

Introduction

A condition with a high mortality rate, penile calcific uremic arteriopathy (CUA) also known as calciphylaxis, is a dire complication of chronic renal failure distinguished by medial calcium overload and intimal fibrosis of medium and small arteries with associated gangrene of affected tissues. The condition has been majorly reported to include distal extremities, thigh and the gluteal muscles. While becoming increasingly more recognized and reported in the literature, few cases of penile necrosis secondary to CUA have been reported in the literature. We present a surviving patient with systemic penile CUA and several severe secondary conditions without parathyroidectomy. Photographic signed consent was obtained from the patient, including IRB approval for the case report.

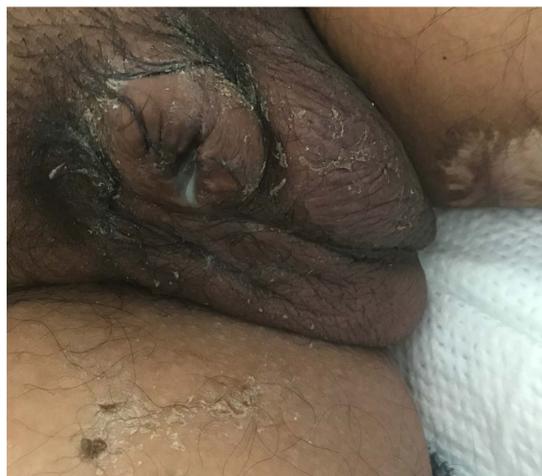


Image 2. Glans penis status post partial penectomy.

Case Presentation

40-year-old male with a past medical history of uncontrolled diabetes myelitis type two, traumatic amputation of toes, hypertension, pneumonectomy status post thoracic gunshot wound one year ago and hyperparathyroidism in the setting of end stage renal disease (ESRD), a history of alcohol and drug abuse, not on dialysis, presented to our facility with two weeks of shortness of breath, productive cough, bilateral lower extremity edema, diffuse rash on upper and lower extremities and severe penile pain and dysuria. The patient stated having sexual encounters with multiple partners and was initially placed on azithromycin and ceftriaxone for gonorrhea treatment and was admitted to the internal medicine service on septicemia, congestive heart failure, and pneumonia diagnosis. However, penile pain did not subside. On hospital day two, a 1cm white lesion with black border on the glans penis surrounding the urethral meatus was noted after observation of a foley catheter (**Image 1**).



Image 1. Glans penis displaying black calcification surrounding the urethral meatus and foreskin.

Discussion

CUA, once termed calciphylaxis, was once thought to be a rare complication of ESRD, but is becoming increasingly recognized in the medical literature. Optimal treatment is not yet known, but some treatment pathways include sodium thiosulfate and non-calcium-containing phosphate binders such as sevelamer, cinacalcet, for patients with elevated PTH, hyperbaric oxygen and sterile maggot therapy. Surgical management in the medical literature suggests that parathyroidectomy may significantly improve survivorship and that survival is independent of the type of local treatment for penile lesions. Pathophysiology is not fully understood, though clinical manifestations result from reduction in arteriolar blood flow, with medial vessel calcification occurring first. Ongoing vascular endothelial injury results in cutaneous arteriolar narrowing and hypercoagulable state that cause tissue infarction. Deep vascular involvement will subsequently lead to limb ischemia. In our case, sloughing of the calcified area did not occur and a partial penectomy was required (**Image 2**).

Conclusions

While CUA was once considered to be a rare complication of ESRD, it has increasingly become more recognized in the medical literature in the past fifteen years. Penile CUA is an extremely rare subset of CUA resulting in penile gangrene, resulting from intimal fibrosis and medial calcification of vasculature. In our case, findings incorporated arteriolar calcifications resulting in necrotic skin lesions including deep vascular calcifications that were identified easily with plain films. Associated with high comorbidity and mortality, clinical suspicion of CUA must remain high in ESRD as early diagnosis in the emergency department and prompt intervention can help prevent amputation, length of hospital stay and mortality. Our case raises awareness in maintaining a high index of suspicion of a condition that may present itself initially with minimally evident physical findings and can progress rapidly if not carefully evaluated.

References

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