



Analysis of sinonasal anatomical variations associated with maxillary sinus fungal balls



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ABSTRACT

Objective: The pathogenesis of MSFB development remains unclear, but it has been suggested that poor sinus ventilation is associated with disease development; such a ventilation is influenced by anatomical variation of the paranasal sinuses. Thus, we sought to determine whether sinonasal anatomical variations were associated with MSFB development.

Methods: Thirty-one patients with MSFB and 28 gender-matched control patients were included in the present study. The presence or absence of Haller cells and a concha bullosa were scored, and the angle of septal deviation and the minimal and maximal lengths of the infundibulum were measured on preoperative computed tomography images.

Results: In the MSFB group, both a concha bullosa (61.3% vs. 28.6%, $p = 0.006$) and Haller cells (41.9% vs. 30.4%) were present at higher frequencies than in the control group, although the between-group difference in Haller cell occurrence was not statistically significant ($p = 0.348$). In addition, MSFB patients had a significantly lower mean infundibular width (3.23 ± 0.69 mm vs. 3.99 ± 1.17 mm, $p < 0.001$) and a longer infundibular length (9.71 ± 1.43 mm vs. 8.23 ± 1.72 mm, $p < 0.001$) than controls.

Conclusions: Sinonasal anatomical variations, especially the presence of a concha bullosa, and/or a narrow and long infundibulum, may play roles in the development of maxillary sinus fungal balls (MSFBs).

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1. Introduction

Fungal rhinosinusitis is traditionally divided into two categories, invasive and non-invasive, based on histopathological

findings. The “invasiveness” refers to invasion of the mucosal layer. Non-invasive fungal rhinosinusitis is subdivided into three types: saprophytic fungal infection, fungal balls, and fungus-related eosinophilic rhinosinusitis (including allergic fungal rhinosinusitis) [1,2]. Of these, a sinus fungal ball is the most common fungal disease entity, usually involving a single sinus, most commonly the maxillary sinus [3].

Computed tomography (CT) reveals partial or complete opacification, and associated bony changes, including bone thickening, erosion, sclerosis, and destruction. *Aspergillus*

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species are the principal causative fungal species. The pathogenesis of maxillary sinus fungal ball (MSFB) remains largely unknown. Although the invasive types of fungal rhinosinusitis are common in immunocompromised patients, non-invasive fungal balls have been found in healthy immunocompetent individuals. This suggests that persistent local factors, such as poor sinus ventilation and/or endodontic treatment of maxillary teeth, may play roles in fungus proliferation and growth in a sinus [4–7].

Many previous studies have suggested that a history of endodontic treatment of maxillary teeth is an independent risk factor for development of MSFB [3,6,8,9]. However, this cannot explain MSFB in patients in whom the fungal ball develops in a remote sinus cavity and who have no history of dental care.

It has also been speculated that MSFB development is associated with a poorly ventilated sinus. Sinus ventilation is influenced by anatomical variation of the paranasal sinuses. Therefore, in the present study, we sought to determine whether sinonasal anatomical variations in the region of the ostiomeatal complex were associated with MSFB development, via radioanatomical analysis.

2. Materials and methods

A retrospective radiological analysis was performed on all patients with histopathologically proven fungus balls attributable to *Aspergillus* species, and who were treated via endoscopic sinus surgery. Between January 2007 and December 2012, 31 patients with unilateral MSFB were included and 28 contemporaneous gender-matched patients (thus with 28 nasal septa and 56 sinuses) with pituitary tumours who underwent transsphenoidal surgery were selected as a control group. Patients were excluded if they showed any evidence of pre-existing conditions including a previous history of sinonasal surgery, a malignancy, immunocompromised status, and/or any other type of fungal infection (e.g., allergic fungal sinusitis or invasive fungal sinusitis).

Using CT images, the presence of sinonasal anatomical variations (including Haller cells and a concha bullosa) was scored; the angle of septal deviation, as well as the minimal width and maximal length of the infundibulum were measured using the appropriate modes of a Picture Archiving and Communications System (PACS) workstation (DEJA-view; Dongeun, Seoul, Korea). The angle of septal deviation was that between the apex of the septal deviation and a straight line crossing the midline of the nasal crest and the crista galli, and was calculated using the angle-annotation PACS mode (Fig. 1A). The direction of septal deviation was also evaluated. If the septum deviated to the lesional side in the MSFB group, the septal angle was considered positive (and, if otherwise, negative). In controls, we first selected one side of the maxillary sinus using a random number table generated using the random number generator running in SPSS (PASW Statistics 18, release 18.0.0, Chicago, IL) and the direction of septal deviation was next noted using the method described above.

The maximal length of the infundibulum was measured at the level of the natural ostium of the maxillary sinus, in the coronal plane, and the minimal width was the distance between the bony margins at the midpoint of the infundibulum (in the same plane); we used the PACS distance-annotation mode to obtain our data (Fig. 1B and C). In the MSFB group, infundibular measurements were taken on the side contralateral to the lesion because many patients exhibited anatomical destruction around the ostiomeatal complex. Additionally, to determine whether the minimal width and maximal length of the infundibulum differed between sides, we compared our control data for one side with those of the other side.

This case–control study was approved by the Institutional Review Board of the Soonchunhyang University College of Medicine as a retrospective medical record review; it was decided that informed consent was not required.

Statistical comparisons were made between data of the MSFB and control groups. The significance of anatomical variations (the presence of Haller cells and a concha bullosa) was explored via Chi-squared analysis. The significances of differences in the angle of septal deviation, and the minimal width and maximal length of the infundibulum, between the two groups, were analysed using unpaired *t*-tests. A *p*-value <0.05 was assumed to reflect statistical significance.

3. Results

A total of 31 MSFB patients, aged 18–74 years (mean age: 56.8 years), were included. Eleven patients were males (35.5%). The control group consisted of 10 males (35.7%) and 18 females (64.3%) of mean age 44.4 years (range: 22–74 years). The sex ratio did not differ between groups (*p* = 0.985), but the mean age of the control group was less than that of the MSFB group (*p* = 0.002) (Table 1).

The mean angle of septal deviation in the MSFB group was $-0.22 \pm 8.71^\circ$ and that in the control group $-1.88 \pm 6.36^\circ$ (Table 2); these values did not differ significantly (*p* = 0.411).

In terms of sinonasal anatomical variation and infundibular measurements, a concha bullosa was present significantly more frequently in the MSFB group than controls (61.3% vs. 28.6%, *p* = 0.006). Haller cells were more often present in MSFB patients than controls (41.9% vs. 30.4%), but the difference was not significant (*p* = 0.348) (Table 3). In addition, MSFB patients had a significantly lower mean infundibular width (3.23 ± 0.69 mm vs. 3.99 ± 1.17 mm, *p* < 0.001) and a longer infundibular length (9.71 ± 1.43 mm vs. 8.23 ± 1.72 mm, *p* < 0.001) than controls (Table 3).

No significant between-side difference in mean infundibular width (right vs. left; 3.94 ± 1.13 mm vs. 4.05 ± 1.22 mm; *p* = 0.742) or mean infundibular length (right vs. left; 8.05 ± 1.74 mm vs. 8.43 ± 1.71 mm; *p* = 0.406) was found in controls (Table 4).

4. Discussion

The aetiopathogenesis of sinus fungal balls caused by *Aspergillus* species has been intensively debated and three possible theories have been advanced to date [10]: the

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