MENINGITIS AND ENCEPHALITIS

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OVERVIEW
This chapter will cover the following 4 topics:
1. Bacterial Meningitis
2. Herpes Encephalitis
3. West Nile Virus Encephalitis
4. Ventriculostomy and Neurosurgical associated infections
5. Bacterial Central Nervous System Space Occupying lesions

BACTERIAL MENINGITIS

EPIDEMIOLOGY
There has been a significant reduction up to 94% in the number of cases of Haemophilus influenzae in the United States since the introduction of the Hib vaccine in 1990[1]. This vaccine is primarily given to children, as children are the primary demographic group that acquired Haemophilus influenzae meningitis (or H.flu) worldwide. Because of the significant reduction of H.flu meningitis in the US and the industrialized world, what was once the primary cause of bacterial meningitis is now relegated to a much lower incidence and Streptococcus pneumoniae (or pneumococcus) is now the most common cause of meningitis in the United States. It too is decreasing, secondary to the multivalent pneumococcal vaccine, but it has the highest case fatality rate at approximately 20%[1].

Group B Streptococcus is the most common cause of meningitis in newborns, Neisseria meningitides is the most common cause of meningitis in children, teens and young adults and pneumococcus is the most common cause of meningitis in adults.

CLINICAL FEATURES
The presenting signs and symptoms of meningitis are: headache, fever, stiff neck, and altered sensorium, presenting in over 80% of patients. The classic signs of meningitis, such as Kernig’s sign and Brudzinski’s sign, are less common at only about 50%. Although bacterial meningitis, by definition, is an inflammation of the meninges, in almost all cases of bacterial meningitis, there is associated inflammation of the underlying brain tissue. This can involve cortical as well as brainstem structures. Because these structures can be affected with the inflammatory process, and because of several other issues such as; secondary venous sinus thrombosis, vasculitis or cranial nerve inflammation, focal neurologic deficits are seen in about 20% of patients upon presentation. Papilledema upon presentation is uncommon (<1%).
DIAGNOSIS

The diagnosis of bacterial meningitis is primarily made based on both the clinical presentation and the gold standard: CSF examination. With regard to the results of the CSF evaluation, almost all patients will have an increased opening pressure (i.e., opening pressures greater than 15 cm of H₂O). In the analysis of CSF WBC, ten percent of the patients will have lymphocytic predominance, 25% will have a normal CSF white blood cell counts, the remainder will have elevated white blood cell count with predominance of neutrophils. In bacterial meningitis glucose is almost always low, although the CSF:serum ratio is more accurate. In bacterial meningitis this ratio should be CSF:serum is < 0.3. When trying to differentiate bacterial meningitis from viral meningitis there is a 99% certainty that the patient has bacterial meningitis (as long as the clinical scenario supports this), if the glucose is < 34 mg/dl, if the serum CSF to erum glucose is <0.23, if the protein is 20 mg/dl, if the white blood cell count is greater than 2000/mm³, or if there are greater than 1180 neutrophils/mm³[2]. C reactive protein, either in the serum or the CSF, or procalcitonin, and have also been demonstrated to be very specific in diagnosing bacterial meningitis vs viral meningitis. Table 2 demonstrates typical findings in the CSF of patients with bacterial meningitis.

Although a CT scan of the brain is typically not required and not helpful in the diagnosis of bacterial meningitis it is useful in excluding other diagnoses. In particular it is useful for excluding mass occupying lesions that might complicate the lumbar puncture done for diagnosis. The literature supporting the necessity of doing a CT scan of the brain prior to LP is minimal. There have been several large consecutive studies of patients with meningitis that suggests that somewhere between 1 and 2% of patients with meningitis develop herniation syndromes[3, 4]. A much smaller percentage of these patients herniate within several hours of the lumbar puncture. Conversely there are studies demonstrating that normal CT scans do not eliminate the risk for herniation nor do abnormal CT scans guarantee herniation after LP[5]. In 2004 Tunkel and colleagues[6] published the Practice Guidelines for the Management of Bacterial Meningitis. In the these guidelines the authors recommend CT prior to LP in patients with suspected bacterial meningitis who have or present with a) a history of central nervous system disease, b) those patients that are immunocompromised, c) patients with a seizure at onset of this particular disease process, d) patients with papilledema, e)those with abnormal level of consciousness and f) those with a focal neurologic deficit.

TREATMENT

Treatment of bacterial meningitis should not be delayed while waiting for a CT scan of the brain. Empiric treatment of the patient should be provided based on the patient’s relative risk for specific organisms (Table 3). Most nonimmunocompromised adults are at highest risk in community acquired meningitis for *Streptococcus pneumoniae*. Those older than 50 are also at risk for *Listeria*. Antibiotics should be administered base on this epidemiology. Care should also be given with regard to the risk for resistant organisms. Drug resistant pneumococcus is becoming more and more prevalent in the community (over 15%) and so Vancomycin should be used as an empiric treatment in the appropriate patient population.
Once the gram stain is obtained and organisms identified by morphology and staining characteristics the antibiotics can be modified to be more specific (Table 4). Finally once the organism has been identified and the sensitivities have been obtained the antibiotics can be changed once again if necessary. Antibiotic treatment durations are also based on the organism found. Both meningococcus and H. flu can be treated successfully for 7 days. Strep. pneumonia requires 2 weeks of treatment while Listeria and other gram negative organisms require at least 3 weeks of treatment.

Adjunctive treatment for bacterial meningitis with corticosteroids has been controversial over the last 2 decades. This has changed since the publication of a randomized controlled trial of Dexamethasone in bacterial meningitis[7]. This trial enrolled 301 patients with the treatment group receiving 10 mg of Dexamethasone every 6 hours for 4 days. It is important to note that the first dose was given prior to antibiotic administration. The trial demonstrated significant improvement in outcome in those patients receiving Dexamethasone. This improvement in outcome and decrease in mortality was almost exclusively in the group of patients that were identified as having pneumococcus. Therefore the current recommendations are to administer Dexamethasone 10 mg every 6 hours for 4 days with the first dose of the corticosteroid prior to antibiotic administration[6]. Finally, with regard to the treatment of bacterial meningitis it is important to note that good supportive intensive care medicine is important in these patients. While no randomized clinical trial has been published with regards to treatment of these patient’s increased intracranial pressure there is clear evidence that this problem exists in this patient population. It is unclear whether monitoring of intracranial pressure or treatment with hypertonic agents such as Mannitol or hypertonic saline is useful in this patient population.

**HERPES SIMPLEX ENCEPHALITIS**

**EPIDEMIOLOGY**

*Herpes simplex* viruses (HSV) are distributed world wide and humans are the sole reservoir of this virus. In fact, between 70-95% of humans are seropositive for *Herpes simplex* virus by adulthood. *Herpes simplex* encephalitis (HSE) is the most cause of sporadic fatal encephalitis in the United States. It is estimated to occur in 1/250,000-1/500,000 in the United States or approximately 250-500 cases per year. There is no seasonal age, sex or racial variation with regards to the patient population that developed HSE. Thirty percent of the patients are under 20 years old and 50% are older than 50.

**PATHOPHYSIOLOGY**

It is not clear whether herpes simple encephalitis represents a primary or recurrent infection. Certainly the virus does lie dormant in neurons and ganglia once the patient has been infected, however, it has been demonstrated in the past that patients with HSE and concomitant cutaneous *Herpes simplex* may have two different strains of HSV. There have been no triggering factors identified.
Immunosuppression does not predispose patients to HSE however patients who are immunosuppressed are at risk for a more aggressive disease with a worse outcome.

**CLINICAL FEATURES**

The common clinical manifestations of HSE are a change in personality, altered mentation and decreased level of consciousness. Because *Herpes simplex* encephalitis always affects the cortical structures symptomatology is therefore based on cortical lesions. Fever, as this is an infection, is common. Focal neurologic findings may represent any cortical abnormality including aphasia or neglect. Findings that are not cortically specific, but represent cortical dysfunction such as dysphagia and hemiparesis, are also seen. Headache, papilledema, nausea and vomiting as well as focal and generalized seizures (seen in two thirds of patients are also common).

**DIAGNOSIS**

The diagnosis of *Herpes simplex* encephalitis relies on the clinical course as well as adjunctive laboratory evaluation. The common laboratory evaluations are lumbar puncture, EEG, brain imagining with CT and MRI, brain biopsy and PCR of cerebral spinal fluid. The CSF white blood cell count in patients with *Herpes simplex* encephalitis typically ranges from the 10s to the 100s with values up to 1000-2000 cell/mm$^3$. Although the differential of these white blood cells is predominantly and most frequently lymphocytes, 10-25% of patients early in their course can have high numbers of neutrophils in their CSF. The CSF in HSE also frequently contains red blood cells. About 50% of patients will have red cells in the range of 10s to 1000s/mm$^3$. Xanthochromia will also be seen in these patients. While CSF red count is not pathognomonic for *Herpes simplex* simplex encephalitis, it is the only common infectious encephalitis in which red cells are frequently seen. CSF protein may be mild to moderately elevated in approximately 50% of patients. Glucose is usually normal but may be slightly reduced in a small number of patients. Opening pressure can be elevated in approximately one third of patients.

Historically the EEG was one of the diagnostic tests of choice. However, electroencephalograms though sensitive (84%) in HSE, are very nonspecific (32% specificity). The characteristic EEG demonstrates spike and slow activity with PLEDs. As mentioned above, this is not specific and can be seen in many other acute brain injuries.

CT scan rarely may show hypodensities in the region of the temporal lobe or frontal lobes in a severe case of herpes encephalitis. The MRI is well regarded as a test for diagnosing herpes encephalitis. It is common to see hyperintensities on T2 and gadolinium enhancement around the lesion in the lobe that is infected. As the temporal lobe is the most common cortical structure to be infected by herpes this is frequently well delineated with gadolinium enhancement.
Brain biopsy is of historical interest but is rarely done at this time. It, of course, is the gold standard with 99% sensitivity and 100% specificity. But, because of the risks of the procedure, as well as other tests with similar sensitivity and specificity, this is infrequently done now.

The polymerase chain reaction (PCR) is both highly sensitive and specific. Its sensitivity is 96% with a specificity of 99%[8]. Very early in the disease (<24 hours) the test may be negative. With treatment, the sensitivity may also decrease. The CSF PCR for herpes simplex is now the test of choice for diagnosis.

**TREATMENT**

The treatment of *Herpes simplex* encephalitis is both supportive and pharmacologic. Acyclovir is the drug of choice and the dose is 10 mg/kg every 8 hours for 21 days. Higher doses and longer duration may help prevent relapse. The prognosis of HSE though significantly improved since the introduction on Acyclovir is still unsatisfactory. The mortality in Acyclovir treated patients is 19% at 6 months and 28% at 18 months[9]. This does compare favorably with a mortality of 70% in placebo treated patients but definitely represents a current high mortality rate. Morbidity is quite high in survivors of herpes encephalitis. Predictors of poor outcome include those patients that are greater than 30 years old and those that were comatose or semi-comatose on presentation with a GCS of <6. In these patients the mortality rate with treatment is 70%.

**WEST NILE VIRAL ENCEPHALITIS**

**CHARACTERISTICS OF THE WEST NILE VIRUS**

The West Nile Virus is a single stranded RNA virus which belongs to the family Flavivirus. This family of viruses includes the Japanese encephalitis virus, the St. Louis encephalitis virus and the Kunjin virus. There is serologic cross reactivity with all of these viruses. This virus was thought to have originated in the West Nile Valley of Uganda. Genetic linkage of the West Nile virus epidemic in the United States suggests that this stereotype of the virus comes from the Middle East. Clinically only the US and Israeli West Nile virus infections have caused severe human disease and bird deaths.

**EPIDEMIOLOGY**

The West Nile virus infection is now considered epidemic in the United States. Between 1999 and 2006 there have been a total of 26,741 cases and 982 deaths (3.5% mortality). Of these cases, 9,814 have been West Nile virus encephalitis (approximately 35% of the total). There is a 10% mortality in those patients who develop West Nile virus encephalitis. West Nile viral infections clinically present as a spectrum, with encephalitis being the most the severe version. The West Nile virus encephalitis increases significantly in those patients older than 50 years old. There is a 10x higher risk of meningitis or encephalitis in those patients that are 50-59 years old and a 43x higher risk of encephalitis in those patients who were greater than 80 years.
In patients who are immunocompromised secondary to malignancy, transplant or HIV there is also an increased risk of encephalitis.

**CLINICAL PRESENTATION**

West Nile virus infection *without* encephalitis is typically either asymptomatic or a “flu-like illness” with fever, malaise, myalgias and headache. In those patients who develop the West Nile virus encephalitis (WNVE), fever, fatigue, nausea, vomiting, headache and altered mental status are common. Additionally up to 48% may develop objective weakness[10]. This weakness has been demonstrated in some patients to represent damage to the spinal cord; more specifically the anterior horn cells[11]. The clinical presentation in these patients may be single or multiple limb weakness; additionally these patients may have severely compromised respiratory function or frank respiratory failure. A subset of WNVE patients have also presented with new and acute onset movement disorders such as parkinsonism due to the involvement of the [12]basal ganglia.

**DIAGNOSIS**

West Nile virus is most accurately diagnosed by evaluating for IgM antibodies in the serum or CSF. Since IgM does not cross the blood brain barrier, the finding of IgM antibodies specific for West Nile virus in the CSF suggests an acute infection with the virus. Although vaccines to Yellow Fever and Japanese encephalitis are cross reactive with the antibodies to West Nile virus one would expect IgG rather than IgM. Other diagnostic modalities, such as screening blood values, are typically unhelpful and nonspecific. Serum white cells are either normal or slightly elevated, although at times it has been reported there is a lymphopenia. There may be a serum hyponatremia in those patients who develop encephalitis. CSF analysis is nonspecific with up to 2000 white cells, predominantly lymphocytes, a mild to moderately elevated protein and normal glucose. Imaging studies are helpful but nonspecific. CTs are typically normal though one third of patients have been seen to have enhancement of the meninges or periventricular areas on MRI. There are also abnormalities seen on MRI such as those involving the cortex, brain stem, basal ganglia or spinal cord[13, 14].

**PROGNOSIS**

The majority patients who develop West Nile virus encephalitis have sequelae. Up to 50% of patients do not return to premorbid function. In those that develop motor neuron damage up to two thirds of those have continued weakness.

**INFECTIONS AFTER NEUROSURGICAL PROCEDURES**

**BACKGROUND**

Infections after neurosurgical procedures, either intracranial monitoring devices or drains or after craniotomy, are rare.
However, bacterial infections within the ventricles, in the subarachnoid space, or of the brain parenchyma itself, can be devastating. With regards to intracranial pressure monitoring and drainage devices, the external ventricular drain or ventriculostomy was developed in the early 1950’s. During the 1960’s Lundberg published extensively on his ventriculostomy drainage system in Lund Sweden[15]. He reported a 6% positive CSF culture rate with these devices and 0% of those patients had clinical infection. Remarkably, these devices were in place for up to 8 weeks.

**External Ventricular Drains**

In general, the epidemiology of infections of external ventricular drain (EVD) devices has been reported in the literature anywhere from to 0 – 22%. There are certainly some outliers in these varied, mostly retrospective, reports. More commonly the infection rates are reported to be 5 – 10%.

In a review of the literature on ventriculostomy related infections by Lozier, et al[16], reported a combined infection rate of between 8 and 9%. One of the difficulties in assessing this data is the variety of definitions of ventriculostomy associated infection. In the same Lozier paper he outlines a description of a variety of possible scenarios based on the certainty of infectious ventriculitis. He describes these as ventriculostomy-related infection, suspective ventriculostomy-related infection, ventriculitis, ventriculostomy colonization and contamination. None of the papers that he reviewed were so stringent in their definitions. For example, based on Lozier’s definitions, a positive culture from cerebral spinal fluid drawn via the EVD does not automatically result in a definitive diagnosis of infection. If the other parameters in evaluating the CSF are normal, such as white count and protein, then a positive culture may represent colonization or contamination. This uncertainty in what the definition of a diagnosis of a ventriculostomy associated infection represents, makes interpreting the literature and interpreting individual clinical scenarios difficult.

Most studies suggest that gram negative bacilli and gram positive cocci the most common species infecting ventricular drains. While this is certainly supported by the literature, the relative ratios of those bacterial species found in the CSF of patients with ventriculostomies can be quite different depending on the study and the hospital[17, 18].

In Lozier’s review of the literature, the risk factors for cerebral fluid infection in patients who underwent ventriculostomy include: intraventricular hemorrhage, subarachnoid hemorrhage, operative depressed cranial fracture, basilar cranial fracture with CSF leak, neurosurgical operation, ventriculostomy irrigation, systemic infection, and duration of catheterization.

**Intraparenchymal Pressure Monitors**

The infection rate from intracranial pressure monitors (“bolts”) is typically quoted as lower, in the 0 – 4% range, than ventriculostomy associated infection. This is likely due to a “set it and forget it” use of these devices.
While ventriculostomies may be sampled and drugs injected into them (this increased the infection rate) intraparenchymal monitors are typically inserted with no subsequent manipulation. However, there are many fewer studies assessing the risk of these devices. A single evaluation of Lumbar drain infection reported less than a 3% infection rate[19].

**Craniotomy Infections**

Meningitis or ventriculitis in the antibiotic era has been reported in the 1 – 9 % range an averaging approximately in 4% of craniotomies[20, 21]. Again the bacteria associated with these infections are typically skin flora such as Staph. and Strep. as well as gram negative bacilli. Risk factors for developing a craniotomy infection include the duration of surgery, re-operation during the same admission, bleeding complications, CSF leaks and the surgeon performing the craniotomy.

**DIAGNOSIS**

As described above in Lozier’s review, the diagnosis of ventriculostomy or craniotomy associated infections is quite difficult. For instance, Lozier and colleagues suggested that a true ventriculostomy related infection have the following characteristics: progressively declining CSF glucose level, increasing CSF protein profiles, advancing CSF pleocytosis, one or more positive CSF cultures or gram stains, and a paucity of clinical symptoms other than fever. The definition of ventriculitis includes similar laboratory characteristics except a gram stain or culture is not necessary and ventriculitis does have clinical signs of meningitis.

Other methods for characterizing a true infection include low CSF to serum glucose ratio. A ratio of .4 gives a sensitivity of 77% and a specificity of 87%[22]. Other methods include CSF lactate, cytokine analysis and cell index studies. None of them have demonstrated significant sensitivity and specificity in large cohorts.

At many centers CSF is routinely drawn from the EVD to help diagnosis and predict the onset of infection. This has been investigated in retrospective studies and typically the routine analysis of CSF to help predict infection has proven to be unhelpful[23, 24] and cannot predict infection better than other clinical and laboratory values such as clinical signs, fever and white count. Other studies have demonstrated that frequent accessing of the EVD may promote infection.

**PREVENTION**

Many centers use antibiotic prophylaxis to help prevent both EVD, intraparenchymal and post craniotomy infections. There is a decent retrospective literature looking at this however, no firm conclusions can be made with a variety of outcomes in the retrospective literature. One study [25] was a randomized controlled trial looking at both continuous prophylactic versus periprocedure use of ampicylin/sulbactam plus aztreonam that demonstrated a significant reduction in infection using antibiotic continous prophylaxis. However, the mortality rate in the patients who did get infected, was higher in the continuous prophylaxis group.
This study also changed the catheter every 5 days. Antibiotic prophylaxis for craniotomies demonstrates in both a randomized controlled trial and metanalysis suggests that there is benefit, although there are some large perspective none randomized controlled trials which demonstrate no benefit.

With regard to changing the monitor or EVD on a routine basis, retrospective data suggests that between days 5 and 9 there is an increased risk of infection, however, in one randomized controlled trial of 103 patients in which the catheters were changed every 5 days there was no significant difference between those that were changed and those that were not[26]. There is also conflicting evidence with regards to the relationship between the duration of catheter insertion and risk for infection. There have been 10 studies comprising 2046 EVD’s in 1698 patients which reported a positive association between duration and infection. Conversely there have been 7 studies comprising 2199 EVD’s in 2113 patients reporting no association.

TREATMENT

In patients in which there is clear evidence of infection, treatment should begin immediately. If cultures are available, the antibiotics should be directed at the appropriate organisms. Without available cultures or gram stains, antibiotic choice should be directed at both gram negative bacilli (including pseudomonas) as well as both streptococcus and staphylococcus (including Staph. aureus).

BACTERIAL CENTRAL NERVOUS SYSTEM SPACE OCCUPYING LESIONS

BACKGROUND

Examples of space occupying lesions of the central nervous system include parenchyma abscesses, epidural abscesses, and empyemas. The most common sources of brain abscesses are from hematological spread (most common lung), direct extension from para-meningeal source, head trauma or surgery, or congenital heart disease (PFO, PDA, etc). Rare causes of brain abscess have been reported to occur with tongue piercing and endovascularly treated aneurysm using Guglielmi detachable coils [27].

Clinical Presentation of Bacterial CNS Space Occupying lesions

Parenchymal brain abscess present with a myriad of symptoms. The triad of fever, headache and focal neurological signs is helpful only occurs in 50% of patients. Other manifestations including symptoms and signs of the original infection (otitis or sinusitis) may be present and more impressive. Although the average course of brain abscess from the time of symptom presentation to hospital admission can be as short as 5 days[28], the course can be much more indolent. In subdural empyema there is also the classical triad (sinusitis, fever, and neurologic deficit) but the signs of cortical inflammation such as focal deficits (75%), seizures (50%) and raised intracranial pressures such as headache, vomiting, and papilledema (50%) are also present.
Spinal epidural abscess is a neurosurgical emergency and back pain, malaise and fever are the classic presenting symptoms but as with the other infectious processes, evolution can be rapid or more indolent.

**Diagnosis of space occupying lesions**

Fever with or without a peripheral white count and symptoms of new CNS deficit suggest an intracranial infection. Other signs and symptoms such as headache, vomiting and papilledema are not specific to these diagnoses. Cranial imaging with a CT or MRI are sensitive tests for these syndromes, with MRI able to provide more information and better resolution[29]. Evidence suggests that MRI with diffusion weighted imaging may be more specific for the diagnosis of intraparenchymal abscess, demonstrating high signal on DWI and low apparent diffusion coefficient (ADC) in the abscess cavity[30, 31]. In subdural empyema, there may be a disproportionate amount of underlying cortical and white matter edema and enhancement with respect to the size of the fluid collection. In patients with spinal epidural abscess there may evidence of discitis and vertebral body infection which is difficult to conclusively diagnose with imaging. Lumbar puncture should not be performed in those with brain abscesses given the risk of herniation. Likewise, lumbar puncture is contraindicated in spinal epidural abscess if there is a suggestion of involvement in the lumber area (low back pain, tenderness to palpation). Performing an LP through an abscess may introduce organisms into the CSF.

**Treatment of space occupying lesions**

Treatment of brain abscess is both surgical and medical. Abscesses greater than 2.5 cm in diameter or those associated with mass effect require CT guided aspiration or surgical excision. Brain imaging may be followed with repeat scans every 1 to 2 weeks to monitor for re-accumulation.

In patients not considered surgical candidates, such as those with associated ependymitis or meningitis, hydrocephalus requiring shunting, or those with inaccessible abscesses, medical treatment alone can be attempted. Broad-spectrum antibiotics are used with, initially, frequent brain imaging. For example, starting with weekly imaging then this can be spaced out to every 2 weeks during the remainder of the 6-8 week antibiotic course, with follow-up scans every 2 to 4 months for the following year to assess for recurrences. Empiric drug regimens for immunocompetent patients should include coverage for methicillin resistant Staphylococcus aureus, anaerobes and gram negative bacilli (including Pseudomonas). For example a regimen such as vancomycin, metronidazole, and cefotaxime initially, then more sensitivity specific antibiotics, typically for 6 to 8 weeks. Even if no anaerobic organisms are cultured, the continuation of anaerobic coverage is recommended as it is difficult to culture these bacteria.
The treatment of subdural empyema is primarily neurosurgical. Multiple surgical approaches exist from craniotomy to burr holes with evacuation. Early treatment may be beneficial in that treatment within 72 hours of symptom onset resulted in less than 10% disability among patients, whereas, 70% of patients died or were disabled if the treatment was prolonged beyond 72 hours [32]. Broad spectrum empiric antibiotics (similar to described above for brain abscess) should be started early. Once bacterial identification is made, antibiotics can be tailored to the organism and continued for 3-4 weeks after drainage.

As mentioned above, spinal epidural abscess is a neurosurgical emergency. Without treatment, severe and permanent spinal cord damage can occur in just a few hours. Treatment includes surgical decompression and drainage, a search for the infectious source (if possible) and antibiotics (empiric as described above, then specific antibiotics based on the pathogens).

**Prognosis**

Reported mortality of patients with brain abscess varies from 20 – 30% [33]. The most pertinent predictive factors in these recent retrospective series were clinical presentation on admission (comatose, poor mental status) and distant metastatic focus of infection. Factors such as age, multiple abscesses, conservative management, and steroid use appeared to have no affect on outcomes. The prognosis of spinal epidural abscess is dependant on how quickly the diagnosis is made and surgical decompression is achieved. Also, neurological outcome with respect to spinal cord disease is related to severity the of the spinal cord symptoms at the time of decompression. The more symptoms at the time of decompression, the worse the outcome, although compared to similar neurological presentations in traumatic spinal cord injury, those with epidural abscess tend to do better[34].

In a recent review of 45 cases of subdural empyema, Tewari et al, found the majority of the cases to be in infancy through the third decade (60%) [35]. In this series, mortality was 4.4% and good recovery was seen in 77.8%. In other series mortality in subdural empyema is in the 10-40% range. Mortality and outcome is also linked to neurological status at presentation. Additionally, since infectious sinus thrombophlebitis is almost always associated with subdural empyema, the extent of the disability and the risk of dying is dependant on the extent and severity of the sinus thrombosis.

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<td>Child to Adult (2-50 years)</td>
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<td>&gt;50 years or very ill</td>
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Note: Antibiotic choice needs to be narrowed once specific organisms are identified.
REFERENCES


