

which could be explained effectively as being caused either by sulphone or by methaemoglobin-aemia and grave anoxaemia.

REFERENCES

1. Belmont, A.: Dapsone-induced nephrotic syndrome. *JAMA* 200: 262, 1967.
2. Browne, S. G.: Desinsitization for Dapsone dermatitis. *Br Med J* 11: 664, 1963.
3. Catalone, P. M.: Dapsone agranulocytosis. *Arch Dermatol* 104: 675, 1971.
4. Hedstrand, H.: A case of acute side-reactions to diaminodiphenyl sulphone (DDS). *Acta Dermatovener (Stockholm)* 48: 528, 1968.
5. Hubler, W. R. & Solomon, H.: Neurotoxicity of sulphones. *Arch Dermatol* 106: 598, 1972.
6. Millikan, L. E. & Harrell, E. R.: Drug reactions to the sulphones. *Arch Dermatol* 102: 220, 1970.
7. Saqueton, A. C. et al.: Dapsone and peripheral motor neuropathy. *Arch Dermatol* 100: 214, 1969.

Keratoderma Blennorrhagica in a Woman with Relapsing Polychondritis

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This 62-year-old woman who in 1967 had had an operation on a serous ovarian cystoma without signs of malignancy suffered bilateral pareses of u. abducens in August 1972. Medical examination revealed, besides the pareses, an erythrocyte sedimentation rate of 100 mm/h, which had probably been present even in 1967, and hepatomegalia. X-ray of the stomach showed deformation of the duodenal bulb, without ulceration. The patient appeared again in November 1973, with acute iridocyclitis and secondary glaucoma on both eyes and a few days after the eye symptoms had started, her right ear became swollen, red and very painful. Penicillin gave no cure. After one month the other ear was also affected. On betamethasone therapy, 3 mg/day, the condition of ears and eyes improved and she was symptom-free within 2 months. She interrupted steroid therapy one month later due to epigastric pains. This was followed by fever, 39–40°C, without other symptoms. On betamethasone 1.5 mg/day she was afebrile. In the autumn of 1974 cutaneous lesions appeared with scal-

ing crusty erosions on acral areas and on elbows and knees.

In March 1975 on admission to the dermatological ward the lesions corresponded to those of keratoderma blennorrhagica (see Figs.). Livedo reticularis and ecchymoses were present in addition. The right ear was red, scaling, enlarged and painful on palpation. Physical examination revealed the liver 5 cm below the costal arc and weakness of the right leg, with peroneal palsy. The right knee was swollen with hydrops.

Laboratory examinations. *Blood:* Haemoglobin 12.8 g/100 ml, white blood cell counts 12 100 (neutrophils 73%, eosinophils 1%, lymphocytes 25% and monocytes 1%), thrombocytes 185 000. Erythrocyte sedimentation rate 110 mm/h. *Serum:* iron 9.6 μ M; transferrin 22 μ M; creatinine 0.8 mg/100 ml; uric acid 5 mg/ml; liver tests (bilirubin and enzymes) normal; proteins: normal immunoglobulins IgG, A and M, orosomucoid 129 mg/ml, anti-nuclear antibody titre homogeneous 1/25–1/100, rheumatoid factor negative (before steroids positive); seroreactions for syphilis and gonorrhoea



Fig. 1.

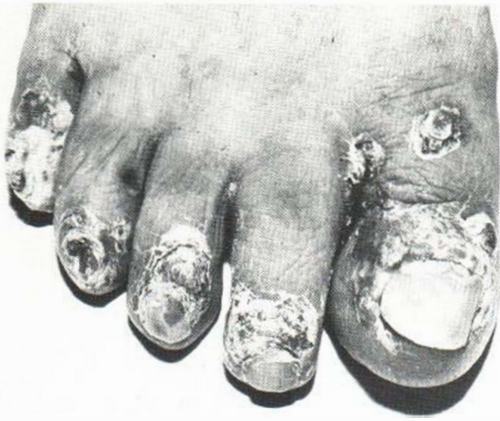


Fig. 2.

were negative. Urine analysis showed no proteins, no glucose, and a normal cell count; screening for aminoacids, α -ketoacids, phenolic acids and mucopolysaccharides gave negative results. Bacterial cultures from urine displayed anaerobic flora; from the mouth, the usual flora; from skin lesions, *Staphylococcus epidermidis*, *S. aureus*, alpha streptococci and *Candida albicans*, and from the right knee exudate, negative. Chlamydia culture from cervical secretion proved negative. Intermittently, blood was found in the stools.

Histological examinations. *Ear:* Cartilage destruction with fibrosis, no focal calcification and a sparse infiltration with plasma cells and lymphocytes. *Skin:* Epidermal hyperplasia with parakeratosis, spongiosis and exocytosis and with subcorneal pustules (Kogoj) were shown in lesions from the elbow and forearm. A confluent infiltrate was present below the epidermis, consisting of a mixture of lymphocytes, plasma cells and polymorphonuclear leukocytes. Bone marrow was normal. Lymphocytes belonged to HLA-27.

X-ray examination did not show signs of arthritis of the joints of the extremities; only an extensive spondylosis deformans was present in vertebral column, and in both knees. There were no signs of cartilaginous destruction of the nose or of trachea. The lungs appeared normal apart from a primary complex and the heart was normal in size and configuration.

Examination by an otorhinologist and ophthalmologist revealed normal findings, apart from the findings on the right ear. An ECG was normal. EMG displayed damage to the peripheral motor neuron units of both anterior tibial muscles.

Subsequent course. The skin lesions disappeared following topical treatment with a salicylic acid ointment and fluorinated steroids. The patient did well on a treatment alternate mornings with prednylidene 24 mg chosen to minimize the effects on her old gastric condition.

Comments. This case conforms well with symptoms and signs of relapsing polychondritis (1). It is the first one to present with the keratoderma blennorrhagica type of lesions. The triad of Reiter's disease was not fulfilled although she belonged to the HLA-27 group. Pearson et al. have presented a case of this disease in which pustular psoriasis also was present (2).

REFERENCES

1. Dolan, D. L., Lemmon, G. B. & Tertilbaum, S. L. Relapsing polychondritis. Analytical literature review and studies on pathogenesis. *Am J Med* 41: 285-299, 1966.
2. Pearson, C. M., Kline, H. M. & Newcomer, V. D.: Relapsing polychondritis. *N Engl J Med* 263: 51-58, 1960.