INFECTIONS OF THE CNS
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Spinal Epidural Abscess; Risk factors- diabetes, iv drug abuse, chronic renal failure, alcoholism. Maybe postoperative complication of spinal surgery/manipulation. Can be (a) acute-less than 16 days, epidural pus, sepsis. (b) chronic-longer than 16 days to months. Associated with vertebral osteomyelitis.
Epidemiology; Earlier years (70's) about 0.2-1.2 per 10,000 hospital admissions but increasing. Common age 50-60's. 50% of cases thoracic., 35% lumbar, 15% cervical.
Presentation; local pain, tenderness, radicular symptoms, sphincter disturbances, limb weakness. Fever. Meningismus. Spinal cord presentation may be due to mechanical compression or vascular compromise. Differential diagnosis-meningitis, acute transverse myelitis (paralysis more rapid, radiographic studies normal), intervertebral disc herniation, spinal cord tumours, pseudomeningocele when postop.
Source of infection; (a) haematogenous-most common upto 50% from skin infections (furuncle), parenteral injections, bacterial endocarditis, UTI, otitis media, sinusitis, pneumonia, pharyngeal or dental abscess. (b). direct extension- decubitus ulcer, psoas abscess, penetrating trauma, pharyngeal infections, mediastinitis, pyelonephritis with perinephric abscess. (c). following spinal procedures lumbar dscectomy (0.5%), spinal epidural, lumbar puncture. (d) back trauma (e) idiopathic (50%) (f) associated conditions- apprx 60% of cases-diabetes mellitus, iv drug abuse, chronic renal failure, alcoholism, UTI, Pott's disease, HIV.
Diagnostic culture-staph aureus (50%) followed by aerobic and anaerobic streptococci, e coli, pseudomonous aerogenous, Diplococcus pneumoniae, Serratio marcenscens, Enterobacter, chronic infections (TB- associated with vertebral osteomyelitis, fungal-cryptococcus, aspergillosis, brucellosis. Parasitic-echinococcus), multiple organisms (10%), anaerobes (8%).
Diagnostic tests- leukocytosis WBC >15,000. ESR>30. LP-at site distant to clinical site to aspirate pus if possible-elevated csf protein and WBC glucose normal. Blood culture. HIV tests.
Radiographic studies; plain films-lytic lesions, demineralisation, scalloping of end plates in osteomyelitis. MRI-eliminates need for spinal tap, can r/o transverse myelitis and cord infarction, TIWI-hypo/isointense epidural mass, vertebral osteomyelitis seen as diminished signal in bone, T2WI-high intensity epidural mass enhancing with gadolinium in late cases (granulation tissue) but not in early stages (Pus). Myelogram- extradural compression-paint brush appearance. CI/C2 puncture to demonstrate upper limit of complete block. CT-helpful after myelography (intraspinal gas)
Treatment; Immobilisation-halo vest for cervical infection,TLSO for thoracic to sacral involvement, antibiotics when no neurological deficits/spinal deformity.
Nonsurgical management may be reserved for (a) those with prohibitive operative risk factors, (b) involvement of an extensive length of spinal canal, (c) complete paralysis for more than 3 days, (d) absence of significant neurological deficits
Surgery; Aims-establish microbiological diagnosis, drain pus, debridement of granulation tissue, bony stabilisation when necessary. Laminectomy adequate for most cases. Patients with vertebral osteomyelitis may develop instability after laminectomy (anterior abscess with osteomyelitis-Pott's disease): Posterolateral extracavitary approach avoids transabdominal/transthoracic approach in very sick patients, removal of devitalised bone, posterior instrumentation and fusion-Strut grafting with autologous bone (rib or fibula) has little risk of bone infection in Pott's.

Specific antibiotics; If organism and source unknown, treat empirically for staph aureus; (a) 3rd generation cephalosporin e.g. cefotaxime (claforan) plus (b) vancomycin, until methicillin resistant S.aureus can be ruled out (MRSA) then switch to synthetic penicillin (nafcillin or oxacillin) plus (c) rifampin PO
Antibiotics may be modified according to culture results or knowledge of source. Give 4 weeks of IV antibiotics and 4 weeks of oral antibiotics. In case of vertebral osteomyelitis 6-8 weeks IV antibiotics guided by serial ESR. Immobilisation for at least 6 weeks during antibiotic therapy.
Outcome; 20% mortality (from primary source of infection, or complication of residual paraplegia-pulmonary embolism). Severe neurological deficits rarely improve.
Differential Diagnosis of Destructive lesions of the spine-
(1) neoplastic-(a) spinal cord tumours, (b) metastatic tumours with a predilection for bone-prostate, breast, renal cell, lymphoma, thyroid, lung, (c) primary bone tumours-chordomas, osteoid osteoma, haemangioma
(2) Infection (a) vertebral osteomyelitis (b)discitis,
(3) chronic renal failure-destructive spondyloarthropathy that resembles infection,
(4) ankylosing spondylitis.
Destruction of disc space suggestive of infection; spared in malignancy.
Vertebral osteomyelitis.
2-4% of all cases of osteomyelitis. Has similarities to spinal epidural abscess.
Tuberculous vertebral osteomyelitis (tuberculous spondylitis, Pott's disease); commonest lower thoracic and upper lumbar, many levels, vertebral body, psoas abscess. Neurologic deficits in 10-47% of patients due to medullary and radicular inflammation, epidural granulation tissue, fibrosis, kyphotic bony deformity. Good result with medical treatment. Surgery indicated in cord compression, abscess or sinus formation. Imaging-Plain x-ray changes seen in 2-8 weeks. MRI; T1WI-low signal from vertebral body and disc space, T2WI-increased intensity from vertebral body and disc space.
Brain Abscess
Brain abscesses arise by several mechanisms including hematogenous spread, penetrating trauma, surgery, or local spread from the paranasal sinuses, mastoid air cells or emissary veins. The peak incidence is in young men due to the occurrence of middle ear and paranasal sinus infections in addition to congenital heart disease. Other predisposing factors include Osler-Weber-Rendu syndrome with pulmonary arteriovenous fistulae, endocarditis, congenital heart disease, dental work and immunosuppression. Symptoms consist of headache, fever, seizures and/or neurological deficit. The majority of brain abscesses are solitary.
Aerobic and anaerobic bacterial abscesses occur. Abscess cultures in one third of patients grow multiple organisms. Common organisms based on the site of origin include Streptococcus species from the frontal/ethmoid sinus; Bacteroides fragilis from chronic mastoiditis/otitis; Staph. aureus or enterobacteriacea following penetrating trauma or surgery; Strep. viridans and Strep. pneumonia in cases of congenital heart disease; Staph aureus and Strep. pneumonia in cases of endocarditis. In immunosuppressed patients, toxoplasma gondii, nocardia, mycobacteria, yeast and fungal abscesses occur. In Africa, tuberculomas, cysticercosis, echinococcus, schistosomiasis and strongoloidiasis are more common.
In the first stage of brain infection, there is inflammation of the brain, termed early cerebritis. This stage occurs in the first 3-5 days after inoculation. The CT scan appearance of cerebritis is that of an ill-defined hypodense contrast enhancing area. This coalesces to a late cerebritis stage during days 4-13 with irregular rim enhancement. This is followed, at approximately day 14, by

a collagen reticulum encapsulation with a necrotic center (early capsule stage). On CT scan or MRI scan this appears as a ring enhancing mass often with the abscess wall facing the ventricle appearing the thinnest. The final stage is the late capsule stage in which there is a three-layer capsule: an outer gliotic layer, a middle collagenous layer and an inner granulation layer. These can persist for months on imaging studies before ultimate resolution.
Antibiotics are the mainstay of treatment in all cases. Empiric treatment of a presumed bacterial abscess requires coverage for both aerobes and anaerobes. Surgery is usually indicated for diagnosis and drainage of the abscess, and for culture and sensitivity of specific organisms. Stereotactic aspiration is the treatment of choice. Aspiration may need to be repeated before resolution occurs. Often two to three weeks of antibiotic treatment are needed before a size decrease is seen on imaging studies. In general 2 weeks of intravenous antibiotics are often used, followed by 4 weeks of oral antibiotics. Patients with nocardia abscesses, or patients in whom treatment has failed after the third aspiration, should consider surgical resection when accessible. Often, aspiration alone can treat significant mass effect and prevent rupture of the abscess into the ventricular system. Ventricular rupture of a bacterial brain abscess is often fatal.