

Beer Potomania Masquerading as Adrenal Insufficiency

L Hopkins, V Stokes, S Chatterjee

Endocrinology Department, Stoke Mandeville Hospital, Aylesbury, Buckinghamshire, UK HP21 8AL

Abstract

A 71 year old male ex-pubican presented to the Medical Emergency Unit suffering from lethargy, weight loss, dizziness on standing and dyspnoea on exertion. He had a past medical history of hypertension, ischaemic heart disease and alcoholic liver disease and he admitted to drinking 100 units of beer per week. His antihypertensive medications included lisinopril and hydrochlorothiazide. On examination BMI was 35kg/m², blood pressure 85/65mmHg and there was no buccal or palmar hyperpigmentation. Admission venous blood tests revealed glucose 2.8mmol/l and sodium 113mmol/l. This led to a working diagnosis of Addison's disease. An MRI head, short synacthen test, ACTH and other baseline pituitary function tests were organised. MRI head revealed a structurally normal pituitary gland. The short synacthen test showed a low baseline cortisol of 109nmol/l which rose to 510nmol/l after 30min. ACTH was 14ng/l. All other tests were normal. Lisinopril and hydrochlorothiazide were stopped and he was commenced on intravenous hydrocortisone. This led to resolution of his hypotension and hyponatraemia. He was given a diagnosis of possible ACTH deficiency and discharged on replacement dose hydrocortisone to be followed up in the endocrine clinic. A repeat short synacthen test 3 months later showed a normal baseline cortisol of 513nmol/l rising to 588 nmol/l after 30min. Hydrocortisone therapy was stopped and hyponatraemia improved with changes to his antihypertensives and a reduced alcohol intake. The cause of the patient's initial presentation was a combination of beer potomania and thiazide diuretic use. Beer potomania is an under-recognised condition characterised by hyponatraemia secondary to water intoxication. Recognition of this condition and careful fluid administration are required in the initial phase to avoid serious complications such as central pontine myelinolysis. Our case demonstrates that other causes of euvoalaemic hypotonic hyponatraemia may obscure the actual diagnosis of beer potomania resulting in incorrect management.

Presentation

A 71 year old male ex-pubican presented with weight loss, lethargy, dizziness on standing and dyspnoea on exertion. He had a past medical history of hypertension, ischaemic heart disease and alcoholic liver disease. He was on medications for these including lisinopril, hydrochlorothiazide, thiamine and vitamin B supplements.

He admitted to drinking 6 pints of beer a day (approximately 100 units a week) and lived alone after his wife died 10 years ago. Following retirement his social life revolved around the pub and the friends he had made there.

On examination he was obese with a BMI of 35kg/m² and hypotensive with a blood pressure of 85/65mmHg. There was no buccal or palmar hyperpigmentation. Examination was otherwise unremarkable.

Patient's Blood Results on Presentation

Blood Test	Result	Normal Values
Haemoglobin	15.0 g/dL	13-18.1 g/dL
White Blood Count	12.5 x 10 ⁹ /L	3.7-11x 10 ⁹ /L
Sodium	113mmol/L	135-145mmol/L
Potassium	6.3mmol/L	3.5-5.1mmol/L
Urea	13.2mmol/L	3.2-7.4mmol/L
Creatinine	175µmol/L	63-115µmol/L
Albumin	44g/L	35-50g/L
Alanine Transferase	58IU/L	10-35IU/L
Alkaline Phosphatase	94IU/L	40-150IU/L
Bilirubin	21µmol/L	3.4-20.5µmol/L
C-reactive Peptide	5mg/L	0-5mg/L
Random Glucose	2.8mmol/L	3-7.8mmol/L
Random Cortisol	555 nmol/L	101-536 nmol/L (Values for 7-9am)

With the symptomatic low sodium and low random cortisol a working diagnosis of Adrenal Insufficiency was made.

Investigations

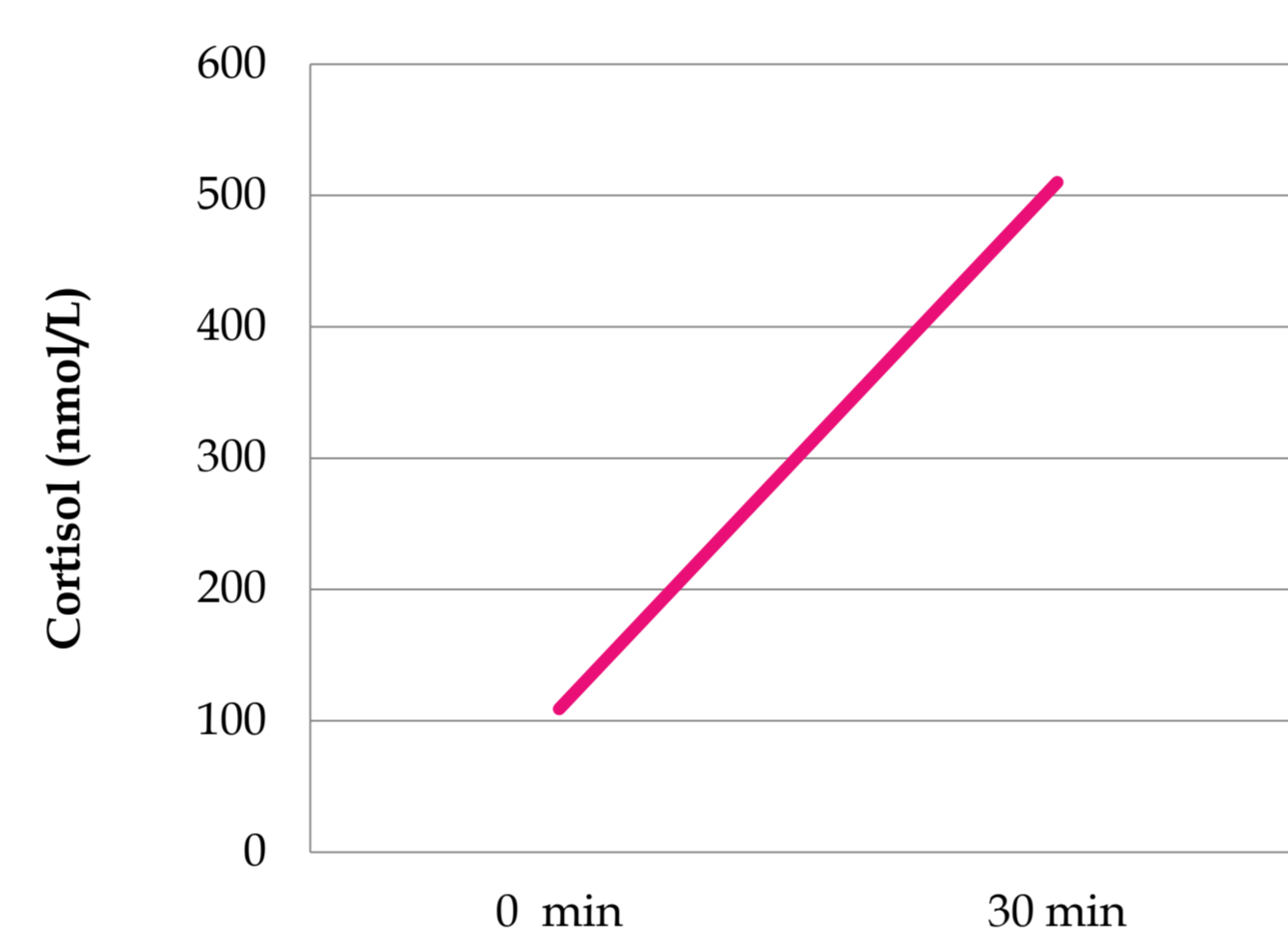
Other baseline pituitary function tests were organised to investigate this patient further:

- MRI head revealed a structurally normal pituitary gland.



Healthy looking pituitary (patient's own)

- The Short Synacthen Test showed a low baseline cortisol of 109nmol/L which rose to 510nmol/L after 30min.

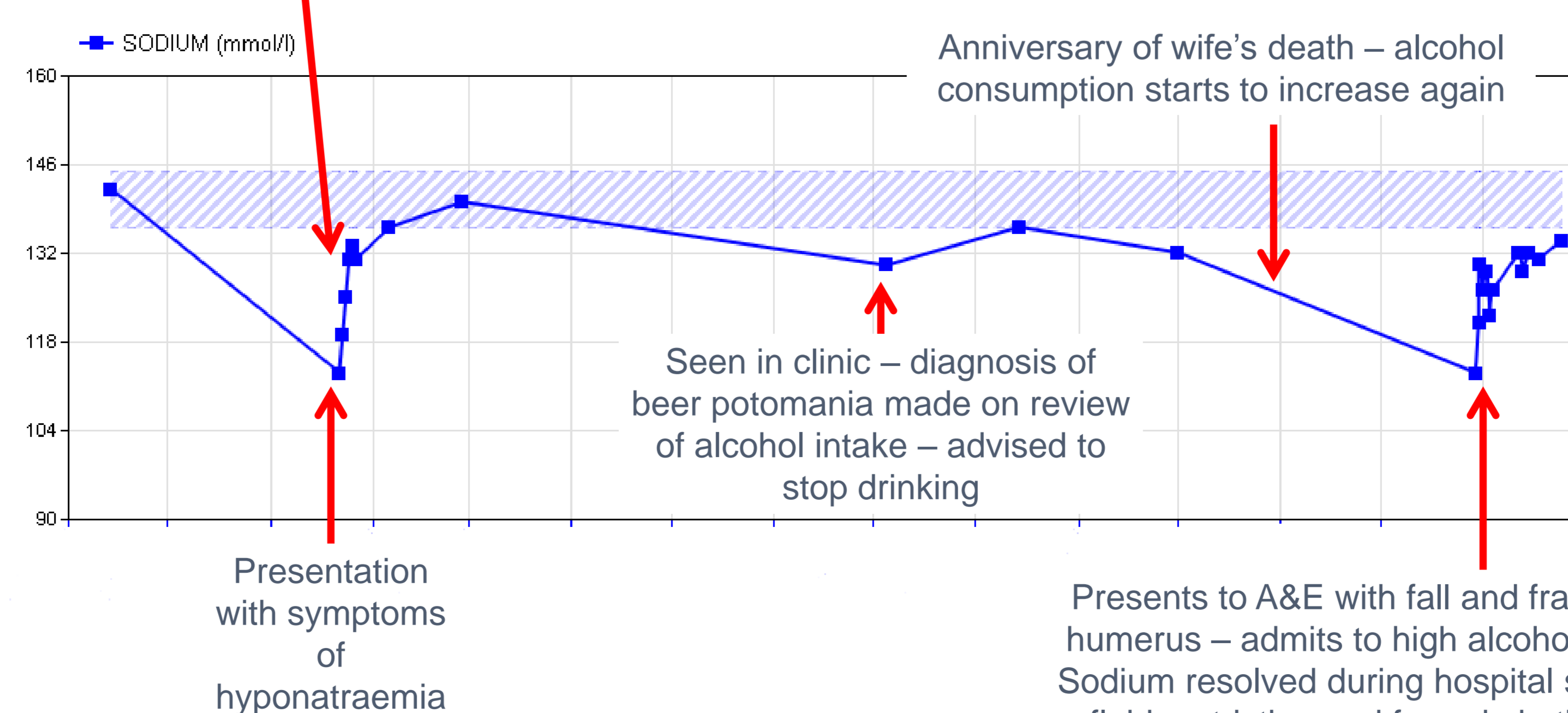


- ACTH was 14ng/l.

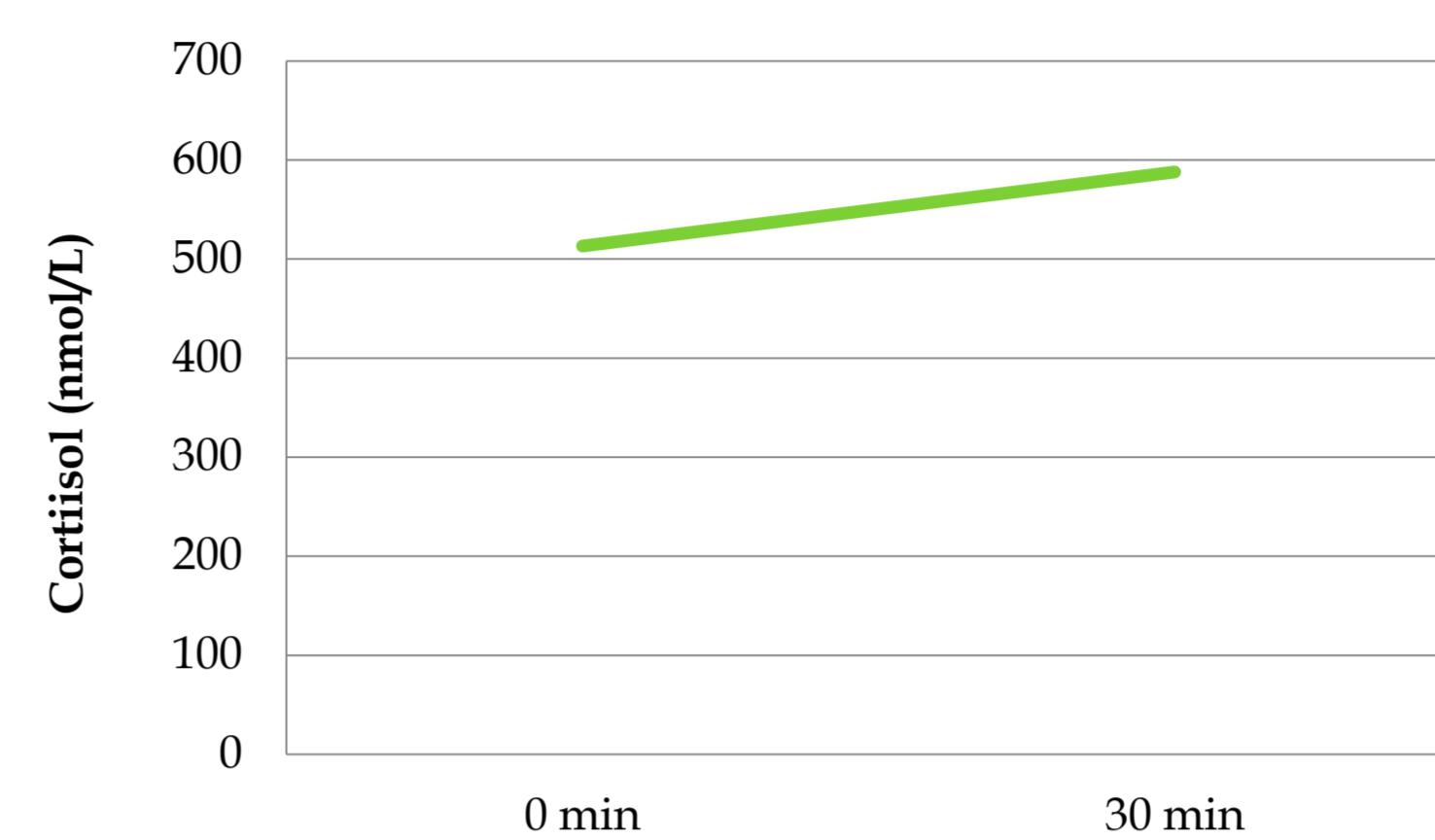
Lisinopril and hydrochlorothiazide were stopped and he was commenced on intravenous hydrocortisone. This led to resolution of his hypotension and hyponatraemia.

He was given a diagnosis of possible ACTH deficiency and discharged on replacement dose hydrocortisone to be followed up in the endocrine clinic.

Lisinopril and hydrochlorothiazide stopped, hydrocortisone therapy started pre-discharge



Follow Up



A repeat Short Synacthen Test 3 months later showed a normal baseline cortisol of 513nmol/L rising to 588 nmol/L after 30min.

Hydrocortisone therapy was stopped and hyponatraemia improved with changes to his antihypertensives and a reduced alcohol intake (see sodium trend table below).

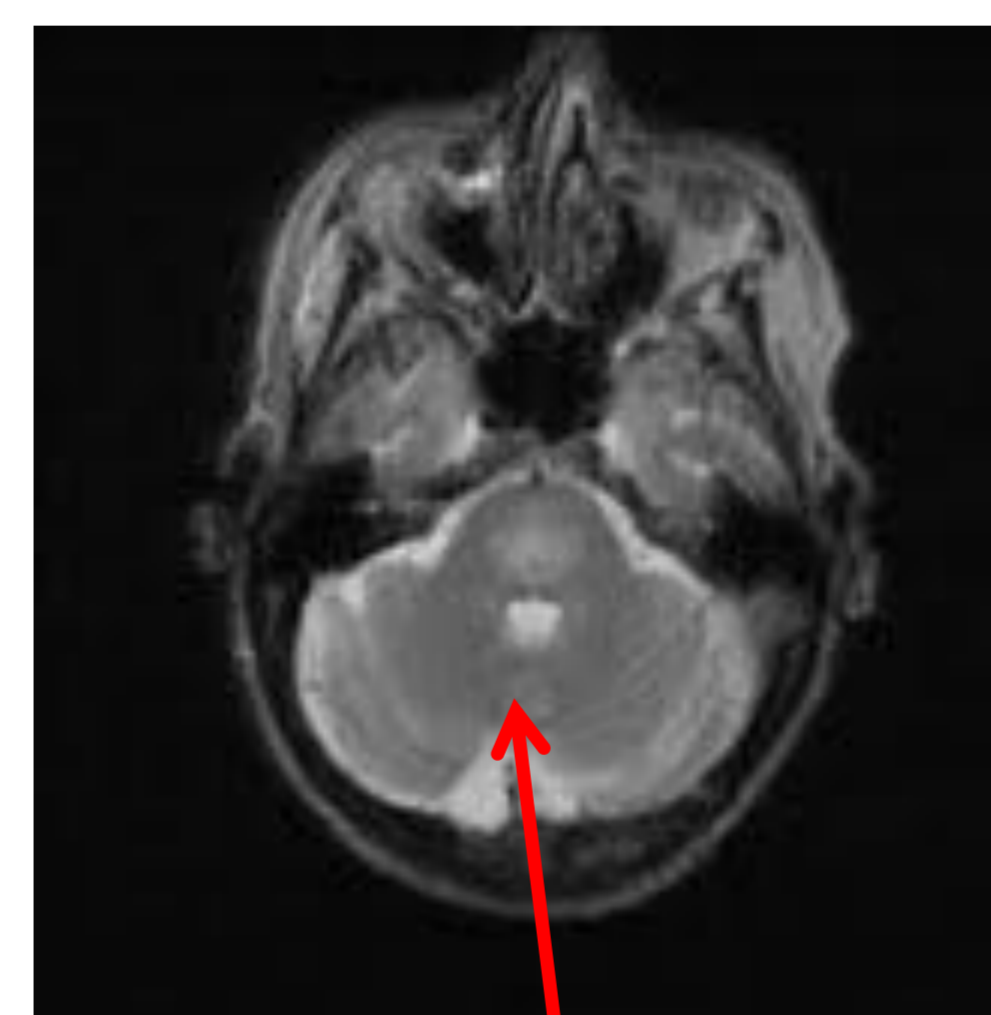
Conclusion

The cause of this patient's initial presentation was considered to be a combination of beer potomania and thiazide diuretic use.

Beer potomania is an under-recognised condition characterised by hyponatraemia secondary to water intoxication. Recognition of this condition and careful fluid administration are required in the initial phase to avoid serious complications such as central pontine myelinolysis.¹

Our case demonstrates that other causes of euvoalaemic hypotonic hyponatraemia may obscure the actual diagnosis of beer potomania resulting in incorrect management.

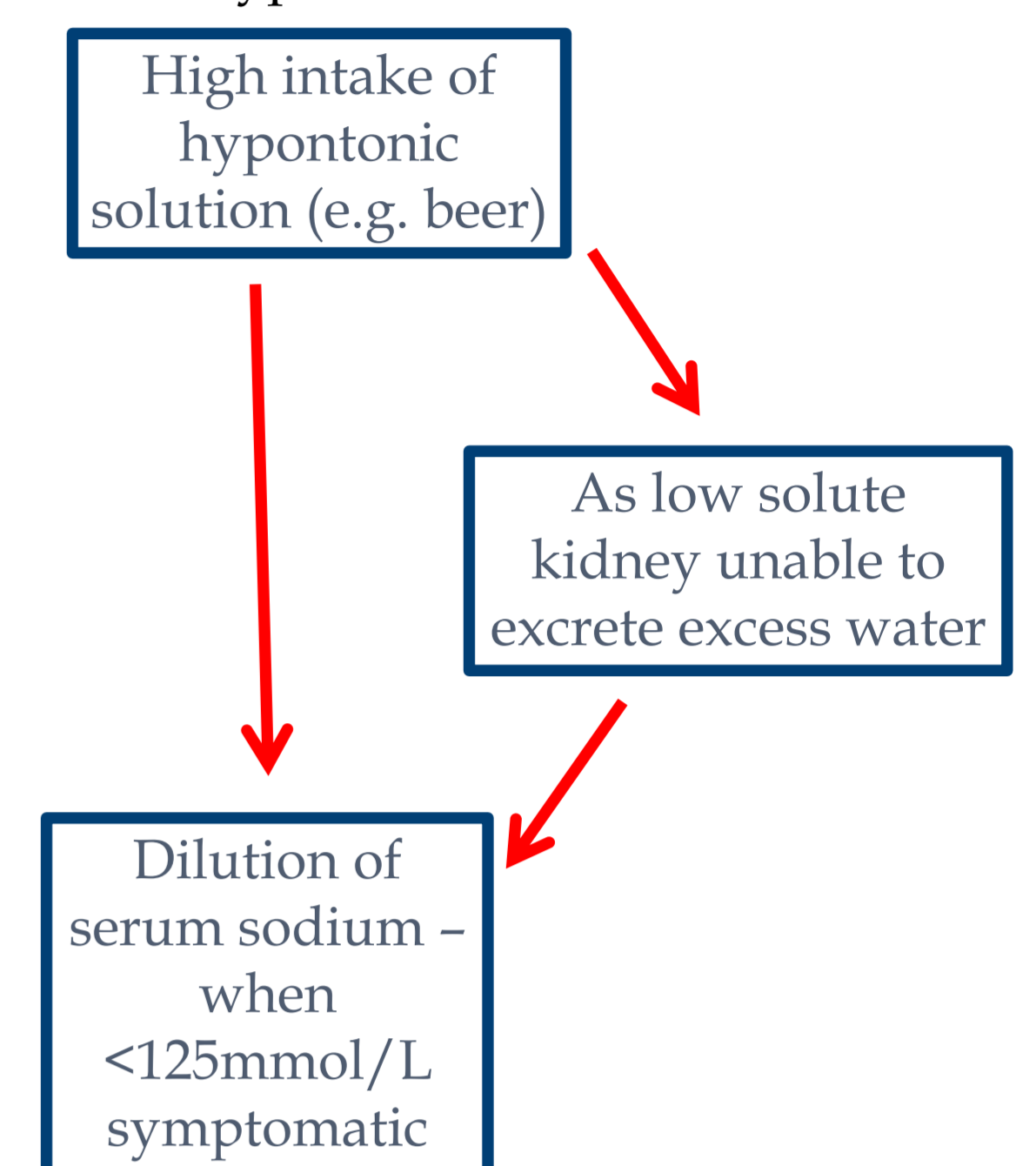
The consequences of an incorrect diagnosis – central pontine myelinolysis



DWI restriction visible in pons

Discussion

Beer potomania was first described in 1971.² It is a rare disorder due to consumption of large amounts of hypotonic solution such as beer.^{1,2}



It is important to determine whether the hyponatraemia is chronic or acute. Overly aggressive treatment may cause irreversible neurological damage or death.¹ There is no effective therapy after the development of central demyelination. Prevention is key.³ Correction of sodium should not exceed 1-2mmol/h and 8mmol/day.^{1,3,4}

When treating chronic hyponatraemia which is the more likely scenario with beer potomania, removal of the underlying cause and fluid restriction are the best course of treatment.⁵

Differential Diagnosis for Euvoalaemic Hyponatraemia⁴

Thiazide diuretics

Hypothyroidism

Adrenal insufficiency

Syndrome of Inappropriate Anti-Diuretic Hormone secretion (cancer, CNS disorders, drugs, pulmonary disease, nausea, post-operative pain, HIV, infection, Guillain-Barre syndrome, acute intermittent porphyria)

Decreased solute ingestion (beer potomania/tea and toast diet)

References

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4. F Assadi, Hyponatraemia: a problem-solving approach to clinical cases, *J Nephrol* 2012 25:4: 473-480
5. P Reddy, A D Mooradian, Diagnosis and management of hyponatraemia in hospitalised patients, *Int J Clin Pract* 2009 63;10: 1494-1508



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