ATYPICAL CUTANEOUS HERPES SIMPLEX INFECTION ASSOCIATED WITH ACUTE MYELOGENOUS LEUKEMIA

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It is well known that persons with malignant reticuloses are more susceptible to infection with the zoster virus than normal persons (1, 4). An altered immunologic status is thought to be responsible. Cutaneous herpes simplex infection also seems to be more common in persons with debilitating diseases including malignant reticuloses; however, the absolute incidence has not been established. We recently observed a patient with acute myelogenous leukemia who developed an unusual vesicular eruption which appeared to be an atypical manifestation of zoster, but a cutaneous biopsy and tissue culture of fluid proved it to be an atypical example of herpes simplex infection, which is being reported to emphasize unusual features that may be present in patients with malignant reticuloses treated with corticosteroids and antimitotic agents.

Case Report

A 68 year old white woman was diagnosed as having acute myelogenous leukemia in October of 1966. One month later, she developed a vesicular eruption of the perianal area and buttocks. At that time she was receiving 60 mg. of prednisone daily, and two weeks later she was given 6-mercaptopurine with subsequent worsening of the eruption. Amphicillin and the use of

topical neomycin ointment failed to alter its progress.

She was admitted to the Internal Medicine Service of the University of Minnesota Hospitals in January of 1967 with multiple weeping, infected ulcerations over both buttocks and the perianal region. The ulcerations were from 1 mm. to 10 cm. in diameter and vesiculation was present at the periphery of the ulceration. The remainder of the physical examination was within normal limits except for a palpable spleen. Past medical history and review of systems were noncontributory.

The laboratory studies were within normal limits except for an ESR of 61 mm. per hour, 4+ sugar in the urine and a gammaglobulin level of 0.4 gm%.

Biopsy at the periphery of a cutaneous ulcer was interpreted as compatible with a viral bulla. Tissue culture of vesicle fluid identified the herpes simplex virus. The antibody titer to herpes simplex was 1:8 on 1/19/67 and 1/26/67.

Because of the diagnosis of herpes simplex and the patient's leukemic disease being in remission, the prednisone dose was decreased to 15 mg. per day and the 6-mercaptopurine was decreased to 50 mg. per day. The eruption improved, but on the sixth hospital day the patient developed a pneumonitis and mild respiratory distress; her condition deteriorated rapidly and she

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Fig. 1. Buttocks and perianal area.

died on 1/28/67. The autopsy revealed death due to bilateral interstitial pneumonitis.

Comment

Stroud (4) in 1961 reported a patient with primary systemic amyloidosis and recurrent herpes simplex similar to the present case. He discussed five variables which may have accounted for the course of the herpetic infection in his patient: (1) administration of systemic steroids, (2) abnormal total serum and gammaglobulin levels, (3) amyloidosis per se, (4) nephrotic syndrome, and (5) general deterioration of the patient's health. Because of the recurrence of the herpetic eruption after each increase in steroid dosage, he felt that this therapy represented the most important variable.

Park et al. (2) described unusual cutaneous viral infections in two patients receiving immunosuppressive therapy for the prevention of homograft rejection following organ transplant. One patient developed a persistent varicella infection while taking azathioprine, prednisone and cactinomycin. Another patient had hemorrhag-7-337-1088. Acta Derm. 49: 1

ic herpes simplex involving the lips during a course of azathioprine, prednisone and cactinomycin. The eruption was unusually persistent in contrast to most cutaneous infections with herpes simplex. It is not clear whether immunosuppressive therapy makes patients more susceptible to new pathogens or whether the infections represent reactivation of organisms already present.

In our patient the unique clinical picture and her unchanging herpes simplex antibody titer cannot be explained on the basis of anyone of the previously mentioned factors; multiple influencing factors are more likely, the altered immunologic mechanism in acute myelogenous leukemia undoubtedly being one. In addition, the use of corticosteroids and 6-mercaptopurine probably influenced her response to the herpetic infection. To evaluate the role of a particular factor is difficult.

With the increased use of new therapeutic agents, either alone or in combination with corticosteroids, in patients with an acquired alteration in their immunologic status, atypical cutaneous expressions of herpetic infection can be expected to occur frequently. For aid in the diagnosis of atypical infections, viral culture should be

attempted rather than depending only on clinical impressions.

SUMMARY

Factors possibly responsible for an atypical cutaneous herpes simplex infection in a patient with acute myelogenous leukemia are briefly discussed. The number and variety of atypical herpetic patterns can be expected to increase in patients whose disease and therapy may contribute to increased susceptability and lessened resistance to the herpetic viruses. Without the aid of laboratory studies, the clinician cannot be certain of his diagnosis in such cases.

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