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CASE OF SEVERE HYPOTHYROIDISM AS A CAUSE OF REVERSIBL ACUTE KIDNEY INJURY Sapna Jariwala, Priya Deshpande, Lori Wang, Lyudmila Shvets, Mount Sinai Doctors Brooklyn Heights, Brooklyn, NY USA

Hypothyroidism is thought to be a cause of reversible acute kidney injury (AKI) that can be rapidly improved with thyroid hormone replacement. We describe the case of TO, an 18-year-old girl with history of obesity, Cri-du-chat syndrome, and mental retardation, who was referred to renal by her endocrinologist for an elevated serum creatinine (Cr) level. Six months prior to the renal visit, she developed weight gain, cold intolerance, dry skin and bilateral lower extremity edema. One month prior to the renal visit, TO was found to have an elevated serum Cr of 1.49mg/dL, a severely elevated thyroid stimulating hormone (TSH) level of 802.5uIU/mL (normal 0.45-4.5) and a low free T4 of 0.04ng/dL (normal 0.93-1.60). On evaluation, vital signs were significant for borderline hypotension, and physical exam revealed 3+ non-pitting edema bilaterally in the lower extremities.

TO was diagnosed with severe hypothyroidism (secondary to Hashimoto's thyroiditis), and admitted to the hospital where adrenal insufficiency was ruled out prior to starting thyroid hormone therapy. In the hospital, her Cr was 1.7mg/dL, urinalysis was negative for proteinuria and hematuria, and her renal sonogram was normal. She was started on levothyroxine 100ug daily. Three weeks after discharge, patient's labs showed that her Cr improved to 1.02mg/dL and her TSH was 14uIU/mL. One year after hospitalization her Cr was 0.85mg/dL and her TSH was 1.16uIU/mL.

Thyroid hormone has been shown to impact renal development, water excretion (i.e. through its effect on aquaporin channels), and can influence the glomerular filtration rate (GFR). The hemodynamic effects of hypothyroidism, such as decreased cardiac output and lower systemic blood pressure, result in reduced renal blood flow causing AKI. There are also some reports of patients with end-stage renal disease attributed to severe hypothyroidism. Studies have shown that initiation of thyroid hormone replacement therapy normalizes Cr and GFR. Karanikas et al [1] looked at Cr-EDTA renal scans in thyroidectomized patients with severe hypothyroidism and found that a decrease in serum Cr after thyroid replacement was associated with improved GFR by Cr-EDTA clearance. Our case demonstrates that clinicians should assess thyroid function in patients with AKI of unclear etiology. [1] Karanikas G, Schutz M, Szabo M, Becherer A, Wiesner K, Dudczak R, Kletter, K. Isotopic renal function studies in severe hypothyroidism and after thyroid hormone replacement therapy. Am J Nephrol 24:41-45, 2004.

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HYPONATREMIA: PROPOSED NEW CLASSIFICATION BASED ON URINE OSMOLARITY & PATHOPHYSIOLOGY: Chandra Mauli Jha, Burjeel Hospital, Abu Dhabi, UAE; H. Dara Dastoor, Rahba Hospital, Abu Dhabi, UAE; Samra Abouchacra, UAE; Hatem Abeid Al Dein, UAE.

Present classification of Hypotonic Hyponatremia based on Urine Osmolarity (Uosm) & volume status of patient considers Syndrome of Inappropriate ADH (SIADH) if patient is euvolemic & Uosm > 100 mOsm/kg. This classification (1) over-diagnoses SIADH by not recognizing that many older patients may not reduce Uosm < 100 Osm/kg even without ADH; (2) fails to account the Syndrome of Hyper Responsiveness to ADH, and (3) it misses the possibility of "Reset Osmostat" as a cause of Euvolemic Hyponatremia. The recent European Best Practices on Hyponatremia have also highlighted shortcomings of the current classification.

We propose to divide Hyponatremia based on U_{osm} relative to Plasma Osmolarity (P_{osm}) and further classification based on patho-physiologic mechanisms of Hyponatremia. In the proposed classification the hyponatremia is classified into two broad groups (1) $U_{osm} < P_{osm}$ [ADH Independent] and (2) $U_{osm} > P_{osm}$ [ADH dependent]. ADH Independent state would further be classified into (A) those due to due to decreased filtrate delivery to Distal Nephron (B) those due to defect in "Restrictive Water Permeability" of distal Nephron and (C) those due to hyper-responsive state to ADH, reset Osmostat state or water intake relatively more while concomitant ADH suppression is modest etc.

This classification would prompt clinician to consider states of "Reset Osmostat" and "Decreased Residual Water Permeability" etc. while investigating hyponatremia and it would decrease over-diagnosis of SIADH. This would reflect in modified treatment pattern and unnecessary over-use of drugs like Demeclocycline.

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RITUXIMAB ADMINISTRATION ASSOCIATED PNEUMOCYSTIS JIROVECII PNEUMONIA IN A RENAL ALLOGRAFT PATIENT: Chandra Mauli Jha, Burjeel Hospital, Abu Dhabi, UAE; H. Dara Dastoor, Rahba Hospital, Abu Dhabi, UAE; Samra Abouchacra, UAE; Hatem Abeid Al Dein, UAE.

A 62 year old male Indian with living unrelated renal Transplant received 1 gm Rituximab, methylprednisolone & Plasma Exchanges for drug induced Thrombotic microangiopathy (TMA). TMA subsided but four weeks later there was dyspnoea, unproductive cough &fever. He had low Oxygen Saturation on Room air (88%), WBC $4.3x10^9$/L, $CD4^+$ T cells 243 /µl; HIV: negative; CXR: bilateral patchy infiltrates; CT Chest: bilateral Ground glass patchy infiltrates in lower lobes. The patient was treated empirically with Bactrim for Pneumocystis Pneumonia. Broncho-alveolar Lavage confirmed Pneumocystis organism. Treatment with Bactrim for 4 weeks was followed by prophylaxis against Pneumocystis empirically decided for 6 months.

Pneumocystis is an opportunistic serious infection among immunosupressed. Classically helper T-cells ($CD4^+$) is known to defend against Pneumocystis with high risk when CD4 T cells are < 200/µL. Rituximab is a monoclonal antibody which deplete B-cells. It was introduced as a treatment for non-Hodgkin's lymphoma which by now has found use in many immunological disorders like SLE, Rheumatoid arthritis and glomerulonephritis etc. Pneumocystis was not reported in original drug trial and a prophylaxis against Pneumocystis is not considered whenever Rituximab is administered, as was in our case too. But, literature search shows that there are experimental proofs of B-cells requirement for T-cell memory & effector function against Pneumocystis. There are two case reports of Pneumocystis ***jirovecii pneumonia (PJP)*** after Rituximab administration in patient of Lupus and Rheumatoid arthritis. There was no report of post-Rituximab Pneumocystis in solid organ patient. It is 1st such report.

We would recommend considering Pneumocystis prophylaxis in patients who are going to receive Rituximab and it would be strongly indicated in those who would remain on multiple immunosuppressives after Rituximab administration.

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ASSOCIATIONS BETWEEN ELEVATION OF RESIDENCE AND ERYTHROPOIETIN STIMULATING AGENT DOSE IN HEMODIALYSIS PATIENTS: Yue Jiao, William Wacker, Zuwen Kuang, John Larkin, Len Usvyat, Peter Kotanko, Jeffrey Hymes, Franklin W Maddux, Fresenius Medical Care North America, Waltham, MA, USA, Renal Research Institute, New York, NY, USA

It has been previously reported that elevation of residence can affect patterns of erythropoietin stimulating agent (ESA) dosing in incident hemodialysis (HD) patients (Brookhart, et al. 2008). We investigated whether the elevation of residence is associated with differing patterns of ESA dosing levels in the general HD population.

We used ESA dosing data from all chronic in-center HD patients treated at Fresenius Medical Care North America between Jun 1, 2015 and Aug 31, 2015. Data obtained from the United States Geological Survey was utilized to determine the patients' elevation of residence. Spline regression models were constructed to determine the associations between the mean ESA dose per patient per HD treatment and elevation of patient residence.

The study analysis included 98,303 chronic HD patients. Overall, the mean ESA dose per patient per ESA administration was observed to decrease with increasing patient elevation of residence. As compared to patients residing at sea level, there was approximately a 3%, 7%, 9%, 13%, and 17% decrease in the ESA dose per patient per treatment for patients residing at 250 meters (820 feet), 500 meters (1640 feet), 750 meters (2461 feet), 1000 meters (3281 feet), and 1250 meters (4101 feet) above sea level, respectively.

This study indicates that ESA doses are lower in chronic HD patients residing at higher elevations. Whether decreased ESA dosing at higher elevations are attributable to increased endogenous erythropoietin production or increased ESA responsiveness is yet to be elucidated and further studies are warranted.