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Mischievous malakoplakia: A potential pitfall of mpMRI of the prostate?

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ABSTRACT

Interpretation of multiparametric magnetic resonance imaging (mpMRI) for prostate cancer diagnosis and staging can be challenging and, in some cases, benign prostate disease can mimic locally advanced malignancy. We present the case of a 57 year-old male with biopsy-proven Gleason 3 + 4 prostate cancer and a preoperative mpMRI showing extraprostatic extension who was later found to have infiltrating malakoplakia on final surgical pathology. This case highlights the importance of recognizing that malakoplakia of the prostate can present as a PI-RADS 5 lesion with extracapsular extension on mpMRI. Such cases can result in wide-excision, non-nerve sparing radical prostatectomies that may be unwarranted.

Introduction

Multiparametric magnetic resonance imaging (mpMRI) has become the imaging modality of choice for evaluation and staging of prostate cancer (PCa). mpMRI can be used to identify clinically significant PCa, localize and target suspicious lesions for biopsy, assess for seminal vesicle invasion and extraprostatic extension (EPE) and evaluate for nodal disease.[1] However, as mpMRI of the prostate is a relatively new tool, many pitfalls can be encountered in its interpretation. For example, benign prostate disease, such as a bulging hyperplastic nodule or granulomatous prostatitis, can mimick T3 PCa and alter treatment choice and surgical planning.[2] As a consequence, false-positive mpMRI for locally advanced PCa can result in patients undergoing wide-excision, non-nerve sparing radical prostatectomies that may be unwarranted. Below, we present a patient with biopsy-proven Gleason score 3 + 4 PCa and mpMRI demonstrating EPE, who was later found to have Gleason group 2, Gleason score 3 + 4 PCa with less than 5% of pattern 4 tumor glands and infiltrating malakoplakia on surgical pathology.

Case presentation

A 57 year-old male was seen locally for lower urinary tract symptoms (IPSS 20) and rising prostate specific antigen (PSA 3.1, 4.1–4.3 ng/mL) over three years. Prostate exam was benign. Post void residual was 89 mL and urine analysis was negative. Prostate health index showed a PSA of 3.7 ng/mL, Free PSA 0.8 ng/mL and proPSA 18.3 pg/mL. He had no family history of PCa or other risk factors. The patient was re-assessed one-year later. Repeat testing showed a PSA 3.9 ng/mL with 19% free. 4Kscore showed a 13% probability of Gleason Score ≥7 on biopsy.

A 12-core biopsy was performed. This showed Gleason 3 + 3 at the left lateral base and lateral mid-gland, Gleason 3 + 3 at the right base, as well as Gleason 3 + 4 PCa at the right lateral base (tumor involving 65% of core with 5% Gleason 4). Bone scan was negative. A pelvic MRI showed marked signal abnormality of the right peripheral zone consistent with malignancy as well as a focal area of EPE measuring 7 mm along the right peripheral zone.

Upon referral to our academic center, repeat PSA was 4.7 ng/mL, IPSS 23, and SHIM 25. Given the low PSA value, normal DRE, and small volume of Gleason 4 on biopsy, findings from the outside MRI were questioned. The original MRI was performed 14 days after biopsy so there was concern for retained blood products limiting the evaluation. The patient was counseled that safely preserving his right neurovascular bundle during surgery would be not possible and that post-operative erectile dysfunction (ED) would be likely. This was distressing to the patient so the decision was made to wait 6 weeks and obtain an mpMRI to confirm a wide excision would be necessary. This confirmed a PI-RADS 5 lesion in the right peripheral zone with extensive EPE and involvement of the right neurovascular bundle (Fig. 1). The patient underwent robotic-assisted laparoscopic prostatectomy with a wide margin on the right and nerve-spare on the left, and a pelvic lymph node dissection. The operation was notable for induration surrounding the seminal vesicles.

Pathology showed pT2N0, Gleason group 2, Gleason score 3 + 4 PCa with less than 5% of pattern 4 tumor glands (right and left posterior

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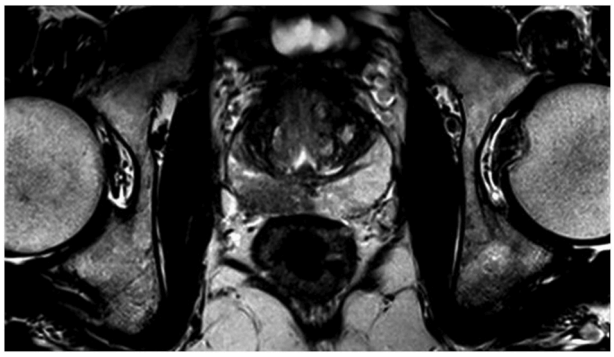

Fig. 1. mpMRI (T2 series) showing a 2.7 × 1.3 × 2.6cm hypointense mass (PI-RADS 5) extending outside the contours of the prostate gland and involving the right neurovascular bundle.

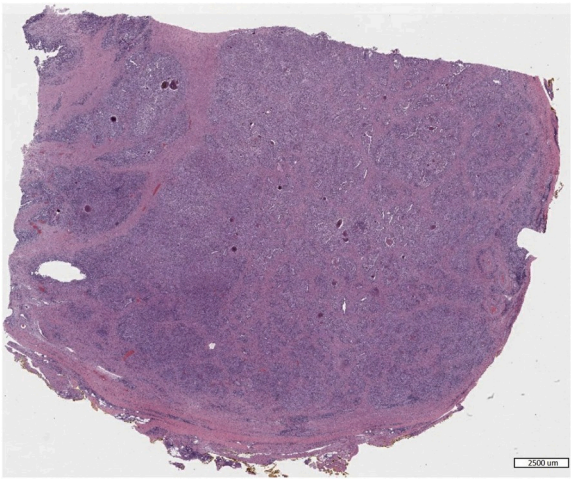

Fig. 2. A cross section of the prostate reveals diffuse acute and chronic inflammation diffusely involving both prostatic glands and stromal tissue. Corpora amylacea can be seen in the lumen of some prostatic glands.

gland, 3% of prostate involved) and extensive acute and chronic prostatitis with numerous eosinophilic granular histiocytes causing mass effect consistent with malakoplakia (Figs. 2–3).

Discussion

Malakoplakia is a rare, chronic granulomatous condition that commonly affects the genitourinary tract, particularly the bladder. This non-neoplastic lesion is often caused by a urinary tract infection due to *E. coli* or other gram-negative bacteria (70–80% of cases).[3] Michaelis-Gutmann inclusion bodies are the hallmark histologic finding in malakoplakia and represent incomplete intracellular degradation of engulfed bacteria by macrophages.[3] The radiographic appearance of malakoplakia is variable, but can be similar to malignancy. It can present has a polypoid, vascular, solid tumor or plaque-like lesion, or as a mass infiltrating into surrounding structures.[3]

Malakoplakia of the prostate is rare, but when present can mimic PCa. In 2017, Heah et al. described a patient who was found to have a PI-RADS 5 lesion on mpMRI during workup for elevated PSA. This patient was diagnosed with malakoplakia of the prostate after undergoing an MRI-guided biopsy.[4] In 2018, Velasquez et al. also found malakoplakia of the prostate after MRI-guided biopsy of a PI-RADS 4 lesion.[5] Neither of these patients had evidence of PCa nor required additional intervention.

Unlike the reports mentioned above, our patient had a standard 12-core biopsy prior to his mpMRI. This showed Gleason 3 + 4 PCa at the right lateral base, which was consisted with a PI-RADS 5 lesion with EPE visualized on mpMRI. Surprisingly, the patient's final pathology was a very low volume of disease (only involving 3% of prostate), Gleason group 2, Gleason score 3 + 4 PCa with less than 5% of pattern 4 tumor glands and extensive inflammatory histology consistent with malakoplakia.

In retrospect, our patient would have been a candidate for active surveillance or nerve sparing surgery given his low grade disease. The large PI-RADS 5 lesion with EPE seen on mpMRI prompted a wide, nerve-sacrificing resection on the right side, which will likely compromise his erectile function. Unfortunately, ED was one of the patient's primary fears heading into surgery. A wide resection was not necessary as the mass emanating from the patient's prostate was not cancer, but infiltrating malakoplakia.

Michaelis-Gutmann bodies were not identified in the pathology specimen. These inclusion bodies are pathognomonic for malakoplakia, but are not required for diagnosis as they can be absent in early and late phases of the inflammatory reaction and after culture-directed antibiotics.

Conclusion

mpMRI of the prostate has revolutionized diagnosis and local staging for PCa but many pitfalls can be encountered in its interpretation. Benign prostate disease can mimic locally advanced PCa, which can alter treatment choice and surgical planning. This case demonstrates that malakoplakia of the prostate can present as a PI-RADS 5 lesion with EPE on mpMRI. Such cases can result in patients undergoing wide-excision, non-nerve sparing radical prostatectomies that may be unwarranted.

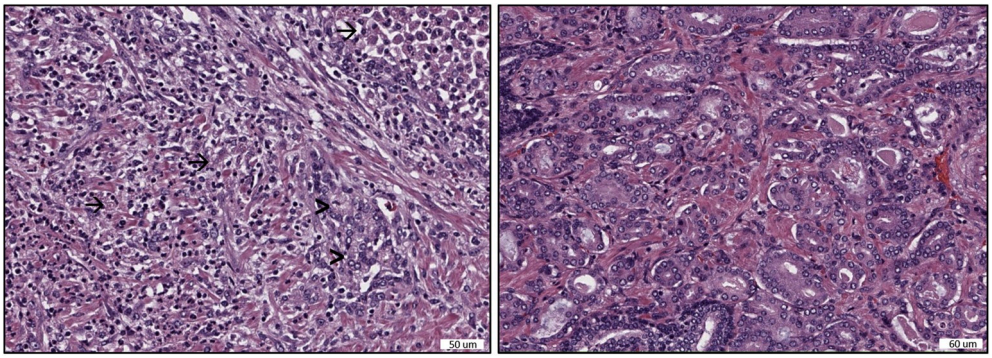

Fig. 3. A). Extensive foamy histiocytic infiltrate with eosinophilic granular cytoplasm involves both prostatic glands (>) and stroma (→), suggestive of malakoplakia. However, no Michaelis-Gutmann bodies are identified in the histiocytic cytoplasm. Scattered lymphocytes, eosinophils and neutrophils are also present admixed with histiocytes. B). A small volume of prostatic adenocarcinoma, predominantly Gleason score 3, is identified.

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Declaration of competing interest

The author(s) declare(s) that there is no conflict of interest regarding the publication of this article.

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