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Case Report

A fatal convergence: necrotizing fasciitis, critical limb ischemia, and severe secondary hyperparathyroidism in a patient with end-stage renal disease

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ABSTRACT

This case report details the clinical course of a 39-year-old female with a complex medical history, including type 2 diabetes mellitus, hypertension, and end-stage renal disease (ESRD) on hemodialysis, who presented with rapidly progressive necrotizing fasciitis (NF) of the left hand. The case exemplifies a catastrophic synergy between severe peripheral arterial disease (Rutherford 6), a polymicrobial soft tissue infection, and profound metabolic disarray secondary to renal failure. Critical laboratory findings included persistent hyperphosphatemia, hypocalcemia, and a markedly elevated parathyroid hormone (PTH) level of 270.42 pg/mL, confirming severe secondary hyperparathyroidism. This renal osteodystrophy contributed to metastatic calcification and vascular calciphylaxis, creating a non-viable tissue environment that thwarted healing and necessitated a transradial amputation. Despite surgical intervention, patient faced a high risk of contralateral limb amputation due to the underlying calcific uremic arteriolopathy, highlighting devastating systemic consequences of untreated secondary hyperparathyroidism in ESRD.

Keywords: End-stage, Renal disease, Hemodialys, Necrotizing fascitis

INTRODUCTION

Patients with ESRD on dialysis face a significantly increased risk of morbidity and mortality from cardiovascular events and infections. A key driver of this pathology is chronic kidney disease-mineral and bone disorder (CKD-MBD), characterized by dysregulation of calcium, phosphate, and vitamine D metabolism. This leads to secondary hyperparathyroidism (sHPT), where chronically elevated PTH levels attempt to normalize serum calcium but ultimately lead to bone resorption and metastatic calcification in soft tissues and the blood vessels.[1-5]

This vascular calcification, known as calciphylaxis, manifests as painful, necrotic skin ulcers and accelerates peripheral arterial disease, rendering tissues ischemic and highly susceptible to infection. NF is a life-threatening soft-tissue infection requiring urgent surgical debridement. Its progression is markedly worsened in an ischemic environment, creating a clinical scenario with extremely high limb loss and mortality rates.[5-8]

CASE REPORT

History and presentation

A 39-year-old female with a past medical history of type 2 diabetes mellitus (since 2005, on insulin), hypertension (since 2019), and ESRD (KDIGO stage V, on hemodialysis since 2023, Tuesday/Thursday/Saturday) presented in late August 2025. The illness began after a puncture wound to the left middle finger, which

progressed to pain, itching, edema, local warmth, and subsequent discoloration. An x-ray was performed showing total calcificaicon of the limb (Figure 1).

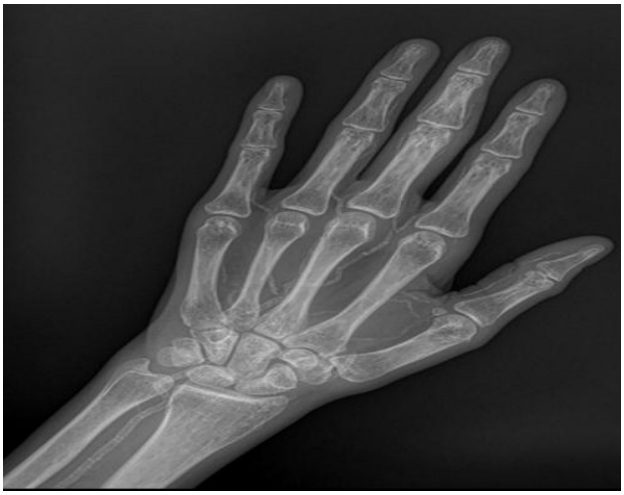

Figure 1: Total calcification of the upper limb.

After initially leaving the emergency department without treatment, she underwent an amputation of the left middle finger at a private facility on September 5, 2025 (Figure 1). She re-presented on September 9, 2025, with a worsening infected surgical site.

Figure 2: Left upper extremity after 3rd left finger amputation.

Physical examination on admission (09/09/2025)

The patient was awake, alert, and oriented. The left upper extremity showed a surgical absence of the middle finger with a necrotic bed, and necrosis of the index and ring fingers (Figure 2).

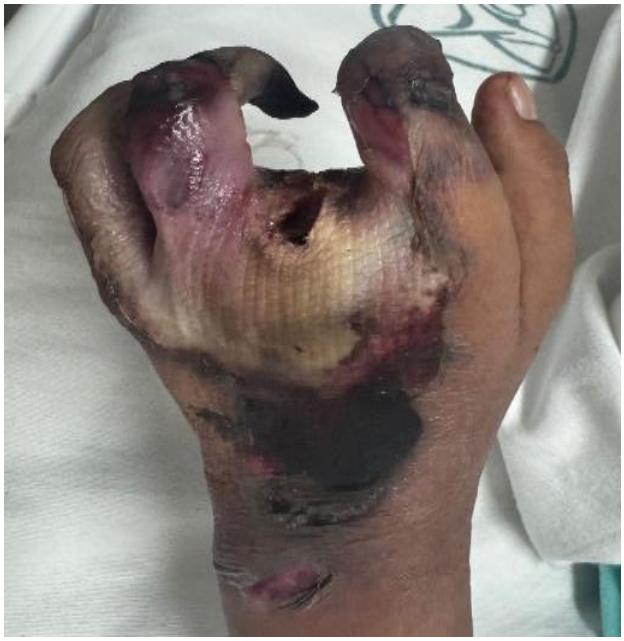

Figure 3: Upper left extremity at admision.

The area was cold, with non-palpable interdigital, radial, and ulnar pulses, and was fetid. Similar necrotic digital ulcers were present on the right hand. Bilateral lower extremities also showed necrotic ulcers on the distal fifth toe (left) and plantar surface (right), with diminished pulses.

Relevant laboratory evolution (Chronologically)

- *22-25 March 2025:* Early evidence of renal failure metabolic disarray: Hyperphosphatemia (9.26 mg/dL), Hyperkalemia (6.84 mmol/L), hyponatremia (131 mmol/L), elevated BUN/creatinine.
- *23 July 2025:* Worsening renal parameters (Creatinine 4.63 mg/dL). Hypocalcemia (8.28 mg/dL) and hyperphosphatemia were present, establishing the classic pattern for sHPT.
- *03 September 2025:* Admission to ER with critical values: Severe hyperglycemia (632.2 mg/dL), acidosis (low bicarbonate inferred from anion gap), hyperkalemia (6.9 mmol/L), hypocalcemia (7.83 mg/dL), and hyperphosphatemia.
- *09 September 2025:* Markers of infection and inflammation: Leukocytosis (17.45 K/µL), elevated Procalcitonin (2.56 ng/mL -high risk for sepsis), coagulopathy (INR 1.48). Renal function: creatinine 5.26 mg/dL.
- *17 September 2025: critical PTH result:* Parathyroid Hormone was drastically elevated at 270.42 pg/mL (ref: 15.00-68.30), providing definitive evidence of severe sHPT. Concurrently, vitamin D3 was deficient at 8.2

pg/mL. Chemistry showed persistent hyperphosphatemia (6.45 mg/dL) and hypocalcemia (7.36 mg/dL).

19 September 2025: Continued pattern of hyperphosphatemia (7.5 mg/dL) and hypocalcemia (7.24 mg/dL).

Hospital course, interventions, and outcome

The patient was diagnosed with necrotizing fasciitis (LRINEC score 6) and critical limb ischemia (Rutherford category 6). Despite broad-spectrum antibiotics, extent of necrosis and underlying vascular disease mandated urgent surgical intervention. On September 12, 2025, she underwent left transradial amputation (Figure 3).

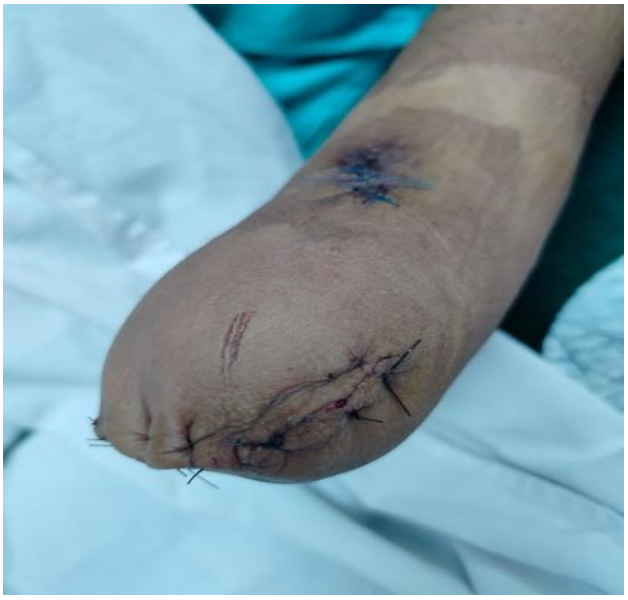

Figure 4: Post operative outcome.

Operative findings described "intraluminal calcifications" in the radial and ulnar arteries, a hallmark of the vascular calcification driven by her sHPT and hyperphosphatemia.

Post-operatively, she remained hemodynamically stable but was noted to have progressive necrotic lesions on the contralateral right hand and both feet. The clinical team urgently requested angiographic studies to assess for revascularization possibilities but faced delays. The patient was deemed at "very high risk for amputation of the remaining limbs." Plans were made for transfer to a tertiary care center for specialized vascular surgery consultation.

Pathophysiology of sHPT and its role in this case

The patient's ESRD is the cornerstone of this pathophysiology. The failing kidneys cannot excrete phosphate, leading to hyperphosphatemia. Concurrently, impaired renal production of active vitamin D (Calcitriol) leads to reduced intestinal calcium absorption, causing hypocalcemia. Both hyperphosphatemia and hypocalcemia are potent stimulators of PTH secretion, leading to sHPT. The profoundly elevated PTH (270.42 pg/mL) has two major detrimental effects:

Bone resorption

In an attempt to normalize serum calcium, PTH leaches calcium and phosphate from the bones, worsening renal osteodystrophy.

Metastatic calcification

The chronic excess of calcium and phosphate products, despite low serum calcium, leads to deposition of calcium-phosphate crystals in soft tissues, including the media layer of blood vessels (arteriolar medial calcification). This process, calciphylaxis, results in:

Vascular stenosis and ischemia

The calcified vessels become rigid and narrowed, severely compromising blood flow to the extremities.

Tissue necrosis

The resulting chronic ischemia makes tissues vulnerable to breakdown and incapable of healing.

Increased infection risk

Poorly perfused tissue is a perfect breeding ground for bacteria.

In this case, the initial puncture wound occurred in an extremity already severely compromised by diabetic vasculopathy and, more critically, by calciphylaxis from sHPT. The minimal blood flow prevented an adequate immune response and delivery of antibiotics, allowing a simple infection to escalate into life-threatening necrotizing fasciitis. The vascular calcification observed during surgery was a direct consequence of this process, making limb salvage impossible and threatening her other limbs.

DISCUSSION

This case tragically illustrates the systemic domino effect of uncontrolled CKD-MBD. The primary issue was not just the necrotizing infection but the underlying tissue environment that allowed it to flourish. The severe sHPT and hyperphosphatemia created a state of functional end-organ ischemia through calciphylaxis.[7-9]

Management of such cases is exceptionally challenging. While urgent surgical debridement is the standard for NF, its success is limited when the underlying blood supply is irreversibly damaged. The focus must be on aggressive prevention and management of CKD-MBD long before

such complications arise. This includes strict phosphate binders, calcimimetics (like Cinacalcet), and judicious use of vitamin D analogues to control PTH levels and mitigate vascular calcification. The delayed procurement of an angiogram highlights systemic barriers in managing these complex patients, where time is of the essence for limb salvage.[9-13]

CONCLUSION

This case underscores secondary hyperparathyroidism as a serious systemic disease, not just a bone disorder. It was a primary contributor to vascular calcification and critical limb ischemia.The convergence of ESRD, sHPT, diabetes, and PAD created a perfect storm, culminating in necrotizing fasciitis and necessitating amputation.

Hyperphosphatemia was a key driver in this pathophysiology, leading to the elevated PTH and subsequent metastatic calcification.A multidisciplinary approach involving nephrology, vascular surgery, infectious disease, and endocrinology is crucial from the early stages of CKD to prevent such devastating outcomes.This case serves as a stark reminder of the importance of aggressive management of mineral metabolism in ESRD patients to prevent the non-renal consequences of the disease, which are often more lethal than the renal failure itself.

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REFERENCES

1. Whitaker CM, Low S, Gorbachova T, Raphael JS, Williamson C. Imaging and Laboratory Workup for Hand Infections. Hand Clin. 2020;36(3):285-99.
2. Misiakos EP, Bagias G, Patapis P, Sotiropoulos D, Kanavidis P, Machairas A. Current Concepts in the Management of Necrotizing Fasciitis. Front Surg. 2014;1.
3. Gawaziuk JP, Liu T, Sigurdson L, Edward B, Thomas EJH, Sarah S, et al. Free tissue transfer for necrotizing fasciitis reconstruction: A case series. Burns. 2017;43(7):1561-66.
4. Lille ST, Sato TT, Engrav LH, Foy H, Jurkovich GJ. Necrotizing soft tissue infections: Obstacles in diagnosis. J Am Coll Surg. 1996;182(1):7-11.
5. Neeki MM, Dong F, Au C, Jake T, Nima K, Carol L, et al. Evaluating the Laboratory Risk Indicator to Differentiate Cellulitis from Necrotizing Fasciitis in the Emergency Department. West J Emerg Med. 2017;18(4):684-9.
6. Gonzalez MH, Kay T, Weinzweig N, Brown A, Pulvirenti J. Necrotizing fasciitis of the upper extremity. J Hand Surg Am. 1996;21(4):689-92.
7. Wronski M, Slodkowski M, Cebulski W, Karkocha D, Krasnodebski IW. Necrotizing fasciitis: Early sonographic diagnosis. J Clin Ultrasound. 2011;39(4):236-9.
8. Melillo A, Addagatla K, Jarrett NJ. Necrotizing Soft Tissue Infections of the Upper Extremity. Hand Clin. 2020;36(3):339-44.
9. Aizawa T, Nakayama E, Kubo S, Nakamura K, Azuma R, Kiyosawa T. Severe Functional Loss of the Hand due to Necrotizing Fasciitis with Underlying Vasculitis Neuropathy. J Hand Microsurg. 2019;11(S 01):S42-5.
10. Bluman EM, Mechrefe AP, Fadale PD. Idiopathic Staphylococcus aureus necrotizing fasciitis of the upper extremity. J Shoulder Elb Surg. 2005;14(2):227-30.
11. Anaya DA, McMahon K, Nathens AB, Sullivan SR, Foy H, Bulger E. Predictors of mortality and limb loss in necrotizing soft tissue infections. Arch Surg. 2005;140(2):151-7.
12. Hsiao CT, Chang CP, Huang TY, Chen YC, Fann WC. Prospective validation of the laboratory risk indicator for necrotizing fasciitis (LRINEC) score for necrotizing fasciitis of the extremities. PLoS One. 2020;15(1):e0227748
13. Shyy W, Knight RS, Goldstein R, Isaacs ED, Teismann NA. Sonographic findings in necrotizing fasciitis: Two ends of the spectrum. J Ultrasound Med. 2016;35(10):2273-7.

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