Hyperbaric oxygen therapy for calcific uremic arteriolopathy: a case series.

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Calcific uremic arteriolopathy (CUA), also referred to as calciphylaxis, is a syndrome of small vessel calcification of unknown etiology causing painful violaceous skin lesions that progress to non-healing ulcers and gangrene. It is observed mainly in patients with end-stage renal disease, is associated with high morbidity and mortality and has no standard treatment at the present time. Although parathyroidectomy (PTX) has been advocated in some cases, other studies have not found this effective. Hyperbaric oxygen therapy (HOT) consists of breathing 100% O2 at higher than ambient pressure, with the patient inside a sealed chamber. HOT has been used with some success in the treatment of selected problem wounds (those that fail to respond to established medical and surgical management). They are often severely hypoxic; restoration of tissue PO2 to normal or above-normal enhances fibroblast proliferation and collagen production as well as angiogenesis. The present is the largest retrospective case series of CUA treated by means of HOT reported so far and comprises 11 chronic uremic patients on dialysis (9 hemo- and 2 peritoneal dialysis, 6 females and 5 males, mean age 56 +/- 7 SD years, time on dialysis 163 +/- 84 SD months). Four patients had biopsy-proven CUA; 3 had diabetic nephropathy as a cause of uremia; 2 were obese and 3 had a consistent increase of serum calcium x phosphorus product; 3 patients had severe secondary hyperparathyroidism (II(nd) HPTH) and two had been submitted to subtotal PTX some years before CUA; two others had already had the limb amputated. Lesions were in the legs, except for one in a hand, and were prevalently ulcers and necrosis. The number of sessions in each HOT cycle ranged from a minimum of 20 to a maximum of 108 (mean 40.6 +/- 29.0). The results of two therapies cannot be evaluated (one was interrupted by the patient after 10 sessions, and one ended with the death of the patient due to ventricular arrhythmia after eight sessions). Eight of the nine remaining had excellent results with healing of the skin ulcers, but the ninth got worse, making it advisable to amputate the foot. In conclusion, CUA appears to result from a multitude of predisposing and/or sensitizing events that are commonly present in the uremic milieu. The specific factors that induce this disorder in an individual patient are not known. The present retrospective study supports a role of HOT in many cases of CUA, especially considering that, in the absence of severe II(nd) HPTH, there are very few therapeutic options.

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Related Links
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- Cutaneous necrosis from calcific uremic arteriolopathy. [Am J Kidney Dis. 1998]

Calcific uremic arteriolopathy: advances in pathogenesis and treatment. [Semin Dial. 2007] PMID:17374090