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Cerebrospinal Fluid Otorrhea after Head Injury (Case Report)

by

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Introduction

Cases of head injury in which a clear watery fluid escaped from the ear were reported as early as 1727 by Stalpartius Van der Weil, many years before the discovery of cerebrospinal fluid (Levinson, 1919). Fracture of the temporal bone constitutes the most common cause of cerebrospinal fluid (CSF) otorrhea next to surgical trauma to the dura, congenital defects and erosion of the dura and skull by tumors or extensive inflammations (Frable et al., 1962). Dandy (1944) reported 4 cases of postmastoidectomy otorrhea, all involving dural defects of the tegmen plate. The earliest case of spontaneous otorrhea was reported in 1897 by Escat, the drainage attributed to a fistula to the external auditory canal, presumably of congenital origin (Frable et al., 1962). The incidence of CSF otorrhea is low. In an analysis of 1800 cases of head injury, Rowbotham (1964) found that 142 exhibited middle ear damage, and of these, only 28 patients had unequivocal CSF otorrhea, giving an overall incidence of 1.4 per cent in a large unselected series of patients (Canniff, 1971).

This report presents a case of CSF otorrhea after head injury in a boy commencing after myringotomy. We believe it to be the first documented one in the Indonesian literature.

Case report

On July 3, 1972, a three years and three months old Indonesian boy was admitted to the Department of Surgery, Dr. Cipto Mangunkusumo General Hospital, Jakarta, for observation. Significant past medical history was having fallen from his bed and hit on his head half an hour prior to admission resulting in drowsiness and profuse vomiting. He had been coughing a couple of days earlier and there was an elevation of body temperature prior to the fall.

Received 19th August, 1974.

Physical examination on admission to the Department of Surgery revealed an acutely ill boy with signs of dehydration attributable to vomiting. Intravenous fluid therapy was given and evidently because of lacking neurologic signs a transfer to the Department of Child Health was resorted to the next day.

Physical examination on admission to the Department of Child Health revealed a conscious, acutely ill boy with a body weight of 10 kg, a body length of 97 cm and a body temperature of 39 degrees centigrade. Eye examination showed no abnormalities. The throat was hyperemic and the tonsils were somewhat enlarged. Heart and lungs were normal. His liver was enlarged to 3 cm below the right costal margin with a sharp edge, smooth surface, normal consistency and no tenderness. His spleen was not palpable. There was no confirmatory evidence of meningeal irritation, increased intracranial pressure or cerebral nerve involvement. He was put on penicillin in adequate dosage.

An ENT consultant found normal right and left auditory canals. The left eardrum was intact revealing hyperemia and bulging. The nasal mucous membrane was hyperemic with visible secretion. The throat was hyperemic and the tonsils somewhat enlarged. There was no clinical evidence of either swelling or redness over the mastoid. A myringotomy was performed releasing thin watery fluid from the left ear. A tampon was inserted, but the flow persisted saturating the pillow and bed linen at night necessitating a dressing over the ear during the day. High dose ampicillin was thenceforth instituted to guard the patient against an eventually complicating meningitis. By the third day of therapy, his spiking temperature began to recede.

A lumbar puncture showed a clear fluid with a count of 5/3 cells, Nonne negative, Pandy negative, NaCl 714 mg/100 ml, Chloride 433 mEq/l, Glucose 66 mg/100 ml and Protein 21 mg/100 ml. Laboratory examination of the aural discharge showed a clear fluid with a count of ⅔ cells, Nonne negative, Pandy negative, NaCl 720 mg/100 ml, Chloride 437 mEq/l, Glucose 66 mg/100ml and Protein 21 mg/100 ml. It proved to be actually cerebrospinal fluid.

A neurosurgical consultant was not able to detect any abnormality on the skull film. A Stenver's photo revealed no trace of abnormality of the petrous bone. A tomogram was not able to reveal any sign of fracture of the left petrous bone. An audiogram performed at the Department of ENT showed no conductive loss in both ears. Culture of the ear drainage fluid resulted in pyocyaneus bacilli, Staphylococcus aureus hemolyticus and Streptococrus anhemolyticus. Gentamycin was added after organism identification and antibiotic sensitivity test. Cultures of the blood and urine yielded no growth. Peripheral

blood examination was done with the following results: hemoglobin value 8.6 gm/100 ml; total leucocyte count 11,200/mm3; differential count showed bands 1%, segments 64 %, lymphocytes 31% and monocytes 4%; platelet count 250.000/mm3. Urine and stool showed no abnormalities.

A second lumbar puncture done 2 weeks after his hospital admission revealed normal findings of the liquor.

Five days after myringotomy, complete stop of the flow of liquor was observed and from then on no more liquor flow has appeared. The patient continued to improve and had no further complaints referable to the left ear until July 27, when he was discharged from hospital. He was advised to return for further evaluation but he did not come up to the moment this paper was finished for publication.

Discussion

The escape of cerebrospinal fluid from the ear signifies a defect of the temporal bone and of the adjacent dura. Fracture of the temporal bone constitutes the most common cause of cerebrospinal fluid otorrhea especially longitudinal fractures, parallel to the long axis of the petrous pyramid. However, fracture lines may be difficult to visualize on roentgenograms, and failure of films to demonstrate such a line does not rule out a fracture (Frable et al., 1962). In 35 per cent of the cases of unequivocal middle ear damage reported by Rowbotham (1964), no fracture was visible (Canniff, 1971). Cerebrospinal fluid otorrhea occurs most frequently as the result of craniocerebral trauma and much less commonly as a complication of surgical procedures in and about the ear (Lang and Bucy, 1962). The majority of cases of cerebrospinal fluid otorrhea are of traumatic origin and frequently heal spontaneously (Leech and Paterson, 1973; Frable et al., 1964). However, if such closures occur spontaneously, one can not be sure that these will remain closed permanently and that recanalization is not possible (Frable et al., 1962). In some of the cases of otogenic liquorrhea the first flow of liquor appeared in connection with a myringotomy (Nenzelius, 1951 and Koch, 1950, as cited by Barr and Wersäl, 1965). It was assumed that the myringotomy in their cases caused a damage from dural hernia caused by congenital malformation of the wall of the middle ear. The present case differs as to the fact that no prolapse of the dura in the cavity of the middle ear could be observed.

Included in the differential diagnosis of colourless, clear aural discharge are salivary fistulae from the parotid gland into the external auditory canal and the escape of fluid from labyrinthine fistulae. However, it must be emphasised that if there is a discharge of sufficient

volume to soak the patient's pillow, it must be assumed to be CSF since no other otorrheic fluid is so massive (Canniff, 1971). The presence of glucose in the spinal fluid has been used as an identification test. A glucose-oxidase test paper such as Clinistix or Testape is very often used. According to experiments carried out by Gadeholt (cited by Gundersen and Haye, 1970), this method must be regarded as unreliable. It is only able to detect the presence of glucose at the level of 50 mg per cent or more and contamination of the fluid could allow bacterial glycolysis to proceed and drop the glucose level below that detectable by the Clinistix method (Canniff, 1971). Furthermore, the CSF must be clear. In our present case, detection of spinal fluid was achieved by comparing the results of microscopic examination and chemical analysis of the aural discharge with those of the cerebrospinal fluid obtained by lumbar puncture.

It cannot be overemphasized that a defect which permits the leakage of cerebrospinal fluid into the external auditory canal is a channel for the passage of virulent bacteria and viruses into the subarachnoid space. For this reason, any persistent discharge of cerebrospinal fluid from the ear should be promptly terminated (Lang and Bucy, 1962). In most cases a possible cerebrospinal fluid otorrhea will cease during the first week (Gundersen and Haye, 1970). Leech and Paterson (1973) stated that all cases of CSF otorrhea should receive chemotherapy from the time of injury until a week after the leak has ceased and an awareness that meningitis may develop should be maintained especially if a fracture is present or if the leak continues for more than seven days. Johnson and Dutt (1947) from their extensive studies of World War II casualties, concluded that meningitis was much less common after CSF otorrhea than after rhinorrhea and that otorrhea is usually of shorter duration.

Summary

A case of cerebrospinal fluid otorrhea after head injury following myringotomy is presented. Detection of cerebrospinal fluid was simply by way of microscopic examination and chemical analysis. Tomographic study and Stenver's projection were unable to reveal any sign of fracture of the petrous bone. The leak stopped spontaneously without neurosurgical attempt.

The relevant literature is briefly reviewed.

Acknowledgements

Our thanks are due to the Subdivisions of Child Radiology and Child Neurology, Department of Child Health, and the Department of Neurosurgery and to Dr. Marliane Wydia, for permission to report this case admitted under her supervision.

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