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RESEARCH ARTICLE

KANAMYCIN INDUCED CARPOPEDAL SPASM IN A PATIENT WITH MULTIDRUG RESISTANT PULMONARY TUBERCULOSIS

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ABSTRACT

Kanamycin is one of the injectable second line anti-tubercular drug used in multidrug resistant tuberculosis. It caused renal electrolyte wasting causing dyselectrolytemia in our patient presenting with carpopedal spasm `which resolved after discontinuation of kanamycin.

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INTRODUCTION

We report a case of patient with Multidrug resistant (MDR) Tuberculosis , on treatment with an injectable aminoglycoside Kanamycin. It caused renal electrolyte wasting including hypocalcemia in our patient leading to carpopedal spasm. On discontinuation of kanamycin symptoms resolved and another second line anti-tubercular drug was added to her regimen. Case report: Nineteen years old female was diagnosed with pulmonary tuberculosis in july 2021. She was on intensive phase of anti-tubercular treatment as per RNTCP (Revised national tuberculosis control programme) guidelines for 1 month. Repeat sputum smear was done in view of no resolution of symptoms and persistent radiological findings on chest xray. It was again positive for acid fast bacilli. It raised a suspicion about drug resistance. Drug sensitivity testing was performed which revealed resistance to isoniazid and rifampicin. As per recent RNTCP guidelines she was labelled as multidrug resistant tuberculosis and was started on second line drugs including Tab. Pyrazinamide 700 milligram (mg) orally twice a day, Tab. Levofloxacin 750mg orally once a day, Inj. Kanamycin (An injectable aminoglycoside) 1gm intravenously once a day. She continued this new regimen for two months. After which she developed sudden onset of bilateral upper limb carpopedal spasm and muscle cramps in lower limbs. She visited the casualty of our hospital for the same. After careful evaluation she was admitted to the ICU (Intensive care unit) as a case of tetany. All her routine blood investigations were done including complete blood count, liver function tests, renal function tests, serum electrolytes. Apart from some electrolyte imbalance no other significant abnormality was found. Among dyselectrolytemia there was hypocalcemia, hypomagnesemia and hypokalemia. She was given Inj. Calcium gluconate 10 ml i.v. , inj. Magnesium surface 1gm i.v. And inj. Potassium chloride 40 milliequivalents (meq) . Investigations were repeated after 24 hours and it showed normal serum calcium, magnesium and potassium levels. Patient was discharged home on oral calcium supplementation. Two weeks later patient presented with similar complaints in casualty and was admitted in ICU again for evaluation. After initial stabilisation with injectable calcium and magnesium, Kanamycin was considered to be the causative agent after exclusion of all other causes of electrolyte imbalance.

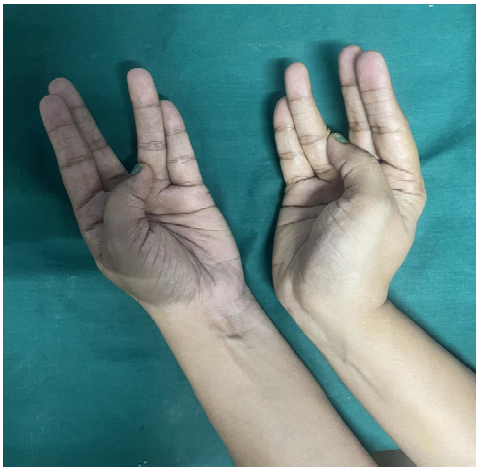
Tables

Electrolyte (Values in milliequivalents	Baseline 23/07/21	first admission 08/10/21	24 hrs after first admission 09/10/21	second admission 20/10/21	48hrs after discontinuation of kanamycin 23/10/21
Na+	138	136	139	138	140
K+	4.2	3.6	3.9	3.7	4.1
Cl-	98	94	99		99
Mg (mg/dl)	1.8	1.5	2.1	1.6	1.9
Ca+2	9.2	7.3	9.0	7.6	9.2

It was discontinued and other second line anti-tubercular drug was added. Patient is now on follow up and symptoms did not recur thereafter.

DISCUSSION

Carpopedal spasm usually seen in cases of tetany is caused by low ionized serum calcium concentration. This results in increased excitability of peripheral nerves, which later perpetuates in forced involuntary contraction of hands affecting wrist, carpometacarpal, distal phalangeal joints. Sometimes in extreme cases may even lead to convulsion, stridor. The total serum calcium of value less than 8.5 mg/dL may be associated with tetany. We report a case of a 19 years old girl with multidrug resistant pulmonary tuberculosis, who was being treated with kanamycin as a part of her treatment for two months. She presented with carpopedal spasm twice in our hospital over a span of two weeks. Her investigations showed hypocalcaemia, hypomagnesemia and hypokalemia on both occasions. On detailed evaluation and literature review it was concluded that an aminoglycoside drug such as kanamycin can cause renal electrolyte wasting including hypocalcaemia which in turn may cause carpopedal spasm. A dose dependent nephrotoxicity is seen with aminoglycoside therapy. It can cause renal electrolyte wasting which is characterised by symptomatic hypocalcemia, hypomagnesemia and hypokalemia. Several risk factors such as volume depletion, sepsis, liver and renal dysfunction, hypokalemia, hypomagnesemia, advanced age, prolonged therapy, type of aminoglycoside, time and frequency of dosing, an elevated serum aminoglycoside concentration, and interactions with other nephrotoxic drugs have been identified for the same. This patient's laboratory findings of serum electrolytes, urea, creatinine, albumin prior to administration of kanamycin were within normal range. Other causes of hypocalcemia like vitamin D deficiency, chronic renal failure, hypoparathyroidism were ruled out on admission. Therefore kanamycin was probably the drug responsible for this rare adverse effect in this case.



Approximately 8-26% of patients who receive an aminoglycoside for several days develop mild reversible renal impairment. The toxicity results from accumulation and retention of aminoglycoside in the proximal tubular cells causing excretion of enzymes of the renal tubular brush border. After several days, there is a defect in renal concentrating ability, mild proteinuria, and the appearance of hyaline and granular casts. The glomerular filtration rate is reduced after several days. The non-oliguric phase of renal insufficiency is thought to be due to the effects of aminoglycosides on the distal portion of the nephron with a reduced sensitivity of the collecting-duct epithelium to endogenous antidiuretic hormone. Although severe acute tubular necrosis may occur rarely. The biochemical events leading to tubular cell damage and glomerular dysfunction involve perturbations of the structure of cellular membranes, inhibition of various phospholipases, sphingomyelinases, and ATPases and they alter the function of mitochondria and ribosomes. Because of the ability of cationic aminoglycosides to interact with anionic phospholipids, these drugs may impair the synthesis of membrane-derived autacoids and intracellular second messengers such as prostaglandins, inositol phosphates, and diacylglycerol. Toxicity correlates with the total amount of drug administered, longer course of therapy.

Continuous infusion is more nephrotoxic than intermittent dosing. High-dose, extended-interval dosing approaches lead to less nephrotoxicity at the same level of total drug exposure than divided-dose approaches.The nephrotoxic potential varies among individual aminoglycosides. Neomycin being highly nephrotoxic and streptomycin being the least. Kanamycin being Intermediate on nephrotoxicity .(causality assessment using Naranjo's Adverse Drug Reaction probability score = score 7).

Conclusion

Kanamycin can have reversible adverse effects on both proximal and distal tubular function, which can lead to renal electrolyte wasting causing significant electrolyte imbalance. Toxicity of kanamycin should be considered in any patient with carpopedal spasm or multiple electrolyte imbalance.

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