Case Report of Renal Tubular Acidosis Presenting with Periodic Paralysis

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Introduction:
PERIODIC PARALYSIS is usually the result of cellular potassium imbalance. Sometimes the cause of hypopotassemia can be diagnosed as in this patient.

Case Report:

P.W.T., a 22 year old Chinese Male, was admitted on 6th January 1975 with complaints of low backache on strenous work and weakness of limbs of sudden onset, and short duration followed by spontaneous recovery. He had another episode on 22nd January and 15th March.

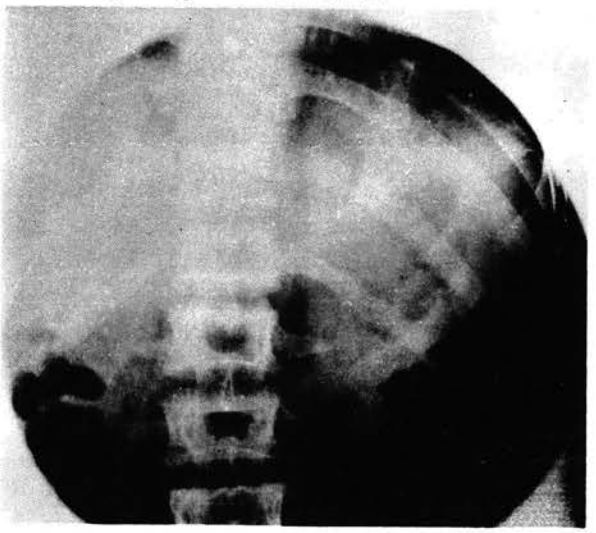

Fig. 1
Cone View of Renal Area Showing Bilateral Nephrocalcinosis and codfish vertebrae.

There were no significant findings on clinical examination except muscle weakness during the acute phase.

Investigations:

Blood

Bl. urea 28 mg%
Serum Uric Acid 5.5 mg%
Serum Calcium 9.6 mg%
Serum Phosphate 3.1 mg%
Serum alkaline phosphatase 9.5 K.A.U.
Serum Sodium 1.38 meq./litre
Serum Potassium 3.9 meq./litre when patient was well and 2.6 meq./litre during acute weakness
Serum Chloride 100 meq./litre
Haemoglobin 15 g%
T.W.D.C. = 7,400 (P = 75%, L = 23%, M = 1%, E = 1%)

Urine

1) PH = 6.5
2) Calcium excretion = 0.13 g/24 hours
3) Phosphate excretion = 4.0 g/24 hours
4) Pitressin test showed impaired concentration
5) S.G. = 1.005

Urine acidification tests were not performed since ammonium chloride could not be obtained.

X-rays – see illustrations

Treatment:
Sodium bicarbonate 10 gram nocte and mist. pot. cit. Patient improved with no recurrence of symptoms.

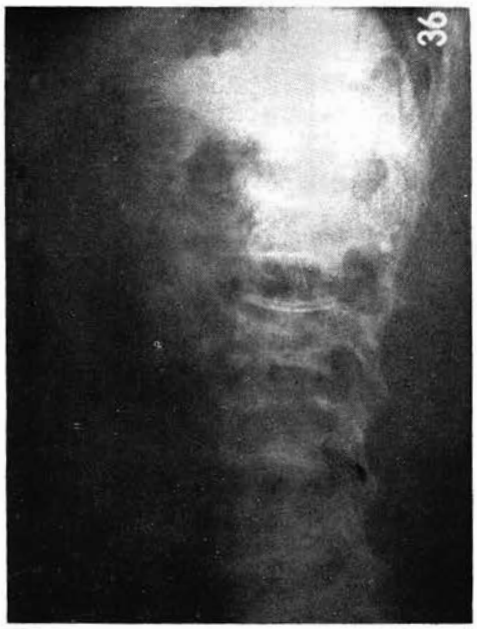

Fig. 2
"Cod fish" vertebrae clearly seen on lateral view of lumbar spine.

Discussion

Renal tubular acidosis was suspected as the cause of periodic paralysis (low serum potassium during acute weakness) from the abdominal X-rays – nephrocalcinosis in kidneys of normal size and cod-fish vertebrae with rarefaction of bone indicating osteomalacia. The other common causes of nephrocalcinosis are hypercalcaemia (e.g. hyperparathyroidum, sarcoidosis, milk alkali syndrome) and medullary sponge kidney. In the former the kidneys are small due to the long standing effects of hypercalcaemia and there are other sites of metastatic calcification. In the later, the kidneys are often large because of congenital dilatation in the collecting tubules and young patients are often asymtomatic (Lindvall, 1959)[3]

Renal tubular acidosis (R.T.A.) has been divided into 2 types (Rodriguez-Soriano J. and Edelmann, C.M. jr 1969)[6] – (1) proximal RTA caused by impaired bicarbonate reabsorption in the proximal tubule and (2) distal R.T.A. secondary to an inability of the distal tubule to normally reduce urine PH. Renal potassium wasting in the latter is probably due to reduction in the $H^+ - Na^+$ exchange; $K^+ - Na^+$ exchange thus increase reciprocally. Corrective alkali therapy by raising urinary pH to values equal to or greater than arterial pH, could remove the gradient restriction on renal H^+ secretion (Gill, J.R. Jr et. al. 1967). The shortage of base with which to excrete acid may lead to a local demand for calcium. The resultant hypercalciuria is probably responsible for the nephrocalinosis and the complication of osteomalacia (Albright and Reifenstein, 1948)[1]

Biochemically in R.T.A. the plasma Co2 combining power is low and the chlorides raised; the serum calcium normal or low and the plasma phosphorus low. If bone changes are marked, the alkaline phosphatase is usually raised. The serum potassium is frequently low. The urine is usually neutral or alkaline but is sometimes slightly acid in the presence of the more severe grades of acidosis. The titratable acidity and the ammonia content of the urine are low, the urine specific gravity is low since the kidneys cannot concentrate normally. Typically there is hypercalciuria (L.N. Pyrah 1960)[5]

For completeness I shall mention other causes of periodic weakness which include aldosteronism, hyperthyroidism, myophosphorylase deficiency, myasthenia gravis, hysteria, paramyotonia congenita, acute intermittent porphyria, idiopathic myoglobinuria, gastrointestinal and renal disease associated with excessive loss of potassium, exposure to exogenous toxins and familial idiopathic periodic paralysis. (Mark Dyen et. al. 1969)[4]

Acknowledgements

I wish to thank the Director General of Medical and Health Services of Malaysia, Tan Sri Datuk (Dr) Abdul Majid Ismail for permission to publish this case report, the Medical Unit for supplying the clinical data and my Radiographers who took the excellent films and especially Mr. Francis Leong Foo Sen for taking the photographs.

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