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Lesson of the Week

Recurrent meningitis due to congenital malformation of the inner ear

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Congenital malformations of the inner ear may be associated with deafness, a cerebrospinal fluid fistula, and recurrent attacks of meningitis.¹ When there is a leak of cerebrospinal fluid and the tympanic membrane is intact the fluid enters the nasopharynx through the eustachian tube, where it may be very difficult to detect.² We report on a case of recurrent meningitis that occurred in a child with congenital deafness. The site of cerebrospinal fluid leakage was not detectable clinically but was accurately predicted by complex motion tomography and confirmed surgically.

Case report

A 5 year old boy was admitted to the Middlesex Hospital on 19 February 1981, having suffered four attacks of pneumococcal meningitis in the previous seven months—in July 1980, September 1980, January 1981, and February 1981. After the second attack he was admitted to a neurosurgical department for investigation. No cause for the attacks was found, and he was discharged without treatment.

The child had had a full term normal delivery. He sat at 1 year and walked at 18 months. A severe bilateral sensorineural deafness and right VI nerve palsy were diagnosed during the first year of life. He had been fitted with bilateral body worn hearing aids and attended a special school for the deaf but could not talk. There was no family history of deafness, and one other child is alive and well. The clinical examination was normal except for deafness, a right VI nerve palsy, and the absence of speech. In particular, there was no evidence of a leak of cerebrospinal fluid from the ears or nose and throat, and none had been seen either at home or on previous hospital admissions.

Recurrent attacks of meningitis in a deaf child may indicate a congenital malformation of the inner ear associated with a cerebrospinal fluid fistula

Preliminary investigations including full blood count, concentrations of urea and electrolytes, nose and throat swabs, immunoglobulin concentrations, skull radiographs, and computed tomography of the head and floor of the anterior cranial fossa gave normal results. Polytomography of the inner ears, however, showed dilated labyrinths on each side with an evagination from the vestibule replacing the lateral semicircular canal. The cochleae were also dilated, particularly on the right, where there was an incompletely formed central bony spiral.

Because of the labyrinthine dysplasia it was thought that there was a fistulous communication between the subarachnoid space, abnormal labyrinth, and middle ear cavity, probably through the oval window. The right abducens palsy and more severe anatomical deformities on the right suggested that surgical exploration of this side should be done.

Tympanotomy showed that the mucosa surrounding the oval window was ballooned and that a small amount of clear fluid had collected just anterior to the stapes footplate. Clearly, a continuous leak of cerebrospinal fluid was escaping from the margin of the oval window and collecting in the middle ear through a small rupture in the ballooned mucosa. The stapes bone was removed and this resulted in a torrential flow of clear fluid under pressure. It was thus impossible to establish the site of communication with the subarachnoid space. The vestibule was packed firmly with a large quantity of temporalis muscle and this was covered with a sheet of temporalis fascia.

After the operation phenoxymethylpenicillin and chloramphenicol elixir were given for 10 days, and then all medication was stopped. There was no postoperative vertigo or nystagmus, which confirmed the preoperative impression of a non-function-

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ing labyrinth. There have been no further episodes of meningitis, and the boy was discharged well one year after surgery.

Comment

Leaks of cerebrospinal fluid from the ear usually follow trauma and have been classified as open or closed, depending on whether or not the tympanic membrane is intact.³ Rarely, a spontaneous leak of cerebrospinal fluid from the ear may occur, and recurrent attacks of meningitis have complicated nearly all the reported cases. When the tympanic membrane is intact and the leak of cerebrospinal fluid is only small or intermittent tracer studies may not show the leak, and failure to locate accurately the anatomical defect may result in fruitless neurosurgical exploration. Most congenital cerebrospinal fluid fistulas in the region of the petrous temporal bone, and especially those that occur spontaneously, are associated with abnormalities of the bony labyrinth and internal auditory meatus.⁴ Complex motion tomography or high resolution computed tomography will show

these abnormalities of the inner ear and should be considered for any deaf child with a suspected leak of cerebrospinal fluid or unexplained meningitis. The high pressure of cerebrospinal fluid against the oval window eventually causes a rupture, allowing the egress of fluid and the ingress of pathological organisms. Once the defect has been located surgery to plug the leak will prevent further attacks of meningitis.

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Clinical Topics

Anticonvulsant drugs and advice about driving after head injury and intracranial surgery

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In 1976 driving licences in the United Kingdom became valid until the driver is aged 70. Since then drivers have had a statutory duty to report both relevant and prospective disabilities—conditions that might affect their fitness to drive.¹ ² Epilepsy is a relevant disability which is, however, compatible with holding an ordinary driving licence under certain conditions that mainly relate to a stipulated period of freedom from attacks. The risk that epilepsy may begin after a head injury or craniotomy may constitute a prospective disability. Patients whose licence is revoked even temporarily in such circumstances, however, may be very resentful; their surgeons often share this resentment³ ⁴ and will sometimes support an appeal against the Driving Vehicle Licensing Centre. The surgeon then usually states that the risk of epilepsy in the patient is minimal, yet it often transpires that he has prescribed prophylactic anticonvulsant drugs.

This paper reports the results of a survey of current practice among a sample of British neurosurgeons in respect of prescribing anticonvulsant drugs and advice about driving.

Methods

A questionnaire was sent to all full and associate members of the Society of British Neurological Surgeons. Replies were received from about half, but these 55 surgeons represented almost all the regional units in the United Kingdom. For several diagnostic categories surgeons were asked whether after performing a craniotomy they prescribed anticonvulsants always, sometimes, or never. When their reply was "sometimes" they were asked what preoperative, operative, and postoperative features influenced the decision to give anticonvulsants. They were then invited to indicate separately what levels of a risk of epilepsy in the first year they considered to justify either prescribing anticonvulsants or advising against driving; bands of risk from which they chose were 5-10%, 10-20%, 20-30%, 30-40%, and more than 40%. Further questions related to details of prescribing practice. The study was designed primarily to discover how neurosurgeons ranked the relative risk of epilepsy in different circumstances, not to canvass opinion about whether or not anticonvulsants should be given or whether they are believed to be effective when there is a risk of epilepsy.

Results

All but one of the respondents always prescribed anticonvulsants for some of their patients. The proportion of surgeons who always gave postoperative drugs in various conditions showed a reasonable correspondence with the relative risks of epilepsy reported for these conditions (table I). Asked what factors other than the diagnosis led them to recommend anticonvulsants, almost all surgeons cited the occurrence of preoperative or early (first week) postoperative epilepsy. Other preoperative features that influenced about half the surgeons included