Clinico-Pathological Cases: Summaries of Papers

CPC-1
Eosinophilic panniculitis – an unusual cutaneous complication of insulin therapy
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We report an original case of eosinophilic panniculitis secondary to injection of zinc-containing insulin.

Eosinophilic panniculitis is seen in association with a wide variety of clinical conditions including arthropod bite, infection, leucocytoclastic vasculitis, malignancy and injection granuloma.

A 65-year-old woman with insulin-dependent diabetes presented with a 1-year history of a plaque on her right thigh and a shorter history of three smaller plaques on the left thigh. She only ever injected insulin into her thighs. She had thought the lesions to be abscesses but had been surprised at the absence of pus at any time. Despite repeated courses of antibiotics and dressings they had failed to heal, prompting referral.

Examination of the right thigh revealed a 10 × 4 cm indurated purplish plaque. The centre was atrophic and on palpation a significant tissue defect was detectable. The left thigh lesions were 3 cm diameter erythematous indurated plaques. All lesions were tender.

Two biopsies were obtained. The first biopsy showed areas of granulomatous inflammation in the deep dermis but was too superficial to assess the fat. The second biopsy showed a predominantly septal panniculitis with a florid eosinophilic infiltrate.

Eosinophilic panniculitis has been reported in association with injection granuloma. Zinc-containing insulin has been shown to cause injection granulomas (Jordaan HF, Sandler M. Zinc-induced granuloma – a unique complication of insulin therapy. Clin Exp Dermatol 1989; 14: 227–9). Our patient was injecting human actrapid insulin (NovoNordisk) which contains zinc chloride. This is the first reported case of eosinophilic panniculitis secondary to injection of zinc-containing insulin. We propose that the zinc component of the insulin has been the trigger for our patient’s lesions.

CPC-02
‘Animal type’ melanoma – a clinicopathological study of 14 cases
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Animal type melanomas are rare melanocytic neoplasms that display prominent pigment synthesis. The largest series in the literature comprises only six cases (Crowson AN, Magro CM, Mihm MC Jr. Malignant melanoma with prominent pigment synthesis: ‘animal type’ melanoma – a clinic and histological study of six cases with a consideration of other melanocytic neoplasms with prominent pigment synthesis. Hum Pathol 1999; 30: 543–50). In part because of their rarity their clinical behaviour is unpredictable. However, the histology closely resembles equine melanomas that classically develop in old, grey horses. In this group the tumours are indolent but metastases are not uncommon (Valentine BA. Equine melanocytic tumors: a retrospective study of 53 horses (1988 to 91). J Vet Intern Med 1995; 9: 291–7).

Over 10 years 14 cases from the consultation files were encountered. There were six females and eight males with an age range of 5–65 years. Lesions were located on the face, trunk and lower limbs and were described as blue-black nodules. The pathology was similar in all cases and comprised a predominantly intradermal, deeply pigmented melanocytic proliferation with confluent nests of spindle-shaped melanocytes containing large hyperchromatic nuclei. Numerous melanophages were seen and mitoses were infrequent. Perineural and intravascular invasion and regression were not apparent. Tumour-infiltrating lymphocytes were absent. One case was a melanoma in situ, but most lesions were dermal nodules. The Breslow thickness ranged from 0.84 to 7.5 mm.

To date, four cases are known to have metastasized to the regional lymph nodes and one has locally recurred.

Animal type melanoma is a distinctive clinicopathological variant of melanoma capable of aggressive behaviour. Whether it behaves in the same manner as a melanoma of similar Breslow thickness has yet to be determined.

CPC-03
Intraoral mycosis fungoides in a 9-year-old boy
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Intraoral lesions of mycosis fungoides (MF) are rare and occur in < 1% of patients with MF. Thirty-four cases have been reported in the literature and are usually associated with aggressive disease and a poor prognosis (the majority of patients dying within 3 years). Subclinical oral cavity involvement is more common, with autopsy reports of an incidence of up to 11%. The youngest reported patient with intraoral disease is 36 years old – it has not previously been reported in a child. Juvenile-onset cutaneous MF is also
uncommon. Four per cent of MF patients have symptoms at <20 years of age but only half of these are diagnosed as having MF before they are aged 20 years. Onset in infancy is rare. We report a unique case of cutaneous MF arising in infancy in association with intraoral MF occurring at 9 years of age. In contrast with the aggressive nature of MF seen in patients with intraoral disease, our patient has now had MF for 11 years and intraoral disease for 3 years, with no evidence of cutaneous or systemic disease progression.

At 6 months of age our patient developed a poikilodermaous rash over his upper inner arms, upper inner thighs and buttocks which has been slowly progressive with frequent flares of psoriasiform lesions on the trunk, arms and legs. As his cutaneous disease has progressed, he has also developed subungual lesions and nail dystrophy. Ultraviolet B treatment caused a disease flare with development of pityriasis lichenoides chronica type lesions. At 9 years of age, he developed an asymptomatic thickened spongy lesion on the right soft palate. Over the next 3 years this increased in size and further lesions developed on his upper right lip and the left lateral border of his tongue. He is currently being offered intraoral radiotherapy.

Biopsies taken from a poikilodermatous area on his right thigh and the infiltrated area on his upper right lip show similar features in keeping with a diagnosis of lichenoid MF. There is a band-like infiltrate in the dermis with apoptotic cells and lymphocytes containing enlarged, hyperchromatic nuclei. The epidermis is acanthotic with hydropic degeneration and Pautrier microabscess formation. Immunohistochemistry demonstrated a CD2+, CD3+, CD4+, CD8−, CD30− phenotype. T-cell receptor gene rearrangement (using polymerase chain reaction/single-strand conformational polymorphism analysis) showed identical T-cell clones in the skin and oral mucosa. Blood was polyclonal. Full blood count and lactate dehydrogenase were normal.

This is the first reported case of intraoral MF in a child.

CPC-04
Isolated cutaneous pseudoinflammatory tumour (plasma cell granuloma)

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We report a 33-year-old man who presented with a 6-month history of a lesion on the right shoulder. On examination, there was a large, firm, indurated reddish plaque with a granulomatous appearance. Full blood count, serum angiotensin converting enzyme levels and chest X-ray were normal. An incisional biopsy revealed a dense lymphoid and mixed inflammatory cell infiltrate extending from the epidermis to the subcutis, splaying of collagen fibrils (suggesting mucin deposition) and a normal epidermis. Immunofluorescence was negative. A reactive lymphoid infiltrate of uncertain origin was diagnosed. Over the next 2 months the lesion grew in size and became increasingly indurated and darker in colour. Erythrocyte sedimentation rate, C-reactive protein, bone marrow aspirate and computed tomographic scan of the chest and abdomen were normal. Further biopsy (including T-cell marker studies) was again consistent with a benign lymphoid hyperplasia. A trial of clobetasol propionate (Dermovate®) ointment under occlusion resulted in an initial reduction in the size and induration of the lesion. A short course of prednisolone 30 mg daily, reducing to zero over 4 weeks, led to further moderate reduction in size. A further biopsy of the lesion revealed a broadly similar process of an inflammatory infiltrate of lymphocytes, plasma cells and occasional eosinophils extending to the subcutaneous fat but with a marked increase in fibrosis. A diagnosis of pseudoinflammatory tumour was made. The patient was unwilling to undergo surgery or have treatment with radiotherapy but remained under close follow-up. The lesion continued to involute without further treatment and only a scar remains 36 months after presentation.

Pseudoinflammatory tumours are a poorly defined group of tumours, with indeterminate malignant potential, which can occur at almost any site of the body. Isolated cutaneous pseudoinflammatory tumours have very rarely been reported. We discuss and review the literature regarding pseudoinflammatory tumours.

CPC-05
Factitious panniculitis masquerading as pyoderma gangrenosum

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Factitious disease of the skin can produce bizarre lesions whose appearance often depends on the initiating insult. We report an unusual case of extensive, widespread ulceration with the typical clinical appearance of pyoderma gangrenosum and the histological features of a panniculitis.

A 35-year-old woman, recovering from an episode of alcohol-induced acute liver failure, presented with extensive, tender, indurated, painful ecchymotic plaques over the lower trunk and legs. Histological examination of several biopsies showed an active lobular panniculitis superimposed upon chronic inflammation with ghost adipocytes surrounded by foamy macrophages. There was no evidence of vasculitis, lymphoma or other malignancy. There was a widespread neutrophilic infiltrate with thickened blood vessel walls and focal stippled calcification of fat due to postinflammatory calcification in areas of necrosis. Laboratory investigations did not reveal any underlying cause for the panniculitis.

Her skin lesions became more extensive and subsequently ulcerated in a pattern persuasive for pyoderma gangrenosum. Treatment with prednisolone 1 mg kg⁻¹ was commenced,
and dapsone 100 mg daily was later introduced. In combination with prednisolone, other treatments over a period exceeding 4 months included topical clobetasol propionate and tacrolimus 0.1% ointment, ciclosporin and mycophenolate motetil, none of which effected an improvement.

Gradually, overt signs of clinical depression emerged and the possibility of a factitious element to her cutaneous lesions was suspected. Immunosuppressive therapy was tailed off and antidepressant therapy was initiated. The ulcerated areas were encased in fibreglass casts for several days at a time. Within a short period, signs of healing appeared, and the ulceration healed completely over the next 12 months with pronounced scarring.

In retrospect, we consider the diagnosis to have been factitious panniculitis (Winkelman RK, Barker SM. Factitial traumatic panniculitis. J Am Acad Dermatol 1985; 13: 988–94), as confirmed by histopathology, although the mechanism of injury is unclear. The artefactual element was initially concealed by the widespread involvement and the clinical impression of pyoderma gangrenosum, which is commonly misdiagnosed as a cause of ulceration (Weeling RH, Davis MDP, Dahl PR, Su WPD. Skin ulcers misdiagnosed as pyoderma gangrenosum. N Engl J Med 2002; 347: 1412–18).

CPC-06
Bullous subacute cutaneous lupus erythematosus with toxic epidermal necrolysis-like expression
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A 59-year-old woman with disseminated uterine leiomyosarcoma developed a blistering skin eruption in a photoaggravated distribution, following a sunny holiday. There were areas of skin that were denuding in sheets. Her palms, soles and mucous membranes were unaffected. She had received chemotherapy with doxorubicin and radiotherapy a year prior to this. A few months after her oncological diagnosis she experienced what was thought to be erythema multiforme (EM), both clinically and histologically. This rash appeared soon after the chemo- and radiotherapy and worsened with each dose of doxorubicin. It eventually responded to topical and systemic corticosteroids. Her extensive medical history included Graves’ disease, for which she had had a total thyroidectomy, pernicious anaemia, alopecia areata and surgically treated breast cancer. She remained clear of breast cancer.

Review of the initial skin biopsy showed features consistent with subacute cutaneous lupus erythematosus (SCLE). Histology from the new rash correlated with a clinical diagnosis of toxic epidermal necrolysis (TEN). Direct and indirect immunofluorescence were negative. She was positive for the anti-Ro/SS-A antibody only. A diagnosis of bullous SCLE was made; we propose that this is the first case of bullous SCLE that has so far been reported.

We propose that this woman’s already activated autoimmune state was further compromised by disseminated carcinoma. Her initial rash, thought to be EM and attributed to chemotherapy, was almost certainly SCLE. The bullous lesions she developed after subsequent ultraviolet exposure and the negative screens for paraneoplastic pemphigus lends support to a diagnosis of bullous SCLE. The presence of the anti-Ro/SS-A antibody leads to increased photosensitive autoreactivity. The TEN-promoter polymorphism 308A that has been linked to TEN is a very important photosensitive regulator in SCLE. There is a higher linkage disequilibrium in people with SCLE and also in those who are positive for HLA-DR3 locus and anti-Ro/SS-A antibody. It is quite possible that this is the common path in developing TEN-like clinical expression in a person with SCLE.

CPC-07
Bullous systemic lupus erythematosus: a shared entity
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Bullous lupus erythematosus (LE) is a form of systemic LE (SLE), with distinct clinical and histological features. It is unusual in that it shares some of the clinical and immunohistological features of dermatitis herpetiformis (DH), bullous pemphigoid (BP) and epidermolysis bullosa acquisita (EBA). A 20-year-old female student presented with a 3-month history of an episodic rash over the palms and upper limbs associated with fatigue and arthralgia. On examination, she was pale with sparse hair, and had a florid papulovesicular eruption over the arms and legs. Investigations revealed anaemia (haemoglobin 9.5 g dL$^{-1}$, erythrocyte sedimentation rate 118 mm in the first hour), with positive antinuclear antibody (1:320) and low C4. Skin histology showed small subepidermal bullae, and direct immunofluorescence confirmed linear IgG, C3 and IgM at the basement membrane. After responding poorly to prednisolone 30 mg daily and hydroxychloroquine 200 mg daily, she responded dramatically to dapsone 50 mg daily, in a similar fashion to those with DH. Her itch and skin lesions settled within days of starting dapsone.

This case demonstrates the overlapping features of DH, BP and EBA which are seen in this rare condition. We discuss the overlapping features of bullous SLE and its relationship with the other primary bullous disorders.

Patients present with widespread nonscarring vesicular lesions on flexural or extensor skin. Blisters may be small and grouped, resembling DH, or large and tense, resembling BP. Pathology shows subepidermal vesicles often containing neutrophil abscesses in the papillary tips, similar to those seen in DH, but immunofluorescence shows linear IgG, IgA or IgM ± C3 in the basement membrane, resembling BP. Interestingly, bullous LE shares the same target antigen as EBA, i.e. type VII collagen. Finally, an important feature of bullous
LE is its DH-like dramatic response to dapsone with cessation of new lesions within 24–48 h.

CPC-08
A novel case of primary metaplastic carcinoma of the skin
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An 85-year-old man presented to the Dermatology Clinic with an ulcerated lesion on his left ear. The lesion had been present for 6 months and was constantly bleeding. The patient had a history of multiple squamous cell and basal cell carcinomas in the scalp, treated over many years. In 2001 he had found to have a mucinous adenoma of the ascending colon and a moderately differentiated oesophageal adenocarcinoma for which he had not received any active treatment. Physical examination revealed a 5 × 3 cm ulcerated lesion on his left ear, which clinically was thought to be squamous cell carcinoma. There were no palpable regional lymph nodes.

Punch biopsy of the skin showed a poorly differentiated squamous cell carcinoma with sarcomatoid areas. Immunohistochemical staining was positive for vimentin but was negative for broad-range cytokeratin. Approximately 50% of the tumour cell nuclei were immunoreactive for Ki-67 and almost all of the tumour cell nuclei were positive for p53. The spindle-cell component was strongly immunoreactive for smooth-muscle alpha-actin, indicating leiomyosarcomatous differentiation.

Metaplastic carcinoma is a biphasic tumour comprising malignant epithelial and heterologous mesenchymal elements. It is a relatively uncommon tumour and has been described in a variety of organs, including female reproductive organs, respiratory and gastrointestinal tracts. It is extremely rare as a primary tumour in the skin, with only 17 cases documented in the literature to date. Cutaneous metaplastic carcinoma, from the limited information available, affects a wide age range although, not surprisingly, those arising in actinally damaged sites predominantly affect the elderly, presenting on the face and scalp. In general, these neoplasms show rapid growth. In contrast to metaplastic carcinomas arising in visceral sites, those primarily arising in the skin do not appear to behave in a very aggressive manner.

In the reported cases osteosarcoma is the most common mesenchymal component of metaplastic carcinoma (Rios-Martin JJ, Parra-Martin JA, Gomez-Pascual A et al. Sarcomatoid carcinoma of the skin: report of a case. J Dermatol 1998; 25: 314–21; Patel NK, McKee PH, Smith NP, Fletcher CD. Primary metaplastic carcinoma (carcinosarcoma) of the skin. A clinicopathologic study of four cases and review of the literature. Am J Dermatopathol 1997; 19: 363–72). Our case is the first in which the mesenchymal component is a leiomyosarcoma.

CPC-09
Systemic sarcoidosis presenting as a granulomatous reaction in a heavily tattooed man after treatment with ribavirin and interferon alfa for chronic hepatitis C
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We report a heavily tattooed patient who presented with numerous nodular, ulcerated lesions distributed throughout his tattoos. There was no predilection for a specific colour as the nodules involved the red, black, green and yellow pigment. There was slight overlap on to normal skin. The lesions had started to develop, within the confines of his tattoos, 4 months into therapy with ribavirin and interferon alfa for chronic hepatitis C virus (HCV) infection. He had contracted HCV as a result of one of his many visits to the tattoo parlour. He had had his last tattoo over 20 years previously and all the pigment was heavy metal-based. He had finished his therapy 2 weeks prior to his dermatology appointment, but had continued to get new lesions.

Histology of the lesions showed a granulomatous reaction and confirmed the clinical diagnosis of cutaneous sarcoidosis. Special stains for fungi, atypical mycobacteria and acid-fast bacilli were negative. Chest X-ray revealed bilateral hilar lymphadenopathy and peripheral parenchymal opacities in keeping with pulmonary sarcoidosis. His serum angiotensin converting enzyme level was three times the upper limit of normal.

Two months after his therapy had stopped he was negative for HCV as tested by HCV RNA polymerase chain reaction. Although treated with potent topical corticosteroids he had noticed a less aggressive nature to the cutaneous lesions 1 month after stopping therapy.

Sarcoidosis is a multisystem granulomatous disease of unknown aetiology. It is recognized that cutaneous sarcoidosis has a predilection for scars and sites of previous trauma. A handful of cases report granulomatous reactions being confined to red, black or green pigment in tattoos, but there has been no case exhibiting reactions in all these pigments. HCV and interferon alfa have been implicated in the manipulation of the immune response and have been associated with a variety of autoimmune conditions. There have been two reported cases of systemic sarcoidosis precipitated by interferon therapy. Alterations in the immune system involving T-helper (Th) 1 cells have also been implicated in the development of sarcoidosis, and it is most likely that the ribavirin and interferon together helped modulate an immune response granting expression of a Th1-mediated granulomatous reaction.
Autoimmune blistering disorders are a heterogeneous group, but typical cases can usually be distinguished with relative ease on combined clinical, histopathological and immunopathological features. However, rarely, there can be considerable overlap, particularly in the clinical presentation of dermatitis herpetiformis (DH) and bullous pemphigoid (BP).

We report a constellation of three cases in which there was overlap between these two diseases. Three patients (two men and one woman) aged 83 and 84 years had immunobullous disease in which there were features compatible with the diagnoses of DH and BP. In each of the three cases the initial clinical diagnosis was DH manifested as an intensely pruritic, papulovesicular eruption most pronounced over the extensor surfaces of the scalp, shoulders, back and buttocks. This diagnosis was confirmed histologically with typical granular IgA deposits. All three patients were initially adequately controlled on dapsone and a gluten-free diet. This disease pattern persisted for 4 months, 1 and 11 years, respectively, after which time the clinical features and response to treatment changed with development of larger bullae. Subsequent reinvestigation confirmed immunopathological features that were compatible with the diagnosis of BP and DH. Two of the three patients required a combination of oral corticosteroid and dapsone in order to achieve an acceptable level of disease control, while the third could be managed on oral corticosteroid alone. It has been suggested that one autoimmune bullous disease can transform into another as a consequence of epitope spreading (Ameen M, Bhogal BS, Black MM. Dermatitis herpetiformis evoking (Ameen M, Bhogal BS, Black MM. Dermatitis herpetiformis evolving into bullous pemphigoid: a probable example of ‘epitope’ spreading. Clin Exp Dermatol 2000; 25: 398–400). This phenomenon describes the way in which targets of immune responses in autoimmunity may alter. Where there is a mixed immunofluorescence pattern it may be that one or other of two apparently coexistent diseases is the active disorder, while residual antibody deposition from a former precipitating immunobullous disease has persisted. A further consideration is the manner in which these overlap or transformation syndromes respond to conventional treatment, suggesting which is the dominant immunopathology. These cases emphasize the necessity for repeat biopsy for routine histology and immunofluorescence studies where a change in morphology of cutaneous lesions has occurred, or in response to treatment.

We report a boy with granulomatous skin nodules in cartilage-hair hypoplasia (CHH) syndrome. Dysmorphic features and short stature were noted at birth and a presumptive diagnosis of hypochondroplasia was made. He had a series of infectious illnesses and failed to thrive. An underlying immunodeficiency was identified with lymphopenia and abnormal T-cell composition. At 1 year of age he developed a purplish nodule at a vaccination site on the thigh. He subsequently developed similar lesions at other sites. The histology of these skin lesions was granulomatous. He was treated as for atypical mycobacteria without improvement. He developed anterior uveitis which has been difficult to treat. His skin lesions have continued to increase and are disfiguring. Therapeutic interventions including ciclosporin, methotrexate, systemic steroids and intravenous immunoglobulin have had limited success.

Skin biopsy showed a dense multinodular infiltrate extending from the superficial dermis into the subcutaneous fat consisting predominantly of T-lymphoid cells. There was an ill-defined granulomatous element. Radiological features of an unusual metaphyseal dysplasia were in keeping with CHH syndrome. Recent genetic investigations have revealed a unique mutation of the RMRP gene on chromosome 9p13-p12. Mutations in this gene are associated with CHH (Ridanpaa M, Sistonen P, Rockas S. Worldwide mutation spectrum in cartilage-hair hypoplasia: ancient founder origin of the major 70A-G mutation of the untranslated RMRP. Eur J Hum Genet 2002; 10: 439–47).

Granulomatous skin reactions are seen in a number of immunodeficiency conditions but this striking cutaneous picture is unusual. CHH syndrome is a rare recessive condition most prevalent in the Old Order Amish and the Finns (Makitie O, Sulisalo T, de la Chapelle A, Ka Kitla I. Cartilage-hair hypoplasia. J Med Genet 1995; 32: 39–43). It manifests clinically as chondrodysplasia, hypoplasia of the hair and impaired T-cell mediated immunity. Granulomatous skin lesions and uveitis are not previously described in this condition. The mutations this boy carries are novel and may account for his unusual clinical picture.
CPC-12
Bullous graft-versus-host disease in a patient with acute myeloid leukaemia
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Acute graft-versus-host disease (GVHD) is a serious complication of bone-marrow transplantation with significant morbidity and mortality. It occurs in 20–30% of allogeneic bone-marrow graft recipients despite immunosuppressive therapy. GVHD primarily involves the skin, gastrointestinal tract and liver. Cutaneous disease tends to precede systemic involvement. Skin manifestations of acute GVHD include initial pruritus, pain on pressure and erythema, initially on the palms and soles, with evolution into a nonspecific maculopapular rash. Follicular erythema, erosive oral lesions and toxic epidermal necrolysis may also occur. Bullous GVHD, however, is extremely rare.

We report a 56-year-old man with acute myeloid leukaemia who developed a morbilliform rash on his trunk, arms and legs, and a blistering eruption 28 days after a mismatched unrelated donor bone marrow transplant. He presented with a 2-day history of tender, tense bullae on his hands, feet, arms, legs, abdomen and scalp. The bullae were at the same stage of development throughout and many had an erythematous base. The skin of the feet was exquisitely tender. There were no oral lesions.

Skin biopsy revealed a moderate upper dermal perivascular infiltrate with extensive exocytosis and some satellite necrosis. There was a subepidermal blister containing fibrin and with degenerate keratinocytes at the base. The appearances were fully consistent with bullous GVHD. Direct immunofluorescence showed a bright linear basement membrane zone staining with IgG and C3. Staining was to the base of the blister. Immunofluorescence findings were considered to be reactive and not pathological.

The patient was treated with methylprednisolone and made a rapid recovery. Early diagnosis of acute GVHD and subsequent timely therapeutic intervention are important to prevent progression to severe disease with potentially fatal outcome. Steroids have remained the standard treatment for acute GVHD and appear to be optimal therapy for bullous GVHD.

CPC-13
Leucocytoclastic vasculitis associated with cutaneous herpes simplex infection presenting as a disseminated haemorrhagic bullous eruption
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Herpes simplex infection commonly presents as a cluster of self-limiting small vesicles that heal without scarring. We observed an unusual case of herpes simplex infection associated with leucocytoclastic vasculitis (LCV) and a disseminated haemorrhagic bullous eruption that healed with scarring.

A 65-year-old caucasian woman presented with a 2-day history of widespread infiltrated vesiculopapular lesions involving the lips, face, trunk and limbs associated with episcleritis and periorbital oedema. This rapidly evolved into disseminated crusted, fungating lesions on the face and an extensive haemorrhagic bullous eruption on the limbs. This was preceded by an influenza-like illness, herpes labialis and pleural effusion. Medical history included end-stage renal failure of unknown origin on haemodialysis. Herpes simplex virus type 1 was isolated from culture and immunofluorescence tests of aspirated fluid from the legs. Investigations revealed positive antinuclear antibodies of 1 : 40, antimitochondrial antibodies 26 U mL−1 (normal 1–10), both of which were normal 6 months prior to this, and elevated rheumatoid factor. Histopathology showed diffuse dermal necrosis and extensive neutrophil infiltration with fibrinoid necrosis of the small vessels, consistent with LCV. Subsequent computed tomographic scan of the chest revealed widespread ground glass opacification probably related to vasculitis.

Treatment with intravenous aciclovir, a single dose of intravenous immunoglobulin (10 g) and flucloxacillin for secondary infection led to resolution of the skin eruption which healed with hyperpigmented scarring over a period of 6 weeks.

Various herpesviruses, e.g. varicella-zoster, herpes simplex virus, cytomegalovirus and human herpesvirus-6, have been associated with LCV. It is, however, an uncommon complication of herpes simplex virus infection (Cohen C, Trapuckd D. Leucocytoclastic vasculitis associated with cutaneous infection by herpesvirus. Am J Dermatopathol 1984; 6: 561–5). Although the histology of our patient did not show multinucleated cells or nuclear inclusions, the resolution of inflammatory markers with clearing of the skin eruption following therapy with aciclovir suggested that the vasculitis was caused by a herpesvirus. The extent of the lesions was probably related to impaired immunity in this patient. The haemorrhagic appearance of the lesions was thought to be secondary to a predominant vasculitic component.