

Intracranial and Orbital Complications of Sinusitis: A Case Series and Review of Literature

¹Vinay Vaidyanathan, ²Kishore Shetty

¹Assistant Professor, Department of ENT, KS Hegde Medical Academy, Mangalore, Karnataka, India

²Professor, Department of ENT, KS Hegde Medical Academy, Mangalore, Karnataka, India

Correspondence: Vinay Vaidyanathan, Assistant Professor, Department of ENT, KS Hegde Medical Academy, Mangalore Karnataka, India, e-mail: drvinayvrao@gmail.com

ABSTRACT

Complications of paranasal sinus infection most often involve the orbit and periorbita. Because of widespread use of antibiotics, intracranial extension of paranasal sinusitis is rarely seen today. Nevertheless, the clinician must be aware of the potential of these complications, as late recognition of this condition and delay in treatment can increase morbidity and mortality rates. An interesting case series of sinusitis with orbital and intracranial complication is presented, which was radiologically evaluated, and was managed by endoscopic sinus surgery with drainage of subdural empyema by appropriate neurosurgical technique. The radiological tools played a very important role in both assessment and timing of surgical intervention. Unparallel role of radiological investigations cannot be overemphasized. The key to successful treatment is aggressive management and the timing for surgical intervention should not be deferred. The patients made full recovery at the time of discharge.

Keywords: Intraorbital complication, Intracranial suppurative complication, Cavernous sinus thrombosis, Superior sagittal sinus thrombosis.

INTRODUCTION

Complications of paranasal sinus infection most often involve the orbit and periorbita. Because intracranial extension of sinus disease is infrequently seen today, the clinician may be unfamiliar with the evaluation and management of this potentially devastating complication. Despite recent advances in treatment and diagnostic imaging, intracranial extension is often not recognized early enough to prevent delays in treatment aimed at reducing morbidity and mortality.^{1,2} In the reported cases, intracranial suppurative complication was detected at variable times after admission even though one of the patient presented with orbital complication. Superior sagittal sinus thrombosis is relatively a rare dural venous sinus complication than cavernous sinus thrombosis both of which were seen in one of the cases. A kaleidoscopic neurological presentation and full recovery made by all the patients at the time of discharge made these cases more interesting. Intracranial complications are most often associated with infections of the frontal, ethmoidal and sphenoidal sinuses. Maxillary sinusitis rarely extends intracranially; however, odontogenic maxillary sinusitis has shown an increased tendency for intracranial spread.³ Complicated sinusitis can lead to brain abscess, subdural empyema, meningitis, cavernous sinus thrombosis, epidural abscess and osteomyelitis, all occurring either alone or in various combinations. In this case series

we have discussed the clinical presentation, which can be quite misleading, the progression of the disease and the vital role played by the radiological investigations in assessing the extent of the disease and surgical treatment.

CASE REPORTS

Case 1

A 20-year-old male patient presented to the outpatient department (ENT OPD) with complaints of protrusion of the left eyeball, lid edema, headache and spikes of high grade fever with chills of one day duration. He gave a history of recurrent episodes of headache with yellowish color, foul smelling postnasal discharge of 2 months duration, for which he took painkillers. On clinical examination, the patient was lethargic but responsive and appeared toxic. He was febrile, his left eyeball was pushed outwards and downwards with lid edema and conjunctival congestion. Extraocular movements of left eye were restricted in all directions but vision was normal. Diffuse swelling of left frontoparietal region was seen and left frontal and ethmoid sinuses were tender on palpation. Examination of nose revealed left alar and vestibular collapse, gross deviated nasal septum (DNS) to left, mucopus in bilateral nasal cavities, and all paranasal sinuses (PNS) were tender. There were no signs of meningeal irritation or any neurological deficits. Fundoscopic examination was normal. Preliminary blood

investigations showed elevated total blood counts with neutrophil predominance. Computerized tomography (CT) scan of PNS showed pansinusitis with left orbital cellulitis with extraperiosteal abscess with pneumocephalus (Figs 1A and B). The patient was admitted and empirical treatment for acute sinusitis with Inj Cefotaxime 1 gm IV BD, Inj Metronidazole 400 mg IV TID, Injection Diclofenac sodium 50 mg IM BD and supportive measures was started. By day 2 in the hospital, the patient developed complete left external ophthalmoplegia with right lower limb grade 0/5 distal motor weakness. PNS were decompressed by endoscopic sinus surgery (ESS). Orbital abscess drained both internally and externally. On 3rd postoperative day (POD) he developed complete internal and external ophthalmoplegia of left eye with grade 0/5 proximal and distal right lower

limb motor weakness with signs of meningeal irritation. Lumbar puncture was inconclusive. Magnetic resonance imaging (MRI) and magnetic resonance venogram (MRV) at this juncture demonstrated minimal subdural collection in left temporoparietal region and partial thrombosis of superior sagittal sinus near the confluence (Fig. 2). Antibiotic (2nd line) was changed to Tazobactam – Piperacillin 4.5 gm IV TID, injection Amikacin 500 mg IV BD, anticonvulsants and diuretics were added. Spikes of fever, headache with motor weakness persisted. CT PNS and brain by 7th and 19th POD reported definite subdural empyema with cerebritis (Fig. 3), which was evacuated by burr hole of left frontal bone. Patient made full recovery over the next one month. He was regularly followed up for 6 months and showed no signs of recurrence or neurological deficit.



Fig. 1A: CT scan image (coronal cut) on admission showing sinusitis with gross DNS to right with orbital abscess (air fluid level with hypodense opacity) displacing the eyeball downward and outward. Intracranial air shadow seen in the left frontal region indicating pneumocephalus

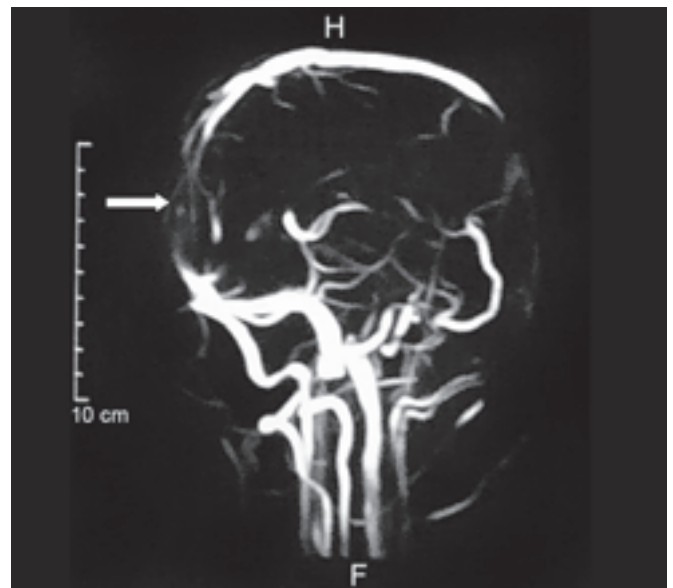


Fig. 2: MRV scan showing filling defect in superior sagittal sinus (white arrow)

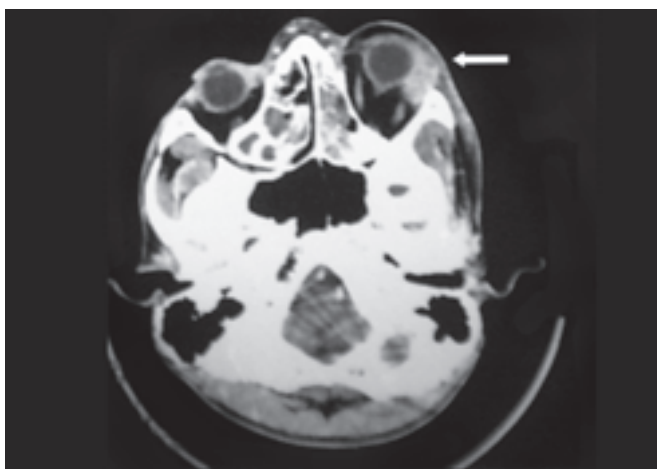


Fig. 1B: CT scan image (axial cut) showing left orbital cellulitis with extraperiosteal abscess displacing the eyeball anterior and laterally

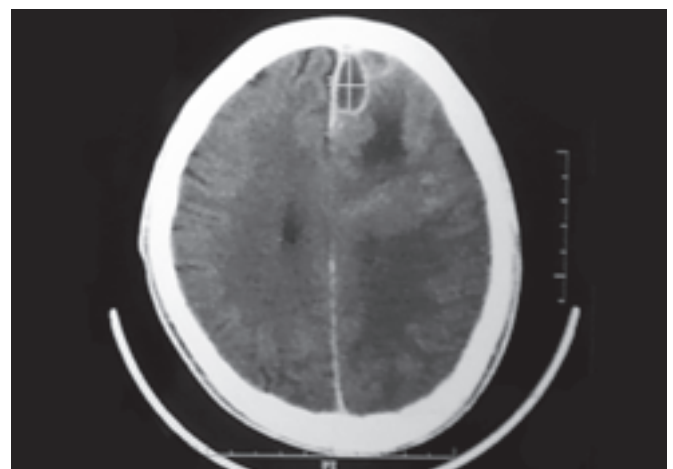


Fig. 3: CT scan image on the 19th POD showing a hypodense collection enveloped by a hyperdense shadow in form of a sac suggestive of an abscess

Case 2

19-year-old male patient presented to the ENT OPD with complaints of continuous unilateral nasal obstruction with recent onset of headache and high grade fever of 2 weeks duration. There was associated foul smelling anterior nasal discharge and episodes of frontal headache in the past but none were so severe. Fever was high grade intermittent type with chills. On examination, patient was febrile with no neurological signs. Diffuse edema was noticed over the left frontal region, on anterior rhinoscopy gross DNS to left with mucosal congestion with mucopus in left middle meatus seen. All sinuses were tender. Thick mucopurulent postnasal discharge was present. Initial blood investigations revealed leukocytosis with neutrophil predominance. CT PNS showed extensive disease of left maxillary ethmoidal sinuses with no bony defect or erosion (Fig. 4A). He was admitted and started on empirical treatment. Like the previous case there was no evidence of intracranial spread, on subsequent serial CT scans a definite epidural abscess in the left frontal lobe was seen (Fig. 4B). His medication was immediately

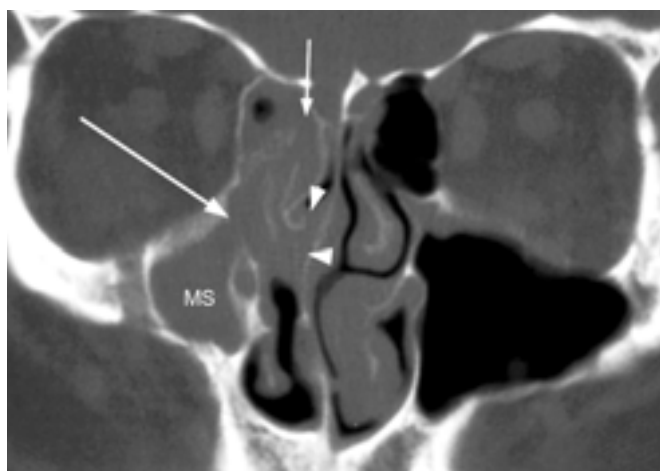


Fig. 4A: CT PNS (axial cut) showing extensive unilateral disease involving maxillary sinus (MS) and ethmoidal sinuses. White arrows are indicating the extent of disease process

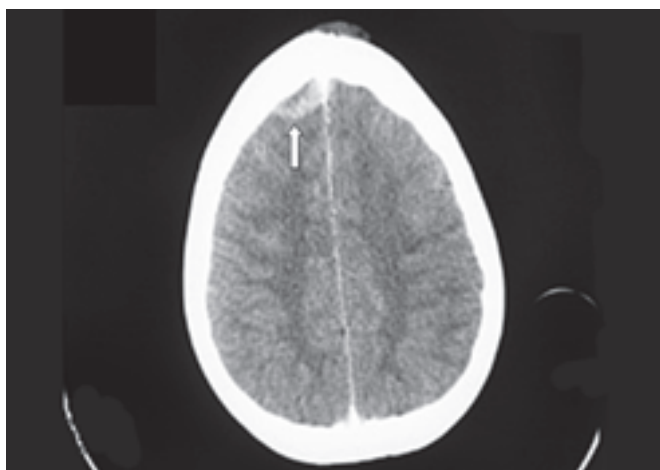


Fig. 4B: CT brain (axial cut) showing epidural collection (white arrow) with minimal soft tissue swelling

changed to 2nd line antibiotics. Surgical decompression of sinuses with evacuation of abscess was done as a staged procedure. He did not have any neurological symptoms or deficits while being in hospital. Patient made full recovery on discharge and remains asymptomatic on subsequent follow-up.

Case 3

A male patient in his late teens presented to the ENT OPD with complaints of bilateral nasal obstruction and high grade fever of 1 week duration. On examination, patient was lethargic but responsive and was febrile. Past history of infrequent nasal obstruction associated with headache secondary to rhinitis was noted. External framework of nose appeared normal. Minimal conjunctival congestion was seen but the vision and extraocular movements were normal. On anterior rhinoscopy, mucopus was present in bilateral nasal cavities both medial and lateral to middle turbinate. There was no septal deviation or any gross anatomical obstruction. Nasal mucosa was congested. Postnasal discharge was present. Immediate CT PNS showed pansinusitis with small extradural abscess in the right frontal lobe unlike the previous cases where the intracranial spread developed later (Figs 5A and B). No obvious bony defect was noticed. He was admitted and started on 2nd line antibiotics. FESS was performed and the sinuses were decompressed. Wait and watch policy was adopted for the extradural collection as per neurosurgical advise. However, the extradural collection failed to resolve and eventually surgical evacuation was done one week later. Very small pus pocket was seen intraoperatively. Patient made full recovery and was asymptomatic on further visits.

DISCUSSION

Sinusitis is a relatively common problem encountered by an otorhinolaryngologist. Despite the advent of newer antibiotics, diagnostic procedures and recent advances in management, it still precipitates with life-threatening intracranial complications.⁴

The suppurative complications of sinusitis can be divided into intracranial and orbital. Orbital complications include preseptal cellulitis, orbital cellulites with/without abscess and cavernous sinus thrombosis (CVT).⁵ Orbit is usually associated with disease involving the anterior ethmoidal air cells, characterized by periorbital edema, chemosis, visual loss, restriction of extraocular movement (the lateral rectus is the earliest muscle to be involved with CVT causing diplopia as the abducent nerve supplying it is closely related) and proptosis.^{6,7} CVT typically presents with orbital apex syndrome (proptosis, ophthalmoplegia, visual

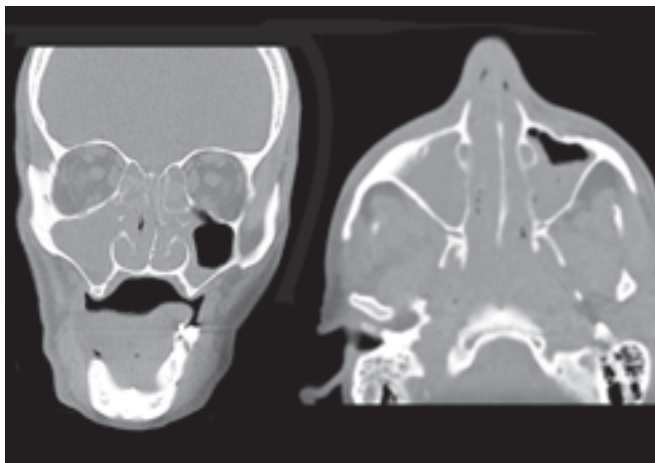


Fig. 5A: CT PNS (coronal and axial cuts) showing features of pansinusitis



Fig. 5B: CT brain (axial cut) showing a small doubtful epidural collection (white arrow). This lesion was not noticed in later scans after the cranial surgery

loss, V1 and V2 facial anesthesia) and venous thrombophlebitis causing episodes of high spiking fever.⁸

Orbital cellulitis and abscess are mainly diseases of children and adolescents, with a peak incidence during the first 15 years of age. In older ages, the disease is more severe and more predisposed to anaerobic infection. In case of orbital infection, clinical examination and prompt treatment are important, since any delay can result in serious complications which include visual loss, intracranial and dural venous sinus spread. Visual loss is thought to be secondary to elevation of the intraorbital pressure caused by the accumulated pus, resulting in retinal ischemia due to central artery occlusion or thrombophlebitis along the valveless orbital veins. Visual loss may also occur because of optic neuritis due to extension of infection. Clinically, it is difficult to differentiate between preseptal cellulitis and abscess, in such conditions CT scan and MRI constitute important diagnostic aids. On CT scan, the presence of abscess is suggested by a low density mass effect without

enhancement, while the presence of air fluid level is a more specific finding of this condition. The medial displacement of medial rectus or the displacement of the periosteum away from the lamina papyracea constitute other characteristics of the abscess, while the swelling of the medial rectus muscle usually indicates orbital cellulitis. MRI is used mainly when intracranial spread is suspected.⁹

Intracranial complications include subdural empyema, epidural abscess, intracerebral abscess, meningitis, CVT and other dural venous sinus thrombosis.

Prevalence of only 3.7% was reported by Claymen et al in his retrospective study to assess the prevalence of intracranial complications of sinusitis (ICS) among all patients hospitalized for sinusitis.¹⁰ The prevalence can vary from 3.7 to 47.6% according to various other studies, however, sinus disease has been implicated as the underlying cause in about 10% of all causes of intracranial suppuration and 45% of these intracranial suppuration coexist with orbital complication.¹¹

Pathogenesis of intracranial complications of sinusitis includes two major mechanisms: Direct and indirect. Direct spread is by osteomyelitis and coexists with underlying epidural abscess. Subdural empyema with epidural abscess would most commonly involve the frontal lobe secondary to direct spread from frontal sinus as a result of osteomyelitis of posterior table. If anterior table is involved, Pott's puffy tumor may occur. Indirect spread or commonly the retrograde thrombophlebitis is via the valveless diploic veins. The infection can pass retrogradely into cavernous sinus and other dural venous sinuses. If the infection reaches the subdural spaces, it spreads easily over the convexities of brain owing to lack of septations.¹² This could have served as the route in the pathogenesis of the present case series where none had any bone erosion.

Most complicated sinusitis occurs in young men in the second or third decade of life as the frontal sinus continues to develop and the vascularity of diploic veins is maximum in that period. In most of the studies, ICS are more commonly seen in males, with sex ratio ranging from 1.3:1 to 4.5:1.^{10,13} The most common presenting complaints are that of headache and fever. Nasal symptoms are prominent in case of gross septal deviations or any other obstructive cause.

Suppuration from paranasal sinuses would commonly cause meningitis but meningitis per se is not commonly due to sinus infection, as it is involved by hematogenous spread. Sphenoid and ethmoid air cell infection are commonly associated with meningitis. Headache and neck stiffness are universal complaints, patients appear toxic and febrile with nuchal rigidity, often lumbar puncture clinches the diagnosis. Mental status at the time of diagnosis and the

type of bacteria causing meningitis are the two most useful prognostic indicators in determining the final outcome. Overall fatality rate increases by 20% in adults if associated with meningitis. Sensorineural hearing loss and mild mental deficits are frequently observed late sequel. Gorse et al¹⁴ found an associated complication rate of 40% in patients undergoing treatment for meningitis, they include sensorineural hearing loss, cranial nerve palsy, optic neuritis and hemiparesis.

Brain abscess occurs in frontal and parietal lobes seen with frontal and ethmoid sinus infection. Chun et al¹⁵ in his study, reported 40% of cases with brain abscess had permanent physical or mental disability, including hemiparesis, dysphagia and blindness. Mental status and degree of orientation at the time of diagnosis are important prognostic indicators as our patients were well oriented and responsive at the time of presentation, it lead to favorable outcome. Clinical features were nonspecific, headache was the most common symptom, early symptoms were due to increased intracranial pressure and often associated with seizures. Cerebritis is the initial stage in the development of brain abscess, which is intense focal intraparenchymal infiltration of inflammatory cells. If left untreated, cerebritis will develop into frank abscess in one or two weeks time. Timely CT with IV contrast detect this early cerebritis in selected patients.^{16,17} This can explain why in the above two cases intracranial extension was not detected early.

Subdural empyema is the collection of frank pus between the dura and the arachnoid layer, most of the time it coexists with epidural abscess. Of all intracranial suppurations, subdural empyema most commonly (41 to 67%) results from paranasal sinusitis (frontal). Other causes being meningitis, otitis media, operative injury, head trauma or bacteremic seeding of a previous subdural hematoma. It is characteristically a rapidly progressive condition. Symptoms are reflective of increased intracranial pressure, meningeal irritation and cerebritis. Most common symptoms are headache, fever and neck stiffness, meningism develops relatively easily. Focal neurological deficits unfold, rapidly including seizures, hemiparesis, aphasia and coma.^{4,6} Kaufman et al¹³ in his study of 17 patients with subdural empyema found coexisting periorbital cellulitis in seven. Lumbar puncture in these cases were consistent with parameningeal infection.

In most cases, CT scan is often the first radiological investigation done in suspected cases of subdural empyema because of cost factor, availability and visualization of the paranasal sinuses and associated bony anatomy. Early changes of meningeal thickening and cerebritis might not be shown in CT till abscess develops, therefore, MRI is preferred over CT as it is more sensitive in picking

parenchymal abnormalities,¹¹ this can delay the radiological confirmation of intracranial pathology as seen in above cases. On CT scan, the empyema appears as a thin, hypodense subdural lesion, with linear enhancement of the medial surface. The grey matter/white matter interface is displaced inwardly. Mass effect is generally caused by edema and ischemia rather than mass effect from the abscess. The edema can cause effacement of the basilar cisterns and flattening of the cortical sulci. The sinuses might appear opacified, with air fluid levels and bony erosion evident in some cases. MRI appearance is similar; T1-weighted images show mass effect and hypointense areas of purulence, which are hyperintense on T2-weighted images. The abscess usually has a hyperintense rim on noncontrast T1-weighted images. Most empyemas are located within the frontal lobe.^{18,19}

Lumbar puncture is contraindicated in patients with subdural empyema if mass effect is present on CT or if patient has papilledema as this can lead to tentorial herniation and even death.¹³ Complications associated with subdural empyema are because of fatal dural venous thrombosis and cortical infarctions, which appears to correlate with poor outcome. Mortality rate is up to 20% in such patients, and 30% of those who survive will be neurologically impaired.²⁰ Subdural empyema is a true neurosurgical emergency, treatment includes antimicrobial therapy against aerobic, anaerobic cocci and bacilli.

Third generation cephalosporins and metronidazole offers broad coverage and good CSF and abscess penetration. At least two weeks of intravenous therapy should be given, followed by oral therapy for up to 6 to 8 weeks. If adjacent osteomyelitis is present, prolonged intravenous therapy for a minimum period of 6 to 8 weeks should be considered.¹⁰ Surgical management is an integral part and should not be delayed. The goals of surgical intervention are decompression of brain and complete evacuation of purulence. Vision impairment, as well as worsening of periorbital erythema or edema, proptosis and restriction of eye movements and lack of response to an initial trial of appropriate antibiotics are absolute indications for surgical drainage. The route of surgical drainage is determined by localization of the orbital subperiosteal abscess, endoscopic orbital decompression or external approach can be employed. In addition, definitive management of infected sinuses should be done preferably at the same time.^{9,11}

REFERENCES

1. Calstrey S, Pfleiderer AG, Moffat DA. Persisting incidence and mortality of sinogenic cerebral abscess: A continuing reflection of late clinical diagnosis. *J R Soc Med* 1991;84:193.

2. Wood MW, Cardenas R, Moore C. Influence of CAT scanning on diagnosis and mortality rate of brain abscess. *J Tenn Med Assoc* 1980;73:325.
3. Courville CB, Rosenfold LK. Intracranial complications of infections of the nasal cavities and accessory sinuses. A survey of lesions observed in a series of fifteen thousand autopsies. *Arch Otolaryngol* 1938;27:692.
4. Robert W Dolan, Chowdhury Khalid. Diagnosis and treatment of intracranial complications of paranasal sinus infection. *J Oral Maxillofac Surg* 1995;53:1080-87.
5. Goldberg AN, Oroszlan G, Anderson TD. Complications of frontal sinusitis and their management. *Otolaryngol Clin North Am* 2001;34:211-25.
6. Jones NS, Walker JL, Bassi S, Jones T, Punt J. The intracranial complications of rhinosinusitis: Can they be prevented? *Laryngoscope* 2002;112:59-63.
7. Singh B, Van Dellen J, Ramjettan S, Maharaj TJ. Sinogenic intracranial complications. *J Laryngol Otol* 1995;109:945-50.
8. Giannoni Carla M, Stewart Michael G, Alford Eugene L. Intracranial complications of sinusitis. *Am J of Otolaryngol H & N Surg* 1997;107(7):863-67.
9. Eleftherios Vairaktaris, Marilita M Moschos, Stavros Vassiliou. Orbital cellulitis, orbital subperiosteal and antraorbital abscess. Report of three cases and review of the literature. *J of Cranio Maxillofac Surg* 2008;1-5.
10. Clayman GL, Adams GL, Paugh DR, Koopmann CF Jr. Intracranial complications of paranasal sinusitis: A combined institutional review. *Laryngoscope* 1991;101:234-39.
11. Osborn Melissa K, Steinberg James P. Subdural empyema and other suppurative complications of paranasal sinusitis. *Lancet Infect Dis* 2007;7:62-67.
12. Wackym PA, Canalis RF, Feurman T. Subdural empyema of otorhinolaryngol origin. *J Laryngol Otol* 1990;104:118-122.
13. Kaufman DM, Miller MH, Steigbigel NH. Subdural empyema: Analysis of 17 recent cases and review of the literature. *Medicine* 1975;54:485-98.
14. Gorse GJ, Thrupp LD, Nudleman KL. Bacterial meningitis in the elderly. *Arch Intern Med* 1984;144:1603.
15. Chun CH, Johnson JD, Hofstetter M. Brain abscess. *Medicine (Baltimore)* 1986;65:415.
16. Heineman HS. Bacterial brain abscess: Infectious diseases and medical microbiology (2nd ed). Philadelphia, PA, Saunders 1986:1080-85.
17. Wenig BL, Goldstein MN, Abramson AL. Frontal sinusitis and its intracranial complications. *Int J Pediatr Otorhinolaryngol* 1983;5:285.
18. Tsai YD, Chang WN, Shen CC. Intracranial suppuration: A clinical comparison of subdural empyemas and epidural abscesses. *Surg Neurol* 2003;59:191-96.
19. Weisberg L. Subdural empyema: Clinical and computed tomographic correlation. *Arch Neurol* 1986;43:497-500.
20. Haines SJ, Mampalam T, Rosenblum ML. Cranial and intracranial bacterial infections. In: Youmans JR (Ed): *Neurological Surgery. A Comprehensive Reference Guide to the Diagnosis and Management of Neurosurgical Problems* (3rd ed) Philadelphia, PA, Saunders 1990;6:3707-35.