CASE REPORT

Transient renal medullary hyperechogenicity in a term neonate

Radhakrishnan Hemachandar,¹ Vinoth Boopathy²

¹Department of Nephrology, Mahatma Gandhi Medical College and Research Institute, Puducherry, India

²Department of Medical Gastroenterology, Mahatma Gandhi Medical College and Research Institute, Puducherry, India

Correspondence to

Dr Vinoth Boopathy, drbvinoth@gmail.com

Accepted 28 July 2015

SUMMARY

Although neonatal renal medullary hyperechogenicity can occur in a variety of pathological conditions, it has been reported to occur transiently with or without renal abnormalities in term as well as in preterm neonates. Variably described in the literature as Tamm-Horsfall nephropathy, or stasis nephropathy, the pathogenesis is unknown, but has an excellent prognosis. The authors describe a 3-day-old neonate with transient renal medullary hyperechogenicity, with no other abnormalities, which resolved within 2 weeks.

BACKGROUND

Renal medullary hyperechogenicity has been reported in neonates with severe perinatal renal injury, kidney malformations and nephrocalcinosis, as well as in neonates with transient renal failure. However, very rarely, it may be transient without any biochemical abnormalities, as in our case, and does not need intervention.

CASE PRESENTATION

A 3-day-old girl was referred to the department of nephrology for evaluation of nephrocalcinosis based on ultrasound finding of hyperechogenic medullary pyramids. She was born of full term through a normal vaginal delivery, with a birth weight of 2.8 kg; she cried immediately after birth. She was breastfed immediately after birth. She started passing urine within 6 h after birth. The perinatal period was uneventful. There was no history of drug intake either during pregnancy or after birth. Antenatal ultrasound at 28 weeks showed bilateral pelviectasis with normal parenchymal echogenicity. The infant passed urine at the rate of 2.3 mL/kg/h. Physical examination was normal with heart rate of 140 per minute, blood pressure 70/40 mm Hg, respiratory rate 30 per minute and temperature of 98.6 F.

INVESTIGATIONS

Laboratory data showed haemoglobin 162 g/L, blood urea 4.2 mmol/L, serum creatinine 51.27 µmol/L, serum sodium 142 mmol/L, serum potassium 3.8 mmol/L, uric acid 285.53 µmol/L, calcium 2.08 mmol/L, pH 7.38, pCO₂ 5.45 kPa and serum bicarbonate 25 mmol/L. Urine analysis was normal. There was no hypercalciuria. Follow-up ultrasound after birth showed normal cortical echogenicity of both kidneys with hyperechogenic medullary pyramids without acoustic shadows (figure 1).

Figure 1 Ultrasound picture of right kidney showing hyperechogenicity of medullary pyramids without acoustic shadowing.

TREATMENT

The parents were reassured and advised to feed the infant breast milk on demand to ensure she had adequate hydration.

OUTCOME AND FOLLOW-UP

Review ultrasound 2 weeks later revealed normal cortical and medullary echoes (figure 2).

DISCUSSION

In neonates, the renal medulla is relatively hypoechoic compared to the cortex. However, renal medullary hyperechogenicity has been reported to occur in neonates undergoing furosemide therapy, and in those with nephrocalcinosis and transient oliguric renal failure.¹ Nephrocalcinosis is common in term as well as in premature infants who have had perinatal renal injury. Other causes of neonatal nephrocalcinosis include William's syndrome, distal renal tubular acidosis, neonatal Bartter's syndrome, oxalosis, vitamin D therapy and primary hyperparathyroidism.¹

Figure 2 Ultrasound picture of right kidney after 2 weeks showing complete resolution of medullary hyperechogenicity.

CrossMark

To cite: Hemachandar R, Boopathy V. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2015-211285

Transient echogenic renal medullary change in neonates was first described in 1982.² It has been described in preterm as well as in term neonates with or without renal dysfunction within a few days of birth.^{2,3} The incidence of renal medullary hyperechogenicity without renal impairment has been reported to be 3.9–37% within 7–10 days after birth.⁴

Makhoul *et al*⁵ described nine term neonates who, after birth, developed transient renal dysfunction, oligoanuria and bilateral medullary hyperechogenicity, with excellent prognosis. Additional findings included proteinuria (100%), uric acid crystalluria (78%) and hyperuricaemia (44%), with mean creatinine of 1.09 mg/dL. Based on their data, the authors calculated a prevalence of 1 in 1000 term newborns. Riebel *et al*⁶ reported transient medullary hyperechogenicity of varying intensity in 37% of neonates. There was no associated renal impairment and the echogenicity disappeared completely within the first week of life. It occurred twice as often in full-term than in premature babies.

The hyperechogenicity is thought to result from precipitation of an endogenous substance in the tubules in the first few days of life, causing a reversible tubular obstruction. The origin of these densities is implied in several reports to be urinary protein, specifically Tamm-Horsfall protein, or uric acid crystals.⁴ Tamm-Horsfall protein is produced by the kidney in the thick ascending limb of the loop of Henle. It is the most abundant protein excreted in human urine and forms the matrix of cellular casts in urine. Hence the term Tamm-Horsfall nephropathy was coined to denote the deposition of Tamm-Horsfall protein in the renal tubules of neonates in a state of dehydration.⁴ Uric acid can precipitate in the kidney leading to reversible tubular obstruction.⁷

Riebel *et al*⁶ determined the concentration of Tamm-Horsfall protein, calcium, uric acid and osmolality in such neonates. The authors did not find any definite clues to the aetiology of the ultrasound features. Hence they suggested the term Tamm-Horsfall nephropathy should no longer be used. Rather, such transient echogenicity should be regarded as normal. They proposed the term ‘tubular stasis nephropathy’ should be reserved for cases with clinically demonstrable impairment of renal function.

Bouwman *et al*⁴ reviewed the diagnostic features of transient renal medullary hyperechogenicity. It is a diagnosis of exclusion. Nephrotoxic medications such as ACE inhibitors or furosemide to either mother or newborn, should be excluded. Prenatal ultrasound (if performed) should be normal. Physical examination is unremarkable except for dehydration. Biochemical features include increase in serum creatinine, urea and uric acid, in combination with proteinuria and uric acid crystalluria. On ultrasound, the hyperechogenicity is restricted to the tips of the otherwise echolucent pyramids and the size of the kidneys is normal.⁸ In contrast, nephrocalcinosis is characterised by a more diffuse hyperechogenicity of the renal cortex and

pyramids, often with acoustic shadowing. The echogenic lesions completely disappear within 8 days.

Treatment is adequate hydration and reassurance. Renal ultrasound should be repeated after 1–2 weeks to document complete resolution of renal echogenicity. Transient renal medullary hyperechogenicity in neonates is a not-so-rare entity of unclear aetiology, with excellent prognosis. Physicians should be aware of this normal variation that appears to be related to dehydration.

Learning points

- Renal medullary hyperechogenicity in neonates without or with mild renal impairment should be recognised as a normal variant and has an excellent prognosis.
- Physical examination appears normal except for dehydration.
- There are no biochemical abnormalities except for mild renal dysfunction and dehydration.
- Follow-up ultrasound examination should be carried out to document complete resolution of echogenicity.

Twitter Follow Vinodh Boopathy at @drbvinodh

Contributors RH is the treating physician and prepared the manuscript. VB edited the manuscript and performed the literature review.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Schell-Feith E, Kist-van Holthe J, van der Heijden A. Nephrocalcinosis in preterm neonates. *Pediatr Nephrol* 2008;25:221–30.
- 2 Howlett DC, Greenwood KL, Jarosz JM, *et al*. The incidence of transient renal medullary hyperechogenicity in neonatal ultrasound examination. *Br J Radiol* 1997;70:140–3.
- 3 Pleasure J, Wolfson B, Hoffman D, *et al*. Transient renal medullary hyperechoic Foci in the Neonate: normal variation found in the first few days of life. *Pediatr Res* 1999;45:219A.
- 4 Bouwman A, Verbeke J, Brand M, *et al*. Renal medullary hyperechogenicity in a neonate with oliguria. *Clinical Kidney J* 2010;3:176–8.
- 5 Makhoul IR, Soudack M, Smolkin T, *et al*. Neonatal transient renal failure with renal medullary hyperechogenicity: clinical and laboratory features. *Pediatr Nephrol* 2005;20:904–9.
- 6 Riebel TW, Abraham K, Wartner R, *et al*. Transient renal medullary hyperechogenicity in ultrasound studies of neonates: is it a normal phenomenon and what are the causes? *J Clin Ultrasound* 1993;21:25–31.
- 7 Gottlieb RP, Roeloffs S, Galler-Rimm G, *et al*. Transient renal insufficiency in the neonate related to hyperuricemia and hyperuricosuria. *Child Nephrol Urol* 1991;11:111–14.
- 8 Nakamura M, Yokota K, Chen C, *et al*. Hyperechoic renal papillae as a physiological finding in neonates. *Clin Radiol* 1999;54:233–6.

Copyright 2015 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow