SHORT REPORT

Aseptic meningitis after posterior fossa surgery treated by pseudomeningocele closure

C E M Hillier, A Penrose Stevens, F Thomas, J Vafidis, R Hatfield

Abstract

Aseptic meningitis is a recognised complication after posterior fossa surgery. It is often self limiting but occasionally runs a protracted course requiring repeated CSF examination to exclude infection, and treatment with systemic steroids. A patient is described with aseptic meningitis after posterior fossa surgery who underwent posterior fossa re-exploration nearly 3 years after the initial operation. This disclosed a pseudomeningocele, which was closed. The patient remains symptom free almost 2 years after closure. In this case of chronic aseptic meningitis after posterior fossa surgery, closure of the pseudomeningocele found at exploratory surgery led to resolution of the symptoms. (J Neurol Neurosurg Psychiatry 2000;68:218–219)

Keywords: aseptic meningitis; pseudomeningocele; posterior fossa

Aseptic meningitis is a syndrome defined by pyrexia and signs of meningism associated with CSF pleocytosis, increased CSF protein, and sterile blood and CSF culture. It is a recognised complication of posterior fossa surgery and is usually short lived. We describe for the first time a case associated with a postoperative pseudomeningocele which when closed led to the patient becoming symptom free.

Case report

A previously well 13 year old white school boy presented in October 1994 with a 2 month history of giddiness, dizziness, and morning headache associated with vomiting. Examination disclosed only bilateral papilloedema. Cranial CT showed an ill defined mass in the right cerebellar hemisphere, causing obstructive hydrocephalus. He was started on dexamethasone and underwent a posterior fossa craniotomy for posterior fossa re-exploration nearly 3 years after the initial operation. This disclosed a pseudomeningocele, which was closed. The patient remains symptom free almost 2 years after closure. In this case of chronic aseptic meningitis after posterior fossa surgery, closure of the pseudomeningocele found at exploratory surgery led to resolution of the symptoms. (J Neurol Neurosurg Psychiatry 2000;68:218–219)

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Aseptic meningitis, posterior fossa surgery, and pseudomeningocele closure

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demonstrated an enhancing nodule in the distal lumbosacral theca. At an exploratory operation (April 1997), the nerve roots were found encased in fibrotic tissue. The histopathology was in keeping with arachnoiditis: the patient was started on dexamethasone (2 mg four times daily) for 2 weeks with improvement of his headache. Reduction of the dose resulted in recurrence of his symptoms.

In July 1997, nearly 3 years after posterior fossa craniotomy, the patient underwent repair of the pseudomeningocele as a last resort. After this procedure, he became asymptomatic, required no medication, and has not required further admissions.

Discussion

The relation between cranial surgery and aseptic meningitis has been known for a long time. As early as 1928, Cushing and Bailey suggested that waves of pyrexia suggesting meningitis were “not uncommon following cerebellar operations”. Although aseptic meningitis has been noted after supratentorial surgery, it is more frequent after posterior fossa surgery and seems to occur more commonly in children. It occurs with a greater frequency in patients operated on for non-tumour abnormalities compared with those operated on for tumour.

Carmel et al described aseptic meningitis in 70% of 50 children undergoing posterior fossa surgery. The meningitic reaction did not correlate with the underlying lesion, closure of the dura, initial postoperative CSF red cell count, or postoperative drainage of CSF. Symptoms appeared as late as the 2nd or 3rd week postoperatively and could last 3—4 months. Clinical characteristics described included headaches, fever, and meningism, and less commonly impairment of consciousness and neurological deficit. Pleocytosis in the CSF, with a significant mononuclear component (or less commonly polymorphs but later lymphocytes), often predominated.

The underlying aetiology remains uncertain. Cushing and Bailey suggested that the pyrexia might be due to blood in the CSF but Carmel et al demonstrated that red blood cells need not be present for pyrexia to continue. Finlayson and Penfield suggested that a cyst-like cavity may form postoperatively and fill with clot and products of tissue destruction which would discharge periodically, and Matson proposed that breakdown products from the tumour bed might be responsible. Attempts to define CSF substances associated with aseptic meningitis have included blood/brain/tumour and muscle markers (haemoglobin, bilirubin, myoglobin, and creatinine kinase) but no clear pattern has emerged: operative technique, postoperative radiotherapy, or antibiotics did not influence the course of aseptic meningitis.

Treatment of aseptic meningitis by decompression of a related pseudomeningocele has been previously reported by Jacobs et al. They describe a case of aseptic meningitis in an 8 year old boy after removal of a cystic astrocytoma. One month after initial surgery he recurred the placement of a ventriculoaqueductal shunt which became necessary to eliminate the “unsightly bulging of the decompression site.” The symptoms resolved after the operation.

To our knowledge, we describe the first case of aseptic meningitis treated by formal closure of a related pseudomeningocele. The finding that pseudomeningocele formation is more common in surgery of the posterior fossa may explain the increased incidence of aseptic meningitis in this group of patients. In the case described, closure of the pseudomeningocele led to resolution of symptoms. Why this occurred is not immediately obvious but hypotheses include the possibility that alteration of normal CSF flow in and around the meningocele leads to the accumulation of, as yet, undetectable inflammatory mediators that are responsible for the meningitic reaction seen. It may be of interest to sample CSF from the meningocele and lumbar region simultaneously and compare concentrations of inflammatory mediators.

4 Finlayson AI, Penfield W. Acute post-operative aseptic leptomeningitis: reviews of cases and discussion of pathogenesis. Archives of Neurology and Psychiatry 1941;46:259—76.
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J Neurol Neurosurg Psychiatry 2000 68: 218-219
doi: 10.1136/jnnp.68.2.218

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