Computed Tomographic and Magnetic Resonance Imaging Findings in Paranasal Sinus Involvement in Nasopharyngeal Carcinoma

V F H Chong,*, FAMS, MBBS, FRCR, Y F Fan,*, FAMS, MBBS, FRCR, J B K Khoo,*, FAMS, MBBS, FRCR

Abstract

Nasopharyngeal carcinoma (NPC) may spread to the paranasal sinuses. This retrospective study describes the features of paranasal sinus involvement in NPC on computed tomography (CT) and magnetic resonance imaging (MRI). One hundred and fourteen patients with histologically proven NPC underwent staging with both CT and MRI. Maxillary sinus infiltration was demonstrated on MRI in 10 patients; sphenoid sinus infiltration in 24 patients; and, ethmoid sinus involvement in 4 patients. CT could separate inflammatory changes from tumour in all maxillary sinuses but is less helpful in the sphenoid and ethmoid sinuses. Contrast-enhanced MRI could differentiate tumour from inflammatory changes in all sinuses. Using MRI as the standard, the rates of CT separating tumour from inflammation are: maxillary sinus (100%), sphenoid sinus (43%) and ethmoid sinus (25%). Histological confirmation of tumour involvement in the paranasal sinuses is not available. It is important to separate sinusitis from tumour infiltration as prognosis and treatment planning may be affected.

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Introduction

Nasopharyngeal carcinoma (NPC) is an aggressive infiltrative neoplasm. Spread into the paranasal sinuses is often seen but the frequency is documented in only a few series.¹ ² Involvement of the bony structures below the skull base is included in the staging of NPC implying prognostic and therapeutic significance. Ho's system classifies bone involvement below the skull base and the floor of the sphenoid sinus as stage T3a.³ The new American Joint Committee on Cancer (AJCC) and International Union Against Cancer (UICC) staging system classifies tumour invading bone as stage T3. Hence, erosion of sinus wall implies stage T3 disease.⁴ In many instances, inflammatory changes are superimposed on the neoplastic process. It is important to differentiate these entities and define the inflammation-tumour border. Inaccurate tumour mapping such as mistaking inflammatory changes in the ethmoid sinuses for tumour extension may result in potential loss of vision when radiation therapy is administered. This paper describes the magnetic resonance imaging (MRI) and computed tomography (CT) findings in patients with neoplastic infiltration of the paranasal sinuses.

Material and Methods

One hundred and fourteen consecutive patients with histopathologically proven NPC were examined with MRI and CT. Spin-echo (SE) technique was used in the axial coronal and sagittal planes. Axial T1-weighted [TR/TE 700/15; repetition time (TR) msec/echo time (TE) msec] and T2-weighted (TR/TE 2730/80) images were obtained. T1-weighted images were also performed in the coronal plane. Axial, coronal and sagittal sections were performed following the injection of gadopentate dimeglumine (magnevist, 0.1 mmol/kg body weight). Slice thickness are as follows: Axial and coronal images, 5 mm sections with 2 mm interslice gap; and sagittal images, 4 mm sections with 1 mm gap. T1-weighted images were performed with two excitations while T2-weighted images were acquired with a single excitation. The imaging matrix was 192 x 256 in all scanning planes and sequences. Axial and coronal 5 mm section CT was performed following the injection of contrast 50 ml of contrast (Iopromide, 370 mg iodine m/L, Ultravist 370). Axial sections on CT and MRI were performed from a point between the vertex and the dorsum sellae to the C7. Coronal images were acquired from the frontal sinus to the posterior margin of the foramen magnum.

The CT and MRI of all patients were retrospectively reviewed for tumour infiltration of the paranasal sinus. The reading was done independently by two radiologists and differences resolved by consensus. CT images
were viewed at both soft tissue and bone settings. A lesion within a sinus is considered a tumour if it could be seen to be in contiguity with the primary tumour in the nasopharynx. On CT, the affected sinuses were examined to see if tumour could be separated from inflammatory changes on the basis of density. On MRI, the separation of tumour-inflammation margins on T1, T2-weighted and contrast-enhanced images were analysed.

**Results**

The frequency of tumour infiltration of the paranasal sinuses is summarised in Table I. The frequencies shown are based on both MRI and CT detection rates. There were 31 patients with paranasal sinus infiltration. Three (10%) patients had sphenoid-ethmoid sinus infiltration and 7 (23%) showed sphenoid-maxillary sinus infiltration. No patients had a combination of maxillary and ethmoid sinus involvement. The ability to distinguish the tumour-inflammation boundary by MRI sequences and CT in the paranasal sinuses is summarised in Table II.

**Maxillary Sinus**

Tumour involvement of 11 maxillary sinuses in 10 (9%) patients was demonstrated. Thickened mucosa could be seen in association with tumour infiltration in only four sinuses. In these four patients, CT could differentiate thickened mucosa that appears lower in density compared to tumour which is higher in attenuation (Fig. 1). Tumour-mucosa separation on T1-weighted MRI was also possible in all cases in this study as thickened mucosa is lower in signal intensity. This is further aided by contrast-enhanced and T2-weighted images as differentiation of these conditions are well demonstrated in all cases. Using MRI as the standard, the ability of CT separating tumour infiltration from inflammation of the maxillary sinus is 100%.

**Ethmoid Sinus**

The ethmoid sinus is infiltrated by tumour infrequently. Separating tumour from inflammation on CT cannot be accomplished with confidence in all affected sinuses except one (Fig. 2). There is no great difference in the attenuation value of both tumour and thickened mucosa. On T1-weighted MRI, differentiation between tumour and inflammation could not be done in two sinuses. T2-weighted images could detect the boundary between tumour and inflammation/retained secretions in three sinuses. Contrast-enhanced images could distinguish the two entities in all four sinuses with tumour and superimposed inflammation. There was only one sinus with tumour infiltration not associated with inflammatory changes. The pattern of tumour enhancement on MRI is different from inflamed mucosa (Fig. 2). Tumour shows solid enhancement while mucosa maintains the “pencil-outline” pattern. On T2-weighted images, tumour shows appreciably lower signals compared to thickened mucosa or secretions. Using MRI as the standard, the ability of CT separating tumour infiltration from inflammation of the ethmoid sinus is 25%.

**Sphenoid Sinus**

The frequency of sphenoid sinus tumour infiltration is much higher than maxillary or ethmoid sinus involvement. Twenty-nine sphenoid sinuses were infiltrated. In eight (26%) sinuses tumour extension into the sphenoid sinus was not associated with inflammatory changes. On CT, thickened mucosa/retained secretions could be separated from tumour in nine of the 21 sinuses (43%) with concomitant sinusitis. In the remaining 12 (57%) sinuses, there was no perceptible difference in the attenuation value of the tumour and inflammatory tissue (Fig. 3). In these patients, how much of the opacified sinus is due to tumour or inflammation could only be determined on MRI.

The tumour-inflammation boundary can be appreciated in 9 of the 21 (43%) involved sinuses on T1-weighted MRI. In all patients, contrast-enhanced MRI could demonstrate the tumour/inflammation boundary. In contrast-enhanced MRI, tumour showed solid enhancement while the typical enhancement pattern of inflamed mucosa could be seen. However, the mucosa adjacent to tumour may show considerably more enhancement compared to elsewhere suggesting possible invasion of the inflamed submucosa (Fig. 4). In T2-weighted images, high signal intensity fluid and mucosa may be seen adjacent to relatively lower signal intensity tumour. T2-weighted images could detect the tumour-inflammation interface in 20 of the 21 sinuses. In six patients, there was

### Table I: Frequency of Tumour Involvement of the Paranasal Sinuses

<table>
<thead>
<tr>
<th>Sinus</th>
<th>Number of patients</th>
<th>Unilateral involvement</th>
<th>Bilateral involvement</th>
<th>Total number of sinuses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maxillary</td>
<td>10 (9%)</td>
<td>9</td>
<td>1</td>
<td>11</td>
</tr>
<tr>
<td>Sphenoid</td>
<td>24 (21%)</td>
<td>19</td>
<td>5</td>
<td>29</td>
</tr>
<tr>
<td>Ethmoid</td>
<td>4 (4%)</td>
<td>3</td>
<td>1</td>
<td>5</td>
</tr>
</tbody>
</table>

### Table II: Tumour-Inflammation Separation by MRI and CT

<table>
<thead>
<tr>
<th>MRI Sequences</th>
<th>Maxillary sinus n* = 4</th>
<th>Sphenoid sinus n* = 21</th>
<th>Ethmoid sinus n* = 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1-weighted</td>
<td>4</td>
<td>9</td>
<td>2</td>
</tr>
<tr>
<td>T2-weighted</td>
<td>4</td>
<td>20</td>
<td>3</td>
</tr>
<tr>
<td>Contrast-enhanced</td>
<td>4</td>
<td>21</td>
<td>4</td>
</tr>
<tr>
<td>CT</td>
<td>4</td>
<td>9</td>
<td>1</td>
</tr>
</tbody>
</table>

* n = number of sinuses with tumour and superimposed inflammatory changes.
Fig. 1a. Axial contrast-enhanced CT shows tumour (large asterisk) destroying the right pterygoid process with infiltration of the maxillary sinus (arrow). The tumour is denser than thickened mucosa (small asterisks).

Fig. 1b. Axial contrast-enhanced MRI shows enhanced tumour invading the right maxillary sinus and clivus. Inflamed mucosa shows only a superficial rim of enhancement (arrows).

Fig. 1c. Axial T2-weighted MRI shows good differentiation between high signal intensity mucosa (asterisks) and lower signal intensity tumour (star). Note bilateral serous mastoiditis (S) and high signal tumour infiltration of clivus (arrow).

Fig. 2a. Axial contrast-enhanced CT shows opacification of the right ethmoid sinus. The lateral ethmoid air-cells (arrowheads) show a lower density compared to the medial air-cells (E).

Fig. 2b. Axial contrast-enhanced MRI shows low signal intensity fluid within the lateral ethmoid air-cells (arrowheads). Enhanced tumour infiltrating the medial cells noted (E).

Fig. 3a. Axial contrast-enhanced MRI shows tumour enhancement in the sphenoid sinus (arrow). Note the adjacent retained secretions (S).

Fig. 3b. Axial T2-weighted MRI shows high signal intensity retained secretions outlining intermediate signal tumour. The area of relatively low signals in the dependent right sphenoid sinus could be debris.

Further tumour extension into the cavernous sinus or suprasellar region. Using MRI as the standard, the ability of CT separating tumour infiltration from inflammation of the sphenoid sinus is 43%.

Discussion

One of the main challenges faced by the radiologist in the head and neck region is accurate mapping of tumour extent. It is important to identify malignant infiltration of the paranasal sinuses because of its prognostic significance. Although the findings in this study are based on NPC, we believe they are also applicable to other malignancies with paranasal sinus extension or tumour originating within the sinuses themselves. Accurate tumour delineation has a bearing on whether a patient is a potential surgical candidate. Radiation portals may depend on the demonstrated tumour extent.

CT is the established modality in the staging of NPC. It is still by far the most commonly employed modality in staging NPC. Tumour extension into the paranasal
sinuses is often superimposed on a background of sinusitis or as a result of tumour obstructing the drainage pathway. The separation of inflammatory changes from tumour infiltration is often difficult on CT as soft tissue contrast resolution is relatively limited. This diagnostic problem is encountered in the ethmoid and sphenoid sinuses but not in the maxillary sinus. There is often considerable overlap of attenuation values of these tissues in ethmoid and sphenoid sinuses making it difficult to confidently identify the tumour margins. The reason for this observation is unknown. In the ethmoid and sphenoid sinuses, sinus ostia obstruction by tumour leads to retained secretions getting progressively dehydrated. This results in the secretions showing a high attenuation which cannot be separated from tumour.

The frequency of malignant infiltration of the ethmoid sinus based on MRI findings in our study was only 4%. This contrasts with the reported incidence of 18% diagnosed on CT by Sham et al.\(^2\) This disparity may quite possibly be due to the difficulty encountered by CT in separating inflammatory changes from tumour involvement. It is also possible that different diagnostic criteria were used. Unfortunately, in Sham’s paper, no diagnostic criteria on ethmoid sinus involvement were given.

Establishing erosion of the sphenoid sinus floor on CT is usually a straightforward exercise except in early cases where the floor meets the anterior or posterior walls. These areas are much better assessed by MRI in the sagittal plane (Fig. 5). In general, CT is superior to MRI in visualising directly the erosion of the thin sphenoid sinus floor. This is in contrast to the superiority of MRI in delineating infiltration of the marrow containing bone in the skull base.\(^6\)

Although CT is adequate in identifying neoplastic extension into the sphenoid sinus as indicated by erosion of the floor, it often overestimates the degree of involvement. Superimposed sinusitis is difficult to separate from tumour. The tumour component may be quite small in comparison with the entire opacified sinus. Tumour extent is much better identified on T2-weighted or contrast-enhanced MRI. Intracranial extension following sphenoid sinus infiltration either laterally into the cavernous sinus or superiorly into the suprasellar space can be identified in 5% of the patients at initial presentation.\(^7\) Although cavernous sinus involvement can be detected on CT, the entire process is much more elegantly displayed on contrast-enhanced MRI.

Som et al.\(^8\) reported the clear superiority of T2-weighted images in separating tumour from inflammatory tissues in 95% of their patients. In general, tumours show a...
lower signal intensity compared to inflammatory tissues providing a method for identifying tumour boundaries. They noted no distinguishing features between T1-weighted images and inflammatory tissues. This is in contrast to our observations in the maxillary sinus where clear demarcation could be seen between inflamed tissues and the infiltrating NPC. However, the types of tumours reported in their series are different from ours and they could explain the observed differences. The situation is different in the sphenoid and ethmoid sinuses where the majority of tumours cannot be differentiated from the adjacent inflammatory tissue on T1-weighted images. In our study, T2-weighted images have a definite edge over T1-weighted scans in separating the two entities. In the study by Som et al, no contrast-enhanced examinations were performed.

The use of contrast-enhanced MRI for differentiating neoplasms from mucocoeles was reported by Lanzieri et al. They noted that signal intensities of both tumours and mucocoeles vary widely. However, T2-weighted images were often sufficient to differentiate the above entities but in a significant majority of cases, such separation cannot be made with confidence without the injection of contrast material. In general, we found T2-weighted images sufficient for separating neoplasm and inflammation in all paranasal sinuses with exception of single cases in the sphenoid and ethmoid sinuses. However, anatomical details were much better demonstrated on contrast-enhanced images. In addition, intracranial tumour extension from the sphenoid sinuses was better delineated on contrast-enhanced MRI.

CT is adequate in separating tumour from inflammation in the maxillary sinus. Erosion of the thin sinus wall is, as expected, confidently recognised. In the ethmoid and sphenoid sinuses, CT is less capable than MRI in distinguishing tumour from inflammatory changes. T2-weighted images are excellent in differentiating high signal intensity thickened mucosa or retained secretions from lower signal intensity tumour. In contrast-enhanced MRI, tumour enhancement can be differentiated from thickened mucosa with a thin superficial enhancement. Full-thickness irregularly thickened mucosa may be potentially confused with tumour infiltration. However, in general, full-thickness mucosal enhancement is more intense than tumour enhancement seen in this study. Destruction of the ethmoid sinus septation seen on CT may help to separate inflammatory from neoplastic involvement. However, as ethmoid sinus septa are very thin structures, they can also be demineralised in inflammatory processes. Furthermore increased sinus pressures as a result of retained secretions can also destroy the septations.

The major weakness of this study is a lack of pathologic or surgical correlation of tumour within the involved sinuses. Pathological proof of tumour in the nasopharynx is easily obtained via endoscopic biopsy but access to tissue in the paranasal sinuses is often difficult. A substantial amount of tumour may have to be traversed before the biopsy needle can reach the involved area as in the case of the sphenoid sinus. The exact tip of the biopsy instrument may be technically difficult to verify and time consuming especially when multiple biopsies are considered. As the mainstay of NPC treatment is radiation therapy, surgical verification is, as a general rule, not available. To overcome this problem of pathological proof, we have strictly adhered to the criterion that tumour must be seen in contiguity with the primary lesion and share the same signal intensity characteristics before making a positive diagnosis.

In summary, MRI is the preferred modality in evaluating possible tumour infiltration of sinuses with the exception of the maxillary sinus where CT is equivalent. T2-weighted MRI is usually sufficient for tumour mapping but contrast-enhanced MRI provides better anatomical details especially in patients with intracranial extension from the sphenoid sinus.

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REFERENCES