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# Angiosarcoma: A Complication of Varicose Leg Ulceration

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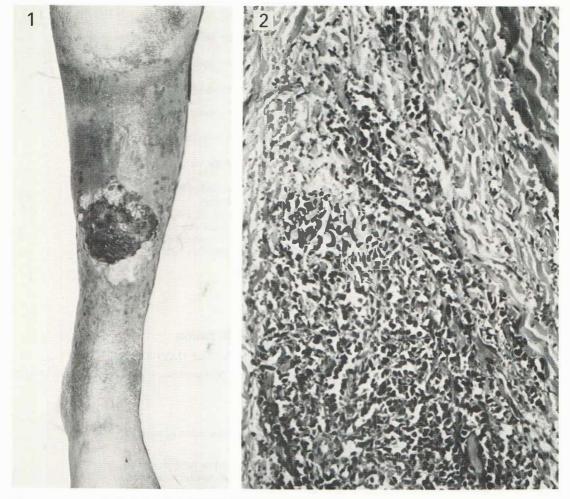
Al-Najjar A A-W, Harrington C I, Slater D N. Angiosarcoma: A complication of varicose leg ulceration. Acta Derm Venereol (Stockh) 1986; 66: 167–170.

Most reports of skin malignancy associated with varicose ulceration have described carcinomatous change (1). The development of fibrosarcoma in varicose ulcers is considerably rare but has also been described (2). We report a case of angiosarcoma developing at the site of longstanding varicose ulceration. *Key words: Malignant changes: Varicose leg ulcer.* (Received October 8, 1985.)

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### CASE REPORT

A 76-year-old man presented in July 1983, with a three-month history of a 4×4 cm painful haemorrhagic ulcer on the mid-anterior aspect of the left leg (Fig. 1). He had suffered from varicose veins with recurrent gravitational ulcers at that site for 15 years. He had bilateral hip replacements in 1972. All investigations were normal except for a high ESR (59 mm/hour Westergren). However, the ulcer rapidly increased in size, and failing to heal with conventional treatment, was biopsied. Light microscopy showed a solid network of anastomosing vascular channels lined by atypical endothelial cells displaying collagen dissection (Fig. 2). A spindle cell component was absent. Electron microscopy displayed tumour endothelial cells resting on a rudimentary basal lamina with intracytoplasmic rod-shaped tubulated Weibel-Pelade bodies in approximately 5% of the tumor cells (Fig. 3). The appearance was considered to represent angiosarcoma. Immunoperoxidase studies (DAKO Laboratory antisera) showed weak patchy positivity in the tumor cells for factor VIII related antigen *Ulex europaeus* I lectin and laminin. This was in contrast to normal stromal blood vessels which displayed complete healing of the ulcer was achieved. In February 1984, recurrences developed at the edge of



*Fig. 1.* Haemorrhagic ulcer on the mid-anterior aspect of the left leg. *Fig. 2.* Ulcer biopsy showing angiosarcoma (Haematoxylin and Eosin  $\times$  30)

the original ulcer, together with inguinal lymphadenopathy. A repeat biopsy revealed that the tumour had become less well differentiated, with more solid epithelial-like areas. Weibel-Pelade bodies were not seen ultrastructurally and immunoperoxidase studies showed negative staining for factor VIII related antigen. Ulex lectin and laminin. Again, satisfactory remission was achieved with electron beam therapy to the leg, combined with cobalt therapy to the groin. A further recurrence of the lesion in June 1984, proved resistant to radiotherapy, and necessitated an above-knee amputation. Gradual deterioration occurred during the following months and the patient died in September 1984. Autopsy showed metastatic spread confined to the liver and thoracic cavity.

# DISCUSSION

The histological appearance of the angiosarcoma in our patient is identical to the more commonly described on the face and scalp of the elderly (3) and in those complicating persistent chronic lymphoedema after mastectomy (4). However, there has been considerable debate as to the actual origin of the malignant endothelial cell in these tumours (3).



Fig. 3. Weibel-Pelade body within cytoplasm of an angiosarcoma cell (Transmission electron micrograph ×87 500).

Immunological and ultrastructural findings related to angiosarcoma of the face and scalp and postmastectomy angiosarcoma have been variable but Weibel-Pelade bodies are not generally seen and it is possible they may be derived from lymphatic vessels (5). However, Weibel-Pelad bodies have been identified in angiosarcoma of the superior vena cava (6) and central nervous system (7) and in the latter case positive staining was seen for factor VIII related antigen, lectin and laminin. In our case, the presence of occasional intracytoplasmic Weibel-Pelade bodies in the tumor cells and the weak positivity for factor VIII related antigen, Ulex lectin and laminin, supports a blood vessel rather than lymphatic origin. The occurrence of angiosarcoma on the leg is extremely uncommon but has been previously described. A common factor in all published acral cases has been the presence of severe persistent pedal oedema (3, 8) and in one case varicose veins were also present (8).

There appears to be only one other case similar to ours on record. Dawson & McIntosh described the development of a fatal granulation tissue sarcoma at the site of chronic varicose ulceration (9). The histological appearance was that of an angiosarcoma and the authors quote Van Henkelen's ability to trace gradual transformation of granulation tissue to sarcoma. Indeed, as capillaries are a major component of granulation tissue, it is perhaps surprising that the complication does not occur more commonly. The main aetiological factor in the causation of angiosarcoma in our case remains uncertain, but could include ulceration, trauma and infection. Tissue oedema was probably of minimal importance, as it was not a significant clinical feature and was absent histologically in the amputation specimen.

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## Skin Lesions as a Sign of Subacute Pentachlorophenol Intoxication

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Lambert J, Schepens P, Janssens J, Dockx P. Skin lesions as a sign of subacute pentachlorophenol intoxication. Aca Derm Venereol (Stockh) 1986; 66: 170-172.

Pentachlorophenol (PCP) and its sodium salt are frequently used in wood preservatives. Little is known about the effects on man when being chronically exposed. Only vague skin symptoms, such as rashes, acne and cutaneous infections were described. We present two cases of pemphigus vulgaris with a known non-occupational chronic PCP exposure. The clinical course and the titer of pemphigus antibodies roughly correlate with the PCP levels in serum. In one case of chronic urticaria the exacerbations also run parallel to the PCP serum levels and increased anti-skin antibodies, without any manifestation of pemphigus vulgaris. The role of PCP as one of the causes provoking pemphigus vulgaris and chronic urticaria with raised anti-skin antibodies is discussed. *Key words: Pemphigus vulgaris; Urticaria; Wood preservatives.* (Received September 3, 1985.)

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Pentachlorophenol (PCP) is a commonly used wood preservative. Its dose dependent acute toxic effects on the phosphorylating process were investigated in animals and the symptoms of acute poisoning in man after massive exposure were described (1, 2). Less is known about minor health problems in people chronically exposed to smaller doses.

Besides conjunctivitis, chronic sinusitis, upper respiratory complaints, recurring headache and neurological complaints, several skin lesions, such as vaguely defined skin irritations and rashes, a possible chloracne and a tendency for cutaneous infections were reported (3, 4).

We report two cases of pemphigus vulgaris and one of chronic urticaria where PCP is a contributing factor.

### CASE REPORT

#### Case 1

A 41-year-old Caucasian man bought a PCP treated bookcase in the summer of 1983, during which he was also highly exposed to the sun. Shortly after, bullae with a diameter of 1 to 3 cm appeared on the