ARACHNOİD GRANÜLASYON: VERTİGONUN NADİR BİR SEBEBİ

Incidental Arachnoid Granulation: A Rare Cause Of Vertigo

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ÖZET

Araknoid granülasyonlar, araknoid membranın dural sinüsler içine doğru büyümesidir. Bu oluşumlar, dural sinüsleri dolduracak ve genişletecek kadar büyüyebilir veya kafatasının iç sahasına açılabilir. Biz burada, bir haftadır başdönmesi şikayeti ile başvuran 56 yaşında erkek bir hasta sunuyoruz. Nörolojik ve göz dibi muayeneleri normal idi. Beyin bilgisayarlı tomografi taraması pariyetal kemiğin iç sahasında bilateral birden fazla litik alanlar gösterirken; bu lezyonlar manyetik rezonans görüntüleme (MRG) kontrast tutmayan tarzda olup dural sinüs tıkanıklığı ile ilişkili değildi. Başdönmesi vestibulosuppressan ilaçlar ile geriledi. Hiçbir müdahale planlanmadı ve hasta MRG çalıřmaları ile takip edildi.

Anahtar kelimeler: Araknoid zar; Litik kemik lezyonu; Manyetik rezonans görüntüleme; Çok kesitli bilgisayarlı tomografi; Baş dönmesi

ABSTRACT

Arachnoid granulations are growths of arachnoid membrane into the dural sinuses. They may grow to fill and dilate the dural sinuses or expand the inner table of the skull. We report a 56-year-old man who presented with a one week history of vertigo. Neurological and fundoscopic examinations were normal. Brain computed tomography scan showed multiple lytic areas on the inner table of the parietal bone bilaterally whereas the lesions were not related to the occlusion of the dural sinuses on magnetic resonance imaging (MRI) without contrast enhancement. The vertigo resolved with vestibulosuppressant drugs. No intervention was planned and the patient was followed up through MRI studies.

Keywords: Arachnoid membrane; Lytic bone lesion; Magnetic resonance imaging; Multidetector computed tomography; Vertigo

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INTRODUCTION

Arachnoid granulations (AGs) are growths of arachnoid membrane into the dural sinuses through which CSF enters the venous system (5). They normally measure a few millimeters but may grow to fill and dilate the dural sinuses or expand the inner table of the skull (9). We present a case of a patient who presented with a one week history of vertigo that is unlikely related to bilateral parietal bone AGs.

CASE REPORT

A 56-year-old man was admitted to the hospital with a one week history of vertigo attack. The patient was having problems while turning to right and left with a vague tinnitus on his left ear. He has not experienced similar attack before. He had no history of either systemic diseases or medication usage. He underwent a complete audio-vestibular test battery revealing no abnormality. The results of the laboratory tests were within the normal ranges. The neurological and general physical examinations were normal. Visual field and acuity were normal. Fundoscopic examination findings were normal. Brain computed tomography (CT) scan showed multiple lytic areas on the inner table of the parietal bone bilaterally (Figure 1). Magnetic resonance imaging (MRI) showed frontal uncalcified AGs bilaterally without contrast enhancement (Figure 2). These lesions were isointense relative to CSF in all images. Occlusion of the dural sinuses was not detected on MRI. Lumbar puncture was performed and the opening pressure was within the normal range (160 mm H₂O). The vertigo resolved with vestibulosuppressant drugs. No intervention was planned and the patient was followed up through MRI studies.

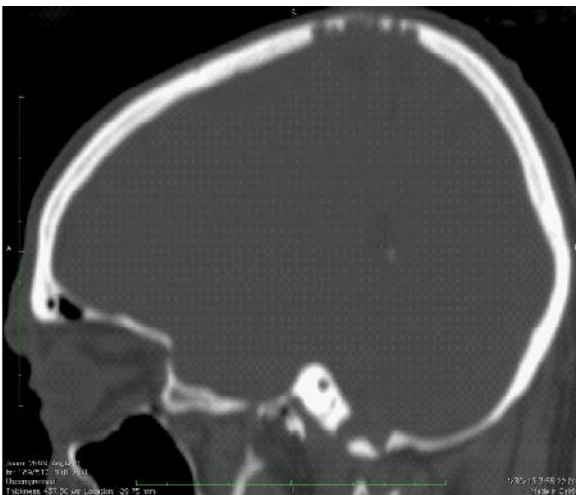


Figure 1. A sagittal reformatted cranial tomography image shows parasagittal defects indenting the inner table of the parietal skull.

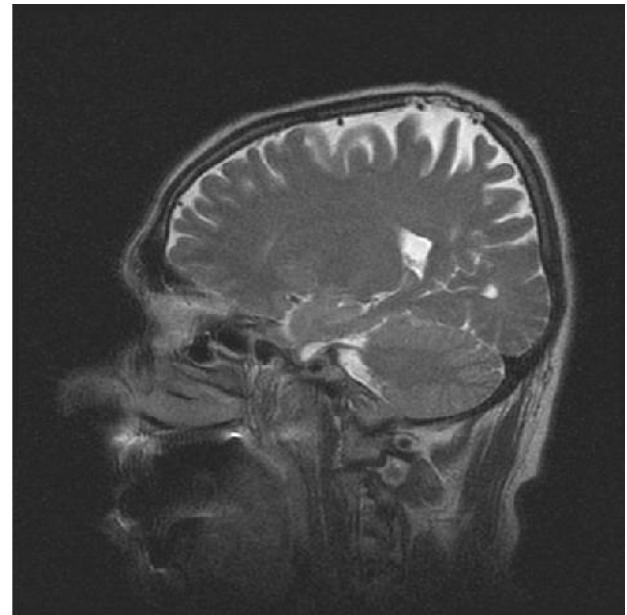


Figure 2. A sagittal T2-weighted magnetic resonance image shows high-signal-intensity lesions protruding into the parietal inner table.

DISCUSSION

The growth of arachnoid membrane into the dural sinus (glandulae conglobatae) was first described by Pacchioni in 1705 (2). These projections are called arachnoid villi or AGs (pacchionian bodies), depending on their size. Arachnoid villi are microscopic, whereas granulation is visible to the naked eye (5). They function as a passive filtration system for cerebrospinal fluid (CSF), providing a pathway from subarachnoid space into the venous system. AGs increase in number and enlarge with age probably in response to CSF pressure and can expand the inner table of the skull (5). Occasionally, they even expand into the diploic space and eventually involve the outer table, mimicking osteolytic lesions (8,9). Erosion of the bone is not clinically significant unless it is located near pneumatized parts of the skull. It can lead to CSF leakage. Erosions are generally in the anterior parietal bone and posterior frontal bone, usually within 3 cm of the midline (8). Similarly, in our case the location of the AGs was the bilateral parietal bone near the vertex. Giant AGs are much larger than normal AGs, approximately 10 mm in diameter (4). Most of the AGs were reported to be spherical or finger-like in shape (5). AGs can be classified into two types: single and lobulated type (5). In our case, AGs had a spherical shape and a lobulated type.

They are generally asymptomatic, which could probably be explained by their slow growth. Moreover, they can be often discovered incidentally in the sinuses in particular transverse and posterior superior sagittal sinus and skull (5). Sometimes, they can grow substantially and occlude the dural sinuses and rarely cause symptoms of increased intracranial pressure prompting immediate management (3). The literature shows that the common complaint of the patients was headache (1,6). Giant AGs were often associated with pseudotumor cerebri with headache, vertigo and blurred vision due to papilledema (1,10). Isolated vertigo attack was not defined before in the literature. Since the opening pressure was within the normal range in lumbar puncture and the size of AGs was not giant, we unlikely explain the patient's vertigo on this ground. So, the mechanism of his vertigo is yet unclear.

Differential diagnosis of an AG can be dural sinus thrombosis, inclusion tumor, meningocele, dermoid, epidermoid, cavernous hemangioma, or meningioma (9). These can be excluded by their characteristic imaging findings. In MRI, AGs appear hypointense or isointense relative to the brain on T1-weighted and hyperintense on the T2-weighted images, showing minimal heterogeneous contrast enhancement (7). Relative to CSF all granulations are isointense on T2-weighted images, while almost all are isointense both on T1-weighted images and fluid attenuated inversion recovery (FLAIR) images. In diffusion-weighted images, all AGs showed isointensity to normal brain tissue, which was higher than the reported signal intensity of arachnoid cysts and lower than that of epidermoids (7). However, it may be difficult to differentiate AGs from dermoids, epidermoids, hemangiomas and other lesions presenting signal intensities similar to CSF. Dermoid and epidermoid cysts frequently involve both the inner and outer tables, hemangiomas and eosinophilic granulomas are located mainly in the intradiploic space and rarely involve the inner table (8). In contrast, AGs determine an impression on the inner table and only in few cases involve the outer table. Similarly, in our case, the inner table of the parietal bone bilaterally was involved. As AGs can involve dural sinuses, differential diagnosis should also include sinus thrombosis and intrasinus tumors. Nevertheless, AGs are not hyperintense on FLAIR images, although lesions usually hyperintense on T2-weighted are thought to be hyperintense on FLAIR images with the exception of epidermoids (7). When suspected prominent AGs are noted, FLAIR images should help to differentiate granulations from the dural sinus or skull lesions, mainly from the epidermoids. In our patient, MRI showed lobulated extraaxial cystic lesions, isointense relative to the CSF in all images, were located on the parietal bone bilaterally near the vertex without contrast enhancement. These lesions were not related to the occlusion of the dural sinuses. Because of the incidental diagnosis and the absence of related symptoms, no intervention was planned and the patient was followed up through MRI studies.

Little is known about the nature of the related symptoms and no long-term follow-up studies which can help to discern if symptoms are related to radiological findings have been performed. Further studies are required to improve our understanding of the possible roles of AGs in pathological conditions.

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