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A Rare Cause of Acute Muscular Paralysis.

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ABSTRACT

A 65 years old woman presented with acute flaccid paralysis, on investigations found to have gross hyperkalemia; prompt correction of this metabolic disturbance totally resolved the weakness. Electrolyte imbalances tend to occur in patient for various reasons ranging from the common diarrhea and vomiting to severe diseases like renal failure. They cause different symptoms including weakness of the muscles. It is common that hypokalemia causes muscle weakness. We present a rare case of unusual electrolyte disturbance causing muscle weakness.

Keywords: muscular paralysis, hyperkalemia, renal failure.

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Case presentation

65 years old Mrs. J, a known hypertensive and a diabetic for the past 10 years. She was on insulin and oral hypoglycemic agents. She presented with weakness of upper and lower limbs of two days duration. Earlier in the week she took oral NSAIDS for severe knee pain.

On examination she was tachypnoeic. Her pulse was 82 per minute and her blood pressure was 220/ 120 . Her capillary oxygen saturation read by a pulse-oximenter was 97%. There was flaccid weakness of both legs and arms.The power of her proximal muscle group of upper limbs and lower limbs was Medical Research Council (MRC) 2/5 while it was MRC 4/5 in the distal muscle groups of both upper and lower limbs. She had sensory disturbance of the lower limbs consistent with diabetic peripheral neuropathy. Her plantar reflex was normal. Examination of her cardiovascular system, respiratory system and abdomen was unimpressive.

She had lab investigations. Her haemoglobin was 12.2 gms %, her liver function tests were normal; her Troponin- T was negative and her plasma acetone was negative. In her blood gas analysis the oxygen Po2/ F1O2 4-22mmhg% . Her presenting blood urea was 96mg /dl and her random blood sugar level on admission was 262mg/dl (Hb A1 c was 9.1%) her Creatinine Phosphokinase Kinase was- 298u/l . her presenting potassium and sodium was 7.46 mEq/L and 136 mEq/L respectively .Also her Thyroid profile was normal. Her urine was reported negative for Myoglobin.

Imaging was done and her CT Brain was normal except for age related atrophy; the ultrasonogram of the abdomen-was normal. Her Electro Cardio Graph showed a wide QRS and tall T waves as seen in figure 1. Her Echocardiograph showed a normal left ventricular function and a mild left ventricular hypertrophy . The nerve conduction study done(figure 2) showed that the nerves of the lower limb could not be stimulatable. i.e. in both lower limbs there was no stimulation possible in motor and sensory nerves. The CMAP of both side Median nerves were prolonged and their conduction velocity was reduced (figure 3). There was a proximal amplitude drop. There was also evidence of demyelination in both side median nerves. Sensory nerve action potential amplitude was slightly reduced in both side median and ulnar nerve.

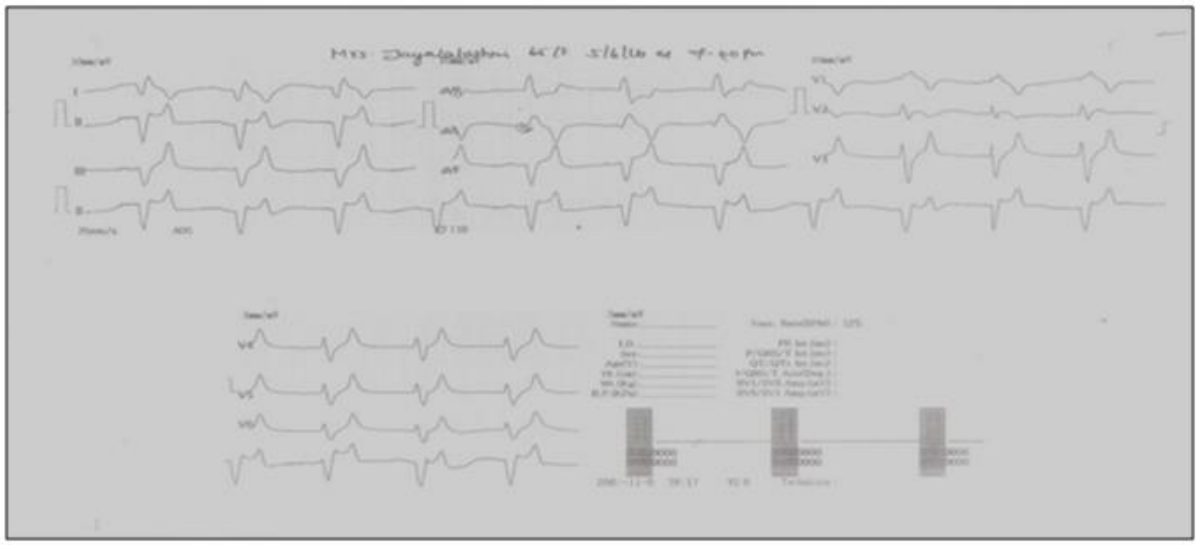

Fig 1: ECG showing tall T waves suggestive of hyperkalemia

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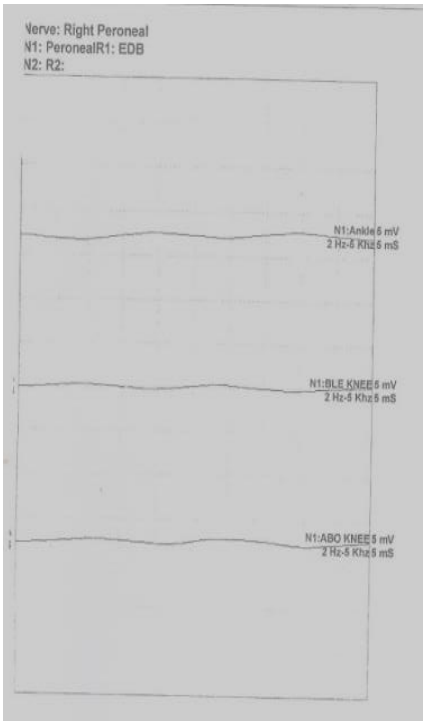

Fig 2: Nerve conduction of peroneal nerve on the right side

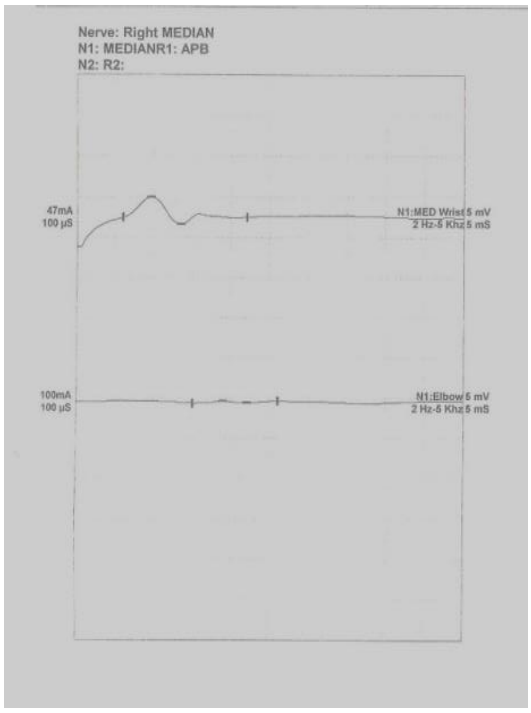

Fig 3: Nerve conduction of median nerve on the right side

Diagnosis

The problems identified in this lady are hyperkalemic paralysis with weakness of upper limbs and lower limbs, hypertension, diabetes mellitus, mild renal failure and diabetic peripheral neuropathy; Acute inflammatory demyelinating polyradiculoneuropathy was ruled out. The patient was started on insulin infusion, albuterol nebulization, K-bind sachet, antibiotics, anti hypertensives, and supportive measures. Haemodialysis through a temporary intravenous access was done in three sittings. With this treatment her

serum potassium (measured daily) gradually came down as represented in figure 4 and there was no gross change in the sodium levels. After her treatment her blood urea was reduced to 68mg /dl and her fasting blood sugar level after treatment was controlled and was 147 mgs /dl. Her power of the lower limbs improved. She was discharged and walked back home.

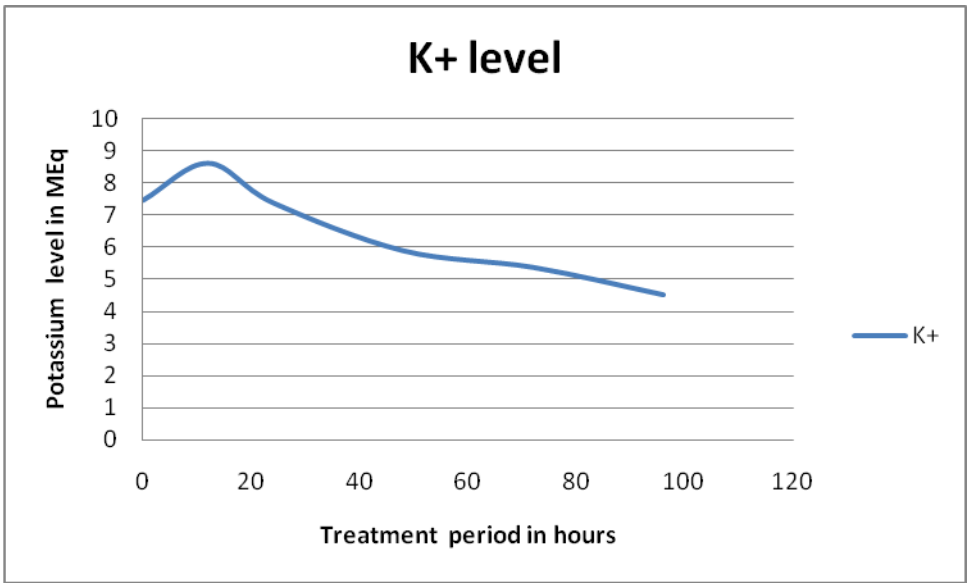

Fig 4: The levels of potassium as measured over the period of days of treatment with dialysis for this patient

DISCUSSION

Hyperkalemia is defined as plasma potassium more than 5.5 m Eq per liter. It occurs in 10% of hospitalized patients. Severe hyperkalemia occurs in 1% and it increases the mortality. Redistribution and reduced uptake can acutely cause hyperkalemia. Reduced Renal Potassium excretion is the most common underlying cause. Drugs that have an impact on Renin - angiotensin aldosterone axis are also a major cause of hyperkalemia. Hypokalemic paralysis is commonly encountered in clinical practice. [1] Hyperkalemic paralysis is a rare presentation. [2] Severe hyperkalemic may be asymptomatic and may predispose to sudden death from asystolic cardiac arrest. Serum potassium > 7 mmol/l is a medical emergency and is associated with ECG changes. Muscle weakness is often the only symptom unless it is associated with metabolic acidosis. Peritoneal dialysis brings down the potassium very slowly.

Our patient had been taking NSAIDS often for her knee pain. This could have precipitated the problem of hyperkalemia in a patient with diabetic kidney disease. In this case haemodialysis is the line of treatment as it will rapidly reduce the serum potassium level.

Muscle weakness in hyper-kalemia affects proximal muscle groups while the bulbar muscles are usually spared. Nerve conduction studies usually shows reduced motor amplitudes. The EMG may be silent in weak muscles. Hyper-kalemia may cause diastolic cardiac arrest. Hence this case was initially stabilized with calcium, insulin, albuterol and then taken for haemodialysis. Patient showed dramatic improvement in metabolic profile which coincided with neurological improvement.

REFERENCES

[1] Vicart S, Sternberg D, Arzel-Hézode M, et al. Hypokalemic Periodic Paralysis. 2002 Apr 30 [Updated 2014 Jul 31]. In: Pagon RA, Adam MP, Ardinger HH, et al., editors. GeneReviews® [Internet]. Seattle (WA): University of Washington, Seattle; 1993-2016. Available from: https://www.ncbi.nlm.nih.gov/books/NBK1338/

[2] Karin Jurkat-Rott, Frank Lehmann-Horn Genotype-Phenotype correlation and therapeutic rationale in hyperkalemic periodic paralysisNeurotherapeutics April 2007, Volume 4, Issue 2, pp 216–224 http://www.ncbi.nlm.nih.gov/books/NBK1338/