Metastatic Prostate Cancer Presenting as a Posterior Mediastinal Mass: A Rare Presentation

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September 14, 2022

Abstract

A 68-year-old African American male who presented to the emergency department with back and abdominal pain. Imaging showed a posterior mediastinal mass interposed between the carina, the left mainstem bronchus, and the descending thoracic aorta. Biopsy of the mass favored a metastatic prostate carcinoma, which is an extremely rare presentation.

Metastatic Prostate Cancer Presenting as a Posterior Mediastinal Mass: A Rare Presentation

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Disclosures: The authors have nothing to disclose

Funding: No financial support

Competing Interests: The authors declare that they have no competing interests

Acknowledgments: Not applicable

Abstract

A 68-year-old African American male who presented to the emergency department with back and abdominal pain. Imaging showed a posterior mediastinal mass interposed between the carina, the left mainstem bronchus, and the descending thoracic aorta. Biopsy of the mass favored a metastatic prostate carcinoma, which is an extremely rare presentation.

Keywords: Prostate, Prostatic Neoplasms, Mediastinal neoplasms, African American, Back Pain

Background

According to American cancer society estimates for 2022, there will about 268,490 new cases of prostate cancer and about 34,500 prostate cancer related deaths in the United states[1]. Patients can have variable symptoms on presentation, including anemia, paralysis from metastasis to the spinal cord, bone pain, and kidney failure from ureteral obstruction. Prostate-specific antigen (P.S.A.) and transrectal ultrasound-guided

biopsy remain the best diagnostic tools, although the use of P.S.A. level for screening remains controversial.[3,4] About 12% of all prostate cancer has already spread to regional lymph nodes at the time of diagnosis, with 5% of cases having distant metastases. The 5-year survival rate is only 29.8% in distant metastatic disease.[1] Skeletal, lung, and liver metastases are the classic site of tumor spread, with other sites being quite rare. Our case is unique, as it presented as a posterior mediastinal metastatic mass.

History of Presentation

A 68-year-old male with a past medical history of hypertension but not compliant with medication presented to emergency department (E.D.) with complaints of back and abdominal for the past 2 months. The patient described the pain as dull, localized to the paraspinal region, non-radiating, with no aggravating factors, and partially relieved on taking ibuprofen. He also noticed having unintentional weight loss and excessive fatigue for the past year. His pain worsened, prompting him to come to the E.D. He denies having fever, chills, night sweats, chest pain, nausea, vomiting, diarrhea, constipation, or a history of cancer.

On examination, his vital signs were recorded as; blood pressure (B.P.) of 223/135 mmHg, a heart rate (H.R.) of 104 beats per minute (B.P.M.), a respiratory rate of 18 cycles per minute (C.P.M.), and oxygen saturation of 99% on room air. On musculoskeletal examination, there was no paraspinal or spinal swelling inspection, and no paraspinal or spinal tenderness on palpation. Respiratory and cardiovascular examinations were normal. His initial working diagnosis was hypertensive urgency, and he was initially treated with intravenous labetalol 20 mg pushes but was switched to a nicardipine drip at 5 mg/hr due to poor control.

Past Medical and Surgical History

The patient is hypertensive but is not compliant with antihypertensive medications. He was on metoprolol 25 mg every 12 hours and nifedipine extended-release 60 mg every 12 hours. He also has benign prostate hyperplasia, for which he took doxazosin 1 mg at bedtime. He was non-compliant to primary care physician visits and had not followed up in years. No surgeries were reported. He denies smoking cigarettes or consuming alcohol, but does smoke marijuana.

Family History

No family history of cancer reported

Investigations

His initial lab results showed a white blood cell (WBC) count of 3.9 K/uL, hemoglobin of 12.5 gm/dL, a platelet count of 187 K/uL, blood urea nitrogen (BUN) of 19 mg/dL, and creatinine (Cr) of 1.6 mg/dL. Other investigations showed an iron level of 57 ug/dl, total iron binding capacity (TIBC) of 227mcg/L, ferritin of 469.4 ng/dL, a lactate dehydrogenase of 286 U/L, an E.S.R. of 31mm/hr, a high sensitivity troponin of 30.8 ng/L, plasma total free metanephrine of 75 pg/mL, plasma normetanephrine of 236 pg/mL, and total plasma metanephrine of 311 pg/mL.

An electrocardiogram (EKG) was done, which showed normal sinus rhythm.

A chest x-ray (C.X.R.) was also done, which showed a 1.3 cm nodule noted projecting over the fifth rib anterolaterally and in the right, mid lung field peripherally, widening of the mediastinum, and sclerotic 1 cm density in the right, and 1.1 cm density in the left humerus shaft [figure1].

Following this result, he underwent a computed tomography angiogram (C.T.A.) of the chest, abdomen, and pelvis (C.A.P.) to rule out aortic dissection. It showed a large enhancing soft tissue mass in the posterior mediastinum measuring 5.0 x 4.3 cm [figure 2 and 3]. The thoracic aorta was unremarkable. There was no pericardial or pleural effusion. The lungs were clear. There was no thoracic lymphadenopathy, and extensive retroperitoneal abdominal lymphadenopathy was noted. Additionally, mild left-sided hydronephrosis and hydroureter were seen due to lymphadenopathy.

A renal sonogram revealed minimal left kidney hydronephrosis,a pre-void bladder volume of 135.3 ccs, and prostatomegaly measuring 5.0 x 5.0 x 5.8 cm and weighing 75.6 gms. The right kidney was unremarkable

on the renal sonogram [figure 4].

Given the findings of the C.T.A., interventional radiology (I.R.) was consulted to biopsy the mass. The biopsy was technically challenging due to the overlying rib, the adjacent descending thoracic aorta, and intercostal vessels in the single available window. The needle was successfully positioned in the posterior border of the mass. No bleeding was incurred.

The biopsy results favored metastatic prostate carcinoma with positive immunostaining of NKX3.1 and positive immunohistochemistry of PD-L1 CPS [Figures 5 and 6].

Management

His symptoms improved with conservative medical management, and he was discharged with instructions to follow up with a hematologist-oncologist on an outpatient basis for further treatment of his mediastinal lesion.

Discussion

Prostate cancer is the third leading cause of cancer death in men.[4] It is the most common non-cutaneous cancer in men in the United States.[5] It typically affects males above the age of 50. However, younger age groups have been identified since using P.S.A. as a screening tool.[6] The five-year survival rate of localized and regional prostate cancer is more than 99%, whereas metastatic prostate cancer is only 30%[1]. At presentation, about 78% of the lesions are localized, and only 6% of cases present with metastasis.[1] Spread to other body parts usually occurs through lymphatic, hematogenous, or direct extension. Bone and lymph nodes are common sites among those with metastatic lesions, with pulmonary, hepatic, retroperitoneal, or abdominal metastasis very uncommon.[6] Mediastinal metastasis is exceedingly rare and can present with mediastinal adenopathy in 0.6% of the cases.[7] Only one other case of prostate cancer is reported presenting with a mediastinal mass.[8] In the United States, significant racial differences exist in the management of prostate cancer.[9]

Management initially begins with screening, either by measuring P.S.A. once a patient reaches above the age of 50 or performing a digital rectal examination to check for a palpable abnormality.[6] Confirmatory tests involve performing an ultrasound-guided transrectal prostate biopsy in a grid-like pattern and staging it based on Gleason's scoring.[10] Even though it is the standard of care, it can miss up to 28% of lesions and undergrade up to 17%.[11] Hence, newer diagnostic tests, biomarkers, and imaging modalities have been developed to improve diagnosis and risk stratification accuracy.[10] One such biomarker that is specific to primary prostatic adenocarcinoma is NKX3.1.[12,13], coded for by a gene located on chromosome 8p, which aids prostate development and acts as a tumor suppressor gene. Loss of this marker correlates with microenvironment inflammation, usually found in the early stages of prostate cancer.[14]

Studies have also shown that NKX3.1 interacts with M.Y.C., and a reduction in the former results in proliferative effects of M.Y.C., leading to cancer progression and relapse.[15] It can also distinguish between high-grade prostate cancer and high-grade bladder cancer.[16] It can be used to identify metastasis and has shown a sensitivity and specificity of 98.6 and 98.7% respectively in a cohort of metastatic tumors.[17] Studies have also shown that it is more specific than P.S.A. when identifying prostate cancer.[18] Based on our discussion, our case also has similar pertinent findings. Our patient was an African American male aged 68 years with a late diagnosis due to patient non-compliance; a biopsy of his metastatic mass was positive for NKX3.1. Even though a digital rectal exam or a prostate-specific antigen due to the absence of genitourinary symptoms and a different presentation, a diagnosis was confirmed based on immunohistochemistry. Since his primary symptoms improved his condition was further managed on an outpatient basis with the hematology-oncology team.

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Consent Statement:

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy

Conflict Of Interest:

There are no conflicts of interest to declare