

An unusual case of concurrent breast and prostate cancer

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WOO TCS, CHOO R, CHANDER S. An unusual case of concurrent breast and prostate cancer. The Canadian Journal of Urology. 2004;11(5):2390-2392.

Breast and prostate cancer occurring concurrently is a rare occurrence. However, the recent literature has reported that there is an increased incidence of male breast

cancer in prostate cancer patients. The authors describe the case of a man who presented with breast cancer, which was preceded by prostate cancer. There are some common features in terms of the etiology, diagnosis and treatment of these two tumors which are discussed.

Key Words: prostate, breast, cancer, concurrent

Introduction

While prostate cancer is the most frequently diagnosed cancer in men, breast cancer accounts for less than 1% of all male cancers. In this case report, we will present the case of a man who had both breast cancer and prostate cancer. Although uncommonly reported together, the literature suggests that breast and prostate cancer might have some common features in terms of their etiology, diagnosis and treatment.

Case report

A 91 year-old Caucasian railroad worker was referred to the Toronto-Sunnybrook Regional Cancer Centre for the management of extensive cutaneous lesions on the chest. Figure 1 His past medical history follows.

At age 72, he was diagnosed with prostate cancer on the basis of the clinical findings of multiple, bilateral, hard nodules in the prostate on per rectal

examination, and a PSA of 20.5 µg/L. At the time of prostate cancer diagnosis, he was asymptomatic, and no further diagnosis or treatment was carried out because of his wish to take a conservative approach and to avoid any side effects of treatment.

At age 90, he presented at a routine follow-up with an extensive cutaneous lesion on his chest. Figure 1 The main complaints related to this lesion were itchiness and bleeding. The lesion was a raised, erythematous plaque involving an area of the skin around the right nipple. The entire breast was indurated with retraction of the right nipple. The lesion extended superiorly to the clavicle and across the midline to the contralateral breast. There was no clinical evidence of supraclavicular, cervical or axillary lymphadenopathy. Staging investigations with CT scans of the neck, chest and abdomen did not suggest any metastatic disease in the liver, lungs or regional lymphatics. However, bone scan showed three areas of increased uptake in the bony pelvis, highly suggestive of metastases. At the same time, he had a significantly elevated serum prostate-specific antigen (PSA) level of 190 µg/L, and digital rectal examination revealed an enlarged, firm nodular prostate gland.

The patient was not troubled by any symptoms of local or metastatic disease.

Deep punch biopsies of the cutaneous lesions in the left breast demonstrated malignant epithelial cells with

Accepted for publication September 2004

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Figure 1. Cutaneous lesions in the left chest wall (breast cancer) before treatment.

rudimentary lumina and gland formation. Immunohistochemistry of the cells was strongly positive for cytokeratin 7, and negative for PSA and thyroid transcription factor (the latter of which is a marker for lung adenocarcinoma). The cells were diffusely positive for estrogen and progesterone receptors. These findings as well as the morphology were consistent with infiltrating ductal carcinoma of the breast. To date, the prostate gland has not been biopsied because of the limited prognosis conferred by the presence of bone metastasis and the locally advanced breast carcinoma.

The patient's only relevant family history was of a cancer of unknown primary in his mother at 80 years of age. He has two siblings, two children and two grandchildren who have not had cancer. His comorbid conditions include frequent recent exacerbations of chronic obstructive pulmonary disease, mild congestive cardiac failure and non-insulin dependent diabetes mellitus.

The decision was made to treat both prostate cancer and the locally advanced breast cancer conservatively with hormonal manipulation, using three-monthly depot injections of goserelin acetate 10.8 mg. After 6 months of androgen suppression, the cutaneous lesions on the chest wall have largely resolved (Figure 2), and the induration in the right breast has greatly reduced. Also his PSA level has now decreased to 0.20 µg/L. No further definitive treatments for breast cancer were planned at the time of this report.

Discussion

Concurrent prostate and breast cancer has rarely been described in the literature. There are a small number

of case studies¹⁻⁵ describing this phenomenon. Most describe patients receiving anti-androgen therapy for prostate cancer who have subsequently developed breast cancer. There has also been a small series of 10 patients from Massachusetts in which breast cancer preceded prostate cancer in eight patients.⁶ Here we describe the case of a man in whom the prostate cancer preceded the breast cancer by 18 years, and who had no previous history of anti-androgen treatments.

A hypothesis frequently given as to why these cancers might occur together is that prolonged exposure to estrogen and anti-androgen therapy for prostate cancer may be a catalyst for growth of breast tissues.^{1,2,4,5} This is exemplified by one case study in which the use of anti-androgens seemed to result in bilateral male breast cancers.¹ In another case report, a patient also had a genetic predisposition to breast cancer because of a BRCA-1 mutation and chromosome 9 inversion.² The authors suggested that where there has been a family history of BRCA mutation - related cancers, anti-androgens should be used with greater care. It is not known whether LH-RH agonists used alone or in combination with antiandrogens might confer the same effect. If this were true, this would be a finding of increasing interest to modern clinical practice, as hormonal treatments are now routinely used in the adjuvant treatment of high-risk prostate cancer.

Genetic predispositions for breast and prostate cancer occurring together are rare, and include the Li-Fraumeni syndrome⁷ in which cancer can occur in many sites, not just the breast and prostate, and BRCA-2 mutations.^{8,9} In BRCA-2 mutation carriers, the risk of prostate cancer has been estimated to be increased by a factor of 4.65.¹⁰ Other conditions that confer a predisposition to hyperestrogenemia and consequently, male breast cancer include Klinefelter's



Figure 2. Resolution of the cutaneous lesions after treatment with LHRH agonist.

syndrome, Reifenstein syndrome and Kallman syndrome.¹¹⁻¹³

In our case, neither environmental nor genetic factors could be identified that would result in both cancers developing. The patient did not have a significant family history of breast cancer to suggest a BRCA – gene mutation. It has recently been reported in the literature that the incidence of male breast cancer is higher in patients who have prostate cancer. An analysis of population data from Sweden of 135,713 persons estimated that the relative risk of breast cancer was 2.01, among men who had prostate cancer.¹⁴ This study was unable to analyze further whether the excess cancers were due hormonal medications being administered, or due to genetic predispositions.

In our case, we did not establish histological proof of a prostate cancer. However, it is self-evident that this patient had two primaries for the following reasons. Firstly, the excessively high PSA level recorded in this case is characteristic of prostate malignancy rather than benign prostatic hypertrophy or breast cancer. It is known that breast cancer can express PSA¹⁵ and that some breast cancer patients have elevated serum PSA. However, the highest PSA level on record for a woman with breast cancer is 16 µg/L.¹⁶ Also digital rectal examination revealed highly suspicious nodular changes in the prostate gland, suggesting malignant involvement. The tumor cells from the breast specimens did not secrete PSA, and their morphology and immunohistochemistry findings confirmed the diagnosis of infiltrating ductal carcinoma of the breast.

Chronic administration of a LH-RH agonist such as goserelin results in a decrease in luteinising-hormone levels, which in turn decreases testosterone production from the testicles. Because most estrogen in men is produced through aromatization of testosterone, goserelin also decreases estrogen levels in men. Therefore, 'pharmacological castration' using a LH-RH agonist provides clinicians with a therapeutic avenue to treat both prostate and breast cancer. For our case, in which factors such as the presence of distant metastasis, the patient's advanced age and co-morbid medical conditions, and positive estrogen and progesterone receptors in his breast cancer needed to be considered, hormonal manipulation using a LH-RH agonist alone was chosen as the treatment of choice. Further surgery and anti-neoplastic agents were not felt to be necessary.

In conclusion, the occurrence of both breast and prostate cancer in the same patient has rarely been reported. Unlike some previous case studies, our case had no clear etiologic factors that could be identified

that would have lead to the development of both breast and prostate cancers concurrently. With the recent report from Sweden of an increased risk of male breast cancer in prostate cancer patients, it will be interesting to see if there will be more cases such as this one being reported. In our case, both cancers were able to be effectively treated using a single agent, goserelin. □

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