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CASE STUDY: TWO CASE REPORTS ON RENAL TUBULAR ACIDOSIS AS A DIAGNOSIS IN PATIENTS PRESENTING WITH FRACTURES WITH NO PRIOR HISTORY OF TRAUMA

Internal Medicine

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ABSTRACT

Introduction: Renal tubular acidosis is a group of disorders in which despite normal renal function there is development of metabolic acidosis due to defective ability of the kidneys to maintain acid base balance. They are mainly of 4 types: type 1(distal RTA), type 2(proximal RTA), type 3(combined) and type 4(hypoaldosteronism). **Case study-** Here we describe two cases of middle aged people who presented with bilateral lower limb fractures. In the first case the patient was admitted with plan for surgery. However since the lab investigations were suggestive of type 1 renal tubular acidosis he was managed conservatively with oral potassium and oral bicarbonate correction. In the second case the patient was worked up and found to have type 2 renal tubular acidosis and was also managed conservatively with oral bicarbonate and potassium correction and IV bisphosphonate. Both patients improved after conservative management. **Conclusion-** From the above two cases we can conclude that in middle aged people who present with bilateral lower limb fractures a diagnosis of renal tubular acidosis should be kept in mind after excluding history of trauma and chronic kidney disease.

KEYWORDS

renal tubular acidosis, metabolic acidosis,

INTRODUCTION

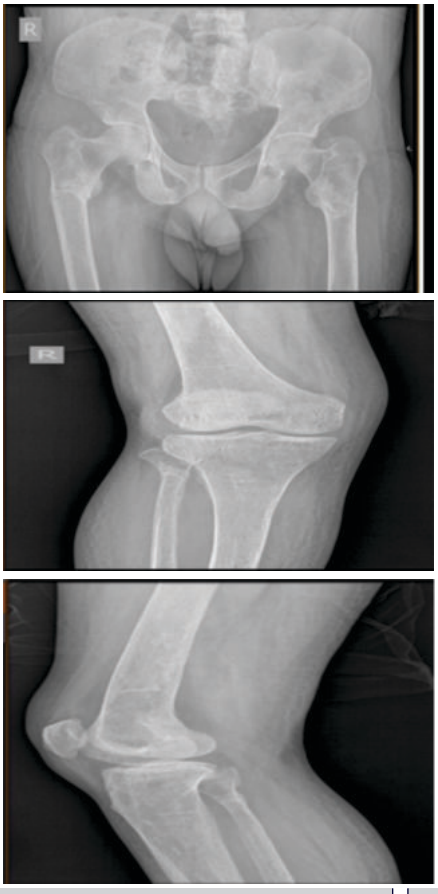
Renal tubular acidosis is a group of disorders in which despite relatively well preserved glomerular filtration rate, metabolic acidosis develops because of defective ability of renal tubules to maintain acid base balance. There is either insufficient secretion of hydrogen ions into the distal tubule or by failure to reabsorb sufficient bicarbonate ions from filtrate in proximal tubule. There are 4 types: type 1 (distal RTA), type 2 (proximal RTA), type 3(combined proximal and distal) and type 4(hypoaldosteronism)[2].

In type 1 RTA the distal tubules and collecting tubules are involved. There is failure of intercalated cells to secrete H+ and reclaim K+[1]. Major causes of new onset distal RTA in adults are autoimmune diseases like Sjogren's syndrome and rheumatoid arthritis, hypercalciuria whereas hereditary distal RTA is most common in children. Other causes of distal RTA are ifosfamide, ibuprofen, toluene etc. It is characterized by severe normal anion gap hyperchloremic metabolic acidosis, hypokalemia, urinary pH persistently 5.5 or higher, reduced urinary ammonium secretion, hypocalcemia, nephrocalcinosis, renal calculi, osteomalacia and rickets, medullary cysts, sensorineural hearing loss and hereditary hemolytic anemia. In type 2 renal tubular acidosis, the proximal tubules are involved. There is failure of the proximal tubular celss to reabsorb HCO3-. Some of the causes for proximal RTA are multiple myeloma and other monoclonal gammopathies, Fanconi syndrome, drugs like acetazolamide, topiramate, tenofovir, ifosfamide, autoimmune disorders like Sjogren's syndrome. It is characterized by normal anion gap metabolic acidosis, hypokalemia, urinary pH 5.3 or less, phosphaturia, glycosuria, aminoaciduria, uricosuria and tubular proteinuria, bone demineralization. Type 3 renal tubular acidosis(combined proximal and distal) is seen most commonly as a result of inherited carbonic anhydrase II deficiency. Due to mutations in gene encoding this enzyme there is autosomal recessive syndrome of osteopetrosis, renal tubular acidosis, cerebral calcification and mental retardation. In type 4 renal tubular acidosis the adrenals are affected. There is either deficiency of aldosterone or resistance to effects of aldosterone(hypoaldosteronism or pseudohypoaldosteronism).It can also be caused by drugs such as NSAIDs, ACE inhibitors, ARBs, spironolactone, trimethoprim, pentamidine. It is characterized by mild normal anion gap metabolic acidosis, hyperkalemia, reduced proximal tubular ammonium excretion. Here are 2 case reports of patients with renal tubular acidosis presenting for the first time as non traumatic bilateral lower limb fractures[1].

Case Study

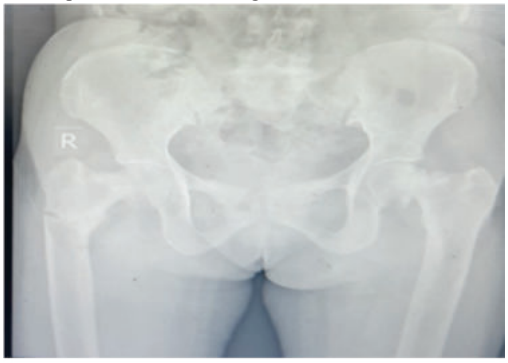
Case 1:

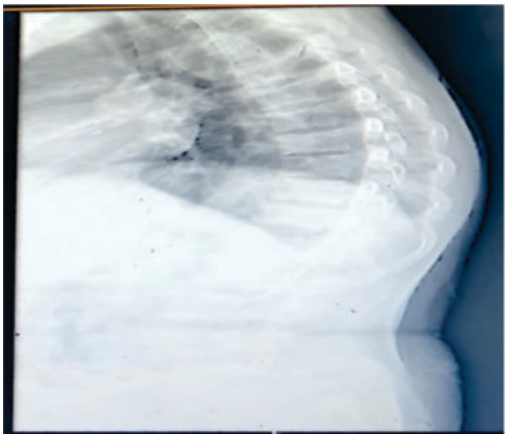
In the first case, a 43 year old male with no known comorbidities and no history of trauma presented bilateral groin pain and right knee pain since 3 years, which had worsened since 3 months. He was diagnosed by X-rays to have bilateral subtrochanteric femur fractures. He was admitted under department of Orthopedics with plan for surgery. Endocrinology opinion was sought in view of suspicion of metabolic bone disease. Clinical examination revealed short stature. Lab investigations showed hemolytic anemia, hypocalcemia, hypopho sphatemia, hypokalemia, vitamin D deficiency. ABG showing normal anion gap metabolic acidosis. Ultrasound of abdomen and pelvis showed nephrocalcinosis. Bone mineral density testing was planned however patient was not willing for the same due to personal reasons. He was diagnosed as type 1 RTA based on the above investigations. The patient was managed conservatively with long term oral bicarbonate and oral potassium citrate correction. As per Orthopedician advice he was advised limited mobilization and traction for the fractures.



Case 2:

In the second case, 42 year old female with no known comorbidities and no history of trauma presented with bilateral hip and lower limb pain since 2½ years. Her Xrays were suggestive of multiple stress fractures. Her laboratory investigations showed anemia, mild deranged renal function tests, hypophosphatemia, hypokalemia, low vitamin D. ABG showed normal anion gap acidosis. Urine routine showed glycosuria and aminoaciduria. Bone mineral density scanning showed T score of -2.9 suggestive of osteoporosis. ANA profile was negative. MRI thoracic spine showed cervical and lumbar spondylosis with grade II anterolisthesis of L4 over L5 vertebral body pseudodisc bulge causing compression of cauda equine nerve roots and bilateral exiting nerve roots at this level. She was diagnosed with type 2 RTA. She was managed conservatively with oral bicarbonate, phosphorus, potassium citrate correction along with IV bisphosphonates for osteoporosis and traction and immobilization for the fractures. Patient condition improved and she is doing well.





Lab parameter	Case 1	Case 2
Hemoglobin(g/dl)	7.5	9.7
Creatinine (mg/dl)	0.98	1.99
Calcium(mg/dl)	7.8	9.4
Phosphorus(mg/dl)	1.7	2.2
Potassium(mmol/L)	3.31	3.48
Vitamin D	14.26	13.31
Arterial pH	7.16	7.3
Bicarbonate	8.2	14.2
Urine pH	7.0	6.0
Glycosuria	negative	present
Aminoaciduria	trace	present
USG abdomen and pelvis	Small right renal calculus(3 mm). Left cortical renal cysts. Moderate splenomegaly	Cholelithiasis. Bilateral grade 1 renal parenchymal changes.

CONCLUSIONS

Normally in patients who present with fractures the preceding cause is found to be trauma. However if the patient is having a pathological fracture the underlying condition needs to be worked up. Most of the pathological fractures tend to occur in elderly people and the cause tends to be malignancies like multiple myeloma, bone secondaries from carcinomas like carcinoma prostate. If pathological fractures occur in middle aged people causes like chronic kidney disease need to be ruled out. However if chronic kidney disease and malignancy is ruled out then the patient needs to be worked up for other causes. From both the above cases, we can learn that renal tubular acidosis should be considered as one of the diagnosis in middle aged adults presenting as fractures with no prior history of trauma. Early diagnosis and initiation of treatment can greatly benefit the patient in overall outcome.

REFERENCES:

1. UptoDate
2. Harrison's Principles of Internal Medicine