Osteomyelitis

Last updated: August 8, 2020

**CRANIAL OSTEOMYELITIS**

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<td><strong>Etiology</strong></td>
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<td>Diagnosis</td>
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<td>Treatment</td>
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**VERTEBRAL OSTEOMYELITIS (S. INFECTIVE SPONDYLITIS)**

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<td><strong>Etiology</strong></td>
<td>2</td>
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<tr>
<td>Clinical Features</td>
<td>2</td>
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<tr>
<td>Diagnosis</td>
<td>2</td>
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<tr>
<td>Treatment</td>
<td>4</td>
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**INFECTIOUS DISKITS**

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<td>4</td>
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<td>Clinical Features</td>
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<td>4</td>
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<td>4</td>
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**VERTEBRAL EPIDURAL ABSCESS**

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</thead>
<tbody>
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<td>5</td>
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**GENERAL FEATURES** of osteomyelitis → see p. 1192 (2.3) >>

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**CRANIAL OSTEOMYELITIS**

**ETIOLOGY**

1. Direct extension from paranasal sinuses, ear (e.g. *malignant external otitis* see p. Ear4 (2.3))
2. Penetrating skull injury
3. Infected craniotomy flap, skeletal traction
4. Hematogenous

**GRADINIEWSKY’S syndrome – optical petrositis** (osteomyelitis) involving CNS & CN6. see p. CNS >>

**CLINICAL FEATURES**

- pain, tenderness, swelling, warmth at infected site.
  - drainage of purulent material if open wound is present.
  - if systemic symptoms are present, underlying subdural / epidural empyema is commonly present.

**DIAGNOSIS**

1. Plain skull film (positive > 50%)
2. CT
3. Technetium bone scans (helpful if skull radiographs are negative);
   - *tale positive in old trauma or previous craniotomy; H. gallium scan* (differentiates infection from other causes of positive technetium scan).

**TREATMENT**

1. Surgical debridement (removal of infected bone)
   - adequate margin of normal bone is removed to minimize risk of recurrence.
   - after at least 1 year with no evidence of inflammation, cosmetic / protective cranioplasty may be performed.
2. Antibiotics
   - MREA is treated with 6 weeks of *VANCOMYCIN*; if hardware is present (e.g. cranial mesh), add *EFAMPIN*.

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**VERTEBRAL OSTEOMYELITIS (S. INFECTIVE SPONDYLITIS)**

- septic disco-vertebral lesion

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**INFECTIVE** of vertebrae usually involve disk space (vs. *malignant lesions*!)

- disseminated via small nutrient arteries, bacteria lodge in metaphysis beneath end-plate of vertebra (usually anteriorly) → quickly extend into adjacent disc and end-plate of opposite vertebra.

In children, because the disk is vascularized, it can be a primary site.

- complications, *paraspinal extension* (along spine, beneath paravertebral ligaments, etc) - paraspinal abscess, anterior epidural abscess.
  - paraspinal masses are large in indolent forms of infection (such as tuberculosis).
**Radiographic changes of spinal tuberculosis**: (on plain films):

1. Lysis destruction of anterior portion of vertebral body
2. Reactive sclerosis on a progressive lytic process
3. Enlarged psoas shadow with or without calcification; fusiform paravertebral shadows suggest abscess formation

In contrast to pyogenic disease, *calcification is common in tuberculous lesions!*

4. Vertebral end plates are osteoporotic
5. Intervertebral disks may be shrunk or destroyed
6. Vertebral bodies show variable degrees of destruction → collapse with anterior wedging
7. Bone lesions may occur at more than one level

**CLINICAL FEATURES**

Obtain bacteria identification ASAP (e.g. blood culture) before starting antibiotics! (else may need IR biopsy)

1. ESR ↑ (73.1%), CRP, WBC (30%)
2. X-ray (changes may take weeks + months to appear!):
   1. Progression narrowing of disk space
   2. Erosion and destruction of adjacent vertebral end-plates → body collapse → wedging
   3. Sclerosis, sharp kyphosis (ribbed bones).
3. Paravertebral soft-tissue masses:
   - Cervical spine - focal swellings of retropharyngeal soft-tissue stripe; thoracic spine - displacement of paraspinal lines; lumbar spine - lost psoas muscle shadow.

**Diagnosis**

- course tends to be subacute (patients with hematogenous spread are spared)
- Spine tenderness unrelieved by rest.
- Fever
- Deep back pain exacerbated by motion (movement restriction by muscle spasm), may be unrelated by rest.

Most common primary sources of infection (can be identified only in 40% patients): urinary tract, skin, lungs.

- well-recognized risk factor - IV drug use.

**Etiology**

- Hemorrhagic spread (rarely, direct extension!):
  1. Pyogenic bacteria - streptococci are most common! (~ 50%)
  2. M. tuberculosis (Pott's disease) – one of the oldest demonstrated diseases of humankind (in 1779, Percivall Pott presented the classic description of spinal tuberculosis).

  - rare in West; still a significant cause of disease in developing countries;
  - affects young adults.
  - 80% patients have no evidence of pulmonary involvement.
  - Most frequent in lower thoracic + upper lumbar vertebrae:
  - Tendency to involve multiple segments (through subligamentous paraspinous spread).
  - Discs frequently are spared until later in course. – “skip” lesions

  *E.g. complications of discography, lumbar puncture*

**Specific MRI findings of tuberculosis:**

1. Intervertebral osteomyelitis (L4-safran T1-MRI):
   - Diffuse low intensity throughout L45 vertebral bodies, and even lower signal from intervening disc space (which is barely visible because of loss of dark line of vertebral endplates); little epidural soft tissue thickening suggesting extraspinal extension.

2. Subligamentous extension:

   - Occasionally, **nondisco genic forms** (involving only vertebral bodies or neural arches) are encountered - difficult to distinguish from neoplasia, metastases!
Infectious spondylitis at T6-7:
A) lateral radiograph - disc space narrowing, erosion of adjacent vertebral end-plates (arrow), reactive sclerosis in inferior vertebra.
B) CT - bony destruction; note extent of associated paraspinal soft-tissue mass (arrow).

Thoracic tuberculous spondylitis:
A) paraspinal soft-tissue mass in AP radiograph; involved disc space is difficult to resolve.
B) disc space obliteration and destruction of adjacent vertebral end-plates.

Tuberculous spondylitis with subligamentous extension (sagittal thoracic tomogram) - obliteration of disc space and destruction of adjacent vertebral end-plates in midthoracic spine; superior and inferior subligamentous extension is manifested by erosions of anterior vertebral body margins over several levels (arrows).

Pyogenic spondylitis:
A) lateral X-ray at L4-L5 - marked narrowing of disc space, loss of sharp vertebral end-plate margins, and mild reactive sclerosis in L4 vertebral body.
B) T1-MRI - extensive abnormal low signal within adjacent vertebral bodies and intervening disc, with loss of hypointense border at vertebral margins.
C) postcontrast T1-MRI - pronounced enhancement of involved vertebra and portions of infected disc; no epidural involvement.
D) fat-suppressed T2-MRI - edema in vertebral bodies, abnormally bright signal in infected disc - corresponding to areas of low signal intensity in postgadolinium MRI (arrowheads).
INFECTIONOUS DISKITIS

ETYMOLOGY
- usually iatrogenic (complication of previous surgery or needle puncture of intervertebral disks) - most often staphylococci!

N.B. TB (Pott’s disease) tends to spare disc space in vertebral osteo - highly aerobic bacteria!

CLINICAL FEATURES
1) severe pain aggravated by palpation; partially relieved by recumbency.
2) muscle spasm
3) fever
• interspace infections must be observed closely – risk of epidural abscesses!

DIAGNOSIS
Early in course:
• X-rays and CT are normal!
• gallium scans may be falsely positive because of recent surgery.

Later in course - destructive changes along edges of disk space, narrowing of intervertebral space.
• CT demonstrates these changes early.

Needle biopsy of involved interspace identifies causative bacteria (cultures are often sterile → direct surgical biopsy).

TREATMENT
1) bed rest, medication for pain and muscle spasms.
2) antibiotic therapy (empirically – against staphylococci).
3) no response to conservative therapy → open surgery (remove infected material from interspace).
VERTEBRAL EPIDURAL ABSCESS

**ETIOLOGY**
- Typically – hematogenous Staph; other – osteodiscitis epidural extension.

**CLINICAL FEATURES**
- acute septic course with severe axial back pain.
- early neurological deficits.

**DIAGNOSIS**
- Tends to spread up and down.
- collection avidly rim-enhances.

**TREATMENT**
1) surgical debridement (unless small collection in neuro intact patient).
2) 4-6 weeks of antibiotics

Long abscesses - skip laminotomies and pediatric feeding tube / EVD irrigation:

**BIBLIOGRAPHY** for ch. “Infections of Nervous System” – follow this LINK >>