microalbuminuria may not be necessarily a component of the metabolic syndrome [3].

Finally, on the basis of other studies, one may speculate that chronic inflammation may be the underlying mechanism that explains the association of hepatic C with microalbuminuria because atherosclerosis itself appears to be an inflammatory disorder [4, 5].

In their study, markers of inflammation such as IL-6 and C-reactive protein were not measured.

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Reply from the Authors

We are very grateful to Dr. Nzerue for his thoughtful comments on our paper [1]. In this correspondence, we respond to thoughtful queries by Dr. Nzerue.

First, in our discussion, we admitted that a single measurement of urine albumin to creatinine ratio to assess for the presence of microalbuminuria is not ideal. Unfortunately, the NHANES III is a cross-sectional study and sequential urinary data were not available.

Second, Dr. Nzerue points out that there is not a consistent relationship between the metabolic syndrome and microalbuminuria in some age groups, and this may explain why we failed to observe an association between hepatitis C and the metabolic syndrome. While this is plausible, more rigorous studies are needed to examine this issue as our results disagree with a previous retrospective cohort study [2], which showed a high prevalence of the metabolic syndrome in patients with hepatitis C.

Third, we accept the possibility that chronic inflammation or immune processes are involved in the pathogenesis of microalbuminuria in hepatitis C subjects. We explored this possibility, but found that cryoglobulin levels were not measured in the NHANES III, and rheumatoid factor data were missing in most patients in the hepatitis C cohort. However, we failed to find any differences in the levels of C-reactive protein in subjects with hepatitis C and controls (0.41 ± 0.62 vs. 0.44 ± 0.86 mg/dL, P = 0.5).

Our study should be viewed as exploratory in nature and our observations require validation in ongoing prospective studies (e.g., NIDDK-funded Virahep-C study).

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Red blood cell deformability and diabetic nephropathy

To the editor: In an interesting study, Brown et al [1] found an impaired red blood cell deformability in 57 adult type 2 diabetic patients. They used a filtration technique using polycarbonate membranes with straight channels of 3 micrometer pore diameter. In patients with diabetic nephropathy they found an increased impairment in red blood cell deformability. The hypothesis that an impaired red blood cell deformability contributes to renal function decline is very attractive and may give rise to therapeutic possibilities. Many studies found an impaired red blood cell deformability in diabetic patients; others, however, did not. Years ago we tested the hypothesis that red blood cell deformability contributes to diabetic nephropathy using both ektacytometry and erythrocyte filtration, with micropores with a diameter of 5 micrometer [2]. In order to imitate local circumstances in the kidney red blood cell deformability was also measured in hyperosmolar solutions. Seventy-one insulin-dependent diabetic patients were included, 25 patients without any sign of organ damage, 21 patients with microalbuminuria, 13 patients with overt nephropathy, and 12 patients with leg ulceration. No decreased red blood cell deformability was found in any of the diabetic groups with either technique, neither
did the total group of 71 diabetic patients have a lower red blood cell deformability when compared to controls. Extraerythrocytic factors, such as leukocytes, plasma fibrinogen, and platelet microaggregates may influence filtration results. I suppose the filtration technique of Brown et al was corrected for this. These results may suggest that the filtration pore size of 3 micrometers used by Brown et al was more sensitive than the 5 micrometer pores filtration technique and ektacytometry that was used by us and that the lack of difference in our patient groups reflects this lack of sensitivity. On the other hand, the question arises whether such subtle differences in red blood cell deformability, if present, contribute to nephropathy in diabetic patients.

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Postprocedural drop in hematocrit versus contrast-induced nephropathy: Eggs or chickens?

To the Editor: In a recent issue of Kidney International, Nikolsky et al suggested that low hematocrit (Hct) predicts contrast-induced nephropathy (CIN) after percutaneous coronary interventions (PCI) [1]. One impressive finding in this study was that the postprocedural drop in Hct (ΔHct = baseline Hct – postprocedural nadir Hct) was highly correlated with CIN. However, we wonder whether ΔHct resulted in CIN or, on the contrary, it was attributed to CIN. Assuming that there was no significant postprocedural change of body weight (BW) or fluid status in the patients receiving PCI, ΔHct might exactly reflect acute blood loss. The most possible route of the blood loss was extravascular loss (e.g., bleeding, hematoma, catheter aspiration, etc.) rather than intravascular hemolysis. Hence, the amount of the procedure-related extravascular blood loss (EVBL) could be estimated by the following equations [2]:

\[ \text{Blood volume} = \frac{\text{plasma volume}}{(1 - \text{Hct})} = \frac{\text{BW} \times (60\% \times 40\% \times 20\%)}{(1 - \text{Hct})}; \]

\[ \text{EVBL (L)} = \frac{\text{BW (kg)} \times (60\% \times 40\% \times 20\%)}{(1 - \text{baseline Hct})} \times \frac{\Delta \text{Hct}}{(1 - \text{baseline Hct} + \Delta \text{Hct})} \]

In a male patient with a BW of 70 kg, baseline Hct 40%, ΔHct 6%, his estimated EVBL would be 510 mL. Actually, the average amount of EVBL after PCI rarely exceeded 100~200 mL. This could not explain the large gap from the estimated data. On the contrary, ΔHct might also be attributed to fluid retention-induced hemodilution secondary to CIN. The proportional increase of serum creatinine must overcome the dilute effect of acute fluid retention before meeting the definition of CIN (i.e., an increase of ≥25% or ≥0.5 mg/dL in pre-PCI serum creatinine at 48 hours post-PCI) [3, 4]. Precise

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