A Case of Aneurysm Induced Obstructive Nephropathy Masked by Polyuria

D Kujubu, J Agarwal, N Gandhi, J Sim, M Idroos, S Rasgon

Citation

D Kujubu, J Agarwal, N Gandhi, J Sim, M Idroos, S Rasgon. *A Case of Aneurysm Induced Obstructive Nephropathy Masked by Polyuria*. The Internet Journal of Nephrology. 2005 Volume 3 Number 1.

Abstract

Arterial aneurysms within the abdominal cavity have been shown to directly cause urinary tract obstruction.1, 2, 3, 4 The majority of these cases involve inflammatory aortic aneurysms associated with perianeurysmal fibrosis that ultimately result in structural compromise of the urinary tract. Indeed ureteral obstruction has been reported to occur in up to 30% of all inflammatory aneursyms.5, 6 Inflammatory iliac artery aneurysms are less common but can occur in association with aortic aneurysms. Even more rarely, obstructive nephropathy secondary to iliac artery aneurysms have been reported to occur.7,8,9,10,11,12 We describe a case of a patient admitted for non-cardiac chest pain and found to be in non-oligoanuric acute renal failure. A renal ultrasound revealed aortic and bilateral iliac aneurysms with minimal evidence of hydronephrosis on ultrasound imaging. Clinically, the patient had a normal to high urine output requiring continuous volume repletion. Urine studies repeatedly showed low osmolarity without other derangements. Only after progressive deterioration of renal function did obstruction surface as a plausible etiology. Bilateral ureteral stent placement and subsequent resolution of renal failure confirmed the diagnosis of obstructive nephropathy. This presentation illustrates a case of bilateral ureteral obstruction and obstructive nephropathy that was masked by high urinary output secondary to water diuresis likely due to nephrogenic diabetes insipidus (DI). Because of the obstruction, the patient did not present with extremely high urinary output and volume depletion. The DI in turn masked the clinical signs of obstruction, specifically oligoanuria.

CASE REPORT

A 53-year-old male presented to our medical center for substernal chest pain. The patient had a past medical history significant for coronary artery disease, hypertension, and tobacco use. He had no prior history of renal disease or use of nephrotroxic medications. His home medications included: aspirin, lisinopril, metoprolol, famotidine, and nitroglycerin. On review of systems, the patient denied gross hematuria, dysuria, polyuria, hesitancy, nocturia, flank or abdominal pain, edema, orthopnea, or dyspnea upon exertion. Physical exam was remarkable for blood pressure of 160/80 mmHg, clear lung fields, non-tender abdomen with no masses or bruits, and no evidence of peripheral edema. Acute coronary syndrome was ruled out and did not receive additional cardiac procedures.

Admission laboratories revealed that the patient had acute renal failure with an elevated creatinine of 2.4 mg/dL (baseline 1.3 mg/dL). The remainder of the laboratory studies were unremarkable: potassium 4.2 meq/L, sodium 142 meq/L, chloride 107 meq/L, CO_2 28 meq/L, and hemoglobin of 11.0 g/dL Urinanalysis indicated a specific gravity of 1.000 with no protein, red or white blood cells per high power field. Urine electrolytes revealed a urine sodium of 26 meq/L. Urine output in the first 24 hours of admission was 4,050 ml with a total of 3,400 ml in oral and intravenous intake. Creatinine phosphokinase levels, urine culture, and 24 hour protein quantification were unremarkable. A renal ultrasound demonstrated a right kidney length of 6.8 cm with no hydronephrosis and a left kidney measuring 13.5 cm with mild dilatation of the intrarenal collecting system. Incidentally, an aortic aneurysm extending down to the iliac bifurcation was noted on the study.

A non-contrast computed tomography (CT) of the abdomen and pelvis was performed given the above findings. The CT revealed an infrarenal aortic aneurysm measuring 4.5 cm in diameter, a right common iliac aneurysm measuring 4.9 cm in diameter, and a left common iliac aneurysm measuring 5.1 cm in diameter (Fig 1). There was mild renal pelvic dilatation/hydronephrosis on the left kidney and a small right kidney with no hydronephrosis. No specific visualization of ureteral compression or compromise was noted. Clinically the patient continued to have urine outputs ranging from 3-4

liters daily. A urological consultation was obtained to evaluate for possible obstruction with resultant obstructive nephropathy. At the time, the mild hydronephrosis was felt to be non significant given the normal to high urine output and thus no intervention was performed. The patient's renal function, however, continued to deteriorate and his creatinine eventually peaked to a level of 4.8 mg/dL. Urine specific gravity continued to range from1.000 to 1.005 suggestive of a water diuresis.

A repeat renal ultrasound was performed but showed no changes in terms of obstruction or external compression. Given the lack of another etiology for the patient's acute renal failure, a therapeutic trial of bilateral ureteral stents was performed despite normal to high urine output. After stent placement, urine output increased to 8-12 liters per day. Workup of the polyuria at that time revealed urine osm 202, serum osm 287, urine Na 90 mEq/L, urine K 4 mEq/L, and urine Cl 88 mEq/L. Isotonic crystalloids were administered at a rate to keep up with the urine output. The polyuria continued for another 11 days post stent placement until it normalized to 2-3 liters per day where the patient was able to orally replenish his fluids. Renal function concurrently improved to a serum creatinine of 1.6 mg/dL.

The patient was eventually taken for surgical repair of the aneurysms. Intraoperative observations revealed the aortic and iliac aneurysms as seen on imaging (fig 1 & 2). The left common iliac aneurysm was inflammatory in nature with perianeurysmal fibrosis. An infrarenal external iliac bypass was performed without complication. Pathology demonstrated an atherosclerotic iliac aneurysm with chronic and follicular inflammation and fibrosis. The patient's serum creatinine one month after discharge was 1.4 mg/dL. Urine output decreased to 3.0 liters per day. A follow up CT continued to show mild hydronephrosis of the left kidney.

DISCUSSION

Inflammatory aneurysms are known to cause obstructive nephropathy by encasing the ureter in perianeurysmal fibrosis. The etiology of the fibrosis, however, is unclear. Early reports suggested that the inflammation may be secondary to small leaks from the anuersym.[13]Studies suggest that the inflammation may represent an auto-immune reaction to atherosclerotic plaquing.[5] Regardless of the etiology, multiple case reports have demonstrated that adhesions to the ureter can occur. Treatment consists of relieving the clinically significant obstruction with ureteral stents or nephrostomy tubes followed by surgical repair of the aneurysm. The need for ureterolysis from adhesions had been previously advocated but recent studies suggest that it is not necessary.[14]

This case illustrates an obstructive nephropathy secondary to an iliac aneurysm that had a very unusual presentation. Our patient did not have oligoanuria but rather had normal to high urine outputs. High urine output has been rarely reported in cases of obstructive nephropathy.[15,16,17,18] Nephrogenic diabetes insipidus (DI) surfaced as the mechanism for the polyuria in these past studies as our case suggests. The pathogenesis of nephrogenic DI from obstruction is not well understood but some suggest that renal tubular resistance to arginine-vasopressin may be the underlying mechanism.[17]Our case suggested a nephrogenic DI based on the persistent high urine output with low urine osmolarity, low urine sodium and low specific gravity. Serum sodium was not elevated but at the same time; the patient was aggressively hydrated with isotonic crystalloids to maintain his volume status to account for the large urine losses. A polyuria workup post stent placement continued to suggest DI and a superimposed solute diuresis. After stent placement and aggressive hydration, urine sodium became elevated to 97 meq/L from pre-stent levels of 26 meq/L. Fluid balance remained even throughout.

A water depravation test was not performed because of the risks of volume depletion and electrolyte derangements in the context of a recent stent procedure. Additionally, the patient was not given an empiric trial of DDAVP because of his presentation with chest pain and his underlying coronary artery disease. Fortunately, the patient's polyuria resolved on its own along with the hypo-osmolar urine. The presentation and clinical course was most consistent with a self limited nephrogenic DI. The aspect of this case that made the diagnosis of obstructive nephropathy difficult was the presence of only minimal hydronephrosis in the setting of significant renal failure and high urine output. Incidentally, repeat ultrasound after stent placement continued to show hypdronephrosis. Significant obstruction causing acute renal failure would be expected to present with greater degrees of hydronephrosis initially and resolve after a therapeutic intervention.

We are reminded by this case that inflammatory aneurysms, both aortic and iliac, may lead to significant obstructive nephropathy. The evaluation of these patients may reveal polyuria and only minimal hydronephrosis. Obstructive nephropathy should be included in the differential diagnosis

even in cases of high urine output acute renal failure.

References

1. Rault R, Kapoor W, Kam W. Perianeurysmal fibrosis and ureteric obstruction: case report and review of literature. Clinical Nephrology 1982; 18: 159-62
2. Brainbridge ET, Woodward DA. Inflammatory aneurysms of the abdominal aorta with associated ureteric obstructionor medial deviation. J Cardiovas Surg 1982; 23: 365-70
3. Radomski SB, Ameli MF, Jewett MA. Inflammatory abdominal aortic aneurysms and ureteric obstruction. CJS 1990; 33: 49-52
4. Korzets Z, Witz M, Glodberg E, Rozin M, Lehmann J, Bernheim J. The patient with asymptomatic advanced renal failure-obstructive uropathy caused by inflammatory abdominal aortic aneurysm. Nephrol Dial Transplant 1998; 13: 1835-7
5. Pennell RC, Hollier LH, Lie JT. Inflammatory abdominal aortic aneurysms a thirty year review. J Vasc Surg 1985; 2: 859-869
6. Lindblad B, Almgren B, Bergqvist. Abdomnial aortic aneurysm with perianeursymal fibrosis: experience from 11 Swedish vascular centers. J Vasc Surg 1991; 13: 231-9
7. Redman JF, Campbell GS. Ureteral obstruction secondary to iliac artery aneurysm. Urology 1975; 6: 212-4
8. Peterson LJ, McAnnich JW, Weinerth JL. Ureteral obstruction of solitary kidneys by iliac artery aneurysms. Urology 1977; 9: 17-21
9. Kaynan A, Rosenberg V, Szuchmacher P. Ureteral obstruction secondary to iliac artery aneurysms. The Mount Sinai Journal of Medicine 1978; 45: 334-41
10. Villani U, Leoni S, Mora A. Unilateral hydroureteronephrosis secondary to iliac aneurysm. Urology 1985; 26: 62-3
11. Minato N, Itoh T, Natsuaki M, Yamamoto H et al. Surgical treatment of isolated iliac artery aneurysm with ureteral obstruction and/or renal failure. J Cardiovasc Surg 1990; 31: 189-93
12. Obrand DJ, Mijangos J, Brassard R, BrockG, Steinmertz OK. Ureteropelvic urinary extravasation due to iliac artery aneurysm. Ann Vasc Surg 1998; 12: 468-70
13. Walker DI, Bloor K, Williams G, Gillie I. Inflammatory aneurysms of the abdominal aorta. Br J Surg 1972; 59: 609-14
14. Speziale F, Sbarigia E, Fiorani P et al. Inflammatory aneurysms of the abdominal aorta involving the ureters: is combined treatment really necessary? J Urol 2001; 165: 27-31
15. Nagar D, Ferris FZ, Schacht RA. Obstructive polyuric renal failure following renal transplantation. American Journal of Medicine 1976; 60: 702-6
16. Kato A, Hishida A, Kaneko E et al. Nephrogenic diabetes insipidus associated with bilateral ureteral obstruction. Intern Med. 1994;33(4):231-3
17. Hong EG, Suh Y, Park RW et al. A csae of nephrogenic diabetes insipidus caused by obstructive uropathy due to prostate cancer. Yonsei Medical Journal 2000; 41: 150-4
18. Yoshioka K, Imanishi M, Fujii S et al. Nephrogenic diabetes insipidus due to hydronephrosis in a patient with solitary kidney. Clinical and Experimental Nephrology 2003; 7: 243-6

Author Information

Dean Kujubu
Department of Nephrology and Hypertension, Kaiser-Permanente Los Angeles Medical Center

Jay Agarwal
Department of Nephrology and Hypertension, Kaiser-Permanente Los Angeles Medical Center

Nirav Gandhi
Department of Nephrology and Hypertension, Kaiser-Permanente Los Angeles Medical Center

John Sim
Department of Nephrology and Hypertension, Kaiser-Permanente Los Angeles Medical Center

Mohammed Idroos
Department of Nephrology and Hypertension, Kaiser-Permanente Los Angeles Medical Center

Scott Rasgon
Department of Nephrology and Hypertension, Kaiser-Permanente Los Angeles Medical Center