CHRONIC OSTEOMYELITIS OF BASE OF SKULL

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[WITH SPECIAL PLATE]

Osteomyelitis of the base of the skull appearing together with or soon after infection of the sinuses or middle ear presents no diagnostic difficulties. This is not the case, however, where chronic osteomyelitis develops without such previous manifestations. Differentiation from neoplastic disease, in particular, is then by no means easy, nor is it clear whether the inflammation is primary or has developed by a process of extension from neighbouring tissues. Radiographs of the skull, even when abnormal, do not give unequivocal information as to the nature of the lesion. Signs and symptoms are equally difficult to relate to an accurate localization. The condition is rare and we have seen only three cases in the past fifteen years; two were successfully operated upon.

Case 1

A 15-year-old girl was admitted to the department of internal medicine in 1948 because of severe headaches, double vision, and vomiting. During the previous four years she had had episodes of fever and headaches, and three years before admission had been under psychiatric investigation for the latter symptom. Double vision appeared six weeks before the present admission, and vomiting a couple of weeks later.

Bilateral paresis of the sixth nerve and also paresis of the right third nerve were noted; there was some congestion of the retinal veins. No other neurological or ophthalmological abnormalities were noted, and the general physical examination was negative. Radiographs of the skull showed gross loss of density of the sella turcica, which could barely be made out. The cerebrospinal fluid contained 65 mg. of protein per 100 ml. and 10 white cells per c.mm., mostly lymphocytes. The erythrocyte sedimentation rate was 70 mm. in the 1st and 105 mm. in the 2nd hour, and there was a mild anaemia but no leucocytosis (R.B.C. 3,700,000 per c.mm.; W.B.C. 7,800 per c.mm.). Wassermann and Kahn reactions negative; urine analysis normal.

The patient complained constantly of severe headache and was generally very restless. Fever ranged from 37° to 39° C. (98.6° to 102.2° F.). Ten days after admission the third-nerve paralysis on the right became complete. Penicillin, 2,000,000 units daily, and parenteral fluids were administered but her condition deteriorated with repeated vomiting and she died 16 days after admission.

At necropsy tissue thought to be neoplastic was found to have invaded the walls of the sella turcica and replaced the pituitary gland (Special Plate, Fig. 1). The same process had destroyed the tip of the right pyramid. Sagittal section showed penetration of the tuber cinereum and the floor of the third ventricle and involvement of the greater wing of the sphenoid on both sides.

Histological examination revealed, however, that the pathological material was chronic inflammatory tissue, probably originating in the neighbourhood of the sphenoid sinus. The mucosa from the sinus showed richly cellular granulation tissue with many eosinophil and plasma cells and occasional giant cells. The inflammatory process had

destroyed the body of the sella turcica, in particular the dorsum, and wherever it had invaded bone it contained many fibroblasts and osteoclasts.

Case 2

An 11-year-old boy with fever, mild exophthalmos, and congestion of the left eye was admitted to hospital because x-ray examination had shown destruction of the orbital roof. His illness had begun six months previously with fever followed by oedema of the eyelids on the left with proptosis and headache. Though the fever and general symptoms responded to treatment with penicillin, the ocular signs did not subside. Repeated radiological examination of the paranasal sinuses showed no abnormality. Later fever returned, this time uninfluenced by penicillin. Further radiography at the end of a six-week period of treatment revealed destruction of the orbital roof on the affected side (Special Plate, Fig. 2).

On his admission there were no abnormal findings on general physical or neurological examination. The left globe was displaced slightly downwards and there was exophthalmos to the extent of 3 mm. Eye movements were unimpaired and the fundus was normal. Laboratory examination gave the following results: W.B.C. 5,200 per c.mm. (polynuclears 29%, lymphocytes 24%, eosinophils 17%, monocytes 8%); R.B.C. 4,700,000 per c.mm.; E.S.R. 18 mm. in the 1st and 42 mm. in the 2nd hour; Kahn, Widal, Weinberg, and Casoni tests negative. Urine analysis and culture were normal.

The various diagnoses considered in the light of the above history and findings included eosinophil granuloma, echinococcus cyst, new growth, and osteomyelitis. Seven weeks after the patient's admission operation was decided upon and a left frontal flap raised. The orbital roof was explored without opening the dura mater, which was firmly adherent to bone. On lifting the frontal lobe, suppurating connective tissue could be seen overlying an ovoid defect in the orbital roof measuring 2.5 by 2 cm. A smooth, normal-looking orbital capsule came into view when the pathological material had been removed by curettage. The rim of the defect was unusually thin, and was removed piecemeal by bone forceps until normal-looking bone was reached. The findings at operation were considered to be a chronic inflammation of the orbital bones, particularly of the roof. The material removed proved bacteriologically sterile. Histological examination showed inflammatory tissue rich in plasma cells and fibroblasts, with many osteoclasts in the bony portions.

Case 3

A 50-year-old man was admitted in 1960 because of attacks of vertigo occurring several times weekly for the previous eight months. More recently he had become aware of tinnitus and deafness in the left ear. Numbness of the left cheek was noticed while shaving and latterly blurring of vision. He was referred from the neurological department with a provisional diagnosis of tumour of the cerebellopontine angle. The physical signs included horizontal nystagmus, hypoaesthesia of the left trigeminal area, and peripheral-type facial weakness on the same side. Deep reflexes were brisker on the left, with an extensor plantar response, and there were also signs of a slight left cerebellar disturbance.

Radiographs of the skull showed partial destruction of the left petrous bone with a defect just lateral to the internal acoustic meatus, the lower wall of which could not be seen (Special Plate, Fig. 3). Hearing of high tones was reduced on the left, as were caloric responses. The usual laboratory investigations all gave normal results with an E.S.R. of 2 mm. in the 1st and 13 mm. in the 2nd hour.

The symptoms and signs as described above were thought to be consistent with a diagnosis of cerebellopontine-angle tumour, though the minimal hearing disturbance, and in particular the site of the bony defect as shown by x-rays, were not appropriate for an acoustic neurinoma.

Suboccipital craniotomy was performed, and when the left cerebellar lobe was raised the dura mater in the region

of the angle was found to be bulging forward over a mass the size of a plum. Several millilitres of pus spurted out when the dura was incised, and the remainder was evacuated by suction. Necrotic bone was removed from beneath the dura. Material sent for examination proved bacteriologically sterile.

After treatment with antibiotics there was a gradual remission of all symptoms and signs.

Discussion

The cases recorded above are examples of chronic osteomyelitis of the skull base without antecedent (or at least not clinically recognized) inflammation of adjacent bony sinuses. The progress of the disease is usually insidious, though it may be marked by acute flare-ups. In the latter the picture is one of an acute inflammatory condition with headache and a neurological syndrome depending on the site of the infection. Examination of the ears and sinuses is negative and radiographs of the skull do not necessarily reveal any change.

Antibiotics given in such circumstances result in disappearance or amelioration of the signs and symptoms and a period of quiescence, perhaps followed later by a further exacerbation. Antibiotic therapy in these cases seems to play a role in their conversion to chronicity; septic material is generally bacteriologically sterile, while histological examination of portions of diseased bone shows chronic inflammatory changes.

The route of infection is difficult to determine in the absence of an acute onset. It is possible that mild respiratory infections lead to infiltrative or metastatic inflammation of the bones of the skull base, and this may have been the situation in our first case, in which post-mortem studies showed involvement of the sphenoid sinus as a possible, though not certain, point of origin. This complication of sphenoid sinusitis is rare, and only a few references have been met with (Proetz, 1949; Smith, 1958; Maxwell and Hill, 1959). In the other two cases there was no evidence pointing to any particular portal of entry. The third patient did not receive any antibiotic treatment during the early part of his illness, but the infection remained chronic and led eventually to abscess formation.

Differential diagnosis is always difficult in these cases, being usually limited to an anatomical localization of some disease process at the base of the skull. Final diagnosis will usually be made only at operation. Surgical therapy should consist of removal of diseased bone and other infected tissues, and be followed by antibiotic therapy. This plan has been pursued in the few cases reported in the literature (Jain *et al.*, 1957; Werner, 1959).

Summary

Three cases of chronic osteomyelitis of the base of the skull are presented, in none of which was there a previous history of inflammation of the ears or paranasal sinuses. Diagnosis was made at necropsy in one case and during surgical exploration in the other two. In these two cases removal of infected bone and granulation tissue was followed by recovery.

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Medical Memoranda

Liver Abscess after Perforation of Liver by a Fish Bone

Pyogenic abscess of the liver is a relatively rare condition. In the series of cases reported by Ochsner *et al.* (1938) liver abscesses accounted for only 0.0077% of hospital admissions. Such abscesses, either solitary or multiple, may arise by spread of infection along the tributaries of the portal vein, by direct extension from infection of adjacent viscera, by secondary infection of an amoebic abscess, or as the result of trauma (Rolleston and McNee, 1929; Weiss, 1935; Ochsner *et al.*, 1938; Bockus, 1946; Sherlock, 1958).

Trauma is among the rarer causes of liver abscess, being responsible for only 2.9% of the 830 cases reviewed by Ochsner and his colleagues. The liver may be damaged either by penetrating wounds or by subcutaneous injury. In very rare instances liver abscesses have been caused by the penetration of foreign bodies from the gastro-intestinal tract. Fish bones seem to be the most common perforating objects, Ochsner *et al.* referring briefly to three such cases; but single cases of liver abscess due to a perforating pin or needle have been described (Wilhelm, 1926). Rolleston and McNee mention that cases due to a piece of glass and a fragment of straw respectively have also been recorded. In Morris's (1940) freak case the patient swallowed a toothpick while sleeping after a meal.

As these accidents are exceptionally rare the present case, in which liver abscess and fulminating septicaemia occurred after perforation of the liver by a fish bone, is deemed worthy of record.

CASE REPORT

A 43-year-old maintenance engineer had had radiotherapy for ankylosing spondylitis between 1950 and 1953. This disease had been arrested, but the patient subsequently contracted pulmonary tuberculosis, which was successfully treated by chemotherapy in 1956. He had been fit and working from 1957 until May 2, 1960, when he complained of nausea and vomited blood-streaked material several times during the day. Although the vomiting lessened, his condition rapidly deteriorated. Initially there was high fever, the temperature reaching 104° F. (40° C.), but this soon subsided and he was admitted to Colindale Hospital on

Photograph showing gross lesions in liver.

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FIG. 1

FIG. 2

FIG. 3

FIG. 1.—*Entamoeba histolytica* in ascitic fluid.

FIG. 2.—First needle biopsy of liver. *Entamoebae histolyticae* (arrowed) among liver cells with vacuoles in cytoplasm; slight inflammatory infiltration.

FIG. 3.—*Entamoeba histolytica* in liver abscess produced experimentally in guinea-pig.

FIG. 4.—Surgical liver biopsy. Amoebic granuloma of liver. Two *Entamoebae histolyticae* (arrowed) with vacuoles in cytoplasm.

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FIG. 1.—Necropsy specimen from Case 1. Disease process has destroyed floor of sella turcica and invaded hypophysis.

FIG. 4

FIG. 2.—Case 2. Radiograph showing destruction of left orbital roof.

FIG. 3.—Case 3. Tomogram showing bony defect in left pyramid.