150–350 mg/dl) and vWF, 130 ± 27.4% of normal value (reference 60–150%). In 160 (67%) patients at least one inflammatory marker was found to be increased. Serum ALT did not show significant correlations with any of the APRs studied in either of the above five groups (all \( P > 0.075 \)), including patients with active liver disease (seropositive with ALT ≥291 U/l). On the other hand, the APRs were consistently and notably associated with each other (all \( P < 0.0003 \) as already reported \cite{4,5}). None of the APRs differed between patients with ALT activity in the top quartile vs the lowest quartile (all \( P > 0.119 \)). Patients with the HCV or HBV marker had almost 3x higher median ALT activity than subjects without ALT vs 15 (5–256) IU/l \( P < 0.0001 \); it is noteworthy that the ALT level in the hepatitis-free patients was comparable to those of 15.6 IU/l and 16.3 IU/l reported in previous studies \cite{6,7}. Our hepatitis marker-positive patients presented with slightly lower vWF levels than patients without the markers \( 124 ± 25.1% \text{ vs } 133 ± 28.3\% , P = 0.017 \); the other APRs did not differ between the positive and negative subjects (all \( P < 0.093 \)). The variation in vWF became non-significant when adjusted for cardiovascular disease prevalence. Finally (Table 1), there were no differences in inflammation markers in HD patients with serologically and biochemically evident viral hepatitis compared to subjects without obvious liver pathology.

Our study shows that liver disease does not clearly contribute to systemic inflammation in HD patients. The disparity between this and previous investigation \cite{1} may be due to methodological limitations as well as conditions that are specific for HD therapy. Namely, our cohort was apparently undersized compared to 1740 subjects with metabolic syndrome \cite{1}, and serum CRP was not quantified with the high-sensitivity method. However, the advantage of the present report could be the variety of APRs studied, including not only CRP but also the long-lived reactants and endothelial injury marker vWF. The specific condition that probably masks the association between hepatic and systemic inflammation is repeated stimulation of the inflammatory response by HD procedures themselves \cite{2}. Another potential confounder is the unusual presentation of liver disease in HD patients, in whom serum aminotransferases are inexplicably lower \cite{6,7}, while liver damage caused by hepatitis C virus is significantly less severe than in subjects without renal failure \cite{8}.

Finally, the link between liver disease and systemic inflammation, although not directly evident, is biologically plausible and of potential importance in maintenance HD patients. Further studies are needed before this new trait is definitely excluded.

Conflict of interest statement. None declared.

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doi:10.1093/ndt/gfh804

Advance Access publication 19 April 2005

**Gabapentin for uraemic pruritus**

Sir,

Gunal et al \cite{1} reported that gabapentin is effective in the treatment of uraemic pruritus. In previous years, we used gabapentin therapy in haemodialysis (HD) patients for restless leg syndrome, chronic pain and peripheral diabetic neuropathy. In our clinical experience, diverse patients suffered from drowsiness when treated with gabapentin 300 mg after each HD session, thus the dose had to be consistently reduced or the drug had to be stopped. Accordingly, previous studies \cite{2,3} have reported the appearance of somnolence/lethargy in some HD patients receiving gabapentin at the same dosage, and there are a number of case reports highlighting the risk of gabapentin-induced neurotoxicity and coma due to its narrow therapeutic window \cite{4–6}.

Similarly to the study of Gunal et al \cite{1}, after having observed the spontaneous remission of uraemic itch in a HD patient receiving gabapentin therapy for peripheral diabetic neuropathy, we started a pilot evaluation aimed at testing the effectiveness and safety of low doses of gabapentin in HD patients with uraemic pruritus \cite{7}. We began this evaluation by cautiously administering gabapentin 100 mg after every HD session and observed no side effects. In addition, the clinical response was as impressive as in the work of Gunal et al \cite{1} in all of the five treated patients \cite{7}. Importantly, we administered gabapentin under nurse surveillance after HD in order to avoid erroneous extra doses of this medication.

In conclusion, we agree with Gunal et al \cite{1} that gabapentin is an effective therapy for uraemic pruritus, but we would suggest that administering a lower gabapentin dose (i.e. 100 mg thrice weekly, after each HD session) under nurse surveillance and slowly titrating it up- or downward may lessen the risk of neurotoxicity and gabapentin-induced coma in HD patients.
Conflict of interest statement. None declared.

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doi:10.1093/ndt/gfh757
Advance Access publication 29 March 2005

**Do we need to change our administration practice with regard to sodium ferric gluconate complex in glucose?**

Sir,

We read with considerable interest the original article by Chertow *et al.* [1] on the relative safety of parenteral iron formulations. They found that adverse drug effects were similar amongst recipients of high molecular weight iron dextran and sodium ferric gluconate complex in sucrose (SFGC), and significantly increased compared with recipients of low molecular weight dextran. In fact, there was even one death with the use of SFGC. This is a matter of concern to nephrologists as it has been accepted over the last couple of years that SFGC is similar to placebo in the incidence of serious anaphylactoid reactions [2]. Based on the study by Michael *et al.* [3], the product monograph of SFGC in the USA and India states that a test dose is not required before administration of the drug. Recently a large study has shown that SFGC could even be safely administered to 98% of patients sensitive to iron dextran [4].

As discussed by the authors, the present study has important limitations. It is retrospective, and there is no detailed clinical information. Adverse drug event reporting has been voluntary, depending on the discretion of the treating physician. Even so, the findings are worrying to the practising clinician. Although the authors have refrained from making any recommendation, we wonder whether any interim changes in the practice guidelines need to be initiated until a large prospective study is published.

Conflict of interest statement. None declared.

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doi:10.1093/ndt/gfh552