Severe hyperkalaemia peripartum with magnesium sulphate

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Summary: A case of severe hyperkalaemia peripartum in the setting of magnesium sulphate infusion is presented. Health professionals involved in the care of high-risk pregnancies should be aware of this rare association.

Keywords: high-risk pregnancy, clinical pharmacology, hypertension

CASE PRESENTATION

A 29-year-old woman, G2 P0 TOP1, was diagnosed with pre-eclampsia at 31 weeks gestation at a routine antenatal visit. Her blood pressure was 155/87 mmHg and there was 4+ proteinuria on dipstick urine testing. She was asymptomatic. Her previous antenatal course had been uneventful with booking blood pressure of 118/72 mmHg at 21 weeks gestation. Investigations revealed an elevated serum urate of 0.41 mol/L, normal liver and renal function, and serum bicarbonate 20 mmol/L with a normal anion gap, consistent with a mild type IV renal tubular acidosis (Table 1). The mother was admitted to hospital; betamethasone was administered for fetal lung maturity. A single dose of slow release nifedipine 20 mg was given when blood pressure rose to 160/106 mmHg, she was commenced on labetalol 200 mg three times a day and transferred to a tertiary care hospital. Upon arrival hyperreflexia and clonus were noted and a magnesium sulphate infusion was commenced at a rate of 1 g/hour.

Ten hours after the commencement of magnesium infusion and following two doses of labetalol, repeat blood tests revealed serum potassium of 6.5 mmol/L and serum sodium 131 mmol. Further tests two hours later showed serum potassium of 5.4 mmol/L and no action was taken. After a second dose of betamethasone labour was induced with prostaglandin E2 but the woman proceeded to lower-segment caesarean section with epidural anaesthesia because of decelerations on cardiotocograph, delivering a live male birthweight 1530 g in good condition.

Blood tests immediately postpartum showed serum potassium of 6.1 mmol/L and medical review was sought. Sodium polystyrene sulfonate 30 g was given orally, calcium gluconate was administered intravenously for cardiac stabilization, the mother was placed on continuous electrocardiographic monitoring by telemetry and labetalol was ceased. Initial serum cortisol was undetectable; however, there were no other symptoms or signs to suggest Addison's disease, there was an adequate response following synacthen stimulation and serum adrenocorticotrophic hormone was undetectable consistent with transient

hypothalamic-pituitary-adrenal axis suppression secondary to the two previous doses of betamethasone. Urine sodium was 89 mmol/L and urine potassium was 32 mmol/L. Serum aldosterone was 501 pmol/L and plasma renin activity 9 mU/L. Eleven hours postdelivery the magnesium infusion was ceased when recognized as a possible aetiological factor in the mother's biochemical abnormalities. Three hours after cessation of the magnesium infusion the mother's serum potassium fell to 5 mmol/L, and her biochemical abnormalities resolved rapidly postpartum, despite serum aldosterone falling to below the detection level of the assay two days postpartum.

DISCUSSION

Three women have been reported previously as developing severe hyperkalaemia peripartum with the use of magnesium sulphate infusion for preeclampsia.^{1,2} Each had mild renal dysfunction (serum creatinine 70–82 mmol/L). Two of the mothers had relatively low values for plasma aldosterone (194 and 277 pmol/L, respectively) and plasma renin activity in pregnancy, leading the authors to conclude that hyporeninaemic hypoaldosteronism may have contributed to impaired renal potassium excretion, given the knowledge that magnesium sulphate infusions reduce plasma renin and angiotensin-converting enzyme activity in women with preeclampsia.³ The third mother, in whom the values for aldosterone and plasma renin activity were not reported, had a hyperkalaemic normal anion gap hyperchloraemic metabolic acidosis (type IV renal tubular acidosis) consistent with hyporeninaemic hypoaldosteronism.

Plasma aldosterone and renin levels rise progressively in uncomplicated pregnancy. In pregnancy complicated by pre-eclampsia, plasma aldosterone and renin are decreased to nearly that of a non-pregnant subject.⁴ The plasma aldosterone value in this case was not consistent with hyporeninaemic hypoaldosteronism as a cause of hyperkalaemia. Two studies demonstrated a consistent significant reduction in urinary potassium excretion after infusion of magnesium, while a third study demonstrated inconsistent results.^{5–7} The mild type IV renal tubular acidosis and low urine potassium in the absence of a low plasma aldosterone raise the possibility of resistance

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Table 1 Electrolytes with time

Time	Day 1	Day 2			Day 3				Day 4	Day 5
	12:00	05:30	07:00	20:00	03:00	06:15	10:00	14:00	09:00	09:00
Sodium	136	131	133	132	133	131	129	131	134	135
Potassium	5.0	6.5	5.4	5.9	6.1	5.6	6.0	5.0	4.7	4.4
Chloride	109	110	110	107	110	109	108	106	105	102
Bicarbonate	20	20	20	22	20	20	21	23	26	27
Creatinine		81	78	80	74	73	71	64	59	54
Aldosterone (pmol/L)							501			<70
PRA (mU/L)							9			4
	↑ Mg commenced			↑ Delivery		Sodium polystyrene sulfonate 30 g		↑ Mg ceased		
	19:00 day 1			01:00		04:00		11:00		

All values in mmol/L except for aldosterone and plasma renin activity (PRA)

to the action of aldosterone as a cause of hyperkalaemia in this case.

While beta-blockers can cause hyperkalaemia due to the lowering of plasma renin activity, it would seem very unlikely that two doses of labetalol contributed to hyperkalaemia in this case. None of the mothers reported in this or other cases received any other medication that may have contributed to hyperkalaemia.

In conclusion, severe hyperkalaemia is a rare complication of magnesium sulphate infusion of which obstetricians, obstetric physicians, anaesthetists and intensive care specialists should be aware.

DECLARATIONS

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