Title
239: Coronary Artery Calcium Score, Inflammation and Mortality in Maintenance Hemodialysis Patients

Permalink
https://escholarship.org/uc/item/9z3747m7

Journal
American Journal of Kidney Diseases, 51(4)

ISSN
0272-6386

Authors
Shantouf, Ronney
Ahmadi, Naser
Rambod, Mehdi
et al.

Publication Date
2008-04-01

DOI
10.1053/j.ajkd.2008.02.249

Copyright Information
This work is made available under the terms of a Creative Commons Attribution License, available at https://creativecommons.org/licenses/by/4.0/

Peer reviewed
239

CORONARY ARTERY CALCIUM SCORE, INFLAMMATION AND MORTALITY IN MAINTENANCE HEMODIALYSIS PATIENTS

Ronney Shantouf, MD\textsuperscript{1}; Naser Ahmadi, MD\textsuperscript{3}, Mehdi Rambod, MD\textsuperscript{1}, Ferdinand Flores\textsuperscript{3}, Jima Tiano\textsuperscript{3}, Matthew Budoff, MD\textsuperscript{3}; and Kamyar Kalantar-Zadeh\textsuperscript{1,2}. \textsuperscript{1}Harold Simmons Center for Kidney Disease Research\& Epidemiology, \textsuperscript{2}Division of Nephrology, and \textsuperscript{3}Cardiology, LBioMed at Harbor-UCLA, Torrance, CA

**Introduction:** Vascular calcification in maintenance hemodialysis (MHD) patients (pts) may be a cause of high mortality. We examined whether the coronary artery calcification scores (CACS) provides incremental value to predict all-cause mortality in MHD pts. **Methods:** Survival analyses were conducted in the 5-year (2001-06) cohort of Nutritional and Inflammatory Evaluation in Dialysis (NIED) Study. **Results:** In 166 MHD pts who underwent CACS (aged 56±13 yrs), the Charlson comorbidity score, C-reactive protein, interleukin-6, TNF-a, and homocysteine were increased in proportion from CACS=0 to 1≤CACS≤99 to 100≤CACS≤399 to CACS>400. CACS>400 and 100≤CACS≤399 were associated with a significantly higher adjusted risk of death than CACS=0 (Figure):

![Graph showing survival analysis with CACS categories: CAC:0, CAC:1-99, CAC:100-399, CAC>400.](image)

**Conclusion:** These results suggest that significant CACS can predict incrementally worsening inflammation and also all-cause mortality of MHD pts independent of conventional risk factors and inflammation.