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Radiation recall reaction with docetaxel administration after accelerated partial breast irradiation

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Abstract
Purpose: as the use of accelerated partial breast irradiation (APBI) becomes more widely used it is important to define and characterize the possible toxicities encountered with this type of therapy to help understand how to prevent these toxicities in the future.
Methods and materials: a 60-year-old woman was treated with APBI using external beam radiation. Three weeks post radiation therapy, she was given a cycle of docetaxel and cyclophosphamide. Within 3 weeks of chemotherapy she developed a radiation dermatitis ulceration in her right axilla and tail of her right breast. Little data exists regarding the association between the actual volume of skin irradiated during external beam APBI and the development of skin toxicity such as RRD, therefore we analyzed the volume of skin getting various prescription doses in our patient and compared them to 30 similar breast cancer patients treated with external beam radiation APBI at the same institution.
Results: our patient's volume of skin getting 100%, 90% and 80% of the prescription dose was below the mean for all three doses when compared to all other patient's treated similarly at our institution.
Conclusions: we present an example of radiation recall dermatitis in a patient receiving APBI with external beam radiation followed by chemotherapy. In our case, volume of skin irradiated did not appear to be associated with the development of RRD, therefore other factors may have led to her development of this rare skin reaction.
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1. Introduction

Radiation recall dermatitis (RRD) is an acute inflammatory condition of the skin in a previously irradiated area, generally triggered by administration of certain agents, such as antineoplastic drugs. Several cases of RRD have

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been described using numerous chemotherapy agents and various methods of radiation delivery, but here we describe a case of radiation recall in a patient who received docetaxel and cyclophosphamide chemotherapy following accelerated partial breast irradiation (APBI) with external beam radiation.

2. Case report

A 60-year-old black female presented with a new suspicious mass in the posterior lateral aspect of the right breast found on screening mammogram on March 17, 2012. Ultrasound guided core biopsy revealed an infiltrating lobular carcinoma with focal LCIS with ductal extension. She underwent right breast lumpectomy and sentinel lymph node biopsy on April 5, 2012. Final pathology revealed a 1.2 cm lobular carcinoma with negative margins and one benign sentinel lymph node. Final staging was reported as pT1cN0M0 (stage IA). The tumor was estrogen receptor and progesterone receptor positive and Her2neu not overexpressed. Post-operatively she was placed on trimethoprim/sulfamethoxazole for redness noted at the incision site, with spontaneous resolution of symptoms.

She was next treated with APBI via external beam radiation beginning on May 10, 2012. The seroma and clips were treated in 10 fractions over the course of five days. She received a total of 38.5 Gy over those five days with treatment twice a day of 3.85 Gy per dose. Her course of radiation treatment concluded on May 16, 2012.

On June 7, 2012, exactly 3 weeks post-radiation therapy, chemotherapy was initiated, consisting of docetaxel 75 mg/m^2 and cyclophosphamide 600 mg/m^2. When she presented for her second course of chemotherapy on June 21, 2012 it was noted that the patient had developed a 2×2 cm^2 area of moderate erythema with edema and moist desquamation her right axilla and tail of the right breast. The chemotherapy was tentatively held at that time and the patient was instructed to keep the area clean and apply duoderm until healed. Cycle 2 was delayed for 6 weeks and was restarted once the skin had healed to a wound measuring 5 mm in her axillary fold. Following the second cycle of chemotherapy the patient suffered from a recurrence of the RRD consisting of desquamation and a non-healing wound in the right axilla. At this time the patient's chemotherapy was switched to weekly paclitaxel 80 mg/m^2. One week after her first cycle of paclitaxel the 5 mm open wound in the right axilla remained unchanged from before the therapy. In August 2012 she began to apply silver sulfadiazine to dress the area with moist desquamation. She was continued on weekly paclitaxel treatments, which she completed on September 27, 2012. The patient began aromatase inhibitor therapy on October 15, 2012.

Our patient had a complicated course of healing from the RRD. At her six month follow-up appointment in February 2013 she presented with right upper extremity and breast lymphedema, which was treated with Flexitouch therapy three times per week. On March 29, 2013 the patient presented to the office febrile, with her right breast warm, erythematous and tender. She was placed on a course of erythromycin with slight improvement. Two weeks later she was placed on ciprofloxacin and rifampin with improvement of the pain and swelling. She applied Aquaphor to the site and also underwent physical therapy, psychological therapy and lymphadema therapy for the dermatitis. The patient's pain was so severe during this time she was placed on a pain regimen, using 4 break-through tabs of 5 mg oxycodone daily with twice daily 30 mg oxycodone ER for maintenance. Patient received steroid injections in her right shoulder and followed up with an orthopedic surgeon and a physical therapist. In January 2013 patient was placed on pentoxifylline and vitamin E in hopes of improving her right axillary and right breast symptoms. The wound eventually healed completely after 18 months and was an open ulceration for at least a year of that time.

3. Discussion

Our case demonstrates an example of RRD precipitated by treatment with docetaxel and cyclophosphamide, following the use of APBI with external beam radiation. There are many proposed hypotheses for the development of RRD, but there are currently no known predictive factors. These hypotheses focus on vascular, epithelial stem cell inadequacy, and epithelial stem cell sensitivity of drug hypersensitivity reactions as the mechanism for RRD [1]. Bostrom et al. have proposed that the radiotherapy induces local vascular permeability or a proliferative change that upon exposure to certain drugs elicits the RRD by affecting the subsequent pharmacokinetics of the drug [2]. Hellman and Botnick have proposed that by irradiating the skin, that region of epithelial stem cells becomes depleted and the stem cell numbers never fully recover [3]. Seymour et al. went on to add that although stem cells were present following the radiotherapy, their ability to proliferate and been permanently altered disabling them from performing their normal function as a stem cell [4]. Based on the speed of onset and drug specificity of RRD Cambridge and Price propose that the mechanism is not based on the triggering drug's cytotoxicity, but on idiosyncratic drug hypersensitivity reactions instead via non-immune activation of inflammatory pathways [1].

While the etiology of RRD still remains unknown, the phenomenon is still relatively rare. Many recorded cases have begun to piece together some common denominators. Many reactions have been documented with cytotoxics, and several chemotherapeutic drugs in particular have been implemented in this type of reaction, including adriamycin, bleomycin, docetaxel, 5-fluorouracil, gemcitabine, paclitaxel, vinblastine, and tamoxifen. The majority of breast cancer patients receiving conventional whole breast external beam irradiation receive their radiation after chemotherapy. However, unlike whole breast irradiation, APBI is typically given before chemotherapy, and the appropriate amount of spacing between treatments has been brought into question. Multiple studies have also focused on the time course that occurs between administration of chemotherapy and radiation. It has been reported that RRD is more prevalent, with higher rates of reactions, in those receiving chemotherapy within 3 weeks of radiation with increasing prevalence as the interval shortens [5]. A case documented with docetaxel administration after APBI with electronic brachytherapy, found that in patients receiving electronic brachytherapy, a minimum surface-to-skin distance

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