

Case – Reflex anuria: A rare complication of retrograde pyelography

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Case report

A 65-year-old male presented to emergency room (ER) with no urine output for more than 24 hours after undergoing cystoscopy, ureteroscopy with bilateral retrograde pyelography. His past medical history was significant for morbid obesity with a body mass index of 43kg/m², hypertension, coronary artery disease with percutaneous coronary interventions in 2003, 2004 and 2009; nonalcoholic steatohepatitis with hepatocellular carcinoma treated with transarterial chemoembolization two years back and IgA nephropathy with stage III chronic kidney disease (CKD).

Three months prior to this admission, he was diagnosed with portal vein thrombosis on routine follow up and was started on rivaroxaban. Three weeks after starting rivaroxaban, he developed gross hematuria requiring cessation of anticoagulation. He had two more episodes of gross haematuria in the following 2 months. Non-enhanced computerized tomography (CT) imaging of the abdomen and cystoscopies performed 10 weeks and again 2 weeks prior to admission failed to determine an etiology of this hematuria.

With background of stage III CKD, contrast enhanced CT scan to image the upper urinary tract was determined to be high risk. He therefore underwent ureteroscopy and bilateral retrograde pyelography (RP) using iohexol as a contrast medium and again no source of hematuria including urolithiasis or tumour was identified. He was hemodynamically stable throughout the procedure.

Twenty-four hours after the procedure he presented to ER with anuria and lower abdominal pain. On examination, his blood pressure was 134/79 mmHg, pulse was 66 beats per minute, and oxygen saturation was 96% on room air. He had mild pedal edema without evidence of pulmonary edema. Bedside bladder scan demonstrated an empty bladder, and there was no urine output after insertion of a foley catheter. He was volume resuscitated with normal saline and given a large dose of furosemide, but the patient remained anuric.

The patient's creatinine was 515umol/L (181umol/L 15 days prior). His renal ultrasound was unremarkable with no hydronephrosis (right kidney 12.1 cm and left kidney

11.6 cm). His renal artery and vein bilaterally appeared patent on doppler study. An abdominal CT scan post volume repletion showed no evidence of urinary tract obstruction. His vasculitis work-up in the form of antineutrophil cytoplasmic antibody and anti-glomerular basement membrane antibody came back negative; complements levels were normal. He remained anuric and became hyperkalemic which was resistant to medical treatment. He underwent a single short run of hemodialysis 48 hours after admission. His clinical course and laboratory trends are depicted in Table 1.

Shortly after dialysis, the patient started making urine. Urine volume doubled to 1600 ml the next day and to 3100 ml the day after. His creatinine plateaued for 2 days post dialysis and then began to trend downwards. His urine sent for analysis was positive for blood and 1+ protein with an unremarkable microscopy. Urine albumin to creatinine ratio was 43.9 mg/mmol 3 days after dialysis, which was similar to his baseline; urine specific gravity was 1.010 and fractional excretion of sodium (FeNa) was less than 1%. He was discharged 1 week after admission with recovering kidney function and final diagnosis of reflex anuria. He was seen in outpatient clinic 1 week after discharge and his creatinine was back to baseline.

Discussion

Discussion of the differential diagnosis surrounding anuric acute kidney injury (AKI) often fail to include 'reflex anuria' (RA) as a possible etiology, even when involving nephrologists and urologists. The literature around it is sparse with PubMed search showing 60 results, not all results are actually pertaining to RA. One of the earliest mentions about this entity was by Morton HM in 1923 where he described temporary suppression of urine following double pyelography.¹ Since then it has been described with various procedures including prophylactic ureteral catheterization during colorectal²⁻³ or gynaecological⁴ surgeries, retrograde pyelography,⁵ unilateral urolithiasis with normal contralateral kidney⁶ and embolization of a unilateral kidney tumour.⁷

The current and widely accepted definition of RA as proposed by Hull⁸ is "cessation of urine output from both kidneys in response to irritation or trauma to one kidney or its ureter, or severely painful stimuli to other organs." Mechanical obstruction of the ureters, acute intrinsic kidney diseases and hypotension as causes of anuria were not included in this definition.

The pathogenesis of RA remains unclear. In all reported cases of RA, the common link is an irritation, instrumentation, or other painful stimulus to a ureter or kidney. There are two proposed mechanisms.⁸ The first is a diffuse spasm of the intrarenal arterioles sufficient to abolish glomerular filtration. Hix in his experiment on conscious dogs demonstrated decrease in ipsilateral and contralateral renal blood flow following ureteral catheterization suggesting ureterorenal reflex as cause for spasm of intrarenal arterioles.⁹ Thomsen et al in his experiment on baby pigs demonstrated that increase in intra-pelvic pressure and intra renal back flow of contrast medium can decrease renal blood flow.¹⁰ However, in both the above-mentioned experiments anuria was never produced casting doubt on the vasospasm theory. The second hypothesis for the pathogenesis of RA postulates spasm of both ureters resulting in a functional obstruction. Shafik in his experiment on dogs showed that distension

of the renal pelvis with large volumes led to an increase in pressure in the contralateral renal pelvis but not in the ureter postulating reno-renal pelvic reflex. But again, no mention about anuria questions the ureteric spasm theory.¹¹

Till the date of writing this case report, 16 cases of RA post retrograde pyelography have been reported within the English language literature. The duration of anuria ranged from 24 hours to 7 days; and time for renal function to return to baseline ranged from 4 days to 1 month.⁵ Only 3 cases have needed dialysis, and all 3 patients began making urine a short time after hemodialysis.^{5,12,13} Compared to previous reported cases, in our case the patient had anuria for 4 days and his renal function returned to baseline after 16 days.

Treatment for RA is largely supportive. Kidney function and electrolytes should be frequently monitored and an indwelling urinary catheter should ideally be inserted to monitor urine output. Hemodialysis may be necessary for severe cases of hyperkalemia or fluid overload. In different case series, patients with ureteral catheter induced RA treated with indwelling stent placement have not needed hemodialysis.^{2,14,15} In light of this, it is unclear if repeat cystoscopy and stent placement would have avoided hemodialysis in our patient. Whether ureteric stenting in such cases should be an integral part of management is controversial, particularly since this procedure may itself predispose to RA. We propose treatment decisions be made on a case-by-case basis and involving consultations with nephrology and urology service.

Conclusions

Reflex anuria is an extremely rare cause of anuric AKI and is often a diagnosis of exclusion. The mechanism of this disease entity is still unclear. Management decisions should be individualized; particularly with respect to further urologic manipulations, hemodialysis and conservative treatment. Fortunately, its prognosis appears to be favourable with renal function recovering to baseline in all cases.

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Figures and Tables

Table 1. Clinical course during hospitalization				
Date	Creatinine (umol/L)	Potassium (meq/L)	Urine output (ml)	Weight (kg)
May 19	181	4.2		
June 3	Ureteroscopy with bilateral retrograde pyelography			
June 4	515	4.6	0	125
June 5	559	5.1	0	126
June 6	791	6	0	127
June 6–7	Hemodialysis			
June 7	684	4.7	800	
June 8	751	4.9	1600	126
June 9	741	4.5	3100	
June 10	684	4.8	3350	122
June 11	607	4.1	3800	119.4
June 12	440	3.8		
	Discharge from hospital			
June 19	191			