EMERGENCY CASEBOOK

Successful resuscitation of hypermagnesaemic asystolic cardiac arrest with the use of early transvenous cardiac pacemaker: a case report

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A 63 year old woman presented to the emergency department (ED) with 1 week of progressive dyspnoea, constipation, and generalized weakness. She had undergone spinal fusion surgery 10 days previously, and had a history of chronic renal insufficiency. The patient had been using milk of magnesia and magnesium citrate in unknown amounts to alleviate her constipation over this time frame. During her ED stay she became progressively hypotensive and bradycardic, and despite aggressive resuscitative measures she suffered an asystolic arrest 1 hour into her ED course. She was resuscitated with conventional therapy, but her haemodynamic profile did not improve significantly until transvenous cardiac pacing was employed. Her magnesium level was 10.4 mmol/l. Treatment of magnesium overload has focused upon haemodialysis, forced diuresis, and the use of intravenous calcium salts. Case reports have previously documented survival of moderately to severely ill patients when these modalities have been used. Likewise, failure of resuscitation despite use of these methods has been previously noted. To our knowledge, this is the first reported case clearly demonstrating the efficacy of transvenous cardiac pacing to successfully resuscitate a patient upon whom multiple vasopressors, fluids, and calcium previously had no clear effect.

A 63 year old woman was brought via the emergency medical services (EMS) to a military community emergency department (ED) complaining of constipation, generalised weakness, and shortness of breath. She had undergone lumbar spinal fusion surgery 10 days previously, and stated that she had not had a bowel movement since the operation. She complained of progressively worsening dyspnoea and generalised weakness over the past 10 days, to the point where she became unable to rise from bed, at which time the EMS was contacted. The patient also reported increasing lower extremity oedema and orthopnoea over the past 3–4 days and denied fevers, chills, haematochezia, melena, nausea, and vomiting. Her past medical and surgical history was remarkable for chronic renal insufficiency and hypertension. She stated she was taking hydrocodone/acetaminophen as needed for pain in addition to furosemide, atenolol, and nifedipine. She denied any recent dosage changes or non-compliance with these medications.

On arrival at the ED, the patient was alert and oriented and appeared in mild distress but responding appropriately in full sentences. Initial vital signs were: blood pressure 113/51 mm Hg, pulse rate 64 beats/min, respiration rate 20 breaths/min, temperature 98.6°F (37°C), and oxygen saturation 94% on room air. Her head, ears, eyes, nose, and throat (HEENT) and

cardiac examinations were unremarkable. Auscultation of her lungs revealed rales bilaterally at the lung bases. Her abdominal examination was significant for diffuse mild tenderness to palpation, but was without peritoneal signs or masses. The lower extremities had symmetrical pitting oedema (score 2+) to the level of the knees. Neurological examination revealed generalised motor weakness to all major muscle groups, symmetrically decreased deep tendon reflexes, and normal mental status and cerebellar functions. Gait was not tested, owing to patient discomfort and weakness. An acute abdominal series, complete blood count, electrolyte panel, blood urea nitrogen, creatinine, and calcium were obtained. Her abdominal radiographs revealed a non-specific bowel gas pattern without any free air or air fluid levels. Chest radiograph displayed diffuse interstitial oedema, but was otherwise normal.

During the subsequent 1 hour, the patient had a progressive decline in her mental status, with ensuing hypotension and bradycardia. One hour into her ED course, she suffered an asystolic arrest. The patient's husband, who had arrived just prior to this episode, added to the history that the patient had been using milk of magnesia and magnesium citrate in unknown amounts to alleviate her constipation. He reported that she had used these formulations on a chronic basis, at least once per week, but that her usage had risen dramatically over the preceding week. At this point, a request for a magnesium level was added to the preceding laboratory studies.

Following cardiac arrest, advanced cardiac life support protocols were instituted. She was intubated, given three rounds (1 mg each) of intravenous epinephrine and two rounds (1 mg each) of intravenous atropine. Eleven minutes into the code, spontaneous circulation returned. At this time, the patient was noted to have a blood pressure of 66/45 mmHg with a pulse of 42 beats/min. ECG showed junctional bradycardia. Subsequent doses of epinephrine and atropine were given, as well as multiple normal saline boluses, with no significant effect on blood pressure or heart rate. Transcutaneous pacing was attempted, but capture could not be obtained even at maximal voltage output. Concurrently, the patient was placed on a dopamine drip, which was titrated to a dose of 20 µg/kg/min. A nor-epinephrine drip was then begun and rapidly advanced to 10 µg/min. These measures failed to improve the patient's haemodynamic profile, and she remained severely hypotensive with systolic blood pressures ranging from 40 to 73 mmHg and heart rate of 32–45 beats/min. An arterial line confirmed these values. During the resuscitation, the patient was also given 5 mg of glucagon intravenously, and 20 ml of 10% calcium chloride for possible beta-blocker or calcium

Abbreviations: ED, emergency department; EMS, emergency medical services; TVP, transvenous pacer

channel blocker toxicity. Neither intervention resulted in haemodynamic improvement.

At this point in the resuscitation, the laboratory values became available. The patient's haemoglobin was noted to be 7 g/dl and creatinine was 4.2 mg/dl. Her magnesium level was 10.4 mmol/L. Initial arterial blood gas was near normal, with a pH of 7.35 and a base deficit of 2. Owing to the critically high magnesium level, an additional 20 ml of 10% calcium chloride were administered as well as additional normal saline boluses (for a total of 8 litres). Systolic blood pressures remained around 70 mmHg and heart rate was 38–45 beats/min. A right internal jugular central venous line was placed, which was followed by passage of a transvenous pacer (TVP). Cardiac capture was obtained at a rate of 80 beats per minute at an output of 3 mA. The patient's blood pressure immediately improved to systolics ranging from 95 to 105 mmHg. She remained stable on the TVP during the remainder of her stay in the ED (3 hours) as she awaited transport to the regional tertiary referral centre's medical intensive care unit.

The patient remained on a combination of ventilator, TVP, and vasopressor therapy for 9 days in the intensive care unit. During this time, she was given large doses of intravenous calcium and had slow improvements in her renal and cardiovascular parameters. The patient had several episodes of recurrent severe hypotension and bradycardia when TVP support was temporarily halted. Her vital signs would quickly stabilise with re-initiation of TVP. She could not undergo dialysis therapy during her stay as the nephrology consultants felt her blood pressures were too tenuous to tolerate the procedure and continuous venovenous haemodiafiltration was not available. The patient made an excellent recovery and had no noticeable neurological sequelae upon discharge from the hospital after 1 month of inpatient therapy.

DISCUSSION

Products containing magnesium are ubiquitous in western society and are commonly available in powdered, liquid, caplet, and rectal forms without prescription. While these products are generally considered quite safe, case reports exist discussing significant adverse health effects and even death, which have occurred secondary to hypermagnesaemia from either inappropriate use of these agents or use in a population group at risk for untoward events.¹²³ Most case reports and toxicology textbooks discuss the use of calcium, intravenous fluids, diuresis with furosemide, and haemodialysis in patients with serious illness resulting from hypermagnesaemia.^{1–3 456} Hypermagnesaemia most commonly occurs in the setting of supratherapeutic administration via intravenous, oral, or rectal routes in patients with impaired renal function.⁴ This case demonstrates the refractory nature of hypermagnesaemic cardiopulmonary arrest that has been noted previously, but also illustrates the efficacy of a TVP in such a patient when vasoactive agents, intravenous fluids, diuresis with volume expansion, and calcium have failed to cause significant improvement. Transcutaneous and transvenous pacemakers are theoretically useful therapeutic modalities and a previous case report has alluded to the use of this intervention in cases such as these,³ but no previous case report has demonstrated such a clear haemodynamic benefit.

Magnesium is an abundant cation in the human body; stored mostly in the bone and intracellularly, and serum magnesium levels correlate poorly with whole body stores of magnesium. However, serum levels exceeding 4 mmol/l usually begin to produce symptoms. At serum levels exceeding 10 mmol/l, these effects are often profound and involve some combination of neurological, cardiovascular, or pulmonary symptoms.^{4–6} In the non-hypermagnesaemic patient, magnesium tends to increase cardiac output and

decrease systemic vascular resistance.^{4 6} In the hypermagnesaemic state, excessive peripheral vasodilation and AV nodal conduction delays cause hypotension and AV blocks.⁷ Magnesium in excessive concentrations also serves to impair neuromuscular function by decreasing acetylcholine release presynaptically and decreasing the responsiveness of postsynaptic receptors. All of these effects were manifested in the patient described in this case: motor weakness, junctional heart rhythm, and respiratory distress and collapse accompanied by hypotension.

Treatment of magnesium overload has focused upon haemodialysis, forced diuresis, and the use of intravenous calcium salts. Case reports have previously documented survival of moderately to severely ill patients when these modalities have been used.^{1–3} Likewise, failure of resuscitation despite use of these methods has been previously noted.²⁸⁹ Transvenous pacing has some advantages over other interventions in that it causes direct stimulation of the cardiac muscle and does not rely upon receptors, ion gradients, or other secondary messenger systems to alter the heart rate. The combination of a pacemaker to increase heart rate in conjunction with attempts to increase preload and systemic vascular resistance with fluid, calcium, and α -1 adrenergic agents makes clinical sense.

To our knowledge, this is the first reported case clearly demonstrating the efficacy of transvenous cardiac pacing to successfully resuscitate a patient upon whom multiple vasopressors, fluids, and calcium had previously had no clear effect.

CONCLUSION

We report the use of transvenous cardiac pacing to successfully treat a patient suffering from hypermagnesaemic cardiac arrest with subsequent cardiac and neurological recovery. Early use of transvenous cardiac pacing in parallel with other suggested therapeutic treatment modalities is recommended in centres with the technical expertise to provide such interventions.

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