RESULTS

Blood levels of C-reactive protein and serum amyloid A protein were both chronically elevated. Serum amyloid P component (SAP)-scintigraphy showed intense uptake in a massively enlarged liver and in the spleen (Figure). An extensive search for an underlying inflammatory process did not show any to be present. Therefore we concluded that morbid obesity most probably was the cause of her AA amyloidosis. Treatment with prednisolone did not substantially reduce the SAA and CRP levels, neither did anakinra (IL1 inhibition) nor tocilizumab (IL6 inhibition). It appeared impossible to reverse the course of the disease and she gradually progressed to complete renal failure within three years. Dialysis was started.

CONCLUSIONS

- This case indicates that longstanding low-grade inflammation seen in morbid obesity may be a potential cause of systemic AA amyloidosis, which observation is also supported by Alisina et al.\textsuperscript{2}
- However it essentially is a diagnosis per exclusionem, since many more usual underlying inflammatory conditions should be ruled out first.