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Case Report

Temporal Bone Metastasis Revealing Progression of Prostate Adenocarcinoma: A Case Report and Literature Review

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A B S T R A C T

Introduction: Temporal bone metastases are rare and usually occur late in the course of the disease. Prostate adenocarcinoma is one of the least frequent primary tumors to metastasize to this region in men, with approximately twenty cases reported in the literature. We report a new case in order to highlight the diagnostic difficulty and the value of a multidisciplinary management.

Case report: A 73-year-old man, with a history of radical prostatectomy five years earlier for prostate adenocarcinoma and currently receiving chemotherapy, was admitted for severe sudden-onset headaches lasting for one month, associated with right-sided hearing loss, tinnitus and vertigo. Cerebral computed tomography (CT), temporal bone CT and cerebral magnetic resonance imaging (MRI) revealed a lytic process of the right petrous apex extending to the occipital basion (30 × 14 mm), compressing the jugular bulb and involving the jugular foramen, the condylar canal and the F3 portion of the facial nerve, without intracranial extension. Pure-tone audiometry showed bilateral mixed hearing loss predominating on the right side. A diagnostic mastoidectomy with biopsy confirmed a bone localization of a poorly differentiated invasive adenocarcinoma (CKAE1/AE3 positive), consistent with progression of the primary prostatic tumor.

Discussion: Temporal bone metastasis from prostate cancer is exceptional and usually presents with hearing loss, tinnitus, cranial nerve palsies or headache. Cross-sectional imaging (CT and MRI) is mandatory to characterize the lesion, but histological confirmation remains required. Treatment is palliative and relies on external-beam radiotherapy, hormone therapy and, in castration-resistant disease, on novel targeted therapies.

Conclusion: Any new otological or neurological symptom occurring in a patient followed for prostate cancer should prompt the search for a temporal bone metastasis, even long after the initial diagnosis.

Keywords: Temporal bone; Petrous apex; Bone metastasis; Prostate adenocarcinoma; Jugular foramen; Hearing loss

Introduction

Prostate cancer is the second most frequent malignancy in men worldwide and one of the leading causes of cancer-related mortality in elderly patients[1]. Its metastatic dissemination is predominantly skeletal and its particular tropism for bone is explained by complex molecular interactions between tumor cells and the bone marrow microenvironment, classically leading to predominantly osteoblastic lesions, less frequently mixed or purely lytic[2,3].

The most common metastatic sites are the spine, the pelvis, the femurs and the ribs. Skull base involvement, particularly of the temporal bone, remains exceptional, accounting for less than 2% of bone metastases from prostate cancer[4,5]. Presenting symptoms are variable and nonspecific: hearing loss, otalgia, tinnitus, vertigo, facial nerve palsy or other cranial nerve deficits, reflecting structural invasion of the petrous bone, jugular foramen or petrous apex[5-7].

We report the case of a 73-year-old man, followed for prostate adenocarcinoma, in whom otological and headache symptoms led to the discovery of a lytic metastasis of the right petrous apex extending to the occipital condyle and jugular foramen. Through this case and a literature review, we discuss the clinical, radiological, pathological and therapeutic features of this rare location.

Case Report

A 73-year-old male patient, with a history of prostate adenocarcinoma diagnosed five years earlier and treated by radical prostatectomy, currently under chemotherapy, was admitted to the ENT department for severe sudden-onset headaches lasting for about one-month, progressive right-sided hearing loss, ipsilateral tinnitus and vertiginous sensations.

ENT clinical examination revealed a free external auditory canal and normal tympanic membranes, with no inflammatory signs or otorrhea. Cranial nerve examination showed no obvious clinical deficit at admission. Pure-tone audiometry revealed bilateral mixed hearing loss clearly predominating on the right side, with a marked drop in the high frequencies.

Radiological evaluation

Emergency cerebral CT showed no parenchymal abnormality but revealed a lytic lesion centered on the petrous portion of the right temporal bone, producing a pseudo-mass extending to the basilar part of the occipital bone and involving the jugular foramen, responsible for a decreased caliber of the ipsilateral internal jugular vein, which remained patent **(Figure 1)**.

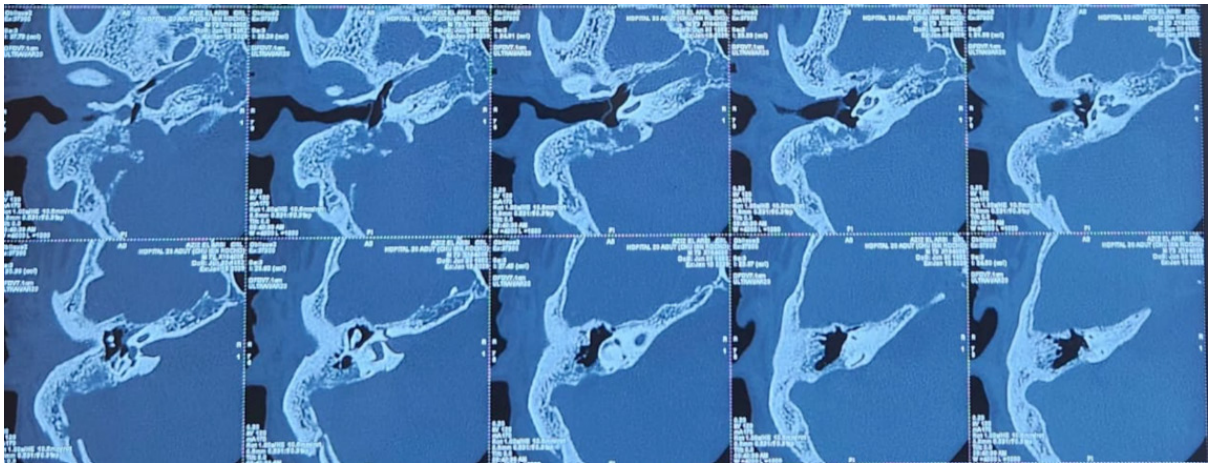


Figure 1: Axial cerebral CT images showing the lytic lesion of the petrous portion of the right temporal bone, extending to the occipital basion, without associated parenchymal abnormality.

Cerebral MRI confirmed a lytic lesional process of the right petrous apex extending to the ipsilateral occipital basion, fairly well-defined, isointense on T1 and T2 sequences, with peripheral enhancement after gadolinium injection, measuring 30 × 14 mm **(Figure 4)**. It was responsible for compression of the right jugular bulb without endoluminal material, reached the sigmoid sinus without signs of extension, extended extra-axially without cerebellar involvement and showed no meningeal thickening or enhancement. Infiltration of the ipsilateral mastoid cells was noted, together with vascular leukoencephalopathy classified as Fazekas 2. The appearance was considered suggestive of a probable secondary origin.

Dedicated temporal bone CT showed diffuse bony demineralization of the skull base and further characterized the lesion on the right side: a lytic process of the right occipital condyle with a few arciform calcifications, extending anteriorly to the petrous bone (including the jugular wall of the tympanic cavity and the mastoid cells), involving the bony wall of the jugular foramen with a mild mass effect on the internal jugular vein **(Figures 2 and 3)**. Medially, it reached the intimate contact of the right hypoglossal canal, the site of a bony discontinuity without intracanal extension. Posteriorly, it extended to the condylar canal. The F3 portion of the facial nerve was encased, whereas the F1 and F2 portions, the ossicular chain, the tegmen tympani and the attic wall were preserved. The tympanic cavity and external auditory canal were well-aerated, with an apparently thickened tympanic membrane compared with the contralateral side. The semicircular canals appeared normal.

Management and histological diagnosis

After multidisciplinary tumor board discussion and given the oncological context and the atypical osteolytic appearance of the lesion, a surgical biopsy was indicated. The patient underwent a diagnostic mastoidectomy with biopsy sampling. Preoperative workup was reassuring: hemoglobin 13.3 g/dL, platelets 232 × 10^3/mm^3, normal renal function and coagulation profile, sinus rhythm ECG without significant abnormality. The anesthesiology consultation classified the patient as ASA II.

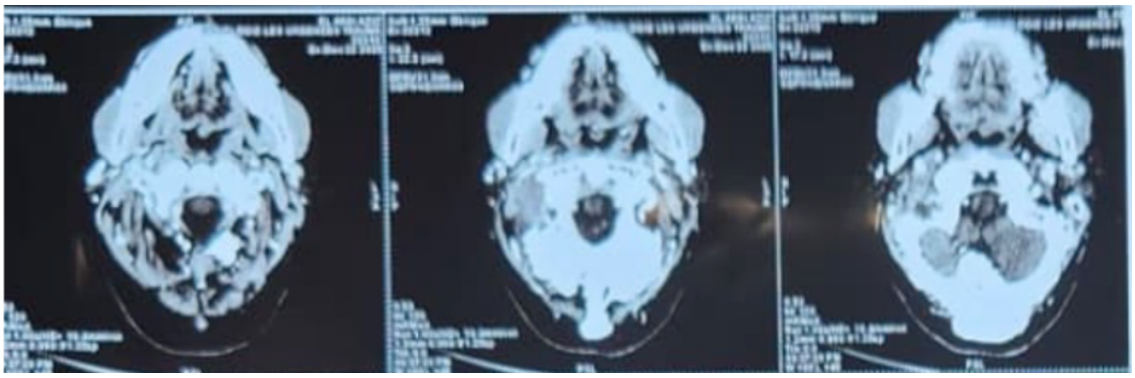


Figure 2: High-resolution temporal bone CT, axial views. Lytic process of the right occipital condyle extending to the petrous bone and involving the bony wall of the jugular foramen.

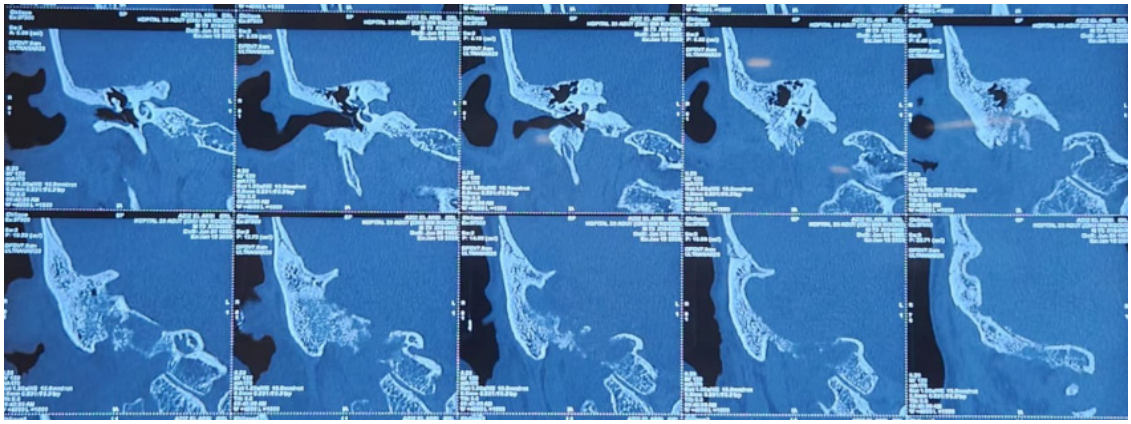


Figure 3: High-resolution temporal bone CT, coronal views. Extension of the lytic process to the occipital basion with involvement of the condylar canal and mass effect on the ipsilateral jugular bulb.

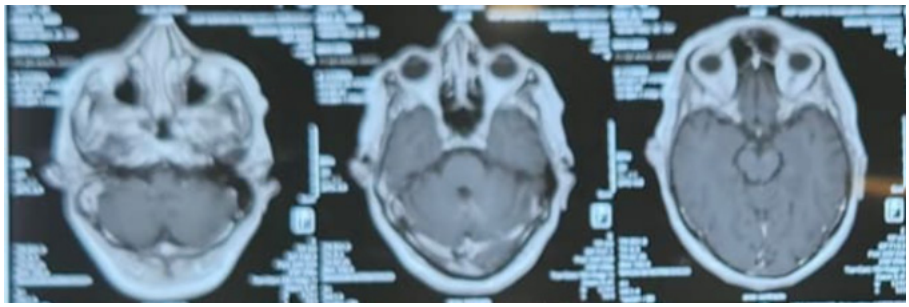
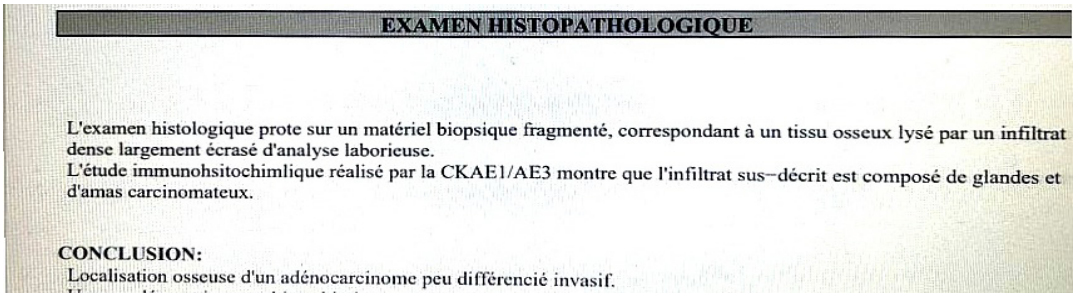


Figure 4: Cerebral MRI, axial and sagittal T1 sequences after gadolinium injection. Right petrous apex lesion with T1 isointensity, peripheral enhancement after injection, measuring 30 × 14 mm, without intracranial extension.

Histopathological examination was performed on fragmented biopsy material corresponding to bone tissue lysed by a dense, largely crushed infiltrate, difficult to analyze. Immunohistochemical study using CKAE1/AE3 cytokeratin showed that the infiltrate consisted of glands and carcinomatous clusters. The final diagnosis was a bone localization of a poorly differentiated invasive adenocarcinoma, with additional immunohistochemistry (PSA, NKX3.1) still pending to confirm the prostatic origin **(Figure 5)**.



EXAMEN HISTOPATHOLOGIQUE

L'examen histologique prote sur un matériel biopsique fragmenté, correspondant à un tissu osseux lysé par un infiltrat dense largement écrasé d'analyse laborieuse.
L'étude immunohsitochimlique réalisé par la CKAE1/AE3 montre que l'infiltrat sus-décrit est composé de glandes et d'amas carcinomateux.

CONCLUSION:
Localisation osseuse d'un adénocarcinome peu différencié invasif.

Figure 5: Histopathological report concluding a bone localization of a poorly differentiated invasive adenocarcinoma, with positive CKAE1/AE3 immunostaining demonstrating an infiltrate composed of glands and carcinomatous clusters.

The patient was jointly referred to the oncology and radiotherapy departments for further management: adjustment of systemic androgen-deprivation therapy and discussion of external-beam radiotherapy for analgesic and decompressive intent on the skull base.

Discussion

Epidemiology

Temporal bone metastases are rare and account for less than 2% of all tumors of this anatomical region[4,5]. In adults, the three most frequent primary tumors are, in decreasing order, breast, lung and prostate cancers[5,7]. In elderly men, prostate adenocarcinoma is the most frequent cause of skull base metastasis, but its involvement of the temporal bone remains exceptional, with only about twenty cases documented in the international literature[5-7].

Dissemination to the temporal bone is mainly hematogenous, via Batson's vertebral venous plexus, which allows tumor cells originating in the pelvis to reach the skull base directly without passing through the pulmonary filter[3,8]. Invasion may also occur by direct extension from an adjacent lesion or by leptomeningeal spread[5].

Clinical presentation

Symptoms are nonspecific and result from the involvement of adjacent anatomical structures. In published series, the most frequent presenting signs are: hearing loss (sensorineural, conductive or mixed), tinnitus, otalgia, vertigo, peripheral facial nerve palsy and, less commonly, otorrhea or posterior fossa syndrome[5-7]. Facial nerve palsy, often recurrent in its course, has been described as the sole presenting feature in approximately ten cases[6,9].

As early as 1981, Greenberg et al. described five clinical syndromes related to skull base metastases: orbital, parasellar, middle fossa, jugular foramen and occipital condyle[10]. The jugular foramen syndrome combines hoarseness, dysphagia and palsy of the IXth, Xth and XIth cranial nerves, whereas the occipital condyle syndrome manifests as unilateral occipital headache radiating to the mastoid and ipsilateral hypoglossal palsy[10,11]. Our case illustrates combined involvement of the jugular foramen and occipital condyle, although at a stage where the clinical neurological symptoms remained subtle, dominated by headache and cochleovestibular manifestations.

Radiological features

Cross-sectional imaging is the cornerstone of diagnosis. High-resolution temporal bone CT allows accurate characterization of the osteolytic nature of the lesion, its extension to the different bony walls of the temporal bone (petrous apex, tympanic cavity, mastoid cells, jugular foramen, facial nerve canal, hypoglossal canal) and its impact on adjacent bony structures[12,13]. MRI, performed with T1, T2, FLAIR, diffusion-weighted sequences and gadolinium-enhanced imaging, provides an evaluation of soft tissues, intracranial extension, cranial nerve involvement and vascular impact (compression of the jugular bulb, thrombosis)[13].

Metastases typically appear isointense on T1, with variable T2 signal and show post-contrast enhancement that is often heterogeneous or peripheral, as in our case. The main radiological differential diagnoses include: jugulotympanic paraganglioma (highly vascularized, with intense contrast uptake and a "salt and pepper" appearance on MRI), chondrosarcoma and chordoma (midline location, calcifications), meningioma (dural implantation), Langerhans cell histiocytosis (young patients), cholesteatoma of the petrous apex (non-enhancing), as well as mucocele and infectious lesions[12,13].

Pathological diagnosis

Histological confirmation is mandatory. Biopsy can be performed via a transtympanic endoscopic approach, a retroauricular approach with mastoidectomy (as in our case) or a percutaneous CT-guided approach depending on the location[5,9]. Histological examination typically shows glandular trabeculae and clusters with adenocarcinomatous morphology, within a variably dense bone infiltrate, frequently crushed and therefore challenging to analyze. Immunohistochemistry is decisive: epithelial markers (CKAE1/AE3, cytokeratins) confirm the carcinomatous nature, while positivity for PSA (prostate-specific antigen) and, especially, NKX3.1 confirms the prostatic origin, with a sensitivity approaching 99% and a specificity above 99% for NKX3.1 in metastatic tumors[14].

Therapeutic management

Treatment of temporal bone metastases from prostate cancer is palliative and requires a multidisciplinary approach. It most often combines external-beam radiotherapy for analgesic and decompressive purposes, with standard systemic therapy for metastatic prostate cancer: hormonal therapy with LH-RH analogues or next-generation androgen receptor pathway inhibitors (abiraterone, enzalutamide), chemotherapy with docetaxel or cabazitaxel in case of castration resistance and, more recently, targeted radioligand therapy with lutetium-177 PSMA in later lines of treatment[5,15,16]. Surgical resection has no role, given the diffuse and frequently multifocal nature of metastatic bone disease.

The prognosis remains poor: the occurrence of a temporal bone metastasis reflects advanced disease and the reported median survival is less than 12 months in most series[5,7,9]. Nevertheless, improvements in systemic therapies and stereotactic radiotherapy techniques have recently modified this prognosis.

Conclusion

Temporal bone metastases from prostate cancer represent a rare but not exceptional entity, whose misleading clinical presentation may reveal metastatic progression. Any new otological or neurological symptom occurring in a patient with a history of prostate cancer should prompt radiological evaluation with temporal bone CT and cerebral MRI. Diagnostic confirmation is histological, often after surgical biopsy and immunohistochemistry (CKAE1/AE3, PSA, NKX3.1) plays a decisive role. Therapeutic management is palliative and multidisciplinary, combining external-beam radiotherapy, hormone therapy and chemotherapy, with promising perspectives offered by novel targeted therapies.

Declarations

Consent

Written informed consent was obtained from the patient for publication of this case report and the associated images, which have been anonymized.

Conflicts of interest

The authors declare no conflicts of interest related to this work.

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