173

AN "ATYPICAL" CASE OF EXIT SITE INFECTION IN PERITONEAL DIALYSIS

Abhilash Koratala, Volodymyr Chornyy, Amir Kazory
Division of Nephrology, Hypertension and Renal Transplantation, University of Florida, Gainesville, Florida, USA.

Exit site infection is a well-known complication among patients receiving peritoneal dialysis (PD). Common responsible organisms include *staphylococcus aureus* and *staphylococcus epidermidis*. Therefore, current guidelines recommend initial empiric antibiotics that are aimed at these organisms. However, infections secondary to uncommon organisms such as atypical mycobacteria and non-diphtheria corynebacteria are being increasingly reported with the widespread use of antimicrobial prophylaxis at the exit site.

A 59-year-old woman with a history of diabetes mellitus and end-stage renal disease treated with PD presented with fever, chills, redness and swelling at her PD catheter exit site for the previous 2 days. She had a failed renal transplant 4 months before and was receiving azathioprine 75 mg/day and prednisone 10 mg/day. Examination revealed erythema and edema at the exit site with serosanguinous discharge. PD fluid cell count was not suggestive of PD-associated peritonitis and Gram stain from the exit site was negative. The diagnosis of exit site infection was made and intravenous antibiotic therapy with vancomycin and cefepime was started, which was followed by rapid clinical improvement. She was discharged home on oral amoxicillin-clavulanic acid. Interestingly, 10 days later, all cultures obtained from the exit site at different time points revealed acid fast bacilli identified as *Mycobacterium fortuitum*. The decision was made to use oral clarithromycin and ciprofloxacin for 2 months. The patient remained asymptomatic with normal exit site on examination. Of note, the patient reported using well water for bathing which could be the potential source for atypical mycobacteria; the culture of the water is pending.

There are two key educational points in this case. First, immunosuppressive agents, even at very low dose, could still increase a patient's susceptibility to uncommon and atypical infections. Second, clinical improvement after initial empiric antibiotic therapy in these patients could possibly be due to partial sensitivity of the organisms, and it is of utmost importance to continue monitoring their culture results despite initial positive response to therapy.

174

HYPERKALEMIA IN PATIENTS WITH CHRONIC HEART FAILURE TREATED WITH RAAS INHIBITION

Abhilash Koratala, Amir Kazory
Division of Nephrology, Hypertension, and Renal Transplantation, University of Florida, Gainesville, Florida, USA

Background: Inhibition of the renin-angiotensin-aldosterone system (RAAS) is a key strategy in treating heart failure (HF). RAAS inhibitors (angiotensin-converting enzyme inhibitors [ACE-I], angiotensin receptor blockers [ARB], aldosterone receptor antagonists [ARA], and direct renin inhibitors [DRI]) are known to increase the risk of hyperkalemia in HF, hence potentially limiting their widespread use. However, the clinical relevance and practical implications of this increased risk remain underexplored.

Methods: Articles cited in PubMed database from 1986 to 2016 using key words: "heart failure", "hyperkalemia", and "renin-angiotensin-aldosterone system" were searched. Those randomized controlled trials on humans that included data on the incidence of hyperkalemia in HF were identified and relevant articles in English language were selected. Articles related to ARA or DRI were excluded. The results of the remaining studies were then reviewed and compared with regards to the risk of hyperkalemia associated with RAAS inhibition.

Results: A total of 113 relevant articles were identified on the role of RAAS inhibition in human HF. Twelve were selected to be included in this study (7 with ACE-I and 5 with ARB) with a total of 45073 patients. Baseline serum creatinine levels were 1.0 to 1.2 mg/dl. The incidence of K > 5.5 mmol/l was 0.9 to 6.4% in the active arm. Four studies reported the incidence of serum K ≥ 6.0 mmol/l, which ranged from 2 to 3%. The RAAS inhibitor discontinuation rate due to hyperkalemia was reported to be as low as 0.1% to 3.4%. There was no strong association reported between hyperkalemia and worse outcomes.

Conclusion: Although RAAS inhibition is associated with an established risk of hyperkalemia in patients with HF, the incidence of clinically significant hyperkalemia (i.e. serum potassium ≥ 6.0 mmol/L) is low in those with normal or near-normal renal function. Moreover, based on the findings of our study, there is little evidence to suggest that the observed increases in serum potassium are associated with adverse clinical outcomes.

175

SEVERE ACUTE KIDNEY INJURY FROM TURP-ASSOCIATED HEME PIGMENT NEPHROPATHY

Anubhav Kumar, Behdad Besharatian, Sidney Kobrin, Matthew Palmer, Jonathan Hogan, University of Pennsylvania, Philadelphia, Pennsylvania, United States of America.

An 82 year-old man with a past medical history of metastatic prostate cancer, urethral strictures, and stage III CKD underwent a trans-urethral resection of the prostate (TURP). Twelve days post-procedure he presented with weakness, anorexia, fatigue, and tremors. He was found to have severe acute kidney injury with creatinine of 31 mg/dL (previous baseline was 2.1 mg/dL), hyperkalemia of 8.8 mmol/L, and new-onset anemia of 11.6 g/dL (previous baseline of 13.8 g/dL). Haptoglobin was undetectably low and creatine kinase was elevated to 959 U/L. LDH was 3019 U/L.

Emergent hemodialysis was initiated. Endoscopy was negative for a source of bleeding. Renal ultrasound was negative for obstruction but showed normal-sized kidneys with increased echogenicity. A kidney biopsy was performed and showed pigmented cast nephropathy, with stains identifying the pigment as hemoglobin. Three months later the patient remains dialysis-dependent.

TURP syndrome is most commonly associated with hypoosmolality due to the use of hypotonic irrigants that can lead to neurological manifestations. As this case demonstrates, heme pigment nephropathy with severe AKI is a rare complication after TURP.

176

POST-ANGIOPLASTY CHANGES IN VENOUS ACCESS PRESSURE RATIO IS A NOVEL DIAGNOSTIC TOOL PREDICTING ACCESS FAILURE.

Lalathaksha Kumbar, Gerard Zasuwa, Mathew Zaborowicz, Andrew Taylor, Jerry Yee. Henry Ford Hospital, Detroit, MI.

Vascular access pressure ratio (VAPR) test identifies dialysis vascular access (VA) dysfunction in patients when 3 consecutive VAPRs are >0.55. We tested if one week mean post-interventional VAPR decline from mean alert diagnose failing access.

Retrospective analysis of all VA procedures from 03/2014 to 06/2016. Data included demographics, comorbidities, VA features, $\%\Delta VAPR = \frac{Pre-Post}{Pre} \times 100\%$, time-to-next procedure, and patency. Area under curve, receiver operating curve, Kaplan-Meier (KM) arteriovenous graft (AVG) and fistula (AVF) survival curves were compared by the log-rank test. A multivariable Cox-proportional hazard (CP) model was used to determine the association of %Δ VAPR with access survival

Analysis of 150 subjects [females 50%; black 85%] included 69 AVF with 114 angioplasties and 81 AVG with 142 angioplasties. Area under the ROC curve (AUC) for fistula failure at 1 year was 0.701, with an optimal cut-off value of 54.7%, sensitivity of 74.3%, and specificity of 40%. AVF with <54.7% decline compared to >54.7% required earlier subsequent procedure (132 vs 266 d), lower survival on KM analysis (p=0.018) and 2-fold greater risk of failure (p=.006). AUC for AVG failure at 2 weeks was 0.612 with cut-off value of 29.6%, sensitivity of 72.4%, and specificity of 42.6%. AVG with post-intervention VAPR decline <29.6% also required earlier subsequent procedure (129 vs 189 d), lower survival on KM (p=0.015) and 72% higher risk for failure. Cut points were significant predictors of overall time to failure in both grafts and fistulas.

Post-intervention reduction of VAPR provides a novel diagnostic tool to predict access failure.

Downloaded for Anonymous User (n/a) at Henry Ford Hospital / Henry Ford Health System (CS North America) from ClinicalKey.com by Elsevier on June 16, 2022. For personal use only. No other uses without permission. Copyright ©2022. Elsevier Inc. All rights reserved.