Case 1
A 71-year-old Indonesian gentleman presented with a skin plaque on the left ankle noted 30 years ago, growing steadily in size. Examination revealed an annular erythematous plaque with a raised creeping ridge-like rim and prominent central clearing. Histology from another dermatology unit showed slight to moderate superficial vasodilatation, with scattered hemosiderin deposition, and was thought to be pigmented purpuric dermatosis [Figure 1].

A diagnosis of porokeratosis of Mibelli was made. Topical imiquimod and tacrolimus were prescribed, with initial improvement after a period of 2 weeks.

As the lesion was slow to improve, a venous stasis was suspected. Venous ultrasound of both lower limbs showed incompetent bilateral saphenous veins as well as left deep vein incompetence. Repeat histology showed dermal fibrosis with markedly increased vascularity, hemorrhage and cellular inflammatory infiltrate composed of lymphocytes, histiocytes, and scattered eosinophils. The epidermis showed acanthosis, spongiosis, and lymphocytic exocytosis with focal parakeratosis. The diagnosis was revised as porokeratosis-like AAD. Regular use of compression stockings was recommended, but the patient was unable to tolerate these. The lesion remained largely unchanged [Figure 2].

Case 2
A 90-year-old Chinese gentleman was seen for discrete and compressible bullae over the lower limbs, associated with lower limb edema. Indurated hyperpigmented skin and varicose veins were also noted [Figure 3].

Serology for BP180, BP230, and indirect immunofluorescence were negative, thus ruling out bullous pemphigoid. Vascular scans showed deep vein incompetence with insufficiency of the great saphenous veins in both legs. Histology of a bulla showed adipose tissue only with focal acute inflammatory infiltrate.

Clinico-pathological correlation led to a diagnosis of a bullous form of AAD. Regular use of compression stockings led to reduction of lower limb edema and resolution of blisters.

AAD is characterized by multiple sharply defined purple-brown papules, plaques or ulcerations, located on the dorsa of the feet and medial lower limbs.[2] These can be unilateral or bilateral, depending on the underlying clinical condition. Chronic venous insufficiency leads to increased venous pressure with resultant proliferation and dilatation of veins and superficial plexus and extravasation of red blood cells, presenting clinically as purple nodules and ulceration.

Both patients had unusual clinical presentations of AAD, namely, one mimicking porokeratosis and another...
Clinically, the non-inflammatory bullae seen in the second case were suggestive of bullous pemphigoid or bullous diabeticorum. Serology and histology excluded these conditions.

Mainstay treatment for AAD is the correction of underlying pathology and use of compression therapy. Drugs such as antimicrobials, topical and oral steroids, and diuretics have been employed.[3] Heller et al. reported good results with a combination of oral erythromycin 500 mg four times a day or dapsone 50 mg twice a day for 3 months with compression therapy.[4]

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

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