Gynecomastia Following Cytotoxic Therapy in a Patient with Testicular Cancer

Testis Kanserli Bir Olguda Sitotoksik Kemoterapi Sonrası Gelişen Jinekomasti

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Abstract

Gynecomastia is the development of abnormal breast tissue in men. Relatively increased estrogen action on tissue level is believed to play a main role in the pathogenesis of the entity. Here, we describe a patient with painless gynecomastia presenting after cytotoxic chemotherapy for testicular cancer. Further investigations showed no evidence of disease progression, recurrence, or metastasis. We suggest that the clinicians should be aware that gynecomastia may follow cytotoxic chemotherapy for testicular cancer and does not reflect the return of malignancy. *Turk Jem 2008; 12: 86-7*

Key words: Gynecomastia, testis, chemotherapy, malignancy

Özet

Jinekomasti erkekte anormal meme dokusu gelişimidir. Antitenin patogenezinde doku düzeyinde rölatif olarak artmış östrojen etkinliğinin ana rolü oynadığına inanılmaktadır. Bu yazıda testiküler kanser için uygulanmış sitotoksik kemoterapi sonrasında ağrısız jinekomasti ile başvuran bir hasta tanımladık. İleri incelemeler sonunda hastalık progresyonu, rekürrensi veya metastazı saptanmadı. Klinisyenlerin testis kanseri için uygulanan sitotoksik kemoterapi sonrasında jinekomasti gelişebileceğinin ve bu durumun malignitenin varlığını yansıtmayacağının farkında olması gerektiğini düşünüyoruz. *Turk Jem 2008; 12: 86-7*

Anahtar kelimeler: Jinekomasti, testis, kemoterapi, malignite

Introduction

Gynecomastia is an often-encountered entity in clinical practice. It is thought to result from an estrogen/ androgen imbalance at breast tissue level, which can be caused by various pathophysiological mechanisms (1). Here we report a case of chemotherapy-related gynecomastia in a patient who had been previously treated for metastatic testicular embryonal cancer.

Case Report

A 35-year-old man was seen in our clinic for a 5-year history of painless bilateral gynecomastia. In April 2000, he had been admitted to urology clinic because of painless left testicular mass and diagnosed as having metastatic testicular embryonal carcinoma with normal beta human chorionic gonadotropin (hCG) levels and markedly elevated levels of α-fetoprotein (9204 IU/mL; normal: 0.5-5.0). He had been treated with cytotoxic chemotherapy including cisplatin (50 mg/m^2), bleomycin (30,000 IU), and etoposide (165 mg/m^2), followed by external radiotherapy for skin metastasis over the right frontal region of cranium. Six months after first course of chemotherapy he was noted to have bilateral, nontender, painless gynecomastia, which became more pronounced with time (Fig. 1). Bilateral gynecomastia with proliferation of fibroglandular tissue was confirmed by ultrasound. He had no erectile dysfunction, and his libido was normal. There was no history of use of drugs associated with gynecomastia. Serum total testosterone was 396 ng/dL (normal: 245-1600 ng/dL), free testosterone 14.4 pg/mL (normal: 12-30 pg/mL), estradiol 48.6 pg/mL (normal: 0-56 pg/mL), prolactin 11.6 ng/mL (normal: 2.5-17 ng/mL), luteinizing hormone 19.8 mIU/mL (normal:0.8-7.6 mIU/mL), follicle-stimulating hormone 58.4 mIU/mL (normal: 0.7-11.1 mIU/mL), and sex hormone binding globulin 29.7 mmol/mL (normal: 18-114 mmol/mL). Hemogram, thyroid, kidney, and liver function tests were within normal ranges. His karyotype was normal (46, XY). He had normal levels of α-fetoprotein and β-human chorionic gonadotropin and no evidence of recurrence of cancer on routine evaluation by the oncologist. The gynecomastia was established to be secondary to previous cytotoxic chemotherapy.

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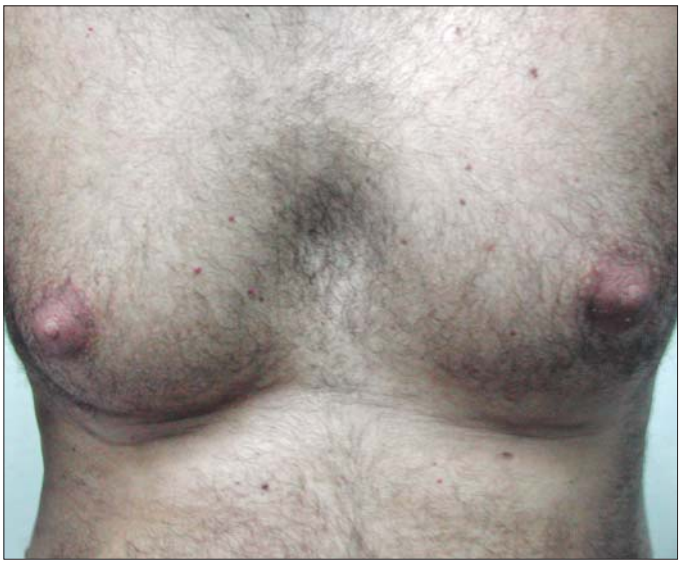

Figure 1. Bilateral gynecomastia of the patient

Discussion

Gynecomastia is the benign enlargement of the male breast due to proliferation of ductal tissue. Disturbed estrogen-androgen balance at the breast tissue level is the likely pathophysiological mechanism of this entity (2). Rarely, gynecomastia may occur in adult males after cytotoxic therapy for testicular cancer (3). Aki et al (4) reported a prevalence of 2.1% among 190 patients with metastatic testicular cancer who were treated by platinum-based combination chemotherapy and achieved complete remission. Toxic effects on Leydig cells and germinal epithelium as well as changes in the peripheral metabolism of testosterone and estrogen are believed to play a key role in the development of chemotherapy-related gynecomastia (3,5). Usually, serum testosterone levels decrease from supranormal to low-normal concentrations, and plasma estradiol rises to high levels (6). Slight increases in levels of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) due to damage of testicular tissue may be detected, as in our case. The stimulatory effect of the supra-normal serum FSH and LH concentrations associated with testicular damage due to cytotoxic chemotherapy on testicular aromatase activity results in increased conversion of testosterone to estradiol and increased testicular secretion of estradiol relative to testosterone (1,4,5).

It is important to recognize that chemotherapy-related gynecomastia does not reflect recurrence of testicular cancer (3). Additionally, chemotherapy-related gynecomastia was found to be associated with a better survival (6). Because gynecomastia may occur following cytotoxic chemotherapy for testicular cancer, and because it does not indicate recurrence of malignancy, a careful approach to diagnosis is suggested in order to avoid unnecessary investigation and treatment.

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