Spinal Infections

Neurosciences Grand Rounds
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Overview

General Principles
  Anatomy
  Pathophysiology
  Epidemiology
  Classification of Spinal Infection

Case Presentation A
  Vertebral Osteomyelitis

Case Presentation B
  Epidural Infection

Conclusion
Introduction

• Infection of the spine recognized throughout history.
• Much of our knowledge stems from tuberculous spondylitis, evidence of which predates 7000 BC.
• Rest and “fresh air”
• Surgical debridement in 1911.
• Decompression of canal in 1956.
• Development of surgical techniques to investigate and treat spinal pathology led to iatrogenic infection.
Anatomy

• Knowledge of the embryology, maturation and circulation of spine is essential to understanding spinal infection and its treatment.
Embryology

- 3rd wk = start spinal development. Notochord forms, then somites form around a segmental artery.
- Separation occurs forming sclerotomes.
- Body starts as an ossific nuclei surrounded by cartilage cells at 10 wks.
- Each body surrounded by notochord remnant and fibrocartilage = disc.
- Cartilage analogue sep into body, superior, inferior ring apophyses.
- 3 nuclei = body, posterior element & pedicle. Fuse at 16 – 20 yrs.
Arterial Circulation

- Arterial circ is analogous in C, T, L spine.
- Vertebral arteries = C spine.
- Intercostals = T spine.
- Lumbar arteries = L spine.
- Arterioles penetrate body.
- 2nd source from posterior spinal branches through intervertebral foramen.
Arterial Circulation

- Within the canal these vessels form an anastamotic network posterior to the vertebral body.
- Network donates nutrient arteries to each vertebral body via centrally located nutrient foramen in the posterior wall of the body.
Venous Circulation

- Venous system forms a valveless plexiform network from the dural sinuses to the sacrum longitudinally.
- Three main divs:
  - Venous channels
  - Epidural veins
  - Venous network
Intervertebral Disc

- Majority of disc is **avascular**, therefore direct inoculation is required for infection.
- Iatrogenic or contiguous spread from body.
- Penetration of Abx must be via diffusion.
- Positive charged nucleus accepts negative charged Abx (i.e., Gentamycin).
- Pyogenic organisms can invade the disc and digest it, mycobacterium (Tb) require $O_2$. 
Pathophysiology

• Hematogenous seeding of bacteria from distant sites.
• Two proposed theories (neither proven):
  1) Valve-less venous route, large capacity, slow flow provide easy and direct flow to the spine for bacteria.
  2) Arterial route near metaphyseal area with rich blood supply from nutrient arteries penetrating bone.
Pathophysiology

• Invasive diagnostic and treatment are associated with iatrogenic infection.
• Lumbar discography complicated by discitis in 0.6% cases.
• Lumbar discectomy 1% patients.
• Posterior and anterior lumbar fusion 3 – 13%.
• Variable rates secondary to procedure, pathology, and host factors.
• Direct inoculation via trauma, stab, GSW, etc.
Epidemiology

- Variety of causative organisms.
- *S. aureus* most common cause pyogenic VO.
- *Staph epidermidis* also common.
- Gram-negatives (*E. coli, proteus, Enterococcus*) common postoperative and immunocompromised.
- Fungal infections are disc sparing multiple lytic lesions also in the immunocompromised.
- Tb (Pott’s) prevalent in 3rd world.
Epidemiology

- Hematogenous seeding as result of sepsis from distant site.
- DM and I.V. drug users higher rate of pyogenic spondylitis.
- Immunocompromised ↑ risk.
- Children have propensity toward bacterial discitis due to immature circulation pattern.
Epidemiology

• Aggressive or longstanding infections may lead to neural compromise.
• Epidural abscess, meningitis, and subdural abscess all shown to cause paralysis.
• Advanced age, S. aureus infection, cervical or more cephalad levels, RA, receiving systemic steroids, or DM at increased risk of pyogenic or fungal osteomyelitis leading to paralysis.
Classification of Spinal Infection

- Described by anatomical location.
- Infection often involves more than one area.
- Location of focus can suggest cause and guide treatment.
- Characterized into three main regions:
  - Anterior spine.
  - Posterior spine.
  - Spinal canal.
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<th>Anatomic Location</th>
<th>Area/Structure Involved</th>
<th>Terminology</th>
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<td>Anterior spine</td>
<td>Vertebral body</td>
<td>Vertebral osteomyelitis</td>
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<td>Spondylodiscitis</td>
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<td>Intervertebral disc</td>
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<td>Paravertebral space</td>
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<td>Psoas abscess</td>
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<td>Retropharyngeal abscess</td>
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<td>Posterior elements</td>
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<td>Subdural space</td>
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<td>Spinal cord</td>
<td>Intramedullary abscess</td>
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Figure 2. Diagram illustrating anatomic areas of potential involvement in spinal infections.
Anterior Spine

- Vertebral body, disc, and paravertebral area.
- Body – vertebral osteomyelitis or spondylitis – commonly hematogenous.
- MR shows signal change in body, disc space and opposite endplate (spondylodiscitis).
- Infection of body with disc sparing often granulomatous (e.g., Tb).
- Discitis common in children, adults discitis iatrogenic involving adjacent endplates.
Anterior Spine

- C spine osteomyelitis can cause retropharyngeal abscess or mediastinitis or vice versa.
- T-spine can progress mediastinitis or mediastinal abscess. Empyema and pericarditis.
- In TL may cause peritonitis, psoas abscess, or subdiaphragmatic abscess.
- Rx with Abx mainstay; surgical debridement req ant approach, occ posterior transpedicular approach.
Posterior Spine

- Most often 2° to acute or chronic postop infection.
- Often assoc with superficial wound infection tracking to bone.
- Pedicle screws can serve as an entrance site for deeper bony infection.
- Ant infection rarely progresses posterior, more often it progresses into the spinal canal.
Spinal Canal

• Commonly an epidural abscess (usually posterior in canal) may form via a hematogenous source.
• Vert osteomyelitis can cause epidural abscess.
• Rx with Abx and debridement via posterior approach with laminectomy. Those from an ant source should be debrided from the anterior.
• Can involve meningitis, Rxd with IV Abx.
• Rare subdural abscess or in the cord as an intramedullary abscess – approach posterior.
Case A

- 46 year old female with severe LBP, Rt/Lt leg weakness, numbness, ambulating with antalgic gait, B&B norm.
- L5/S1 laminectomy with discectomy on 20 June for acute spinal stenosis.
- Postop - Lt leg weakness unchanged, motor deficits, mild sensory ↑ Rt leg, developed complete peroneal anesthesia and lost fxn B & B.
Case A

- Two MRI’s later infection L5 to S2 detected – Rxd with crse Abx. Presented 14d post-op to HGH and Rxd with Abx and an L3,4,5 decompression and debridement.
- MRI 20 July – reaccumulation of pus.
- 2nd surgical debridement of wound infection, septations, and abscess. Penrose, hemovac. Closed with four tension sutures.
Case A

- Sep 12, f/u with MRI.
- Neuro status improving, Lt leg norm, Rt leg weakness and numbness, regained bowel control, some minor incontinence remains.
- Posterior incision open to heal via secondary intention, clean.
- Improving from neuro perspective. Now ambulatory.
Pyogenic Vertebral Osteomyelitis and Post surgical Disc Space Infections

• Pyogenic vertebral osteomyelitis, or discitis, assoc with GU, soft tissue, resp tract infections, and the immunocompromised.
• Disc space infections seen after direct inoculation, penetrating injuries, discograms, LP’s, and surgery.
• 37% unknown source.
• *Staph aureus* most common, *E. coli, Proteus*, and *Pseudomonas* seen with GU infection.
Pathophysiology

- Hematogenous seeding.
- Direct inoculation.
- Neurological injury assoc with **Epidural spread**.
- RF’s include: age, cephalad level of infection, DM, RA (17%), steroids, CA, irradiation, and the immunocompromised.
- Incidence after discectomy is 0.7 – 0.8%, 6 – 8% fol fusion with instrumentation with Abx prophylaxis.
Diagnosis

- Clinical presentation varied, dependant on organism and host. Pain, fever, ROM, and + straight leg raise common, Overt sepsis if late.
- Postop infection 2-6 weeks relief fol by return of back pain, (Discitis), occasionally constitutional SSX. Neuro deficit rare, if present suspect cauda equina or epidural abscess.
- Positive peripheral culture, CT guided needle biopsy.
- CBC often void of leukocytosis, ESR often positive, ESR good guide to treatment effectiveness.
• **Plain radiographs** often normal, later may show decreased disc height and blurring of endplates. Findings often lag 2-3 weeks behind SSX.

• Technetium accuracy 90% in non postop.

• CT with/out myelogram (if no epidural infection).

• MRI test of choice (96% sensitivity), Gad **very** helpful when Dx is equivocal, T1 images reveal contrast uptake in infected disc and body.
Treatment

- Goals of treatment to prevent/reverse neuro compromise, relieve pain, establish stability, eradicate infection, and prevent relapse.

- Prophylaxis is required for operative cases, parenteral Abx, Abx irrigation, re-administer Abx if significant blood loss. 48hrs.

- If infected, parenteral Abx are required for 6 weeks followed by 2-4 months oral Abx.
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- Immobilization with bed rest until clinical improvement, then C collar or halo, TLSO or LSO for 3 – 4 months.
- Indications for surgery: open biopsy for Dx, sepsis, large abscess, refractory to non-operative treatment, cord compression, neuro deficit, instability or significant destruction.
- Role of surgery in postop infection reserved for sepsis, epidural abscess formation, and **advancing neurological deficit**. Surgical approach depends on extent of problem.
• Patients discovered early may benefit from re-exploration.
• In more extensive cases or chronic cases, most effective approach is anterior, there should be a through debridement of necrotic tissue back to viable bone followed by autogenous strut graft.
Sub Conclusion

• Hematogenous and postop infections similar presentation and courses.
• Treatment is primarily conservative with immobilization and Abx.
• Indications for surgery are rare, approach best suited is most often anterior.
• The outcome is favorable with early recognition and appropriate treatment.
• Cases that have formed an epidural abscess require an emergent decompression.
Case B

- 58 year old male ref re. anterior spinal abscess from MUMC.
- Hx RA with ++ prednisone (5yrs) & methotrexate.
- Jan ’01 UGI bleed 2° NSAID’s.
- Apr ’01 LLL pneumonia with empyema (*staph & strep*).
- Jul ’01 reoccurrence with cavitation.
- Sep ’01 admit St. Joe’s febrile sepsis, developed acute pulm edema. Dx’d with mediastinal abscess at T4 with involvement of vertebral body.
Case B

• Seen by thoracic surgery, attempted percutaneous drainage.
• SSx only moderate thoracic back pain. As of yet, NO neurological deficit.
• **Plain X rays** show multiple comp #’s T5 – T9 and significant T4 body destruction.
• **MRI** shows large ant abscess T2 – T5, remnants of T4 impinging on thecal sac, and a large epidural abscess running from C1 to L1.
Case B

- Consultation with Ortho for second opinion, aghast at lack of neuro SSx.
- Agree to attempt percutaneous drainage of anterior abscess and hope it communicates with epidural abscess.
- Unfortunately, new MRI shows worsening in all regions. Pt remains stable and well.
- What next???????
Neurosurgical Care of Spinal Epidural Abscesses

- Rare but potentially devastating.
- Rapid evolution of disease process and assoc illness mortality can be 20%.
- Incidence 2 cases/10,000 hosp admissions.
- Peak incidence in sixth/seventh decade.
- Assoc comorbidities include DM, CRF, ETOH, CA, IV Drug users, and other conditions that impair immunocompetence.
Continued . . .

• 16% to 40% cases source unidentified.
• Skin, soft tissue = 25%, surgery, osteomyelitis, spinal trauma, UTI, Resp infection all common sources.
• Etiology is *Staph aureus* in 60%, fol by *Strep* species. Gram neg in IV drug abusers and occasionally *Mycobacterium Tb*. 
Pathophysiology

- Epidural space is a potential space with individual metameres septated preventing free communication between anterior and posterior space.
- Metameric segmentation may limit longitudinal spread to 3 – 4 segments, extensive abscesses have been reported.
- Hematogenous spread assoc with post infection.
Continued . . .

- Cases associated with discitis or vertebral osteomyelitis typically involve the anterior space.
- Cause of neuro deficit unknown. Many suffer rapid, irreversible impairment.
- Postulate ischemic mechanism from occlusion or venous stasis (vascular thrombosis), other studies show compression as the causative lesion.
- Likely multifactorial.
Diagnosis

- Functional outcome dependant on timely Dx & Rx.
- Most frequent presenting symptom back pain (94%), severe and stabbing. Pain may be radicular followed by weakness and gradual paralysis.
- Fever common and should raise suspicion if assoc with back pain. DDx is abscess, osteomyelitis and leukemia.
- Overt sepsis, hypotension or multiorgan failure, do poorly.
Continued . . .

- CBC, ESR, Cultures often positive (60%).
- Lumbar puncture = risk of neuro deterioration, spread of infection = completely contraindicated.
- Plain radiographs miss 35-65%, findings are nonspecific showing degenerative changes.
- Myelography or CT Myelography very sensitive but danger associated with needling thecal sac.
- Modality of choice now MRI, enhancement may allow differentiation between liquefied pus and granulation tissue.
Treatment

- Management depends on clinical condition.
- Surgical debridement mainstay.
- Medical Rx with appropriate IV Abx if abscess located so cord not at risk, no neuro deficit and + culture for correct Abx – use with caution and close monitoring.
- Disadvantage due to 8-12 wks IV Abx and serial MRI’s. Risks irreversible neuro deterioration and is not cost effective.
Proposed Criteria for Medical Treatment

- Poor surgical candidates because of severe concomitant medical problems.
- Cases in which the abscess involves a considerable length of the spinal canal and who have an epiduritis from the cervical to lumbar level.
- Patients not suffering from severe loss of spinal cord or cauda equina function.
- Patients with complete paralysis for more than 3 days.
Surgical Therapy

• Decompression and drainage is best method to prevent neurological deficit, if done quickly after the onset of deficits, may allow full recovery.

• Time course of disease not helpful in determining the type of infectious material found at surgery (i.e., pus or granulation tissue). Cannot easily estimate extent of procedure, determine intraoperatively.

• Not critical in posterior cases (80%) as easily extended. Laminectomy common and effective.
Continued . . .

- Culture for Abx.
- Copious irrigation
- Most posterior infections are hematogenous therefore few with discitis/osteomyelitis – anterior column therefore stable.
- Closure with drains may be primary, delayed closure prolongs hospitalization and discomfort; delayed closure due to tension is difficult.
Continued . . .

- Anterior much more complicated, disproportionate number assoc with discitis.
- Instability and deformity frequent.
- Anterior approach with discectomy suitable if pus encountered which drains easily.
- Bone must be debrided back to healthy bone, may require two level corpectomy, stabilization may be done at the same time.
Continued . . .

- If done from posterior alone, stabilization will be required (anterior column unstable).
- Method is surgeon dependant; antero-posterior (1 or 2 stage), or in T-spine extracavitary approach.
- Anterior support, likely supplemented with instrumentation, will be needed. Status of posterior elements dictates need for posterior instrumentation.
Continued . . .

• Instrumentation may be placed in situations of active infection as long as adequate debridement conducted, if not feasible, patient will require bed rest until infection controlled and stabilization performed.

• Access possible via percutaneous methods, reasonable for purulent debris.

• Appropriate IV Abx post-op for 4-6 weeks.

• Pts need to be followed for several months to check stability.
Prognosis

- Heavily dependant on clinical and neurological condition of the patient at presentation and delay in Dx.
- If presentation is overt sepsis likely death.
- Preop plegia has poor outcome (time!).
- Appears 36 hours is cutoff.
- Mortality has improved over past several decades, remains ~14%.
- Mortality related to delay in Dx and Tx
References


Plain Thoracic Radiographs
MRI IV
Plain Radiographs