Diagnosis of an ectopic ureter in a girl by differential urine collection after administration of desmopressin acetate

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Urinary incontinence in girls is very common, usually presenting as intermittent, frequent urinary dribbling due to detrusor instability. Rarely, continuous urinary dribbling is caused by a dysplastic, poorly functioning moiety of a duplex kidney, or a unilateral dysplastic kidney draining into a ureter that exits ectopically to the vagina or distal urethra.1–4 Typically, the diagnosis is made after a long delay.2–4 This is partly because the dribbling pattern is not always easy to ascertain,3 but mainly because it may be difficult to confirm the diagnosis preoperatively by examination of the genitalia,24 or using various imaging techniques.1–4 We present a girl in whom the diagnosis was confirmed by chemical analysis of urine collected after the administration of desmopressin acetate.

Patient, method, and results
An antenatal ultrasound diagnosis was made of a female infant with a normal left kidney, but a duplex right kidney with a dilated, dysplastic upper moiety. Postnatally, her renal function was normal, and imaging investigations included ultrasound, micturating cystogram, dimercapto succinic acid and mercaptoacetyltriglycine (MAG-3) scans. These confirmed that she did not have vesicoureteric reflux, and that her left kidney and right lower moiety were normal and contributed 47% and 48% of her total renal function respectively. She had a dysplastic right upper moiety that contributed 5% of the function, and was drained by a dilated pelvis (20 mm anteroposterior diameter) and ureter, but was not obstructed. She was treated daily with trimethoprim (2 mg/kg) for two years. She appeared to be unaffected by her renal problem, apart from having an E coli urinary tract infection at eight months.

By 2.5 years she had established a normal voiding pattern, and she also had continuous dribbling of urine during the day, and consistently damp nappies overnight. We suspected that the right upper moiety ureter drained ectopically, but examination of her external genitalia was normal, and a repeat ultrasound provided no new information. The ureter draining the right upper moiety was still dilated along its length, but the course of its insertion could not be visualised.

Because ectopic ureters are typically associated with dysplasia,5 the kidney tissue is likely to have a reduced capacity to concentrate urine.3 We collected urine serially, alternating between catching voided samples and collecting dribbled urine into a pad, to see if they were different. It took about 20 minutes to collect sufficient dribbled urine to sample each time, and the subsequent voided samples were simply collected when the patient was next ready to pass urine. The voided samples were yellower, but all were relatively dilute, and there were no consistent biochemical differences between them. Though collecting samples after an eight hour thirst produced a greater biochemical difference, it was not consistent. However, repeating the collection two hours after 10 µg intranasal desmopressin acetate revealed a clear difference between the urine types, especially in their creatinine concentrations and osmolalities (fig 1). Following this, she underwent cystoscopy and vaginoscopy, and a right upper pole haeminephrectomy; histology confirmed dysplasia. It was not possible to identify an ectopic ureteric opening, but her dribbling was cured immediately.

Comments
Confirmation of biochemical differences in serially collected alternate dribbled and voided urine is a simple non-invasive way of diagnosing an ectopic ureter in a girl. The use of desmopressin acetate is recommended to exaggerate the biochemical differences. Even when
Zinc deficiency

Zinc is an essential micronutrient, and zinc deficiency affects gene expression, protein synthesis, and immune function as well as taste and appetite. Studies in developing countries on the effect of zinc supplementation have given conflicting results. Now a study in rural Ethiopia (Melaku Umeta and colleagues Lancet 2000;355:2021–6) has shown that zinc supplementation improved growth in infants.

The study included 100 apparently healthy breastfed 6–12 month old infants who were stunted (length for age Z score <−2) and 100 infants matched for age and sex who were not stunted. They were randomly assigned to zinc sulphate 10 mg or placebo daily for six days a week for six months. Over the six months the mean growth in length of stunted infants was 7.0 cm with zinc and 2.8 cm with placebo. Growth of the non-stunted group was 6.6 cm (zinc) v 5.0 cm (placebo). The corresponding increases in weight were: stunted infants 1.73 kg (zinc) v 0.95 kg (placebo); non-stunted 1.19 kg v 1.02 kg. Zinc supplementation reduced the incidences of anorexia, fever, diarrhoea, vomiting, and cough in stunted infants. At the end of the study, serum and hair zinc concentrations were lower in non-supplemented stunted infants than in non-supplemented non-stunted infants (they were not measured at the beginning of the study) and zinc concentrations in supplemented stunted infants correlated with growth in length.

Zinc supplementation may improve growth in some children in developing countries. Whether the effect is a direct effect of zinc on growth or a result of improved appetite, fewer infections, and less diarrhoea is uncertain.

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