CASE REPORT

Signal void and pseudo-pneumatized sinus in fungal rhinosinusitis – Case report

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ABSTRACT

BACKGROUND. Signal void, or the absence of signal on MRI sequences, in the sinonasal region may be encountered in fungal rhinosinusitis cases with the aspect of a pseudo-pneumatized sinus, leading to diagnostic errors.

CASE REPORT. We present the case of a 75-year-old woman referred to our clinic for complete and persistent right-sided nasal obstruction. The patient was evaluated using sinus CT and contrast-enhanced head MRI. Opacification of the right maxillary, ethmoid and frontal sinuses as well as of the right nasal fossa were seen on CT, with maxillary sinus expansion and osseous erosion. The MRI showed T2 signal void in the maxillary sinus with extension to the nasal fossa, creating the appearance of a pseudo-pneumatized sinus, and hyperintense signal in the ipsilateral anterior ethmoid and frontal sinuses. The patient underwent endoscopic sinus surgery. The dual imaging evaluation of the patient aided the preoperative differential diagnosis and choosing the surgical approach.

KEYWORDS: signal void, pseudo-pneumatized sinus, fungal rhinosinusitis.

INTRODUCTION

Signal void is a term used to describe absolute low signal intensity (black appearance) on magnetic resonance imaging sequences. Signal void appearance can be produced by air-filled cavities, secretions with various viscosity, protein concentration or binding of different metals. Signal void (or flow void in this case) can also be observed in fluids (blood, cerebrospinal fluid) moving with a high enough velocity to escape the imaging plane before the previously absorbed radiofrequency energy can be emitted back1.

In the paranasal sinuses, signal void MRI appearance is frequently attributed to fungal rhinosinusitis, case in which a pseudo-pneumatized sinus aspect may be encountered, leading to diagnostic errors2,3. In the course of the disease, fungal rhinosinusitis tends to desiccate, accumulate macromolecular proteins and include iron, calcium, magnesium, manganese and other heavy metals, all contributing to a progressive reduction in signal intensity4,5. Depending on the protein content, sinus secretions can vary in signal intensity. T2 weighted signal progressively decreases from hyperintense as protein content increases, becoming hypointense when protein content reaches 25-30% (Figure 1). T1 weighted signal has a curved distribution, with maximum intensity around 25% protein content and decreasing as it deviates from this value6,7. Other causes of signal void in the extracranial head and neck region include large neoformation vessels within tumours8.

CASE REPORT

A 75-year-old female patient was referred to our clinic with persistent and complete right-sided nasal obstruction, which developed over the course of a year. The patient’s history revealed only cardiovascular comorbidities such as hypertension, ischemic heart disease, NYHA II heart failure, recurrent angor pectoris and use of related medication. No environmental or genetic risk factors were evident.

Clinical and endoscopic examination of the right nostril revealed medialization of the intersinusonasal wall with near-total collapse of the inferior meatus, contact between the inferior turbinate and the nasal
septum and a polypoid growth in the middle meatus. No discharge or apparent necrotic mucosal areas were seen (Figure 2).

The thoracic radioscopy showed no progressive lesions. Preoperative blood tests showed minimally increased GPT, GOT and serum glucose, with a normal blood count and no acute inflammatory markers elevation (CRP, ESR, and fibrinogen). Middle meatus sampling returned negative for both bacteria and fungi.

Imaging included paranasal sinus CT scan and contrast-enhanced head MRI (Figures 3-7).

**Figure 1** MRI T1W and T2W signal depending on protein concentration in paranasal sinus secretion.

**Figure 2** Right nostril rigid endoscopy.

**Figure 3** Native CT scan shows complete opacification of the right-sided maxillary, anterior ethmoid and frontal sinuses and near-total obstruction of the ipsilateral nostril. The lateral nasal wall is eroded by the expansive process which comes in contact with the nasal septum.

**Figure 4** Axial CT scan sections show sinus expansion and pressure thinning of the anterior wall (green arrow) and posterior wall (red arrow) of the right maxillary sinus with osseous remodelling and a clear breach seen in the anterior wall. Pterygopalatine fossa contents are compressed by the expanding process.

**Figure 5** Contrast adjustment reveals a compact, homogenous hyperdense content occupying the right maxillary sinus and ipsilateral nasal fossa (red area) and a secondary fluid retention due to ostiomeatal obstruction in the anterior ethmoid and frontal sinuses.
Our differential diagnosis at this stage included benign and malignant sinonasal tumours and fungal rhinosinusitis.

The dual imaging evaluation strongly suggested fungal rhinosinusitis, and the patient was proposed for endoscopic sinus surgery (Figures 8 – 11).

Operating time was approx. 90 minutes. No significant bleeding was present. The patient had the right nostril lightly packed for 24 hours and was discharged the following day after packing removal.

Two separate specimens were subjected to histopathological examination:

1. Maxillary sinus content revealed allergic mucin with focal calcifications, without hyphae or Charcot-Leyden crystals.
2. Maxillary sinus mucosa revealed a chronic lymphoplasmacytic inflammatory infiltrate, necrotic areas with epithelioid histiocytes, giant multinucleated cells and epithelioid granulomas, which suggested granulomatous chronic rhinosinusitis. No fungal hyphae were observed.
Figure 9 Surgery is carried out with removal of large quantities of clay-like, greyish amorphous material, which detached in the form of sinus walls molds.

Figure 10 The remaining right lateral nasal wall is observed, completely devoid of any bony structure due to pressure resorption from the expanding pathology. Fungal material is still present.

Figure 11 After complete removal of the amorphous material. A large opening between the right maxillary sinus and the nasal cavity is seen bordered by the remnants of the lateral nasal wall (top). Right maxillary sinus view using 70° angled endoscope shows areas of denuded bone and the aspect of the remaining mucosa (bottom).
DISCUSSIONS

While the intraoperative macroscopic aspect has a reported positive predictive value of up to 100% for the diagnostic of paranasal fungus ball\(^9\), differential diagnosis remains difficult in this particular case. The most inconvenient result was the absence of hyphae both in the maxillary sinus content and in the sinusal mucosa. Although necrotic mucosal areas and mucosal granulomas were observed, the absence of intramucosal fungi and patient immunocompetence refutes invasive rhinosinusitis diagnosis. Besides the presence of allergic mucin – which is not unique to allergic fungal rhinosinusitis -, the other diagnostic criteria for allergic fungal rhinosinusitis have not been met (hyphae, Charcot-Leyden crystals, elevated serum IgE). It has been shown that fungal cultures do not contribute to a correct paranasal sinus fungus ball diagnosis, and that a severe inflammatory reaction of surrounding tissues can accompany fungus balls\(^{10}\). Furthermore, routine H&E stains can yield false-negative results as hyphae can appear similar to allergic mucin or necrosis especially on low magnification\(^{11}\). Until further information is acquired, the patient is managed as having a non-invasive and non-allergic chronic fungal rhinosinusitis – no antifungals or systemic corticosteroids were prescribed.

CONCLUSIONS

The CT scan of the paranasal sinuses clearly shows the sinuses involved by the pathology, but may not always differentiate between fungal, other chronic rhinosinusitis and tumours. The MRI more clearly differentiates between fungal rhinosinusitis, other types of chronic rhinosinusitis and masses, but may present the pitfall of the pseudo-pneumatized sinus. Corroborating both imaging techniques helps preoperative diagnosis and surgical planning.

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Contribution of authors: All authors have equally contributed to this work.

REFERENCES