



Painful Abdominal Ecchymotic Lesions

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Case Study

Ms. B is a 57-year-old female who had a 1.5 mm Clark level IV amelanotic melanoma removed from her right eyebrow in 1998. She was disease free until September 2001, when she presented with a mass below her left mandible; an open biopsy confirmed the recurrence of melanoma. She also was found to have a 7 mm lung lesion that was positive for melanoma. The treatment plan included both chemotherapy and surgery. Prior to initiating chemotherapy, Ms. B underwent a wedge resection of the metastatic lesion in her lung followed by two cycles of dacarbazine, vinblastine, cisplatin, interleukin, and interferon. After completing the initial two cycles, the left submental mass was removed. She then received two additional cycles of chemotherapy.

Ms. B's therapy was complicated by some anticipated side effects, including fever, intractable nausea, hypokalemia, and hypomagnesemia. After she was admitted to the hospital for cycle three, she developed a staphylococcus sepsis and thrombus of her central venous catheter (CVC). The CVC was removed, and treatment was initiated with antibiotics and warfarin 5 mg per day at bedtime. The international normalization ratio goal was 2–3, and Ms. B's was maintained at 2.4–2.6 during her therapy. On day six of warfarin therapy, she developed painful ecchymotic lesions with irregular borders across her lower abdomen. Some of the ecchymotic areas had a dark red erythematous flush, and the skin was broken over some of the darkest lesions. The oncologist, oncology clinical nurse specialist, and primary care nurse examined the lesions and



reviewed the medication and patient profile for potential causes of the skin lesions. The patient's laboratory values and other parameters were normal. Warfarin was the only medication that had been added to her therapy that week, which led to the suspected diagnosis of warfarin-induced skin necrosis. The warfarin was discontinued immediately, and Ms. B received vitamin K 5 mg and was started on Lovenox® (Aventis Pharmaceuticals, Bridgewater, NJ) 60 mg twice daily. Protein C and S levels were obtained, and the ecchymotic lesions spontaneously faded over the next two days following the discontinuation of warfarin therapy. Both protein C and S levels were significantly low compared to normal values. These low protein levels, combined with the appearance of the initial lesions and spontaneous fading, confirmed the diagnosis of warfarin-induced skin necrosis. The open lesions eventually healed, and no further sequelae of the skin or subcutaneous tissue appeared. Ms. B was discharged from the hospital several days later following CVC replacement and completion of chemotherapy. Lovenox was discontinued at the time of discharge.

Discussion

Oral anticoagulant therapy can result in several adverse skin manifestations, including ecchymosis, hemorrhagic necrosis, and urticarial eruptions. Warfarin-induced skin necrosis first was described in 1942 and is a rare complication that occurs in 0.1% of the population treated with anticoagulant therapy (Chan, Valenti, Mansfield, & Stansby, 2000; RxList, n.d.; Warkentin, 2001). A brief and simplified review of hemostasis, including the clotting cascade and hemostatic control mechanisms, is necessary to better understand this rare adverse event.

Hemostasis is defined as a sequence of events that stops bleeding. Three mechanisms take place to reduce blood loss: a vascular spasm, platelet plug formation, and blood clotting. The vascular spasm occurs when the arteries and arterioles have been damaged and is triggered by damage to the smooth muscle and stimulation of pain receptors. Platelet plug formation occurs to prevent blood loss in small vessels. Clotting is a complex cascade of reactions that takes place in pathways; each clotting factor activates the next one in a fixed sequence. Twelve clotting factors are known, including calcium ions, several inactive enzymes synthesized by hepatocytes, and

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