

istration, there will be no added radiation dose to the soft tissues surrounding the port.

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Disseminated Intravascular Coagulation in Metastatic Prostate Cancer

TO THE EDITOR: The article by Leong et al. (*J Nucl Med* 1994;35:1662-1664) merits further discussion of the factors to consider in selecting patients who will receive ^{89}Sr therapy for palliation of pain from osseous metastases.

The patient described had pre-existing anemia and thrombocytopenia, which the authors term "low-grade DIC," prior to ^{89}Sr therapy. There is no mention of the active management of the DIC with heparin and blood products in the article. No platelet levels are given between Day 10, when they were 53×10^9 per liter, or Day 31, when the first platelet infusions were given. There is no detail on the progression of the condition. The anemia was actively managed by packed red cell infusions. No evidence, however, is given of the coagulopathy being managed prior to Day 31.

In clinical trials, we have seen no evidence of suppression of the red cell series or of coagulopathy as a result of ^{89}Sr therapy. We do report thrombocytopenia and leukopenia as a result of the marrow suppression by ^{89}Sr . We previously reported two cases of DIC or consumptive coagulopathy (which is typically associated with severe infections, septic shock and advanced malignancy) in patients who were given ^{89}Sr , but this was ascribed to the underlying disease. This represents three known cases in a total of over 12,000 patients treated so far.

Although the authors concede that no causal relationship was established, this article does highlight the need for caution in patients with compromised bone marrow, as mentioned in the package insert and, importantly, emphasizes the need for active specific management of treatable hematological conditions prior to therapy.

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Bisphosphonate Effect on Bone Scintigraphy

TO THE EDITOR: We read with interest the article by Pecherstorfer et al. on the effect of bisphosphonate treatment on bone scintigraphy (1) and their reply (3) to the letter to the editor by Kayano et al. (2). Bisphosphonates are powerful inhibitors of osteoclast-mediated bone resorption. They are used not only for the treatment of hypercalcemia, but also for the treatment of Paget's disease and bone metastasis. Among various bisphosphonates, etidronate is the first generation bisphosphonate and has a unique action for bone metabolism; that is, etidronate action prevents bone resorption in a range of small amount but etidronate produces bone resorption above the threshold amount. Etidronate was reported to disturb the bone scintigraphy (4,5), but clodronate did not (1).

We recently observed that YM-175, a new bisphosphonate 7-10 times as potent as pamidronate, had no significant effect on bone scintigraphy

TABLE 1
Bone Scan Index

Patient no.	Pretreatment	4 wk	8 wk
1	3.47 \pm 2.50*	2.83 \pm 1.55	3.35 \pm 1.58
2	3.10 \pm 1.05	3.66 \pm 1.79	4.03 \pm 2.04
3	3.15 \pm 1.79	3.17 \pm 1.21	3.30 \pm 1.16
4	2.66 \pm 0.71	3.15 \pm 1.33	2.90 \pm 0.73
Average†	3.09 \pm 1.58	3.16 \pm 1.35	3.30 \pm 1.33

*Mean value and 1 s.d. of the ratio of abnormal uptake sites and normal counterparts (5-6 sites in each patient) was shown.

†No statistical significance was detectable by ANOVA.

(6). We intended to treat the bone metastatic pain with YM-175. Some of the patients were monitored by bone scintigraphy. We administered YM-175 (at a dose of 10 mg once a week for 5 wk intravenously). Bone scintigraphy was performed at pre-, 4- and 8-wk. Quantitative bone scintigraphy was performed according to the methods of Pecherstorfer et al. (1). Briefly, a region of interest was placed on bone metastasis and opposite nonmetastatic site, and the ratio of bone metastatic site to nonmetastatic counterpart was calculated. As shown in Table 1, no statistical difference was shown between pre-, 4- and 8-wk bone scintigraphic values. Furthermore, in one patient in whom bone scintigraphy was performed just after administration of YM-175 and no change was shown. Only etidronate appears to interfere with bone scintigraphy.

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Radioiodine Therapy of the Autonomous Thyroid Nodule in Patients with or without Visible Extranodular Activity

TO THE EDITOR: The interesting paper by Clerc et al. (1) describes the outcome of 88 patients ~6 yr after radioiodine therapy (intended dose of 80 Gy) for an autonomous thyroid nodule. Forty-nine patients without scintigraphically visible extranodular activity fulfilled the criteria of scintigraphic decompensation, another 39 did not; 9 of 88 patients had nonsuppressed TSH levels (TSH > 0.1 mU/liter or TRH test > 2 mU/liter). Uptake monitoring and calculation of the doses absorbed by the autonomous thyroid nodule and the heterolateral extranodular area (in patients with and without extranodular activity) are beautifully presented. Unfortunately, the effectively measured results are disappointing in that 72 Gy (in case of extranodular activity) or 57 Gy (no extranodular activity) to the nodule are rather low doses to eradicate an autonomous thyroid nodule [a monoclonal growth due to mutations of the TSH receptor genes (2,3)], whereas doses of 8.5 Gy or 49 Gy absorbed by the extranodular area are too high to preserve full function. Thus, while late hypothyroidism is not prevented, hardly tolerable rates of therapy failures result.

As compared to our results, based on an internationally established dosimetric approach (4), 72 patients treated with 300 Gy to their auto-