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Pediatric Radiology

Extensive Sclerosis of the Base of the Skull Due to Primary Nasal Tuberculosis

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Abstract. An 8 year old black male is presented as a primary nasal tuberculous granuloma whose roentgenograms of the skull revealed extensive sclerosis of the periorbital region involving the frontal, sphenoid, and petrous bones. The tuberculous meningitis and the osseous sclerosis at the base of the skull were cured with anti-tuberculous therapy.

Key words: Tuberculous mastoiditis – Tuberculous osteomyelitis of skull – Meningitis – Nasal tuberculosis (primary infection) – Cerebral tuberculoma

In western countries, involvement of the cranial vault is rare in tuberculosis. A nasal lesion was the portal of entry and source of primary infeection. This report illustrates the latter with subsequent healing and describes the late complications of meningitis from a tuberculoma.

Strauss [8] collected 184 patients with tuberculosis of the flat bones of the skull from the world literature, half under 10 years of age, and three-fourths under 20. Most of these were associated with lymphohematogenous dissemination; rarely was a primary focus in the nasopharynx implicated. Similar observations are reported from African countries [2] where tuberculosis is still a problem. In the USA there is still tuberculous mastoiditis and otitis media [5].

Case Report

A 6 year old black boy had chronic rhinitis, left otitis media and partial left ear deafness. Discovery of a nasal mass led to biopsy of the nasal tissue and to a spinal tap. The microscopy of the nasal tissue contained epitheloid tubercles and giant cells and AFB were cultured from the tissue. The cerebrospinal fluid showed transient increased protein (124 mgs. %) but was otherwise normal. Radiographs of the skull and lungs were initially negative and the EEG was normal. The tuberculin test was positive. Streptomycin for 3 weeks (shortened because of patient's leaving the hospital against advice) and INH with PAS were given, the latter dropped by the patient. Thus, presumably, INH alone was continued for 2 years. He remained well except for left ear deafness.

Two years after this primary infection, he was admitted to Bellevue Hospital because of irritability, lethargy, restlessness and fever with a diagnosis of tuberculous meningitis. Severe left ear deafness was reconfirmed. Neurologic examination revealed nuchal rigidity, bilateral Kernig, negative Babinski, increased deep tendon reflexes, normal eye movements and eye-grounds. The remainder of the physical examination was negative. The cytology and chemistry of the spinal fluid was consistent with the diagnosis of tuberculous meningitis, although cultures for AFB were negative.

Radiographs of the chest were negative. The skull now showed marked sclerosis of the base, including the sphenoids, orbital plates, and frontal bones (Fig. 1a and b).

Following therapy with SM and INH, the neurological signs became localized. A common carotid angiogram suggested a mass extending from the anterior fossa to the proximal portion of the middle fossa. The electroencephalogram, normal 2 years previously, now showed diffuse cerebral dysfunction with shifting lesions of the left temporal and right parasaggital areas.

Following treatment with intramuscular SM, INH and PAS, the patient's condition improved. Fourteen months later, the skull radiographs were normal (Fig. 2a and b). Increased behaviour problems required withdrawal from school and prolonged psychiatric care. There was no evidence of active tuberculosis. Left ear deafness persisted when last seen at age 16.

Discussion

The unusual aspects of this patient are primary infection in the nostrils from which AFB were cultured and radiographic evidence of tuberculous infection of the skull, normal at the onset, and associated with tuberculous meningitis 2 years later. Recovery from

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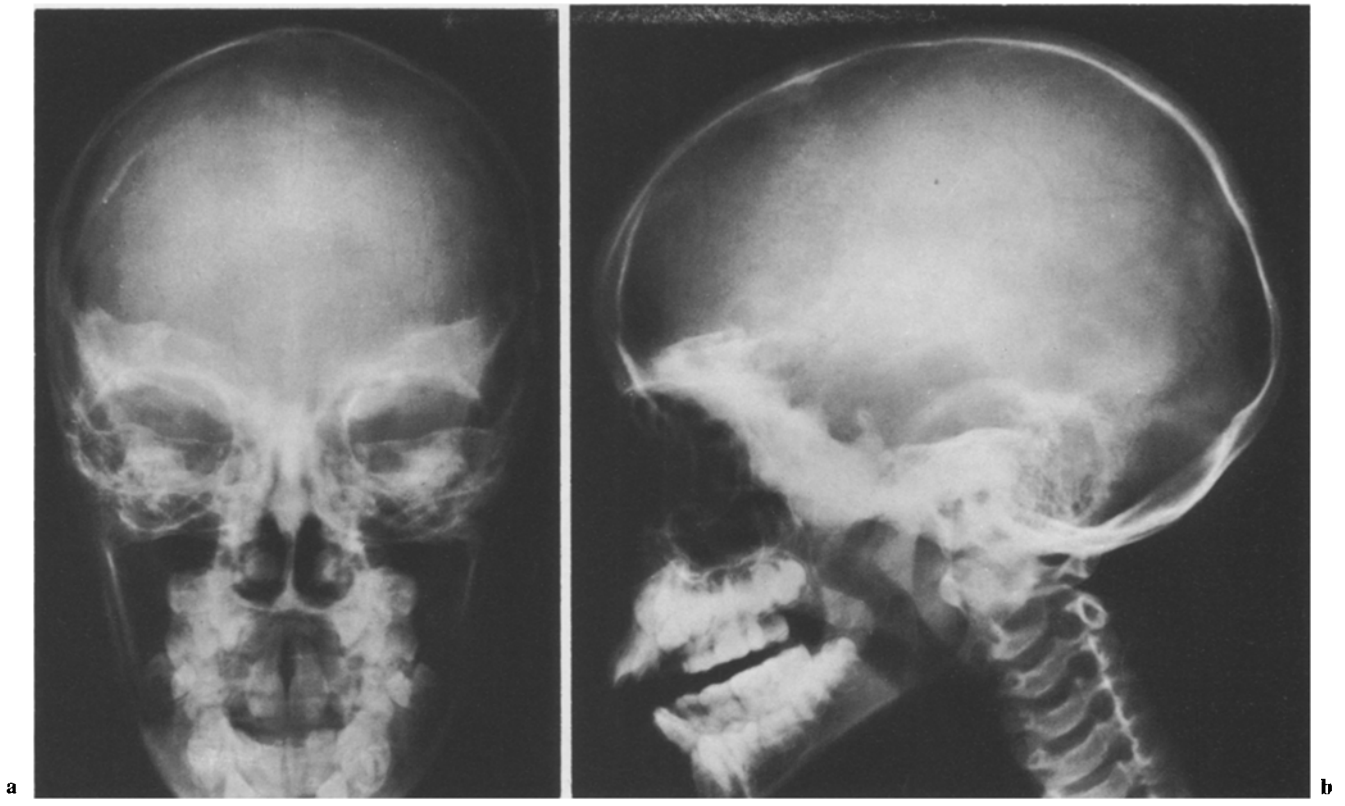

Fig. 1. a PA roentgenogram of the skull demonstrates sclerosis of the inferior aspect of the frontal bone and sphenoid. **b** Lateral roentgenogram of the skull demonstrates marked sclerosis of the base which extends from the anterior floor to the clivus

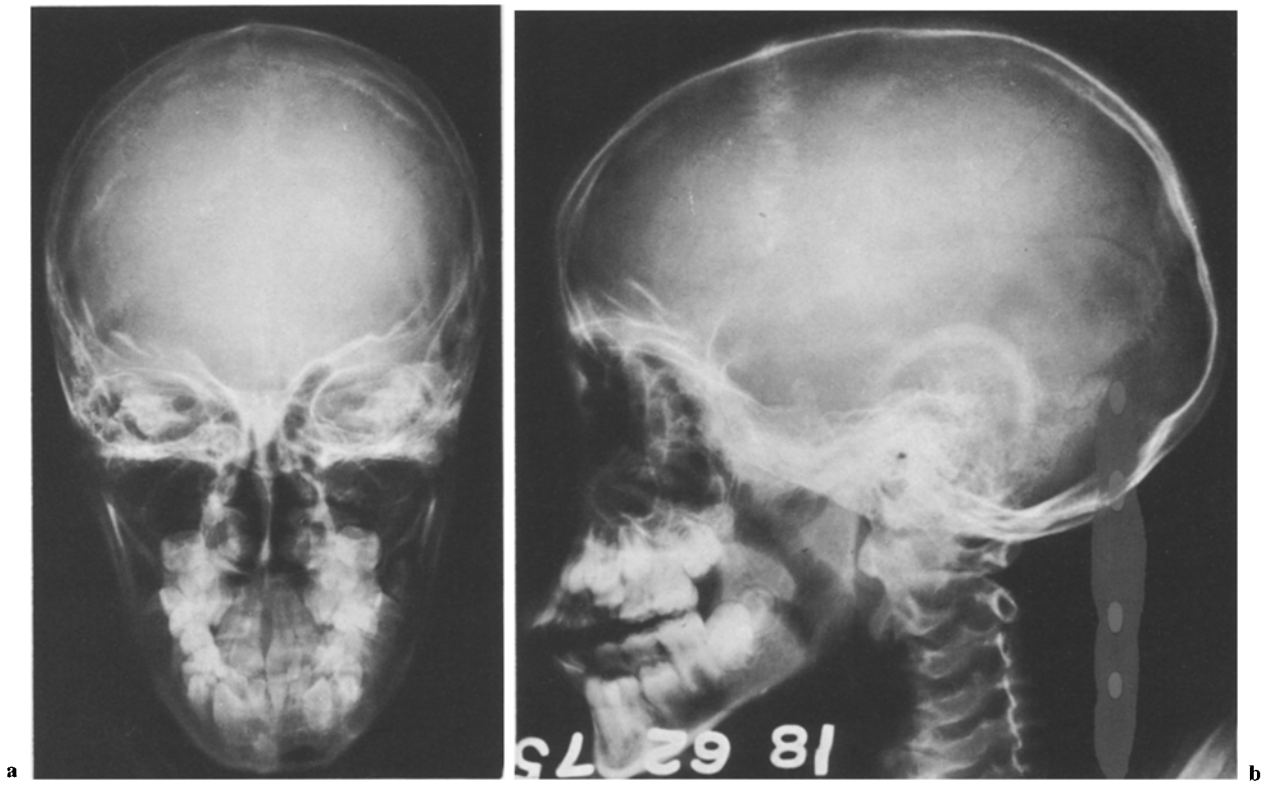

Fig. 2. a PA roentgenogram taken 14 months later shows marked clearing of the sclerosis demonstrated in Figure 1 a. **b** Lateral roentgenogram of the skull 14 months later showing marked clearing of the sclerosis of the cranial floor demonstrated in Figure 1 b

the infection was noted but deafness and psychopathic behaviour remained.

Tuberculosis of the skull was described first by Reid, a German surgeon in 1842 and reported by others [7, 9]. Radiographic features were described [1, 4, 9] as follows: 1. The circumscribed or perforating type (Volkmann) with solitary or multiple round, punched-out defects through the entire thickness of the skull surrounded by a narrow zone of rarifying osteitis [2, 4] and 2. The diffuse infiltration type (Koenig) progressively invading the diploe and the inner table and spreading over the vault producing serpiginous and geographic bone destruction.

Multiple bone tuberculosis in 3 of 6 African young children [1] also showed sclerosis of frontal and zygomatic bones. All had hematogenous tuberculous infection, and other bone lesions in contrast to our patient in which disease was limited to infection of the skull.

Tuberculosis of the skull may result from direct extension from the orbit, nasal cavity and the mastoids. Primary tuberculous infection of otomastoid cells has been reported [6]. Other pathways for infection [3] include direct extension to the temporal bone from intracranial structure or by direct extension from a nasopharyngeal lesion via the submucosa of the Eustachian tube. In our patient, the latter is the probable route of infection of the first mastoiditis from the primary nasal lesion. Extension of infection from an intracranial lesion resulted in the radiographic bone pathology 2 years later. The activation of the previously developed tuberculoma at that time led to the presenting meningitis. After specific therapy, there was recovery from disease and clearing of the bone lesions. In our patient, there was inadequate therapy following the primary infection because of poor compliance throughout his therapy.

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