International Journal of Advances in Medicine
Kant R et al. Int J Adv Med. 2021 Feb;8(2):283-285
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: https://dx.doi.org/10.18203/2349-3933.ijam20210277

Case Report

Type 1 diabetes mellitus presenting as distal renal tubular acidosis (type 1)

Ravi Kant*, Mohit Garg, Mahendra Kumar Meena

Department of Internal Medicine, All India Institute of Medical Sciences, Rishikesh, Uttarakhand, India

Received: 02 December 2021
Accepted: 08 January 2021

***Correspondence:**
Dr. Ravi Kant,
E-mail: drkantr2006@gmail.com

ABSTRACT

A 42 year-old male patient known case of type 1 diabetes mellitus for last 15 years got admitted with bilateral lower limb swelling and poorly controlled diabetes. He was found to have alkaline urinary pH, persistent metabolic acidosis even after the correction of blood sugars without ketonuria or diabetic ketoacidosis and nephrocalcinosis thus he was diagnosed as distal renal tubular acidosis (RTA Type 1) and managed by alkali replacement with blood sugar control. The association of type-1 diabetes mellitus with type-1 RTA has been rarely reported in the literature, but there are various case reports which had linked with distal RTA with autoimmunity and destruction of DCT. As our case which was unusual as every type-1 diabetes doesn't have acidosis due to DKA focused research is required in this field.

Keywords: Type 1 diabetes mellitus, Ketonuria, Distal renal tubular acidosis, Diabetic ketoacidosis, Nephrocalcinosis

INTRODUCTION

Type 1 diabetes mellitus (T1DM) is an auto-immune disease which is characterized by immunological destruction of beta cells leading to absolute insulin deficiency.[1] Insulinopenia is characterized by dysmetabolism and generation of lactic acid which is a high anion gap metabolic acidosis. Patients with T1DM generally presents with diabetic keto-acidosis (DKA) in which acidosis is due to high ketones in body. We present a case that had normal anion gap acidosis in T1DM with uncontrolled hyperglycemia. Distal renal tubular acidosis (dRTA) commonly occurs in condition like amyloidosis, Sjogren syndrome, systemic lupus erythematosus (SLE) and sickle cell disease. Distal RTA presents as nephrocalcinosis and normal anion gap hyperchloremic acidosis that occurs due to inability of distal convoluted tubule to secrete hydrogen (H+) ions.[2] This in turn leads to accumulation of H+ and subsequently loss of potassium (K+) from the body causing hypokalemia and alkaline urine. This alkaline urine causes precipitation of calcium stones in urine and patients presents as having asymptomatic/ symptomatic nephrolithiasis.

CASE REPORT

A 42-year male presented to emergency with complaint of swelling of bilateral lower limb associated with pain/tingling and numbness for 3 months duration. Swelling in bilateral lower limb was gradual, extending up to ankle. He was a known case of T1DM for 15 years and was currently on inj. insulin mixtard twice daily regimen with fluctuating blood sugar levels. No history of pain abdomen, nausea, vomiting, chest pain, shortness of breath, syncope, palpitation, sweating, burning micturition or decreased urine output. No history of hypertension, tuberculosis, asthma in past. Family history was also not significant. Patient left inj. insulin for the past 3 days due to unavailability. Patient was admitted and investigation was sent to rule out the possibility of DKA. Investigations send at the time of admission were Hb-7.49g/dl, Sr. Na+/K+- 124/5.5mEq/L and HbA1c – 12.7; the laboratory reports of day 04 of admission were listed in (Table 1).

Patient was started on intravenous (IV) fluids and in the lines of DKA protocol as the patient was having a very low bicarbonate (HCO3) and high dextrose. He was managed in the ward and overdue course patient improved of symptoms. After 2 days in hospital, patient's repeat ABG was done, showed persistent low HCO3, so urine pH was advised and reported to be 6.0.

Patient was started on intravenous (IV) fluids and in the lines of DKA protocol as the patient was having a very low bicarbonate (HCO3) and high dextrose. He was managed in the ward and overdue course patient improved of symptoms. After 2 days in hospital, patient's repeat ABG was done, showed persistent low HCO3, so urine pH was advised and reported to be 6.0.

Table 1: The laboratory investigations at day of admission and day-4 of admission.

Date	24/01/2019	28/01/2019
Hemoglobin	7.49g/dl	7.3gm/dl
Total leucocyte count	11340/mm3	8500/mm3
Differential Leucocyte count	N89L5M5	N78L20M2
Platelets	1.46 lakh/mm3	1.56lakh/mm3
Hematocrit	23.7	21.3
Blood urea	81mg/dl	22mg/dl
Serum creatinine	1.96mg/dl	0.96mg/dl
Serum Na	124mEq/l	137mEq/L
Serum K	5.5mEq/L	3.2mEq/L
Serum Cl	98mEq/L	106mEq/L
Serum calcium	7.6mg/dl	7.7mg/dl
Serum phosphorus	3.9mg/dl	3.4mg/dl
Total bilirubin	0.46mg/dl	
Direct bilirubin	0.12mg/dl	
SGPT	15U/L	
SGOT	36U/L	
ALP	168U/L	
Total Protein	5.3gm/dl	
Albumin	3.2gm/dl	
Globulin	2.1gm/dl	
pH	7.32	
HCO3	11.1mEq/L	
Anion gap	11mEq/L	
RBS	High	
HbA1C	12.7	
Urine R/M	Glucose ++, protein + and ketones absent	

Patient USG was done in view of deranged kidney function and reported - multiple hyper-echoic calculi seen in upper pole with largest measuring 7.8 mm in the right kidney. Left kidney size normal with multiple hyper-echoic calculi in lower pole and posterior pole longest 9 mm.

Ophthalmology evaluation suggested bilateral moderate non proliferative diabetic retinopathy (NPDR). 2D echo was normal. Patient's periodic special investigations after the admission in hospital are enclosed in Table 2.

Table 2: Periodic special investigations after the admission in hospital.

Date	28/01/2019	30/01/2019	04/02/2019
Urine pH	6.0	6.0	6.0
Urine R/M	Glucose-nil, protein-nil, ketone-nil	Protein+, glucose-nil	Proteins+, glucose–nil
pH	7.29	7.41	
HCO3	15.6 mEq/L,	18.8	
Anion Gap	9 mEq/L	5 mEq/L	
iPTH	14.9 (11-79)		

Patient was evaluated for low HCO3 with high urinary pH. Patient had kidney stones despite low serum calcium. iPTH in our patient was also low which showed that it cannot be due to secondary hyperparathyroidism. We searched literature for the same and most common cause for this presentation was distal RTA.

We finally made the diagnosis of type1 DM with diabetic neuropathy, retinopathy and nephropathy with distal RTA causing nephrocalcinosis.

DISCUSSION

Type 1 diabetes mellitus (T1DM) is a chronic auto-immune disease with genetic susceptibility and progressing to complete destruction of β-cells. Auto-antibodies against the pancreatic cells like islet cell antibodies (ICA) and glutamic acid decarboxylase (GAD) are detected in childhood in T1DM patients.[3] It is usually a disease of childhood/young. According to 2009 census, 6666 of 3.[4] million youth were diagnosed with type 1 diabetes for a prevalence of 1.93 per 1000. The highest prevalence of T1DM (2014 census) was 2.55 per 1000 among white youth and the lowest was 0.35 per 1000 in American Indian youth.[4]

Distal renal tubular acidosis is a syndrome of systemic hyperchloremic acidosis with alkaline urine pH, hypocitraturia and hypercalciuria due to reduced secretion of H+ ions by the cells of the distal convoluted tubules.[5,6] Metabolic acidosis in distal renal tubular acidosis contributes as a predisposing factor to recurrent nephrolithiasis and bone loss6. Patients with distal RTA are unable to lower urine pH normally in the presence of systemic metabolic acidosis regardless of its severity.[7]

15-30% of subjects with T1DM have autoimmune thyroid disease, 4–9% had celiac disease, and 0.5% had Addison's disease3. Distal RTA is also multifactorial; it can develop as a consequence of autoantibodies, most commonly with Sjögren's syndrome and systemic lupus erythematosus (SLE). There are various case reports which had linked distal RTA with autoimmunity and destruction of distal convoluted tubules.[8] There are only few case reports with patients of T1DM who develop distal RTA, in which one patient had due to autoimmunity and other patient had Sjogren syndrome and no cause could be found in some other.[9-11]

Our patient had all features of distal RTA in the form of nephrocalcinosis, acidic urine, low bicarbonate, metabolic acidosis which persist even after the correction of blood sugars. He had proteinuria and slightly deranged creatinine which suggested diabetic kidney disease (DKD); but destruction of glomeruli alone cannot explain the development of distal RTA in our patient. Various studies previously had already shown that diabetic patient have interstitial and tubular injury along with glomerular injury.[12-14]

Additional investigation like kidney biopsy could have been for further confirmation of destruction of distal convoluted tubules to prove tubular damage but our patient refused for procedure.

CONCLUSION

Distal RTA patient improves with alkali therapy and it also prevents the formation of renal stones. Our patient already has developed renal stones; still alkali therapy will prevent the progression of stones. We hence conclude our case which was unusual as every T1DM doesn't have acidosis due to DKA. Also we suggest that if a patient had hypokalemia and nephrocalcinosis, distal RTA should be ruled out by urine pH and blood HCO3 levels. More focused research is required in this field.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Atkinson MA, Eisenbarth GS, Michels AW. Type 1 diabetes. Lancet. 2014;383(9911):69-82.
2. Both T, Zietse R, Hoorn EJ, van Hagen PM, Dalm VA, van Laar JA, van Daele PL. Everything you need to know about distal renal tubular acidosis in autoimmune disease. Rheumatol Int. 2014;34(8):1037-45.
3. Barker JM. Clinical review: Type 1 diabetes-associated autoimmunity: natural history, genetic associations, and screening. J Clin Endocrinol Metab. 2006;91(4):1210-7.
4. Dabelea D, Mayer-Davis EJ, Saydah S, Imperatore G, Linder B, Divers J, et al. Search for Diabetes in Youth Study. Prevalence of type 1 and type 2 diabetes among children and adolescents from 2001 to 2009. J Am Med Assoc. 2014;311(17):1778-86.
5. Hess B (2006) Acid–base metabolism: implications for kidney stones formation. Urol Res. 2006;34(2):134–8.
6. Arampatzis S, Röpke-Rieben B, Lippuner K, Hess B. Prevalence and densitometric characteristics of incomplete distal renal tubular acidosis in men with recurrent calcium nephrolithiasis. Urol Res. 2012;40(1):53-9.
7. Batlle DC, Sehy JT, Roseman MK, Arruda JA, Kurtzman NA. Clinical and pathophysiologic spectrum of acquired distal renal tubular acidosis. Kidney Int. 1981;20(3):389-96.
8. Konishi K, Hayashi M, Saruta T. Renal tubular acidosis with autoantibody directed to renal collecting-duct cells. N Engl J Med. 1994;331(23):1593-4.
9. Maeng M, Pedersen KM. A young woman with metabolic acidosis and recently discovered IDDM without ketouria. A rare autoimmune (?) combination of hypothyroidism, diabetes mellitus and distal tubular acidosis. Ugeskr Laeger. 1998;21;160:5663–566-4.
10. Raddatz V, Alvo M, Durruty P, Orellana L, Garcia de los Rios M. Decompensated diabetes mellitus and hyperchloremic metabolic acidosis: a case with both pathologies. Rev Med Chil. 1998;126:1224-8.
11. Dymot JA, McKay GA. Type 1 (distal) renal tubular acidosis in a patient with Type 1 diabetes mellitus--not all cases of metabolic acidosis in Type 1 diabetes mellitus are due to diabetic ketoacidosis. Diabet Med. 2008;25(1):114-5.
12. Gilbert RE, Cooper ME. The tubulointerstitium in progressive diabetic kidney disease: more than an aftermath of glomerular injury? Kidney Int. 1999;56(5):1627-37.
13. Magri CJ, Fava S. The role of tubular injury in diabetic nephropathy. Eur J Intern Med. 2009;20(6):551-5.
14. Zeni L, Norden AGW, Cancarini G, Unwin RJ. A more tubulocentric view of diabetic kidney disease. J Nephrol. 2017;30(6):701-17.

Cite this article as: Kant R, Garg m, Meena MK. Type 1 diabetes mellitus presenting as distal renal tubular acidosis (type 1). Int J Adv Med 2021;8:283-5.