

Review Article

Sphenoid sinus mucocele: a review

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ABSTRACT

Sphenoid sinus mucocele (SSM) is a benign, encapsulated, expansile, locally destructive mass within the sphenoid sinus, filled with mucous and lined by epithelium. The exact etiology of the sphenoid sinus mucocele is still not known. The proximity of the SSM to the orbit and brain may cause morbidity and potential mortality if left without intervention. SSM has varied clinical manifestations. Headache and visual disturbances are common symptoms. Diagnostic nasal endoscopy and computed tomography (CT) scan and/or magnetic resonance imaging (MRI) are important pre-operative investigations. Diagnosis of SSM is done by computed tomography (CT) scan of the nose and paranasal sinus which shows a hypodense cystic lesion in the sphenoid sinus. MRI is helpful to detect the extension of the mucocele to orbit and cranium. Treatment of SSM is surgical which include endoscopic transnasal sphenoidotomy with sufficient removal of anterior and inferior walls of the sphenoid sinus for allowing adequate drainage and to avoid recurrence. The endoscopic approach is a safe and effective method of treatment for SSM with fewer recurrences than an open approach. Marsupialization of the mucocele via transnasal transsphenoidal approach is another option with a good outcome. The literature review for sphenoid sinus mucocele was performed from the database of Pub Med, Medline, Scopus, and Google scholar search with the term sphenoid sinus mucocele. Here, this review article is attempting to discuss the details of the sphenoid sinus mucocele including its history, epidemiology, etiopathology, clinical presentations, investigations, and current treatment.

Keywords: Sphenoid sinus, Sphenoid sinus mucocele, Headache, Visual disturbance, Endoscopic sinus surgery

INTRODUCTION

Mucocele of the paranasal sinus is defined as accumulation and retention of mucoid secretion inside the sinus resulting in distension, thinning, and erosion of one or multiple of its bony walls.¹ Primary mucocele usually arises from the retention cysts of the mucous glands of the sinus epithelium and secondary mucoceles arises by obstruction of the sinus ostium or due to cystic degeneration of the polyps.² The mucocele occurs due to blockage of the sinus ostium which may happen spontaneously or due to infection, chronic inflammation, trauma, iatrogenic injury, or neoplastic growth.³ Mucocele commonly affects the frontal sinus and

ethmoidal sinuses whereas the maxillary sinuses are less commonly affected, but sphenoid sinus is very rarely affected by the mucocele.³ The exact etiopathology of this SSM is still unclear but usually thought to be caused by blockage of the sphenoid sinus ostium. Other hypotheses include cystic dilatation of glandular structures and cystic development from the embryonic epithelial remnants.⁴ Although SSM is pathologically benign, it may affect vital structures such as dura, pituitary gland, cavernous sinus, optic nerve, internal carotid artery and cranial nerves (oculomotor, abducent and trigeminal nerves).⁵ To date, only a few cases have been reported. The under-reporting of the SSM may be due to its non-specific presenting symptoms with suboptimal physical examinations because of

inaccessibility of the sphenoid sinus. Imaging like CT scans or/and MRI of the nose and paranasal sinuses are helpful for early diagnosis.⁶ Endoscopic transnasal surgery is a useful technique for the treatment of the sphenoid sinus mucocele. Transnasal endoscopic sphenoidotomy along with drainage or marsupialization of the mucocele is safe and effective in the eradication of the disease.

METHODS OF LITERATURE SEARCH

The research articles related to the sphenoid sinus mucocele were searched through multiple approaches. Here, the author performed a literature review of SSM from the database of Pub Med, Medline, Scopus, and Google scholar search with the term sphenoid sinus mucocele. A search strategy using Preferred Reporting Items for Systemic Reviews and Meta-analysis guidelines was developed. Randomized controlled studies, observational studies, comparative studies, case series, and case reports were evaluated for eligibility. Review articles with no primary research data were also excluded. The abstracts of the published articles were identified by this search method and other articles were identified manually from the citations. This review article focuses only on SSM. All the articles were read and analyzed, with relevant data being extracted. This manuscript reviews the details of SSM with its history, epidemiology, etiopathology, clinical presentations, assessment, and management. This review article surely makes a baseline from where further prospective studies can be designed for the SSM which can help to prevent this morbid clinical entity.

HISTORY

Mucoceles of the paranasal sinus were first documented by Langenbeck in 1820 with the name of hydatids and Rollet coined the term mucocele in 1909.⁷ The first case of SSM was documented by Berg in 1889.⁸ Pelaz et al documented two patients of SSM presenting with optic neuritis in 2008.⁹ Gupta et al documented a case of retrobulbar optic neuritis in a patient with SSM.¹⁰ Selvakumar et al documented a case of bilateral optic neuropathy and unilateral sixth cranial nerve paralysis in a patient of SSM where the patient recovered after surgical excision of the mucocele.¹¹

EPIDEMIOLOGY

Mucocele can occur in any paranasal sinuses but the frontal sinus (60 to 89%) is most commonly involved followed by ethmoidal sinus (8 to 30%), maxillary sinus (<5%), and rarely found in the sphenoid sinus.¹² Only 1 to 2% of the paranasal sinus mucoceles are found in the sphenoid sinus.¹³ To date, only 140 cases of SSMs have been reported.¹⁴ SSM is frequently misdiagnosed and can lead to complications due to their vicinity with vital structures. Dealing with SSM, they represent approximately 17% of all inflammatory lesions affecting

the paranasal sinuses.¹⁵ Most common clinical presentation of SSM is headache (frontal or retro-orbital:70% of cases) and the second most common presentation is a visual disturbance (in 65% cases).¹⁶ Cranial neuropathies are seen in approximately 50% of cases of SSM.¹⁷

ETIOPATHOLOGY

The exact cause for the development of the mucocele in the sphenoid sinus is not known. The development of the mucoceles is thought to be caused by blockage of the sinus, but there are still more hypotheses about etiology such as cystic dilatation of glandular structures and cystic development from the embryonic residual tissues.¹⁸ There are certain risk factors associated with the development of the SSM including chronic low-grade inflammation/infection, allergy, previous sinus surgery, radiation therapy, trauma, previous history of sinus surgery, and tumors.¹⁹ The expansile growth of the sphenoid mucocele often follows the path of least resistance that is into the orbit. A study showed that high levels of prostaglandin E2 play a vital role in the osteolytic process in the mucoceles and explain its locally aggressive nature of the expansile mucocele.²⁰ The osteolytic bone resorption is also aggravated by the release of proinflammatory cytokines (IL-1 and TNF- α) into the wall of the mucoceles has been thought to be the inflammatory etiology of the mucocele.²¹ The clinical manifestations of the patients with SSM are directly related to the anatomy of the sphenoid sinus and its contiguous vascular and neurological structures. The contiguous structures of the sphenoid sinus include cranial nerves II to VI, the cavernous sinus, the carotid artery, the sphenopalatine artery, and the nerve of the pterygoid canal, dura, and pituitary gland.²² SSM may extend anteriorly into posterior ethmoidal air cells, cribriform plate, and orbital apex which result in proptosis and anosmia apart from visual disturbances.²³ The extension of SSM and its presentations often mimic other clinical entities of sinonasal pathology. The differential diagnosis of the mucoceles in the sphenoid sinus include encephaloceles, epidermoid cyst, meningioma, cholesterol granuloma, paraganglioma, chordoma, neurofibroma, and malignant neoplasms.²⁴

CLINICAL PRESENTATIONS

SSM may be found at any age group from early adulthood to 70 years and occurs in males and females with equal preponderance.²⁵ The clinical presentations of the SSM are directly related to the sphenoid sinus anatomy and its contiguous structures.²⁶ Patients of the sphenoid sinus mucocele may present with a myriad of symptoms due to the presence of adjacent important structures such as neurological, orbital, and vascular structures.²² Although the SSM is pathologically benign, it may affect vital structures such as the dura, pituitary gland, cavernous sinus, optic nerve, internal carotid artery, and cranial nerve such as oculomotor, trochlear,

trigeminal, and abducent nerves. The third cranial nerve is the commonest cranial nerve affected by SSM.²⁷ Sparing function of the pupil mimics a vasculopathic process in approximately 46.6% of patients with SSM.²⁸ The expansile growth of the mucocele in the sphenoid sinus cause involvement of the orbit.²⁹ The enlarged mucocele may result in lateral and inferior displacement of the orbit, diplopia, restricted ocular motility in upward gaze, sometimes with a characteristic eggshell cracking sensation because of the thinning of the overlying bone.³⁰ Visual disturbance is the second most common symptom in patients with SSM.

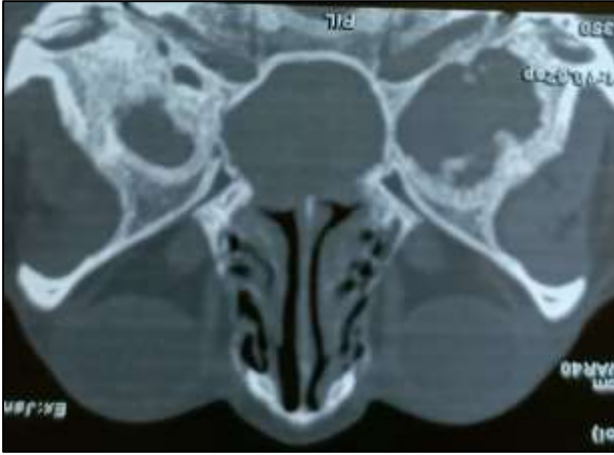


Figure 1: CT scan of nose and paranasal sinus (axial view) is showing sphenoid sinus mucocele.

The most common cause of visual disturbance in SSM is optic nerve involvement. It can result in decreased visual acuity and even blindness, which is usually irreversible.⁵ The visual disturbance may also occur because of III, IV, and VI cranial nerves involvement. The ocular presentations are usually drooping of the upper eyelid, diplopia, and restricted eye movements (external ophthalmoplegia).³¹ SSM usually does not manifest bitemporal hemianopia which is seen in other sphenoidal and sellar lesions such as pituitary macroadenoma.³¹ Sometimes, secondary infection of the mucocele may result in pyoceles which cause rapid expansion and reduced vision.³² Sometimes the patient of SSM are asymptomatic. The common clinical presentations of the patients with SSM may include visual loss, cranial nerve paralysis, and headache.³³ The complications of the SSM may include blindness, meningitis, cavernous sinus thrombosis, and compression of the internal carotid artery.⁵ SSM is an uncommon cystic lesion that can be mistaken by the clinician as pituitary pathology.³⁴ As headache is an important symptom of the SSM, it should be differentiated from other causes of headache which may be due to neurological causes like increased intracranial pressure and ophthalmology cause like acute angle-closure glaucoma.³⁵ The headache in SSM presumably occurs due to stretching of the dura covering of the planum sphenoidale and floor of the anterior cranial fossa. Pain due to SSM can be referred to any area

of the headache as the nerve supply of the sphenoid sinus arises from the ophthalmic division of the trigeminal nerve (posterior ethmoidal branch).³⁶ The cranial nerves involvement in SSM may lead to anosmia, visual disturbances, and ophthalmoplegia (often due to oculomotor palsy) and the cranial nerve involvement may occur by direct inflammatory (neuritis) or vascular process. Extension of the SSM may result in exophthalmos and conjunctival hyperemia, the latter presumably occur by compression of the sphenopalatine ganglion or the nerve to the pterygoid canal.³⁷ Some cases may manifest as pituitary hypofunction. Compression of the cavernous sinus by SSM may cause exophthalmos and periocular pain.³⁸ The clinical manifestations of SSM often mimic chronic sphenoid sinusitis, a fungal ball, benign neoplasms like inverted papilloma, and rarely malignant sinonasal tumors.¹

INVESTIGATIONS

Diagnostic nasal endoscopy and CT scan and/or MRI are important pre-operative investigations. Plain radiography and helical format CT scan are helpful to outline the bony erosion or massive bony destruction that may occur in case of expanding SSM.³⁹ CT scans of the nose and paranasal sinus show isolated opacification and expansion of both sphenoid sinuses with or without bony erosion in SSM (Figure 1).⁴⁰ The imaging picture may include elevation of the planum sphenoidale and tuberculum sellae, erosion of the pituitary fossa floor, petrous apex, orbital walls, optic canals, and floor of the anterior cranial fossa.⁴¹ CT scans usually show low-density cystic lesion, the wall does not enhance but may do if there is active inflammation (in case of pyoceles). Some clinicians suggested CT-guided biopsy for confirmation of SSM for confirmation of diagnosis before performing definitive surgery. SSM should be differentiated from other cystic lesions of the sphenoid sinus in this area such as Rathke cleft cyst, craniopharyngioma, cystic pituitary adenoma, epidermoid cyst, cystic optic nerve glioma, and arachnoid cyst.⁴² Cerebral angiography is helpful to assess the position of the internal carotid artery before surgery.⁴³ MRI may show high or low signal intensity on T1 and T2 weighted images, depending on whether the contents are inspissated or hydrated respectively.⁴⁴ MRI is needed to fully evaluate the extension of the SSM. MRI shows high signal intensity after contrast, and impairment of optic nerve and internal carotid artery can be assessed too. The histopathology report of SSM shows cyst-like lesions lined by respiratory epithelium and retention of mucoid secretions resulting in lining and erosion of the sinus bony walls.⁴⁵

TREATMENT

The objectives of the surgery in SSM are diagnosis, drainage of the cystic contents, and excision or marsupialization of the mucocele. In the majority of cases, the SSM is easily accessible via the

transsphenoidal route.⁴⁶ Endoscopic transnasal sphenoidotomy has largely replaced the conventional open technique with excellent results. The endoscopic approach for SSM avoids external incision and associated morbidity.⁴⁷ This approach avoids the risk due to transcranial surgery. The transcranial approach should be advised in patients of SSM with extensive intracranial and intra-orbital involvement, which may even need a combination of transsphenoidal-transcranial approaches for getting the best results.⁴⁸ Some surgeon advocates bony decompression of the optic nerve if vision disturbances happened which may need an intracranial extradural approach. The surgical approach for SSM is often focused on reducing the size of the mucocele which decreases the compressive effect of the lesion on the optic nerve. The visual acuity and visual field defects are rarely improved after surgical removal of the SSM and only one-fifth of the patients with visual impairment by SSM show improvement of the visual acuity to more than 20/40 following surgery in follow up periods.⁴⁹ However, early surgical intervention is required to limit the complications like visual impairment.⁴⁹ One report showed retrobulbar optic neuritis attributable to the SSM.¹⁰ In this case of retrobulbar optic neuritis, early surgical excision of the SSM brought about improvement in visual acuity and visual field.¹⁰ Another report showed sudden blindness due to isolated SSM where endoscopic excision of the mucocele, total vision, and visual field recovered.⁵⁰ Surgical interventions in SSM should focus on decreasing the size of the mucocele for lowering the compressive effect of the lesion on the optic nerve. Treatment with marsupialization or surgical excision of the mucocele usually results in rapid improvement of the ocular manifestations especially in third nerve palsy, but with a smaller effect on visual impairments.⁴⁹ There are very few cases of optic neuropathy due to SSM where the prompt diagnosis and treatment could recover the optic neuropathy and improve the vision. If the treatment is delayed in optic neuropathy along with SSM, the prognosis will not be promising. Marsupialization or surgical excision of the SSM usually results in a rapid reduction of ophthalmic manifestations particularly third nerve palsy, but with little impact on visual impairments.⁵⁰

CONCLUSION

Sphenoid sinus mucocele (SSM) is a rare clinical entity that results in serious sequelae if diagnosis and treatment are delayed inappropriately. SSM should be kept in mind in case of a patient with a deep-seated headache, visual disturbances, and external ophthalmoplegia. Early surgery should be planned for preventing neurological deficits and permanent blindness. Endoscopic transnasal sphenoidotomy with adequate removal of anterior and inferior walls of the sphenoid sinus is helpful for adequate drainage and avoiding recurrence. Marsupialization of the SSM is another option with good results.

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