Treatment of Basal Skull Fractures With and Without Cerebrospinal Fluid Fistulae

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The primary goal of therapy in fractures of the base of the skull is the prevention of intracranial infection; in all such cases the subarachnoid space must be considered potentially contaminated. Before the advent of antibiotics, this risk was high, and whenever a cerebrospinal fluid fistula was present, many felt that meningitis was inevitable. There are no large studies of the incidence of meningitis in head injuries before the advent of antibiotics. Teacheneor in 1927 was able to lower the mortality from infections in fractures through the frontal sinus from 68% to 13% by early repair of the dura and drainage of the sinus in a small series of patients. Munro reported a 60% mortality in five cases of persistent rhinorrhea following head injury. Calvert noted that 50% of 29 patients with cerebrospinal fluid (CSF) rhinorrhea developed meningitis. With the use of antibiotics, the risk of meningitis in the acute state of basal skull fractures has been markedly reduced. Most neurosurgeons advocate surgical repair of the dura only in cases of persistent cerebrospinal fluid rhinorrhea or otorrhea.\(^1\)\(^,\)\(^2\)\(^,\)\(^4\)\(^\text{-}\)\(^6\)\(^,\)\(^11\)\(^,\)\(^12\) Lewin,\(^9\) on the other hand, advocated operative closure of the dura in all patients with rhinorrhea and basal skull fractures as soon as their condition permits. He based this conclusion on a comparison of 26 patients with rhinorrhea who were not operated on and 55 patients who had craniotomy and dural closure. Six of the 26 unoperated patients developed meningitis, four of whom died, while only one of the 55 operative patients developed meningitis and this was not fatal.

However, if operative treatment is confined to cases with cerebrospinal fluid leak, some cases will not be treated which will later develop meningitis. Of 128 cases of paranasal sinus fracture, Calvert noted six cases of meningitis in patients who had no history of rhinorrhea or otorrhea. It seemed worthwhile to review our own case material to determine the risk of infection in basal skull fractures with and without CSF leak to see if the cases of recurrent meningitis could not be predicted during the acute illness.

Case Material

All cases of basal skull fracture at King County Hospital for the 5-year period from 1953 to 1958 were reviewed and follow-up attempted to determine the incidence of infection in an unselected group of cases with this diagnosis. In addition, all cases of meningitis associated with a previous basal skull fracture over a 15-year period, 1947 to 1962, at the same hospital were reviewed in a further attempt to see how frequently recurrent meningitis occurred following a basal skull fracture. We have included two additional cases to demonstrate our indications for the occasional patient who needs operative repair of a dural defect.

The diagnosis was determined on clinical grounds since only a small portion of basal skull fractures can be located by x-ray. Significant clinical findings included the drainage of blood or cerebrospinal fluid from the nose or ears, bilateral peri orbital ecchymosis, Battle’s sign, and anosmia. Undoubtedly, some patients were treated as basal skull fractures who did not have a fracture because we were using clinical criteria for diagnosis. The treatment was uniform except for the type of antibiotics given. Procaine Penicillin 600,000 units with Streptomycin 0.5 gm twice daily or Chloromycetin 250 mg four times daily were used. Patients were placed on antibiotics as soon as the diagnosis was made and were kept on them for a minimum of 5 days after the leakage had stopped. All patients received a lumbar puncture on admission; frequently the diagnosis of CSF leak was further substantiated.

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by a very low CSF pressure. If rhinorrhea or otorrhea were present, repeat lumbar punctures were done daily or twice daily, each time draining off enough fluid to lower the intracranial pressure to that of the atmosphere or until the patient developed headache. This was done to reduce abnormal drainage of CSF through the fistulous tract and to facilitate natural repair. In addition, the patient was placed in semi-Fowler's position and instructed not to blow his nose. For otorrhea, care was taken not to allow any cotton or bandages to obstruct the external auditory canal.

**Results**

**Group 1.** Of 1250 head injuries seen in the 5-year period from 1953–1958, 303 were basal skull fractures (24%). There were 35 cases with a documented CSF leak; this represented 2.8% of the head injuries and 11.5% of the basal fractures (Table 1). All cases with rhinorrhea or otorrhea ceased to leak within 2 weeks under the above regimen of multiple lumbar punctures and elevation of the head. No case of otorrhea persisted beyond 5 days, while one case of rhinorrhea persisted for 2 weeks and required a total of 13 lumbar punctures. Three cases of meningitis developed during the acute illness but all three patients had entered the hospital more than 2 days after their injury and had the infection on admission. None of the 300 patients who received antibiotics from the day of their injury developed meningitis in the acute period. Setting a minimum follow-up of 1 ½ years, we were able to get detailed information on 77 cases which included 27 cases with CSF leak and 50 cases without a leak. The average interval of follow-up was 5 years, with a range from 1 ½ to 12 years. There were no cases of recurrent meningitis in this group with or without otorrhea or rhinorrhea.

**Group 2.** In the 15-year period from 1947 to 1962, we were able to find nine cases of meningitis which were associated with a previous basal skull fracture. Seven of the nine cases entered the hospital 2 days to 1 week after their injury and had meningitis when admitted. One case with pneumocephalus developed staphylococcal meningitis while on Penicillin and died with an associated subdural hematoma. Only one case over this 15-year period had a delayed meningitis. This 12-year-old girl had a severe diastatic fracture and rhinorrhea. She was decerebrate for 2 to 3 weeks post-injury and was not considered for surgical repair. Eighteen months later she developed meningitis, but repair could not be carried out because she was no longer eligible at the charity hospital. As we review her x-rays in retrospect, we consider that she should have had early operative repair because of the diastasis of the fracture site across the cribiform plate.

We have three indications for operative closure of a dural defect in basal skull fractures not due to a missile: (1) Recurrent meningitis, (2) X-ray evidence of herniation of the brain into the sinuses through a large diastatic fracture, (3) X-ray evidence of a spicule of bone projecting into the brain. In order to demonstrate herniation of soft tissue into the ethmoid air cells, laminography in the frontal plane is essential. We are reporting two recent cases to emphasize these points.

**Case Reports**

**Case 1.** A 42-year-old man was admitted to the Neurological Surgery Service during his fourth episode of meningitis. He had had an auto accident 5 years before in which he had sustained a right frontal skull fracture, but had had no rhinorrhea with his acute injury. Following his third bout of meningitis a right radical mastoidectomy was performed because of a chronic mastoiditis that was considered to be a possible source of infection. Since the accident he had had generalized seizures which were controlled with medication. Physical examination was normal except he was unable to smell out of the right nostril. Skull x-rays showed an old frontal fracture, and frontal laminograms (Fig. 1) demonstrated soft tissue herniation through a defect in the floor of the frontal fossa into the ethmoid air cells. Pneumoencephalography revealed enlargement of the
right frontal horn, and electroencephalography revealed bilateral slow frontal waves. At operation a $1.0 \times 0.7$ cm quadrangular defect was present in the cribriform plate with sclerotic frontal lobe herniating into the ethmoid. Postoperatively the patient has done well with no recurrence of meningitis for 1½ years.

Case 2. A 48-year-old man was admitted to our service during the terminal state of meningitis and cavernous sinus thrombosis. He had been in an auto accident 4 years before, sustaining a ruptured liver in addition to a severe head injury with a linear fracture of the skull. Although there was no mention made of rhinorrhea during the acute illness, he developed meningitis 3½ years later, at which time plain skull x-rays showed a probable mass in the ethmoid air cells on the left. No laminograms were done, however, and he was not seen by a neurosurgeon. Ten months later he was admitted to the hospital in coma with meningitis and died in 3 days. A postmortem examination showed a hole in the roof of the orbit with herniation of sclerotic brain through the defect. In retrospect, it is apparent that frontal laminography would have shown the soft tissue herniation into the ethmoid, and that this patient required operative closure of the dural defect.

Discussion

It has been our experience that most non-missile basal skull fractures with or without cerebrospinal-fluid leak will heal without sequelae. Our method of treatment is to place all patients on moderate doses of prophylactic antibiotics and control the CSF leak by elevation of the head and frequent lumbar punctures to keep the intracranial pressure low. There were no cases of rhinorrhea or otorrhea which persisted beyond 2 weeks on this regimen and no cases of acute or delayed meningitis in our study group.

There is no question of the value of prophylactic antibiotics in the acute state. Over a 15-year period, only one patient developed meningitis while on antibiotics. This patient developed a staphylococcal meningitis but had an associated subdural hematoma.

Fig. 1. Case 1. Frontal laminograph through the cribriform plate showing the mass of soft tissue projecting into the ethmoid sinus.
There were seven other cases of meningitis in the acute period but all had entered the hospital 2 or more days after head injury and had meningitis on admission.

Recurrent meningitis, months or years after a basal skull fracture, is unusual in our experience and does not approach Lewin’s incidence of 25% of patients with rhinorrhea. There have been no cases of recurrent meningitis in 27 cases of rhinorrhea or otorrhea with an average follow-up of 5 years. Likewise, only one case of recurrent meningitis developed following a head injury during a 15-year follow-up in a charity hospital where 250 head injuries are treated yearly. It is hard to explain this difference between our results and those of Lewin. One possible cause for the difference is the antibiotic treatment of his patients, which consisted of sulfonamides by mouth, a regimen we would consider inadequate. Raskind, on the other hand, noted only one case of late meningitis in 35 cases of otorrhea or rhinorrhea.

From a review of our own cases of late meningitis and of other published cases, it is apparent that the fistulous tract responsible for the recurrent infection is not a simple dural tear. The tear is usually complicated by herniation of brain through a large bony defect into the sinuses or cribiform plate or a spine of bone projecting into the brain. Simple dural lacerations are frequently produced during frontal craniotomies in association with rupture of the frontal sinus mucosa, but this rarely results in recurrent meningitis. Otorrhea from a basal fracture is also usually associated with an uncomplicated dural tear because of the anatomy of the petrous bone; it likewise is rarely followed by late infection. Most frequently, recurrent meningitis is associated with a large bony defect in the cribiform plate or the roof of the sinuses. This may occur as an extension of a linear fracture or, in the case of the cribiform plate, may occur without any other fractures in continuity with it. In the latter case it is analogous to a “blow-out fracture” of the orbit. It seems likely that due to a marked increase in intracranial pressure at the moment of injury, a “blow-out” occurs through the thin bone overlying the cribiform plate or roof of the sinuses. While plain skull x-rays may fail to show this, frontal laminograms will almost invariably reveal the soft tissue herniation into the air-filled cavities beneath.

The patients with late meningitis may or may not have a history of CSF leak. The two cases presented above did not. Thus surgical therapy based on the presence of CSF leak would miss some of the cases that would later develop meningitis, while it would submit to craniotomy a large group of patients in whom the chance of late meningitis is small. We believe that the decision to use surgery during the acute illness should be determined by the laminographic demonstration of a complicated fracture overlying the cribiform plate or paranasal sinuses. Every patient with an acute CSF leak, anosmia, bleeding from the nose, or any suggestion of a fracture extending into the paranasal sinuses should have frontal laminograms. If the sinuses are filled with fluid, the x-ray examination should be repeated in 2 weeks, after the sinuses have cleared. This kind of immediate approach to basal skull fractures should result in identification of those cases that need surgical repair of the dural defect.

In cases of recurrent meningitis, laminography provides localization of the dural defect preoperatively and indicates the need for unilateral or bilateral exposure. Of course some cases of recurrent meningitis may have an occult cause that will escape recognition by this examination.

This report concerns only non-missile-induced basal skull fractures. For completeness we should therefore add that any missile injury in which the missile traverses a sinus before piercing the dura should have operative exposure and repair of the dura.

**Summary**

Our treatment of basal skull fracture includes prophylactic antibiotics for 5 days, with frequent lumbar punctures and elevation of the head if there is an associated CSF leak.

Of 1250 cases of head injuries treated over a 5-year period, 24% had a non-missile-inflicted basal skull fracture, and 2.8% had proven CSF otorrhea or rhinorrhea. Follow-up of these 77 cases for an average of 5 years revealed no instances of recurrent rhinorrhea, otorrhea, or late meningitis.

Over a 15-year period we saw 8 cases of acute meningitis following head injury, but
only one developed after admission to the hospital. There was only one case of late meningitis in this 15-year period.

The decision to repair a dural defect during the acute period should be determined by the laminographic demonstration of a spicule of bone projecting into the brain or herniation of brain into a sinus through a diastatic fracture or large bony defect. Surgical therapy based only on the presence of an acute CSF leak is not warranted.

References