CLINICAL PRACTICE

Case series

Diabetes and the skin

Part 4 – diabetic bullae

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Case history – Jane

Jane is showing you her latest 'blister'. The blister is thick walled, doesn't break down and heals without scarring. Jane is 72 years of age and has had type 2 diabetes for 25 years requiring insulin over the past 15 years. She has associated renal impairment and is taking the following medications: frusemide 80 mg per day, perindopril 4 mg per day, simvastatin 20 mg per day, omeprazole 40 mg per day, aspirin 100 mg per day, calcium carbonate 1.2 g per day and twice daily intermediate insulin (isophane, NPH).

'These blisters keep appearing on my legs. They don't hurt and usually go away, but if they break they can take ages to heal'.

Question 1

What are the likely causes for these blisters?

Question 2

What management would you recommend?

Answer 1

Diabetic bullae occur as spontaneous atraumatic lesions mostly on the hands, feet and lower legs, and are often recurrent. They usually arise on a noninflamed base measuring a few mm to 3–5 cm in size and heal without scarring in 2–5 weeks. The bullae can be either subepidermal or intra-epidermal without acantholysis. There is usually long established diabetes and associated peripheral neuropathy. In diabetic bullae there is usually no primary immunological abnormality.

Bullous pemphigoid would be the main differential in an elderly person. Sometimes the bullae can be localised and confined to the lower legs. Biopsy with immunofluorescence is needed to confirm the diagnosis but the immunofluorescence can be negative in some cases of bullous pemphigoid, especially those occurring on the lower legs.

A medication history is very important as bullae may occur as side effects of medication. Bullous pemphigoid and other autoimmune blistering disorders can be precipitated by several medications and pseudoporphyria can be triggered by medication such as naproxen, frusemide, tetracyclines and nalidixic acid. Nonautoimmune bullous drug eruptions can also occur.

Fixed drug eruptions should be suspected if the bullae are occurring in the same site and heal with hyperpigmentation. The most common medications causing fixed drug eruptions are sulphonamides, tetracyclines, NSAIDs and phenolphthalein.

Bullae can occur with renal failure, particularly if the patient is also taking high dose frusemide and this should be changed to an alternative until a diagnosis is made. Bullae can occur in patients with renal failure on dialysis. Sometimes this is related to elevated porphyrin levels, which should be measured.

Other causes would include bullous insect bites or bullae secondary to gross pitting oedema. Other autoimmune blistering diseases such as epidermolysis bullosa acquisita or pemphigus are less likely, but can be excluded on biopsy.

Answer 2

Jane's bullae are recurrent and it would be worthwhile identifying and ideally reversing or treating the cause. The blisters have the clinical characteristics of diabetic bullae (asymptomatic, thick walled with no identifiable precipitants) but a careful medication history might identify a preventable precipitant. A biopsy would confirm the diagnosis of diabetic bullae and exclude the major differential of bullous pemphigoid.

Jane's renal failure may be contributing, but she is not taking high doses of frusemide (80 mg/day compared to doses in renal failure of 250–1000 mg/day) and she is not yet on dialysis. The other causes are easily excluded from the history (insect bites), examination (oedema), and biopsy (other autoimmune blistering diseases).

Management is conservative and symptomatic. If the bullae are intact, there is no need to prick them and an antiseptic cream and protective dressing is adequate. If the skin is broken, an anti-ulcer treatment would be worthwhile such as silver sulfadiazine/chlorhexidine cream (Silvazine) or a hydrocolloid or alginate dressing. Glycaemic diabetic control does not help to reduce the number of bullae occurring but is worthwhile achieving.

Conflict of interest: none.

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