Correspondence

Synovial amyloid deposits and chronic haemodialysis

SIR, In their recent paper Muñoz-Gómez *et al*¹ suggested that localised synovial amyloidosis might be peculiar to chronic renal failure treated with periodical haemodialysis, and they made the assumption that it was the cause of an arthropathy. In fact localised amyloid deposition in synovium and also in articular cartilage is a common aging phenomenon² apparently associated with osteoarthritis, and it is relevant that four of the seven patients studied were aged 50 years or more. This type of aging deposit was actually referred to in the preceding editorial as 'joint capsule and articular cartilage' deposition.³ The nature and extent of synovial amyloid deposits described in the article did not appear to differ from those associated with aging, and I would suggest that the finding of such deposits may represent no more than an accelerated age change. It is of interest in this context that synovial and articular cartilage amyloid deposits in patients with the so called amyloid arthropathy syndrome of multiple myeloma and primary amyloidosis do not appear to differ qualitatively from aging deposits either.⁴ Such patients may, however, show gross soft tissue amyloid deposition around joints.

The finding of synovial amyloid deposits in chronic haemodialysis patients may be unrelated to any arthropathy present and should not prevent other causes for an arthropathy from being sought.

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SIR, Recently, the biochemical nature of amyloid fibrils in haemodialysis associated amyloidosis has been characterised.¹ Amyloid fibrils isolated from tissue containing amyloid obtained during carpal tunnel decompression from a patient undergoing long term haemodialysis were found to contain as a major component a protein

homologous to the normal plasma protein β_2 -microglobulin. This protein has not previously been shown to be a fibril constituent in amyloid deposits. β_2 -Microglobulin is known to accumulate in the circulation of patients undergoing haemodialysis since this procedure does not remove the protein from the plasma,^{2,3} and Gejyo *et al* postulate that sustained increase of plasma concentrations of β_2 -microglobulin leads to the accumulation of protein in tissues, causing the formation of amyloid fibrils.¹ Interestingly, both AL amyloid fibrils⁴ and β_2 -microglobulin derived fibrils appear to have a relatively high affinity for the carpal tunnel, and in both cases precursor proteins are related to immunoglobulins.

It is likely that the amyloid deposits in the joints of patients undergoing long term haemodialysis also contain β_2 -microglobulin as the fibril precursor, and it may be possible to confirm this relatively easily by immunohistochemical techniques. Similarly, although the biochemical nature of amyloid deposits in osteoarthritic joints has not been characterised, it should be possible to determine whether these or any other age related amyloid deposits contain β_2 -microglobulin.

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Combination chemotherapy in rheumatoid arthritis

SIR, Would that the situation as outlined by Dr Bamji were so simple.¹

Hodgkin's disease is a malignancy and in the majority of patients suffering from it the outcome, untreated, is death. Justification for using such toxic regimens is therefore easy and indeed giant strides have been made in improving survival. Rheumatoid arthritis cannot be considered comparable. Whether we are right in considering it to be a non-malignant B lymphoproliferative disease remains to be