

Bilateral Ureteropelvic Junction Obstruction in an Adult Male: A Rare Cause of Hypertensive Crisis

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Background

- Bilateral ureteropelvic junction (UPJ) obstruction as a cause of secondary hypertension commonly occurs in children secondary to a congenital malformation.
- Secondary hypertension in the setting of an acquired bilateral obstructive uropathy among adults, however, is less frequently encountered.
- Prompt recognition and treatment of the obstruction is therefore necessary to resolve the hypertensive crisis.

Case Presentation

- A 41-year-old man with history of hypertension and morbid obesity presented to the emergency department with a four-day history of anuria and abdominal pain without other symptoms.
- His blood pressure was 262/147 mmHg with a heart rate of 102 beats/min, respiratory rate of 18 breaths/min and oxygen saturation of 97% on ambient air. Cardiopulmonary examination was unremarkable. He had generalized abdominal pain on palpation. No costovertebral angle tenderness was elicited.
- Serum creatinine was 14.1mg/dL from his baseline of 1.6 mg/dL, BUN: Creatinine ratio was 4.04, urinalysis showed microscopic hematuria.
- Chest X-ray revealed mild pulmonary edema.



Figure 1: Obstructive 1cm calculus at the left UPJ causing moderate to severe hydronephrosis



Figure 2: Obstructive 1.4 cm calculus at the right UPJ causing moderate hydronephrosis

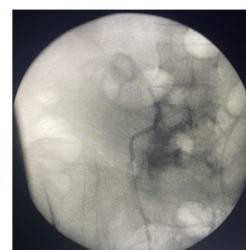


Figure 3: Right renal pelvis dilation with filling defect consistent with calculus noted on sonogram.

Interventions

- He was managed with IV Labetalol 10mg, oral Carvedilol 25mg, and oral Hydralazine 25mg which slightly decreased his high blood pressure by about SBP 40mmHg and DBP by 10mmHg.
- A straight catheter was inserted; however no urine was seen. Bladder scan ruled out urinary retention.
- Ultrasound of the kidneys showed bilateral UPJ obstruction with hydronephrosis from bilaterally obstructing nephrolithiasis. (**Figure 1&2**)
- He underwent bilateral retrograde pyelogram, bilateral ureteral stent placement and cystoscopy.
- After the procedure, the patient's blood pressure improved dramatically and returned to his baseline of 140/80 mmHg.
- The serum creatinine also trended downward to 2.2 mg/dL which is close to his baseline of 1.6mg/dL (**Figure 4**)
- Foley catheter was initially placed and was eventually able to urinate by himself. Definitive management of the bilateral obstructing nephrolithiasis will be performed as outpatient.

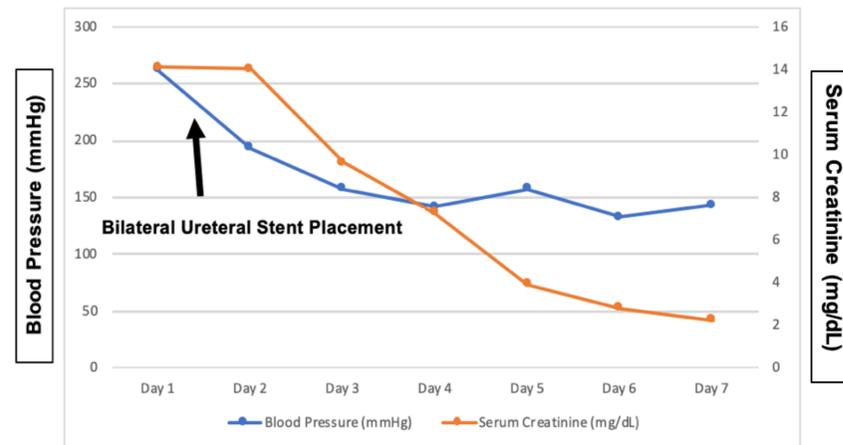


Figure 4: Blood pressure and serum creatinine trend on admission and after Bilateral Ureteral Stent Placement

Discussion

- This case illustrates the importance of prompt management of the underlying secondary cause of a hypertensive crisis.
- In this case, the patient had a bilateral UPJ obstruction causing hydronephrosis secondary to bilateral nephrolithiasis. Although the incidence of nephroliths causing bilateral obstruction simultaneously is less common, it can still occur, and is a plausible cause of secondary hypertensive crisis.
- Two mechanisms are implicated in the development of hypertension in hydronephrotic kidneys, including increased tubuloglomerular feedback (TGF) activity and Renin-Angiotensin-Aldosterone System (RAAS) activation. Nitric oxide, a modulator of TGF, is downregulated in hydronephrotic kidneys. This causes unrestricted vasoconstriction of the afferent arteriole, decreasing the sodium delivery to the macula densa, thereby activating the RAAS and activating its downstream effects. The improvement of hypertension after relief of obstruction suggests that humorally mediated vasoconstriction plays a significant role in the mechanism by which hydronephrosis causes hypertension.
- Therefore, prompt treatment of the underlying obstruction is indicated to reverse the effects of angiotensin II mediated vasoconstriction and the aldosterone effect of water and sodium retention in hydronephrotic kidneys.

Conclusions

- Bilateral Ureteropelvic Junction obstruction is a rare but possible cause of hypertensive emergency in adults.
- Mechanisms implied in the development of secondary hypertensive crises in the setting of bilateral hydronephrosis from obstruction include increased tubuloglomerular feedback (TGF) and Renin-Angiotensin-Aldosterone System activation.
- Prompt management of the obstructive uropathy is required in order to resolve the hypertensive crises and prevent target organ damage.

References

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