

endovascular repair may be necessary in the high-risk group, such as patients with refractory pain or hypertension, an expanding haematoma, or signs of impending rupture.² However, the appropriate management of stable PAU, such as in our patient, is controversial.

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LETTERS TO THE EDITOR

Clinical-scientific notes

Pyoderma gangrenosum developing over an arteriovenous fistula scar

A 47-year-old man was admitted to our hospital for a radiocephalic arteriovenous fistula (AVF) in the right arm for dialytic treatment. His past medical history included Wegener granulomatosis diagnosed 8 years previously and chronic kidney disease for which he was receiving kidney replacement therapy in the form of haemodialysis for 7 years. Twenty-four hours after the operation, the patient complained of pain, fever, general discomfort and inflammation in the surgical area. He was admitted to hospital for monitoring and observation. The AVF was dismounted and ligatured and the aneurysmatic area removed and debrided. Response was positive for the first 24 h. However, 48 h after the operation, he began suffering from intense pain and discharge from the operated area. After 15 days, he began to develop a painful ulcer, which gradually increased in size. The skin examination showed a 15 \times 8-cm ulcer with irregular, raised, congestive, violet coloured erythematous edges, along with abundant granulation tissue (Fig. 1a). Dermatologists were consulted and they suggested pyoderma gangrenosum (PG) as a possible diagnosis according to the clinical findings. This diagnosis was confirmed by a skin biopsy, which revealed inflammatory infiltration, consisting of neutrophil accumulation, abscess formation, disintegration of the matrix and areas of necrosis, presence of necrotizing perivasculitis and leukocytoclastic vasculitis of the peripheral tissue, lymphocytes and macrophages around the microvascularization. Treatment was started with corticosteroid therapy, cleansing with saline solution and topical application of betamethasone valerate with gentamicin, with significant improvement. From then on, the PG began to progress positively, with no need for immunosuppressive therapy, leaving a 2-cm ulceration, which closed completely with the topical application (Fig. 1b). PG is a very uncommon cutaneous





Figure 1 (a) Ulcer with irregular, raised, congestive, violet-coloured erythematous edges, along with abundant granulation tissue. (b) After 8 weeks of treatment

ulceration of unknown aetiology, described as a painful, progressive, ulcerative, neutrophilic dermatosis.¹ The atypical feature of our case is the unusual localization of PG occurring on the arteriovenous dialysis shunt.

The pathergy phenomenon could explain the fact that PG sometimes occurs after minor injuries or surgical operations in patients with or without associated systemic autoimmune disease.² If it is caused by an injury, the lesions usually appear on the lower limbs.³ Some authors have proposed PG in the differential diagnosis of complications in postoperative wounds,⁴ emphasizing the fact that its early diagnosis can facilitate appropriate treatment, avoiding unnecessary and ineffective procedures.⁵ In our patient, PG was suspected because antibiotic therapy showed no improvement. Despite the unusual localization, the diagnosis of ulcerative PG was considered based on the medical history, laboratory and histopathological findings and excluding other causes of skin ulcers. Ulcerative PG usually occurs on legs and is

thought to be related to underlying vascular insufficiency of the legs. In our case, the ulcerative PG developed on the arteriovenous dialysis shunt. Furthermore, our patient responded quickly to corticosteroid therapy, which is the usual treatment for ulcerative PG. We present an atypical case of PG and we think every physician should keep in mind PG in patients with refractory ulcers of unclear origin. To our knowledge, this is the first case of PG occurring on an arteriovenous dialysis shunt.

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Combination antiretroviral therapy as treatment for human immunodeficiency virus-associated mucosa-associated lymphoid tissue type lymphoma of the nasopharynx

Optimal therapeutic strategies for mucosa-associated lymphoid tissue type (MALT) lymphoma in association with human immunodeficiency virus (HIV) infection are unknown. We present a case illustrating that anti-retroviral therapy alone may be a potential treatment modality.

A 48-year-old man with a distant past history of injecting drug use presented with a 3-year history of nasal and ear obstruction with a middle ear effusion. He reported weight loss and sicca symptoms. Fibre-optic nasendoscopy revealed a submucosal mass involving

the left wall and roof of the nasopharynx. Biopsy revealed nasopharyngeal lymphoid tissue infiltrated with small cells consistent with the diagnosis of an extra nodal marginal zone B cell lymphoma (MALT type) as per the World Health Organization classification system. Staining was positive for CD20 and negative for CD5, CD10 and cyclin. Helicobacter pylori infection was not specifically excluded and staging investigations revealed no extension of the tumour (stage 1E). He tested positive for antibodies to HIV. The HIV viral load was greater than 100 000 RNA copies/mL and he was immunodeficient with a CD4 lymphocyte count of 140/μL (7%). He had previously been treated for hepatitis C virus infection (genotype 3a) with treatment response but relapse of viraemia. Combination antiretroviral therapy (cART) including zidovudine, lamivudine and lopinavir/ritonavir was commenced given the