

Erythema Ab igne

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DESCRIPTION

A man in his late 80's with chronic end-stage kidney disease (ESKD), presumed secondary to renovascular causes and a horseshoe kidney, presented to an Australian tertiary hospital with a 1-month history of worsening back pain and a new non-pruritic, painless rash across his lower back. This coincided with receiving an iron infusion prior to presentation. Other than being recently commenced on a buprenorphine patch with paracetamol and oxycodone for back pain 2 weeks prior to admission, there were no other changes to regular medications. On examination, there was evidence of a brown, hyperpigmented, non-blanching, smooth, web-like rash spanning across his lower back (figure 1).

A CT scan of his lumbar spine revealed a vertebral fracture at L3 and multiple spinal lytic lesions. Full blood examination demonstrated a microcytic anaemia: haemoglobin 83 g/L (125–175 g/L) and mean corpuscular volume 69 fL (78–98 fL). Eosinophils were $0.04 \times 10^9/L$ (0.00 – $0.50 \times 10^9/L$) suggesting this rash was not secondary to a drug reaction. Biochemistry revealed ESKD with serum creatinine 834 $\mu\text{mol/L}$ (60 – $100 \mu\text{mol/L}$), eGFR 4 mL/min ($>90 \text{ mL/min}$) and mild hypercalcaemia 2.75 mmol/L (2.10 – 2.60 mmol/L). Free lambda light chains were significantly elevated at 27 456 mg/mL (5.7 – 26.3 mg/mL) while free kappa light chains were 23.4 mg/mL (3.3 – 19.4 mg/mL); a kappa/lambda ratio of <0.001 . Serum protein electrophoresis showed an IgA monoclonal gammopathy. Total IgA and beta-2-microglobulin levels



Figure 1 Clinical photograph demonstrating erythema ab igne rash spanning across the lower back.

were significantly elevated at 46.2 g/L (0.8 – 4.5 g/L) and 75.5 mg/mL (1.4 – 3.2 mg/mL), respectively, with a low albumin 25 g/L (32 – 47 g/L). A bone

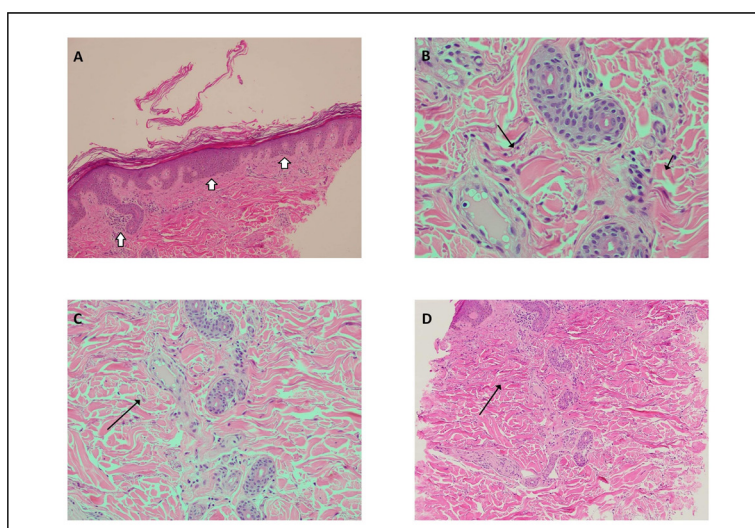


Figure 2 Histopathology of biopsy. (A) Epidermis with thinned suprapapillary plates, epidermal atrophy and some subtle patchy basal vacuolar degenerative change (open arrows) with associated overlying surface hyperkeratosis. (B) Sweat glands in the skin with elastic fibres arranged in strands and globules indicative of prominent periadnexal elastosis within the dermis (arrows). (C, D) Sparse perivascular lymphocytic infiltrate (arrows) and subtle pigmentary incontinence.



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Images in...

marrow aspirate and trephine confirmed diagnosis of IgA multiple myeloma with presence of clonal bone marrow plasma cells of 80%.

A skin biopsy performed of his lower back demonstrated histopathological findings of basal vacuolar degeneration and epidermal atrophy with overlying surface hyperkeratosis (figure 2) typically seen with erythema ab igne (EAI). Eosinophilic and neutrophilic infiltrates were not seen, making the diagnosis of drug reactions and neutrophilic dermatosis, respectively, less likely. On targeted history, the patient admitted to applying a hot water bottle directly onto his back daily to relieve his pain over the preceding month. The patient was advised to avoid further use of hot water bottles or direct heat exposure.

Management of his myeloma was conducted by the haematology team. Unfortunately, the patient died shortly after commencing treatment with bortezomib and dexamethasone due to complications of ESKD and a superimposed hospital acquired pneumonia. Resolution of EAI was unable to be realised. Given that the rash preceded commencement of bortezomib and dexamethasone and that the patient was not on any

other chemotherapy regimens, drug reactions to those medications and flagellate pigmentation were thought to be an unlikely cause.

EAI typically presents as an asymmetrical, hyperpigmented and reticulated rash that results from repeated heat contact on the skin.¹⁻³ There may be punctuate hypopigmented or hyperpigmented scars in between the hyperpigmentation, however, this was not seen in our case.^{2,4} Differential diagnoses can include livedo reticularis, livedoid vasculitis, flagellate pigmentation, cutaneous T-cell lymphoma and dermatomyositis.⁵ It is often self-limiting over weeks to months and is managed by avoiding further direct heat exposure to prevent irreversible skin changes such as focal keratinocyte atypia and even reactive angiomatosis.^{1,3} In some reports, the hyperpigmented skin persisted for years and topical tretinoin, hydroquinone and laser treatments were used successfully.^{6,7}

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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Patient's perspective

My family and I initially thought that the rash was due to the iron infusion that was given to me two weeks ago and I feared that this would be permanent. We still couldn't believe that a hot water bottle can cause this sort of looking rash, but I am relieved that it will resolve spontaneously.

Learning points

- Erythema ab igne typically presents as an asymmetrical, hyperpigmented and reticulated rash.
- Erythema ab igne results from repeated direct heat contact on the skin. Commonly reported sources of heat include hot water bottles, hot towels, laptop computers, fireplaces, stoves, heating pads, car heaters, electric space heaters and even videogame devices.
- It is often self-limiting over weeks to months which is contingent on the complete elimination of the chronic heat source. Otherwise, continued heat exposure may lead to irreversible skin changes such as focal keratinocyte atypia and even reactive angiomatosis.

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