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P439 -Lessons from hyponatraemia management in a paediatric transplant recipient with extra-pontine myelinolysis and neurological sequelae.

Dr Victoria Harkins[1], Dr Gillian Cassels[1], Dr Sarah Abernethy[1], Dr David Hughes[1], Dr Deepa Athavale[1]
[1]*NHS Greater Glasgow and Clyde, Glasgow, United Kingdom*

Hyponatremia correction and management in extra-pontine myelinolysis (EPM) is rare in paediatric practice. We report our management of a case in a seven year old, renal transplant patient who presented with severe hyponatraemia, associated influenza A and subsequently developed significant neurological sequelae secondary to extra-pontine myelinolysis.

Following a history of vomiting and diarrhoea, a seven year old, known renal transplant patient with history of polyuria, presented with disorientation. Previous background included a history of hyporeninaemic hypoaldosteronism as well as episodes of severe hyponatremia (109 and 110mmol/l), occurring pre-transplant in the presence of impaired renal function. Both episodes resulted in complete recovery with rapid hyponatremia correction without any adverse affect. Prior to this presentation, his daily fluid target of 2.8 litres had been achieved by additional water administration via his gastrostomy. Serum sodium was 108 mmol/l and on presentation he was clinically dehydrated. He received fluid resuscitation and correction of hyponatremia was initiated to a rise of 131mmol/l the following day. Neurological symptoms initially improved, in addition to a confirmed diagnosis of Influenza A with serum sodium normalising to 136mmol/l. However, further neurological deterioration occurred with neuro-imaging consistent with basal ganglia involvement and extra-pontine myelinolysis. A clinical decision was undertaken to gradually re-lower the sodium to 128mmol over the next 24 hours as seizure activity, dystonia and speech problems developed. After lowering, sodium levels once again were gradually normalised and a remarkable neurological recovery ensued over the course of the following year.

Hyponatremia management remains challenging with risks of significant neurological impairment. Despite tolerating previous severe hyponatremia episodes with relatively rapid correction, neurological symptoms ensued in this presentation suggesting perhaps previous episodes (pre-transplant) with concurrent uraemia, supported in literature animal models, may have been protective. Presence of Influenza A is likely to have played a multifactorial element to symptomology. Gradual re-lowering of sodium was challenging but with minimisation of neurological sequaelae adds weight to limited literature evidence available suggesting this is a protective management strategy.

Lessons learnt include slow, gradual raising of serum sodium, after initial resuscitation measures, in presence of symptomatic hyponatremia may be more beneficial and in presence of ongoing neurology suggestive of EPM, gradual re-lowering is a strategy to employ.