STIR(ing) appearance of rhabdomyolysis

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A 30-year-old man was admitted unconscious to a peripheral hospital after consuming 120 mg of methadone to celebrate his release from prison. On arrival his creatinine was 1.93 mg/dl and his creatine kinase 68645 (IU/l). After reversal of his opiate intoxication, he began complaining of burning pain and weakness in both legs. Over the next 36 h his creatinine increased to 11.4 mg/dl with worsening metabolic acidosis, and he became anuric. He was transferred to our unit for dialysis. Due to persistent bilateral leg pain and a right-sided foot-drop an MRI (Figure 1) was performed, which demonstrated bilateral gluteal oedema (panel C, arrows) consistent with rhabdomyolysis, causing compression of the right sciatic nerve at the sciatic notch (panel D, arrow). He required seven dialysis sessions before his renal function began to recover, and when reviewed 1 month post-discharge his creatinine was 1.5 mg/dl and he was receiving physiotherapy for a persistent foot drop.

Rhabdomyolysis describes the breakdown of striated muscle fibres, which results in the release of intracellular contents [1]. Of the many compounds liberated, myoglobin in particular is implicated in the development of renal failure, through precipitation and obstruction of tubules and free radical generation [2].

MRI has become the imaging modality of choice to define the extent of muscle damage in rhabdomyolysis, especially when fasciotomies are considered [3]. Short tau-inversion-recovery (STIR) sequences provide excellent differentiation between affected (hyperintense signal) and unaffected tissues with greater sensitivity than other imaging techniques and without exposure to radiation.

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References


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Fig. 1. Coronal (A and C) and axial (B and D) short tau-inversion recovery MRI sequences from a normal individual on the left (A and B) and showing high signal intensity in the glutei bilaterally in the patient on the right (C and D).