Prostate Tuberculosis as Predisposition for Prostate Cancer

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Abstract

The prostate is one of the most common sites for cancer and in contrast prostate tuberculosis seems to be a rare disease. Mycobacterium tuberculosis and cancer look like antagonists; based on this principle BCG-therapy for bladder cancer was established. However sometimes tuberculosis may predispose cancer. We present a patient, 72 year of age, who was cured from pulmonary and prostate tuberculosis. Seven years after recovery he presented with dysuria and elevated PSA (11ng/ml). Prostate biopsy revealed prostate cancer and the patient underwent radical prostatectomy. After surgery huge caverns with calcified caseation and glandular cancer in the caverns’ wall were found. Only post-tuberculous fibrosis but no active TB inflammation was detected. In this case prostate tuberculosis may have predisposed prostate cancer. In any case high PSA levels are an indication for prostate biopsy, especially if the patient has had a long-term history of an infectious-inflammatory process in the prostate.

INTRODUCTION

The World Health Organization (WHO) recognizes tuberculosis (TB) as a global problem. In 2013, an estimated 9.0 million people developed TB and 1.5 million died from the disease, 360,000 of whom were HIV-positive [1].

Prostate cancer is one of the most common types of cancer and it is the second commonest cause of cancer-related death among men in the western world. Silberstein et al. (2013) have found microscopic prostate cancer in up to 30% of men between 20–40 years old [2]. Of 115,881 men attending the prostate cancer screening in Japan, 2,320 of 6,099 secondary screening patients underwent prostate biopsy, and 1,073 men were diagnosed with PCA [3]. Although adenocarcinoma of the prostate and genitourinary tuberculosis are seen frequently, their concomitance is extremely rare [4,5].

A recent study showed that chronic prostate inflammation accelerates prostate cancer progression [6], promotes initiation of diverse malignancies, enhances basal-to-luminal differentiation, and accelerates initiation of prostate cancer originating from basal cells [7-9]. Here we present a case in which the chronic inflammation of a prostate TB may even have predisposed prostate cancer.

CASE PRESENTATION

A 72 years old man was presented to our Institute in March 2011 with complaints of frequency and urgency, weakening urine stream, night sweats, bladder and perineal pain. In 1987 the patient had had a pulmonary TB and had been treated during 10 months with three anti-TB drugs (isoniazid, rifampicin, and streptomycin) with good result. In 1990 the patient presented with hemospermia, strong perineal pain and was referred to a TB-urolgist. Full examination revealed pyospermia, hemospermia and caverns of the prostate. Growth of MTB was found both in ejaculate and prostate secretion. MTB was sensitive to all anti-TB drugs. The pulmonary TB was not reactivated. The patient was treated during 10 months with four anti-TB drugs (isoniazid, rifampicin, streptomycin and pyrazinamide) again with good efficacy: pain and hemospermia disappeared and pyospermia decreased significantly. The prostate caverns, however, remained as complete healing of prostate tissue destructions is not possible. In 1993 the patient complained again of dysuria, perineal pain, and painful ejaculation. A relapse of prostate TB was finally diagnosed. MTB did not grow, but could be detected in the ejaculate by polymerase chain reaction (PCR). The pulmonary TB remained inactive. The patient received isoniazid, rifampicin, streptomycin, PAS and pyrazinamide for 4 months, than isoniazid and rifampicin only for the rest of the year and in addition tocopherol, thiamin, phytotherapy, dimexid, and non-steroid inflammatory drugs. The patient also received rehabilitation courses annually for 5 years in a special anti-TB sanatorium. The patient was healed and remained well until March 2011, when
pain and dysuria appeared again. Pyospermia with growth of *Enterobacter* sp. in prostatic secretion was found, but MTB was not detected by any method. X-ray examination showed huge caverns of the prostate with calcification (Figure 1).

As the PSA level was 11ng/ml, prostate biopsy was performed and a solid-glandular cancer was found by histological examination. There was, however, no active TB inflammation in the biopsies. The patient underwent radical prostatectomy. The section of the prostate gland revealed huge TB caverns filled with stones (actually calcified caseation) as demonstrated in (Figure 2).

Histo-patho- histological investigations revealed a proliferation of the glandular prostate cancer with invasion of the capsule in the right and left lobes. The cavity walls of the caverns were lined with transitional epithelium with dense calcium salts in the lumen. In the seminal vesicles glandular tumor structures were growing into the muscle layer. Active TB inflammation was not found.

**DISCUSSION**

Although male genital TB seems to be a rare disease, 77% of men who died from TB of all localizations had prostate TB, mostly overlooked during their lifetimes [10]. In a recent study [10] 93 patients suspicious of prostate TB underwent ultrasound guided core prostate biopsy. Probes were investigated by PCR, pathomorphology and culture. MTB was found by PCR in 10.7%, but MTB culture was only positive in 6.9%. Patho-histology revealed inflammation in 94.6% of probes, fibrosis in 65.6%, intra prostatic neoplasia in 9.7%, cancer in 5.4%, and TB in 24.7% [10].

Clinical features of urogenital TB (UGTB) are variable, non-specific, and sometimes also mimic prostate and other urogenital cancer [10,11,12]. Kho and Chan [13] reported about a 20-year-old man who presented with a slow-growing painless scrotal tumor for 2 months, initially suspicious for a right paratesticular tumor. The patient underwent operation and pathohistology revealed TB.

Another case history was described by López Barón [14]. A 65 year old man presented with symptoms of frequency, dysuria and weight loss within the last 6 months, without pulmonary symptoms and negative ELISA test for HIV. Digital rectal examination revealed a high volume, irregular and hard prostatic gland. Ultrasound investigation showed a prostatic volume of 39 cm³, without sign of malignancy. The prostate biopsy showed multiple granulomas and the Zhiel-Neelsen staining was positive for MTB.

Thus, UGTB should be considered as differential diagnosis for both, neoplastic and infectious–inflammatory diseases. An incorrect diagnosis leads to unnecessary surgical intervention, as UGTB should be treated by drugs only, at least if it has been diagnosed in time. Prostate TB like any other chronic infectious inflammation may predispose for prostate cancer, which is indicated by the case presented here. In any case high PSA levels are an indication for prostate biopsy, especially if the patient has a long-term history of an infectious-inflammatory process in the prostate.

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This study had been approved by the local ethics committee of Novosibirsk Research TB Institute and the patient gave his written consent that his medical history is published in a medical scientific journal. Kurt G. Naber reviewed the manuscript.

**REFERENCES**


