Recurrent Late Onset Post-Traumatic Meningitis

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Recurrent meningitis is an uncommon clinical problem which results from post-traumatic osteomeningeal defect. Streptococcus pneumoniae accounts for 80% of central nervous system infections associated with bony anatomical defects in the skull[1]. Despite vigorous use of antibiotics and aggressive supportive care, about 50% survivors have disabling neurological sequelae[2]. Pneumococcal meningitis is associated witha mortality ofabout 32%[3] which is even higher in individuals with bacteraemia[4]. We report a case of recurrent late onset post-traumatic meningitis with some unusual features, who underwent definitive surgical treatment and made an uneventful recovery.

Case Report

A 32 year old male was admitted with recurrent bacterial meningitis having been treated for the same condition 12 months previously. As a child in 1962 he had suffered recurrent it is media and a year later underwent tonsillectomy, adenoidectomy and central lavage. After clearance of these problems he was subsequently discharged from the ENT clinic. Nine years before the most recent admission he had been assaulted, resulting in a head injury. He suffered abrasions over the right side of the forehead associated with profuse nasal bleeding and was brought to the accident and emergency department where X-rays of the skull excluded bony injury After overnight observation he was discharged. There was no reference subsequently to rhinorrhoea or otorrhoea. Eight years after this assault he was admitted to the same hospital with acute meningitis caused by haemophilus influenzae from which he recovered uneventfully. A year later (nine years following initial head injury), he was admitted on this occasion with rapid onset of headache, drowsiness, nausea, vomiting and fever. On examination he was drowsy and had a temperature of 38.1°C. Signs of meningeal irritation were present with marked neck rigidity and a positive Kemig\'s sign. The rest of the neurological examination was normal. Otological and nasopharyngeal examinations were also unremarkable. Lumbar puncture revealed turbid cerebrospinal fluid (CSF) with a raised pressure of more than 50 cms of CSF. containing 4000 leukocytes per cubic millimeter with 95% polymorpho nuclear cells. Gram stain preparation of CSF. demonstrated intracellular and extracellular gram positive diplococci. Cultures of the C.S.E and blood later confinned S. pneumonia. X-rays of the skull, mastoids and paranasal sinuses were normal. Computed tomography (CT) scan of brain was subsequently performed and was normal. Serum immunoglobulins and complement levels were normal. In view of the past medical history, a second CT scan of the head six weeks later with high definition thin axial sections revealed an old fracture involving the anterior aspect of the right loculus of the frontal sinus and a deficiency at anequivalent locationonthe posterior wall of the sinus (Figure).

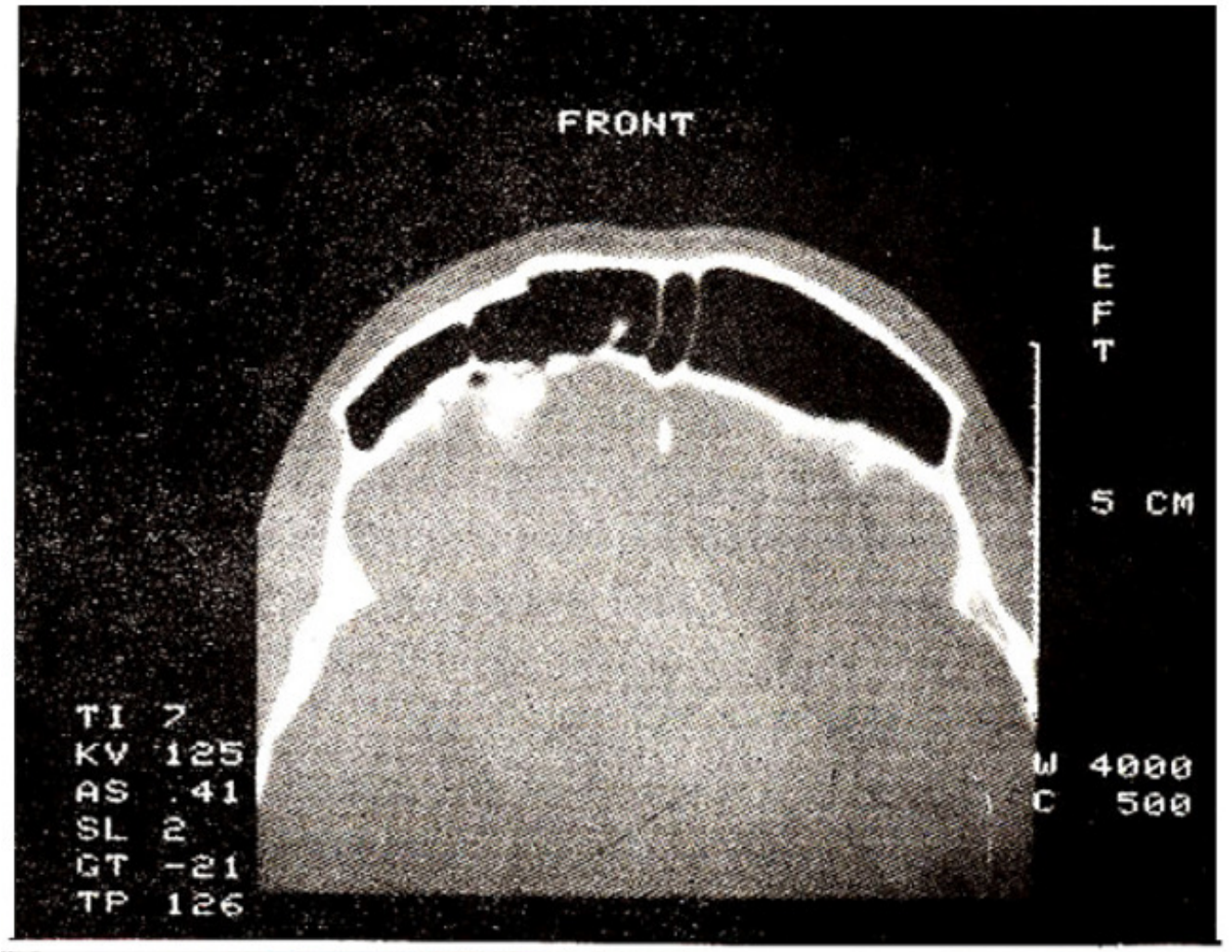

Figure. High definition axial CT scan of the head showing fractures through anterior and posterior wall of right frontal sinus.

High dose antibiotics resolved the acute infection and later he was referred to the neurosurgical unit. At craniotomy afascial patch was applied to seal the defect. The patient made a full recfoveiy and returned to his work as an ODA in the hospital.

Discussion

Basilar and related skull fractures form a significant proportion of head injuries accounting for 15.6 to 24% of all fractures[5-7]. The association of such fractures with dural tears resulting in cerebrospinal fistulae provides a potential portal of entry for micro-organisms which may lead to meningitis. Persistence of such fistulae is the commonest cause of recurrent meningitis in adults. The frequency of osteomeningeal defects in skull fractures has been variably reported. Although the tme incidence of post-traumatic meningitis is not known, there isaconsensus that the risk is high. The initial episode of meningitis is generally seen within two weeks of the injury[1-8]; late disclosure is rare. Furthermore, it rarely occurs without cerebrospinal fluid leakage, and when rhinorrhoea is present the incidence may be as high as 40%[5]. However, the presence of CSF leakage is not a necessary clinical prerequisite for recurrent post-traumatic meningitis. The diagnosis of these defects is difficult and shoul be considered

when there is a history of head trauma, with or without CSF leakage rhinorrhoea or otorrhoea, associated with recurrent bacterial meningitis and radiological or CT scan evidence of a fracture. The location and demonstration of osteomeningeal defects may prove difficult because of their small size and/or situation. For instance, defects in the cribriform plate or ethmoidofrontal junction (the most likely sites of anatomical defect) may not be visualised on plain skull X-rays, tomograms or conventional CT scans. Fluorescent or radio-isotope tracer studies do not commonly detect C.S.F. defects[8]. A detailed study of the skull is therefore warranted with high definition thin section CT scans. Two millimeter direct coronal CT scan is particularly useful for the anterior cranial fossa defects of even 1.5 mm in size[9]. Prophylactic antibiotics are largely said to be ineffective[7] and surgical techniques for repair have been described with excellent prognosis[10] and minimal mortality in expert hands[11]. Two cases of post-traumatic late onset meningitis, one 31 years after head injury and the other 20 years later reported by Hand1 and Sengupta[12] had gross head trauma with radiological evidence of skull fracture and both these patients suffered injury during the growing period, that is at 11 and 7 years of age respectively. It has been suggested that a vascular cuff of meninges may have become trapped between the fracture lines, not only providing a potential portal of entry for the micro-organisms but preventing the fracture healing as well. Over the period with the expansion and growth of the skull bones, the gap may have widened as accounted by the theory of growing skull fractures of childhood[13] with the result that there has been herniation of the meninges with or without brain tissue at some stage, as seen in the case of Sengupta[12]. However, the theory of growing skull fractre is inapplicable in our case which demonstrates that other factors must be involved. The case demonstrates a number of salient features; a very trivial forgotten head injury in an adult that could not be detected by ordinary X-rays and initial CT scan; the long time interval of eight years between the head injury and initial meningitis; recurrent meingitis with different organisms in the absence of CSF leakage. Osteomeningeal defects can present with meningitis many years after and apparently trivial and forgotten head injury even without CSF leakage. Every case of meningitis should be carefully questioned about previous head injury and those with a potential history, irrepective of time interval between the head injury and occurrence of meningitis should be thoroughly investigated, as small osteomeningeal defects are liable to e missed on ordinary routine investigation.

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