

## Dendrocytoclasis in Henoch-Schönlein Purpura

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**We studied by immunohistochemistry the fate of factor XIIIa-positive dermal dendrocytes in Henoch-Schönlein purpura. We have observed apoptosis affecting most of the perivascular dendrocytes. Such an aspect of dendrocytoclasis has never previously been reported in inflammatory disorders in the absence of vasculitis.**

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Leukocytoclastic (necrotizing) vasculitis is currently considered to result from the interplay between immune complexes, polymorphonuclear neutrophils and endothelial cells (1). Recently, dendrocytes – or at least a subset thereof – have been shown to be closely related to the microvasculature (2, 3). As these cells display phagocytic functions (4, 5), and could therefore play a role in the processing of immune complexes, we were interested to study their fate in necrotizing pediatric vasculitis.

### PATIENTS AND METHOD

Skin biopsies were taken from early lesions of Henoch-Schönlein purpura occurring in children less than 13 years of age. We selected for this study 15 cases where perivascular deposits of immunoglobulins, mostly of the IgA type, and of complement were documented by immunofluorescence technique.

Controls consisted of 141 biopsies from normal skin (38), contact dermatitis (14), psoriasis (7), scleroderma (26), leprosy (9), peritumoral inflammatory reactions (47). Biopsy specimens were fixed in formalin and paraffin-embedded. Histological sections were stained with hemalum-eosin.

Other sections 5 µm thick were dewaxed in xylene, hydrated in graded alcohols and preincubated with 0.05% pronase E (protease XXV, Sigma Chemical Co., St Louis, Mo, USA). Endogenous peroxidase was blocked with 3% hydrogen peroxide in methanol for 5 min at room temperature. PAP technique was used with factor XIIIa rabbit polyclonal antibody (Behring-werke, Marburg, FRG) at a dilution of 1:300 for 30 min. The 3-amino-9-ethylcarbazole was used as chromogen.

### RESULTS

In all samples, leukocytoclastic vasculitis was present with extravasation of erythrocytes. The immunostaining with the factor XIII antibody revealed numerous dendrocytes in the inflammatory infiltrate. Many of them were plump. There were also signs of cytoplasmic fragmentation of dendrocytes, most probably originating from dendrites, as the diameter of these globules was small and as dendrites were almost unrecognizable (Fig. 1). Such dendrocytoclasis was prominent in all histological sections and was never seen in normal skin and in dermatitides without vasculitis (Fig. 2).

### DISCUSSION

Our findings reveal that dendrocytes become altered during leukocytoclastic vasculitis, but this aspect of

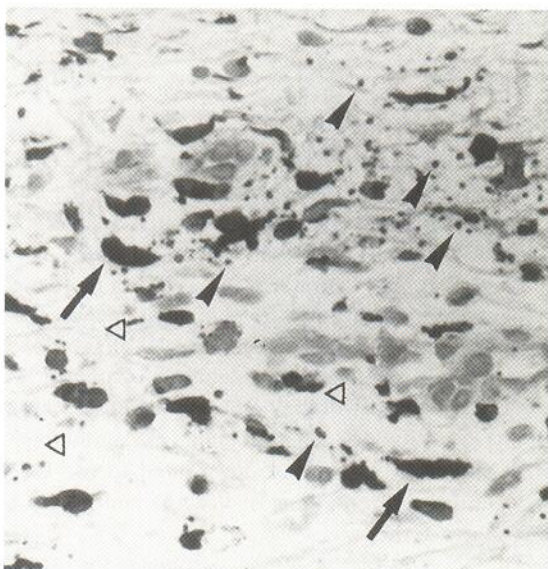


Fig. 1. Leukocytoclastic vasculitis. Some Factor XIIIa-positive dendrocytes are plump (↗). Fragmentation of the cytoplasm of dendrocytes (▲, 3-amino-9-ethylcarbazole-positive black dots) and nuclear fragments (△, hematoxylin-positive grey dots) are also present (anti-Factor XIIIa immunoperoxidase, ×400).



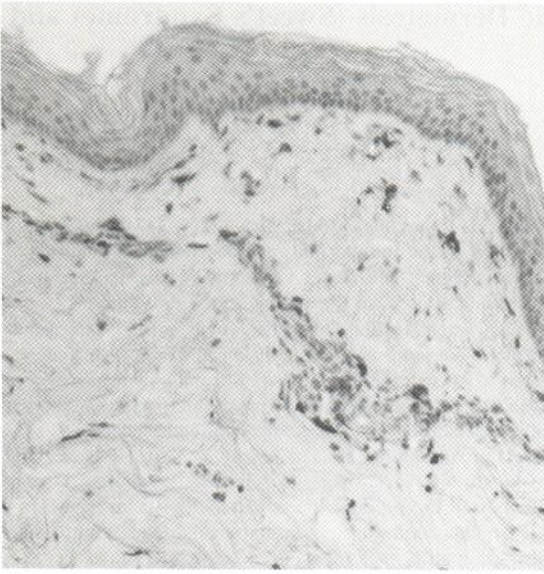


Fig. 2. Dendrocytes in normal skin do not present signs of apoptosis (anti-Factor XIIIa immunoperoxidase,  $\times 100$ ).

dendrocytosis was never seen in other inflammatory diseases (3–5) including our controls. It was observed, however, though to a lesser extent, in some PUVA-treated patients (6).

The cytoplasm (Fact- XIII-positive) fragmentation of dendrocytes is easily distinguished from the nuclear (Haematoxylin-positive) fragmentation of neutrophils. The dendrocytic alterations reminds one of a process of cell death called apoptosis (7–9). Apoptosis affects single cells and is characterized by disintegration of the cell into smaller particles, apoptotic bodies, which are then phagocytosed. They are present in physiological involution of tissues such as during the hair cycle (10). Diseases such as graft-versus-host reaction, lichen planus, fixed drug eruptions, pseudopelade, regression of warts and neoplasms, and phototoxic reactions all induce apoptosis in the epidermis (11–20). It seems that dendrocytosis is the first example of the apoptotic process occurring in dermal cells.

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