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Case Report

Case Report of an Atypical Evolution of Urine Obstruction, is this Acute on Chronic Kidney Disease?

De la Vega Méndez FM[1], Alvarado Villarreal FJ[1], Purón-González E[1], Martínez Jímenez JG[1,2,3], and Rizo Topete L[1,2,3]*

[1]Fellow of the Internal Medicine Department, Hospital Christus Muguerza Alta Especialidad, UDEM, Monterrey, Nuevo León, México

[2]Internal Medicine and Nephrology Profesor at Hospital ChristusMuguerza Alta Especialidad, UDEM, Monterrey, Nuevo León, México

[3]Hospital Universitario "José Eleuterio González", UANL, Monterrey, Nuevo León, México

Abstract

Acute kidney injury (AKI), previously known as acute kidney failure, is defined as an abrupt (within hours) reduction of renal function in which both structural damage and loss of function are encompassed. Classification of AKI includes pre-renal, intrinsic kidney diseases (renal) and post-renal [1]. The term 'acute-on-chronic renal failure' has been used when AKI occurs in the background of pre-existing chronic kidney disease [2].

Post-renal AKI occurs after acute obstruction of the urinary flow, which increases intra-tubular pressure and thus decreases glomerular filtration rate. In addition, this acute obstruction generates an inflammatory process that contributes to the reduction of glomerular filtration. It can occur after an obstruction at any level of the urinary system (from the renal tubule to the urethra). The obstruction of the urinary tract is a common problem but a rare cause of AKI, currently there is no epidemiological data on its incidence, however it is more common in men and increases with age mainly due to prostate disease, pelvic cancer and urolithiasis [3,4]. The timing of the reversal of the obstructive cause is crucial because if not done in time can lead to permanent kidney damage. It is clear that complete or prolonged partial urinary tract obstruction can lead to tubular atrophy, interstitial fibrosis and, eventually irreversible renal injury [5]. Also is important to notice that obstruction of urinary tract may occur in the setting of preexisting Chronic Kidney Disease (CKD).

We present an atypical case (time of evolution) of a urinary obstruction with extremely high levels of plasmatic creatinine.

***Corresponding author:** Rizo Topete L, Fellow of the Internal Medicine Department, Hospital Christus Muguerza Alta Especialidad, UDEM, Monterrey, Nuevo León, México, Tel: +52 1 81 8029 7718; E-mail: marili18@hotmail.com

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Case Report

A 58-year-old man came to the emergency department with oral intolerance. He has a medical history of a perianal abscess, treated and solved. Patient refers to have presented 2 weeks before arriving to our emergency department with nausea, postprandial fullness, weight loss of eight pounds and progressive asthenia and malaise which interfered with his daily activities. The patient goes to consult where he is prescribed omeprazole for 3 weeks without improvement. Patient presented sudden vomiting and oral intolerance and arrives to our emergency department. With intentional interrogation patient refers frequency of urination, interruption of urine stream and weakness of urine stream. On physical examination: hypertensive with 177/100 mmHg, uremic breath, with presence of asterixis, oriented in time, place and person with attention preserved, without muscle weakness or sensory disturbances. Globose abdomen dull to percussion in hypogastrium, palpable mass of 15 x 13 cm in mesogastrium and hypogastrium that was mobile and not painful to palpation. Laboratories: Hemoglobin: 7.6 mg/dl, Creatinine 36.87 mg/dl, Blood urine nitrogen 211.7 mg/dl, Uric acid: 12.3 mg/dl, Albumin 4.6 g/dl Sodium: 140 mEq/L, Potassium: 6.86 mEq/L Chloride: 95.7 mEq/L. Calcium 8.6 mg/dl, Phosphorus 8.3 mg/dl. Venous Gasometry: metabolic acidosis with pH: 7.28 and HCO3: 11.6 mEq/L, Lactate 0.5mEq/L. Urine analysis: with hemoglobin +++ and Erythrocytes 30-40 x field. An EKG was obtained in which the only relevant observed was a sinus tachycardia and QT interval prolongation (Figure 1). CT (computerized tomography) of abdomen without contrast: Hydroureteronephrosis grade IV (Figure 2). Bladder with an estimated volume of 2 liters. Increased prostate of size 6. 5 x 5. 6 x 6 cm with an estimated volume of 115 cc that causes compression of the bladder floor.

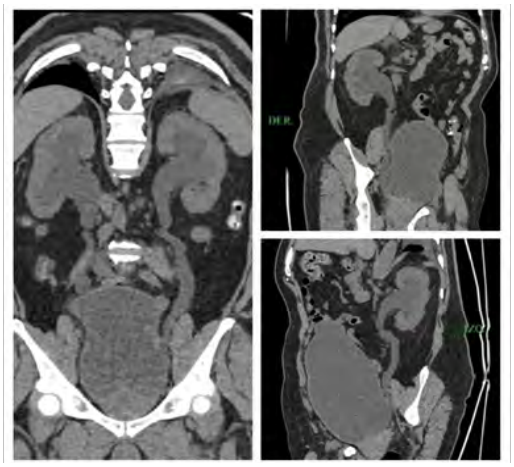

Figure 1: Simple CT with grade IV hydronephrosis showing right kidney with measurements of 13.4 x 7.8 x 6.4 cm with increased dimensions and cortical thinning caused by the presence of hydronephrosis. Left kidney with measures of 12.3 x 7.1 x 5 cm with increased dimensions and cortical thinning caused by the presence of hydronephrosis.

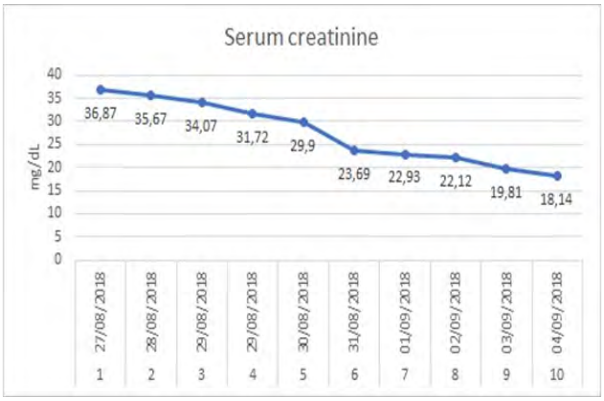

Figure 2: Evolution of creatinine levels during the days of hospitalization.

The patient's obstruction was resolved on the first day of hospitalization with a urinary catheter but electrolyte abnormalities (hyperkalemia) persisted and hemodialysis was started.

Discussion

The clinical presentation of AKI due to a urinary tract obstruction varies according to the site of the obstruction, the degree of the obstruction (partial or complete) and the speed at which it occurs. It usually manifests with pain, changes in the urinary pattern, hypertension, hematuria and elevation of serum creatinine [6]. When pain is present, it is secondary to bladder distention, infection or lithiasis; hydronephrosis by itself is asymptomatic [7]. In our patient due to the large mass that was palpated in mesogastrium, it was decided to perform a CT as the first option because of the possibility of ruling out other causes of urinary retention besides increased prostate size. Although our patient had alterations in urinary volume this was not perceived by our patient as something annoying and only after an intentional interrogation is that he referred urinary symptoms. Drawing to our attention the significant elevation of the serum creatinine that normally occurs in bilateral obstruction which is the first indicator of obstruction. Obstruction of the upper and lower urinary tracts may occur in the setting of preexisting CKD whether CKD is due to an obstructive or another cause, and should be considered in all patients presenting with acute-on-chronic [8]. This may be the reason why our patient seemed to have good clinical tolerance to the considerably high levels of urea.

There are no absolute criteria for when to initiate dialysis in these patients, but it is recommended to initiate before complications occur [6,9]. It is important to remember that the serum creatinine level does not correlate with Glomerular Filtration Rate (GFR) in a patient with AKI, so the clinical manifestations will govern the decision of renal replacement therapy. It is for this reason that in the beginning this treatment was not initiated in our patient, in spite of having a potassium greater than 6.5 mEq/L and urea greater than 30mEq/L because it did not cause significant electrocardiographic changes and was not accompanied with alterations in the mental status or symptoms of severe uremic encephalopathy however despite bladder catheterization, the hyperkalemia persisted and renal replacement therapy was decided to start.

Conclusion

Although postrenal acute kidney injury is not a frequent cause of AKI could be a complication in a chronic obstruction and could drive to chronic kidney disease. The clinical presentation of an urinary obstruction should be recognized in a timely manner because it requires relatively simple treatments that can promptly restore renal function, as in this case where hydronephrosis get better but because of the length of time the diagnosis and probably the unknown chronic damage the function do not recover and the patient is still at chronic renal replacement therapy. Most of the times function recovery in AKI occurs in the first 7 to 10 days after treatment of the cause of the obstruction. However, some patients with severe renal failure may require dialysis even when the obstruction is resolved and generally creatinine levels stabilize at a level greater than 3mg/dL in those patients [9], the evolution of serum creatinine in our patient is showing in (Figure 2). Hence, it is extremely important not to forget that acute post renal kidney injury even in a setting of acute on chronic kidney disease can have severe consequences on residual renal function and urgent intervention is critical to limit renal damage and preserve residual renal function.

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