"I just can't pee!"

Michelle Strasberg, BSc (Hons); and
Sam G. Campbell, MB, BCh, CCFP(EM,) CHE

George's case

George, 66, presents to the ED with abdominal pain and a decreased urine output of 3 days duration (< 400 mL in 24 hours).

Past medical history

- Hypertension treated with: 10 mg of ramipril q.d., 12.5 mg of hydrochlorothiazide q.d. and 5 mg of amlodipine q.d.
- Hypercholesterolemia treated with: 20 mg of rosuvastatin q.d.

Examination

George's physical exam shows:
- Temperature: 37.9°C
- BP: 182/110 mmHg
- Heart rate: 72 bpm
- Respiratory rate: 18 breaths per minute
- Oxygen saturation: 99%

George's abdomen is not distended, there is no hepatosplenomegaly, but bowel sounds are present. He complains of tenderness upon palpation of the mid-lower abdomen and the bladder is palpable. A rectal exam demonstrates an enlarged, firm, non-tender prostate.

Investigations

Clinical investigations reveal:
- Hemoglobin: 145 g/L
- White blood cell count: 7.6×10^9/L
- Platelets: 236×10^9/L
- Sodium: 136 mmol/L
- Potassium: 3.3 mmol/L
- Bicarbonate: 25 mmol/L
- Glucose: 5.5 mmol/L
- Creatinine: 350 µmol/L

Turn to page 2 for more on George.

Questions & Answers

1. *What are the different categories of RF and how can they be distinguished?*

Renal failure (RF) can be categorized by route cause into:
- Pre-renal RF
- Intrinsic RF
- Post-renal RF (obstructive uropathy)

Pre-renal RF

Pre-renal RF results from the response of a normal kidney to hypoperfusion. It can be caused by hypovolemia, decreased cardiac output, renal vasoconstriction or large vessel occlusion. Untreated, pre-renal RF can result in intrinsic RF.

Intrinsic RF

Intrinsic RF occurs as a result of damage to the kidney itself. The three main etiologies of instrinsic RF are:
- Acute tubular necrosis: This is caused by ischemia or toxins and is evidenced by the presence of pigmented granular casts and red blood cells (RBCs) in the urine
- Acute interstitial nephritis: This may be caused by medications, kidney infections (pyelonephritis) or an infiltrative process. Urine will contain white blood cells (WBCs), WBC casts and possibly RBCs
- Renal vascular disease (glomerulonephritis): This is caused by multiple small vessel insults including:
  - Disseminated intra-vascular coagulation
  - Preeclampsia

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George's case cont'd...

Abdominal ultrasounds (Figures 1 and 2) find:

- Aorta within normal limits
- Bilateral hydronephrosis not resoved post-void
- Thick and trabeculated bladder wall
- Large post-void residual urine volume
- Significantly enlarged prostate

Go to page 4 for George's treatment.

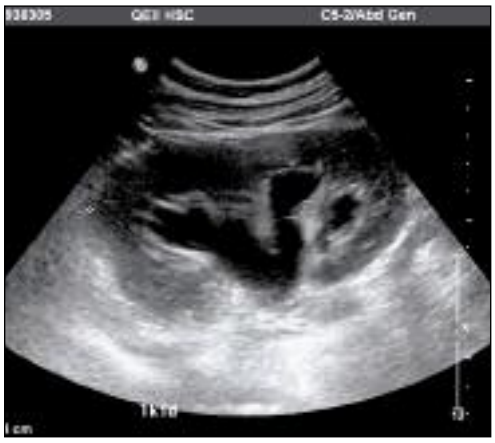

Figure 1. Ultrasound of the right kidney showing the distended renal calyces of hydronephrosis.

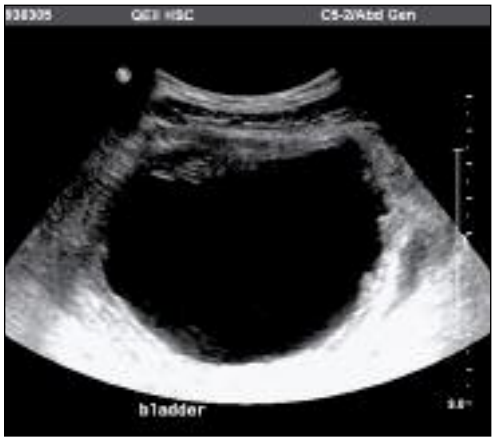

Figure 2. Post-void ultrasound showing the bladder distended by 1 L of fluid, plus bladder wall thickening.

- Cholesterol emboli
- Endocarditis
- Hypertensive crisis

In glomerulonephritis, dysmorphic RBCs and RBC casts are present in the urine.

Post-renal RF

Post-renal RF is caused by an obstruction of the bladder neck, urethra, or renal tubules.

2. What is George's diagnosis?

George has acute post-renal RF (obstructive uropathy) due to an enlarged prostate pressing on his bladder neck, obstructing urine outflow through the urethra.

3. What is the acute treatment of obstructive uropathy and what should you be aware of?

The acute treatment of obstructive uropathy is decompression of the bladder. This is usually achieved by the insertion of a catheter into the bladder. If this cannot be achieved, a suprapubic catheter is urgently indicated.

A major complication after the treatment of obstructive uropathy is excessive diuresis. The urine output, after a catheter is inserted, can exceed 10 L q.d., resulting in volume depletion. Accordingly, the patient should be monitored carefully for clinical signs of volume depletion, including hypotension and increased blood urea nitrogen. Fluid should be replaced appropriately.

George's treatment

Treatment

Several attempts to catheterize George's urethra with a Foley catheter fail and he requires the placement of a suprapubic catheter, which drains 1,000 mL of urine.

He is admitted to hospital for observation and experiences significant hematuria as his urinary system decompresses.

His BP is gradually controlled with an increased dose of amlodipine (10 mg q.d.) and the addition of 2 mg of doxazosin q.d.

George undergoes a transurethral prostatic resection and is able to void normally the following day. The suprapubic catheter is removed and he is discharged. At discharge, his creatinine is 168 mmol/L.

One week later, his creatinine is 145 mmol/L and he feels fine.

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4. *Are there any other interventions in this case?*

Other renal insults should be minimized wherever possible. In this case, George should discontinue the use of ramipril. ACE inhibitors have been known to alter the hemodynamics within the renal glomerulus. They prevent the conversion of angiotensin I to angiotensin II, causing vasodilatation of the efferent arterioles. Subsequently, hydrostatic pressure is decreased, leading to a decrease in filtration through the glomerulus.

Hydrochlorothiazide should also be discontinued to decrease the risk of post-obstructive diuresis.

George's BP should also be monitored. It is likely to settle spontaneously, but medication dose increase or additional antihypertensives may be required. Dx

Ms. Strasberg is a Fourth Year Medical Student, University of Toronto, Toronto, Ontario.

Dr. Campbell is an Associate Professor of Emergency Medicine, Dalhousie University, Halifax, Nova Scotia.

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Return undeliverable Canadian addresses to:
STA Communications Inc.
955 boulevard St-Jean, Suite 306
Pointe-Claire, QC, H9R 5K3