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CPD

Dietary deprivation during the COVID-19 pandemic producing acquired vulval zinc-deficiency dermatitis

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A 51-year-old woman of Chinese origin with no relevant medical history presented to the outpatient dermatology department with a severe dermatitis of the vulva, perineum and groin. This was initially thought to be irritant, with superimposed candidal infection. Contact dermatitis was also considered, as the patient had been using topical haemorrhoid treatments. Topical corticosteroid/antifungal treatment was instituted, resulting in partial improvement, but the condition then progressively and rapidly deteriorated. Mycology swabs were negative. A single bacteriology swab grew *Pseudomonas aeruginosa*, presumed to represent colonization. Escalation of topical corticosteroid and oral antimicrobials failed to produce improvement. The patient returned in extreme pain, prompting her admission for management.

Physical examination revealed gross oedema and an erythematous, eroded dermatosis (Fig. 1) over the whole perineum. The medial thighs and interlabial sulci demonstrated hyperkeratosis and desquamation. The periphery of the dermatosis developed a dusky, purpuric appearance with satellite lesions. There was no perioral or acral involvement, but the patient had received recent ophthalmology review for conjunctival erosions and madarosis. She was systemically well and denied weight loss or diarrhoea. A collateral history from the patient's husband revealed that their Chinese takeaway business had closed 6 months previously due to the COVID-19 pandemic. Under financial pressure, they had resorted to a restricted diet comprising processed, refined foods. The patient had experienced constipation and developed prolapsed haemorrhoids, then subsequently narrowed her intake further with the aim of minimizing discomfort on defecating.

A trace element panel revealed that the patient's serum zinc level was low (6.9 μmol/L; normal levels 11.0–19.0 μmol/L), and this, in the context of normal inflammatory markers, led to a diagnosis of acquired zinc-deficiency dermatitis (ZDD). Copper level was also low (11.6 μmol/L; normal levels 12.0–25.0 μmol/L), selenium was borderline low (0.97 μmol/L; normal levels 0.9–1.67 μmol/L) and vitamin D was deficient. Renal and liver profiles, full blood count and blood glucose levels were normal.

Intravenous replacement of trace elements, including zinc, was commenced on the advice of biochemistry colleagues. A biopsy of the outer, purpuric region of dermatitis showed changes consistent with late-stage ZDD, including cytoplasmic vacuolation and necrosis, resulting in intraepidermal vesicles and subcorneal neutrophilic pustules (Fig. 2). Once treatment was started, the dermatitis improved dramatically within 48 h of admission.

Malabsorption screens were negative and dietary restriction was deemed to be responsible. The patient received dietetics advice regarding foods rich in trace elements, taking into account her financial limitations. She was discharged on oral vitamin/mineral supplementation. Follow-up 2 weeks later revealed complete resolution of active dermatitis, with evidence only of postinflammatory dyspigmentation. Her ophthalmological issues were also noted to be improving. Repeat zinc level was normal at 17.4 μmol/L.

Zinc, which concentrates in the epidermis, is a vital nutrient for skin health. It supports epithelial structure and is important for wound healing, cell survival and inflammation prevention.[1] It follows that deficiency

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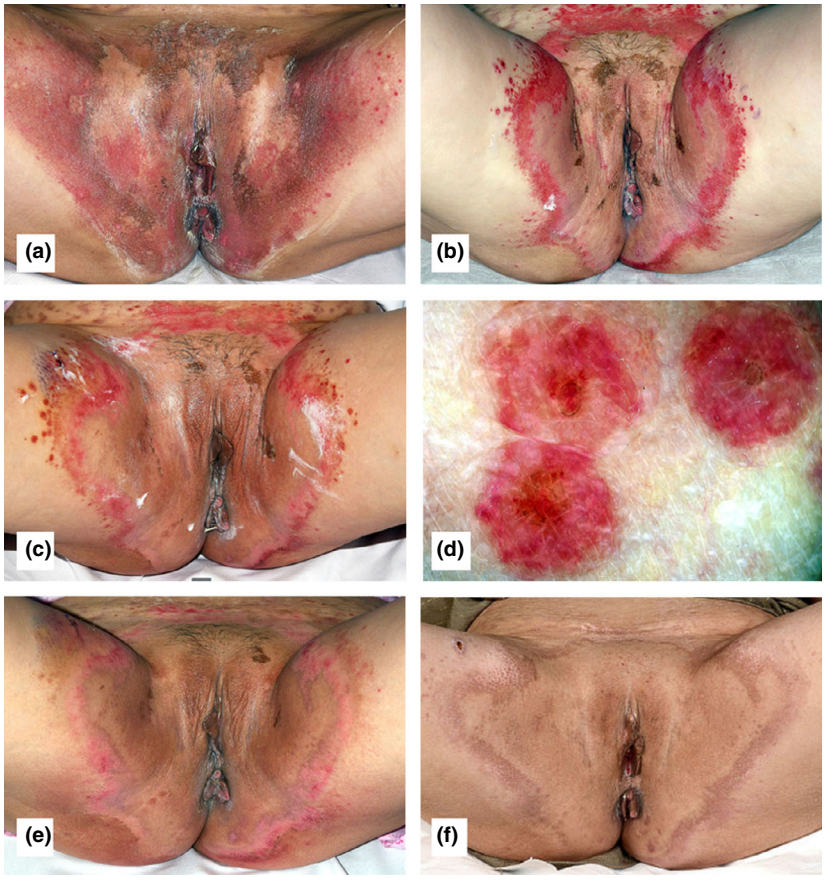

Figure 1 (a) Widespread erosions and erythema 10 days prior to hospital admission; (b) florid, palpable satellite purpura with pustular-appearing centres and ongoing vulval erosions, taken on day of admission; (c) improvement in purpuric rash and erosions (biopsy site on right inner thigh), taken after 2 days of intravenous (IV) zinc therapy; (d) closer image of satellite lesions in part (c); (e) dramatic improvement after five doses of IV zinc; (f) postinflammatory dyspigmentation 2 weeks following discharge.

often presents as a contact dermatitis (in response to often normal substances such as faeces/saliva) due to poor defences and healing. Clinically, ZDD is seen as sharply demarcated erythematous plaques/erosions, which may develop vesicles or pustules. It is most often located periorally, acrally or anogenitally and has specifically been described as a cause of vulval dermatitis.[2] In developed countries, risk factors for acquired zinc deficiency include vegetarianism, alcoholism, eating disorders and malabsorptive disorders.[3] Individuals with increased requirements (such as preterm infants) are also at risk. A number of studies[4–6] have described ZDD in such groups.

This case study demonstrates a particularly topical cause of malnutrition, related to sequelae of the COVID-19 pandemic. The dermatosis may have been multifactorial (i.e. stemming from irritant or contact dermatitis) but was undoubtedly exacerbated by the zinc deficiency, which would compromise skin integrity and healing. This case also highlights the importance of measuring zinc levels in patients with resistant anogenital dermatitis and those at risk of nutrient deficiency.

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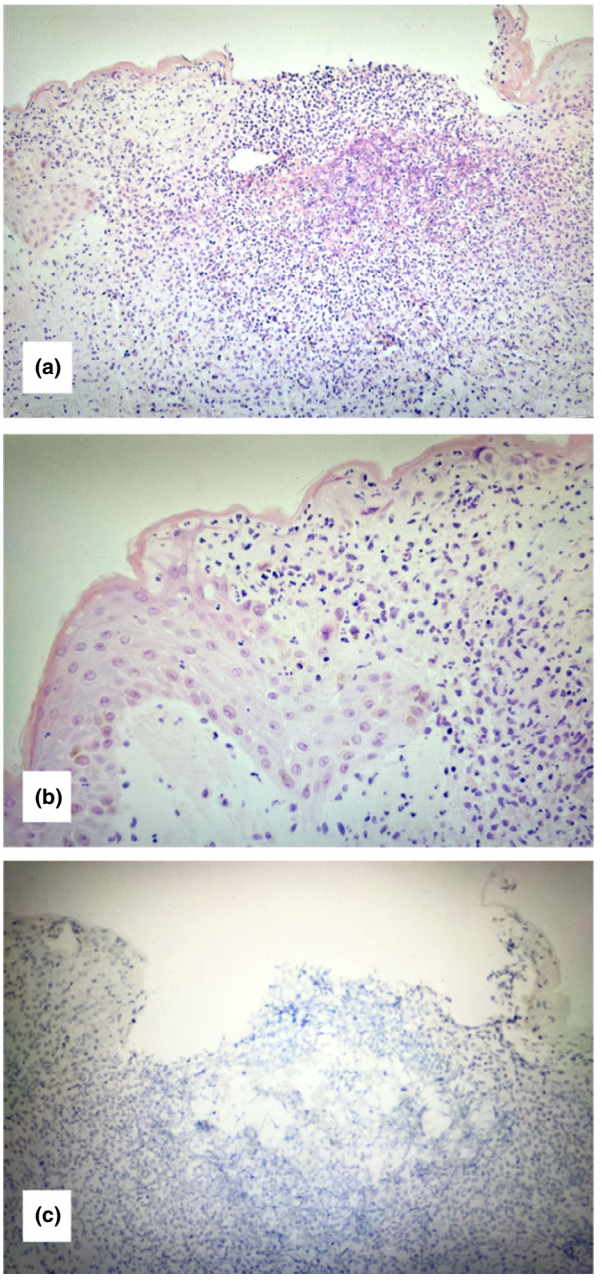

Figure 2 (a) This section demonstrates the late stages of the skin manifestations of zinc-deficiency dermatitis with cytoplasmic vacuolation, focal ulceration and epidermal necrosis; (b) intraepidermal vesicles and subcorneal pustules filled with neutrophils. Haematoxylin and eosin stain, original magnification (a) × 40; (b) × 200. (c) Immunohistochemistry for CD1a showed that the skin sample was negative for Langerhans cells, as can be seen in zinc-deficiency dermatitis (original magnification × 19).

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CPD questions

Learning objective

To demonstrate knowledge of the investigations for and the clinical manifestations of zinc deficiency.

Question 1

Which additional test is it essential to run alongside a zinc level assay, and why?

(a) Serum full blood count, because zinc deficiency commonly results in peripheral leucocytosis.

(b) Serum C-reactive protein (CRP), because zinc is a negative acute-phase reactant and therefore a normal CRP is supportive of true zinc deficiency.

(c) Serum calcium, because dietary calcium is an essential co-factor for enteral zinc absorption and hypocalcaemia can therefore trigger/exacerbate zinc deficiency.

(d) Genetic testing, because zinc deficiency is most commonly due to a congenital defect in the intestinal zinc transporter.

(e) Thyroid function tests, as zinc deficiency is commonly associated with hyperthyroidism.

Question 2

Aside from a periorificial and anogenital dermatitis, which cutaneous features are frequently observed in zinc deficiency?

(a) Eczematous plaques at sites of friction, diffuse thinning of scalp hair, paronychia, and horizontal linear indentations of the nails.

(b) Eczematous plaques at sites of friction, scarring alopecia, paronychia and Terry nails.

(c) Flexural psoriasiform dermatitis, diffuse thinning of scalp hair, ragged cuticles and transverse white bands on the nails.

(d) Flexural psoriasiform dermatitis, loss of eyebrow/eyelash hairs, onycholysis and horizontal linear indentations of the nails.

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(e) Generalized pustular psoriasiform dermatitis, diffuse thinning of scalp hair, paronychia and transverse white bands on the nails.

Instructions for answering questions

This learning activity is freely available online at http://www.wileyhealthlearning.com/ced

Users are encouraged to

- Read the article in print or online, paying particular attention to the learning points and any author conflict of interest disclosures.
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