BARTTER SYNDROME

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INTRODUCTION

Bartter syndrome is an uncommon renal tubular disorder.

The first case was reported in 1960 with subsequent three cases in 1962 and thereafter several cases have been reported. Bartter syndrome is characterized by hypokalemic, hypochloremic alkalosis, normal blood pressure despite elevated serum levels of angiotensin and marked hyperplasia of juxtaglomerular apparatus.

There is failure to thrive, polyuria, polydipsia, constipation, muscle weakness, salt craving, tetany and sometimes—renal osteodystrophy and renal insufficiency. Biochemical changes include hypokalemia, hyponatremia, hypomagnesemia and hypochloremic metabolic alkalosis. Urinary potassium and chloride excretion is increased inspite of low serum levels. Endocrine changes include very high plasma renin and angiotensin levels and increased aldosterone secretion rates with a normal urinary aldosterone excretion. The diagnosis is suspected by finding characteristic biochemical changes already described and confirmed by histologic demonstration of hyperplasia of juxta-glomerular apparatus.

CASE REPORT

A three months old male baby was referred to us from a peripheral hospital with the history of fever, fits, loose motions, vomiting and irritability. On examination he was found to be unwell, irritable, febrile, dehydrated with marked carpopedal spasm. His B.P was 75/50 mm Hg. Our initial diagnosis was sepsis / meningitis with electrolyte imbalance (hypocalcemia). His initial investigations showed serum sodium (Na⁺) 135 mmol/L, Potassium (K⁺) 2.2 mmol/L, Calcium (Ca²⁺) 10.5 mg%, Chloride (Cl⁻) 90 mmol/L, blood urea 30 mg%, creatinine 0.6 mg% and urine specific gravity 1.086. His cerebrospinal fluid (C.S.F) analysis showed a clear fluid with glucose 93 mg%, Protein 20 mg%, white cell count (W.B.C) 12/cmm with a differential count of 4% polymorphs and 96% lymphocytes. C.S.F culture yielded no growth. The patient was resuscitated with intravenous fluids and electrolytes along with parenteral ceftriaxone and calcium gluconate infusion but the baby was still dehydrated, febrile and lethargic with carpopedal spasm inspite of the overnight fluids and calcium gluconate infusion. Repeat investigations revealed serum Na⁺ 148mmol/L, K⁺ 2.9 mmol/L, Ca²⁺ 11.5 mg% and Cl⁻ 90.6 mmol/L. Calcium gluconate infusion was stopped. Arterial blood gases (A.B.G.s) were done and


