

Prevention of Cardiovascular Complication of Hemodialysis Patients III:  
Hyperlipidemia

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Dyslipidemia is well-established cardiovascular (CV) risk factor in the general population. High total cholesterol (TC), high low-density lipoprotein cholesterol (LDL-C), high triglyceride (TG), and low high-density lipoprotein cholesterol (HDL-C) are all clearly associated with CV events. Dyslipidemia has been a major concern in patients with end-stage renal disease (ESRD) because of extremely high CV mortality.

Dyslipidemia is common but not universal in patients treated with hemodialysis (HD). Lipid profile may differ quantitatively and qualitatively by presence of co-morbid conditions including diabetes, amount of proteinuria, and nutritional status. HD patients tends to have low HDL-C, high TGs but normal or low TC and LDL-C. However, this spectrum hides more atherogenic profile, which includes increased apolipoprotein B, lipoprotein(a), intermediate- and very-low-density lipoprotein, and small dense LDL particles. LDL particles are likely to be oxidized, forming oxidized LDL. Just LDL-C level dose not reliably discriminate between patients at low or high risk of CV events.

In HD patients, the association between dyslipidemia and CV outcomes was not robust in many observational studies. A lower TC level rather than a higher TC level was associated with a higher mortality, so called “reverse epidemiology”. Furthermore, recent large randomized trials showed that effects of lipid-lowering treatment on CV risk reduction were not evident as in the general population. The 4D study including 1255 HD patients with type 2 diabetes failed to demonstrate reduction of composite CV events by atorvastatin vs. placebo [relative risk (RR) 0.92; 95% confidence interval (CI) 0.77–1.10]. AURORA Study which assigned 2776 HD patients to receive rosuvastatin vs. placebo also showed that primary CV endpoint was not reduced [hazard ratio (HR) 0.96; 95% CI 0.84–1.11]. The SHARP conducted in 9270 patients with chronic kidney disease (CKD) and reported that simvastatin/ezetimibe therapy was associated with a significant 17% RR reduction of major atherosclerotic events compared to placebo (HR 0.83; 95% CI 0.74–0.94). However, the benefit was not observed in the subgroup of over 3000 patients treated with HD. The clinical benefit of statins with/without ezetimibe is uncertain in prevalent HD patients.

Although an essential vascular pathology may not differ in the HD population

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from the general population, it is difficult to explain why the difference is observed. There might be a point in the impairment of the renal function in which beneficial effects of lipid-lowering treatment are modified by uremia. The survival bias also might attenuate the effect of lipid-lowering treatment, which means significant portion of vulnerable patients are died in CKD stage before reaching ESRD.

At this point in time, initiation of lipid-lowering treatment is not recommended for most prevalent HD patients (2013 KDIGO guideline). The physician, however, might reasonably choose the treatment at individual base. It would be reasonable to continue lipid-lowering treatment in patients already receiving it at the time of HD initiation.