CASE REPORT

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Pneumatization of the concha inferior as a cause of nasal obstruction*

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SUMMARY

Chronic nasal obstruction is a common disorder. Hypertrophy of the inferior turbinates is responsible for nasal obstruction more frequently than it is commonly thought. A pneumatized inferior turbinate has recently been described as a cause for nasal obstruction and only two cases have been reported until now. Inferior nasal turbinate develops by endochondral ossification of components of the mesethmoid and ectethmoid. The chondral framework of the inferior turbinate consists of a double lamella and two separate ossification centers that develop between the fifth and seventh month of fetal life. The separate ossification centers meet by the eighth fetal month. During ossification, the inferior turbinate detaches from the ectethmoid and becomes an independent bony structure. During that time the epithelium may misinvaginate into double lamellas and such double lamellas formed by the inferior turbinate may become persistent.

A patient was referred to our clinic with headaches and nasal obstruction. A CT scan was performed which showed that the right lower concha was pneumatized. The headache of the patient disappeared after partial resection of the lower and middle turbinate.

Key words: inferior concha, nasal obstruction, headache

INTRODUCTION

The inferior turbinate is a part of the lateral nasal wall. The physiological function of the turbinate is to contribute to warming, humidification and filtration of inspired air. In addition, the turbinate regulates the flow of inspired air. Therefore, the inferior turbinate is an important structure in the nasal cavity. The anterior part of the inferior turbinate is located in the vicinity of the narrowest part of the nose isthmus nasi (Hilberg et al., 1990). Airflow through each nasal cavity is altered by the swelling and shrinkage of the inferior turbinates, which contribute to a nasal cycle in the majority of individuals for 3-4 hours. Changes in the skeleton of the turbinate or increase in the volume of the erectile mucosa may influence nasal patency (Hilberg et al., 1990). Although it is not a life-threatening condition, nasal obstruction can interfere with the quality of life (Passali et al., 1999). Chronic nasal obstruction is a common disorder and if septal defects are excluded, the main structure contributing to this problem is the inferior turbinate (Hilberg et al., 1990; Passali et al., 1999). Nasal obstruction rarely results from the pneumatization of the inferior turbinate. To date, only two cases have been reported. We here present the third case related to pneumatization of the inferior turbinate.

CASE REPORT

A 35-year-old man presented to our department with headaches and nasal obstruction. On anterior rhinoscopy, both the left and right inferior turbinates were hypertrophic. After application of adrenaline, the volume of the left inferior turbinate decreased but that of the right turbinate remained unchanged. The examination was supplemented with nasal endoscopy. For evaluation of the headaches, a coronal CT was obtained. In CT, it seemed that the right inferior and the middle turbinate were pneumatized and the middle turbinate was hypertrophic (Figure 1a and b). The paranasal sinuses were normal. The patient underwent partial inferior turbinectomy under local anesthesia. Following this operation, the nasal obstruction was relieved but the headache continued. The headache was evaluated as due to physical contact and therefore a partial middle turbinate resection was

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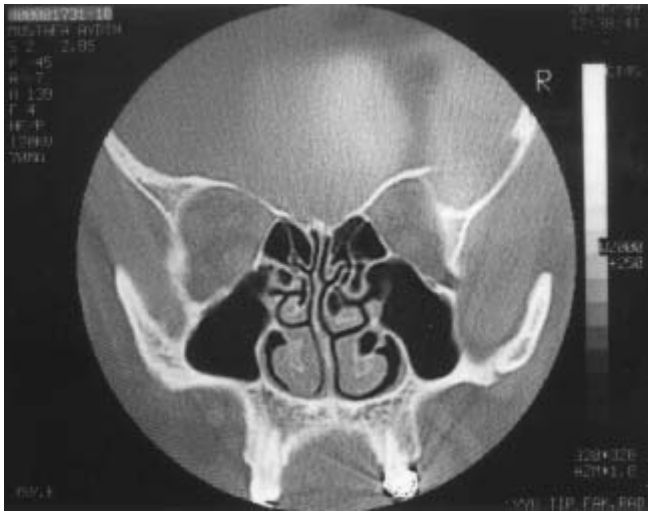

1a.

1b.

Figure 1. In CT, the pneumatized right inferior (a) and middle turbinate (b).

performed 3 months later. Following the second operation, the headache disappeared.

DISCUSSION

Extensive pneumatization of the middle turbinate, also referred to as concha bullosa, has been implicated as a possible etiologic factor in recurrent sinusitis due to its postulated negative influence on paranasal sinus ventilation and mucociliary clearance in the middle meatus region (Bolger et al., 1991). Initially described by Santorini in 1739, such pneumatization is believed to present an anatomic variation of ethmoid air cell development, and not the result of a prior pathologic intranasal process (Bolger et al., 1991). Less frequently, aeration of the superior turbinate may occur, whereas aeration of the inferior turbinate is infrequent. The first cases of pneumatization of the inferior turbinate were reported by Dogru et al. (1999) and Dawlatly (1999), who suggested that a pneumatized inferior turbinate might be of congenital origin. The inferior nasal turbinate develops by endochondral ossification of components of the mesethmoid and ectethmoid. The chondral framework of the inferior turbinate consists of double lamella and two separate ossification centres, which appear between the fifth and seventh month of fetal life. The separate ossification centres meet by the eighth fetal month. During ossification, the inferior turbinate detaches from the ectethmoid and becomes an independent bony structure (Moss-Saletjin, 1991). During this time, the epithelium may misinvaginate into double lamella and double lamellas forms of the inferior turbinate may become persistent. All cases support the idea of congenital origin because the lower turbinate pneumatization has occured at the same side with the middle concha bullosa and there was no sinusitis.

Chronic nasal obstruction is a common disorder and the inferior turbinate hypertrophy is responsible for nasal obstruction more frequently than it is commonly thought but it does not cause headache and facial pain (Ophir et al., 1992). Some of the common causes of the inferior turbinate hypertrophy include compensatory enlargement resulting from a long-standing septal deviation as well as generalized nasal mucosal diseases in allergic and vasomotor rhinitis caused by thickening of the mucosa without hypertrophy of the underlying bone (Passali et al., 1999; Ophir et al., 1992). A pneumatized inferior turbinate is a recently described cause for nasal obstruction. Dogru et al. (1999) and Dawlatly (1999), believed that it may result in contact headache and nasal obstruction. Contact headache of the paranasal sinus and nasal origin in the absence of sinus inflammatory disease is a clinical entity, which acts as a mechanical stimulus initiating an axon reflex with resultant referred pain (Clerico, 1996). Septal spurs with contact points on the lateral nasal wall were the most frequent cause and various intranasal abnormalities or anatomical variations have been implicated in this disorder, including enlargement of the ethmoid bulla, septal deviations, the middle turbinate abnormalities (Chow, 1994; Clerico, 1996). The inferior turbinate hypertrophy does not cause headache, because the mucosa of the turbinates is not so sensitive (Chow, 1994). Facial pain and chronic headache are therefore not likely to be cured by inferior turbinectomy (Ophir et al., 1992). In our case, the symptom of nasal obstruction was relieved after partial resection of the inferior turbinate, but the headache was not. Consequently, we performed a partial resection of the middle turbinate, as a second operation. Postoperatively, the headache disappeared. These findings suggest that the middle turbinate is more important than the lower turbinate in causing headaches. Finally, pneumatization of the inferior turbinate should be considered when lower turbinate hypertrophy does not respond to vasoconstrictors.

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