RESIDENT'S CORNER

Malakoplakia of prostate as a complication of transrectal needle biopsy

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We report a case of a 72-year-old male that underwent two sets of transrectal prostatic needle biopsy (TPNB) within 9 month period. Pathology showed unremarkable benign prostatic tissues in the first group of biopsies while extensive diffuse inflammation with the characteristic features of malakoplakia in the second set. Three cores in the repeat biopsy contained foci of prostatic adenocarcinoma as well. Occurrence of malakoplakia several months after TPNB in our case suggests that microorganisms may have been inoculated to the prostate during the biopsy procedure. We believe that malakoplakia must be added to the list of complications after TPNB.

Key Words: transrectal ultrasound guided prostate needle biopsy, adenocarcinoma, complication, malakoplakia, prostate,

Introduction

Ultrasound guided transrectal prostate needle biopsy (TPNB) is the standard practice for the evaluation of patients when there is a suspicion of prostate carcinoma in a patient. The procedure is known to have minor frequent complications such as hematuria, rectal bleeding and hematospermia. More serious complications such as prostatitis, epididymitis, septicemia and even death due to infection have been rarely reported. To our knowledge, there has been no account of prostatic malakoplakia developing after TPNB. In this article, we report first such case that appears to arise as a complication of needle biopsy procedure. Characteristic microscopical features

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Address correspondence to Dr. Dilek Ertoy Baydar, Department of Pathology, Hacettepe University Hospital, Sihhiye/Ankara 06100 Turkey of malakoplakia were observed in the second set of TPNB's performed in a course of 9 months after the first biopsy that had showed no signs of significant inflammation. In addition to malakoplakia, three cores contained small foci of prostatic adenocarcinoma.

Case presentation

A 72-year-old man who had lower urinary tract symptoms for the last 10 years presented to an outside health institution in 2005. His past medical history was insignificant. Alpha-1 blocker tamsulosin-hydochloride was started, and was effective to relieve his problems. In August 2009, he presented to our institution with the recurrent complaints. His total PSA measured was 19.23 ng/mL, with free/total PSA ratio of 35%. The digital rectal examination revealed an enlarged prostate. The 10-core transrectal ultrasound-guided prostate needle biopsy was performed. Pathological examination of all the cores showed benign prostatic tissues without any

significant finding, Figure 1a. Prophylactic amoxicillin (2 x 1 gr/day) was administered to the patient for 5 days starting the day before biopsy. The patient reapplied 8 months later with the complaints suggestive of prostatitis. He had acute urinary retention and needed urinary catheterization. His serum creatinine and potassium were 17 mg/dL and 7 mEq/lt respectively. There was also a boost in serum PSA levels, the highest values reaching 100 ng/mL. With the appropriate treatment and antibiotics, his symptoms subsided and creatinine turned to normal. When the patient was re-seen 1 month after, his total PSA was 30.19 ng/mL with 14% free/total ratio. The high total PSA and the fall in free/total value necessitated another attempt for the prostate biopsy.

Twelve cores of prostate were received from the transrectal biopsy. Ten out of 12 revealed dense inflammatory infiltration in the prostatic fibromuscular stroma diffusely, surrounding the glands, Figure 1b and 1c. The infiltrate consisted of lymphocytes and epithelioid histiocytes mainly, as well as scattered polymorphs. On high-power view under the microscope, the epithelioid cells were noted to contain blue-grey targetoid inclusions of variable sizes, Figure 2a. Upon the suspicion, the special histochemical

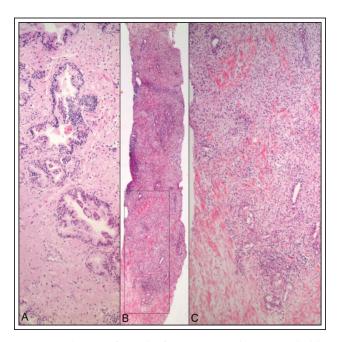


Figure 1. a) Acore from the first TPNB with unremarkable features (H&Ex100). **b)** Repeat biopsy showing intense inflammation (H&Ex40). **c)** Higher power picture of the area marked by a rectangle in panel B. Diffuse inflammation composed of histiocytes and lymphocytes (H&Ex100).

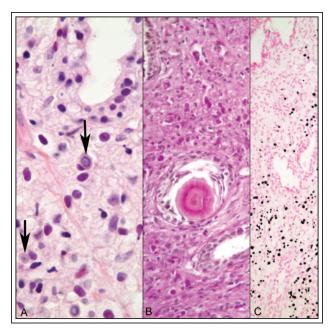


Figure 2. a) Characteristic Michaelis Gutmann bodies in the form of targetoid inclusions (pointed by arrows) (H&Ex1000). **b)** PAS stain highlights the inclusions (PASx400). **c)** von Kossa staining shows that inclusions contain calcium, which is typical of Michaelis Gutmann bodies (von Kossax200).

studies were done, which supported the diagnosis of malakoplakia. As expected, the inclusions, Michaelis Gutmann bodies were PAS positive, and they contained calcium and iron, Figure 2b and 2c.

In three tissue cores, there were foci of prostatic adenocarcinoma adjacent to the masses of malakoplakia, Figure 3. The diagnosis of cancer was verified by the absence of the basal cell layer immunostaining around the neoplastic glands with p63 and 34β E12 antibodies. All three had the Gleason score of 3+3=6.

After the diagnosis of malignancy, the abdominal CT was taken that revealed an enlarged prostate containing a hypodense area and prominent parailiac lymph nodes. The patient underwent non-nerve sparing retropubic radical prostatectomy and bilateral pelvic lymphadenectomy. The prostate weighed 65 grams and its pathological examination showed large areas of inflammation, typical of malakoplakia throughout the prostate. Prostatic adenocarcinoma was located at the left lobe as a single focus from apex up to the middle portion of the gland. It was an acinar type, Gleason score 3 + 3 = 6 tumor without extracapsular extension or seminal vesicle invasion. Tumor occupied less than 5% of the prostate. All surgical margins were negative and there was no lymph node metastasis.

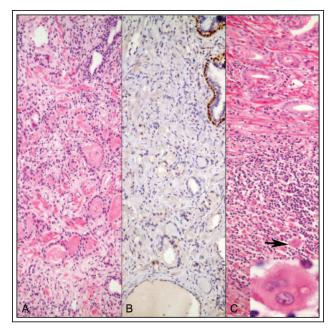


Figure 3. a) Gleason score 3 + 3 = 6 cancer composed of small glands containing eosinophilic secretions (H&Ex200). **b)** Absence of basal cells in the small glands supports the malignancy. Normal glands at the upper right constitute intrinsic positive control for the stain (Anti-p63 Abx200). **c)** Malakoplakia type infiltrate adjacent to prostatic carcinoma. Inset shows a higher magnification of typical von Hansemann histiocyte pointed by arrow above (H&Ex200; inset H&Ex1000).

The patient otherwise was a healthy male without a chronic disease like diabetes mellitus, HIV infection or previous malignancy that would lead to immune depression.

Discussion

Malakoplakia is a rare chronic inflammatory condition known since 1902,³ reported in various sites such as skin, bone, colon, uterus, liver, stomach and lung, but most commonly observed in the urinary tract of middle aged women as a result of recurrent infections. It is thought to be an aberrant or defective lysosomal host reaction to an infectious agent. The most common organism related to disease is Escherichia coli. Klebsiella pneumonia, rhodococcus equi, pseudomonas aeruginosa, staphylococcus aureus, and mycobacterium bovis have also been isolated.⁴ Microscopically, masses of large eosinophilic histiocytes, named as von Hansemann histiocytes are seen, many of which contain basophilic inclusions. These inclusions, called Michaelis-Gutmann bodies can be found both

intracellularly and extracellularly, are characteristic sharply spherical 5-10 μ m target-like structures and thought to be accumulations of incompletely digested bacterial fragments. They are highlighted with PAS and special stains for iron and calcium.

Bladder is the most common organ involved in the case of urinary tract disease. Malakoplakia of the prostate, first described in 1959,⁵ is a rare diagnosis. Only less than 50 prostatic cases have been reported so far. It may occur in immunocompromised patients, may also coexist with other serious diseases such as malignant lung lesions, E. coli septicemia, pulmonary abscess or prolonged urinary infections. Our patient had an attack of acute prostatitis between the two sets of transrectal prostate biopsies which were 9 months apart. The cores obtained in the first attempt were free of inflammation, but the repeat TPNB showed prostatic tissues occupied by diffuse malakoplakia, to suggest the disease arising as a complication of the first biopsy. Otherwise, he was an immune competent healthy man without a chronic debilitating disease or a previous malignancy. His full check up before the operation did not reveal a clue of additional organ involvement by malakoplakia.

Well-known complications of transrectal prostate biopsies include hematospermia, hematuria, rectal bleeding that may be self-limiting or that may rarely require surgical intervention, fever and urinary retention. Urinary tract infection and sepsis have also been reported. Our case is the first that reports the development of malakoplakia after a transrectal prostate needle biopsy.

Malakoplakia in prostate can imitate cancer both microscopically and macroscopically. It is palpated hard in the digital examination and causes significant PSA elevation. Histiocyte groups may be confused with high grade prostatic carcinoma under the microscope. The coexistence of prostatic adenocarcinoma and malakoplakia of the prostate is extremely rare. Only 3 such cases have been reported so far, ours is being the 4th.8-10

As a summary, we present the first case of prostatic malakoplakia in which previous prostate transrectal needle biopsy looks like a predisposing factor. We suggest that malakoplakia may need to be added to the list of complications after TPNB. When there is unexpected elevation of serum PSA or significantly different digital rectal examination findings in a man a few months after a TPNB, malakoplakia of the prostate is possibly one of the diseases to consider. This issue is also important to remember so that malakoplakia is one of the benign mimickers of the malignancy and has the potential of misdiagnosis as prostatic carcinoma microscopically.

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