

Advanced glycation end products in clinical nephrology.

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As a result of oxidative and carbonyl stress, advanced glycation end products (AGEs) are involved in the pathogenesis of severe and frequent diseases and their fatal vascular/cardiovascular complications, i.e. diabetes mellitus and its complications (nephropathy, angiopathy, neuropathy and retinopathy, renal failure and uremic and dialysis-associated complications), atherosclerosis and dialysis-related amyloidosis, neurodegenerative diseases, and rheumatoid arthritis. They are formed via non-enzymatic glycation which is specifically enhanced through the presence of oxidative and carbonyl stress, and their ability to form glycoxidation products in peptide and protein structures finally modulating or inducing biological reactivity. Food can be another source of AGEs; however, high serum AGEs in hemodialysis patients might reflect nutritional status better. Several methods of renal replacement therapy have been studied in connection with the AGE removal, but unfortunately the possibilities are still unsatisfactory even if high flux dialysis, hemofiltration, or hemodiafiltration give better results than conventional low flux dialysis. AGEs are currently being studied in the patients on peritoneal dialysis as their precursors can be formed in the dialysis fluid. AGEs can cause damage to the peritoneum and so a loss of ultrafiltration capacity. Many compounds give promising results in AGE inhibition (inhibition of formation of AGEs, inhibition of their action or degradation of AGEs), are tested for these properties, and eventually undergo clinical studies (e.g. aminoguanidine, OPB-9195, pyridoxamine, antioxidants, N-phenacylthiazolium bromide, antihypertensive drugs, angiotensin-converting enzyme inhibitors and angiotensin II receptor-1 antagonists). Copyright 2004 S. Karger AG, Basel

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