A Case of Intra Muscular Sodium Stibogluconate Induced Hypokalemia

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ABSTRACT

Introduction: Cutaneous leishmaniasis has been detected in the Kashmir valley since the last decade[a]. The pentavalent antimonials meglumine antimoniate and sodium stibogluconate are the gold standard of treatment.

Case Report: A case of cutaneous leishmaniasis in a native Kashmiri is presented.He was put on intravenous sodium stibogluconate for the same. Eight days into therapy he developed recalcitrant hypokalemia not responding to potassium supplementation till discontinuation of therapy. In addition, he also developed new onset cardiac arrhythmias.

Conclusion: Sodium stibogluconate can cause refractory hypokalemia.

Keywords: Intra Muscular Sodium, Stibogluconate, Hypokalemia

INTRODUCTION

Cutaneous leishmaniasis has been detected in the Kashmir valley since the last decade.[1] The pentavalent antimonials meglumine antimoniate and sodium stibogluconate are the gold standard of treatment. The common side effects are nausea, pancreatitis and myalgias. If localized it can be treated by intralesional sodium stibogluconate. However, for disseminated disease, systemic therapy is recommended.[1-3]

Sodium stibogluconate is administered in intramuscular form with close monitoring of ECG recommended. Known side effects are cardiac arrhythmias. We report a case of sodium stibogluconate induced hypokalemia.

CASE REPORT

A seventy five year old Kashmiri Gujjar, known hypertensive, nondiabetic, euthyroid, non smoker presented with a five years history of nonhealing ulcer right cheek. He had been evaluated two years back and been prescribed ATT for six months. However, no response was seen.

On reevaluation, in view of his residence (the patient belonged to the Leishmaniasis belt of Kashmir Uri[1]), a possibility of cutaneous leishmaniasis was thought of.

A skin biopsy from the lesion revealed granulomas with a neutrophilic infiltrate. Slit skin smear of the lesion revealed amastigote LD bodies twice on Giemsa staining. In view of the clinical picture and supportive lab findings a diagnosis of cutaneous leishmaniasis was made.

A decision to put the patient on intramuscular sodium stibogluconate was made. Hemogram, liver and renal biochemistry profile was done. On the basis of his body weight, 850mg sodium stibogluconate intramuscularly was started.

In view of his age and hypertensive condition cardiology clearance was sought prior to start of treatment. A first degree heart block and left ventricular hypertrophy was seen on baseline investigations. After cardiology clearance with an advice to monitor electrolytes and ECG daily, im sodium stibogluconate was started after in hospital admission. The patient was doing well clinically as well as lab wise with normal parameters till the morning of the eighth injection. On the morning of the ninth dose, tests revealed falling potassium levels. Also, the ECG revealed ventricular premature contractions and atrial flutter. Medical opinion was sought. Potassium chloride oral supplementation was started. This failed to elevate the serum levels of potassium. The patient was referred to nephrology for resistant hypokalemis. Renal parameters were normal.

In view of new onset hypokalemia after a drug was started and no comorbid conditions explaining this the nephrologist advised to stop Sodium stibogluconate. Potassium supplementation was continued. The level of potassium returned to normal within one and a half week after discontinuation of the drug.

Lack of preexisting electrolyte imbalance, presence of a reasonable temporal relationship, lack of any other cause and complete resolution on discontinuation are in favour of sodium stibogluconate induced hypokalemia. Re-challenge was not done as patient refused it. The causality assessment score was found to be 4 as per Naranjo probability scale[2] and the severity assessed as possible ADR.

DISCUSSION

Pentavalent antimonials like meglumine antimoniate and sodium stibogluconate are the gold standard of treatment of Leishmaniasis. The common side effects include nausea, pancreatitis, and myalgias. Reversible ECG changes are

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Analyte↓	Baseline before starting sodium stibogluconate	On 9th day of therapy	Stopped the drug	3rd day after stopping	4rd day after stopping (morning)	4th day after stopping (evening)	5th day of stopping	10th day after stopping
pH	7.38	7.43	-	7.47	7.45	7.40	7.39	7.31
Na	143	142	-	141	140	142	144	140
K	3.5	2.8 oral potassium suppl started	-	2.5	2.5	2.6	2.6	4.1
Ca	1.01	1.02	-	0.88	0.88	0.79	0.99	1.1
HCO3	29	30.1	-	24	25.7	27.4	28.5	28.2

Table-1: Serial Blood gas analysis & elevtrolytes

seen in 30-60% of the cases.[3,4] These may occur without evidence of myocardial damage. Hypokalemia is defined as serum potassium of less than 3.5 meq/l. Mildly low levels may cause tiredness, leg cramps, weakness and constipation. At more severe drop it is a potentially life threatening condition in view of its cardiac risks. Severe hypokalemia with potassium levels less than 2.5 can cause bradycardia or cardiac arrest. Causes include starvation, diarrhea, steroids, furesamide, dialysis, hyperaldosteronism, hypomagnesemia and diabetic ketoacidosis. Sodium stibogluconate induced torsades de pointes has also been reported as cause of sudden unexplained death presumably due to ventricular arrhythmias and QT interval prolongation.[5] A case of polymorphic ventricular tachycardia has been reported.[6] Only three cases of stibogluconate induced hypokalemia have been seen in world literature.[7,8]

Lack of preexisting electrolyte imbalance, presence of a reasonable temporal relationship, lack of any other cause and complete resolution on discontinuation are in favour of stibogluconate induced hypokalemia. Rechallenge was not done as patient refused it. The causality assessment was found to be 4 as per Naranjo probability scale and the severity assessed as possible ADR.

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