Title  
Acute kidney injury in the neonatal unit at risk of chronic kidney disease – under recognized and lack of follow-up

Presented by  
Claire McFaul

Background

Acute kidney injury (AKI) occurs in 40-70% of critically ill infants. It is independently associated with increased morbidity and mortality. Emerging evidence suggests those who survive AKI are at increased risk of chronic kidney disease (CKD). There is no nationally agreed framework for follow up of neonates with AKI. The Nottingham Children’s Hospital (NCH) has guidance on management and follow up of neonatal AKI.

Aim

The aim of the study was to retrospectively identify all neonates diagnosed with AKI and review the severity of AKI, follow up arrangements and prospectively review for evidence of CKD.

Methods

Electronic records were searched for all neonates who were diagnosed with “acute kidney injury” from the time of search (February 2020 to 2011). Data was collected including their lowest previous, peak and discharge creatinine. The Neonatal acute kidney guideline from NCH was used to categorize the severity of AKI and those who could have been followed up.

Results

67 infants were identified. 7 were excluded due to congenital disease. 41 infants survived and 19 infants died. Stage 1 n= 27, stage 2 n=4, stage 3 n = 3. For 5 infants there was insufficient information. 5 infants were referred for nephrology follow up. Following NCH guidance, 19 infants could have been referred for follow up. On prospective review 16/41 had a repeat creatinine. 29/41 had no documented BP. 38/41 had no documentation of urinalysis or Protein: Creatinine ratio. Where data was available, none showed evidence of CKD.

Conclusion

Limited data was available to comment on those who may have developed signs of CKD. However, the risk of CKD in infants who survive AKI has been under recognized in our department. A framework to identify and follow-up those at risk of CKD should be developed.
Background

Individuals with Down Syndrome are at increased risk of developing thyroid disease. Given thyroid disorders represent a preventable cause of neurodevelopmental impairment, early detection and treatment are essential to maximise cognitive abilities. This service evaluation sought to assess the efficacy of the Down Syndrome Hypothyroid Screening programme in its uptake and subsequent diagnosis of hypothyroidism.

Methods

A report of children with Down Syndrome was obtained from the Greater Glasgow and Clyde (GG&C) Down Syndrome database. Children were excluded if they were <2 years or had been a resident for <2 years. Electronic Patient Records were accessed for baseline characteristics and venous Thyroid Function Tests (TFTs). Data on TSH capillary screening were obtained from Scottish Newborn Bloodspot Screening. Data were collected on each child’s previous 3 screens and the time between screening was calculated. Children referred to Endocrinology following abnormal screening and those subsequently commenced on Levothyroxine therapy were identified.

Results

Of the 248 children with Down Syndrome, 228 were included within our study (114 male). The mean age was 9.9 years, range (2.1-22.7). 3 children had never been screened. Of those screened, 92% received screening within the last 1.0 years (207/225) and a further 3.1% (7/225) received screening within 1.5 years. 7 of the 225 children had been screened once (n=218). 74 children (33.9%) had ≤1.0 years between their previous screenings. A further 118 children (54.1%) had ≤1.5 years between each screen. 21 children (9.3%) had abnormal screening, with 20 referred to Endocrinology. Of the 21 children with abnormal screening, 16 (76.2%) were commenced on Levothyroxine therapy.

Conclusion

Within GG&C, the hypothyroid screening programme is effective in monitoring and detecting thyroid disease. The majority of children with Down Syndrome receive hypothyroid screening annually. Of those screened, 9.3% had abnormal screening, with 76.2% of these children commenced on Levothyroxine therapy.
Title 10 years outcome of Pulmonary Artery Band Balloon dilatation; Single surgical centre experience and literature review.

Presented by Precyilia Fernandes

Background

Pulmonary Artery banding (PAB) is an operation carried out for congenital heart defects, essentially to control increased pulmonary blood flow. This is performed to protect pulmonary vasculature from long term irreversible damage leading to pulmonary hypertension while awaiting complete repair or as destination therapy. There is limitation of data in literature regarding the outcomes of pulmonary artery balloon dilatation (dPAB).

Aim

Literature review and review the outcomes of balloon dilatation of pulmonary artery banding in our surgical centre over 10 years.

Methodology

We conducted extensive literature search in Medline, Embase, Web of Science, Google Scholar citation using mesh words pulmonary artery and balloon dilatation, de-bandung, balloon catheter to study the available outcomes on the procedure. We then conducted a 10 year retrospective observational analysis of patients who underwent dPAB in our surgical centre.

Results

In the literature search 14/32 studies were reviewed. 7 published case-reports demonstrated successful de-bandung with no or few complications and need for re-intervention. The largest case series of 33 patients was published in 2009 by Holmstrom et.al with 42 percutaneous de-bandung procedures. They reported good efficacy with 4.8% risk of procedure related complications. In our 10 years’ experience we performed 11 dPAB. 6 for muscular ventricular septal defects (VSD), 3 for perimembranous VSD and coarctation of aorta, 1 for unbalanced AVSD and 1 for TGA with subarterial VSD. 9/11 Balloon dilatations were successful with relief of PAB gradient to RV pressure less than two third systemic. In two procedures there was no change in gradient due to non-dilatable band. Overall we had 1/11 intraoperative and 1/11 post-operative complication and 9/11 successful PAB balloon dilatation over 5 years.

Conclusions

We report good efficacy of balloon dilation of pulmonary artery banding with low complication and need for reintervention. There is need multi-centre study for larger cohort of patient which is currently ongoing.
Title: An Investigation Of Androgen-responsive Non-coding RNAs In Boys With Atypical Genitalia Without Genetic Variants in the Androgen Receptor (AR)

Presented by: Malika Alimussina

Background

Introduction: Transcriptome analysis of peripheral blood mononuclear cells (PBMC) RNA has identified a set of androgen-responsive non-coding RNAs(1).

Aim

To quantify the androgen-responsive gene expression and investigate its relationship to the testosterone (T) rise following hCG stimulation in boys with no genetic evidence of androgen insensitivity.

Methods

Boys with suspected DSD who were evaluated in Glasgow from 2018 to 2021 were also included. Information on clinical, biochemical and genetic assessment was obtained from clinical records. PBMC RNA was collected before and after hCG stimulation of the testes on day 4(D4) and day 22(D22) and gene expression was quantified using QuantStudioTM 3D Digital PCR.

Results

Twelve XY boys with atypical genitalia, a median age of 0.8 yrs (0.5,3.4) and no detected AR gene variants, were included. The median baseline and peak T was 0.5 nmol/l(0.5,6.8) and 20.6 nmol/l(1.2,42.1), respectively. Within this group, there was one patient who did not show a T response to hCG at all on D4 and a minimal response on D22 (1.2nmol/l). The median fold change in SNORD5 and RNY5 on D4 in this patient was 0.09 and 0.05, respectively. The median fold change for the two genes on D22 was 0.14 and 0.04, respectively. In the rest of the cohort, the median post-hCG T on D4 and D22 was 15.3 nmol/l(2.5,42.1) and 24.3 nmol/l(17.4,37.3), respectively. In this group, the median fold change in SNORD5 expression on D4 and D22 was 2.7(0.25,13.9) and 2.0(0.1,5.6), respectively. The median fold change in RNY5 expression on D4 and D22 was 0.95(0.8,37.6) and 1.2(0.2,7.7), respectively.

Conclusions

Expression levels of RNY5 and SNORD5 can be quantified accurately and show androgen dependency. Further research in genetically confirmed cases of androgen insensitivity plus those with no response to hCG stimulation is required to determine the diagnostic role of non-coding RNAs in XY DSD.
Title What makes children thin? Behavioral correlates of wasting: a pilot study with children attending a tertiary feeding clinic

Presented by Eunice Nortey

Background

It is not clear whether tube fed children or children with severe food refusal regulate energy intake in the same way as healthy children.

Objective

To describe energy compensation patterns in children with severe feeding problems compared to healthy children. METHOD: A cross-over experimental study was carried out among thin children (BMI <9th centile) attending the Glasgow feeding clinic. A standardized methodology was used to assess the ability to compensate for a drink taken before a meal. Caregiver-child dyads came twice to the University metabolic laboratory. Children were randomly assigned to two conditions: high energy preload drink supplying 10% of daily energy requirements or low energy supplying 2kcal/100ml, after 2+ hours fast. After 30 minutes they were served identical ad libitum lunches. Calories consumed from preload and lunch meals were calculated and the proportion of the preload compensated (COMPX) was calculated their parent completed the ICFET eating behaviour questionnaire.

Results

Ten children (mean age=57 ±17 months) were studied, of whom 3 were currently and 6 previously tube fed, mean (SD) heightZ -1.63 (1.03) and BMIz -2.15 (1.18). All but one child ate less after the high energy preload. Overall energy intake was similar for both preload conditions (Low = 248 ±61 kcal, high= 259±75 kcal). The mean compensation COMPX was 93.3 ± 49% with 4 children over-compensating (>100%) and 1 child having no compensation (<1%). Their mean Avidity Z score was low (-1.5; -3.3 to -0.1) and their refusal score above average (0.6 (-2.0 to 3.6); COMPX was not significantly related to avidity or refusal.

Conclusion

The mean COMPX for this high-risk group of children was higher than the published 50-70% COMPX for healthy children and 4/10 had their appetite excessively suppressed by a high energy drink. This may explain the difficulty they faced in stopping tube feeding.
Introduction

Elevated serum lactate during cardiopulmonary bypass (CPB) results from inadequate tissue oxygenation and is associated with increased postoperative morbidity and mortality. The aim of this study was to identify patient and CPB factors that predict hyperlactaemia in children undergoing cardiac surgery using cardiopulmonary bypass.

Methods

Retrospective data of 160 consecutive paediatric patients [M:F 3:2, Age(years)=4.2 +/-4.7] was collected from the CPB record. Hyperlactaemia during CPB was defined as lactate >3 mmol/l and/or an increase of >1mmol/l. Demographic and CPB variables were analysed for their prediction of hyperlactemia using a multi-variate linear model.

Results

Hyperlactemia occurred in 39 patients (24%). Gender, age, weight or BSA was not associated with elevated lactate. Significant predictors of increased lactate included: increased glucose during CPB (r= 0.52, p= <0.001), longer duration of CPB (r= 0.23, p= 0.0016), reduced heart lung machine (HLM) generated blood flow rates, as a % of BSA predicted flow (r= -0.16, p= 0.032) and lower base excess (r= -0.18, p= 0.039). Elevated baseline lactate, prior to CPB, also predicted intra operative Lactates of >3 mmol/l (r= 0.74, p= <0.001).

Conclusion

This study identified factors associated with increasing lactate during CPB relate primarily to longer duration of CPB, lower % predicted flow rate delivered during bypass and concurrent markers of the patient stress response (glucose + base excess). Because CPB duration relates to the surgical procedure it is therefore a non-modifiable risk factor. However, recalibrating flow rates to increased % predicted flow, employing techniques to aid in flow delivery and control of the stress response may reduce lactate production by enhancing tissue oxygenation.