features of sepsis. Diagnosing DKA in this age group is crucial as any delay in initiating right treatment may result in significant morbidity and mortality.

P77 REYE-LIKE SYNDROME AND SEVERE CARNITINE DEFICIENCY IN A BOY WITH AUTISM SPECTRUM DISORDER AND RESTRICTED DIET

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Case Description A 9-year old boy presented to the Emergency Department with an acute history of vomiting, ataxia and reduced consciousness in the setting of a respiratory illness. In the two months prior he had intermittent episodes of vomiting, without headaches or visual disturbance.

On examination, he was encephalopathic (GCS 9/15), with marked hepatomegaly. Height and weight were on the 0.4th-2nd percentile and 9th percentiles respectively.

He had a background of high-functioning autism spectrum disorder (ASD) and a long history (since aged 18 months) of rigid eating behaviour, with a diet restricted to corn-based crisps ('Rancheros'), rich-tea biscuits (2-3/day), French-fries and Coca-Cola (10% glucose, drinking 1 L/day). He had recently commenced a multivitamin preparation.

Blood-work showed anaemia, elevated hepatic transaminases and creatinine kinase (6000U/l), low albumin, hypoglycaemia (2.7 mmol/l) and deranged electrolytes. CT brain showed moderate, communicating hydrocephalus without pressure effects. An extra-ventricular drain was inserted but later removed as this was deemed to be a coincidental finding of arrested congenital hydrocephalus.

Management and Investigation He required intubation and ventilation for respiratory compromise (RSV infection). Given the Reye-like scenario and abnormal biochemistry, a metabolic disorder was suspected. Acylcarnitine profile showed undetectable free carnitine and amino acid levels were low, as were vitamin levels. He received carnitine supplementation and very cautious nasogastric feeding; biochemistry and neurological status improved. Extensive investigations including carnitine uptake studies did not reveal an inherited metabolic disorder.

Detailed nutritional analysis of the patient's diet revealed very low daily protein (0.37 g/kg/day) and fat (0.77 g/kg/day) but with normal total daily caloric intake of 1200kCal, indicative of protein-energy-malnutrition (Kwashiorkor). He received intensive inpatient dietetic and psychological input and by discharge from hospital he was consuming an expanded range of foodstuffs. Carnitine is synthesised from essential amino acids lysine and methionine, but primarily it is obtained directly from protein in the diet. Cereal-based diets such as above are particularly low in the amino acids methionine and lysine.

Diagnosis and Follow-up On follow-up 2 years later, carnitine levels remain normal without supplementation. Protein intake is normal and diet is varied. Vertical growth has improved significantly with height now on the 91st centile and hepatomegaly has resolved. The final diagnosis was dietary protein-energy-malnutrition with profound secondary carnitine deficiency resulting in Reye-like syndrome.

This case reminds us that severe malnutrition exists in the developed world, and emphasises the need for detailed nutritional assessment of children on self-restricted diets, especially those with ASD.

P78 AN UNUSUAL CAUSE OF AN ACUTE CONFUSIONAL STATE IN AN ADOLESCENT; TRANSIENT GLOBAL AMNESIA

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Background Transient global amnesia (TGA) is a syndrome that has been known to occur predominantly within the adult population. It's an entity characterised by sudden loss of memory with confusion that lasts less than 24 hours. The event itself is never associated with focal neurological findings and the patient remains conscious throughout the event. Some of the precipitants documented within the literature have included swimming, immersion in cold water, extreme physical exertion or emotional stress. There has been much debate to date over the etiology/pathogenesis of TGA, including migraine, epilepsy, emboli, ischaemia of hippocampal regions or even venous congestion have all been suggested causes. While TGA is more prevalent amongst those >60 years of age, there has been very few instances of TGA in childhood.

Aim To describe a case of transient global amnesia in a teenage boy.

Methods We report the clinical presentation, results of investigations and outcome to date in a fourteen year old boy who presented in an acute confusional state, the ultimate cause for which was transient global amnesia.

Results A 14 year old boy presented to the Paediatric Emergency Department (PED) for evaluation of acute confusion following a hurling match. A previously well, neurodevelopmentally appropriate child, he had no significant family history nor did he have any regular medications or allergies.

Being an active sportsman, he had just participated in an intense hurling match that had lasted almost two hours in exceptionally cold weather. On cessation of the game, he had approached his coach disoriented and claiming 'I do not know where I am or why I am here'.

He continued to ask bizarre repetitive questions, and had no memory of the match for the following 12 hours. His neurological exam was normal, with no loss of consciousness or head injury. Basic blood work, toxicology screen, CXR and ECG were normal. Twenty fours later he had completely recovered. CT brain, MR brain with angiography and EEG were normal.

At follow-up six weeks later, he had remained perfectly well with normal neurological assessment.

Conclusion Childhood transient global amnesia is rare. Recurrence has been reported in those with a history of recurrent migraine. Our case is interesting in that it deviates from such reports whereby this episode seems solely provoked by Val-salva-like activity (extreme exertion)

P79 KAWASAKI DISEASE

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Introduction Kawasaki Disease (KD) is a relatively rare disease with a reported current, annual incidence in Europe of about 5-10/100,000 children younger than 5 years¹. The highest