

(5.1–61). Serum lactate median was 2.1 (0.1–11.2). Neurological findings were determined in 22.4% of the patients. Normobaric oxygen treatment was given to 75.5% of the patients, while 24.5% were given hyperbaric oxygen treatment. A positive correlation was determined between Lactate and COHb levels ($P=0.01$, $r=0.228$). A negative correlation was determined between Glasgow Coma Scale and COHb levels ($p=0.01$, $r=-0.383$). Patients with neurological findings had an average COHb level at $35.7\% \pm 16$, patients without neurological findings had an average COHb level at $22.5\% \pm 7.7$ ($p=0.001$). Blood lactate level was higher in patients with neurological findings than patients without neurological findings ($p=0.01$).

Results COHb level in carbon monoxide poisoning cases is an important indicator for the existence of neurological findings. Since increase in lactate is correlated with the poisoning level and neurological findings, high lactate levels should be taken into consideration.

370 A RANDOMISED TRIAL OF DEXAMETHASONE VERSUS PREDNISOLONE IN THE TREATMENT OF ACUTE PAEDIATRIC ASTHMA EXACERBATIONS

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^{1,2}J Cronin, ^{1,2}S McCoy, ²S Nally, ³U Kennedy, ²G Crispino-O'Connell, ¹S Walsh, ^{1,2,4}R O'Sullivan. ¹Paediatric Emergency Research Unit, Our Lady's Children's Hospital, Crumlin; ²National Children's Research Centre, Our Lady's Children's Hospital Crumlin; ³Department of Emergency Medicine, St James's Hospital; ⁴Department of Paediatrics, University College Dublin, Dublin, Ireland

Introduction The aim of this open-label trial is to examine whether a single dose of dexamethasone is non-inferior to a 3-day course of prednisolone in the treatment of exacerbations of asthma in children who attend the Emergency Department (ED).

Methods Participants were identified on ED presentation, eligibility was confirmed and informed consent was obtained. Patients were randomised to receive either a stat dose of oral dexamethasone 0.3mg/kg (max 12mg) or prednisolone 1mg/kg/day (max 40mg) for 3 days. Otherwise standard treatment was administered.

Following discharge from the ED, patients were clinically reviewed on Day 4, and by telephone on Day 14. The primary outcome measure was the Pediatric Respiratory Assessment Measure (PRAM score) at Day 4. Secondary measures include relapse rate, requirement for more steroids, number of salbutamol therapies, and vomiting.

A sample size of 210 subjects will be sufficient to reject the null hypothesis - that the population means of both groups are equal with a probability of 0.9. The Type I error probability is 0.05.

Results 201 individual asthma exacerbations (101 prednisolone, 100 dexamethasone) have so far been enrolled. Demographic details and exacerbation severity are equal across both groups. We will complete enrolment in May 2012.

Conclusion The results of this randomised trial may have a significant impact on the management of acute asthma in children. At current rates we will complete recruitment in May 2012 and will present full results at the conference.

371 RARE PRESENTATION OF ACUTE DEMYELINATING ENCEPHALOMYELITIS IN A CHILD WITH EXCESSIVE FATIGUE AND SLEEPINESS

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A Silwal, A Morris. Calderdale and Huddersfield NHS Trust, Leeds, UK

The encephalopathic child is a paediatric emergency and presents a considerable challenge.

We present a 4 year old boy who was admitted with 3 week history of excessive fatigue and sleepiness, waking for only 4–5 hours a

day. He had occasional episodes of vacant stares and "wanted to go home", when was at home.

He had gastroenteritis after exposure to snails at nursery and was treated for otitis media by general practitioner. There was no history of fever or foreign travel.

He was sleepy with normal neurology and an aphthous ulcer over lower lip. He was managed as encephalitis and treated with acyclovir, ceftriaxone and clarithromycin. He had neutrophilia with normal lumbar puncture and viral PCR's.

After a week of admission, he deteriorated with generalized pain, bedwetting, ataxia and upper motor neuron signs. MRI scan showed asymmetric, bilateral white matter changes suggestive of acute demyelinating encephalomyelitis (ADEM) or other viral encephalitis.

Repeat lumbar puncture showed pleocytosis with neutrophilia. Viral PCR's were negative. ASOT was raised. MRI with contrast showed 4 mm high signal focus behind C2 body and C2–3 disk.

After completing a 10 day course of acyclovir, he was started on methyl prednisolone for ADEM and drastically improved. He was discharged after 3–4 days of the treatment with follow up.

This case initiated a lot of discussions- presence of fever to diagnose encephalitis? When to start and stop acyclovir in suspected encephalitis with negative viral PCR's? To start steroids or not - why the delay?

372 ACTIVATED CHARCOAL FOR GHB INTOXICATION IN CHILDREN: AN IN VITRO STUDY

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¹RW Neijzen, ²MA Sikma, ¹AC Egas, ¹FFT Ververs, ¹EM van Maarseveen. ¹Department of Pharmacy; ²Intensive Care, University Medical Centre Utrecht, Utrecht, The Netherlands

Background and Aims Treatment of intentional (mainly by adolescents) and unintentional (mainly by children) intoxications with the increasingly popular drug gamma-hydroxybutyrate (GHB) primarily consists of symptomatic treatment. The usually rapid absorption and the need for intubation argue against activated charcoal (AC) treatment in GHB intoxications. However, the use of AC has been suggested in several guidelines and in literature, but it has never been demonstrated to what extent GHB binds (adsorbs) to AC. Reduction of GHB absorption by AC administration could be clinically relevant, especially in children. Therefore, binding of GHB to AC in an in vitro model was tested.

Methods Different quantities (2.5, 5, 7.5, or 10 grams) of AC were mixed with a dose of 800 mg GHB at 37°C in 100 mL simulated gastric (pH 1.2) or intestinal (pH 7.2) fluid, respectively. Subsequently, after 15 minutes of incubation the AC was separated from the liquid by centrifugation and the remaining GHB quantified by gas chromatography. GHB binding to AC was plotted in a binding curve.

Results Binding of GHB to AC was dose-dependent. At gastric pH, binding was higher than at intestinal pH, with a maximum binding of 84.3% and 23.3%, respectively, with 10 grams of AC, corresponding with a high adult dose.

Conclusions AC has GHB binding capacity, which is pH dependent. In case of (un)intentional intake of GHB by children, rapid treatment with AC may be considered.

373 CHARACTERISTICS AND COMPLICATIONS IN ORAL CAUSTIC INGESTION IN CHILDREN

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¹M Rafee, ²M Shoaran, ³F Javadzadeh. ¹Pediatric Gastroenterology, Tabriz University of Medical Sciences, Liver & Gastrointestinal Research Center; ²Pediatric, Tabriz University of Medical Sciences; ³Dentistry, Tabriz University of Medical Sciences, Aras International University, Tabriz, Iran