

Intracranial Subdural Empyema – A Mini Review

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Abstract

Intracranial subdural empyema is a rare but devastating infection of the brain. With improvements in investigations and treatment, the previously seen mortality of 100% has now been reduced to 4-9%. The underlying factor in this improvement is the reduction in time to treat. To achieve this, the first barrier is identifying the possibility of the condition. There have been multiple case reports, series, and retrospective studies on the matter, but over time the clinical epidemiology has changed. In this report, we give a up to date review of the infection and highlight the important features clinicians should consider when assessing patients with possible intracranial subdural empyema.

Introduction

Intracranial subdural empyema (ISE) is a rare but serious infection of the brain with the purulent collection located within the space between dura and arachnoid mater. Prior to the advent of antibiotics, morbidity, and mortality affected near 100% of patients within 24-48 hours of presentation¹⁻⁷. The combination of improvement of investigations and management strategies have decreased mortality to 4-9%⁶⁻⁹. This is largely credited to decreased time to diagnosis and a subsequent reduction in the time to treatment^{7,10,11}. The aim of this report is to summarize the key clinical features of ISE and assist in management for clinicians faced with this challenging condition.

Epidemiology

ISE can occur in any age group, gender or population^{3,6,8,12-15}. Studies have shown two distinct populations at risk of ISE. The first group are children and young adults (<20 years of age) with males being at a higher risk^{2-4,6,8,15-17}. This demographic has a strong association with sinusitis and otogenic infections (41-78% of cases) highlighting the likely pathogenic roles of these diseases to ISE^{6,13,15-18}. Other, but less frequently mentioned causes included meningitis, neurosurgical procedures, and head trauma^{15,19,20}. The relationship of the younger population, sinus/otogenic infections and ISE can be explained by a combination of anatomical and behavioral risk factors. The younger male population possesses a relatively higher risk to develop sinusitis, meningitis, otitis media and undergo trauma compared to their older and female counterparts^{4,17,21}. Some authors deduct this to be a possible consequence of the male population's greater risk of neglecting infections and higher risk taking behaviour^{3,6,22}. Anatomical differences in the greater vascularity of the diploic veins in the younger male may also increase the risk of septic embolism and subsequent ISE compared to the older and female counterparts¹².

The second group are older with dissimilar associated risk and again a slight male predisposition^{2-4,6,8,9,11,17,18,20,23}. In this group patients tend to be older (age 40-50) and associated more with neurosurgical procedures compared to diseases of the ear, nose and throat^{2-6,8,17,20}. This group was likely under-recognized secondary to the volume of younger patients presenting with ISE. When isolating the population statistics of trauma/surgery associated ISE in previous studies the older population also becomes more readily evident^{4,6}. As the general population grows older, and more patients survive neurosurgical procedures there may be a shift in the epidemiology of patients with ISE^{2-5,9,11,18,20,23,24}

Other risk factors for developing ISE include smoking, diabetes, low socioeconomic status, and low health literacy²⁵⁻²⁷. However, the weight of these risk factors is yet to be established. Regardless, understanding the difference between the two population groups are important as it helps identify and assess them both uniquely.

Pathogenesis and Clinical Presentation

Entrance of pathogens into the intracranial compartment can occur through multiple pathways. One such pathway is haematogenic from emboli via intracranial vessels such as diploe veins or endolymphatic channels^{3,28,29}. As explained above, due to the anatomical differences, the younger male patient may be at a higher risk developing ISE from this pathway. The second pathway is from the suppurative process within the sinus and ear. The progressive infection causes osteomyelitis and direct invasion into the intracranial compartment^{2,3,22}. This pathway is evident with the common location of the ISE being placed in the temporal lobe and cerebellum with identifiable areas of bone and dural defects adjacent to the ISE^{2,3,8,21}. The third is direct inoculation, as seen in ISE associated with trauma and neurosurgical procedures.

The symptoms experienced are secondary to the presence of the infective collection causing mass effect to adjacent brain structures with associated inflammation. Within the subdural compartment, due to lack of physical constraints, the empyema is relatively free to spread over a larger area. The infection then poses a significant risk of venous stasis, venous thrombosis, cerebritis, and stroke over a large area of brain^{11,13,30}. This can lead to rapid deterioration with symptoms presenting as early as 1 day or late as 6 weeks, with mean time of 15 days prior to clinical presentation^{1,5,8,13,31}. Patients commonly experience headaches (77-83%), fevers (72-96%), altered sensorium (56-67%), vomiting (50%) and seizures (29-56%)^{4-6,8,16,17,20,29}. The clinical triad commonly described of fevers, headaches and vomiting is only present in half of patients^{1,3,5,29}.

Depending on the location of the disease, mass effect

can lead to symptoms including papilledema, cranial nerve palsy and hemiparesis (48-96%)^{4-6,8,15-17,20,29}. In contrast to the subdural empyema, epidural abscesses are walled off by strong dural adhesions and therefore tend to present later and are associated less with fevers, loss of consciousness and seizures²⁰.

The pathogenesis and clinical presentation of precursor infections and associated conditions is outside the scope of this report. It is however, important to understand that the risk of developing ISE from sinusitis and otogenic infections remains significantly low < 0.1%^{2,3,22,28}. Regardless, in cases of sinus and otogenic complications cranial imaging and thorough examination of ear, nose and throat should be carried out to exclude concurrent intracranial infections^{32,33}.

Investigations

Laboratory analysis of blood tests are non-specific and can show raised white cell count, C-reactive protein and erythrocyte sedimentation rate^{4,9,18,34}. Blood cultures, although recommended, can have poor yield as low as 5%. This is likely a consequence of the blood brain barrier and significant localization of infection within the intracranial compartment^{5,8}. As a result, it is recommended that intra-operative sampling and cultures are performed in all surgeries. These too can render a negative result and fail to yield growth in 7-53% of cases but, if not acquired might preclude vital pathogenic information. Failure of such growth can be secondary to sterile empyema from early administration of antibiotics or improper use of culture medium^{6,16,33}. Invasive procedures such as lumbar puncture (LP) are not recommended as previous reports have shown significant risk of neurological deterioration and herniation^{6,17,32}. When LP is indeed done, cerebrospinal fluid (CSF) gram stains, cultures, and cell counts indicate inflammation but lack any specific pattern for ISE^{14,15,17,32,35}.

Diagnosis is confirmed radiologically with computed tomography (CT) scans being the initial recommended modality as they provide a prompt assessment. If an ISE is evident on CT scans there is usually appreciable visualization of the collection including associated brain injury, bony destruction and adjacent sinus and otogenic pathology. The empyema appears as a thin, hypodense subdural lesion with linear enhancement of the medial surface. The mass effect that is present is usually disproportionate to the volume of the abscess as the cerebritis and cerebral ischemia contribute greatly to oedema and subsequent mass effect than the volume of abscess itself³⁴. Unfortunately, the CT scans can be normal in 63% of cases^{4,31,36}. The false negative scans can be a consequence of early symptomology without appreciable evidence of change. In these cases, a routine repeats CT scan or magnetic resonance imaging (MRI) is warranted. MRI has greater sensitivity (93%) and is therefore the

gold standard in all patients with suspicion of ISE³¹. It provides accurate information regarding the extent of infection as more than half of patients will have more than one intracranial complication at time of diagnosis^{3,22,31}. MRI appearances show similar patterns as shown in CT scans. The empyema is represented as a T2 hyperintense collection in the subdural compartment with associated T1 hypointensity. Addition of gadolinium highlights the surrounding membrane of the empyema and diffusion weighted imaging typically demonstrates restricted diffusion³⁷. Simultaneously, a magnetic resonance venography (MRV) can be done to assess for venous sinus thrombosis. The extent of cerebral oedema may also be more apparent on the MRI^{15,38}.

Management

Like many infections of the central nervous system, implementation of antibiotics should not be delayed and patients should be promptly treated with broad spectrum antibiotics in accordance to local hospital guidelines^{2,8}. Prior to the advent of antibiotics mortality reached near 100% within 24-48 hours of presentation even after surgical drainage¹⁻⁷. Introduction to antibiotics have decreased mortality and reduced the rates of complications in ISE to 15-41%²⁻⁴. Further improvements of investigations, such as CT scans, has reduced this lower to 4-15% which has been credited to assisting in early diagnosis^{1,7,8}.

Infections are commonly polymicrobial. *Streptococcus milleri* group is over-presented in cultures but other streptococcus species, staphylococcus species and anaerobic gram positive and negative species are also present and have been cultured^{14,32,35,39,40}. In certain population groups, fungal empyema are also a possibility²¹. In Australia, most departments would use a combination of a third-generation cephalosporin with metronidazole and vancomycin. This would provide broad spectrum cover for most pathogens including aerobic and anaerobic organisms⁴¹. In tropical areas such as our department in the Northern Territory, Australia, the prevalence of fungal infections and melioidosis has prompted the use of meropenem, amphotericin and vancomycin. In cases of negative cultures, continuation of empiric antibiotics may be required which is typically a period of 6 weeks, with a minimum of 2 weeks of intravenous antibiotics^{8,29,35,41}.

Other medical management strategies include implementation of prophylactic anticonvulsants and medical therapy to maintain normal intracranial pressure^{1,15,29}. This could include but not exclusive to elevation of head of bed, normocapnia and normotensive parameters^{1,41}. Medical management alone however is infrequently adequate in the management of ISE^{15,35}. It has been successful in small number of cases and may be suitable in minor collections with the option to transition

to surgery in the event of early therapy failure¹⁰. Many surgeons will attest the mainstay of treatment of any abscess is and remains as surgical drainage. The drainage of the collection provides a substantial step towards source control and accelerates recovery. Depending on factors including collection characteristics, surgical expertise and patient's clinical status, the surgical procedure can be in the form of burr-holes or craniotomy^{35,41-43}. If the abscess is easily accessible, thin and non loculated, then burr-holes may be an option. However, patients treated with burr-holes have an increased risk of requiring additional procedures and/or conversion to craniotomy^{15,35}. Our department approaches ISE more aggressively and we believe a craniotomy is warranted in most if not all cases. Craniotomy has the benefit of providing the option of assessment of loculations, removal of membranes, repair of dural breach as well as an adequate washout in the subdural compartment.

Intraoperatively, decision to remove the bone flap should be done by case to case basis. If there is contamination of bone with evident osteomyelitis, we would recommend closure without bone. This is the same if there are signs of raised intracranial pressure (ICP)^{29,43}. In cases of concurrent sinus or otogenic infections we would recommend definitive treatment of underlying pathology preferable at the same time as the ISE treatment³.

Conclusion

Intracranial subdural empyema is an uncommon intracranial infection. Like other cranial infections, the morbidity and mortality are significantly high, but this can be reduced with prompt diagnosis and subsequent implementation of antibiotics and surgical drainage. The time to diagnosis is dependent on a high degree of suspicion and a good understanding of the clinical risk factors associated with ISE. Over the years the epidemiology and risk profile for our patients have shifted and understanding the reason behind this can help the clinician's approach their patients on a case by case basis and improve patient outcomes.

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