Editorial

Glycated albumin for glycemic control of diabetic patients on hemodialysis

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Regular assessment of glycemic control in diabetic patients is very important, because it reduces the incidence of complications and determines prognosis and quality of life of the patients.[1]

Uncontrolled and advanced diabetes may lead to complications, such as kidney disease, heart attacks, and stroke, which are on the increase worldwide. Patients with diabetic nephropathy account for almost 50% of dialysis patients in the US, while cardiovascular disease is the main cause of death of patients with complicated diabetes.[2]

Assessment of glycemic control status in diabetic patients may be performed by determining the glycation of several proteins, which is increased in diabetic patients as compared with nondiabetic patients. Among these glycated proteins, glycated hemoglobin (HbA1c) is the gold standard index for glycemic control in the treatment of patients with diabetes.[3] HbA1c is the condensation product of hemoglobin A and glucose, and its level is defined as the percentage of hemoglobin in the circulation that is bound to glucose.[1,2]

Measurement of HbA1c concentration in diabetic patients with nephropathy may be inaccurate (yield lower values) due to shortened erythrocyte life span, which causes decreased chemical binding of glucose to erythrocytes, resulting in low HbA1c concentrations.[2]

Glycated albumin (GA) may be an alternative marker for glycemic control in diabetic patients undergoing hemodialysis that is more accurate than HbA1c.[1,2,4] Since human serum albumin turnover is faster (with a half life of 15-20 days) than that of hemoglobin, determination of glycated albumin yields a glycemic control index over a shorter period of time (2 weeks) in comparison with HbA1c (4 months). HbA1c is not suitable for evaluation of glycemic control in the short term, due to the long erythrocyte life span of 120 days.[5] HbA1c is also inaccurate for determination of glycemic control status in patients with anemia and variant hemoglobins.[3]

In hemodialysis patients with low hemoglobin concentrations, administration of high doses of erythropoietin may shorten the erythrocyte life span, thus also lowering HbA1c levels. This is in contrast with glycated albumin, which is not associated with hemoglobin turnover or erythropoietin dose.[2] A variety of data indicate that HbA1c is not an ideal index for glycemic control in diabetic patients undergoing hemodialysis and receiving erythropoietin, as approximately 90% of dialysis patients receive erythropoietin therapy.[1] Fukuoka et al. reported that glycated albumin (GA) was correlated with life span in Japanese dialysis patients with diabetes and end stage renal disease (ESRD).[6]

Glycated albumin is a ketoamine formed by the binding of albumin and glucose in a nonenzymatic oxidation reaction. Measurement of glycated albumin is not influenced by hemoglobin metabolic disorders. The percentage (%) of glycated albumin is calculated from (glycated albumin/total serum

albumin) x 100. Determination of this parameter is a more accurate test for glycemic control in diabetic patients on hemodialysis and the percentage of glycated albumin is an accurate predictor of morbidity and mortality in diabetic patients undergoing hemodialysis.[2]

In the study by Inaba et al. on hemodialysis patients with diabetes, the degrees of glycemic control based on HbA1c values were categorized as follows: excellent (HbA1c ≤6%), good (6.0 <HbA1c ≤7%), fair (7.0 < HbA1c ≤8.0%), and poor (HbA1c > 8%). Since GA values were three times greater than HbA1c, glycemic control based on GA was categorized as: excellent (GA ≤18%), good (18.0 < GA ≤21.0%), fair (21.0 < GA ≤24.0%) and poor (GA >24.0%).[1]

Glycated albumin is used for glycemic control in diabetic patients on hemodialysis, because GA is not affected by renal anemia.[3] GA also contributes directly to vascular damage and is therefore better than HbA1c in predicting the development of vascular complications in diabetic patients on hemodialysis, although determination of GA also has its limitations, e.g. in liver disorders and metabolic disorders of albumin.[1]

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