CASE REPORT

Not the typical Tornwaldt's cyst this time? A nasopharyngeal cyst associated with canalis basilaris medianus

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ABSTRACT. We report a patient with a cystic structure in the nasopharynx mimicking a Tornwaldt's cyst, which was felt to represent a different entity owing to the lack of the distinct features of a typical Tornwaldt's cyst. It was associated with a bony cleft in the basiocciput that was considered to be a canalis basilaris medianus (CBM), thought to represent an embryological vestige of the cephalic end of the notochord along its course within the basiocciput.

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Persistent canalis basilaris medianus (CBM) is a rare and typically asymptomatic congenital skull base defect believed to arise from notochordal remnants [1, 2]. Clival defects from this vestige can be associated with several anomalies. MRI may detect accompanying cysts and may help differentiate between possible aetiologies. We present an adult with a midline clivus defect that is most likely a CBM, associated with a midline nasopharyngeal cystic lesion.

Case report

A 45-year-old man underwent MRI as part of a work-up for Parkinson's disease. A tiny defect was noted in the midline basiocciput, composed of both a bony intracanal cleft and an associated midline T_2 hyperintense cystic structure measuring $7 \times 8 \times 10$ mm that protruded anteriorly into the nasopharynx. Posteriorly, the cleft appeared to end blindly at the dural surface. The tract and cyst were hyperintense on T_2 weighted images (Figure 1a–d), and fluid-attenuated inversion recovery (FLAIR) and pre-contrast T_1 weighted images (T_1 WI) without a definite contrast enhancement on post-contrast T_1 WI (not shown).

Sagittal multiplanar reformats of <1 mm axial thin section CT were obtained to further evaluate for a bony tract of a cephalocele, which confirmed a thin bony defect (4 mm length, <1 mm diameter). The cleft was extending from the pharyngeal surface of the basiocciput into the posterior third of the clivus (Figure 1e,f). Owing to its course, the tract was thought to be a notochordal remnant from a residual CBM, with the differential for the cystic structure being a small cephalocele protruding through the nasopharyngeal end (the foveola pharyngica)

that communicated with the subarachnoid space; a Tornwaldt's cyst (not communicating with the subarachnoid space); or a cystic-appearing remnant of notochordal tissue (ecchordosis physaliphora).

Discussion

The CBM was first described by Gruber in 1880 as a rare anatomical variant of the basiocciput occurring in approximately 2–3% of adults [1, 2]. The CBM is a well-defined corticated tubular structure, usually ≥ 2 mm in diameter, originating on the intracranial surface of the basiocciput in close proximity to the anterior rim of the foramen magnum. It is thought to represent an embryological remnant demarcating the cephalic end of the notochord (a midline collection of cells that aids embryonic long-axis and neural plate development) and corresponds to the course of the notochordal canal in the caudal basiocciput [1, 3]. Several variants of CBM have been described, namely six subtypes: a complete CBM that includes superior, inferior and bifurcatus forms; and an incomplete CBM that includes a long channel running through the basiocciput and posterior sphenoid, a superior recess within the basiocciput and an inferior "foveola pharyngica" recess [2, 4]. A complete CBM traverses entirely through the basioccipital bone, while an incomplete CBM can extend partially through either the cranial or the pharyngeal portions of the basiocciput, but does not extend all the way through to the other surface. Detailed illustrations of the six subtypes have been previously described in the literature and are beyond the scope of this report [2, 4].

Although the exact mechanisms involved are not explicitly understood, a few proposed theories should be mentioned regarding the embryogenesis of this defect. One, less likely, theory has been that the channels and foramina along the surface of the basiocciput are of vascular origin (emissary canals), while the other, more probable, theory attributes the presence of channels and

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Figure 1. (a–d) Axial fat saturated T_2 weighted images. (a) Image through the cystic lesion (arrows) in the posterior midline nasopharynx. (b) Slice more superiorly demonstrating the defect in the clivus. Arrows denote the partially hyperintense defect in the midline. (c) Inverted T_2 weighted image through the same level as (b) with better depiction of the canal within the bone. (d) More superior slice demonstrating the junction of the canal with the dural surface. (e and f) Thin slice CT images. (e) Axial and (f) sagittal images thin images from a dedicated skull base CT depicting the bony canal within the clivus. Arrow in (e) depict the broader anterior part of the canal which could be a so-called “foveola pharyngica”. Arrowheads in (e) point to the sclerotic posterior aspect of the defect. Arrows in (f) denote the broader anterior aspect; cranial communication could not be definitively demonstrated.

pits to their being postnatal vestiges of the cephalic end of the notochordal canal (canalis chordae) [2, 4]. The event causing this aberrant development occurs early in the fifth gestational week of embryonic life, when the notochord follows a “sigmoid-like” caudal-cranial curve that crosses the clivus approximately three times, initially perforating the occipital plate ventrally near the CBM, and then, when directly in contact with the endoderm of the pharynx, follows a rostral trajectory that penetrates the sphenoid plate near the dorsum sellae (Figure 2) [5, 6].

At the time when the notochord is in contact with the endoderm of the primitive pharynx, a focal adhesion may occur, causing the notochord to carry along a segment of the pharyngeal mucosa with it during its

rostral ascension back to the developing skull base. This creates a potential space known as the “pharyngeal bursa” (*i.e.* a Tornwaldt’s cyst), which is a midline bursa lined by pharyngeal mucosa [7]. Embryologically, a Tornwaldt’s cyst is a persistent communication between the roof of the nasopharynx and the notochord [8].

In our patient, the appropriate term for the cystic structure protruding into the nasopharynx may be a matter of debate, depending on nomenclature. Although it carries some of the features typical for a Tornwaldt’s cyst [7, 9, 10], a Tornwaldt’s cyst lacks osseous involvement, and corresponds to a superior level in the clivus, while in our patient it lies at the mid-clival level and is associated with a bony midline defect in the basiocciput

Figure 2. A different, normal patient with a sagittal view of the skull base showing a superimposed diagram of the craniocervical component of the expected embryonic notochordal trajectory in the adult (coloured line). The cephalic end of the notochord may be embedded in the bony sphenoid, while the segment in the basioccipt level (arrows) may lie free in the nasopharyngeal wall.

with an anterior opening at the level of the pharyngeal fossa. Therefore, although it has a very similar appearance, we propose that this lesion is not a Tornwaldt's cyst, but rather a different type of cystic lesion mimicking a Tornwaldt's cyst (Figure 1). Regarding the bony defect, it is most consistent with the incomplete (or nearly complete) CBM subtypes, one of which can have a foveola pharyngica [10, 11].

In the differential diagnosis of the cystic lesion, a cephalocele might also be considered. However, the appearance is atypical since the larger portion of the tract was on the nasopharyngeal side. The presence of intracranial extension was difficult to assess, and it could not be determined with certainty whether the cleft communicated with the subarachnoid space; a CT myelogram was suggested, but not performed. A final consideration is ecchordosis physaliphora, which is unlikely since that entity is usually intradural within the prepontine cistern, and is usually attached to the dorsal wall of the clivus via pedicles [12, 13]. The only way to confirm with certainty would be a biopsy, which was considered to be inadvisable by the staff neuroradiologists as it was felt clinically unnecessary and a skull base resection of such a site could be quite complicated.

Conclusion

We believe that the cystic lesion found in our patient could well be a Tornwaldt's cyst associated with an incomplete inferior variant of CBM (a foveola pharyngica) but also a totally different entity of indeterminate aetiology. To our knowledge, a CBM in association with a Tornwaldt's type cyst has not yet been reported in the literature. This case demonstrates the potential overlap of these entities, which appear to be inter-related based on the embryological development and regression of the notochordal axis. The overlap in embryogenesis makes the task of correctly classifying this cystic defect quite difficult.

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