Bacterial Cerebral Abscess

Sorayouth Chumnanvej, M.D.

Introduction

Central nervous system (CNS) infection is defined as the infection in both of the brain and the spinal cord. There are several organisms which could invade CNS including bacteria, virus, fungus or protozoa. CNS infection must be concerned to be the emergent situation with diagnostic and therapeutic issue. This article will focus only on the bacterial cerebral infection which can affect both meninges and brain parenchyma particularly bacterial cerebral abscess. When there is a bacterial cerebral infection, this might be occurred epidural, subdural or intracerebral region. When it ruptures into the subarachnoid space, meningitis will be revealed with fever, nuchal rigidity and alteration of consciousness or if it ruptures into the ventricular system, ventriculitis is diagnosed. There could be even a diffuse or focal mode of inflammation and infection. Surgical treatment for diffuse bacterial infection such as bacterial meningitis or bacterial encephalitis, is rarely contributing. Neurosurgical procedure will play a great role in the intracranial focal bacterial purulent conditions including cerebral abscess because it is a severe threatening pathology. Bacterial cerebral abscess is a focal intraparenchymal bacterial infection that can cause neural tissue damage through inflammation, edema and compression.

Before the 19th century, bacterial cerebral infection was a nearly constantly grave condition. It was difficult to diagnose until autopsy was performed. The Roman surgeon named Claudius Galenus, used trepanation; the procedure is used for relieving intracranial pressure and drainage of phlegmatous lesions, to drain pus beneath the skull. He was credited to be the first surgeon who attempted to treat intracranial purulent collection(1). In 1872, Weeds reported the successful drainage of a posttraumatic cerebral abscess(2). A few years later, Macewen published his work; the pyogenic

infective diseases of the brain and spinal cord. He reported the treatment of 25 brain abscesses with very satisfied result. So far, there are several techniques which have been described to drain cerebral abscess, including tube drainage, marsupialization, excision and tapping with aspiration(3). Recently, the minimally invasive procedure by Dandy which has been refined in the modern era via using stereotactic techniques is very well acceptance.(4-6). The further crucial step in the treatment of intracranial abscess conditions was the development of antibiotic drug which was introduced in the mid 20th century such as penicillin or chloramphenicol. Nicolosi et al reported a large retrospective epidemiological study of patients in Olmsted County, Minnesota. The study found 38 cases of intracranial abscess over a 47-year period and stated an incidence of 1.3 cases of cerebral abscess per 100,000 person-years(7). In Thailand, there is a scanty report about bacterial cerebral infection and abscess. There were 4-6 cases per year of the community-acquired bacterial meningitis in adults and commonly in male rather than female(8). According to the cerebral abscess, there were 8.4 cases per year of cerebral abscess in children that was reported in Thailand and the congenital heart disease is the most common cause (9). S. pneumoniae is the most common pathogen of its cause(8). There were 107 cases of intracranial abscess and granuloma in adult during 2001-2011 be treated in Ramathibodi Hospital. Up to date, the advent of several new antibiotic drugs and neurosurgical knowledge permit the treatment of bacterial infection particularly in CNS with reasonable risk. Finally, the developments of neuroimaging techniques, including CT scan and MRI studies, have extremely improved. This is not only the advantage in preoperative diagnosis for bacterial cerebral infection, but also for the preoperative plan, intraoperative and monitoring of treatment results. So the fundamental that actively contributes to the sufficient and success treatment in bacterial cerebral abscess is to understand the pathogenesis and pathophysiology of bacterial CNS infections.

Pathophysiology of Bacterial Cerebral Abscess

The central nervous system (CNS) is isolated. It is so sterile area and against bacterial attack by the skull, the leptomeninges, and the blood–brain barriers. There is no lymphatic system in the CNS, on the other hands, cerebrospinal fluid (CSF) acts as the antibodies and transports the metabolic waste products. When the bacteria invade into the CNS, the inflammation occurs. If the infection localizes focally, meninges is infected and inflamed. So far after the meninges are ruptured by the bacterial infection the brain parenchyma is infected with a focal bacterial purulent. Finally the cerebral abscess is formed.

The causes of bacterial cerebral abscess might be classified into four etiologic classes: 1) contiguous infections; 2) hematogenic infections; 3) trauma or postoperative causes; and 4) immune compromise host infection(10). Whenever there is an bacterial infection occurred within the adjacent area such as air sinus or the petrous bone, it will cause a localized osteitis nearby the brain. Then it might invade and spread intracranially. The direct extension or contiguous infection occurs by adjacent osteomyelitic bone or retrograde spread by the phleblitis. Commonly the bacterial cerebral abscesses resulting from contiguous infections tend to be solitary. The hematogenous infection might be from a distant infection and commonly occurs through the invasion and destruction of the skull, meninges and cerebral parenchyma which envelopes by pathogens. This will proceed through septic thrombophlebitis of intra-extracranial venous channels. These infections are frequently polymicrobial and multiple lesions might be identified. According to trauma or postoperative causes such as penetrating head injury, is the increasing cause of bacterial cerebral abscesses presently. Retained foreign bodies are the main concern with dural tear. For the last group, the immunologically compromised patients account for an increasing numbers of cerebral abscess. Transplant patients or during chemotherapy and also acquired immunodeficiency syndrome patients develop cerebral abscesses with varieties of pathogens from the previous etiologies however fungal and parasitic pathogens are predominate.

Initially, after there is a bacterial infection, it will be confined to the epidural space. However it will break the dura and spread subdurally further. Commonly, the primary infections which are the causes of the secondary cerebral abscesses are from direct or contiguous spread. Contiguous spread of bacterial infections including from the paracranial sinuses, chronic otitis media or dental abscess remains the major pathophysiologic mechanism in bacterial cerebral abscess formation. The distant or hematogenous spread infection causes bacterial cerebral abscess could be from lung infection, congenital cyanotic heart disease or gastrointestinal infection. Because of these reasons, when the bacterial cerebral abscess is diagnosed the physicians always have to look for the possible primary cause of it.

So far, whenever it spread into the subarachnoid space, there will cause meningitis. In bacterial cerebral infections, bacterial meningitis is both the most common and most serious bacterial infection of the CNS before the abscess is formed. It is characterized by acute purulent infection of the meninges affecting the pia, arachnoid, and subarachnoid space. If the infection and inflammation process is still going on the cerebritis occurs. After an initial cerebritis stage during the first 3 days of the infection, a hypodense lesion with mild mass effect is visualized by CT brain scan. There is an

edema formation, diffuse microglial activation, and astrocytosis occurring around. During the next 6-7 days, this cerebritis stage begins to consolidate. A thick ring-like enhancing lesion is seen on CT brain, reflecting hypervascularity, and the mass effect will cause the increase intracranial pressure. Fibronectin is deposited around the central necrotic area as an introduction to the appearance of myofibroblasts concomitantly with collagen formation. The encapsulation process continues during the days 10–13, this mass effect decreases and the capsule becomes thinner. In the fully formed abscess after 2 weeks, the capsule is rather thin and the amount of edema has decreased significantly. Hypervascularity of the capsule is maintained during this period. The direction of growth of the abscess is through the less vascularized white matter, where it may lastly rupture within the ventricular system. Another direct inoculation means are from trauma or surgery: However the infections following open traumatic injuries or surgery are infrequent.

About 60 % of cases which related to middle-ear infection and 20 % of cases accounts for frontal sinusitis are the primary source of infection commonly for the cerebral abscess(7,9). There are 10%-20% of cases which there is no primary source of infection identified and also known as the cryptic abscess without obvious source(7,9,10).

According to the hematogenous spread, it is the result of bacteriemic episodes secondary to infectious pathologies in other organs such as endocarditis, or breaches including dental extractions, or invasive diagnostic studies introducing micro-organisms into the blood stream, and is favored by underlying conditions shunting the pulmonary filter such as arteriovenous fistulas or persistent foramen ovale. This is a frequent source of brain abscesses that tend to be monomicrobial and polymicrobial.

The likely primary sources of bacterial cerebral abscess are as followings:

1. Direct extention primary sources

   -Paranasal sinus: streptococci, Bacteroides, Haemophilus, and Fusobacterium spp.

   -Otitis media, mastoiditis: streptococci, Enterobacteriaceae, Bacteroides, and Pseudomonas spp.

   -Dental sepsis: occurs in frontal lobe; mixed Fusobacterium, Bacteroides, and Streptococcus spp.

2. Hematogenous spread or distant site of infection

   - Congenital heart disease: Streptococcus, Haemophilus spp.
   - Endocarditis: S. aureus, Viridans streptococci
   - Lung: Streptococcus, Actinomyces, Fusobacterium spp.

- Urinary tract: Enterobacteriaceae, Pseudomonadaceae
- Intra-abdominal: Streptococcus, Enterobacteriaceae, anaerobes

3. Penetrating or postoperative causes

-Penetrating head injury: Staphylococcus aureus, Clostridium species, Enterobacteriaceae spp.

-Postoperative: Staphylococcus epidermidis and S. aureus, Enterobacteriaceae, and Pseudomonadaceae

4. Immunocompromised host

Clinical Presentation

The clinical presentation of a patient with a cerebral abscess varies significantly depending on the size, location, and number of abscesses. Although a minority of patients will have focal neurological deficits, the majority of signs and symptoms are nonspecific. In many case series, headache was the most common symptom and motivation in search of medical attention. Fever may not be present and is not reliable in making a diagnosis. Most series report about 50% of patients will have fever at presentation, though other series report fever in as few as 20% and other series in as many as 79% of patients(2,9,11-14). In Thailand, The classic triad of brain abscess; including headache, fever, and focal neurological deficits, was found in only 9.4%(9). The five most common initial signs and symptoms were fever, headache, vomiting, alteration of consciousness and focal neurological deficit are the clinical manifestations that might help to make diagnosis. Nausea and vomiting are also common in the setting of increased intracranial pressure. Also other signs of elevated ICP, with papilledema and alteration of consciousness, may be present and specify an immediate medical attention. Another varieties of other nonspecific signs and symptoms, including malaise, meningismus, and photophobia, may be presented in patients with cerebral abscess.

Clinical Management

1. Clinical diagnosis

1.1 Lumbar puncture is definite contraindicated in patients with suspected cerebral abscess.

1.2 Prior to the routine use of the brain imaging, the clinical diagnosis required suspicion of a focal neurological deficit. Because the clinical features of patients with cerebral abscess are nonspecific. The differentiating cerebral abscess from other CNS diseases was difficult. Moreover, in the absence of focal neurological deficits, localization of the abscess would be also not easy. The

routine use of CT brain determines these equivocal problems, allowing for accurate diagnosis and localization of cerebral abscess. MRI brain also can supplement and potentially replace CT for the diagnosis and follow-up.

2. Laboratory Test and imaging studies

2.1 Laboratory Test

-CBC: White blood cell (WBC) counts are elevated in 60% of patients.

-ESR is usually elevated, but may be normal.

-C reactive protein is usually elevated but the measurement technique is inexpensive and is available only in the clinical laboratories of some hospitals.

- Blood cultures are most often negative. Some studies reported 10% positive.

-Gram stain and culture of material aspirated yield; after surgical drainage, is 100%.

2.2 Imaging studies

- MRI brain is the diagnostic procedure of choice. It provides superior detail compared with CT brain. According to MRI brain, there is a higher sensitivity and specificity than CT brain, but not always in immediately availability.

- CT brain with intravenous contrast is still an excellent test. There is 95% to 99% sensitivity.

- Serial CT or MRI brain scanning is recommended to follow the response of the clinical therapy.

3. Medical treatment

Characteristics of bacterial cerebral abscess regarding to sources and locations, the most common causative organism and the recommended empirical treatment is classified as followed.

Source	Abscess Location	Frequent Organisms	Empirical Treatment
Paranasal sinuses	Frontal	Streptococci (aerobic and anaerobic), Haemophilus sp. Bacteroides sp., Fusobacterium sp.	Ceftriaxone 3-4 g/d + Metronidazole 500 mg/8h
Otic infections	Temporal Lobe Cerebellum	Streptococcus sp. Enterobacteriaceae, Bacteroides sp. Pseudomonas aeruginosa	Ampicillin 2g/8h+ Metronidazole 500 mg/8 h + Ceftazidime 2g/ 8 h
Hematogenous spread/ cryptogenic	Mainly middle cerebral artery territory, but any region can be involved Multiple abscesses	• Endocarditis Staphylococcus aureus Streptococcus viridans • Urinary tract Enterobacteriacea Pseudomonas • Intra-abdominal Enterobacteriacea Streptococcus sp. Anaerobes • Pulmonary abscess Streptococcus sp. Actinomices sp. Fusobacterium sp.	Endocarditis: Benzolpenicillin 1.8-2.4 g/6 h Ceftriaxone 3-4 g/24 h + Metronidazole 500 mg/8 h
Trauma	Depends on site wound	Staphylococcus aureus Clostridium Enterobacteriaceae	Cloxacillin 2g/4h or Ceftriaxone 3-4/g/24 h
Neurosurgery	Depends on operated area	Staphylococcus aureus Staphylococcus epidermidis Enterobacteriaceae Pseudomonas	Vancomicyn 1 g/12 h + Ceftazidime 1 g/8 h

Ref: Michael J. A., Franc¸ O.B., and Dick F. S.,Handbook of Clinical Neurology, V96, 2010.

4. Neurosurgical management

In management of cerebral abscess, there are some concerns including life saving, preserving, restoring function, and also preventing sequels from brain damage and recurrence of infection. Additionally, the methods of treatment have a tendency to significantly differ in different neurosurgeons. Jooma et al emphasized that, in conclusion, there is no hard and fast rules can be laid down in definite treatment of cerebral abscess. In some cases with high intracranial pressure, operative decompression is necessary to save life, and in these it is usually advisable at a later stage, or even at the time of the first operation, to excise the abscess. In other cases, where the abscess is situated in an eloquent area of the brain and the abscess is solitary, repeated aspiration is the better method of treatment. But in these regions, as in other parts, an abscess is occasionally encountered which does not collapse after repeated aspiration, and must be excised to obtain a cure(15).

Conclusion

Bacterial cerebral abscess can create an important diagnostic problem while signs and symptoms are nonspecific. So a doubt needs to be reserved in mind, especially in immunocompromised patients, a past history of vulvular heart, , dental, otic or nasosinusal pathologies, bacteremic conditions or surgical or traumatic previous circumstances.

Neurosurgical treatment option is an essential tool in managing of cerebral abscesses to identify the microorganism, diminish the risk of complications, ease mass effect and improved the clinical conditions. Treatment of the primary causes which is the most important mean including vulvular heart, dental caries, otic or nasosinusal pathologies, when present is necessary. Increased intracranial pressure due to bacterial cerebral abscess represents the neurosurgical emergency, so craniotomy and evacuation are required as soon as it is diagnosed. Epidural abscesses need to be evacuated to eradicate the infection. Bacterial infections following neurosurgical procedures indicate for surgery when there is a purulent collection. In these cases, surgical removal of foreign bodies and wound debridement offers a more rapidly and safer improvement. According to the small abscess or its location is in eloquent area, to manage with antibiotics alone is possible after stereotactic aspiration and known causative organisms.

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