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Occipital Lobe Abscess Causing Homonymous Hemianopsia After a Dental Procedure

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Occipital Lobe Abscess Causing Homonymous Hemianopsia After a Dental Procedure

Abstract

Background: A homonymous hemianopsia visual field defect is defined as vision loss on the same side of the vertical midline in both eyes. Although the vast majority are caused by an ischemic stroke, other neurologic etiologies must be considered and ruled out. This case highlights the importance of a rapid and thorough investigation of a patient's symptoms using both clinical examination and imaging to reduce the risk of permanent complications and potential fatality.

Case Report: A 67-year-old Caucasian male presented to the eye clinic with sudden onset reduced peripheral vision to his right side in both eyes. Entering visual acuities were 20/40 OD and 20/60 OS. Humphrey visual field (HVF) testing confirmed a complete right homonymous hemianopsia. Magnetic resonance imaging (MRI) revealed a large, left occipital lobe intracranial mass. Neurosurgical intervention was necessary to confirm the diagnosis of a brain abscess, thought to be related to a prior dental procedure with tooth extraction.

Conclusion: A brain abscess is a focal area of necrosis within the brain parenchyma that typically results from an infectious process. Though rare, the condition has potentially devastating neurologic complications which can be mitigated with early detection and treatment. In this case, the abscess caused a right homonymous hemianopsia field defect, which prompted his urgent request for an appointment with the eye clinic.

Keywords

Homonymous hemianopia, brain abscess, craniotomy, magnetic resonance spectroscopy

INTRODUCTION

A brain abscess is a focal area of necrosis within the brain parenchyma that typically results from an infectious process. Potentially, 4% to 7% of all pyogenic brain abscesses are caused by a dental source[1] with advancements in antimicrobial therapy and imaging capabilities, the diagnosis and treatment of a brain abscess has significantly improved. The purpose of this retrospective case review is to remind the reader of the brain abscess diagnosis when considering potential causes of homonymous hemianopic visual field defects in patients. The overall outcome and quality of life in patients after brain abscess resolution is specific to each case and dependent on time to diagnosis and treatment. Complications often include seizures, loss of mental acuity, and focal neurological defects.

CASE REPORT

A 67-year- old Caucasian male presented to the eye clinic with a symptomatic history of reduced peripheral vision to his right side OU, first noticed upon awakening two weeks prior to his visit and stable since its onset.

The patient's medical history was positive for hypertension**,** viral hepatitis C, liver cirrhosis, hyperlipidemia, and tobacco abuse. The patient's ocular history included a corneal scar OS, nuclear sclerotic cataracts OU, epiretinal membrane OS and glaucoma suspicion OU. His ocular family history was positive for macular degeneration. He had no known drug allergies. His current medications included atorvastatin and chlorthalidone.

The patient was oriented to person, place, and time with a normal affect. Entering Snellen acuity was 20/40 OD and 20/60 OS, improvement to 20/25 OD and 20/40 OS with potential acuity. Pupils were equal, round, and reactive to light, without a relative afferent pupillary defect. Confrontation visual fields demonstrated constriction inferior temporal OD and inferior nasal OS. Extraocular motility was full in all gazes OU. Slit lamp exam was remarkable for a papilloma on his right upper eyelid and grade 2 nuclear sclerotic cataract OU. Intraocular pressures with Goldmann applanation tonometry were 19 mm Hg OD, OS. Manual blood pressure was 164/82 in office. Dilated fundus exam revealed asymmetric cup to disc ratios of 0.40 OD and 0.60 OS, and a diffuse epiretinal membrane OS, all of which were stable from previous exams. HVF 30-2 threshold test was performed, and results demonstrated a right homonymous hemianopsia (Fig. 1). A brief neurological exam revealed CN II-XI grossly intact. On review of systems, the patient denied all other systemic symptoms including malaise, dizziness, confusion, weakness, numbness, tingling, facial droop, easy bruising, headache, night sweats, fevers, chills, or recent

unintentional weight loss. His wife stated she had noticed the patient experiencing an increasing clumsiness with occasional stumbling the previous few weeks.

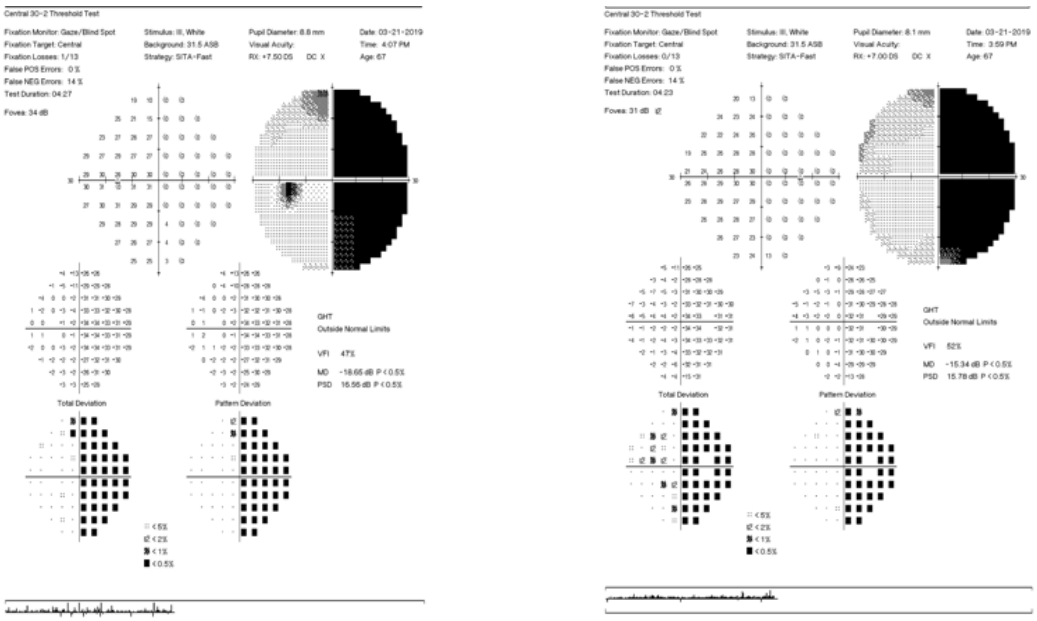

Figure 1: HVF 30-2 threshold test, right homonymous hemianopsia on initial exam.

The patient was escorted to the emergency department the same day and a computed tomography (CT) of the head without contrast was completed. A focal, round area of hypoattenuation in the left parieto-occipital region measuring 1.9 cm x 1.6 cm in size was found on the initial CT (Fig. 2). No definite acute intracranial hemorrhage was present. Given the focal appearance of the finding in the left parieto-occipital lobe, an MRI with and without contrast was recommended for evaluation of the underlying intracranial mass.

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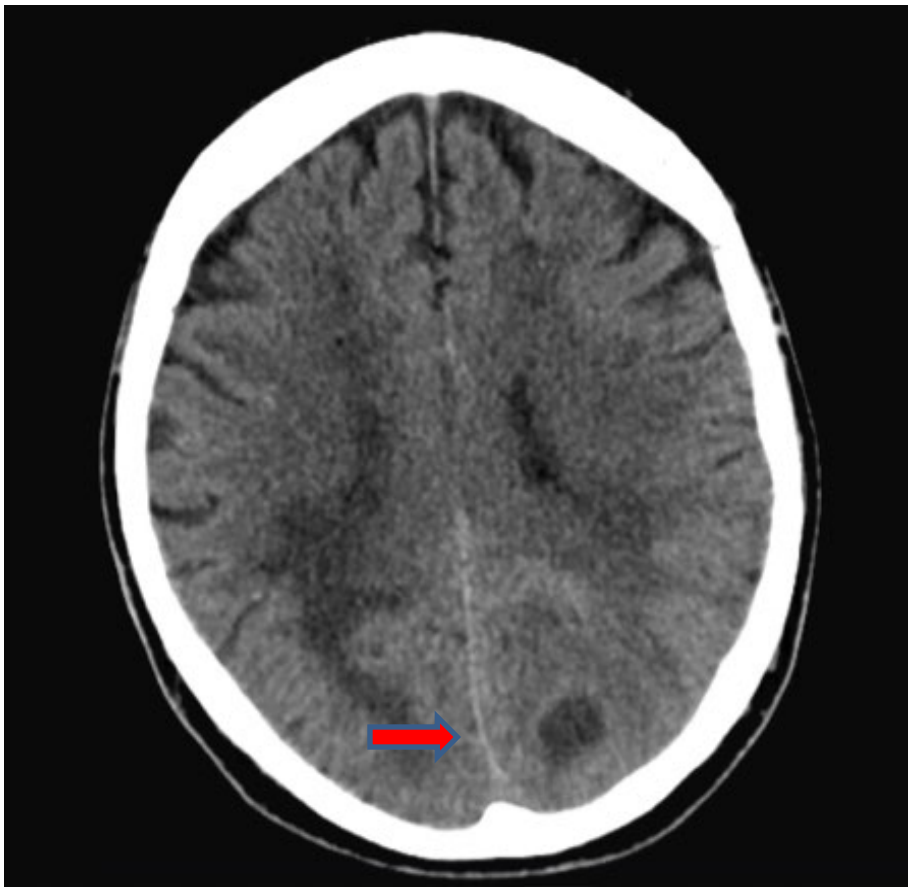

Figure 2: A focal, round area of hypoattenuation in the left parieto-occipital region measuring 1.9 x 1.6 cm in size on CT of the head without contrast.

The following day, an MRI with and without contrast was performed. A well-defined, round area measuring 2 cm x 3 cm in the left occipital lobe was discovered (Fig. 3). Associated surrounding edema with slight mass effect upon the adjacent brain parenchyma was present. The interpretation by radiology indicated a necrotic neoplasm.

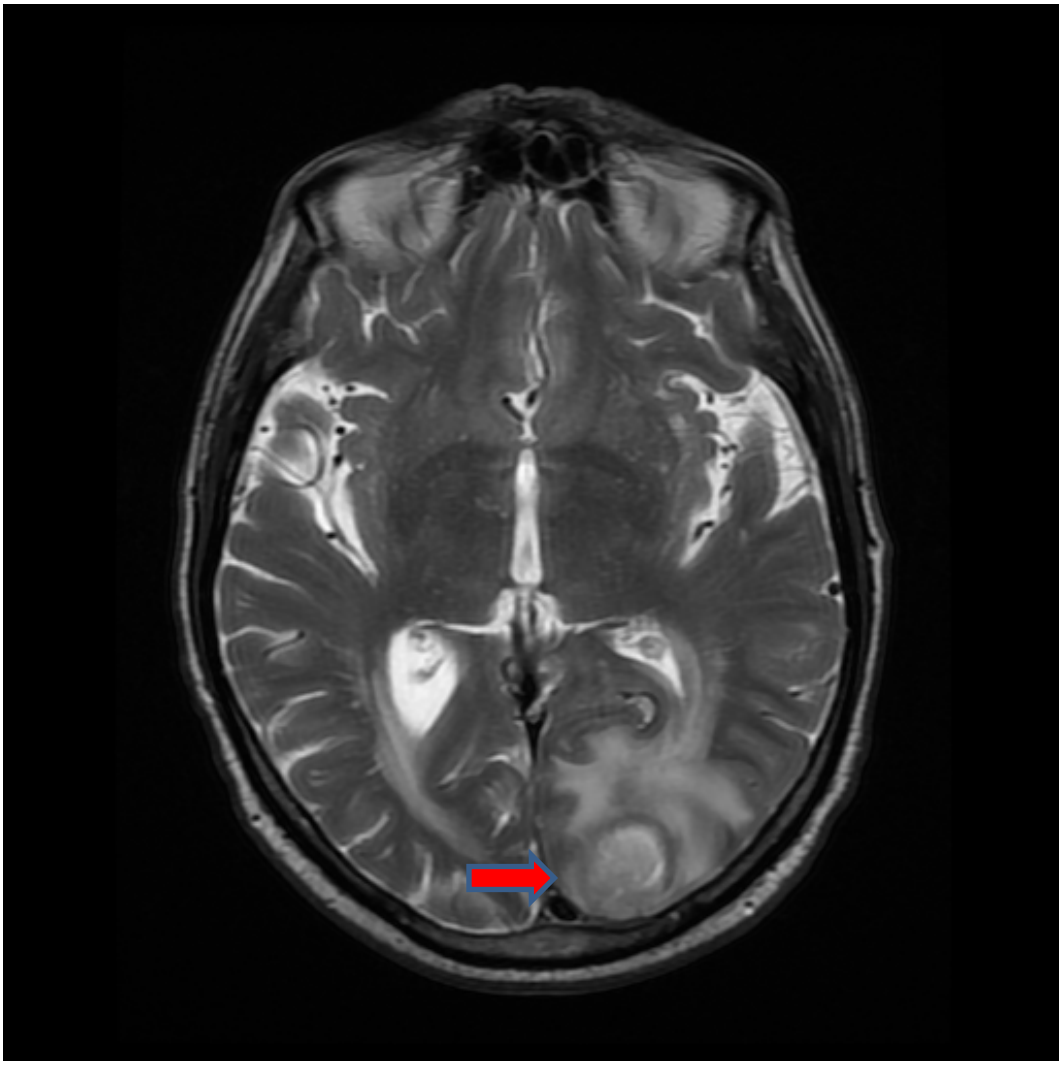

Figure 3: A well-defined, rounded area measuring 2cm x 3 cm in the left occipital lobe seen on MRI.

The patient was transferred to a tertiary care facility with neurosurgical capabilities. CT with contrast of abdomen, pelvis, and chest were performed to rule out metastatic disease. Three days later a repeat CT of the head without contrast and magnetic resonance spectroscopy (MRS) were completed. The CT confirmed a rounded focal hypodense lesion in the left parieto-occipital region measuring 2.2 cm x 2.3 cm x 2.5 cm with adjacent white matter edema and no mid-line shift. The MRS identified a necrotic area consistent with both tumor and abscess. The lesion exhibited characteristics of both an abscess due to the extremely bright diffusion

signal and a tumor given the thickness and irregularity of the wall. Due to the suspicion for abscess, broad spectrum IV antibiotics were immediately initiated: ceftriaxone, vancomycin, and metronidazole.

Eight days after the MRS, the patient's condition deteriorated, and he began to suffer from seizures and mild cognitive impairment. Further investigation into the patient's medical history revealed a recent dental procedure with tooth extraction a few weeks prior to his symptomatic visual field loss. A repeat MRI was performed and indicated significant enlargement of the mass, which now measured 6.7 cm x 2.9 cm x 4.0 cm. There was moderate to severe mass effect and a 1.1 cm left to right midline shift. Given these findings, the decision was made for neurosurgical intervention.

The next day the patient had a stealth-guided 6 cm x 6 cm left occipital craniotomy performed for drainage versus resection of the occipital mass. The neurosurgeon visualized edematous brain tissue. A 1 cm corticectomy was performed and immediately a foul smelling, purulent material was expressed and a minimum of 30 ml of the material was drained. A yellow exudate and some of the abscess wall were removed with gentle dissection. The purulent material, exudate and portion of the abscess wall were sent for stat Gram stain, aerobic and anaerobic cultures, mycobacterium, and fungal cultures.

The diagnosis following his craniotomy was confirmed to be a large, left occipital abscess. Gram stain showed rare gram-positive cocci and gram-negative bacilli, in addition to cultures with late growth of microaerophilic strep species. The patient was to continue IV ceftriaxone, vancomycin, and metronidazole for 6 weeks and begin Keppra for seizure prophylaxis for 10 days with a scheduled repeat MRI in two weeks to determine if further debridement was needed.

Twenty-eight days after admission to the tertiary care facility, no further debridement of the lesion was required, and the patient was discharged to begin inpatient rehabilitation.

The patient returned to the eye clinic sixteen days after the stealth-guided left occipital craniotomy. Entering Snellen acuity was 20/30 OD and 20/50 OS, with potential acuity improvement to 20/20 OD and 20/30 OS. Pupils were equal, round and reactive to light, without a relative afferent pupillary defect. Confrontation visual fields continued to show a constriction inferior temporal OD and inferior nasal OS. Extraocular motility was full in all gazes OU. Intraocular pressure with Goldmann applanation tonometry was 14 mm Hg OD and 15 mm Hg OS. Dilated fundus exam was stable. A repeat Humphrey visual field 30-2 was performed and

results demonstrated a residual incomplete right homonymous hemianopsia (Fig. 4), which indicated superior visual field improvement, right eye greater than left eye. Based on serial MRI results, the improved visual field loss was most likely attributed to the resolution of parenchymal edema and abscess which was more localized to the occipital-parietal lobe versus the temporal lobe.

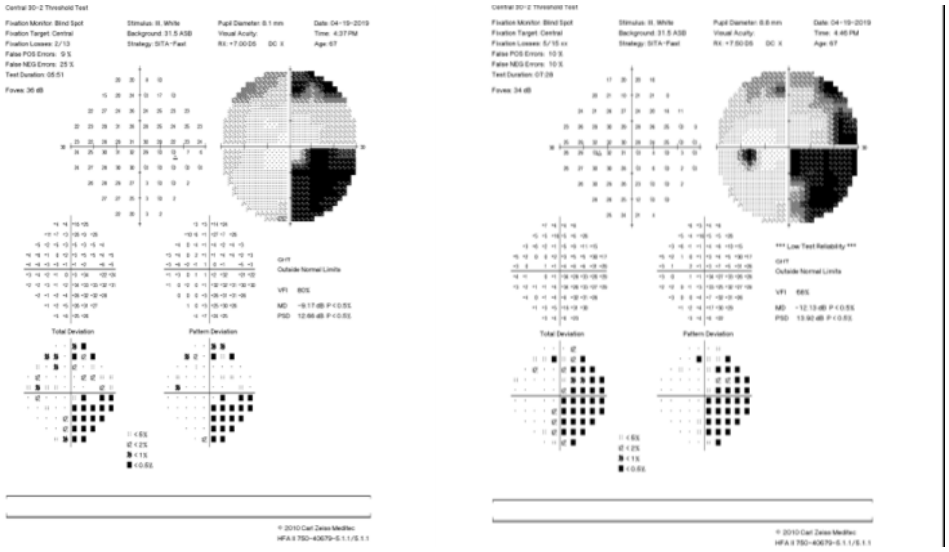

Figure 4: HVF 30-2 threshold test with improved right homonymous hemianopsia sixteen days after stealth-guided left occipital craniotomy.

Repeat MRI with and without contrast was performed 1, 4 and 10 months after the open craniotomy. Signal abnormality and soft tissue changes remain stable in the occipital region 10 months after surgery (Fig 5). There has been no evidence of brain abscess recurrence or abnormal contrast enhancement.

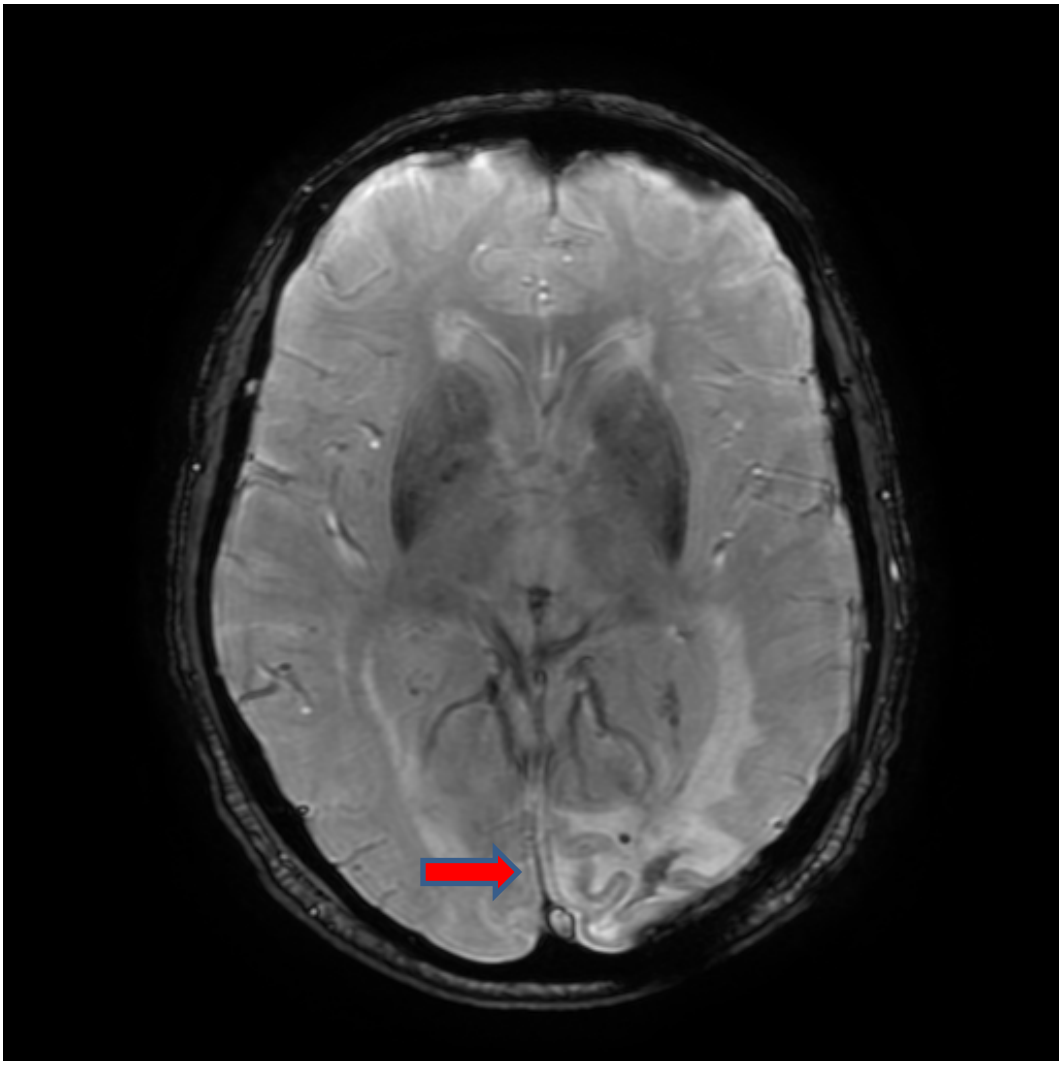

Figure 5: 10 months post craniotomy MRI.

DIFFERENTIAL DIAGNOSIS

Homonymous hemianopsia results in a visual field defect in the two right or the two left halves of a patient's visual field and is caused by a unilateral lesion(s) of the visual pathway posterior to the optic chiasm[2] which includes lesions located in the optic tract, lateral geniculate nucleus, temporal lobe, parietal lobe, and occipital lobe. Differential diagnosis is aided by the characteristic of the field defect (type, form, size, and congruity), the patient's neurological signs and symptoms[2] onset of visual symptoms, expected clinical course and imaging results. Differential

diagnoses considered in this case included cerebral infarct, intracranial hemorrhage, intracranial mass, infection, and central nervous system disease.

In this case, based on initial clinical exam and presentation, the likely cause of the visual field defect was a cerebral infarct due to the sudden onset of peripheral vision defects. His risk factors included elevated blood pressure at his eye exam, as well as a ½ pack per day smoker with a 40+ pack year history of tobacco use. However, our patient denied having stroke related symptoms, including sudden numbness or weakness in the face, arm, or leg especially on one side of the body, sudden confusion, dizziness, or severe headache. The only associated symptom reported by his wife, was increased clumsiness with occasional stumbling the previous few weeks which did not allow any potential differential diagnoses to be ruled out. The HVF result localized the source of the defect to the occipital lobe. The etiology remained elusive as all potential differentials have similar symptoms, therefore imaging was indicated to further identify the underlying lesion. Only after the visualization of the brain lesion with imaging, was the diagnosis more apparent and defined. As additional neurologic symptoms manifested a gradual onset of intensity, further diagnostic techniques were necessary to confirm the diagnosis.

DISCUSSION

A brain abscess is a suppurative infection within the brain parenchyma[3] that is caused by bacteria, mycobacteria, fungi, or parasites.[4] Brain abscess is uncommon with an incidence of 0.4 to 0.9 cases per 100,000 population[4] Intracranial abscesses are a serious and life-threatening infection that are most common in males and occur more frequently in the first four decades of life.[5]

The abscess infection enters the brain through direct extension, trauma, cryptogenic or hematogenous spread from a distant focus. The most common entrance is through direct extension, which occurs in 45-50% of cases, and is commonly associated with otitis media, mastoiditis, sinusitis and congenital heart disease.[5,6] Hematogenous spread occurs in approximately 25% of cases, often leads to multiple brain abscesses, and includes etiologies related to dental extractions and manipulations.[6] Traumatic spread of the infection, usually from a penetrating head injury or post-neurosurgery, are the least common and occur in 10% of all cases.[5,6] Finally, approximately 15% are cryptogenic with no recognized focus.[6]

Dental sources make up 4-7% of all pyogenic brain abscesses.[1] A dental procedure is the most likely underlying etiology in our patient case. The time between his dental care and the onset of symptoms, approximately two to four weeks,

corresponded to the amount of time required for the development of a brain abscess.[1] In addition, the organisms cultured from his brain abscess are commonly found in the oral cavity.

Brain abscess development consists of cerebritis lasting 1-10 days and capsule formation within 11 to 14+ days.[7] Mass effect or necrosis eventually leads to focal neurological signs.[1] These neurological signs depend on the location of the abscess and can often be subtle. As the abscess enlarges, the neurological signs become more evident.

Approximately 2 out of 3 patients will experience symptoms for 2 weeks or less.[5,8] Headache, mental status changes, or focal neurological deficits occur in 65-70% of patients; the triad of these occurring together is present in less than 50% of patients.[5] An intense headache, located on the ipsilateral side of the abscess, is the most common clinical manifestation of a brain abscess.[4,5] Other common symptoms are fever, seizures, nausea, vomiting and nuchal rigidity.[5]

The development of acute symptoms prompts a person to seek medical treatment, leading to the need for expedited cranial imaging. Low-attenuation abnormalities with mass effect are seen with a non-contrast CT.[7] With the addition of contrast, the brain abscess will have a hypodense center with a peripheral uniform enhancement ring.[5] CT with contrast can quickly identify the size, number, and location of abscesses.[4,5]

MRI is more sensitive than CT and can better recognize cerebritis and cerebral edema. With contrast, the MRI can better distinguish the abscess from the enhancement ring and surrounding cerebral edema. Despite advancements in radiographic technology, differentiating intracranial lesions is difficult. Advanced forms of MRI imaging are sometimes used as an adjunct to a conventional MRI. A diffusion-weighted MRI is very good at differentiating an abscess from a necrotic tumor, with a sensitivity and specificity of over 90%.[7] MRS has also been shown to differentiate a brain abscess from necrotic tumors or other cystic lesions.[9] MRS is a non-invasive procedure that determines the concentrations of brain metabolites such as N-acetyl aspartate, choline, creatine and lactate in the tissue being examined, a specific pattern of metabolites can be seen with an untreated bacterial brain abscess.[9]

Once an abscess is suspected, immediate treatment with antimicrobial agents is recommended. If antimicrobial therapy is delayed a poor outcome may result.[4] In this case, the patient began treatment with IV ceftriaxone, vancomycin, and metronidazole on the same day the brain abscess was suspected. The length of time

between the start of symptoms and diagnosis is critical.[6] Early antimicrobial therapy during the cerebritis stage may prevent abscess formation.[6] If medical therapy is started within one week from the start of symptoms a favorable response is more likely.[6]

Antimicrobial therapy is initially targeted to the most likely cause of the abscess. The microbial flora of the brain abscess is often polymicrobial.[4,5] Sinus and dental infections are associated with aerobic and anaerobic streptococci, anaerobic gram-negative bacilli, and microaerophilic streptococci.[5] These pathogens are empirically treated with third-generation cephalosporin combined with metronidazole to cover anaerobes.[4,6] Staphylococcal infection is treated by the addition of vancomycin.[4] Intracranial abscess formation may lead to serious disability or even death if misdiagnosed or managed improperly.

The treatment depends on the location and size of the abscess. Small abscess (usually classified as less than 2.5 cm) and cerebritis may respond to antimicrobial treatment alone. However, larger abscesses require surgical options, aspiration, or complete excision of the abscess. Aspiration can rapidly relieve intracranial pressure, confirm the diagnosis of an abscess, and obtain a sample to identify the causative organism(s).[3] Stereotactic aspiration, using CT-guided stereotaxy, can be performed multiple times and is used often in deep-seated abscesses.[3] Complete removal of the purulent material and the capsule is done with an open craniotomy, reducing the need for additional treatment and antibiotic therapy.[3]

Microbiologic evaluation of the purulent material from the abscess includes Gram staining and aerobic and anaerobic cultures.[4] Antibiotic selection is geared towards the organism(s) cultured. The length of IV antimicrobial therapy in patients with bacterial brain abscess is usually 6 to 8 weeks and repeat imaging after surgery should immediately be performed with any signs of clinical deterioration.[4]

Seizures, loss of mental acuity, visual defects, hemiparesis, cranial nerve palsy, and hydrocephalus are among the long-term effects after brain abscess resolution.[5, 7] Seizures occur in almost 70% of patients following a brain abscess.[7] Neurological damage varies from 20-79% and directly correlates to how quickly the diagnosis is made and how soon the antibiotics are initiated.[6]

It is critical to remember that visual field loss can have a profound impact on activities of daily living. Often a referral to a low vision specialist and rehabilitation support team is warranted to provide management of symptoms such as reading difficulty, mobility and restricted or loss of driving privileges. Vision rehabilitation can be an asset to helping the patient cope with their vision loss. Computerized

saccadic training programs have been reported to increase reading duration and decrease reading and scanning errors.[2] Also, the use of hemianopic mirrors or prisms to project images from the blind hemifield into the intact hemifield can greatly help with mobility, though are not used to reinstate driving privileges.[2]

CONCLUSION

Brain abscesses account for only 1-2% of all intracranial masses in westernized countries and can be very difficult to diagnoses because of vague, non-pathognomonic symptoms.[8] The expected clinical course of a slow onset and delayed symptoms in this potentially fatal condition highlights the need for vigilance and expedited care once symptomatic. Thorough patient examination, case history, prompt referral for imaging, and early detection are critical to reducing morbidity and mortality for these patients. Once treated, residual neurological deficits often vary from mild to severe and it is imperative to refer to the appropriate specialist for rehabilitation to best manage the patient's activities of daily living.

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