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Cisplatin Induced Hypomagnesemic Hypocalcemia-A Case Report and a Review of the Pathophysiology

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Abstract

We present a case of a 62 year old man with history of small cell lung cancer status post radiation and chemotherapy with cisplatin and etoposide who presented to the emergency room with complaints of generalized repetitive muscle contractions. His physical examination was notable for involuntary rhythmic muscle contractions of face, neck, upper arms, thigh and hyperreflexia. Laboratory data was significant for calcium 4.6 mg/dl (corrected 5.64 mg/dl), albumin 2.7 g/dl, and magnesium 0.7 mg/dl. He was treated aggressively with intravenous magnesium and calcium with resolution of symptoms. Hypomagnesemia with subsequent hypocalcemia is a common side effect of cisplatin therapy, an understanding of its pathophysiology is imperative for clinician.

Case Presentation

We present a case of a 62 year old man with history of smoking, COPD, coronary artery disease, small cell lung cancer status post radiation and chemotherapy who presented to the emergency room with complaints of generalized repetitive muscle contractions. He was three days out from his fourth cycle with cisplatin, etoposide and decadron. Patient reported that symptoms stated slowly, prominent on his checks, upper arms and thigh and progressed to the point where he could barely hold a cup of water or ambulate freely. Review of systems was positive for increasing dyspnea on exertion but negative paroxysmal nocturnal dyspnea, orthopnea and lower extremity edema. He denied palpitations, chest pain, syncope, numbness or paresthesias. He was not taking any diuretics, glucocorticoids or calcium supplementation at presentation. His physical examination was notable for involuntary rhythmic muscle contractions of face, neck, upper arms, thigh and hyperreflexia. Laboratory data was significant for calcium 4.6 mg/dl (corrected 5.64 mg/dl) (pre-chemotherapy serum calcium 8.8 mg/dl), albumin 2.7 g/dl, magnesium 0.7 mg/dl (pre-chemotherapy serum magnesium 2.4 mg/dl), creatinine 1.2 mg/dl, hemoglobin 8.2 g/dl, hematocrit 23.3% and platelet 83 k/UL. Serum PTH and urinary cAMP was not checked. EKG showed sinus rhythm with rate of 89, prolonged QTc 503 (pre-chemotherapy QTc 449) and chest X-ray without infiltrates but showed right upper lobe mass. Patient was admitted to a cardiac monitored floor and treated with intravenous magnesium sulfate and calcium gluconate. Serum calcium and magnesium was checked every six hours with repletion therapy tailored towards resolution of symptoms. Initially, calcium gluconate three (3 g) grams was given intravenously over 10 minutes, followed by a continuous infusion at a rate of 2 mg/kg/hr. Magnesium sulfate five (5 g) grams in 500 cc of normal saline was given over 5 hours and repeated twice daily until normal serum magnesium value. It was initially difficult to maintain normal levels of serum magnesium. He received a total of twenty-five grams of intravenous magnesium and was subsequently transitioned to oral magnesium oxide. Patient was given a total of eighteen (18 g) gram of calcium gluconate intravenously. There was complete resolution of his symptoms by hospital day three. He was discharged with oral magnesium, calcium and vitamin D3 (cholecalciferol) supplementation.

Discussion

Hypomagnesemia is a common side effect of cisplatin occurring in 90% of patients by the fourth cycle of treatment caused by renal tubular damage leading to increased renal wasting of magnesium if no corrective measures are taken [1]. Cisplatin causes damage to calcium and magnesium sensing receptors at level of thick ascending loop of henle and distal convoluted tubules during the early phases and in the later phase, causes patchy tubular cell necrosis which leads to renal magnesium wasting [1]. The subsequent hypocalcemia is believed to be secondary to hypomagnesemia induced functional hypoparathyroidism and Parathyroid Hormone (PTH) resistance [2]. The release of PTH from the parathyroid gland and the end –organ effect of PTH is based on magnesium dependant Cyclic Adenosine Monophosphate (C-AMP) generation which is defective in severe hypomagnesemia [3]. This leads to decreased calcium reabsorption from the kidneys, decreased calcium release from bones, decreased vitamin D production leading to decreased calcium absorption from the gut. Understanding the proposed mechanism is crucial in the management of severe symptomatic hypocalcemia associated with hypomagnesemia. Initial focus should be placed in preventing hypomagnesemia associated hypocalcemia in patients treated with cisplatin with prehydration and posthydration intravenous magnesium or oral magnesium supplementation or a combination.

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