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Paracetamol overdose complicated by PRES – A case report

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Objectives: To describe a case of paracetamol ingestion in a patient who developed hepatorenal toxicity complicated by posterior reversible encephalopathy syndrome (PRES).

Case report: A 21 year old female presented to the emergency department (ED) after having ingested 40 g of immediate release paracetamol 8 hours prior to hospital transfer. She had a past medical history of depression and bipolar disorder including previous paracetamol overdoses requiring N-acetyl-cysteine (NAC) treatment. On examination, she was alert and afebrile. Her heart rate was 78 beats per minute with a blood pressure (BP) of 102/66 mmHg. Investigations on arrival revealed a paracetamol concentration of 261 mg/L with an ALT of 73 U/L. Her initial renal function and coagulation studies were all within normal limits. She was treated with NAC infusions but went on to develop hepatotoxicity (peak ALT of 13,000 U/L and peak INR of 3.1). The renal function began to deteriorate on day 3, finally peaking around day 11 (Creatinine 1030 umol/L and eGFR of 4). She was managed conservatively by way of intravenous fluids in the absence of dialysis. Unfortunately, this coincided with the patient developing hypertension (BP 150/100 mmHg) and new-onset seizures which resolved spontaneously. She was commenced on amlodipine and levetiracetam. A CT scan of her brain revealed bilateral parieto-occipital hypo-attenuation changes. A subsequent MRI of the brain revealed patchy cortical & subcortical white matter changes consistent with PRES. Despite her complicated hospital stay, she was eventually discharged on day 25 with a Creatinine of 161 umol/L (eGFR 40). Her discharge medications included amlodipine 5 mg daily and levetiracetam 250 mg twice daily. A follow-up MRI one month later revealed complete resolution of her radiological PRES features.

Conclusion: PRES is commonly associated with eclampsia, hypertensive encephalopathy, renal failure and immunosuppressive treatments. Paracetamol is known to cause acute liver and kidney injuries. However, there remains a paucity of literature implicating paracetamol to PRES. A case series of 15 patients in 1996 reported 1 patient who developed paracetamol-associated PRES. The present case highlights the importance of considering a diagnosis of PRES when treating patients with paracetamol overdose who develop hepatorenal toxicity.