PRACTICE | CLINICAL IMAGES

Uremic tumoral calcinosis of the cervical spine

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65-year-old man with end-stage renal disease secondary to lupus presented to the emergency department with severe neck pain, progressive bilateral lower extremity weakness and upper motor neuron findings. Weakness had been progressive over the preceding 2 weeks and was quantified as American Spinal Cord Injury Association impairment scale C with a C6 root level. On physical examination, painless periarticular soft tissue masses were identified along the ulnar aspect of his left wrist and left first metatarsophalangeal joint. Corresponding multilobulated calcium depositions and vascular calcification were visible on radiography (Appendix 1, Supplementary Figure 1A and Figure 1B, available at www. cmaj.ca/lookup/doi/10.1503/cmaj.200104/tab-related-content).

Computed tomography (CT) of the cervical spine showed an erosive calcified mass at C5/C6 causing spinal stenosis (Figure 1A). Dual-energy C-spine CT ruled out tophaceous gout. Laboratory abnormalities included hyperparathyroidism and hyperphosphatemia (Appendix 2, available at www.cmaj.ca/lookup/doi/10.1503/ cmaj.200104/tab-related-content). Based on the laboratory abnormalities and radiographic findings of periarticular soft tissue calcifications with a negative dual-energy CT scan, we diagnosed uremic tumoral calcinosis. The spine surgery team proceeded with decompression and stabilization with a C5-C7 laminectomy and C4-T2 posterior instrumentation with no direct mass excision. We started medical treatment with sevelamer, pamidronate and 4 hours of hemodialysis with low-calcium dialysate 5 times per week. Five weeks later, our patient's neurologic status had improved, and he was ambulatory. A repeat CT of the cervical spine showed a large reduction of the calcified mass (Figure 1B).

Uremic tumoral calcinosis occurs in 0.5%–3% of patients with end-stage renal disease who are undergoing hemodialysis and is the result of metabolic bone disease.¹ Hyperparathyroidism and hyperphosphatemia contribute to ectopic periarticular deposition of calcium phosphate, although the mechanism is poorly understood.^{1,2} Vascular calcification and calciphylaxis can also occur in patients with metabolic bone disease from end-stage renal disease.³

Differential diagnosis includes gout, pseudogout, calcific tendinitis, synovial osteochondromatosis, sarcoma and myositis ossificans.^{1,2} As recurrence can occur after surgical resection, surgery should be generally reserved for cases in which medical management is unsuccessful or with progressive neurologic deficit.¹ Medical management focuses on reversal of the underlying electrolyte abnormalities and includes phosphate restriction, non-calciumcontaining phosphate binders and frequent hemodialysis with a low-calcium dialysate.^{1,2} Other adjuncts include calcimimetics, sodium thiosulfate, bisphosphonates and parathyroidectomy.^{1,2}

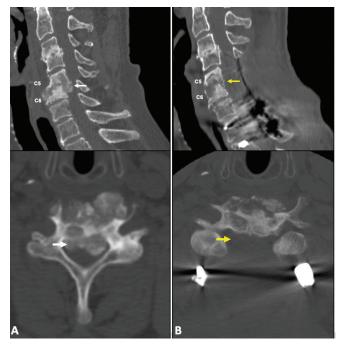


Figure 1: Computed tomography of the cervical spine in a 65-year-old man with end-stage renal disease. Midsagittal (top) and axial (bottom) views showing (A) multilobulated amorphous calcium deposition (white arrows), most notable at the C5/6 level and (B) the corresponding images 5 weeks later after cervical posterior decompression and instrumentation without mass excision, and medical management for uremic tumoral calcinosis. A substantial reduction in the burden of the periarticular calcific mass deposit is visible (yellow arrows).

References

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