

pyloric basal lamina and submucosa causes inflammatory reaction and consecutive obliteration by granulation- and scarring tissue.

Thus, pyloric atresia can be considered a sequel to epitheliolysis of the mucosa within the Herlitz syndrome and not—as previously discussed—a random association of two different disorders or a pleiotropic effect of a single mutant gene (7, 8).

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Granulomatous Vasculitis as a Complication of Potassium Iodide Treatment for Sweet's Syndrome

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A case of Sweet's syndrome treated with potassium iodide is hereby described. The patient responded well a few days after the initiation of therapy, but the evolution was complicated with a severe clinical deterioration two weeks later. Systemic vasculitis was diagnosed on the basis of significant impairment of renal function, involvement of the eyes (papillitis) and the skin biopsy which showed a leukocytoclastic vasculitis. This systemic vasculitis was attributed to the potassium iodide therapy. *Key words: Drug therapy; Angiitis; Erythema.* (Received October 23, 1986.)

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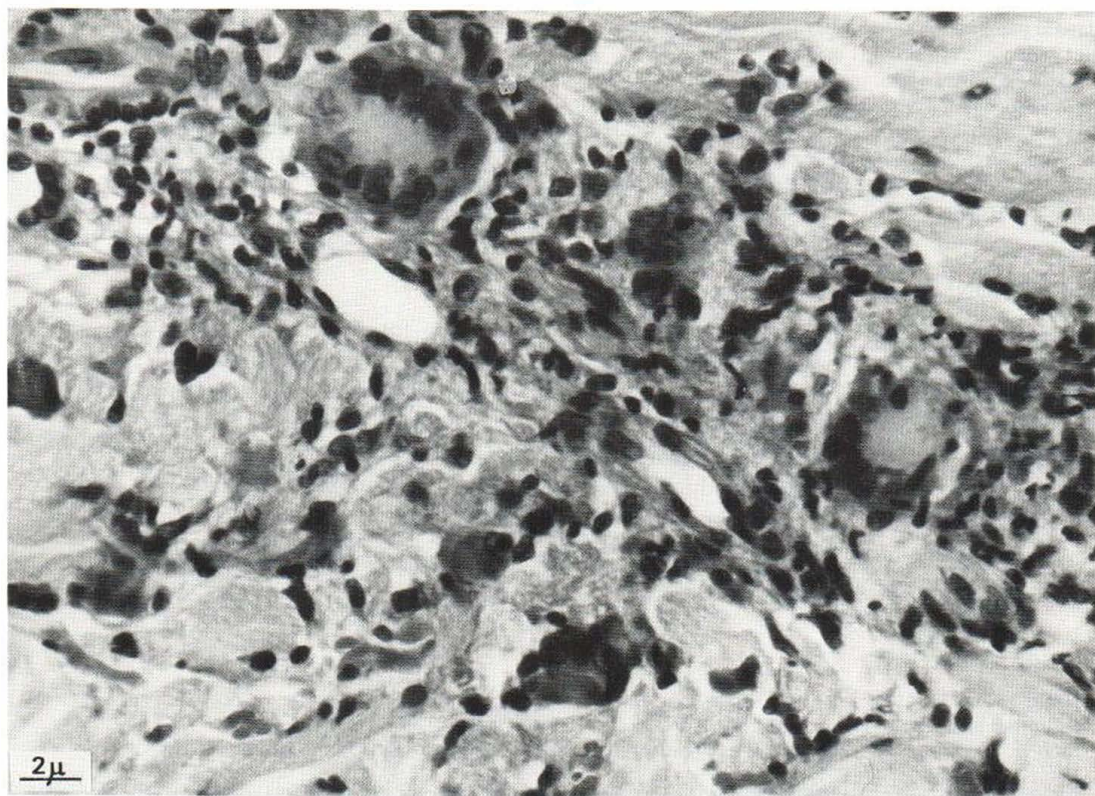


Fig. 1. Skin biopsy specimen of the left hand. Leucocytoclastic vasculitis and several giant cells of the Langhans type. (Hematoxylin-eosin stain, $\times 347$.)

Potassium iodide has been successfully used in the treatment of Sweet's syndrome and other erythematous dermatoses (1). A vasculitis-like syndrome has twice been described as a manifestation of potassium iodide sensitivity (2, 3). A patient with Sweet's syndrome, who developed a granulomatous vasculitis after potassium iodide treatment is described.

CASE REPORT

A 37-year-old female presented with fever and arthralgias. She had an unremarkable medical history, until fifteen days before this admission, when a mild headache, diffuse myalgias, a sore throat and fever appeared. A week before admission she developed diffuse arthralgias affecting the wrist, elbow, ankle and knee joints.

Physical examination disclosed a normal blood pressure (130/80 mmHg), high fever (39.6°C) and multiple, dark red, indurated patches on the left forearm, left foot and the face. Initial laboratory values were as follows: the leukocyte count was $9900/\text{mm}^3$, with 88% neutrophils, fibrinogen 912 mg/dl, erythrocyte sedimentation rate (ESR) 116 mm/h, creatinine 79.2 $\mu\text{mol/l}$, blood urea nitrogen 4.6 mmol/l.

The diagnosis of Sweet's syndrome was based on clinical, biochemical and histopathological grounds. Treatment with potassium iodide (3 \times 300 mg, daily) resulted in a rapid regression of the skin lesions. Two days later she was afebrile with ESR 68 mm/h and fibrinogen 490 mg/dl.

On the twelfth day of potassium iodide treatment, she was readmitted because of malaise, diminished vision, severe arterial hypertension (160/120 mmHg) and fever (38.3°C). A new patch had appeared on her left hand. Laboratory investigations revealed a marked renal function impairment, in comparison with initial laboratory values (creatinine 184.4 $\mu\text{mol/l}$, blood urea nitrogen 6 mmol/l, creatinine clearance 43 ml/min, proteinuria 0.9 g/24 h). Fundoscopic examination demonstrated

bilateral papillitis. A skin biopsy from the hand revealed leukocytoclastic vasculitis, however, several giant cells were observed (Fig. 1).

A kidney biopsy specimen and renal and mesenteric angiography were normal. Oral prednisolone therapy (40 mg, daily) was started and the patient became afebrile after a few days, with improvement of vision. The skin lesions resolved within one week. Corticosteroid therapy was tapered, following ESR, and eventually discontinued after 11 months. At the same time renal function gradually normalized. One year after initial treatment, creatinine clearance was 114 ml/min and ESR 13 mm/h. The patient remained well with no relapses until present.

DISCUSSION

This patient who was initially treated with potassium iodide for Sweet's syndrome, developed an iatrogenic "granulomatous vasculitis"-like syndrome, which responded well to corticosteroid therapy. A relapse of Sweet's syndrome is not likely for neither dermal giant cells, nor systemic hypertension, nor renal failure as papillitis are present in Sweet's syndrome (4). All these features have been described in cases of potassium iodide sensitivity (2, 3). Curd et al. suggested that hypocomplementemia might serve as a clue to potassium iodide sensitivity (5). In this patient complement levels were normal. This case report illustrates the unpredictable outcome with the use of potassium iodide in the therapy for erythematous dermatoses.

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Delayed Tissue Necrosis Due to Mitomycin C

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Aizawa H, Tagami H. Delayed tissue necrosis due to mitomycin C. *Acta Derm Venereol (Stockh)* 1987; 67: 364-366.

We report about a patient having received intravenous mitomycin C without evidence of tissue injury until three months following its extravasation. After drinking alcoholic beverages 3 months later he developed a severe ulcer at the site of the previous extravasation. There are only a few reports about such extravasation ulcers occurring a long time after the injection. (Received December 16, 1986.)

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Mitomycin C (MMC) is a toxic antibiotic which acts as an alkylating agent that, after being activated intracellularly by reductive metabolism, crosslinks covalently to DNA to produce altered replication and transcription (1). The frequency of its use as a chemothera-