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Glycosuria: A Review

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Introduction

Glycosuria is a term that defines the presence of reducing sugars in the urine, such as glucose, galactose, lactose, fructose, etc. Glucosuria connotes the presence of glucose in the urine and is the most frequent type of glycosuria and is the focus of this review. It happens when the glomerulus filters more glucose than the proximal tubule can reabsorb. In normal individuals, glucosuria can be up to 0.25 mg/ml. More than 0.25 mg/ml in random fresh urine is considered increased glucosuria and can be due to elevated plasma glucose or renal glucose absorption impairment, or both. Physiologic glucosuria is a condition where individuals consume an excessive amount of carbohydrates [1].

Small amounts of glucose present in the urine are considered normal, but the term glucosuria usually refers to pathologic conditions where the amounts of urine glucose are more than 25 mg/dl in random fresh urine. Normally, the renal tubule will reabsorb almost all (leaving less than 25 mg/dl urine glucose) glucose present in the normal glomerular filtrate [2]. When the glucose filtrated by glomerular exceeds the capacity of the renal tubule to absorb it, the loss of balance occurs. It can happen due to elevated plasma glucose as in diabetes mellitus or when the ability of the tubule to absorb glucose is impaired, e.g., Fanconi syndrome with impairment in the absorption of phosphate, amino acids, or isolated glucosuria as an inherited disorder termed Familial Renal glucosuria.

Mechanism

In healthy individuals, the renal system filters approximately 180 g of glucose daily. The glucose that entered the tubular system is reabsorbed along with the segments of the proximal convoluted tubule (PCT). In diabetic patients, as a result of the increase in plasma glucose, the filtered glucose exceeds the capacity of the tubular system and results in glucosuria. The majority of glucose uptake of greater than 90 percent occurs in the proximal tubule, mediated by SGLT2, a low-affinity/high capacity transporter. The remaining glucose will then be reabsorbed by the distal parts of the proximal tubule via the high-affinity/low-capacity SGLT1 [3].

Kidneys play a significant role in maintaining glucose homeostasis and preventing an individual from developing hypoglycemia. The maintenance of glucose homeostasis by the kidney includes glucose reabsorption in the PCT, gluconeogenesis, and the clearance of important hormones such as insulin.

Pathophysiology

Glucose filters through glomeruli, and then it is reabsorbed by the proximal renal tubule. Less than 0.1% of glucose is not reabsorbed by the kidneys (less than 0.25 mg/ml), and most of the standard test does not detect this level. The causes of glucosuria can be grouped under two classes: the inability of PCT to reabsorb glucose and an increase in the concentration of glucose in the circulating blood [4].

Defects in the PCT, either primary or secondary, can result in glucosuria. Examples include pregnancy, Fanconi syndrome, and acute tubular necrosis. In a normal condition, when the plasma glucose level is increasing, renal tubular reabsorption of glucose will rise linearly until its maximum is reached (ranges from 0.9 to 2.0 mmol/min). As mentioned before, SGLT1, SGLT2, and GLUT2 are the membranes protein that is responsible for glucose reabsorption; mutations in one of these membrane proteins will cause glucosuria. Mutation in SGLT1 is associated with glucose-galactose malabsorption, a mutation in SGLT2 is associated with familial renal glucosuria (FRG), and mutation in GLUT2 is associated with Fanconi-Bickel syndrome.

Glucosuria can also occur in an increased concentration of glucose in the circulating blood. This phenomenon can also occur in normal individuals who consume excess carbohydrates, known as 'alimentary glycosuria.' It also presents in diabetic patients. In diabetes mellitus, with increasing duration, glomeruli can be damaged, resulting in albuminuria and a decrease in the glomerular filtration rate [5]. In diabetic patients, the kidneys are more susceptible to the effects of hyperglycemia; many of the kidney cells are unable to decrease glucose transport rates and unable to prevent intercellular hyperglycemia in an increased glucose concentration state.

Some conditions are known to raise the renal threshold for glucose, such as age, renal disease (diabetic glomerulosclerosis), heart failure, and chronic hyperglycemia. Also, some conditions are known to decrease it, such as hyperthyroidism, pregnancy, fever, and exercise. A number of substances are also known for their capability to cause glucosuria, such as chloride, iodide, bromide, and nitrate of sodium. Glucosuria can also occur in a condition where there is a lack of oxygenation of the PCT.

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