Imaging of nasopharyngeal inflammatory pseudotumours: differential from nasopharyngeal carcinoma

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ABSTRACT. The purpose of this study was to describe the MRI findings of inflammatory pseudotumours (IPTs) involving the nasopharynx and to differentiate IPTs from nasopharyngeal carcinoma (NPC). The medical records and imaging studies of 7 patients (6 men, 1 woman; age range, 32–71 years; mean age, 52.4 ± 18 years) with IPT involving the nasopharynx were reviewed retrospectively. The MRI findings were compared with those of seven patients with advanced NPC with skull base invasion. All patients with IPT involving the nasopharynx presented with single or multiple cranial neuropathies; six reported pain; and four patients had a recent history of otitis media. Three were initially misdiagnosed as having NPC according to MRI findings, and all underwent nasopharyngeal biopsy to exclude malignancy. 7/7 (100%) patients received systemic corticosteroid treatment; 7/7 (100%) showed initial rapid resolution of clinical symptoms or radiographical findings; 3/7 (42.9%) had resolution of all signs and symptoms; 3/7 (42.9%) still have limited residual symptoms; and 1/7 (14.3%) suffered recurrence 3 years after remission. The characteristic MR findings of IPT include an infiltrative growth pattern, minimal to mild mass effect, hypointensity on T₂ weighted images, and moderate homogeneous enhancement after contrast administration. Intact nasopharyngeal mucosa, internal carotid artery encasement and narrowing, extensive pachymeningeal thickening and a relative paucity of associated neck lymphadenopathy are additional MR findings that favour the diagnosis of IPT rather than NPC. In conclusion, IPT involving the nasopharynx has characteristic MR findings which, together with clinical and laboratory presentations, are helpful in differentiating IPT from malignant tumours, especially NPC.

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Inflammatory pseudotumour (IPT) is an idiopathic inflammatory lesion characterised by the proliferation of myofibroblastic spindle cells with mixed inflammatory infiltrates of plasma cells, lymphocytes, eosinophils and histiocytes [1]. IPT has been described in various locations in the body including the lungs, lymph nodes, trachea, spleen, mesentery and skin [2–8]. IPT of the head and neck region most commonly occurs in the orbits [9, 10], although IPT of the larynx, paranasal sinuses, parapharyngeal space and salivary glands [11–15] has also been reported. It is known as Tolosa–Hunt syndrome when IPT involves the cavernous sinus and causes painful ophthalmoplegia [16].

IPT occurring in the nasopharynx and the skull base is less frequently reported, and could be a primary lesion or extension of orbital disease. It has a propensity for locally aggressive growth, frequently with bony erosion and cranial neuropathy, which are clinically and radiographically indistinguishable from invasive neoplasm [17–19]. The diagnosis of IPT is usually clinical, but the combination of blood tests, radiographic studies and biopsies can be helpful [9, 20]. The relative inaccessibility

of the skull base when obtaining biopsy specimens often accentuates the diagnostic dilemma. Understanding the presentation and making a more accurate differentiation between IPT and aggressive tumour through MRI is important, because they require completely different treatment strategies.

The purpose of our study was to describe the MR findings in seven patients with IPT involving the nasopharynx and skull base, and to discuss their characteristic imaging findings, associated clinical presentations and differentiation from aggressive malignant tumours.

Methods and materials

Patients

We reviewed retrospectively the medical records and imaging studies of seven patients with IPT involving the nasopharynx encountered in our practice in the past four years. The diagnoses of IPT were made on the basis of biopsy and clinical response to systemic corticosteroids. The demographic and clinical data of these patients are summarised in Table 1. The patients were 6 men and 1 woman who ranged in age from 32 years to 71 years

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(mean, 52.4 years). The clinical presentation, method of biopsy, treatment modality and treatment response were reviewed for each patient. Follow-up MRI studies were obtained in five of the seven patients (Cases 1, 2, 4, 5 and 7).

As controls for the study of IPT involving the nasopharynx and skull base, we selected seven cases of pathologically proven nasopharyngeal carcinoma (NPC) with skull-base and intracranial involvement encountered in our practice in the past two months. The medical records and imaging studies were reviewed for comparison.

Imaging

MRI studies were performed on a 1.5-T magnetic unit (Signa; GE Healthcare, Milwaukee, WI; Magnetom Vision or Sonata; Siemens Medical Solutions, Erlangen, Germany) with a dedicated head coil. Our imaging protocol included axial T_1 weighted spin-echo, T_2 weighted spin-echo with or without fat saturation, and coronal and sagittal T_1 weighted spin-echo sequences. All patients had intravenous administration of gadodiamide (gadolinium diethylenetriamine pentaacetic acid bismethylamide (GdDTPA-BMA)) (Omniscan, GE Healthcare, Cork, Ireland) at a dose of 0.1 mmol kg^{-1} . Subsequently, axial and coronal T_1 weighted spin-echo sequences with or without fat saturation were obtained.

The MR images were evaluated for margins, homogeneity and signal intensity of affected areas. Contrast-enhanced MR images were compared with the unenhanced images. All images were evaluated for the precise location and extent of affected areas. Attention was also paid to the presence of mastoiditis, neck lymphadenopathy, vascular encasement and narrowing, as well as the presence and extent of intracranial invasion. The MRI findings of seven cases of IPT and seven cases of locally advanced NPC are compared.

Results

Clinical features of IPT

The demographic and clinical data of the patients with IPT involving the nasopharynx are summarised in Table 1. Four patients had a recent history of otitis media (Cases 1–4). All patients presented initially with either single or multiple cranial nerve (CN) neuropathies, including diplopia, visual loss, facial numbness, facial palsy, tinnitus, dysphagia, hoarseness and hemitongue atrophy. Four patients had single CN neuropathy (Case 1, CN VIII; Case 3, CN V; Cases 5 and 6, CNXII), and three patients had multiple CN neuropathies (Cases 2: CN II, III, IV, V, VI, VII, VIII; Case 4: CN II, III, V, VII, IX, X, XII; Case 7: CN II, V, VI, VII, XI). The trigeminal nerve was the most commonly involved CN ($n=4/7$). Six patients complained of pain other than that of the cranial neuropathies.

After the MRI study, three lesions were initially suspected to be NPC; the other four lesions were adjacent to the nasopharynx and skull base, and so histological examination was indicated. Tissue biopsy was obtained in all seven patients, nasopharyngeal punch biopsy in only five patients, and both nasopharyngeal punch biopsy and

CT-guided biopsy in two patients. The histological results were consistent with non-specific inflammatory cell infiltration and fibrotic change, without evidence of malignancy.

All seven patients received steroid treatment. Three patients (Cases 2, 3 and 4) received pulse therapy with intravenous high-dose Solu-medrol (methylprednisolone sodium succinate) followed by high-dose prednisolone, while four patients (Cases 1, 5, 6 and 7) received high-dose oral prednisolone only. All patients showed good response to steroid treatment, as evidenced by rapid relief of clinical symptoms or resolution of the lesion on MRI. Three patients (Cases 3, 5 and 6) reported complete resolution of clinical symptoms after treatment, and are free of disease at recent follow-up. Three patients (Cases 1, 2 and 4) showed improvement both clinically and radiologically, but still suffered from mild residual pain eight months, one month and 11 months after treatment, respectively. One patient (Case 7) responded to the treatment initially with an improvement of symptoms, but suffered from recurrence of IPT three years later. This patient was lost to follow-up after recurrence, and so the current disease status is unknown.

Radiologic features of IPT

The imaging findings of IPT involving the nasopharynx in these seven patients are listed in Table 2. All lesions showed an ill-defined infiltrative border without a distinct margin, and all cases showed minimal or mild mass effect. The soft-tissue mass lesions were slightly hypointense to isointense relative to the brainstem at the same level on T_1 weighted images, were hypointense relative to the brainstem on T_2 weighted images, and showed moderate homogeneous enhancement on contrast-enhanced T_1 weighted images (Figures 1–3). All of the lesions involved the nasopharynx but were sub-mucosal, with variable bulging of the nasopharyngeal contour. The overlying nasopharyngeal mucosa was not significantly thickened, and the hyperintense nasopharyngeal mucosa could be delineated from the hypointense lesion on T_2 weighted images (Figures 1–3). The lesions infiltrated the adjacent structures and skull base to different extents. Adjacent structures that were involved included the Eustachian tube (E-tube), carotid space, parapharyngeal space, clivus, occipital condyle, jugular fossa, hypoglossal canal, Meckel's cave, foramen ovale, orbital apex, infratemporal fossa and cavernous sinus. The involved skull base showed loss of normal fatty marrow with mild enhancement (Figure 3).

All of the lesions had encased the internal carotid artery in the upper cervical segment or the petrous or cavernous segments, and six lesions had caused narrowing of the carotid artery (Figures 2 and 3). Carotid artery narrowing was defined as more than a 20% reduction in the vessel diameter on T_2 weighted images when compared with the adjacent non-involved segment.

All patients had ipsilateral mastoid effusion. Three of the seven patients (Cases 2, 4 and 7) had a wide area of pachymeningeal thickening that enhanced after administration of contrast (Figure 1). All patients were screened for neck lymphadenopathy (LAP) except for one patient (Case 1), in whom the scanning coverage was

Table 1. Demographics and clinical findings in seven patients with inflammatory pseudotumours of the skull base

Case	Age (years)/sex	Presenting symptoms	Cranial nerve neuropathies	Method of tissue proof/pathology	Treatment	Response to treatment	Follow-up MRI	Current status
1	66/F	OM, tinnitus, otalgia	VIII	NP bx/lymphoid hyperplasia; CT-guided bx/fibrosis	Oral CS	Yes	Yes	9 months; improved but residual pain
2	32/M	OM, ptosis, diplopia, trigeminal neuralgia, facial palsy	II, III, IV, V, VI, VII, VIII	NP bx/chronic inflammation	Pulse steroid;oral CS	Yes	Yes	8 months; improved but residual pain
3	71/M	OM, trigeminal neuralgia	V	NP bx/lymphoid hyperplasia	Pulse steroid; oral CS	Yes	No	5 months; NED
4	70/M	OM, dysphagia, hoarseness, orbital pain	II, III, V, VII, IX, X, XII	NP bx/chronic inflammation	Pulse steroid; oral CS	Yes	Yes	1 month; improved but residual pain
5	32/M	Hemicrania pain, hemi tongue atrophy	XII	NP bx/chronic inflammation; CT-guided bx/lymphoplasmacytic infiltration	Oral CS	Yes	Yes	7 months; NED
6	37/M	Hemicrania pain, hoarseness	XII	NP bx/lymphoid hyperplasia; LN bx/reactive hyperplasia	Oral CS	Yes	No	8 months; NED
7	59/M	Visual loss	II, V-1, VI, VII, IX	NP bx/chronic inflammation	Oral CS	Yes	Yes	Initial improvement but retrobulbar recurrence 3 years later, lost follow-up.

M, male; F, female; OM, otitis media; NP, nasopharynx; CS, corticosteroid; NED, no evidence of disease; Bx. biopsy

Table 2. Lesion location and associated findings in seven patients with inflammatory pseudotumours of the skull base

Case	Side	Lesion location	Associated findings					
			NP mucosal thickening	ICA encasement	ICA narrowing	Extensive pachymeningeal enhancement	Lymphadenopathy	Mastoid effusion
1	Left	NP, CS, E-tube, tympanic cavity	No	Yes	Yes	No	Not screened	Yes
2	Right	NP, PPS, CS, Meckel's cave, E-tube, SOF, cavernous sinus	No	Yes	Yes	Yes	No	Yes
3	Right	NP, CS, clivus, occipital condyle, pterygoid process	No	Yes	Yes	No	No	Yes
4	Bilateral	NP, CS, hypoglossal canal, jugular fossa	No	Yes	No	Yes	No	Yes
5	Left	NP, PPS, CS, E-tube, clivus, hypoglossal canal, occipital condyle	No	Yes	Yes	No	No	Yes
6	Right	NP, CS, clivus, occipital condyle, hypoglossal canal, jugular fossa	No	Yes	Yes	No	Yes	Yes
7	Right	NP, PPS, CS, lateral pterygoid muscle, clivus, pteryoid plate, cavernous sinus, orbital apex, optic nerve	No	Yes	Yes	Yes	No	Yes

NP, nasopharynx; PPS, parapharyngeal space; CS, carotid space; E-tube, Eustachian tube; SOF, superior orbital fissure; ICA, internal carotid artery

limited. Only one patient (Case 6) presented with neck LAP, an excisional biopsy of which showed reactive hyperplasia.

Comparison of radiologic features of IPT and NPC

A comparison of the radiological features of IPT and NPC is summarised in Table 3. The most striking feature that differed between IPT and NPC was that all IPT lesions ($n=7/7$, 100%) showed hypointensity on T_2 weighted images when compared with the brainstem, and six of NPC lesions ($n=6/7$, 86%) showed mild hyperintensity on T_2 weighted images (Figure 4). The remaining NPC lesion showed isointensity on T_2 weighted images.

With regard to the location of the lesions, IPT was located submucosally in the nasopharynx with direct extension to the adjacent carotid space, without abnormal thickening of the nasopharyngeal mucosa. In contrast, all cases with NPC presented with lesions arising from the nasopharyngeal mucosa, and a mass lesion contiguous with thickened well-enhancing mucosa was observed on MRI. Mass effect, as manifested by nasopharyngeal bulging and displacement of adjacent muscles and the fat plane, was more obvious in patients with NPC (Figure 4).

Internal carotid artery encasement and narrowing were more common in IPT (encasement $n=7/7$, 100%; narrowing $n=6/7$, 86%) than in NPC (encasement $n=4/7$, 57%; narrowing $n=1/7$, 14%). Extensive pachymeningeal thickening was seen only in cases of IPT ($n=3/7$, 43%) and was not present in cases of NPC. LAP was also

a remarkable feature that was more prevalent in NPC. Only one patient ($n=1/7$, 14.3%) with IPT showed evidence of LAP, but 85.7% ($n=6/7$) of patients with advanced NPC had enlarged LAPs (Figure 4).

Discussion

Clinically, an IPT involving the nasopharynx and skull base causes symptoms, including CN neuropathies, pain and hypopituitarism, as the mass grows and compresses the surrounding tissues [17, 19, 21, 22]. The disease may also present with features suggestive of malignancy, such as disease recurrence, bony erosion/destruction or infiltration of surrounding tissues [19, 23–25]. It can be difficult to distinguish between IPT and aggressive malignancy in most cases, because they may have similar clinical or radiographical appearances.

IPT is an idiopathic inflammatory lesion, the aetiology of which remains unclear. In a review article, Mangiardi and Har-El [20] summarised three current theories. Firstly, and most likely, IPT is an autoimmune reaction that has been linked to viral infection or sinus infection. Secondly, IPT is an infectious process resulting from sinusitis or syphilis. Thirdly, IPT results from the aberrant production of fibrogenic cytokines, as may be inferred from the pathological findings of fibroproliferative disorders.

We did not find any systemic autoimmune disease or predisposing infection in our patients with IPT. Four of our patients (57.1%) did have a recent history of otitis media prior to the presentation of CN neuropathies; however, because of the anatomical proximity of the

Figure 1. Case 2: a 32-year-old man with right ptosis, trigeminal neuralgia and facial palsy 2 days after an episode of acute otitis media treated with tympanocentesis. (a) Axial T_1 weighted image shows an intermediate signal intensity mass lesion involving the right nasopharyngeal submucosal region (arrows). (b) Axial T_2 weighted image shows low signal intensity change of the lesion (arrows). Note the right mastoiditis change. (c) Coronal contrast-enhanced T_1 weighted image shows extension of the lesion to the right skull base, cavernous sinus and Meckel's cave (black arrows). Enhancement of the thickened pachymeninges is also present (white arrows). (d) Axial T_2 weighted and (e) contrast-enhanced images obtained 2 months after a course of intravenous high-dose pulse solumedrol therapy followed by oral corticosteroid medication show the mass lesion to be significantly reduced in size. The pachymeningeal enhancement has nearly disappeared.

E-tube and skull base, it is probable that an early lesion of IPT impairs the function of the E-tube and results in otitis media, as is true in many cases of NPC with initial presentation of otitis media. Unilateral otitis media with multiple CN palsies may be a warning sign for either IPT or NPC; further characterisation by imaging is necessary to distinguish between IPT and NPC.

Both hyperintensity and hypointensity on T_2 weighted images of the IPT lesion involving the skull base region have been reported in the literature [17–19, 26]. In each of our seven cases, the lesion showed hypointensity compared with the adjacent brainstem on T_2 weighted images. This hypointensity may be explained by a relative lack of both free water and mobile protons within the fibrotic lesions [17]. Hypointensity of IPT lesions on T_2 weighted images serves as an important clue to differentiating IPT from aggressive malignancies.

The differential diagnosis of IPT includes primary malignant tumours of the central skull base (e.g. chordoma and chondrosarcoma), direct invasion from NPC, metastatic malignant tumour, primary skull base

lymphoma and plasmacytoma. Chordoma and chondrosarcoma are usually bulky masses with hyperintensity on T_2 weighted images. Lymphoma and plasmacytoma usually show homogeneously slight hyperintensity on T_2 weighted images, with more obvious mass effect and marked enhancement.

NPC, which is the most common malignancy of the skull base region in Asian populations, is the most important disease to be differentiated from nasopharyngeal IPT. In a prospective study of 262 patients with newly diagnosed NPC, 32.1% of these patients had stage T4 lesions, and 13.0% had CN involvement at presentation [27]. The major dilemma in diagnosing IPT is its overlap with advanced NPC in both clinical presentation and imaging findings with regard to lesion location and extension.

Informative imaging characteristics in the differential diagnosis of IPT and NPC in our study group included:

1. *Signal intensity on T_2 weighted images.* In our study, all seven patients with IPT involving the nasopharynx and skull base showed hypointensity when compared

Figure 2. Case 5: a 32-year-old man with left hemicrania of 5 months' duration and hemitongue atrophy of 1 months' duration. (a) Axial T_1 weighted image and (b) axial T_2 weighted image show an ill-defined hypointense lesion in the left nasopharyngeal submucosal region with extension to the carotid space and skull base. The left internal carotid artery is encased and narrowed (white arrow). Involvement of the hypoglossal canal (black arrow) is evident. The mass lesion is distinguishable from the nasopharyngeal mucosa on T_2 weighted images. (c) Axial contrast-enhanced T_1 weighted image with fat saturation shows moderate enhancement of the left nasopharyngeal lesion, with extension around the left internal carotid artery into the skull base, and involving the prevertebral muscles and hypoglossal canal (black arrow). (d) Axial T_2 weighted and (e) contrast-enhanced T_1 weighted image obtained 2 months after a course of oral corticosteroids show nearly complete resolution of the mass lesion.

with the brainstem on T_2 weighted images, whereas six patients with NPC presented with slight hyperintensity on T_2 weighted images.

2. *IPT involving the nasopharynx is submucosal rather than mucosal.* In the seven patients with IPT, the nasopharyngeal mucosa was not thickened, and the hyperintense mucosal layer could be delineated from the hypointense submucosal IPT lesion on T_2 weighted images. Although NPC could also manifest as a minimal mucosal lesion with early extension to the skull base, NPC usually manifests with focally thickened mucosa with good contrast enhancement.
3. *Internal carotid artery encasement and resultant narrowing are common features in cases of IPT but not NPC.*
4. *Extensive pachymeningeal thickening and enhancement is a feature of IPT and is not seen in NPC.*
5. *Presence of LAP is more commonly encountered in patients with NPC than with IPT.* These imaging features could serve as criteria to help differentiate IPT from aggressive malignancy.

The diagnosis of IPT is usually made clinically, but blood tests (*e.g.* complete blood count (CBC), electrolytes, erythrocyte sedimentation rate (ESR), antinuclear antibody (ANA), anti-double-stranded DNA, anti-neutrophil cytoplasmic antibody (ANCA), angiotensin-converting enzyme, rapid plasma reagin (RPR) and serum protein electrophoresis), radiographical studies and biopsy may be helpful for establishing the diagnosis [20]. The diagnosis of IPT is often difficult in that it cannot be made definitively by current radiological techniques, and fine-needle aspiration or biopsy is often non-specific.

The administration of corticosteroids may be considered a diagnostic trial for orbital IPT. Mombaerts et al [28] concluded that the use of a corticosteroid trial as a diagnostic test for IPT had 78% sensitivity, with a cure rate of 37% and a recurrence rate of 52%. For orbital IPT, the mainstay of therapy is high-dose corticosteroids. Response is observed as quickly as 2–4 days after treatment. Radiation therapy, surgery and chemotherapy are appropriate in cases refractory to steroids [9, 29]. Char and Miller

Figure 3. Case 6: a 37-year-old man with right hemicrania of 2 months' duration. (a) Axial T_2 weighted image shows hypointensity and (b) axial contrast-enhanced T_1 weighted image shows moderate enhancement of the lesion in the right nasopharyngeal submucosa and carotid space (white arrows), which encases the right internal carotid artery with extension to the skull base. There is high signal change on T_2 weighted images and abnormal contrast enhancement in the bone marrow of the right occipital condyle (black arrows). (c) Contrast-enhanced MR angiography shows segmental narrowing of the right upper cervical internal carotid artery (white arrows) owing to the encasement by the carotid space lesion.

[29] showed that high-dose oral corticosteroids were effective in approximately 31% of patients, although a poor steroid response was observed in patients with mass lesions and in those with fibrotic disease. However, one should be cautious when interpreting the results of steroid treatment because malignancies with a large inflammatory component, as well as lymphoproliferative disease, may undergo a reduction in size, giving the false impression of a confirmatory response to the diagnostic trial.

Although there is no large series examining the precise effects of systemic steroid therapy on IPT involving the skull base, it has been observed that systemic steroid therapy may help to stabilise or alleviate pain and CN dysfunction [18, 21, 24]. In a review of literature on orbit-sparing IPT of the skull base, Mangiardi and Har-El [20] observed that IPT in this region may be quite responsive to steroid treatment. Although some authors advocate radiotherapy or surgical resection as the treatment of choice [19, 30, 31], many authors believe that these therapeutic options should be considered in patients who fail to

respond to steroids, or who relapse soon after the treatment [20, 23]. In our study, all seven patients initially had a good rapid response to steroid treatment; three patients (42.9%) were disease-free after treatment, and three patients (42.9%) had marked improvement in clinical symptoms and only mild residual pain. One patient (14.3%) responded rapidly to the treatment initially, but suffered from recurrence three years later. In our experience, systemic steroid treatment provides an overall good outcome without significant complications. Our conclusion that systemic steroids should be the treatment of choice in patients with IPT involving the nasopharynx and skull base region is consistent with the literature.

There are some limitations to our study. Firstly, our study is a retrospective one with an inherent selection bias. The relatively small number of patients selected may make it difficult to generalise all of the MRI findings of IPT involving the nasopharynx and skull base. Nevertheless, we believe that our results merit reporting because the imaging characteristics for distinguishing

Table 3. Comparison of clinical and imaging characters in seven patients with IPT involving the skull base and seven patients with T4 stage NPC

	IPT Mean $\pm$ SD (range) n (%)	NPC Mean $\pm$ SD (range) n (%)
Age (years)	52.4 $\pm$ 18 (32–71) 7 (100)	52.4 $\pm$ 13.6 (34–73) 7 (100)
Clinical presentations		
Cranial neuropathy	7 (100)	4 (57.1)
Pain	6 (85.7)	3 (42.9)
Image characteristics		
Hypointensity on T_2 weighted images	7 (100)	0 (0)
Nasopharyngeal mucosa thickening	0 (0)	7 (100)
Internal carotid artery narrowing	6 (85.7)	1 (14.3)
Extensive pachymeningeal thickening	3 (42.9)	0 (0)
Presence of lymphadenopathy	1 (14.3)	6 (85.7)

NPC, nasopharyngeal carcinoma; IPT, inflammatory pseudotumour.

Figure 4. A case of locally advanced nasopharyngeal carcinoma in a 44-year-old man with right-ear tinnitus and hearing impairment of 1 year's duration. (a) Axial T_1 weighted image shows a bulging right nasopharyngeal mass with extension to the skull base, partially encasing the right internal carotid artery. The right parapharyngeal fat is obliterated. (b) Axial T_2 weighted image shows mild hyperintensity of the right nasopharyngeal mass. The signal intensity of the mass is similar to that of the nasopharyngeal mucosa, and the margin between the normal mucosa and the mass could not be delineated. Note the presence of right mastoiditis change. (c) Axial and (d) coronal contrast-enhanced T_1 weighted images with fat saturation shows vivid enhancement of the nasopharyngeal mass. There is skull-base and cavernous sinus invasion with encasement of the right internal carotid artery, which is not narrowed (white arrows). Bilateral neck lymphadenopathy (black arrows) is prominent.

between IPT and aggressive malignancy have not been described in detail in any previous report. Secondly, follow-up MR studies were not available for each patient because clinical follow-up was usually determined by the response to treatment, the relief of clinical symptoms and signs, and the overall health of the patient, rather than on the imaging findings alone.

In summary, patients with IPT involving the nasopharynx and skull base often present with a recent history of otitis media and clinical complaints of cranial neuropathies and pain. The clinician should be aware of this disease and carefully investigate patients who present with the combination of a suggestive clinical picture, imaging characteristics and possible histological evidence in suspected cases to make a correct diagnosis, thereby distinguishing between IPT and aggressive malignancy.

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