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Leptomeningeal Metastasis from Castration-resistant Prostate Cancer: A Case Report

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Abstract

Leptomeningeal carcinomatosis is an exceedingly rare complication of prostate cancer. However, its incidence may increase as prostate cancer patients survive longer with new therapies even after castration resistance. The clinical presentation is varied and nonspecific. The diagnosis of leptomeningeal metastatic disease is made either by identifying malignant cells in cerebrospinal fluid or by gadolinium-enhanced MRI. Several treatments, including radiation therapy, intrathecal chemotherapy, steroids, debulking surgery, and best supportive care have been suggested. Although, the prognosis is extremely poor not exceeding weeks. We report a case of leptomeningeal carcinomatosis in a 64-year-old patient with metastatic castration-resistant prostate cancer with a review of the literature.

Keywords: Leptomeningeal carcinomatosis • Prostate cancer • Radiation therapy

Introduction

Leptomeningeal Metastases (LM) are defined as the infiltration of the leptomeninges, including the pia mater, arachnoid, and subarachnoid space, from a primary solid tumor. They are relatively uncommon events with dismal prognosis and Overall Survival (OS) ranging from weeks to months, regardless of the type of treatment [1,2]. Solid tumors with a significant risk of leptomeningeal recurrence are melanoma, non-small-cell lung cancer, and breast cancer. The incidence of LM is increasing due to the improvement of tools for diagnosis and monitoring and the availability of more active targeted therapies to control systemic disease while being less effective in CNS due to the presence of the blood-brain barrier [1,2]. Therefore, the prognosis is poor and patients do not survive more than a few weeks to months following the diagnosis of leptomeningeal metastases [3]. We here report a case of LM originating from Metastatic Castration-resistant Prostate Cancer (mCRPC) who presented with headache and weakness.

Case Presentation

A 64-year-old male was first diagnosed with de novo metastatic Gleason 6 (3+3) adenocarcinoma in 2018, with extensive bony disease on bone scan without any visceral metastasis. The patient was initially treated with gasoreline resulting in a good biochemical response (nadir = 2.6 ng/ml *vs.* 100 ng/ml) and symptomatic improvement, then he developed a castration resistance 14 months later. He ultimately progressed through several therapies, including 6 cycles of docetaxel, 4 cycles of etoposide and cisplatinum, both associated with zoledronic acid, and most recently, to abiraterone plus prednisone. He presented with a headache and gradually worsening generalized weakness for 4 weeks, associated with vomiting one week before admission. On physical examination, the patient has a performans status 2 with gait disturbance, he was not very reactive to questions and he was lethargic with no cranial nerves nor neurologic deficits. Laboratory workup on admission showed a prostate-specific antigen level of 1891ng/ml with a testosterone dosage of 0.05 ug/ml. MRI of the brain showed a right extra-axial process associated with a diffuse contrast-enhanced and nodular pachymeningeal thickening (Figure 1). CT scan showed additional liver metastasis.

Given the patient's poor performans status and the risk of engagement, no lumbar punction nor intrathecal chemotherapy was administered. We decided then to perform whole-brain radiation. The patient received EBRT to a total dose of 20 Gy delivered in 5 fractions to the whole brain followed by a boost to the macroscopic disease of 9 Gy in 3 fractions after immobilization by a head and neck thermoplastic mask (Figure 2). Treatment was well tolerated and the patient experienced no radiation-induced acute toxicity. After a month, the patient's performans status improved with regression of the symptoms and the PSA level to 100 ng/ml. MRI showed also regression of the right parieto-occipital extra-axial lesion and pachymeningeal thickening (Figure 3). Hence, we decided to rechallenge Abiraterone low dose (the patient being unfit to receive cabazitaxel). Unfortunately, the neurological symptoms progressed leading to the patient's death three months later.

Discussion

Leptomeningeal (LM) from genitourinary tract cancers is very rare, especially in prostate cancer. It is usually a late manifestation of systemic disease. Common symptoms include headache, seizures, sensory deficits, gait abnormalities, altered mental status, and memory problems. Other possible symptoms are nausea and vomiting, fatigue, pain, incontinence, and confusion. The most commonly affected cranial nerves are III, V, VI, VII, and VIII [3-6] . Contrast-enhanced magnetic resonance is the first mandatory diagnostic procedure. MRI findings include sulcal and foliar enhancements, linear ependymal and cranial nerve root enhancement but also leptomeningeal enhancing nodules. Of note, about 20–30% of patients with LM have a normal or false-negative MRI [1]. The sensitivity of MRI is equivalent to that of CSF with rates around 76%, but the specificity of CSF examination appears to be less specific than MRI. Nevertheless, MRI provides strong support in the diagnosis of LM, especially in patients with cancer who have negative results on CSF cytology [7]. After exclusion of hydrocephalus, diagnosis of LM is confirmed by malignant cells in the CSF. It should be noted that the first cerebrospinal fluid sample obtained is only diagnostic in about 50% and should be repeated

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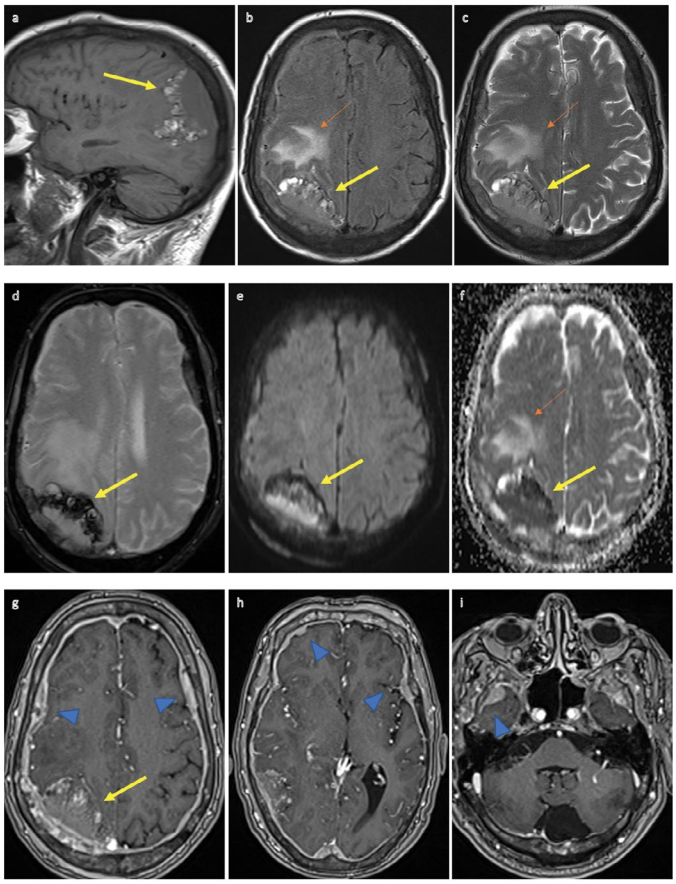

Figure 1. Cerebral MRI with T1-weighted sagittal (a), axial FLAIR (b), T2 SE (c), T2 EG (d), diffusion (e) with ADC cartography (f), and 3D T1 (g-i). EG images showing right extra-axial process with bony attachment (yellow arrow) and a heterogeneous signal containing hemorrhagic zones in hyper signal T1 and T2. This process compresses the cerebral parenchyma and is responsible for vasogenic edema (orange arrow). It is also associated with a diffuse contrast-enhanced and nodular meningeal thickening (arrowheads).

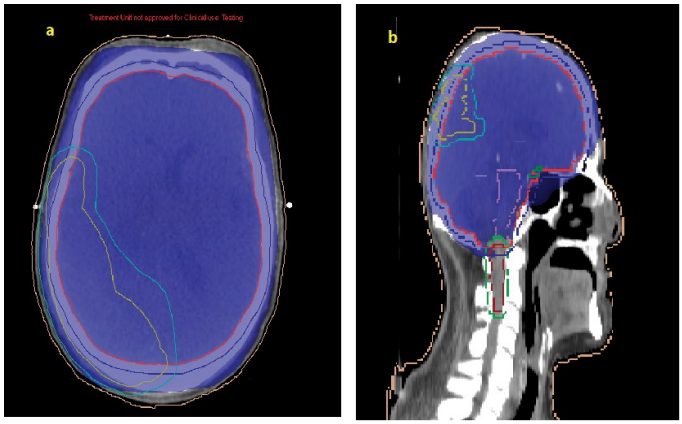

Figure 2. Axial view of the dose distribution to the whole brain (a) and sagittal view of the dose distribution of the boost (b).

if deemed necessary [1,6]. This should be taken into consideration especially when the risk of engagement is high. Many reports suggest that when there is strong evidence of LM on MRI, cytological confirmation is not necessary, and physicians can proceed with the treatment in this context [6,8,9].

Treatment for the leptomeningeal disease is palliative and aims to improve quality of life and maintain neurological function. There is currently no standard treatment owing to the rare cases reported in the literature, and treatment choices are largely based on expert opinions, local practices, and patients' performans status. Primary hormonal treatment, corticosteroids, radiotherapy, intrathecal chemotherapy, debulking surgery, and best supportive care, are all

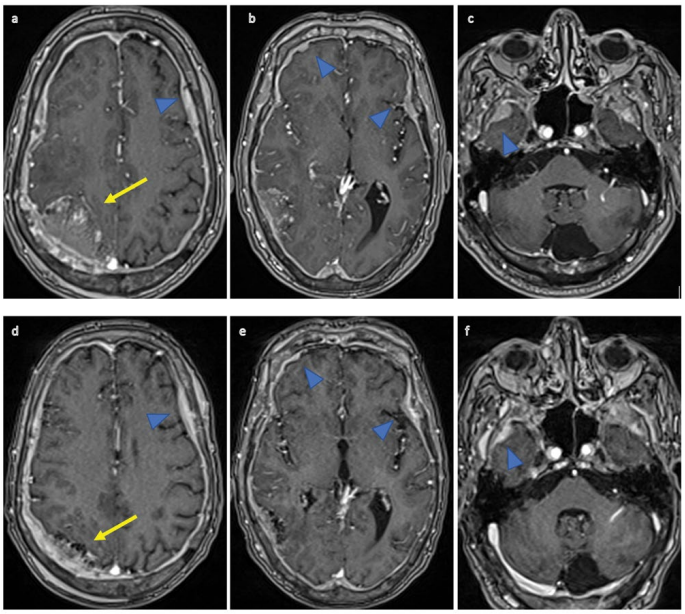

Figure 3. Regression of the right parieto-occipital extra-axial lesion (yellow arrow) and meningeal thickening (arrowheads) in the post-radiotherapy MRI (d-f) as compared to initial MRI (a-c).

treatment options but are generally associated with poor results and survival not exceeding a few weeks. Intrathecal chemotherapy is effective for most LM from solid tumors, especially for 1–2mm layers or freely circulating tumor cells, but might not be suitable for controlling disease in the plaque or nodular phase. Furthermore, prostate cancer has relatively low sensitivity to available intrathecal agents and may not be the best option of choice [1,9].

Local radiotherapy is strongly recommended in case of nodule or mass. Also, it may be an option for the treatment of CSF flow blocks [1]. Lin and al, reported 4 cases of LM of mCRPC, three of them were treated with radiation therapy leading to symptom relief initially [9].

Craniospinal irradiation to the whole neural axis has not been evaluated in this setting and is generally not recommended because of the associated toxicity such as bone marrow suppression and nausea [5,9]. Because of the extent of intracranial macroscopic disease that presents our patient, we choose to include the whole brain in the treatment volume with a boost delivered to the parieto-occipital mass. The patient experienced no remarkable acute toxicity and achieved symptom relief after the radiation therapy. The prognosis for prostate cancer patients with LM is poor with survival ranging from a few weeks to months following the diagnosis [4,5].

Conclusion

LM from prostate cancer is a rare and devastating complication that occurs later in the course of prostate cancer with an extremely poor prognosis. Prompt recognition of metastases may allow for early palliative treatment and interventions. There is no standard treatment for LM. However, radiation therapy may be of some benefit in symptom relief and improving quality of life.

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