Paediatric Intensive Care Group/British Association for Paediatric Nephrology

G44   A PROSPECTIVE QUESTIONNAIRE ASSESSMENT OF KNOWLEDGE AND APPLICATION OF ESTIMATED GLOMERULAR FILTRATION RATES AMONGST PAEDIATRIC TRAINEES

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Aims To determine awareness of estimated glomerular filtration rates (eGFR) and its application in prescribing and managing paediatric patients.

Methods This was a cross-sectional, prospective, questionnaire based study. The questionnaires were distributed at the regional deanery teaching for Paediatric trainees at levels ST1–8 in Autumn 2012.

Results 40 questionnaires were completed, with a 50% response rate. Trainee groups were categorised as junior trainees, ST1–3 (n = 17), senior trainees, ST4–8 (n = 16) or trainees specifically with renal experience (n = 7). Almost a third of the total number of participants had never performed an eGFR, three-quarters of whom were junior trainees. Not surprisingly therefore, one-third of junior trainees had never adjusted medication in consideration of renal impairment. Likewise, three-quarters of trainees were unable to suggest a formula for calculating an eGFR. Of significance, from the 25% who were able to give a formula, 30% wrote it incorrectly.

In the clinical scenarios 80% correctly identified an abnormal creatinine but less than half realised the need to dose adjust acyclovir. In the second case 75% appropriately indicated the use of Gentamycin in renal impairment but there was no clear consensus on the dosing regime and when levels should be checked. Only 20% were aware of the 24-hour dosing schedule with almost half opting to look it up or discuss with the renal team. Overall those with more training (ST4–8) or with renal experience (ST1–8) were more knowledgeable.

Conclusions There is significant room for improvement in the knowledge and appropriate use of eGFR amongst Paediatric trainees, particularly at the junior level. An increasing awareness of these simple tests would improve prescribing practise; potentially preventing the inappropriate use of nephrotoxic drugs in those with renal impairment or conversely denying a patient gentamicin when it is the most suitable antibiotic and safe if used appropriately. We hope to establish simple regional guidelines through the local nephrology network, develop practical prescribing modules to improve prescribing in renal impairment and consequently improve clinical decision-making and reduce the burden on tertiary nephrology services.

G45   THE DIAGNOSIS OF URINARY TRACT INFECTION IN YOUNG CHILDREN (DUTY) STUDY: THE DEVELOPMENT OF A CLINICAL ALGORITHM TO IMPROVE THE RECOGNITION OF URINARY TRACT INFECTION (UTI) IN PRE-SCHOOL CHILDREN PRESENTING TO PRIMARY CARE

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Aim To develop a clinical algorithm based on symptoms, signs and urine dipstick results to assist the identification of children who require urine sampling, antibiotic treatment and/or laboratory analysis.

Methods We conducted a diagnostic cohort study of children <5 years presenting acutely (≤28 days) unwell to primary care in the UK. We collected detailed information on the presence/absence and severity of presenting symptoms and signs, as well as socio-demographic and past medical history data. Urine was sampled by clean catch (preferred) or nappy pad, 'dipsticked' and sent to (i) the local NHS laboratory (the priority sample) and (ii) a reference laboratory. Blind to children's clinical symptoms and signs, the NHS and reference laboratories processed urine samples according to their standard operating procedures.

Results (preliminary) 7,163 children were recruited with NHS and research urine sample results available for 6,328 (88%) and 5,257 (73%) respectively. Of the 5,017 children without missing data and with urine results from both laboratories: mean age was 2.2 years (s.d. = 1.4); 49% were male; 54% urines via clean catch, 45% via nappy pads and 1% via bag. UTI rates were 2.8% and 3% from clean catch and pad samples respectively. Among clean catch samples, the following were independently associated with UTI: history of UTI; parental report of smelly urine; pain/crying while passing urine; clinician's global impression of illness severity; and on dipstick: nitrites, leukocytes and blood (area under the ROC = 0.87 (95% CI 0.82 to 0.92). Among the nappy pad samples, the factors were: female gender; age; smelly urine; darker urine; and on dipstick: nitrites, leukocytes and blood (AUROC = 0.78 (0.72 to 0.83)).

Conclusions Symptoms, signs and dipstick testing have diagnostic utility for UTI. These results will be developed into an algorithm to help clinicians select which should have: a urine sample obtained; a sample sent for laboratory culture and receive immediate antibiotic treatment.

G46   DEVELOPING A HUMAN PROXIMAL TUBULAR CELL MODEL FOR CYSTINURIA

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Aims The inherited stone-forming condition cystinuria is most commonly caused by mutations in the SLC3A1 or SLC7A9 genes. These encode the two subunits, rBAT and $b^{0,+}AT$, of the heterodimeric cystine transporter, which is located on the apical membrane of proximal tubular cells (PTC). Defects in this transporter cause multiple, bilateral and recurrent cystine stones. Trafficking of the $rBAT/b^{0,+}AT$ transporter has been shown to be disrupted by mutations of SLC3A1 and SLC7A9 in other cell lines, but this has not yet been demonstrated in a relevant cell type. Our aim is to interrogate cystine transport in vitro to identify new therapeutic targets at the molecular level.

Methods PTC from urine of healthy patients were conditionally immortalised using a temperature sensitive SV40 construct. Fluorescent tags for wild type and mutated SLC3A1 and SLC7A9 sequences were generated, and used to study the trafficking of the $rBAT/b^{0,+}AT$ heterodimer, and quantify cystine uptake using radiolabelled cystine assays.

Results Constructs for wild type and mutated rBAT and $b^{0,+}AT$ were transfected into human conditionally immortalised PTC, and imaged in real time to demonstrate transporter trafficking. Functional assays of cystine transport are underway to quantify the effects of clinically relevant cystinuria mutations.