

Calcific Uremic Arteriopathy (Calciophylaxis): Case report

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Introduction

The calcific uremic arteriopathy (CUA) is one of the several types of extra-osseous calcification that may occur in patients with end-stage renal disease (ESRD). CUA is a serious disorder characterized by calcification of the arterioles that leads to ischemia and subcutaneous necrosis. The pathogenesis is poorly understood, and the optimal treatment is not known.

Case report

We present the case of a 64-year-old female with end-stage renal disease on chronic hemodialysis since 2010 and with several secondary hyperparathyroidism. She is hypertensive, had a heart attack in 2006.

She was referred to our hospital with extensive 2 ulcers on the left leg with necrotic eschars.



Laboratory investigations showed elevated levels of phosphorus 2,46 mmol/l, total calcium 2,73 mmol/l, [Ca]x[P] 6,7 mmol²/l² and iPTH 2529 pg/ml (150-300 KDOQI).

Ultrasound revealed multiple enlarged parathyroid glands

Dual energy X-ray absorptiometry revealed osteopenia only in neck -1,2 SD; ,in total hip -0,6 SD, L1-4 -0,4 SD.

The clinical diagnosis was severe secondary hyperparathyroidism and Calcific Uremic Arteriopathy .

Considering the severity of CUA, was recommended a total parathyroidectomy. However, due to the patient's cardiovascular status, there were certain contraindications.

Treatment

Initially, we canceled the warfarin, recommended a hypophosphatic diet, analgesics, intravenous antibiotics, and assessment by surgeons for wound care.

After that:

1. she started intensive dialysis on a daily: three-hour-sessions with low calcium (1,25 mmol/L) dialysate for 2 weeks, later 3 times a week, followed by 24 mg of sodium thiosulfate intravenous administration at the end of every session.
2. She was also started on non-calcium and aluminium-based phosphate binders (sevelamer 2400 mg/day) and calcimimetics (cinacalcet 30-60 mg/day) for better control.

After 3 months:



Laboratory investigations were done during the treatment - phosphorus 2,14 mmol/l, total calcium 2,13 mmol/l, [Ca]x[P] 4,55 mmol²/l², iPTH 2086 pg/ml.

We added other phosphate binders (Aluminium-based) for a short period (during 1 months).

After 6 months later, the first CUA lesion was healed:



Laboratory investigations: phosphorus 1,7 mmol/l, total calcium 2,2 mmol/l, [Ca]x[P] 3,74 mmol²/l², iPTH 1690 pg/ml.

After year later the second CUA lesion was healed.

Conclusion

Our case study shows that only a multi-interventional strategy is likely to be more effective in treating CUA in patients on hemodialysis and with several secondary hyperparathyroidisms.

