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CASE REPORT

HYPERKALEMIA CAUSING SEVERE MUSCLE WEAKNESS IN A PATIENT WITH RENAL INSUFFICIENCY

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ABSTRACT

In individuals with renal insufficiency, hyperkalaemia is one of the most common electrolyte imbalances, whereas it is uncommon in healthy people. When potassium is given or combined with a potassium-sparing diuretic, it happens quickly. It usually does not produce any signs and symptoms and is identified with normal blood investigations. Hyperkalemia causes faulty heart conduction and muscle weakness, among other symptoms. Muscle weakness as a clinical manifestation, on the other hand, is infrequent in clinical practise. This could be due to the fact that cardiac symptoms frequently appear earlier than weakness of muscles, necessitating the implementation of suitable interventions even before potassium concentration reaches a level that causes weakness. We present a case in which a patient with renal insufficiency had acute weakness of muscles as a result of extreme hyperkalaemia that acquired fast recovery after potassium and potassium-sparing diuretic administration.

KEY WORDS: Hyperkalemia; Muscle Weakness; Paralysis: Renal Insufficiency; Potassium.

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CASE REPORT

In the end of year 2019, a 51-year-old woman received a cadaveric kidney transplant, but as a consequence developed acute tubular necrosis. She was subsequently kept on haemodialysis for the initial couple of months after the transplant, after which she was slowly weaned off and her serum creatinine levels steadied at 3 mg/dL around the beginning of year 2020. Subsequently, he underwent periodic medical check-ups. She had a small spike in her creatinine to 3.7 mg/dL at her routine check-up in March 2020, 20 weeks after her transplant, from her usual normalized values. After an abdominal scan the transplanted kidney showed blockage at the uretero-vesical junction. She was hospitalized for re-constructive surgery, which was worsened by hypo-tension during the procedure. She had reduced urine output after the operation for which she was started on diuretics. Because of the increased dosages of furosemide, she had azotaemia and her potassium levels appeared to be low. To sustain diuresis and attain normal potassium values, aldactone was ad-ministered. Nonetheless, potassium levels remained low. After that the patient had paralytic ileus, which could have been caused by low serum potassium values. The patient was asked not to take any food or water and IV fluids were started with potassium reinforcement (80 mEq/L per day) for the treatment of paralytic ileus. On the subsequent day, the potas-sium values were corrected to 4.6 mEq/L. Potassium supplementation was lowered. Her serum potassium values were elevated to 6.6 mEq/L the very next day, therefore the potassium complement was removed and the aldactone was kept. She began to have weakness in both the legs later in the day, which proceeded to the upper extremities, trunk, and neck muscles by night time. She was afebrile, attentive and completely aware on physical examination. Her cra-nial nerves had not been damaged. Both legs had a power of 0/5, while both arms had a power of 1/5. Her plantar reflexes were flexors since she had areflexia. The potassium level was 9.4 mEq/L when a second blood sample for potassium was taken. A sine wave pattern was visible on the ECG. She was given 40 mL of 10% calcium gluconate on IV and transferred

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to hemodialysis right away. Following half an hour of dialysis, the patient was able to move her hands, and she progressively improved.

DISCUSSION

In individuals with renal impairment, symptoms and indicators of hyperkalaemia vary according to the severity of the condition. Hyperkalemia causes a variety of abnormalities, including faulty cardiac conduction and muscle aches, which can result in potentially fatal arrhythmias and paralysis. Even though both cardiac and neurological problems have been reported, cardiac symptoms are more common in clinical practise. Tall T waves, expanded QRS, prolonged PR interval, and disappearance of P wave on an electrocardiogram might give a rough notion of the extent of hyperkalaemia.[1,2] These modifications could quickly lead to deadly arrhythmias. Thankfully, this did not occur with our case.

Neurological symptoms are frequently under-reported. Alterations in neuromuscular conduction seem to be the cause of muscle weakness linked with hyperkalemia. The ratio of intracellular K+ concentration to that in the ECF falls when plasma K+ level elevates, causing a drop in the amplitude of resting membrane potential. However this would enhance membrane excitability (since an action potential requires a depolarizing stimulation), the impact seen in individuals is distinct. Consistent depolarization inhibits sodium channels in the cell membrane, leading to a net reduction in membrane excitability and muscular weakening or paralysis.

Weakness of muscle normally does not appear until the plasma potassium levels reaches 9 mEq/L. Patients with periodic paralysis, on the other hand, might develop symptoms at plasma concentrations as low as 5.6 mEq/L, owing to the fact that aberrant membrane activity is the major problem in this condition.

The clinical manifestation of hyperkalemia is vague muscle soreness. Weakness of muscles follow, with the feet being the first to be affected, followed by the trunk and arms, and finally the facial and respiratory muscles.[3,4] Despite muscle paralysis, the patient is normally awake till a cardiovascular incident happens. After getting the potassium level back on track, these modifications reverse, which was the precise chain of actions in our case.

CONCLUSIONS

The current example demonstrates that hyperkalemia should be thought a potential cause of paralysis in all incidences of paralysis. Potassium supplementation and potassium-sparing diuretics can produce hyperkalemia, especially in people with kidney impairment. Not only does prompt diagnosis and treatment totally recover paralysis, but it also prevents catastrophic heart arrhythmias.

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CONFLICT OF INTEREST
Authors declare no conflict of interest.
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Conception or Design:	PHA, NA
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All the authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.