disorders in AYAs are lacking. This is an area that warrants further investigation.

I believe that there is enough evidence to support education in AYAs about sleep hygiene and the potential effects of smartphone use at bedtime and at night and I applaud the author's advice that good sleep hygiene would 'include not having these devices within the bedroom'. ■

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Endocrine abnormalities in lithium toxicity

Editor – Shanks *et al*[1] describe salient physiological lessons from their patient with severe sequelae of lithium toxicity. I wish to suggest further lessons that may be learned from their report to minimise harm in future similar cases. It appears that nephrogenic diabetes insipidus (DI) was not considered until day 10 of the admission. That sodium remained elevated despite fluid resuscitation clearly implicates impaired renal salt handling and the patient's chronic lithium treatment was known. It is pertinent that 0.9% sodium chloride failed to correct the hypernatraemia from the start and so earlier suspicion of DI may have helped. There is no reason to stick doggedly to saline infusions to treat hypercalcaemia and 5% dextrose may have been the preferable resuscitation fluid.

One may justly wonder if the patient would have benefited from earlier ITU admission. Serum sodium was 172 mmol/L at day 4 – a severe medical emergency especially in a young patient – yet she was not transferred to ITU until almost a week later on day 10. This case must surely demonstrate that close monitoring and chasing of fluid and electrolyte goals is very hard on Level 1 wards. I would suggest that the refractory severe hypocalcaemia seen in this case was a known complication of unnecessary bisphosphonate treatment, as calcium will typically drop to safe levels (<3 mmol/L) with sufficient fluids alone. It would be pertinent to know how much improvement in fluid balance and what correction in serum calcium was achieved before the IV bisphosphonate was administered. Finally, with regards the patient's neurological disorder, did the authors consider the possibility of thyrotoxic periodic paralysis? ■

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Regular and frequent feedback of specific clinical criteria delivers a sustained improvement in the management of diabetic ketoacidosis

Editor – Notwithstanding the statement made by the authors of this paper that 'Fluid replacement is the most important initial management [in diabetic ketoacidosis]',[1] the caveat is that intravenous fluid (IVF) replacement is contraindicated when pulmonary oedema is present on admission in a patient with diabetic ketoacidosis (DKA).[2,3] In some of these cases, advanced chronic renal failure is an associated feature.[2] Pulmonary oedema may also be a feature when non-ketotic hyperglycaemia occurs in a patient with chronic renal failure managed by haemodialysis.[4] Both in the context of DKA[2] and non-ketotic hyperglycaemia,[4] one of the underlying causes of pulmonary oedema is the osmotic shift of fluid from the intracellular to the extracellular fluid compartment as a consequence of severe hyperglycaemia. This may overwhelm the pulmonary circulation when there is impaired excretion of that sudden additional extracellular fluid load. In some of these patients the sole use of insulin to correct hyperglycaemia may be instrumental in the resolution of pulmonary oedema.[2,4]

Pulmonary oedema may also be present on admission in a DKA patient with coexisting congestive heart failure.[3] In that context IVF replacement can be withheld, and DKA can be managed solely with intravenous insulin infusion.[3] The associated pulmonary oedema resolves after intravenous administration of frusemide.[3] Also in the context of cardiogenic pulmonary oedema a potential alternative treatment strategy is the use of intravenous nitrate infusion,[5] the latter a well-tried strategy in the management of pulmonary oedema complicating myocardial infarction.[6] The advantage of the latter strategy is that, in a DKA patient concurrently managed with an insulin infusion, hypokalaemia is less likely to be an outcome than might be the case when diuretics are coprescribed with insulin infusion. ■

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Response

We thank Mr Jolobe for his comments. It raises an interesting question relating to the fluid management of patients in DKA who are at an increased risk of overload (those with heart failure, renal failure or liver failure). The Joint British Diabetes Society guideline[1] does not suggest any alternative fluid management strategies for patients with end organ damage. To our knowledge, there has not been any work done to assess the management of DKA specifically in these patient cohorts to see if clinicians opted for personalised fluid strategies for these patients and if so, whether this had an impact on prognosis.

To explore this further, on our original dataset [2] we conducted a subgroup analysis to assess for any differences in the management of fluids of patients with end organ failure, which was presented at the 2017 Diabetes UK conference.[3] Using blood test results and clinical documentation we identified four groups:

- patients without established organ damage (ie those not at an obvious risk of fluid overload) (n=197)
- patients with heart failure (n=8)
- patients with liver failure (n=11)
- patients with renal failure (n=39).

In comparison, median values of appropriateness of fluid management in comparison to the JBDS guidelines were 100%, 90%, 80% and 80% in the four groups respectively. Using unequal t-tests these values were not significantly different to one another, suggesting there was not actually variation in the practice of early fluid prescription during the management of DKA. Although limitations persist in terms of case identification and sample size, this is an attempt to assess whether clinicians are considering the risk of fluid overload in these high-risk patients. We certainly believe further research should be done to explore the appropriate management of fluid in patients with end organ damage. ■

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