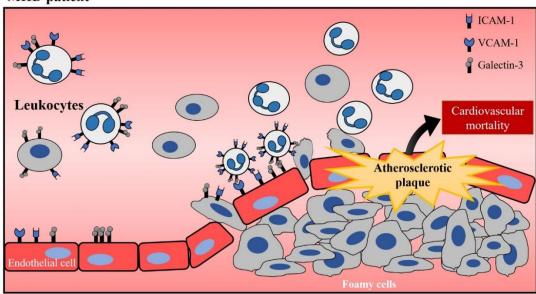
All patients undergoing
maintenance hemodialysis treatment
for at least 3 months were eligible
for inclusion in the study.

Exclusion:
Severe cardiac failure (New York
Heart Association class IV),
terminal illness, active infections,
active malignancy, protein-energy
wasting, incomplete data or
personal willingness.

Eligible for study (n=86)

Supplemental Figure 1. Flowchart of patient enrollment.

MHD patient



Supplemental Figure 2. Schematic of joint effects of galectin-3 and VCAM-1 on pathogenesis of leukocyte trafficking and atherothrombosis. Atherosclerotic plaque formation involves enhanced expression of various adhesion molecules on endothelial cells, including ICAM-1 and VCAM-1. Galectin-3 on the cell surface augments attachment between leukocytes and endothelial cells. Fatty streaks are the first signs of atherosclerosis that consist of lipid-containing foam cells in the arterial wall just beneath the endothelium. The mass of the "atheroma" is composed of a mixture of lipid and inflammatory cells, leading to progressive spatial occupation of arterial lumen. Once a plaque ruptures, the subsequent thromboembolic event increases the risk of cardiovascular mortality. ICAM-1, intercellular adhesion molecule-1. VCAM-1, vascular cell adhesion molecule-1.