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Case report of prostate ductal adenocarcinoma presenting with hematuria

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Introduction

Prostate cancer is the most common malignancy in males, typically considered to have an indolent course. The World Health Organization 2016 classification divides epithelial tumors of the prostate into several subcategories including acinar and ductal adenocarcinoma. Almost all prostate cancers are acinar adenocarcinomas, developing from the glands themselves. Pure ductal adenocarcinoma originates in the ducts and represents 0.2–0.4% of all prostate cancers. It is usually mixed with the acinar subtype.[1] The incidence of ductal adenocarcinoma has been increasing over each decade, at approximately the same rate of increase as acinar carcinoma.[2] However, it is likely underreported in prostate specimens.[1] Many studies report more aggressive features than acinar prostate cancer, including higher stage and metastases at presentation as well as a higher mortality rate.[1,3] This is a case report of a gentleman who was diagnosed with mixed ductal and acinar prostate adenocarcinoma and his clinical course.

Case presentation

An 85-year-old Caucasian man presented with two months of intermittent painless macroscopic hematuria. His medical history included benign prostatic hypertrophy (estimated prostate volume 50 cc) and Grave's disease status post I-131 ablation; he was otherwise fit and in good health. His laboratory findings demonstrated mild anemia and a pretreatment PSA of 5.63 ng/ml, which had remained relatively stable in the 4–6 ng/ml range for at least the past 5 years.

During cystoscopy, a papillary lesion in the prostate with associated hemorrhage was observed (Fig. 1). Transurethral resection of this specimen revealed prostatic adenocarcinoma with mixed acinar and ductal types, Gleason 4 + 5 = 9 (Fig. 2). Digital rectal examination confirmed prostatic hypertrophy with tumor extending from seminal vesicles to prostatic apex.

An MRI demonstrated a 3.1 cm infiltrative tumor with multifocal extension to the bladder with pelvic adenopathy concerning for tumor involvement. A bone scan and CT chest, abdomen, pelvis were negative for distant disease. He therefore was staged with cT4N1M0 disease. Given his locally advanced disease, the patient was not deemed a surgical candidate, and so he was treated with definitive radiation via intensity-modulated radiation therapy (IMRT) to 5040 cGy to pelvic lymphatics, prostate, and seminal vesicles, followed by a boost to 7920 cGy total to the prostate and seminal vesicles. He also received 10 months of concurrent and adjuvant androgen deprivation therapy with Degarelix and Lupron.

The patient had good clinical, radiographic, and PSA response to treatment. He ceased to have hematuria, his PSA became undetectable, and repeat MRI demonstrated resolution of pelvic adenopathy and regression of the prostate tumor. 1.5 years after his initial diagnosis, he presented again with intermittent painless macroscopic hematuria, which was initially treated as hemorrhagic radiation cystitis. However, repeat cystoscopy and biopsy confirmed recurrent disease at the right ureteric orifice.

Restaging scans obtained two years after his initial diagnosis included bone scan and CT chest, abdomen, and pelvis, which were negative for distant disease with the exception of increased uptake on bone scan at the distal right tibia and fibula. This location was moderately painful and so was further evaluated with MRI, which showed extensive osseous metastatic disease in the distal lower extremity. This was treated with palliative radiation.

The patient was then restarted on Lupron and began Zometa for bony metastases. His PSA had increased to 0.23 ng/ml by the time of starting androgen-deprivation therapy. He initially declined

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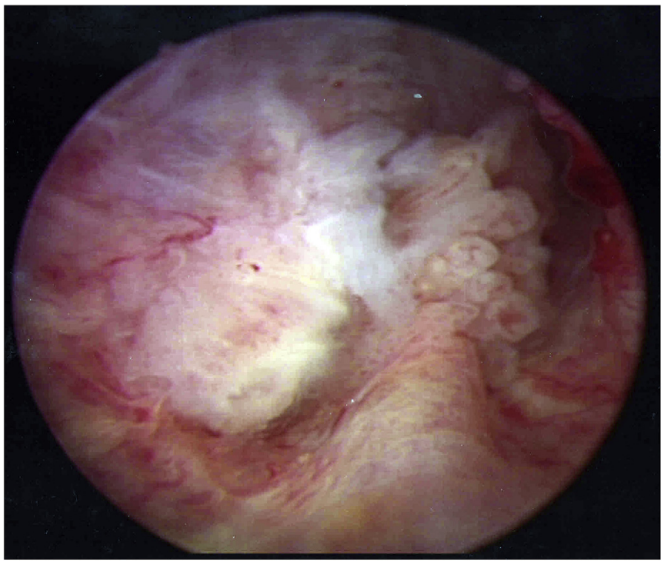

Fig. 1. Cytoscopic Image of Bladder Lesion. This is the papillary lesion of prostate ductal adenocarcinoma seen during cystoscopy.

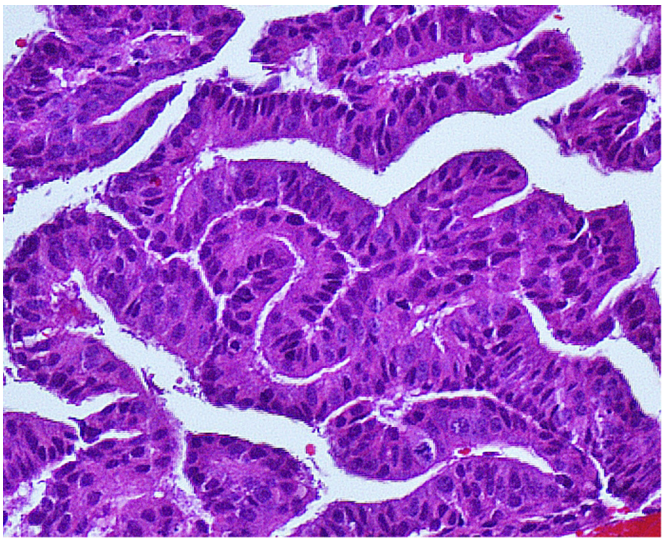

Fig. 2. Microscopic Image of Bladder Lesion. This bladder lesion was diagnosed as prostatic ductal adenocarcinoma, Gleason 4 + 5.

chemotherapy and developed progressive disease over the next six months with widespread osseous scleroticmetastases, dural metastasis along the falx, innumerable pulmonary nodules, and lymphangitic carcinomatosis. Although he attempted one cycle of Docetaxel 60 mg/m2, he was unable to tolerate it and passed away approximately three years after his initial diagnosis.

Discussion

Ductal adenocarcinoma of the prostate, with or without a mixed acinar component, represents less than 5% of all prostate cancers. Tumors are characterized by tall, columnar, pseudostratified epithelium with papillary architecture. Macroscopically, ductal adenocarcinoma tends to appear exophytic with villous growth, and can involve the prostatic urethra, seminal vesicles, or periprostatic soft tissue, including the bladder. Men with PDA are more likely to present with locally advanced disease, although lymph node metastases seem to be comparable between ductal and acinar pathology.[2]

Studies have shown that men with PDA undergo similar rates of radical surgery as their acinar counterparts. However, PDA tends to have a high short-term failure rate after radical prostatectomy.[4] PDA can also be treated with definitive radiotherapy, as with our patient. PDA response to hormone therapy is debatable. Some studies have found an excellent long-term response to endocrine control based on PSA level. However, while PDA tumor cells do produce PSA, this lab value is not elevated in all patients.[3] In our patient, PSA was discordant with disease progression and hormone therapy did not control his disease. His PSA trend during hormonal therapy may suggest the presence of castrate-resistant cancer as an evolutionary escape method of the tumor.

PDA tends to metastasize to unusual locations. For example, our patient initially developed solitary distal extremity osseous metastases. This was followed by dural metastases to the falx and lymphangitic carcinomatosis. Visceral metastases are also relatively common with PDA. This may be due to the propensity of PDA to disseminate via hematogenous rather than lymphatic routes.[5]

Conclusion

This case is an example of how PDA is a more aggressive form of prostate cancer. PSA may not be a sufficient tumor marker for local recurrence of disease progression. The location of distant metastases in PDA tends to be less predictable. More efforts are required to contribute knowledge of this tumor biology and to assess its response to therapy.

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