Case Report

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Nasopharyngeal Carcinoma Spreading Along the Eustachian Tube: The Imaging Appearance

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Nasopharyngeal cancer (NPC) is one of the most common cancers in Taiwan. It can spread anteriorly, laterally, posteriorly, superiorly and inferiorly. Skull base invasion is the common presentation, in up to one-third of cases. We report a rare spreading pathway of NPC, *via* the eustachian tube. A 44-year-old male suffered from NPC and got radiotherapy about 2 years ago. He suffered from middle ear effusion and facial nerve palsy on the right side recently. The computed tomography and magnetic resonance image could clearly depict the infiltrating tumor over the mastoid region and tympanic cavity on the right side, spreading along the eustachian tube. It could result in eustachian tube dysfunction and middle ear effusion. The nature of the right facial palsy was most likely due to tumor infiltration.

Key Words
eustachian tube;
middle ear effusion;
nasopharyngeal cancer

Nasopharyngeal carcinoma (NPC) is one of the most common malignancies in Taiwan. The common spreading pathways are through the foramen lacerum, foramen ovale, sphenoid sinus or ethmoid sinus.

A middle ear effusion is found in approximately 38% of patients with NPC,[1,2] and auditary syndromes are the main presenting complaints in 18% of patients with NPC. Mechanisms by which an effusion is produced are thought to include mechanial or functional obstruction and abnormal patency of the eustachian tube (ET).[3] Computed tomography (CT) has been used to study the ET and its involvement in NPC. More recently, improved depiction of the small anatomic structures, including the palatini muscles, pharyngobasilar fascia and ET, has been achieved using magnetic resonance imaging (MRI).

NPC extended along the ET is an uncommon spreading path. We report a case with this unusual spreading pathway of NPC.

CASE REPORT

A 44-year-old male patient suffered from NPC (T4N2M0), and received radiotherapy and chemotherapy about 2 years ago. Recently, the patient suffered from progressive right side facial nerve palsy for 2 months. Then he came back to our hospital for further evaluation. Under the impression of right mastoiditis with right side facial palsy, he was admitted. During the hospitalization, CT was performed, revealing suspected soft tissue mass at the right mastoid region with bony destruction (Fig. 1). Otoscopy revealed amber color of right eardrum and serous fluid collection in middle ear cavity. Otherwise, the physical examination and laboratory data were normal.

MRI was then performed for further evaluation. There was an infiltrative soft tissue mass noted in the right mastoid and tympanic cavity spreading along the ET. This mass showed low signal intensity (SI) on axial

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section T1-weighted image (T1WI) (Fig. 2A), high SI on T2-weighted image (T2WI) with fat-saturation technique (Fig. 2B) with strong contrast-enhancing pattern on post-contrast axial section T1WI (Fig. 2C). Also, the infiltrative lesion surrounding the ET demonstrated similar image characteristics as the mass lesion in the mastoid area and tympanic cavity.

Right-side modified radical mastoidectomy with facial nerve decompression was performed, and the pathological report was metastatic squamous cell carcinoma (Fig. 3). The final diagnosis was NPC with right side mastoid, tympanic cavity extension *via* the ET, which result in right side mastoiditis and middle ear serous effusion.

DISCUSSION

MRI can identify and distinguish the bony from the cartilaginous ET. The main body of the cartilaginous ET lies below the skull base before continuing forward, medially and inferiorly, toward the nasopharyngeal orifice, where it expands to form the torus tubarius (TT), which lies directly anterior from the fossa of Rosenmuller.

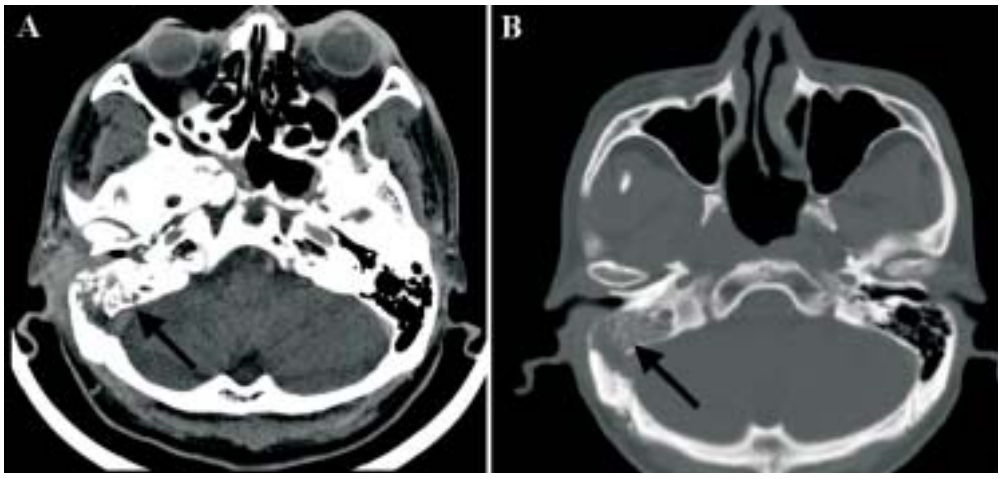

Fig. 1. CT of temporal bone (**A**); bone window setting (**B**). A soft tissue lesion was noted over the right side mastoid region with bony destruction (arrow) and bony cortex disruption.

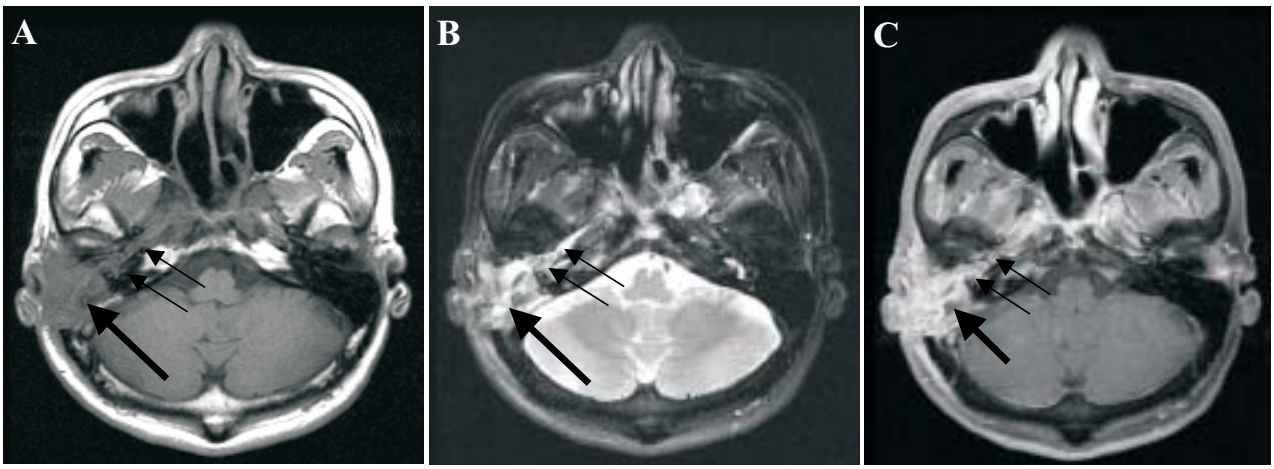

Fig. 2. MRI of head, axial section. 2A: T1WI (TR/TE/NEX/MATRIX: 500/19/2/256X256); 2B: T2WI (4000/86/2/256X256); 2C: post-gadolinium enhancing T1WI (516/25/2/256X256). One relatively low SI soft tissue lesion was noted over the right side mastoid region (thick arrow) on T1WI (**A**), high SI on T2WI (**B**) with contrast enhancement on post-gadolinium images (**C**). An infiltrative lesion along the right side eustachian tube (double thin arrows) with similar image characteristic was also identified. The lesion was in contact with the right side mastoid lesion.

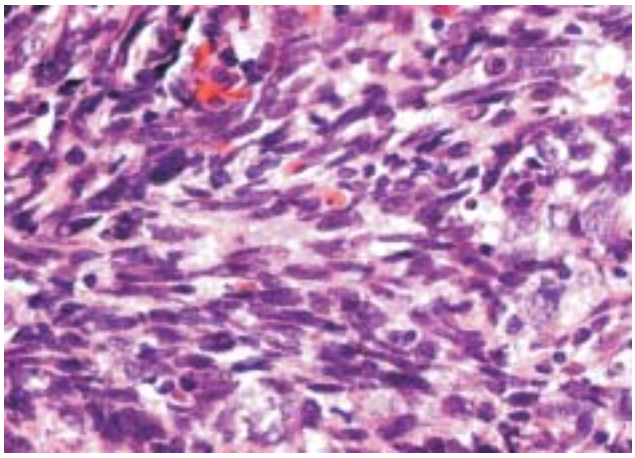

Fig. 3. Microscopic view of the specimen removed from the right side mastoid (HE stain 400x) revealed high mitotic squamous cell carcinoma with hypercellularity.

MRI also identifies the pharyngobasilar fascia just below the level of the skull base, the fatty tissue adjacent to the lateral margin of cartilaginous eustachian tube (CET). The levator palatini muscle (LP) lies inferiorly and medially from the ET, and the tensor palatini muscle (TP) lies laterally and superiorly from the ET.

Middle ear effusion is one of the most common presentations of NPC patients. There are many theories concerning the etiology of a middle ear effusion. Effusions are seen most frequently in advanced disease that has spread outside the confines of the nasopharyngeal region into the parapharyngeal space and TP muscle. Besides, the tumor involving the fossa of Rosenmullar, ET orifice, TP muscle, LP muscle or CET could result in middle ear effusion due to ET dysfunction.

Advanced disease invading outsides the confines of the nasopharynx, into the parapharyngeal fat space and TP muscle, is reported to be the most common pattern of tumor spread associated with an effusion.[4,5] The TP muscle plays a major role in ET function for actively dilating the ET during swallowing, and experiments have shown paralysis of this muscle results in an effusion. Patients have been shown to be electroencephalogram abnormality in the tensor muscle of 67% of symptomatic ears. The results suggest a neurogenic paralysis caused by invasion of the nerve as it passes through the superior aspect of the parapharyngeal fat space.

Advanced invasion also caused a middle ear effusion by disrupting the attachment of the ET to the skull base,[6] interfering with supporting tissue such as Ostmann's fatty tissue,[7] and altering the compliance of the ET. In our case, the nature of the middle ear effusion was the result of ET tumor infiltration with tube dysfunction.

In general, NPC spreads along well-defined routes. Anteriorly, it spreads into the nasal fossa. Erosion of the nasal and infratemporal wall of the maxillary sinus may be observed. The tumor may infiltrate the pterygopalatine fossa through the sphenopalatine foramen. Then it can infiltrate the middle cranial fossa *via* the inferior orbital fissure, optic canal and superior orbital fissure to the cavernous sinus. It can also invade the anterior cranial fossa through the ethmoid sinus and cribrifrom plate anteriorly. Laterally, it can be recognized by the partial or complete effacement of the fat-filled parapharyngeal space. Further lateral spread takes the tumor into the masticator space. When the medial or lateral pterygoid muscles are infiltrated, the patient may complain of trismus. Posteriorly, it can spread to the prevertebral muscle, including the longus colli muscle. Inferiorly, it can invade to the oropharynx. Superiorly, skull base erosion and destruction is frequently detected, in up to one-third of patients. It most commonly spreads *via* the foramen lacerum, which then invades to the skull base and middle cranial fossa. Another common spreading way to the middle cranial fossa is through the foramen ovale. Otherwise, it also commonly invades the posterior cranial fossa *via* the jugular foramen. In summary, NPC extended through the ET to the mastoid region is a rare spreading path. The definite reason of the rare spreading pathway *via* ET is still unknown. Maybe it is due to the relative anti-neoplastic nature of cartilage.

In our case, MRI could clearly depict the infiltrating soft tissue along the ET. It had low SI on T1WI, high SI on T2WI with fat saturation technique, and strong contrast-enhancing pattern on post-gadolinium images. Additionally, bony destruction could also be clearly defined on CT images. Sometimes the infiltrating high SI lesion along the ET on T2WI is due to improper fat saturation technique. But in our case, the fat saturation technique was excellent and its thickness was greater than 5 mm. So it is more likely to be the true pathological tumor lesion, not due to the improper fat saturation technique.

The clinical presentation of mastoiditis in NPC patients is relatively common. But the presentation of facial nerve palsy is relative rare; usually it is a late compli-

cation of radiotherapy. In our case, the nature of facial nerve palsy is more likely to be tumor infiltration. In the NPC patient with the presenting syndrome of mastoiditis and facial palsy, the clinician should always be alert to the possibility of infiltrative tumor along the ET in addition to the late complications of radiotherapy. Further imaging study may be necessary.

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