Induction of Premature Ventricular Complexes by Hemodialysis?

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Dear Sir,

With interest we read the article of Quereda et al., recently published in Nephron [1], wherein the authors agreed with the main conclusion of our study [2] that premature ventricular beats occur primarily in hemodialysis patients with coronary artery disease. However, the authors disagreed with our second conclusion, that chronic hemodialysis or related methods do not increase the risk of ventricular ectopies, and they provided evidence in 35 of their hemodialysis patients, that Lown 2–5 ectopies occurred in 1/35 patients prehemodialysis but in 8/35 patients in the immediate posthemodialysis period. A similar observation was made by Quellhorst et al. [3], who reported an increase of higher grade ventricular arrhythmias following acetate hemodialyses (without control of ultrafiltration) as compared to hemofiltration where weight loss is perfectly controlled gravimetrically and symptomatic hypotension occurred less frequently.

In our study [2], volumetrically controlled ultrafiltration was used in all dialysis procedures and thereby arterial hypotension could be avoided. Thus, differences in dialysis technique and prevention of dialysis hypotension may account for the observed differences in ventricular ectopic activity.

To reassess the influence of hemodialysis on ventricular ectopic activity, we further investigated 105 patients (33 females, 72 males; mean age 45 years) with a Reynolds Pathfinder System. In respect to the frequency of premature ventricular complexes (PVCs), there was no statistical difference between the period on dialysis (11.4 ± 39 PVCs/h), the immediate postdialysis (0–4 h posthemodialysis) period (12.5 ± 37 PVCs/h) and the late (4–16 h) postdialysis period (12 ± 32 PVCs/h). As indicated by the high standard deviations from the mean values, all variations of cardiac rhythm were observed in the individuals. Thus, the parameter ‘frequency of PVCs’ appears not to be influenced by dialysis if the study group is large enough. Quereda et al. [1] focused on the parameter ‘severity of PVCs’, and found that in their patients, most complex arrhythmias occurred during the last hours of dialysis and immediately afterwards. However, arrhythmias of Lown grade 2 were included into their classification of complex ventricular arrhythmias and the percentage of grade 2 or 3 arrhythmias from the total of 8/35 patients, in whom ‘complex’ PVCs were observed during dialysis, was not given. We therefore cannot follow the argument that serious arrhythmias are induced by dialysis, since we consider PVCs of grade 2 or 3 of less clinical importance. On the other hand, we agree with the authors

[1] that the contributions of arrhythmias to mortality do not seem to be important in dialysis patients without concomitant heart diseases.

It is of clinical interest that, in all patients reassessed by our group, neither electrocardiographic nor echocardiographic parameters (n = 105) were different between patients who had Lown grade 3–5 arrhythmias (n = 30) and those who had no ventricular ectopies (n = 27). In 57% of our dialysis patients, left ventricular hypertrophy was demonstrated using echocardiography and a subgroup of hypertrophic cardiomyopathy could be identified in nearly half of these patients. We did not find evidence that pre-existing ‘cardiopathy’ per se facilitates the origination of ventricular ectopies. However, 9 out of 10 patients, who had significant coronary artery disease, diagnosed by angiography, had grade 3–5 ventricular arrhythmias. Therefore, ‘cardiopathy’ has to be specified and in case ventricular ectopies occur in a patient on hemodialysis, coronary artery disease should be excluded or confirmed. In the latter case, the primary disorder should be treated consequently, which includes optimizing dialysis therapy by reducing dry weight and thereby preload [4].

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