Psoriasis following adjuvant anastrozole therapy for breast cancer: a case-based review

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Summary

Anastrozole is a selective nonsteroidal aromatase inhibitor (AI), widely used in the treatment strategy for postmenopausal women with hormone-sensitive breast cancer. It induces estrogen depletion by decreasing androgen-to-estrogen conversion in the peripheral tissues. There are only few reports concerning cutaneous side effects of aromatase inhibitors including anastrozole. Here, we describe the first case of psoriasis induced by anastrozole in a menopausal woman. It is plausible that in our patient the administration of anastrozole, suppressing plasma estrogens levels, may have interfered with the state of immune tolerance shifting the inflammatory response toward a Th1 cytokine profile, responsible for the occurrence of psoriasis in a predisposed subject.

KEY WORDS: anastrozole; psoriasis; breast cancer.

Introduction

Anastrozole is a selective nonsteroidal aromatase inhibitor (AI), widely used in the treatment strategy for postmenopausal women with hormone-sensitive breast cancer. It induces estrogen depletion by decreasing androgen-to-estrogen conversion in the peripheral tissues. There are only few reports concerning cutaneous side effects of aromatase inhibitors in-

Case report

A 61-year-old woman was referred to our Department for a diffuse asymptomatic cutaneous eruption. She underwent a left radical mastectomy for a breast cancer 4 months previously, treated with radiotherapy, followed by anastrozole 1 mg tablet taken once a day, started 6 weeks before our evaluation. Physical examination showed reddish, slightly elevated patches covered with silvery-white scales symmetrically distributed on the trunk and thighs (Figure 1). The lesions were sharply demarcated, varied in size from 1 to 10 millimetres in a pattern clinically suggestive for guttate psoriasis (Figure 2). She had neither a personal or familial history of psoriasis, nor any exposure to any of the known exacerbating factors of the disease at the time of the event.

Discussion

The etiology of guttate psoriasis is not well understood although, as in other types of psoriasis, genetic
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Predisposition seems to play an important role in the development of the lesions. Factors identified as precipitating are infections, especially Group A streptococcal tonsillopharyngitis, and several drugs. Anastrozole is a selective nonsteroidal AI used as an adjuvant therapy for postmenopausal women with hormone-sensitive breast cancer. It markedly suppresses plasma estrogens levels by inhibiting aromatase, the enzyme responsible for the synthesis of estrogens from androgenic substrates in the peripheral tissues (1). Common adverse effects associated with AI’s include arthralgias, mialgias, other musculoskeletal disorders and an increased risk of bone fracture (2), but only few cutaneous side effects have been reported in literature. In particular, anastrozole has been responsible for the induction of subacute cutaneous lupus erythematosus (3), erythema nodosum (4), cutaneous vasculitis (5) and an unspecific pruritic micropapular eruption (6). Our case is the first description of psoriasis induced by an AI. Several lines of evidence suggest that psoriasis may fluctuate or be influenced by sex hormones such as estrogens and progesterone, but the mechanism has not yet been identified: a peak onset of disease at menarche, precipitation of psoriasis by oral contraceptives, amelioration during pregnancy and both improvement and exacerbation with menses have been described (7). The perimenarchal increase in the prevalence of psoriasis is explained by some authors by the increased levels of estrogens, which are known to promote keratinocyte proliferation via specific receptor-mediated mechanisms (7). Estrogens and progesterones levels increase throughout pregnancy until antepartum period and pregnancy is generally associated with improvement of psoriasis in over 50% of patients. On the other hand a decrease in estrogens during menopause is believed to be a major factor in the occurrence or exacerbation of psoriasis: this may explain the tardive peak of incidence of the disease (55-60 years in women) and the worsening of the skin condition that often occurs in menopausal women (8). Furthermore, recent in vivo and in vitro studies suggest that sex steroid hormones may regulate several immunological mechanisms in the skin. In particular, 17β-estradiol (E2) inhibits IL-12 and TNF-α production and antigen-presenting capacity in dendritic cells. It also downregulates the production of neutrophils, T-cells, and macrophages attracting chemokines by keratinocytes, while stimulates anti-inflammatory IL-10 production in T cells and dendritic cells (9). Therefore, a fall in estrogens concentration in postmenopausal women may lead to insufficient Th1 cell-mediated response inhibition, playing a major role in the exacerbation of psoriasis. It is plausible that in our patient the administration of anastrozole, suppressing plasma estrogens levels, may have interfered with the state of immune tolerance shifting the inflammatory response toward a Th1 cytokine profile, responsible for the occurrence of psoriasis in a predisposed subject.

Conflict of interest

The Authors declare that they have no conflicts of interest.

References