Severe Necrosis Due to Paclitaxel Extravasation: Case Report

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Paclitaxel (Taxol) is an agent with antineoplastic properties that is derived from the bark of the Pacific yew tree, Taxus brevifolia. It has been shown to have activity against many tumors including lung, breast, and ovarian carcinomas. Its mechanism of action involves the induction of microtubular assembly and stabilization. Ultimately, this leads to cell death. In the past, the extravasation of paclitaxel has been known to have qualities considered to be similar to those of local irritants. Recent reports have suggested that its extravasation may cause vesicant reactions. Local venous effects including immediate or delayed reactions have been described. Patients have experienced local reactions including pain, swelling, erythema, induration, and hyperpigmentation, without ulceration. In this article, the authors report on a patient who experienced a delayed vesicant reaction to a paclitaxel extravasation that resulted in severe necrosis.

The patient in this report was a 53 year old woman with a history of stage IIB adenocarcinoma of the right breast. She was initially treated with breast conservation and adjuvant doxorubicin and cyclophosphamide for four cycles followed by tamoxifen. After three years of tamoxifen therapy, metastatic disease was detected in her lumbar and thoracic spine. Her treatment plan was for four cycles of paclitaxel in preparation for high-dose chemotherapy with peripheral blood cell transplant. The patient received 250 mg/m2 of paclitaxel (total dose 400 mg diluted in 1000 ml normal saline) infused over 24 hours through a peripheral line. She was premedicated with dexamethasone, diphenhydramine, and cimetidine as preventative measures against hypersensitivity reactions. No immediate reactions or complaints were reported during the infusion. However, two to three days later, the woman developed erythema and pain at the infusion site. Not seeking medical attention, by day 11 the pain had worsened, and two elliptically shaped necrotic areas, 1 x 4 cm in size were present, as well as a surrounding area of cellulitis with a central eschar region. Following debridement of the necrotic tissue, the patient was treated empirically with cephalaxin and silver sulfadiazine. The wound was surgically closed. A culture obtained from the necrotic area on day 11 grew methicillin-sensitive Staphylococcus aureus and Peptostreptococcus asaccharolyticus. Histopathologic examination of a tissue sample showed ulceration with reactive granulation tissue, fat necrosis, and fibroblastic proliferation. When the necrotic area had resolved (day 32) the patient received a second course of paclitaxel through a central venous catheter without complications. The infusion wound site was completely healed by day 50.

At the current time, there is no antidote for the condition. Treatment is primarily palliative with the application of compresses (warming and/or cooling). Other case reports have described quicker resolution of symptoms with cooling alone when compared with the addition of hyaluronidase.

The authors recommend against prolonged paclitaxel infusions through peripheral lines due to the potential for necrosis if extravasated.