CLINICAL RECORDS

CEREBELLAR ABSCESS

By WILFRED LEACH (London)

SERIOUS intracranial complications arising from a chronic ear used to occur frequently enough not to warrant recording any particular case. The standard textbooks still detail stage by stage the classical progress of a neglected chronic ear which leads to intracranial complications and most otologists can recall several instances before the days of antibiotics. The rarity of this occurrence with present-day surgical and antibiotic treatment prompts one to record a recent clinical case which, although classical in its evolution, should rank equally worthy of inclusion amongst the rare, isolated and unusual records we read in these pages as a salutary reminder of such a possibility despite modern treatment.

Clinical Record

P.S. had suffered intermittent left otorrhœa since 1934 and it was not helped by the "water torture" he suffered while a prisoner of war in a Japanese camp. This consisted of a continuous drip of cold water on to the upturned ear for hours at a time. He suffered an episode of ataxia in *November, 1961*, and sought advice at St. Thomas' Hospital the following month.

The left ear showed chronic attic suppurative disease but his hearing was not badly affected, some 25–30 db. conduction hearing loss for speech frequencies being recorded. A left modified radical mastoidectomy was therefore performed on *February 5th, 1962*, and no fistula into the labyrinth was noted. On *April 1st*, he had a sudden episode of characteristic vertigo when objects seemed to move from left to right, therefore his nystagmus was presumably to the right, away from the diseased ear and thus paralytic. This was confirmed when he was eventually examined and he was also found now to be stone deaf in the left ear though he had had a relatively undisturbed post-operative hearing threshold. Ten days later his giddiness had disappeared.

On *June 13th, 1962*, he was readmitted with left temporal headache, neck stiffness, nystagmus to both sides and tenderness over the upper end of the left sterno-mastoid. Observation over a few days revealed an intermittent pyrexia, but repeated blood cultures were sterile. A swab from pus in his left mastoid cavity grew *Proteus vulgaris*. A thrombosis of the left sigmoid sinus was diagnosed. Six days after admission the mastoid cavity which had previously been exenterated was re-opened and the middle and posterior fossa dura widely exposed. No thrombosis of the sigmoid sinus was found but some

https://doi.org/10.1017/S002221510006196X Published online by Cambridge University Press

diseased peri-labyrinthine cells and a necrosed posterior half of the lateral semi-circular canal were removed. After this his headaches improved temporarily and he was sent home. He again complained of violent headache in August and of some unsteadiness of gait but his mastoid cavity was pronounced virtually dry.

The patient was readmitted to hospital on *October 6th* with violent headaches, vomiting, transient diplopia, giddiness and ataxia. He was seen to have neck stiffness, a right sixth cranial nerve palsy and coarse nystagmus to the left side. Later he became increasingly confused and disorientated and co-ordination was impaired on the left. A lumbar puncture showed 50 white cells/c.mm. and 140 mg. per cent. of protein.

A diagnosis of a left posterior fossa abscess was made and he was transferred to the National Hospital for Nervous Diseases, Maida Vale, where a left cerebellar burr hole was made, the abscess found and evacuated and the cavity filled with a mixture of Penicillin, Streptomycin and Thorotrast. Subsequently the abscess cavity was recanulated and the pus replaced with this mixture daily for 3 days while the progressive diminution in size of the abscess was followed radiologically. *Pseudomonas pyocyaneus* and *Streptococcus fæcalis* were grown from the pus. After 2 weeks, when the abscess cavity had shrunk sufficiently and the culture of pus was sterile, the former was excised at a posterior fossa craniectomy. The patient recovered uneventfully and was discharged exactly 1 month after admission.

He was kept under observation in the E.N.T. out-patient department at St. Thomas' Hospital and apart from a slight discharge from his left mastoid cavity was well until *November 20th, 1962*, when he again developed giddiness and unsteadiness of gait, exhibiting nystagmus to the left and poor co-ordination on the left. On re-admission to the National Hospital for Nervous Diseases, there was a discharge from the craniectomy wound. He developed increasing headache and neck stiffness and a lumbar puncture revealed an increased fluid cell content. Ventriculography revealed that the abscess had recurred and treatment as before was recommenced. The abscess seemed to diminish in size as previously but unfortunately he became comatose and died fairly suddenly on *November 30th*.

An autopsy showed a recurrent cerebellar abscess and in addition an extra-dural collection of pus on the posterior aspect of the petrous temporal bone related to the original mastoid sepsis. Thus this reservoir of infection had caused the abscess to recur and also produced a localized posterior fossa meningitis during his last illness.

Discussion

While the incidence of intracranial complications of middle-ear disease has fallen from round about 2 per cent. in the pre-antibiotic era (Hegener, 1908; Fraser and Halliday, 1936) to less than 0·15 per cent. in Jeanes' (1961) series of patients attending the Royal National Throat, Nose and Ear Hospitals between 1951 and 1960, once such complications have developed there is still a high mortality incidence, especially for brain abscess. It was 40 per cent. for the latter in Jeanes' series and Dawes (1961) gave a figure of 50 per cent. from Newcastle upon Tyne between 1944 and 1960.

https://doi.org/10.1017/S002221510006196X Published online by Cambridge University Press

The above record illustrates how insidious the development of a brain abscess can be. From the ear the labyrinth was first infected and in spite of the diseased portion thereof being removed surgically, the infection then spread towards the posterior fossa, first forming an extra-dural abscess and finally transgressing the meningeal barrier to produce a cerebellar abscess. Had this ear not been exposed and drained twice already, the natural sequence would have been for the craniectomy to be followed by a mastoidectomy.

Conclusions

Brain abscess is an insidious and highly dangerous disease still to be reckoned with as a complication of chronic suppurative otitis media. Its development can be greatly protracted, it quite often proves fatal and recovery may be followed by permanent mental changes—all this in spite of modern surgical and antibiotic treatment. Otologists should however bear in mind that these last remarks apply equally to non-otogenic brain abscesses.

I am grateful to Mr. G. H. Bateman for allowing me to publish these details of his patient's case.

REFERENCES

DAWES, J. D. K. (1961) *Proc. roy. Soc. Med.*, **54**, 315.
FRASER, J. S., and HALLIDAY, G. C. (1936) *J. Laryng.*, **51**, 619.
HEGENER, J. (1908) *Z. Ohrenheilk.*, **56**, 3.
JEANES, A. (1962) *J. Laryng.*, **76**, 388.

8 Barnstaple Mansions,
Rosebery Avenue,
London, E.C.1.

https://doi.org/10.1017/S002221510006196X Published online by Cambridge University Press