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Renal Tubular Acidosis

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SUMMARY

The clinical laboratory and X-Ray findings of 16 patients with distal (type-1) renal tubular acidosis diagnosed at Mayo and Sheikh Zayed Hospital, Lahore during an eight year period (1984 to 1992) are presented. Twelve patients (75%) were male and four female (25%). Their ages ranged between 1.5 and 41 years (mean age 12.8 years). Eight patients (50%) had radiologically evident bone disease in the form of rickets or osteomalacia. Two patients had pathological fractures. Growth retardation was noted in 10 patients (63%). Polyuria and polydipsia were noted in 6 patients (31%). Nephrocalcinosis and or nephrolithiasis was evident in 12 patients (75%). Muscle weakness was noted in 7 patients (44%). Biochemical derangements observed were hyperchloremic metabolic acidosis mean blood pH = 7.259±0.055, but urine pH remained alkaline in all patients during all grades of metabolic acidosis with a mean value of 6.69 (range 5.8 to 8.0). Serum Chloride level was mean 111±5.288 mmol/l. Hypocalcemia, hyponatremia and hypokalemia was noted with mean values of 8.44±1.24 mg/dl, 134±3.56 mmol/l and 3.68±0.53. Treatment with alkali therapy resulted in general well being, reduced bone pains, muscle weakness and improved growth significantly.

INTRODUCTION

The kidney's primary role in acid base homeostasis is to stabilize the serum bicarbonate concentration. The daily acid production in adults averages 1 mEq/kg and in children 2 mEq/kg body weight[1-3]. Body must get rid of this load and bicarbonate lost in the process of neutralizing this acid must be replenished. The kidney must not only resynthesis what is lost during metabolic acid production but also must reclaim the daily filtered load of bicarbonate 4500 mEq of bicarbonate is filtered daily and most of it is reabsorbed by the renal tubules[1]. If the tubular function is not intact, the urinary loss of even small percentage of this could rapidly deplete body stores of bicarbonate and cause severe metabolic acidosis.

The proximal convoluted tubule takes the responsibility of reabsorbing the filtered bicarbonate, the distal tubule secretes hydrogen ions and regenerates bicarbonate[5,6]. Failure of the renal tubules to do this will result in renal tubular acidosis. On the basis of this physiological division renal tubular acidosis is classified into various types. Commonest being type 1 (Classic) or distal renal tubular acidosis and less common type 2 or proximal renal tubular acidosis.

Patients with distl renal tubular acidosis have an inappropriately alkaline urine in the presence of metabolic acidosis and fail to acidify the urine in response to an acid load. The resulting hyperchloremic metabolic acidosis with normal anion gap, results in growth retardation, late rickets, Nephrocalcinosis, Nephrolithiasis, polyuria, polydipsia and at times osteomalacia and pathological fractures[7]. Diagnosis of renal tubular acidosis is very simple provided the disease is in the physician's mind and clinically suspected in the setting of a hyperchloremic metabolic acidosis and inappropriately alkaline urine. Simple tests like urine pH, analysis of serum electrolytes and plain x-ray abdomen give a clue to the diagnosis.

Most of the literature available on this subject is published abroad and only few cases have been reported in this part of the world even though the disease is not so rare[8].

MATERIAL AND METHODS

The study was performed to see the (1) mode of

presentation of patients with renal tubular acidosis, the severity and nature of biochemical derangements in these patients and to evaluate (2) the effect of treatment on the subjective symptomatology, growth rate and biochemical profile during the follow up period. The study consists of 15 patients admitted to North Medical and Paediatrics departments of Mayo Hospital and Sheikh Zayed Hospital, Lahore. Patients were included in the study on the basis of following criteria.

Inclusion criteria

1. Symptoms referable to renal tubular acidosis.
2. Inappropriately high urine pH.
3. Presence of metabolic acidosis.

Exclusion criteria

1. Presence of high urine pH due to urinary tract infection with urea splitting organisms.
2. Hyperchloremic metabolic acidosis due to causes other than renal tubular acidosis.

A detailed history was taken from the patients or their relatives according to the following proforma:-

- Age & Sex.
- Duration of illness and onset of symptoms.
- Growth retardation.
- Polyuria & polydispsia.
- Gastro intestinal symptoms like nausea and vomiting.
- Musculoskeletal symptoms: Muscle weakness & periodic paralysis Bone pains, and joint pains.
- Symptoms referable to Nephrolithiasis.
- Any family history of such disease.

These patients were thoroughly examined with special attention to the presence of growth retardation and signs of rickets and bone disease.

Following investigations were done to establish the diagnosis:-

- Urine pH, Specific gravity, microscopy & Culture Sensitivity.
- Serum Sodium, Potassium, Chloride, Calcium, Phosphorus.
- Arterial blood gases.
- Blood urea and creatinine estimations.
- X-Rays of bones for signs of rickets, osteomalacia, fractures & bone age.
- Plain X-Ray abdomen to see Nephrocalcinosis.
- Intravenous pyelography (where indicated).
- Ultrasonography of kidneys.
- Confirmatory tests (when indicated).
  - Ammonium chloride loading test.
  - Bicarbonate loading test with
  - Urine - blood PCO2 ratio.

RESULTS AND OBSERVATIONS

A total of 16 patients are included in this study. All were found to have distal (Type 1) renal tubular acidosis. Thirteen (13) patients were sporadic in nature, 2 had a family history of similar disease and in one patient self administration of vitamin-D resulted in hypervitaminosis-D and later renal tubular acidosis.

Their age ranged between 1.5 to 41 years (mean 12.8 years). Twelve patients were male and four were females (Fig. 1).

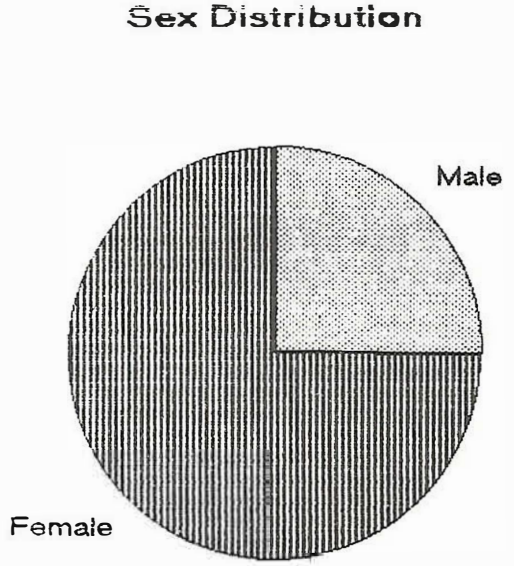

Fig. 1: Showing sex distribution.

Clinical data

Rickets and osteomalacia was noted in 8 patients (50%) with classical architect changes (widening of ends of long bones, loss of normal trabecular bone pattern alongwith bowing of long bones) and generalized loss of bone mineral. Two patients even had pathological fractures involving shafts of tibia and femur. Growth retardation was noted in 10 patients (63%). Two children below the age of 10 years were on 3rd and 5th percentile of

growth. Extensive calcification within the renal parenchyma or in the renal pelvis was noted in 12 patients (75%) on plain x-ray abdomen and was ultrasonographically confirmed in the form of Nephrocalcinosis / Nephrolithrasis. History of renal angle pain was present in majority of these patients and 3 patients had passed stones per urethrum on more than one occasion and had received prior treatment elsewhere. Polyurea and polydipsia was noted in 5 patients (31%) and muscle weakness co-related well with serum potassium level (Fig. 2).

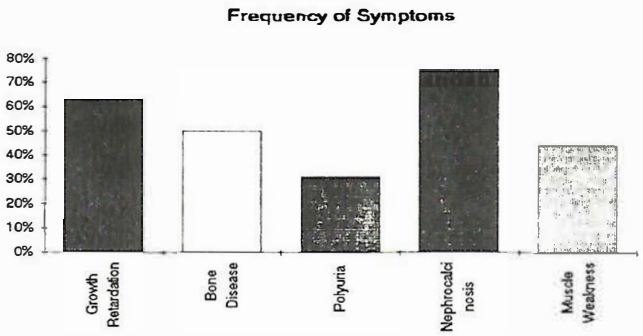

Fig. 2: Showing frequency of symptoms.

Biochemical data

Analysis of arterial blood gases showed that all patients had mild to moderate degree of metabolic acidosis with pH range of 7.340 to 7.160 (mean 7.259±0.055) inspite of this metabolic acidosis, urine was irappropriately alkaline in all the patients with range 5.80 to 8.00 (mean 6.69±053).

Serum bicarbonate levels were correspondingly low ranging from 11.9 to 20.3 mEq/l in all patients (mean 15.631±2.529). Serum chloride levels were high with a range of 107 - 129 mmol/l (mean 111±5.228). Both serum calcium and phosphorus levels were low because of renal bone disease with mean values of 8.44 mg/dl and 3.95 mmol/l respectively. Serum sodium and potassium levels were also low mean sodium 134±3.56 mmol/l and mean potassium value of 3.68±0.53 mmol/l. Treatment with alkali therapy (Baking Soda) 50 - 100 ml/day given to two patients with 3rd and 5th percentiles improved their growth to 7th and 10th percentiles respectively over a follow up of 6 months period.

DISCUSSION

Renal tubular acidosis is nearly half a century old disease Lightwood in 1935[9] gave the first description of a renal acidosis quite distinct from that associated with renal failure. Initial case reports drew attention of many research workers and since then a substantial amount of literature has been published on this disease. Micropuncture techniques have brought about major breakthrough in the understanding of its pathophysiology and as knowledge about renal tubular acidosis has expended newer form of renal tubular acidosis have been discovered. The older definitions have been replaced by the new ones. For instance, older definitions of renal tubular acidosis included hypokalemia, but ever since the discovery of hyperkalemic (type 4) renal tubular acidosis, this word has been excluded from the definition[10]. The most recent definition of renal tubular acidosis defines it as " A clinical syndrome of disordered renal acidification characterized biochemically by minimal or no azotemia, hyperchloremic metabolic acidosis, reduced venous CO2 content, high urine pH and decreased urinary excretion of titrable acid and ammonium ". However in type-II (Proximal) renal tubular acidosis, urine pH may be low in the presence of severe metabolic acidosis[11,12].

Prototypic type 1 (distal) renal tubular acidosis is the only type in which urine pH is always high, even during severe metabolic acidosis[13]. In healthy kidney Hydrogen ions are continually exchanged for sodium at distal tubules to maintain the hydrogenion concentration in the lumen that is 800 times that of plasma. In type 1 (distal) renal tubular acidosis, the maximum lumen to plasma gradient is only 80:1. This is as a consequence of reduced net rate of H+ secretion in the distal nephron or an increased rate of passive back leak of secreted Hydrogen ions from lumen to cell. Active transport of hydrogen ions might be reduced due to" too few" hydrogen ion secretory pumps or "weak pumps"[4]. Figure 3[12] depicts clinical features and pathophysiology of distal renal tubular acidosis.

In type 2 (Proximal) renal tubular acidosis reabsorption of bicarbonate is reduced by at least

15% at normal plasma bicarbonate concentration. Urine pH is high and bicarbonaturia occurs during mild to moderate but not during severe metabolic acidosis. Distal nephron acidification mechanism is intact. There is common occurrence with Fanconi' Syndrome[11].

In type 4 renal tubular acidosis there is reduction in renal potassium excretion much greater than would be expected for mild accompanying renal insufficiency[14]. The proximal tubule is not at fault, urine become acidic during acidosis and is free of aminoacids, glucose and phosphates. The urinary excretion of ammonium is however greatly reduced even when the urine is very acidic. The defect lies in the cation exchange segment of distal nephron and thereby causes a reduction in secretion rate of both hydrogen and potassium ions. Severity of impairment is determined by the accompanying hyporenenemic hypoaldosteronism[15,16].

Confirmatory biochemical tests are done in doubtful cases Ammonium Chloride loading test: Ammonium Chloride which is metabolized to urea and HCl by the liver is the acidifying agent of choice. NH4Cl 100 mg/kg body weight is given in capsules form orally over 45 minutes and one hourly urine samples are taken for 8 hours. Arterial or free flowing venous blood is drawn at start and 3 hours after NH4Cl load. Inability to lower urine pH below 5.4 is diagnostic of distal rental tubular acidosis[2]. Bicarbonate loading test: This is done to differentiate proximal from distal renal tubular acidosis. Enough bicarbonate is given to elevate and sustain serum bicarbonate at 23 - 28 mEq/l for several days orally. Parentral dose of 1 mEq/kg body weight will rapidly achieve this target. Urinary PCO2 levels should rise to > 32 mmHg in normal individuals. Failure to increase urinary PCO2 during bicarbonate loading is indicative of distal nephron acidification defect. Hence urine, Blood PCO2 values of < 32 mmHg indicate distal renal tubular acidosis.

Distinguishing features between type 1 (distal) and type 2 (Proximal) renal tubular acidosis are presented in Table 1[17].

In distal renal tubular acidosis in addition to hyperchloremic normal anion gap metabolic acidosis, there is renal sodium wasting with consequent fluid volume depletion, renal potassium wasting with consequent muscle weakness and paralysis. Hypercalciuria, hypocitraturia, nephrocalcinosis and renal stone formation. Rickets and osteomalacia may be present due to systemic acidosis.

Table 1: Distinguishing features in proximal versus distal renal tubular acidosis.

Feature	Distal (Type I)	Proximal (Type II)
Biocarbonate Tm	Normal	Decreased
Urinary bicarbonate losses	Small	Large
Serum bicarbonate concentration	May be very low less than 16 mEq	Usually more than 16-18 mEq/l
Response to alkali therapy	Good	Poor
Dose of alkali needed	Small	Large
Serum potassium	Low	Very low
Response to potassium therapy	Good	Poor
Urinary citrate	Low	Normal
Fanconi syndrome	No	Yes
Nephrocalcinosis	Common	Rare
Urine pH - Random	Inappropriately alkaline	Inappropriately alkaline
- First AM	Never less than 6.0	Commonly less than 6.0
- Post acid load	Never less than 5.3	Less than 5.3
Urine PCO_2 minus blood PCO_2	Less than 20	20 or more
Urine PCO_2 to Blood PCO_2 ratio	1:1	2:1

Tm = transport maximum

Source reference 17.

Hypokalemia commonly seen in type 1 and type 2 renal tubular acidosis in due to gradient restriction of Hydrogen ion secretion which reduces the rate of H+-Na+ exchange with resultant increased K+-Na+ exchange, increased urinary sodium loss leading to hyperaldosteronism. Hyperaldosteronism further promotes Potassium excretion and hypokalemia of varying degree results[15,18,19].

Table 2: Orally administered preparations for alkali therapy.

Drug		How supplied	Dosage Equivalent
Bicitra	Solution	5 ml = Citric acid 300 mg Sodium citrate 500 mg	1 ml = 1 mEq Base
Calcium carbonate	Tablet Powder	420, 650 mg, 1000 mg 1/2 teaspoon	1000 mg = 22.3 mEq Base
Polycitra	Solution	5 ml = Citrate Acid 334 mg potassium citrate 550 mg sodium sitrate 500 mg	1 ml = 2 mEq base and 1 mEq potassium
Polycitra-K	Solution	5 ml = potassium Citrate 1,100 mg Citric acid 334 mg	1 ml = 2 mEq base and 2 mEq potassium
Sodium Bicarbonate	Tablet Solution	325 mg, 650 mg 1 ml = 1 mEq base	325 mg = mEq base 650 mg = mEq base
Shohl's solution	Solution	1000 ml = Citrate Acid 140 gms Sodium citrate hydrated Crystalline 90 gms	1 ml = 1 mEq base

Source reference 30.

Hypokalemic periodic paralysis may occur spontaneously or as a result of treatment of acidosis with alkali and glucose containing fluids causing intracellular shift of potassium[3]. In our study 7 patients (44%) experienced muscle weakness and had evidence of hypokalemia on laboratory investigation.

Polyuria and polydipsia is due to poor handling of sodium and water by the kidneys, impaired concentrating ability and hypercalciuria which promotes diuresis and stimulate thirst mechanism. In our study 6(31%) patients had these symptoms.

Nephrocalcinosis and Nephrolithiasis is a reliable marker of distal renal tubular acidosis[20]. Several metabolic abnormalities associated with renal tubular acidosis predispose to calcium deposition in the kidneys. Hypercalciuris and alkaline urine are the prerequisites. Hypercalciuria is due to mobilized calcium from the bone consequent to metabolic acidosis. Excess of Hydrogen ions are taken up by bone which acts as a buffer and calcium carbonate is released. Hypercalciuria and hyperphos-phaturia results. Secondary hyperparathyroidism is also said to play a role in its causation[21]. A more alkaline urine favours calcium phosphate crystal formation in the kidneys. Citrate acts as a natural inhibitor of calcium deposition as calcium phosphate in the urine. It chelates calcium by forming soluble complexes with it therefore Hypocitraturia in patients with distal renal tubular acidosis predisposes them to stone formation[19,22-25]. In our study of 16 patients with distal renal tubular acidosis all these features were noted in one or the other patient. Growth retardation, Nephrocalcinosis/ephrolithia-sis, bone disease in the form of Rickets or osteomalacia remained to be prominent features of the disease. Twelve patients (75%) had symptoms pertaining to nephrolithrasis and nephrocalcinosis which was ultrasonographically or radiologically evident. Brenner at al in 1982[20] showed an over all incidence of 29% in all types of renal tubular acidosis and 56% incidence of nephrocalcinosis in type 1 renal tubular acidosis patients. Earlier Courey and Pfister in 1972[26] reported 21 patients with renal tubular acidosis with an incidence of 49% Nephrolithiasis and 38% Nephrocalcinosis. The higher incidence of these two conditions in our study could be either longer duration of the disease before diagnosis or otherwise higher incidence of stone formation in our stone belt area.

In some patients tubular acidification defect is a consequence of Nephrocalcinosis rather than its cause. In one of our adult patients there was a strong history of self administration of vitamin-D for many years and renal tubular acidosis and Nephrocalcinosis in this patient were assumed to result from Hypervitaminosis-D.

In both children and adults bone disease may occur. In children it takes the form of rickets and in adults osteomalacia, pathological fractures may occur too.

The causes of bone disease are acidosis induced bone mineral loss and inadequate production of 1,25

Dihydroxyvitamin-D3[27]. Harrington at all in 1983[19] reported an incidence of 27% bone disease in patients of renal tubular acidosis. In our study the incidence of bone disease was 50%. Two patients had spontaneous pathological fractures.

In untreated patients of both proximal and distal renal tubular acidosis impairment of growth is characteristic. There is good evidence that bicarbonate wasting and resultant metabolic acidosis is the cause. Bone disease also contributes to growth retardation. Serum Bicarbonate level is also considered to be a critical determinant of human growth hormone release[13].

Correction of acidosis with alkali results in improvement in growth and healing of bone lesions[28,29]. In our study 10 patients (63%) had growth retardation and 2 children below age 10 years were in 3rd and 5th percentiles on growth chart. There was improvement noted and in 6 months follow up period their growth improved to 7th and 10th percentiles respectively.

The main stay of treatment in type 1 and 2 renal tubular acidosis is the administration of alkali in amounts necessary for correction of metabolic acidosis and bicarbonate solution can be prepared at home very easily. Eight ounces of Baking Soda dissolved in 2.88 litres of distilled water and yields a bicarbonate concentration of 1 mEq/ml[15]. Patients unable to tolerate soda bicarbe can be given Shohl's solution as an alternative. Table 2 provided a list of soda bicarb preparations marketed alongwith their dose equivalents[30]. Some patients may require potassium supplements which can be given by replacing half of sodium citrate in Shohl's Solution with potassium citrate or by adding potassium supplements in the form of potassium gluconate or potassium acetate[31].

Present study stimulates us to investigate all children with growth retardation in detail especially when they have symptoms referable to acidosis, muscle weakness or nephrolithiasis keeping diagnosis of renal tubular acidosis in mind. Early diagnosis and treatment minimizes complications and promotes growth.

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