Case Report

Parafalcine Subdural Empyema-A Rare Entity

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Subdural empyema (SDE) is a significant cause of neurological morbidity and mortality. It should be recognized early and treated as an emergency. Although convexity subdural empyema is not so uncommon but parafalcine subdural empyema is quite rare. A recent case of parafalcine subdural empyema managed at Prince Abdullah Bin Abdulaziz Hospital, Bisha (KSA) is discussed. The epidemiology, pathophysiology, bacteriology, clinical features, radiographic findings and treatment of this life-threatening entity are reviewed.

Key Words: Parafalcine, Subdural empyema, Pathophysiology, Treatment

Case Summary: SSAB is a 12 years old male who presented to the ER with 4 days history of headache, vomiting and fever. He had an episode of focal fits to the left leg. There was H/O chronic sinusitis as well.

On examination, he was conscious, oriented & febrile (39° C) & had weakness of the left leg (grade 0). Neck stiffness & Kernig's sign were positive. White blood cell count was elevated (20.7) with 87% PMNs & 58% band forms.

CT scan brain (plain & with contrast) showed a right parafalcine subdural fluid collection that extended from frontal to occipital region and inferiorly to the tentorium with leptomeningeal enhancement (Figure 1). Extensive (maxillary) sinus disease was also noted.

A diagnosis of parafalcine subdural empyema was made, and the patient was started on broad-spectrum antibiotics and antiepileptic treatment. He was taken for urgent surgery for craniotomy and drainage of pus. He underwent two separate craniotomies (Right frontal & parietal). Thick pus was drained from the right parafalcine space & washed thoroughly with normal saline (with the help of a thin soft catheter) (Figure 2). Drains were left in the interhemispheric fissure in the frontal & occipital regions and were removed after 48 hours. Gram staining of the pus did not show any organisms and culture & sensitivity showed "no growth". He made a good postoperative recovery. Follow up CT scans did not show any collections (Figure 3). Antibiotics were continued for 6 weeks & antiepileptic treatment for one year.

Introduction

Infections involving the central nervous system (CNS) present in a variety of ways and unless diagnosed and treated promptly can result in serious morbidity and mortality. Intracranial infections encountered in neurosurgical practice include bacterial meningitis, brain abscess and subdural empyema. Subdural empyema is a collection of pus between the dura and the leptomeninges and frontal sinusitis is the most common cause of a subdural empyema[1,3]. Contrary to prevailing opinion, SDE is not a rarity (about one-third as frequent as cerebral abscess)[4]. It often occurs as a complication of paranasal sinusitis, otitis media, meningitis and osteomylitis. SDE may also develop as a complication of head trauma or following neurosurgical procedures[2,3,5,7,8,9,10,11]. Pyemia from the sources such as lung abscess or infected heart valves is a rare cause of subdural empyema[4,8]. SDE accounts for about 13% to 20% of all cases of intracranial infections[11]. The early description of subdural empyema as a pathological entity has generally been attributed to Richter in 1773 and Courville gave an excellent historical background of this disorder in 1944. Other early cases of SDE have been reported by Schutz, Paulsen, Carver and Macewen[7].

History & Epidemiology: Despite advances in imaging and the availability of antibiotics, subdural empyema remains a disease process that carries significant morbidity and mortality. Prior to 1948, any case of subdural empyema was considered fatal, but antibiotics, combined with neurosurgical intervention, have helped to decrease the mortality rate to 15-30%. A male predominance exists, and 70% occur within the second and third decades of life. The majority is found supratentorially and usually occurs over the hemispheres but rarely in the parafalcine space[3,4,8].

Pathophysiology: Frontal sinusitis is the most common cause of subdural empyema[1,3,8,10,11]. Initial investigators proposed thrombophlebitis spreading from chronic suppuration of the ear as the cause of subdural empyema, but further analysis of the pathophysiology has shown that the thrombophlebitis originates most commonly in the paranasal sinuses[3,4,7,8,10]. The infection begins with a pansinusitis that leads to a progressive thrombophlebitis that then spreads through mucosal veins to emissary veins.

As the emissary venous network is valveless, this allows for subdural spread to occur[4,8,11]. Sometimes, it may be by direct spread from the frontal sinus or the ear by an osteitic process[3,4,8,10]. Cerebral convexities and interhemispheric fissure are the most common locations[1]. Which part of the subdural space gets involved depends mainly on three things: point of origin, gravity, and anatomical barriers. The falx and tentorium tend to limit the spread to an area over one hemisphere. Parafalcine spread may progress posteriorly or under the falx to the opposite hemisphere[10]. Occasionally, SDE is restricted to the parafalcine area[8].

SDE is often associated with other intracranial infections including epidural empyema (40%), cortical thrombophlebitis (35%), and intracranial abscess or cerebritis (>25%)[8]).

Bacteriology: The organisms responsible for subdural empyema vary with the primary source of infection[10] (Table 1). The majority of the cases are of sinus origin and in these cases streptococci (nonhemolytic and viridans) are the most common organisms, followed by anaerobic streptococci (often Strep. milleri) or Bacteroides. Less often Staphylococcus aureus, E. coli, Proteus, and Pseudomonas are the causative organisms[3,4,5].

Table 1: Initial Infections and Organisms Commonly Isolated in Subdural Empyema[4,5,8,10]

Source of initial infection	Organisms
Paranasal sinuses	Alpha-hemolytic streptococci
	Anaerobic streptococci
	Nonhemolytic streptococci
	Streptococcus milleri
	Haemophilus influenzae
	Staphylococcus aureus
Trauma	Staphylococcus aureus
	Staphylococcus epidermidis
	Gram-negative bacilli
Postneurosurgical wound infection	Staphylococcus aureus
	Staphylococcus epidermidis
	Gram-negative bacilli
Meningitis	Haemophilus influenzae
	Escherichia coli
	Meningococci
	Streptococcus pneumoniae
Otitis media	Alpha-hemolytic streptococci
	Anaerobic streptococci
	Haemophilus influenzae
	Gram-negative anaerobes
	Bacteroides

Clinical Features: When making the diagnosis of subdural empyema, one must pay close attention to the patient's history. Any patient presenting with a history of sinusitis and neurologic symptoms should raise the index of suspicion for subdural empyema because the most common predisposing factor for SDE is paranasal sinusitis. In patients who are secondary to paranasal sinusitis, periorbital edema and erythema may be present[4,7,8,9]. These patients are systemically unwell, febrile, complaining of headache and usually have distinct nuchal rigidity, with rapidly progressive neurological signs, a depressed level of consciousness and focal deficits[2,3,4,6,7,8,10,11]. Epilepsy occurs in most patients (approximately 60% of patients) and can often progress to status epilepticus[4, 7, 8, 9, 10]. The combination of fever and seizures with a background of sinusitis is usually diagnostic of this lesion[5].

Invariably, the patient with subdural empyema will have fever, but the clinical triad of sinusitis, acute febrile illness, and neurologic deficit remains a classic presentation for subdural empyema[9]. Because the infection involves the leptomeninges, patients may present with headache (usually frontal), meningeal signs, focal findings, seizures and signs of increased intracranial pressure[3,8]. When focal signs are evident they usually herald deterioration in the mental status that can quickly progress to coma, if left untreated.

When the collection of pus is parafalcine, the patient may present with what is called as "falx syndrome". It is manifested by motor and/or sensory dysfunction of contralateral lower limb, particularly distally. It includes focal deficits and irritative dysfunction with seizures starting from the foot. Interhemispheral pus on one side may also track under the falx to involve the corresponding area on the opposite side, in which case the patient may present with paraparesis or paraplegia[8].

Raised ICP may result in third and sixth cranial nerve palsies and in signs of brainstem dysfunction. In infants, subdural empyema commonly develops secondary to infected subdural effusion associated with purulent meningitis[3]. Thus a past history of meningitis is of particular importance. There may be enlargement of the head, bulging of the fontanelles & separation of the sutures[7,8].

Diagnosis: Routine laboratory studies will usually demonstrate a leukocytosis[4,8,10] with a shift to the left, and blood cultures may or may not reveal an organism.

Lumbar puncture may be dangerous, is contraindicated, and adds very little critical information[3,4,8,10]. Nevertheless, if CSF is obtained, there is a fair amount of CSF data. The CSF pressure is high, the sugar content is normal, and there is pleocytosis in the range of 50 to 1000/mm^3, polymorphonuclear cells predominating and elevated

protein content (75 to 300 mg/dl). Commonly, gram stain will not demonstrate any organisms, and CSF cultures are usually negative[4,8,9,10].

Radiographic Findings: The diagnosis of subdural empyema is strongly based on the clinical presentation, but radiographic studies are mandatory to a complete work up[8]. Despite being a surgical emergency, diagnosis is often delayed as the collection on CT scanning is usually subtle to the untrained eye and frequently missed[5].

Plain films will sometimes show opacification of the sinuses, mastoiditis, diastasis of the sutures in an infant and a shift of the pineal gland in an adult. These may be the first clue to the diagnosis of SDE[8,9,10].

Both CT and MRI give information that is diagnostic. Hemispheric empyemas appear as extra-axial, crescent-shaped, hypodense or isodense areas on CT that enhance along the margins because of the leptomeningeal inflammation along with mass effect[1,2,4,7,8,10,11].

It must be kept in mind that occasionally CT scan, even with enhancement, may be inconclusive, even when clinical findings are well developed. Therefore, discarding a likely diagnosis of SDE as a result of inconclusive findings on the CT (or on any other single test) is unwarranted. Other tests may have to be used, or repeat CT scan if time permits. It is unlikely that all the tests would be false-negative[8]. Cerebral angiography was a very useful diagnostic tool before the advent of CT scan[8].

Recently, MRI has become the diagnostic modality of choice because of its ability to detect collections earlier than CT. Several studies have found MRI to be superior to CT scanning in demonstrating the presence and extent of a subdural empyema because of its ability to scan in multiple planes[1,3,4,7]. MR images demonstrate subdural collections that have a low signal on TI-weighted sequences and a high signal on T2-weighted sequences. Also, the edema shows up well on the T2 images. MRI provides greater clarity than CT and has a higher sensitivity for small subdural empyemas not detectable by CT. Cranial MRI is also extremely valuable in identifying sinusitis, other focal CNS infections, cortical venous infarction, cerebral edema and cerebritis[3].

Under unusual circumstances, when none of the above mentioned tests are readily available and there is compelling suspicion for a diagnosis of SDE, emergency burr holes judiciously placed may be both diagnostic and therapeutic[8].

Differential Diagnosis: To think of its possibility is often halfway to the diagnosis of SDE and early diagnosis and prompt and intensive treatment do much to improve the prognosis. But it still carries a formidable morbidity and mortality (as high as 40% in some series)[8].

Differential diagnosis includes extradural abscess, brain abscess, pyogenic meningitis, herpes simplex encephalitis, cavernous or lateral sinus thrombosis, necrotizing hemorrhagic leukoencephalitis, septic embolism (due to bacterial endocarditis) and cortical thrombophlebitis[3,4,8,10].

Treatment: SDE is a neurosurgical emergency[8,11]. The treatment is as for brain abscess but the collection requires a formal craniotomy for drainage with exploration of the interhemispheric fissure[9]. Once the diagnosis of subdural empyema has been made, emergent neurosurgical evacuation of the empyema, either through burr-hole drainage or craniotomy, is the definitive step in the management of this infection.

Empirical antibiotic therapy should include a combination of a third-generation cephalosporin (e.g., ceftriaxone or cefotaxime) to cover aerobic streptococci, vancomycin (to cover staphylococci) and metronidazole (for anaerobes). Cultures taken at the time of surgery will help modify the antibiotic regimen. Parentral antibiotic therapy should be continued for a minimum of 4 weeks[2,3,4,7]. The decision to switch to oral medications should be based on the patient's neurologic status, the absence of fever and evidence by CT or MRI of resolution of the collection. Antibiotic therapy should continue for a total of six weeks[4,7,8,10].

The fact that appropriate antimicrobial therapy has now been identified and established for use after surgical drainage does not mean that chemotherapy alone can be relied upon to arrest the progression of inflammatory process to frank pus formation with its associated rise in intracranial pressure[10].

Some debate exists in the neurosurgery literature concernng the best surgical procedure for drainage but craniotomy has been shown to result in less morbidity and mortality than the placement of burr holes for drainage. Craniotomy provides a better exposure resulting in a more complete evacuation of the empyema under vision while treatment with burr holes results in a higher incidence of reoperation secondary to inadequate initial drainage[7,8,9,10].

Parafalcine collections frequently extend along the entire length of the falx and may require clearance by two separate routes to effect adequate drainage. The best approach is probably a combination of a small craniotomy at the frontal pole extending well up to the midline to provide a clear view backwards into the parafalcine space, together with separate drainage of the occipital loculus[10].

It is preferable to leave a drain, to be removed after 48-72

hours. The use of subdural antibiotic irrigation is controversial[9].

The primary source must be treated surgically as a matter of urgency to prevent reinfection. The frontal sinus must be drained, and in the case of an otogenic source, a radical mastoidectomy is carried out[2,8,10].

Adjunctive Therapy: Other medical therapies appropriate to consider are drugs to control cerebral swelling and anticonvulsants. Whether or not seizures have occurred, anticonvulsants are always required, as subdural pus is highly epileptogenic and dilantin can both prevent and control resultant seizure activity. Anticonvulsant treatment should therefore probably be continued for at least 5 years (2 years in some studies) after surgery[7,8,10].

The empyema can cause thrombosis of the cerebral veins resulting in considerable edema, and corticosteroids and mannitol will help curtail the swelling. According to some other studies, there is no clear rationale for the use of steroids in subdural empyema and their use must be weighed against their adverse effects on host resistance to infection[8,10].

Outcome: Prognosis is influenced by the level of consciousness of the patient at the time of presentation, the size of the empyema and the speed with which therapy is instituted. Long-term neurologic sequelae, which include seizures and hemiparesis, occur in up to 50% of cases and serious, disabling morbidity in approximately 20% [3,9].

Complications include refractory status epilepticus, cortical vein/venous sinus thrombosis and infarction[9,11]. Despite recent improvement in surgical technique and antibiotics, the mortality remains high (25% to 40%)[7,8,10,11].

Without massive antimicrobial therapy and surgery, most patients die, usually within 7 to 14 days. On the other hand, patients who are treated promptly may make a surprisingly good recovery, including full or partial resolution of their focal neurologic deficits[4].

Conclusion: Several factors influence the prognosis in subdural empyema (Table 2), but prompt diagnosis and treatment remain the most important indicators for a favorable outcome. It should be remembered that the diagnosis of subdural empyema is strongly based on close attention to the patient's history and clinical presentation as well as a high index of suspicion. Subdural empyema is a neurosurgical emergency that left untreated will invariably become fatal. It should be at the top of the differential diagnosis in any patient presenting with a history of sinusitis and a change in mental status. Compared to burr-hole drainage, craniotomy is a better option as it helps in better drainage of pus under vision, and so a better outcome. Antibiotics must be continued for at least 4 to 6 weeks and anticonvulsants for at least 2 years.

Table 2: Factors Influencing Outcome in Subdural Empyema[7]

Good prognosis	Patient age: 10-20 years
	Paranasal sinuses as site of initial infection, Aerobic streptococci isolated
	Craniotomy performed
	Patient awake and alert at the time of Presentation
Poor Prognosis	Patient age: 10 years or younger, or elderly, Cultures sterile
	Patient comatose on presentation

References:

1. Sutton D., Stevens J. and Miszkiel K.: Intracranial Lesions (2), in Sutton D. (ed.): Textbook of Radiology & Imaging, Churchill Livingstone (USA), 2003 (7th Edition), Vol.2, p 1785.
2. Allen C.M.C., Lueck C.J.: Neurological Disease, in Haslett C., Chilvers E. R., Boon N. A., Colledge N. R. (ed): Davidson's Principles and Practice of Medicine, Churchill Livingstone (USA), 2002 (19th. Edition), p. 1200.
3. Roos K.L. & Tyler K.L.: Bacterial Meningitis and other suppurative infections, in Braunwald E., Fauci A. S., Kasper D.L., Hauser S.L., Longo D. L. Jameson J. L. (ed): Harrison's Principles of Internal Medicine, Mc Graw-Hill; Medical Publishing Division (USA), 2001 (15th. Edition), Vol.2, pp 2469-2470.
4. Victor M., Ropper A.H.: Infections of the Nervous System, in Adams and Victor's Principles of Neurology, Mc Graw-Hill (USA), 2001 (7th. Edition), pp 749-750.
5. Kerr R.C.S. & Maartens N.F.: The Cranium (scalp, skull, brain), in Russell R.C.G., Williams N.S. & Bulstrode C.J.K. (eds): Bailey & Love's Short Practice of Surgery, London: Arnold, 2000, pp 555-557.
6. Girolami U.D., Anthony D.C., Frosch M.P.: The Central Nervous System, in Cotran R.S., Kumar V., Collins T. (ed): Robbin's Pathologic Basis of Disease, Harcourt Asia PTE, LTD (India), 1999 (6th. Edition), p. 1316.
7. Haines S.J., Mamplam T., Rosenblum M.L. and Nagib M.G.: Cranial and intracranial bacterial infections, in Youmans JR (ed.): Neurological Surgery, Vol 6. Philadelphia: W.B. Saunders, 1990, pp 3720-3722.
8. Smith B.H.: Infections of the dura and its venous sinuses, in Joynet R.J. (ed.): Clinical Neurology, Philadelphia: J.B. Lippincott Company, 1988, pp 8-12, volume 2, chapter 23.
9. Renaudin J.W.: Cranial epidural abscess and subdural empyema, in Wilkins R.H., Rengachary S.S. (eds): Neurosurgery, Vol. 3. New York: McGraw-Hill, 1985, pp 1961-1963.
10. Strong A.J. & Ingham H.R.: Surgical and microbiological management of subdural and extradural abscesses, in Symon L., Thomas D.G.T. and Clarke K. (ed.): Rob & Smith's Operative Surgery; Neurosurgery Volume, London: Butterworth & Co. (Publishers) Ltd. 1983 (4th. Edition) pp 94-100.
11. Zee C-S., Go J.L., Kim P., Segall H.D., Ahmadi J.: Cerebral Infections and Inflammation, in Haaga J.R., Lanzieri C.F.,

Gilkeson C. (ed): C.T. and M.R. Imaging of the Whole Body, Mosby (USA), 2003 (4th. edition), volume-1; p 208.

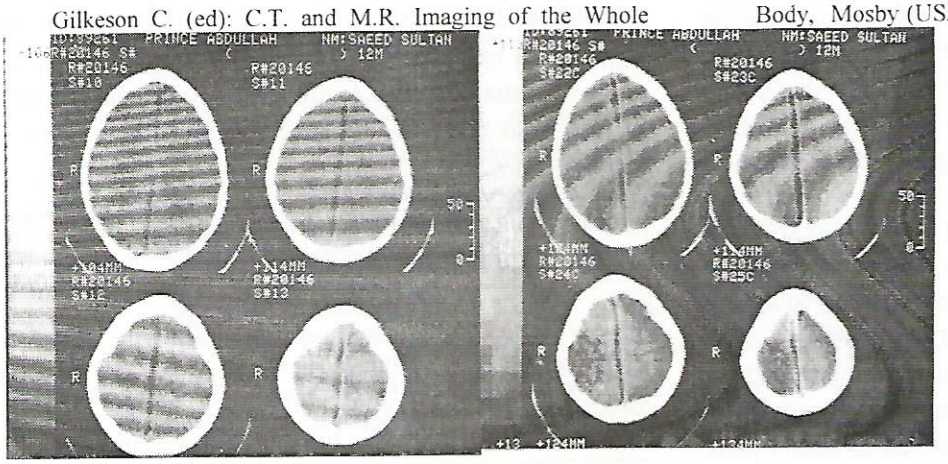

Figure 1: CT scan brain (plain & with contrast) demonstrating parafalcine subdural empyema on right side.

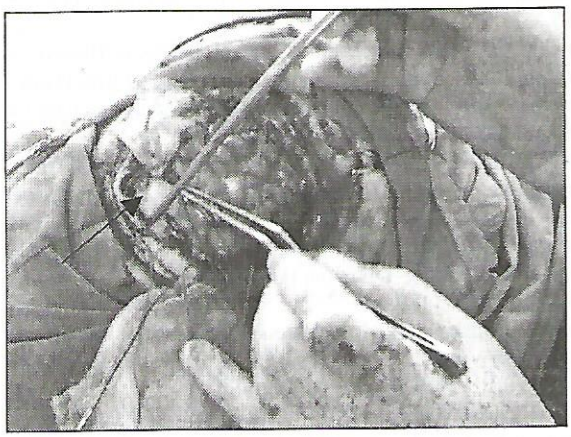

Figure 2: Peroperative picture showing thick pus coming out from interhemispheric fissure (arrow) in frontal region.

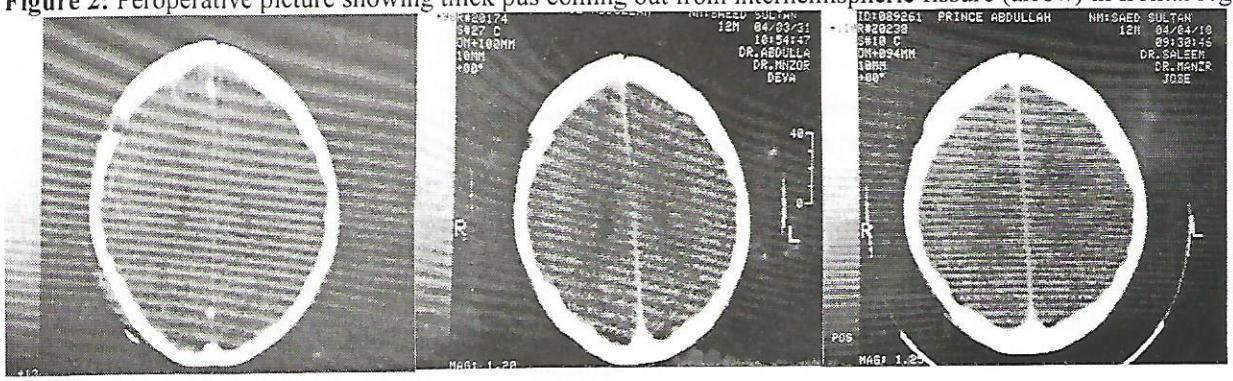

Figure 3: CT scans brain (with contrast) at 1, 2 & 4 weeks postoperatively demonstrating no parafalcine subdural collection.