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Compression of the Brain Stem and Cerebellar Peduncles: Nasopharyngeal Carcinoma Can Be Incriminated Too A Case Report

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Abstract **Case Report**

Nasopharyngeal carcinoma (NPC) is a distinct head and neck tumour associated with Epstein Barr virus infection. This case report describes a 60-year-old woman with a history of NPC who developed after an initial treatment, extensive skull base and intracranial invasion, causing compression of the brainstem and cerebellum, and cranial nerve palsies. Intracranial invasion is a prognostic factor indicating advanced disease, however direct compression of the brainstem by the tumour is not a frequent occurring.

Keywords: Nasopharyngeal carcinoma (NPC), neck tumour, initial treatment, intracranial invasion, brainstem and cerebellum.

INTRODUCTION

Nasopharyngeal carcinoma is a particular Head and Neck tumour, frequent in some areas of the world. It's most frequent form is unlike other head and neck tumours, not related to alcohol/tobacco consumption, but closely linked to Epstein Barr virus. It can manifest with various symptoms according to the extent. Neurological symptoms are quite frequent, whether intracranial involvement is present or not. in this article, we report the case of an intracranial invasion of a nasopharyngeal carcinoma, responsible of brain stem and cerebellar compression.

CASE REPORT

A 60-year-old woman treated in 2019 for nasopharyngeal carcinoma (Undifferentiated Carcinoma Nasopharyngeal Type) was examined in the oncology clinic for a control. She presented with the following symptoms: left oculomotor paralysis, decrease of sight, headache and serous otitis media.

A cranio facial magnetic resonance (May 2023) was conducted and showed remarkable progression of her tumour (Figure 1); particularly in its intracranial component compared to a previous examination 6 months prior (July 2022) (Figure 2). The latest MRI shows tumour centred in the skullbase, extending to the apex of petrous bone, the sphenoid bone, the clivus with intracranial involvement through the clivus and the left skull base foramina.

Intracranial extension was responsible of complete obliteration of the prepontine cistern, and compression (T2 and FLAIR hyperintensity) of the pons and the left middle cerebellar peduncle. The petrous bone was largely involved abutting the cochlea, the internal auditory canal, invading the temporal meninges that were thickened, compressing the temporal lobe superiorly. Both cavernous sinuses (predominantly left) along with internal carotid arteries in their (petrosal and cavernous segments were involved). Anterior extension involved the parapharyngeal and infratemporal spaces on the left. Opacification of the left mastoid cells and tympanic cavity were also noted. On the previous MRI, (July 2023), the intracranial involvement was significantly lower and manifested as meningeal thickening, mild cavernous sinus infiltration on the left and lesser petrous extension. The patient had had her final radiotherapy session in 2019, then was diagnosed in April 2021 with papillary carcinoma of the thyroid. The patient has not resumed her NPC controls for over a year. The MR conducted in July 2022 was the first after this period (Figure 2). Then a petrous bone CT was conducted in February 2023 (Figure 3), showing extensive lytic lesions of the skull base (clivus, petrous apex reaching the anterior wall of the left porus, the

sphenoid bone reaching the inferior aspect of the sella turcica). The patient had still at this point refused treatment. Finally, after the last MRI held (May 2023), a pluridisciplinary meeting has been held and the patient was sent for chemotherapy after nasopharyngeal biopsy.

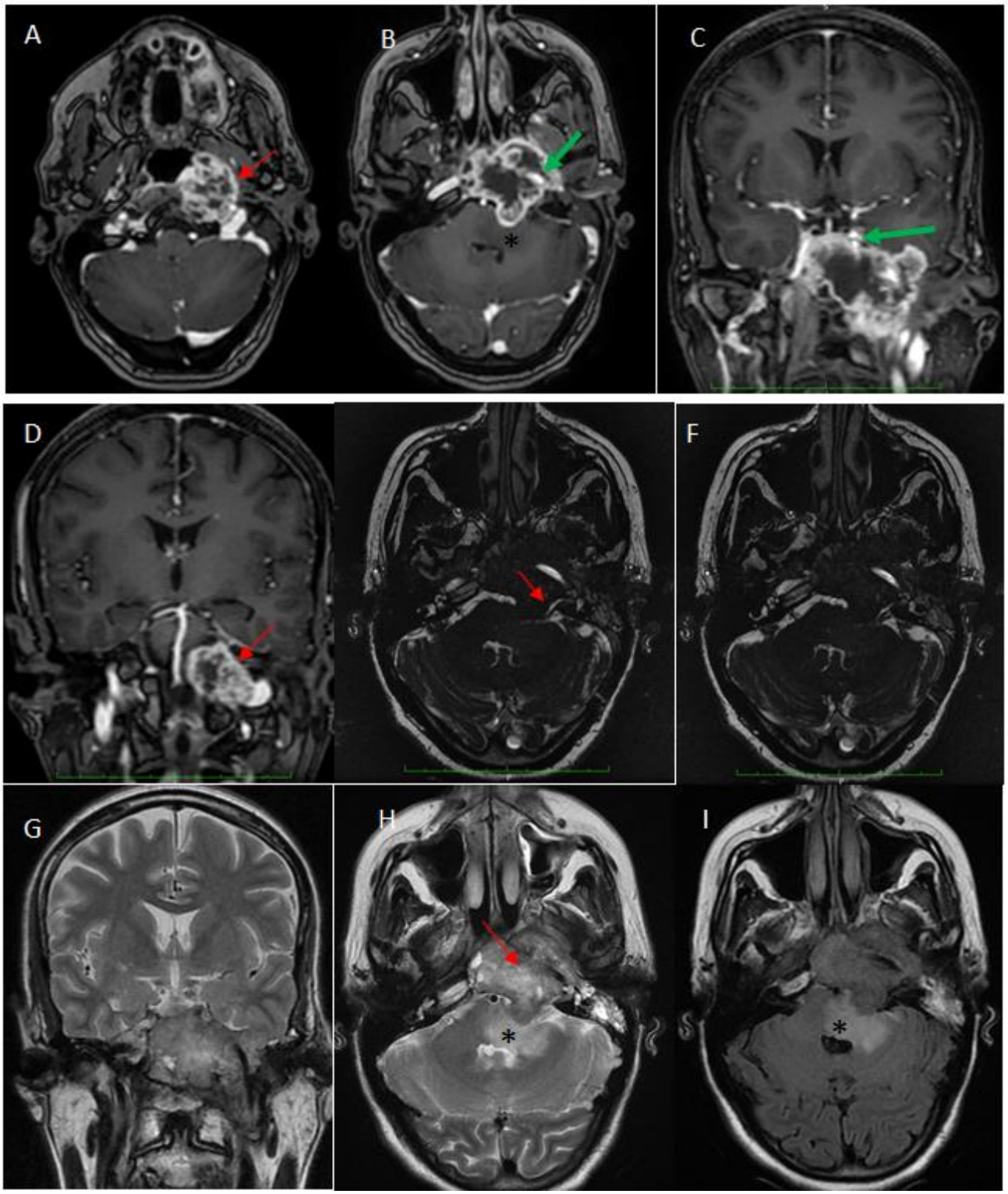

Figure 1: Cranio-facial MRI (May 2023): T1WI post Gadolinium contrast injection coronal and axial views (A-D), 3D FIESTA (E, F), T2WI in coronal and axial views (G, H), FLAIR (I) sequences. Enhancing lesion (red arrow) with intra cranial involvement, mass effect on the pons and left cerebellar peduncle and T2 hyperintensity in both structures (asterisks), left temporal lobe, filling of the cerebellopontine angle, abutting the left porus. The left internal carotid artery in its petrous and cavernous segments in within the lesion (green arrow)

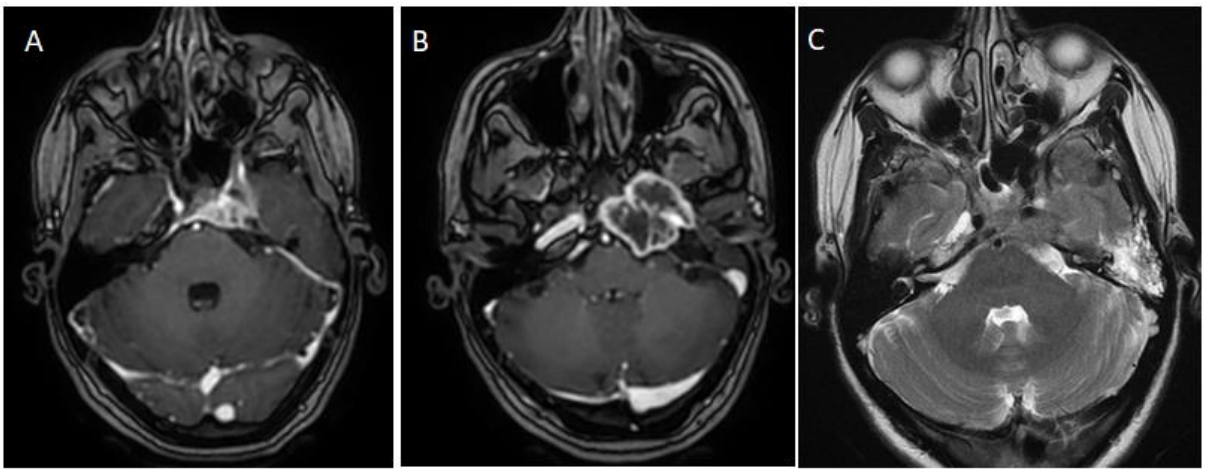

Figure 2: Cranio-facial MRI (November 2022): T1WI post Gadolinium injection and T2WI in axial view: the lesion is invading the skull base with less intracranial extension (mildly in the cerebellopontine angle), no compression of the pons nor cerebellar peduncle. The left internal carotid artery is within the lesion

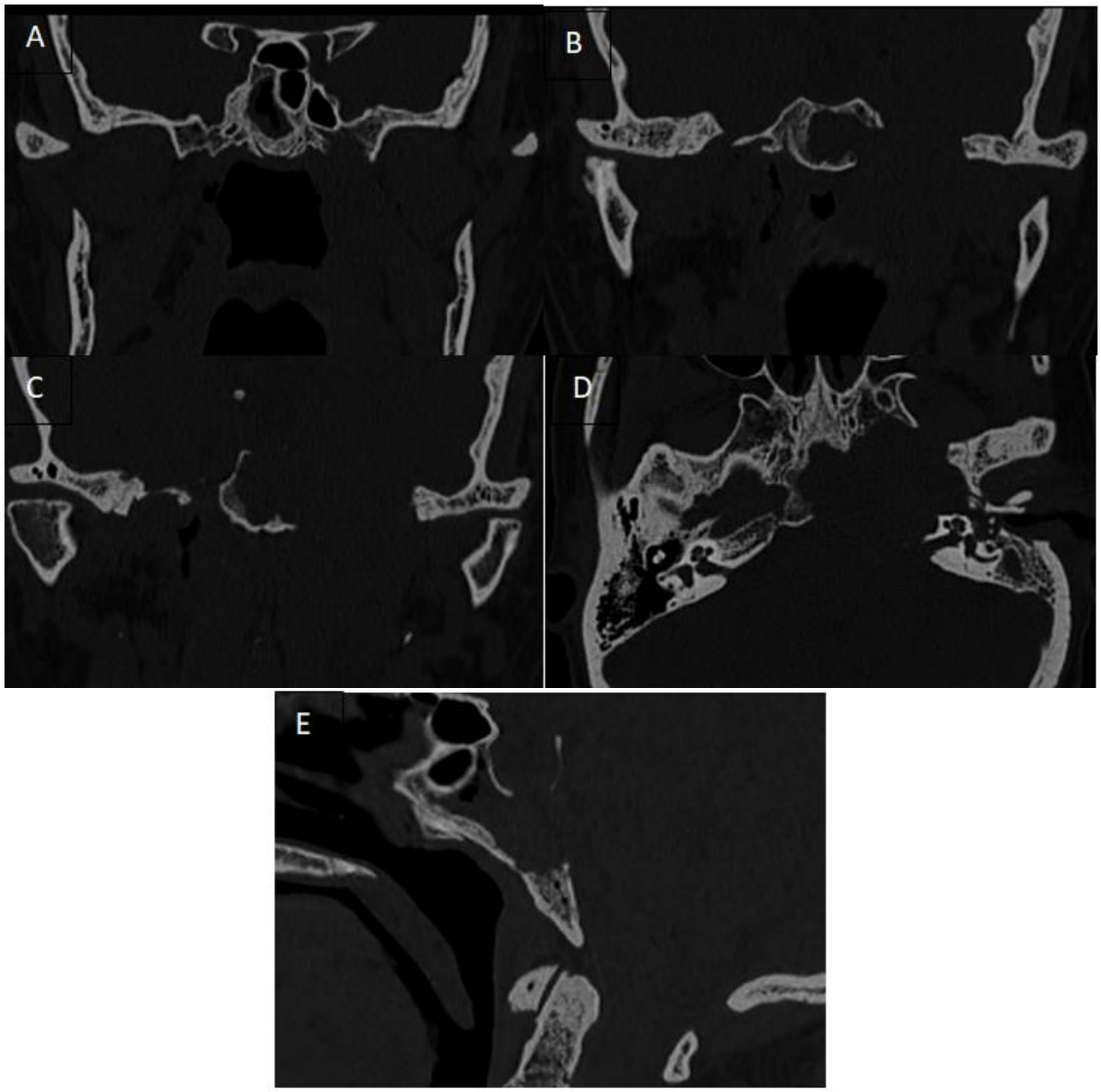

Figure 3: CT of petrous bone in coronal (A-C), axial (D) and sagittal (E) views: Extensive lytic lesions of the skull base (clivus, sphenoid, and left petrous apex), extending to the foramen rotundum (A), foramen ovale (B), foramen lacerum (C) and the inferior aspect of the sella turcica (E)

DISCUSSION

Nasopharyngeal carcinoma (NPC) is a relatively rare tumour on a global scale, but more frequent in South and East Asia (China) North Africa, and the Arctic region [1]. Unlike other Head and Neck tumours, whose contributing factors are alcohol-tobacco consumption, only one form shows this connection: Keratinizing NPC (K-NPC). The other forms, Non-Keratinizing NPC (both differentiated and undifferentiated) are closely linked to Epstein Barr virus, and have the racial distribution cited earlier. The incidence is twice to three times higher in men [2]. Endoscopic biopsy is the first step for evaluating NPCs. MRI is the best imaging modality for soft tissue and nodal extension (better contrast resolution), and CT for bone invasion [3]. MRI protocol for NPC should include: T1WI, T2WI with fat saturation, and T1WI post gadolinium injection and fat saturation. Axial, coronal and sagittal planes should be included. A head and neck antenna is used, and large matrix of 256 x 352 or more [4].

Clinical signs are predominantly related to the obstruction of the Eustachian tube, resulting in a conductive hearing loss. Neurological symptoms occur due to infiltration of cranial nerves. Epistaxis and other rhinological symptoms may occur, as well as enlarged lymph nodes. The unilaterality of the signs is more in favour of the diagnosis of NPC [5].

The 8th edition of UICC/AJCC Staging system is used to classify the patients according to the degree of extension of the tumour into four stages for treatment planning and prognosis [6].

NPC commonly begins in the fossa of Rosenmüller and tends to spread submucosally, infiltrating the palatal muscles, particularly the levator veli palatine muscle, responsible of serous otitis. Then it extends first to loose regions of the pharyngo basilar fascia [7]. The pathways of extension can be divided into six routes. Anterior extension is toward the choanes , the nasal fossae, the paranasal sinuses and the . Inferior extensions towards the oropharynx, laterally into the parapharyngeal spaces and further to infratemporal and masticatory spaces, with possible infiltration of the maxillary nerve, reaching the foramen rotundum and the mandibular nerve to the foramen ovale. Posteriorly, it can infiltrate the retropharyngeal and pre-vertebral spaces with possible vertebral destruction and invasion of the hypoglossal nerve.

Superior extension can involve the foramen lacerum, the sphenoid sinus and the clivus, and further to the intracranial space. Nodal invasion is also very frequent, starting from retropharyngeal nodes to jugular nodes [7].

Intracranial invasion is a prognostic criterium and classes the NPC into T4. It can occur through different pathways. Direct extension via the clivus, the petrous apex and the sphenoid bone. It can also invade the orbital apex through the inferior fissure, then further to the endocranium via the superior fissure. Perineural extension through the mandibular nerve (foramen ovale), maxillary nerve (f.rotundum), pterygoidian nerve (petrous apex). It can also extend hematogeneously, following the bloodstream pathway of metastases [8].

Endocranial extension manifests as meningeal thickening or infiltration of the cavernous sinus responsible of oculomotor nerve palsies.

Meningeal involvement appears as nodular leptomeningeal thickening and concerns more frequently the middle cranial fossa and the temporal lobes. Posterior invasion is less frequent and rarely, we may find invasion of the cerebellopontine angle through the jugular foramen resulting in palsies of the VIIth and VIIIth in cranial nerves [9-11]. Direct invasion of the cerebral parenchyma is rare. Compression of the brain stem caused by clival invasion is also rare [12].

In our case we have found patent compression of the brain stem and left cerebellar peduncles.

CONCLUSION

NPC (NK-UCNT) is a treatable disease when diagnosed during early stages. Intracranial invasion is a relatively frequent occurring in NPCs and an important prognostic factor that classes the tumour as T4. Direct compression of the brainstem has rarely been reported, as the tumour usually shows good response to radiotherapy.

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