Average weight was 57.1 Kg (range 39 – 69; S.D. 6.9) and average urea distribution volume (V) was 29.28 (range 21.7- 40.98; S.D. 3.44).

We included 48 patients with Qd 500 mL/min and 23 with Qd 400 mL/min (67.6% and 32.3% respectively).

There were 36 withdrawals: 13 patients died (18.3%), 12 patients (16.9%) changed to PD, 6 recovered renal function (8.45%), 1 (4.23%) was lost from RTS network and lost for follow up and 2 patients (2.85%) received renal transplantation.

The main cause of death was cardiovascular: 33.8% (7 patients: myocardial infarction, congestive heart failure, cerebrovascular event), 5 patients (38.4%) died because of sepsis and one patient died by cancer (7.69%)

Annual mortality rate in our population was 16.6%. Bivariate analysis at two year of follow up was not statistically different (p=0.427) between Qd 400 mL/min and 500 mL/min. A Kaplan Meier analysis at two year of follow up was not statistically different (7.69%).

5 patients (38.4%) died because of sepsis and one patient died by cancer (2.85%) received renal transplantation.

There were no differences in Kt/V, phosphate, calcium and parathyroid hormone (PTH) between Qd 400 vs 500 mL/min.

There was no statistically significant difference between Qd with outcome of mortality adjusted to age, gender, hemoglobin, serum phosphate, PTH, diabetes mellitus, hypertension, vascular Access and time in dialysis. Table 1.

**Table 1. Adjusted HR for mortality vs clinical variables (n=71)**

<table>
<thead>
<tr>
<th>Variable</th>
<th>HR</th>
<th>IC 95%</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male gender</td>
<td>1.44</td>
<td>0.4-43</td>
<td>0.53</td>
</tr>
<tr>
<td>Age</td>
<td>0.93</td>
<td>0.9-1.03</td>
<td>0.94</td>
</tr>
<tr>
<td>Body weight</td>
<td>0.95</td>
<td>0.87-1.02</td>
<td>0.21</td>
</tr>
<tr>
<td>Kt/V</td>
<td>0.76</td>
<td>0.16-3.7</td>
<td>0.76</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>0.79</td>
<td>0.59-1.05</td>
<td>0.11</td>
</tr>
<tr>
<td>Phosphate</td>
<td>1.03</td>
<td>0.62-1.72</td>
<td>0.87</td>
</tr>
<tr>
<td>Albumin</td>
<td>0.42</td>
<td>0.18-0.95</td>
<td>0.038</td>
</tr>
<tr>
<td>PTH</td>
<td>0.99</td>
<td>0.99-1.3</td>
<td>0.69</td>
</tr>
</tbody>
</table>

Conclusions: The lowering of Qd in patients receiving chronic maintenance HD with high efficiency membranes allows an adequate dose of dialysis without effects on mortality, but with interesting savings of potable water: 24 Liters were saved in each session per patient. Our calculated 5 years spare of water data is presented in Table 2.

When extrapolating our results to 100 patients, reduction of Qd would result in an annual saving of 345,600 L of water, that is remarkable compared to WHO minimum for basic health protection of at least 20 L per person/day; our saving equals the minimal amount of water for 1 adult for 47 years.

We present our results as part of the Blue Planet dialysis initiatives to reduce the ecological impact of renal replacement therapies and to present HD as an affordable therapy in places with scarcity of water.

**SAT-343**

PROGRESS TOWARDS ENVIRONMENTALLY SUSTAINABLE RENAL CARE IN AUSTRALIA AND NEW ZEALAND

Baraclough, K*,1 Knight, J*,2 Sypek, M*,3 Agar, J2

1 Royal Melbourne Hospital, Nephrology, Parkville, Australia, 2 University of New South Wales, The George Institute for Global Health, Sydney, Australia, 3 Barwon Health, Nephrology, Geelong, Australia

**Introduction:** The healthcare industry, together with its supply chain, contributes significantly to greenhouse gas emissions and natural resource depletion. Dialysis programs have a particularly large carbon footprint, with a recurrent, per capita resource consumption and waste generation profile that is second to none in healthcare. Recognising this, and the need for change, the Australia New Zealand Society of Nephrology (ANZSN), in partnership with the Renal Society of Australasia (RSA; the peak Australasian body for renal nursing and related allied health professionals) and Kidney Health Australia (KHA; the Australian body for consumers and carers), convened a working group in early 2017 to promote and support a transformation to environmentally sustainable care in Australia and New Zealand.

**Methods:** Expressions of interest were sought from environmental sustainability-passionate clinicians, nurses, administrators, and consumers in Australia and New Zealand. This resulted in the formation of the Green Nephrology Action Team (GNAT), whose membership includes 4 nephrologists, a renal technician, two renal nurses, a representative from KHA and a consumer.

**Results:** GNAT has developed a position statement on environmental sustainability and renal care which can be viewed on the ANZSN website. GNAT’s primary focus to date has been on raising awareness within the ANZ renal community about the environmental problems related to dialysis, because willingness to solve them can come only after there is realisation that they exist. To this end, an environmental symposium and workshop were held at the 2018 ANZSN and RSA annual meetings, respectively, and social media platforms for posting/discussing ideas are slowly building. A draft ‘Green Dialysis’ website (which improves, updates and expands an existing website - www.greendialysis.org) is near completion - this aims to serve as a resource for all those in the renal community keen to address the environmental impact of their own practice. Recognising the need for a strong evidence base to guide practice change, GNAT has also developed a list of ‘green’ research priorities and funded two Environmental Research Prizes, one each for RSA and ANZSN, which were awarded for the first time at the 2018 RSA and ANZSN annual meetings.

**Conclusions:** Climate change, resource consumption and waste management are issues that impact us all. As a nephrology community, we have a responsibility to minimise the adverse effects of our own practice and to protect and promote a safe and healthy environment for the sake of our patients and the broader global community. GNAT seeks to encourage the Australia and New Zealand nephrology community to adopt resource conservation measures and environmental sensitivities, and to show other nephrology communities and healthcare sectors how to do the same.

**POSTER SESSION: PODOCYTES, MESANGIAL CELLS AND GLOMERULAR FUNCTION**

**POS27**

14/04/2019

Exhibition hall (Doors 7 & 8)

12:00-13:15

**SUN-001**

A NEURO-RENAL SYNDROME WITH NEUROFASCIIN ANTIBODIES

BUKHARI, S*1, Cathro, H3, Solorzano, G2, Gwathmey, K4, Bowman, B1

**Abstract:**

We present our results as part of the Blue Planet dialysis initiatives to reduce the ecological impact of renal replacement therapies and to present HD as an affordable therapy in places with scarcity of water.

**Table 2. Water consume and saving in our population**

<table>
<thead>
<tr>
<th>Water saving (L)</th>
<th>500 mL/min</th>
<th>400 mL/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>One single HD Session</td>
<td>120</td>
<td>96</td>
</tr>
<tr>
<td>Monthly per patient*</td>
<td>1,440 (1.44 m³)</td>
<td>1,152 (1.15 m³)</td>
</tr>
<tr>
<td>Yearly per patient*</td>
<td>17,280 (17.28 m³)</td>
<td>13,824 (13.8 m³)</td>
</tr>
<tr>
<td>23 patients / year</td>
<td>397,440 (397.4 m³)</td>
<td>317,952 (317.9 m³)</td>
</tr>
</tbody>
</table>

**Prizes**

One each for RSA and ANZSN, which were awarded for the first time at the 2018 RSA and ANZSN annual meetings.

**Conclusions:** Climate change, resource consumption and waste management are issues that impact us all. As a nephrology community, we have a responsibility to minimise the adverse effects of our own practice and to protect and promote a safe and healthy environment for the sake of our patients and the broader global community. GNAT seeks to encourage the Australia and New Zealand nephrology community to adopt resource conservation measures and environmental sensitivities, and to show other nephrology communities and healthcare sectors how to do the same.
INTRODUCTION: We present a case of focal segmental glomerulosclerosis (FSGS) and acute-onset chronic inflammatory demyelinating polyneuropathy (CIDP) associated with rare neurofascin autoantibodies, supporting a newly defined neuro-renal syndrome.

METHODS: A 48-year-old African-American male presented to the hospital with lower extremity paresthesias that progressed rapidly to bilateral upper and lower extremity weakness with areflexia. He underwent an electrodagnostic study that demonstrated sural sparing, absent F waves, conduction block and markedly reduced recruitment of normal appearing motor unit potentials, consistent with acute inflammatory demyelinating polyneuropathy (AIDP) and received 2 g/kg of intravenous immunoglobulin. He also developed new-onset proteinuria with 10 g on spot urine albumin:creatinine ratio (UACR). Urine microscopy revealed 10-20 RBCs/HPF with few dysmorphic RBCs and a fine granular cast. A limited kidney biopsy with 3 glomeruli without tubulointerstitial damage on light microscopy (LM) and severe podocyte foot process effacement on electron microscopy (EM) was interpreted as minimal change disease (MCD). He was started on 1mg/kg prednisone daily; however, subsequently developed respiratory failure due to neuromuscular weakness requiring mechanical ventilation and was treated with plasmapheresis for 7 sessions. The patient was later extubated and discharged to acute rehabilitation. He was readmitted 2 weeks later for a suspected AIDP relapse with respiratory failure, again prompting intubation and plasmapheresis for 5 sessions. Proteinuria worsened to 24 gm despite continued treatment with prednisone. A repeat kidney biopsy showed 1/12 glomeruli with segmental sclerosis and acute tubular damage on LM, no deposits on immunofluorescence and severe podocyte foot processes effacement on EM, resulting in a new diagnosis of primary FSGS. In light of waxing and waning symptoms and protracted course he was diagnosed with acute-onset CIDP. A detailed work up returned positive for NF antibodies, NF140 cytosis and urinary protein (Kidney Int 2011). Patient was treated with 2 g/kg methylprednisolone and tacrolimus resulting in marked improvement of both proteinuria of 100 mg and polyneuropathy over 6 months of follow up.

RESULTS: FSGS and CIDP may share immunopathogenic mechanisms. NF is a protein encoded by NFASC gene and belongs to the L1 family of immunoglobulin cell adhesion molecules that are involved in neuronal cell adhesion and function. The presence of NF antibodies and concurrence of severe proteinuria and polyneuropathy suggests a strong neuro-renal interface. NF antibodies are typically of the IgG4 subclass.

CONCLUSIONS: Physicians should have a high clinical suspicion for sampling errors on kidney biopsy if the disease in question does not seem to respond to adequate treatment. Our patient was first diagnosed with MCD and AIDP; however, despite adequate treatment suffered a relapse and was later diagnosed with FSGS and a rare subtype of CIDP. The pathophysiological role NF 140 and NF 155 antibodies in FSGS and their prognostic significance needs validation in further studies.

SUN-002
INSERTIONS AND DELETIONS (INDELS) IN THE NON-TRANSLATED HUMAN GENOME UPSTREAM OF ZHX2 ALTER ZHX2 MRNA EXPRESSION IN MCD AND FSGS

CHUGH, S1

1Rush University Medical Center, Internal Medicine, Chicago, USA

INTRODUCTION: ZHX proteins, especially ZHX2, play a critical role as transcriptional regulators of human and experimental podocyte disease. Multiple groups conducting whole exome sequencing were unable to identify recurrent mutations in these genes in human glomerular disease. We sequenced the genome upstream of ZHX2 and the intronic sequence looking for insertions and deletions that may induce DNA conformational changes, resulting in altered ZHX2 expression.

METHODS: The 1.3 million bp region between the beginning of the immediate upstream gene HAS2 and the end of ZHX2 was sequenced in 28 patients with nephrotic syndrome (8 MCD, 2 FSGS tip lesion, 8 FSGS with mutations in slit diaphragm genes, 4 recurrent FSGS, 2 recurrent non-S1A collapsing glomerulopathy, and 4 Hodgkin disease with nephrotic syndrome) and 27 healthy controls using Agilent Custom capture and high throughput illumina sequencing to obtain about 8 million sequences per sample. The Qiagen Biomedical Genomics Workbench software was used to identify InDels > 3 bp and > 20 sequences present exclusively in the patient population. One of the InDels identified was replicated in cultured podocytes using CRISPR Cas9 technology to study changes in ZHX2 expression.

RESULTS: We identified 5 InDels (size range 6 to 67) shared by than one patient and 40 others (size range 4 to 133) present in a single patient. These InDels were absent in controls and the 1000 genomes project. Patients with MCD and FSGS tip lesion had a high percentage of deletions (approximately 80%), whereas those with other forms of FSGS had mostly insertions (approximately 66%). Significance of these indels was verified by inserting one of these indels upstream of ZHX2 in single cell derived immortalized human podocytes by CRISPR-Cas9 approach. Podocytes carrying this InDel developed reduced ZHX2 expression.

CONCLUSIONS: Insertions and deletions upstream of the ZHX2 gene are commonly present in patients with MCD and FSGS patients and alter ZHX2 mRNA expression.