Eur Radiol (2008) 18: 1232–1243
DOI 10.1007/s00330-008-0882-4



**Ilona M. Schmalfuss**
**Morgan Camp**

# Skull base: pseudolesion or true lesion?

Received: 8 November 2007
Revised: 28 November 2007
Accepted: 14 December 2007
Published online: 13 February 2008


I. M. Schmalfuss
North Florida/South Georgia Veterans Administration,
Gainesville, FL, USA

I. M. Schmalfuss (✉) · M. Camp
Department of Radiology,
University of Florida,
P.O. Box 100374,
1600 SW Archer Road,
Gainesville, FL 32605, USA
e-mail: schmai@radiology.ufl.edu
Tel.: +1-352-3761611
Fax: +1-352-3781267

**Abstract** The skull base is a complex anatomical structure. Therefore, radiologists often use "side-to-side" comparison for detection of abnormalities. This approach is compromised by the high frequency of anatomical variations involving the skull base and the common presence of flow-related artifacts within vessels at the skull base that might mimic true lesions. The spectrum of "pseudolesions" ranging from different anatomical variations, such as unusual arachnoid granulations, asymmetric pneumatization and/or appearance of neurovascular foramina, to flow-related artifacts will be discussed in this review article, and tips for their distinction from a true lesion in a similar location will be given.

**Keywords** Skull base · Normal variants · Pseudolesions · Symmetric pneumatization · Symmetric skull base foramina

## Introduction

Radiologists often use "side-to-side" comparison to determine if a structure is normal or abnormal in the head-and-neck region. This is in particular true when an anatomical structure is complex in nature and when the radiologist is unfamiliar with its anatomy. This is frequently the case with the skull base as true lesions in such a location are relatively uncommon. Thus, trainees get limited experience with these problems during training, even in excellent neuroradiology teaching programs. The issue is even more complicated by the fact that the skull base is predisposed to a variety of anatomical variations and flow-related artifacts within the vessels coursing through the skull base.

Asymmetric pneumatization is one of the most commonly encountered challenges, raising the question if a lesion is present or not and if pneumatization beyond the "mother" bone could become clinically significant and when. Flow-related artifacts and asymmetric size of neurovascular foramina are certainly the second most common problem with which the radiologist is faced. If the patient is asymptomatic, the distinction from a true lesion may be easier, or one would recommend a follow-up examination in ambivalent cases to ensure stability. Conversely, when the patient is symptomatic, the interpreting radiologist is in a dilemma, because most skull base "lesions" are not accessible to percutaneous biopsy. This review article will discuss the common location of flow-related artifacts, as well as common anatomical variations, their frequency and clinical significance, and will provide tips for the interpreting radiologist to answer the question: pseudolesion or true lesion?

Variations in pneumatization

*Temporal bone*

Significant variations in temporal bone pneumatization are commonly seen. Non- or poorly pneumatized mastoid air cells are most frequently encountered. Sometimes this is mistaken for an underlying lesion-even if asymmetric in distribution-as benign or malignant bony lesions infrequently involve this particular portion of the skull base. Differentiation from an infectious process is typically not a dilemma either as the poorly pneumatized mastoid air cells have the typical appearance of dense bone on CT or are lower in attenuation on T2-weighted images than expected for fluid/pus.

Pneumatization of the petrous apex can be observed in 35% of patients [1]. Usually, it is bilateral and symmetric in distribution. Only when asymmetric it may cause a diagnostic dilemma, in particular on magnetic resonance imaging (MRI). Typically, the non-pneumatized petrous apex is incorrectly interpreted as a petrous apex lesion, such as cholesterol granuloma or cyst, congenital cholestatoma or petrous apicitis. The distinction between normal and abnormal can usually easily be made with computed tomography (CT) where the persistent internal bony structure, preservation of the cortex and lack of mass effect are readily visualized. On MRI, the presence of signal intensity within the non-pneumatized petrous apex may be confusing (Fig. 1). Hence, close attention to the signal characteristics is critical to lead to the correct diagnosis. The typically seen increased T1 signal intensity of fatty bone marrow within the non-pneumatized petrous apex leaves cholesterol granuloma as the only differential diagnostic option that can be usually quickly ruled out due to lack of mass effect and/or lack of suppression on fat-suppressed sequences (Fig. 2). In addition, the T1 signal intensity of a cholesterol granuloma is often even higher in signal intensity than the surrounding fatty bone marrow (Fig. 2). Occasionally, a pneumatized petrous apex that is filled with fluid and/or mucosal thickening may be mistaken for a petrous apicitis (Fig. 3). On CT, the preservation of septations in the petrous air cells, with lack of fragmentation, usually yields the correct diagnosis (Fig. 3). In addition, patients with petrous apicitis are

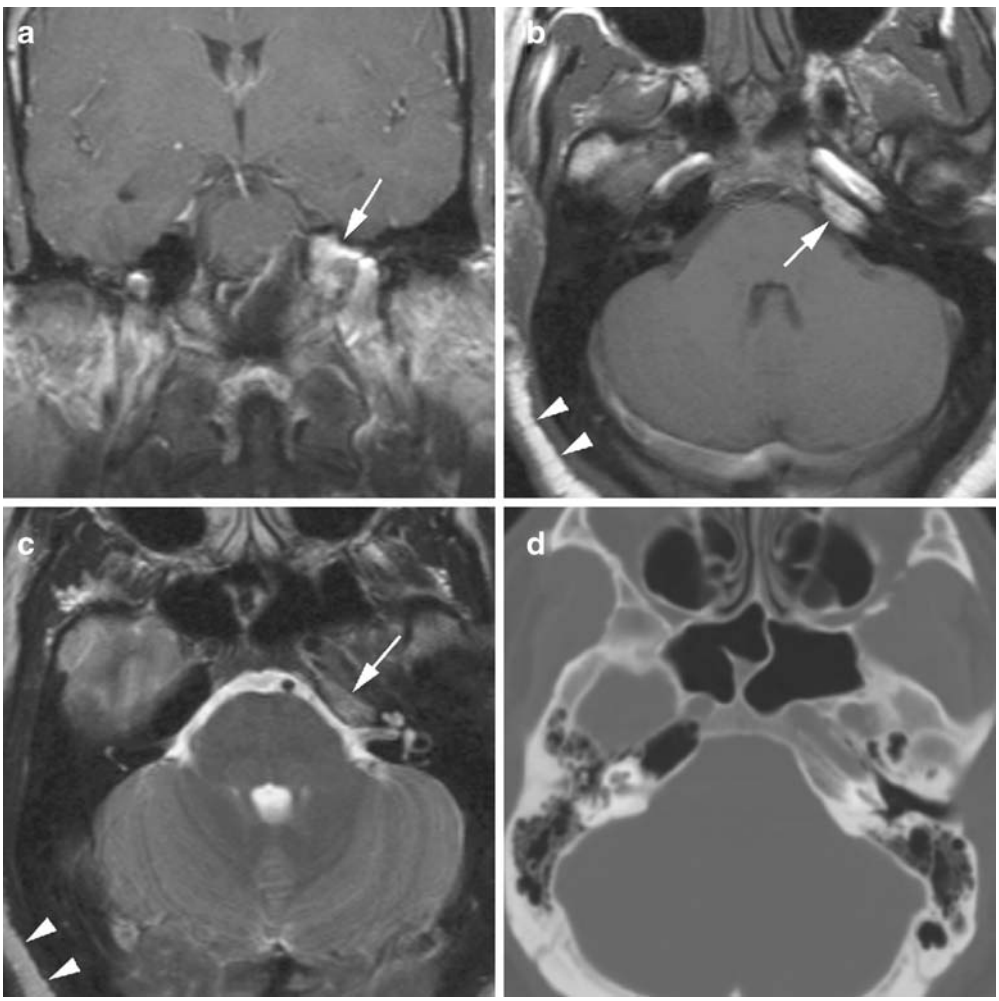


**Fig. 1** The coronal gadolinium-enhanced T1-weighted image (**a**) demonstrates very asymmetric appearance of the petrous apices with an "enhancing lesion" (arrow in **a**) seen on the left side. Careful evaluation of the axial non-contrast-enhanced T1- (**b**) and T2- (**c**) weighted images clarifies that this appearance is caused by asymmetric aeration of the petrous apices. An "enhancing mass" can be clearly excluded as increased T1 signal intensity (arrow in **b**) and intermediate T2 signal intensity (arrow in **c**) are seen on the non-contrasted T1- (**b**) and T2- (**C**) weighted images-signal characteristics similar to visible subcutaneous fat (arrowheads in **b** and **c**) and therefore consistent with fatty bone marrow. The lack of a petrous apex mass and the asymmetric aeration were confirmed with CT (**d**)

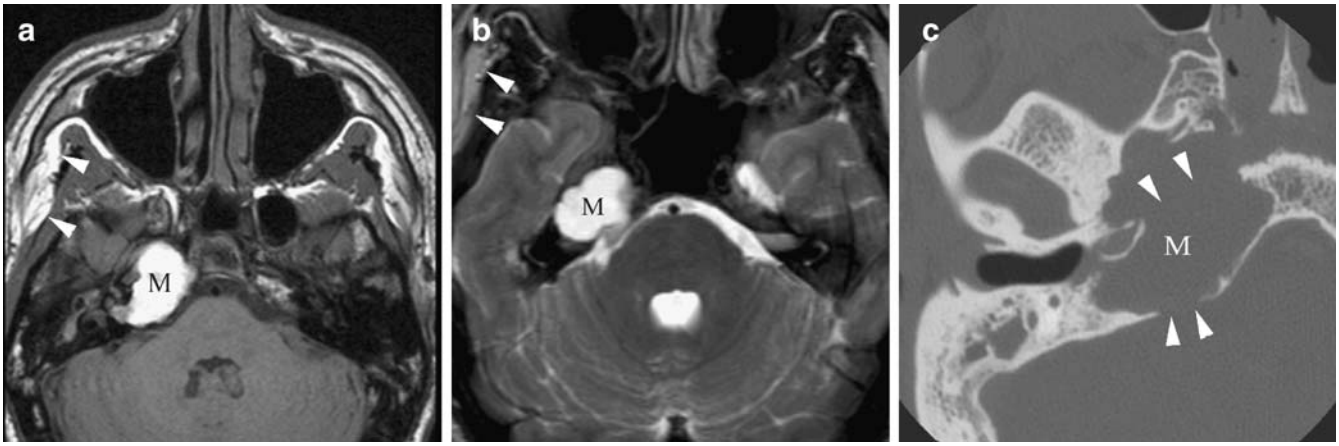


**Fig. 2** Axial non-contrast-enhanced T1 (**a**), T2 (**b**) and CT (**c**) images through the petrous apex in a patient with a pathologically proven cholesterol granuloma (M for mass) nicely demonstrating the distinguishing features to non-aerated petrous apex filled with bone marrow as seen in Fig. 1. The differentiating imaging characteristics include: higher T1 and often T2 signal intensity in comparison to normal fat (arrowheads in **a** and **b**) and presence of mass effect, which is associated with bony disruption (arrowheads in **c**), as best seen on the axial CT image (**c**)

typically very symptomatic and usually show associated involvement of the middle ear cavity, mastoid air cells and/or adjacent dura (Fig. 3). On MRI, the intermediate T1 and high T2 signal intensity of a fluid-filled, pneumatized petrous apex may be mistaken for chondrosarcoma that usually shows similar signal characteristics on those sequences. However, the lack of mass effect and of contrast enhancement should lead to the correct diagnosis.

*Clinoid and pterygoid processes*

Pneumatization of the clinoid or pterygoid processes by the paranasal sinuses, most commonly from the sphenoid sinuses and rarely from the ethmoid air cells, is a well-recognized process occurring in approximately 10% of patients [2, 3]. Typically, it is of no clinical significance; however, it may be mistaken for an orbital apex, cavernous sinus or suprasellar cistern lesion if asymmetric pneumatization occurs. On CT, this anatomical variation is easily recognized in contrast to MRI, where most commonly the non-pneumatized process or the pneumatized process containing sinus disease is mistaken for a soft-tissue mass. In case of a non-pneumatized process, observation of high T1 signal intensity of fatty bone marrow without mass effect is very helpful. In the instance of a pneumatized process that is involved by sinus disease, the evaluation of the signal characteristics can be misleading as dried-out secretions, fungal colonization or proteinaceous material may assume variable signal intensities on the different sequences. Here, evaluation of the extent of disease may lead to the correct diagnosis, as isolated involvement of a pneumatized process without ipsilateral sphenoid sinus and/or ethmoid air cell disease rarely occurs.

In patients that require clinoidectomy for paraclinoid or upper basilar artery lesion resection, it is imperative to report that a clinoid process is pneumatized. During such surgery, the clinoid process is removed to allow better access to the paraclinoid and/or the upper basilar artery region. Therefore, if a pneumatized clinoid process is resected, the patient will be placed at risk to develop post-surgical cerebrospinal fluid leakage via the persistent connection of the pneumatized clinoid process to a paranasal sinus [2].

*Paranasal sinuses*

An enlarged, air-filled paranasal sinus is easily recognized on the various imaging modalities and extremely rarely mistaken for an underlying lesion; however, it raises the question of what degree of pneumatization is normal and if excessive pneumatization is of clinical significance. A hyperpneumatized paranasal sinus can be caused by a spectrum of entities, ranging from hypersinus, pneumosinus dilatans to pneumocele. The terminology, in particular of the last two entities, is confusing as both terms are often used interchangeably [4]. Clarification of the definitions is, however, critical as each of these abnormalities carries different clinical significance. CT is the study of choice to help differentiate these entities from each other.

Hypersinus represents an air-filled sinus that is larger than typically seen, but does not extend beyond the normal boundaries of the "mother" bone and shows normal thickness of its bony walls [5]. It is clinically asymptomatic and does not extend intracranially or cause encroachment upon adjacent anatomical structures [5, 6]. Therefore, it truly represents an anatomical variation and is of no clinical significance.

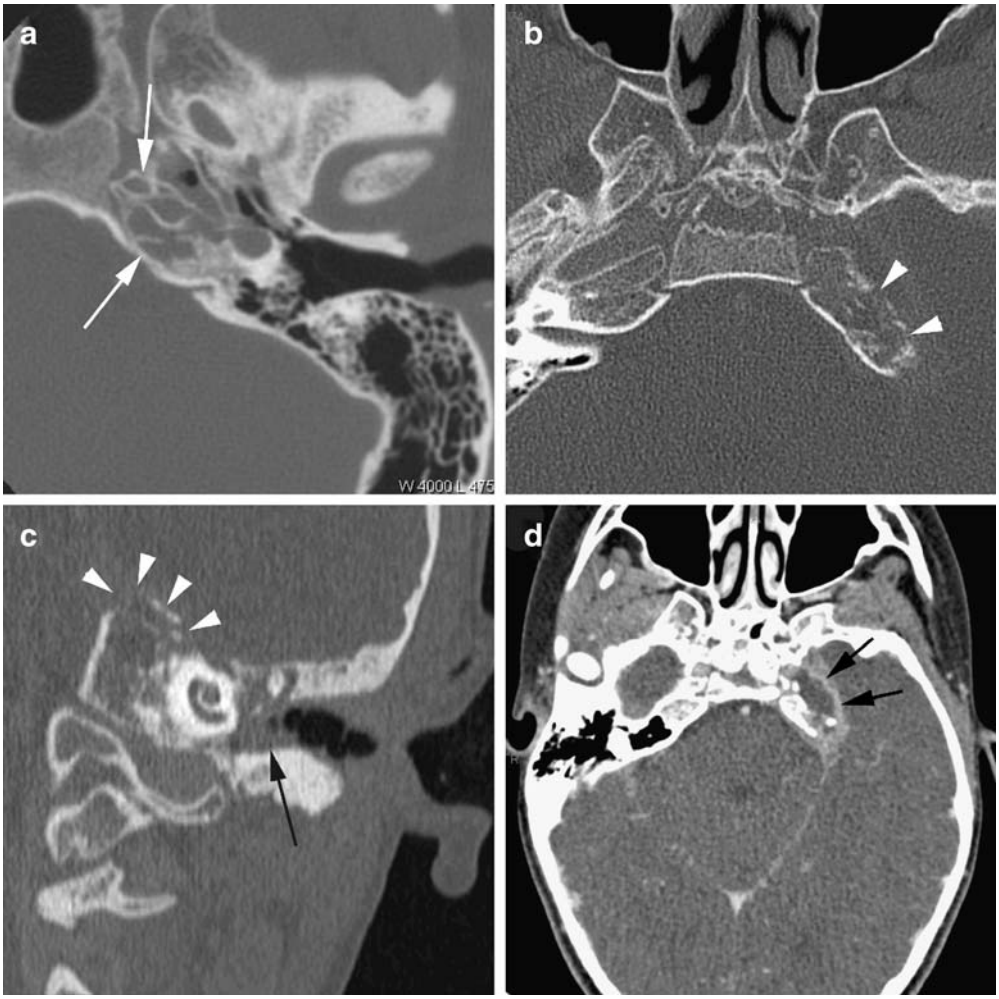


**Fig. 3** Axial CT image in bone window (**a**) demonstrates almost complete opacification of the petrous air cells with fluid and/or mucosal thickening (arrows in **a**). The bony trabecula between the different air cells as well as the cortex remain preserved in contrast to the axial (**b**) and coronal CT (**c**) images of a patient with petrous apicitis showing marked fragmentation of the petrous apex cortex and adjacent air cells (arrowheads in **b** and **c**). Patients with petrous apicitis often have intracranial involvement at the time of presentation as in this case with presence of a small epidural abscess (arrows in **d**) on this contrast-enhanced axial CT image (**d**). Also note the associated middle ear involvement (arrow in **c**)

Pneumosinus dilatans is an abnormally expanded, air-filled paranasal sinus that extends beyond the margins of the "mother" bone, but shows normal thickness of the sinus walls [7]. Most commonly, this is an idiopathic condition; however, it has also been described in association with other disorders, such as cerebral hemiatrophy or large middle cranial fossa arachnoid cyst, in particular following surgical drainage (Fig. 4) [8, 9]. In these cases, the

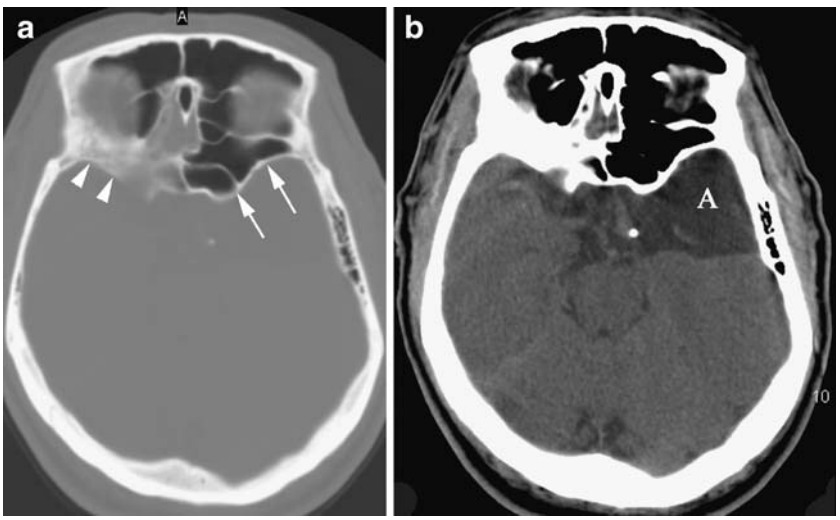


**Fig. 4** Axial CT images displayed in bone (**a**) and soft tissue (**b**) windows show marked hyperaeration of the left frontal sinus that is extending beyond the "mother bone" to involve portions of the greater sphenoid wing (arrows in **a**). There is no evidence of thinning of the involved walls. This is consistent with pneumosinus dilatans associated with an underlying arachoid cyst in this particular patient (**a** in **b**). Arrowheads in A = normal greater sphenoid wing on right

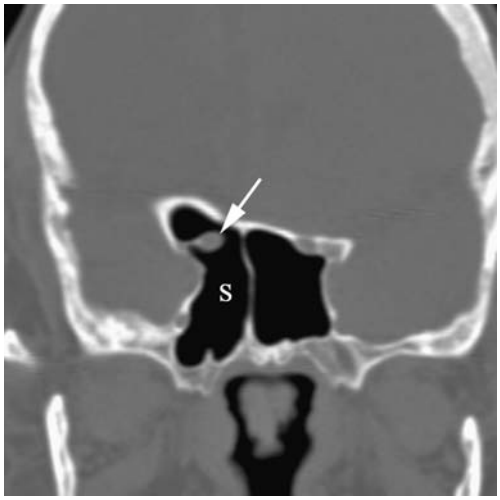


**Fig. 5** Coronal CT image displayed in bone window shows extensive pneumosinus dilatans on the right side with the hyperaerated sphenoid sinus (S) almost completely surrounding the optic nerve (arrow), placing the patient at increased risk for visual damage during surgical intervention in this area

abnormal expansion of the involved paranasal sinus(es) is likely due to the desire of the skull to maintain its normal shape and to compensate for the intracranial volume loss by skull thickening, elevation of the orbital roof, sphenoid wing and petrous ridge as well as hyperaeration of the paranasal sinus and mastoid air cells as classically seen in Sturge-Weber and/or Dyke-Davidoff-Masson syndromes [10]. Rarely, it can represent an indirect sign of a meningioma originating in or close to the optic nerve canal [11, 12]. Therefore, the radiographic diagnosis of pneumosinus dilatans should initiate the search for an underlying meningioma and/or for the etiology of brain tissue loss. Occasionally, pneumosinus dilatans may engulf the internal carotid artery or the optic nerve (Fig. 5)-a critical radiographic observation when surgery for an underlying meningioma or associated sinus disease is planned.

*Pneumocele* is a rare condition characterized by abnormal expansion of an air-filled sinus that extends beyond the normal margins of the bone of origin and shows focal or generalized thinning of the bony sinus walls [5]. This is a true disease entity as it clinically presents with facial bossing and dull pain. A "ball valve type" mechanism of sinus obstruction at the sinus ostium is considered the most likely etiology leading to trapping of air and increased pressure inside the involved sinus [13]. If untreated, pneumoceles typically slowly progress and may cause displacement of adjacent structures, e.g., optic nerves or cavernous sinus, leading to additional symptoms such as diplopia or vision loss. On MRI, a mucocele with superimposed dried-out secretions and/or fungal colonization may mimic a pneumocele as dried-out secretions and/or fungal manganese deposition results in significant signal drop off on all sequences, assuming the imaging appearance of an expanded, air-filled sinus as seen in pneumoceles.

## Asymmetric size of neurovascular foramina and/or canals

At the skull base level, asymmetric size of venous vascular foramina can be routinely seen as a normal anatomical variation. The jugular foramen represents the best example as it is larger on the right side in the majority of patients [14]. Minor differences may not be noticed or may be considered an anatomical variant; however, significant discrepancies may be misinterpreted as an underling mass

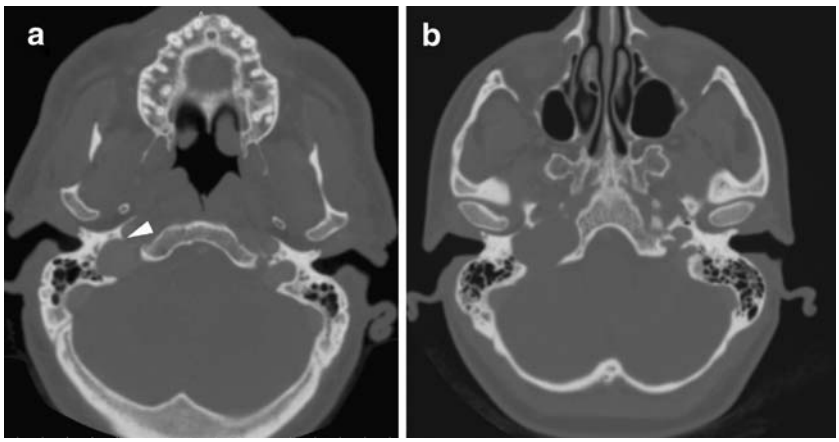


**Fig. 6** Two axial CT images displayed in the bone window of two different patients show asymmetric size of the jugular foramen. The asymmetry in **a** is caused by anatomical variation in size of the jugular fossa with the right side being typically larger, as in this patient. The enlargement of the right jugular foramen in **b** is, however, due to an underlying benign schwannoma. Note that the difference in the size of the jugular foramen is a non-reliable distinction between the anatomical variation and true lesion. Observation of preservation of the jugular spine in normal anatomical variation (arrowhead in **a**) and absence in underlying lesion is usually more accurate

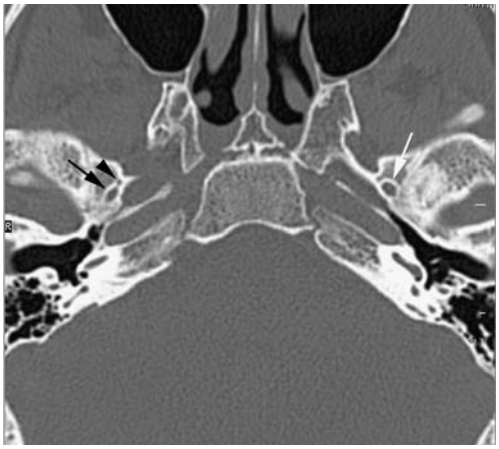

**Fig. 7** Axial CT image through the skull base displayed in bone window shows a normal foramen spinosum on the left (white arrow) and a duplicated foramen spinosum on the right with a smaller anterior foramen (black arrowhead) and a larger, ovoid posterior foramen (black arrow)

(Fig. 6). Evaluation for intact bony boundaries is certainly important in these cases to exclude an aggressive underlying process, such as jugular foramen paraganglioma, which typically shows "mouth-eaten" bony boundaries or an aggressive infection or underlying malignancy causing frank bone destruction. In comparison, the bony boundaries in benign tumors (such as jugular foramen schwannomas) remain intact, making a distinction between a normal anatomical variation and an underlying benign lesion challenging. Here, the assessment of the jugular spine is critical. The jugular spine is a small pointy bony protuberance along the anterior bony margin that partially divides the jugular foramen into a pars venosa and pars nervosa (Fig. 6). It is often foreshortened and/or remodeled in patients with a significant large jugular foramen schwannoma, while it remains preserved in cases of anatomical variants (Fig. 6).

On the contrary, arterial vascular foramina are rarely involved by anatomical variations. Foramen spinosum is likely the most commonly affected arterial vascular foramen. Its appearance is influenced by variations in the course and the origin of the middle meningeal artery. In case of early bifurcation of the middle meningeal artery, a duplicated foramen spinosum (Fig. 7) is present, while it is absent in aberrant origin of the middle meningeal artery, e.g., from the ophthalmic artery or in persistent stapedial artery [15]. In the latter case, the persistent stapedial artery becomes the middle meningeal artery by entering the middle ear cavity through the facial hiatus [14]. Within the middle ear cavity, it runs between the crura of the stapes and enters the facial nerve canal distal to the geniculate ganglion to cause enlargement of the facial nerve canal at the tympanic segment [14, 15]. An enlarged foramen spinosum as an anatomical variation has not yet been described.

Asymmetric size of neural skull base foramina is also rarely seen as an anatomical variation and has been most commonly reported to affect the foramen ovale [15]. Asymmetric size of the foramen ovale has been reported in approximately 30% of individuals with no cases being attributable to disease of the mandibular division of the trigeminal nerve [15]. Several explanation for such an asymmetry of the foramen ovale have been provided, including confluence of the foramen Vesalius (also called sphenoid emissary foramen) with the foramen ovale, coursing of the sphenoid emissary vein through the foramen ovale in the absence of the ipsilateral foramen Vesalius and transmission of an unnamed venous plexus through the foramen ovale [15–18]. Overall, it is currently thought that the amount of venous plexus within the

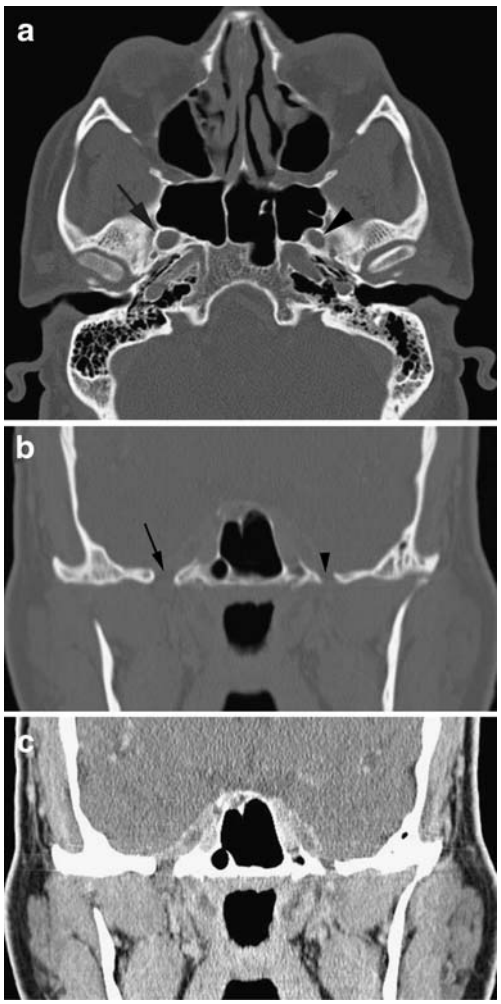


**Fig. 8** Axial (**a**) and coronal (**b**) CT images displayed in bone window demonstrate a markedly enlarged foramen ovale on the right side (arrows in **a** and **b**) in comparison to the left (arrowheads in **a** and **b**), suggesting an underlying mass. The enlargement in this asymptomatic patient is assumed to be due to a prominent perineural venous plexus [15]. This is supported by the symmetrically preserved small fat planes at and below the foramen ovale on the coronal CT image displayed in soft tissue window (**c**)

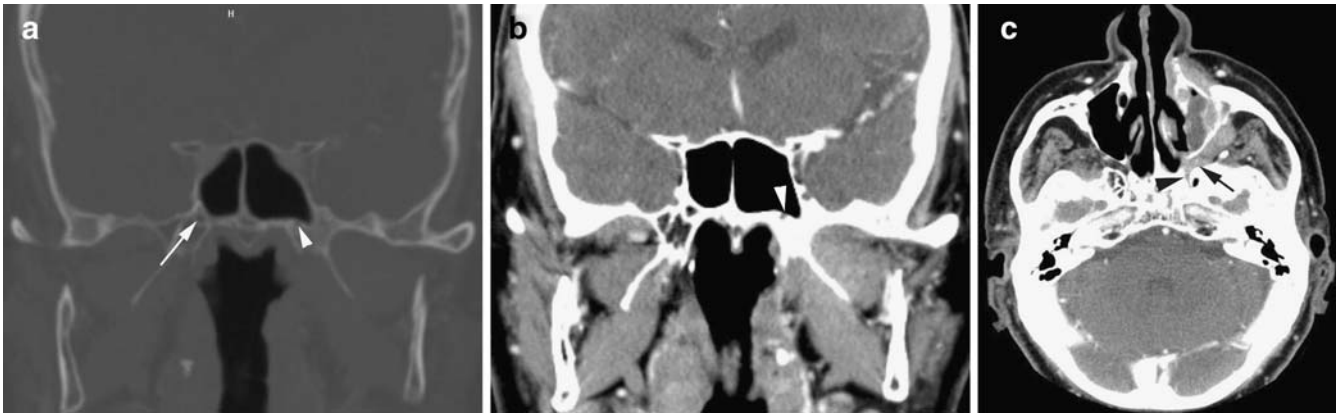


**Fig. 9** Coronal CT image in bone window (**a**) shows similar situation as in Fig. 8 with markedly larger vidian canal on the right (arrow in **a**) when compared to the left (arrowhead in **a**), suggesting that the right vidian canal represents the abnormal neural foramen. The evaluation of the soft tissue windows in coronal (**b**) and axial (**c**) plane, however, reveals complete obliteration of the fat plane in the smaller vidian canal on the left side (arrowheads in **b** and **c**) with associated complete obscuration of the fat plane in the adjacent pterygoid palatine fossa (arrow in **c**). This was consistent with perineural tumor spread from an adenoid cystic tumor arising along the superior alveolar ridge (not shown). These images nicely demonstrate the importance of evaluation of adjacent fat planes and not only of the size of an individual foramina. Courtesy of Dr. F. A Pameijer, Antoni van Leeuwenhoek Hospital, Amsterdam

foramen ovale is the primary influencing factor for foraminal size rather than variations in the size of the cranial nerve itself (Fig. 8).

Enlargement of a skull base foramen can represent a dilemma for the interpreting radiologist. This issue is even more complicated as small defects within the bony walls of different skull base foramina have been previously described with variable frequency [15]. In general, skull base foramina containing venous structures are markedly more likely to be asymmetric in size and shape than other types of foramina. Therefore, an asymmetric arterial vascular or a neural foramen should raise the question of an underling vascular (e.g., arteriovenous malformation, fistula, aneurysm) or cranial nerve lesion (e.g., neurofibroma, perineural tumor spread). Evaluation of the adjacent anatomical structures, in particular on soft tissue windows in case of CT examination, plays in these cases a critical role as a tangle of or enlarged vessels may be seen in case of vascular lesion and obliteration of the fat pad within or just below the neural foramen in case of neurofibroma or perineural tumor spread (Fig. 9). Search for signs of denervation of muscles that are supplied by the suspected abnormal nerve may also be beneficial.

Occasionally, usually non-visible very small channels may be more prominent in appearance and therefore may become noticed by the interpreting radiologist, who may mistake these as small fractures. Emissary vein channels as well as mastoid and inferior tympanic canaliculi are the

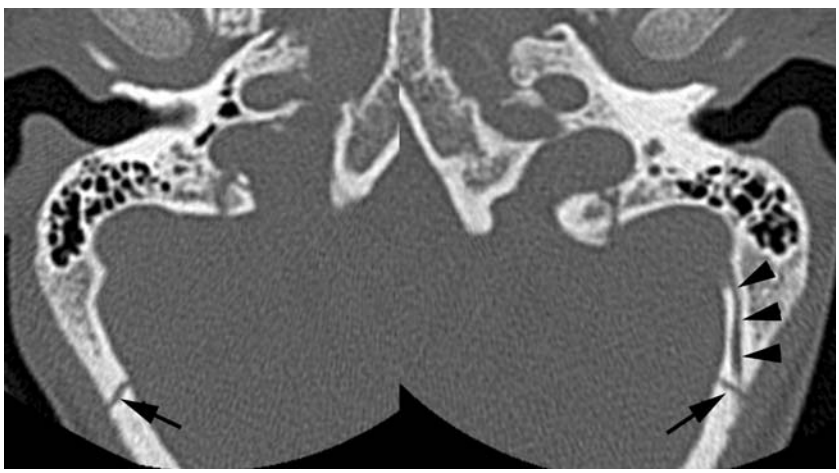

**Fig. 10** Composite of axial CT images displayed in bone window demonstrates a prominent mastoid emissary vein canal running obliquely through the posterior mastoid bone on the left (arrowheads), while it is absent on the right. This asymmetry should not be mistaken for fracture as the walls of the emissary vein canal are well corticated and smooth in appearance. In addition, there is no evidence of associated soft-tissue hematoma. The black arrows indicate the occipitomastoid sutures that course markedly less obliquely through the skull base than the mastoid emissary veins

most common examples [14]. The mastoid canaliculus can be seen in 28% of patients and contains the auricular branch of the vagus nerve [14]. The inferior tympanic canaliculus is more rare and seen in only 6% of patients. It lies in the lateral part of the jugular fossa within the bony ridge dividing the carotid canal from the jugular fossa. It contains the tympanic branch of the glossopharyngeal nerve. Both the mastoid and inferior tympanic canaliculi can be seen in specific locations, while the small channels carrying the emissary veins may occur essentially in any location (Fig. 10). Therefore, close evaluation of the bony margins and adjacent soft tissues for hematoma or fluid within the neighboring air cells is a very important step in accurate classification (fracture versus normal venous channel) of a lucent line within the skull base.

## Variations in position or appearance of neurovascular foramina and/or canals

Dehiscence and more anterior location of the sigmoid sinus can be seen in 2–3% of patients [14]. More anterior location of the sigmoid sinus has been defined as a sigmoid sinus to external auditory canal distance of <10 mm [19]. If that small, it predisposes the patient to complications during mastoid surgery [19]. It has been noticed that the sigmoid sinus to external auditory canal distance is in particular short in patients with non-pneumatized mastoid air cells [19].

Dehiscence of the internal carotid artery canal is rare with 1.4% [19]. It usually involves the right side [19]. In these cases, the bony separation between the vertical segment of the petrous portion of the internal carotid artery and the jugular fossa or between the internal carotid artery and the middle ear is absent, placing the patient at increased risk for injury during middle-ear surgeries [19].

Exceedingly rarely, an aberrant internal carotid artery can be seen (Fig. 11). It is caused by disturbed differentiation of the third branchial artery, resulting in an underdeveloped or absent cervical segment of the internal carotid artery [14]. In such cases, there is an enlarged inferior tympanic artery anastomosis with the horizontal petrous part of the internal carotid artery with the aberrant internal carotid artery coursing through an enlarged inferior tympanic canaliculus into the middle ear cavity [14, 15, 20]. Most commonly, the aberrant internal carotid artery is

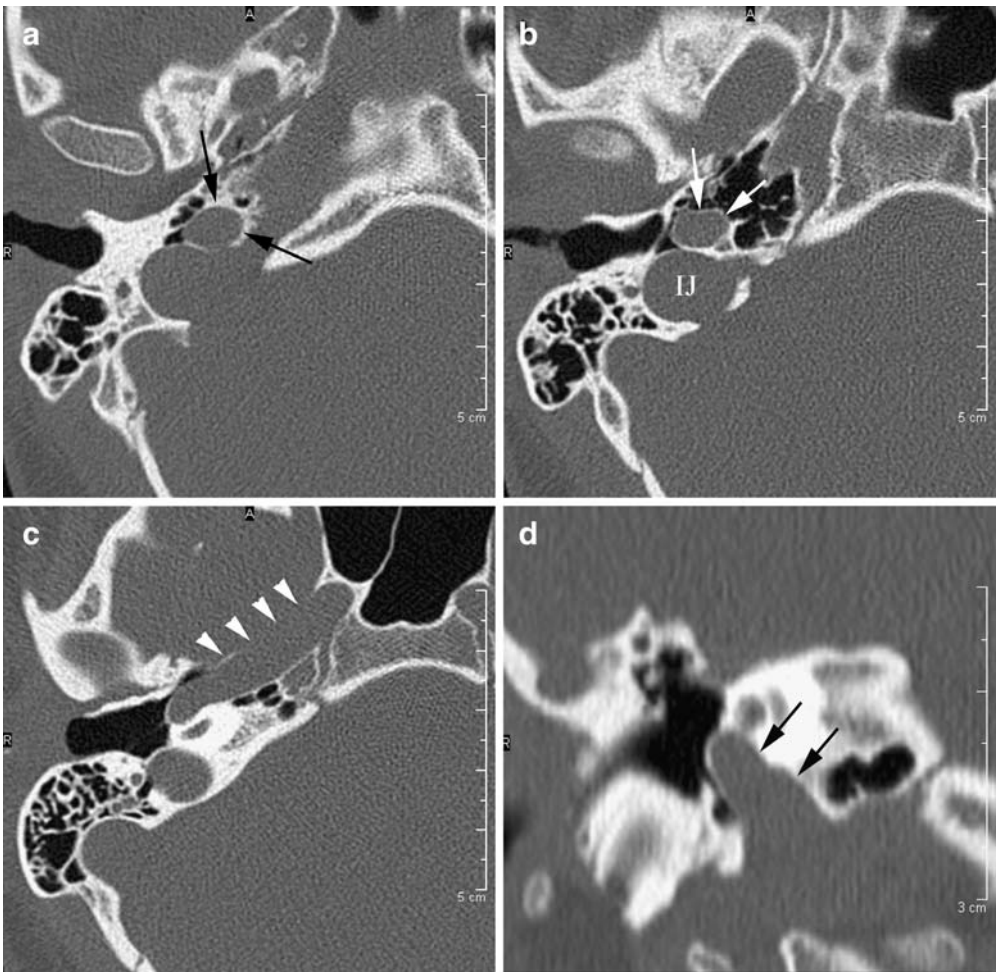


**Fig. 11** Axial CT images (**a**, **b** and **c**) and coronal reformatted CT image (**d**) displayed in bone window show a markedly enlarged inferior tympanic canaliculus (arrows in **a** and **d**) that extends superiorly (arrow in **b**) just anterior to the internal jugular bulb (IJ in B) to enter the middle ear cavity. Within the mesotympanum, it courses anteriorly to continue within the internal carotid canal (arrowheads in **c**). This appearance is classic for an aberrant internal carotid artery, a rare congenital arterial anomaly

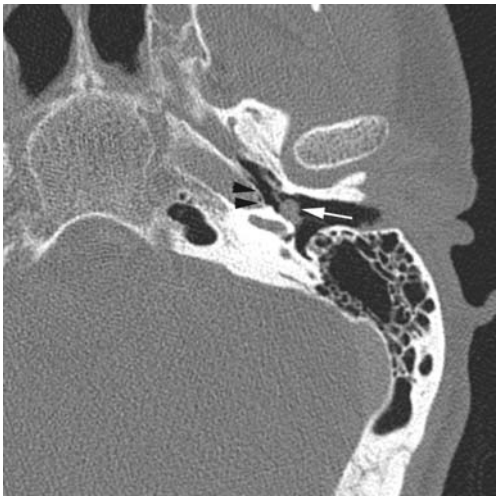

**Fig. 12** Axial CT image displayed in bone window shows a small mass overlying the promontory (white arrow). The intact posterior margin of the internal carotid artery canal (black arrowheads) helps to exclude an aberrant carotid artery as the etiology of this middle ear mass, which was proven to be a small glomus tympanicus

mistaken for a glomus tumor, a dangerous mistake as biopsy/removal of an aberrant carotid artery can yield severe complications, including death from uncontrollable bleeding or major brain infarction from vascular injury. Therefore, careful evaluation of the course and intactness of the internal carotid artery canal is critical (Fig. 12). In ambiguous cases, MRA or CTA might also be indicated.

## Internal jugular vein pseudolesions

The internal jugular vein represents the most common cause of flow-related artifacts on MRI at the skull base level (Fig. 13). This is usually amplified when certain normal anatomical variations of the jugular vein coexist. When the anatomical variant is unilateral, it may be easily mistaken for a soft tissue lesion on MRI examination. CT is not affected by such artifacts and therefore is often helpful for clarification.

A high-riding jugular bulb is the most common anatomical variation involving the internal jugular vein

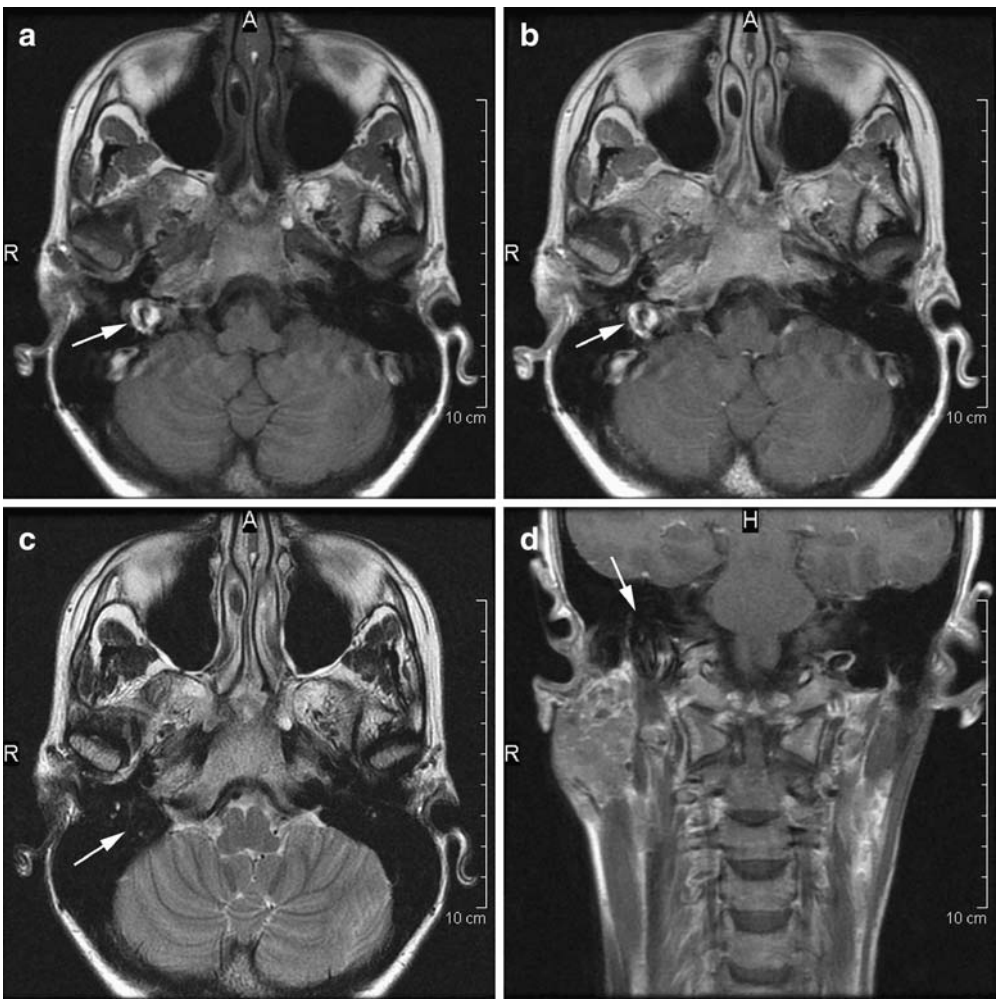


**Fig. 13** Axial T1 (**a**) and gadolinium-enhanced T1 (**b**) weighted images demonstrate a small round mass in the petrous apex region on the right side (arrows in **a** and **b**). This appearance is caused by flow artifacts from a high-riding jugular bulb on the right that mimic a pseudolesion in this location. The artifactual nature can be confirmed on the axial T2 (**c**) and coronal gadolinium-enhanced T1 (**d**) weighted images that clearly demonstrate a flow void (arrows in **c** and **d**) in this location

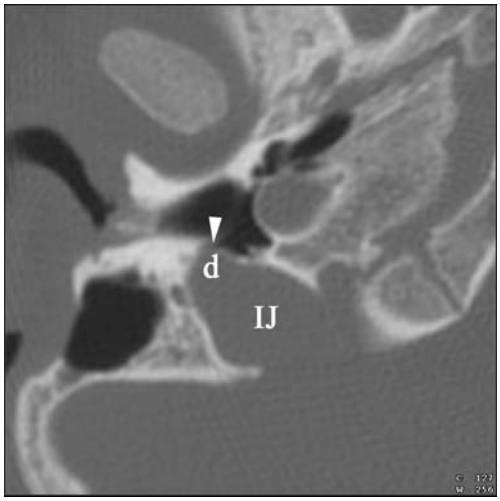


**Fig. 14** Axial CT image displayed in bone window shows a focal outpouching (d) along the anterior lateral aspect of the internal jugular vein (IJ) on the right side towards the middle ear cavity with partially dehiscent bony coverage (arrowhead) over it. This appearance is consistent with jugular vein diverticulum and may result in pulsatile tinnitus or be mistaken for a vascular mass on clinical examination

(Fig. 13). It is seen in 20% of patients and defined as a jugular bulb that rises above the inferior aspect of the bony annulus of the basal turn of the cochlea [19]. In two thirds of the patients, the right side is involved [19]. Bilateral involvement is rarely seen [19]. High-riding jugular bulb may become clinically significant when interfering with translabyrinthine surgery once reaching the level of the internal auditory canal (in 7%), or in patients with pulsatile tinnitus [19]. Jugular bulb diverticulum is the second most common venous variant and is seen in 8% of patients (Fig. 14) [19]. As with high-riding jugular bulb, the right side is most commonly involved, and bilateral involvement is very rare [19]. Typically, the diverticulum arises from the dome of the jugular bulb and may protrude through a bony dehiscence into the middle ear cavity, causing pulsatile tinnitus or conductive hearing loss [19]. It may also present with dizziness or facial nerve palsy due to exertion of pressure upon the posterior semicircular canal, facial nerve, vestibule or vestibular aqueduct [19]. Unfortunately, it is often difficult to determine if either a high-riding jugular bulb or a jugular bulb diverticulum is responsible for such symptoms as these are so commonly seen in asymptomatic patients. Therefore, the diagnosis of a symptomatic high-riding jugular bulb or jugular diverticulum should only be made after other potential causes are excluded.

The main dilemma of such jugular vein variations is, however, that they may be mistaken for a true lesion, especially on MRI (Figs. 13 and 15). Usually, close evaluation of all images in the different planes and on the various sequences reveals inconsistency of the flow pattern with the classic flow void seen in at least one plane or on one sequence, usually the T2-weighted sequences (Figs. 13 and 15). In ambiguous cases magnetic resonance venography and/or even conventional venography may be necessary for clarification.

## Arachnoid granulations

Arachnoid granulations are usually seen as incidentilomas on CT and/or MRI [21]. They most commonly present as small indentations of the inner table called granular foveolae that are typically located over the high convexities

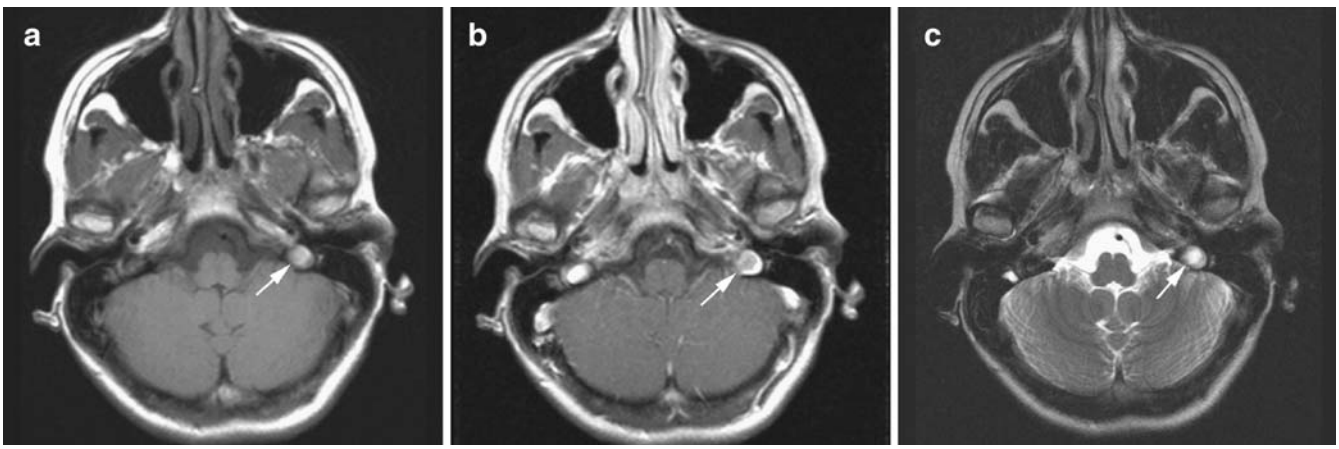


**Fig. 15** Axial T1 (**a**) and gadolinium-enhanced T1 (**b**) weighted images reveal similar situation as in Fig. 13 with a mass present in the jugular fossa on the left side (arrows in **a** and **b**). Careful evaluation of the axial T2 (**c**) weighted image, however, shows that there is lack of flow void (arrow in **c**) within the internal jugular vein on the left side. This appearance is therefore inconsistent with flow artifacts within an internal jugular vein. These imaging findings were caused by a thrombosed internal jugular vein on the left

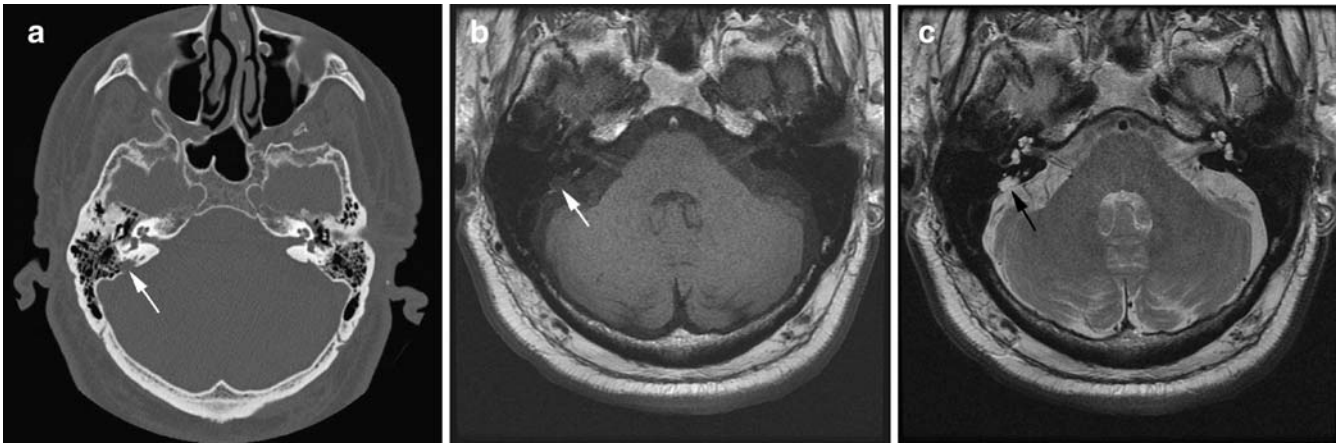


**Fig. 16** Axial CT image displayed in bone window (**a**) shows a focal disruption of the posterior cortex of the petrous bone with adjacent lytic lesion on the right side (arrow in **a**). The non-enhanced axial T1 (**b**) and T2 (**c**) weighted images reveal that this focal lesion follows the signal characteristics of cerebrospinal fluid (arrows in **b** and **c**) on both sequences. No gadolinium enhancement or restricted diffusion was present on additional images (not shown). This imaging appearance is characteristic for arachnoid granulations in ectopic location

in parasagittal location adjacent to the superior sagittal sinus. On imaging, these are usually small in size, show no disruption of the cortex, follow cerebrospinal fluid attenuation and may show capsule-like enhancement [21–23]. Occasionally, they can, however, mimic a true lesion, e.g., metastatic disease in a cancer patient, in particular when occurring in an atypical location (Fig. 16) or when showing unusual attenuation [21]. In such cases, they may be associated with thinning of the outer skull table, bulging into the paranasal sinuses or mastoid air cells, and therefore potentially result in otorrhea/rhinorrhea or otitis media. In these cases, the patients are placed at increased risk for septic meningitis [24]. In addition, they may mimic an intradiploic epidermoid cyst (Fig. 17). In such situations, diffusion-weighted imaging might be beneficial for the differential diagnosis (Fig. 17) [25].

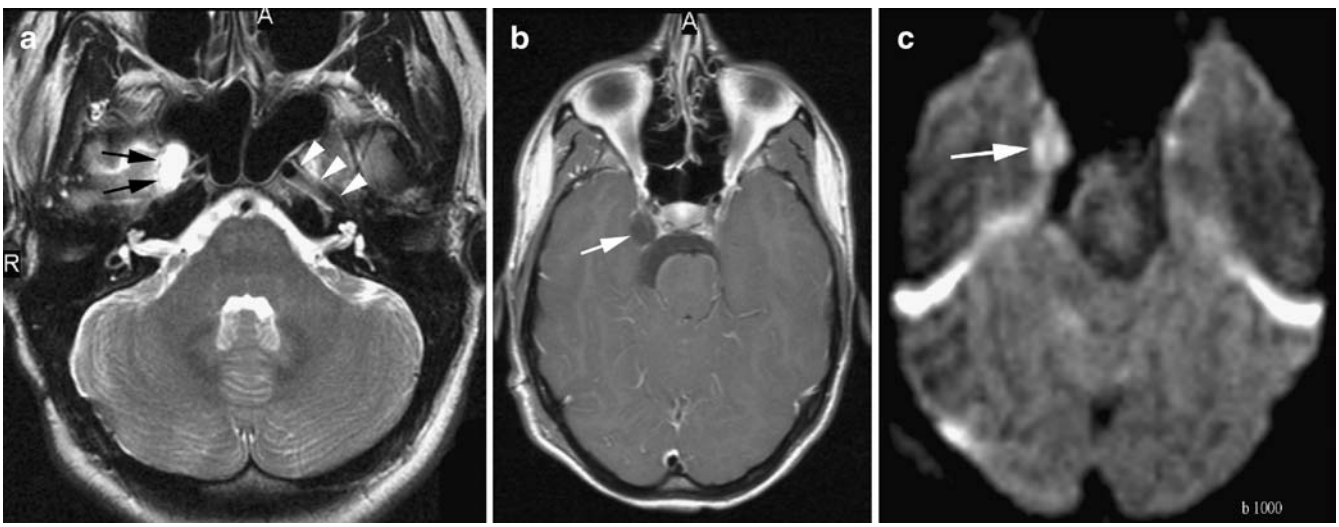


**Fig. 17** Axial T2 (**a**) and gadolinium-enhanced T1 image (**b**) demonstrates a non-enhancing fluid attenuation lesion (arrow in **a** and **b**) just anterior and superior to the petrous apex involving the cavernous sinus and its bony floor. It has similar signal characteristics to the lesion in Fig. 16. However, in this case, the diffusion-weighted image (**c**) reveals restricted diffusion within the lesion (arrow in **c**), suggesting a small epidermoid tumor that was later pathologically proven. Arrowheads in B mark the normal horizontal course of the internal carotid artery canal on the left, while this path is disturbed by the mass effect from the epidermoid tumor on the right

In summary, the radiologist is quite often faced with the problem of determining if an asymmetric appearance of the skull base is caused by a true lesion, in particular in symptomatic patients. The here provided discussion of normal anatomical variations in conjunction with their potential pathological mimics will hopefully provide helpful hints in answering the question: *pseudolesion or true lesion?*

## References


1. Virapongse C, Sarwar M, Bhimani S, Sasaki C, Shapiro R (1985) Computed tomography of temporal bone pneumatization: 1. Normal pattern and morphology. AJR 145:473–481
2. Mikami T, Minamida Y, Koyanagi I, Baba T, Houkin K (2007) Anatomical variations in pneumatization of the anterior clinoid process. J Neurosurg 106:170–174
3. Morton ME (1983) Excessive p neumatization of the sphenoid sinus: a case report. J Maxillofac Surg 11:236–238
4. Walker JL, Jones NS (2002) Pneumosinus dilatans of the frontal sinuses: two cases and a discussion of its etiology. J Laryngol Oto 116:382–385
5. Acar M, Yucel A, Degirmenci B, Yilmaz MD, Albayrak R (2004) Pneumocele vs. pneumosinus dilatans: Review of the literature with a case of frontal sinus pneumocele. Tohoku J Exp Med 202:295–297
6. Urken ML, Som PM, Lawson W, Edelstein D, Weber AL, Biler HF (1987) Abnormally large frontal sinus. II. Nomenclature, pathology and symptoms. Laryngoscope 97:606–611
7. Haetinger RG, Navarro JAC, Liverti EA (2006) Basialr expansion of the human sphenoidal sinus: an intergrated anatomical and computerized tomography study. Eur Radiol 16:2092–2099
8. van Schayck R, Niedeggen A (1992) Pneumosinus dilatans after prolonged cerebrospinal fluid shunting in young adults with cerebral hemiatrophy. A report of two cases and review of the literature. Neurosurg Rev 15:217–223
9. Sener RN (1997) Arachnoid cyst and pneumosinus dilatans. Comput Med Imaging Graph 21:125–129
10. Corey SA, O'Donovan CA (2005) Sturge-Weber Syndrome and Accompanying Dyke-Davidoff-Masson Syndrome. Arch Neurol 62:1928–1929
11. Hirst LW, Miller NR, Hodges III FJ, Corbett JJ, Thompson S (1982) Sphenoid pneumosinus dilatans a sign of meningioma originating in the optic canal. Neuroradiol 22:207–210
12. Vauterin T, Mombaerts I, Jorissen M, Sciot R, Legiud E (2005) Pneumosinus dilatans and orbital meningioma in neurofibromatosis type 2. B ENT 1:89–92
13. Suryanarayanan R, Abbott G (2007) Pneumosinus dilatans: demonstrated by sinus expansion on serial sinus X-rays with discussion of possible aetiology. J Laryngol Otol 121:96–99
14. Koesling S, Kunkel P, Schul T (2005) Vascular anomalies, sutures and small canals of the temporal bone on axial CT. Europ J Radiol 54:335–343
15. Ginsberg LE, Pruett SW, Chen MYM, Elster AD (1994) Skull-base foramina of the middle cranial ossa: Reassessment of normal variations with high-resolution CT. AJNR Am J Neuroradiol 15:283–291
16. Shapiro R, Robinson F (1967) The foramina of the middle fossa: A phylogenetic, anatomic and pathologic study. AJR 101:779–794
17. Henderson WR (1966) A note on the relationship of the human maxillary nerve to the cavernous sinus and to an emissary sinus passing through the foramen ovale. J Anat 100:905–908
18. Lindblom K (1936) A roentgenographic study of the vascular channels of the skull, with special reference to intracranial tumors and arteriovenous aneurysms. Acta Radiol (Suppl) 30:1–146
19. Atilla S, Akpek S, Uslu S, Ilgit ET, Işik S (1995) Computed tomographic evaluation of surgically significant vascular variations related with the temporal bone. Eur J Radiol 20:52–56
20. Verbist B, Hermans R, Devlies F, DeFoer B, Baert AL (1996) Quiz of the month. Partial agenesis of the internal carotid artery with collateral circulation through an enlarged inferior tympanic artery. Eur Radiol 6:939–940
21. Kuroiwa T, Kajimoto Y, Ohta T, Tsutsumi A (1996) Symptomatic hypertrophic pacchionian granulation mimicking bone tumor: case report. Neurosurg 39:860–862
22. Okamoto K, Ito J, Tokiguchi S, Furusawa T, Nishihara M (1997) Arachnoid granulations of the posterior fossa: CT and MR findings. Clin Imaging 21:1–5
23. Celli P, Cervoni L, Quasho R (1999) An asymptomatic hypertrophic pacchionian granulation simulating osteolytic lesions of the calvaria. Neurosurg Rev 22:149–151
24. Perry BP, Rubinstein JT (2000) Meningitis due to acute otitis media and arachnoid granulations. Ann Otol Rhinol Laryngol 109(9):877–879
25. Yamaguchi S, Hirohata T, Sumida M, Arita K, Kurisu K (2002) Intradiploic arachnoid cyst identified by diffusion-weighted magnetic resonance imaging-case report. Neurol Med Chir 42:137–139