CASE REPORT

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A Case with Renal Glycosuria

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ABSTRACT Glycosuria is defined as the detectable amount of urinary glucose excretion. When this laboratory finding is noted, fasting plasma glucose level should be checked. In the absence of hyperglycemia, renal glycosuria should be thought. Female patient with the age of 33 years old applied to our hospital with the complaint of constipation. Laboratory tests revealed glycosuria without hyperglycemia. Twenty four hour urine collection was made and 47.8 g glucose was detected. Her physical examination and other laboratory tests including HOMA- insulin resistance, arterial blood gas levels, phosphate and uric acid levels were completely normal. So the case was diagnosed as familial renal glycosuria. Renal glycosuria is usually harmless condition and does not need any specific therapy except prevention of dehydration and candidial infections. A satisfactory diagnosis of renal glycosuria can not be reached without elimination of diabetes mellitus, Fanconi syndrome, drug history and pregnancy.

Keywords: Familial renal glycosuria; innocent polyuria; euglycemia

Under normal conditions, kidneys assist glucose homeostasis by synthesizing glucose via gluconeogenesis and preventing urinary glucose loss via tubulary glucose reabsorption.[1] Reabsorption of glucose which is freely filtered in the glomeruli almost completely occur in proximal tubules only. This glucose reabsorption is mostly (90%) carried out by sodium-glucose cotransporters type 2 (SGLT-2) and glucose transporters 2 (GLUT2). So in patients with normal kidney function, significant glycosuria does not generally occur until the plasma glucose concentration exceeds 180 mg/dL (10 mmol/L).[2]

Glycosuria may be due to either the inability of the kidney to reabsorb filtered glucose in the proximal tubule despite normal plasma glucose concentration, or to an overflow scenario related to high plasma glucose concentrations overwhelming the capacity of the renal tubules to reabsorb glucose. The commonest cause of glycosuria is uncontrolled diabetes mellitus. Abnormally elevated levels of glucose in the blood result in high amount of filtered glucose waiting to be reabsorbed through overactive SGLT2 which execute reabsorption eventually. When glycosuria occurs with a normal plasma glucose, a primary defect of proximal tubule reabsorption (renal glycosuria) needs to be considered.[1,2] Renal glycosuria is a rare and long

known condition.[3] Although there is no current epidemiological information about incidence of renal glycosuria, it was proposed to be approximately 0.29 %.[4]

Here, 33 years old woman with an unusual cause of glycosuria was presented. The patient gave consent for publishing her medical information.

CASE REPORT

A 33 years old female patient applied to gastroenterology clinic with the complaint of constipation and bloating. Routine urine analysis revealed glycosuria. So patient was directed to our nephrology department for further analysis. In her medical history, since her childhood dry mouth, polydipsia, polyuria and frequent perineal candidal infections have been present. There was no consanguinity between her parents. Parents of the patient were screened and no glycosuria was found. Except vaginal topical gel which includes clorhexidine + hyaluronic acid (gynelaude mucus) she had not taken any medicine. In her physical examination, arterial blood pressure was 110/70 mmHg (measured from both arms) and no pathological finding was detected. Urine analysis was repeated and glucose was present with the concentration of 1000 mg/dL, while density of urine was 1.037. Pregnancy test was negative. Biochemical analysis revealed normal findings: fasting blood glucose: 86 mg/dL, HbA1c: 5.4%, C-peptide: 2.21 ng/mL (0.9-7.1), insulin level: 8.35 mIU/mL (<29.1), HOMA-IR (insulin resistance): 1.77 mg/dL (<2.5), serum uric acid level: 2.8 mg/dL, phosphorus level: 3.54 mg/dL, bicarbonate level: 25.5 mmol/L, Na: 137 mEq/L, K: 4.3 mEq/L, BUN:14 mg/dL, creatinine: 0.56 mg/dL, glomerular filtration rate with formula of CKD-EPI: 124 mL/min/1.73 m^2, creatinine clearence with 24-hour urine collection: 131.1 mL/min, proteinuria 96.7 mg/day, albuminuria <13.2 mg/day, fractitional uric acid excretion 10.1%, phosphorus excretion 10.7%. The glucose level in a 24-hour urine sample was found to be 47.8 g/day. Daily glomerular glucose filtration of the patient was calculated as 153.6 g. Glucose excretion was estimated as approximately 31%. By ultrasonography and direct urinary system graphy nephrolithiasis was not detected. Nephrolithiasis was also absent in her past and familial history. So no cysteinuria analysis was required.

DISCUSSION

Plasma glucose is filtered freely through glomerular barrier.[2] Under normal circumstances, kidneys filter and reabsorb nearly 100% of filtered glucose, approximately 180 g (1 mole) each day.[1] This way kidneys contribute to glucose economy of the body in addition to renal gluconeogenesis (15-55 g/day).[5] In the urine of healthy adults, less than 0.5 g/day glucose is excreted thanks to glucose transporters expressed in the renal proximal tubule[1]. So with proper renal functioning, the expected urinary glucose amount is 0 to 0.8 mmol/L (millimoles per liter). When glycosuria occurs, it is due to either glomerular hyperfiltration of glucose exceeding proximal tubule reabsorptive capacity which results from diabetes mellitus or usually inherited deficiency of tubule membrane glucose transport proteins which is defined as renal glycosuria. Renal glycosuria is the urinary excretion of abnormal amount of glucose, while maintaining a normal glucose concentration.[6] This identity can be recognized among people with familial renal glycosuria which is a rare tubular disorder or in patients receiving drugs such as tenofovir.[7] Mutations with the renal sodium glucose cotransporter 2 (SGLT2) gene (in other words SLC5A2 gene) lead to familial renal glycosuria.[4] This gene is mapped to chromosome 16p11.2.[8] Renal glycosuria is harmless and it is also called as diabetes innocens[3]. Renal glycosuria was also reported not to be associated with atherosclerotic cardiovascular diseases.[9] However, episodic dehydration and ketosis during pregnancy and starvation, polyuria, enuresis and an increased incidence of urinary tract infections were reported with this situation in the literature.[10,11] For proper diagnosis of familial renal glycosuria; diabetes mellitus, Fanconi syndrome (amino aciduria, phosphaturia, bicarbonaturia, cystinuria, proteinuria), pregnancy, drug history and acute tubular necrosis should be searched in differential diagnosis.[9]

In our case, diagnostic work out revealed isolated renal glycosuria in the absence of other abnormalities listed above. With this case we aim to remind clinicians a very rare cause of glycosuria.

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Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

Authorship Contributions

Idea/Concept: *Kübra Kaynar, Canan Şehit, Arif Mansur Coşar, Gül Cebecioğlu Hasançebi;* ***Design:*** *Kübra Kaynar, Canan Şehit, Arif Mansur Coşar, Gül Cebecioğlu Hasançebi;* ***Supervision/Consulting;*** *Kübra Kaynar; Canan Şehit;* ***Data Collection and/or Processing:*** *Kübra Kaynar; Canan Şehit;* ***Analysis and Interpretation:*** *Kübra Kaynar; Canan Şehit;* ***Source Browsing:*** *Kübra Kaynar; Canan Şehit;* ***Writing of the Makalene:*** *Kübra Kaynar; Canan Şehit;* ***Critical Review:*** *Kübra Kaynar; Canan Şehit;* ***Resources and Funding:*** *Kübra Kaynar; Canan Şehit;* ***Materials:*** *Kübra Kaynar; Canan Şehit.*

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