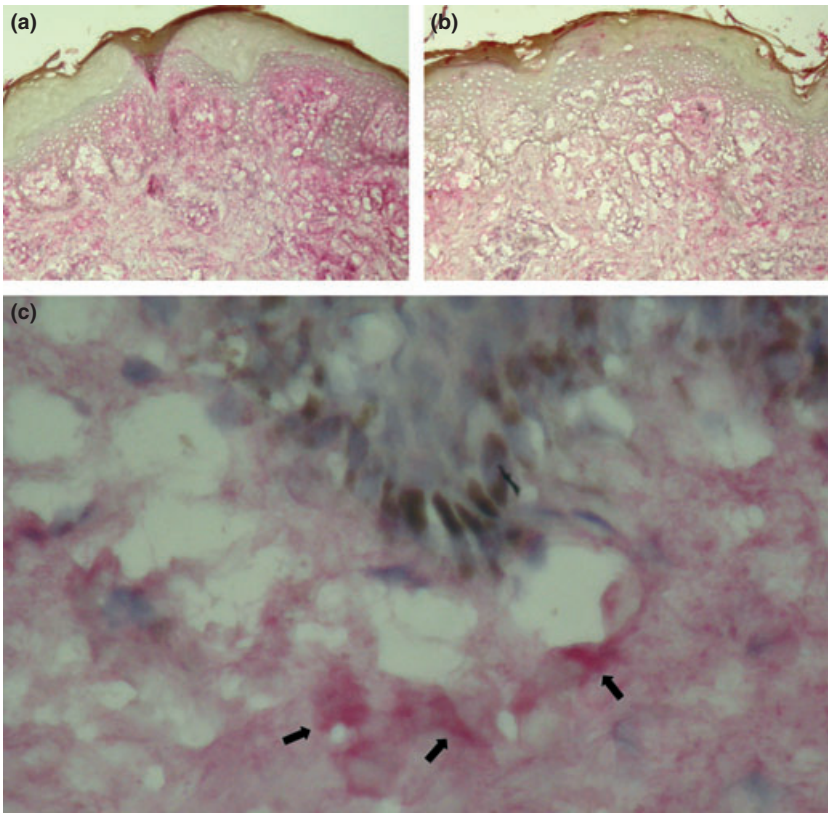

Figure 1 (a) Positive staining for Fas in the basal and suprabasal layers of the epidermis and on many cells infiltrating the dermis of a Gottron papule (original magnification × 100). (b) Several FasL-positive cells infiltrating the dermis of a Gottron papule (original magnification × 100). (c) FasL-positive cells (arrows) can be seen underneath the dermoepidermal junction in a lesional dermatomyositis section (original magnification × 200).

Perivascular/periadnexal infiltrating cells expressed Bax, Bcl-2, Fas, FasL (Fig. 1b,c) and caspase 3, and there were no significant differences between DM and CLE.

This study confirmed that the cutaneous lesions of DM and CLE have an abnormal increase in apoptotic phenomena.[3,4] Moreover, as we found that pro-apoptotic markers were also slightly overexpressed in the sun-protected healthy skin of patients with DM and CLE, we hypothesize that dysregulation of apoptosis may represent a 'constitutive' feature in DM and CLE, i.e. that it is at least partly independent of ultraviolet radiation exposure and immune inflammation. Although either the mitochondrial or death-receptor pathway may be involved in triggering apoptosis in CLE lesions[3–5] (the former putatively induced by GrB[1] among other factors), our findings also suggest that FasL-positive lymphocytes rather than Bax may play an important role in inducing death of basal keratinocytes in DM. The fact that Bax seems to have lower expression in DM than in CLE makes sense in light of the scarce production of GrB in DM.[1]

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A rare case of vulval pustulation in rosacea fulminans

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Rosacea fulminans (RF) is characterized by the sudden onset of papules, pustules and nodules, occasionally with

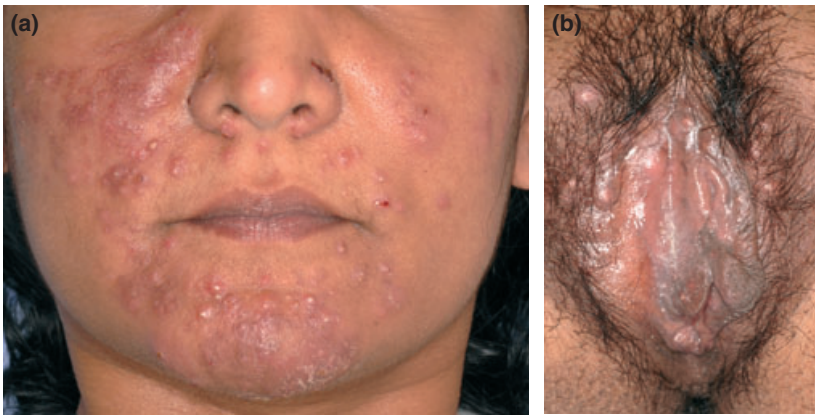

Figure 1 (a) Florid centrofacial eruption comprised of erythematous pustules, nodules and cysts coalescing to form oedematous plaques; (b) multiple tender pustules and nodules on the labia majora and minora.

sinuses and facial oedema. Extrafacial lesions are rare, but there are reports of ear, neck, chest, axillary, groin and limb involvement.[1] We report a case of RF with extensive vulval disease.

A 35-year-old woman presented to the genitourinary medicine department with a 2-month history of vulval soreness, especially after sexual intercourse. On physical examination, superficial vulval ulceration was noted and the patient was therefore treated empirically for herpes simplex virus (HSV) infection with oral aciclovir but without demonstrable benefit. Laboratory investigations including PCR testing for HSV were all normal or negative. The vulval symptoms evolved rapidly over 4 weeks to tender discharging pustular lesions, and the patient was referred to the dermatology department.

In addition to the existing symptoms, the patient had a 2-year history of a facial rash, which her general practitioner had been treating as acne. She reported a history of easy flushing. but denied any systemic or gastrointestinal symptoms. Despite numerous treatments including oral antibiotics (flucloxacillin 250 mg four times daily for 2 weeks; lymecycline 408 mg once daily for 3 months; doxycycline 100 mg twice daily for 6 months) and oral cocyprindiol (Dianette®; Schering Healthcare Ltd, West Sussex, UK) for 1 month, the facial rash had deteriorated rapidly over 6 weeks with tender discharging nodules, coinciding with the deteriorating vulval symptoms.

On physical examination, a florid centrofacial eruption was seen, comprised of erythematous pustules, nodules and cysts coalescing to form oedematous plaques (Fig. 1a). The vulva had similar lesions affecting the labia majora and minora (Fig. 1b). The patient had no fever.

Laboratory testing showed a mildly elevated white blood cell count (12.0×10^9/L; normal range $4–11 \times 10^9$/L) with neutrophilia. The erythrocyte sedimentation rate was raised (38 mm/h; 0–20 mm/h) but C-reactive protein was normal (4 mg/L). Results for angiotensin-converting enzyme, bone profile, anti-neutrophil antibody, complement, and skin swabs were all normal or negative.

Histological examination of skin biopsies from the facial and vulval lesions found an acute on chronic cell infiltrate with pseudoepitheliomatous hyperplasia and granuloma formation. A neutrophilic infiltrate was present within the epidermis associated with hair follicles (Fig. 2). Periodic-acid–Schiff, Gram and Ziehl–Neelsen stains were negative.

Oxytetracycline 1 g twice daily and a tapering course of oral prednisolone (20 mg daily reducing over 7 weeks) was

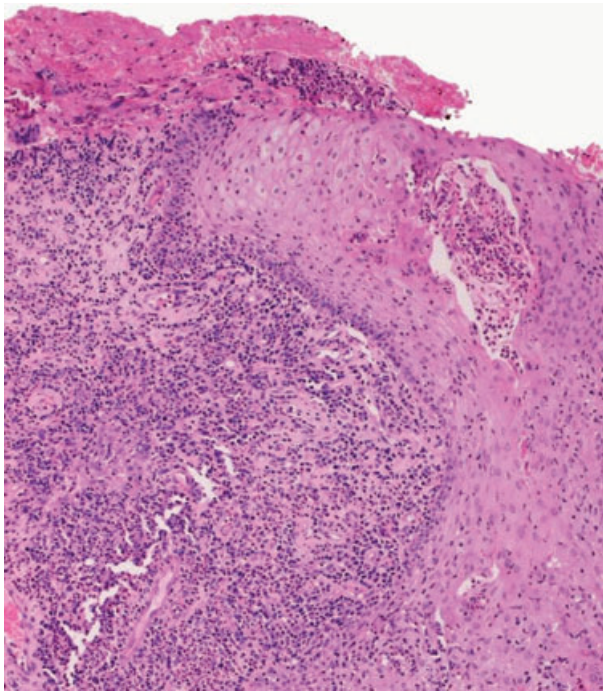

Figure 2 Acute on chronic cell infiltrate with pseudoepitheliomatous hyperplasia and prominent granuloma formation, with a neutrophilic infiltrate in the epidermis associated with hair follicles (haematoxylin and eosin, original magnification × 40).

started. Within 3 weeks, the vulval symptoms improved significantly and the patient was able to resume sexual intercourse. Complete clearance of the facial and vulval lesions was achieved after 2.5 months, and the oxytetracycline was continued at 500 mg twice daily for 6 months to prevent disease relapse.

RF predominantly affects women, usually with a history of rosacea and can be a devastating condition. Patients are usually affected well past adolescence,[2] although there are reports of young children developing the disease.[3] RF peaks a few weeks to months after onset, with regression within a year. Scarring is common, and therefore prompt and aggressive treatment is warranted. Treatments used include oral contraceptives with antiandrogens in women, antibiotics (especially tetracyclines), corticosteroids, retinoids and dapsone.[3] Relapses are rare and the overall prognosis is good.

The pathogenesis of rosacea is unclear and thought to be multifactorial.[4] Factors include vascular instability, cell-mediated and humoral immune responses, and presence of *Demodex* mites. Hormonal influences may play a role as RF mainly presents in women; indeed, RF has been reported in pregnancy[5] and with use of oral contraceptives. Although extrafacial involvement is rare in rosacea, it has been reported in up to 15% of cases, usually on the upper body.

To our knowledge, this is the first case of rosacea fulminans RF presenting with vulval involvement in an otherwise healthy woman. This case illustrates that patients with rosacea can rarely present with vulval disease to other specialties, which could delay diagnosis and definitive treatment.

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Anti-cytokeratin CAM5.2 is not a monoclonal antibody against cytokeratin 8/18

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We read with great interest the contribution by Tanese *et al.*[1] in a recent issue of *Clinical and Experimental Dermatology*. However, one aspect of this paper requires clarification, as the positioning of the brackets in the fifth paragraph might suggest that anticytokeratin CAM5.2 is a monoclonal antibody to cytokeratins 8 and 18.[1]

We would like to comment that Becton Dickinson (BD) Biosciences developed and manufactured the anticytokeratin CAM5.2 (clone CAM5.2) reagent, which reacted with human cytokeratin intermediate filament proteins of 48 kDa and 52 kDa in size, which were identified as cytokeratins 7 and 8, respectively. In 1997, BD Biosciences revised the data sheet (BD 23-3190-01) to indicate that anti-cytokeratin CAM5.2 had a primary reactivity with CK8, and a weaker but distinct reactivity with CK7. It had no reactivity with CK18 or CK19.[2–5] We therefore emphasise the crucial point that CAM5.2 is different from CK8 and CK18, and that the anticytokeratin CAM5.2 reagent is not an antibody against cytokeratin 8/18.

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