

Nasopharyngeal Carcinoma

A Report of Two Cases with Unusual Extension

NAZOFARENKS KANSERİ NADİR YAYILIMLI İKİ OLGU SUNUMU

Bülent SATAR*, Fuat TOSUN*, Yalçın ÖZKAPTAN**

* Asst.Prof., Dept. of Otolaryngology, Head and Neck Surgery, Gülhane Military Medical Academy,

** Clin.Prof., Dept. of Otolaryngology, Head and Neck Surgery, Gülhane Military Medical Academy, Ankara, TURKEY

Summary

This report of two cases is of two aims, first is to remind of probable misdiagnosis of middle ear invasion by nasopharyngeal cancer because of its polyp-like appearance, and the other to emphasise that nasopharyngeal cancer may invade even facial nerve in its tympanic segment and cochlea.

Two cases of nasopharyngeal cancer admitted to Dept. of ORL in Gülhane Military Medical Academy were evaluated with clinical, histopathological and radiological findings.

Two cases had middle ear invasion by nasopharyngeal cancer by the way of Eustachian tube. In addition to that, left facial paralysis and left total hearing loss possibly arisen from cochlear aqueduct involvement were seen in first case. In both cases, pterygopalatine and infratemporal fossas were attacked.

It should be kept in mind the fact that nasopharyngeal carcinoma may invade middle ear causing polyp-like tissue likened to chronic otitis media. With middle ear invasion, tympanic segment of facial nerve is hit as well as extratemporal part rather seen. Also, even cochlea having solid bone is likely to be attacked by aggressive nasopharyngeal cancer.

Key Words: Nasopharyngeal carcinoma, Eustachian tube, Facial nerve, Cochlear aqueduct

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Özet

Bu çalışmanın amacı, orta kulağa invazyon göstermiş nazofarenks kanserinin, neden olduğu polibe benzer görünümlü nedeniyle yanlış teşhis edilebileceğini vurgulamak ve kohlea ve fasiyal sinir timpanik segmentinin invazyonunun olabileceğini göstermektir.

Gülhane Askeri Tıp Akademisi Kulak Burun Boğaz Kliniği'ne başvuran nazofarenks kanserli iki olgu muayene bulguları, histopatolojik bulgular, ve radyoloji bulguları ile değerlendirildi.

İki olguda da nazofarenks kanseri Östaki tüpü yoluyla orta kulağı invaze etmişti. İlk olguda sol fasiyal paralizi ve muhtemelen aquaduktus tutulumuna bağlı olarak total işitme kaybı mevcuttu. Yine her iki olguda pterigopalatin ve infratemporal fossa tutulumu söz konusu idi.

Nazofarenks kanserinin orta kulağı invaze ederek oluşturduğu polibe benzer görüntünün kronik orta kulak iltihabıyla karışabileceği unutulmamalıdır. Sıklıkla görülen ekstratemporal parçasının tutulumunun yanısıra orta kulak invazyonuyla fasiyal sinirin timpanik parçası da tümör tarafından atake edilebilir. Aynı şekilde, agresif seyreden nazofarenks kanseri solid kemik dokusuyla çevrili kohleaya da infiltre olabilir.

Anahtar Kelimeler: Nazofarenks kanseri, Östaki tüpü, Fasiyal sinir, Kohlear aquaduktus

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Although nasopharyngeal carcinoma originating from epithelial cells of the nasopharynx is rarely

seen in most of the world, it constitutes approximately 18 per cent of all malignant tumors in eastern and southeastern Asians. That is to say, its incidence shows some racial and geographical differences (1). Hearing loss and lump in the neck are the most common presentations. Because nasopharynx is located in the vicinity of skull base, the carcinoma erodes that region and attacks cranial nerves. Nasopharyngeal carcinoma with involvement

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Yazışma Adresi: Dr.Bülent SATAR
Gülhane Askeri Tıp Akademisi
Kulak Burun Boğaz AD
06018, Etlik, Ankara, TURKEY

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of cranial nerves comprises 20-25 per cent of all nasopharyngeal carcinomata (2,3). Most commonly the fifth and sixth cranial nerves are affected (2-4). Involvements of the seventh and eighth cranial nerves and invasion of the temporal bone are rarely seen (3,5,6). Cochlear invasion has not been reported so far. To our knowledge, there is a single case with involvement of the VIIIth cranial nerve reported by McGill (7).

The nasopharyngeal carcinoma may extend to the intracranial region by different pathways. One of them is the Eustachian tube. Cundy et al (1973) reported that obstruction of the Eustachian tube by the nasopharyngeal carcinoma was a well-known entity, but nasopharyngeal carcinoma extension to the middle ear via Eustachian tube has only been reported histologically in one temporal bone examination (8).

In this article, we present two cases of nasopharyngeal carcinoma with middle ear invasion via the Eustachian tube, one of whom with facial paralysis and total hearing loss. Within this framework we elaborated on the extension of nasopharyngeal carcinoma.

Case Reports

Case 1

A 63-year-old man (Mr.H.C.) referred with the complaints of tinnitus, decreased hearing in the left ear, left facial weakness and difficulty in swallowing. On admission, left peripheral facial paralysis and lymphadenopathies on the left parotid and upper jugular regions were found. The otoscopic examination revealed serous otitis media in the left ear. In pure tone audiogram, moderate conductive hearing loss was seen. The computerized tomography (CT) scan showed a mass filled the roof and the left side of the nasopharynx with extension to the nasal cavity as well as parotid and cervical lymphadenopathies. The histopathological examination of the biopsy taken from the nasopharynx disclosed a nonkeratinizing epidermoid carcinoma. Thereafter, he underwent radiotherapy. One and a half-month later, he complained of total hearing loss and discharge from the left ear. Peripheral facial paralysis still persisted on the left side. The otoscopic examination revealed a polypoid tissue in the left outer ear canal

and tympanic membrane perforation. Soft tissue invasion on the left parotid region was noticed. Biopsy from polypoid tissue in the outer ear canal led to the conclusion of infiltration of nonkeratinizing carcinoma. In the neurological examination, hypoesthesia and paralysis were found in the mandibular division of the fifth cranial nerve and the seventh, ninth and tenth cranial nerves. The audiogram revealed a total sensorineural hearing loss. The Schirmer's test was symmetrical on both sides.

CT of the nasopharynx revealed a tumor spread through the roof and the left posterolateral wall of the nasopharynx, invading nasal cavity and filling up the pterygopalatine fossa. The Rosenmüller's fossa and the nasopharyngeal opening of the Eustachian tube were obstructed. The mass infiltrated through the temporal muscle planes and subcutaneous tissues in the infratemporal fossa. The parotid gland was infiltrated by the tumor. The CT of the temporal bone revealed the findings as follows: The anteromedial face of the petrous bone was eroded on the left side. The mastoid aeration was significantly decreased. The outer ear canal was filled up by soft tissue (Figure 1). The cochlear aqueduct was widened. The ganglion geniculatum and the tympanic segment of the facial nerve were destroyed. The anterior wall of the tympanic cavity was completely destroyed. The magnetic resonance



Figure 1. CT view of the tissue filling up outer ear canal, middle ear space and Eustachian tube on the left (thick arrows). Mastoid aeration of the left ear is significantly decreased (Case-1).

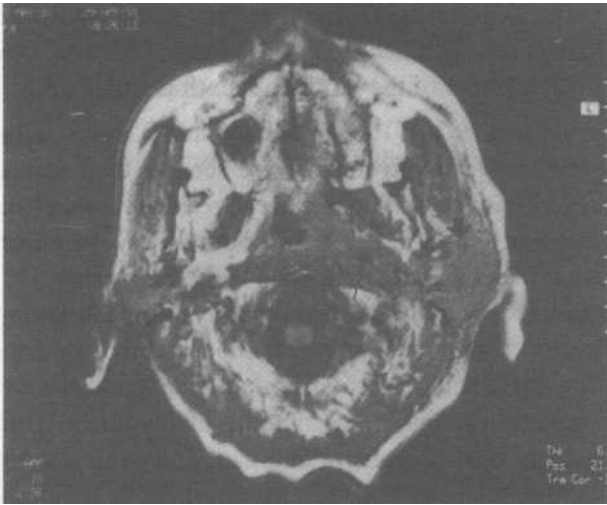


Figure 2. MRI view reveals invasion of pterygopalatine fossa, masseter muscle and subauricular region. Nasopharynx airway passage appears asymmetric on the left (thick arrow). Left internal carotid artery is displaced towards anteromedial direction (thin arrow) (Case-1).



Figure 3. CT view shows the soft tissue present in middle ear and outer ear canal (thick arrow) as well as the decreased mastoid aeration of the left ear and sclerosis (Case-2).

imaging (MRI) of posterior fossa and nasopharynx showed the invasion of the dura mater in the posteroinferior face of the left temporal lobe as well as the invasion of the masseter and temporal muscles and the subcutaneous soft tissue (Figure 2). Additionally, density of the soft tissue in the mastoid antrum was apparent. The bone scintigraphy demonstrated increased activity in the left temporal region in all of three sections. Because of the fact that radiotherapy had been previously applied, chemotherapy was instituted. Unfortunately, the patient died in the fifth month after the first diagnosis, while chemotherapy was going on.

Case 2

A 43-year-old woman (Mrs.S.A.) was admitted in September 1994 with a history of facial pain, otalgia and discharge on the left side. Her report revealed that she had undergone radiotherapy with a diagnosis of nasopharyngeal undifferentiated carcinoma and bilateral cervical lymphadenopathies at another center in 1992. The radiotherapy had led to the complete regression of the tumor.

In the examination, trismus was observed. The otoscopy exhibited polypoid tissue in the outer ear

canal through the tympanic membrane perforation after aspirating purulent secretion. In the nasopharyngeal examination, a verrucous mass of 7x8 mm in diameter was seen above the torus tubarius on the left side. Biopsy from the mass in the nasopharynx revealed nonkeratinize carcinoma in the nasopharynx. In the histopathological examination, infiltration of the nonkeratinize carcinoma in the nasopharynx into the polypoid tissue in outer ear canal was observed. There was no palpable lymphadenopathy in the neck. In the cranial nerve examination, only hypoesthesia and paresthesia were confirmed on the left lower lip. There was no corneal hypoesthesia.

The CT of nasopharynx and temporal bone revealed a soft tissue density in the posterior wall of the nasopharynx, the pterygopalatine fossa and the infratemporal fossa on the left. The anterior surface of petrous bone was eroded. The soft tissue was present in the left tympanic cavity (Figure 3). The bone scintigraphy demonstrated the increased activity in the nasopharynx region. Because of the previous radiotherapy application, a chemotherapy regime consisting of cisplatin, cyclophosphamide and bleomycin was instituted six times with 28-day intervals. The examination in the third months after chemotherapy surprisingly revealed that there was

no mass in the nasopharynx. Tympanic membrane was found to be intact but adhesive in appearance. The audiogram still showed moderate conductive loss in the involved ear. For the time being, she is followed up with 6-month intervals.

Discussion

Nasopharyngeal carcinoma constitutes approximately the 2% of all head and neck tumors. The symptoms depend on the location and extension of the tumor. The spread of nasopharyngeal carcinoma is divided into six main directions. In the anterior extension, pterygoid process, pterygopalatine fossa, nasal cavity, maxillary sinus and ethmoid sinus are involved by the tumor. In the posterior extension, clivus, cervical vertebra, spinal canal and posterior fossa are affected. In the superior extension, sphenoid sinus and cavernous sinus are invaded. In the posterolateral extension, foramen lacerum and cavernous sinus are involved. If tumor extends to superolateral direction, foramen ovale, foramen spinosum and middle cranial fossa are affected. The most common directions of spread are anterior and posterolateral directions respectively (2, 3, 8-11).

Thomas and Waltz (1965) pointed out that the Rosenmüller's fossa, the classic site of origin of the nasopharyngeal carcinoma, lies beneath foramen lacerum (5). The foramen lacerum and foramen ovale are preferred routes of entry into the cranial cavity. The cranial neuropathy results from superior extension of the tumor, and petrosphenoidal crossway formed by the foramen lacerum and foramen ovale provides a pathway from the Rosenmüller's fossa into the cranium (9,12). In either case, infratemporal fossa was attacked thereby involving mandibular nerve.

Cranial nerves leaving the skull through the posterior fossa are susceptible to damage from such a tumor. These lesions are extracranial and caused either by spread of tumor into the retroparotid space or by pressure from metastasis in the retropharyngeal lymph nodes. In addition, the tumor may spread posterolaterally with infiltration of jugular foramen (13). Thomas and Waltz (1965) reported that involvement of the posterior nerves in their series accounted for 26 per cent of all cranial

nerve lesions (5). In first case, retroparotid extension of this aggressive tumor caused the involvements of the ninth and tenth cranial nerves.

Chen et al (1989) stated that 37 cases with facial nerve involvement were seen in their series of 109 patients with nasopharyngeal carcinoma (6). Thomas and Waltz (1965) reported only 14 cases with facial paralysis in their series covering 111 cases (5). In first case, facial paralysis on admission was probably due to extratemporal involvement. The existence of parotid lymph adenopathies may account for this extratemporal involvement. But later, tympanic segment and ganglion geniculatum were attacked as well. The involvement of ganglion geniculatum and tympanic segment is rarely seen in nasopharyngeal carcinoma. It is entrapped in its extratemporal part with parotid spread of the tumor. Vestibulocochlear nerve involvement is so rare that even it is ignored in most cases. The only attacked site of it is internal acoustic canal (IAC). In coexistence of seventh and eighth cranial neuropathies, tumor may be thought to extend to the IAC. However, in first case, IAC was intact. Schirmer's test was bilaterally symmetrical. Therefore, the involvement of facial nerve must have been after ganglion geniculatum. Schuknecht et al (1968) proposed that sensorineural hearing loss arising from damage of cochlear nerve fibres, compression of the cochlear nerve in the IAC and unusual invasion of the otic capsule may be seen in the nasopharyngeal carcinoma (14). However, compact and solid bone structure in this region prevents cochlea from the tumor invasion. Okan et al (1993) reported a case on whom seventh and eighth cranial nerves lesions were seen along with the intact IAC (15). However, no widening of IAC is likely due to the rapidity with which the disease progressed (16). Cundy et al (1973) could not explain the sensorineural component of the hearing loss in their case on the basis of temporal bone findings (8). In 1976, McGill published a report consisting of 12 cases with totally degenerated cochlear and vestibular nerves, but normal temporal bones. Therefore, McGill proposed the term of carcinomatous neuropathy of involved the eighth cranial nerve that was explained with secondary nonmetastatic paraneoplastic syndrome (7). As opposed to this, Leslie and Zinreich (1991)

stated that lesions of the ganglion geniculatum cause findings of seventh and eighth cranial nerve involvements similar to those in IAC (17). The tumor cells might pass by way of CSF to the inner ear or petrous portion of the temporal bone. The cochlear aqueduct constitutes a connection between perilymph and CSF. In our first case, the inner ear and IAC appeared normal in the CT and the MRI, dilatation of the cochlear aqueduct excepted, which may explain the hearing loss. An explanation of this condition is that dura invasion of middle cranial fossa leading to meningitis carcinomatosa and thereby causing cochlear aqueduct involvement. More detailed investigation of the subtle route of the metastasis to the temporal bone may help to explain these conditions.

The Eustachian tube can be used by the nasopharyngeal carcinoma as a portal of entry to the middle ear and mastoid but such an extension is again unusual (8). Çuhruk et al (1988) reported three cases with nasopharyngeal carcinoma invaded middle ear via Eustachian tube among 214 patients (18). The CT of the two cases presented in this article demonstrated involvements of middle ear and outer ear canal through the Eustachian tube by nasopharyngeal carcinoma. It was thought that the polypoid mass in the outer ear canal was the secondary invasion of the primary tumor in the nasopharynx. The biopsy from nasopharynx and polypoid tissue in the outer ear canal disclosed the diagnosis of nonkeratinizing epidermoid carcinoma in first case. In the other case, interestingly the biopsy from nasopharyngeal mass revealed undifferentiated carcinoma, whereas the biopsy from the polypoid tissue in the outer ear canal displayed nonkeratinizing epidermoid carcinoma. An interesting finding is that we have seen regression of tumor in outer ear canal and intact tympanic membrane in second case after chemotherapy. Returning of the tympanic membrane to intact appearance is probably due to spontaneously healing of the tympanic membrane perforation and its adhesion to promontorium after regression of the tumor. But, Eustachian tube disfunction still persisted. Instead of invasion through eustachian tube, the other possibility for the spread of tumor through the outer ear canal might be the erosion of anterior wall of outer ear canal bypassing middle ear. However, in both

cases, CT's prove clearly outer ear canals not to be destroyed.

The symptoms of patients with both primary and secondary temporal bone tumors include aural polyp or chronic infection, pain, cranial nerve paralysis, haemorrhage, hearing loss, vertigo tinnitus, hemotympanum, acute otitis media or mastoiditis (3,10). Maddox (1967) reported that in more than one third of the cases (37%) with involvement of temporal bone, mastoiditis with subperiosteal abscess and facial nerve paralysis were seen as initial impressions (19). Facial paralysis, otalgia and periauricular swelling must be the triad to alert the physicians to metastatic temporal bone disease (10,19). Okan et al (1993) also reported that acute mastoiditis and facial paralysis must be the signs of metastatic tumor of the temporal bone (15). First case exhibited these three signs, whereas the second case had presented only mastoiditis.

Conclusion

The nasopharyngeal carcinoma invasions into adjacent areas and structures are well-known despite the fact that the extension via Eustachian tube to the middle ear is rarely seen. This extension may imitate chronic otitis media because of otorrhea, otalgia and polyp-like appearance. Therefore, this fact may lead to misdiagnosis.

Another emphasis of this article is on that it presents the involvement of cochlear aqueduct as well as the involvement of facial nerve in its segments after geniculate ganglion not just extratemporal part.

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