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POLYURIA IN A PATIENT WITH HYPONATREMIA: RESIDUAL WATER PERMEABILITY - A CONCEPTUAL EXPLANATION: H. Dara Dastoor, Rahba Hospital, Abu Dhabi, UAE; Chandra Mauli Jha, Burjeel Hospital, Abu Dhabi, UAE; Samra Abouchacra, Tawam Hospital,UAE; Hatem Abeid Al Dein, UAE

A consult was sought for a patient of hyponatremia in a 71-year-old diabetic, hypertensive lady who had, gingival carcinoma treated with 3 cycles of Paclitaxel + Cisplatin followed by radiotherapy in 2012 and hysterectomy for poorly differentiated uterine adenocarcinoma in 2014. Review revealed that she had a chronic Hyponatremia - Plasma Sodium (P_{Na}) 127 and 118 mmol/l one month and 8 days earlier respectively. She had presented with dizziness, nausea, vomiting and a fall without loss of consciousness. Her vitals were normal and she was suspected to be mildly hypovolemic to euvolemic by admitting physician. Her medications included: Metformin, Levothyroxine, amlodipine, rosuvastatin and Nystatin. Her serum electrophoresis, thyroid function test, liver function test, lipid & glucose, urea & creatinine were normal.

She received N. Saline @ 60 to 100 ml/hour. Her daily serum Na trend was as: 113 → 117 → 124 → 125 →124 mmol/L. On day five she developed polyuria (5.4 L ; trend 1.3 → 2.2 → 2.3 → 5.4 L/day). Her 24-hour urinary osmole excretion on the day of polyuria was 956 mOsmole. This polyuria could only be explained by "Restrictive Water Permeability". In the absence of ADH, luminal Osmolarity in medullary collecting duct (MCD) is lesser than interstitial osmolarity. This gradient drives fluid reabsortion which can exceed >5 litres/day. This can cause vasopressin independent hyponatremia. If these patients are subsequently exposed to a high solute content (IV NaCl, increased protein load) in the absence of vasopressin, the osmolar gradient change results in less fluid absorption and diuresis with risk of rapid rise of serum sodium and central pontine demyelanosis,

While Hyponatremia remains commonest electrolyte disorder, it has been observed that less attention is paid to possibility of states of Osmostat resest, inability to dilute urine in old age and mechanism of Restrictive Water Permeability etc. while investigating and treating a patient of hyponatremia. We would recommend change in practice Habit.

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NEW KIDNEY ALLOCATION SYSTEM HITS THE 305: A. Dejman; F. Cabeza; A. Torres; J. Gaynor, A.Schneegans, P. Ruiz, D.Roth,W. Kupin,G. Burke, G. Ciancio, L. Chen, R.Vianna, A. Mattiazzi, G.Guerra. Kidney Transplant, Miami Transplant Institute, Miami, FL, USA.

The new national Kidney Allocation System (KAS), effective 12/4/14, was designed to improve limitations of the prior system: higher discard rates of kidneys, inequity of access to transplants by blood type, sensitization level, geographic location and high rates of unrealized graft years and re-transplantation due to long potential longevity kidneys allocated to candidates with shorter potential longevity and vice versa. We evaluated the impact of the KAS in patient characteristics and 6-month clinical outcomes of deceased donor kidney transplants in our center.

We performed a retrospective chart review of patients transplanted from 12/2/2014 to 4/31/2015 (N=79). Patient demographics: Mean recipient age was 52.6 years, with 48% in the 50-64 years range. African-Americans represented 70.9% (56/79) of the population followed by Hispanics at 17.7% (14/79). No significant gender difference noted. Pre-transplant diabetes was 25% (20/79). Patients with an Estimated Post Transplant Survival (EPTS) of >20% represented 81% (64/79), and Kidney Donor Profile Index (KDPI) <20% were used in 15.2% (12/79) of them. 6.3% (5/79) had a calculated panel of reactive antibodies of ≥99%. 67.1% (50/79) were locally procured kidneys and 32.9% (29/79) were imported. Cold Ischemia Time >24 hours was 53.2% (61/79). Mean time on dialysis was 120 months, with no preemptive transplantation. 40.5% of our patients were on dialysis for ≥120 months. Clinical outcomes: Delayed graft function (DGF) rate was 33% (26/79) and slow graft function (SGF) 12.7%. 60% (47/79%) of patients required at least one hospitalization for rejection and infections with 19.0% (15/79) and 25.3% (20/79) respectively.

Based on this analysis KAS is achieving its goals. However, our center had increase DGF and SGF and high EPTS scores that led to increase in re-hospitalization rates and greater economic burden.

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SEVERE LUPUS NEPHROPATHY (WITHOUT ANY PRIOR HISTORY OF SYSTEMIC LUPUS ERYTHEMATOSUS) PRESENTING AS SEVERE PREECLAMPSIA

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A 35-year-old white female G3P0020 presented with 29 weeks of gestation with progressive lower extremity (3-4+ edema). Past medical history was significant for hyperthyroidism, treated with propylthiouracil. Physical examination revealed hypertension with a blood pressure 154/92 mmHg. Initial laboratory studies showed a BUN of 49 mg/dl, and a serum creatinine of 1.6 mg/dl and uric acid of 8.6 mg/dl. The urine examination revealed 3+ protein and blood on dipstick analysis, and a calculated protein-to-creatinine ratio 5.2. Clinical diagnosis of severe preeclampsia was made that resulted in an emergent caesarean section. Postpartum, she developed oliguric acute kidney injury, and required renal replacement therapy. Subsequent evaluation showed low C3, C4, and positive ANA (1:1280), dsDNA of 210 IU/ml, with a negative C-ANCA and positive P-ANCA 1:640. Renal biopsy demonstrated diffused proliferative crescentic glomerulonephritis with "full house" immunofluorescence consistent with class 4 WHO classification. Patient responded to treatment with corticosteroids, mycophenolate and plasmapheresis. Patient had renal recovery with serum creatinine returning to baseline. The unique feature of our patient was the presentation as pre-eclampsia with no prior history of SLE.

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NEVER TOO LATE FOR RECURRENT DISEASE

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Recurrent primary renal disease is emerging as an important cause of allograft dysfunction. We present a case of recurrent lupus nephritis that presented 26-years after transplantation.

A 58-year-old Caucasian female presented with edema and new onset proteinuria with dysmorphic hematuria. She was a recipient of a living related renal transplant from her brother for end stage renal disease due to lupus nephritis 26 years prior. Since her transplant she has had stable renal allograft function with a serum creatinine ranging from 0.8-1.0 mg/dL and urine protein excretion < 100 mg/day. Her immunosuppression consisted of azathioprine 100mg /day and prednisone 10 mg/day. At presentation 24-hour urine protein was 1.8 gram, urine albumin to creatinine ratio was 2.4g/g. Serological testing revealed an anti dsDNA >1000, consumed C3 and C4. Renal allograft biopsy showed a proliferative glomerulonephritis with features consistent with recurrent lupus class IV with moderate activity and chronicity.

She was treated with prednisone 60 mg/day for 2 weeks; tapered over 6 weeks and azathioprine was substituted with mycophenolate mofetil (MMF). Serology, proteinuria and hematuria resolved over the following 9 months.

This case represents the latest recurrence of lupus nephritis and the efficacy of MMF in controlling recurrence.