Tubular stasis nephropathy

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Section: Paediatric radiology
Imaging Technique: Ultrasound
Case Type: Clinical Cases
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Patient: 2 days, male

Clinical History:

Oliguria. Clinical examination was unremarkable, but the renal parameters (blood urea and serum creatinine) were elevated.

Imaging Findings:

The newborn presented with oliguria. Clinical examination was unremarkable, but the renal parameters (blood urea and serum creatinine) were elevated at the time of presentation. Ultrasound revealed echogenic renal pyramids, with normal cortex and normal renal lengths in both kidneys. The serum calcium and the urinary calcium were within normal limits. The patient's urine output and the renal parameters improved gradually over a week. A repeat ultrasonogram after a week revealed resolution of the pyramidal echogenicity.
Discussion:

Tubular stasis nephropathy was first described by Avni et al in 1982. This affects the neonates and the features include a) transient renal impairment b) bright echogenic renal pyramids on ultrasonogram c) normal renal cortex d) gradual reversal of medullary hyperechogenicity in 7-30 days.

The sonographic appearances of the neonatal kidney differ considerably from that of older children and adults. Infant kidneys have prominent hypoechoic renal pyramids in contrast to echogenic renal cortex. The newborn kidney also lacks sinus echogenicity. These features (hyperluscent pyramids, lack of sinus echogenicity) in newborn kidneys are likely to lend to ultrasound a greater sensitivity for reflecting the pathological changes that occur in the renal medulla.

The cause of the tubular stasis nephropathy is unknown. It has been proposed that transient medullary hyperechogenicity may represent transient tubular blockade caused by Tamm-Horsfall protein (THP) forming aggregates within the renal tubules. THP is a macroglobulin normally found in the urine, amniotic fluid and serum. Stasis nephropathy should be differentiated from the other causes of renal medullary hyperechogenicity. In the first week after delivery, increased medullary hyperechogenicity is demonstrable in the course of renal vein thrombosis, nephrocalcinosis after administration of frusemide or of high doses of vitamin D and/or calcium, fungal infection and occasionally with autosomal recessive polycystic kidneys. However transient increase in the echogenicity of the medullary pyramids could represent the spectrum of normal findings (Riebel et al).

Hypercalciuria and hypercalcaemia should be excluded in all newborns presenting with bright echogenic renal pyramids on ultrasonogram. A repeat ultrasonogram in 10 days to look for the resolution of the medullary hyperechogenicity is often helpful to confirm the diagnosis of stasis nephropathy and to exclude the other pathological causes of bright echogenic renal pyramids.

Differential Diagnosis List: Tubular stasis nephropathy

Final Diagnosis: Tubular stasis nephropathy

References:

Riebel TW, Abraham K, Wartner R, Muller R.

Transient renal medullary hyperechogenicity in ultrasound studies of neonates: is it a normal phenomenon and what are the causes?


Hernanz-Schulman M.

Hyperechoic renal medullary pyramids in infants and children


Shultz PK, Strife JL, Strife CF, McDaniel JD.

Hyperechoic renal medullary pyramids in infants and children

Greenberger M, Skoog SJ.

Renal medullary hyperechogenicity of the neonate.


Figure 1

**Description:** Ultrasound showing bright echogenic renal pyramids. **Origin:**
Description: Follow up scan after 7 days showing complete resolution of the echogenicity. Origin: