Do brain abscesses have a higher incidence of odontogenic origin than previously thought?

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Objective. The aim of this study was to answer the following clinical question: "In patients admitted to a tertiary care hospital with a diagnosis of brain abscess, how common is odontogenic etiology?"

Study Design. We designed a retrospective study of patients with brain abscesses diagnosed at the Massachusetts General Hospital between 1980 and 2017. Inclusion criteria were complete medical records outlining clinical course, relevant dental history, and radiographic and microbiologic data.

Results. Of 167 intracranial abscesses, 88 (52.7%) originated from a head/neck source, and 12 (13.6%) were of odontogenic etiology. Dental radiographs in 7 cases showed active dental infection. The remaining 5 patients reported recent dental procedures. Frontal lobe localization was the most common (7 of 12 [58.3%]). Presenting signs included headache (66.7%), mental status changes (41.6%), visual deficits (41.6%), and speech difficulties (33.3%). Computed tomography (CT) or magnetic resonance imaging (MRI) confirmed all diagnoses. Drainage via open craniotomy was performed in 6 (50%) of 12 patients, and stereotactic CT-guided drainage in 4 (33.3%). The most common pathogens were *Streptococcus milleri* (45.5%), *Staphylococcus* species (27.3%), and *Fusobacterium* (27.3%). All cases had favorable outcomes. Five had residual neurologic deficits, 4 had persistent visual complaints, and a recurrent abscess developed in 1 case.

Conclusions. These findings showed a higher subset (13.6%) of brain abscesses that could be attributed to odontogenic etiology than previously reported in the literature and highlight the need to rule out dental sources in cryptogenic cases. (Oral Surg Oral Med Oral Pathol Oral Radiol 2020;130:10−17)

Although rare, brain abscesses are associated with significant morbidity and mortality. Overall, the incidence has dramatically decreased over the last 50 years. Between 1935 and 1981, approximately 2.7 to 3.9 per 100,000 people were diagnosed with brain abscesses, whereas more recent findings in 2015 have suggested that the rate has fallen to 0.3 to 0.4 per 100,000.[1] Approximately 1500 to 2500 cases are reported annually in the United States, and, with the advent of advanced imaging and broad-spectrum antibiotics, mortality rates have declined to less than 15% over the last few decades.[2,3]

Although multiple etiologies have been cited, the most frequent ones include ENT (ear, nose. and throat) infections (sinusitis and otitis) as well as invasive neurologic procedures. Less frequent etiologies include dental infection, cardiac anomalies and associated endocarditis, and pulmonary infections.[4] A few case reports of brain abscesses have been attributed to an odontogenic origin, and in most of those cases, the incidence has been reported to be less than 5%.[5] Over the last 2 decades, however, an increasing proportion has been attributed to odontogenic sources.[4] The reasons for this are speculative and include heightened awareness of the predilection of oral infections to spread in this manner, maintenance of a greater number of retained teeth in older populations, and the increasing number of foreign bodies being implanted in the oral and maxillofacial area (dental and zygomatic implants; hardware, including plates and screws, etc.). Microbial resistance patterns to multiple antibiotics may also play a role.

Approximately 30% of brain abscesses have an unclear etiology.[6-9] Determination of the definitive source can be problematic, but variables, such as microbiology, may be suggestive of odontogenic infections. Ruling out the more common etiologies, such as sinus infection or otitis, and identifying a definitive dental pathology are imperative. To prove that an abscess has truly developed from an odontogenic source, 3 criteria should be met: (1) No alternative source of bacteremia is found; (2) the bacteria responsible for the abscess are typically found in the oral microflora; and (3) clinical signs of active dental disease are present.[10] Given these conditions, validating an odontogenic source can be dif-

Statement of Clinical Relevance

Odontogenic sources of brain abscesses are more common than previously reported and may account for "cryptogenic" sources. Considering this potential source of infection will help with timely diagnosis and initiation of therapy that may include treatment of active dental disease.

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ficult. A few studies have shown preliminary associations among the microbiology of the infection, the localization of the abscess, and the routes of oral infection spread.[10] However, better documentation is needed.

The purpose of this study was to answer the following clinical question: "Among all patients admitted to a tertiary care hospital with a diagnosis of brain abscess, how many appear to be of odontogenic origin?" The secondary aims were better understanding of the clinical course of the infection and treatments received by patients and addressing the following questions: (1) "Are the organisms cultured similar to those represented in cultures from odontogenic infections, and are resistance patterns to antibiotics similar?" (2) "What treatment and outcome information is available?" We hypothesized that the proportion of brain abscesses of odontogenic etiology is higher than previously reported in the literature and needs to be taken into account in all cases without a clear etiology.

MATERIALS AND METHODS

Study design and study sample

To answer our clinical question, we designed a retrospective, single-center, cohort study. The study population consisted of patients diagnosed with brain abscesses at the Massachusetts General Hospital (MGH, Boston, MA) between 1980 and 2017. Patients were identified by using the appropriate International Classification of Diseases, 9th revision (ICD-9) and ICD-10 coding for brain abscesses in the MGH Research Patient Database Registry. Inclusion criteria were diagnosis of brain abscess and availability of complete clinical, radiographic, and microbiologic data. Incomplete records resulted in exclusion of the case. Institutional review board (IRB) approval was obtained (Protocol 2018 P-000165), and the study followed the tenets of the Helsinki Declaration.

A total of 167 patients were identified. Of these, in 88 cases, abscesses were determined to arise from a head and neck primary source of infection. As mentioned above, the following criteria were then used to determine the odontogenic etiology: (1) no alternative source of bacteremia identified; (2) culture data consistent with oral microflora; and (3) clinical signs of active dental disease or recent dental treatment of active disease.

Study variables

The study variables used in this investigation are listed in Table I. These included demographic data, medical history, presenting signs and symptoms, vital signs and laboratory findings at presentation, imaging information, culture data, and treatment specifics. The primary outcome variable was the diagnosis of a brain abscess of odontogenic origin.

Table I. Data intake form

Demographic characteristics	Age
	Gender
Health history	Active medical problems
	Medications
	Allergies
	Past medical problems
	Surgical history
	Trauma history
	Habits—cigarettes, alcohol, drug use
	Dental history, if recorded
Dental history	History of prior dental procedures
	Swelling or pain
Presenting signs and symptoms	Headache
	Seizures
	Visual impairment
	Ataxia
	Neurosensory changes (paresthesia, hemiplegia)
	Confusion
	Dizziness
	Speech impairment
	Memory/cognitive impairment
Vital signs at time of presentation	Blood pressure
	Respiratory rate
	Temperature
	Heart rate
Laboratory values at time of presentation	Complete blood count (CBC)
	Inflammatory markers
	Other
Imaging	Computed tomography (CT)
	Magnetic resonance imaging (MRI)
	Panoramic radiography
	Periapical radiography
Suspected mechanism of infection	Hematogenous spread
	Contiguous spread
	Lymphatic spread
	Foreign bodies
Microorganisms involved in infection	Gram staining of organisms
	Antibiotic-resistant strains
	Aerobic versus anaerobic
Treatment	Surgical, medical, and supportive care
	Pertinent follow-up instructions

Data collection and analysis

Medical records were assessed for our study variables, as described above. Data were subsequently extracted and compiled into an electronic data extraction document. Descriptive statistical data (mean, frequency, range, and standard deviation) were computed, using Microsoft Excel, version 15.26, for each study variable, when appropriate.

RESULTS

Demographic characteristics and medical history

The study sample consisted of 12 patients identified as having a brain abscess of odontogenic origin. Mean age was 47.8 years (range 25−66 years), and 50% were females. Of these 12 patients, 5 were immunocompromised (4 had diabetes and 1 had hepatitis C). Five patients reported excessive alcohol consumption, and 5 were active smokers. Psychiatric diagnoses were reported in 3 cases.

Presenting signs and symptoms

Symptoms were present, on average, 1 week before admission, although 2 patients reported headache and mental status changes of 2 to 3 weeks' duration before presentation. Of the 12 cases, headache (8 cases) was the most common symptom reported, followed by altered mental status (5 cases), ocular changes (5 cases), flu-like symptoms (4 cases), speech deficits (4 cases), and facial pain (4 cases). Specifics are summarized in Table II.

Dental history

Five of the 12 patients reported a recent dental visit (within 2 months of presentation). Procedures included deep scaling, dental restorations, extractions, and implant placement. The remaining 7 patients had active dental infection, most commonly a periapical abscess with associated radiolucency noted on dental radiographs. Molar teeth in both the maxilla and the mandible were the most common sites of origin. One maxillary canine tooth was also found to be etiologic. Details are summarized in Table III.

Laboratory tests and radiologic examinations

Elevated temperature and white blood cell (WBC) counts were the most common abnormalities found. Average temperature at presentation was 99.24 ± 1.99° F, and average WBC count was 12.4 k ± 4.02 k. For 11 of the 12 cases, WBC differentials were available. The average polymorphonuclear count was 79.1%. Erythrocyte sedimentation rate (ESR) and cross-reactive protein (CRP) determination were not uniformly obtained. Seven patients had an elevated ESR (average 68; range 25−127). CRP was documented for 4 patients and averaged 78.4 (range 51.6−142.7). These elevated values are consistent with active infection and associated inflammation.

Computed tomography (CT) or magnetic resonance imaging (MRI) was used to confirm the diagnosis. For 7 cases, the results of both imaging studies were available. Seven of 12 patients had abscesses localized to the frontal lobe. The parietal and temporal lobes were each involved in 3 patients, and the occipital lobe in 2. In 4 patients, the abscess involved more than 1 lobe. One patient had generalized and widespread involvement. Four patients had panoramic imaging performed, and 3 had periapical dental radiographs identifying the offending teeth that were thought to be etiologic. Examples of CT and MRI images are shown in Figures 1 and 2.

Table II. Presenting signs and symptoms

Signs and symptoms (No. of cases)	*Specific presentation of change (No. of cases)*
Headache (8)	Generalized (8)
Mental status changes (5)	Confusion (3)
	Somnolence (1)
	Physical and mental agitation (1)
Visual changes (5)	Eyelid edema (3)
	Ophthalmoplegia (1)
	Homonymous hemianopsia (1)
Flu-like symptoms (4)	Chills and night sweats (3)
	Rhinorrhea, fever (1)
Speech impairment (4)	Broca aphasia (2)
	Generalized difficulty speaking (1)
	Gerstmann syndrome (1)
Facial pain (4)	Ipsilateral ear pain (2)
	Dental pain (1)
	Trismus and dysphagia (1)

Mechanism of infection and mode of spread

In 4 (58.3%) of 12 cases, the infections were thought to have spread contiguously. In these cases, the site of odontogenic infection was ipsilateral to the abscess, and imaging demonstrated bony dehiscence, as well as a direct pathway from the oral cavity to the brain. In the remaining 5 cases, the infections were thought to have spread hematogenously. Of these, 3 patients had recent dental work and presumptive bacteremia as a consequence. One case of hematogenous spread led to multifocal brain involvement. The details are provided in Table III.

Microbiology

Culture data were as follows: in 6 cases, the infections were polymicrobial, in 5 cases, they were monomicrobial, and 1 case demonstrated gram-positive cocci in pairs that were not speciated. Three infections were found to contain both aerobic and anaerobic bacteria. The most common pathogens isolated belonged to the *Streptococcus* species (5 of 11 cases), 4 of which were *S. milleri* and one *Streptococcus constellatus*. All *Streptococcus* species were susceptible to ceftriaxone as well as to penicillin G. Other common isolates included *Staphylococcus* (3 of 11 cases), 2 with *Staphylococcus epidermis* and 1 with *Staphylococcus aureus*. *Fusobacterium* (3 of 11 cases) was the most common anaerobic species isolated. *Nocardia* was found in 1 case, an immunocompromised subject with type 2 diabetes and severe liver and kidney disease. Culture data are summarized in Table III.

Table III. Dental source, microbiology, localization, and route of spread

Age (years), gender	*Dental source*	*Microorganisms*	*Site of abscess*	*Presumptive route of spread*
69, F	Mandibular molar abscess	*Staphylococcus epidermis, Fusobacterium*	Temporal	Contiguous
44, M	Periapical abscess of tooth #3	*Streptococcus milleri*	Multifocal	Hematogenous
43, M	Left maxillary abscess	*Staphylococcus aureus*	Frontal	Contiguous
25, F	Periapical abscess of tooth #1	Anaerobes	Frontal	Contiguous
23, F	Periapical abscess of tooth #30	*S. milleri*	Frontal, parietal	Hematogenous
23, M	Left maxillary canine abscess	*Eikenella corrodens, Fusobacterium, Streptococcus constellatus*	Frontal	Contiguous
59, M	Mandibular bone infection	*Streptococcus anginosis,* anaerobes	Parietal, occipital	Contiguous
53, M	Prophylaxis 2 weeks earlier	Gram-positive cocci in chains	Frontal	Hematogenous
51, M	Full-coverage restoration	*S. epidermis*	Temporal	Contiguous
58, F	Dental restorations	*S. milleri, Haemophilus aphrophilus*	Frontal, temporal	Hematogenous
66, F	Prophylaxis with noncompliance to premedication antibiotics	*Nocardia*	Parietal, occipital	Hematogenous
60, F	Bilateral maxillary dental implant placement	*Propionibacterium acnes, Fusobacterium, Peptostreptococcus*	Frontal	Contiguous

F, female; *M*, male.

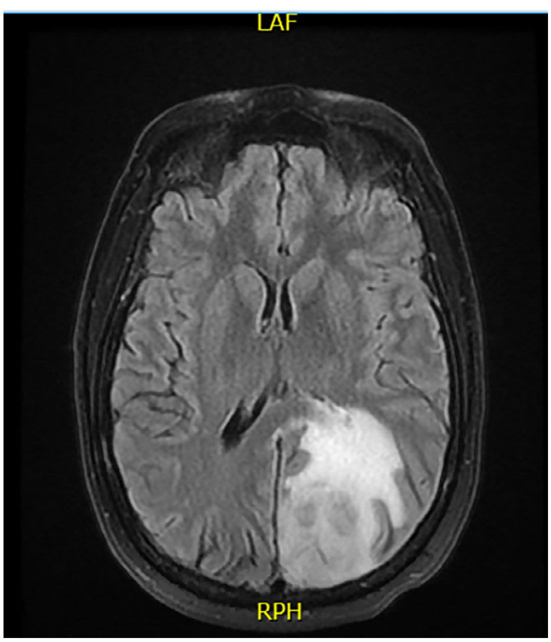

Fig. 1. Magnetic resonance imaging (MRI) depicting a multilobulated, peripherally enhancing mass in the left parieto-occipital region measuring 3.5 × 2.8 × 4.5 cm with a thick, irregular enhancing rind and extensive surrounding T2/FLAIR (fluid-attenuated inversion recovery) signal abnormality consistent with an abscess. There is no significant midline shift, herniation, or hydrocephalus.

Fig. 2. Head computed tomography (CT) scan demonstrating a 4-cm rim-enhancing hypodense lesion in the left temporal lobe, with moderate surrounding edema and local mass effect consistent with an abscess.

Treatment

In the 12 cases, combination antibiotic treatment (10 cases) was far more common than monotherapy (2 cases). The most commonly used antimicrobials were intravenous (IV) ceftriaxone (8 cases), metronidazole (7 cases), and vancomycin (5 cases). IV steroids were used in 2 of the 12 cases. Antiseizure medication was initiated in 2 of 12 cases because of intraoperative seizure activity. Surgical treatment was performed in 10 of the 12 patients. Open craniotomies were performed in 6 of 12 cases, and stereotactic CT-guided drainage in 4. Treatment specifics are summarized in Table IV.

Treatment outcomes

In all 12 cases, there was ultimate improvement in clinical and radiographic findings after treatment. Residual encephalomalacia was seen in 2 follow-up CT scans. Acute kidney injury as a sequela of antibiotic therapy was seen in 2 patients. Dilantin-induced hepatitis was

Table IV. Antibiotic specifics, surgical treatment, and patient outcomes

Age (years), gender	*Antibiotic specifics*	*Surgical treatment*	*Patient outcomes*
69, F	• Vancomycin • Ceftriaxone • Metronidazole	Craniotomy	Resolution on imaging and clinically
44, M	• Ceftriaxone	N/A	Residual encephalomalacia; neurologic deficits (mild deficits in attention, memory, and executive function)
43, M	• Ceftriaxone • Clindamycin	Craniotomy	Residual sinusitis; visual deficit (left eyelid lag); Dilantin-induced hepatitis
25, F	• Clindamycin • Ceftriaxone • Metronidazole	Craniotomy	Residual sinusitis; visual deficit (right eye medial hemi-vision loss)
59, M	• Vancomycin • Ampicillin and sulbactam • Meropenem	Craniotomy	Resolution on imaging and clinically
60, F	• Levofloxacin • Gentamicin • Metronidazole	Craniotomy	Recurrent brain abscess; neurologic deficits (residual upper and lower extremity weakness)
23, F	• Vancomycin • Penicillin G • Metronidazole	Stereotactic aspiration	Neurologic deficit (persistent foot numbness); speech deficit; Dilantin-induced hepatitis
53, M	• Ceftriaxone • Metronidazole	Stereotactic aspiration	Neurologic deficit (recurrent seizures)
51, M	• Vancomycin • Ceftriaxone • Metronidazole	N/A	Visual deficit (persistent right eye vision loss)
23, M	• Vancomycin • Ceftriaxone • Metronidazole	Craniotomy	Neurologic deficit (recurrent seizures); acute kidney injury (AKI)
58, F	• Ceftriaxone	Stereotactic aspiration	Resolution on imaging
66, F	• Imipenem • Trimethoprim/ Sulfamethoxazole	Stereotactic aspiration	Residual encephalomalacia; visual deficit (left eye visual field cut); AKI

diagnosed in 1 case. Both problems resolved with discontinuation of the offending medication. Four of 5 patients with visual symptoms at presentation experienced residual visual side effects, which included eyelid lag, visual field cuts, and unilateral blindness secondary to cavernous sinus thrombosis in 1 case. Five patients, 4 of whom had mental status changes at presentation, had long-term neurologic sequelae, which included memory and executive function deficits, extremity weakness, and recurrent seizures. One patient experienced a recurrent brain abscess which resolved 2 months after its onset. Follow-up information is summarized in Table IV.

DISCUSSION

The purpose of this study was to answer the following clinical question: "In all patients admitted to a tertiary care hospital with a diagnosis of a brain abscess, how common is odontogenic etiology?" Our specific aims were to identify the putative source of infection and to obtain a better understanding of the clinical presentation, treatment specifics, and outcome. We hypothesized that odontogenic sources in the head and neck region are more common than previously reported and that in the large number of cases designated as cryptogenic, an odontogenic source should always be considered.

Clear-cut identification of odontogenic infection as the etiology of a brain abscess can be difficult. Recent studies have found that up to 43% of intracranial abscesses are characterized as "cryptic" without a specific etiology identified.[7,9,11] It is possible that a subset of these cases involve an odontogenic infection that was overlooked at presentation. Other studies have cited a primary dental, sinus, ear, or mastoid infection as etiologic in 14% to 58% of brain abscesses.[7] Odontogenic infections alone have been cited as being responsible for as little as 5% of such cases.[12] In our study, 12 (13.6%) of 88 abscesses were attributed to odontogenic sources, a higher percentage than previously reported in the literature and supporting our hypothesis. This may reflect early input from both dental and oral and maxillofacial surgery (OMFS) consultants available at our institution, allowing for diagnosis of odontogenic infections. However, because the number of cases is small, firm conclusions are difficult; this would best be addressed by multicenter studies in which data can be pooled and perhaps allow for statistical evaluation.

Demographic characteristics and medical history

Among our cohort of patients, both genders were equally represented, and the average age was 47.8 years. Prior systematic reviews have reported a similar mean age but a much higher proportion of male patients (82.1%).[11] The reason for this distinction in our cohort is unclear. The role of immunosuppression in the development of brain abscesses has been noted by other authors. Studies have shown that up to 20% of patients with a predisposing medical condition (particularly diabetes and alcoholism) have increased susceptibility to central nervous system (CNS) infections.[5,13] This was noted in our study as well. Increased susceptibility to certain organisms, such as *Listeria, Cryptococcus*, and *Nocardia*, has been reported in this patient population.[7] Nocardial infection was found in 1 of our patients who had multiple comorbidities. Nocardial brain abscesses are rare, accounting for less than 2% of brain abscesses and occur as a result of disseminated infection and lack of cell-mediated immunity.[14] In immunocompromised patients, hematogenous spread is the most common mode of spread.[15] This was also found in our sample of immunocompromised patients with diagnoses of diabetes, alcoholism, and active smoking. Studies also report poorer prognosis in immunocompromised patients, including a 2.8-fold increased risk of mortality or severe disability.[10] Follow-up showed that our subject with a nocardial abscess experienced persistent right eye vision loss, chronic kidney dysfunction, decreased dexterity, and self-care deficits in grooming and dressing.

Presenting signs and symptoms

The classic clinical triad suggestive of brain abscess is fever, headache, and focal neurologic deficits, although recent studies have reported that less than 20% of patients present with all 3 manifestations.[1,7,13] This triad was found in 5 (41.6%) of our 12 patients. Earlier symptoms are often nonfocal and include headaches and nausea.[7] In such cases, inflammatory markers may be helpful in early detection, although they are not diagnostic. Patients often present with moderately elevated ESR and CRP.[7,9,10,16] Approximately 60% of patients have an elevated leukocyte count and CRP.[13] In our cohort of patients whose ESR and CRP were measured, the values were above the normal range, not a surprising finding. Fever and neurologic deficits commonly present later and are associated with a more guarded prognosis.[17] This was also noted in our study. Of the 5 patients who initially presented with mental status changes, 3 had accompanying visual changes that persisted after resolution of the abscess, and 1 had residual deficits in attention and executive function.

Dental history

A systematic review of previous reports and case studies by Moazzam et al. identified 60 cases of brain abscesses attributed to odontogenic sources and found recent dental work reported in 41.7% and active dental pathology in 86.7%.[11] Our findings were similar. Five of the 12 patients (41.6%) in our cohort had undergone dental procedures within 2 months of presentation, and active dental disease was found in 7 (58.3%). Moazzam et al. found infection in the molars to be responsible for the abscess in 31.7% cases, whereas our cohort demonstrated a much higher prevalence (83.3%). We believe our finding is more realistic because dental infections are more prevalent in the posterior teeth.[18] The current literature cites equal involvement of maxillary and mandibular teeth, consistent with our cohort, in which 4 (33%) of the 12 patients had maxillary involvement, 3 (25%) had mandibular involvement, and 5 (41.7%) had involvement of both jaws.[11,12]

Localization and mode of spread

Findings regarding abscess localization in our patients are consistent with those reported by other studies. Of the brain abscesses attributed to a head and neck source, most were localized to the frontal lobe, temporal lobe, or cerebellum.[19] In particular, frontal lobe localization was the most common in reports of odontogenic etiology.[16] Paranasal sinusitis most commonly spreads to the frontal lobe, whereas sphenoid sinusitis and mastoiditis most commonly spread to the temporal lobe.[6,8] Unifocal localization (often involving areas of contiguous lobes), as opposed to generalized abscess formation, has been reported in 87% of cases.[4] Our results are consistent with these findings. Odontogenic infections can spread centrally via a hematogenous route through the low pressure, valveless venous system or via direct extension through the paranasal sinuses.[20] To distinguish between the 2 mechanisms, localization of the abscess to the ipsilateral side of the odontogenic source suggests contiguous extension, whereas contralateral involvement suggests a hematogenous route.[12] Moazzam et al. suggest that hematogenous spread is more likely because location is often unrelated to the side of dental pathology.[11] Haymaker et al. report maxillary and mandibular teeth to be equally etiologic, further supporting a greater likelihood of a hematogenous route of spread because infections of mandibular teeth presumably do not spread contiguously.[12] Our cohort, however, showed an almost even distribution of spread, with a slight predilection for the contiguous route (58.3%). Furthermore, we found that in 2 of our cases, mandibular infections had spread contiguously to the brain through the parapharyngeal spaces.

Microbiology

A recent review of severe orofacial infections treated at our institution identified the *Streptococcus anginosis* as the most common isolate and found a 32% resistance rate to clindamycin in these organisms.[21] This was the most commonly cultured bacterial species in our current cohort of patients, and similar resistance patterns were found. All isolates were sensitive to both penicillin and ceftriaxone. The presence of anaerobic gram-negative bacilli and *Bacteroides* spp. is also strongly indicative of odontogenic etiology.[22,23] This is consistent with the findings in our cohort, in which anaerobic bacteria, most notably *Fusobacterium*, were isolated in42% of cultures.

Treatment

The gold standard of treatment for a brain abscess is a combination of surgical drainage, IV antibiotics for a minimum of 6 weeks, and interval CT or MRI imaging.[20] Both direct drainage via craniotomy and CT-guided stereotactic drainage were associated with favorable outcomes in our cohort. This finding is consistent with those reported by other studies citing no significant differences in patient outcomes with use of open drainage and aspiration.[9] The 2 patients who relied on antibiotics without surgical treatment experienced poorer outcomes: One developed a recurrent brain abscess 6 months after initial treatment, and the other presented with executive function deficits on follow-up. All patients in our cohort received IV antibiotics for at least 6 weeks, most notably a combination of IV vancomycin, ceftriaxone, and metronidazole.

Treatment outcomes

The current literature suggests that severely impaired mental status on admission is associated with higher morbidity.[24] In our group, in 4 (80%) of the 5 patients who presented with mental status changes, long-term sequelae or recurrent infections developed, as described. Seizure activity is a reported long-term risk in patients with brain abscesses, yet the reported incidence of seizures varies greatly from 9% to 70% in the literature.[25] This appears to be greater in those treated with craniotomies.[21] Research also suggests that both the size of the hypodense area surrounding the abscess and the frontoparietal localization are associated with seizure development.[26] This is consistent with our data. The 2 patients who developed persistent seizures had abscesses localized to the frontal lobe. Treatment included antiepileptic prophylaxis for at least a year.[8,20] Although seizures are the most common long-term sequelae of brain abscesses, persistent focal neurologic deficits, such as loss of vision, are not uncommon. Recent studies have reported at least 1 seizure during the postoperative period in 11% and residual neurologic deficits (including hemiparesis and vision loss) in 7.9%.[7,26]

CONCLUSIONS

Our findings highlight a subset of brain abscesses originating from a head and neck source (12 of 88 [13.6%]) that are of odontogenic etiology. This percentage is higher than previously reported in the literature (5%−8%) and highlights the need to rule out dental sources in cases considered cryptogenic. Of note is that the total number of cases identified from our database from all etiologic sources (including cardiac and pulmonary) was 167, thus making the overall percentage of odontogenic cases 7.2% (12 of 167). *S. milleri*, a common isolate in odontogenic infections, was cultured in 42% and demonstrated a greater than 30% resistance to clindamycin. Distinct from other studies, molar (both maxillary and mandibular) etiology was the most common in this cohort. Furthermore, contiguous spread is as prevalent and harmful as hematogenous spread in causing brain abscesses of odontogenic origin. Frontal lobe localization was found to be the most common. Residual visual and neurologic deficits were found in 6 of our 12 patients. Early initiation of appropriate therapy was associated with a better outcome. Because an odontogenic source of infection could be a potential etiologic factor, the need for timely dental examination is a crucial consideration in the management of brain abscesses.

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