Case report

Thalamic bacterial abscess presenting with hemiparesis and expressive aphasia

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A B S T R A C T

Brain abscesses are relatively rare in the developing world, with an incidence of 2% of all space occupying lesions. Deep-seated abscesses such as thalamic and basal ganglia abscesses are much rarer than abscesses in other locations of the brain, comprising 1.3–6% of all brain abscesses. These abscesses may present with hemiparesis, and subcortical aphasia has only been reported in a few cases throughout the literature. Here we present and discuss a case of thalamic brain abscess caused by S. anginosus that presented with subcortical aphasia.

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Introduction

The Streptococcus anginosus group includes three taxonomically distinct species: Streptococcus anginosus, Streptococcus intermedius and Streptococcus constellatus. These organisms are found as commensal flora of the oropharyngeal, gastrointestinal and urogenital tract, but they can become pathogenic and unlike most other alpha-hemolytic streptococci cause contiguous or metastatic infection after mucosal disruption [1]. Of the three species, S. anginosus shows the greatest propensity for metastatic dissemination.

Brain abscesses are relatively rare in the developing world, with an incidence of 2% of all space occupying lesions [2]. Deep-seated abscesses such as thalamic and basal ganglia abscesses are much rarer than abscesses in other locations of the brain with reported incidences ranging from 1.3 to 6% of all brain abscesses [3–7]. We present and discuss a case of thalamic brain abscess caused by S. anginosus that presented with subcortical aphasia.

Case presentation

A 46 year-old African American man with a history of uncontrolled diabetes mellitus, hypertension, chronic kidney disease, and polysubstance abuse (including alcohol, tobacco, marijuana and cocaine) was brought to the emergency room with confusion and difficulty speaking. On initial examination temperature was 37.6 °C (99.7 °F) and his blood pressure was 154/93 mm Hg. The patient was only oriented to self, and had evidence of expressive (Broca’s) aphasia, with non-fluent speech, and impaired naming and repetition. He was able to follow two-step commands (good comprehension). He also had evidence of dysartria, and right-sided hemiparesis. Laboratory investigations showed leukocytosis of 12.86 × 103/µL without bandemia. Computer tomography showed a 3.1 cm left thalamic mass with 5 mm rightward midline shift. Magnetic resonance imaging confirmed the computer tomography findings (Figs. 1 and 2).

The patient was admitted to the neurological intensive care unit. Empiric intravenous antimicrobial therapy with vancomycin and meropenem was instituted and he underwent a stereotactic aspiration which yielded 15 mL of purulent material. Gram stain of the aspirated fluid revealed Gram positive cocci in chains suggestive of streptococci, and pure culture of the aspirated fluid grew Streptococcus anginosus. The patient had negative blood cultures and a transthoracic echocardiogram resulted negative for valvular vegetations. Computer tomography imaging of chest, abdomen, and pelvis revealed an area of sigmoid colitis. Inpatient endoscopic evaluation to further elucidate the sigmoid lesion was deferred per Gastroenterology team given patient’s altered mentation and active infection. The antibiotic regimen was switched to ceftriaxone 2 g every 12 h for 8 weeks. Unfortunately, the patient left against medical advice after 15 days of intravenous
antimicrobials. He was seen by Neurosurgery as an outpatient for wound assessment one month after presentation. There was no documentation regarding his aphasia though persistence of his hemiparesis was stated.

Discussion

Thalamic abscesses are deep-seated metastatic lesions of hematogenous origin. They may occur from congenital heart disease with right to left shunts, intra-thoracic and/or abdominal sepsis, of local origin from dental caries, otitis media or sinusitis, or be cryptogenic [3]. The most common reported organism isolated from thalamic abscesses are streptococci, particularly the S. anginosus group, and anaerobes [5]. The propensity for abscess formation by S. anginosus may be explained by production of toxins with leukocidin-like activity and thrombin-like activity, allowing these microorganisms to resist phagocytosis [1]. S. anginosus can cause brain abscesses by two different mechanisms: by contiguous spread from a juxta-cranial site of infection, such as sinusitis, otitis media, mastoiditis, meningitis, orbital cellulitis, and odontogenic infections; and via hematogenous spread from a remote site such gastrointestinal or respiratory tract [1]. S. anginosus can be responsible for pyogenic invasive infections with abscess formation, but are a very infrequent cause of infective endocarditis compared to other streptococci.[1]

Important risk factors for thalamic abscesses due to S. anginosus include liver cirrhosis, malignancy, diabetes mellitus, malnourishment and immunosuppression [1,5,7]. Periodontal disease is also an independent risk factor for brain abscesses, including thalamic abscesses [8,9]. Most patients present with fever, leukocytosis, as well as headache, altered sensorium, and hemiparesis [3]. Chang et al. collected data from CSF, blood, pus, and cultures of 8 adult patients with deep-seated bacterial brain abscess over a period of 14 years. On analysis, 6/8 were male, mean age was 61 years, and of the clinical presentations, hemiparesis was the most common (6/8). Simone et al. analyzed the features of the only 11 well documented cases of disseminated S. anginosus group infections found in PubMed at that time, and their own case report. They demonstrated that 83% patients were male, mean age was 47.58 years (SD +/- 20.92 years), and 42% of patients had dental infections, malignancy, gastrointestinal or respiratory diseases as underlying comorbidities. No cases of endocarditis were encountered.

Our patient presented with acute encephalopathy, contralateral hemiparesis and expressive aphasia. Of note, there are only a few cases in the literature where deep-seated brain abscesses caused aphasia. A PubMed search, current as to the date this case report was written, of “thalamic abscess AND aphasia” yielded 5 cases only, and our case could be an addition to these; however, one of the cases was a thalamic abscess after hypertensive thalamic hemorrhage not the result of contiguous spread or disseminated pyogenic disease. Of note, another search was done for “basal ganglia abscess AND aphasia”. The latter yielded 3 cases, for a total of 8 reported cases of deep-seated brain abscesses causing aphasia. There are two theories that attempt to explain these cortical disturbances. Olsen et al. describe an ischemic penumbra or an area of hypoperfusion adjacent to cerebral infarct where blood flow to this area is sufficient for tissue viability but not enough for normal function. Another potential explanation is the cortical diaschisis theory, where cortical neuronal activity is decreased due to loss of input from thalamocortical projections.[9]

For diagnosis, computer tomography with contrast has become the mainstay of rapid diagnosis of brain abscesses, allowing early detection [6]. This is essential, as the differential diagnosis is broad, including fungal, nocardial, or tubercular abscesses, neurocysticercosis, toxoplasmosis, glioblastoma, metastasis, infarction, resolving hematoma, lymphoma or radionecrosis [3,4,6]. Deep-seated abscesses are complex due to difficult access and are associated with an increased risk of intraventricular rupture and antibiotic resistance [3]. Treatment options include stereotactic aspiration with or without continuous drainage, free hand
aspiration with burr hole, stereo-endoscopic aspiration, ultrasound guided aspiration, surgical transventricular approach, and pharmacological management alone [1,3–7] Controversy exists as to which of these is the best surgical intervention. Nonetheless, stereotactic aspiration remains the preferred option for many neurosurgeons as it drains the abscess, reduces mass effect, carries less risk of intraventricular rupture and confirms the diagnosis [3–5]. Medical therapy alone should be reserved for patients with multiple and/or small abscesses, specifically those smaller than 2 cm or 2.5 cm in diameter, or when patients are at high risk for surgical complications [3]. In the literature, length of antimicrobial therapy varies from 4 to 8 weeks pending resolution of abscesses as evidenced by repeat imaging [1,7,8].

Eradication of the primary focus is paramount in determining the clinical outcome [6]. Callovini et al. reviewed 18 cases, and in 10 of these (55.5%) the origin of the infection was not identified. Inability to define a primary focus risks further abscess formation and systemic morbidity [5]. The mortality rate from a thalamic abscess is 9–14%, rising to 80% with intraventricular rupture, and survivors at a risk of permanent neurological damage [4,7]. If intraventricular rupture occurs, aggressive ventricular drainage in the very early stages and direct intraventricular administration of antibiotics are required to attain a favorable outcome.

**Conclusion**

Deep-seated abscesses such as thalamic and basal ganglia abscesses are much rarer than abscesses in other locations of the brain. Hemiparesis is a common presentation of this abscesses but subcortical aphasia as a result of a deep-seated abscesses has only been reported in a few cases throughout the literature. Thalamic abscesses carry a high morbidity and mortality, and rapid diagnosis via computer tomography or magnetic resonance imaging, drainage, and eradication of the primary focus are crucial in determining clinical outcome. Despite the controversy as to which neurosurgical approach is best for abscess drainage, many authors support stereotactic aspiration followed by culture directed antibacterial therapy for at least 6 weeks. If intraventricular rupture occurs direct antimicrobial instillation is recommended. Follow up imaging is required to define the therapeutic end point and to assure resolution of disease.

**Contributorship**

The idea for reporting this case was that of Jarelys M. Hernandez Jimenez, MD. Further intellectual content and editing was done by all authors. Jarelys M. Hernandez Jimenez, MD assumes responsibility for the integrity of the contents herein.

**Patient consent**

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

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**Conflict of interest**

The authors have no financial or personal conflicts of interest to disclose.

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