A Rare Case of Acute Myeloid Leukemia Presenting as Central Diabetes Insipidus

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Introduction: Central Diabetes Insipidus (CDI) is an uncommon condition, with an overall incidence of approximately 1:25,000 and is usually associated with neurosurgery, trauma, autoimmune and vascular disease, inflammatory disorders, hypoxic brain injury and brain metastasis. Patients with acute myeloid leukemia (AML) mostly present with symptoms of pancytopenia, noted to have hematologic abnormalities and are subsequently diagnosed with AML after bone marrow biopsy. Here we describe a unique case of a patient who presented with symptoms of CDI and incidentally diagnosed with AML.

Case Description: A 64-year-old male with a history of coronary artery disease presented to his primary physician complaining of polyuria and polydipsia which affected his work as a truck driver. Labs were notable for mild anemia (Hgb 11 g/dL) with macrocytosis, thrombocytopenia (platelet 667 K/μL), serum sodium was 146 mmol/L, Hgb A1c 5%, prostate-specific-antigen 1.4 ng/dL, normal lipid panel, and normal thyroid function. No definitive diagnosis was made and he underwent evaluation by hematology. Peripheral smear showed increased (44%) blasts/promyelocytes, consistent with acute leukemia. Cytogenetic analysis showed an abnormal karyotype of cells with an inverted chromosome 3 and monosomy for chromosome 7. He was admitted for induction therapy and presenting symptoms worsened (~10 L urine output per day) along with hypotension which peaked at 160 mmHg, serum osmolality 319 mOsm/kg and urine osmolality of 174 mOsm/kg despite large oral and peripheral free water supplementation. MRI brain was normal. Hypertension was noted in the context of CDI, empiric treatment with intravenous fluid administration and hypertonic saline (4.2%) was initiated. Unfortunately this translocation has been associated with poor clinical outcome. With this case, we suggest screening patients with CDI for who have an unclear reason of developing of polydipsia and polyuria and if hematologic abnormalities are noted, should undergo prompt evaluation for AML.

A Case of Ketamine-Induced Diabetes Insipidus

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Introduction: To our knowledge there have been six previously published case reports describing central diabetes insipidus (DI) related to ketamine. We present a unique case of central DI associated with ketamine infusion in a critically ill patient with acute respiratory failure.

Case Description: A 52-year-old African American man with medical history of bipolar disorder, polysubstance abuse, chronic obstructive pulmonary disease, hypertension, and deep vein thrombosis was admitted to the medical intensive care unit with hemoptysis and acute respiratory failure. Due to agitation and refractory hypoxemia he required multiple sedating agents. Within hours of starting a ketamine infusion his urine output increased from a mean of 71 mL/hr to 305 mL/hr. Over 48 hours serum sodium (Na+) rose from 142 to 159 mmol/L. Urine osmolality (Uosm) was 132 mOsm/kg. 4 mcg intravenous (IV) desmopressin was administered. 90 minutes later Uosm had increased to 646 mOsm/kg. Urine output fell to 49 mL/hr. About 28 hours after the initial dose of desmopressin polyuria recurred and Uosm fell to 272 mOsm/kg. IV desmopressin was re-administered at 2 mcg with a similar response to the first dose. Na+ normalized with free water replacement. Ketamine was stopped. Urine output, Uosm, and Na+ remained stable without further intervention. Alternative etiologies for central DI such as hypoxic brain injury were considered but felt to be less likely due to the strong temporal relationship with ketamine. The Naranjo adverse drug reaction (ADR) likelihood score was 5 indicating a probable ADR.

Discussion: This case reinforces the association between ketamine and central DI which has been described in prior case reports. A hypothesized mechanism is ketamine’s antagonism of N-methyl-D-aspartate receptors in the posterior pituitary thus inhibiting arginine vasopressin production. Ketamine is being used with greater frequency in critical care. In this case, we want to recognize this rare but potentially serious complication. Monitoring of Na+, Uosm, and urine output should be considered. When central DI related to ketamine is identified, withdrawal of the drug appears to be corrective.

Multi-Electrolyte Storm Associated with Non-Exocrine Manifestations of Sjögren’s Syndrome

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Introduction: Kidneys are one of the most commonly affected non-exocrine glands by Sjögren’s syndrome. Renal involvement includes glomerulonephritis, interstitial nephritis, or both. Chronic TIN is the most common renal manifestation in Sjögren’s syndrome. Clinical manifestations of TIN present as abnormalities of tubular function with Fanconi syndrome, Distal RTA and DI.

Case Description: 26 yo Hispanic female with no known past medical history with a one year history of polyuria and fatigue. Workup was significant for elevated creatinine with proteinuria and pyuria, normal anion gap metabolic acidosis, hypokalemia, and hypophosphatemia. Urine studies revealed an inappropriately alkaline urine with impaired renal reclamation of potassium and phosphorous. Serological tests were significant for ANA, SSA, and SSB antibodies. Kidney biopsy revealed acute tubulointerstitial nephritis. The patient was started on an IV steroid course with oral taper and hydroxychloroquine with improvement in Cr from 2.2 to 1.2 mg/dL with a potassium, phosphate and sodium bicarbonate supplementation regimen.

PO1157

Estimating 24-Hour Urinary Excretion Using Spot Urine Measurements in Kidney Stone Formers

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Background: One limitation of the use of the 24-h collection, a key element in the management of kidney stone (KS) disease, is impracticability. To overcome this limitation, we analyzed the performance of spot urine measurements to estimate 24-h excretion in patients with KS.

Methods: 74 adult KS patients from two centres were instructed to perform a 24-h urine collection. A sample of the last micturition (fasting, upon awakening) was sent for spot urine analysis. Twenty patients were asked to collect two additional spot urine samples, one before dinner (pre-prandial) and the other after dinner (post-prandial).

Results: The performance of estimation of urine measurements with spot urine samples to estimate 24-h excretion in KS patients.

Conclusions: Utilizing measured or estimated 24-h urinary creatinine substantially increases the utility of spot urine samples in estimating 24-h excretion of urinary analytes in KS formers.

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Evidence for Abnormal Linkage Between Urine Oxalate and Citrate Excretion in Human Kidney Stone Formers

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Background: Animal models have demonstrated an interactive relationship between the epithelial anion exchanger SLC26A6 and transporter NAD1 that regulates citrate and oxalate homeostasis. This relationship is a potential mechanism to protect against kidney stones as higher urine oxalate is accompanied by higher urine citrate but it has not been explored in humans.

Methods: We examined 24-hour urine data on 13,155 kidney stone forming patients (SF) from separate datasets at the University of Chicago and Litholink, a national laboratory, and 143 non-kidney stone forming participants (NSF) to examine this relationship in humans. We developed a Bayesian linear regression models to examine the association between oxalate and citrate in all study participants and separately in SF and NSF.

Results: Higher urinary oxalate was associated with higher urinary citrate in both SF and NSF. In NSF, the multivariate adjusted urinary citrate excretion was 3.0 (1.5 to 4.6) (mmol)/(mmol) (per oxalate (mmol)/(mmol) creatinine (mmol)). In SF, the multivariate adjusted urine citrate excretion was 0.3 (0.2 to 0.4) (mmol)/(mmol) creatinine (mmol) per oxalate (mmol)/(mmol) creatinine (mmol).

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