Abscess, Empyema

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INTRACEREBRAL ABSCESSES

ETIOPATHOPHYSIOLOGY

Predisposing conditions

- Accounts for 2% of intracranial mass lesions.
- Male/female ratio ≈ 2:1.
- Median age at presentation 30-45 years; 25% cases occur in children < 15 years!

ETIOPATHOPHYSIOLOGY

- Rare disease in immunocompetent individuals.

PREDISPOSING CONDITIONS

1. Immunocompromise: AIDS, organ (esp. bone marrow) transplant recipients, chronic corticosteroid therapy, neutropenia, lymphoma / leukemia.
   - HIV is associated with brain abscess caused by Toxoplasma gondii, Mycobacterium tuberculosis.
   - Fungi are responsible for up to 90% of cerebral abscesses among recipients of solid-organ transplants.
2. Congenital heart disease (with right-to-left shunt), pulmonary A-V fistulas - infected venous blood bypasses pulmonary filter (gains access to cerebral arterial system).
3. IV drug abuse.

SOURCES

1. Infections focus elsewhere:
   - Direct osteomyelitis of the sphenoid or ethmoid sinuses.
   - Exogenous bacteria (predominantly Staphylococcus aureus) are common in cranial osteomyelitis.

ETILOGIC AGENTS

(Defy primary infective process and immune state of host).

1. 30.60% abscesses are mixed infections!

1) Streptococci (esp. Streptococcus intermedius group – S. anginosus, S. constellatus, S. milleri) are identified in 50-70% brain abscesses.
2) Invasive bacteria (predominantly Bacteroides species) are common in chronic otitis media or pulmonary disease.

3) Staphylococcus aureus and Clostridium are known common after cranial penetration from surgery or trauma.

N.B. pneumococci, meningococci, Haemophilus influenzae (major causes of bacterial meningitis) are rarely recovered from brain abscess!

4) Fungi are common in immunosuppressed.

Aspergillus fumigatus – after organ transplantation, granulocytopenia.

Candida – in chronic corticosteroid therapy, granulocytopenia, after bone marrow transplantation, IV drug abusers.

5) Parasites are common in immunosuppressed.

- Intact brain parenchyma is relatively resistant to infection - in order for brain abscesses to form, there must be pre-existing compromised area (ischemia, necrosis, hypoxia) in brain tissue.
Abscess formation evolves through four stages (regardless of infecting organism):

**INFLAMMATION → NECROSIS → SUPPURATION → CAPSULE**

1) **EARLY CEREBRITIS** (days 1 to 3) - perivascular infiltration of PMNs, plasma cells, and mononuclears; marked surrounding cerebral edema.

2) **LATE CEREBRITIS** (days 4 to 9) - well-formed necrotic center reaches its maximum size and is surrounded by inflammatory infiltrate of macrophages and fibroblasts; rapid new vessel formation around developing abscess; thin capsule (fibroblasts and reticular fibers) gradually develops; area is surrounded by cerebral edema.

3) **EARLY CAPSULE FORMATION** (days 10 to 13) - necrotic center decreases in size; inflammatory infiltrate contains increasing number of fibroblasts and macrophages; mature collagen evolves from reticulin precursors, forming capsule that is better developed on cortical than ventricular side of lesion.

4) **LATE CAPSULE FORMATION** (day 14 and later) - well-formed shrinking necrotic center surrounded by dense collaginous capsule.

- depending on etiologic organism and immunologic status, there may be delayed / incomplete encapsulation, or abscess may enlarge more quickly.
- encapsulation is more complete (more mesenchymal cells forming tougher capsule) on cortical side (than on ventricular side) - propensity of abscesses to extend and rupture into ventricular system.
- encapsulation is less extensive in hematogenous abscesses.

**LOCATION**:

Frontal = temporal = parietal > occipital > brainstem, intrasellar, basal ganglia, thalamus

**Otogenic abscesses** – temporal lobe (adults), cerebellum (children).

**Sinogenic abscesses** – frontal areas.

**Hematogenous spread** – following characteristics:

1) multiple* brain abscesses (although solitary lesions may also occur)
2) distribution of middle cerebral artery - parietal lobe predominates (highest blood flow).
3) initial location at gray matter-white matter junction.

*another cause of multiple abscesses – immunosuppression.

Trichrome stain – light blue connective tissue in wall of organizing abscess. Normal brain is at right and abscess at left:

Biopsy specimen (hematoxylin and eosin) - abscess with collections of neutrophils (prominent in lower right corner) and macrophages within gliotic brain tissue:

Staining with Gomori methenamine silver highlights fungal organisms (black):

*Daughter* abscess, posterior to main abscess, had ruptured into lateral ventricle as terminal event in this case.
Abscess, Empyema

**Clinical Features**

- Rapidly expanding infectious mass lesion (most patients have subacute course with symptoms progressing during ≤ 2 weeks; may be indistinguishable from meningitis or encephalitis).
  
1. **ICP** - Prominent hemicranial or generalized headache (most common symptom!) - 70-90% patients, alterations in consciousness, vomiting, papilledema (rare finding in meningitis?).

2. **Focal neurological deficit** (75% patients!) - Seizures (focal or generalized) are particularly prominent!
3) Infection – fever ≤ 50% (i.e. may be minimal or absent!!); mucosal rigidity is present in 25-50% patients.

Abrupt neurologic deterioration:
- **Abscess rupture** into ventricular system → **ventriculitis** & **hydrocephalus**, **shock** & death.
- **Abscess rupture** into subarachnoid space → meningitis (sudden rise of CSF pressure, cell count up to 50,000/mm³, decrease in sugar content).
- **Brain herniation**
- **Spontaneous hemorrhage**

**DIAGNOSIS**

**Contrast-enhanced CT / MRI** - low-density lesion with sharply demarcated, dense, uniform* ring of contrast enhancement surrounded by hypodense region of edema. *markedly irregular wall suggests tumor! CSF - **aseptic meningeal reaction** (pressure↑, 0-1000 PMNs, protein slightly↑, normal sugar)

**MRI** is study of choice for initial detection and subsequent monitoring.

- **Abscess, stroke, and lymphoma** (high cellularity) have diffusion restriction (bright on DWI, dark on ADC), whereas gliomas and metastases do not restrict diffusion! Same as epidermoid cyst (bright DWI) vs. arachnoid cyst (normal DWI)

**Cerebritis stage** (MRI is superior to CT): area of hypointensity (hyperintensity on T2) with indistinct margins and patchy contrast enhancement in periphery.

- Enhancing ring may appear at late cerebritis stage before true capsule has been formed!
- **Delayed scan** (obtained 30 min. after IV contrast) - contrast diffusion into low-density center of abscess (vs. stage of formed true capsule - no inward diffusion of contrast).
- **MRS** – **↑ acetate, lactate, amino acids, alanine** (and ↓ NAA) - highly suggestive of cerebral abscess (but adds little value in addition to DWI)

**Encapsulated stage**: low T1 intensity (T2 hyperintense) lesion with diffusion restriction surrounded by edema.

- glucocorticoid use may alter appearance - only 40-60% reveal ring enhancement.

**Fungal abscess in 48-year-old diabetic**:

- Axial T2-MRI: central hyperintense abscess cavity with surrounding vasogenic edema.
- Coronal post-gadolinium T1-MRI: large multiloculated abscess cavity with enhancement of capsule and abscess wall. Note mild mass effect + relative thinness of medial wall compared with thicker, more irregular, lateral component.

**T1-MRI** with gadolinium: necrotic mass with peripheral enhancement and surrounding edema. Ependymal enhancement in lateral and third ventricles (ventricular rupture, ventriculitis), enhancement of subarachnoid space (meningitis), mass effect with midline shift.

**Diffusion restriction:**
**MRI of small brain abscess:**

**Abscess in right-to-left cardiac shunt:**

A) CT - marked mass effect on left frontal lobe; ringlike isodense areas surrounded by low-density edema.

B) same CT after IV contrast - enhancement of periphery of multiloculated abscess cavity.

**Brain abscess (MRI):**
Brain abscess (MRI):

2. EEG - focal slowing.

3. Etiological organism identification
   a) procedure of choice - CT / MRI guided stereotactic abscess aspiration (abscess is often sterile by time of operation).
   b) blood cultures (positive in – 10% cases*).
   c) serum should be sent for antitoxoplasma IgG (in patients with AIDS).

   - when hematogenous dissemination from remote site of infection is likely etiology

   - pulmonary infection is found in – 10% cases (chest X-ray is mandatory for all patients!).


N.B. in significant number (≈ 40%) of patients, laboratory criteria for infection are lacking!

Distinguishing brain abscess from brain tumor:
   1) C-reactive protein (CRP)↑
   2) indium-111-labeled leukocyte scintigraphy (detects areas of active inflammation); false-positive results - leukocytic infiltration into brain tumor (esp. with severe necrosis).

TREATMENT

1. Decreasing mass effect – corticosteroids (only for profound cerebral edema with impending herniation!); may decrease penetration of antibiotics! - discontinue when edema and mass effect improves.

2. Antimicrobial therapy
   - antibiotics for 6-8 weeks (at least 1-2 weeks should be intravenous)
   - empirical therapy for AIDS patients – after results of neuroimaging (focal mass lesion without impending herniation) and toxoplasma serology:
     A) > 1 enhancing lesion OR positive toxoplasma serology = presumptive diagnosis of TOXOPLASMA ENCEPHALITIS → start PYRIMETHAMINE (+ leucovorin) plus:
       a) SULFADIAZINE – first choice
       b) CLINDAMYCIN – second choice
       c) ATOVAMUNE
       d) AZITHROMYCIN
     B) 1 enhancing lesion AND negative toxoplasma serology → brain biopsy.

   N.B. rarity of toxoplasmosis in children may warrant brain biopsy without any preceding studies.

   - severely ill / immunocompromised / transplant patients - MEROPENEM is first-line choice.
   - empirical therapy for immunocompetent patients (must cover streptococci & anaerobes):
     A) PENICILLIN G 4 MU 4h + CEFTRIAXONE 2 g q12h + METRONIDAZOLE 500 mg q4h.
     B) PENICILLIN G* + METRONIDAZOLE**

* covers streptococci and anaerobes
** covers Bacteroides fragilis
Sequelae

Mortality

3. **Toxoplasma gondii**: Draining pus lesions typically occur in the CNS and eye (immunologically privileged sites) → meningoencephalitis & chorioretinitis.

Prognosis

CT diagnosis has been responsible for significant reduction in morbidity / mortality.

**Sequelae**

- **Cerebral abscess** - loss of motor or sensory deficits.
- **Focal abscesses** - drainage catheter is not recommended.
- **Cerebral abscesses** with thick, well-defined wall & central cavity* may resolve completely (usually 6-8 weeks).

**Response to antibiotics** is best monitored by serial CT / MRI:

- failure to demonstrate abscess shrinkage in 4 weeks constitutes antibiotic failure → abscess healing is indicated by decrease in its size.

**Empiric treatment of children**:

- **Amoxicillin**: brain biopsy, meningitis, neurosurgical patient
- **Ceftriaxone**: abscesses, cerebritis & ventriculitis
- **Cefotaxime**: meningitis, neurosurgical patient
- **Vancomycin**: neurosurgical patient
- **Metronidazole**: abscesses, HIV/AIDS
- **Trimethoprim-Sulfamethoxazole**: abscesses

**SPECIAL SITUATIONS**

**Toxoplasma gondii** - most common cause of focal infectious CNS lesions in AIDS patients.

- **AIDS** patients are at increased risk of brain abscess.

- **Toxoplasma gondii** abscess:
  1. **Cerebral abscess** - grey-white junction
  2. **Thalamus and basal ganglia**
  3. Less often - cerebellum and brain stem; rarely - spinal cord.

**Prognosis**

- Mortality: 5-20% (if untreated = 100%).
- **Sequelae**: seizure disorder (80-90% patients!), hydrocephalus, motor or sensory deficits, behavior and learning problems.
- **Recurrent abscesses**

**Brain biopsy** - Toxoplasma gondii cysts in microglial nodules with variety of inflammatory cell types (patient with AIDS).
Clinically – acute + chronic meningocerephalitis with **Focal Features** (multiabscesses).

**Diagnosis:**

1) **Positive serology** - antitoxoplasma IgG in serum (only indicates exposure, but not active infection).

2) **Contrast neuroimaging** (MRI is superior to CT) - like pyogenic abscesses or lymphoma; multiple lesions enhance in ringed or diffuse pattern; relatively small (1–4 cm), surrounded by edema.
   - **Thallium SPECT** (± CSF PCR for EBV) - distinguishing toxoplasmosis from primary CNS lymphoma (focal increased uptake is seen in lymphoma) - similar CT/MRI appearance.
   - **Cerebral calcifications** are not found in postnatally acquired infections!

3) **CSF** - protein↑, mononuclear pleocytosis (< 100 /mm³), glucose normal or ↓. Presence of CSF antibodies may be sensitive indicator of CNS infection.

4) **Definitive diagnosis:**
   a) 1-2 week trial of antitoxoplasma therapy (objective response must be seen on imaging; small lesions may disappear completely in matter of weeks).
   b) **Brain biopsy** - organism detection (both free tachyzoites and encysted bradyzoites may be found at periphery of necrotic foci).

**Typical toxoplasma abscesses and response to treatment** (T2- A:C,D; T1- B):

- Multiple masses of varying sizes with propensity to involve basal ganglia and gray–white matter junction; perilesional edema.
- High signal on T1-MRI due to hemorrhage.

**C.D. Response to toxoplasma therapy** - reduced size of lesions and surrounding edema; responding lesions may show increased intensity and surrounding low signal rim due to hemosiderin (arrow).

**Enhancement in toxoplasma abscess** (T2- A: T1- B): irregular rim enhancement is frequent; perilesional edema but not mass itself is involving corpus callosum.

**Treatment** - for at least 6 weeks. see above >>

- For mass effect – corticosteroids (discontinue as soon as possible).

**Prognosis**:

- Relapses occur in 50% AIDS patients and 15–25% non-AIDS patients.
- Large lesions (reduce in size and have less surrounding edema) may continue to enhance for > 2 years.

**Primary & secondary prophylaxis** in HIV-infected patients see p. 269 >>

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**Source of picture:** *WebPath - The Internet Pathology Laboratory for Medical Education* by Edvard C. Klatt, MD
- immunosuppressed* patient with unremitting fever.

**ANGIOINVASIVE**: multiple thrombotic infarctions / SAHs from ruptured mycotic aneurysms → multiple brain abscesses (in major vascular territories).

- radiologically similar to pyogenic abscesses.

Branching hyphae of *Aspergillus* invading cerebral vessel:

- Aspergillus causes 50% brain abscesses after bone marrow transplantation

*Source of picture: "WebPath - The Internet Pathology Laboratory for Medical Education" (by Edward C. Klatt, MD)

Bilateral infarction and hemorrhage (in territories of lenticulostriate perforating arteries) caused by angioinvasive *Aspergillus*:

- chest X-ray - pulmonary infiltrates; bronchoscopy may identify infecting organism in some cases.
- rapid diagnosis - *Aspergillus* antigen test in blood.
- treatment - liposomal AMPHOTERICIN B (0.8 - 1.0 mg/kg/d) + FLUCYTOSINE (25 mg/kg q6h).

New drugs for invasive aspergillosis:

- **VORICONAZOLE** (loading 6 mg/kg IV q12h for two doses → maintenance 4 mg/kg IV q12h).
- **CASPOFUNGIN** (70 mg IV over 60 min single loading dose on day 1 → 50 mg/d IV).

**CANDIDA** - see p. Inf1>>

**INTRAMEDULLARY SPINAL CORD ABSCESS**

Only < 100 cases have been reported since 1830:
- males > females
- peak incidence in 1st and 3rd decades of life.
- particular high risk factor – IV drug abuse.
- most common etiology: *Staphylococcus* and *Streptococcus* species, followed by Gr- organisms.
- solitary abscesses most likely appear in thoracic cord; abscesses may occur in areas of infarction (explaining septic spread to lower half of thoracic cord).
- **holocord abscesses** have been reported in 5 patients.
- spinal cord abscesses do not destroy fiber tracts (abscess displaces fiber tracts and spreads along axonal pathways!).

- since inflammatory process involves surrounding vasculature, spine cord infarction may lead to irreversible paraplegia.

**Chronic cases**: **RHOMI INTRAMEDULLARY TUMOR** - gradually progressing neurological symptoms predominate over those of systemic infection.

**CLINICAL FEATURES**

Acute cases - similar to **EPIDURAL ABSCESES** (but percussion tenderness is not noted) - extremely ill patients presenting with:

1) **symptoms of infection** - acute onset of back pain, fever, chills, malaise.
2) **neurological symptoms** - weakness + paraplegia, areflexia, bladder and bowel incontinence.

- since inflammatory process involves surrounding vasculature, spine cord infarction may lead to reversible paraplegia.

**DIAGNOSIS**

Neurosurgical decision of choice - gadolinium-enhanced MRI

1) **mass** (homogenous spinal cord enlargement on T1-MRI but high signal intensity on T2-MRI);
2) abscess margin enhances brightly with gadolinium.

CSF (can be within normal ranges) - protein+, pleocytosis.

Identification of infecting organism - cultures from abscess aspirate (aerobic and anaerobic bacteria, fungi, and tuberculosis) during laminectomy.

Myelography - only widening of spinal cord.

TREATMENT
1. Antibiotics - empirically broad-spectrum antipenicillilwase penicillin, minimum 4 weeks following surgery.
2. Steroids (DEXAMETHASONE) 4-10 mg q6h during entire course of treatment – to reduce spinal cord swelling.
3. Surgical drainage of abscess cavity - LAMINECTOMY one level above and below abscess edges:
   - open dura
   - identify area of spinal cord involvement (swelling, hemorrhage, distorted veins).
   - abscess aspiration for culture & stain (Gram, India ink).
   - myelotomy over length of abscess.
   - irrigate (wound and abscess cavity) with antibiotic solution.
   - closure in anatomical layers.
   - drain is optional.

PROGNOSIS
• MORTALITY 10-20%.
• significant percentage of patients have abscess recurrence - repeat MRIs are essential in long-term follow-up care (enhancement of cavity will likely continue for several weeks).

SUBDURAL EMPYEMA (CRANIAL AND SPINAL)
- pus collection in space between dura mater and arachnoid.
  - INTRACRANIAL >> SPINAL (only 50 cases reported in literature)
  - 13-20% of localized intracranial infections.
  - most common in children & young adults (70% patients are in 2-3rd decade of life).
  - males > females (3:1).

ETIOPATHOPHYSIOLOGY
• primary causes - sinusitis (esp. frontal) 50-80%, otitis media 10-20%, superficial infections of scalp and skull, craniotomy, meningitis (very rarely*), suppuration of subdural hematoma.
  *vs. in infants subdural empyema represents infected subdural effusion (complicating bacterial meningitis).
  - in spine: hematogenous spread from distant site (most commonly), trauma, spine surgery, dural sinus.
  - pathogenesis:
    a) direct spread via erosion of bone adjacent to dura mater.
    b) septic thrombophlebitis of mucosal veins (e.g. of sinuses) → retrograde extension with drainage of bacteria into regional dural veins → superior sagittal sinus → subdural space.
    - brain beneath pus is molded in manner similar to that seen in subdural hematoma.

PROGRESSION
• subdural space has no barriers (hence, empyema not abscess!)
• empyema evolution is remarkably fast (along falk and over convexities);
• subdural empyema may breach arachnoid (arachnoid is not very strong barrier) → meningitis.
• septic thrombophlebitis extends from dural sinuses to cortical veins → cortical venous infarction of gray and white matter drained by thrombosed vessels → brain abscess (20% patients!).
• with successful treatment, thickened dura may be only residual finding.

CLINICAL FEATURES
- patient is acutely ill (entire clinical picture may evolve in as little as few hours or as long as 10 days):
  1) frontal sinistis - periorbital edema and erythema, local pain and tenderness, etc.
  2) fever, chills.
  3) severe headache (often localized initially to side of infection), nuchal rigidity (70-80%).
  4) MASS EFFECT - progressive disturbance of consciousness, increase in infant head size with bulging fontanel.
  5) FOCAL NEUROLOGICAL DEFICITS (80-90% patients; caused by cortical vein thrombophlebitis): seizures (30-60% patients), hemiparesis, aphasia.

Spinal subdural empyema - fever with rapidly progressive spinal cord compression.
• backache is not as characteristic as in spinal epidural abscess.
• tenderness along spine is often absent (vs. spinal epidural abscess).

DIAGNOSIS
• marked peripheral leukocytosis.
• MRI (procedure of choice) - hypodense crescent adjacent to inner border of skull or adjoining falk with mildly (markedly on T2-MRI) increased signal intensity compared with CSF.

N.B. empyema is denser than CSF, vs. benign subdural effusion - isointense on MRI T1- and T2 with CSF
• contrast enhancement of empyema margin (fine, intense line).
• underlying parenchymal edema.
• mass effect.
• empyema extent is limited by attachments of dura (way to distinguish epidural from subdural suppurative process).

Cerebral arteriography* (formerly was used routinely) should be employed on emergent basis when MRI is unavailable and subdural empyema is strongly suspected despite normal CT - subdural avascular mass.
• *myelography for spinal empyema.

Subdural tap may be diagnostic in infants.

Lumbar puncture should be avoided; CSF is as in cerebral abscess (aseptic meningeal reaction):
• clear and colorless.
• neutrophilic pleocytosis may be absent;
**TREATMENT**

- surgical emergency!
  - anticonvulsants should be administered prophylactically.

1. **Intravenous antibiotic therapy** (same as that for brain abscess) – against organisms typically isolated from chronic sinusitis / otitis:
   - aerobic streptococci (30-50%)
   - anaerobic and microaerophilic streptococci (15-25%)
   - staphylococci (12-25%: majority of cases of spinal subdural empyema)
   - aerobic G- bacilli (3-10%)

   Empiric therapy: doses in → see p. Inf1 >>
   1) **PENICILLIN G** or 3rd-generation cephalosporin (CEFTRIAXONE or CEFOTAXIME)
   2) **METRONIDAZOLE**
   3) **Nafcillin** or **Vancomycin**

   • IV for at least 3 weeks after surgical drainage → PO to complete 6-week course.

2. **Management of increased ICP** – use of steroids (tapered rapidly after surgery) is common but remains controversial.

3. **Immediate surgical drainage**
   
   A. **CRANIAL** – via craniotomy (esp. for posterior fossa subdural empyemas) or multiple burr holes:
      • drains are left in subdural space for several days.
      • postoperatively, repeat CT / MRI scans – **reoperation** (drainage of loculated pockets) is typically necessary.
   
   B. **SPINAL** – **lamincetomy** → dural incision → drainage.

   • although extensive antibiotic irrigation of subdural space at time of surgery (BACTRACIN + VANCOMYCIN or GENTAMICIN) is common, there are no data on benefits of this practice.

**PROGNOSIS**

- mortality 10-40% (almost fatal if untreated).
- in 8-46% patients chronic epilepsy results; disabling hemiparesis or aphasia (5-25% survivors).

**CRANIAL EPIDURAL ABSCESS**

- suppurative infection in epidural space (between dura and bone).

Epidural abscesses: SPINAL >> CRANIAL (9:1)

- **etiology & pathogenesis** = subdural empyema.
  - almost always associated with **overlying infection in cranial bones** (e.g. penetration from chronic sinusitis or mastoiditis; most common cause is craniotomy complicated by wound infection).
  - hematogenous spread to epidural space from remote site of infection is extremely rare (vs. extremely common cause of spinal epidural abscess!).
  - rare in young children; median – 6th decade.
  - **MORBIDITY & MORTALITY** are low.
  - remains well localized due to tight adherence of dura to overlying cranial bones.
  - abscesses rarely dissect beyond base of skull.

**CLINICAL FEATURES**

- **slowly growing mass** (does not produce sudden major neurologic deficits unless complicated by deep extension*) - insidious clinical presentation:
  - subdural empyema, meningitis, intraparenchymal abscess

1) unrelenting hemicranial headache or persistent fever (patient may otherwise be asymptomatic?!

2) without treatment, **intracranial hypertension** and focal neurologic signs ultimately develop (when infection extends into subdural space).

**DIAGNOSIS**

MRI - superficial, circumscribed lenticular-shaped lesion of diminished density*, with **inner rim of contrast enhancement** (thicker and more irregular than with subdural empyema).**

*but higher signal intensity (on T1- and T2) than CSF
**infused dye

- MRI is free from bony artifacts adjacent to inner table of skull.
- MRI readily differentiates abscesses from sterile effusions or chronic extraxial hematotomas.
Lumbar puncture is certainly to be discouraged until after imaging has established that significant mass effect is not present:
- modest aseptic CSF reaction.

Hyponatremia is found in 30% cases.

**TREATMENT**

Antibiotic therapy → see SUBDURAL EMPYEMA

Surgical drainage - depending on extent of lesion and involvement of overlying bone:
- a) burr holes
- b) craniotomy
- c) craniectomy (debridement of infected bone)

- dural grafting may be necessary (if dura has been breached by infection).
- communications between sinus cavities and epidural space may require later surgical closure.

**SPINAL EPIDURAL ABCCESS**

- any infectious phlegmon involving epidural space, even without demonstrable contained pus (true abscess):

**Spinal epidural space**

TRUE space – posteriorly, AP width greatest where spinal cord is smallest (T4-8 and L3-S2).
- mid thoracic region (TE-8) – largest amount of epidural fatty tissue – most common location for spinal epidural abscess!

POTENTIAL space – anteriorly (because dura is adherent to posterior surface of vertebral bodies).
- anterior abscesses usually occur at cervical levels.

Can occur at all ages (60% patients are 20-50 years of age).

**ETIOPATHOPHYSIOLOGY**

a) most common (2/3) ETIOLOGY (vs. cranial epidural abscess) - HEMATOMGENOUS SPREAD from remote site:
- immune suppression (most commonly AIDS or diabetes mellitus) is predisposing condition in 50% cases.
- small hematoma (mild blunt trauma) provides locus minoris resistentiae - may allow for hematogenous spread of infection.
- hematogenous spread to vertebral body / disc (ostemyelitis / discitis) may also occur → subsequent extension into spinal epidural space.

b) DIRECT EXTENSION (1/3) from vertebral osteomyelitis is esp. common in IV drug abusers.
- other sources of direct extension - discitis, spondylitis, infected abdominal wounds, psoas abscesses, periphereal and retropharyngeal abscesses, iatrogenic complication of lumbar puncture.
- tuberculous epidural abscess is usually associated with vertebral osteomyelitis.

Spinal epidural abscess extends ≈ 3 spinal segments (may extend for as long as 13 segments).

**Spinal cord lesion**
- gross appearance of spinal cord is usually normal.
- direct compression of neural tissue & inflammatory thrombosis in intravascular vessels → infarction → MYELOMALACIA.
- microscopically - scattered areas of softening, vacuolization of cord, areas of necrosis (disappearance of cells, loss of myelin, axonal swelling).

**CLINICAL FEATURES**

1. Fever.
2. Back pain & tenderness (on percussion & movement) at affected spinal level.
- erythema and swelling in area of back pain.
- stiff neck and headache are common.

3. Progressive compression of spinal cord (appears within hours = weeks of initial symptoms):
   - radicular pain
   - paresis & loss of sensation below level lesion + bowel & bladder dysfunction
   - complete paraplegia & loss of all sensory modalities below level of lesion (i.e. transection syndrome)

**DIAGNOSIS**

MRI (procedure of choice) – abscess isointense to CSF (hyperintense on T2).
- contrast enhancement of lesion occurs.
Lumbar puncture (demonstrates subarachnoid block) should not be performed:
1) risk for infection spread to subarachnoid space.
2) risk for herniation from decompression below area of obstruction
   • if meningitis is suspected - high cervical tap is often safest approach.
Normal plain CT alone does not exclude diagnosis (if MRI is unavailable → use CT myelography).

Myelography (not necessary if abscess is diagnosed by MR1/CT) - extradural block (complete in 80% patients)
   • performed by cervical puncture.
   • needle is advanced slowly and suction applied with syringe as epidural space is approached - if abscess has extended to level of puncture, pus is withdrawn → terminate procedure (use pus for culture).

X-rays show osteomyelitic findings in 1/3 cases.

TREATMENT
Immediate surgery: laminectomy - decompression - drainage of epidural space.
   • granulation tissue is commonly found in association with epidural abscesses and may require excision during course of decompression.

Antibiotics 4-6 weeks IV → 2-3 months oral.
   • empiric antibiotics should cover:
     1) S. aureus (etiologic agent in majority of cases!!!) – NAPCILLIN.
     2) M. tuberculosis (ISONIAZID, RIFAMPIN, PYRAZINAMIDE & STREPTOMYCIN or RIFABUTIN or CLOFAMINE).
     3) most authorities would provide additional Gr coverage (3rd-generation cephalosporin, quinolone, or aminoglycoside).

PROGNOSIS
- relates inversely to amount of neurologic dysfunction at time of diagnosis:
  - only pain → recovery without deficit.
  - some weakness → 50% will achieve complete resolution.
  - paralysis < 36 hours’ duration → < 50% will show some return of motor function.
  - in tuberculous extradural abscess motor recovery has been reported even after paralysis lasting for weeks.

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