CASE REPORT

Confusion after starting citalopram in a renal transplant patient

Hersharan Sran,¹ Mysore K Phanish²

¹Department of Renal Medicine, Epsom & St Helier University Hospitals NHS Trust, Carshalton, Surrey, UK
²Department of Renal Medicine, South West Thames Renal and Transplantation Unit, St Helier Hospital, Carshalton, Surrey, UK

Correspondence to
 Dr Hersharan Sran,
 hersharan@doctors.org.uk

SUMMARY

The UK NICE (National Institute for Clinical Excellence) guidelines on management of depression in adults recommends use of selective serotonin re-uptake inhibitor (SSRI) as a first choice agent. Citalopram, an SSRI antidepressant, is a preferred antidepressant in those patients with concomitant chronic physical health problems. Hyponatraemia because of syndrome of inappropriate antidiuretic hormone secretion has been reported with SSRI antidepressants but is usually mild and responds to fluid restriction and cessation of the drug. We report a case of severe hyponatraemia because of citalopram in a renal transplant patient who required treatment with hypertonic (3%) saline, with a good outcome avoiding any neurological complications.

BACKGROUND

The increasingly common usage of selective serotonin re-uptake inhibitor (SSRI) antidepressants, especially in elderly patients with multiple comorbidities, has revealed an increased risk of adverse events such as hyponatraemia. Although this is usually mild and corrects with cessation of the drug, in rare cases severe hyponatraemia can occur.

CASE PRESENTATION

A 77-year-old woman presented with confusion and unsteadiness 10 days after starting citalopram for depression. The patient's medical history included end-stage renal disease of unknown cause in 1982 and subsequent cadaveric renal transplantation in 1985 with excellent graft function to date, hypertension and depression. Other medications included prednisolone, ciclosporin A, irbesartan and doxazosin. On examination, she was euvoelaemic, and had no focal neurological signs but was confused and disoriented. She had a previous history of a transient mild hyponatraemia after starting of hydrochlorthiazide, which resolved on stopping the drug, 18 months prior to this presentation.

INVESTIGATIONS

Laboratory investigations revealed severe hyponatraemia with serum sodium of 104 mmol/L, potassium 4.2 mmol/L, creatine 57 µmol/L, bicarbonate 23 mmol/L, serum osmolality 227 mmol/L, inappropriately concentrated urine with an osmolality of 341 mmol/L and urine sodium 30 mmol/L. Serum sodium was 137 mmol/L 1 month prior to this presentation. Thyroid function, serum glucose and lipid profile were normal, as was a subsequent

short synacthen test. CT of the brain, chest, abdomen and pelvis was normal. The history of starting citalopram prior to the onset of the patient's symptoms, and the serum and urine findings in the context of euvoelaemic state (inappropriately concentrated urine with a dilute serum), confirmed the diagnosis of syndrome of inappropriate antidiuretic hormone secretion.

TREATMENT

The citalopram was stopped and a 500 mL/day fluid restriction was instituted. In view of the severity of hyponatraemia and associated confusion and potential risk of other neurological complications, the patient was transferred to the high dependency unit and was given hypertonic (3%) saline via a central venous catheter. The rate of administration of hypertonic saline was calculated as follows

$$\begin{aligned} \text{Sodium deficit} &= \text{Total body water (TBW)} \\ &\times (\text{desired serum Na} \\ &- \text{actual serum Na}) \end{aligned}$$

TBW is $0.5 \times \text{body weight}$ for a woman. For this patient who weighed 48 kg, to achieve a rise in serum Na of 10 mmol/L over 24 h, the calculated sodium deficit = $0.5 \times 48 \times (114 - 104) = 240$ mmol. As hypertonic 3% saline contains 513 mmol/L of sodium, 480 mL would contain approximately 246 mmol of sodium. Therefore, this volume of 3% saline (480 mL) was administered at an initial rate of 20 mL/h and subsequently reduced to 10 mL/h titrated to the rise in serum sodium measured at regular intervals over the next 48 h.

OUTCOME AND FOLLOW-UP

The serum sodium rose to 123 mmol/L 48 h later at which point she was transferred to the renal ward and started on slow sodium tablets in conjunction with a 1 L fluid restriction. The serum sodium continued to rise slowly to 130 mmol/L over the next 8–10 days. Figure 1 illustrates the rate of rise in serum sodium over a period of time. The patient's confusion resolved over several days.

DISCUSSION

Our patient was elderly and was on a salt restricted diet because of hypertension both of which increase the risk of development of hyponatraemia on SSRIs. The incidence of hyponatraemia because of citalopram reported in the literature varies from 0.5% to 32%¹ and has been reported to be three times higher than with other antidepressants.² Most of the other reported cases of hyponatraemia

To cite: Sran H, Phanish MK. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-010511

Figure 1 The rate of rise in serum sodium over a period of time.

Learning points

- ▶ Hyponatraemia is a known but uncommon potential serious adverse effect of commonly used selective serotonin re-uptake inhibitor antidepressant citalopram.
- ▶ Severe hyponatraemia may necessitate use of hypertonic 3% saline.
- ▶ The rate of administration of hypertonic saline can be estimated by calculating the sodium deficit to achieve a rise of around 10–12 mmol over 24 h.
- ▶ It is essential to titrate the infusion against regular measurements of serum sodium in a controlled setting in order to avoid over-rapid correction and the risk of osmotic demyelination.

thought to be because of citalopram are associated with milder degrees of hyponatraemia which improved on stopping the drug or with fluid restriction. Two other cases report hyponatraemia of less than 110 mmol/L.^{2 3} Hypertonic saline requires caution because of the risk of osmotic demyelination particularly in high-risk groups which include patients with alcoholism, malnutrition, hypokalaemia, burns and elderly women on thiazide

diuretics.⁴ Although calculation of the sodium deficit and rate of administration as described above and by Adroque and Madias⁵ is essential for the management of these patients, this may not reliably predict the rate of rise of serum sodium and therefore, frequent measurements of serum sodium are required to reduce the risk of overly rapid correction of hyponatraemia. This case illustrates a known but uncommon potential serious adverse effect of commonly used SSRI antidepressant citalopram. Both primary and secondary care physicians need to be aware of this potential adverse effect of this commonly prescribed drug particularly in the elderly.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Jacob S, Spinler SA. Hyponatremia associated with selective serotonin-reuptake inhibitors in older adults. *Ann Pharmacother* 2006;40:1618–22.
- 2 Kirpekar VC, Joshi PP. Syndrome of inappropriate ADH secretion (SIADH) associated with citalopram use. *Indian J Psychiatry* 2005;47:119–20.
- 3 Bez Y, Aktolga S, Balci M, et al. Citalopram-induced SIADH in a hypertensive patient on salt restricted diet. *J Psychopharmacol* 2007;21:665–7.
- 4 Lauriat SM, Berl T. The hyponatremic patient: practical focus on therapy. *J Am Soc Nephrol* 1997;8:1599–607.
- 5 Adroque HJ, Madias NE. Hyponatremia. *N Engl J Med* 2000;342:1581–9.

Copyright 2013 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow