

Cutaneous Involvement in Hodgkin's Disease

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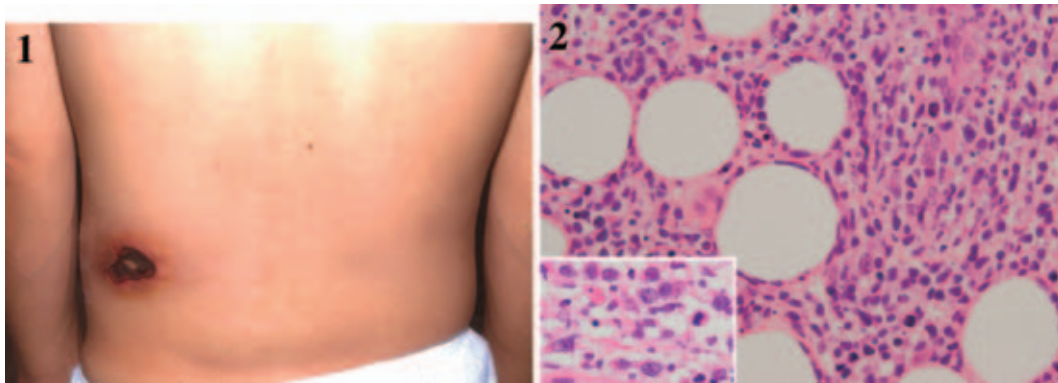


Figure 1. Patient with Hodgkin's disease showing an ulcerated skin lesion in the left lower back.
Figure 2. Section from the back skin lesion showing a subcutaneous infiltrate consisting of atypical large cells, histiocytes, and lymphocytes. Classical Reed-Sternberg cells are not present but mononuclear cells with prominent nucleoli are seen (HE stain).

A 44-year-old man was hospitalized because of fever, night sweats, and cervical lymphadenopathy. An abdominal computed tomography scan revealed paraaortic lymphadenopathy and hepatosplenomegaly. He was seronegative for HTLV-I. A neck lymph node biopsy demonstrated lymphocyte-depletion Hodgkin's disease. He was given six courses of combination chemotherapy (cyclophosphamide, vincristine, prednisolone, procarbazine) and complete remission was achieved. However, 32 months after diagnosis, relapse occurred with hepatomegaly and paraaortic lymphadenopathy. Chemotherapy was resumed with doxorubicin, bleomycin, vinblastine, and dacarbazine with partial remission. Then he developed a 2.5-cm ulcerated nodule in the skin of the left lower back (Fig. 1). A biopsy of this skin lesion revealed multifocal infiltrates of atypical cells intermixed with histiocytes, lymphocytes, and mitotic figures in the dermis and subcutaneous tissue. The atypical cells had

large hyperchromatic nuclei, some of which showed prominent nucleoli, resembling Hodgkin's cells (Fig. 2). While the back skin lesion was being treated with etoposide, ifosfamide, and vindesine, diffuse infiltration of the facial skin appeared; this infiltration responded poorly to electron beam irradiation. The patient declined further chemotherapy and died 5 months after the skin lesion was first noted on his back. Lymphomatous involvement of the skin is seen relatively frequently in non-Hodgkin's lymphoma but it is rare (0.5-3.4%) in Hodgkin's disease. Cutaneous Hodgkin's disease is manifested as papules, nodules, plaques, or ulcerative lesions, usually in late stages of the illness. The proposed mechanisms are retrograde lymphatic spread from a proximal nodal focus, direct extension from an underlying nodal focus, and hematogenous spread of tumor cells. Cutaneous involvement, which represents stage IV disease, generally indicates an ominous prognosis.

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