Case Study

A Rare Case Of Renal Glycosuria In A 3 Years 2 Months Old Child

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ABSTRACT

We present a 3 year old male child otherwise healthy and developmentally normal incidentally found to have 3+ urine glucose on dipstick. His blood sugar & HbA1C was normal. There was no other evidence of any tubulopathy. Hence the possibility of Non-diabetic Renal Glycosuria was kept, which is a benign & self-limiting condition.

CASE PRESENTATION

A 3 year old male child born to a non-consanguineous parents came to our O.P.D with complaint of fever. Routine blood & urine investigations were sent. Child was otherwise healthy with normal developmental milestones. His weight and height were plotted on growth charts & found to be with in normal limits. Clinical examination was also normal. To our surprise his urine sugar came 3+, rest all investigations were normal. Child was treated symptomatically for viral fever & when the fever subsided urine routine examination was repeated, which again showed 3+ sugar by dipstick while all other parameters like leucocytes, nitrates, urobilinogen, blood, protein & even ketones was negative. So his fasting sugar & HbA1C was done which came as 85mg/dl & 4.6% respectively which were normal. Additional investigations were then performed which includes blood urea, creatinine, electrolytes and uric acid. All of which were with in normal limits.

Since the child was asymptomatic now, so he was kept in follow up. IN follow up after about a month. His blood sugar & Hba1C was repeated along with urine routine examination. His blood sugar was 90mg/dl & Hba1C was 4.5% but, urine again showed 3+ sugar in it. Urine was also analysed for phosphate, creatinine, & sodium also but, all were within normal limits. Both the parents were also tested for glycosuria which was absent on dipstick testing.

In view of glycosuria in the absence diabetes and without any features of tubulopathies, the diagnosis of benign renal glycosuria was made.

DISCUSSION

With normal renal tubular function, glucose will only be excreted in urine when blood glucose concentrations are elevated, and the renal tubular re-absorptive capacity for glucose is exceeded. Glucose transport in human kidney and intestine is mediated by sodium-coupled glucose transporters, termed SGLT, and glucose transporters, termed GULT. Glucose is transported across the brush border in to the epithelial cell via SGLT, and across the baso-lateral aspect out of epithelial cell by GLUT. The intestine and kidney share high-affinity Na+/glucose co-transporters (SGLT1), but a low affinity Na+/glucose co-transporter (SGLT2) is kidney specific. In the normal nephron, filtered glucose is reabsorbed in the proximal convoluted tubule (PCT), about 90% being reabsorbed in the first segment of PCT by high-affinity SGLT1, and the remainder in the second and third segments of the PCT by low-affinity SGLT2[1].

Renal glycosuria (also known as benign glycosuria or non- diabetic glycosuria) is a benign, inherited condition in which glucose is excreted in urine despite normal blood glucose concentrations. The condition is asymptomatic and self-limiting, and is usually only discovered incidentally. However, glycosuria may be associated with other

tubulopathies, such as Fanconi's syndrome, Cystinosis, Wilson'sdisease , hereditary tyrosinaemia and oculocerebrorenal syndrome (Lowe's syndrome). These other tubulopathies, however, are associated with growth failure, muscle dystonia, polyuria, polydipsia, dehydration or ocular defects (glaucoma, cataracts). In renal glycosuria no other renal tubular dysfunction is present[5] .

Benign glycosuria can be of two types:

Type A: Classic glycosuria caused by a reduced tubular threshold and maximal reabsorptive rate for glucose .

Type B: Normal maximal reabsorpitive rate for glucose, but reduced tubular threshold.

Plasma glucose, glucose tolerance, insulin and HbA1c are all normal, and all other renal tubular abnormalities are absent in both types[5].

The molecular mechanism giving rise to benign renal glycosuria has been discovered to be due to a defect in the low-affinity SGLT2 in the proximal convoluted portion of renal tubules[3,4]. Inheritance is autosomal recessive, but because the condition is asymptomatic, a family history is often non-contributory.

Suggested investigations for suspected cases of renal glycosuria include blood tests for urea, creatinine and electrolytes, calcium, phosphate, uric acid, glucose, and measurement of the HbA1c. Urine should be sent for urinalysis and microscopy, and measurement of phosphate, and the tubular maximum of phosphate reabsorption should be calculated. In cases where another tubulopathy is suspected on the basis of abnormal laboratory results or clinical findings, a 24-hour urine specimen should be collected and sent for amino acid analysis. Refferal to a pediatric nephrologist is then recommended[5].

Renal glycosuria requires no treatment or special dietary restrictions, and the prognosis is excellent. Once diabetes mellitus and other renal tubular disorders are excluded, all that remains is to explain the condition, and reassure the parents5.

CONCLUSION

The case illustrates an unusual cause of glycosuria, the commonest cause of which is uncontrolled diabetes mellitus. In young non-obese children type 1 diabetes mellitus is the commonest type of diabetes, where the absence of insulin production and secretion from the beta cells in pancreatic islets causes hyperglycemia and ketosis. Renal glycosuria is a benign autosomal recessive condition caused by a defect in SGLT2 in the PCT of the nephron, which allows renal glycosuria to occur in the absence of a raised serum glucose level. Benign renal glycosuria is not associated with other renal tubular abnormalities. The condition is benign, and asymptomatic, and no treatment or diteary restriction is necessary.

REFERENCES

1. Kanai Y, Lee WS, You G, Brown D, Hediger MA. The human kidney low affinity Na+/glucose cotransporter SGLT2. Delineation of the major renal reabsorptive mechanism for D-glucose. The Journal of clinical investigation. 1994 Jan 1;93(1):397-404.
2. Burtis CA, Ashwood ER, Bruns DE. Tietz textbook of clinical chemistry and molecular diagnostics-e-book. Elsevier Health Sciences; 2012 Oct 14.
3. van den Heuvel LP, Assink K, Willemsen MA, Monnens L. Autosomal recessive renal glucosuria attributable to a mutation in the sodium glucose cotransporter (SGLT2). Human genetics. 2002 Dec 1;111(6):544-7.
4. Magen D, Sprecher EL, Zelikovic I, Skorecki K. A novel missense mutation in SLC5A2 encoding SGLT2 underlies autosomal-recessive renal glucosuria and aminoaciduria. Kidney international. 2005 Jan 1;67(1):34-41.
5. Wilgen U, Cobb H. An unusual cause of glycosuria in a 5-year-old child. South African Journal of Child Health. 2008;2(3).