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CASE REPORT

Pediatrics

Abdominal distension and lower extremity edema

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Abstract

Posterior urethral valves are a congenital malformation in males that results in the obstruction of urinary outflow from the proximal urethra. Presentation depends on severity of disease, with patients presenting antenatally with oligohydramnios and hydronephrosis, to postnatally with delayed or poor voiding, urinary tract infections, or renal anomalies on ultrasound. Our case illustrates an 11-day-old male who presented to the emergency department with poor feeding, vomiting, significant abdominal distension, and pitting edema of the lower extremities. The patient was found to have hypoglycemia, hyponatremia, hyperkalemia, and acute renal failure. Point of care ultrasound showed severe bilateral hydronephrosis with a significantly distended bladder that was compressing the inferior vena cava. Early recognition of the obstruction via ultrasound led to rapid decompression of the bladder using catheterization and immediate involvement of urology, nephrology, and neonatology consults in the pediatric emergency department.

KEYWORDS
emergency medicine, point of care ultrasound, posterior urethral valves

1 | INTRODUCTION

Posterior urethral valves (PUV) are a congenital malformation in males, occurring in 1 in 5000 to 8000 births, which results in urinary outflow obstruction from the proximal urethra.[1] Depending on the type and configuration of the membranous obstruction within the urethra a range in presentation and severity is seen.[2,3] Presentation of postnatally diagnosed PUV consists of delayed voiding, poor urinary stream, failure to thrive, lethargy, urosepsis, or pulmonary hypoplasia.[1] Renal and bladder ultrasound may show a thickened bladder wall and a dilated and elongated posterior urethra.[4] Diagnosis is confirmed by voiding cystourethrogram, which usually shows thickened trabeculated bladder, dilatation of the posterior urethra, circumferential filling defect at the level of the pelvic floor, and prominence of the bladder neck.[5,6]

2 | CASE PRESENTATION

An 11-day old male presented to the pediatric emergency department (ED) because of concern for abdominal distension, vomiting, and poor feeding. He was born at 38 weeks via cesarean section to a mother with gestational diabetes. He was discharged from the nursery at 48 hours of life. At home the mother reported he was feeding well, urinating small amounts 5–6 times a day, and stooling appropriately.

On the morning of arrival to the pediatric ED he was no longer feeding well and had one episode of non-bilious emesis. Upon arrival to the pediatric ED he was noted to have significant abdominal distension and tenderness, a sunken fontanelle, as well pitting edema to the lower extremities and poor tone. Initial vital signs showed mild tachycardia, but he was otherwise afebrile, respiratory rate of 46, and blood pressure of 85/55.

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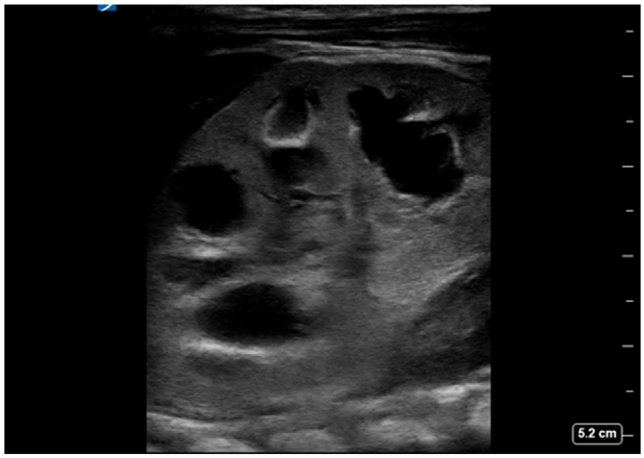

FIGURE 1 Point of care ultrasound image of the right kidney showing severe hydronephrosis. Image obtained using the linear probe on the Sonosite X-porte machine

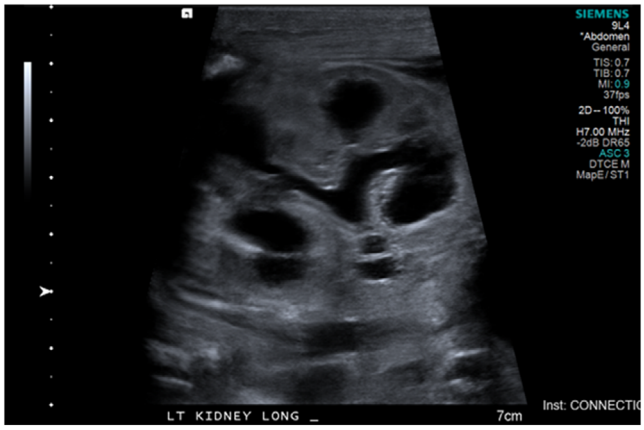

FIGURE 2 Radiology obtained ultrasound image of the left kidney showing severe hydronephrosis

Using the Sonosite X-porte (Fujifilm SonoSite, Inc., Bothell, WA) with a curvelinear probe we were able identify severe bilateral hydronephrosis (Figures 1 and 2) and a significantly distended bladder (Figure 3) that was compressing flow in the inferior vena cava. As a result, straight catheterization of the bladder was performed, which drained 260 cc of urine. Immediate improvement of abdominal distension was noted. Improvement of lower extremity edema was noted 24 hours after admission.

Initial laboratory studies showed hypoglycemia (21), hyponatremia (113), hyperkalemia (8.7), hypochloremia (76), and renal failure (blood urea nitrogen [BUN] 71 and creatinine 6.8). Hypoglycemia was corrected with dextrose infusion. Urology and nephrology were consulted.

The patient was admitted to the neonatal intensive care unit (ICU) where he was started on broad spectrum antibiotics and fluids of 100 mL/kg/h. Electrolytes and urine output were monitored hourly. His hyponatremia and hypoglycemia corrected rapidly, along with a quick reduction in serum creatinine and BUN. PUV were confirmed by fluoroscopy urethrocystogram and cystoscopy on day 3 of admission and he had resection of the valves during the procedure.

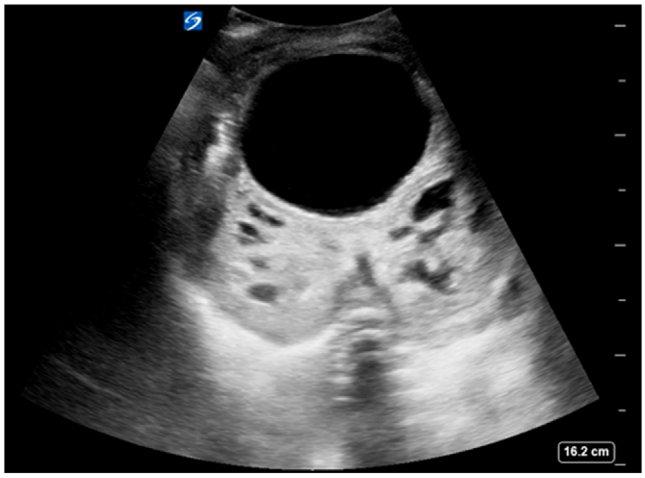

FIGURE 3 Point of care ultrasound image showing a full bladder before decompression with foley catheter. Image obtained using the curvilinear probe on the Sonosite X-porte machine

3 | DISCUSSION

PUV are often found antenatally or while infants are still in the nursery because of poor urine output or urine stream. Outside of the nursery infants often present to the ED for urinary tract infections, failure to thrive, or in urosepsis.[1] The infant in this case had been gaining weight appropriately per his pediatrician, which was likely secondary to fluid accumulation rather than appropriate weight gain. This led to his case being missed until abdominal distension was large enough to cause more profound effects.

On initial exam this infant was noted to have significant abdominal distension. Point of care ultrasound was able to see a large volume of urine in the bladder and associated bilateral hydronephrosis. The distended bladder caused compression of the inferior vena cava, which resulted in lower extremity edema. This presentation has not been previously documented in infants with PUV. Previous studies have shown bladder hypertrophy, hydronephrosis, and hydroureter.[6]

The ability to evaluate the bladder via point of care ultrasound led to rapid decompression of the bladder and early urology consult. Early detection is important as prolonged obstruction and hydronephrosis can lead to worse long-term prognosis and chronic kidney disease.[7] Even after the PUV are ablated, children can develop long-term consequences including chronic kidney disease and bladder dysfunction.[5,7]

The electrolyte abnormalities (hyponatremia and hyperkalemia) in this case were attributed to severe hydronephrosis resulting in compression of the renal calices causing a secondary (transient) pseudohypoaldosteronism. Once the obstruction was relieved with the aid of a urinary catheter, abnormalities improved. This phenomenon has been previously reported in the literature associated with urinary tract malformations but usually presents with urinary tract infection.[8]

4 | CONCLUSION

PUV are a common cause of urinary tract obstruction. Although often caught neonatally, it can be missed if infants are able to have good urinary output initially. Accumulation of urine within the bladder results in abdominal compression, renal injury, as well as electrolyte derangement. In this case, point of care ultrasound was able to identify a distended bladder that was compressing the inferior vena cava leading to extremity edema, a finding not previously reported in the literature. The rapid recognition of this phenomenon led to immediate decompression of the bladder as well as urology and nephrology consultation.

CONFLICT OF INTEREST

None

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