

developed fever, headache and altered sensorium with signs of raised intracranial pressure. On neuroimaging, he had multiple tuberculomas with communicating hydrocephalus. Child was started on previous ATT regimen that we had put him on initially, and he was treated with dexamethasone along with 3% sodium chloride for his raised intracranial tension. Subsequently, his DST report arrived which showed resistance to rifampicin, isoniazid, km, ofloxacin, E and eto with sensitivity to moxifloxacin, Z, linezolid, PAS, amikacin, clofazimine and capreomycin. Thus as per the medicines that he was receiving from DOTS, the child would be receiving only two drugs (Z and km) to which the DST showed sensitivity with all the other drugs being resistant. This could have led to worsening of his clinical condition. Though it may be argued that appearance of tuberculomas may suggest a paradoxical reaction, the child even after one month of hospitalization was bed ridden, and needed a ventriculoperitoneal shunt for his

hydrocephalus suggesting that he developed CNS TB as part of his worsening of TB.

Thus, there is a need for revision of national guidelines for management of DR-TB patients to avoid worsening of the disease condition.

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## Bartter Syndrome with Nephrogenic Diabetes Insipidus and Vitamin D Resistant Rickets

A 1-year-old boy presented with polyuria, polydipsia and poor weight gain noticed since three months of age. He weighed 5 kg (-3SD), Height 64 cm (-3 SD), and had features of some dehydration. Hyponatremia (165 mEq/L) and metabolic acidosis (pH 7.27, bicarbonate levels 17 mEq/L) were detected. Serum creatinine, potassium, calcium, magnesium and chloride levels were within normal reference ranges. Serum osmolality and urine osmolality were 355 mOsm/L and 145 mOsm/L, respectively. He was diagnosed as nephrogenic diabetes insipidus after a vasopressin challenge test failed to increase the urine osmolality levels. Renal ultrasonography was normal. He was treated with spironolactone.

At the age of 3 years, he presented with rickets and hypocalcemic tetany (ionized calcium 2.2 mg/dL) in association with hypophosphatemia (2.2 mg/dL) and secondary hyperparathyroidism (PTH levels 180.2 pg/mL). The rickets was refractory to therapy with Vitamin D; and the child developed fractures of bilateral ulnae and femur requiring hip spica and plaster casts. He was still showing poor weight gain (weight 7.9 kg, -3SD).

Triangular facies, prominent eyes and forehead, and large ears were appreciated. Blood pressure was normal. At this juncture, he was found to have hypokalemia (2.5 mEq/L), metabolic alkalosis (pH 7.52, bicarbonate levels 35.2 mEq/L) and hypercalciuria (spot calcium: creatinine ratio 1.4). Serum magnesium and creatinine levels were normal; urine chloride was >20 mEq/L. Plasma renin activity was high (38.9 ng/mL/h), confirming Bartter syndrome. Wrist X-ray showed metaphyseal cupping and splaying. Serum 25 hydroxycholecalciferol levels were 31.4 ng/mL. He is currently on potassium chloride (8 mEq/kg/day), indomethacin (2 mg/kg/day), enalapril (0.5 mEq/kg/day), and calcium supplements. At the last follow up at age of 4 years, his serum potassium, sodium, creatinine, calcium and phosphate levels are normal, and he is showing satisfactory weight gain.

The presentation of this child with Bartter syndrome is unusual for two reasons. The first being the initial paradoxical presentation with hypernatremic dehydration and metabolic acidosis; the second being the association with vitamin D resistant rickets (leading to secondary hyperparathyroidism). The former presentation has been anecdotally reported in the literature [1,2]. Bettinelli, *et al.* [1] reported a child who presented with severe hypernatremia, who was initially diagnosed as nephrogenic diabetes insipidus, but on follow up was diagnosed as Bartter syndrome. They concluded that in a few cases of Bartter syndrome, hypokalemia and/or metabolic alkalosis may be absent during the initial few

years of life. Instead, atypical presentations such as hypernatremia and/or metabolic acidosis may be encountered. The latter atypical presentation in our case is the development of vitamin D resistant rickets in Bartter syndrome. This complication has been reported only twice in published literature long ago [3,4], and is a largely forgotten entity. It has been attributed to the calcipenic effect of hyperprostaglandinemia, or renal phosphate loss [3]. It is pertinent to note that in spite of deranged vitamin D metabolism, overt rickets is uncommon in Bartter syndrome [5].

Complications such as hypernatremia, metabolic acidosis and rickets can confound the clinical presentation of Bartter syndrome, and lead to a delayed diagnosis since pediatricians may not be familiar with such atypical presentations. The objective of this report is to create awareness regarding such atypical presentations, which highlight the phenotypic variability of Bartter syndrome.

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