Case Report

ANNALS OF MEDICINE & SURGERY

OPEN

A case report on metastatic prostate cancer with normal PSA level diagnosed by immunohistochemistry and its management

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Introduction and importance: Metastatic prostate cancer (mPCa) is an advanced form of cancer that spreads beyond the prostate to distant organs such as bones and lymph nodes. While prostate-specific antigen (PSA) testing is commonly used for diagnosis, rare cases with normal PSA levels complicate detection. This case highlights the crucial role of immunohistochemistry (IHC) in diagnosing mPCa with normal PSA and its management in resource-limited settings.

Case presentation: A 63-year-old male presented with 2 months of persistent back pain without neurological deficits. Initial diagnostics, including PSA levels, CT, MRI, and TRUS guided prostate biopsy, revealed no prostate abnormalities. However, an MRI and Bone scan showed a suspicious vertebral lesion, and a subsequent biopsy confirmed malignancy. IHC demonstrated overexpression of alpha-methyl acyl-CoA racemase (AMACR), leading to the diagnosis of mPCa. The patient underwent bilateral subcapsular orchiectomy and received external beam radiotherapy (EBRT) to manage symptoms and control disease progression.

Clinical discussion: This case highlights the diagnostic challenges of mPCa in patients with normal PSA levels. Standard diagnostics, including imaging and biopsy, may fail to detect prostate cancer, making IHC, specifically AMACR, an essential tool for diagnosis. Early surgical intervention followed by EBRT offered significant symptomatic relief and disease control.

Conclusion: This case demonstrates the importance of IHC in diagnosing atypical presentations of mPCa with normal PSA. A multidisciplinary approach combining surgery and radiotherapy can improve outcomes and quality of life, even in resource-limited settings

Keywords: alpha-methylacyl-CoA racemase, immunohistochemistry, metastatic prostate cancer, prostate-specific antigen

Introduction

Prostate cancer (PCa) is the most common cancer in men worldwide[1]. The progress made in the treatment of primary tumors has now made way for metastasis prostate cancer to lead the rising mortality trend associated with prostate cancer[2]. The bones, lymph nodes, liver, thorax, kidneys and adrenal glands are the most common sites of metastasis, resulting in specific organ-related complications[3]. Commonly diagnosed via a combination of laboratory tests, imaging modalities and biopsy, metastatic prostate cancer can also present atypically remaining insidious to screening tests such as prostate-specific antigen (PSA) and transrectal ultrasound (TRUS)[4]. In such cases, immunohistochemistry (IHC) can be crucial in narrowing the diagnosis of unknown metastatic foci[5]. The prognosis for metastatic PCa (mPCa) is not optimistic[6]; however, numerous therapies have been introduced to ease the process of palliation. These include medical and surgical castration to reduce the androgen load, radiotherapies and chemotherapies[7]. Here, we present a rare case of a 63-year-old man with metastatic prostate cancer with normal PSA levels who was diagnosed via immunohistochemistry and its management at a tertiary care center.

Case summary

History

A 63-year-old man presented to our outpatient department with chief complaints of back pain for 2 months. The pain was insidious in onset, progressive, and did not aggravate upon movement or a change in posture. There was no history of fever, burning micturition, trauma, a palpable mass, changes in urinary urgency or frequency, or changes in bowel movements. His past medical and surgical history did not reveal anything significant. He had a 20-pack-year smoking history but had left smoking for 1 year. On examination, his vital signs were within normal limits. Systemic examinations of the respiratory, cardiovascular, abdominal, and neurological systems were conducted, but the findings were insignificant. There was no focal tenderness in the back region, and a straight leg raise test was also performed, which was negative. The patient revealed that he had taken pain medications and had undergone regular physiotherapy, but there was no improvement.

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Annals of Medicine & Surgery (2025) 87:364–371

Received 10 October 2024; Accepted 19 November 2024

Published online 9 January 2025

http://dx.doi.org/10.1097/MS9.0000000000002783

Investigations

X-ray of the lumbar spine in the anteroposterior and lateral views revealed no abnormalities. The patient was then advised to have a Computed Tomography (CT) scan of the abdomen, which revealed an approximately 15 × 12 × 12 cm well-defined heterogeneous soft tissue density mass involving all the segments of the right lobe of the liver. The lesion showed peripheral discontinuous nodular enhancement in arterial phase imaging with centripetal filling and progressive enhancement in subsequent phases. The lesion showed an exophytic component along the visceral surface of the liver and abutted the right kidney posteriorly. The branches from the right hepatic artery supplied the lesion. The prostate gland was normal in size, with an outline and attenuation, and no focal lesions were observed. An approximately 31 × 26 mm heterogeneously enhancing soft tissue density lesion was also noted involving the right side of the L1 vertebral body and the right pedicle, which destroyed the cortex. This lesion extended to the right paravertebral region (Fig. 1). On the basis of the CT findings, the lesion on the liver was suspected to be a hemangioma; however, the soft tissue density lesion in the vertebrae was more likely to be malignant. As a result, an MRI (magnetic resonance imaging) of the lumbar spine (post gadolinium) was performed. MRI also revealed a soft tissue mass (26 (transverse) × 36 (anteroposterior) × 38 (craniocaudal) mm in the L1 vertebral body on the right side. The pedicle and the posterior element were also involved. The mass extended into the surrounding soft tissue and infiltrated into the right psoas muscle. There was minimal epidural extension into the spinal canal. The lesion had a low signal intensity on the T1W image, heterogeneously high enhancement on the T2W image, and heterogeneous postgadolinium enhancement. The MRI findings reinforced the suspicion of a metastatic lesion (Fig. 2). A ^{99M}TC-MDP whole body bone scan was performed which revealed increased tracer uptake in the L1 vertebra confirming the osteoblastic nature of the tumor. A subsequent biopsy from the lesion of the L1 vertebra was performed. The specimen consisted of multiple pieces of gray–brown colored tissue. Microscopically, the section had clusters and cords of polygonal tumor cells. These cells had a moderate amount of granules to clear the cytoplasm. The nuclei were round to oval with small prominent nucleoli, and mild nuclear pleomorphism was noted. Further, these cells were arranged in tubular architecture and the basal cell layer was absent. This confirmed that the lesion was metastatic and highly suggested the prostatic origin of the tumor because of the absence of the basal cell layer (Fig. 3). Then, immunohistochemistry tests and immunoassays were performed. The results are presented in Tables 1 and 2. The baseline test reports are mentioned in Table 3.

Based on AMACR positivity (Fig. 4) and other negative markers, prostate cancer was the most likely diagnosis. A TRUS-guided biopsy of the prostate was also performed, but it did not reveal any malignant lesions. Elevated AMACR was highly unusual because the patient's PSA levels were normal, and the CT scan and TRUS biopsy also revealed no lesions on the prostate gland. AFP and CEA levels were also normal ruling out other carcinomas. Combining the fact that the lesion was osteoblastic and metastatic, the biopsy revealed the absence of basal cell layer and histopathological features of carcinoma and that the IHC revealed AMACR positivity and other markers returning negative made a strong foundation for diagnosing prostate cancer. Further IHC tests with the use of NKX3.1, ERG, p63, and AR would have definitely confirmed the diagnosis. However, the patient's family refused further investigations.

Management

Oncology and surgery department consultations were performed for the patient, and a multidisciplinary approach was discussed. The patient was provided the diagnosis of metastatic prostate cancer after thorough counseling of his imaging and histopathological reports. The patient was advised to undergo further IHC tests to confirm prostate carcinoma however, the patient's family argued against it since the lesion was metastatic and there was already a strong reason to suspect its prostatic origin, they opted not to conduct further confirmatory tests because they were under a strong financial burden. They wanted to proceed with palliative therapy as soon as possible because further confirmatory tests would not change the patient's prognosis; and even if they were performed, the ultimate outcome would still be palliation. The surgery team decided to proceed with the available evidence, which was strong enough because in resource-limited countries like ours, it is not always possible to demand the best available tests to confirm a diagnosis. The patient was scheduled for subcapsular orchiectomy since the cancer had already metastasized, and treatment was shifted toward symptomatic management and palliation. It was concluded that subcapsular orchiectomy could rapidly lower testosterone levels and provide symptomatic relief of bone pain in the back and prevent further growth of the cancer. Intraoperatively, an incision was made in the midline of the scrotum to reveal the tunica albuginea bilaterally. The contents inside the tunica albuginea were removed, and the tunica albuginea was plicated. After gradual recovery, the patient was scheduled for palliative radiotherapy for bone metastasis. The patient received external beam radiotherapy (EBRT) at 20 Gy in 5 fractions in 5 days via a linear accelerator. Following radiotherapy, the patient was prescribed zoledronic acid (4 mg IV) dissolved in 100 ml NS for 30 min monthly. The patient has undergone regular follow-up, and there are no new issues except mild weakness in the bilateral limbs.

Discussion

The objective of our case report is to highlight the challenges of diagnosing and managing metastatic prostate cancer in resource-limited settings, particularly in atypical presentations where conventional diagnostic methods like PSA testing may fail. The strategic use of immunohistochemistry, even in the absence of more advanced diagnostic tools, can provide a definitive diagnosis when it is combined with findings from other diagnostic modalities. In settings where access to state-of-the-art diagnostics is constrained, relying on targeted, cost-effective methods can still yield accurate and actionable results, enabling affordable management and improving patient outcomes.

Metastatic prostate cancer is an advanced stage of prostate cancer in which cancer cells propagate beyond the prostate gland to other parts of the body, such as the bones (84%), lymph nodes (10.6%), liver (10.2%), and thorax (9.1%)[3]. Compared with PCa without metastasis, mPCa is more serious and life-threatening and requires comprehensive treatment and

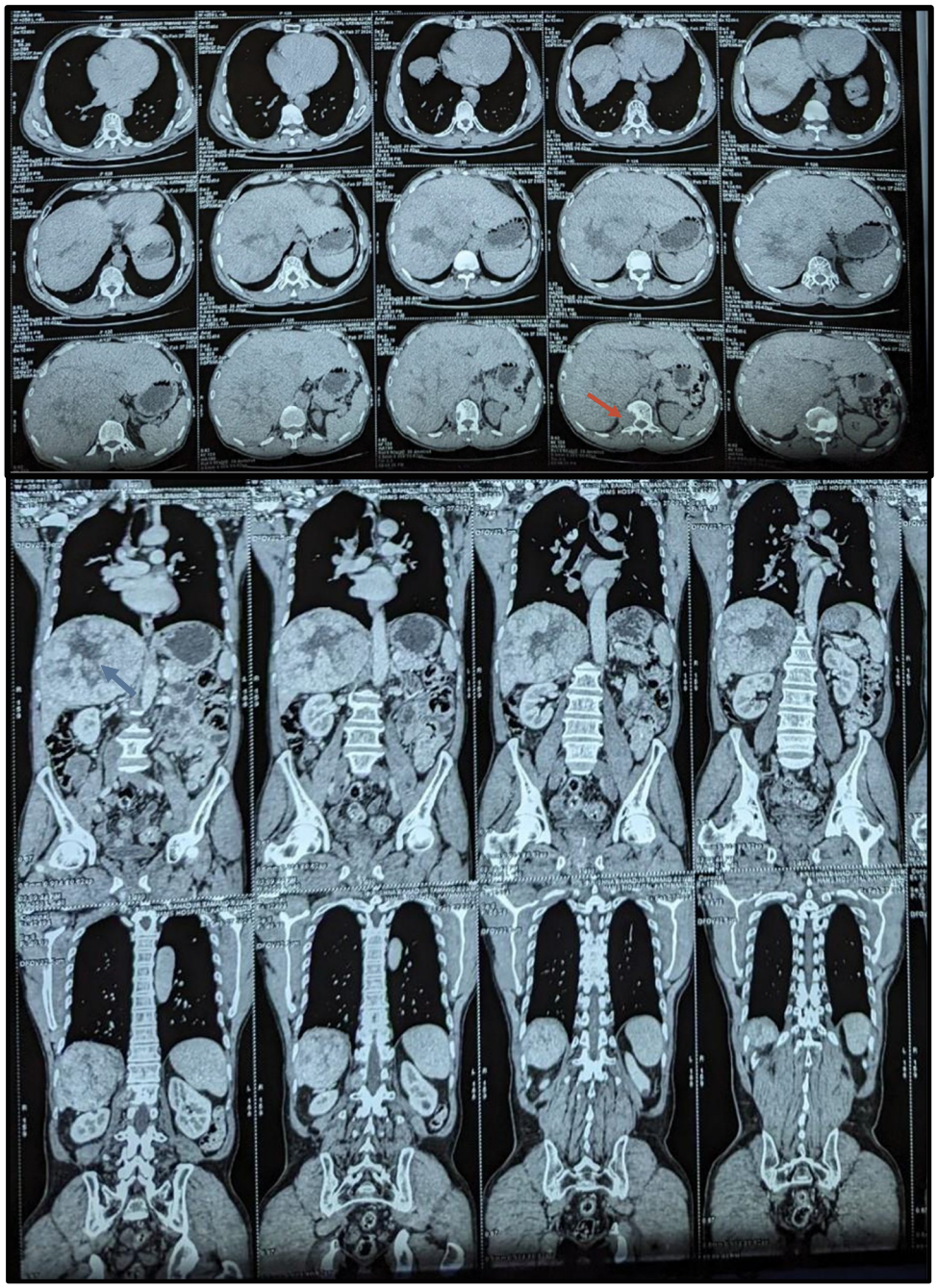

Figure 1. CT scan of the patient (up: transverse view; down: coronal view). An approximately 15 × 12 × 12 cm well-defined heterogeneous soft tissue density mass (blue arrow) involving all the segments of the right lobe of the liver. The lesion showed peripheral discontinuous nodular enhancement in arterial phase imaging with centripetal filling and progressive enhancement in subsequent phases. The lesion showed an exophytic component along the visceral surface of the liver and abutted the right kidney posteriorly. The branches from the right hepatic artery supplied the lesion. The prostate gland was normal in size, with an outline and attenuation, and no focal lesions were observed. An approximately 31 × 26 mm heterogeneously enhancing soft tissue density lesion was also noted involving the right side of the L1 vertebral body (red arrow) and the right pedicle, which destroyed the cortex. This lesion extended to the right paravertebral region.

management strategies. It is associated with increased symptoms, challenges in treatment, and impacts quality of life.

PCa is one of the most common cancers in men worldwide and is diagnosed around the age of 65–74[1]. Statistical analysis models show that there has been a 0.9% rise in average age-adjusted rates for new PCa each year from 2012 to 2021; however, the age-adjusted death rates have fallen on average by 0.5% per year from 2103 to 2022[8]. In 2024, it is predicted that there will be an estimated 299 010 new cases and 35,250 deaths due to PCa[8]. The estimated age-standardized rate of PCa was seen to be the highest in Oceania (79.1/100 000 people) and North America (73.7), followed by Europe (62.1). The incidence

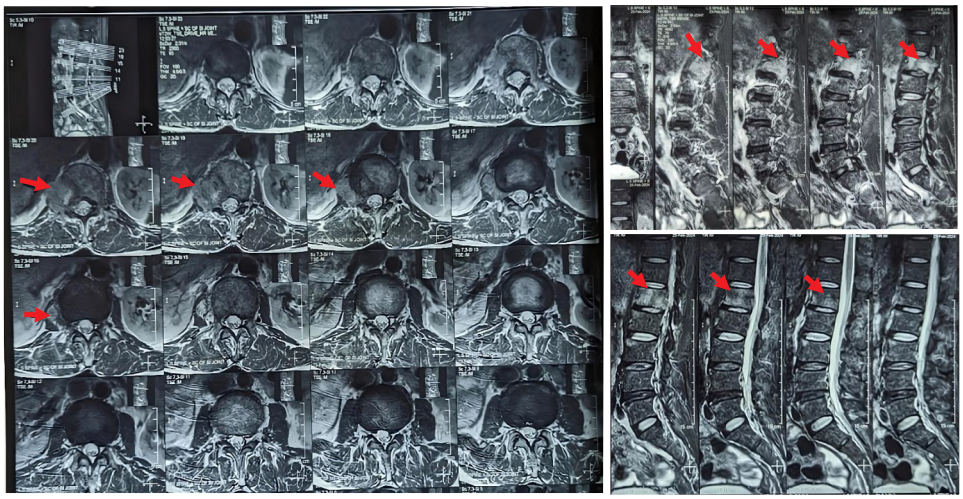

Figure 2. MRI of the lumbar vertebrae of the patient (left: coronal, right up and right down: sagittal section) showing a soft tissue mass (26 [transverse] × 36 [anteroposterior] × 38 [craniocaudal] mm) in the L1 vertebral body (red arrow) on the right side. The pedicle and the posterior element were also involved. The mass was seen extending into the surrounding soft tissue, and infiltration into the right psoas muscle. There was a minimal epidural extension into the spinal canal.

rates are lower in Africa (26.6) and Asia (11.5)[1]. This variation in the distribution of PCa incidence around the globe can be attributed, in part, to a variation in the level of PSA tested[9]. From 2004 to 2018, the SEER database recorded a total of 836 282 patients with PCa; among which 26 642 (56.5%) distant mPCa cases were reported in men aged 45 to 74 years, and 20 507 (43.5%) cases were reported in men aged 75 years or older[10].

PCa is an adenocarcinoma and is most common in the peripheral zone[11]. Studies have demonstrated that prostate cancer carcinogenesis resembles organogenesis embryologically and that androgen receptor signaling plays a crucial role in the progression and survival of these cancer cells[11,12]. Androgenic hormones such as testosterone and dihydrotestosterone (DHT) bind to androgen receptors and initiate nuclear translocation of the receptor, initiating a cascade of events that ultimately leads to the transcription of genes that regulate proliferation, apoptosis and functional differentiation[12]. Another important mechanism of tumor progression and aggressiveness of PCa is the epithelial-to-mesenchymal transition, which is commonly referred to as lineage plasticity[13]. Eventually, these cancerous cells multiply and spread to the surrounding prostate tissue, where they form a tumor. They then invade other organs (metastases), with the most common sites being the bones, lymph nodes, liver, thorax, brain, kidney and adrenal glands[3]. The survival of cancer cells also depends upon transcription factors and proteins. Normal prostate cells accumulate zinc. Zinc acts as an antiproliferative agent and induces apoptosis in abnormal cells, and its levels are regulated by the transporter protein ZIP1. These transporter proteins also act as tumor suppressors. In cancerous prostate cells, the gene encoding ZIP1 is silenced; thus, these cells lack the zinc transporter, leading to the absence of zinc in the cells[11]. RUNX2 is a transcription factor that prevents apoptosis in cancer cells. It also contributes to the development of prostate cancer[11].

MPCa presents with a wide range of clinical features depending on the spread of cancer to various parts of the body. Prostate cancer largely metastasizes to the bone and this has been

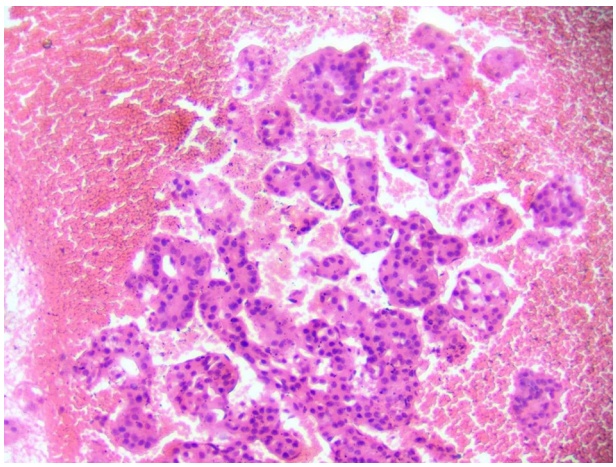

Figure 3. Sections show closely packed tubular glands lined by single layer of cuboidal epithelium with moderate cellular pleomorphism, eosinophilic to clear cytoplasm, vesicular nuclear chromatin and prominent nucleoli. Mitotic figures are infrequent.

Table 1

Immunohistochemistry results of the biopsy from the vertebral lesion of the patient.

IHC markers	Result
Inhibin	Negative
Synaptophysin	Negative
CK7	Negative
CK20	Negative
CD10	Negative
Glypican-3	Negative
AMACR	Positive

IHC, immunohistochemistry; AMACR, alpha methyl acyl CoA racemase; CK, cytokeratin.

Table 2

Immunoassay test results of the patient.

Immunoassay test	Value (ng/mL)	Normal range (ng/mL)
Alfa-feto protein (AFP)	3.42	0–10
CEA	3.38	<5
PSA total	3.97	<4

The following interpretations were made from these results:

· Inhibin: A negative result rules out adrenal cortical carcinomas and sex cord-stromal tumors.

· Synaptophysin: A negative result suggests that the tumor is not neuroendocrine in origin.

· CK7 and CK20: Negative results are less specific but can help narrow the differential diagnosis. For example, many lung, breast, and gastrointestinal tumors are positive for one or both of these markers.

· CD10: A negative result suggests that the tumor is not a renal cell carcinoma, acute lymphoblastic leukemia, or endometrial stromal sarcoma.

· Glypican-3: Negative results rule out hepatocellular carcinoma and some testicular germ cell tumors.

Table 3

Relevant lab details of the patient.

CBC	
Hemoglobin (HB)	(10.6) g/dL
Platelet count	(139,000)/cumm
White blood cell (WBC) count	(4, 780)/cumm
Red blood cell (RBC) count	(3.40) mill/mm^3
Basophil	0.4%
Eosinophil	2.3%
Monocyte	5.0%
Lymphocyte	13.4%
Neutrophils	78.9%
Absolute basophils	(0.02) thou/mm^3
Absolute eosinophils	(0.11) thou/mm^3
Absolute monocytes	(0.24) thou/mm^3
Absolute lymphocytes	(0.64) thou/mm^3
Absolute neutrophils	(3.77) thou/mm^3
Mean platelet volume (MPV)	(11.3) fL
Red cell distribution width (RDW)	(12.0)%
MCHC	(32.9) g/L
Mean cropuscular hemoglobin (MCH)	(31.2) pg
Mean corpuscular volume (MCV)	(94.7) fL
Packed cell volume (PCV)	(32.2)%
RFT	
Urea	26 mg/dL
Creatinine	0.88 mg/dL
Na^+/K^+	140/4.1 mmol/L
Urine R/M/E	Color: light yellow
	Crystal: nil
	Cast: nil
	Epithelial cells: 0–1/HPF
	RBC: Nil/HPF
	WBC: 1–2/HPF
	Sugar: nil
	Protein: nil
	Reaction: acidic
	Transparency: clear
Total protein	5.80 g/dL
Albumin	3.50 g/dL

attributed to cancer cell osteomimicry. MINDIN, an extracellular matrix protein, has been shown to induce this osteomimicry, however, the exact mechanism is yet to be elucidated[14]. Carrion et al[15] postulated that MINDIN downregulates the expression of NHERF-1, a scaffold protein that is responsible for enhancing a variety of cellular processes including cell proliferation and migration. Fatigue (73%), urinary symptoms (63%), sexual function symptoms (62%), and bone pain (52%) were the most commonly reported symptoms of metastatic prostate cancer reported by Drudge-Coates et al[16]. in their survey, which included patients with advanced prostate cancer. Pain preceded the diagnosis in 73% of the patients in their study. Holmstrom et al[17] reported similar findings in their survey involving 22 patients, with fatigue, bone pain, anxiety, stress, depression and interference with daily activities being the most commonly reported symptoms. Since bones, particularly the pelvis, ribs, and spine, are among the most common sites of metastasis[18], patients often experience persistent pain in these areas. Pathologic fractures can also occur due to the weakening of bones. When cancer spreads to the vertebrae, it can compress the spinal cord, causing neurological deficits, weakness, numbness and even paralysis in severe cases[19]. It can also cause obstructive urinary symptoms such as difficulty urinating, retention of urine, and a weak urine stream[19]. Our patient had similar complaints of back pain for 2 months and did not have any neurological deficits or bowel/bladder dysfunction.

The diagnosis of MPCa involves a combination of laboratory tests, imaging studies, and biopsy confirmations. The primary tests used are PSA tests, digital rectal examination (DRE) and TRUS-guided biopsy. More than 60% of asymptomatic PCa patients are diagnosed with a normal DRE and elevated PSA[20]. However, it is not unusual for patients with prostate cancer to have a normal or lower PSA level. Thompson et al[4]. demonstrated that 6%–27% of men with PSA levels less than or equal to 4.0 ng/mL had biopsy-proven prostate cancer. Therefore, the level of 4.0 ng/mL, which was previously used as a cutoff value to specify normal or abnormal levels of PSA in the blood, has now been rendered obsolete[21]. Further, It has been suggested that PCa with normal or low PSA levels are more likely to be benign/low grade and confined, and have a Gleason score <7 and negative margins[22]. However, our patient presented with MPCa in bones with normal PSA levels, which is unusual. Thus, the use of serum PSA as a screening test has faced significant criticism. Additionally, PSA testing struggles to accurately distinguish between low-risk, intermediate-risk, and high-risk aggressive forms of the disease[23]. Imaging techniques such as X-rays, CT scans, MRIs, and PET scans can be used to aid in the diagnosis of PCa. Bone X-rays can be performed to look for bone metastases. CT and MRI can help visualize the extent of cancer spread to bones, lymph nodes, and other organs. PET scans with radioactive contrasts can demonstrate the activity of cancer cells[11]. MRI via dynamic contrast-enhanced and diffusion-weighted imaging alongside T2W imaging can be used accurately to detect PCa. This technique has 89% sensitivity and 73% specificity[24]. CT has limited sensitivity, ranging from 25% to 78%, for detecting prostate cancer, particularly in the lymph nodes, but has greater specificity, ranging from 77% to 98%[25]. Because of the low sensitivity of CT imaging, PET-CTs using various radiotracers are being employed. Three radiotracers have been approved for use in PET–CT for prostate cancer: C-choline, F-fluciclovine and F-sodium fluoride[26]. C-choline PET-CT can detect varying degrees of metastases on the basis of the site of disease (local,

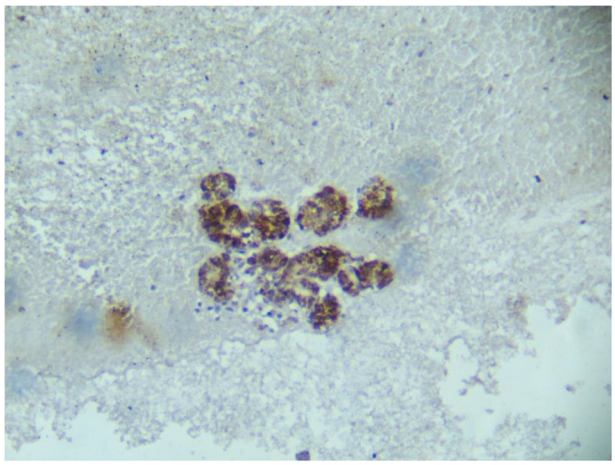

Figure 4. AMACR (alpha methyl acyl CoA racemase) positive stained cells from the vertebral biopsy specimen.

nodal, or distant) and the PSA level and has a sensitivity ranging from 38% to 98% and a specificity ranging from 50% to 100%. F-fluciclovine PET-CT has 89%–100% sensitivity and 67% specificity for detecting metastatic prostate cancers. The use of F-sodium fluoride PET-CT is limited to bony metastases and has a sensitivity of 87%–89% and a specificity of 80%–91%[26]. Our patient demonstrated heterogeneous soft tissue density in the vertebrae on both CT and MRI; however, the prostate appeared normal on CT. Because of financial constraints and the lack of PET–CT facilities at our center, a biopsy of the vertebral metastatic lesion was performed as a means to determine the source of the metastasis. A TRUS biopsy is the standard test to confirm the diagnosis, but it still has a false negative rate of 15%–46% on the basis of the needle position relative to the tumor location[20]. Similarly, our patients' TRUS biopsy results also revealed no malignant findings.

Immunohistochemistry (IHC) markers are mainly used in patients with atypical and suspicious lesions to rule out/validate a diagnosis of malignancy[5]. The types of IHC markers used are mentioned. Synaptophysin is an integral-membrane glycoprotein of presynaptic vesicles and is a specific and highly sensitive marker for neural or neuroendocrine tumors of low and high grades of malignancy. Cytokeratin is expressed in specific subtypes of ovarian, breast and lung adenocarcinomas. CK7 is useful for distinguishing between transitional cell carcinoma and prostate cancer, which are positive and negative, respectively. The combined use of CK7 and CK20 is useful for distinguishing ovarian carcinomas from colonic adenocarcinomas. Glypican 3 is a sensitive marker for certain testicular cancers and is overexpressed in hepatocellular carcinoma, ovarian clear cell carcinoma, pancreatic acinar cell carcinoma, and urothelial carcinoma but is not expressed in prostate cancer. CD10, also known as membrane metalloendopeptidase (MME), marks the cell membrane and is expressed in approximately 75% of precursor B-cell acute and small subsets of T-cell acute lymphoblastic leukemia. It is also used in the evaluation of lymphomas, most often diffuse large B-cell lymphoma $^{(NOS)}$[27]. AMACR is a peroxisomal enzyme and is involved in the metabolism of branched-chain fatty acids. This enzyme is overexpressed in metastatic prostate cancer cells compared with normal prostate cells[28]. The AMACR is a sensitive (82%–100%) and relatively specific (70%–100%) marker for prostate cancer[27]. AMACR was the only positive marker in our patient, and as a result, our patient was diagnosed with metastatic prostate cancer.

The treatment for metastatic PCa is multifaceted and aims to control the spread of the disease, alleviate symptoms, and improve quality of life[7]. Androgen deprivation therapy (ADT) is the gold standard treatment, both as a curative intent for localized disease with intermediate to high risk and palliation for metastatic prostate cancer[7]. Huggins and Hodges in 1972 demonstrated that androgen deprivation is an excellent method to achieve temporary control of PCa growth[29]. ADT leads to a decrease in testosterone production in the body and can be performed surgically via bilateral orchiectomy, or medical castration can be achieved via LHRH agonists, LHRH antagonists, and antiandrogens[7]. A retrospective study involving 83 patients who underwent subcapsular orchiectomy for metastatic PCa demonstrated a median tumor remission time of 29 months and an overall survival time of 36 months[30]. A few hours postsurgery, testosterone levels rapidly decrease, which plays a crucial role in providing symptomatic relief to patients with ostealgia. It also significantly reduces the risk of fractures or compression of the spinal cord due to metastatic prostate cancer. In a study comparing bilateral total orchiectomy with bilateral subcapsular orchiectomy, the latter was as effective as the former in achieving castration[22]. Bilateral subcapsular orchiectomy offers significant advantages in terms of patient satisfaction and acceptance because it helps reduce the psychological impact associated with an empty scrotum[31]. Thus, castration seems to be an effective therapy in terms of both curation and palliation. However, the majority of patients usually progress into an evolved state of cancer known as castration-resistant prostate cancer (CRPC)[32]. In CRPC, the androgen receptor (AR) pathway is activated and becomes the main driver of castration resistance. For these patients, AR-directed therapies need to be provided, which include the suppression of androgen biosynthesis and the blockade of AR[32]. Chemotherapy can be used to kill rapidly dividing cancer cells. Common chemotherapeutic drugs for metastatic prostate cancer are docetaxel and cabazitaxel[7]. Targeted therapies are also used where targeted drugs that inhibit the PARP or androgen receptor pathways are used. These drugs target specific molecular pathways involved in cancer progression. Radiation therapy is used to control pain and other symptoms caused by bone metastasis. Lutz et al[33] reported that a radiotherapy dose of 30 Gy in 10 fractions, 24 Gy in 6 fractions, 20 Gy in 5 fractions, or a single fraction of 8 Gy can provide excellent pain control. Bisphosphonates also act effectively in combination with radiotherapy[33]. While a single fraction can be more convenient for patients, a fractionated longer course has a lower incidence of repeat treatment to the same side. Our patient also received fractionated radiotherapy (20 Gy) in 5 fractions. Immunotherapy, such as Sipuleucel-T, stimulates the immune system to identify, attack, and kill cancer cells.[7] Treatment plans are highly individualized depending on the patient's overall health, extent of metastasis, previous treatments, and preferences. Our patient initially underwent bilateral subcapsular orchiectomy and was later treated with EBRT.

The prognosis of metastatic prostate cancer varies widely depending on factors such as the extent of metastasis, the aggressiveness of the cancer, the response of the patient to treatment

and the patient's overall health. PCa patients who are receiving treatment at early stages have a 5-year survival rate of 97%, whereas the 5-year survival rate of patients with metastatic prostate cancer is approximately 30%[6]. Despite the many advances in treatment, metastatic prostate cancer is incurable. However, with effective treatment, patients' quality of life and survival rate can be improved.

Our case has several strengths, including the effective use of immunohistochemistry (IHC) in combination with other lab reports to diagnose metastatic prostate cancer despite normal PSA levels and inconclusive TRUS biopsy results. The involvement of a multidisciplinary team ensured comprehensive management through surgical and radiotherapeutic interventions, providing symptom relief and disease control. Additionally, the approach was cost-effective, focusing on essential diagnostic and therapeutic measures within the patient's financial constraints. However, the method had its weaknesses, such as the inability to perform further confirmatory IHC tests like NKX3.1 or AR markers due to financial limitations, leaving a slight margin for diagnostic uncertainty. Relying on a vertebral biopsy rather than a direct prostate biopsy limited the direct evaluation of the prostate, and the absence of advanced imaging techniques like PET-CT restricted the assessment of the disease's full extent. These limitations highlight the challenge of balancing resource constraints with the need for diagnostic precision, emphasizing the importance of optimizing care delivery in resource-limited settings.

Conclusion

In conclusion, this case highlights the diagnostic challenges and complexities associated with metastatic prostate cancer, particularly in patients with normal PSA levels and atypical presentations. The use of immunohistochemistry, and the detection of AMACR positivity, was crucial in confirming our diagnosis when conventional methods, such as TRUS biopsy and PSA testing, were inconclusive. The multidisciplinary approach, including subcapsular orchiectomy and palliative radiotherapy, provided significant symptomatic relief. This case emphasizes the need for comprehensive diagnostic evaluations in metastatic prostate cancer, which has been rising in incidence in recent years.

Ethical approval

Not applicable.

Consent

Written consent was obtained from the patient for publication of the patient's details.

Sources of funding

None.

Author's contribution

All the authors read and approved the final manuscript.

Conflict of interest

None.

Research registration unique identifying number (UIN)

Not applicable.

Guarantor

Kritick Bhandari.

Provenance and peer review

Not commissioned, externally peer-reviewed.

Data availability statement

All data generated or analyzed during this study are included in this published article.

Statement

This article has been written in line with the SCARE guidelines.[34]

Acknowledgments

We would like to express our sincere gratitude to the Department of Oncology, and Surgery of our institute for their invaluable contributions to the management of this patient. Their expertise and collaborative efforts were crucial in the diagnosis and treatment process, and their insights greatly enhanced the quality of care provided.

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