LETTER TO THE EDITOR

Comments on ‘‘Endoscopic Transpterygoid Approach and Skull Base Repair After Sphenoid Meningoencephalocele Resection’’

Comentario sobre ‘‘Abordaje endoscópico transpterygoideo y reparación de base de cráneo tras resección de meningoencefalocele esfenoidal’’

Dear Editor,

A recent article by Martinez Arias and colleagues, ‘‘Endoscopic transpterygoid approach and skull base repair after sphenoid meningoencephalocele resection’’, assessed the effectiveness of the transpterygoid approach in the treatment of meningoencephaloceles of the lateral recess of the sphenoid (LRS) sinus.1 Their conclusion that the transpterygoid approach is an appropriate intervention for cerebrospinal fluid (CSF) rhinorrhea is justified based on the data provided, however their association of CSF leaks in the LRS with a congenital persistence of Sternberg’s canal is an inaccurate assertion that we would like to see removed from the literature.

The presence of a lateral craniofaryngeal canal as a result of failure of fusion in the alisphenoid, basiphenoid, and preshenoid ossification centers was first postulated in 1888 by Maximilian Sternberg. As first described the canal runs medial to the superior orbital fissure, so encephaloceles and CSF leaks lateral to the maxillary nerve (cranial nerve V2) cannot be ascribed to a patent Sternberg’s canal.

In a recent study of consecutive computed tomography (CT) scans, only one defect/canal in 1000 sphenoid bones was identified that satisfied the original description by Sternberg.2 Such a small incidence of radiographic evidence consistent with Sternberg’s canal in the general population leads to the assertion that the canal is very rare, small, and not a significant cause of spontaneous LRS CSF leaks and encephaloceles.

Another study established that among patients presenting with LRS CSF leaks/encephaloceles, radiographic signs as well as CSF pressure measurements indicate intracranial hypertension is the underlying etiology.3 In the study 93% of patients had arachnoid pits, a finding that is normally only found in 25% of the population. This suggests the etiology of LRS CSF leaks involves lateral recess pneumatization, attenuated sphenoid sinus recess roof and skull base, and the development of arachnoid pits from intracranial hypertension. We would also argue that since your success rate is really only 80% during the follow up period, your relapse was secondary to undiagnosed intracranial hypertension. Identification of elevated pressure with lumbar pressure measurements would have provided you with useful data. Long-term acetazolamide should have been considered in these patients.

We encourage the authors of the manuscript to read the original descriptions and treatise regarding Sternberg’s canal. The mention of a ‘‘congenital’’ cause of LRS defects attributable to this canal should be eradicated from the literature.

References


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