Recurrent fulminating meningitis
20 years after head injury

Case report


Regional Neurological Centre, Newcastle University Hospitals, Newcastle General
Hospital, Newcastle upon Tyne, England

A case of recurrent fulminating meningitis 20 years after head injury is reported.
Herniation of brain tissue into the frontal sinus through a growing fracture at the base
of the skull was responsible for the infection.

KEY WORDS • long delayed meningitis • herniation of brain tissue • growing fracture of the skull

MENINGITIS many years after a related head injury is extremely rare. Only two cases of recurrent meningitis, each 31 years after head injury, have been reported, but the mechanism of delayed infection remains unclear. We are reporting the case of a man who developed fulminating meningitis 20 years after a head injury. The source of the infection was identified and surgically corrected.

Case Report

This 27-year-old man was admitted as an emergency case in May, 1970, for progressive headache and malaise of 36 hours’ duration. During the night before admission he had become delirious, restless, and confused.

Examination. The patient was unconscious, restless, and feverish, with profuse sweating. The pulse was 60/min and blood pressure was 180/90 mm Hg. Both pupils were moderately dilated and fixed to light. There was a divergent squint on the right. A star-shaped opacity was noted in the right lens but there was no papilledema. There was marked neck stiffness and a positive Kernig’s sign. A diagnosis of meningitis was made, and this was confirmed by lumbar puncture which produced purulent cerebrospinal fluid (CSF) under high pressure (250 mm H$_2$O) from which pneumococci were isolated. In spite of intensive therapy with systemic chloramphenical and sulfonamide and intrathecal penicillin, he remained gravely ill for about 3 days. His respiration became irregular and at one point he stopped breathing; he was intubated and later required tracheostomy and assisted respiration. With daily instillation of intrathecal penicillin and systemic dexamethasone, his level of consciousness started to improve. By the end of the fourth day he was obeying commands. Repeat lumbar puncture at this time showed
Recurrent fulminating meningitis

![Fig. 1. Anteroposterior view of skull showing bone defect in left frontal region (arrows). Note absence of opacity in the frontal sinus.](image)

![Fig. 2. Diagram, lateral view, showing herniation of brain into the frontal sinus as seen at operation.](image)

a protein content of 110 mg%, sugar 56 mg%, cell count 250/cu mm, 80% polymorphs, and 20% lymphocytes. There was no growth on culture. By the end of 1 week it was possible to close the tracheostomy, and he continued to make a total recovery. He was discharged home on the 20th day and was able to return to work after a short time.

He was readmitted 5 months later with a 24-hour history of headache, restlessness, and shivering. His mother had noted that he was drowsy and confused; there had been no convolution.

**Second Examination.** The patient was uncooperative and drowsy. The pupils were equal and normal in size, and responded to light. There was marked neck stiffness. The rest of the neurological examination was normal. Lumbar puncture produced purulent CSF which grew pneumococci on culture. The CSF protein content was 570 mg%, sugar 20 mg%, chloride 700 mg%, and cells 5750/cu mm, mainly polymorphs. With systemic penicillin and sulfonamide as well as intrathecal instillation of penicillin he made a quick recovery within 4 days.

To assess the cause of the recurrent meningitis, further investigation was considered necessary. On further inquiry into the past medical history the patient's mother revealed that at the age of 7 years he had had a depressed fracture in the left frontal bone caused by being kicked by a horse. He had not lost consciousness at the time and there was no CSF rhinorrhea. Following operation to elevate the depressed fragments of bone, he had recovered uneventfully and remained in good health. With this past history of head injury in mind a number of tests were carried out. Skull films revealed an area of translucence in the left frontal region close to the frontal sinus. The margins of this defect were scalloped (Fig. 1). The changes were thought to be due to an osteitis. The sinus, however, was not opaque. Echoencephalography did not reveal any shift. Electroencephalography showed diffuse areas of theta activity and occasional delta wave formation, changes considered to be compatible with recent meningitis. A brain scan with Tc⁹⁹ did not show any abnormal uptake. A lumbar route air-encephalogram was also normal. On the basis of the localized bone abnormality near the left frontal sinus, it was decided to explore the left anterior fossa of the skull.

**Operation.** A left frontotemporal craniotomy was performed. No evidence of bone infection was found. When the left frontal lobe was retracted it became clear that part of the frontal lobe was indurated and was herniating into the left side of the frontal sinus through a defect at the junction of the floor and the anterior wall of the anterior cranial fossa (Fig. 2). The dura was firmly
adherent to the margins of the defect. The herniated part was excised. The dural defect was then repaired with fascia lata. The patient made an uneventful recovery following the operation and has remained well.

Discussion

The case reported here presents several interesting points, namely, the interval of 20 years between head injury and meningitis, the absence of CSF rhinorrhea, and the presence of a skull defect with a related herniation of the brain into the frontal sinus detected and treated at operation.

Meningitis following head injury usually develops due to a dural tear associated with a fracture of the base of the skull involving the paranasal sinuses. In most cases there is a history of CSF rhinorrhea and this CSF leak provides an easy route for the bacterial infection. In such cases the onset of meningeal infection is early. However, in the absence of a CSF leak, meningeal infection is still possible and a small sealed-off vascular cuff of meninges is thought to be trapped in the fracture line providing an avenue of entrance for the organism. In these circumstances the onset of infection may be delayed as communication between the meningeal spaces and the nasopharynx is less free. However, a delay of 20 years or more for the development of the infection is extremely rare, and the mechanism of infection after such a long interval may not be the same as in the earlier cases. Bøe and Huseklepp referred to the possibility of "an unknown" factor responsible for delayed infection.

Review of the case reported by Hand and Sanford, the one by Stenger, and ours reveals some important similarities. Stenger's case had pneumococcal meningitis five times in the course of 3 years starting 31 years after a head injury. The patient did not have any CSF leak or demonstrable fracture of the skull. At operation, however, a defect in the posterior wall of the frontal sinus and an adjoining fracture line were found. In the case reported by Hand and Sanford, the patient had four attacks of pneumococcal meningitis 31 years after head injury. This patient also did not have CSF rhinorrhea. Although the patient had surgical correction of the skull defect, the actual operative findings were not mentioned.

In our case, the patient had two attacks of pneumococcal meningitis 20 years after head injury, at which time a depressed fracture of the skull vault was found but no fracture of the skull base was demonstrable, and the patient did not have CSF rhinorrhea. At operation 20 years later, a defect 8 x 4 mm was found. Through this defect brain tissue was herniating into the frontal sinus. The herniated portion of the brain was devoid of any meningeal covering, and the dura was firmly adherent to the margins of the defect.

Thus it seems that in the three cases of long-delayed meningitis there was no detectable fracture of the skull base at the time of injury, nor was there any subsequent CSF rhinorrhea. All had pneumococcal organisms, and in two cases exploration revealed a skull defect that allowed herniation of cerebral tissue.

In the absence of CSF leak or demonstrable fracture of the skull base at the time of injury, it is unlikely that a defect in the floor of the anterior cranial fossa with herniated brain had been present for all this time without giving rise to any complications. On the other hand, a narrow fracture line sealed with torn dura and not demonstrated by x-ray may grow after some years like the well-described "growing fracture" of the skull. In this condition a fracture of the skull in childhood, usually in the parietal vault, fails to repair due to interposition of the meninges. In the adult period when the growth ceases, the fracture reveals itself as a translucent area with scalloped margins, as in the skull films of our patient. Cerebral herniation following trauma can result in the formation of a growing fracture. In this case, however, it is unlikely that the herniation occurred at the time of injury without causing any complications for 20 years. Although a growing fracture of the base of the skull is unusual, the interposition of the torn dura at the narrow fracture line can prevent repair, causing a significant defect and allowing the gradual herniation of cerebral tissue. Because of the sealing off of the meningeal space at the margin of the defect, an actual CSF leak is prevented. Infection with pneumococci following a flu-like illness suggests that when the body resistance is lowered the organisms succeed in starting meningitis. It is possible that the two long-delayed cases of meningitis reported before had a similar mechanism, namely, a growing fracture.
Recurrent fulminating meningitis

The problem of ignoring a history of head trauma has been mentioned by many authors, and apart from the medicolegal aspects, lack of this information may create difficulty in the management of such patients. Lacking knowledge of the previous head injury at the time of our patient's first admission, we interpreted the radiological appearances of a growing fracture of the skull as possible osteomyelitis, and did not discover the true nature of the lesion until operation. So, in agreement with Bøe and Huseklepp, we feel that in cases of recurrent meningitis (particularly pneumococcal), a history of head injury, however trivial and with or without recognizable CSF leak or skull fracture, may lead to the identification of the source of the infection, and early surgical treatment can prevent further attacks of meningitis.

Acknowledgments

We are grateful to Mr. L. P. Lassman for allowing us to report the case history of this patient who was admitted under his care. The diagram was drawn by Dr. J. D. Whitby, F.F.A.R.C.S., who also gave anesthesia in this patient.

References

6. Stenger HH: cited in reference 1

Address reprint requests to: R. P. Sengupta, F.R.C.S., Regional Neurological Centre, Newcastle General Hospital, Newcastle upon Tyne NE4 6BE, England.