Scalp, epidural, subdural and brain abscesses caused by *Streptococcus intermedius* secondary to sinusitis: A case report

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ABSTRACT

Introduction: Brain abscess and subdural empyema are the most common surgically treated intracranial infections. Involvement of all layers from scalp, epidural and subdural region in brain abscess has not been reported. The clinical significance of the anatomy of emissary veins, sinuses and brain parenchyma is illustrated.

Case presentation: A 25-year-old man with a history of sinusitis presented with headache, confusion, fever and a left frontal scalp swelling. Computed tomography (CT) scan of brain revealed sinusitis and abscesses involving scalp, epidural, subdural regions

and brain parenchyma. The patient underwent surgical drainage and antibiotic treatment. No complication occurred and the patient recovered with no neurological deficit.

Conclusion: Brain abscess is a potentially fatal complication of sinusitis. The presentation of scalp abscess could be a sign of deep infection. Clinicians should have a high degree of awareness in order to prevent neurological complications. Prompt diagnosis and treatment is emphasized.

Keywords: brain abscess, emissary veins, epidural abscess, scalp abscess, sinusitis

Cite This Article: Cheng, M.F., Yeow, S., Chan, W.H., Chow, K.H. 2020. Scalp, epidural, subdural and brain abscesses caused by *Streptococcus intermedius* secondary to sinusitis: A case report. *Indonesian Journal of Neurosurgery* 3(2): 52-58. DOI: [10.15562/ijn.v3i2.118](https://doi.org/10.15562/ijn.v3i2.118)

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INTRODUCTION

There is a wide range of clinical, radiological and surgical characteristics in different intracranial infections.^{1,2} The incidence of brain abscesses ranges from 0.4 to 0.9 per 100,000 persons per year.³ Higher prevalence in developing countries and immunocompromised patients is observed.⁴ The classical presentation of focal neurological deficits, headache and fever is uncommon. This triad occurs in fewer than 40% of patients.^{5,6} The most common causative organisms were *Streptococcus* species. However, no clear source could be identified in up to 30% of cases.⁷ Intracranial extension secondary to acute or chronic sinusitis is found in 3.7% to 11% of hospitalized patients.⁸ We report a case of abscesses involving from the scalp to brain parenchyma due to *S. intermedius* secondary to sinusitis. Physicians should have high degree of suspicion for intracranial infection in the presence of scalp swelling, fever and confusion. Early recognition and prompt treatment can reduce potential neurological disability.

CASE PRESENTATION

A 25-year-old man with e-antigen negative chronic hepatitis B presented with headache, confusion

and fever for two days. He had nasal obstruction, mild headache and mild fever before developing confusion. His headache was intermittent but was worsening over two days. No neck pain, vomiting or photophobia was reported. There was no history of trauma. On the day of admission he developed confusion and fever up to 39 degree Celsius. Blood pressure was 120/81 mmHg and heart rate was 105 per minute. His Glasgow Coma Scale on arrival was E4V1M6. Pupils were 3 mm equal and reactive. On examination, the patient was febrile with a left frontal scalp tender swelling. No neck stiffness was detected. The remainder of the physical examination was normal.

Laboratory data revealed leukocytosis with 23,100 cells per milliliters (Normal range: 3700 – 9200 cells per milliliters). HIV screen was negative. Other hematological and biochemical parameters were normal. Head CT scan (Figure 1, 2 and 3) revealed features of sinusitis. Besides features of sinusitis, there were scalp, epidural, subdural and brain abscesses. The patient underwent surgical drainage. Six milliliters of pus from brain abscess and five milliliters of pus from scalp abscess were drained. Microscopic examinations of the abscess aspirate showed a large number of

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Figure 1. Mucosal thickening and retention changes in left ethmoid and maxillary sinuses.

Figure 2. A 4.3 cm rim-enhancing brain abscess in the left frontal lobe and extensive surrounding oedema. Right parafalcine extra-axial subdural abscess and left epidural abscess seen on contrast CT scan. A scalp abscess measured 6.5 cm is also seen.

polymorphonuclear leukocytes and gram-positive cocci in chains. Abscess and blood culture showed *Streptococcus intermedius*, which was sensitive to penicillin. Minimum inhibitory concentration (MIC) of penicillin was 0.03 microgram per milliliters. Smear for acid-fast bacilli and culture of abscess aspirate were negative. Abscess culture was negative for fungus, *Nocardia* and *Actinomyces*. The patient underwent two more surgeries for drainage of the abscesses. The patient was initially given intravenous (IV) penicillin G 4 million units every four hours and IV metronidazole 500 milligrams (mg) every eight hours. He developed generalized macular rash. Penicillin allergy was confirmed when penicillin G was replaced with vancomycin 500 mg IV and resolution of rash was observed. He completed total six weeks of antibiotics treatment and head CT scan showed resolution of brain abscess. The patient had a complete recovery eventually.

It is a case of brain abscess secondary to sinusitis. The involvement of all layers from scalp, epidural and subdural regions with abscess formation was attributed to by the presence of emissary veins.

DISCUSSION

Brain abscess is a focal pus collection in the brain parenchyma. Invasion of bacteria to brain tissue is either by direct spread or through hematogenous seeding.⁹ Direct local spread accounts for at least 50% of cases.⁷ Less commonly, penetrating brain trauma is a cause of brain abscess.⁷

Direct spread of organisms from a contiguous region to brain parenchyma usually produces a single brain abscess. Brain abscesses can result from primary infections in head and neck region, such as dental infection, frontal or ethmoid sinusitis, otitis media and mastoiditis.¹⁰⁻¹⁴ Inferior temporal lobe and cerebellar brain abscesses are characteristically secondary to otitis media and mastoiditis. Infection from frontal and ethmoidal sinuses spreads to frontal lobes. Dental infection also usually causes frontal lobe brain abscesses.

Foreign bodies are important but uncommon sources of direct spread of infection.^{15,16} Neurosurgical procedures can also cause delayed manifestation of brain abscess secondary to direct spread of organisms.^{17,18}

Brain abscesses related to bacteraemic spread of organisms usually produce multiple abscesses. The most common source of infection is lung infections, such as empyema and lung abscesses. Cyanotic congenital heart diseases in children are associated with brain abscesses.¹⁹ It should be noted that 1 – 7% of infective endocarditis is complicated by brain abscesses.²⁰ Other extracranial sources

Figure 3. A rim-enhancing scalp abscess and a brain abscess in the left frontal lobe are seen.

include skin, pelvic and intraabdominal infections. Extracranial sources with bacteraemic spread leading to metastatic seeding of the brain usually result in multiple brain abscesses.⁷ The abscesses are commonly located along the distribution of the middle cerebral artery and usually at the gray-white matter junction.²¹ Brain abscesses could be associated with or located within a tumour, however it is a rare occurrence. Reported tumours include glioblastomas, astrocytomas, meningiomas and metastases.²²⁻²⁶ The mortality of such presentation is high. No underlying condition or primary infection can be identified in 20 to 40% of brain abscesses cases.²⁷⁻²⁹

Subdural empyema is a pyogenic infection located between the arachnoid mater and dura. Subdural empyema alone is uncommon and comprises 15 – 20% of localized intracranial infections.³⁰ Both scalp abscess and extradural abscess can result from transcranial extension of frontal sinus infection.³¹ The significance of emissary veins in scalp infection was recognized nearly a century ago. Sir Frederick Treves stated that if there were no emissary veins, injuries and diseases of the scalp would lose half their seriousness.³² The emissary veins are valveless.³³ They play important functions in equalizing intracranial pressure and

selective blood cooling.^{34,35} Emissary veins form the venous communication between the veins outside the cranium with the intracranial venous sinuses.³⁶ There is great individual variation in the anatomy of emissary veins.³³ The valveless emissary veins allow bidirectional blood flow between intracranial and extracranial regions. The emissary veins may serve as pathways that infections are carried in both directions.³⁷⁻³⁹

The initial manifestation of brain abscess is usually non-specific.^{6,27,40} Symptoms are present for 2 weeks or less in about two-thirds of patients. Mean times from symptom onset to presentation at a medical center range from 7 to 25 days.⁵ The classical triad is present in about fewer than 40% of patients.^{5,6} Fever is present in only 50% patients.⁴¹ Focal neurological deficits are observed in up to 50% of cases and seizure is the first manifestation of brain abscess in 25% of patients.⁴² Grand mal seizures are common in frontal abscesses. Nuchal rigidity is associated with occipital lobe abscess or an abscess that has leaked into a lateral ventricle. Suddenly worsening headache and signs of meningism could represent rupture of abscess.

Laboratory investigations and imaging studies are important in evaluation of brain abscess. Increased inflammatory markers of bacterial infection are usually observed. However, normal results of white blood cell count, C-reactive protein and erythrocyte sedimentation rate have been reported.⁴¹

Imaging findings are related to the stage of the infection. On computed tomography (CT) early cerebritis appears as an irregular low-density area which may not enhance, or may show patchy enhancement. A more conspicuous rim-enhancing lesion would show as cerebritis evolves.⁴³ Patchy enhancement in early cerebritis would evolve to a rim of enhancement in late cerebritis, and later on brain abscess forms. Rim enhancement in late cerebritis is not associated with collagen deposition as in abscess. Late abscess show decreased oedema and mass effect. Brain abscess wall is usually 1 mm to 3 mm thickness with surrounding parenchymal oedema. The ring of enhancement may be relatively thin on the ventricular or medial surface in the deep white matter as they are the less vascular areas. The presence of gas suggests gas-forming organisms. CT scan may be more easily obtained on an emergency basis.

Magnetic resonance imaging (MRI) is better than CT scan in differentiating between different brain masses. MRI is the imaging study of choice. It is more sensitive for satellite lesions and early cerebritis. MRI provides better assessment of brainstem. Contrast between cerebral oedema

and the brain is greater on MRI. Assessment of spread of inflammation into the subarachnoid space and ventricles is more sensitive.^{44,45} Classically an abscess appears a contrast-enhanced rim surrounding a necrotic core. The rim is T1 isointense to hyperintense and T2 hypointense, relative to white matter. Variable T2 hyperintensity in central necrosis depends on the protein content. Central necrosis of the abscess is T1 hypointense.

Brain abscess can be distinguished from other ring-enhancing lesions on diffusion-weighted MRI (DWI).⁴⁶ Abscesses are usually hyperintense on DWI, indicating restricted diffusion, characteristic of viscous materials, such as pus. Neoplastic lesions are hypointense or of variable hyperintensity which are lower than the intensity shown with an abscess on DWI.⁴⁷ Proton MR Spectroscopy reveals elevated succinate level, which is specific for an abscess. Elevated signals of lactate, acetate and alanine are detected. MR spectroscopy may be used to differentiate anaerobic from aerobic metabolism as elevated succinate and acetate peaks are typically observed in anaerobic infection. In strict anaerobe infections lactate peaks are lowest on MR Spectroscopy due to metabolic lactate consumption.

Bacteria, fungi and parasites have been reported as the causes of brain abscess.^{9,48} *Streptococcus anginosus* group is the most commonly isolated organism in brain abscess cases.⁹ *Streptococcus intermedius*, along with *Streptococcus anginosus* and *Streptococcus constellatus*, is a gram-positive round-shaped coccus of *Streptococcus anginosus* group (also known as the *S. milleri* group).⁴⁹ These viridans were first isolated from dental abscesses and described by Guthof in 1956.⁵⁰ It is a part of normal flora in stool, upper respiratory, gastrointestinal and female urogenital tracts, as well as in oral cavity, with the ability to cause systemic infections and abscesses.⁵¹⁻⁵⁴ There are other groups of pathogenic streptococci, such as *S. pyogenes* and *S. agalactiae*. *S. anginosus* group is unique that it can cause abscesses.⁵⁵ *S. anginosus* group members produce pyrogenic exotoxins.⁵⁶ Intermedilysin production is a unique feature of *S. intermedius*. Intermedilysin is a cytolytic toxin specific to human cells and it is related to deep-seated abscesses.^{57,58} Interaction between *S. anginosus* group and polymorphonuclear granulocytes may also contribute to abscess formation.⁵⁹ *S. anginosus* group infections commonly involve head and neck region, the gastrointestinal tract or the oral cavity. It is observed that *S. anginosus* group members can enter systemic circulation in the absence of obvious focus of trauma or infection.^{60,61}

Streptococcus, together with *Bacteroides* and *Peptostreptococcus*, are most commonly identified

in brain abscesses secondary to contiguous spread.⁶²⁻⁶⁴ *Streptococcus* and *Peptostreptococcus* are mostly isolated in brain abscess patients secondary to cardiac disease.⁶⁴ *Staphylococcus* and *Streptococcus* are commonly found in patients with previous surgery. In open head trauma cases, *Staphylococcus*, *Streptococcus*, *Clostridium* and *Enterobacteriaceae* are commonly found.^{62,64,65} Among brain abscess cases in immunocompromised patients, and patients on chemotherapy, steroid therapy, or after organ transplantation; fungal infections, *Toxoplasma*, *Staphylococcus*, *Streptococcus* and *Pseudomonas* are frequently identified.^{62,64,66}

For pyogenic abscesses greater than 2.5 cm, surgical drainage is recommended.⁴⁶ In pyogenic abscesses less than 2.5 cm, antimicrobial therapy is the first-line treatment. *Streptococcus intermedius* is sensitive to penicillins, cephalosporins and lincosamides. A combination of IV therapy of ceftriaxone and metronidazole demonstrate good brain penetration in *Streptococcus intermedius* brain abscess cases.⁴⁸ *Staphylococcus aureus* and *Staphylococcus epidermidis* brain abscesses should be covered with vancomycin. Vancomycin is also effective in brain abscess related to clostridium species. Linezolid, daptomycin or trimethoprim-sulfamethoxazole can be used in cases of vancomycin resistance. Fungal infections such as *Aspergillus*, *Candida* and *Cryptococcus* should be treated with amphotericin B. Intracavitary amphotericin B following removal of the central infection material was reported to be effective.⁶⁶ Voriconazole can be considered in *Pseudallescheria boydii* cases. Pyrimethamine and sulfadiazine can be used in *Toxoplasma gondii* cases, with HAART combination therapy in HIV positive cases. The duration of antimicrobial treatment should be from 6 to 8 weeks. The treatment should be prolonged considering late presentation, encapsulated abscess, multiloculated abscess and immune status of the patient.⁶²

Timely diagnosis and prompt treatment reduces mortality from 40% to 10%.⁶⁷ Successful management of brain abscess patients require an integrated approach with collaboration of healthcare professionals.⁶⁸

CONCLUSION

High degree of suspicion and awareness of associated risk factors and symptoms are required for brain abscess treatment. Clinicians should be aware that sinusitis, which is supposed to be minor illness, could develop into an extensive and serious disease. The knowledge of significance of emissary veins would give insight of potential development

of abscesses involving all layers from the scalp to brain parenchyma. The management of the extensive involvement of scalp, epidural, subdural and brain abscesses should include a combination antimicrobial therapy and neurosurgical intervention. Prompt diagnosis and treatment can lead to satisfactory recovery, as illustrated in this case.

CONFLICT OF INTEREST

There is no conflict of interest related to the materials or methods used in this study.

AUTHORS' CONTRIBUTIONS

MFC and SY drafted the manuscript and did literature review. WHC contributed to data collection. KHC made critical revision of the manuscript. All authors read and approved the final manuscript.

FUNDING

This article received no funding from any public, commercial, or not-for-profit organization.

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