- 6 **Saland JM**, Mahony L, Baum M. Perinatal renal ischaemia resulting in hypertensive cardiomyopathy. *Pediatrics* 2001;**107**:185–187.
- 7 **Landing BH**, Ang SM, Herta N, et al. Labeled lectin studies of renal tubular dysgenesis and renal tubular atrophy of postnatal renal ischemia and end-stage kidney disease. *Pediatr Pathol* 1994;**14**:87–99.
- 8 **Allanson JE**, Pantzar JT, MacLeod PM. Possible new autosomal recessive syndrome with unusual renal histopathological changes. *Am J Med Genet* 1983;**16**:57–60.
- 9 **Gribouval O**, Gonzales M, Neuhaus T, et al. Mutations in genes in the renin-angiotensin system are associated with autosomal recessive renal tubular dysgenesis. *Nat Genet* 2005;**37**:964–968.
- 10 **Daikha-Dahmane F**, Levy-Beff E, Jugie M, et al. Foetal kidney maldevelopment in maternal use of angiotensin II type I receptor antagonists. *Pediatr Nephrol* 2006;**21**:729–732.
- 11 **Bush KT**, Sakurai H, Steer DL, et al. TGF- β superfamily members modulate growth, branching, shaping, and patterning of the ureteric bud. *Dev Biol* 2004;**266**:285–298.
- 12 **Tufro-McReddie A**, Romano LM, Harris JM, et al. Angiotensin II regulates nephrogenesis and renal vascular development. *Am J Physiol* 1995;**269**:F110–F115.
- 13 **Karihaloo A**, Karumanchi SA, Cantley WL, et al. Vascular endothelial growth factor induces branching morphogenesis/tubulogenesis in renal epithelial cells in a neuropilin-dependent fashion. *Mol Cell Biol* 2005;**25**:7441–7448.
- 14 **Guron G**, Frieberg P. An intact rennin-angiotensin system is a prerequisite for normal renal development. *J Hypertens* 2000;**18**:123–137.
- 15 **Barr M Jr**, Sedman AB, Heidelberger KP. Renal tubular dysgenesis in twins. *Pediatr Nephrol* 1998;**12**:408–413.
- 16 **Barr M Jr**, Cohen M Jr. ACE inhibitor fetopathy and hypocalvaria; the kidney-skull connection. *Teratology* 1991;**44**:485–495.
- 17 **Genest DR**, Lage JM. Absence of normal-appearing proximal tubules in the fetal and Neonatal kidney: prevalence and significance. *Human Pathol* 1991;**22**:147–153.
- 18 **Johal JS**, Thorp JW, Oyer CE. Neonatal haemochromatosis, renal tubular dysgenesis and hypocalveria in a neonate. *Pediatr Dev Pathol* 1998;**1**:433–437.
- 19 **Marcussen N**. Atubular glomeruli in renal artery stenosis. *Lab Invest* 1991;**65**:558–565.
- 20 **Rapola J**. The Kidneys and urinary tract. In: Wigglesworth JS, Singer DB, eds. *Textbook of Fetal and Perinatal Pathology*. Massachusetts: Blackwell Science, 1998:982–1012.
- 21 **Edgill EL**, Bingham C, Ellard S, et al. Mutations in hepatocyte nuclear factor-1 α and their related phenotypes. *J Med Genet* 2006;**43**:84–90.
- 22 **Kolatsi-Joannou M**, Bingham C, Bulman MP, et al. Hepatocyte nuclear factor-1 beta: a new kindred with renal cysts and diabetes and gene expression in normal human development. *J Am Soc Nephrol* 2001;**12**:2175–2180.
- 23 **Bellanne-Chantelot C**, Chuveau D, Goutier JF, et al. Clinical spectrum associated with hepatocyte nuclear factor-1 beta mutations. *Ann Intern Med* 2004;**140**:510–517.
- 24 **Daftary AS**, Patole SK, Whitehall J. Hypertension-hyponatremia syndrome in neonates: case report and review of literature. *American Journal of Perinatology* 1999;**16**:385–389.

IMAGES IN NEONATAL MEDICINE.....

doi: 10.1136/adc.2006.114090

Neonatal subcutaneous fat necrosis with characteristic rash and hypercalcaemia

A 3.305 kg baby girl was born at term following an emergency caesarean section for reduced fetal movements and meconium staining of liquor. Cord blood gases showed pH of 7 and base deficit of 13.9. She fed poorly and developed symptoms of hypoglycaemia. Her lowest recorded temperature was 35.5°C. On day 4, she developed an extensive rash over her back, upper thighs and groin area. The rash was erythematous, indurated and tender on palpation, consistent with extensive subcutaneous fat necrosis (SCFN) (fig 1A). Investigations for sepsis revealed negative cultures.

When reviewed at 4 weeks of age the baby was failing to thrive and was hypercalcaemic with adjusted calcium level of 3.99 mmol/l. She received intravenous fluids, oral furosemide and spironolactone. She was given low calcium formula but her adjusted calcium level remained high at 4.0 mmol/l. A renal ultrasound showed medullary echogenicity consistent with nephrocalcinosis. She was given prednisolone (1 mg/kg/day) and diuretics were discontinued. The calcium level normalised and prednisolone was weaned over five weeks. At 9 months of age her weight remains below the 0.4th percentile. Serum calcium and creatinine are normal and the skin changes are less extensive, but areas of induration remain (fig 1B) and ultrasonographically nephrocalcinosis persists.

Subcutaneous fat necrosis is rare, but it requires careful investigation and follow up for potentially serious sequelae.^{1,2} Treatment of hypercalcaemia is controversial.³

Figure 1 Baby girl with subcutaneous fat necrosis at age (A) 5 days and (B) 9 months. Parental/guardian informed consent was obtained for publication of this figure.

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Competing interests: None.

REFERENCES

- 1 **Bartrop D**. Hypercalcaemia associated with neonatal subcutaneous fat necrosis. *Arch Dis Child* 1963;**38**:516–8.
- 2 **Cunningham K**, Paes BA. Subcutaneous fat necrosis of the newborn with hypercalcaemia. A review. *Neonatal Netw* 1991;**10**:7–14.
- 3 **Alos N**, Eugene D, Fillion M, et al. Pamidronate: treatment for severe hypercalcaemia in neonatal subcutaneous fat necrosis. *Horm Res* 2006;**65**:289–94.