Successful treatment of calciphylaxis and severe hypocalcemia after renal transplant

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**Background:** Calciphylaxis (or calcific uremic arteriolopathy) is a poorly understood syndrome of vascular calcifications in the skin, fat tissue, bursae, or joints, causing intense pain and necrosis due to calcium deposits in the medium sized arteries. These are mostly seen in ESRD patients who are on prolonged hemodialysis, also occur in rare renal transplant patients, patients with hyperparathyroidism, hypoparathyroidism receiving high dose calcitriol and calcium treatment, and malignancy. Known risk factors increasing the incidence include the use of calcium-based phosphate binders, steroid, chronic liver disease, and hypoalbuminaemia. The mortality associated with non-ulcerating type of calciphylaxis is approximately 30%. Here we present a case of non-ulcerating calciphylaxis in a patient with history of subtotal parathyroidectomy, 18 years of hemodialysis and recent renal transplantation, presenting with left wrist pain and intractable hypocalcemia and hyperphosphatemia, which resolved upon treatment with recombinant parathyroid hormone.

**Case description:** 53 year old male with no past medical history of joint disease, presented with severe left wrist pain with no overlying skin lesions. His past medical history is notable for chronic hepatitis and hypoalbuminaemia (2.6 mg/dl). He had parathyroidectomy 13 years ago for secondary hyperparathyroidism secondary to end stage renal disease, requiring high dose calcitriol (1.5 mcg twice daily), calcium (2 g three times a day), and sevelamer (800 mg three times a day) for maintenance that was ineffective in increasing PTH level in response to hypocalcemia. Prior to transplant with deceased kidney, he received hemodialysis for the past 17 years. Post-transplant treatment included prednisone 5 mg daily. Progressive hypocalcemia and hyperphosphatemia developed during the four weeks post-transplant, starting with serum calcium 6.9 mg/dl and serum phosphorous 4.7 mg/dl and escalating over next 2 weeks to serum calcium < 5 mg/dl (normal 8.4-10.2), serum phosphorus 9.4 mg/dl (normal 2.5-4.5), serum magnesium 1.9 mg/dl (normal 1.6-2.3), intact PTH 17.7 pg/ml (normal 12-65). Graft recovery was slow. X-rays revealed heterotopic soft tissue calcification at the right lateral upper thigh and extensive vascular calcifications along the ventral aspect of the wrist without any fluid collection or joint swelling. Wrist joint aspiration ruled out infection inflammation, or crystals. Treatment with recombinant PTH injections (rhPTH 1-34) 20 mcg subcutaneously twice a day quickly resolved the pain and normalized parameters of calcium metabolism, allowing to taper the dose of calcitriol and calcium and phosphate binder. Patient continues treatment with parathyroid hormone injections as outpatient.

**Conclusion:** Our case reflects on risk factors for calciphylaxis in setting of the rare combination of postsurgical hypoparathyroidism, prolonged hemodialysis, hepatitis C and hypoalbuminaemia, phosphate binder treatment, post-transplant steroid treatment and severe hypocalcemia. Treatment with replacement parathyroid hormone is addressing hyperphosphatemia in this setting, decreasing the precipitation of calcium phosphate product. The rapid resolution of severe pain is surprising.

**References:**