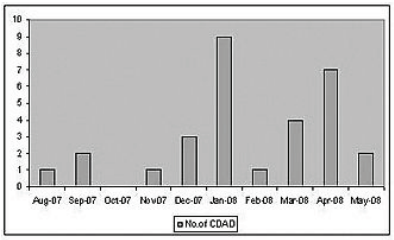

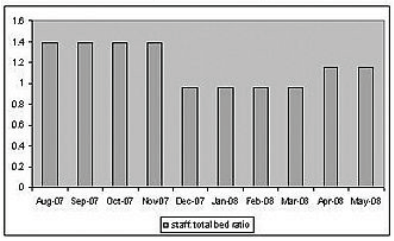

Conclusion:

Environmental change, reduction in nursing staff levels and reduction in isolation facilities are important but under-recognised factors in the spread of Clostridium difficile infection.

Disclosure of Financial Relationships: nothing to disclose

PUB638

Multi-Frequency Bioelectrical Impedance Analysis and Cellular Health in Mobile CKD Patients L. Mason,[1] M. I. C. Kingsley,[1] J. K. Rees,[1] P. A. Ali,[1] E. Rees,[1] I. Hilldrup,[2] A. Mikhail.[2] *[1]Swansea University; [2]Morriston Hospital, Swansea, United Kingdom.*

Large differences exist within CKD patients on haemodialysis, from relatively active transplant candidates (mobile CKD patients) to those who are sedentary and depend on healthcare support. Even the healthiest patients display frequent changes in hydration status and may show signs of cell membrane damage. Multi-frequency bioelectrical impedance analysis (BIA) measures impedance or resistance to currents at specified frequencies. Unlike high frequency current (200kHz), low frequency current (5kHz) cannot penetrate intact cell membranes. Consequently, impedance values at 5kHz should be high and the Illness Marker (the ratio of impedance values at 200 and 5kHz) should be low. The aim of this study was to compare impedance values and the Illness Marker in mobile CKD patients with healthy matched controls (Con).

Seven mobile CKD patients on haemodialysis (Age: 53.4±10.7years, Mass: 79.1±10.7kg, Height: 1.67±0.11m) and seven matched controls (Age: 50.3±9.8 years, Mass: 76.4±12.5kg, Height: 1.69±0.09m), volunteered. Anthropometric measurements were taken and BIA (Quadscan; Bodystat, Isle of Man, UK) was conducted at 5, 50, 100 and 200kHz. These data were used to calculate hydration status and the Illness Marker. All measurements were taken following an overnight fast and within 18 hours of receiving dialysis for CKD patients.

Wilcoxon signed ranks tests revealed that the Illness Marker was significantly higher in CKD patients compared with matched controls (CKD: 0.809±0.024, Con: 0.780±0.024, P=0.046). Total body water (CKD: 37.1±8.7 L, Con: 40.2±7.8L, P=0.249) extra-cellular water (CKD: 16.4±2.9L, Con: 17.6±2.3L, P=0.237), and impedance values at all individual frequencies ($P \geq 0.128$) were similar for both groups.

These findings show that, even when the healthiest group of CKD patients is considered, CKD patients have elevated Illness Markers, indicative of poor cellular health. Interestingly, this finding was independent of body composition, absolute impedance values, total body water and extra-cellular water. This simple measure may offer a novel approach to evaluating and tracking cellular health in the haemodialysis population.

Disclosure of Financial Relationships: nothing to disclose

PUB639

Transfusion and ESA Use in Medicare Beneficiaries with Chronic Kidney Disease (CKD) Shuling Li,[1] Hassan N. Ibrahim,[1] Thomas J. Arneson,[1] David T. Gilbertson,[1] David A. Zaun,[1] Brian Bradbury,[2] Allan J. Collins.[1,3] *[1]Chronic Disease Research Group, MMRF, Mpls, MN; [2]Amgen Inc., Thousand Oaks, CA; [3]Medicine, Univ of MN, Mpls, MN.*

Anemia is a common and important consequence of CKD. Few studies have examined patterns of transfusion and ESA use in the CKD population during the transition period to ESRD or to death. We sought to describe transfusion and ESA use in the 12 months prior to these outcomes, as well as in those who survived.

We identified 36,640 elderly Medicare pts with CKD diagnosed in 2002 who remained continuously enrolled in Medicare Parts A/B and free of ESRD to the end of 2003. We followed these pts from Jan 1, 2004 to the earliest of ESRD diagnosis, death, payer change, or the end of 2005. We then categorized pts into 3 groups: those who progressed to ESRD (1,465), those who died (8,757), and those who survived free of ESRD (26,418). Monthly ESA use and transfusion rates were estimated for each of the last 12 months of observation in each group.

Among those who progressed to ESRD, 39.2% received an ESA in the preceding 12 months. The proportion was much smaller in those who died (12.1%) and those who survived (7.6%). ESA use increased steadily from 17.3% to 28.2% over the 12 months for those who progressed to ESRD, but little or no increase occurred in those who died (5.4%-6.4%) and those who survived (3.6%-4.4%). Of those who progressed to ESRD, 17.3% received a transfusion. The proportion was a bit higher in those who died (21.7%) and much lower in those who survived (6.6%). The transfusion rates (per 1,000 pt-month) were similar for the 6 to 12 months prior to death or ESRD, ranging from 21-29 and 14-21, respectively, and showed a sharp increase in the 5 months prior to death (35-72), but only in the 3 months prior to ESRD (20-46). Those who survived showed little or no increase (7-11).

Death was more common than progression to ESRD in CKD pts. Throughout the 12 months preceding death/ESRD, the transfusion rate was substantially higher than among CKD pts surviving free of ESRD. In contrast, ESA use among the pts who died showed a low, stable pattern very similar to those who survived, while pts who progressed to ESRD had a higher and increasing pattern of use.

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PUB640

The Association of Metabolic Syndrome and Chronic Kidney Disease in Thai Army Population Ouppatham Supasyndh,[1] Natee Mayteedol,[1] Bancha Satirapoj,[1] Amnart Chaiprasert,[1] Duangporn Phulsuksombuti,[2] Darunee Utainnam.[2] *[1]Division of Nephrology, Department of Internal Medicine, Phramongkutklao Hospital and College of Medicine, Bangkok, Thailand; [2]Armed Forces Research Institute of Medical Sciences, Bangkok, Thailand.*

Background: Metabolic syndrome (MS) is a common risk factor for cardiovascular disease. MS have been shown to be increased risk of chronic kidney disease (CKD). However, a large study from Thai population is scarce.

Objective: To demonstrate the association between MS and CKD in the Thai army population.

Method: A cross-sectional study was conducted in Thai army population aged 35-60 years in Bangkok and perimeter. MS was defined by National Cholesterol Education Program Adult Treatment Panel III modified criteria (mod MET-NCEP). CKD was defined as a glomerular filtration rate below 60 ml/min/1.73 m^2.

Results: A total of 22,120 subjects were examined, but only 20,001 subjects were recruited in the study. There were 90.0% males, mean age of 41.29±6.39 year, body mass index (BMI) of 24.08±2.64 kg/m^2, waist circumference of 83.70 ±8.91 cm, systolic blood pressure of 131.05±17.06 mmHg, and diastolic blood pressure of 82.56±11.58 mmHg. Mean of fasting plasma glucose, triglyceride, HDL-cholesterol and serum creatinine were 102.03±31.73 mg/dl, 159.48±131.16 mg/dl, 56.14±17.07 mg/dl and 1.03±0.28 mg/dl, respectively. The prevalence of MS was 18.0 % and 7.0 % had CKD as a co-morbid disease. MS was associated with increased risk for developing CKD (odds ratio [OR] 1.21; 95% CI 1.04 to 1.41). The components of MS associated CKD were hypertension (OR 1.33; 95%CI 1.18 to 1.51) and hypertriglyceridemia (OR 1.15; 95%CI 1.02 to1.30).

Conclusion: These findings indicate that MS defined by mod MET-NCEP is associated with CKD in Thai population. The important components of MS which seem to be risk factors of CKD are hypertension and hypertriglyceridemia.

Disclosure of Financial Relationships: nothing to disclose

PUB641

The Renal Function Evaluation in Epileptic Children under Long Term High Dose Valprorate Treatment Ling-Mei Chiang,[2] Pu Duann.[1] *[1]Medicine/Nephrology, UMDNJ-Robert Wood Johnson Medical School, New Brunswick, NJ; [2]Pediatric/Pediatric Neurology, Chang Gung Memorial Hospital, Keelung, Taiwan.*

Valprorate is a safe, broad spectrum, effective anti-epleptic medicine. Either valproric acid or vaprorate is obligatory hepatic (>95%) excretion thus kidney is pharmacologically spare from aberrant effects. However, renal complications such as Fanconi syndrome, tubular interstitial nephritis and hyponatremia were reported. The mechanism of nephrotoxicity is not yet clear but only observed in victims on prolonged high dose usage thereby highly suggested that an overwhemed dosage exhausts hepatic disposal capacity