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Case report

Brain abscess with pyogenic ventriculitis

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ABSTRACT

The authors present the case of a 57-year-old male with a large polymicrobial brain abscess complicated by eruption into the intraventricular space. He was treated with parenteral ampicillin, cefepime, and metronidazole and adjuvant intraventricular vancomycin/gentamicin as well as surgical debridement. The authors discuss the diagnosis, treatment, and prognosis of brain abscesses, with a focus on prior cases with pyogenic ventriculitis and those treated with intraventricular antimicrobials.

Introduction

Brain abscesses are rare in occurrence, but may result in significant morbidity and mortality [1]. Typically, brain abscesses are the result of direct spread from a contiguous infected site such as the sinuses, ears, or mouth; however, they can also result from hematogenous spread or iatrogenic inoculation [2]. Microbial etiology is dependent on the primary source of infection with the most frequently identified organisms being Staphylococcus and Streptococcus species [2]. In the case of an otogenic source, the most encountered pathogens are Enterobacteriaceae, streptococci, *Pseudomonas aeruginosa*, and *Bacteroides* [3]. Patients typically present with focal neurologic deficits, fever, or headaches. A brain abscess may erupt into the brain's ventricular spaces in severe cases. This can result in widespread meningitis and ventriculitis, which are associated with worse outcomes [1]. Treatment of brain abscesses usually entails a combination of surgical drainage and long-term antimicrobial therapy. The role of intraventricular antimicrobials has not been well defined, particularly when widespread ventriculitis is present. Herein, we present the case of a patient with a brain abscess that had erupted into the ventricular space, and who achieved near-normal recovery with a combination of surgical debridement with parenteral and intraventricular antimicrobials. We review the literature on brain abscess diagnosis, microbiology, treatment, and prognosis.

Case report

A 57 year old male with a past medical history significant for type 2 diabetes mellitus (A1c 6.8%), alcohol use disorder in remission, and chronic bilateral otitis media with a history of myringotomy tubes presented with chills and malaise of 10 days duration. Home medications included metformin, metoprolol, omeprazole, and simvastatin. He lived at home with his wife and worked as a mechanic, with no significant recent travel, animal, water, or other noteworthy exposures. He was initially diagnosed presumptively with a viral infection, but then developed progressive ear pain and difficulty speaking before presenting to a tertiary medical care center.

On presentation, he became progressively agitated and confused, leading to emergent intubation and sedation. He was given 10 mg of dexamethasone and started on intravenous vancomycin, metronidazole, and cefepime. Laboratory values were significant for a white blood cell (WBC) count of 22,900 cells/μL, hemoglobin of 14.1 gm/dL, platelet count of 292,000/μL, creatinine of 0.85 mg/dL, and glucose of 286 mg/dL. A magnetic resonance imaging (MRI) study of the head demonstrated a 5 cm abscess in the left temporal lobe (Fig. 1). The abscess extended to the surface of the left lateral ventricle and had ruptured into the ventricles causing a ventriculitis and meningitis (Fig. 2). The MRI also showed evidence of a left middle ear/mastoid cholesteatoma with superimposed

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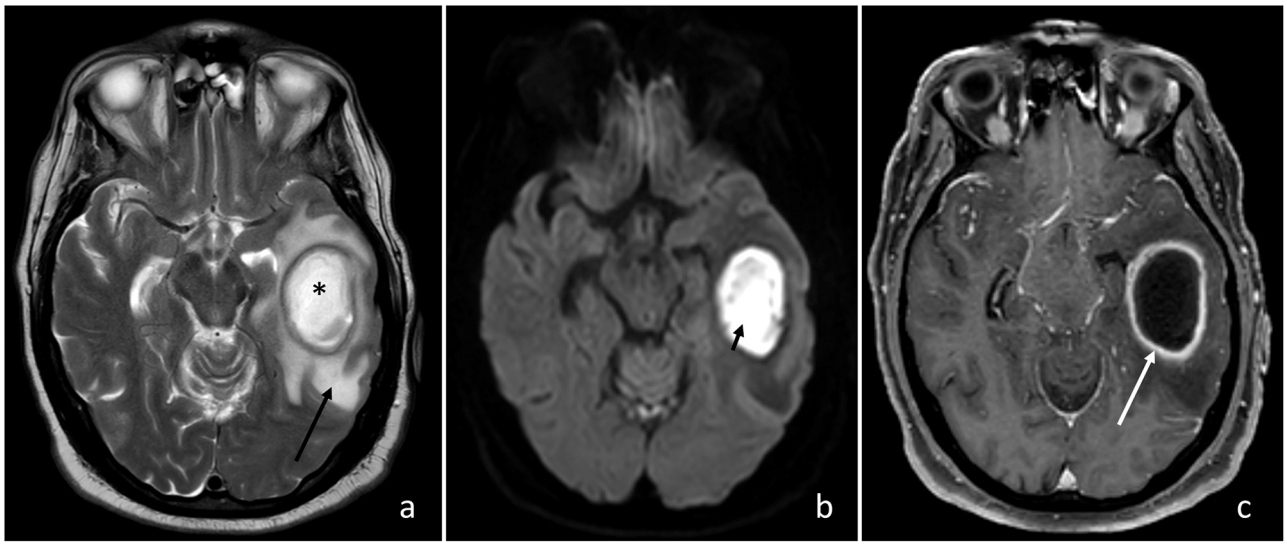

Fig. 1. Axial MRI of the brain indicative of cerebral abscess. (a) T2-weighted image shows a large T2 hyperintense lesion (asterisk) with surrounding edema (black arrow). (b) Diffusion weighted image shows marked restricted diffusion centrally within the lesion (black arrow). (c) T1-weighted post-contrast image shows a thin rim of peripheral enhancement (white arrow).

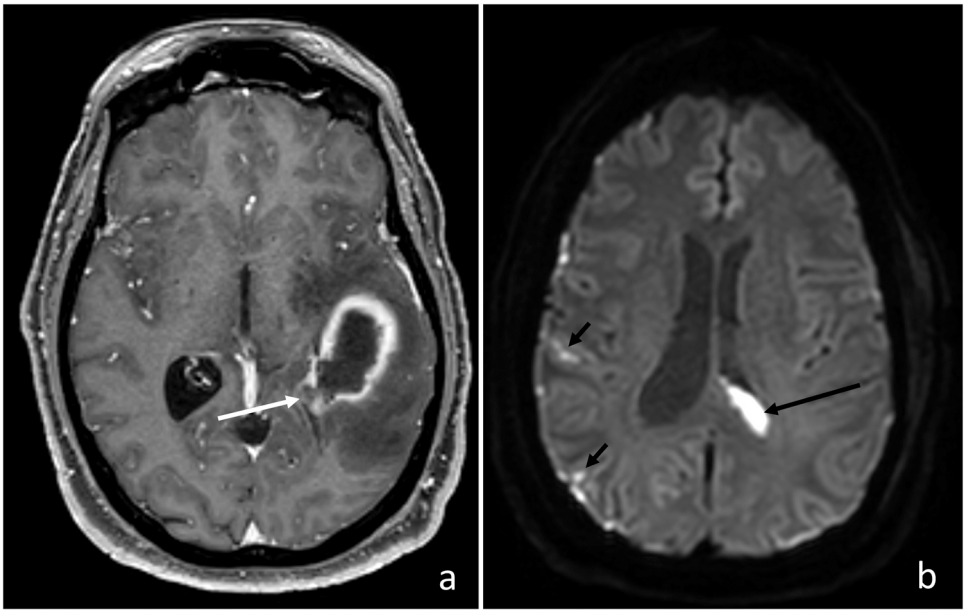

Fig. 2. Axial MRI of the brain. T1-weighted post-contrast image (a) shows discontinuity in the peripheral enhancement of the abscess where the abscess has ruptured into the left lateral ventricle. Diffusion weighted image (b) shows marked restricted diffusion dependent within the left lateral ventricle (long black arrow) compatible with ventriculitis as well as small foci of restricted diffusion in the sulci (short black arrows) compatible with meningitis.

mastoiditis (Fig. 3), presumably the source of the intracranial infection. Neurosurgery, otolaryngology, and infectious disease (ID) were consulted.

On hospital day 1, a stereotactic aspiration of the left temporal intracerebral abscess was performed. Over 40 mL of purulent fluid was removed. The gram stain and culture revealed many polymorphic neutrophils (PMNs) and mixed gram positive and gram negative organisms. Blood cultures were negative. A left auditory canal wall mastoidectomy was performed to drain the mastoiditis and resect the cholesteatoma. Brain abscess cultures grew *Enterococcus faecalis* and *Clostridium ramosum*. Parenteral antimicrobials were changed to ampicillin 2 g IV every 4 h, cefepime 2 g IV every 8 h, and metronidazole 500 mg IV every 6 h.

On hospital day 5 the patient had a head CT and repeat head MRI, which showed recurrent left temporal lobe abscess as well as worsening of meningitis and ventriculitis. On hospital day 6, a mini craniotomy was performed to drain the recurrent abscess and a transcranial drain was left in place within the drained abscess cavity. An external ventricular drain was also placed for intraventricular antimicrobials. Thereafter, the patient was started on adjuvant intraventricular vancomycin (10 mg every 24 h) and gentamicin (5 mg every 24 h) therapy, as well as continued on parenteral ampicillin, cefepime, and metronidazole.

Over the next several days he steadily improved. The patient was extubated on hospital day 9. On hospital day 19, the patient's neurologic status had markedly improved. Intraventricular antimicrobials and his external ventricular drain were discontinued (after approximately 2 weeks of intraventricular therapy). Interval head imaging showed improvement in the size of the abscess. He was continued on parenteral antimicrobials for a total of 8 weeks. He

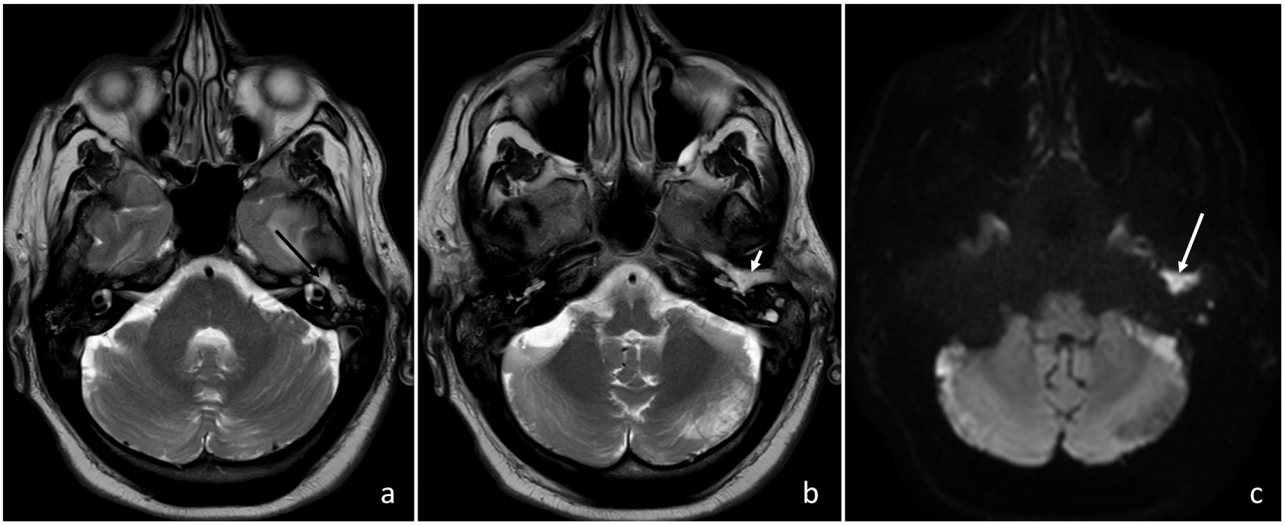

Fig. 3. Axial MRI of the brain. T2 weighted image at the level of the mastoid temporal bone (a) shows fluid in the left mastoid air cells (black arrow) which is seen in the setting of mastoiditis. T2-weigthted image at the level of the middle ear (b) and diffusion weighted image at the same level (c) show fluid/debris in the left middle and external auditory canal (short white arrow) with corresponding restricted diffusion compatible with the surgically proven cholesteatoma.

continued to work with physical therapy and speech language pathology.

Discussion

Brain abscesses arise most commonly from direct spread of bacteria or fungi into the cerebral cortex [2]. Direct spread from contiguous sites may result from otitis media, mastoiditis, or dental infection, and accounts for 25–50% of cases and typically presents with a single abscess [4]. Hematogenous or iatrogenic seeding has also been described, accounting for 20–35% of cases and often presenting with multiple abscesses [2]. The mean age of presentation is 33.6 years with a male-to-female predominance of 2.4:1 [1]. Once a brain abscess occurs, it causes localized edema and acute inflammation that leads to liquefactive necrosis of brain tissue and eventual fibrosis. Much of the damage is caused by the immune inflammatory response rather than by the microorganisms [5].

Only approximately 20% of patients present with the classic triad of brain abscess symptoms including headache, fever, and focal neurological deficits [1]. MRI is the diagnostic test of choice. The classic findings on MRI are a T2 hyperintense, ring-enhancing lesion with central restricted diffusion and surrounding edema [6]. If possible, brain abscesses are drained for definitive diagnosis and culture. Blood cultures should be obtained before initiating antibacterial treatment [2]. Lumbar puncture is not typically required for diagnosis [7].

Brain abscesses are typically caused by *Staphylococcus* or *Streptococcus* species and little published literature exists on *Enterococcus faecalis* brain abscesses, as was seen in our case [2]. This is because *E. faecalis* is infrequently encountered in the central nervous system and is typically found in the context of iatrogenic or nosocomial brain abscesses such as from prior neurosurgery, congenital malformations, or immunodeficiency [8]. Our patient had a history of chronic otitis media and diabetes, which may predispose him to a brain abscess from contiguous spread from acute otomastoiditis. However, neither of these disease states are frequently documented as risk factors for an *E. faecalis* brain abscess. A case report exists of a patient with hereditary hemorrhagic telangiectasia who presented with *E. faecalis* brain abscess following a dental procedure [9], and another of a patient with mitral valve *E. faecalis* endocarditis who developed a brain abscess as a sequela [10]. When susceptible, high-dose parenteral ampicillin is felt to be the treatment of choice.

Most patients with brain abscesses also require surgical intervention [11]. The majority of these abscesses are aspirated both for culture and decompression. Relatively few are excised due to increased risk of neurologic deficits [1]. Surgical excision is typically reserved for multiloculated abscesses, encapsulated fungal abscesses, and traumatic brain abscesses [11]. In our case, the patient's brain abscess continued to increase in size despite intravenous antimicrobials and aspiration, which suggested the need for further surgical intervention.

A feared complication of brain abscesses is rupture into the ventricular system, causing pyogenic ventriculitis, with a mortality rate approaching 80% [12]. Our patient was initiated on intraventricular antimicrobials given the lack of clinical improvement and imaging evidence of worsening ventriculitis and meningitis. The precise role of intraventricular antimicrobials in brain abscesses and ventriculitis has not yet been well defined. [13]. Several case reports exist documenting improved clinical outcomes with the use of intraventricular antimicrobials, but there are no large-scale reviews or randomized prospective data. A case report from 1995 noted the use of intraventricular gentamicin in a case of *Streptococcus intermedius* brain abscess complicated by rupture. The patient survived with minimal deficits [12]. In 2018, a study of 19 patients with gram negative pyogenic ventriculitis treated with intraventricular antimicrobials demonstrated a 74% cure rate with 12 of 14 surviving patients regaining consciousness [14]. Another case report in 2021 described a patient with a ruptured *S. intermedius* brain abscess treated with intraventricular vancomycin as an adjunct to parenteral antimicrobials. The patient's clinical status improved, but he subsequently died several weeks later [15].

The prognosis of patients suffering from brain abscesses is improving. The rate of patients making a full neurologic recovery increased from 33% to 70% between 1970 and 2013 [1]. The most common neurological sequela of brain abscess is seizure, with approximately 32% of 30-day survivors of brain abscess developing new-onset epilepsy according to a 1982–2016 cohort study [16]. Poor prognostic factors include coma, rapid onset of symptoms, and rupture into the ventricle [2]. The development of pyogenic ventriculitis, or pyocephalus, secondary to abscess eruption into the intraventricular space is associated with a particularly poor prognosis [17]. Between the years 1953 and 1995, there were 20 survivors

of intraventricular rupture of brain abscess out of 129 published cases [12].

We present the case of a patient with a large brain abscess and subsequent pyogenic ventriculitis who achieved a return to near-normal neurologic function with a combination of surgical debridement with parenteral and intraventricular antimicrobials. Further research is needed into the precise role of intraventricular antimicrobials.

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CRediT authorship contribution statement

Nina Feinberg: Conceptualization, Patient care, Writing – original draft, Writing – review & editing. **Brendan Campbell**: Conceptualization, Patient care, Writing – original draft, Writing – review & editing. **Michael Bazylewicz**: Conceptualization, Patient care, Writing – original draft, Writing – review & editing. **William D. Brown**: Conceptualization, Writing – original draft, Writing – review & editing. **Devika Singh**: Conceptualization, Patient care, Writing – original draft, Writing – review & editing. **Timothy Whitman**: Conceptualization, Patient care, Writing – original draft, Writing – review & editing. **Wallace Kemper Alston**: Conceptualization, Patient care, Writing – original draft, Writing – review & editing. **Adam Ulano**: Conceptualization, Patient care, Writing – original draft, Writing – review & editing. **Benjamin Sawatzky**: Conceptualization, Patient care, Writing – original draft, Writing – review & editing. **Andrew J. Hale**: Conceptualization, Patient care, Writing – original draft, Writing – review & editing.

Conflict of interest statement

None of the authors report any conflicts of interest.

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Authorship verification

All co-authors have seen and agree with the contents of the manuscript and have contributed significantly to the work.

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