NDT Plus (2011) 4: 443–444
doi: 10.1093/ndtplus/sfr120

NDT PLUS
Nephrology Dialysis Transplantation

Nephroquiz
(Section Editor: M. G. Zeier)

Unusual skin lesions in a haemodialyzed patient

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Keywords: haemodialysis; Kaposi's sarcoma; skin lesions

Case

A 71-year-old woman of Turkish origin presented in July 2009 to our haemodialysis unit with multiple painless, non-pruritic, reddish-purple skin lesions on legs and feet. She had been on peritoneal dialysis from July 2006 to December 2008 for presumed diabetic nephropathy and then shifted to haemodialysis due to recurrent peritonitis. She was under low-dose steroids since February 2008 for seropositive polyarthritis. Steroids were increased in May 2009 following a third sub-occlusive episode due to sclerosing encapsulating peritonitis.

At presentation, she was taking methylprednisolone (12 mg per day), aspirin, omeprazole, atorvastatin, gabapentin, clonazepam, calcium carbonate and insulin.

Clinical examination showed multiple infracentimetric dark red to violaceous non-tender macules, papules and nodules on legs and feet (Figure 1). No ulceration or necrosis was visible. No inguinal lymph nodes were palpable. Physical examination revealed no other new finding.

Blood tests showed normal C-reactive protein, Complements C3 and C4 serum levels, a normal eosinophil count and negative anti-nuclear and anti-neutrophil cytoplasmic antibody (ANCA) titres. Platelet count and coagulation tests were normal. Serologic tests for HIV, Hepatitis C and Hepatitis B were negative.

Question

What is the aetiology of the skin lesions?

Answer

Kaposi's sarcoma

Discussion

Histopathology of a skin lesion showed a dermal proliferation of neoplastic spindle-shaped cells with slit-like vascular spaces, surrounded by lymphocytes and plasma cells (Figure 2A). Immunohistochemical staining was strongly positive for CD31, CD34 and human herpes virus 8 (HHV-8) in spindle and endothelial cells (Figure 2B).

Kaposi's sarcoma (KS) was thus diagnosed. Serologic tests for HHV-8 were positive both in 2008 (retrospectively, before haemodialysis initiation) and at diagnosis of KS. Thoracoabdominal computed tomography (CT) did not detect visceral KS, but no gastrointestinal endoscopy was performed. Systemic doxorubicin was not administered because of the potential cardiotoxicity, co-morbidities and absence of visceral involvement. The patient was treated with intra-lesional injections of vincristine sulphate every 3 weeks.

After 3 months of treatment, regression of cutaneous KS was noted and visceral involvement still not detected. Two months later, the patient died suddenly.

End-stage renal disease has been associated with many distinctive skin problems. Generalized itching or pruritus is frequent (up to 50–90% of patients) but its pathogenesis (partially secondary to xerosis) remains largely unclear. Pruritis can lead to secondary skin lesions such as prurigo nodularis and excoriations. Drug-induced allergic rashes are most often secondary to antibiotics (and heparin) and can be associated with hypereosinophilia. Diabetic patients are more vulnerable to bacterial skin infections.

Other differential diagnoses include calciphylaxis (very painful necrotic lesions), skin vasculitis (ANCA-associated, cryoglobulinemia), cholesterol emboli and venous stasis [1]. Our patient presented with painless and non-pruritic skin lesions. The nodular aspect of the lesions, their violaceous colour and the patient's Mediterranean background all suggested KS.

KS is a malignant tumour involving blood and lymphatic vessels, affecting predominantly the dermis and less frequently the gastrointestinal tract and regional nodes. HHV-8 has been implicated in the pathogenesis of all forms of KS [classic (European and Mediterranean), endemic (HIV

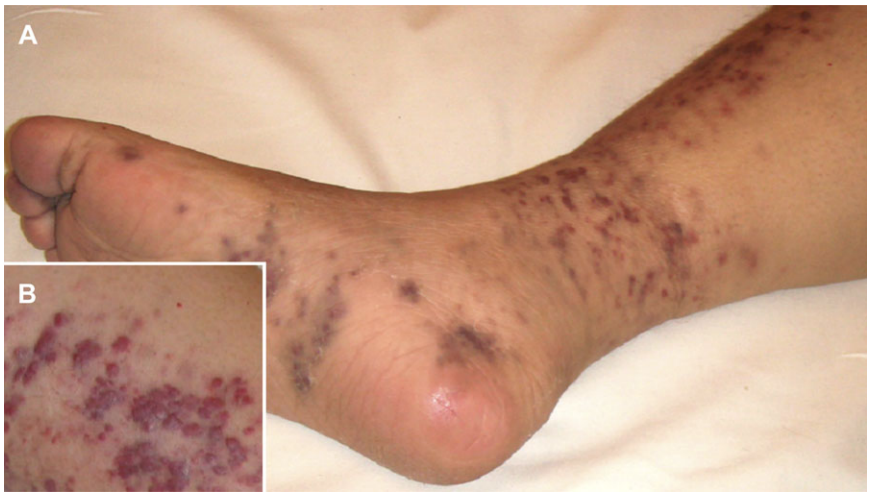

Fig. 1. Multiple dark red to violaceous macules, papules and nodules on the right leg and sole of the foot (Panel A), with firm angioma-like nodules on close-up (Panel B).

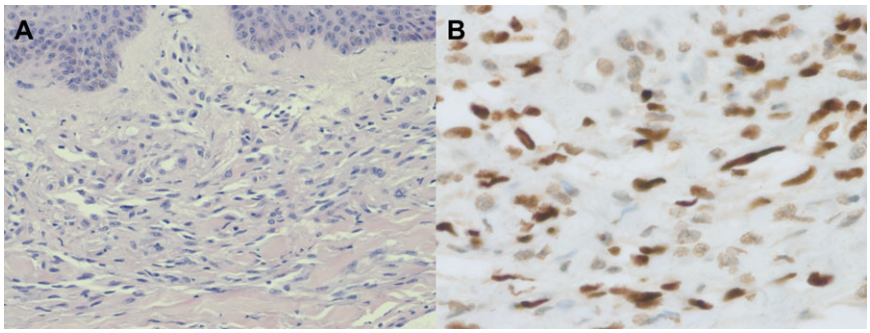

Fig. 2. Panel A: proliferation of spindle-shaped cells leading to the formation of abnormal vascular slits (haematoxylin and eosin, objective × 20). Panel B: nuclei positive for HHV-8 by immunochemistry (objective × 40).

negative, African), epidemic (AIDS related) and iatrogenic (immunosuppressive therapy)] [2]. KS has been infrequently reported in haemodialysed patients [3, 4].

Our patient probably developed a Mediterranean form of KS, further promoted by her immunodepressed status (long-term steroids, dialysis, diabetes). Indeed, ethnic groups at risk for classic KS, see their risk increased by the initiation of immunosuppressive therapy [2].

KS mostly follows a chronic course and metastases are rare. Treatment decisions take into account the type and extent of KS, the organs involved and the virologic and immune status. Treatment options include surgery, radiation therapy, chemotherapy (systemic and intra-lesional) and interferon-alpha, treatment of HIV and dose reduction or withdrawal of immunosuppressive agents [2].

Though rarely reported in haemodialysed patients, KS must be correctly diagnosed to permit a prompt and appropriate treatment.

Conflict of interest statement. None declared.

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Received for publication: 27.12.10; Accepted in revised form: 24.8.11