CASE REPORT

A Case of Intranasal Meningocele

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INTRODUCTION

Intranasal meningoceles occur as a result of herniation of the meninges into the nasal cavity through a defect in the cribriform plate. These are commonly found in the pediatric population due to a congenital defect in the skull base. Intranasal meningoceles are very rare in adults, and tend to arise only when engendered by trauma. Here, we present the case of a patient with seemingly unprovoked intranasal meningocele.

CASE REPORT

Mr. M. is a 23-year-old labourer at a lignite factory who presented with left-sided nasal obstruction and watery discharge which he had been experiencing on-and-off for the past 5 years. He attributed the obstruction to a mass in his left nostril, and said he would often breathe through his mouth. Other than an episode of pyogenic meningitis 9 years prior to the present complaint, this patient had been relatively healthy. He had no history of epistaxis, head or nasal trauma, anosmia, headache or visual disturbances. Constitutional symptoms such as anorexia and weight loss were absent.

On rhinoscopic examination, the left nasal cavity was found to contain a pale polypoidal mass with prominent blood vessels. The mass appeared to arise from the roof of the nasal cavity with no other points of attachment, and almost completely obstructed the nasal cavity. It descended to below the level of the inferior turbinate. No active discharge was observed. In addition, there was septal deviation to the right.

At this point, the clinician would suspect the following as part of the differential diagnosis: antrochoanal polyp, inverted papilloma, angiofibroma, intranasal menigocele, nasal glioma and congenital tumours such as neuroblastoma (1). The clinical presentation strongly suggested the diagnosis of polyp for this nasal mass.

A CT scan of the paranasal sinuses showed that they were clear. A cystic mass attached to the roof of the nasal cavity and causing right septal deviation could be seen. No bone erosion was apparent, and no abnormalities at the intracranial or orbital levels were noted. Routine blood investigations turned out to be normal.

TREATMENT

Due to the possibility of this nasal mass being a meningocele, certain precautionary measures were taken as part of endoscopic surgery to treat this patient. Of particular importance was irrigation of the nose with a solution of clindamycin to decrease bacteria within the operative field so as to reduce the potential for intracranial seeding from nasal flora.

An aspirate of the nasal mass was taken and sent for analysis. Rather than grasping and avulsing the mass, the upper portion of the polyp was cut, leaving a small sleeve. Once the mass was surgically excised, the source of the fluid leak was closed in layers: fat, then temporalis muscle/fascia. The temporalis fascia was used to seal the defect and served as a scaffold for fibrous ingrowth. Through the measurement of glucose content of the mass aspirate, it was determined that the fluid was cerebrospinal fluid (CSF). Pathological analysis of the tissue was characteristic of the meninges. These two findings correlated with the diagnosis of intranasal meningocele.

Post-operative management of this patient consisted of antibiotics (amoxicillin/clavulinic acid), acetazolamide and bedrest in the head-up position. The nasal pack was removed after 5 days. Diagnostic

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endoscopy on the 7th post-operative day showed that the graft was in place. No CSF leak was observed, and the patient did not complain of rhinorrhea subsequent to surgery.

DISCUSSION

Intranasal meningocoeles occur secondary to meningeal herniation through a defect in the floor of the anterior cranial fossa. In addition, there is a disruption in the arachnoid and dura mater, leading to a CSF leak. In a patient with intranasal meningocele, this is manifested as CSF rhinorrhea (2).

A CSF pressure gradient that is greater than the tensile strength of the disrupted tissue is a contributing factor in meningeal herniation. This can occur in the context of intracranial hypertension and results in exertion of hydrostatic pressure at anatomically weakened sites within the skull base (3). When elevated intracranial pressure is present, the weakest anatomic points in the periphery of the central nervous system (optic nerve sheath, the cribriform plate, the sellar diaphragm, and any other bony dehiscences in the anterior or middle cranial fossae) can act as potential release valves for the high pressure (4).

There are various possible etiologies of CSF leaks: spontaneous, accidental trauma, surgical trauma, congenital, and tumours.

The most common etiology of CSF leaks is accidental trauma from closed head injury; such leaks occur in around 1% to 3% of all closed head injuries. In such instances, CSF leaks usually begin within 48 hours, and 95% of them manifest within 3 months of injury (5).
The detection of CSF fistulae is best made these days using Computed Tomographic Cisternography (CTC) (5). Such instrumentation is not readily available in hospitals of developing countries, thus the visualization and detection of herniation in this case was made using CT scanning.

Other methods used to localize CSF leakage include plain skull films, positron emission tomography, introduction of dyes by lumbar puncture, digital fluoroscopy and magnetic resonance imaging (5).

Intraoperatively, one can identify the site of a leak through the immunoelectrophoretic identification of CSF-specific marker proteins such as B2-transferrin. The sodium fluorescein test perioperatively enables precise localization of the defect and allows for confirmation of complete sealing of the leak (6).

Intranasal meningocele can be complicated by meningitis, as the intracranial contents are exposed to intranasal organisms such as S. pneumoniae and H. influenzae.

The patient's own episode of pyogenic meningitis 9 years ago was likely attributable to such a communication with intranasal contents. Intranasal meningoceles, as mentioned earlier, present most commonly in childhood as the result of a congenital weakness in the cribriform plate. It is probable that our patient had such congenital weakness to a lesser extent than most individuals. It is also possible that some minor trauma contributed to herniation through a pre-existing weak cribriform plate. The herniation through the cribriform plate defect was likely minimal when the patient suffered an episode of pyogenic meningitis, which might be why the patient did not feel nasal obstruction at the time.

Surgical repair of CSF leak using a frontal craniotomy approach was started by Dandy in 1926. This was followed by extracranial and transnasal approaches in the 1950s. Endoscopic technique, which was being used principally for sinus surgery, was first attempted in the repair of CSF leaks in 1981 (8, 9). This has evolved to the present-day endoscopic technique which offers greater benefits by way of visualization, no scar remnant, lower morbidity and no adverse effect on the sense of smell. The literature shows that the success rate with such an approach is 86 to 97% (10, 11).

Certain medical centers in the U.S. have begun to implement computer-assisted image-guided surgery techniques, which have ensured more accurate and precise closure of cribriform plate defects.

With respect to post-operative management, nasal packing is kept in place in order to compress the fascia against the recipient bed. Bedrest in the head-up position ensures that CSF pressure at the anterior skull base is decreased. Acetazolamide is used to decrease ICP, thus preventing herniation. It is a diuretic that diminishes CSF production by 48% (12).

In summary, patient presentation with nasal mass and rhinorrhea has its own set of differential diagnoses. Intranasal meningocele, although relatively rare, comes under this differential. CSF rhinorrhea is a diagnostic challenge; if not recognized, it can lead to devastating complications.

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