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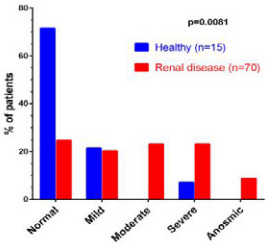
OLFACTORY DEFECTS IN RENAL DISEASE: Sagar U. Nigwekar, Jeremy M. Weiser, Sarah M. Dougherty, Joshua L. Wibecan, Sahir Kalim, Kristin M. Corapi, Nwamaka D. Eneanya, Dennis Brown, Ravi I. Thadhani, Teodor G. Paunescu, Massachusetts General Hospital and Harvard Medical School, Boston, MA, USA

Malnutrition and cachexia are prevalent in renal patients and are associated with increased morbidity and mortality. We set out to investigate whether olfactory defects that could lead to food aversion exist in chronic kidney disease (CKD) and end stage renal disease (ESRD) patients, in order to assess their putative contribution to the pathogenesis of malnutrition.

We quantified odor identification and threshold in CKD (n=28, 60.3±2.5 years old, mean±SEM) and ESRD patients (n=42, 57.4±2.5 y.o.) in comparison to healthy volunteers (n=15, 55.3±1.8 y.o.), using the standardized University of Pennsylvania Smell Identification Test (UPSIT) and Smell Threshold Test (Sensonics, Haddon Heights, NJ).

Odor identification is normal in healthy volunteers, who obtained 87.0±2.8% correct answers on the UPSIT test, while renal patients identified 70.5±1.9% of the odorants correctly (p=0.0004). The CKD and ESRD patient subpopulations scored 75.4±2.5% and 67.4±2.6% correct answers respectively, significantly less than healthy volunteers (p=0.0071 and p=0.0001).

Subjective assessments of smell and taste were similar among the control, CKD, and ESRD groups, thus not correlating with the UPSIT scores. No statistically significant odor threshold differences were observed among these cohorts.



The results of our study indicate that CKD and ESRD patients have olfactory deficits and that the UPSIT test is an appropriate tool to quantify them. Mitigating these olfactory defects may improve dietary intake and nutritional status in renal disease patients.

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ANURIA RESULTING FROM BILATERAL URETERAL OBSTRUCTION MISSED ON ULTRASONOGRAPHY AND COMPUTED TOMOGRAPHY: Chukwunweike Nwosu, Sindu Yenigalla, Shaik Abdul Samad, Ritesh Rampure, Abington Jefferson Health, Abington PA, USA

Most causes of anuria in adults are obstructive distal to the bladder. It may also result from ureteric obstruction of a solitary kidney. Bilateral synchronous involvement of kidneys and ureters is rare and obstruction commonly results in dilatation upstream with hydronephrosis which is usually evident on US and CT imaging of the urinary tract.

A 70 year old female with history of renal calculi presented with anuria. She visited the emergency room a day prior for left flank pain, had CT abdomen and pelvis which showed bilateral non obstructing renal calculi without hydronephrosis, and was subsequently sent home on Ibuprofen 600mg 6hrly as needed. Her physical exam was unrevealing. She had hematuria and her serum creatinine had increased from 1.2mg/dl the day before to 4.4mg/dl. Retroperitoneal US scan done had similar results as the CT. Further lab tests for Ibuprofen nephropathy and glomerular disease were sent; nephrology and urology consults were placed. The urologist was hesitant pursuing an invasive investigation as both CT and US imaging did not show obstruction. The nephrologist wanted renal biopsy after ruling out obstruction. She eventually had cystoscopy with bilateral retrograde pyelography which revealed left distal ureteral stone and right ureteral stricture. She subsequently had bilateral double stent insertion and immediately started making urine. Her creatinine returned to normal limits within few days and she later had the stones removed.

Our unusual case of anuria from bilateral pelvicalyceal system obstruction was missed on CT and US imaging. This questions their reliability and emphasizes the fact that obstruction must not necessarily induce dilatation upstream. If present, there might not be a marked dilatation upstream on evaluation with imaging techniques and direct opacification of the urinary tract can help confirm presence of obstruction. Early diagnosis and intervention is critical to preventing uremia, irreversible renal injury, rupture of the collecting system and consequently urinary extravasation.

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ACCELERATED ACUTE KIDNEY INJURY DURING VANCOMYCIN EXPOSURE: A DISTINCT ENTITY. Ndidiamaka Obadan , Juan Carlos Velez, Department of Nephrology, Medical University of South Carolina, Charleston, South Carolina, USA.

Vancomycin nephrotoxicity has spurred renewed attention due to reports of increased incidence which coincide with recommendations for higher therapeutic trough levels. However, distinct clinical features of vancomycin-associated (VA) acute kidney injury (AKI) have not been reported.

We report the case of a 56 year old African American woman with ischemic cardiomyopathy, type 2 diabetes mellitus, hypertension and prior methicillin-resistant *Staphylococcus aureus* bacteremia who presented with wound dehiscence and pus extravasation from her pacemaker site. She was normotensive and had a BMI 35 kg/m^2. She was started on vancomycin 1 g IV bid. After the 5th dose, her serum creatinine rose from 1.0 to 4.0 mg/dL over 48 hrs. A trough level was 50.3 mcg/ml. Serum CPK was normal. Urinalysis was unremarkable and renal ultrasound showed no hydronephrosis. None of her outpatient medications had been changed. Three days later, her serum creatinine rose to 7.2 mg/dL, she became oliguric, required dialysis but recovered renal function.

A review of existing literature on VA-AKI over the past decade identified 7 articles with 2 cases. Both had morbid obesity, cumulative vancomycin dose of 5g and quadrupling of serum creatinine within 48hrs. In total, we compiled 3 cases of VA-AKI characterized by large rises in serum creatinine [mean (SD) = 3.0 (0.1) mg/dL] without evidence of rhabdomyolysis.

VA-AKI may present as “accelerated” AKI, i.e., with an initial daily rise of serum creatinine larger than the standard daily rise in serum creatinine of around 1.0 mg/dL per day. Because vancomycin is secreted through an organic cation transporter in the proximal tubular epithelia, we speculate that vancomycin may decrease urinary creatinine secretion, leading to a larger rise in serum creatinine during AKI. However, further prospective studies are needed to confirm the emergence of this distinct form of AKI, as well as its potential link to morbid obesity.

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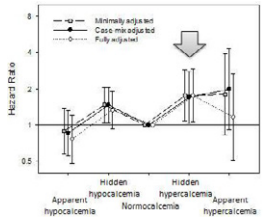
HIDDEN HYPERCALCEMIA AND MORTALITY IN INCIDENT HEMODIALYSIS PATIENTS. Yoshitsugu Obi[1]; Elani Streja[1]; Matthew B. Rivara[2]; Vanessa Ravel[1]; Melissa Soohoo[1]; Wei-Ling Lau[1]; Rajnish Mehrotra[2]; Kamyar Kalantar-Zadeh[1]. [1]*Harold Simmons Center, UC Irvine, Orange, CA;* [2]*Division of Nephrology, University of Washington, Seattle, WA.*

Ionized calcium (iCa) is the bioactive component of serum calcium, but albumin-corrected total calcium (tCa_{ALB}) poorly correlates with iCa in patients with end-stage renal disease. Additionally, among these patients, the association of iCa with survival remains unclear. We hypothesized that hemodialysis patients with hidden hypercalcemia (iCa >1.32 mmol/L and normal tCa_{ALB}) and those with apparent hypercalcemia (iCa >1.32 mmol/L and tCa_{ALB} >10.2 mg/dL) have a similar higher risk for death compared to patients with normocalcemia.

We examined a 5-year cohort of incident hemodialysis patients receiving care from a large U.S. dialysis organization (Jan 2007–Dec 2011), and identified 869 patients in whom iCa was measured during the first 91 days of dialysis. Associations of iCa with mortality were examined using Cox regression models with adjustment for case-mix and laboratory covariates.

Patients were 63±15 years; 43% female, 35% Black, and 56% diabetic. Hypercalcemia (iCa >1.32 mmol/L) was observed in 74 patients (8.5%), and was associated with higher mortality after adjustment for demographics and comorbidities. Among them, only 9 patients (12%) had tCa_{ALB} >10.2 mg/dL. Hidden hypercalcemia in the remaining 65 patients was independently associated with higher mortality, the effect size of which was not significantly different from apparent hypercalcemia (p=0.72, Fig). Further adjustment for laboratory variables did not change the results.



In conclusion, tCa_{ALB} is in the normal range in some hemodialysis patients with high iCa, and the death risk with hidden hypercalcemia is the same as those with apparent hypercalcemia.