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Letters

Epignathic Teratoma Associated with Craniopharyngeal Canal

Recently we reported an unusual case of epignathic teratoma in a male neonate [1]. At that time, we did not recognize the craniopharyngeal canal (CPC) that was seen later. The infant had a mass of 8–9 cm arising from the hard palate, no soft palate, and a hypoplastic mandible. Fluid dripping from the lesion had a glucose level similar to that of CSF. At surgery, the tumor was followed down via its broad stalk to the roof of the mouth, where dissection to the level of the sphenoid bone failed to reveal any cerebral communication. Microscopically, the wall of the mass was composed of a mixture of skin, respiratory epithelium, tooth germ, choroid plexus, salivary gland, and abundant neural tissue, establishing the diagnosis of teratoma.

Five years later the child had CT again at our institution as a part of a routine follow-up examination. The scans (Fig. 1) showed a bony defect with smooth sclerotic margins and internal bony septations, extending from the sella turcica inferiorly through the sphenoid bone into the region of the palate where the teratoma previously had been resected. The bony defect was widest at its superior margin; only a small opening was seen on the more inferior scans. On physical examination, no evidence of a defect within the roof of the mouth could be found.

We think that this bony defect is a form of CPC. The embryogenesis of this canal has been debated in the literature [2–4]. One theory proposes that this structure is the remnant of the Rathke pouch. The other theory places its origin at a later stage in fetal life, after the seventh embryonic week, when the passageway formerly occupied by the Rathke pouch has been filled in by cartilage. Proponents of the latter theory state that in the process of ossification of this cartilage, periosteal vessels grow through the future sphenoid bone and that in 0.4% of human neonates these former vascular channels persist as CPCs [2].

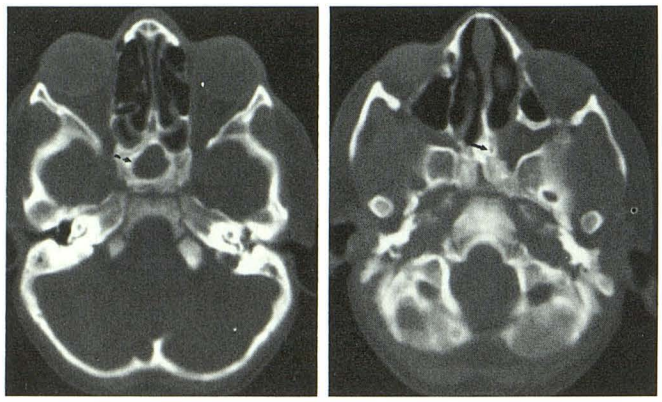

Fig. 1.—*A* and *B*, Serial axial CT scans show smoothly marginated bony canal (*arrows*) extending through basisphenoid. More inferior slice (*B*) shows only a small opening.

Currarino et al. [3] described a form of large CPC similar to that seen in our patient. They classified large CPCs into those associated with nasopharyngeal masses and those unassociated with such masses. The nasopharyngeal masses mentioned in their series were either parts of the third ventricle and pituitary gland or a variety of meningoencephalocele. In addition, they noted a high prevalence of craniofacial anomalies of the type seen in our case.

Review of the literature revealed only one other report of a facial teratoma associated with CPC [4]. In that case, a strand of meninges was found extending into a patent CPC, allowing the subarachnoid space to communicate with the facial mass. No extension of cortex into the canal was evident, but histologic examination showed remnants of pituitary tissue scattered along the canal.

Although our patient did not die, no cerebral communication was identified at surgery or on follow-up examination 5 years later. It is possible that such a communication was overlooked at the initial surgery, as CSF-like fluid was found dripping from the teratoma, and that palatoplasty covered over the inferior part of the bony defect. It is also possible that the CPC was not patent and that the CSF-like fluid came from the cystic mass itself.

In view of the complex embryogenesis in this region, it is not surprising that all of the elements found in the cyst wall were derived from tissues originating near the palate. Whether teratoma should be included in the list of nasopharyngeal masses associated with CPC awaits confirmation.

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