Session P03 - Vascular

Itinerary

P507 / P507 - Atherosclerosis Progression in Chronic Kidney Disease

September 16, 2017, 1:30 - 3:00 PM | Pacific A-K

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Disclosures

H.A. Perez: None. **L. Aballay:** None. **L.J. Armando:** F. Ownership Interest (includes any stock, stock option, partnership, membership or other equity position in an entity regardless of the form of the entity, or any option or right to acquire such position, and any rights in any patent or other intellectu; Significant; Vascularis, Inc. **J. Spence:** F. Ownership Interest (includes any stock, stock option, partnership, membership or other equity position in an entity regardless of the form of the entity, or any option or right to acquire such position, and any rights in any patent or other intellectu; Significant; Vascularis, Inc. **N.H. Garcia:** None.

Abstract

Cardiovascular risk is very high in CRF, but the underlying mechanisms are not well understood. Traditional cardiovascular risk factors (RF) do not explain the increased risk, and observational studies have observed paradoxical or absent associations between classical RF and mortality in ESRD. CRF studies found that statin therapy does not reduce CV events; these may be the results of “resistant atherosclerosis” observed in these patients. We investigated if carotid total plaque area (TPA) is increased at progressively lower creatinine clearance and whether or not TPA progression is increased in CRF patients not on dialysis. Methods: The Blossom DMO Argentina ethics committee approved the study and informed consent was obtained from each participant. We performed a cohort study in 201 patients with Normal Renal Function (NRF), Stage 2 and 3 CRF. Clinical, laboratory tests and TPA were determined at time 0 and after 1 year. TPA was measured using carotid ultrasonography. Renal function (eGFR) was determined by the MDRD equation. The Study population was divided into quartiles of eGFR. Results: 1st Quartile, (51±1yo, eGFR 89±2 ml/min) had a blood pressure (BP) of 136±2/81±1 mmHg, BMI 31±1, Total Chol (tChol) 196±6 mg/dl, HbA1c 6.7±0.4% and had the lowest Chol 192±5 mg/dl, HbA1c 6.2±0.1% and TPA 47±6mm^2; 3rd Quartile, (59±1yo, eGFR 63±1 ml/min) BP 133±2/82±1, tChol 192±5 mg/dl, HbA1c 6.2±0.1% and TPA 47±6mm^2; 4th Quartile (60±2yo, eGFR 52±1 ml/min) BP 140±3/84±1, tChol 209±5 mg/dl, HbA1c 6.2±0.1% and TPA 76±11mm^2. After one year, the 4th Quartile had the most progression of TPA ($p < 0.005$); it was not influenced by age, hypertension, smoking, dyslipidemia or diabetic status. Conclusions: In CRF, TPA increases as renal function decreases; its progression is not associated with traditional risk factors. Other mechanisms are responsible for the observed excess of cardiovascular disease in CKD. Determination of TPA should be used to measure effects of antiatherosclerotic therapy to decrease the enormous cardiovascular event rate observed in this population.