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Severe Hypocalcemia after Renal Transplantation: An Extreme Case of Hungry Bone Syndrome

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Abstract **Case Report**

Hungry bone syndrome is a rare but potentially lethal complication that is characterized by rapid, severe, long-lasting hypocalcemia and hypophosphatemia secondary to increased bone metabolism. Hereby, we present a case of a 38-year-old patient who received a kidney transplant 09 months after a subtotal parathyroidectomy. She developed severe hypocalcemia and hypophosphatemia 15 days after renal transplant and required high-dose of calcium for 4 months thereafter. The aim of this case report is to highlight and raise awareness for the importance of strict nutritional and electrolyte management in the post transplantation period. A prompt diagnosis and correction of hungry bone syndrome are imperative to prevent the associated significant morbidity and mortality [1].

Keywords: Hungry bone syndrome; hypocalcemia; parathyroidectomy; renal transplantation.

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INTRODUCTION

The Hungry Bone Syndrome (HBS) is defined by persistent hypocalcemia for several days after the collapse of PTH levels, through an active bone remineralization process. It is a complication of parathyroidectomy performed in primary hyperparathyroidism, or autonomous in renal failure.

HBS can be observed minimally after renal transplantation [2]. We describe here an extreme case of HBS in the aftermath of renal transplantation.

CASE PRESENTATION

A 38-year-old female, chronic hemodialysis since 2008, who received a kidney transplant from a related living donor (her mother) 09 months after a subtotal parathyroidectomy (diffuse hyperplasia). On the day of transplantation, corrected Ca = 87 mg/l and PTH = 29 ng/ml (n = 15-65 pg/ml) with normal 25-OH D3 levels. The evolution was marked by an immediate recovery of the renal function with a creatinine level at 9mg/l at the tenth day

She consulted on day 15 of the transplantation for cramps, tremors, spasms of the face and the neck. the biological check-up finds : Corrected serum calcium level at 56 mg/L, inappropriate parathyroid hormone level = 35 pg/mL, transient hypophosphatemia was 18mg/L. Bone anabolism markers are discreetly elevated (bone alkaline phosphatase at 107mg/l).

The correction of hypocalcemia had required intravenous calcium combined with high doses of oral calcium (12g per day) for more than a month. Calcium was stopped progressively and suspended four months after renal transplantation.

DISCUSSION/CONCLUSION

The conjunction over time of two etiologies of HBS, parathyroidectomy and renal transplantation, resulted in profound and symptomatic hypocalcemia requiring massive doses of IV calcium in addition to prolonged oral calcium and active vitamin D intakes. Close monitoring of serum calcium and calciuria should be performed following renal transplantation after a recent parathyroidectomy [3].

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