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Isolated Brain Metastasis as the Initial Presentation of Prostate Adenocarcinoma: A Case Report and Literature Review

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Abstract | **Case Report**

Prostate cancer is among the most common cancers in men worldwide and usually metastasizes to the pelvic lymph nodes, axial skeleton, and lungs [1,2]. Brain metastases from prostate cancer are rare and are most often identified during post-mortem examinations [3]. According to previous reports, the incidence of brain metastases in prostate cancer patients ranges from 0.16% to 0.63% with median survival following their detection ranging from 2.8 to 4.5 months [4,5]. Consequently, it was long assumed that the brain parenchyma was capable of resisting the development of metastatic lesions originating from prostate adenocarcinoma cells. [6]. Given that most patients with prostate cancer who develop brain metastases may initially present with or without urinary tract symptoms, these lesions are frequently overlooked in clinical practice or may be misinterpreted on routine head CT scans as meningiomas, abscesses, or subdural hematomas [7]. In this report, we present a rare case of a patient with prostate adenocarcinoma who developed brain metastasis. We also provide a review of the existing literature on brain metastasis in prostate cancer, highlighting its principal characteristics.

Keywords: Prostate cancer, Brain metastasis, Adenocarcinoma, Androgen deprivation therapy, Rare malignancy, Case report.

INTRODUCTION

Brain metastases are a common complication of systemic malignancies, most frequently arising from lung, breast, melanoma, renal, and colorectal cancers. In contrast, intracranial involvement from prostate adenocarcinoma is uncommon, typically occurring late in the disease course and often in association with widespread systemic metastases. Even rarer is the presentation of an isolated brain metastasis as the initial clinical manifestation of prostate cancer. Such atypical presentations pose diagnostic and therapeutic challenges, may delay identification of the primary tumor, and raise important questions about tumor biology, patterns of spread, and optimal management strategies.

This report describes a patient whose first clinical and radiologic sign of malignancy was a solitary intracranial lesion subsequently identified as metastatic prostate adenocarcinoma. We present the clinical course, diagnostic workup, histopathologic and immunohistochemical findings, and treatment decisions for this case. To contextualize the rarity and clinical implications, we complement the case with a systematic review of the literature addressing incidence, pathophysiology, diagnostic pitfalls, imaging and pathological characteristics, and outcomes of patients with brain metastases from prostate adenocarcinoma—particularly those presenting as an isolated intracranial lesion. Our aim is to increase awareness of this uncommon presentation, highlight key diagnostic clues, and discuss management considerations to inform clinicians who encounter similar diagnostic dilemmas.

CASE PRESENTATION

We report the case of an 84-year-old man presenting with a three-month history of severe headache. The symptoms had an abrupt onset and were characterized by a diffuse, throbbing pain without clinical evidence of increased intracranial pressure, although they were partially alleviated by analgesic therapy. The patient was subsequently referred to a neurosurgeon, and brain MRI revealed a secondary

metastatic lesion. Extensive diagnostic investigations were undertaken to identify the primary tumor.

Although, the patient had a remote history of lower urinary tract symptoms specifically pollakiuria and dysuria that had been effectively managed with alpha-blocker therapy, resulting in good clinical improvement. However, he had no history of infection, hematuria, lower-limb edema, or lower back or bone pain. He also denied palpitations, diaphoresis, cough, dyspnea, or chest pain. On general examination, he was conscious, non-pale, and non-icteric. Abdominal examination was unremarkable. Digital rectal examination revealed a soft, grade II prostate with a preserved median sulcus and a freely mobile rectal mucosa.

The results of the complete blood count, liver function tests, electrolyte panel, lipid profile, renal function tests, and HbA1c were all within normal limits. Furthermore, metastatic screening including chest X-ray and chest computed tomography revealed no abnormalities.

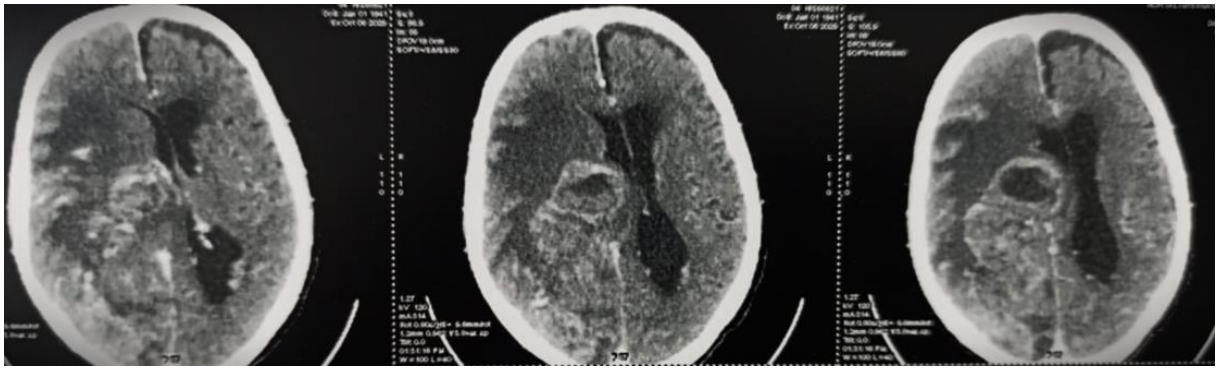

Fig.1. A CT scan showing evidence of supratentorial and infratentorial brain lesions, the largest of which is in the right parietal lobe with edema and subfalcine herniation.

KUB ultrasound revealed no signs of bladder outlet obstruction and an enlarged prostate 33 X 48 X 56 mm.
PSA results was 12 ng/dl.

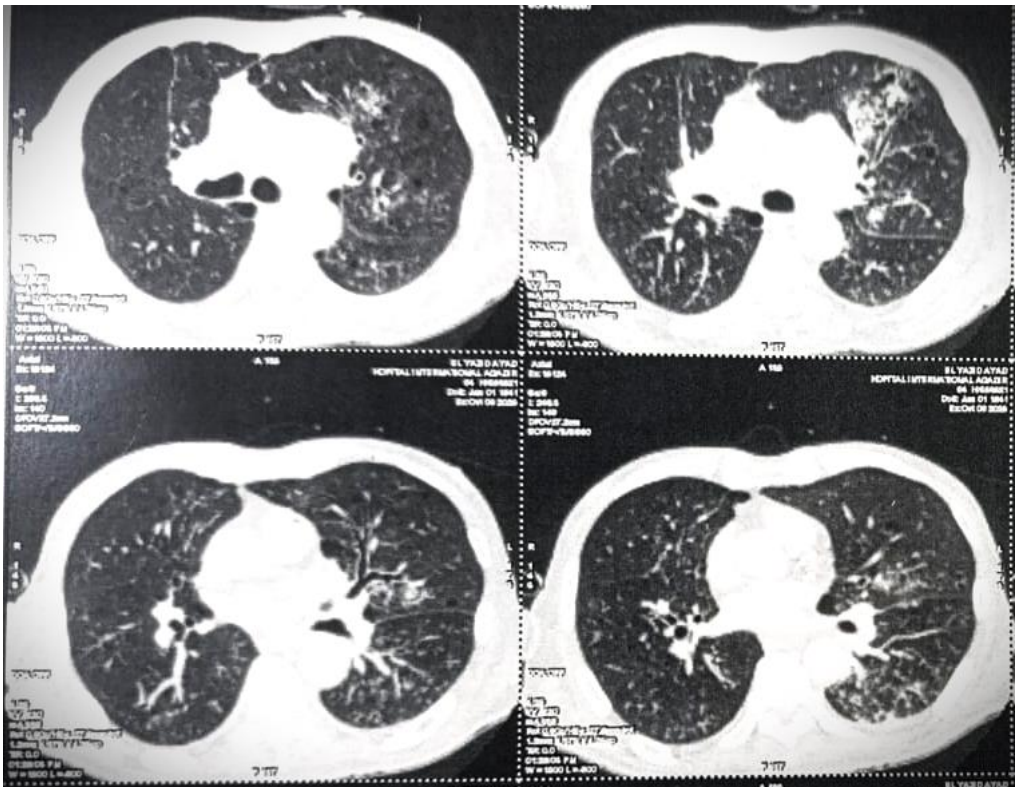

Fig.2. A thoraco-abdomino-pelvic CT scan revealed bilateral pulmonary micronodules and infectious-appearing pulmonary foci, as well as conglomerated mediastinal and peritoneal lymphadenopathy, most consistent with secondary [metastatic] involvement.

Accordingly, the patient underwent a core prostate biopsy, which demonstrated adenocarcinoma of the prostate with a Gleason score of $3 + 3 = 6$, corresponding to ISUP grade 1.

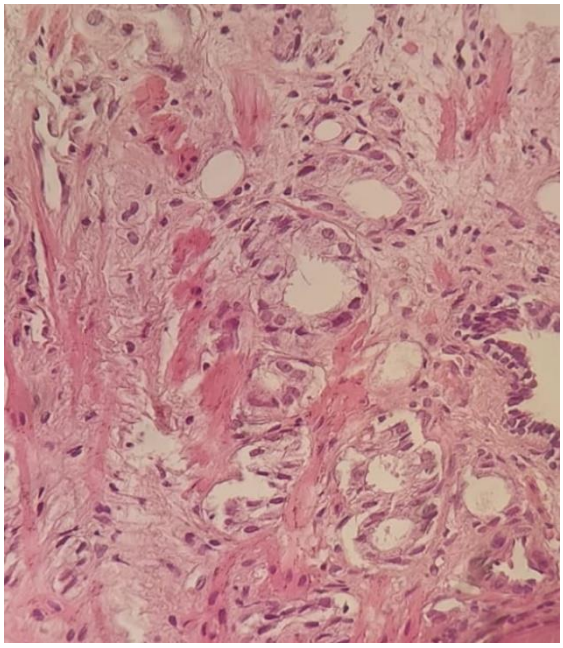

Fig.3. Photomicrograph of a core needle biopsy of prostate shows a small focus of prostate adenocarcinoma. Malignant glands have an infiltrative growth pattern and exhibit malignant cytological features, such as enlarged nucleoli.

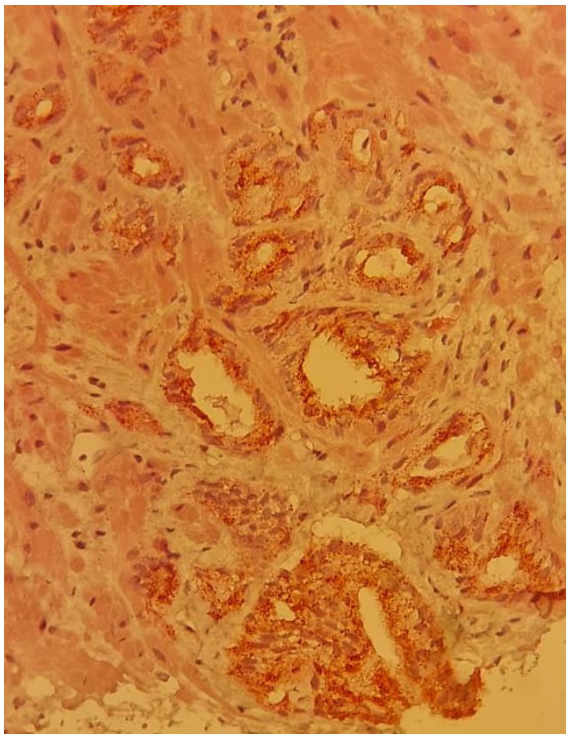

Fig.4. Photomicrographs of immunochemistery cocktail confirms the absence of basal cells highlighted by the absence of the basal cell markers as p63 antibody. The neoplastic glands show a strong immunostaing for AMACR- P504S antibody.

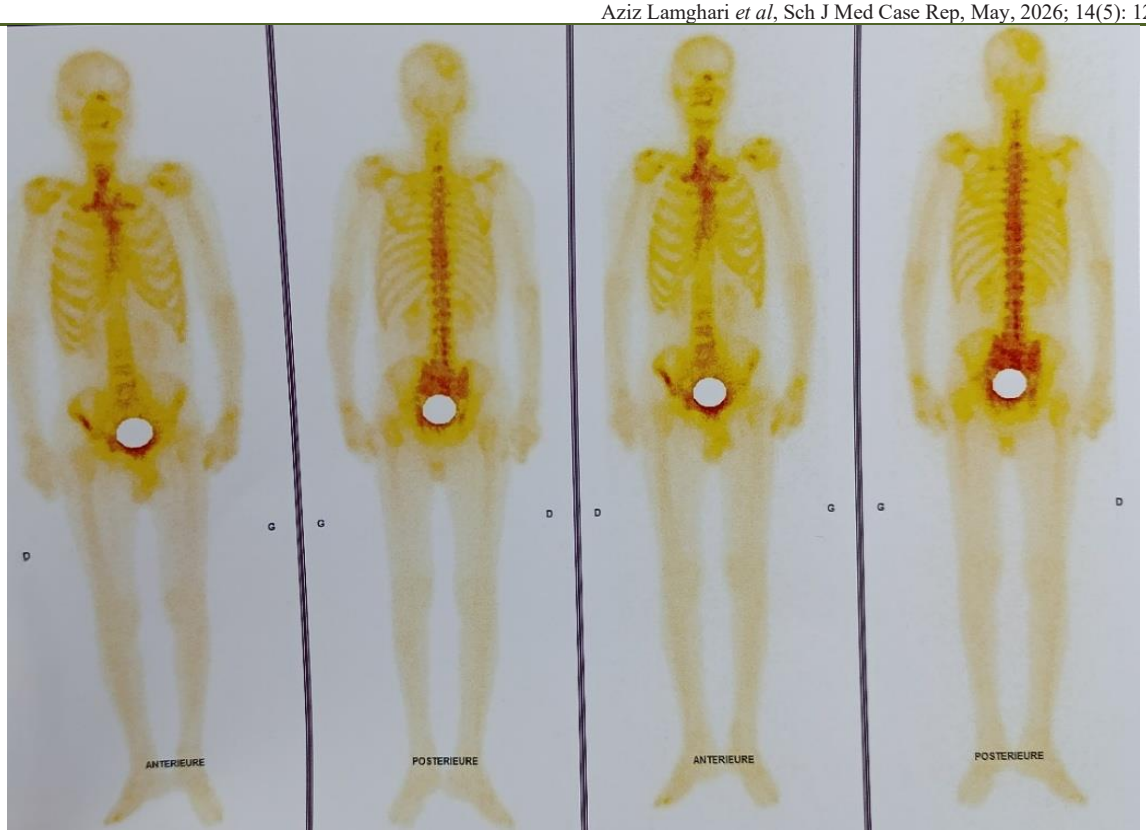

Fig.5. Bone scintigraphy did not reveal any evidence of secondary osseous lesions.

Given the discrepancy between the histology of the prostate biopsy and the negative results of the staging examination, apart from the brain lesions.

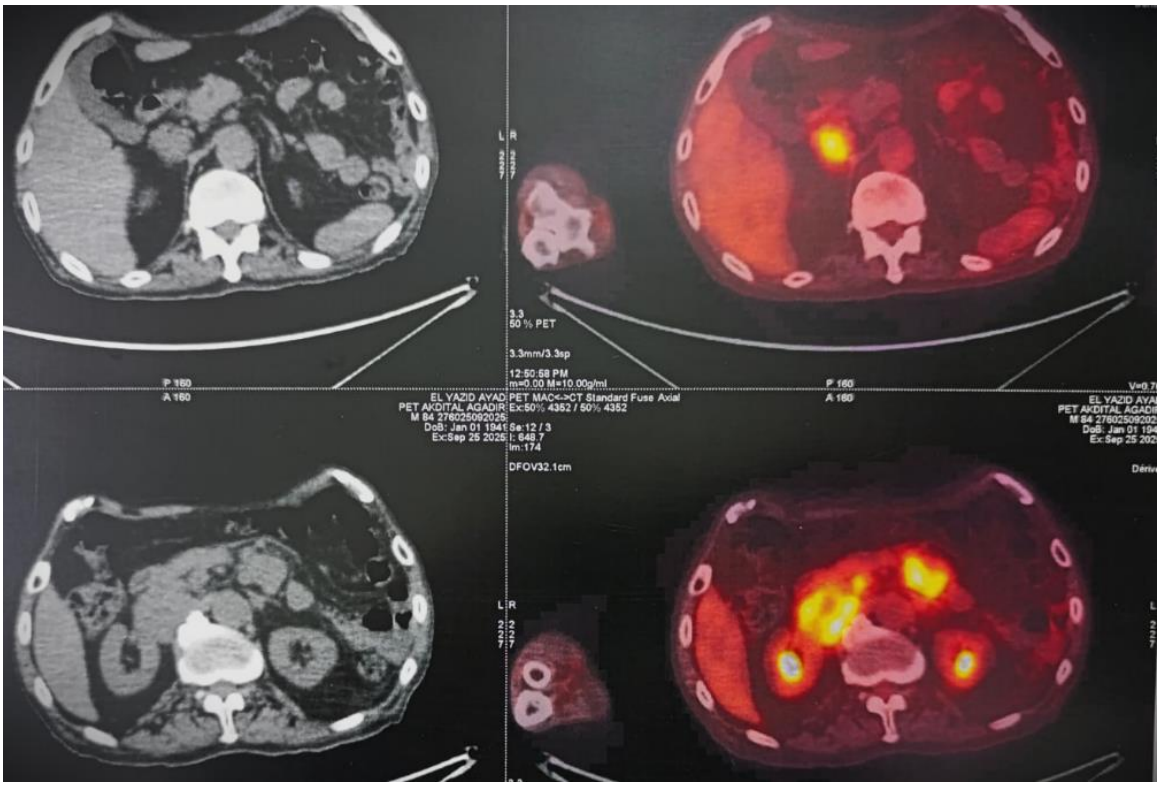

Fig.6. A PET-Scan 18F- FDG was performed, which confirmed: Three hypermetabolic intraperitoneal abdominal tissue lesions associated with interaortocaval lymphadenopathy and a cluster of mediastinal lymphadenopathies.

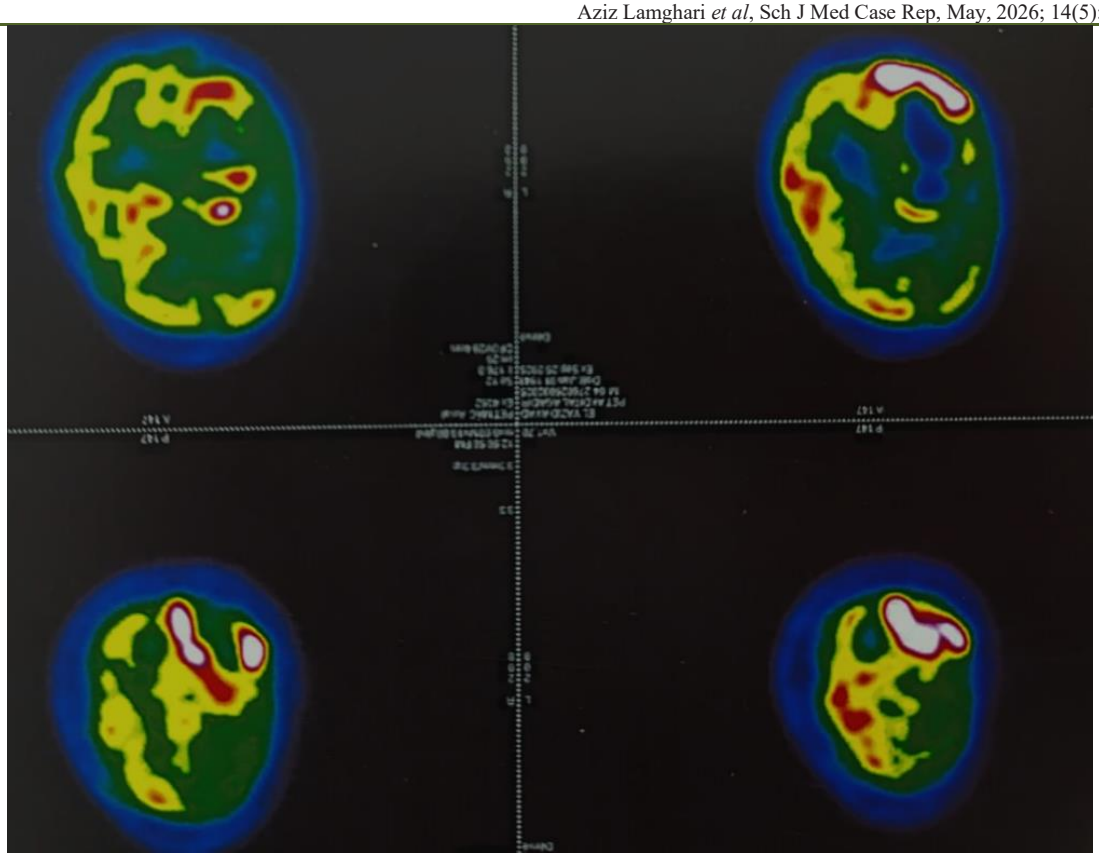

Fig.7. Multiple brain lesions suggestive of secondary locations were also observed.

TNM staging showed extra-prostatic extension bilaterally, metastatic inguinal lymph nodes brain mets with stage T3aN1M1 and diagnosis of advanced prostate cancer with metastases to the brain was made.

The patient was counseled regarding the available therapeutic options based on his performance status, and he made an informed choice. Following androgen deprivation therapy [ADT], his serum PSA decreased from 12 ng/mL to 0,2 ng/mL. One month later, his headache had completely resolved. Plans were made for chemotherapy and craniotomy with tumor resection if his symptoms had not improved. During subsequent follow-up, the patient remained free of headache and neurological deficits, and his PSA was undetectable. He was able to resume his normal daily activities.

CLINICAL DISCUSSION

Brain metastasis from prostate cancer is uncommon, and the reported proportion of intracranial metastases attributable to prostate cancer is infrequently updated. [8,9] Brain metastases occur more frequently in patients with neuroendocrine variants of prostate cancer, whereas they remain rare in those with conventional adenocarcinoma [10,11].

Similar to other solid tumors, prostate cancer [PC] is a heterogeneous disease composed of cells exhibiting diverse molecular profiles and genetic alterations [12]. This biological heterogeneity likely contributes to the variable patterns of brain metastatic spread observed in PC. As prostate cancer typically disseminates through lymphatic or hematogenous routes, primary tumor cells may reach the brain microenvironment through several mechanisms [7]. Tumor embolism may bypass the pulmonary circulation by traversing a patent foramen ovale. Alternatively, malignant cells may pass through the pulmonary capillary bed, enter the left heart, and subsequently disseminate via the arterial circulation to the brain.[7]

Furthermore, some studies have proposed that prostate cancer cells may disseminate to the brain through secondary spread from a pre-existing metastatic site, such as the liver or lungs [7]. Brain metastasis may also occur as a late event, potentially facilitated by immune system deterioration or disruption of the blood–brain barrier [13].

Thus, these disseminated cells may remain dormant for an indeterminate period, and patients who eventually develop brain involvement from such quiescent cells often present with more favorable clinical outcomes [14,15]. In contrast, in patients who develop secondary brain metastases originating from a prior metastatic site, the tumor cells being genetically unstable and poorly differentiated may have undergone multiple molecular alterations before reaching the brain microenvironment. These patients typically present with more advanced disease and acute clinical symptoms [16].

Distinguishing primary intracranial lesions from metastatic prostate cancer can be challenging, particularly in cases presenting with a solitary lesion. Although MRI characteristics specific to prostate cancer metastases have not been comprehensively described in the literature, several reports have noted mixed cystic and solid features, ring-enhancing patterns, and hemorrhagic components on imaging [17].

Differentiating primary intracranial lesions from metastatic prostate cancer can be challenging, particularly when only a solitary lesion is present. Although MRI features specific to prostate cancer metastases have not been comprehensively characterized, several studies have described mixed cystic and solid components, ring-enhancing patterns, and hemorrhagic features on imaging [17]. The diagnosis of brain metastases in patients with prostate cancer may be difficult in the absence of neurological symptoms [7]. Despite recent advances in diagnostic modalities such as integrated positron emission tomography/magnetic resonance imaging [PET/MRI] systems utilizing prostate-specific membrane antigen [PSMA] with 18F- or 68Ga-labeled PET tracers symptomatic patients may present with a spectrum of clinical manifestations. These vary according to the location of the metastatic focus and include headache, seizures, and focal neurological deficits, along with more nonspecific symptoms such as confusion and memory impairment [7].

In our patient, prostate cancer was diagnosed at an advanced stage, after it had already disseminated, yet without producing overt neurological symptoms. In cases of metastatic prostatic disease involving the brain, biopsy of the intracranial lesion is generally not recommended. Histopathological confirmation from the primary tumor remains the preferred diagnostic approach; however, in patients with limited prognosis, invasive confirmation may not be necessary. Historically, treatment of solitary brain metastases has included radiation therapy often whole-brain radiotherapy [WBRT] followed by craniotomy with surgical resection [3].

In published cases of prostate cancer with brain metastasis, reported survival times typically range from 2.8 to 4.5 months after diagnosis [18,19]. However, survival has improved with advances in therapeutic strategies and diagnostic modalities [20,21,22]. Treatment approaches for brain metastases from prostate cancer mirror those used for other intracranial metastases and include corticosteroids, surgical resection, and radiotherapy, with stereotactic radiosurgery serving as an option particularly in cases of recurrence. These interventions have been shown to extend survival. In a retrospective ten-year study involving 31 patients with brain metastases from prostate cancer, median survival times of 4.6 months and 13 months were reported for those treated with stereotactic radiosurgery and combined radiotherapy plus surgical resection, respectively [21]. These findings are consistent with our case, in which the patient remains alive and clinically well after one year of follow-up. In contrast, patients who did not receive any form of treatment had a median survival of only one month [23].

Our patient was managed with androgen deprivation therapy and closely monitored for neurological manifestations. Over a 12-month follow-up period, no neurological symptoms developed. Although a follow-up brain MRI was planned at six months.

CONCLUSION

Brain metastasis from prostate cancer is rare. However, with recent advances in oncologic therapies, the life expectancy of patients with prostate cancer has increased substantially, which is expected to lead to a higher frequency of clinically detected brain metastases. Therefore, clinicians should consider intracranial metastatic disease in all men over 50 years of age with a history of malignancy who present with new neurological symptoms. When brain metastasis is suspected, gadolinium-enhanced MRI remains essential for confirming or excluding its presence and for monitoring disease progression. Furthermore, additional studies and case reports on prostate cancer brain metastasis are needed, as they provide valuable insight into the mechanisms underlying cerebral dissemination.

Declarations

Ethics approval and consent to participate

All authors equally contributed to the analysis and writing of the manuscript.

Consent for publication

Informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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