Spinal Epidural Abscess

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Abstract:

Spinal Epidural Abscess (SEA) is a rare but severe condition that results in mortality if it has not been diagnosed or is diagnosed in the later stages. The prognosis of SEA is important before neurological deficit develops. The diagnosis is difficult for SEA as the symptoms are common with other diseases. Most patients have symptoms of fever and sever back ache. Gadolinium-enhanced magnetic resonance imaging is the most accurate method of diagnosis. Surgical drainage together with systemic antibiotics is the treatment of choice for SEA. The consequence of SEA is greatly influenced by the severity and duration of neurological deficits prior to surgery. Increased awareness of the disease is necessary for successful treatment.

Introduction:

Spinal epidural abscess (SEA) is a severe infection of the epidural space. It is a rare but serious condition with significant morbidity and mortality [1]. It is the rapid accumulation of purulent material in the space between the dura matter and the Osseo-ligamentous confines of the vertebral canal may injure the spine by direct compression or local ischemia [2]. It is really important that SEA is diagnosed early before neurological deficits develops. Despite the advances of modern diagnostic and management methods the diagnosis of SEA is challenging because of its rarity and nonspecific presentation, such as back pain.

Epidemiology:

Incidence:

Although SEA is rare its incidence has doubled in the past two decades. In 1975, Baker et al. [3] reported an incidence of SEA ranging from 0.2 to 1.2 per 10 000 hospital admissions per year and a peak incidence in the sixth and seventh decades of life [4]. The current annual incidence is estimated to be 2.5–3 per 10 000 hospital admissions.

Risk Factors:

Risk factors such as diabetes mellitus, higher age or intravenous drug use, chronic renal failure, alcoholism and anesthetic interventions contribute to the incidence rate of SEA.

Pathophysiology:

Bacteria gain access to the epidural space by three ways:

i- per continuitatem from a neighbouring infected structure
ii- through haematogenous dissemination from a remote focal infection
iii- through iatrogenic inoculation [1].

Most Spinal Epidural Abscess result from haematogenous spread of bacteria usually from a cutaneous or mucosal source. In Haematogenous spread of infection the sources of bacteraemia included furuncles, pharyngitis, and dental abscesses [5] In such cases the abscesses are often located in the dorsal aspect of the spinal canal.

The direct spread of infection into the epidural space could be from a source adjacent to the spine like spondylitis or discitis. In such cases the abscesses are often located in the ventral aspect of the spinal canal and are thought to originate from the adjacent vertebral body.

Iatrogenic causes of SEA include all kinds of invasive procedures, such as surgery, lumbar puncture, peridural anesthesia, epidural analgesia and nerve blocks, and are estimated to be responsible for 15% of all cases [1]. These infections are usually due to the procedure itself or through microorganisms in the skin flora when a device is left there. On such catheters there is formation of a biofilm like that found on intravascular catheters. Another possible iatrogenic cause of SEA is the paraspinal injection of analgesics and steroids.

Neurological impairment due to spinal cord lesion may occur which is due to the direct cause of mechanical compression by the inflamed mass. Mechanical compression and vascular occlusion may occur at different phases of the disease and cause adverse effects. Usually neurological improvement is seen after surgical depression of the mass.

Clinical features:

The progression and symptoms of SEA can be best explained in the following stages:

Stage 1- Spinal pain at the level of abscess
Stage 2- Nerve root pain from the affected spine level
Stage 3- Motor weakness, sensory deficit, and bladder and bowel dysfunction
Stage 4- Paralysis [6]

Fever, spinal pain and tenderness, and radiating root

pain followed by limb weakness are the most common symptoms seen in patients with SEA. Spinal pain and fever are the first symptoms to appear. The duration of the symptoms before hospital admission and the rate of progression from one stage to another are highly variable because abscesses are more likely to develop in larger epidural spaces that contain infection-prone fat, they are more common in posterior than anterior areas and in thoracolumbar than cervical areas [6].

Pathogens:

Although SEA is caused by many organisms Staphylococcus aureus accounts for 60-90% of cases with methicillin-resistant S. aureus (MRSA) accounting for an increasing number. Less common causative pathogens include coagulase-negative staphylococci, such as S. epidermidis associated with spinal procedures and gram-negative bacteria, particularly Escherichia coli in patients with urinary tract infections and Pseudomonas aeruginosa especially in injection drug users. Other pathogens such as mycobacteria, including Mycobacterium tuberculosis, tend to target immunosuppressed patients, while staphylococcal species other than S. aureus and fungi such as Candida species are often associated with spinal instrumentation or injection [7].

Diagnosis:

In SEA, WBC count, CRP and erythrocyte sedimentation rate (ESR), are generally elevated. Leukocytosis is found in 60–80%, and an ESR >20 mm/h in up to 95% of cases. Lumbar puncture plays a less important role in diagnosing SEA, and should not be performed routinely. Neither Gram staining (generally negative), nor cultures of CSF (growth in 6–28%) reveal results with acceptable sensitivity [1].

MRI with gadolinium (Gd-MRI) detects SEA with great sensitivity. CT scans can also be utilized to detect SEA.

Treatment:

Retrospective studies show that surgical drainage together with systemic antibiotics is the treatment of choice for SEA. Because the preoperative neurologic stage is the most important predictor of the final neurologic outcome, and because the rate of progression of neurologic impairment is difficult to predict (with some patients becoming paralyzed within hours after the onset of neurologic deficit), decompressive laminectomy and debridement of infected tissues should be done as soon as possible [6].

Antibiotics should be used after laboratory procedures and tests are performed. To increase the sensitivity of microbiological results, medical treatment can be slightly delayed and given immediately after the invasive procedure. Intravenous therapy must include anti-staphylococcal activity, as well as antimicrobial activity against streptococci and Gram-negative bacilli [1].

Surgical treatment involves removal of the pus with decompression of the spinal cord and nerve roots.

Conclusion:

The outcome of SEA is often assessed based on mortality and recovery from neurological deficits [1]. Though the mortality rate has fallen from 80%-20% many patients still die because of this disease. Mortality is due to sepsis and occurs patients having many complications. Patients who diagnose SEA early have a higher chance of surviving and acquiring less neurological deficits. Repeated spinal and neurological examinations are essential in any patient who has an unknown source of infection and severe spinal pain.

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