

Challenging Case

Necrotic Plaque on the Penis

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Case presentation

A 58-year-old man with a past medical history of hypertension, long-standing diabetes mellitus, and end-stage chronic kidney disease (CKD) on hemodialysis (HD) for 3 months presented with a 1-month history of lesion on the glans penis. He reported that it was initially whitish, mildly painful, and progressively increased in size and darkened.

Physical examination

A 2-cm adherent necrotic plaque was observed on the glans penis, with well-defined irregular erythematous–violaceous borders and no apparent urethral involvement (Fig. 1). No inguinal lymphadenopathy or similar cutaneous lesions were detected elsewhere.

Histopathology

Initial histologic examination revealed extensive necrosis, along with a neutrophilic inflammatory infiltrate and karyorrhexis. A subsequent



Fig. 1.

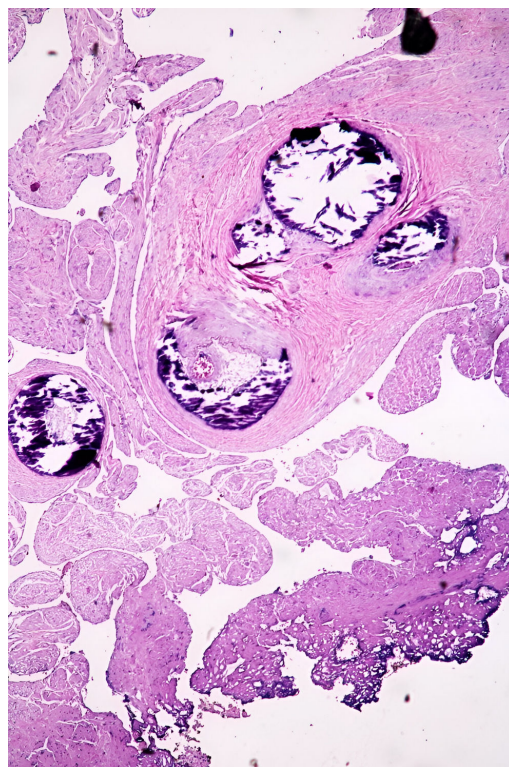


Fig. 2.

biopsy showed, in addition to large areas of tissue necrosis with bacterial colonies, medium-caliber vessels with intraluminal wall calcification and associated myointimal hyperplasia (Fig. 2).

Additional tests

Blood tests showed elevated parathyroid hormone levels, with normal calcium (Ca) and phosphorus (P) levels and a Ca × P product of 54.6 (< 55). Infectious serologies were negative. Angio-CT revealed extensive calcification of pudendal and penile vessels (Fig. 3).

What is your diagnosis?

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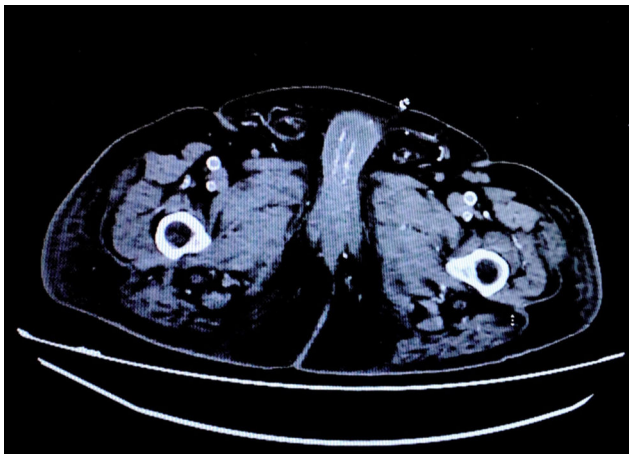


Fig. 3.

Diagnosis

Penile calciphylaxis.

Course and treatment

Intravenous and topical sodium thiosulfate (STS) was administered, along with hemodialysis and optimization of calcium–phosphorus metabolism. Despite these measures, the lesion progressed, leading to sepsis, and total penectomy was performed. Despite surgical and medical treatment, the patient had an unfavorable outcome and died 3 months after symptom onset.

Comments

Calciphylaxis (Cx) is a rare condition resulting from dystrophic calcification of the intimal and medial layers of small- and medium-sized dermal and hypodermal arterioles, leading to ischemic tissue necrosis.¹ When it occurs in the setting of end-stage CKD, it is classified as uremic calciphylaxis, whereas nonuremic calciphylaxis occurs in patients with preserved renal function.² Its incidence ranges from 1% to 4% in patients with end-stage CKD undergoing hemodialysis.³ Associated risk factors include end-stage CKD (particularly in patients on peritoneal dialysis), disturbances in calcium–phosphorus metabolism, vitamin K deficiency (often related to anticoagulant therapy), obesity, rapid weight loss, diabetes mellitus, and female sex.²

Penile calciphylaxis (CxP) is rare due to the rich vascular supply of the penis. Clinically, it presents with dysesthesia and the appearance of painful erythematous–violaceous nodules or reticular plaques on the glans, which may progress to ulcers with necrotic eschar.⁴ Differential diagnosis is broad and includes conditions presenting with ulcers and/or

necrosis, such as Fournier gangrene, squamous cell carcinoma, arterial ischemia, and pyoderma gangrenosum. A definitive diagnosis may be clinical when presentation is typical.⁵ Imaging modalities (ultrasound, pelvic radiography, or angiography) may support the diagnosis. Skin biopsy should be reserved for cases with diagnostic uncertainty and, if performed, should be deep, as histologic findings are located in the dermis and hypodermis.¹

Prognosis is poor. One-year mortality in uremic calciphylaxis ranges from 45% to 80%.³ In a series of 34 patients, mortality associated with CxP reached 64%, with a mean survival time of only 2.5 months.⁴

Treatment is based on 3 main pillars: correction of calcium–phosphorus metabolism (increased dialysis sessions, treatment with cinacalcet, parathyroidectomy), inhibition of calcification and restoration of blood flow (intravenous, topical 25%, or intralesional STS, bisphosphonates, discontinuation of vitamin D, revascularization), and management of local pain and wound care (antibiotic therapy, hyperbaric oxygen therapy, penectomy, among others).^{2,5,6}

In conclusion, CxP is a rare condition that should be suspected in the presence of necrotic genital lesions in patients with end-stage CKD.

Conflict of interest

The authors declare that they have no conflict of interest.

Acknowledgments

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