Rare Presentation of Metastatic Prostate Adenocarcinoma as a Meningioma Mimic

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Abstract

Background  Dural lesions in the anterior skull base may occur secondary to benign or malignant pathology that may be difficult to differentiate on imaging. Detailed clinical evaluation in many cases will narrow the differential diagnosis. In spite of using all the available information, in certain cases the underlying etiology of a lesion remains unclear.

Participant  We report a rare case of metastatic prostate adenocarcinoma to a meningioma in a 67-year-old-man who presented with progressive confusion and mental status alterations with no prior history of malignancy. Neuroimaging revealed a large anterior skull base lesion.

Results  The lesion was surgically resected, and histopathology revealed a collision tumor, in which prostate adenocarcinoma was found admixed with a World Health Organization grade I meningioma.

Keywords
► collision tumor
► anterior skull base
► meningioma
► prostate cancer

Conclusion  Anterior dural skull base lesions can be either benign or malignant. Although infrequently reported, a benign-appearing dural-based lesion may be a manifestation of an underlying malignancy, and a thorough clinical, radiologic, and pathologic examination may be necessary, especially in the elderly.

Introduction

Collision tumors occur when two pathologic tumor types are intermingled and identified histopathologically from a single specimen.1,2 Metastasis from prostate adenocarcinoma commonly involves the bone and other organ systems.

Case Report

A 67-year-old-right-handed man presented with a history of progressive confusion, abulia, and urinary incontinence. Magnetic resonance imaging revealed a 5.3 × 4.8 × 4.2 cm extra-axial lesion arising from the anterior skull base with associated vasogenic cerebral edema in the frontal lobes bilaterally (►Fig. 1). On examination, although his affect was dull, there was no focal neurologic deficit. On digital rectal examination, his prostate was enlarged and firm. The prostate-specific antigen (PSA) level was 49.6 ng/mL (normal: 0–4). Given the large extra-axial tumor, with vasogenic edema, mass effect, and neurologic symptoms, a bifrontal craniotomy was performed, with gross total resection of the lesion.3

Histopathology revealed the presence of a World Health Organization grade I meningothelial meningioma. An adenocarcinoma was dispersed widely within the meningioma.
Discussion

Intracranial dural-based metastasis is relatively uncommon in comparison with the intraparenchymal disease burden. Several neoplastic and nonneoplastic lesions have been reported to mimic both the radiologic imaging and clinical features of meningioma.4

Prostate carcinoma has a predilection to metastasize to the bones and lymph nodes; central nervous system involvement is rare.5 Brain metastases in patients with prostate cancer range from 0.2% to 0.63%. Dural-based metastases from prostate carcinoma have been reported and may mimic meningioma.5–8 Most uncommon is metastasis of a prostatic adenocarcinoma to a preexisting intracranial tumor, a so-called collision tumor. Döring reported the first case of prostate cancer metastasizing to a meningioma in 1975, subsequently followed by other reports.9,10 There has been a report of metastatic prostate cancer presenting with a subdural hematoma.11 Elderly men with known prostate carcinoma have been reported to have intracranial metastatic disease on routine surveillance and autopsy.12 Intra-axial brain metastases are a rare and typically terminal event in patients with advanced systemic metastases. Dural spread could be hematogenous or because of direct colonization of tumor cells, with another speculation being the increased tumor vascularity from the anterior skull base meningioma potentially having a higher predilection for metastatic cells to develop within a primarily meningeal tumor.13 Only five cases of prostate cancer collision tumors have been reported previously.14 All the previous reports of a collision tumor have been in patients with a known prostate cancer that had already metastasized widely and systemically prior to central nervous system involvement. To our knowledge, this is the

(Fig. 2): the adenocarcinoma cells stained immunohistochemically for antibody directed against the PSA.2

Fig. 1 Neuroimaging. (A) Sagittal postcontrast T1-weighted magnetic resonance (MR) scan demonstrating a contrast-enhancing mass (white arrow) arising from the anterior skull base with mass effect on the adjacent frontal lobes. (B) Coronal MR demonstrating a contrast-enhancing lesion (arrowhead) with heterogeneous areas within the tumor mass appearing to arise from the region of the olfactory groove characteristic of an olfactory groove meningioma. (C) Axial contrast-enhancing MR images demonstrate lobulated lesion (white arrowhead) with mass effect and adjacent perilesional edema (black arrowheads). (D) Axial fluid-attenuated inversion recovery images revealing significant bifrontal edema (arrowheads).
first case where metastatic prostate adenocarcinoma has presented initially and primarily with neurologic symptoms. Although collision tumors are rare, the imaging characteristics make it difficult to discern different dural-based pathology, with malignant lesions posing as benign tumors thus mandating a thorough clinical, laboratory, and imaging work-up of elderly patients.

References

Fig. 2 Microscopic views of the lesions. (A) A World Health Organization grade I meningothelial meningioma with a focally disordered architecture and psammoma bodies was present. (B) Nests of atypical-appearing cells consistent with metastatic adenocarcinoma were observed within the meningioma. (C) The metastatic carcinoma showed focal areas of glandular differentiation. (D) Focal positive immunoreactivity with antibody to prostate-specific antigen was observed in the metastatic tumor. The metastasis additionally demonstrated positive staining with antibodies to chromogranin and neuron-specific enolase, indicative of neuroendocrine differentiation. The tumor stained with cytokeratin 7 and 20 antibodies and did not stain with antibody to thyroid transcription factor 1.