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Non-conventional therapy of a triple acid base disorder

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LATE-ONSET GROSS HEMATURIC AKI FROM IGAN:

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IgA nephropathy(IgAN) the most common glomerulonephritis (GN) worldwide has a variable presentation and course making the clinical diagnosis challenging. We present an unusual case of IgAN with AKI 3-4 weeks after onset of infection.

A 67 y/o Caucasian obese male with h/o HTN, Parkinsons disease burnt his right foot resulting in cellulitis requiring debridement and a 10 day course of oral amoxicillin/clavulanate. He subsequently presented with new onset painless gross hematuria and AKI 2 weeks after completing treatment and resolution of infection. Laboratory findings: creatinine 8.8mg/dl(baseline 1.1mg/dl), urine protein/creatinine ratio 12.6g/g. Urine sediment showed many RBC's, few dysmorphic but no casts. Work up including viral, autoimmune, infectious etiologies, CT chest, abdomen, pelvis, Xray foot were negative. His AKI rapidly progressed to anuria requiring hemodialysis. Renal pathology showed mesangial expansion, thinning of tubular epithelium, extensive blood in tubules, protein casts and IF revealed heavy mesangial IgA deposits. A diagnosis of IgAN was made.

Due to the timing of hematuria 3-4 weeks after onset of infection, Infection related GN (IRGN) was high on our differential however pathology revealed no infiltration of neutrophils or subepithelial electron dense deposits (unlikely IgA-dominant post infectious GN) and no glomerular deposition of IgG/C3 predominance was noted (unlikely IRGN). Our case was unusual in that 1) hematuria was not intercurrent with onset of infection as expected with synpharyngitic IgAN and occurred atleast 2 weeks after complete recovery from infection 2) no prior episodes of hematuria and it was de novo gross hematuria in his late 60's 3) no crescents were noted despite the rapid progression and severity of AKI. Mechanical obstruction by extensive tubular red blood cell casts, associated with cytotoxic effects of oxidative stress from heme and iron released by RBC's explains the gross hematuria and AKI.

AKI from gross hematuria of IgAN can occur as a late finding 2-4 weeks after the onset of a cellulitic infection.

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NON-CONVENTIONAL THERAPY OF A TRIPLE ACID

BASE DISORDER: **Maria Andrievskaya, MD¹**, Daniel Peisach, MD, PhD¹, Jerry Yee, MD¹. ¹Henry Ford Hospital, Detroit, MI, USA

Severe electrolyte derangements are difficult to correct and standard approaches are not always successful. We present a case in which an unconventional treatment approach led to resolution of a severe, mixed acid-base disorder. A 74-year-old female with a history of stroke, seizures, and chronic hyponatremia presented with confusion, gait difficulty, and poor oral intake. Initial vital signs: T 36.3 °C, BP 97/52 mm Hg, HR 70 bpm, RR 16 min-1, BMI 19 kg/m². Admission lab data revealed: Na 118, K 3.2, Cl 89, TCO₂ 23 mmol/L, BUN 11, SCr 0.76, Mg 1.5, Pi 1.7 mg/dL. ABG: pH 7.76, pCO₂ 12.5 mm Hg. Diagnoses of ketolactic acidosis from starvation with metabolic and respiratory alkaloses were established by elevations of serum lactate (3.9 mmol/L) and beta hydroxybutyrate (1.07 mmol/L) levels.

To prevent aggravation of hyponatremia by free-water glucose infusions yet treat the ketosis, a concentrated sugar solution comprised of addition of 20 grams of glucose to 20 oz (contains 77 grams of total carbohydrates) of phosphorus-free Mountain Dew was administered. Cerebral perfusion compromise from hyperventilation and life threateningly high pH were offset by intubation with addition of dead space via increased tubing length. Hypokalemia and hypophosphatemia, from dietary deprivation, were addressed with potassium phosphate administered at 0.5 mmol/kg/day. Hyponatremia, attributed to SIADH with superimposed volume depletion, was gradually corrected at 6-8 mmol/L/day.

Within 24 hours, ketone production was suppressed and arterial pH lowered to 7.41 with pCO₂ of 31.8 mm Hg. This case demonstrates efficacy of a sugar-sweetened beverage to treat starvation ketosis, and mechanical hypoventilation to correct severe respiratory alkalosis. In addition to utilizing traditional protocols, physicians must be creative and consider non-conventional approaches to patients.

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DIABETIC FIBRILLOSIS: AN UNEXPECTED FINDING:

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Diabetic fibrillosis is a rare finding that was first described in 1970 by Sohar et al when they observed small sized fibrils in the expanded mesangium of diabetic patients with nodular glomerulosclerosis. This is a report of a patient with diabetic kidney disease whose renal biopsy showed unusual mesangial fibrillary structures consistent with diabetic fibrillosis.

A 56 year old African American woman with DM type 2, diabetic neuropathy, hypertension, dyslipidemia, obesity admitted with symptomatic hypoglycemia. She reported dyspnea, lower extremity edema, and progressive orthopnea that started 2 weeks prior to admission. On admission, her creatinine was 2.85 mg/dL (baseline creatinine 1.0-1.2 mg/dL seven months ago) and peaked at 4.5 mg/dL. Nephrology was consulted for AKI on CKD and nephrotic syndrome. Her labs were significant for UA showing WBC 0-2, RBC 2-5, no casts, urine eosinophils (+). The urine protein/creatinine ratio was 16.2 g/day. Serology workup for her proteinuria showed ANA, Rheumatoid factor, RPR, ANCA screen, anti-Glomerular basement membrane, Phospholipase A2 receptor AB, SPEP, UPEP, HIV, Hepatitis B and C were all negative.

Kidney biopsy showed classic diabetic nodular glomerulosclerosis (KW), Acute Interstitial Nephritis in addition to advanced interstitial fibrosis and tubular atrophy (up to 70%), with moderate arteriosclerosis and arteriolar hyalinosis on light microscopy. The Congo Red stain was negative. Surprisingly on electron microscopy, organized fibrillary deposits were seen in the mesangium, measuring 12-15 nm (Fig.1) and characteristic of an uncommon finding: DIABETIC FIBRILLOSIS.

This case raises awareness of this potential findings on electron microscopy that should not be confused with other fibril deposits that can look similar, for example in conditions like Amyloidosis, Fibrillary GN and Immunotactoid GN. This finding does not have any correlation with the long-term kidney prognosis.

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PAGE KIDNEY AND DECREASED RENAL FUNCTION SECONDARY TO RENAL SUBCAPSULAR HEMATOMA AFTER EXTRACORPOREAL SHOCK WAVE LITHOTRIPSY

(ESWL): Waleed Asfar¹, David Rodriguez¹, Mohammed Mohialdeen¹, Ruth Campbell¹. ¹Medical University of South Carolina, Charleston, SC, USA

Page kidney refers to hypertension that develops following long-standing compression of renal parenchyma by subcapsular renal collection like hematoma, seroma and urinoma. Compression of the renal parenchyma results in compression of the renal vessels which leads to decreased blood flow and activation of the renin-angiotensin system. The renin-angiotensin system activation results in a rise in blood pressure and development of hypertension. This phenomenon was first described by Irvine H Page in 1939 when he discovered that hypertension could be produced in a dog by wrapping one or both kidneys in cellophane.

We describe a case of page kidney in a 44 year old female who developed a large right sided subcapsular hematoma after a stented ESWL. She had continued pain and was found to have a large subcapsular hematoma a week after her procedure. She developed new hypertension with blood pressure in the 180s systolic requiring medical management. She was followed with repeat imaging 2 weeks post op that showed mild worsening of hematoma size from 3.5 to of 3.8 cm of the right kidney. Her hematoma was found to be stable in size 4 weeks post op and she maintained normal kidney function with creatinine of 0.63. She underwent nuclear medicine renal scan that showed renal function discrepancy with decreased perfusion and function of the right kidney at 36%, but without evidence of obstruction. Patient was not taking any anti platelet medications or blood thinning medications.

Given evidence of decreased renal function on renal scan and the onset of new Hypertension it was felt that patient has page kidney and decision was made to drain patient's hematoma in order to preserve her renal function and treat hypertension.

We encourage keeping a high index of suspicion following procedures that can lead to subcapsular hematoma such as kidney biopsy and ESWL, this includes close hemodynamic monitoring and follow up imaging.

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NEPHRECTOMY: AN OLD TREATMENT FOR A COMMON DISEASE:

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