Sternberg’s Canal and the Controversies Surrounding It

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Although Maxmillan Sternberg described this anatomical entity (the lateral craniopharyngeal canal or the Sternberg’s canal) as far back as in 1888, it has only recently stirred much controversy in world medical literature.

Materials and methods: A review of contemporary world literature on this subject has been made and applied to the authors’ personal surgical experience of managing 21 spontaneous cerebrospinal fluid (CSF) leaks over a period of 6 years, with special focus on lateral sphenoid CSF leaks, in an attempt to understand their philosophy and clinical course.

Results: Due to the rarity of such spontaneous CSF leaks (in all the published world series as well as the authors’ personal experience), it is difficult to comment on the exact site of congenital ‘weakness’, i.e. Sternberg’s canal. Similar body profiles of all the patients in most series further complicates the scenario.

Conclusion: While one school of thought based on clinical and cadaveric studies hypothesizes, the Sternberg’s canal to be present lateral to the foramen rotundum, the other based entirely on radiological analysis of 1000 CT scans of the parasanal sinuses proposes it to be medial to the foramen rotundum. Most patients with lateral sphenoid sinus CSF leaks (in world literature as well as the authors’ personal experience), are obese middle-aged women irrespective of whether the leak arises medial or lateral to the foramen rotundum. Thus, two strong and valid theories as regards the origin and existence (in adult life) of the Sternberg’s canal are present and a multicentric, combined clinicoradiological, meta-analytical approach may probably serve as a solution to this controversy.

Keywords: Cerebrospinal fluid, Sphenoid sinus, Lateral recess, Sternberg’s canal, Benign intracranial hypertension, Lateral craniopharyngeal canal.

INTRODUCTION

Spontaneous cerebrospinal fluid rhinorrhea is an increasingly recognized entity today with numerous series of the same being published in world literature. The term ‘spontaneous’ means arising from natural impulse without external stimulation or having a self-contained cause or origin, or arising from or entirely determined by the internal operative or directive forces of the organism (Shorter Oxford English Dictionary, 1962). These spontaneous CSF leaks are usually seen in females, commonly in the 4th to 5th decades of life, and are induced by coughing, sneezing or other ‘trivial’ causes of raised intracranial pressure. The common etiologies could be old ‘forgotten’ injuries, altered fluid dynamics and anatomical variations. One such entity thought to be strongly associated with the latter cause is the Sternberg’s canal.

In 1888, Maxmillan Sternberg described the ‘lateral craniopharyngeal canal’ as a bony congenital defect in the lateral wall of the sphenoid sinus. This resulted as a failure of fusion of the ali-, basi-, and presphenoid ossification centers in the lateral sphenoid sinus. In his original description, he described the canal to extend from the junction of the body of the sphenoid bone and the posterior root of the lesser sphenoid wing, just medial to the superior orbital fissure and inferiorly to connect with either the pharynx at the processus vaginalis, or when sufficient pneumatization has occurred, into the lateral wall of the sphenoid sinus. Sternberg estimated that the lateral craniopharyngeal canal/Sternberg’s canal was an absolute constant anatomical entity on the skulls of 3- or 4-year-old children, but only seen in 4% of adult skulls.

MATERIALS AND METHODS

In our series of 21 spontaneous CSF leaks over a period of 6 years (2006-2012), 11 were from the sphenoid of which three were from the lateral recess, whereas eight were from other areas (4 from the lateral wall, 3 from the roof and 1 from the posterior wall) (Table 1). Except two of our patients, the remaining were females, ages ranging between 38 to 56. Eighteen of these patients (all females) had a body mass index more than 27. All patients were treated by an endoscopic endonasal approach, and followed-up at 2 and 6 weeks, 3 and 6 months and 1 year following surgery. Two of these patients had a recurrent spontaneous leak (one at the same site, while the other at a site different from that of the primary repair).
DISCUSSION

Although various series in the world literature have described CSF leaks from the Sternberg’s canal, the latter two series have provided a detailed insight and proposed hypothesis for the development and existence of this canal.

The study published by Tomazic and Stammberger in 2009 states that developmentally, the sphenoid bone has two cartilaginous precursors, namely the presphenoid and postsphenoid. The former comprises the sphenoid body, lesser wings and tuberculum sellae, whilst the latter comprises the greater wings, dorsum sellae and pterygoid plates. Only certain parts of the medial portion of the pterygoid process are formed by membranous ossification. The ossification centers appear around the third month of fetal life and, at the time of birth, all the parts fuse, except for a weak cartilaginous union between the greater wings, presphenoid and basisphenoid, corresponding to the future lateral wall of the sphenoid sinus. During the neonatal period, their fusion starts anteriorly. Incomplete fusion of the posterior part leads to a bony gap, called as the lateral craniofaryngeal canal, which is located in the posterior part of the lateral sphenoid sinus wall, inferior and lateral to the maxillary nerve. After resorption of cartilage, the Sternberg’s canal is closed by connective tissue, thus being a potential point of weakness at the skull base. As pneumatization of the sphenoid sinus reaches into the greater wings of the sphenoid forming the lateral recess, a connection between the sphenoid sinus and the Sternberg’s canal may occur.

In controversy with the above-mentioned findings and in the same year as Tomazic and Stammberger, Baranano et al published their description of the Sternberg’s canal which was different from that seen above. In their interpretation and, we quote, ‘by definition and embryologic development, the canal must be present medial to the superior orbital fissure and, thus, medial to the foramen rotundum and V2’. They also categorically stated that ‘the location of Sternberg’s canal in anatomic studies is inconsistent with the majority of lateral sphenoid sinus CSF leaks because they are nearly all located lateral to the second branch of the trigeminal nerve. In addition, an encephalocele origination through this theoretical canal must not only transverse the cavernous sinus, but, also penetrate two layers of duramater before exiting the skull base’. In support of their theory, their radiological findings revealed that in patients with a lateral recess, pits were detected in less than 1/4th (25%) of the population, whereas all patients (100%) with lateral recess CSF leaks had identifiable pits. This further supports the theory that erosive arachnoid pits rather than a congenital dehiscence lead to the development of encephaloceles and CSF leaks in this cohort. Also, in a case report published by Schick et al (2000), of a case of Sternberg’s canal as a cause of CSF leak, the image shows the meningoencephalocele to originate in the lateral wall of the sinus and occupying the lateral recess, but not arising from the latter.

It is worthy to note that while the former study was based predominantly on clinical findings, the latter was based entirely on radiological review of a thousand CT scans of the paranasal sinuses. The former authors (personal communication) have also attempted to validate their hypothesis with cadaveric studies. Since, the methodology in both the above-mentioned studies is different, it is difficult to compare their results. However, such stark differences most certainly warrant a combined, multicentric, clinicoradiological approach to studying this anatomical entity.

What cannot be argued upon is that all sphenoid sinus lateral recess leaks result from a combination of lateral recess pneumatization and an attenuated skull base due to the development of arachnoid pits from underlying intracranial hypertension. An increasingly common patient profile is the association of obesity in middle-aged women. This can probably be explained by the extraordinary production of estrone from androstenedione in the excessive adipose tissue due to their higher body-fat content. This could lead to a relative hypoadrenalism thus, altering the drainage channels and vacuolar transport across the arachnoid villi and in effect leading to an increased resistance to CSF outflow.

In our personal experience, we have had leaks from the lateral sphenoid recess (three in number, see Figs 1 and 2) and from the lateral wall of the sphenoid sinus, medial to the foramen rotundum (four in number, see Figs 3 and 4). The management of the recess leaks is far more challenging, as a transthyroid-transpterygoid approach was employed to access the lateral extent of the meningocele, by removing the posteromedial wall of the maxillary sinus (Fig. 5), and then drilling away the pterygoid root (Fig. 6), and traversing the superomedial part of the pterygopalatine fossa, so as to remove the anterior wall of the sphenoid sinus lateral recess (Fig. 7). Branches of the sphenopalatine artery were routinely encountered in this approach and tackled with diathermy. The mucosa of the entire lateral recess had to be denuded so as to either obliterate the recess (two patients) or for allowing the fascial graft to adhere to the edges of

<table>
<thead>
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<th>Site</th>
<th>Posterior table of frontal sinus</th>
<th>Craniiform area</th>
<th>Ethmoid fovea</th>
<th>Sphenoid roof</th>
<th>Sphenoid posterior wall</th>
<th>Sphenoid lateral wall</th>
<th>Sphenoid lateral recess</th>
</tr>
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<tbody>
<tr>
<td>No. of patients</td>
<td>0</td>
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<td>3</td>
<td>3</td>
<td>1</td>
<td>4</td>
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Table 1: Sites for spontaneous CSF leaks (n = 21, 2006-2012)
Fig. 1: Coronal CT scan of the paranasal sinuses showing a skull base defect in the roof of the lateral recess of the right sphenoid sinus, in a patient with a CSF leak

Fig. 2: T2-weighted MRI scan of the same patient (Fig. 1) showing the meningoencephalocele exiting the skull base from the roof of the lateral recess of the right sphenoid sinus

Fig. 3: Coronal CT scan of the paranasal sinuses of a patient showing a defect in the lateral wall of the right sphenoid sinus in a patient with a CSF leak

Fig. 4: T2-weighted MRI scan of the same patient (Fig. 3) showing the CSF in the right sphenoid sinus

Fig. 5: Intraoperative endoscopic view showing a part of the posterior right maxillary wall removed (black arrow)

Fig. 6: Intraoperative endoscopic view of the same patient (Fig. 5) showing removal of the root of the pterygoid process (black arrow)
the bony defect (all three patients). The sphenoid sinus lateral wall defects were easier to tackle, with a fat ‘bath plug’ technique (three patients) and obliteration of the sphenoid sinus (one patient).

All of our patients had similar body profiles, and all showed radiologic evidence of arachnoid pits on the skull base.

One of the patients with a sphenoid CSF leak also had a pneumatized lateral recess; however, the leak did not originate from the recess (Fig. 8), but from the roof (Fig. 9). Whether this indicates that despite the pneumatized recess, the ‘weakest’ part of the skull base was not lateral to the foramen rotundum (and possibly a congenital weakness), or whether this was an incidental finding, would be difficult to comment, since the sample size is too small.

**SUMMARY**

The principles of management of sphenoid CSF leaks are consensual in most series of the world literature, including the authors. The challenges posed in managing lateral recess leaks are also universally accepted, as are their management principles. The correct interpretation of Sternberg’s canal and its adult existence leaves much to argument.

Probably, a multicentric meta-analytical evaluation of clinical and radiological data would help to shed further light on this entity. Nevertheless, as the principles of the surgical management of lateral sphenoid sinus CSF leaks are well defined (for CSF leaks arising both medial and lateral to the foramen rotundum), this controversy would probably remain one of nomenclatures, rather than of treatment philosophy.

**REFERENCES**