Dialysis-related amyloidosis: role of advanced glycation end product-β2-microglobulin in joint inflammation

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Dialysis-related amyloidosis is a serious complication for patients undergoing long-term hemodialysis. Amyloid deposits composed of β2-microglobulin as the major constituent protein are mainly localized in joints and periarticular bone and lead to destructive arthropathy. The pathobiology of dialysis-related amyloidosis is still incompletely understood. Although recent histologic studies have shown the accumulation of monocytes/macrophages around amyloid deposits, the factor(s) causing their infiltration and pathologic involvement has yet to be fully elucidated. Accumulating evidence suggests that β2-microglobulin modified with advanced glycation end products has a key role in recruitment and activation of macrophages through an advanced glycation end product receptor-mediated pathway. Thus, dialysis-related amyloidosis arthropathies may result from progressive accumulation of advanced glycation end products in long-lived amyloid linked to a heightened cellular response. Antagonism of the interaction between advanced glycation end products and their receptors may be a relevant strategy for cellular inflammation in dialysis-related amyloidosis. (Hong Kong J Nephrol 2002;4(2):73-77)

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