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Maxillary sinus hypoplasia*

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SUMMARY

Maxillary sinus hypoplasia (MSH) is an uncommonly encountered condition by otolaryngologists. The computerized tomography (CT) scans provide valuable data about the anatomic details of the paranasal sinuses. MSH may be misdiagnosed as an infection or a neoplasm of the maxillary sinuses. Variations of the other paranasal structures, especially the uncinate process associated with MSH were defined. MSH shows three distinct hypoplasia patterns. Type I MSH characteristics are mild hypoplasia of the maxillary sinus, normal uncinate process and a well-developed infundibular passage. Significant hypoplasia of the maxillary sinus, hypoplastic or absent uncinate process and absent or pathologic infundibular passage are seen in Type II MSH. Type III MSH is characterized by the absence of an uncinate process and cleft-like maxillary sinus hypoplasia. In this study a series of 18 patients with MSH were presented. Twelve cases of unilateral and 6 cases of bilateral maxillary antrum hypoplasia were evaluated and 13 MSH type I, 7 MSH type II and 4 MSH type III were detected. Three ethmomaxillary sinuses, an overpneumatized posterior ethmoid cell into the orbit and the maxillary sinus were determined. Our series showed that the uncinate process anomalies related to MSH may lead to inadvertent orbital complications and therefore should be kept in mind.

Key words: maxillary sinus hypoplasia, anatomic variation, ethmomaxillary sinus, CT scan, classification

INTRODUCTION

Maxillary sinus hypoplasia (MSH) may be misdiagnosed as an infection or a neoplasm of the maxillary antrum. This may lead to unnecessary and difficult surgical attempts. MSH is an uncommon clinical entity that has been reported in 1.73% to 10.4% of patients with sinus symptoms (Karmody et al., 1977; Bassiouny et al., 1982; Bolger et al., 1990; Khanobthamchai et al., 1991; Sirikci et al., 2000). Asymptomatic cases may have lower ratios than patients with sinus symptoms. The MSH term describes an anatomic abnormality spectrum ranging from mild sinus hypoplasia to cleft-like sinuses.

The maxillary sinus develops in the third month of fetal life as a mucosal evagination of the middle meatus of the nasal cavity (Bassiouny et al., 1982; Vinson and Collette, 1991). During this evagination process, simultaneous resorption of the maxillary bone occurs.

The maxillary sinus volume is 6-8 mm^3 at birth and increases to several directions such as the infraorbital wall, nasal cavity, zygomatic process and alveolar process. Until the age of 8 years, the maxillary antrum volume increases by 2 mm per year in the vertical and lateral dimensions and by 3 mm per year in the anteroposterior dimension (Kosko et al., 1996). At the age of 10 years, the lower boundary of the maxillary sinus is at the level of the nasal cavity floor. The following growth is principally in an inferior direction and after the eruption of the upper teeth, the maxillary sinus size reaches its maximum volume at about the late adolescence ages (Kosko et al., 1996).

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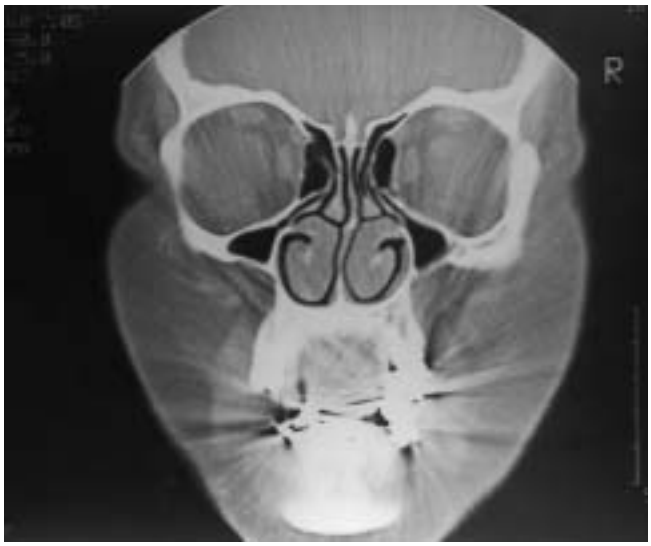

Figure 1. Bilateral type I MSH well developed infundibular passage with mild MSH.

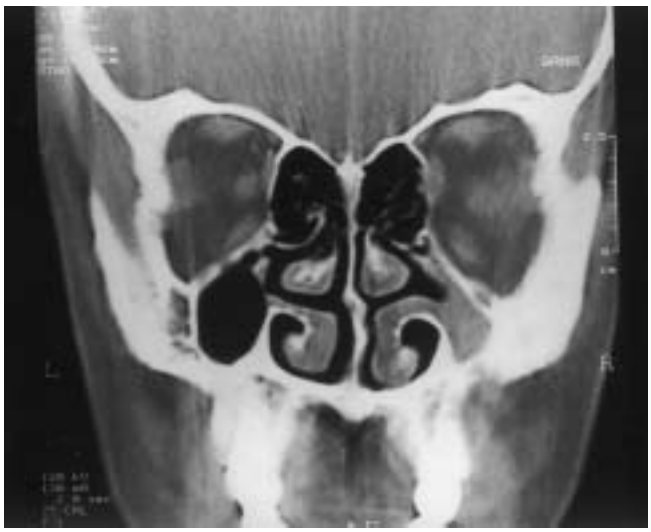

Figure 2. Type II MSH, hypoplastic uncinate process and pathologic infundibular passage with significant MSH.

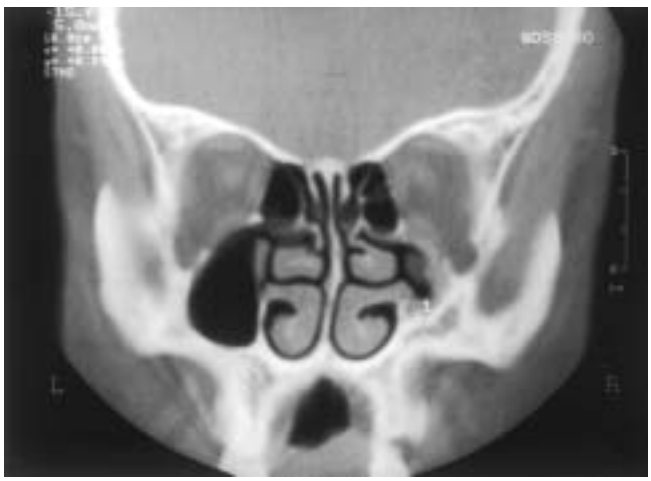

Figure 3. Type III MSH, slit-like maxillary sinus.

The first attempt to classify the MSH was introduced by Bassiouny (Bassiouny et al., 1982). Later Bolger et al. (1990) proposed a new classification, which is currently widely accepted. According to this classification MSH shows three distinct hypoplasia patterns. Type I MSH characteristics are mild hypoplasia of the maxillary sinus, normal uncinate process and well-developed infundibular passage. Significant hypoplasia of the maxillary sinus, hypoplastic or absent uncinate process and absent or pathologic infundibular passage are seen in Type II MSH. Type III MSH is characterized by the absence of uncinate process and cleft-like maxillary sinus hypoplasia (Bolger et al., 1990).

The aim of this study was to present a series of 18 patients with MSH in the light of the relevant literature and derive clinical implications.

MATERIALS AND METHODS

The paranasal sinus computerized tomography scans of 280 patients with sinus symptoms applying for medical care to the Malatya Inonu University School of Medicine and Izmir Ataturk State Hospital were enrolled our study.

Computerized tomographic examinations were performed on either GE prospeed helical CT or W 950 SR x-ray system CT. Scans were obtained by 5 mm increments from the glabella to posterior boundary of the sphenoid sinus in coronal planes. Transverse sections were added to evaluate the posterior ethmoid cells and sphenoid sinuses. All CT scans were evaluated in both bone and soft tissue windows.

The Bolger's MSH classification system was used for classification of our patients with MSH.

RESULTS

MSH was detected in 18 (10 female and 8 male) of 280 patients (6.4%), with a total of 24 MSH in these 18 patients. MSH was presenting unilaterally in 12 cases and bilaterally in 6 cases. As for the severity, 13 MSH type I, 7 MSH type II and 4 MSH type III were detected (Figures 1 to 3). Among 6 bilateral maxillary sinus hypoplasia, 3 cases with bilateral type I MSH, 2 cases with type I MSH on one side and type III MSH on the other side, and 1 case with type I + type II MSH were observed.

MSH was found on the left side in 9 cases and on the right side in 15 cases. The mean age of the patients was 32 (ranging from 21 to 56) years. Infundibular pathology and developmental abnormalities of the uncinate process were observed in all patients with MSH type II and III. Three overpneumatized posterior ethmoid cells called ethmomaxillary sinus were discovered (Figure 4).

Any local or systemic reason such as the Treacher-Collins syndrome, Crouson's syndrome or Apert's syndrome, thalassemia, Paget's disease or cretinism leading to MSH were not observed.

DISCUSSION

Embryogenic developmental abnormalities or acquired reasons such as infection or trauma leading to arrest of sinus pneumatization may be the cause of MSH (Weed and Cole, 1994). A developmental abnormality may also be the reason of MSH during the initial stages of in-utero sinus development (Bolger et al., 1990). Bolger et al. (1990) suggested that developmental abnormalities of the uncinate process may lead to MSH. Hypoplastic or absent uncinate processes were observed in all our patients with MSH type II and type III.

An uncinate process may play an important role for aeration of the maxillary sinus during the expiration phase by altering the direction of exhaled air into the maxillary sinuses (Sirikci et al., 2000). Maxillary sinusitis was detected in all of our cases with absent uncinate process. In our series all patients except those patients with MSH had a well developed uncinate process ending at the lamina papyracea, the skull base or the middle concha and some of them had a free ending.

Milczuk et al. (1993) noticed the relation of MSH and chronic maxillary sinusitis. However, it is not known whether the sinusitis is a primary or secondary condition to the MSH. Mucosal thickening of the maxillary sinus was observed in 4 of 13 MSH type I and in all MSH type II and III cases. In our series, hypoplastic or an absent uncinate process may be the reason of the sinusitis because of impeding the mucociliary clearance and lack of altering effect of the well developed uncinate process on the direction of the expired air (Milczuk et al., 1993; Sirikci et al., 2000). Weed et al. (1994) observed a clinically absent natural maxillary ostium associated with a poorly developed uncinate process and infundibular passage in all 4 patients with MSH. Salib et al. (2001) emphasized the relationship between MSH and maxillary sinusitis and suggested a posteriorly placed middle meatal antrotomy to avoid the inadvertent orbital complications.

Paling et al. (1982) suggested that acquired MSH may be secondary to the sinusitis leading to new bone formation and partial or total obliteration of the maxillary sinus. MSH may be secondary to accidental or surgical trauma during the developmental period (Kosko et al., 1996). Kosko et al. (1996) reported five children with MSH secondary to functional endoscopic sinus surgery for chronic refractory sinusitis. They proposed that surgical intervention may lead to MSH, due to activation of bone formation and removal of pneumatization centers. The patients did not give any history of accidental or surgical trauma in our series.

Weed et al. (1994) suggested an analogous mechanism as hypoventilation between the MSH and chronic otitis media with effusion since a similar thick mucoid effusion may be seen in these diseases. Choanal atresia has been accused as the etiologic agent of MSH. The idea that nasal ventilation may be important for paranasal sinus pneumatization was proposed in similarity to what middle ear ventilation does for temporal bone pneumatization. But Behar et al. (2000) found that the maxillary sinus development is independent of posterior nasal ventilation.

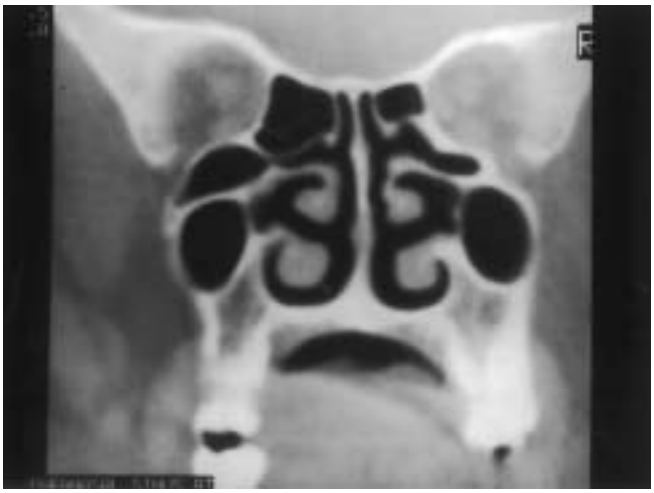

Figure 4. Bilateral ethmomaxillary sinus draining into the superior meatus.

Sirikci et al. (2000) emphasized that there may be an association between a low-lying roof of the ethmoid and MSH. In their series, more than one-third of the MSH cases had presented with a low-lying roof of the ethmoid. This may lead to inadvertent intracranial complications during functional endoscopic sinus surgery. We found a low-lying ethmoid roof in two of our cases. One of them had type I and the other has type III MSH.

An association between maxillary sinus hypoplasia and orbital enlargement has been reported (Bierny and Drayden, 1977; Modic et al., 1980; Geraghty and Dolan, 1989; Weed and Cole, 1994). Thus, orbit displaces inferiorly.

Khanobthamchai et al. (1991) reported 8 ethmomaxillary sinuses seen above the maxillary sinus draining into the superior meatus. The ethmomaxillary sinus is a posterior ethmoidal air-cell enlarged at the expense of the maxillary sinus. An ethmomaxillary sinus should be differentiated from a septated maxillary sinus, with the latter draining into the ethmoid infundibulum. We found three cases with an ethmomaxillary sinus in our series.

There are several classifications for the MSH. The first attempt to classify the MSH was achieved by Bassiouny et al (1982) as isolated, associated with regional abnormalities such as Treacher-Collins Syndrome, Crouson's Syndrome and Apert's Syndrome, and related to systemic disorders such as thalassemia and cretinism. Bolger et al. (1990) classified the MSH based on the degree of maxillary sinus pneumatization and lateral nasal wall abnormalities. Type I MSH characteristics are composed of mild MSH, a normal uncinate process and a well-developed infundibular passage. Significant MSH, an hypoplastic or absent uncinate process and an absent or pathologic infundibular passage are seen in Type II MSH. Type III

Table 1. The rates of MSH according to Bolger's classification.

	Bolger	Sirikci	Erdem
Type I MSH	14 (66.7%)	7 (33.3%)	13 (54.2%)
Type II MSH	6 (28.6%)	6 (28.6%)	7 (29.2%)
Type III MSH	1 (4.8%)	8 (38.1%)	4 (16.7%)

MSH is characterized by the absence of an uncinate process and cleft-like MSH. Sirikci et al. (2000) suggested a new classification of the MSH. While they accepted Bolger's classification, they added the orbital enlargement to the type II and type III MSH and proposed a new definition of the mild and severe MSH by comparing the maxillary sinus size with orbital size. Table 1 shows the distribution of MSH according to these types.

CONCLUSION

MSH is a rare, but important condition that may lead to incorrect diagnosis and inadequate treatments including surgery. The diagnosis and differential diagnosis of MSH may be accomplished by paranasal sinus CT scans. As uncinate process anomalies are often presented with MSH, the probability of inadvertent orbital complications during surgery should be kept in mind. It is suggested that this anatomical configuration may result in additional dangers at surgery or need to pay special attention to the anatomy.

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