CASE REPORT

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Brain abscess as a complication of sinusitis in an 8-year-old female patient – case report

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Abstract

Introduction. Brain abscess is accumulation of pus in brain parenchyma. Predisposing factors include head trauma and spreading the inflammatory process from remote locations or from surroundings. The most common location is the frontal lobe. Frequent pathogens that cause paediatric BA are S. pneumoniae, viridans and S. aureus. Headache is a common symptom. The first imaging test performed is head CT. This is crucial for correct diagnosis and treatment. BA is a serious health problem, with a mortality rate up to 24%.

Case Report. The medical history is presented of an 8-year-old female patient who was admitted to hospital in a serious general condition with non-specific symptoms. Imagining diagnostics showed inflammatory changes of the sinuses communicating with the right orbit and nasal cavity. Broad-spectrum antibiotic therapy was administered, but later the patient's condition deteriorated. The lesion had progressed. She underwent endoscopic surgery, but a few days later her condition was again serious, despite regression in imaging studies.

Key words

inflammation, complication, sinusitis, brain abscess

INTRODUCTION

Brain abscess is a rare disease. It is estimated to occur in 0.4–0.9 per 100,000 people per year (more often in females), of which approximately 42% of cases are reported among paediatric patients [1]. Deep, focal accumulation of purulent content in brain parenchyma, called an abscess, is usually not a primary condition, but a consequence of the spread of infection from surrounding structures (40–50% of cases). For this reason, brain abscesses are typically located in the lobes adjacent to the sites of primary infections. The most common location is the frontal lobe as a consequence of sinusitis, followed by the parietal lobes as a consequence of otitis media or mastoiditis. Slightly less frequently, pathogens spread through the blood from more distant infection foci, such as lung infections or endocarditis [2]. The most common pathogens causing brain abscesses include Gram-positive bacteria, in particular aerobic and anaerobic *streptococci* (30–60%) and *staphylococci* (up to 20%). Among Gram-negative bacteria, *enteric bacilli* are relatively frequently detected (23–33%) [3]. Brain abscess is a serious health problem with a mortality rate of up to 24%, and as a consequence, may lead to a serious complication – perforation of the abscess into the ventricular space of the brain [4].

This report presents the case of an 8-year-old female with a brain abscess in which the disease was unusually mild, despite a diffuse inflammatory process.

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CASE REPORT

An 8-year-old female patient was admitted to the Intensive Care Unit (ICU) in a serious general condition, sleepy, confused, with a Glasgow Coma Scale (GCS) score of 8, presenting with vomiting and headache. In the neurological examination, right eye exophthalmos, positive meningeal symptoms and cutaneous hypersensitivity were observed. The computed tomography (CT) scan of the head showed inflammatory changes of the sinuses with the fluid space of the sphenoid sinus and part of the ethmoid sinuses, communicating with the right orbit and nasal cavity, with destruction of the surrounding bony structures. Broad-spectrum antibiotic therapy (Meropenem, Vancomycin, Metronidazole), fluid therapy, antioedematous, anticoagulant, analgesic and antifungal prophylaxis were administered. Blood and cerebrospinal fluid (CSF) cultures came back positive for *Streptococcus pneumoniae*.

Central nervous system and orbital magnetic resonance imaging (MRI) (Tab. 1, I) revealed a superinfected lesion of the right sphenoid sinus/posterior ethmoidal cells, communicating with the pericerebral space and infiltrating the right frontal lobe, with possible formation of microabscesses (Fig. 1, 2). The lesion was consistent with meningeal herniation within meningal infection as a consequence of acute sinusitis. Conservative treatment, a follow-up head CT scan after 2 weeks, and deferred surgical treatment were recommended.

After clinical improvement, the patient was transferred to the Paediatric Neurology Department. Two days later, her general condition deteriorated again, with the appearance of neurological symptoms and elevated inflammatory

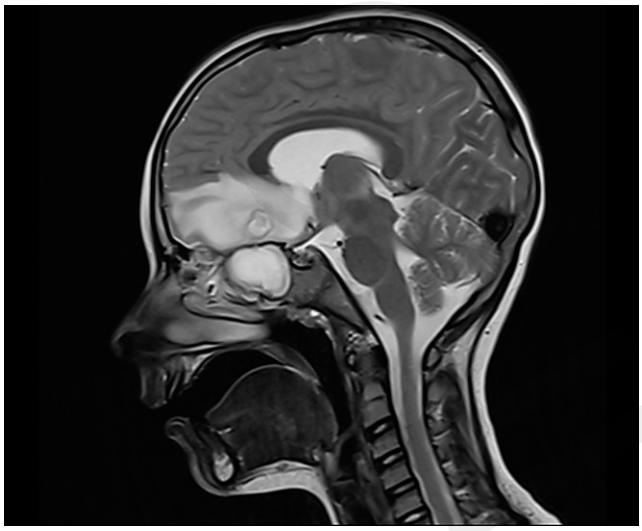

Figure 1. T2 MRI of the head. Sagittal section of the brain showing inflammatory infiltrate, edematous changes and abscess of the right frontal lobe and inflammatory changes of the sinuses with the fluid space of the sphenoid sinus

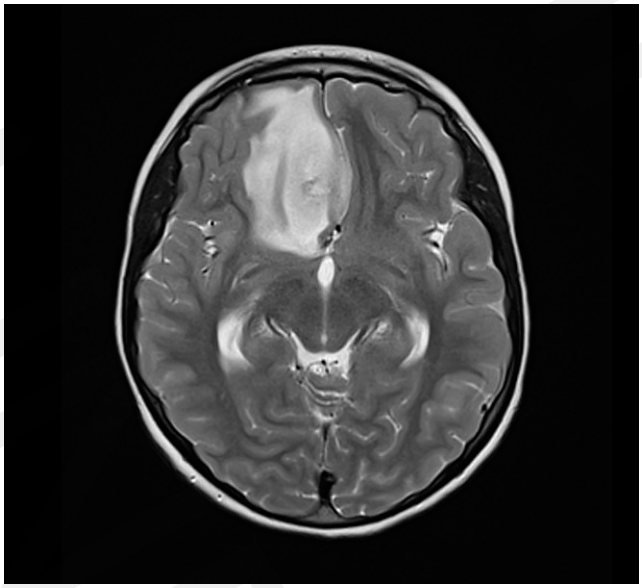

Figure 2. T2 MRI of the head. Transverse section of the brain showing inflammatory infiltrate, edematous changes and abscess of the right frontal lobe

markers. A follow-up head CT scan showed progression of the central nervous system (CNS) lesions. After neurosurgical consultation, an urgent MRI scan of the head was performed, which showed significant progression of inflammatory infiltration and swelling of the right frontal lobe with a formed abscess. Dilatation of the ventricular system with the presence of cerebrospinal fluid was noted (Tab. 1, II).

The patient was qualified for urgent endoscopic surgery of the sphenoid sinus in the Department of Otolaryngology. After the operation, the patient was transferred to the ICU. In the ICU, vital signs were monitored and post-operative analgesia, fluid therapy, antibiotic therapy (Meropenem, followed by Ceftriaxone, Vancomycin and Metronidazole), antithrombotic and antifungal prophylaxis were administered. On the 4th day of the ICU stay, the patient was transferred, in good general condition and fully alert, to the Paediatric Neurology Department for further treatment. During her stay in the ward, the girl remained in good condition, active, without any abnormalities in the neurological examination. Antibiotic therapy was continued as recommended by the consulting epidemiologist, as well as antifungal prophylaxis.

On the 12th day after surgery, a follow-up MRI of the head was performed. Examination showed partial regression of the inflammatory infiltrate and oedematous lesions. The size of the inflammatory infiltrate and abscess decreased, the ventricular system was not dilated, and there was no CSF seepage (Tab. 1, III). During the stay, the patient was consulted by an ENT specialist, neurosurgeon and epidemiologist with the diagnosis of chronic sinusitis. Antibiotic therapy (Vancomycin, Metronidazole) was continued.

On the 20th day after surgery, the patient developed vomiting and abdominal pain. Laboratory tests revealed elevated liver enzymes: ALT 61 U/l (normal: <39 U/l), AST 157 U/l (normal: <51 U/l), normal lipase and amylase levels, stool virological tests showed no viral antigens. Fluid therapy was recommended and Vancomycin was discontinued. A follow-up MRI performed one week later showed significant regression of the lesions (Tab. 1, IV). The patient was discharged in good general condition with a recommendation for follow-up in the neurology clinic and antibiotic therapy with cefuroxime.

A few weeks later, the patient was re-admitted to the Paediatric Neurology Department because of headaches, fever and vomiting that had been present for several days. Head MRI showed cerebral oedema, inflammatory infiltration of the left frontal lobe due to perforation of the inflammatory process from the sphenoid sinus to the anterior cranial fossa. A new lesion at the base of the left frontal lobe, and widening of the pericerebral fluid space were also observed (Tab. 1, V). Physical examination, laboratory parameters and the cerebrospinal fluid analysis were normal.

In order to determine the cause of the disease, PCR tests were performed to detect the 14 main pathogens causing meningitis/encephalitis, as well as adenovirus, *Borrelia burgdorferi*, Epstein-Barr Virus, HHV 6, HHV 7,Tick-borne encephalitis virus (TBEV). All tests came back negative. It was decided not to proceed with surgical intervention and to maintain the conservative treatment. After a week of hospital observation, the patient was discharged with the recommendation of antibiotic therapy with rimethoprim / sulfamethoxazole.

After one month, a follow-up MRI showed regression of the oedematous changes and inflammatory infiltration of the brain (Tab. 1, VI) (Fig. 3). Further antibiotic therapy for one month and a follow-up MRI after 3 months were recommended.

DISCUSSION

Brain abscess (BA) is a condition in which pus collects in the brain parenchyma. The most common locations are frontal–temporal, frontal–parietal, parietal, cerebellar, and occipital lobes, and cerebellum. A rare location is in the brainstem and is established to be 1% of cases [1]. The most common pathogens that cause BA among children are *Streptococcus pneumoniae*, *viridans* and *Staphylococcus aureus* [5].

Predisposing factors can be identified in 56–86% cases of brain abscess in the paediatric population [6]. These factors

Table 1.

	I.	II.	III.	IV.	V.	VI.
	One week before the surgery	Day of the surgery	Two weeks after the surgery	Four weeks after the surgery	Four months after the surgery	Five months after the surgery
Inflammatory infiltrate and oedematous changes at the base of the right frontal lobe	38x8x18 mm	60x36x33 mm	59x35x25 mm	Significant regression, only visible features of slight swelling at the longitudinal fissure of the brain	Area of oedematous lesions and inflammatory infiltration of the brain in the midline area at the longitudinal fissure of the brain in the area of the base of the right frontal lobe with discrete increased intensity	Area of oedematous lesions and inflammatory infiltration of the brain in the midline area at the longitudinal fissure of the brain in the area of the base of the right frontal lobe of lesser severity
Abscess of right frontal lobe	Diameter about 8 mm	18x12x14 mm	12x11x13 mm	Total regression	Absent	Absent
Contrast enhancement of abscess capsule, adjacent meninges and nasal sinus lesion	Slight post-contrast enhancement of meninges at the base of the frontal lobe.	Prominent post-contrast enhancement of the abscess capsule, adjacent meninges and nasal sinus lesions	Clear contrast enhancement of the abscess capsule and nasal sinus lesions, slightly weaker enhancement of the adjacent meninges,	Slight regression of the dimension of the mucocele-like lesion involving the cuneiform sinus and partially the posterior situs on the right side, with a clear capsule undergoing peripheral enhancement	Stronger post-contrast enhancement and thickening of the meninges near the base of the right frontal lobe, and near the base of the left frontal lobe with widening of the paracentral fluid space to about 6 mm, possible features of meningitis. A lesion measuring about 23x25x21 mm, comparable in size to the previous study, with a more homogeneous high signal in T2-dependent images - features of a fluid lesion, weaker marginal post-contrast enhancement, only in the cephalic region an area of strong meningeal postcontrast enhancement is visible. In the caudal-right part of the lesion, a fluid area with numerous septa is visible. Ethmoidal cells with slightly thickened mucosa.	The area of meningeal enhancement near the base of the right frontal lobe and near the base of the left frontal lobe is less intense, with no features of widening of the parocerebral fluid space in this area. A focal lesion of the cuneiform sinus and ethmoidal cells on the right side measuring approximately 25x22x21 mm shows weak marginal post-contrast enhancement. In the caudal-right part of the lesion, a fluid area with numerous septa comparable to the previous examination. Ethmoidal cells with less intense thickening of the mucosa.
The mass effect	Absent	Displacement of the basal part of the right frontal lobe to the left side about 10mm (modeling the left simple curve), modeling the anterior cerebral arteries, modeling the frontal horns of the lateral ventricles, discrete modeling of the right optic nerve directly in front of the optic chiasm.	Significant reduction in severity: Displacement of the basal part of the right frontal lobe to the left approx. 4 mm, slight modeling of the anterior cerebral arteries, discrete modeling of the right optic nerve directly in front of the optic chiasm	Discrete segmental displacement of the base of the right frontal lobe to the left side for about 1.5mm.	Discrete, segmental shift of the base of the right frontal lobe to the left for about 2mm.	Absent
Ventricular system of the brain	Non-displaced, symmetrical and non-expanded	Widened lumen (LR dimension of right lateral ventricle shaft 13mm, left 9-10mm, width of ventricle III 8mm), asymmetry of lateral ventricles (P>L)	Non-displaced, symmetrical, not dilated, width of chamber III 5mm	Non-displaced, symmetrical and non-expanded	Non-displaced, symmetrical and non-expanded	Non-displaced, symmetrical and non-expanded

include head trauma, localized infection in the surrounding tissues, or spreading of the inflammatory process from remote locations [7]. Children with congenital heart defects are also more prone to brain abscess, as the infected microbacterial embolism is not eliminated by the pulmonary filter. Congenital and acquired immunodeficiency also increases the risk of the disease [6]. Recently, prevalence of paediatric brain abscess has been reduced due to better management of the predisposing factors, such as otorhinolaryngological infections or congenital heart defects [2].

The classic triad of symptoms in brain abscess consists of headache, fever and focal neurological deficits [8]. However, all three are observed only in 20% of patients [9]. Vomiting, nausea, encephalopathy and seizures may also occur [8]. The most common symptom is headache [1]. The clinical presentation depends on the number of abscesses, their location and the size of the lesions, as well as the causative microorganism [2]. Behavioural change may indicate frontal lobe involvement. Lower cranial nerve palsies, ataxia, and encephalopathy may be characteristic of lesions in the

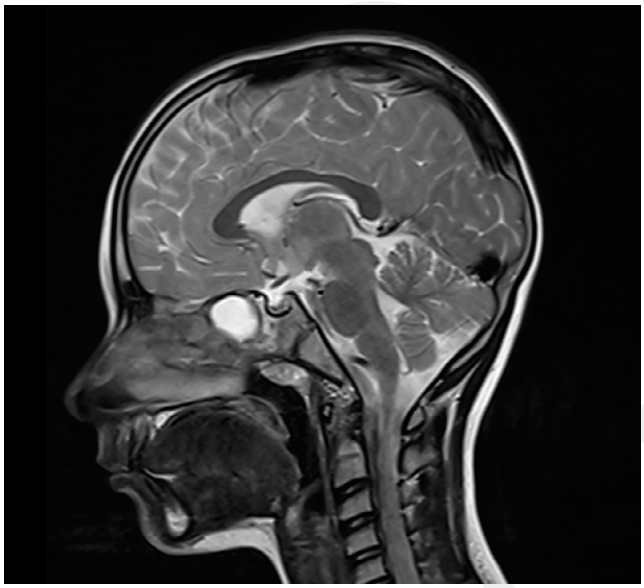

Figure 3. T2 MRI of the head. Sagittal section of the brain showing the results of treatment. The brain abscess is absent and there is a focal lesion of the cuneiform sinus and ethmoidal cells on the right side

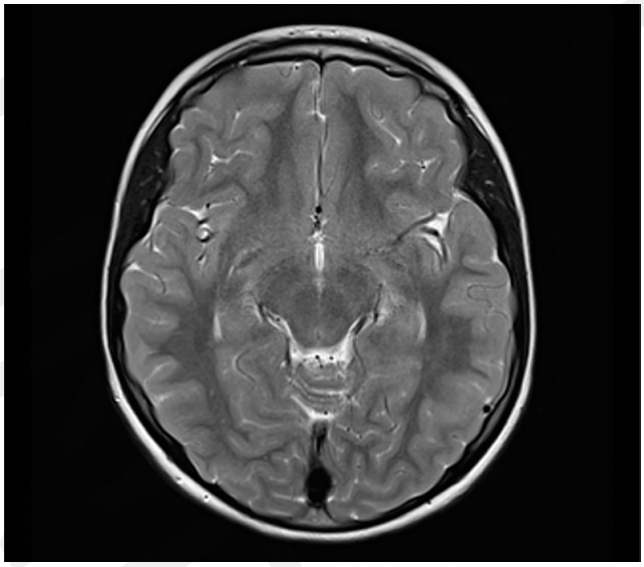

Figure 4. T2 MRI of the head. Transverse section of the brain showing the results of treatment. There is a focal lesion of the cuneiform sinus and ethmoidal cells on the right side

posterior fossa [8]. Abscesses located in the brainstem can cause nerve paralysis and disturbances in afferent and efferent long tracts, leading to hemiparesis. The nerves most frequently involved include facial (VII) and abducens (VI). However, among paediatric patients with involvement of the brainstem, all cranial nerves can be palsied, except for the hypoglossal nerve (XII) [1]. The medium time from the onset of the symptoms to the diagnosis is 7–11 days [2].

The medical history is described of an 8 year old female patient who presented with headache, vomiting, right exophthalmos, and positive meningeal symptoms. Imaging scans of the head revealed inflammatory changes of the sinuses and fluid space in the sphenoid sinus, and parts of the ethmoid structures in communication with pericerebral space and infiltration of the right frontal lobe. This may indicate the spread of the infection through the sinuses.

Only 4%-20% of acute sinusitis cases result in complications [10]. Intracranial abscess is associated initially with inflammation of the frontal sinus, then the ethmoid, sphenoid and maxillary sinuses [11]. Open cranial sutures in children can be potential sites for the spreading of the inflammation [10]. The most frequent presentation of sphenoid sinusitis is headache, cranial nerve deficit, visual deficits and dysfunction of trigeminal nerve. Nasal obstruction or rhinorrhea happens rarely due to its deep anatomical situation [12].

Due to the rare occurrence and non-specific symptoms of paediatric abscesses, diagnosis is a major challenge, and a delay in diagnosis may negatively affect the subsequent prognosis [13]. Brain imaging is crucial for correct diagnosis and treatment. In most cases, the first test performed is CT of the head. Introduction of this method allowed a decrease in brain abscess-related mortality from 40 to 20% [2]. The scan can quickly detect the location, size and number of the lesions [8]. Brain abscess on CT scan manifests as a hyperdense external and internal rim that is surrounding a centre of low depletion material. This may indicate central necrosis of the lesion. Vasogenic oedema might be suspected when low attenuation accompanies the lesion. Ependymal hyperdensity is when ventriculitis occurs due to extension of the infective foci [14]. However, CT has lower sensitivity and specificity compared to MRI. Unfortunately, using CT, it is impossible to distinguish between abscess and tumour [1]. Therefore, MRI imaging has become the gold standard in the diagnosis of brain abscess as it allows location of the lesions with greater accuracy, and distinguish them from brain tumours [15]. Contrast-enhanced CT is recommended when MRI is unavailable [16]. Advanced MRI sequences, such as diffusion-weighted imaging (DWI), can improve sensitivity and play an important role in differentiating intracranial pathologies. This imaging test enables the assessment of the orbits and bone structures of the skull. The pus in pyogenic abscess is ordinarily hyperintense. A subdural empyema presents a picture resembling a parenchymal abscess and DWI allows it to be distinguished from sterile subdural effusions [17].

In paediatric brain abscesses, the first line of treatment is empiric antibiotic therapy [5], which should be chosen based on potential sources of infection [18]. Long-term pharmacotherapy is essential and should not be delayed due to the lack of microbial culture results. Broad spectrum antibiotics, such as 3rd or 4th generation cephalosporin, metronidazole, and vancomycin can be administered [19]. Therapy should be maintained for the period of 6–8 weeks. Administration for less than 45 days is linked to greater risk of relapse. If improvement is not noted after 14 days of pharmacotherapy, then neuroimaging is advisable [20]. Abscesses bigger than 2.5 cm are removed surgically or aspirated [19], whereas those under 2,5 cm are only aspirated for diagnostic purposes. Small abscesses can be treated only pharmacologically. In these patients, clinical improvement is observed earlier than the regression of the lesions in the neurological imaging tests [7].

The etiology of brain abscess can be determined with the culture of the extracted pus. The sample of the pus can be obtained by stereotactic biopsy or aspiration, rarely by craniotomy with excision. Neurosurgical intervention should be implemented when empirical antibiotic therapy does not bring improvement within 1–2 weeks. Surgical intervention is efficient in reduction of mass effect [2]. It should be performed

the very moment when the patient's condition deteriorates, or neuroimaging shows enlargement of the lesions [1]. The gold standard of surgical treatment is aspiration of the abscess. This is associated with a lower mortality rate compared to open surgery. Stereotactic aspiration can be performed on abscesses ≥ 2.5 cm, when lesions are deep or numerous, and when there is a high risk of complications [2]. Minimally invasive techniques are safer, they allow simultaneous aspiration of the pus and the performance of a biopsy [1]. Open surgery (excision) is perfomed when the abscess is located in the posterior cranial fossa, in post-traumatic abscesses, or when the lesions are multi-lobulated or superficial, and when aspiration has failed [2].

CONCLUSIONS

Early diagnosis and implementation of appropriate therapy is crucial in the treatment of brain abscess. In the case of a focal lesion in the CNS, it is essential to perform further diagnostic imaging. The gold standard of imaging tests is MRI which allows location of the lesion and differentiate brain abscess from a tumour. Additional types of scans are CT, which can be used when MRI is unavailable, and DWI, which has higher sensitivity. In the presented case, the correct diagnosis was made at an early stage based on the MRI result. However, it did not correlate with the atypical clinical picture the patient presented. Successful treatment of brain abscess and meningitis is possible due to the cooperation of specialists from different medical fields: neurologist, neuroradiologist, neurosurgeon, ENT specialist, as well as an infectious disease specialist. In the treatment process, a combination of surgical methods and antibiotic therapy is preferred.

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