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Solid Facial Edema as a Complication of Acne vulgaris: Treatment with Isotretinoin and Clofazimine

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Helander I, Aho HJ. Solid facial edema as a complication of acne vulgaris: treatment with isotretinoin and clofazimine. Acta Derm Venereol (Stockh) 1987; 67: 535–537.

We present two patients, a 20-year-old female and an 18-year-old male, who suffered from persistent solid facial edema as a complication of acne vulgaris. They were treated with isotretinoin with moderate response and thereafter with lymph massage with further response. The female patient also received clofazimine with good response. (Received February 27, 1987.)

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Persistent solid facial edema, as a complication of acne vulgaris is rarely seen and infrequently reported in the literature (1, 2, 3). It consists of periorbital, centrofacial, occasionally erythematous nonpitting swelling.

We present two patients, a 20-year-old female and an 18-year-old male, who were treated with isotretinoin with moderate response and thereafter with lymph massage with good response. The female patient also received clofazimine with moderate response.

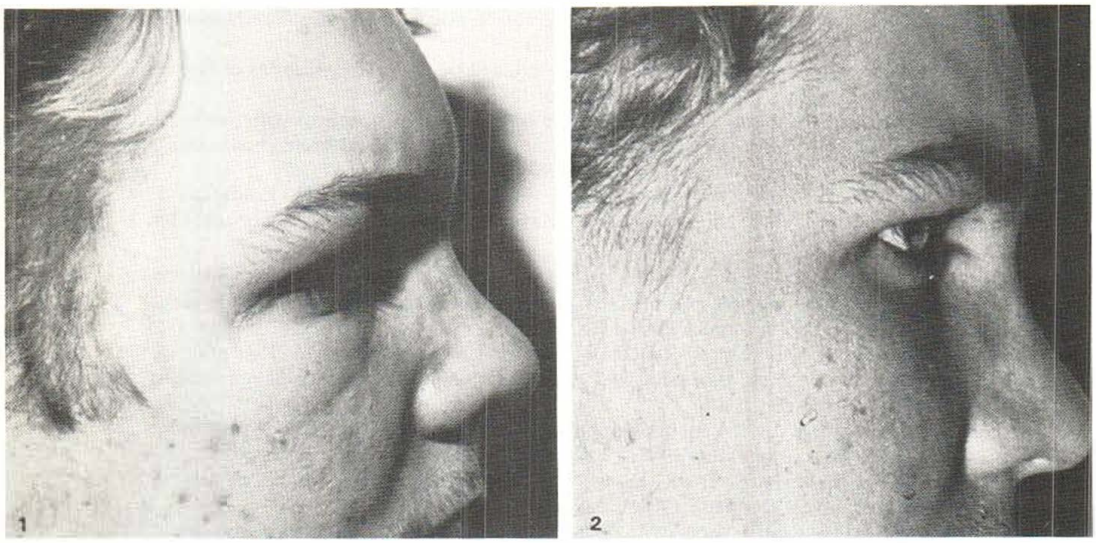

Fig. 1. Eighteen-year-old man (case 2) with solid edema of the forehead, nasal bridge, lower eyelids and the cheeks.

Fig. 2. Case 2 after the treatment. Slight edema was found only on the forehead.

CASE REPORT

Case 1

A 20-year-old female patient was referred to the Department of Dermatology in February 1984 for evaluation and treatment of a persistent swelling of the middle forehead, lower eyelids, middle areas of the cheeks and nasal bridge of four months duration. She had a 4-year history of facial acne vulgaris.

Results of the blood investigations including autoantibodies and complement studies and sinus X-ray were normal. The teeth were in excellent condition. A 4 mm biopsy specimen from the edema showed moderately dense inflammatory infiltrates composed of mononuclear and neutrophile leukocytes around blood vessels and between the collagen bundles in the deep reticular dermis.

The first clinical impression was Melkersson-Rosenthal Syndrome. The patient responded to prednisone 30 mg/day, but the edema reappeared in a couple of days after the cessation of cortisone therapy. The patient then received tetracycline 4×250 mg daily for one week followed by 250 mg twice daily for two months without any response in edema. Chloroquine phosphate was given for three months with no response. In December 1984 treatment with clofazimine (Lampren® 100 mg four times a week was begun. In March 1985 the swelling from lower eyelids had disappeared, otherwise the edema was unchanged. Clofazimine treatment was stopped. The patient was then treated with isotretinoin 30 mg daily (0.6 mg/kg). Three weeks later eyelid swelling reappeared and her acne flared up. Isotretinoin 30 mg/daily was continued and clofazimine 100 mg four times a week was added to the treatment. During the following 20 weeks the swelling disappeared from eyelids and diminished approximately 50% from the forehead, nasal bridge and cheeks. There were no acne lesions. Isotretinoin treatment was stopped and clofazimine was still continued.

There was an estimated 75% reduction in the edema of the forehead and cheeks at the control visit two months later. Clofazimine was stopped and the patient was referred to lymph massage once a week for 15 times. Edema disappeared from the forehead and diminished clearly from the nasal bridge and the cheeks. In May 1986, four months after the cessation of the lymph massage, the edema had still decreased and was just noticeable only on the forehead. The condition was unchanged at the control visit in October 1986.

Case 2

An 18-year-old male patient was referred to the Department of Dermatology in December 1985 for evaluation and treatment of persistent swelling of the forehead, nasal bridge, cheeks and lower eyelids of nine months duration (Fig. 1). He had a 4-year history of facial acne. He had been treated for acne

with numerous courses of topical (e.g. clindamycin) and systemic antibiotics (e.g. tetracycline, erythromycin) with reduction of inflammatory acne lesions, which soon after the courses flared up. Results of the blood investigations including autoantibodies and complement studies and sinus X-ray were normal. The teeth were in good condition.

The clinical impression was that of persistent solid facial edema secondary to acne. The patient was treated with isotretinoin 50 mg daily (0.8 mg/kg) for the first 12 weeks and then 40 mg daily for the following 12 weeks. There was a marked reduction in the number of inflammatory acne lesions after 12 weeks of isotretinoin. The edema of the lower lids had disappeared and approximately a 25% reduction in the edema of the forehead, nasal bridge and cheeks was noted. After 24 weeks of isotretinoin treatment acne had improved and approximately a 50% reduction in the edema was noticed. Isotretinoin treatment was discontinued and lymph massage daily for fifteen times was begun. At the control visit in October 1986 slight edema was found only on the forehead (Fig. 2).

DISCUSSION

Our two cases of centrofacial edema in acne are similar to those reported by others (1, 2, 3). The edema which developed approximately four years after the onset of acne did not respond to oral antibiotics, but responded partially to cortisone and to isotretinoin as in previously reported patients (1, 3). When acne lesions had first improved with isotretinoin, lymph massage reduced the remaining edema effectively.

Atypical Melkersson-Rosenthal syndrome was first suspected in the female patient. She was treated with clofazimine, because responses to that drug have been observed in Melkersson-Rosenthal Syndrome (4). The edema decreased moderately during the three months therapy with clofazimine and rapidly flared up after discontinuation of the drug. Clofazimine is a phenazine iminoquinone derivative. The mechanism of its action is not fully known. It is thought to be related to its ability of stimulating phagocytosis and macrophage functions (5).

The pathogenesis of solid facial edema associated with acne is unknown. Histopathologically seen deep inflammatory reaction was possible due to suppuration in the neighbourhood or to an immunological reaction. It has been speculated, that chronic cutaneous inflammation from acne can produce limited cellulitis and subsequent progressive edema of the face (1). The lack of beneficial effect with the antibiotics speaks against the bacterial etiology. The improvement of the edema with isotretinoin and clofazimine, both of which are known to stimulate macrophage functions and phagocytosis (5, 6), suggests some kind of immunological response to the mediators of acne.

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