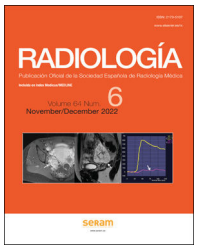
Radiología 66 (2024) 577–587

RADIOLOGÍA

www.elsevier.es/rx



ARTICLE FROM A RESIDENT

Complications of acute rhinossinusitis. A clinical radiological review

P. Carvalho dos Santos[a,*], P. Costa[b], I. Carvalho[a], C. Sousa[a]

[a] *Departamento de Otorrinolaringología, Cirugía de Cabeza y Cuello, Centro Hospitalar Universitário do Porto, Instituto de Ciências Biomédicas Abel Salazar, Universidade do Porto, Porto, Portugal*
[b] *Departamento de Radiología, Unidade Local de Saúde de Matosinhos, Matosinhos, Portugal*

Received 24 October 2021; accepted 7 March 2022

KEYWORDS
Pott Puffy Tumor;
Absceso cerebral sinusitis;
Orbital cellulitis;
Cavernous sinus thrombosis;
Brain abscess

Abstract Acute rhinossinusitis is defined as a symptomatic inflammation of the nasal fossa and paranasal sinuses. The diagnosis of this disease is clinical and usually does not require imaging evaluation. However, when there is a suspicion of a complication or even for surgical planning, imaging is of primordial importance.

The aim of this paper is to provide a concise pictorial review with clinical and imagiological correlation of the most common complications in acute rhinossinusitis to provide relevant clinical data and highlight the most important imaging findings.

PALABRAS CLAVE
Tumor inflamatorio de Pott;
Sinusitis;
Celulitis orbitaria;
Trombosis del seno cavernoso;
Absceso cerebral

Complicaciones de la rinosinusitis aguda. Una revisión clínica radiológica

Resumen La rinosinusitis aguda se define como una inflamación sintomática de las fosas nasales y los senos paranasales. El diagnóstico de esta enfermedad es clínico y no suele requerir una evaluación por imágenes. Sin embargo, cuando existe la sospecha de una complicación o incluso para la planificación quirúrgica, los estudios de diagnóstico por imagen son de importancia primordial.

El objetivo de este artículo es proporcionar una revisión concisa de las imágenes con correlación clínica e imagenológica de las complicaciones más frecuentes en la rinosinusitis aguda para proporcionar datos clínicos relevantes y destacar los hallazgos más importantes de las técnicas de imagen.

* Corresponding author.
E-mail address: pedrosantos288@gmail.com (P. Carvalho dos Santos).

2173-5107/

Introduction

Acute rhinossinusitis (ARS) is defined as a symptomatic inflammation of the nasal fossa and paranasal sinuses.[1] Its diagnosis is clinical and defined as the presence of at least two of the following symptoms: nasal obstruction, anterior or posterior nasal discharge, headache or facial pain and hyposmia.[1]

Complicated ARS, which accounts for a minority of cases, is mostly of bacterial etiology, with the most common causative agents being *Streptococcus pneumoniae*, *Haemophilus influenza* and *Moxarella catarrhalis*''.[2]

There is a subgroup of patients where ARS can assume an aggressive and rapidly progressive status, whose main pathological agent is fungal and not bacterial (invasive fungal rhinosinusitis, IFRS).[3,4] It occurs mostly in elderly immunocompromised patients with poorly controlled diabetes.[4] Patients with AIDS, with a surgical or oncologic history, or patients in treatment with biologics are also at risk for IFRS and therefore should be approached with a high degree of suspicion. This subtype of ARS has a particular clinical and radiological presentation[5] whose description is beyond the scope of this paper.

Regarding bacterial ARS, its diagnosis is established when, in the setting of ARS, purulent nasal discharge, localized facial pain, high fever or ''double-peak pattern'' is present.

From a practical standpoint, we can consider ARS as complicated when the sinonasal infection spreads beyond the sinonasal bony confines. Although antibiotic use has decreased the incidence of these complications, host factors, bacterial pathogenicity and late presentation continue to adversely impact the outcome of sinonasal infections.[3]

ARS complications occur more commonly in male pediatric patients (1 in 12 000 vs 1 in 36 000 adults) with orbitary complications accounting for the majority of treated patients (60–75%), followed by intracranial (15–20%) and osseus complications (5–10 %).[6]

The extension of infection to adjacent structures can occur by way of direct bone penetration, either across congenital dehiscent regions of the orbital wall, throughout acquired bone dehiscence (trauma or previous surgery), across the anterior or posterior ethmoidal foramen, or by means of thrombophlebitis of the ophthalmic venous system.[3]

ARS is a clinical diagnosis, however, as will be further shown, imaging plays a key role when there is a suspicion of a complication.[2]

This paper aims to be a useful pictorial review, with clinical and imagiological correlation of the most common complications in ARS in both children and adults, to provide radiologists with relevant clinical data and highlight the most important imaging findings.

Imaging considerations

Uncomplicated ARS usually does not require imaging evaluation, although CT of the paranasal sinuses, mainly without intravenous contrast, is usually appropriate in cases of possible surgical candidates, namely in the setting of recurrent cases.[7] In fact, CT is considered the mainstay of surgical planning,[8] mainly due to its high-spatial resolution, allowing excellent delineation of bone anatomy, namely of the ethmoidal sinuses, osteomeatal complex and anatomic variations that increase surgical risk.[2,7] Thus, CT images provide an anatomical roadmap that should always be available in the operating room. New technologies such as intraoperative navigation systems have been increasingly used and are strongly recommended, whenever available.[9]

Moreover, contrast-enhanced CT (ceCT) plays a vital role towards the clinical suspicion of ARS complications. Although MRI presents a high accuracy in the identification and characterization of orbital and intracranial complications, CT frequently is the only cross-sectional imaging technique available in the emergency setting.[2,7] CT is also useful as a complement to MRI when better bone definition is required.[7] It should be kept in mind that CT uses ionizing radiation, an important concern especially in children. However, low dose and ultra-low dose acquisition techniques are now available, allowing a great reduction in the ionizing radiation, with calculated effective doses as low as 0,045 mSv (8 times lower than standard protocol), without compromising its diagnostic ability.[10,11]

In the setting of high clinical suspicion of intraorbital and intracranial complications, MRI with and without intravenous contrast is most appropriate, due to its excellent soft-tissue resolution.[2,7] However, its limited availability and need for sedation in pediatric patients may impair its use. From a practical stand point, the fact that most of these complications are approached in emergency setting (where MRI is often not readily available) makes ceCT the most performed first-line imaging modality by far.[2,4]

A standard recommended CT protocol includes images obtained with submilimeter slice thickness[2,8,12] with (if complications are clinically suspected and MRI cannot be performed) or without (if the goal is surgical planning) intravenous contrast, with a maximum of 1,5-mm thick reconstruction in the axial, sagittal and coronal planes, using both bone and soft-tissue filter algorithms.[13]

When using ceCT, dual-phase CT is not usually required.[7] and images are acquired 2 min after injection of 100 ml of iodinated contrast[2]; in children aged more than 2 years, 2 mL/kg body weight after the age of two years should be used.[14]

Exposure settings usually recommended for standard protocols are 120 kVp and 55 mAs[2]. although these settings can be adjusted in low-dose protocols (further information can be found, among others, in the works of Hagtvedt,[15] Bulla,[16] Hoxworth,[17] Bodelle[10]).

MRI protocol[2,7] should include T1, T2-weighted images (WI) and DWI. Fat-saturated T2WI is also useful to detect inflammatory changes[2,12] and fluid attenuated inversion recovery (FLAIR) images are needed in cranial examinations.[2,13] Contrast-enhanced fat-saturated T1WI are also indicated whenever an orbital or intracranial ARS complication is suspected.[2,12,13] Images are acquired after administration of 0.2 ml/kg body weight of gadolinium-based contrast agent.[18] A field of view of 13–15 cm is usually sufficient. Slice thickness should be 3 mm, with an interslice distance of 1 mm. Imaging acquisition in the three planes is recommended.[2,13]

Table 1 Chandler's classification system.

Group I	Inflammatory edema (preseptal cellulitis)
Group II	Orbital cellulitis
Group III	Subperiosteal abscess
Group IV	Orbital abscess
Group V	Cavernous sinus thrombosis

Orbitary complications

All paranasal sinuses share boundaries with the orbit, making it the most common place for contiguous spread of sinonasal infectious processes.[8,19] The ethmoidal sinus is the most frequently implicated sinus in this type of complications, followed by the maxillary, and frontal sinus.[1] Orbitary complications occur in 6% of cases of ARS, more commonly in the pediatric population.[2]

Chandler[6] described these complications as a progression of the infectious process through the orbital region, which correlates with a progression of its severity arranged into five categories, described in Table 1.

Preseptal and orbital cellulitis are usually treatable with medical therapy alone, namely intravenous antibiotherapy and corticosteroid.[12] Nevertheless, more severe complications (Chandler 3, 4 and 5) usually require surgical drainage coupled with intravenous antibiotherapy,[1] although medical treatment alone may be successful in selected cases of subperiosteal abscess.[20]

With early treatment, orbital complications have a good prognosis, although some symptoms, such as proptosis or orbitary edema, could take up to two to three months to fully recede.[3]

Differently from the above, septic cavernous sinus thrombosis is associated with a poor prognosis. In the preantibiotic era SCST was considered universally fatal (80–100%). Nowadays, although the overall incidence, morbidity, and mortality have greatly declined with the introduction of antibiotics, mortality rates still remain as high as 8% in children[21] and up to 30% in adults.[1] Moreover, SCST accounts for important morbidity associated with damage of structures that traverse or are in close relation to the cavernous sinus, including cranial nerves (CN) I, III, IV, V1, V2 and VI, internal carotid artery, ophthalmic veins and the optic quiasm.[21] Thus, prompt diagnosis and early treatment is essential to avoid its fatal outcome and other ominous consequences.

Preseptal cellulitis

Preseptal cellulitis, consists of inflammatory edema of the eyelid and is the most common complication of ARS in children. Progression to abscess or cutaneous fistula is rare due to the protective effect of the orbital septum, which acts as a barrier to the spread of infection.[3] Clinically, it presents as palpebral swelling and erythema, without ocular symptoms.[2]

CT shows thickening and densification of the eyelid,[2,12] with no changes in the postseptal area, namely extraocular muscles, postseptal fat and lacrimal glands (Fig. 1).[12] Although rarely performed in the emergency setting, MRI presents high signal intensity of the eyelid on fluid sensitive images, with corresponding hypointense reticulation on T1WI and enhancement after gadolinium administration.[12]

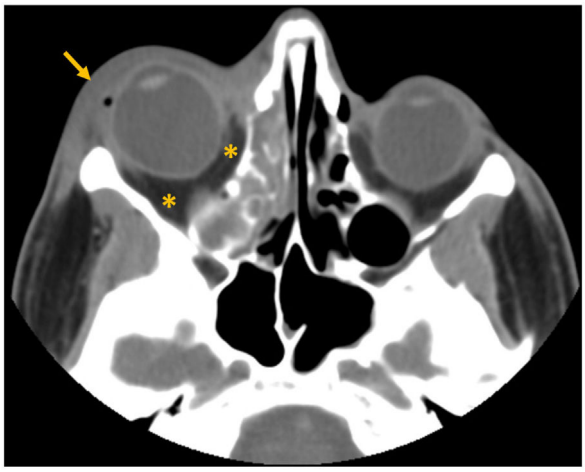


Figure 1 **Preseptal cellulitis**: Axial unenhanced CT image, soft tissue window, shows thickening and fat densification of the right eyelid (arrow), without any changes in the postseptal (asterisks). Complete opacification of the right ethmoidal cells is also seen, confirming the clinical diagnosis of acute rhinosinusitis.

In the rare cases of abscess formation, a fluid collection with peripheral enhancement will be seen in both CT and MRI.[2,12]

Orbital cellulitis

Orbital cellulitis results from extension of the infectious process to the orbital soft tissues, with bacterial proliferation and inflammatory tissue in the intra-orbital fat.[3]

Clinically, this manifests as fever, palpebral swelling, ophthalmoplegia, pain with eye movements, chemosis and even proptosis. Although rare, it can lead to elevated intraocular pressure and progressive vision loss.[2,22]

CT shows intraorbital fat densification, involving extraconal and/or intraconal structures, frequently in association with preseptal cellulitis (Fig. 2A–C).[2,3] MRI is also very sensitive to detect inflammation, mainly with fluid-sensitive sequences, as fat-saturated T2WI, where high signal intensity is usually easily recognized (Fig. 2D). MRI may also help in the differential with lymphoproliferative disorders, where DWI plays a major role,[2,12] since high cellular lesions (as lymphoproliferative disorders) are typically hyperintense on DWI and hypointense on ADC map, while inflammatory changes do not present restricted diffusion.[23] Contrast-enhanced images (both CT and MRI) show enhancement without discrete fluid collections.[12]

Subperiosteal abscess

Subperiosteal abscess is defined as a purulent accumulation between the bone and its periosteum, most frequently affecting the medial or superior orbital walls.[12] Patients present with chemosis and, less frequently, proptosis and ophthalmoplegia. Visual impairment is not frequent at this stage.[2,24]

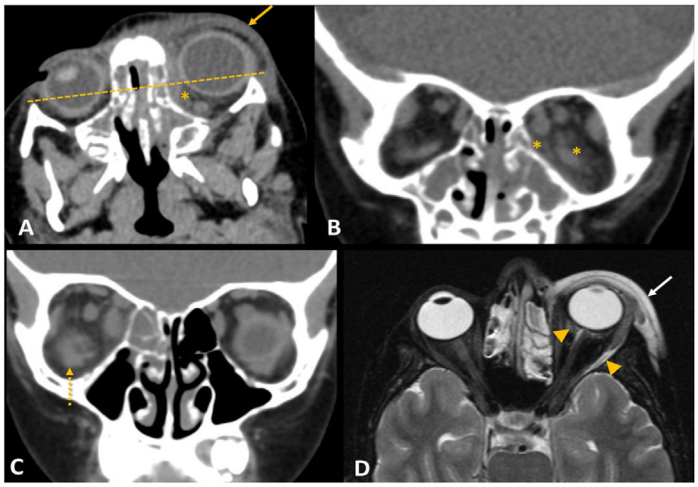


Figure 2 **Orbital cellulitis**: Axial (A) and coronal (B) non-enhanced CT images, soft tissue window, present opacification of the ethmoid and maxillary sinuses bilaterally, associated to mild proptosis and densification of the left pre- and postseptal fat (arrow and asterisks, respectively), in keeping with preseptal and orbital cellulitis. Note that the orbital fat densification occurs predominantly alongside the lamina papyracea and orbital roof. No fluid collections are seen. (C) Coronal non-enhanced CT images, soft tissue window, of another patient demonstrate opacification of the right ethmoidal cells, associated to postseptal fat-stranding and enlarged ill-defined medial and inferior rectus muscles (white arrow), suggestive of periorbital cellulitis with myositis. (D) Axial fat-saturated T2WI of a different patient shows thickening and hyperintensity of the preseptal fat (white arrow) and high signal intensity in the intraconal and extraconal orbital fat (arrowheads), without discrete collections.

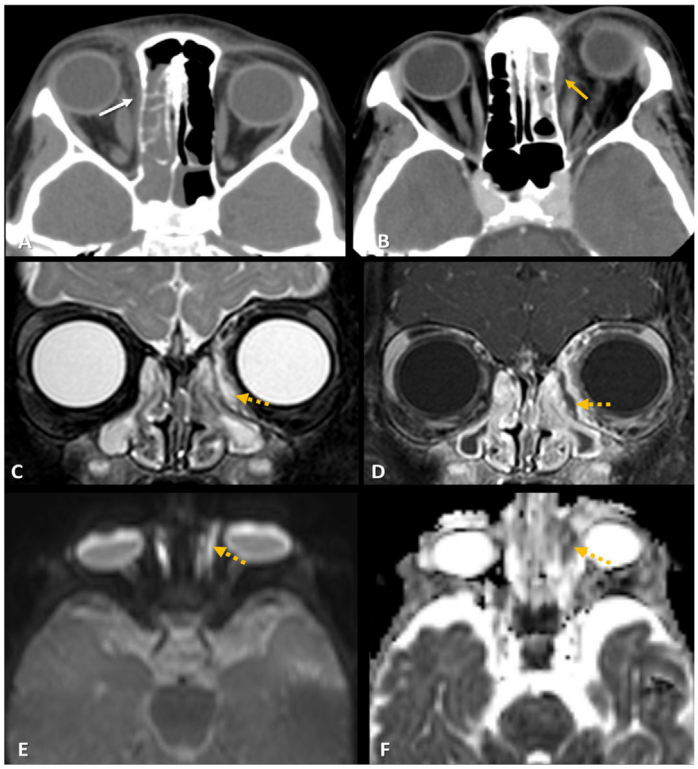


Figure 3 **Subperiosteal abscess**: (A) Axial non-enhanced CT images, soft tissue window, present opacification of the right ethmoid and sphenoid sinuses, associated to preseptal and orbital cellulitis, with more pronounced fat densification adjacent to the medial orbital wall (white arrows) and irregularity of the lamina papyracea contour, in keeping with early subperiosteal abscess formation. (B) Axial ceCT, soft tissue window, of a different patient shows opacification of the left ethmoidal cells and a conspicuous rim-enhancing crescent-shaped collection (yellow arrows), forming obtuse angles with the adjacent medial orbital wall. Coronal SPAIR (A) and post-contrast fat-saturated T1WI (B), axial DWI (C) and ADC map (D) of another patient show mucosal thickening and filling of the maxillary and ethmoid sinuses bilaterally, associated to a rim-enhancing fluid collection (dotted arrows), adjacent to the medial orbital wall, with restricted diffusion, compatible with subperiosteal abscess.

CT findings can be subtle, with only minimal densification of the fat adjacent to the medial or superior orbital wall associated with a fluid-filled paranasal sinus (Fig. 3A).[2] With progression, a crescent-shaped collection will become conspicuous, demonstrating rim enhancement and obtuse angles with the orbital wall (Fig. 3B).[2,3] Contrast-enhanced MRI can also clearly depict subperiosteal inflammatory changes, that present high signal on fat-saturated T2 and hyperenhancement (Fig. 3C).[2] If a conspicuous collection is present, rim-enhancement will be seen (Fig. 3D). DWI is a useful tool in the detection of abscesses, wherever the location, due to the typical restricted diffusion of the purulent content (Fig. 3E–F).[2,24]

Orbital abscess

Differently from the latter complication, orbital abscess consists of purulent accumulation within the orbit, emerging as a complication of orbital cellulitis or ruptured subperiosteal abscess, and is most often located in the extraconal compartment.[2,22]

In this setting, ocular symptoms are more evident, frequently with proptosis and ophthalmoplegia, which can lead to rapidly progressive vision loss.[3]

At this stage, ceCT demonstrates a fluid collection with sharp angles with the orbital wall (Fig. 4A and B). Regardless of location, abscesses present on ceMRI as rim-enhancing T1 isointense/T2 hyperintense fluid-collections with restricted diffusion (Fig. 4C).[2]

Cavernous sinus thrombosis

SCST is a rare, yet severe, process typically arising from infections of the paranasal sinuses and less commonly, otogenic, odontogenic, and pharyngeal sources.[25]

It may be considered an orbital or intracranial complication, resulting from retrograde thrombophlebitis of the ophthalmic vein or direct extension of sphenoidal sinusitis.[1]

Clinical symptoms of SCST arise from orbital venous drainage obstruction and compression of the CN within the cavernous sinus, resulting in proptosis, chemosis, ptosis and diplopia, with the latter two symptoms caused by affliction of CN III, IV and VI.[3]

Rarely, SCST can progress to septic thrombophlebitis of the internal jugular vein. If bacteriemia occurs, infection can spread hematogenously, leading to septic emboli, most frequently in the lungs, a condition known as Lemierre's syndrome.[26]

Imagiological signs of cavernous sinus thrombosis can be difficult to recognize. Non-enhanced CT can be normal, although a high-density thrombus is seen in the affected cavernous sinus in about 25% of cases.[2] On non-enhanced MRI, thrombus can be represented as absent flow voids, and present different signal intensities depending on its temporal evolution and the age of the thrombus.[2,27]

Indirect signs suspicious for thrombosis include cavernous sinus (and occasionally superior ophthalmic vein) enlargement and convex contour, intraorbital fat densification, proptosis and enlargement of extraocular muscles.[12] Cavernous sinus asymmetry is an important clue (Fig. 5), however not useful in case of bilateral thrombosis.

Ideally, the presence of filling defects representing the occluding thrombus is demonstrated on either ceCT (Fig. 5A) or contrast-enhanced MRI, although MRI is considered superior.[12] On ceCT, care should be taken not to misinterpret filling defects as fatty deposition (a normal finding), and vice versa.[2]

Clearly depict filling defects can be challenging (Fig. 5B). Indirect signs should always be pursued, namely those described above for the unenhanced imaging studies, which along with the appropriate clinical context allow for the diagnosis of cavernous sinus thrombosis.[27] Associated dural enhancement along the lateral margin of the sinus (Fig. 5C)[2,12] and superior ophthalmic vein thrombosis (Fig. 5D) also reinforce this diagnosis, the latter suggesting a retrograde spread.[25]

Osseous complications — osteomyelitis

Osteomyelitis constitutes about 9% of ARS complications,[3] and occurs mainly due to extension of frontal sinusitis to the diploic region of the frontal bone. The infectious process can spread into osseous structures both by direct extension or through means of venous drainage, as frontal sinus mucosal veins drain into diploic veins.[28]

When affecting the posterior wall of the sinus, it may lead to an epidural abscess or progress to other intracranial and intraparenchymal complications.[29] Once again, venous drainage may play a role, as the valveless diploic veins communicate with the dural venous system.[28]

If the infection involves the anterior wall, it can result in a subperiosteal abscess, presenting clinically as a frontal tumefaction named Pott's puffy tumor (PPT) that can fistulize to the skin. PPT is a very rare entity in the post-antibiotic era, occurring mostly in the adolescent age group and showing a clear male predominance Although rare, PPT should be promptly recognized, as it is frequently associated with intracranial complications (reported rates ranging from 29% to 85%), which can be asymptomatic in the early stages and may develop during intravenous antibiotherapy Thus, when suspecting PPT, imaging should be performed both at diagnosis and during the course of treatment.[28]

Less commonly, ARS may cause central skull base osteomyelitis (SBO), typically involving the clivus, usually seen in the setting of sphenoidal or ethmoidal invasive sinusitis.[3]

Typically, patients with osteomyelitis in the setting of ARS present with headache, fever, purulent rhinorrhea, periorbital swelling and forehead tumefaction.

CT is usually the first imaging modality in the emergency setting.[4,30] It confirms the presence of ARS, with opacification, mucosal thickening and air-fluid levels in the sinuses, and is the modality of choice to depict cortical bone erosions and/or trabecular demineralization, the main signs of osteomyelitis (Fig. 6B and E).[31]

When the anterior wall of the frontal bone is involved, CT can also show thickening and densification of the forehead soft-tissues[32] or even a conspicuous fluid collection with peripheral enhancement in cases of subperiosteal abscess formation (Fig. 6C and D).[33]

MRI allows superior soft-tissue evaluation and should be used to assess intracranial or intraorbital complications.

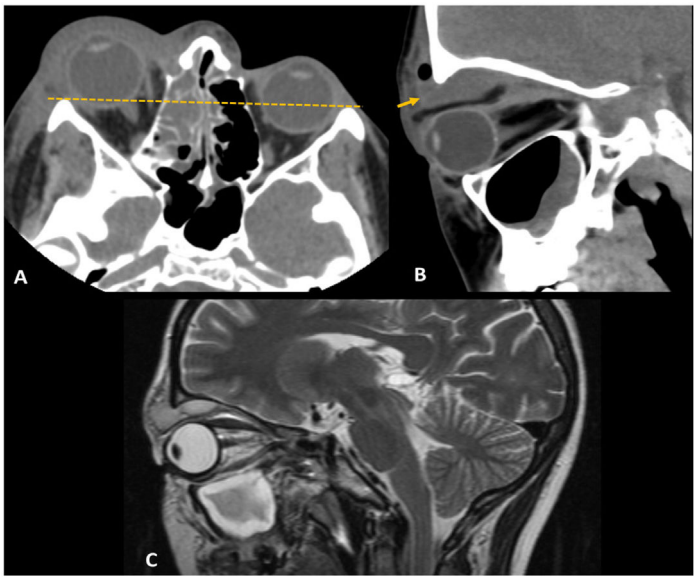


Figure 4 **Orbital abscess**: Axial (A) and sagittal (B) non-enhanced CT images, soft tissue window, present opacification of the left ethmoidal cells, associated to severe proptosis and a fluid collection with sharp angles with the orbital roof, in keeping with an orbital abscess. (C) Sagittal T2-weighted images of another patient shows a discrete fluid collection in the orbital roof (white arrow) associated to extensive orbital inflammatory changes and maxillary occupation (not shown).

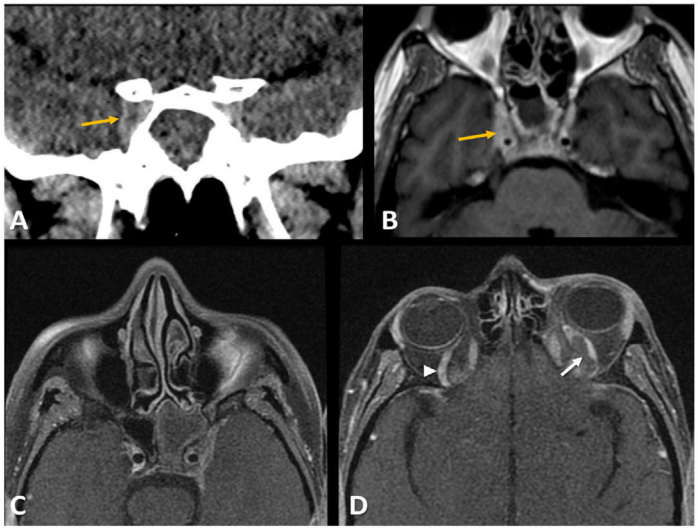


Figure 5 **Cavernous Sinus Thrombosis**: (A) Coronal enhanced-CT, brain window, and (B) post-contrast T1WI shows sphenoid sinus opacification and cavernous sinuses asymmetry, due to enlargement of the right side (arrows), which presents a convex contour, suggestive of thrombosis. On CT, filling defects on the right cavernous sinus are also seen, although less evident on MRI, due to thrombus high T1-signal. Axial post-contrast fat-saturated T1WI in two levels (C and D) in a different patient demonstrate left sphenoid sinus filling and mucosal thickening, associated to asymmetric enlargement, convex contour and enhancement of the lateral margin of the left cavernous sinus, as well as enlargement and partial occlusion of the ipsilateral superior ophthalmic vein (white arrow; arrowhead showing the normal right superior ophthalmic vein), in keeping with cavernous sinus thrombosis, probably due to retrograde spread.

Osteomyelitis with bone marrow involvement can also be depicted on MRI, as high signal intensity on short tau inversion recovery (STIR) and, consequently, loss of the normal fat high-signal on T1WI and, as well as heterogeneous enhancement on post-contrast images (Fig. 7B–D).[31] Progression may lead to necrosis and abscess formation, which will present as a rim-enhancing fluid collection with restricted diffusion.[31,34]

In the rare cases of SBO, signal abnormalities will be centered to the sphenoid bone and clivus (Fig. 7B–D), although they can extend to the petrous apices; associated symmetric thickening and edema of preclival soft-tissues may also be seen.[31]

Regarding treatment response, imaging re-assessment will show improvement of soft-tissue findings, although

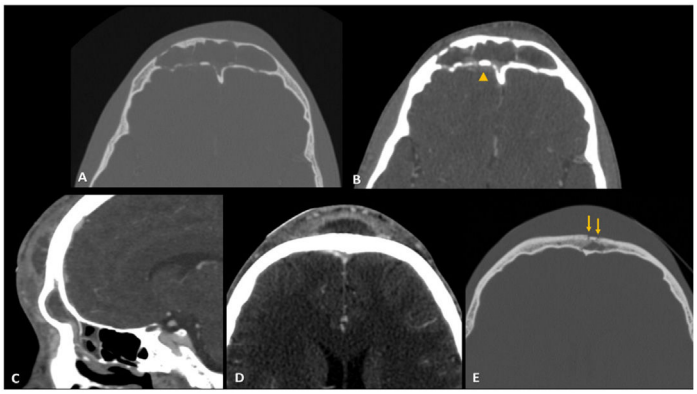


Figure 6 **Frontal bone osteomyelitis**: Axial ceCT, bone window (A) soft-tissue window (B), show frontal sinus opacification and cortical erosion of the posterior wall of the frontal bone, in keeping with osteomyelitis. Posteriorly to the cortical bone erosions, there is linear hyperenhancement (arrowhead), without obvious fluid collections, suggestive of focal meningitis. Sagittal (C) and axial (D and E) ce-CT, soft-tissue window (C and D) bone window (E) of another patient, show frontal sinus opacification associated with frontal tumefaction corresponding to a subperiosteal abscess (Pott's puffy tumor). Bone window allows easy identification of cortical erosions in the anterior wall of the frontal bone, in keeping with osteomyelitis (arrows).

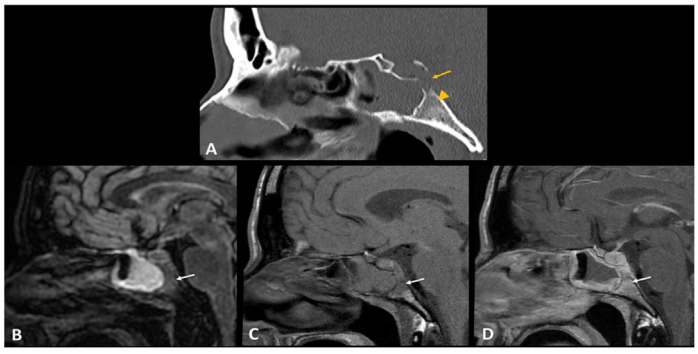


Figure 7 **Clivus osteomyelitis**: (A) Sagittal non-enhanced CT, bone window, demonstrates extensive cortical bone erosion and trabecular bone demineralization of the clivus and sella turcica. Sagittal fat-saturated T2WI FLAIR (B), pre-contrast T1WI (C) and post-contrast T1WI (D) shows high T2-signal, low T1-signal and heterogeneous enhancement of the clivus adjacent to the sphenoid sinus, in keeping with central skull base osteomyelitis.

bone abnormalities may take several weeks to clear up on imaging.[31,35]

The prognosis is usually good, if diagnosed and treated early.[3,28]

Intracranial complications

With the exception of cavernous sinus thrombosis, the frontal sinus is the most frequent origin site of intracranial complications.[1]

The sinus infection can extend to the intracranial compartment directly through bony dehiscences or by means of osteitis, followed by osteomyelitis, epidural empyema, subdural empyema, meningitis, cerebritis and, ultimately, cerebral abscess.[3] Thus, the primary intracranial complications are epidural and subdural empyemas, both presenting as extra-axial fluid collections, whose distinction may be challenging.[2]

In contrast to CT, MRI performs better in detection, localization and characterization of intracranial collections as well as parenchymal changes, allowing for earlier diagnosis of these complications and a more reliable distinction between them.[3]

Intracranial complications are more frequent in the second and third decades.[1] Clinical manifestations include severe headache, photophobia, seizures, or focal neurologic findings.[3]

Treatment of intracranial complications requires sinus drainage, allied with neurosurgical intervention if empyema or brain abscess are present.[2]

Epidural empyema

This complication results from the accumulation of pus between the skull and the dura-mater, mostly caused by contiguous infection spread from adjacent bone.[36] It is almost exclusively derived from frontal sinusitis, due to the poor adhesion of the dura onto the frontal wall of the anterior fossa. Although extradural by definition, it should be noted that epidural empyema frequently occurs in association with or immediately previous to the development of a subdural empyema.[37]

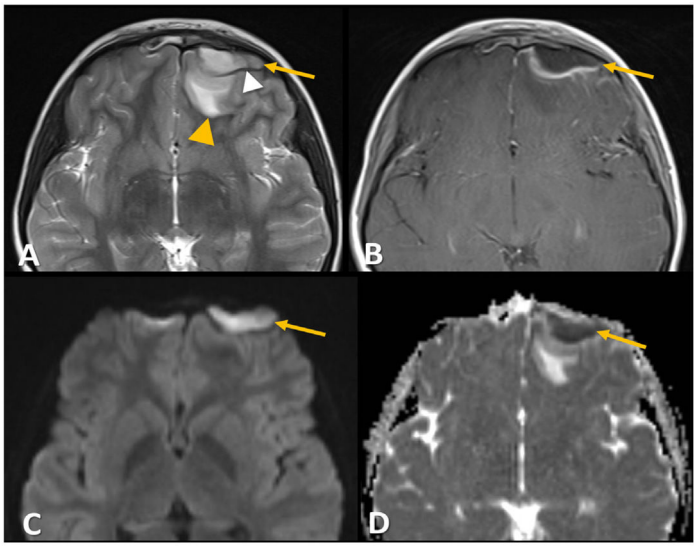


Figure 8 **Epidural empyema**: Axial T2WI (A), post-contrast T1WI (B and F), DWI (C) and ADC map (D) show two rim-enhancing extra-axial fluid collections, one with biconvex morphology, suggestive of epidural empyema (yellow arrows in A–D). Note the hypointense rim at the interface with the brain in epidural empyema (white arrowhead), representing the displaced dura, and the marked restricted diffusion (C and D), indicative of purulent content. Cerebral edema is also seen (yellow arrowhead), probably due to mass effect.

Clinically, it courses with mild symptoms, headache and fever, with minimal or absent neurological symptoms, as dura mater is still acting as a barrier between the infection and the brain parenchyma.[4]

Imaging shows a biconvex fluid collection with strong rim-enhancement, better depicted on MRI than CT (Fig. 8A and B).[2] This lesion does not cross suture lines, although it may extend across the midline in the frontal region. Due to its frequent association with frontal sinusitis, epidural empyema may be differentiated from subdural empyema when it crosses the midline, anteriorly to the falx cerebri.[2,36] Moreover, epidural empyemas usually exhibit a hypointense rim at the interface between the collection and the brain on both T1 and T2WI (Fig. 8A), that stands for the displaced dura.[37,38] The presence of fluid with restricted diffusion on MRI is highly suggestive of purulent content (Fig. 8C and D) and helps in the differential with sterile fluid-containing lesions.[2]

Epidural empyema is frequently associated with osteomyelitis that, in the setting of rhinosinusitis, occurs mostly in the frontal bone. Thus, the overlying tissues should be evaluated for the presence of abscesses, such as Pott's puffy tumor or cutaneous fistula.

Careful evaluation of the underlying meninges is essential to identify signs of meningitis. Although dura mater functions as a natural barrier, cerebral edema can be present due to mass effect (Fig. 8A).[38]

Subdural empyema

Subdural empyema consists of purulent content located between the dura and arachnoid. It represents 10% of intracranial complications of rhinosinusitis and is frequently caused by frontal sinusitis, followed by ethmoidal and sphenoidal sinusitis.[3] The progression of infection occurs mainly by thrombophlebitis of the emissary veins to the subdural space, although direct extension may also play a role.[36,37]

Conversely to epidural empyema, in subdural empyema there is no dura acting as a barrier between the infectious process and the brain tissue. Moreover retrograde phlebitis can reach the cortical veins, leading to edema and infarction, and creating a direct spreading route for the infectious process.[37,39] Thus, subdural empyema is considered a neurosurgical emergency, characterized by a rapid progression.[40]

Clinically, there is rapid deterioration of the patient's neurologic status, with high fever, headache, neck stiffness and focal neurological deficits. Clinical symptoms overlap with those of meningitis,[36] thus imagiological evaluation is mandatory to detect extra-axial fluid collections.

Imaging typically shows a crescentic fluid collection with peripheral enhancement, not crossing the midline and located on one side of the falx.[2,36] MRI is the modality of choice to detect both subdural empyema and its complications.[36,37] Parenchymal edema and early cerebral abscess formation present as areas of increased signal intensity on water-sensitive sequences.[2] DWI should not be overlooked, as it allows easier identification of a small empyema, often difficult to recognize on the other sequences, and aids in differentiation between sterile subdural effusions and true empyemas.[39]

Towards a subdural empyema, immediate intervention should be performed with drainage of both the empyema and the involved sinuses, in order to prevent and restrict cortical damage as well as developing neurologic deficits.[37,40] Antibiotherapy with CSF penetration is the mainstay of treatment and should be promptly started.[40] Mortality ranges from 20 to 35%, with a lower rate in children.[34]

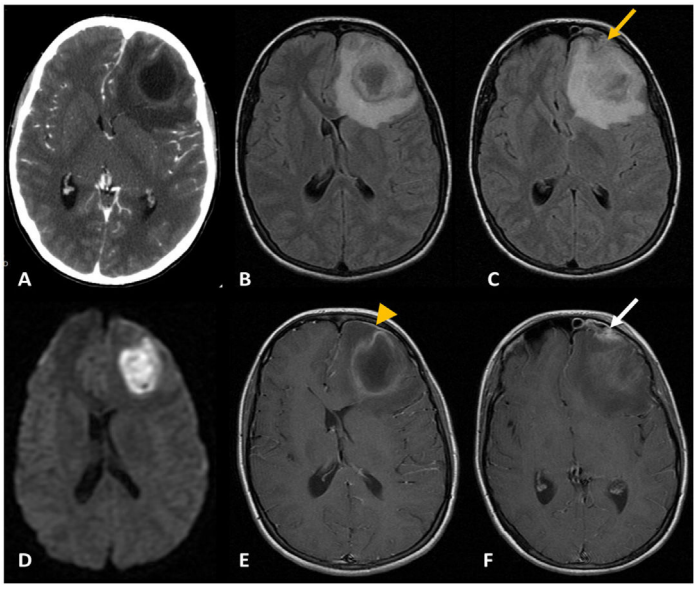


Figure 9 **Cerebral abscess**: Axial ceCT, soft-tissue window (A), shows a rim-enhancing frontal lobe collection with perilesional edema, causing mass effect with midline shift, suggestive of cerebral abscess. T2WI FLAIR (B and C) in two different planes, DWI (D) and post-contrast T1WI (E and F) in two different planes confirm the presence of a frontal lobe abscess with extensive perilesional edema, associated to crescent-shaped high T2/FLAIR-signal (yellow arrow), suggestive of subdural empyema, and leptomeningeal enhancement (arrowhead) suggestive of focal meningitis. At a lower level (C and F), it is demonstrated the presence of mucosal thickening of the left frontal sinus in continuity with the intracranial complications (white arrow in F).

Cerebral abscess

In this case, the purulent collection is located within the brain parenchyma and is either caused by contiguous extension from an infected sinus or by hematogenous spread.[36] Between 14–34% of cerebral abscesses in adults have a sinonasal origin, most often due to frontal and, to a lesser degree, sphenoidal sinusitis.[3] This complication is rarer in children, probably due to the underdevelopment of both frontal and sphenoid sinuses. The frontal lobe is the most commonly involved, given its proximity to the frontal sinuses.[40]

In the first stages of abscess formation, patients may not present with neurologic symptoms.[40] The early stages of cerebral edema and cerebritis usually manifest as headache, lethargy, agitation and altered mental status. With progression to abscess formation, there is worsening of mental status.[3]

Progressive enlargement of the abscess may lead to rupture into the ventricular system, a catastrophic complication with a high mortality rate.[40]

Diagnosis is attained by CT or MRI. Both will show a rim-enhancing fluid collection, in addition to marked oedema of the surrounding parenchyma due to mass effect (Fig. 9).[2,36] Similar to extra-axial collections, ceMRI is the preferred modality for evaluation of parenchymal complications. On MRI, a brain abscess presents increased signal intensity on DWI, in keeping with purulent content within (Fig. 9D),[2] and a moderately to markedly hypointense rim on T2WI, that differs from subdural empyema.[37]

An area of focal softening of brain parenchyma (cerebritis) that emerges prior to abscess formation[12] usually appears on ceCT as a focal ill-defined hypodensity or an area of patchy enhancement, which can be difficult to recognize. On the contrary, MRI typically shows a conspicuous area of increased signal intensity on T2WI and FLAIR.[2]

Treatment includes antibiotherapy and drainage of the involved sinuses and of the cerebral abscess. Intravenous treatment should be maintained for 2–4 weeks. The initial phase of cerebritis can be treated conservatively and medical therapy should include corticosteroids, anti-intracranial hypertension measures and anticonvulsants. The high mortality rate that ensued some years ago has now decreased considerably to 5%, due to advances in early diagnosis. Epilepsy is the most common sequel, and occurs in 30% of cases.[3]

Meningitis

In the pre-antibiotic era, meningitis was considered the most common complication of ARS, but its frequency has decreased in relation to other intracranial complications.[1]

The most frequently implicated sinuses are the frontal, sphenoid, and posterior ethmoid.[3,12]

Clinically, the most important symptoms are headache and neck stiffness, with drowsiness preceding coma. Diagnosis is established by lumbar puncture, that demonstrates high leucocyte and protein counts.[3]

Imaging findings of meningitis are usually nonspecific on non-enhanced CT however subarachnoid sulci and cisternal effacement due to iso to hyperattenuation of cerebral spinal fluid are suspicious findings. On ceCT, linear leptomeningeal thickening and enhancement (Fig. 6B) should suggest the diagnosis.[41,42]

As for other intracranial complications, ceMRI is the most suitable imaging modality MRI findings depend on the stage

of disease and may be normal initially.[2] As the disease progresses, thickening and edema of the pachymeninx and/or leptomeninges results in abnormal meningeal enhancement on post-contrast images and subarachnoid hyperintensity on FLAIR images (Fig. 9C and E),[2,4] that can be associated with subdural effusions and ventricular debris. Leptomeningeal enhancement is seen extending into the sulci and cisterns and can be easier to detect using post-contrast 3D T2-FLAIR.[12]

With disease progression, meningitis may result in cerebral edema, presenting as areas of high signal intensity on water-sensitive sequences as described previously, or even ischemia, best depicted on DWI as areas of restricted diffusion[2]. Other complications include accumulation of pus in the subarachnoid space, that is also hyperintense on DWI, as well as hydrocephalus.[2,12]

Treatment includes surgical drainage of the involved sinus, antibiotherapy corticosteroids and anticonvulsants.[3]

Conclusion

Although ARS complications can often present with mild and unspecific symptoms, they can be life-threatening, especially in high-risk patients. Thus, high clinical suspicion is the most important factor to achieve a correct and timely diagnosis.

Regarding imaging evaluation of clinically suspected ARS complications, contrast-enhanced MRI or CT are mandatory. Although ceCT is more frequently performed in the emergency setting, MRI performs better in the identification and characterization of orbital and intracranial complications.

In this setting, a good multidisciplinary coordination between the clinic and the radiologist is of the utmost importance.

Financial disclosure

The authors declare no financial disclosure.

Authorship

Responsible for study integrity: PS
Study conception: PS
Study design: PS
Data acquisition: PS, PC
Data analysis and interpretation: PS
Statistical processing: N/A
Literature search: PS, PC, IC
Drafting of the manuscript: PS, PC
Critical review of the manuscript with intellectually significant contributions: IC, CA
Approval of final version: IC

Key points

- Acute rhinossinusitis is a common clinical problem in both pediatric and adult patients, whose complications can be severe and life threatening. Therefore, the radiologist should be familiar with the spectrum of sinus disease and its potential complications.
- All paranasal sinuses share boundaries with the orbit, making it the most common place for contiguous spread of sinonasal infectious processes. The ethmoidal sinus is the most frequently implicated sinus in this type of complications, followed by the maxillary, frontal and, more rarely, the sphenoidal sinus.
- Acute uncomplicated rhinosinusitis usually does not require imaging evaluation, although CT and/or MRI are mandatory when there is suspicion of complications or in the setting of pre-operative planning.
- Due to its high availability, CT frequently is the first imaging technique performed in the emergency setting. The excellent bone and high-spatial resolution make CT the preferred imaging examination in presurgical planning. However, contrast-enhanced MRI is far superior in soft-tissue evaluation and should be considered whenever an orbitary or intracranial complication is suspected.

Conflict of interest

The authors declare no conflict of interest.

References

1. Fokkens WJ, Lund VJ, Hopkins C, Hellings PW, Kern R, Reitsma S, et al. European position paper on rhinosinusitis and nasal polyps 2020. Rhinology. 2020;58 Suppl S29:1–464, http://dx.doi.org/10.4193/Rhin20.600.
2. Dankbaar JW, van Bemmel AJM, Pameijer FA. Imaging findings of the orbital and intracranial complications of acute bacterial rhinosinusitis. Insights Imaging. 2015;6:509–18, http://dx.doi.org/10.1007/s13244-015-0424-y.
3. Subtil J, Barros E. In: Vilhena Mendonça MT, Arraiano Lopes MdoC, editors. Rinologia multidisciplinar. 1st ed. Lisboa: Edições Círculo Médico; 2019.
4. McCann MR, Kessler AT, Bhatt AA. Emergency radiologic approach to sinus disease. Emerg Radiol. 2021;28:1003–10, http://dx.doi.org/10.1007/s10140-021-01945-y.
5. Gavito-Higuera J, Mullins CB, Ramos-Duran L, Sandoval H, Akle N, Figueroa R. Sinonasal fungal infections and complications: a pictorial review. J Clin Imaging Sci. 2016;6:23, http://dx.doi.org/10.4103/2156-7514.184010.
6. Chandler JR, Langenbrunner DJ, Stevens ER. The pathogenesis of orbital complications in acute sinusitis. Laryngoscope. 1970;80:1414–28, http://dx.doi.org/10.1288/00005537-197009000-00007.
7. Expert Panel on Neurologic Imaging, Kirsch CFE, Bykowski J, Aulino JM, Berger KL, Choudhri AF, Conley DB, et al. ACR appropriateness criteria® sinonasal disease. J Am Coll Radiol. 2017;14(11S):S550–9, http://dx.doi.org/10.1016/j.jacr.2017.08.041.
8. O'Brien WTS, Hamelin S, Weitzel EK. The preoperative sinus CT: avoiding a ''CLOSE'' call with surgical complications. Radiology. 2016;281:10–21, http://dx.doi.org/10.1148/radiol.2016152230.
9. Galletti B, Gazia F, Freni F, Sireci F, Galletti F. Endoscopic sinus surgery with and without computer assisted navigation: a retrospective study. Auris Nasus Larynx. 2019;46:520–5, http://dx.doi.org/10.1016/j.anl.2018.11.004.
10. Bodelle B, Wichmann JL, Klotz N, Lehnert T, Vogl TJ, Luboldt W, et al. Seventy kilovolt ultra-low dose CT of the paranasal sinus: first clinical results. Clin Radiol. 2015;70:711–5, http://dx.doi.org/10.1016/j.crad.2015.03.002.
11. Abul-Kasim K, Strömbeck A, Sahlstrand-Johnson P. Low-dose computed tomography of the paranasal sinuses: radiation doses

and reliability analysis. Am J Otolaryngol. 2011;32:47–51, http://dx.doi.org/10.1016/j.amjoto.2009.08.004.
12. Velayudhan V, Chaudhry ZA, Smoker WRK, Shinder R, Reede DL. Imaging of intracranial and orbital complications of sinusitis and atypical sinus infection: what the radiologist needs to know. Curr Probl Diagn Radiol. 2017;46:441–51, http://dx.doi.org/10.1067/j.cpradiol.2017.01.006.
13. Som PM, Curtin HD. Head and neck imaging. Missouri: Mosby Elsevier; 2011, 2.
14. Sorantin E, Weissensteiner S, Hasenburger G, Riccabona M. CT in children —dose protection and general considerations when planning a CT in a child. Eur J Radiol. 2013;82:1043–9, http://dx.doi.org/10.1016/j.ejrad.2011.11.041.
15. Hagtvedt T, Aaløkken TM, Nøtthellen J, Kolbenstvedt A. A new low-dose CT examination compared with standard-dose CT in the diagnosis of acute sinusitis. Eur Radiol. 2003;13:976–80, http://dx.doi.org/10.1007/s00330-002-1652-3.
16. Bulla S, Blanke P, Hassepass F, Krauss T, Winterer JT, Breunig C, et al. Reducing the radiation dose for low-dose CT of the paranasal sinuses using iterative reconstruction: feasibility and image quality. Eur J Radiol. 2012;81:2246–50, http://dx.doi.org/10.1016/j.ejrad.2011.05.002.
17. Hoxworth JM, Lal D, Fletcher GP, Patel AC, He M, Paden RG, et al. Radiation dose reduction in paranasal sinus CT using model-based iterative reconstruction. Am J Neuroradiol. 2014;35:644–9.
18. Ibrahim M, Hazhirkarzar B, Dublin A, Retrieved January 3, 2022 from https://www.ncbi.nlm.nih.gov/books/NBK482487/, 2021.
19. Mossa-Basha M, Blitz AM. Imaging of the paranasal sinuses. Semin Roentgenol. 2013;48:14–34, http://dx.doi.org/10.1053/j.ro.2012.09.006.
20. Gavriel H, Jabarin B, Israel O, Eviatar E. Conservative management for subperiosteal orbital abscess in adults: a 20-year experience. Ann Otol Rhinol Laryngol. 2018;127:162–6, http://dx.doi.org/10.1177/0003489417751155.
21. Smith DM, Vossough A, Vorona GA, Beslow LA, Ichord RN, Licht DJ. Pediatric cavernous sinus thrombosis: a case series and review of the literature. Neurology. 2015;85:763–9, http://dx.doi.org/10.1212/WNL.0000000000001886.
22. Al-Madani MV, Khatatbeh AE, Rawashdeh RZ, Al-Khtoum NF, Shawagfeh NR. The prevalence of orbital complications among children and adults with acute rhinosinusitis. Braz J Otorhinolaryngol. 2013;79:716–9, http://dx.doi.org/10.5935/1808-8694.20130131.
23. Ferreira TA, Saraiva P, Genders SW, Buchem MV, Luyten GPM, Beenakker J-W. CT and MR imaging of orbital inflammation. Neuroradiology. 2018;60:1253–66, http://dx.doi.org/10.1007/s00234-018-2103-4.
24. El Mograbi A, Ritter A, Najjar E, Soudry E. Orbital complications of rhinosinusitis in the adult population: analysis of cases presenting to a tertiary medical center over a 13-year period. Ann Otol Rhinol Laryngol. 2019;128:563–8, http://dx.doi.org/10.1177/0003489419832624.
25. Caranfa JT, Yoon MK. Septic cavernous sinus thrombosis: a review. Surv Ophthalmol. 2021;66:1021–30, http://dx.doi.org/10.1016/j.survophthal.2021.03.009.
26. Costa P, Santos P, Silva AC, Silva C. Lemierre syndrome: an overlooked diagnosis in otherwise healthy patients. Acta Radiol Port. 2021;33:29–31.
27. Razek AAKA, Castillo M. Imaging lesions of the cavernous sinus. Am J Neuroradiol. 2009;30:444–52, http://dx.doi.org/10.3174/ajnr.A1398.
28. Akiyama K, Karaki M, Mori N. Evaluation of adult Pott's puffy tumor: our five cases and 27 literature cases. Laryngoscope. 2012;122:2382–8, http://dx.doi.org/10.1002/lary.23490.
29. Velasquez N, Strober W, Shaffer A, Stapleton A. Clinical and radiologic characterization of frontal sinusitis in the pediatric population. Ann Otol Rhinol Laryngol. 2021;130:923–8, http://dx.doi.org/10.1177/0003489420987969.
30. Álvarez Jáñez F, Barriga LQ, Iñigo TR, Roldán Lora F. Diagnosis of skull base osteomyelitis. Radiog Rev Publ Radiol Soc North Am Inc. 2021;41:156–74, http://dx.doi.org/10.1148/rg.2021200046.
31. Chapman PR, Choudhary G, Singhal A. Skull base osteomyelitis: a comprehensive imaging review. Am J Neuroradiol. 2021;42:404–13, http://dx.doi.org/10.3174/ajnr.A7015.
32. Ozcan H, Avcu S, Lemmerling M. Radiological findings in a rare case of eyelid swelling: Pott's puffy tumor. JBR-BTR. 2012;95:27–8, http://dx.doi.org/10.5334/jbr-btr.559.
33. Morón FE, Morriss MC, Jones JJ, Hunter JV. Lumps and bumps on the head in children: Use of CT and MR imaging in solving the clinical diagnostic dilemma. RadioGraphics. 2004;24:1655–74, http://dx.doi.org/10.1148/rg.246045034.
34. Giannoni C, Sulek M, Friedman EM. Intracranial complications of sinusitis: a pediatric series. Am J Rhinol. 1998;12:173–8, http://dx.doi.org/10.2500/105065898781390127.
35. Abou-Al-Shaar H, Mulvaney GG, Alzhrani G, Gozal YM, Oakley GM, Couldwell WT. Nocardial clival osteomyelitis secondary to sphenoid sinusitis: an atypical skull base infection. Acta Neurochir (Wien). 2019;161:529–34, http://dx.doi.org/10.1007/s00701-018-3768-5.
36. Foerster BR, Thurnher MM, Malani PN, Petrou M, Carets-Zumelzu F, Sundgren PC. Intracranial infections: clinical and imaging characteristics. Acta Radiol. 2007;48:875–93, http://dx.doi.org/10.1080/02841850701477728.
37. Weingarten K, Zimmerman RD, Becker RD, Heier LA, Haimes AB, Deck MD. Subdural and epidural empyemas: MR imaging. AJR Am J Roentgenol. 1989;152:615–21, http://dx.doi.org/10.2214/ajr.152.3.615.
38. Kastrup O, Wanke I, Maschke M. Neuroimaging of infections of the central nervous system. Semin Neurol. 2008;28:511–22.
39. Tsuchiya K, Osawa A, Katase S, Fujikawa A, Hachiya J, Aoki S. Diffusion-weighted MRI of subdural and epidural empyemas. Neuroradiology. 2003;45:220–3, http://dx.doi.org/10.1007/s00234-003-0949-5.
40. Lal D, Hwang PH. In: Lal D, Hwang PH, editors. Frontal sinus surgery: a systematic approach. Phoenix: Springer International Publishing; 2019.
41. Mohan S, Jain KK, Arabi M, Shah GV. Imaging of meningitis and ventriculitis. Neuroimaging Clin N Am. 2012;22:557–83, http://dx.doi.org/10.1016/j.nic.2012.04.003.
42. Osborn AG, Hedlund GL, Salzman KL. Meningitis. In: Osborn AG, Hedlund GL, Salzman KL, editors. Osborn's Brain. Elsevier; 2018. p. 349–50.