Review the signs and symptoms of impending intracranial complications. Compare and contrast the presentation of the different intracranial complications. How do you work these patients up?

Spread of infection occurs through 3 routes, namely, direct extension, thrombophlebitis, and hematogenous dissemination. Extracranial complications are usually direct sequelae of localized acute or chronic inflammation. The complications of otitis media include the following:

- **Chronic suppurative otitis media**[^3,4]
- Postauricular abscess
- Facial nerve paresis
- Labyrinthitis
- Labyrinthine fistula
- Mastoiditis
- Temporal abscess
- Petrositis
- Intracranial abscess
- Meningitis
- Otitic hydrocephalus
- Sigmoid sinus thrombosis
- Encephalocele
- Cerebrospinal fluid (CSF) leak

The following signs or symptoms are suggestive of intracranial complications:

- Fever associated with a chronic perforation
- Lethargy
- Focal neurologic signs (eg, ataxia, oculomotor deficits, seizure)
- Papilledema
- Meningismus
- Altered mental status
- Severe headaches

Presentation of intracranial complications includes the following:

- Brain abscess - Fever, possibly seizures or focal neurologic signs, headache
- Meningitis - Fever, meningismus
- Otitic hydrocephalus - Headache, signs of increased intracranial pressure in the setting of otitis media
- Sigmoid sinus thrombosis - Spiking fever, otitis media, edema and tenderness over mastoid cortex, headache

**Intracranial complications**

A brain abscess may occur in the temporal lobe or cerebellum, typically from chronic otitis media. An epidural abscess may occur as a result of bony destruction and extension...
from coalescent mastoiditis or cholesteatoma.

Meningitis may be associated with acute or subacute/chronic infection. Acute otitis media is the most common cause of meningitis. Extradural granulation tissue or frank pus may be found.

In adults and children, meningitis in the setting of chronic suppurative otitis media may be secondary to the direct extension of infection through the dura, through a previous stapedectomy site, or through a cholesteatoma-induced labyrinthine fistula.

A sigmoid sinus thrombosis or subdural abscess/empyema may be associated with otitis media. Otitic hydrocephalus may occur as a result of increased intracranial pressure secondary to middle ear infection and complicated by sigmoid sinus thrombosis with total occlusion.

Work-up includes

Imaging: CT first, then MRI

Monitoring of visual acuity and visual fields is essential in otitic hydrocephalus. If deterioration is progressive, fenestration of the optic nerve sheath may be warranted.

Lumbar puncture is indicated in suspected meningitis. Avoid this procedure until imaging studies are performed to exclude a brain abscess. Brain herniation can result in the face of sudden release of intracranial pressure if a brain abscess is present.

Obtain cultures from the septic focus to guide therapy; however, they may fail to reveal the true pathogen in at least 25% of cases of otorrhea

CBC for leukocytosis

What is the treatment of lateral sinus thrombosis? What is the role of anticoagulation?

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At Emory, six cases of lateral sinus thrombosis (LST) were seen over the last ten years — two children and four adults.

The picture and bacteriology of LST have changed with the advent of antibiotics as has the usefulness of various diagnostic tests. Less is it a disease of children in association with acute otitis media. More often it is seen in the adult patient after a long history of chronic ear disease. Fever and mastoid and neck tenderness are still universal signs of the affliction. However, rarely patients do present with progressive anemia, emaciation and
evidence of septic emboli. Since antibiotics are commonly used during the prodromal ear infection, cultures are often negative. If they do identify an organism, it is usually a mixed flora rather than *beta hemolytic streptococcus*. Spinal fluid results are variable and seldom is there evidence of increased spinal fluid pressure. Arteriography, venography, and digital subtraction venography are the most reliable tests to prove and delimit the thrombus.

Early management involves high dose broad spectrum, intravenous antibiotics including chloramphenicol. Surgical intervention involves a mastoidectomy, exposure of the sinus, incision and drainage, but not necessarily removal of the thrombus. Internal jugular vein ligation should be reserved for those cases in which septicemia and embolization do not respond to initial surgery and intravenous antibiotics.

In selected cases of lateral sinus thrombosis, medical therapy alone with intravenous antibiotics may be successful.

Most authors agree that anticoagulants have no place in the management of lateral sinus thrombosis. Anticoagulants have been advocated to prevent extension of the thrombus to the distal sinuses. However, they are rarely used now because most infections can be controlled with antibiotics and surgery, and this tends to prevent the thrombus from propagating. The risks of anticoagulation include releasing septic emboli from clot breakdown and uncontrollable hemorrhage at the bleeding site. Anticoagulants arrest the spread of thrombosis but may increase the risk of venous infarctions and are therefore no longer used.

Systemic anticoagulation is not necessary unless the clot is shown to involve the sagittal sinus, or signs of increased intracranial pressure persist despite medical management.

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**What is the etiology and management of brain abscess, epidural abscess, subdural empyema?**

**Intracranial complications of otitis media: 15 years of experience in 33 patients**

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Brain abscess more commonly related to chronic OM, with cholesteatoma. Interestingly, otogenic brain abscess related to otitis media is always located adjacent to the temporal bone, almost exclusively at the temporal lobe and cerebellum2-7 (Figs 1 and 2). In our view, this characteristic location is also indicative of the main mechanism of infection that progresses through the osseous limits of the temporal bone by means of osteitis and erosion of bone, as mentioned above. This position of the abscess allows, in a reasonable number of cases, simultaneous drainage through the mastoid approach, avoiding an additional craniotomy.
Lumbar or occipital puncture for a diagnosis of meningitis deserves careful attention. The possibility of occurrence of more than 1 ICC is common, and the presence of intracranial hypertension or a mass effect would promote encephalic herniation with acute and life-threatening repercussions. It is recommended that lumbar or occipital puncture should be conducted only after a cranial imaging study.

The occurrence of lateral sinus thrombosis (LST) and empyema were less frequent in our series, and these findings agree with those reported in the literature. Nevertheless, it is our impression that the presence of LST can, in many circumstances, be underestimated. Some patients with this complication may follow a course without major clinical expression or concomitant with another evident ICC, and definitive identification of the LST would only be possible through an angioresonance study with venous acquisition or on angiography, both of which are examinations that are not routinely performed.

During this study we also had the opportunity to identify the presence of sinus thrombosis only after the initial treatment, during imaging used as a control in a follow-up period, as mentioned by other researchers (Fig 3). This finding should alert clinicians to the possibility of the presence of a clinically unexpressed lateral sinus thrombosis and the benign course of this complication on many occasions. Because of this characteristic, the treatment of LST is under critical analysis, and one can observe a tendency toward a more conservative approach. Surgery should be directed to the primary infectious disease, using sinus decompression in association with puncture to confirm the diagnosis.

More aggressive surgical maneuvers, such as ligature of the jugular vein or a large opening of the lateral sinus, must be considered only in those few cases with evident progression of the infectious thrombus-embolic disease. The use of anticoagulants for long periods has also been questioned.

Epidural abscess: Intracranial epidural abscess often has an insidious onset, with symptoms developing over several weeks to months. Symptoms of the initiating infection might dominate the picture. Signs and symptoms are as follows:

- Usually, the patient presents with headache that is either diffuse or localized to one side with scalp tenderness. Headache may be the only presenting symptom. The patient may have persistent fever that develops during or after treatment for sinus or middle ear infection. Purulent discharge from the ears or sinuses, periorbital swelling, and brawny edema of the scalp might accompany.
- Because the epidural abscess usually enlarges slowly, the following signs do not develop until the infection has reached the subdural space, resulting in subdural empyema, at which time the patient might present with neck stiffness, nausea, vomiting, lethargy, and hemiparesis. Seizures might very well be the first
presenting symptom in some cases.

- Symptoms and signs of increased intracranial pressure (ICP) include nausea, vomiting, and papilledema. Rarely, when the epidural abscess develops near the petrous bone and involves the fifth and sixth cranial nerves, the patient may present with ipsilateral facial pain and weakness of the lateral rectus muscle (ie, the so-called Gradenigo syndrome). Many times, scalp cellulitis, sinusitis, or skull fracture may draw the attention of the physician to such an extent that the diagnosis of epidural abscess may be missed.
- One should consider the diagnosis of intracranial epidural abscess when a patient presents with unresolving frontal sinus symptoms. Also consider this diagnosis in patients with new neurologic symptoms after trauma or cranial surgery, even if months or years have elapsed since operation or trauma.
- Onset can be acute, especially in patients without any history of previous cranial neurosurgery. They often present with acute symptoms of encephalopathy and focal neurological deficits.

Early diagnosis and treatment of epidural abscess cannot be overemphasized as neurologic outcome mainly depends on the patient’s neurologic status immediately prior to surgery.

- Prehospital management
  - Rapid transport and early stabilization are highly essential in the prehospital setting.
  - Endotracheal intubation and hyperventilation may be required in some patients who are critically ill.
- Initial management (depends upon the type of clinical presentation)
  - The presence of seizures and focal neurological deficits requires emergent intubation, anticonvulsant therapy, hyperventilation, and hemodynamic stabilization before proceeding with diagnostic tests.
  - Patients who are not critically ill or who have a subtle presentation may undergo CT scanning after initial clinical evaluation. Neurologic status should be monitored closely.
- Antibiotic therapy
  - Until the culture and sensitivity report of the infectious agent becomes available, the choice of empiric antibiotic therapy should be based on the underlying etiology. For example, when an intracranial abscess is thought to be due to extension of infection from paranasal sinuses involving staphylococcal, aerobic, and anaerobic bacteria, more than one antibiotic is necessary. Likewise, an antistaphylococcal agent would be an appropriate choice for infection occurring after a neurosurgical procedure.
  - For patients presenting in the emergency department (ED) with cranial epidural abscess, empirical antibiotics are the first-line pharmacologic therapy. These antibiotics must cover a broad spectrum of both aerobic and anaerobic bacterial organisms.
  - Usually, length of therapy is determined by the patient's response to treatment and by resolution of the epidural abscess on follow-up MRI and/or CT scanning. As a general rule, antibiotic therapy should be
continued for a minimum of 8 weeks if surgery is not undertaken and for at least 4 weeks if the abscess is drained. Antibiotics have been administered from 6 weeks to 6 months. In general, follow-up CT scanning or MRI should be obtained 10-14 days after antibiotic therapy has been discontinued.

- Seizure therapy
  - Prophylactic seizure therapy is not generally recommended. If cranial epidural abscess is not associated with subdural empyema, seizures are unlikely to ensue. In the event of the administration of anticonvulsant therapy, consider weaning patients off anticonvulsant therapy if patients remain seizure free for more than 2 years and the EEG findings do not show any evidence of seizure disorder.
  - Discontinuing anticonvulsant therapy suddenly can be risky because it can lead to recurrent seizures, which may be prolonged. This is true even if the medication was not successfully controlling the seizures. Weaning patients off the drug gradually after fully understanding the potential possibility of recurrent seizure(s) and related consequences, including losing a driving license and the possible impact on employment, is strongly advised. If seizures do recur, resuming the previous medication immediately usually results in the same level of seizure control as before. However, in rare instances, the original antiepileptic medication may not be as effective, even if previously successful; alternative therapy should be considered.

Surgical intervention is an integral part of treatment for epidural abscesses in patients with neurologic symptoms or who have not responded to medical management.

- Optimal management of an intracranial epidural abscess should include neurosurgical drainage; Gram stain, India ink, and acid-fast bacilli (AFB) staining of the purulent material; and administration of appropriate intravenous antibiotic(s). In case of small abscesses, adequate appropriate antibiotic therapy alone might suffice, without the need for surgical intervention.
- The goal of therapy is to eradicate the infection and prevent further complications. Surgical exploration, decompression, and debridement, along with antibiotic therapy, are the mainstays of surgical treatment in cranial epidural abscess.
- The type of emergency surgery for cranial epidural abscess depends on the extent of the lesion and involvement of the overlying skull bone.
  - When burr holes cannot provide sufficient drainage or when debridement with drainage is indicated, craniotomy is undertaken.
  - When the dura is affected by infection, a dural graft may be required. During anesthesia, anesthetics that can cause intracranial vasodilation should be avoided because this might result in further increase in intracranial pressure, heralding herniation.

A patient with subdural empyema could present with any of the following symptoms:

- Fever - Temperature above 38°C (100.5°F)
• Headache - Initially focal and later generalized
• Recent history (< 2 wk) of sinusitis, otitis media, mastoiditis, meningitis, cranial surgery or trauma, sinus surgery, or pulmonary infection
• Confusion, drowsiness, stupor, or coma
• Hemiparesis or hemiplegia
• Seizure - Focal or generalized
• Nausea or vomiting
• Blurred vision (amblyopia)
• Speech difficulty (dysphasia)
• History of intracerebral abscess (recent or in the past)

Antibiotic therapy\(^4\) alone may be adequate for small subdural empyema (ie, < 1.5 cm diameter). Because of the aggressive nature of this disease, however, this option is not widely utilized. This is an option for patients with major contraindications to surgery or significant mortality risks.

Other medical interventions may include medications for seizure treatment or prophylaxis. Treatment for increased intracranial pressure also has been advocated.

Immediate neurosurgical drainage\(^5\) of the subdural empyema should be considered. The primary surgical option is craniotomy, which allows wide exposure, adequate exploration, and better evacuation of the purulent collection than other procedures. Stereotatic burr hole placement with drainage and irrigation is another option but is less desirable because of decreased exposure and possible incomplete evacuation of the purulent material.

Drainage and debridement of the primary source of infection may be necessary. Samples should be collected for Gram staining, culture, and sensitivity tests.