CORRECTION OF REFRACTORY HYPOKALEMIC HYPOMAGNESEMIA BY INITIATING HEMODIALYSIS

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Background: Magnesium is the second most abundant intracellular cation and the fourth most abundant cation in the body. it plays many physiological role achieved through two important properties including the ability to form chelates by ligation to intracellular anions and the ability to compete with calcium for binding sites on proteins and membranes. Thus, magnesium is essential for the synthesis of nucleic acids and proteins. It's required for metabolism, neurotransmission, hormone receptor binding and post receptor activation. Renal handling of magnesium, like other electrolyte, is adaptable unless creatinine clearance declines to less than 20 ml/min. Hypomagnesaemia is mostly observed in hospitalized patient leading to various clinical conditions, refractory electrolyte deficiency and hormonal imbalance. Our case illustrates the importance of correcting hypomagnesaemia.

Case presentation: This 50 year old man is a known case of two sibling nephronophtisis who has been transplanted three times (1 live and 2 cadavers). He was referred to nephrology clinic for decreased appetite and weakness. His interrogatory is significant for chronic renal rejection, three episodes of thrombophlebitis, eventration , rapamucin induced bronchiectasis, destabilizing polyneuropathy and growth retardation. He admits to smoking one pack of cigarettes a week for the past 10 years and he denies alcohol or drug use. He denies use any medication out of the counter apart of his regular medication which includes cyclosporine , prednisolone ,furosemide 60 mg, atorvastatin , trimethoprim/sufamethoxazol , mycophenolate mofetl , 2 capsules of potassium chloride TID, pantoprazole, calcium carbonate and high dose pregabalin. Physical examination reveals an alert and oriented patient , having normal vital signs. The rest of physical examination is not contributory. Laboratory investigations show a decline in his creatinine clearance estimated by MDRD clearance of 12 ml/min with upper limit potassium level , mild inflammatory signs and anemia as confirmed by HB level =103g/dl , VGM = 85.5 fl and ferritin level = 343 ug/ml and normal phosphor –calcic product corrected to hypoalbuminemia. However, there is a dramatic low level of magnesium at 0.12mmol/l (0.7 – 1) and erythrocyte magnesium level at 2.10mmol/l (2.21-3.51) with normal TSH and intact PTH. Blood gas parameters , HBA1C , B12 , folic acid , homocysteine , zinc , aluminum and serology profile were all negative. Plasma protein electrophoresis is inflammatory as confirmed by the level of inflammatory profile. Prealbumine level = 0.19 g/l (0.2-0.4). Electrocardiogram tracing shows no abnormalities . Patient convinced to start twice weekly hemodialysis under a dialysate bath concentration of Na 103 , K 3 ? Mg 0.75 mmol:l , calcium 1.5 mmol:l chloride 110 mmol:l and glucose 1 g/l. Magnesium level regains

its normal level after quitting cyclosporine and hemodialysis twice weekly for six months. His potassium intake decreases gradually after the correction of hypomagnesaemia. His nutritional status is markedly improving gaining weight of twenty kilograms. Erythrocyte content of magnesium shows gradual increment faster than the normalization of blood level. After one year from the start of renal replacement magnesium level was at 0.5 mmol/l for erythrocyte of normal value at 2.72 mmol/l. Moreover, serum magnesium level normalized by the second year of hemodialysis.

Discussion: severe hypomagnesaemia is well known to be associated with clinical neuromuscular disorders, electrocardiographic abnormalities and resistant electrolyte homoeostasis. The sole observed persisting abnormality observed in our case was chronic hypokalemia mandating an increase intake of potassium despite clinical, histological feature of chronic renal graft rejection eventually leading to renal replacement. The cyclosporine treated transplanted patients usually exhibit hypomagnesaemia requiring magnesium supplementation. The mechanism by which cyclosporine induces hypomagnesaemia seems to be related to renal wasting of this cation. Unfortunately, we don't know either it is cause of cyclosporine toxicity or a consequence of chronic cyclosporine nephropathy. In our patient, cyclosporine level was at the lower limit of the therapeutic range for renal transplanted patient and histological study didn't show the feature of cyclosporine nephropathy. Though serum magnesium does not reflect well the total body magnesium. Initiation of hemodialysis and the discontinuation of cyclosporine provide a prompt improvement of serum level of magnesium while other drugs were maintained. We didn't observe any variation of intact parathyroid hormone while replacing hypomagnesaemia that is well known to exacerbate secondary hyperparathyroidism. This event was probably avoided in the presence of residual renal function and combined cholecaceferol /calcium intake.

Conclusion: correction of hypomagnesaemia by hemodialysis allows better control of recurrent hypokalemia. The presence of resistant electrolyte deficiency should alert the physician to check underlying hypomagnesaemia.