Unusual Case of Maxillary Antrum Obliteration Secondary to Cyst Removal-A Case Report and Review of Literature

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Abstract
Intraluminal osteogenesis of maxillary antrum is very rare. We report a case of Extrinsic cyst (residual cyst) involving the maxillary antrum which was enucleated by Cadwell-luc approach. Six month follow-up showed osteogenesis of the maxillary antrum.

Keywords: Maxillary antrum; Cyst Enucleation; Maxillary sinus obliteration; Osteogenesis; Maxillary sinus Hypoplasia (MSH); Silent sinus syndrome (SSS)

Introduction
Maxillary sinus develops from invagination of the mucous membrane of middle meatus of the nasal cavity at about the 3rd month of intrauterine life. Fully development reaches with the age of 16 years. Enlargement of the sinus is uncommonly encountered, and is produced by air (pneumocele) and mucus (mucocele) entrapment, or by benign tumors. Reduction in size and volume is more frequent. It can be due to Heredo-familial syndromic conditions, neoplastic changes, systemic disorders like sickle cell anemia and osteopetrosis, Fibro-osseous disorders, Midfacial fractures involving the sinus, iatrogenic cause, like direct surgical intervention (Caldwell-Luc procedure), benign odontogenic cysts as they enlarge produce size and shape distortions by external impingement. In this report we present a rare case of florid ossification secondary to residual cyst enuculation by Cadwel-Luc operation. Our literature search has not shown any similar case.

Case Report
A 42 year old female patient fit and healthy reported to the department of maxillofacial surgery with the history mild asymmetry of the face on the left side. She had a history of extraction of 26 four years back at another clinic. She has been noticing a swelling on left side of face since then which was slowly increasing in size. CT scan PNS region showed a residual cystic lesion from 26 involving the left maxillary antrum.

Biopsy was done and Histopathology report was of a radicular cyst. The cyst was enucleated by Cadwell Luc approach under General anesthesia with inferior meatus nasal antrostomy. The area healed uneventfully. The patient was evaluated at 1months, 3 months and 7 months. During her 7 months review it was noted that she had slight odema on the left side of face. A CT scan was done which showed obliteration of the maxillary antrum

A repeat biopsy was done which showed bone on exploring the antrum with lining which was histopathologically reported as fibrotic tissue with normal oral mucosa. The patient is under follow-up.

Discussion

Intraluminal osteogenesis of maxillary antrum has been reported very rarely. The reduction of the size of the maxillary sinus is multifactorial and includes congenital and acquired variants. Among the latter are
1. Fibro osseous lesions,
2. Tumors of maxillary antrum,
3. Trauma resulting in displaced bony fragments
4. Cysts involving the maxillary antrum

We only address those disorders which cause a reduction of the actual bony configuration and anatomic borders of the sinus in this discussion section

Although complete aplasia of the maxillary sinus can occur, it is extremely rare, whereas maxillary sinus hypoplasia (MSH) is a well-known clinical entity and is classified by CT scan. There are three types: MSH-Type I has normally developed uncinate process and well-defined infundibulum with mild-to-moderate hypoplasia, MSH-Type II is characterized by a hypoplastic uncinate process and an ill-defined or absent infundibular passage, and soft tissue opacification of the sinus on CT scan. MSH-Type III shows absence of uncinate process with profound hypoplasia of the sinus, seen only as a shallow cleft. Unilateral and bilateral hypoplasia occurs in less than 10% of patients [6].
Syndromal etiologies of MSH can be broadly divided into lack of development from failure of midfacial skeleton growth and obliteration of the sinus cavities from osteosclerosis. Many forms of craniofacial dysostoses exist, most common being mandibular dysostosis (Treacher-Collins syndrome), Acrocephalosyndactyly (Apert’s syndrome), craniofacial dysostosis (Crouzon’s syndrome), and maxillo-mandibular dysplasia (Binder’s syndrome). The size of the maxillary sinus is reduced by unilateral hyperplasia of the lateral maxilla and zygoma in Goldenhar’s syndrome. In Williams syndrome, or Elfin Face syndrome paranasal sinuses are small bilaterally due to failure in prenatal and post-natal growth.

MSH is considered to stem from changes in embryologic development, silent sinus syndrome (SSS) is considered to be an acquired disease. Usually the patient is asymptomatic and they suddenly develop enophthalmos or hypoglobus secondary to the collapse of the orbital floor. Obstruction of the natural ostium is thought to be the initiating factor, resulting in hypoventilation and generating a negative pressure causing demineralization and bowing of the walls into the sinus, causing collapse of sinus.

Hematologic conditions like sickle cell anemia and thalassemia affects the marrow space of the facial bones. As with all marrow spaces in the body, the medullary spaces of the maxilla are widened to accommodate the need for increased production of blood cells, leading to a decrease in the volume, or obliteration, of the sinus itself. Maxillary sinus hypoplasia has been reported with primary and acquired hypothituitarism and hypothyroidism. Osteopetrosis (Albers-Schonberg disease, Brittle Bone disease) is a family of heredofamilial disorder, in which a proliferation of abnormally dense bone replaces the normal medullary bone, resulting in narrow space and sinus cavity obliteration [5]. Our patient was free of any systemic disease which could have lead to obliteration of the sinus.

Another greatest source of obliteration of the maxillary sinus was malignant neoplasms arising in the sinus, mainly squamous cell carcinomas. The tumor proliferates in the cavity resulting in the destruction of the wall of the maxillary antrum. As the tumors advances there may be no recognizable sinus, only an irregular soft tissue mass. Malignant tumors arising from the maxillary alveolus may grow into the sinus but generally destroy only its floor, whereas intracavitary tumors have multidirectional growth. The main feature of malignant tumors is an irregular appearing sinus due to the loss of its bony walls, with soft tissue inside.

Benign tumors of the maxillary sinus are extremely rare and include salivary gland neoplasms, inverted papillomas and osteomas, which produce an intracavitary mass which can expand the sinus if large. An exception is the pseudo tumor, which is a chronic inflammatory lesion that can produce extensive destruction and distortion of the maxillary sinus.

Odontogenic growth like radicular cysts arising from the roots of nonvital teeth elevates the antral floor, while developmental (dentigerous, keratocysts) cysts displace the posterior and lateral walls. They don’t destroy the walls of the antrum but expands and create what appears to be a duplicate sinus. Same is the case with Benign odontogenic tumors (myxoma, cementoma, odontoma) which narrow the maxillary sinus by external compression, Only exception being the Ameloblastomas. Although histologically they are benign, but locally invasive and can infiltrate the sinus. Radiographically, the sinus walls may be remodeled, or destroyed. The calcifying epithelial odontogenic tumor (Pindborg tumor) 2 presents similarly. Our case had a radicular cyst initially for which tooth extraction was done after which slowly the cyst enlarged involving the maxillary antrum and destroying the walls of antrum.

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Fibro-osseous disorders may broadly be divided into ossifying lesions (osteomas, ossifying fibroma), fibrous dysplasia, and Paget’s disease. The ossifying fibroma (also cementifying fibroma) is an encapsulated benign bony neoplasm that encroaches upon the antrum by mass effect. It arises from the alveolar segment of the maxilla and expands internally towards the sinus rather than deform the outer surface bones. Fibrous dysplasia is an idiopathic skeletal disorder which occurs as a monostotic and polyostotic in which medullary bone is replaced by poorly organized weak fibro-osseous tissue. In the maxilla, the abnormal “woven” bone gradually replaces the normal medullary bone, enlarging the maxilla and progressively obliterating the maxillary sinus cavity. By contrast, in Paget’s disease, or osteitis deformans, the maxilla is enlarged bilaterally, especially in its posterior aspect, by the formation of abnormal bone formed by both osteoclastic and osteoblastic activity. In replacing the normal medullary bone, both sinus cavities become progressively obliterated [1].
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Midfacial fractures commonly change maxillary sinus shape and volume by telescoping, or rotation, of bony fragments into the sinus. This is especially true with zygoma fractures. Orbital floor “blowout” fractures also destroy the roof of the maxillary sinus, and many times lowers the roof into the antrum. There may be areas of bony sclerosis and irregularities in the antrum post reduction of maxillary and zygoma fractures.

The paranasal sinuses possess the idiosyncratic ability to auto-obliterate by osteoneogenesis and fibrosis, following disturbance and removal of their lining especially in Caldwell-Luc operations. This varies in degree with different sinuses, but is most marked in the maxillary and frontal sinuses. In the maxillary sinus, the process progresses medially from the zygomatic recess in varying degrees, producing lateral narrowing to near total obliteration. The condition can be distinguished from developmental hypoplasia by the presence of a surgical defect in the lateral wall and the dense bony sclerosis of the remaining walls. Our case had cyst removal through Caldwell-Luc approach which resulted in florid bone deposition. Our case was unique because the amount of bone deposition was large and florid following a peculiar pattern along the displaced and dissected periosteum. This case proves that periosteum in the maxilla has high regenerative capacity and if used properly can be used for Osseo integration of Implants. The high regenerative capacity of the sinus mucosa and periosteum is made use in sinus lift procedures [4]. Considering the fact that this technique reduces the risks of morbidity related to bone graft harvesting or eliminates the cost of allogenic or synthetic grafting materials.

Calcification with a nodular or linear shape was found with both fungal and non fungal sinusitis; however, fine punctate calcification was found only in fungal sinusitis, while smooth-margined, round, or eggshell type calcification was found exclusively with the non fungal variety [3]. Our patient was not immunocompromised nor did not have any fungal sinusitis.

Conclusion

The maxillary sinus either expands or compresses its walls in response to an internal or external slow growing mass. Aggressive infiltrating lesions will produce bone destruction and will show a mixed radiological pattern of remodeling and erosion. Lesions arising from fibro-osseous disorders, hereditary anemias and bone dysplasias will involve the medullary spaces and progressively obliterate the sinus.

The high frequency of variation seen in the normal and abnormal anatomy of the maxillary sinus, and how this sinus preserves its morphology across polyethnic groups, continues to intrigue the author. While volume differences may vary in diverse populations, there is a relatively uniform response to a multiplicity of stresses across these ethnic boundaries.

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Bibliography


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