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Microalbuminuria as a marker of kidney damage in patients with diabetes mellitus.

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Abstract

Diabetes mellitus has increased its incidence and prevalence gradually in recent years throughout the world. Currently, diabetes mellitus and high blood pressure are the main causes of chronic kidney failure in its different stages. Nephropathy is one of the most serious complications affecting diabetics. Its onset is usually insidious, progresses without symptoms, and generally becomes clinically evident after 5 to 10 years of diabetes progression. This complication in early stages (incipient nephropathy) can be diagnosed through microalbuminuria, which is currently the first marker that exists to detect the existence of the condition. This review aims to summarize the importance of the determination of microalbuminuria in diabetic patients with the aim of making an early diagnosis of the disease and the control of risk factors.

Keywords: *diabetes mellitus, nephropathy, microalbuminuria.*

Introduction

Diabetes is a chronic disease characterized by the inability of the pancreas to produce insulin in sufficient quantities or by its inefficient use. It is classified as a worldwide epidemic. [1,2].

Diabetes mellitus as a cause of chronic kidney disease has increased its incidence and prevalence gradually in recent years throughout the world. Calculations have been made regarding the global number of diabetic patients, it has been estimated that they could reach 366 million people, especially at the expense of type 2 diabetes mellitus. According to estimates by the World Health Organization, by 2030 this figure could double and group between 85-90% of cases of diabetes mellitus. In the United States of America, diabetes mellitus is the most frequent cause in more than 40% of patients admitted to dialysis. In the Latin American region, it reaches between 58 and 60% in Mexico, 42% in Venezuela, 35% in Colombia and 28% in Cuba. [3,4,5]. This last figure is the figure with a tendency to a progressive increase in diabetics who reach terminal chronic renal failure.

Between 25 and 40% of diabetic patients will have some degree of nephropathy throughout their evolution, a prevalence that will depend on numerous factors involved in its pathogenesis (genetics, time of evolution of diabetes, degree of glycemic control, adequate management or not of blood pressure, dyslipidemia, smoking, appearance of microalbuminuria and progression towards macroalbuminuria), which will mark the evolution towards established nephropathy.[2] In the past, nephropathy was considered a non-immunological disease, but studies have shown that inflammatory and immune processes are involved in the pathogenesis of this disease. Several mechanisms involved in the appearance of nephropathy in diabetic patients are known, such as: advanced glycosylation products, oxidative stress, polyol pathway, protein

kinase C, renin angiotensin aldosterone system, growth factors, inflammation and epigenetics. [3,4]. The best mechanism to prevent progression would be to maintain an adequate glycemic level, when this is not achieved, a pathological biological response is triggered that will lead to the formation of products that cause damage at the microvascular level.

Early diagnosis of diabetic nephropathy continues to be a problem for specialists, because patients come late for diagnosis. Diabetic nephropathy is classically defined by the presence of proteinuria.

The presence of a higher-than-normal amount of albumin in the urine is due to an increase in the passage of albumin through the glomerular barrier that exceeds the tubular capacity to reabsorb albumin. This seems to be due, more than to a specific kidney injury, to a diffuse endothelial injury, which favors the increase in endothelial permeability in vessels of different locations, behaving as a facilitating factor for the development of arteriosclerosis, thus explaining the increase in risk of developing cardiovascular disease and retinopathy [6]. microalbuminuria is the earliest marker of kidney disease and increased cardiovascular risk. [1,2]. Microalbuminuria is the urinary excretion of albumin between 30 and 300 mg/24 hours (20-200 ug/min), 20-200 mg/L or an albumin/creatinine ratio of 20 mg/mmol or 30-300 mg/g. According to the Canadian Diabetes Association, after 2-3 abnormal samples in a 3–6-month period.[3]. Microalbuminuria is considered to be a risk factor for developing macroalbuminuria (albuminuria greater than 30-300 mg/g), the amount of which correlates with the involvement of the podocyte processes and the size of the filtration pore and marks the beginning of the loss of kidney function. In this phase there is a progressive decrease in the glomerular filtration rate with evolution to End-Stage Renal Disease. It is said that the higher the albuminuria, the higher the risk of kidney disease progression, complication and mortality.[4]. These aspects motivated the realization of the following bibliographical revision with the objective of summarizing the importance of the determination of microalbuminuria in diabetic patients to carry out an early diagnosis of diabetic nephropathy and the control of risk factors.

Developing

The term microalbuminuria was introduced by Viberti et al to describe the presence of albumin in urine in values higher than those considered normal, but which are not quantifiable by usual laboratory methods. Microalbuminuria is present in a wide variety of physiological and pathological processes; in some of the latter, it is considered a prognostic indicator of kidney damage. Albumin is a negatively charged protein that is filtered in small quantities in the glomerulus and that under normal conditions appears in the urine in concentrations below 20 mg/day, thanks to its tubular absorption [7,8].

Microalbuminuria may increase or appear transiently (intermittent albuminuria) in relation to poorly controlled hypertension, hyperglycemia, physical exercise, urinary tract infections, pregnancy, hypervolemia or protein overload. Other circumstances in which an increase in microalbuminuria has been shown are related to age, race or obesity, and a circadian rhythm in albumin excretion has been described, which decreases during the night, attributable to a drop in blood pressure (PA). [9,10,11].

Several semi-quantitative tests have been described for the determination of MA based on: turbidity, colorimetry and the agglutination of latex particles, for the latter a sensitivity and specificity of 95% are indicated. There are also reagent strips that detect other proteins and a specificity of 80-90% and a sensitivity of 90-95 are declared, the choice of the type of system depends on the equipment available and the experience of the laboratory staff.[12].

Urine samples for albuminuria should not be collected after exercise or after an acute fluid load. Nor should the determination be made if the patient has poor diabetic control, as this increases the rate of albumin excretion. Albumin excretion should not be assessed if the patient has a urinary tract infection. Acute illness with fever also increases the rate of albumin excretion. Additionally, it is recommended that patients not be tested during menstruation, or when experiencing any other vaginal discharge, due to likely sample contamination.

Among the ways used to determine microalbuminuria

• Albumin determination in a 24-hour urine sample: represents the reference value for urinary albumin determination, since it accurately reflects the amount of albumin eliminated in 24 hours. It has the drawback that the collection of the entire amount of urine emitted in 24 hours is complex and incomplete collection is frequent, which can lead to errors. Microalbuminuria is considered at values of 30 to 300 mg/24 hours (20 to 200 µg/min).

• Albumin determination in a 12-hour or overnight urine sample: Urine collection over a known period of time allows the daily albumin excretion to be calculated fairly accurately. Compared to the 24-hour collection, it has the advantage that since it is the

shorter period, it is easier for the collection to be complete, and the margin of error is smaller. In addition, if the period collected is at night, the values obtained are more realistic, since the effect of physical exercise and standing on the urinary elimination of albumin is avoided. The drawback is that when multiplying by a correction factor to get the 24-hour value, any errors made are multiplied by that factor. Microalbuminuria is considered at values of 30 to 300 mg/24 hours (20 to 200 µg/min).

Determination of albumin in an isolated urine sample: the determination of microalbuminuria in an isolated urine sample often leads to errors due to the influence, as we have already mentioned, of the urinary concentration on the microalbuminuria values. To avoid these, attempts have been made to evaluate microalbuminuria in the first morning urine. Although this value correlates best with 24-hour urinary albumin excretion, it continues to vary widely. Microalbuminuria is considered at values of 20 to 200 mg/L.

• Determination of the albumin/creatinine ratio (Alb/Cr) in an isolated urine sample: when the urinary albumin concentration is corrected for creatinine excretion, the problem of the influence of urinary volume is obviated, since its effect is similar on the numerator and denominator. This estimate correlates significantly with 24-hour urinary albumin excretion. It has the advantage that it avoids the collection of several urine samples and therefore the error to which urine collection over a long period of time may be subject. If it is also performed in the first morning urine, the correlation with albumin excretion is even higher. It has the disadvantage that depending on two determinations, it can increase the errors depending on the technique; in those subjects whose creatinine excretion differs from the average (muscular or cachetic), protein losses may be underestimated or overestimated. [13].

To classify nephropathy in the early stages (1 and 2), kidney damage markers such as albuminuria and hematuria are required, since renal function may be normal or slightly decreased.[4].

The finding of microalbuminuria is therefore an indication to search for renal and vascular disease for aggressive intervention on risk factors. Some studies show that glycemic control by maintaining glycosylated hemoglobin levels below 7% is a good argument for reducing kidney damage. [14,15,16].

On the other hand, it has been shown that converting enzyme inhibitors (ACE inhibitors) administered to diabetics with microalbuminuria and normal blood pressure can normalize urinary albumin levels in some patients, while in subjects with established nephropathy they reduce proteinuria and slow evolution. [17,18,19]. Hence the importance of reducing intraglomerular pressure to hinder the loss of albumin.

Conclusions

Diabetic nephropathy is a common complication of diabetes mellitus, and is the main cause of chronic renal failure, it also has a high morbidity and mortality. Early detection in the microalbuminuria stage is very important since management of risk factors at this stage prevents or delays progression to clinical nephropathy.

Recommendations

Carry out research on educational interventions or strategies in Primary Health Care with the aim of reducing the percentage or reducing complications in patients with diabetic nephropathy and thus reduce chronic renal failure, which results in a better quality of life for patients.

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