Radical resection of thyroid cancer infiltrating the trachea, with circular tracheal resection and termino-terminal anastomosis, followed by radioiodine therapy should be considered as the treatment of choice.

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A. Filipovic[1], L. Vuckovic[2], M. Mijovic[3]

[1]Department of Endocrine Surgery, [2]Department of Pathology, [3]Department of Chest Surgery, Clinical Centre of Montenegro, Podgorica, Montenegro

Correspondence to: Aleksandar Filipovic, MD, PhD.
E-mail: a.filipovic@t-com.me

Recurrent episodes of recall dermatitis of irradiated breast after LHRH agonist administration

Dear Editor,

Radiation recall dermatitis has been defined as an inflammatory reaction of a previously irradiated area, in response to the administration of certain drugs. Although this phenomenon is relatively well known in the literature, the exact cause has not been documented. We report a case of radiation recall dermatitis in a breast cancer patient receiving adjuvant treatment with an LHRH analogue (LHRHa).

In April 2008 a 47-year-old lady underwent right breast quadrantectomy and axillary lymph node dissection for management of a signet ring breast adenocarcinoma, pT1cN0Mx, ER/PgR positive and HER2 overexpressing (+3 IHC). Staging was negative for distant metastasis, so she received adjuvant chemotherapy from May to September 2008 with 6 cycles of docetaxel, carboplatin and trastuzumab. In October 2008 she had conventional-fractionation external beam radiation therapy to the entire breast (45 Gy in 23 fractions) and continued on trastuzumab every 3 weeks. The patient was put on hormonal therapy with daily tamoxifen and intramuscular LHRHa goserelin 10.75 mg every trimester.

In January 2009 she presented to our department with pain, swelling, tenderness and erythema on the irradiated breast, fever 39° C and chills, 48 h after having

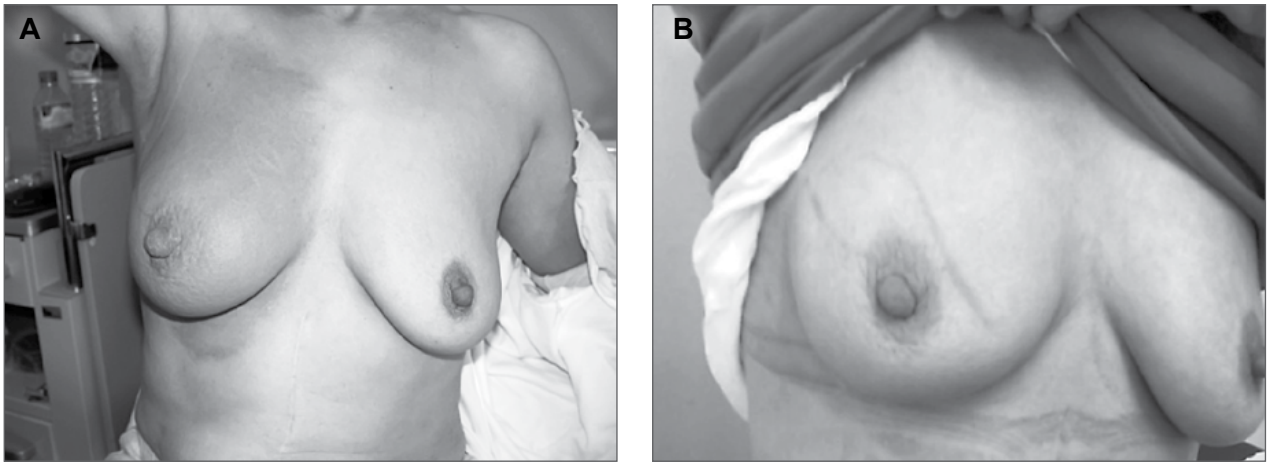

Figure 1. A: recall dermatitis of the right breast after the LHRHa injection. **B:** same patient 72 hours after her hospitalization.

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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L. Kostadima, G. Pentheroudakis, N. Pavlidis

Department of Medical Oncology, Ioannina University Hospital, Ioannina, Greece

Correspondence to: Dr. Lida Kostadima. E-mail: lidakosta@gmail.com

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].