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Non-dilated obstructive uropathy

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SUMMARY

Recent Urinary tract obstruction leading to acute kidney injury (AKI) is commonly associated with hydro ureter and hydro nephrosis, often accompanied by oliguria. AKI and the syndrome of Non-Dilated Obstructive Uropathy (NDOU) is well reported, however, it is not common, accounting for less than 4% of obstructive uropathy cases. Our recent experience with two cases of NDOU seen within the span of six months may imply otherwise. We present two cases of obstructive uropathy without sonographic features of obstructive uropathy with acute renal failure. Two elderly male patients presented at Royal Bahrain Hospital Nephrology Centre with an acute rise in serum creatinine, without active urinary sediment or proteinuria. Imaging studies showed normal size kidneys without hydronephrotic changes. Initially, they were diagnosed with pre-renal azotemia secondary to volume depletion, and they were treated with intravenous fluids which failed to improve the kidney function. Repeated imaging showed bladder fullness with prostate hypertrophy, therefore NDOU was diagnosed. Both patients' urinary obstruction was relieved resulting in recovery of the kidney injury.

Keywords: Microbiome; Endometrium; *Lactobacilli*; Gynecologic conditions.

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INTRODUCTION

Acute kidney injury secondary to obstruction is typically characterized by bilateral hydronephrosis [1]. However, under certain clinical conditions, renal imaging may still fail to demonstrate hydronephrosis [2-6]. This medical presentation can be attributed to a variety of clinic-pathologic conditions including volume depletion, severe oliguria, acute early obstruction, and inability of the collecting system to dilate because of infiltrative abdominal-pelvic disease such as metastatic cancer or retroperitoneal fibrosis [4-9]. Our recent experience with two cases of NDOU at Royal Bahrain Hospital within a six month-period prompted this report.

CASE PRESENTATION

Case 1

A 63-year-old male patient with longstanding history of hypertension, well controlled with Angiotensin receptor blockers (ARBs), and a recent serum creatinine of 90 µmol/L presented to our clinic with worsening anorexia, nausea, vomiting, and oliguria. One week ago, he was diagnosed and treated for urinary tract infection (UTI). Blood pressure was 155/89 mmHg, and he was not orthostatic. Serum creatinine was 390 µmol/L and potassium 5.5 mmol/L. Urinalysis showed 5– 10 WBC PHF and 2–5 RBC PHF without proteinuria. Alanine transaminase (ALT), aspartate transaminase (AST), and total creatine kinase (CK) levels were normal. Renal ultrasound and doppler showed normal appearing kidneys with post-void urinary bladder scan showing 50 mL of residual urine and a large prostate. The next day, he had increased vomiting, oliguria, with serum creatinine trending upto 455 µmol/L, with potassium elevated at 5.7 mmol/L. A non-contrast computerized tomography (CT) examination was normal except for fullness in the urinary bladder. Foley catheter was inserted with an immediate output of 400mL. The patient was diagnosed with acute kidney injury secondary to obstructive uropathy, and urology team was consulted. The patient's symptoms stabilized after a few days with recovery of his kidney function. He was treated conservatively with alpha blockers and eventually underwent transurethral resection of the prostate. After 8 weeks, his serum creatinine trended down to 109 µmol/L. The pathology report revealed low grade prostate cancer.

Case 2

A 67-year-old man with a history of coronary artery stenting, peripheral arterial disease (PAD), hypertension, type II diabetes mellitus, benign prostatic hyperplasia and chronic kidney disease (baseline serum creatinine of 135 μmol/L) presented to our clinic complaining of headache, malaise, fatigue, urinary frequency and decreasing urine output. He was vitally stable and afebrile. His blood work showed an increase in serum creatinine of 330 μmol/L, hyperkalemia (3.6 mmol/l) and hyperphosphatemia (5.1 mg/dl.). Renal sonogram revealed normal size kidneys without hydronephrotic changes. NDOU was suspected following a nephrology consultation ruling out renal disease. As a result, he underwent a post-void bladder ultrasound which confirmed urinary retention. Foley catheter placement quickly drained 350 cc of amber-colored clear urine. Hyperkalemia, and hyperphosphatemia, together with serum creatinine, improved and serum creatinine decreased to 150 μmol/L four days later. The Foley catheter was removed against medical advice. The next two day, his serum creatinine increased to 170 μmol/L and 205μmol/L respectively – a very clear affirmation of the presence of NDOU, most likely from an enlarged prostate. Because medical treatment failed to control his obstructive uropathy he underwent TURP, and his kidney function recovered to baseline.

DISCUSSION

Acute kidney injury is characterized by abrupt deterioration in renal function, manifested by a rise in serum creatinine level with or without decrease in urine output. The spectrum of renal injury ranges from mild to severe, sometimes requiring renal replacement therapy. The diagnostic evaluation can be used to classify acute kidney injury as pre-renal, renal, or post-renal [10,11]. The initial workup includes the patient history to identify the use of nephrotoxic agents or the presence of systemic disease that might cause poor renal perfusion or directly impair renal function. Physical examination should assess intravascular volume status and identify signs of systemic illness. The initial laboratory evaluation should include measurement of complete blood count, serum creatinine level, and urinalysis. Ultrasonography of the kidneys should be performed, particularly in older men, to rule out obstruction.

Most physicians don't suspect post renal AKI unless hydronephrotic changes are present. However, post renal AKI can present without hydronephrotic changes, as noted in the early stage of obstruction, or with retroperitoneal fibrosis [11,12]

CONCLUSION

The syndrome of NDOU and AKI has been frequently reported but not well studied. The literature suggests that it is a rare syndrome. In this report, we describe our recent experience with two cases of NDOU seen within the span of 6 months. Despite the absence of dilatation on renal imaging, strong suspicion for NDOU led to decompression procedures with prompt recovery of kidney function in both patients. We believe that NDOU may be more common than previously speculated, and a high index of suspicion, coupled with a high level of collaboration between Nephrology, Urology, and Radiology, can lead to a higher rate of detection of such cases. Furthermore, we noted the relative ease of reversing uremia by early decompression therapy, and the prevention of potentially irreversible end-stage renal disease, if left untreated.

CONFLICTS OF INTEREST

The authors declare no competing interests.

All authors declare that the material has not been published elsewhere, or has not been submitted to another publisher.

DATA AVAILABILITY

Authors declare that all related data are available concerning researchers by the corresponding author's email.

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