

The patient's condition continued to improve although small amounts of blood were intermittently passed through the rectum. In an attempt to localize the source of blood loss, various tests were performed; barium enema, gastroscopy, and technetium scan of the abdomen revealed no abnormality. Coagulation screen and platelet count had remained normal throughout. During this period the skin had been steadily improving, and although a further significant hemorrhage was clearly a possibility, laparotomy was not considered advisable in view of the patient's general condition.

However, the lower abdominal pain and perineal pain recurred suddenly, followed by a further severe episode of rectal bleeding. On this occasion the hemorrhage persisted, and despite the replacement of large quantities of blood the patient became severely hypotensive. An emergency laparotomy was then arranged.

At operation the source of hemorrhage was identified as a single spurting vessel located just within the anorectal junction and arising from otherwise normal-looking mucosa. Ligation of this vessel resulted in arrest of the hemorrhage. At this stage, blood began to well up from the pelvic cavity, and despite all efforts the hemorrhage could not be arrested. Clotting was not seen to occur, and it was considered that this terminal, uncontrollable hemorrhage was caused by a failure of coagulation resulting from massive transfusion.

The postmortem examination failed to reveal a specific cause for the hemorrhage. In particular, there was no evidence, either on routine investigation or on direct immunofluorescence examination, of pemphigus vulgaris involving the bowel.

Discussion. Pemphigus vulgaris is a disorder that, prior to the advent of systemic corticosteroid therapy, was largely untreatable and frequently fatal.¹ Despite modern treatment it remains a serious disease with a significant mortality rate. The most common cause of death is infection, with septicemia and pneumonia being responsible for the majority of fatalities.^{2,3}

Significant hemorrhage from the large bowel is extremely unusual in patients with pemphigus, and when it does occur, there is usually some other associated abnormality to account for it.³

This case is the first, to our knowledge, in which a patient suffering from pemphigus vulgaris has died as a direct consequence of uncontrollable hemorrhage from a rectal vessel in this fashion. We suggest that the possibility of this association be borne in mind in the management of patients with pemphigus and large-bowel hemorrhage of uncertain origin.

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Acquired ichthyosis in a patient with acquired immunodeficiency syndrome and Kaposi's sarcoma

To the Editor: We have observed a case of acquired ichthyosis in a patient with the acquired immunodeficiency syndrome (AIDS) and Kaposi's sarcoma. To our knowledge, the association of acquired ichthyosis with AIDS-related Kaposi's sarcoma has not been previously reported. Acquired ichthyosis has been reported in association with the classic form of Kaposi's sarcoma (multiple idiopathic hemorrhagic sarcoma).¹⁻³

Case report. A 32-year-old bisexual man was admitted to the San Diego Veterans Administration Medical Center in October 1985 with a diagnosis of AIDS. His symptoms had begun approximately 18 months prior to admission, with the onset of fatigue, diarrhea, a 60-pound weight loss, and abnormal neurologic behavior. Six months prior to admission, the patient noticed reddish brown papules on his arms and the onset of generalized dry skin. There was no prior personal or family history of ichthyosis. The patient's past medical history included treatment of syphilis and gonorrhea, as well as hepatitis B. He was taking no medications known to induce ichthyosiform skin changes.

Examination of the patient's skin revealed multiple 0.5-cm, reddish brown papules scattered on his left arm and the upper part of his back. In addition, there was generalized ichthyosis with large and adherent scales on the trunk and extremities, consistent with a diagnosis of acquired ichthyosis

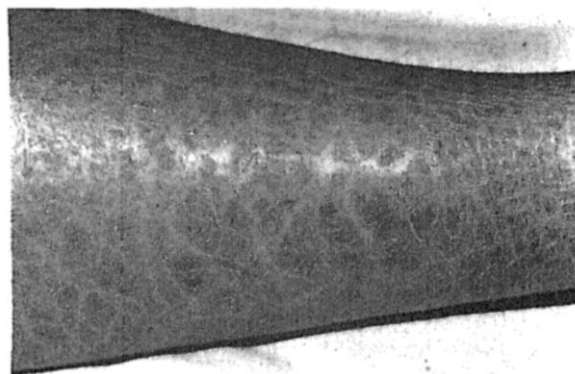


Fig. 1. Ichthyotic skin on the patient's right leg.

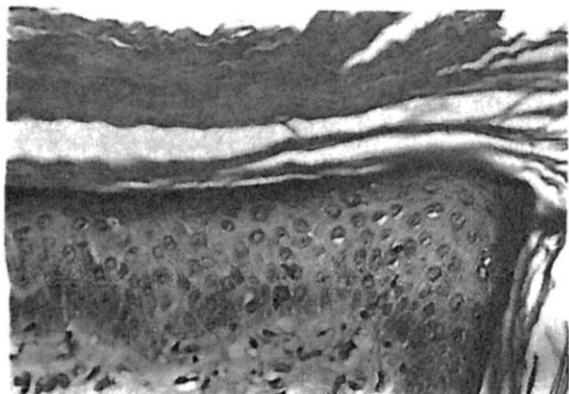


Fig. 2. Low-power view of skin biopsy specimen, showing compact hyperkeratosis, an intact granular layer, slight acanthosis, and a normal-appearing dermis. (Hematoxylin-eosin stain; $\times 100$.)

(Fig. 1). The ichthyosis spared the flexures. The patient was also noted to have oral candidiasis, and *Cryptococcus* organisms were cultured from his stool. Results of thyroid function tests were normal.

Histopathologic examination of representative skin papules confirmed the diagnosis of Kaposi's sarcoma. A biopsy specimen of representative ichthyotic skin (Fig. 2) showed hyperkeratosis, an intact granular layer, slight acanthosis, and an unremarkable underlying dermis, consistent with a diagnosis of ichthyosis.

Discussion. Acquired ichthyosis is most commonly reported in association with Hodgkin's disease and has also been reported in association with reticulolymphosarcoma, aplastic anemia, mycosis fungoides, spindle-cell sarcoma, multiple myeloma, breast carcinoma, and metastatic lung carcinoma.⁴ Acquired ichthyosis has also been reported in association with several cases of the classic form of Kaposi's sarcoma.¹⁻³ Although there has been one previous report of acquired ichthyosis in association with AIDS,⁵ to our knowledge there have been no previous reports of acquired ichthyosis associated with AIDS-related Kaposi's sarcoma. The temporal relationship between the onset of this patient's Kaposi's sarcoma and the onset of acquired ichthyosis leads us to believe that the two conditions were related.

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Norwegian scabies in acquired immunodeficiency syndrome: Report of a case resulting in death from associated sepsis

To the Editor: The crusted (Norwegian) form is an uncommon and distinctive variant of scabies. It is usually associated with immunosuppression or neurologic impairment, although it has rarely been reported in normal persons. Its incidence is increasing, perhaps as a result of the increasing use of immunosuppressive agents. In contrast to the common form of scabies, in which less than a score of adult female mites are present,¹ hundreds to thousands of *Sarcoptes* mites infest patients with this form.² It is highly contagious, even through fomites. Patients have hyperkeratotic plaques on erythematous bases. Lesions may be generalized but are especially prominent on the neck, scalp, and trunk. Subungual spaces may be involved.³ Itching is usually minimal. Associated findings include lymphadenopathy, eosinophilia, elevated levels of IgE,⁴ and decreased levels of IgA.⁵ We describe a case of crusted scabies in a patient with acquired immunodeficiency syndrome (AIDS) who died with bacteremia, pericarditis, and pneumonia.

Case report. A 35-year-old homosexual man presented to the University of California, San Diego, Medical Center because of increasing weakness, shortness of breath, and a generalized eruption with painful fissuring. He had had multiple male sexual partners, and he had a 1-year history of fever and chills. Three months prior to his presentation, skin lesions appeared as linear, red, intensely pruritic "welts." Pruritus gradually lessened as the lesions thickened and coalesced to cover most of his body. Several days prior to the patient's admission to the medical center, painful fissures developed in the flexures.

The patient was acutely ill with shaking chills and dyspnea. His oral temperature was 96° F, blood pressure was 116/60 mm Hg, and pulse was 100 beats/min. There was jugular vein distention, and S₃ heart sound, hepatosplenomegaly, pitting edema to the mid portion of the thigh, and cervical and axillary lymph node enlargement.

There were striking gray-brown hyperkeratotic plaques