Subdural Empyema: A Rare Complication of Chronic Otitis Media

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Introduction

Subdural empyema is a rare infection characterized by a purulent collection in the subdural space [1]. It is a serious intracranial infection with a mortality rate of approximately 35%. Although meningitis, sepsis, penetrating skull wounds, osteomyelitis and rupture of intracerebral abscess have all been implicated as causes of subdural empyema, otolaryngologic infections have been reported to be the most common cause [2]. Approximately 70% of subdural empyemas are due to extension of paranasal sinus infections, while another 20% are due to extension of otologic infections. We report one case of subdural empyema with transverse sinus thrombosis as a result of otologic infection.

Case Report

A seven year old male child, presented with purulent foul smelling discharge from right ear over earlier 2 years. He had developed intermittent high grade fever associated with chills and rigors, projectile vomiting more so early in the morning on awakening and pain right ear associated with a postaural swelling for the earlier two weeks. Child also complained of anorexia, headache, weakness and lethargy. He had undergone incision and drainage elsewhere for the swelling behind the ear and was administered an irregular course of antibiotics before he reported to us. On examination child looked ill, toxic and pale but was conscious. He had deep tenderness on the right side of the neck along with neck rigidity, papilloedema and a positive Kernig's and Brudzinski's sign. Examination of the right ear revealed purulent, foul smelling discharge and florid cholesteatoma. There was a soft fluctuant swelling in the right postauricular region with a small palpable bony defect on the posterior aspect of the mastoid. Clinically he was diagnosed as a case of chronic otitis media giving rise to acute pyogenic meningitis with a possibility of brain abscess and transverse sinus thrombosis. CSF confirmed pyogenic infection (gram positive cocci and gram negative diplococci). Pus swab from the ear

isolated *Pseudomonas* organism. He was nursed in paediatric intensive care unit and was administered oral glycerol and high doses of intravenous antibiotics against gram positive, gram negative and anaerobic organisms. HRCT temporal bone (Fig 1) revealed right sided mastoiditis with an abscess under the temporalis muscle. There was cerebritis, transverse sinus thrombosis with empyema along the inferior border of the tentorium cerebelli on the right side (Fig 2). Neurosurgical consultation warned of considerable risk in draining a small subdural empyema and recommended continuation of conservative therapy. After a week of antibiotic cover child was taken up for a tympanomastoid exploration under general anaesthesia. By a postaural approach the mastoid was exposed. There was an abscess pocket below the temporalis muscle that was fully evacuated and granulation tissue debrided. The surface of the mastoid did not reveal any erosion. Some pus was seen to be draining from the region of the mastoid emissary vein. A canula was inserted in the foramina and suction applied. Mastoidectomy was performed

Fig. 1 : Abscess below right temporalis muscle and thrombosis of the ipsilateral transverse sinus (arrow)

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Fig. 2 : Arrow showing abscess under right tentorium cerebelli

and a large cholesteatoma with extensive granulation tissue was found in the attic and antrum. Tegmen plate was eroded and a large dural defect was noticed. The sinus plate was intact. Facial canal showed no erosion. Temporalis muscle was used to support the dural defect. Meatoplasty was done and the cavity packed. He had an uneventful recovery. MRI brain on the fifth post operative day revealed complete disappearance of the cerebritis and resolution of the subdural empyema. MR angiography at the same time showed patent intracranial venous sinuses (Fig 3). He was given intravenous antibiotics for four weeks and thereafter oral antibiotics for another four weeks. Cavity healed well and follow up six months later revealed no other complications.

Discussion

Otitis media associated with severe earache and high fever could cause death, was recognized as early as 400 BC by Hippocrates, by Celsus in 25 AD and Avicenna (980-1037 AD). Brain abscess was the first complication of otitis media to be recognized and surgically treated successfully by Morand in 1768. Purulent meningitis was the most dreaded complication and before the antibiotic era, the outcome was invariably fatal. Lateral sinus thrombophlebitis was first recognized by Hooper in 1826 and the first surgery was attempted in 1880 by Zaufal. The advent and developments in antimicrobials have progressively reduced the incidence and fatalities associated with otogenic intracranial complications.

Complications of otitis media either acute or chronic occur when the infective process spreads beyond the confines of the middle ear. Intracranial otogenic complications include meningitis, being the most common, brain abscess, extradural abscess, lateral sinus thrombosis and subdural abscess. Factors influencing the spread of infection beyond the middle ear space

Fig. 3 : MR angiography showing normal flow in the intracranial venous sinuses

are, a) the type and virulence of the infecting organism b) the resistance of the host and c) the adequacy of treatment[3]. At present only infrequent reports of CNS complications of ear infections appear. These complications have declined markedly and many contemporary otolaryngologists have not been exposed to these complications. Once the focus of infection has been removed surgically complications of otitis media now usually can be arrested with antibiotic therapy, and progression to a more severe stage of complications can be prevented. A suppurative process in the ear has access to the CNS by direct extension through bone, by either cholesteatoma or chronic osteomyelitis, through spread by thrombosis of small venules from the dura, transverse sinus and beyond; and through preformed pathways, such as labyrinth, the endolymphatic channels and developmental or traumatic bony defects. During extensive review of literature [4,5,6,7] we have come across many cases of subdural empyema as a complication of otitis media. This case confirms these reports and also establishes the pathways of spread of infection retrograde from the sigmoid sinus through the mastoid emissary vein to the surface, presenting extracranially as an abscess under the temporalis muscle.

Extradural abscess is the commonest complication and relatively simple to treat. By way of a mastoidectomy the pus is evacuated and enough bone is removed for an area of healthy dura to be exposed all around the dural defect. Subdural empyema must be managed in close cooperation with a neurosurgeon. Apart from massive doses of antibiotics, the subdural space is drained and ear condition surgically treated. Lateral sinus thrombophlebitis requires early neurosurgical intervention as it continues to infect other intracranial

structures. A radical mastoidectomy is done and the sinus exposed. A normal looking sinus giving a free flow of blood through a needle prick is left alone. In the past a sinus whose lumen was obliterated was opened and the pus, clot and necrotic material evacuated in both directions. It is now generally believed that an organized thrombus need not be evacuated. In case of profuse bleeding the lumen of the sinus is obliterated with ribbon gauge. Internal jugular vein ligation should be reserved for the very rare cases in which septicaemia does not respond to antibiotics and surgery and for patients showing signs of embolisation.

Our case clearly demonstrates that more than one intracranial complication may be present at any one time. We conclude by emphasizing the role of appropriate and prolonged antibiotic therapy and appropriate timing of surgery in the management of intracranial complications of otitis media.

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