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Case Report

A CASE OF BARTTER SYNDROME-ADULT VARIANT

Sasi Sekhar T.V.D., *Sravya K., M. Indrakleela Girish, Jagadeeshwara Rao
Department of General Medicine, Dr. Pinnamaneni Siddhartha institute of Medical Sciences, Chinoutpalli, Gannavaram, Vijayawada
**Author for Correspondence*

ABSTRACT
Bartter syndrome is a rare disease which presents in childhood which is characterized by hypokalemia, metabolic alkalosis, hyperaldosteronism and normal blood pressure. We report a case of 42year old male who presented with quadriparesis and weakness of neck. Investigations were suggestive of Bartter syndrome. We report this case because adult variant Bartter is rare.

***Keywords*:** *Bartter Syndrome, Hypokalemia, Metabolic Alkalosis, Hyperaldosteronism, Hyperreninemia*

INTRODUCTION
Bartter syndrome, originally described by Bartter and colleagues in 1962, represents a set of closely related autosomal recessive renal tubular disorders characterized by hypokalemia, hypochloremia, metabolic alkalosis and hyperreninemia with normal blood pressure[1 3]. The underlying renal abnormality results in excessive urinary losses of sodium, chloride and potassium. Bartter syndrome has traditionally been classified into 2 main clinical variants: neonatal (or antenatal) Bartter syndrome, classic Bartter syndrome[1].

CASES
A 40 year old male presented with history of weakness of all 4 limbs of 2 days duration followed by weakness of neck.
On Examination: - BP:-110/80mmhg; Neurological examination:-Power of muscles of both upper limbs and lower limbs 3/5; Reflexes: - Normal; Sensory system:-Normal; Cranial nerves: - Normal

Investigations

Serum potassium:-1.8meq/l, Serum Sodium:-137meq/l, Serum Creatinine:-0.8, RBS: - 100 mg/dl
Initial investigation showed Hypokalemia.
We followed the algorithm (fig 2) and evaluated the patient for the cause of Hypokalemia.
Serum Magnesium:-2.12 mg/dl, Serum Calcium:-7.5mg/dl, Hematological profile normal. Serum Osmolality:-289.6, Trans tubular potassium gradient:-7, ABG: showed metabolic alkalosis with pH of 7.58, pCo2:-31mmhg, pO2:-100, Hco3:-30.1mmol/l, Urinary calcium creatinine ratio:-0.31, Serum aldosterone: 294 Pmol / L (55-250Pmol/L), Plasma rennin: 10.8ng/ml/hr(0.29- 3.7ng/ml/kg)
Urinary Potassium: 40mmol/l (<15 mmol), Urinary Sodium:-95mmol, Urinary Osmolality: 306 mos/kg,

DISSICUSION
Bartter syndrome is a group of closely related hereditary tubulopathies characterized by renal salt wasting, hypokalemia, metabolic alkalosis and hyper-aldosteronism hyperrenemic with normal blood pressure[1,4]. The renal salt loss in Bartter syndrome is caused by impaired transepithelial transport in thick ascending loop of henle. The resultant defect in sodium transport leads to reduction in trans tubular potential difference resulting in decrease in paracellular calcium reabsorption in thick ascending Loop of henle[3,1]. Reduction in intravascular volume also induces aldosterone mediated metabolic alkalosis.
Some patients have an autosomal recessive mode of inheritance in classic Bartter syndrome, although many cases are sporadic. In classic Bartter syndrome, the defect in sodium reabsorption appears to result from mutations in the chloride-channel gene.
The consequent inability of chloride to exit the cell inhibits the sodium chloride/potassium chloride cotransporter.

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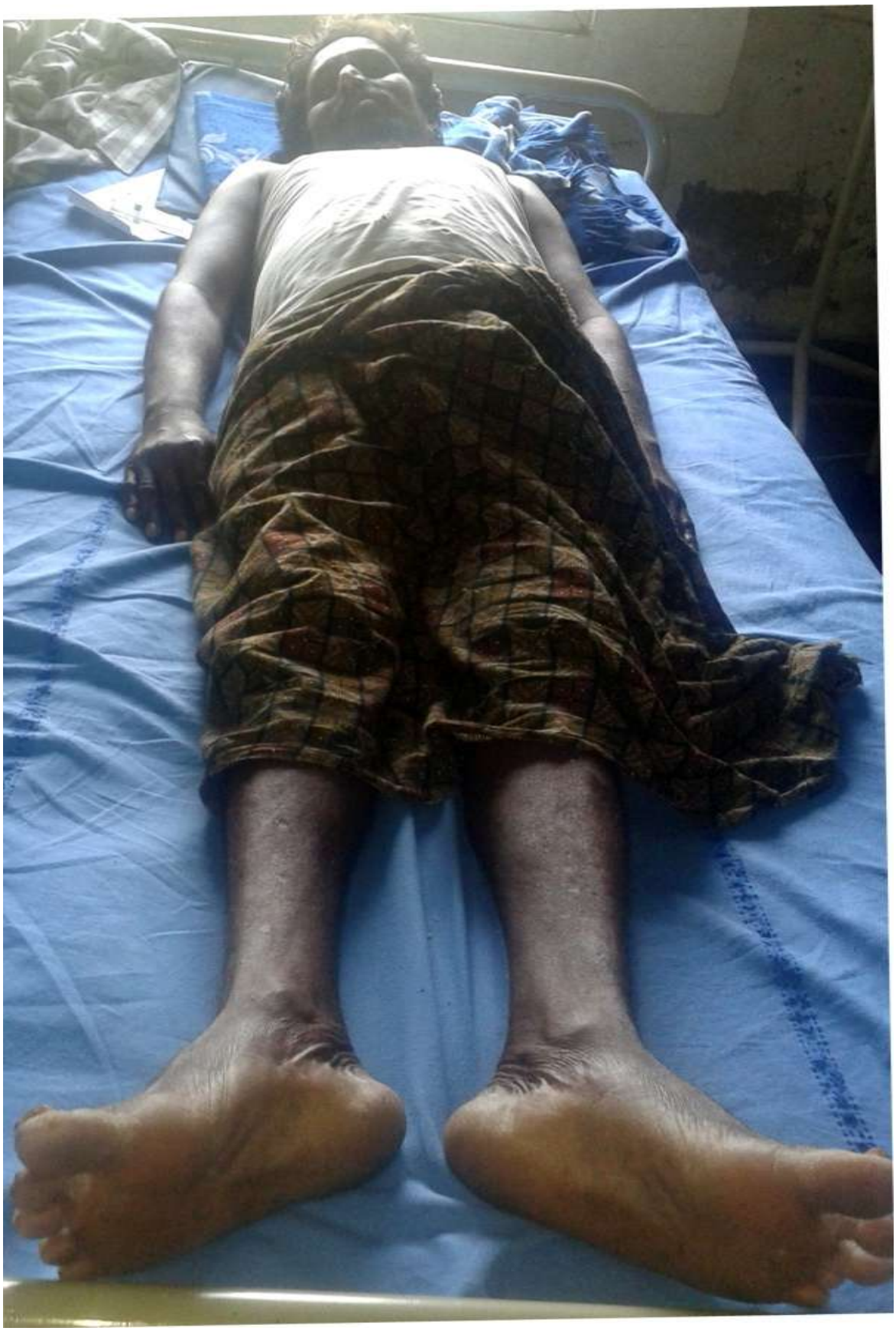

Figure 1: Showing Quadriparesis

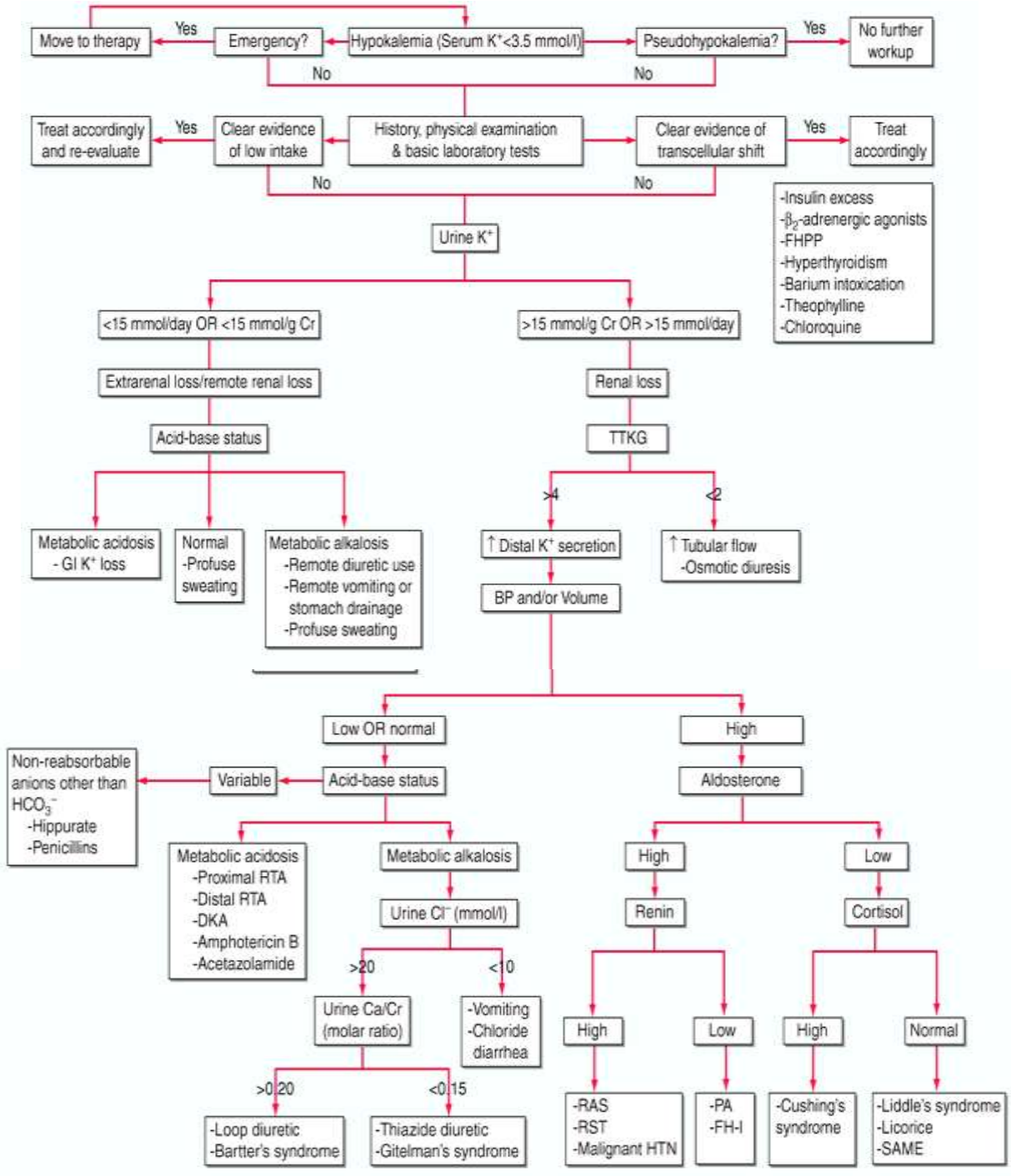

Figure 2: Approach to a patient with hypokalemia

Increased delivery of sodium chloride to the distal sites of the nephron leads to salt wasting[3], polyuria, volume contraction, and stimulation of the renin-angiotensin-aldosterone axis. These effects, combined with biologic adaptations of downstream tubular segments, specifically the distal convoluted tubule (DCT) and the collecting duct, result in hypokalemic metabolic alkalosis

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Bartter syndrome differs from Gitelman syndrome in presence of hypercalciuria, nephrocalcinosis , nephrolithiasis. Treatment is aimed at inhibition of renin angiotensin aldosterone axis, prostaglandin kinin and potassium supplements.

Our patient had hypokalemia, metabolic alkalosis, increased renin ,aldosterone levels, elevated 24hrs Urinary calcium creatinine ratio, with normal blood pressure, normal levels of magnesium. Although hypomagnesemia is a finding of Bartter syndrome only 20% had hypomagnesemia[3]. We reviewed the literature and very few cases of adult onset Barters were reported in literature[7 8 9]. Our patient had similar presentation and investigation. Our patient improved with potassium supplementation. We also started the patient on inhibitors of prostaglandin kinin.

CONCLUSION

Patients with Hypokalemic paralysis should be evaluated for the cause of Hypokalemia. So that they can be managed effectively.

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