Quantifying Paediatric High Dependency Care: Does the Paediatric Critical Care Minimum Dataset Accurately Capture Workload?

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Aims The structure of paediatric critical care (PCC) in the UK is under review. This study aimed to evaluate the paediatric high dependency unit (PHDU) workload at our district general hospital, mapping it to the Paediatric Critical Care Minimum Dataset (PCCMDS).

Methods The demographics, diagnostic category, interventions required and outcomes of all PHDU admissions were recorded prospectively over a 4 month period (winter 2016–2017). Demographic data was compared to previous local PHDU audits and the intensity of interventions each patient received was assessed using the PCCMDS.

Results 105 patients were admitted with a mean age of 4.9 years (median 2.6 years, range 12 days–16 years 3 months). Age distribution was similar to previous years but number of admissions has greatly increased (n=34 in 2000–2001 vs 55% in 2016–2007). Mean length of stay was 1.6 days (range 3 hours–12 days). 90 patients were discharged to the ward or home and 9 transferred to a tertiary centre (6 to paediatric intensive care, 1 to PHDU, 2 to wards). Outcome data was not recorded for 6. Regarding intensity of care: 66 patients received PCCMDS basic interventions; with 2 further patients meeting the suggested new criteria. 11 patients had intermediate interventions (8 also had basic interventions). 34 patients received no PCCMDS interventions. These were mainly neurology (n=13) and poisoning (n=9) admissions. 3 of this sub-group received volume resuscitation and 4 were transferred to a tertiary centre.

Conclusions Paediatric high dependency workload is increasing, particularly respiratory admissions. The PCCMDS improves understanding of the PHDU workload and will enable comparison of work between units. However 32% of patients admitted to our PHDU received no PCCMDS interventions prompting review of our PHDU admission criteria and highlighting that some interventions are not recognised by the PCCMDS (including volume resuscitation and observation after prolonged seizures) leading to potential underestimation of workload. We recommend further modification of the PCCMDS to account for this.

Reference

Bulimia Nervosa – The Silent Killer – A Case Report From Paediatric Intensive Care

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A 14 year old boy was admitted to the paediatric intensive care unit following an out of hospital cardiac arrest. He was found to be in Ventricular Tachycardia by the ambulance service and one DC shock was administered at the scene and cardioversion achieved.

On arrival to hospital a capillary blood gas revealed a severe hypochloraemic hypokalaemic metabolic acidosis. PH 7.89, PCO2 2.4, Na+126, K+1.5, Cl82. BE +ve 18. Bloods on admission showed an acute kidney injury creatinine 129, urea 8.0. The patient was gradually hydrated and electrolyte disturbance was corrected over 48 hours.

An ECG on arrival to accident and emergency showed a prolonged QTc of 675 ms. An echocardiogram confirmed that the heart was structurally normal. The patient was unresponsive on admission to hospital (GCS 4) he was therefore intubated and ventilated and transferred to PICU.

The differential diagnosis in this case was broad and included Bartters syndrome, Gitelmann syndrome, gastric outlet obstruction, HNF-1 beta, renal, metabolic and endocrine causes. The patient was extubated twenty-four hours following admission. He was assessed by the neurology team and he was found to have no residual neurological deficit. On waking the patient disclosed that he had struggled with body image and perceptions about his weight for the last 3 years. His enteral intake had been good but he would induce vomiting after every meal. He denied laxative use but admitted to drinking highly caffeinated drinks to keep his energy levels up. The combination of severe hypokalaemia <1 and high caffeine intake may have lead to Torsades de pointes and ultimately cardiac arrest.

A history of chronic vomiting fits with the profound hypochloraemic hypokalaemic metabolic acidosis observed on admission. Metabolic and genetic investigations were normal. Interestingly the blood gas taken in accident and emergency was taken thirty minutes after a return of spontaneous circulation had been achieved. Therefore the patients PH and potassium levels may have been higher and lower respectively at the time of cardiac arrest. Theoretically the patient will have started to correct his alkalosis and profound hypokalaemia as cardiac arrest increases serum potassium levels and induces acidemia.

Eating disorders in male paediatric population is an increasing problem with associated high morbidity, mortality and poor prognosis. This case highlights the importance of early intervention and support for affected individuals.