

Osteomyelitis

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CRANIAL OSTEOMYELITIS

ETIOLOGY

1. Direct extension from paranasal sinuses, ear (e.g. *malignant external otitis* see p. Ear40 >>)
2. Penetrating skull injury
3. Infected craniotomy flap, skeletal traction
4. Hematogenous

GRADENIGO'S syndrome – *apical petrositis* (osteomyelitis) involving **CN5 & CN6**. see p. CN5 >>

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);
 - **false-positive** in *old trauma* or *previous craniotomy*; H: **gallium scan** (differentiates infection from other causes of positive technetium scan).

TREATMENT

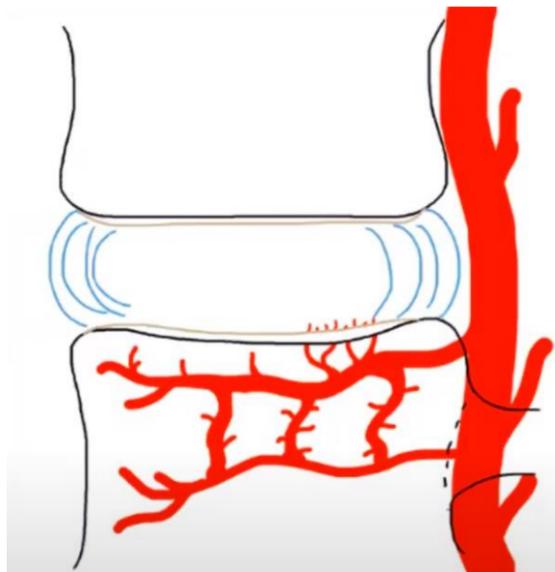
1. **Surgical debridement** (removal of infected bone)
 - adequate margin of normal bone is removed to minimize risk of recurrence.
 - *after at least 1 year* with no evidence of inflammation, **cosmetic / protective cranioplasty** may be performed.
2. **Antibiotics**
 - MRSA is treated with 6 weeks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

VERTEBRAL OSTEOMYELITIS (S. INFECTIVE SPONDYLITIS)

- **destructive disco-vertebral lesion**.

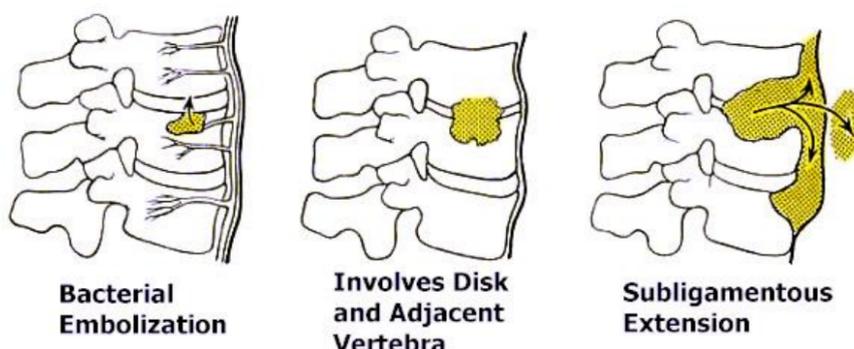
Infections of vertebrae usually involve disk space (vs. **malignant lesions!**)

- disseminated via small nutrient arteries, bacteria lodge in *metaphyses* 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).



- occasionally, *NONDISCOGENIC FORMS* (involving only **vertebral bodies** or **neural arches**) are encountered - difficult to distinguish from neoplasia, metastases!

ETIOLOGY

- **hematogenous spread** (rarely, direct extension*):

- Pyogenic bacteria - *staphylococci* are most common! ($\approx 50\%$).
- 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 paraspinal spread).
 - discs frequently are spared until later in course – “skip” lesions
*e.g. complications of discography, lumbar puncture

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

- well-recognized risk factor - **IV drug use**.

CLINICAL FEATURES

- course tends to be subacute (patients with *pyogenic* spondylitis usually present while infection is still confined to one disc space):

- Deep back pain** - exacerbated by motion (**movement restriction** by muscle spasm), may be unrelieved by rest.
- Spine tenderness** over involved spine segment.
- Fever** (25%).

N.B. all signs of infection may be absent and course may be indolent!

Neurological involvement - late and inconstant feature ($\approx 1\%$ cases; but in 40% of cases caused by **tuberculosis!**) by:

- intraspinal extension of infection (epidural abscess)
- spine instability and fractures

DIAGNOSIS

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

- ESR \uparrow (!!!), CRP \uparrow , WBC \uparrow (30%)
- X-ray** (changes may take weeks ÷ months to appear!):
 - progressive *narrowing of disk space*
 - erosion and *destruction of adjacent vertebral end-plates* \rightarrow *body collapse* \rightarrow wedging, subluxations, sharp kyphosis (GIBBUS).
 - 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.

Radiographic changes of spinal TUBERCULOSIS on plain films:

- Lytic destruction of anterior portion of vertebral body
- Reactive sclerosis on a progressive lytic process
- Enlarged psoas shadow with or without calcification; fusiform paravertebral shadows suggest abscess formation
In contrast to pyogenic disease, **calcification is common** in tuberculous lesions!
- Vertebral end plates are osteoporotic.
- Intervertebral disks may be shrunk or destroyed.
- Vertebral bodies show variable degrees of destruction \rightarrow collapse with anterior wedging
- Bone lesions may occur at more than one level.

- MRI** (**diagnostic method of choice** – highly sensitive and specific! – changes are seen 2–3 weeks earlier than on plain X-rays):
 - low T1 signal (high signal on T2) throughout disc and in adjacent vertebral bodies.
 - thinning, fragmentation (and eventual loss) of dark line of vertebral end-plates.
 - IV Gd-DTPA \rightarrow diffuse enhancement in areas showing signal change.

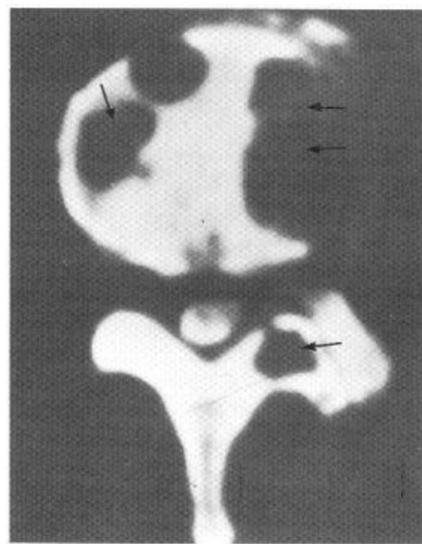
N.B. in *degenerative disk disease*, changes are less uniform, disk is desiccated and bone destruction is absent, no paravertebral soft-tissue masses.

Specific MRI findings of TUBERCULOUS spondylitis:

- thin and smooth enhancement of the abscess wall (vs. pyogenic spondylitis - thick and irregular enhancement of abscess wall)
 - well-defined paraspinal abnormal signal (vs. pyogenic spondylitis - ill-defined paraspinal abnormal signal).
- CT** (less sensitive and specific; better tolerated by some patients with severe back pain): punched-out erosions of bone adjacent to involved disc (“moth-eaten” appearance), small dense sequestra, prominent sclerosis.
 - Bone scan** with technetium, gallium.
 - CT-directed needle biopsy** of affected vertebrae; **open biopsy** may be necessary to obtain adequate tissue for culture.

Discovertebral osteomyelitis, L4-5 (sagittal T1-MRI):
diffuse low intensity throughout L4/5 vertebral bodies, and even lower signal from intervening disc space (which is barely visible because of loss of dark line of vertebral end-plates); little epidural soft-tissue thickening suggesting extraosseous extension.

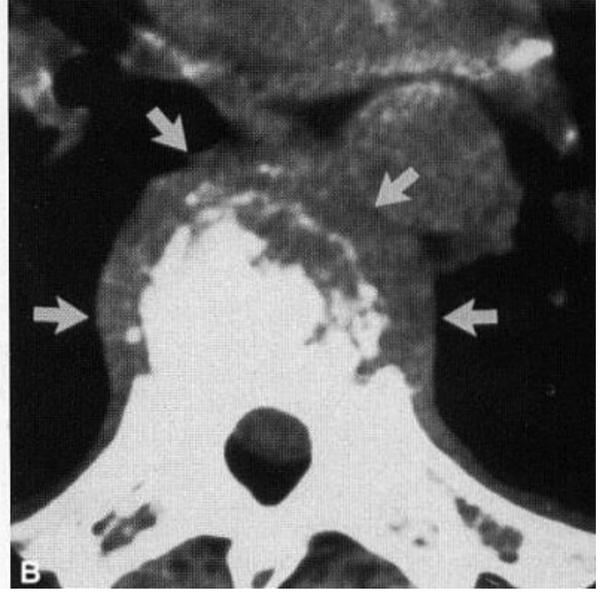
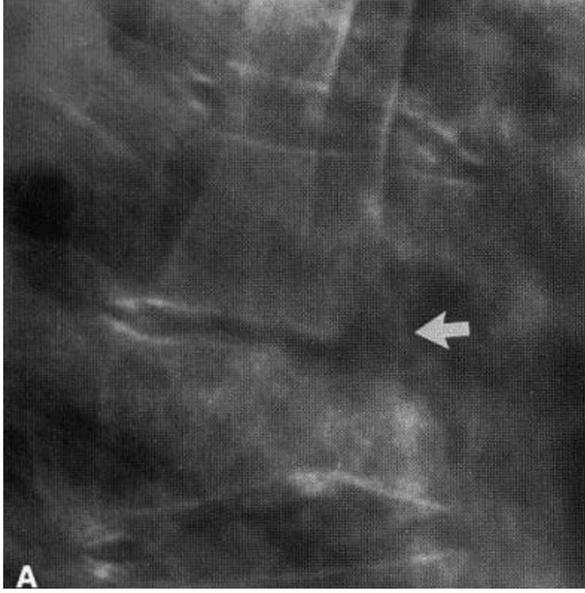
Staphylococcal discovertebral spondylitis (axial CT):
vertebral end-plate shows typical *moth-eaten appearance* (upper arrows) and another focus of infection is visible in inferior articular process (lower arrow):



Source of picture: Ronald G. Grainger, David J. Allison "Grainger & Allison's Diagnostic Radiology: A Textbook of Medical Imaging", 4th ed. (2001); Churchill Livingstone, Inc.; ISBN-13: 978-0443064326 >>

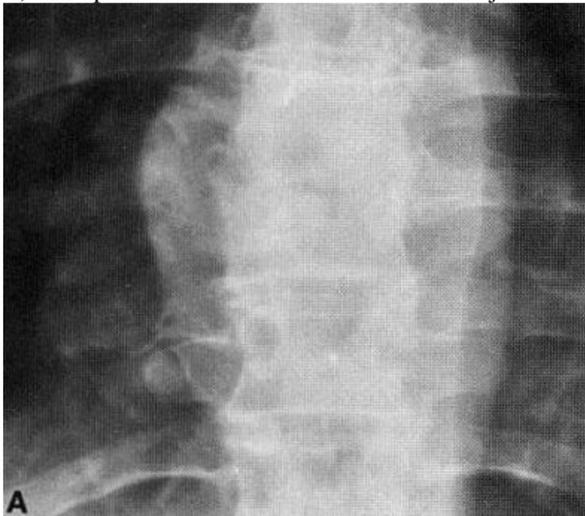
Infectious spondylitis at T6-7:

- 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 (*arrows*).

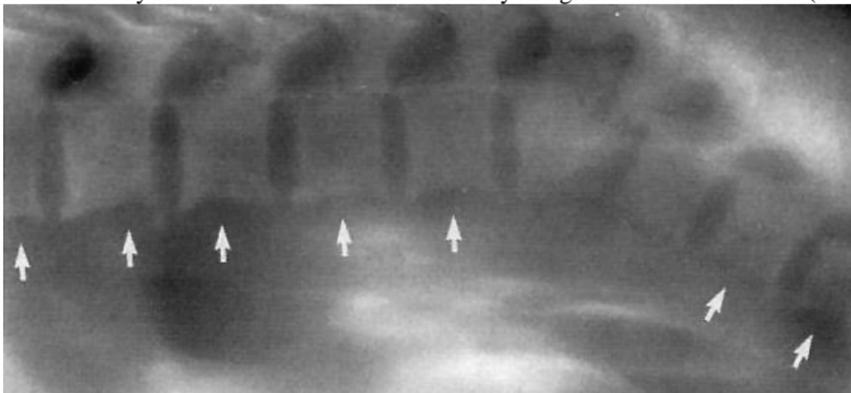


Thoracic tuberculous spondylitis:

- 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*).

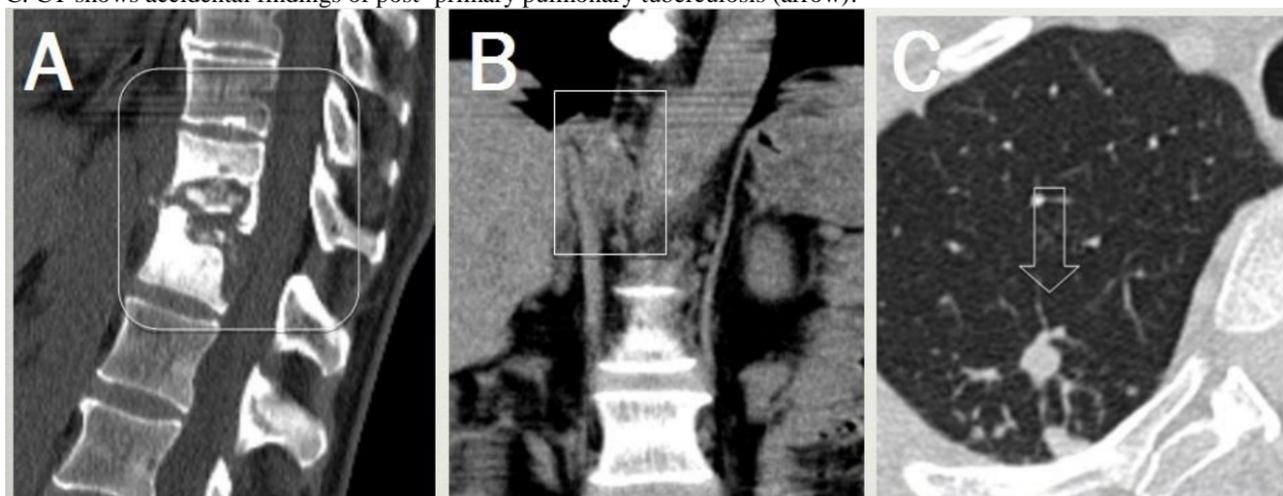


Tuberculous spondylitis in 46-year old male.

A. CT shows contact interdisk Th11-Th12 vertebral body destruction, with large and spongy sequestrars, prevertebral and epidural extension (surrounds a large square).

B. CT shows paravertebral tight, with calcination abscess near the right crus of diaphragm (surrounds a small square).

C. CT shows accidental findings of post- primary pulmonary tuberculosis (arrow):



TREATMENT

- Infection control
 - MRSA is treated with 6 weeks of **VANCOMYCIN**; if hardware is present (e.g. cranial mesh), add **RIFAMPIN**.
- Patient comfort and prevention of further deformity (bed rest, brace)
- Operative debridement – limited indications (e.g. for epidural extension as abscess with neuro deficits, kyphosis)
 - just pain, including radicular pain (tends to get better with abx) are not surgical indications.
 - instrumentation*** up to **corpectomy** may be indicated for instability / kyphosis.
 - *modern instrumentation is titanium – does not need to be isolated from site of infection.

INFECTIOUS DISKITIS

ETIOLOGY

- 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

- severe **pain** aggravated by palpation; partially relieved by recumbency.
- muscle spasm**.
- 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

- bed rest**, medication for pain and muscle spasms.
- antibiotic therapy** (empirically – against staphylococci).
- no response to conservative therapy* → **open surgery** (remove infected material from interspace).

- when infection is controlled, interspace will eventually narrow → **spontaneous fusion**.

VERTEBRAL EPIDURAL ABSCESS

ETIOLOGY

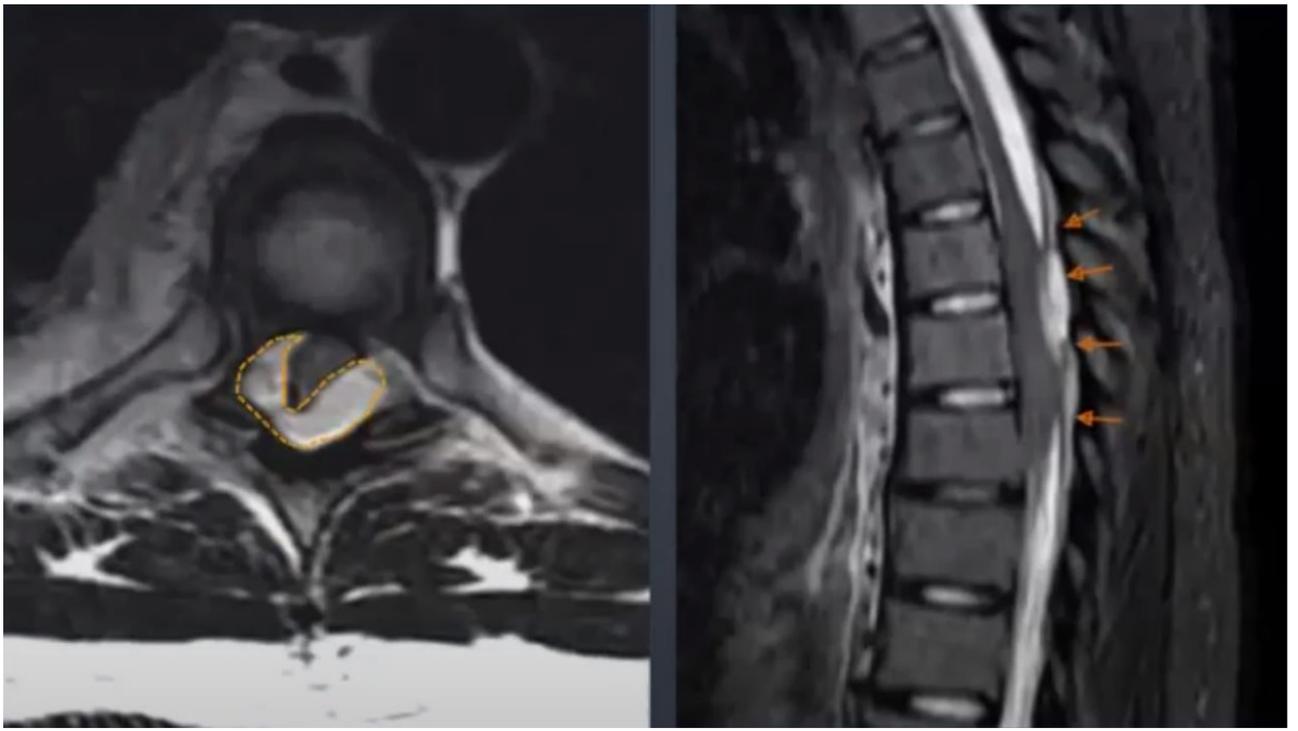
Typically – **hematogenous Staph**; other – osteodiscitis **epidural extension**.

CLINICAL FEATURES

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

DIAGNOSIS

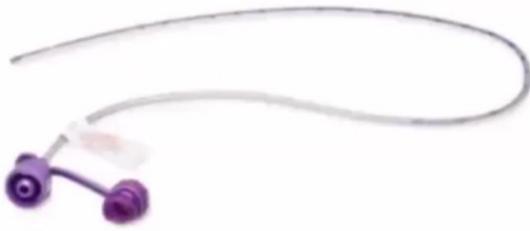
- tends to spread up and down.
- collection avidly rim-enhances.



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 >>](#)