Dear Sir,

In a recent letter to the Editor of this journal, Sethi et al. [1] report 2 cases of β2-microglobulin (β2-M) amyloidosis of the sternoclavicular joint (SCJ) in hemodialysis patients, presenting as acute osteoarthritis. No evidence of bacterial infection could be demonstrated histologically, but unfortunately it appears that the biopsy material has not been cultured. The authors also claim that, to the best of their knowledge, the involvement of the SCJ has not been previously described with dialysis amyloid. However, the presence of β2-M amyloid deposits in the SCJ in 9 chronic hemodialysis patients has recently been reported by our group at the 20th meeting of the American Society of Nephrology (Washington, D.C., December 1987) and at the 4th meeting of the British Society of Rheumatology (London, UK, November 1987). Our preliminary findings have been published in abstract form [2].

In our study, amyloid deposits of the SCJ had been systematically sought during neck surgery in 22 hemodialysis patients. Of the 22 subjects included in the study, 13 had been on chronic hemodialysis for more than 10 years. Eight of them (62%) had amyloid deposits in their SCJ biopsy that reacted with an anti β2-M antibody, stressing the high incidence of β2-M amyloidosis in patients undergoing hemodialysis for a long period of time. The 2 cases reported by Sethi et al. had both been on hemodialysis for at least 10 years (10 and 15 years, respectively). Hence their observations are in line with the prevalence of β2-M amyloidosis in our patients. However, in our experience, the finding of SCJ amyloidosis was not associated with local inflammatory signs in any case, in contrast to the clinical presentation of the patients of Sethi et al.

Thus, the authors’ assumption of a causal relationship between β2-M amyloidosis and the occurrence of acute arthritis remains questionable. Moreover, the absence of patent, acute inflammation of other joints, though massively involved [3], during β2-M amyloidosis rather pleads against such a hypothesis. However, clinically latent, micro-inflammatory processes, may be involved in the pathogenesis of this serious complication of long-term hemodialysis.

References
