Correspondence

Neonatal urological ultrasound

Sir,

It is a cause for concern that I find the statement ‘all patients underwent renography and cystography, with anatomical localisation by intravenous urography and sometimes antegrade pyelography’, in a paper published in 1989.1 The obsessive desire for full assessment should not override management based on a primary division of patients into those requiring early intervention and those requiring careful follow up. For example, dilatation of one renal tract in a neonate may resolve spontaneously without surgery. There is no single imaging pathway that is correct for all neonates with urinary abnormalities. Prudence in the use of ionising radiation dictates the need for individual assessment by surgeon and radiologist followed by the use of investigations relevant to the individual problem.

R K LEVICK
Paediatric Radiology Department, Sheffield Children’s Hospital, Western Bank, Sheffield S10 2TH

Reference


Dr Clarke comments:

While we agree that not all cases with urinary tract dilatation at birth require early surgical intervention, we maintain that in order to be sure which cases are best managed conservatively, accurate diagnosis must first be made.

Regarding the example quoted by Dr Levick, we would certainly not advocate the full investigation of a neonate with a transiently dilated urinary tract. If the dilatation persists, however, the information provided by an ultrasound scan is insufficient to provide accurate diagnosis and plan appropriate management. This fact is confirmed by the results of our paper.

We do tailor our investigations to the individual child but it is inappropriate to dispense with definite imaging as this is the only way to make specific diagnosis. To settle for anything less is to adopt a policy of ‘best guess’. This is neither appropriate nor safe in current urological practice.

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Sir,

Clarke et al are quite correct to emphasise that ultrasound alone is rarely enough to initiate treatment of renal tract abnormalities detected antenatally.1 Their paper, however, does not define the precise role of ultrasound and underestimates its diagnostic capabilities in experienced hands. The role of antenatal ultrasound is twofold. Firstly to detect abnormalities incompatible with a normal life expectancy so that termination of pregnancy can be offered. A recent study has shown good specificity of ultrasound in this situation.2 Secondly to forewarn of structural renal abnormalities so that these can be investigated after birth. The specific cause for renal tract dilatation at this stage is not important.

Postnatally the situation is quite different. Ultrasound clearly shows pelvicaliceal and ureteric dilatation, bladder volume and wall thickness, the presence of diverticula and urertereceles, and dilatation of the posterior urethra in cases of posterior urethral valves. Dilatation of collecting systems does not necessarily infer obstruction and further imaging is necessary to exclude reflux or non-obstructed dilated systems. Pelviureteric junction obstruction often has a characteristic appearance on ultrasound, however, and it is surprising that Clarke et al found this abnormality to be accurately diagnosed in only 14 of 35 cases. We are...
Correspondence

not told what the ultrasound diagnoses in the other 21 was, presumably dilatation of unknown cause. Cystic renal dysplasia can be accurately diagnosed by ultrasound in most cases if the strict criteria of non-communicating cystic spaces, which vary in size and number, is adhered to. It is well recognised that in a few cases it may be difficult to differentiate from hydronephrosis but this is readily achieved either by cyst puncture or radionuclide scintigraphy. It is, however, good practice in all cases to perform a dimercaptosuccinic acid (DMSA) scintigram to confirm total absence of function.

The place of postnatal renal tract ultrasound is quite clear. It should act as a guide to the next examination, although in many cases the specific diagnosis will be apparent. Tudor and Whitaker have recently described a protocol for the detection and management of the dilated fetal urinary tract, which is to be commended.3 The ultrasound scans should be performed by individuals skilled in paediatric ultrasound. In a tertiary referral centre one would expect all the scans to be repeated by such an individual before the initiation of treatment or further investigation.

References


Gall stones in homozygous sickle cell disease

Sir,

We recently communicated detailed findings on the prevalence and risk factors for gall stones in children with homozygous sickle cell disease.1 The study has been repeated to include other sickle genotypes for which there are few data,2 and this has enabled comparison of gall stone prevalence between genotypes. It has also enabled us to examine the role of haemolytic rate and mean serum bilirubin concentration, which is the prime determinant of gall stone prevalence in homozygous sickle cell disease.1

The children who participated in the Jamaican cohort study of sickle cell disease were aged between 6 and 14 years at the date of the study. Gall stone prevalence was determined by real time ultrasound in a cross sectional study with informed consent in every case. Details of the number of children with each genotype, the total examined, and the prevalence of gall stones are given in the table. The mean serum total bilirubin concentration for each genotype was calculated from steady state values obtained at each child’s birthday after four years; this is because of the rapid change in haematological indices before this age.3

<table>
<thead>
<tr>
<th>Genotype</th>
<th>No of children in cohort study</th>
<th>No of children screened</th>
<th>No (%) with gall stones</th>
<th>Mean (range) serum bilirubin (μmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SS</td>
<td>242</td>
<td>218</td>
<td>30 (14)</td>
<td>35 (27–38)</td>
</tr>
<tr>
<td>SB⁺</td>
<td>11</td>
<td>9</td>
<td>2 (22)</td>
<td>27 (20–35)</td>
</tr>
<tr>
<td>SC</td>
<td>152</td>
<td>138</td>
<td>4 (3)</td>
<td>19 (16–22)</td>
</tr>
<tr>
<td>SB*</td>
<td>33</td>
<td>25</td>
<td>0</td>
<td>15 (12–17)</td>
</tr>
</tbody>
</table>

Gall stones were more common in homozygous sickle cell (SS) than sickle cell/haemoglobin C (SC) disease ($\chi^2=9.7, p<0.01$), but other differences in prevalence were not significant. Gall stone prevalence was unreliably estimated in sickle cellβ (SB) thalassaemia due to small patient numbers. Mean serum bilirubin concentrations varied by genotype in a similar manner to gall stone prevalence, and data from the cohort study have shown differences by genotype in major haemoglobin and reticuloocyte count consistent with variation in haemolytic rate.4 Our data indicate that haemolysis, mean bilirubin, and gall stone prevalence vary by sickle genotype, and that the risk of gall stones is low in children with SC disease and SB* thalassaemia.

References


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Haemophilus influenzae type b disease in the Oxford region

Sir,

Tudor-Williams et al report an incidence of systemic Haemophilus influenzae type b disease in children less than 5 years of age of 33-4/100 000.1 This was determined from a detailed laboratory based study in the Oxford region and they urge others to set up similar prospective studies.

I have examined Hospital Activity Analysis (HAA) data for all deaths and discharges for H influenzae meningitis (ICD Code 320-0) and acute epiglottitis (ICD Code 464-3) in the South Western region from 1976–86 and 1979–86 respectively. These two groups accounted for 80% of all

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