Sympathetic Activity and Clinical Outcome in Dialysis Patients

To the Editor:

We feel that the meta-analysis allows for additional interpretation and recommendation for the clinician and definition of future research. Treatment should not only be aimed at blood pressure lowering, per se, but at correcting mechanisms most likely contributing to the pathophysiology. In the case of dialysis patients, it should at least include interventions addressing fluid overload and overactivities of the renin-angiotensin system (RAS) and sympathetic nervous system. Indeed, 3 of the reviewed studies compared a regimen with and without RAS blockade (see References 11–13 in the article by Agarwal and Sinha). However, study medication (carvedilol in References 10 and 15) was added to “conventional” treatment, ie, RAS blockade. In a nonreferenced study, telmisartan was added to chronic treatment with a RAS inhibitor. In addition, in Reference 12 of the article by Agarwal and Sinha, the study group receiving candesartan also used (although not significantly) more angiotensin-converting enzyme inhibitors and β-blockers. These data suggest that more profound inhibition of the renin/sympathetic system may be beneficial. RAS blockers and carvedilol are sympatholytic agents in disease conditions with renin/sympathetic activation. RAS blockers in presently advised dosages reduce but do not normalize sympathetic hyperactivity. Sympathetic hyperactivity seems to be even more pronounced in dialysis patients (Figure). It is clearly related to poor clinical outcome and is most likely caused by kidney ischemia. It is tempting to hypothesize that such “intensive” treatment may provide a better inhibition of sympathetic overactivity, resulting in a better clinical outcome. Aims for future research are: 1) how to identify dialysis patients who will especially benefit from higher dosages of RAS blockade or the addition of carvedilol; 2) it is conceivable that renin/sympathetic activation will decrease or cease to exist in the course of dialysis life because of progressive kidney tissue loss. However, it is uncertain whether this indeed occurs and how to monitor it.

Disclosures

None.

Figure. Regression lines of muscle sympathetic nerve activity (MSNA) in chronic kidney disease (CKD) patients not on dialysis (mean creatinine clearance: 54±31 mL/min; n=74), hemodialysis patients (HD; n=17), and controls (n=80). Data are taken from References 4 and 5.

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