Introduction:

Bartter's Syndrome is characterized by renal potassium wasting with hypokalemia, metabolic alkalosis, increased renin-angiotensin-aldosterone system, normal blood pressure, resistance to the pressor effects of angiotensin II and juxtaglomerular cell hyperplasia. Most of the cases have been noted in the pediatric age group and adult-onset cases are very rare. In 1962, Frederic Bartter and his colleagues wrote their seminal paper based on two patients with hypokalaemic metabolic alkalosis, hyperaldosteronism, normal blood pressure, decreased pressor responsiveness to angiotensin II infusion and hyperplasia of the juxtaglomerular apparatus.The syndrome comprising the above mentioned observations was hence named after him. We report a case of adult-onset Bartter's syndrome.

Case Report :

A 52 year old Gentleman with past history of Pulmonary Koch came with breathing difficulty along with fever.In view of severe respiratory distress, he was intubated and put on Mechanical Ventilation and treatment initiated after sending all relevant investigations.From history and from past medical records it was found that he was Normotensive in matters pertaining to his Blood Pressure.Initial routine investigations revealed low serum Potassium levels for which intravenous Potassium Chloride was given. Despite correction,his serum Potassium did not rise and maintained in the range of 2.4-2.9 mEq/L.Hence cause for persistent hypokalemia was evaluated and Arterial Blood Gas analysis which was already being done to asses ventilatory status, correlated to look for any respiratory cause of hypokalemia.ABGs showed relatively persistent Metabolic Alkalosis.Obvious cause of Metabolic Alkalosis such as diueretics,renal failure were also ruled out.Ultrasonography of Abdomen revealed tiny Right renal calculi, hypoechoic areas in bilateral Adrenal glands and Common Bile Duct Stone.In view of renal stone, urinary Calcium was evaluated which was found to be high.Suspecting Bartter Syndrome as the above mentioned findings pointed towards it, his serum Aldosterone level was assessed which was also high.Serum Renin levels were not assayed due to Laboratory constraints.Adrenal Biopsy was planned after patient stabilization in view of ?Adrenal Mass.Renal Biopsy was also not done.Meanwhile the patient was tracheostomised due to difficult weaning and subsequently the patient expired.

Discussion:

The constellation of Hypokalemia, Normotension, Metabolic Alkalosis, increased Aldosterone level and increased Urinary Calcium substantiated thedifferential diagnosis of Bartter's syndrome.However, as stated, Renin Activity could not be assesed and Renal Biopsy was not done.Also since Adrenal gland biopsy could not be done,an Adrenal glandtumour contributing to increased serum Aldosterone and hypokalemia could also be not ruled out.Hence the possibility of any other differential diagnosis contributing to the above constellation of findings is open for debate. Terms such as Bartter-like syndrome do little to help the clinician identify the specific metabolic defect and treat the patient's illness correctly. It may be better to sub-classify Bartter syndrome by renal pathophysiological abnormality. By this method, Bartter syndrome falls into four subgroups: (i) antenatal Bartter syndrome (hyperprostaglandin E2 syndrome); (ii) the Gitleman variety of Bartter syndrome (Gitleman syndrome); (iii) classical Bartter syndrome; and (iv) pseudo-Bartter syndrome.Often while assessing a cause of Hypokelemia we might miss that the hypokalemia might represent anyone of the constellation of findings correlating with any above mentioned syndrome.Bartter's syndrome may be mimicked by magnesium deficiency, diuretic use or vomiting. Magnesium depletion causes kaliuresis, diuretics cause potassium and volume depletion and vomiting causes renal potassium wasting and volume depletion.The primary aim of the treatment of the Classical Bartter syndrome is correction of hypokalaemia and alkalosis. Therefore administration of potassium chloride is always necessary. The dose of KCl supplementation should individually be titrated in accordance to the patient's needs and must balance the amount lost by the kidney. However, this mode of supplementation therapy is almost totally ineffective by itself, since administered potassium is lost through the kidney in a short period of time. It may seem logical that potassium-sparing agents such as spironolactone would be aneffective additiveto supplementation therapy at this stage. Indeed these groups of medication offer an effective but transient control of hypokalaemia.The most beneficial group of medication in treatment of classical Bartter syndrome is the prostaglandin synthetase inhibitors. Indomethacin (2–5 mg/kg/day), acetylsalicylic acid(100 mg/kg/day), and ibuprofen (30 mg/kg/day) have all been tried. But the most frequently used is indomethacin.

Conclusion:

Although rare a strong suspicion for this syndrome should be there while dealing

with difficult cases of hypokalemia.

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