Association of Vascular Calcification and Residual Renal Function in Hemodialysis Patients

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Introduction

- Vascular calcification (VC) is common and may contribute to cardiovascular mortality in patients with end-stage renal disease.
- Little is known about the effect of residual renal function (RRF) on VC in patients on hemodialysis (HD).

Table 2. ABPM, PWV, and abdominal aorta calcification scoreIn HD patients according to KRU.

| Variables | KRU <0.9 ml/min/1.73m ² (n = 53) | KRU ≥0.9 ml/min/1.73m² (n =53) | P- value |
|-----------------------------|--|-----------------------------------|----------|
| ABPM (mmHg) | | | |
| Daytime mean blood pressure | 108.9±14.4 | 104.5±12.4 | 0.095 |
| Nighttime blood pressure | 102.6±15.4 | 100.4 ± 14.3 | 0.474 |
| Non-dipper, n (%) | 41 (80.4) | 38 (79.2) | 0.999 |

- We hypothesis that RRF was associated with VC and affected cardiac function and cardiovascular events.
- Therefore, we investigated the correlation between RRF expressed as GFR and VC in patients on HD and conducted echocardiography. Furthermore, new cardiovascular events were evaluated after study enrollment.

Methods

- One hundred six patients with RRF on maintenance HD for 3 months were recruited between January 2014 and February 2015 from 3 different HD centers.
- We used residual renal urea clearance (KRU) to measure RRF.

Urea clearance = $\frac{2 \times (\text{urine urea concentration} \times \text{urine volume})}{\text{urine collection duration} \times (\text{BUN1} + \text{BUN2})}$

* BUN1: sampled at the end of the first dialysis session of the week

| baPWV (cm/s) | 1836.1±250.4 | 1676.8±311.0 | 0.005 |
|--------------|----------------|---------------|-------|
| AACS | 4.0 (1.0–10.0) | 3.0 (0.0–8.0) | 0.050 |



Figure 1. The association of abdominal aorta calcification score and KRU.



- * BUN2: sampled immediately before the next session
- To assess VC severity, we conducted abdominal aortic calcification score (AACS) analysis, ambulatory blood pressure monitoring, and brachial-ankle pulse wave velocity. We also performed echocardiography to evaluate cardiac function.

Results

Table 1. Baseline characteristics according to KRU.

| Variables | KRU <0.9 ml/min/1.73m ² (n =53) | KRU ≥0.9 ml/min/1.73m² (n =53) | P- value | |
|--|---|-----------------------------------|----------------|--|
| Age (years) | 58.2±9.1 | 60.0 ± 12.7 | 0.394 | |
| Male, n (%) | 26 (49.1) | 29 (54.7) | 0.697 | |
| HD duration (months) | 37.4 (19.5–56.6) | 13.6 (7.0–42.9) | <0.001 | |
| Diabetes, n (%) | 31 (58.5) | 29 (54.7) | 0.695 | |
| Coronary artery disease, n (%) | 23 (43.4) | 20 (37.0) | 0.994 | |
| Interdialytic weight gain (kg) | 1.9±1.3 | 1.1±1.0 | <0.001 | |
| Residual renal urine (cc) | 250 (120–400) | 1000 (800–1575) | <0.001 0.03 | |
| Diuretics, n (%) | 32 (60.4) | 42 (79.2) | 0.03 | |
| Resistance to ESAs, n (%) | 8 (15.1) | 2 (3.8) | 0.046 | |
| Hemoglobin (g/dL) | 10.1 ± 1.1 | 10.2±1.3 | 0.854 | |
| Albumin (g/dL) | 3.8±0.4 | 3.7±0.5 | 0.862 | |
| Calcium (mg/dL) | 8.7 (8.1–9.1) | 8.6 (8.2–8.9) | 0.915 | |
| Phosphate (mg/dL) | 4.6±1.4 | 4.4±1.1 | 0.513 | |
| Ca X P (mg ² /dL ²) | 40.5 (34.2–45.1) | 38.3 (30.8–43.8) | 0.281 | |
| Total cholesterol (mg/dL) | 138 (107.0–160.0) | 140.0 (110.5–164.5) | 0.382 | |
| LDL cholesterol (mg/dL) | 72.0 (53.0–89.0) | 86.0 (62.0–103.0) | 0.024 | |
| CRP (mg/L) | 0.9 (0.4–3.0) | 0.5 (0.3–1.1) | 0.029 | |
| Parathyroid hormone (pg/dL) | 243.8 (102.4–415.2) | 196.6 (118.8–346.0) | 0.649 | |
| β2-microglobulin (mg/L) | 22.4±6.7 | 17.3±5.8 | 0.008 | |
| KRU (mL/min/1.73m ²) | 0.3 (0.2–0.6) | 2.5 (1.5–3.0) | <0.001 | |
| Single-pool Kt/V | 1.6±0.4 | 1.5±0.3 | 0.591 | |

Figure 2A. Kaplan-Meier analysis of CV events in HD patients according to KRU. CV events were comparable patients with KRU <0.9 and KRU ≥0.9 ml/min/1.73m².



| No at risk | | | | | | | |
|-------------------------------------|----|----|----|----|----|---|--|
| RRF > 0.9 ml/min/1.73m ² | 23 | 23 | 23 | 23 | 12 | 0 | |
| RRF < 0.9 ml/min/1.73m ² | 23 | 22 | 18 | 16 | 7 | 0 | |

Figure 2B. Kaplan-Meier analysis of CV events in non-diabetic HD patients according to KRU. CV were significantly lower in patients with KRU ≥1.0 ml/min/1.73m².

Conclusion

- Increased AACS was independently associated with RRF deterioration. In particular, in non-diabetic patients on HD, CV events were higher in patients with a low RRF.
- This result suggests that preservation of RRF may prevent VC. In addition, the effort to protect RRF may be more important in nondiabetic than in diabetic patients on HD.

