

was conducted in the third month of the study.

Pre-dialysis albumin was 32.8 ± 4.1 g/L in Australian indigenous patients and 36.2 ± 4.5 g/L in non-indigenous patients ($p = 0.003$). The difference between pre-dialysis and post-dialysis albumin was -4.0 ± 2.8 g/L in indigenous patients and -1.0 ± 1.9 g/L in non-indigenous patients ($p = 0.000$). Intra-dialytic weight gain was 3.0 ± 2.6 kg in indigenous patients and 2.0 ± 0.8 kg in non-indigenous patients ($p = 0.010$). Serum sodium and bicarbonate were 136.2 ± 2.7 mmol/L and 22.3 ± 1.7 mmol/L in indigenous patients and 138.5 ± 2.3 mmol/L and 25.2 ± 5.8 mmol/L in non-indigenous patients ($p = 0.010, 0.003$). Glucose, HbA1c and triglyceride were 9.7 ± 4.6 mmol/L, $7.0 \pm 1.7\%$ and 2.2 ± 1.3 mmol/L in indigenous patients and 7.0 ± 2.8 mmol/L, $6.2 \pm 1.0\%$ and 1.4 ± 0.7 mmol/L in non-indigenous patients ($p = 0.005, 0.047, 0.016$). No differences were observed in dry weight, PG-SGA, post-dialysis albumin, serum potassium, urea, creatinine, parathyroid hormone, phosphate, calcium, C-reactive protein or hemoglobin.

This study indicates that differences exist between Australian indigenous and non-indigenous maintenance hemodialysis patients, with indigenous patients less likely to be meeting recommendations.

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257 GUANIDINOACETIC ACID(GAA) IN PATIENTS WITH CHRONIC KIDNEY DISEASE(CKD) AND DIABETES MELLITUS(DM)

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GAA is the precursor of creatine, an essential in the energy metabolism of muscle and nerve tissue. GAA is mainly produced in the kidney. GAA production was reported to be suppressed in the streptozotocin-induced DM rats. However, GAA metabolism has not been really investigated in CKD or DM patients. In this study, we determined serum level(S) and urinary excretion(U) of GAA and creatinine(Cr) in patients with chronic glomerulonephritis (CGN) and DM. The subjects were 15 healthy adults, 92 patients with CGN and 27 patients with non insulin-dependent DM nephropathy. S and U-GAA were determined with HPLC. As shown in the Table describing mean values, U-GAA of early stage CKD patients was significantly lower than healthy subject. And S-GAA decreased with loss of renal function or with U-Cr, especially in DM patients.

	N	S-Cr	U-Cr	S-GAA	U-GAA
		(mg/dL)	(mg/day)	(μ g/dL)	(mg/day)
Normal	15	0.9	1633	38.2	103.0
CGN(Ccr > 90mL/min)	12	1	1742	41.1	41.3*
CGN(Ccr < 90mL/min)	24	1.5*	1385*	37.7	39.2*
CGN(Ccr < 30mL/min)	56	7.1*	715*	24.4*	5.9*
DM(Ccr > 60mL/min)	16	0.9	775*	27.8*	46.2*
DM(Ccr < 60mL/min)	11	5.0*	581*	22.1*	2.3*

In conclusion, GAA production in the kidney decreased in CKD patients, suggesting GAA deficiency was a reason of muscle wasting of CKD and DM patients.

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258 EFFECTS OF GUANIDINOACETIC ACID(GAA) SUPPLEMENTATION IN RATS WITH CHRONIC RENAL FAILURE(CRF)

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GAA is the precursor of creatine(CRT), an essential in the energy metabolism of muscle and nerve tissue. GAA is mainly produced in the kidney. We reported the GAA deficiency in CRF patients. This study was designed to assess the effects of GAA supplementation(10 mg,100 mg/day orally for 4 weeks respectively) for the muscle capabilities in CRF rats prepared by 5/6 nephrectomy. Muscle power was assessed by the sliding angle of the inclined screen test, and physical strength was evaluated by the time to survive in water (forced swimming method). GAA and CRT concentrations in serum, muscle and other organs were significantly

decreased in CRF rats. GAA administrations significantly increased CRT content in muscle, and improved muscle capabilities dose-dependently.

	muscle power	physical strength
	sliding angle; $^{\circ}$	swimming time; min
Sham	55.2 ± 0.9	52.0 ± 3.8
CRF(GAA 0)	$42.3 \pm 1.9^*$	$21.9 \pm 1.2^*$
CRF(GAA 10 mg)	$47.4 \pm 1.3^*\$$	$25.5 \pm 1.1^*\$$
CRF(GAA 100 mg)	$48.8 \pm 2.1^*\$$	$29.0 \pm 3.3^*\$$

*; $p < 0.05$ vs Sham \$; $p < 0.05$ vs GAA 0

In conclusion, we demonstrated a deficiency of GAA and CRT, and muscle weakness in CRF rats. However, oral GAA supplementation could recover muscle content of CRT and muscle capabilities in these rats.

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259 IMPACT OF IDPN ON Kt/V

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The administration of intradialytic parenteral nutrition (IDPN) and the impact it has on Kt/V has not been thoroughly examined. The available literature addressing the influence of IDPN on Kt/V is limited and the observations are conflicting. Some studies have observed little or no significant change with IDPN administration. In contrast, one randomized, crossover study by McCann et al. of patients receiving IDPN observed a significant decrease. When amino acid (AA), dextrose and lipid components were administered a significant reduction in mean Kt/V resulted. When solutions containing only AA were administered, Kt/V was significantly less than when IDPN was withheld. Administration of IDPN without AA resulted in a mean Kt/V not significantly different from the mean Kt/V when IDPN was withheld. AA containing IDPN reduced mean Kt/V by 18–23%. By personal communication with McCann, it was thought the amount of carbohydrate (CHO) determined whether the Kt/V was impacted when they studied further (unpublished). The authors found that by increasing the CHO, the Kt/V was not impacted (lower) and theorized that inadequate CHO resulted in catabolized protein generating urea. To investigate this further, a retrospective analysis was performed of the Pentec Health IDPN internal data base consisting of 489 patients that met inclusion and exclusion criteria. For this group: the age was 66.23 ± 13.38 years; BMI was 25.79 ± 5.6 ; 239 males (49%) and 250 females (51%); 304 patients with DM (62%); 430 patients with HTN (88%). Baseline albumin was 2.98 ± 38 g/dL and baseline creatinine was 6.03 ± 2.10 mg/dL. Results are below Table

Kt/V (mean + SD)

Baseline	Month 1	Month 2	Month 3
$1.65 \pm .34$	$1.44 \pm .36$	$1.43 \pm .35$	$1.44 \pm .34$

A repeated measures ANOVA indicated a statistically significant difference ($p < 0.0001$). A Tukey's adjustment resulted in non-significant differences between month 1 and month 2 ($p = 0.995$), month 1 and month 3 ($p = 0.987$), and month 2 and month 3 ($p = 0.936$). In conclusion, the initial decrease in Kt/V was followed by a period of stabilization, and the mean Kt/V never dropped below the KDOQI recommendation of 1.2.

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260 A FULL COLOR VISUAL EDUCATING TOOL TO IMPROVE RENAL DIET COMPLIANCE

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The objective is to simplify Renal diet education of a newly diagnosed, illiterate/non-English speaking CKD patient by using a 5 step concept that is explained by a fully illustrated color manual. The 5 steps to be followed are as follows:

Step 1. What foods are eaten daily? The renal dietitian is faced with many challenges when taking a diet history & educating a newly diagnosed CKD patient. It is of utmost importance to tailor education to specific patient's