

# Cerebellar abscess

## A review of 47 cases

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**SYNOPSIS** Forty-seven cases of cerebellar abscess have been reviewed, 93% of which were secondary to otogenic disease. There has been little change in the annual incidence during the period of time under review. The overall mortality was 41%, but with successive decades the mortality has increased. Three factors appear to be of importance in determining survival: the patient's ability to control his infection; reduction of the effect of the posterior fossa mass, preferably by complete excision of the abscess under antibiotic cover; and, in the case of otogenic abscess, an adequate radical mastoidectomy with bone removal to the site of attachment of the abscess to the dura mater.

Macewen (1898) in managing cerebellar abscess had a mortality of 55% in his nine cases, thus demonstrating the lethal nature of this condition, which even now remains treacherous. Samson and Clark (1973) in their review of the current status of brain abscess concluded that the mortality relates mainly to the rise in intracranial pressure, with or without associated brain distortion, rather than the infective element. They, therefore, recommended reduction of the effect of the mass, preferably by total excision of the abscess under antibiotic cover. Pennybacker (1948) came to a similar conclusion about the specific treatment of cerebellar abscess, as in his 18 patients who underwent a variety of surgical decompressive procedures, there was a marked degree of improvement in the mortality rate after

the introduction of antibiotic therapy (Table 1). However, Schreiber (1941) reported nine children with cerebellar abscess, treated only by aspiration through a trephine hole, with long-term success in seven patients.

A retrospective review of the cerebellar abscesses presenting in Glasgow was therefore undertaken in an attempt to resolve this apparent contradiction.

### METHODS

**INCIDENCE** Over the period December 1950 to December 1973, 47 cases of cerebellar abscess were seen at the Institute of Neurological Sciences, Glasgow (Table 2). The male:female ratio was 32:15. There was a slightly increased incidence in the teenage and 40 to 59 years age groups (Table 2).

**TABLE 1**  
EFFECT OF ANTIBIOTIC ON MORTALITY\*

|               | <i>Alive</i> | <i>Dead</i> | <i>Total</i> |
|---------------|--------------|-------------|--------------|
| Antibiotic    | 8            | 1           | 9            |
| No antibiotic | 2            | 7           | 9            |
|               | 10           | 8           | 18           |

\* Data from Pennybacker (1948).  
 $\chi^2 = 7.92$ ;  $P < 0.005$ .

**TABLE 2**  
INCIDENCE

| <i>Age (yr)</i> | <i>1950-59</i> | <i>1960-69</i> | <i>1970-73</i> | <i>Total</i> |
|-----------------|----------------|----------------|----------------|--------------|
| 0-9             | 1              | 3 (1)          | —              | 4 (1: 25%)   |
| 10-19           | 5              | 6 (3)          | —              | 11 (3: 27%)  |
| 20-29           | 2              | 3 (2)          | —              | 5 (2: 40%)   |
| 30-39           | 3              | 1              | 1              | 5 (-: 0%)    |
| 40-49           | 1 (1)          | 3 (1)          | 4 (2)          | 8 (4: 50%)   |
| 50-59           | 3 (2)          | 4 (2)          | 3 (2)          | 10 (6: 60%)  |
| 60-69           | 1 (1)          | —              | 3 (2)          | 4 (3: 75%)   |
|                 | 16 (4: 25%)    | 20 (9: 45%)    | 11 (6: 55%)    | 47 (19: 41%) |

Deaths and mortality in parentheses.

**SOURCE OF INFECTION** Forty-four cases (93%) were secondary to infective otogenic disease, there being no difference in incidence between the two sides (Table 3). Thirty-five patients had chronic ear disease

TABLE 3  
PRIMARY SOURCES

| Site             | Cases (no.) |
|------------------|-------------|
| Otogenic disease | 44          |
| Right 22         |             |
| Left 22          |             |
| Tonsils          | 1           |
| Chest            | 1           |
| Septicaemia      | 1           |
|                  | 47          |

with previous histories extending from 2 to 33 years. Of these patients, 32 experienced an acute exacerbation of their disease at or about the time they developed their cerebellar complication. Seven patients had acute ear disease only. In the remaining two patients, the ear disease was not diagnosed until postmortem examination.

Three cases were metastatic in origin. One complicated acute tonsillitis, another a chest infection in a patient with Fallot's tetralogy, and the third in a case of septicaemia which complicated catheterization for acute retention of urine.

The commonest organism found at the site of primary infection was *Proteus mirabilis* (Table 4).

**CEREBELLAR INFECTION** Twenty-five of the abscesses were situated in the right cerebellar hemisphere and 22 in the left one. There were no cases of multiple abscesses. *Proteus* and other Gram negative bacilli were the commonest organisms found (Table 4). A mixed flora was found in 11 cases, and no organisms found in seven cases.

**SYMPTOMS AND SIGNS** All 47 patients experienced headache, which was of greater than one week's duration in 26 of them. It was commonly associated with vomiting, drowsiness, and unsteadiness (Table 5).

At the time of admission 14 patients were fully alert, 24 were drowsy but capable of obeying simple commands, six responded only to deep pain, and two were totally unresponsive. No record is available of the final patient's level of response (Table 6). Nystagmus, neck stiffness, and incoordination of

TABLE 4  
ORGANISMS IDENTIFIED

|                                 | Primary site |                 | Cerebellar abscess |                 |
|---------------------------------|--------------|-----------------|--------------------|-----------------|
|                                 | Culture      | Microscopy only | Culture            | Microscopy only |
| <i>Proteus mirabilis</i>        | 11           | —               | 9                  | —               |
| Coliforms                       | 4            | —               | —                  | —               |
| Gram negative bacilli           | —            | 4               | —                  | 10              |
| <i>E. Coli</i>                  | —            | —               | 2                  | —               |
| Gram positive cocci             | —            | 3               | —                  | 9               |
| <i>Staph. aureus</i>            | 3            | —               | 6                  | —               |
| <i>Staph. albus</i>             | 1            | —               | 5                  | —               |
| <i>Strep. pneumoniae</i>        | 1            | —               | 5                  | —               |
| <i>Strep. viridans</i>          | —            | —               | 1                  | —               |
| Non-haemolytic anaerobic strep. | —            | —               | 4                  | —               |
| Diphtheroids                    | 2            | —               | 1                  | —               |
| Micrococcus                     | 1            | —               | —                  | —               |
| No report                       | 10           | —               | —                  | —               |

TABLE 5  
CLINICAL FEATURES

|                           | Cases |     |
|---------------------------|-------|-----|
|                           | (No.) | (%) |
| <i>Symptoms</i>           |       |     |
| Headache                  | 47    | 100 |
| Vomiting                  | 39    | 83  |
| Drowsiness                | 32    | 66  |
| Unsteadiness              | 23    | 49  |
| Confusion                 | 16    | 34  |
| Ipsilateral limb weakness | 6     | 13  |
| Visual disturbance        | 4     | 8   |
| Blackout                  | 3     | 6   |
| <i>Signs</i>              |       |     |
| Nystagmus                 | 35    | 74  |
| Neck stiffness            | 31    | 66  |
| Incoordination            | 27    | 57  |
| Papilloedema              | 21    | 45  |
| Pyrexia                   | 16    | 34  |
| Sixth nerve palsy         | 2     | 4   |

TABLE 6  
CLINICAL RESPONSE LEVELS

|                             | Alive | Dead | Total |
|-----------------------------|-------|------|-------|
| Alert                       | 10    | 4    | 14    |
| Drowsy/simple commands      | 17    | 7    | 24    |
| Painful stimuli/no response | 1     | 7    | 8     |
| No record                   | —     | 1    | 1     |
|                             | 28    | 19   | 47    |

$\chi^2 = 10.84$ ;  $P < 0.02$ .

the limbs on the affected side were the commonest physical signs (Table 5).

**INVESTIGATIONS** Ten patients underwent carotid angiography. In seven, dilated lateral ventricles were revealed, and in three this study was unhelpful. Of the seven cases, two proceeded to vertebral angiography, one of which revealed a right cerebellar mass and in the other the basilar artery was seen to be displaced anteriorly.

Ventriculography was performed in 43 cases—25 with air, seven with iophendylate (Myodil, Glaxo), five with meglumine iothalamate 35% w/v (Retro-Conray, May & Baker), three with meglumine iothalamate and iophendylate, two with air and iophendylate, and one air and meglumine iothalamate. In only one case was the ventriculogram normal. In the remaining 42 studies positive localization was obtained.

Ventricular cerebrospinal fluid was sent for analysis in 18 cases. The white cell count varied from 0 to 1 890 cells/mm<sup>3</sup> (mainly lymphocytes in all cases), the protein estimation was 4–100 mg/dl, and the sugar 50–144 mg/dl. Analysis of the lumbar cerebrospinal fluid was available in 13 cases; the white cell count varied from 1–1 750 cells/mm<sup>3</sup>, the differential count being similar to that of the ventricular fluid; the protein from 37–100 mg/dl, and the sugar 22–73 mg/dl.

**TREATMENT** Forty-one patients underwent surgical treatment, all of whom received postoperative antibiotics (Table 7). One patient underwent a negative posterior fossa exploration, the postoperative diagnosis of acute haemorrhagic leucoencephalitis being supported on biopsy. Postmortem examination three weeks later revealed a right sided cerebellar abscess. In the five patients who died without undergoing posterior fossa surgery, the tentative diagnosis was of a purulent meningitis in three cases and a

TABLE 7  
EFFECT OF OPERATIVE TREATMENT ON MORTALITY

|                                 | Alive | Dead | Total |
|---------------------------------|-------|------|-------|
| Aspiration                      | 11    | 5    | 16    |
| Aspiration + late excision      | 2     | 2    | 4     |
| Partial excision                | 1     | 1    | 2     |
| Complete excision               | 14    | 5    | 19    |
| Negative exploration/no surgery | —     | 6    | 6     |
|                                 | 28    | 19   | 47    |

$\chi^2 = 11.27$ ;  $P < 0.025$ .

posterior fossa tumour in two cases, each of whom died before exploration. In those patients with otogenic abscess the dural attachment was usually found to be superior, anterior, and medial to the internal auditory meatus.

**TREATMENT OF PRIMARY SOURCE** Of the 44 patients whose primary source of infection was otogenic, 29 underwent a radical mastoidectomy for their disease (Table 8). These 29 cases include eight patients whose mastoidectomy preceded their cerebellar surgery, and of whom five developed their intracranial symptoms after mastoid surgery. Two of these patients

TABLE 8  
TREATMENT OF OTOGENIC SOURCE

|                             | Alive | Dead | Total |
|-----------------------------|-------|------|-------|
| Radical mastoidectomy       | 25    | 4    | 29    |
| Local cleaning + antibiotic | 2     | 11   | 13    |
| None                        | —     | 2    | 2     |
|                             | 27    | 17   | 44    |

$\chi^2 = 20.26$ ;  $P < 0.001$ .

required further surgery to their mastoid cavities. Only two of the cases undergoing other types of treatment for their mastoid disease would have been unfit for mastoid surgery at some time after their presentation. The two patients receiving no treatment were not recognized in life as having otogenic disease.

The primary source, in those patients with metastatic cerebellar abscesses, was treated with appropriate antibiotics, and, in the case of the chest infection, physiotherapy.

**RECURRENT CEREBELLAR ABSCESS** Ten patients developed recurrent cerebellar abscesses. All were secondary to otogenic disease and occurred between the 11th and 28th postoperative days, except for one which recurred at three months (Table 9). One case was re-aspirated and the remainder underwent excision, which was incomplete in two cases because of adherence of the capsule to the pons (Table 10).

**MORTALITY** Nineteen patients died, which gives an overall mortality of 41%. Two patients died late; one at 14 months postoperatively from recurrent *Proteus mirabilis* meningitis complicating the ventriculoperitoneal shunt, which had been required for the treatment of subsequent hydrocephalus, and one

TABLE 9  
RECURRENT ABSCESS

| Treatment of cerebellar abscess | No. | Recurrence |
|---------------------------------|-----|------------|
| Aspiration                      | 16  | 4          |
| Aspiration + late excision      | 4   | —          |
| Partial excision                | 2   | 2          |
| Complete excision               | 19  | 4          |
|                                 | 41  | 10         |

TABLE 10  
TREATMENT OF RECURRENT ABSCESS

|                   | Alive | Dead | Total |
|-------------------|-------|------|-------|
| Aspiration        | —     | 1    | 1     |
| Partial excision  | 1     | 1    | 2     |
| Complete excision | 7     | —    | 7     |
|                   | 8     | 2    | 10    |

at four months in whom the cause of death is not known, but who, postoperatively, had severe lower cranial nerve deficits. Of the three patients with metastatic cerebellar abscesses, two died, the survivor being the patient who had had tonsillitis.

**MORBIDITY** Twelve of the 28 survivors (43%) made a complete recovery from their cerebellar abscess, though one patient developed a complete seventh nerve palsy after mastoid surgery. A second of these patients developed trigeminal neuralgia on the same side as the abscess, some 14 months after surgery. A further 13 patients (46%) had a very mild ataxia or nystagmus which did not interfere with their normal activities. One of these patients also had a complete seventh nerve palsy after the mastoid surgery. Three patients (11%) have a major degree of ataxia, such that they require aid when walking, or have limitations on the use of their upper limbs.

Four patients (14%) have not returned to their work, three of these being the patients with a marked degree of ataxia.

#### DISCUSSION

Wright and Grimaldi (1973) have reported that, in Oxford, there has been a dramatic fall in the number of otogenic brain abscesses over the successive past three decades, though there was no change in relative incidence of temporal lobe

to cerebellar abscesses. In Glasgow, the yearly incidence of cerebellar abscess has varied from nought to five, but the variation between decades is less marked (Table 2). Otogenic disease was the primary source of infection in 94% of the abscesses, which is in keeping with previous workers' experience (Macewan, 1893; Eagleton, 1922; Atkinson, 1934; Schreiber, 1941; Pennybacker, 1948, 1951, 1961; Krayenbühl, 1967). This is in contradistinction to supratentorial abscess, which recent series (Carey *et al.*, 1972; Samson and Clark, 1973; Martin, 1973) suggest are more frequently metastatic and post-traumatic than otogenic, though this would not be our experience in Glasgow. Of our cerebellar abscesses, 16% resulted from acute otogenic disease, whereas the remainder resulted from chronic disease. This is similar to Atkinson's figures (1934) of 20% from acute, and 80% from chronic disease. All Dawes's (1969) cases complicated chronic disease.

Previous series in which bacteriological studies have been available (Schreiber, 1941; Pennybacker, 1948) have shown that the commonest infecting organisms were the pneumococcus and both the haemolytic and non-haemolytic streptococcus. In the present series the commonest organisms are the Gram negative coliforms and, in particular, *Proteus*. There has not been any tendency towards change in the period under review. In addition, mixed flora have become more common. As all our cases occurred in the antibiotic era, whereas this was not the case with the previous series, it would seem likely that this change is due to the widespread use of antibiotics in infective ear and upper respiratory tract disease.

The clinical features have been well summarized by Dow and Moruzzi (1958), but it is worth emphasizing that cerebellar abscess remains treacherous, because signs of impaired cerebellar function frequently appear late in the natural history of the disease.

Ventriculography was the usual method of investigation used in this series. In the majority of cases air was used, but more recently meglumine iothalamate is the contrast medium of choice. In only one case subsequently shown to have a cerebellar abscess was the ventriculogram normal. This patient, on biopsy during a posterior fossa exploration, was diagnosed as having an

acute haemorrhagic leucoencephalitis. Neither ventricular nor lumbar cerebrospinal fluid examination appears to be of help in the diagnosis of cerebellar abscess, as they may both be normal or abnormal.

Dow and Moruzzi (1958) have reviewed the opinions concerning the routes of infection through the petrous temporal bone to the cerebellum. The two most important channels were considered to be, direct extension from a suppurative labyrinthitis, and by retrograde thrombosis from the lateral, the inferior petrosal, or superior petrosal sinuses. Other much less frequent pathways were the preformed canals—the internal auditory meatus, the facial canal, the subarcuate fossa, the vestibular aqueduct and the cochlear canaliculus, and direct extension from the air cells.

In our series, and those of Pennybacker (1948) and Krayenbühl (1967), the dural attachment of the abscess was commonly superior, anterior, and medial to the internal auditory meatus. This is the situation of venous connections between the inferior and superior petrosal sinuses. The former receives veins draining the vestibular aqueduct, the middle ear, the diploic tissue around the semicircular canals, and the subarcuate vein, while the latter receives tributaries from the cochlear aqueduct, the vestibular aqueduct, and the internal auditory vein (Kalbag and Woolf, 1967), in addition to draining veins from the rostradorsal aspect of the cerebellar hemisphere (Kaplan and Ford, 1966). As bony necrosis was seen in the posterior fossa in only three cases in this series, this would suggest that an infective thrombophlebitis involving the inferior and superior petrosal sinuses and their connections is the most important pathway of the four possibilities that have been advanced.

The overall mortality in our series remains at 41%. When considered in decades there has been a gradual worsening in mortality; 1950–1959, 25%; 1960–1969, 45%, and 1970–1973, 55% (Table 2). Using the chi-squared test for significance and accepting a value of 0.05 as being significant, various factors were analysed to see if they influenced the mortality. Neither the use of antibiotics before cerebellar surgery, nor the ability to grow organisms from the primary site influenced the outcome. On considering symptomatology, no significant correla-

TABLE 11  
DURATION OF SYMPTOMS

| <i>Duration (weeks)</i> | <i>Alive</i> | <i>Dead</i> | <i>Total</i> |
|-------------------------|--------------|-------------|--------------|
| Under two               | 26           | 11          | 37           |
| Over two                | 2            | 8           | 10           |
|                         | 28           | 19          | 47           |

$\chi^2 = 8.26; P < 0.005.$

tion was found with any particular symptom, but if the duration of intracranial symptoms was less than two weeks the outlook was better than if it exceeded this period (Table 11). Similarly, the prognosis was bad if the patient was either unresponsive or would respond only to painful stimuli (Table 6).

Complete excision as a primary procedure is marginally better than aspiration (Table 7). This superiority is more apparent if the recurrent abscesses are also considered, and would help to support the views of Pennybacker (1948) and Krayenbühl (1967) that excision is the treatment of choice. However, a complete excision is more likely to be attempted and achieved if the abscess has a well-developed capsule (Table 12). This

TABLE 12  
CAPSULE

|              | <i>Alive</i> | <i>Dead</i> | <i>Total</i> |
|--------------|--------------|-------------|--------------|
| Present      | 16           | 10          | 26           |
| Absent       | 4            | 8           | 12           |
| Not recorded | 8            | 1           | 9            |
|              | 28           | 19          | 47           |

$\chi^2 = 6.60; P < 0.05.$

feature in itself improves the survival rate and, combined with the finding that a short history of intracranial disease also improves the prognosis, suggests that it may be the patient's ability to control his own infection which is the important feature.

Analysis of the effect of treatment of the primary source of infection upon the mortality rate leads to the conclusion that, if the primary

source is the ear, patients who have undergone radical mastoidectomy have a low mortality rate, whereas other forms of treatment are uniformly disastrous (Table 8). Eagleton (1922) found that many cerebellar abscesses occurred either as the direct result of the operative manipulation during mastoidectomy, or from postoperative secondary infection. That this argument does not apply to the present series is suggested by the fact that only eight patients underwent their mastoid surgery before the posterior fossa surgery, and of these only five developed intracranial symptoms after their mastoid surgery. Presumably, the use of antibiotic cover during mastoidectomy has reduced the incidence of the situation described by Eagleton.

In addition, the deterioration from decade to decade, in part, reflects a temporary change in the attitude to the treatment of the otogenic source. During the period 1950–59, when the mortality was 25% (Table 2), 75% of the patients with otogenic disease underwent radical mastoidectomy. In the second decade, when the mortality rose to 45%, the percentage undergoing radical mastoidectomy fell to 55%. Over the last four years, radical mastoidectomy was performed on 70% of the patients. Additional factors are, therefore, necessary to explain the increased mortality of 55% during this time interval. Between 1950 and 1959, one case of cerebellar abscess was not diagnosed in life (6%); there was a further case between 1960 and 1969 (5%), while in the last four years there were four cases (36%). On looking at the age distribution during the three periods of time under review, patients were noticeably older during the latest of the three periods (Table 2). Furthermore, if the age group birth to 39 years is considered, six patients died out of a total of 25 (24%), whereas in the over-40s, 13 out of 22 died (59%) ( $P < 0.02$ ). Therefore, it would seem that the mortality in the last four years is related to the difficulty in diagnosis of a posterior fossa mass lesion in the older age groups. This may result from a degree of brain atrophy, which delays the development of the signs of raised intracranial pressure. We would, therefore, support the contention of Wright and Grimaldi (1973) that radical mastoidectomy should be undertaken as soon as the intracranial mass of the abscess has been controlled. Unlike Wright

and Grimaldi (1973), we have no experience of using the same anaesthetic for both the mastoid and the intracranial surgery.

#### CONCLUSIONS

The incidence of otogenic cerebellar abscess in Glasgow remains constant, and would seem to be secondary to the high incidence of chronic ear disease in the West of Scotland.

The overall mortality from cerebellar abscess remains high at 41%. Furthermore, the mortality has continued to rise over successive decades. The reasons for this are discussed.

The commonest infecting organisms have changed from the pneumococcus and the streptococcus to the Gram negative bacilli.

Under appropriate antibiotic cover, the posterior fossa should be decompressed urgently, and preferably by complete excision of the abscess.

The patient with otogenic disease should undergo radical mastoidectomy, to control the infective element, once the intracranial pressure is under control. In addition, bone should be resected to the point at which the abscess was found attached to the dura mater. This becomes mandatory if extradural pus has been demonstrated, or is suspected, at the time of the intracranial surgery.

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