her LHRHa injection (Figure 1A). She recalled having a similar episode 3 months before, 48 h after her first LHRHa injection, with spontaneous resolution within 72 h. Full blood count and serum biochemistry did not reveal any abnormalities. Although blood and urine cultures were negative, oral antibiotics, anti-inflammatory drugs and analgesics were administered. MRI mammography showed inflammation of the skin without any pathological sign in the parenchyma. A fine needle aspiration demonstrated presence of inflammatory reactive cells and absence of malignant cells. Resolution of clinical signs and symptoms followed within 72 h from hospitalization (Figure 1B). One week later she underwent a programmed total hysterectomy because of uterine leiomyofibromas. She subsequently continued her treatment schedule with trastuzumab until May 2009 and is currently on regular follow-up without relapse of mastitis or cancer.

In view of the consistent appearance of two episodes of recall dermatitis of the irradiated breast within 48 h from LHRHa injection, spontaneous resolution within 72 h and lack of relapse upon LHRHa discontinuation, our final diagnosis was recall dermatitis induced by the LHRHa.

Recall dermatitis has been reported with a variety of drugs used in the treatment of breast cancer such as docetaxel, paclitaxel, doxorubicin, gemcitabine, methotrexate, tamoxifen and herceptin [1,2] but this is the first reported association of LHRHa with recall dermatitis. The exact cause is not known but several hypotheses have been proposed including increased sensitivity to drugs, changes of vascularization, DNA repair and radiation-impaired epithelial function of stem cells. In future, physicians should be aware of this rare association, rule out malignant relapse and seek alternate modalities of chemical castration.

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BRCA-deficient and triple negative breast cancers: is olaparib effective in both subtypes?

Dear Editor,

Olaparib (AZD-2281) is an inhibitor of PARP, an enzyme involved in DNA repair. It acts against cancers in people with hereditary BRCA1 or BRCA2 mutations, which include many ovarian, breast and prostate carcinomas. The study by Tutt and colleagues [1] indicated that oral poly (ADP-ribose) polymerase inhibitor olaparib has activity in BRCA-deficient breast cancer patients. We would like to address that olaparib may show a favorable therapeutic index for novel targeted treatment strategy not only in patients with tumors that have loss of BRCA-associated DNA repair but also in triple negative breast cancer (TNBC) patients without BRCA1 mutations.

Ninety percent of breast cancers in women with germline BRCA 1 mutations are TNBC [2]. On the other hand, it is found that up to 10% of the patients with TNBC bear this mutation [3]. However, in the study by Tutt et al. [1] most of TNBC patients had BRCA 1 mutations (11 of 13 in a cohort assigned to olaparib 400 mg, and 11 of 16 in a cohort assigned to olaparib 100 mg). Therefore, the question arises whether olaparib will have activity with TNBC without a BRCA1 mutation.

Histologically and transcriptionally, TNBCs have many similarities to BRCA1- associated breast cancers, which suggests that dysfunction in BRCA 1 or associated pathways occurs in this subset of sporadic cancers. There are many phenotypical and molecular features shared, including ER negativity, high nuclear grade, high Ki-67 staining, CK 5/6 expression, EGFR expression, high degree of genomic instability and p53 tumor suppressor gene mutation [3]. In addition, clustering analyses of microarray RNA expression data have shown that familial BRCA1 mutant cancers strongly segregate with TNBCs, suggesting similar carcinogenic pathways or causes of these two subtypes [4].

Results of another study [5] showed that BRCA1 messenger RNA expression was two-fold lower in basal-like breast cancers compared to matched controls (p=0.008). ID4, a negative regulator of BRCA1, was expressed at 9.1-fold higher levels in basal-like breast cancer ($p < 0.0001$), suggesting a potential mechanism of BRCA1 downregulation.

In conclusion, these findings suggest that even if BRCA1 is rarely mutated in sporadic TNBCs, BRCA1 or associated pathways can become inactivated in TNBCs via some mechanisms. Since there are limited treatment options for TNBC, olaparib is a promising agent for future trials in patients with TNBC.

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