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Increased Monocyte-to-High-Density Lipoprotein Ratio is Associated with Recurrent Vascular Access Stenosis

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Objectives: Previous studies indicate that increased monocyte count and decreased high-density lipoprotein (HDL) cholesterol levels are associated with chronic kidney disease, and increased risk for cardiovascular diseases, indicating its possible role in pro-inflammatory and pro-oxidant states. We tried to investigate the clinical significance of circulating monocyte count-to-serum HDL cholesterol ratio (M/H ratio) in predicting recurrent vascular access stenosis after angioplasty in hemodialysis patients.

Methods: M/H ratio at access creation and prior to angioplasty was measured in a total of one-hundred and fifty hemodialysis patients in Incheon St. Mary's hospital from July, 2006 to September, 2017. The impact of M/H ratio in predicting recurrent access stenosis was evaluated retrospectively by using Kaplan-Meier, Cox regression, and ROC curve analyses.

Results: The patient group comprised of 67% male and 58% diabetes, aged 62±14 years old (n=150). Baseline M/H ratio at access creation was not different between those with vascular access stenosis (n=71) and those without stenosis (n=79) (10.74 vs. 12.42, p = 0.11). Among patients with vascular access stenosis, there also was no difference in baseline M/H ratios between those with recurrent stenosis (n=33) and those without recurrence (n=38) (9.98 vs. 11.4, p = 0.24). However, pre-angioplasty M/H ratio increased significantly when compared to that of baseline (10.75 vs. 17.95, p < 0.001). Delta M/H ratio, as defined by a difference between baseline and pre-angioplasty ratios was calculated and mean delta M/H ratio was 1.9. Increased delta M/H ratio (delta M/H ratio greater than or equal to 1.9) was associated with recurrent stenosis (HR 4.16, CI 1.43-12.12, p = 0.009). Moreover, increased delta M/H ratio was clinically significant in predicting recurrent vascular access stenosis (AUC 74%, p = 0.001).

Conclusions:

Increased delta M/H ratio may play a role in pro-inflammatory and pro-oxidant environment and predispose vascular access for recurrent stenosis after angioplasty.