

Brain abscess of odontogenic origin – a case report

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ABSTRACT:

Odontogenic brain abscess is a rare life-threatening entity accounting for up to 5% of brain abscesses. Brain abscess mortality rate has decreased substantially in the last decades, but still has a significant mortality rate and potentially severe consequences. The infection can be spread to the brain directly or hematogenously. The most common site for direct intracranial spread is the frontal sinus. We present a case of a young male with odontogenic sinusitis and brain abscess, due to untreated periapical process.

KEYWORDS: odontogenic brain abscess, brain abscess imaging, intracranial sinusitis complications

SAŽETAK

ODONTOGENI APSCES MOZGA – PRIKAZ SLUČAJA

Odontogeni apsces mozga rijetko je životno-ugrožavajuće stanje, oko 5 % apscesa mozga odontogenog su porijekla. Iako je danas smrtnost od moždanih apscesa znatno manja nego ranije, i dalje je značajna, a posljedice apscesa mozga mogu biti ozbiljne i značajno smanjiti kvalitetu života pacijenata. Infekcija se u mozak može proširiti direktno ili hematogeno. Najčešći izvor infekcije kod direktnog širenja je frontalni sinusitis. Prezentirat ćemo slučaj mladog muškarca s odontogenim sinusitisom i apscesom mozga, uzrokovanim neliječenim periapikalnim procesom.

KLJUČNE RIJEČI: odontogeni apsces mozga, radiološka dijagnostika apscesa mozga, intrakranijalne komplikacije sinusitisa

INTRODUCTION

Odontogenic infections are among the most common diseases, but they mainly remain localized and are self-limiting. They can spread to head and neck spaces, or rarely disseminate to more distant sites. Some of the most severe, possibly even life-threatening complications of an odontogenic infection are airway obstruction, mediastinitis, endocarditis, sepsis, necrotizing fasciitis, Lemierre syndrome and brain abscess (Burgos-Larraín LF, 2022) (Neal TW, 2022).

Brain abscesses have an incidence of around 1 per 100.000 per year (Burgos-Larraín LF, 2022) (Neal TW, 2022), but have a mortality of 10-15%, with severe sequelae present in 20-30% of patients (Brouwer MC, 2014). Brain abscesses are commonly caused by a direct spread of the infection from adjacent structures (mastoiditis, otitis media, sinusitis), resulting mainly in one solitary abscess. Abscesses caused by direct extension comprise 12-25% of all brain abscesses by Brouwer and al. Hematogenous spread is also a common pathway, with abscesses often being multiple and usually found in the middle cerebral artery territory. A brain abscess can also be caused by penetrating head trauma, or it can be iatrogenic, i.e. after a neurosurgical procedure. (Brouwer MC, 2014)

Brain odontogenic abscesses account for a very small number of brain abscesses – only 2 - 5%. (Burgos-Larraín LF, 2022). The spread of the dental infection to the brain can be contiguous or hematogenic (Brouwer MC, 2014).

Brain abscesses develop in four stages. The first stage, called the early cerebritis, takes place within the first three days and is characterized by an ill-defined edema and hyperemia due to the infection, without the capsule. The next stage, the late cerebritis, starts from the second to third day after the infection, lasting for a week to ten days. It is characterized by an irregular, ill-organized rim surrounding the necrotic foci beginning to merge, and the edematous surrounding brain. Early capsule stage starts after approximately a week, with a well-defined rim around the now coalescent necrotic core. It is the stage with the most prominent ring-like capsule enhancement and a very high diffusion restriction in the core of the abscess, corresponding to the pus. The last stage is the late capsule stage, where the wall thickens, and the inflammation and the edema gradually reduce (Osborn AG, Hedlund GL, 2017).

CASE REPORT

A 28-year-old male patient has been admitted to the hospital due to a severe pulsating occipital headache (VAS 9/10) with nausea, vomiting and photophobia, lasting for three days prior to the admission. The patient mentioned having a previous “flu” two weeks ago, along with a strong headache and sinusitis. CRP has been significantly increased – 183, and white blood cell count was $20 \times 10^9/L$ with 89% neutrophils, showing a bacterial infection.

An emergency CT scan showed an ill-defined hypodensity in the right frontal brain lobe (Figure 1.). The right maxillary sinus, middle and superior nasal meatus, ipsilateral anterior ethmoid cells and the right side of frontal sinus were opacified, filled by purulent content, confirming the right-sided sinusitis. Deep cavities and untreated periapical processes of teeth 16 to 17 were present, thus an odontogenic sinusitis with intracranial extension was suspected (Figure 2.). MRI scan was performed, showing a very high T2 signal in the center of the right frontal lobe lesion with an irregular iso- to hypointense T2 rim, surrounded by a hyperintense perifocal edema. There were SWI hypointensities and slight T1 precontrast hyperintensities of the rim, corresponding to petechial hemorrhages. The findings corresponded to a brain abscess in a late cerebritis stage (Figure 3.). A small layer of debris, showing signs of restricted diffusion, was seen in the occipital ventricular horns, therefore a possible ventriculitis was suspected (Figure 4.). There was an extraaxial thin fluid collection frontally, adjacent to the inflamed frontal sinus, surrounded by a thickened, strongly enhancing dura. The collection showed restricted diffusion, corresponding to an empyema (Figure 5.). The patient underwent an osteoplastic frontal craniotomy the same day and the abscess has been evacuated. Intraoperatively, the damaged dura was found frontobasally, along with leptomeningitis, confirming the spread of the infection from the frontal sinus. The teeth 16 to 18 extraction, right maxillary sinus antrostomy and right ethmoidectomy were performed two days later. The bacterial cultures taken intraoperatively from the abscess showed *Streptococcus viridans*, *Staphylococcus aureus* and coagulase-negative *Staphylococci*, as well as anaerobic *Parvimonas micra*, *Peptostreptococcus sp.*, *Fingoldia magna* and *Cutibacterium sp.* The patient received antibiogram-targeted antibiotic therapy (empiric antibiotic therapy with Medazol has been replaced by Mero-penem, Vancomycin continued) and his clinical and neurological status have improved significantly (GCS 15, no neurological deficit, no psychological symptoms).

The patient has been clinically well for three weeks after the operation, after which he started complaining about a severe frontal headache and sniffing, after which he became somnolent. Due to such rapid clinical deterioration, another emergency CT scan has been performed. It showed mildly widened lateral ventricles, a progression in comparison to previous examinations, so ventriculitis has been suspected. The next day the patient's condition progressed with a mild left arm paresis and neck stiffness. An osteoclastic recraniotomy, with an implantation of ventricular drainage has been performed, and the previously clinically suspected rhinoliquorrhoea has been confirmed and attended to. The right arm hemiparesis persisted for another several days, after which the patient fully recovered.

More than a year after the hospitalization the patient is without any neurological deficit. Cranioplasty is being planned within 6 months.

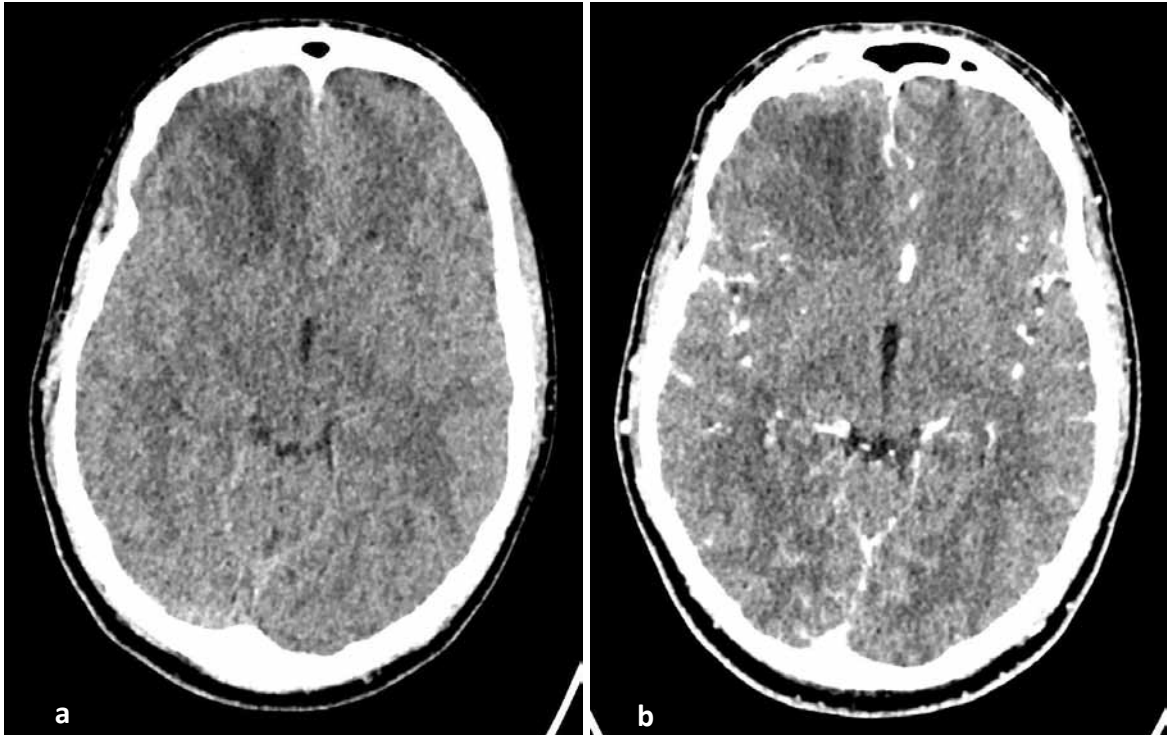


Figure 1. Precontrast (a) and post-contrast (b) brain CT demonstrating an ill-defined right frontal lobe hypodensity, no evident post-contrast enhancement.

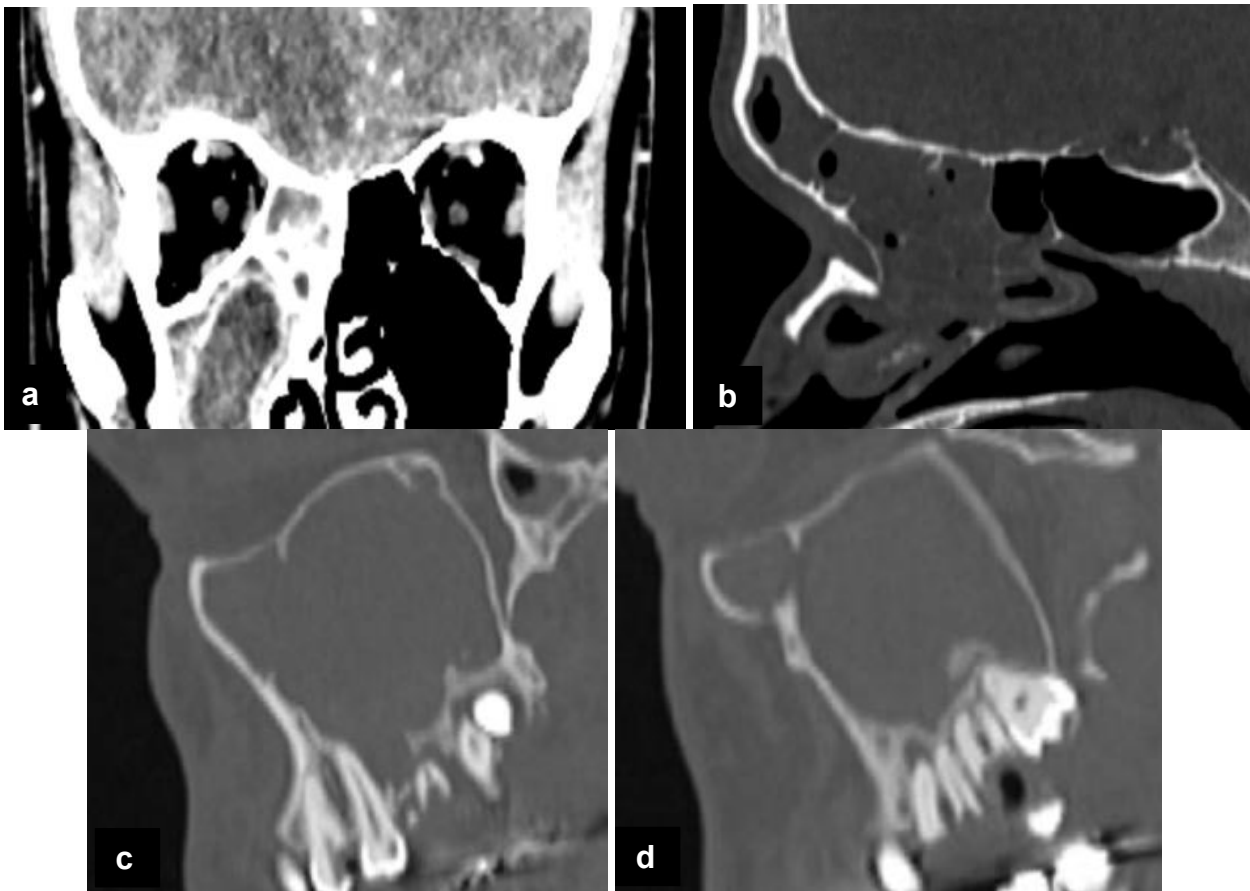


Figure 2. CT showing the unilateral inflammatory content in paranasal sinuses (a), opacification of right maxillary sinus and anterior ethmoid cells (b), opacified right maxillary sinus (c, d) with a floor defect adjacent to periapical process of tooth 16 (c), deep cavities of teeth 16 and 17 with periapical processes (d).

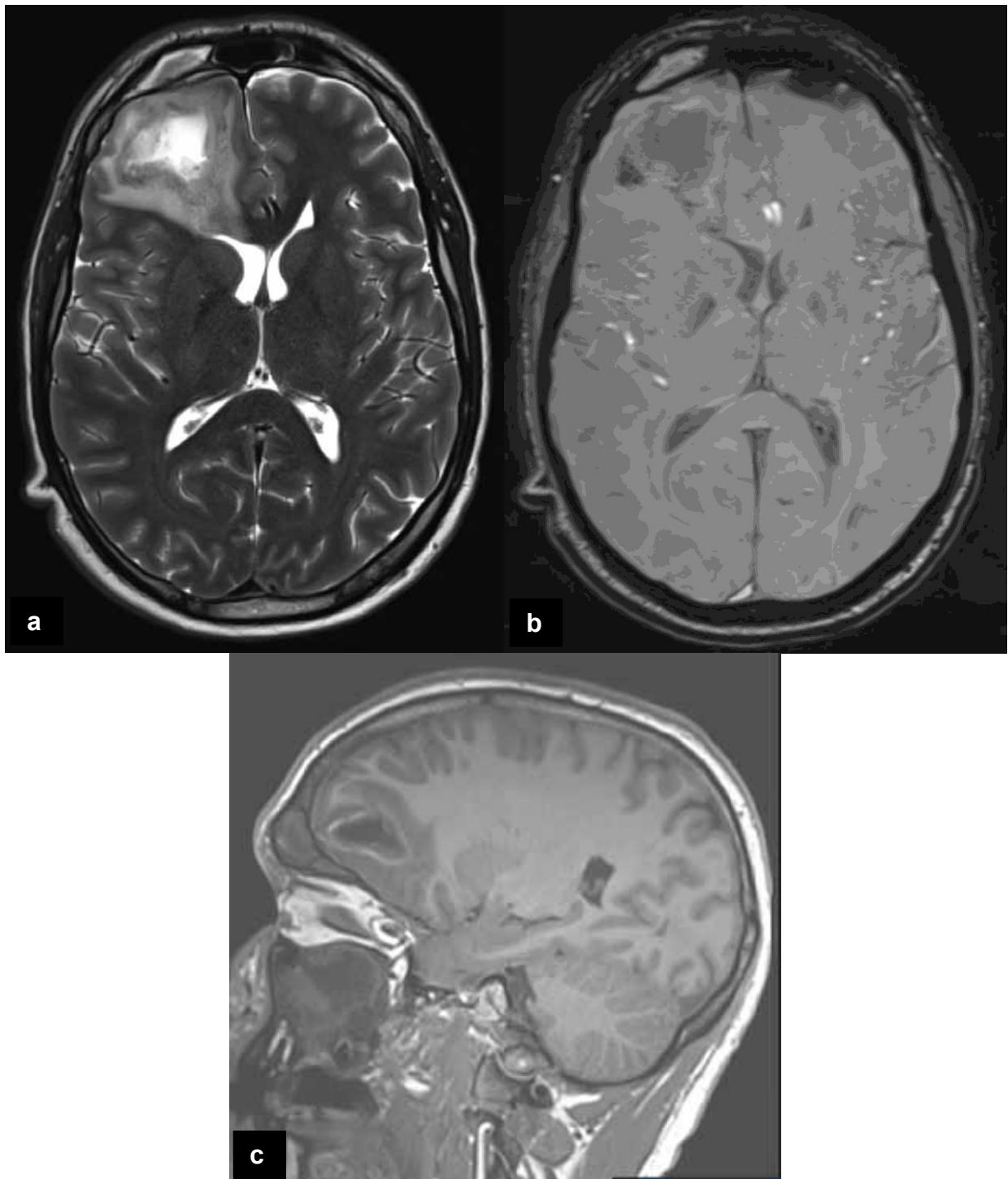


Figure 3. Brain MRI showing a very hyperintense lesion center on T2-weighted image, surrounded by an iso- to hypointense rim and a perifocal hyperintense edema (a), hypointense dots in the forming capsule on SWI sequence (b) and precontrast T1-weighted image rim hyperintensities (c), corresponding to a late cerebritis stage of a brain abscess. Frontal (a - c) and maxillary sinus (c) opacification. Right frontal ventricular horn compression (a, b).

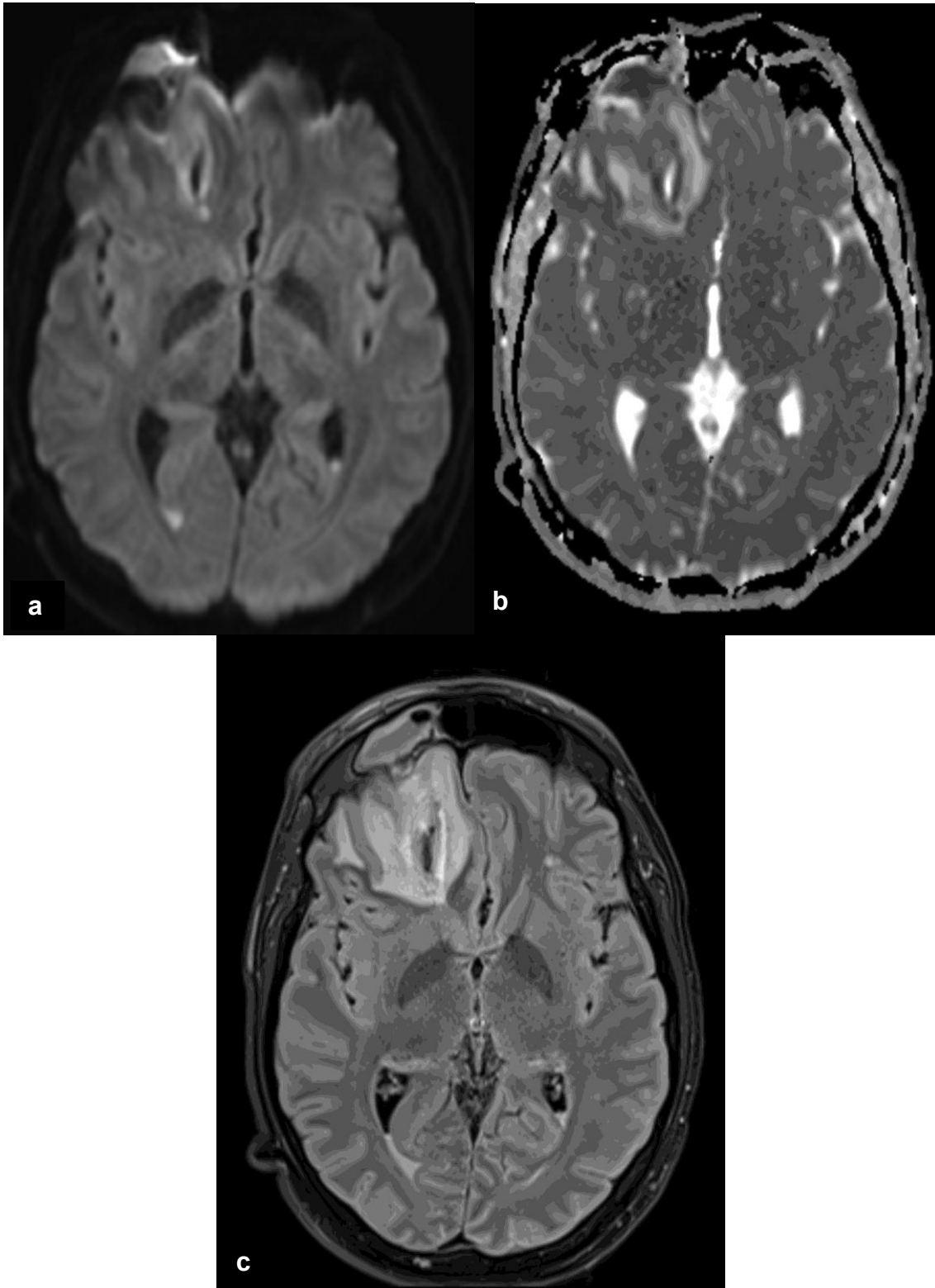


Figure 4. Debris in occipital horns of lateral ventricles showing signs of restricted diffusion (a, b) and the corresponding FLAIR hyperintensity (c). The right frontal lobe abscess and the frontal sinusitis are present (a - c).

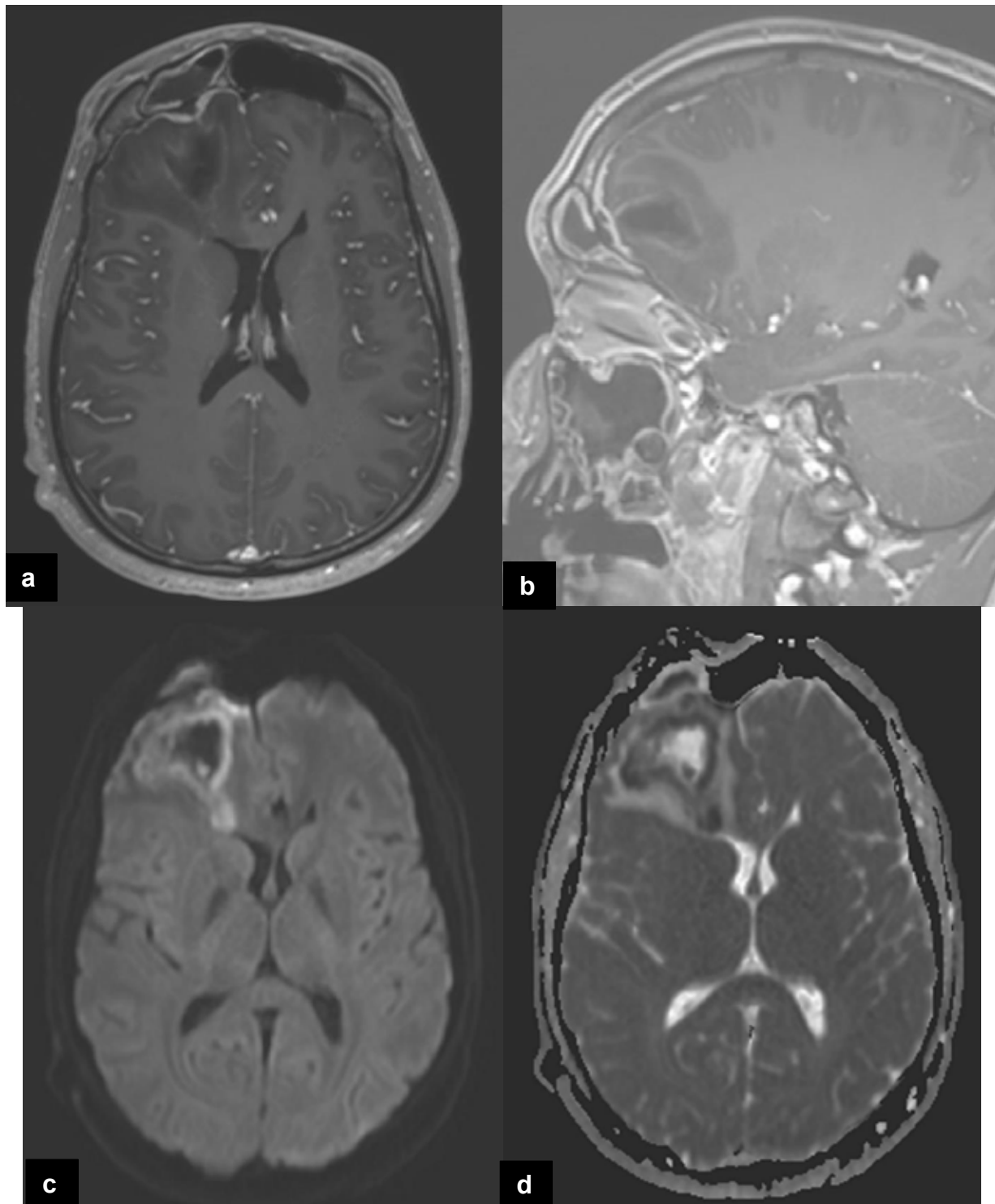


Figure 5. Right frontal thin extraaxial fluid collection, adjacent to the right frontal sinus, surrounded by thickened, strongly enhancing dura on axial (a) and sagittal (b) T1-weighted images, with signs of restricted diffusion on DWI (c) and ADC (d), corresponding to empyema. Signs of restricted diffusion in the abscess, corresponding to late cerebritis stage (c, d). Maxillary sinusitis (b).

DISCUSSION

Most common cause of unilateral sinusitis is odontogenic (Loureiro RM, Naves EA, Zanello RF, Sumi DV, Gomes RLE, Daniel MM, 2019), usually arising from an untreated periapical process (Whyte A, Boeddinghaus R, 2019). Ipsilateral mucosal thickening, or the opacification of all ipsilateral paranasal sinuses in more advanced cases is seen, confirming the suspicion of odontogenic sinusitis. The disruption of sinus wall next to the periapical process can sometimes be visualized on imaging. Our patient had periapical processes of maxillary right molars with a clear adjacent sinus floor defect. There were signs of unilateral, right maxillary, ethmoid and frontal sinus inflammatory changes, all in concordance with odontogenic sinusitis.

Brain infections can be caused by a hematogeneous spread of bacteria (predominantly *Streptococcus pneumoniae*, *Neisseria meningitidis* and *Hemophilus influenzae*), but also by a direct spread of the infection from the adjacent structures, most commonly from sinuses or mastoid cells, resulting in a solitary brain abscess (Brouwer MC, 2014). Our patient was male, in his twenties, with a solitary frontal lobe abscess which originated from the frontal sinus. Intracranial complications are most common in male adults in their 2nd and 3rd decades and adolescents. The frontal sinusitis is the most common cause of intracranial complications (Koontz NA, 2017), due to the emissary valveless Behcet veins connecting the mucosa of posterior frontal sinus to emissary diploic veins and dura (Yong A, Gomati A, Khor K, KheiHu M, Kanodia AK, 2020). Therefore, the bone defect in the posterior frontal sinus is not necessarily seen. The frontal lobe is also the most commonly affected brain area (Burgos-Larraín LF, 2022), like in our patient.

Odontogenic brain abscesses are rare, accounting for up to 5% of all brain abscesses (Burgos-Larraín LF, 2022). Most common bacterial species cultivated from odontogenic brain abscesses are *Streptococcus sp.*, *Fusobacterium*, and *Porphyromonas* (Burgos-Larraín LF, 2022) (Neal TW, 2022). Odontogenic infections are mostly mixed aerobic-anaerobic, which was also the case in our patient. *Streptococcus viridans*, *Staphylococcus aureus* and coagulase-negative *Staphylococci*, as well as anaerobic *Parvimonas micra*, *Peptostreptococcus sp.*, *Fingoldia magna* and *Cutibacterium sp.* were cultivated from the abscess. *Streptococcus viridans* is a large group of aerobic bacteria, very common in the mouth, with *Streptococcus mutans* being the most common cause of caries. *Staphylococcus aureus* is common on skin, but also in pharynx and nose cavity and is the second most common bacteria isolated in pus samples of odontogenic origin, followed by coagulase-negative *Staphylococcus*, as reported by Bahl and al. (Bahl R, Sandhu S, Singh K, Sahai N, Gupta M, 2014). *Parvimonas micra* is commonly isolated from dental plaques causing periodontal disease (Shimizu K, Horinishi Y, Sano C, Ohta R, 2022), *Fingoldia magna* can be found on skin and in the mouth (Neumann A, Björck L, Frick IM, 2020), and *Cutibacterium*, previously known

as *Propionibacterium*, is usually found on skin, but can also cause periodontal disease (Brotfain E, Koyfman L, Saidel-Odes L, Borer A, Refaely Y, Klein M, 2015). Therefore, all of the bacteria cultivated from our patient's brain abscess can be connected to the odontogenic origin of the infection.

In our patient, CT and MRI confirmed the diagnosis of a brain abscess and an empyema frontally, adjacent to the inflamed frontal sinus. The suspected findings were confirmed intraoperatively. Earlier stages of brain abscess are more difficult to distinguish from other conditions which can resemble them on imaging, due to their ill-defined margins and CT hypodense and T2 MR hyperintense areas. They include neoplasms, ischemia or encephalitis. In the later phases of brain abscess formation, when the capsule around the necrotic core forms, the differential diagnosis is those of typical ring-enhancing brain lesions. The most common ones, apart from the brain abscess, include glioblastoma and metastases, and the less common consist of demyelination and resolving intracerebral hematomas. The DWI and ADC sequences are very important because the abscesses typically show a very high level of restricted diffusion. MR perfusion can also be performed, showing an increased cerebral blood volume (CBV) for tumors and low CBV for abscesses. MR spectroscopy can help narrow the differential diagnosis, showing a peak of lactate, cytosolic amino acids, acetate and succinate in an abscess, in contrast to a choline peak and NAA decrease in glioblastoma (Osborn AG, Hedlund GL, 2017) (Thurnher MM, 2018). Our patient had clear clinical history and findings suggestive of sinusitis, with laboratory findings of a bacterial infection which, given the imaging findings on CT and MRI, made the diagnosis of a brain abscess highly probable. The defect in the maxillary sinus floor, the ipsilateral sinusitis, and the confirmation of bacterial cultures from the brain abscess, compatible with an odontogenic infection, confirmed the suspected diagnosis of an odontogenic brain abscess in our patient.

Ventriculitis is a possible, serious complication of a brain abscess, occurring in approximately up to 35% of brain abscesses, more commonly in males. These patients show a rapid clinical deterioration and only 40% survive with a good functioning outcome (Osborn AG, Hedlund GL, 2017). Our patient had signs of ventriculitis, but there was only a small amount of debris intraventricularly and only a slight enlargement of the lateral ventricles, without the typical periventricular hypodense/T2 hyperintense halo. Our patient recovered and is now without any neurological deficit.

CONCLUSION

Odontogenic infections are very common but rarely have life-threatening sequelae. They are the most frequent cause of unilateral sinusitis. Sinusitis is also quite common but causes intraorbital or intracranial complications in a small number of patients. Frontal sinusitis most commonly causes intracranial complications, mostly affecting young males. Brain abscesses of odontogenic origin only make up 2 to 5% of all brain abscesses but can

have devastating consequences. Clinical suspicion and findings, appropriate imaging features and the confirmation of bacterial cultures compatible with an odontogenic origin of an infection are vital for establishing the right diagnosis. Communication between the clinician and radiologist is essential for choosing the right imaging protocol on CT and MRI to confirm the diagnosis of odontogenic sinusitis with intracranial complications.

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