

Correspondence

Acquired reactive perforating collagenosis associated with urticarial vasculitis in pregnancy

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We read with interest the report by Healy *et al.*¹ in a recent issue of *Clinical and Experimental Dermatology* describing the first report of acquired reactive perforating collagenosis (ARPC) in pregnancy. There has only been one previous report of an association of ARPC with vasculitis.² We report another case of ARPC in pregnancy, but also in association with urticarial vasculitis.

A 31-year-old woman presented at 30 weeks into her first pregnancy with a burning skin eruption over the thighs, which had developed 1 week after a urinary tract infection. Physical examination showed urticarial weals over the thighs and lower legs, which lasted for 4 days and left residual post inflammatory hyperpigmentation. The patient was diagnosed clinically as having an urticarial vasculitis. On histological examination of a skin biopsy of the urticarial eruption, a lymphocytic vasculitis was seen, evidenced by endothelial swelling and extravasated red blood cells. (Fig. 1a). The results of a vasculitis screen were negative, and the patient was treated symptomatically with chlorpheniramine.

Three weeks later, the patient presented again, this time with a new skin eruption over the sites of resolving vasculitis. On physical examination, multiple papules with central keratotic crusts were seen at the sites of the resolving vasculitis (Fig. 1b, Fig. 2a). On histological examination of a skin biopsy taken from a dome-shaped papule, a central cup-shaped depression in the epidermis was seen, filled with a plug consisting of parakeratotic keratin, collagen, elastic fibres and inflammatory debris, characteristic of ARPC (Fig. 2b). The perforating lesions resolved after 6 weeks of treatment with clobetasol propionate 0.05% ointment (Dermovate®; GSK Pharma UK, Uxbridge, Middlesex, UK), and the patient later delivered a healthy baby girl at term.

ARPC is an uncommon disease in which altered collagen bundles and elastic fibres are eliminated through the epidermis.³ This condition is often associated with diabetes and renal disease.⁴ The underlying pathogenesis of ARPC is unknown. A number of theories have been proposed, and the most widely accepted hypothesis is that

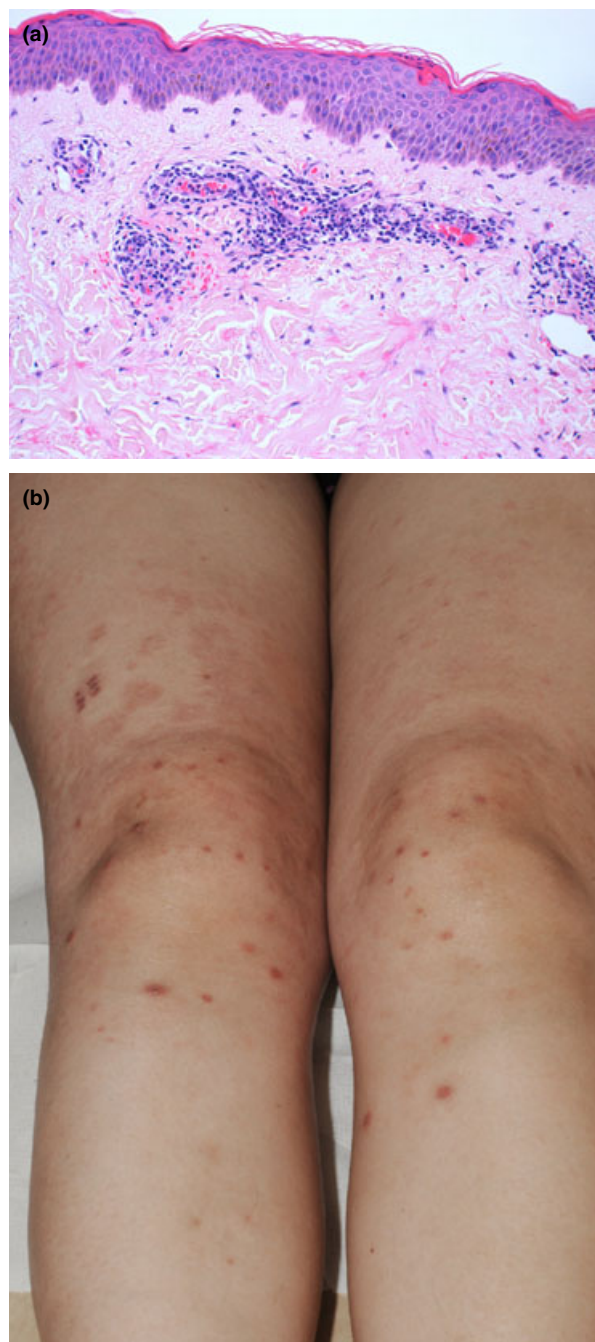


Figure 1 (a) Perivascular lymphocytic infiltrate with extravasated red blood cells and endothelial swelling in keeping with lymphocytic vasculitis (haematoxylin and eosin, original magnification $\times 100$). (b) Multiple papules and nodules developing on sites of resolving urticarial vasculitis.

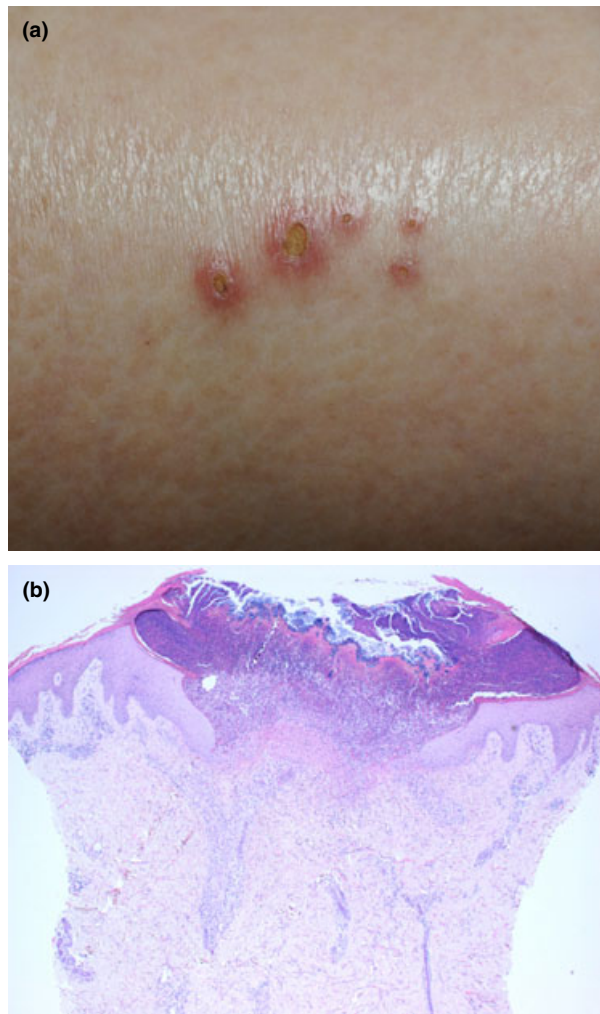


Figure 2 (a) Multiple papules with a central keratinous plug over the knee. (b) Central cup-shaped depression in the epidermis filled with a plug consisting of parakeratotic keratin, collagen, elastic fibres and inflammatory debris, in keeping with acquired reactive perforating dermatosis (haematoxylin and eosin, original magnification $\times 40$).

mild superficial trauma in genetically susceptible individuals leads to necrobiosis of dermal collagen, resulting in transepidermal elimination.³ There is only one reported case of a leukocytoclastic vasculitis occurring in association with perforating dermatosis,² and the authors speculated that the vasculitis caused release of oxidative products into the dermis, triggering altered collagen and transepidermal elimination.

In conclusion, we report a case of ARPC associated with urticarial vasculitis occurring during pregnancy, the first such case to our knowledge.

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