Successful Treatment of Thrombocytopenia Due to Marrow Metastases of Breast Cancer with Weekly Docetaxel

Patients with cytopenias due to marrow metastases of breast cancer *(1)* are generally excluded from clinical trials because of the eligibility requirements for near-normal hematologic parameters. Moreover, for patients with endocrine-insensitive disease *(2),* the myelosuppressive effect of most cytotoxic drugs complicates attempts at chemotherapy *(3–5).*

The principal dose-limiting toxicity of full-dose docetaxel (typically 75–100 mg/m^2 every 3 weeks) is neutropenia; however, there is less myelosuppression with lower dose (typically 25–35 mg/m^2 every week) schedules *(6).* Many clinicians have now routinely adopted this low-dose weekly docetaxel schedule for the treatment of patients for whom neutropenia would pose a substantial risk for life-threatening complications. We have observed sustained platelet recovery, without intervening neutropenic complications, in two patients with transfusion-dependent thrombocytopenia due to marrow metastases of breast cancer using this low-dose approach.

In the first case, a 47-year-old woman diagnosed with primary breast carcinoma in 1986 was treated with mastectomy, radiotherapy, and 6 months of tamoxifen therapy. The patient was treated with radiotherapy and tamoxifen therapy for spinal metastases in 1996 and with anastrozole and pamidronate for progressive bony metastases in 1998. On January 15, 2000, the patient's platelet count was 15×10^9/L; a bone marrow biopsy revealed metastatic breast carcinoma. The patient was treated with low-dose (30 mg/m^2) weekly docetaxel therapy. The patient's platelet count rose steadily, reaching 80×10^9/L within 3 weeks (without any complicating neutropenia) and 159×10^9/L within 6 weeks. Weekly docetaxel therapy was continued until May 15, 2000, when the patient was switched over to docetaxel (75 mg/m^2 every 3 weeks) for an additional two treatments and was continued thereafter on pamidronate (90 mg every 4 weeks). In September 2000 the patient's platelet count began to drop (132×10^9/L), and by October, 2000, it had dropped to 70×10^9/L. The patient was treated unsuccessfully with capecitabine (1250 mg/m^2 twice daily for 10 days), and her platelet count continued to decline, reaching a nadir of 45×10^9/L. Weekly docetaxel (30 mg/m^2) was reinitiated in November 2000. The patient's platelet count recovered and remained within the normal range until her death with progressive cancer in June 2001.

In the second case, a 64-year-old woman presented in February 2001 with estrogen receptor-negative metastatic breast cancer that involved bone, marrow (histologically proven), and pleura. The patient's platelet count was 24×10^9/L. Weekly treatment with docetaxel (25 mg/m^2) was initiated on February 19, 2001, and continued for 6 consecutive weeks. Within 1 week of the initiation of docetaxel therapy, the patient was platelet-transfusion independent; because this patient lived in a geographically remote area, a further transfusion was administered on March 6, 2001, when the patient's platelet count was 40×10^9/L. The patient's leukocyte count nadir was 2.6×10^9/L, and she never developed any neutropenic com-

plications. On the day of her sixth scheduled treatment, the patient's platelet count was 82×10^9/L, and on April 23, 2001, following a scheduled treatment break, her platelet count was 138×10^9/L. The patient's platelet count remained within the normal range for 2 months, during which time she developed progressive malignant pleural effusion. The patient died on June 25, 2001, with a platelet count of 99×10^9/L.

Interestingly, both patients achieved prompt platelet independence without neutropenic complications using a low-dose schedule of docetaxel. Low-dose docetaxel appears, therefore, to be an appropriate candidate for formal prospective studies in patients with thrombocytopenia due to marrow metastases of breast cancer, a potentially morbid and expensive clinical scenario *(7)*. The marrow-sparing effect of low-dose docetaxel therapy, together with the broad activity of taxanes, suggests a basis for prospective studies in other poor-risk, clinical trial-underrepresented populations of cancer patients.

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NOTES

Editor's note: J. Crown has accepted honoraria from Aventis Pharmaceuticals (Bridgewater, NJ), the makers of docetaxel.

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