



EXCEPTIONAL CASE

Hard water syndrome: a case series of 30 patients from a London haemodialysis unit

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ABSTRACT

Severe and life-threatening hypercalcaemia can develop in haemodialysis patients dialysed against a dialysate with a high calcium concentration, the so-called hard water syndrome. Here we describe the development of hard water syndrome in 30 patients following sequential failure of the reverse osmosis unit and water softeners. Serum calcium levels rose from 2.43 ± 0.19 to 3.92 ± 0.51 mmol/L after exposure. All patients required emergency haemodialysis and four acutely deteriorated, one of whom was 24 weeks pregnant. This is the largest reported series of patients affected by this rare and severe condition. This event led to the introduction of processes to minimize future risks.

Keywords: chronic renal failure, haemodialysis, hard water syndrome, hypercalcaemia, hypertension

BACKGROUND

Hard water syndrome was first described in 1967, when failure of a dialysis unit water softener [1] led to 12 patients being dialysed against hard water, causing severe symptomatic hypercalcaemia and hypermagnesaemia. Following its initial description, further case reports of life-threatening hypercalcaemia in haemodialysis patients dialysed against a dialysate with a high calcium concentration have been reported [1–4], though its overall incidence remains rare. Modern dialysis units have been designed to minimize the risks of dialysis from exposure to hard water, toxins and infective organisms with the utilization of both reverse osmosis (RO) units and water softeners to ensure water quality. Here we describe the acute onset of hard water syndrome in 30 patients dialysed in a London dialysis unit.

CASE REPORTS

Thirty patients received haemodialysis during an afternoon session and the following morning session in March 2016 at Guy's Hospital, London. During the afternoon dialysis session, a

71-year-old male presented with fever, hypertension, tachycardia and headache 90 min into dialysis. He had developed new-onset atrial fibrillation and his serum corrected calcium was 3.86 mmol/L. His deterioration was initially thought due to a prescription for 1.5 µg of 1-alfacalcidol daily and pneumonia. The following morning, several patients became unwell in rapid succession. A 27-year-old female who was 24 weeks pregnant developed headache, vomiting and hypertension (204/116 mmHg) during dialysis. She was treated initially for suspected severe pre-eclampsia. Three other patients then developed severe vomiting and hypertension. The symptoms of hard water syndrome were recognized and the dialysis unit was closed with patients diverted to satellite dialysis units or inpatient beds. All exposed patients were called in for assessment and emergency haemodialysis.

Thirty patients [median age 69 years (range 27–86)] were affected and 13 (43%) exhibited symptoms of nausea (8), headache (4), vomiting (6) and drowsiness (2). Three patients required admission: the pregnant patient identified above, a 63-year-old with a urinary tract infection and a 43-year-old who deteriorated while receiving emergency dialysis the following day,

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requiring intensive care unit admission with cholecystitis. All patients survived to discharge. The pregnancy continued to term without complications.

Serum corrected calcium concentrations rose from 2.43 ± 0.19 mmol/L (mean \pm SD) pre-dialysis to 3.92 ± 0.51 mmol/L post-dialysis ($P < 0.001$) and serum magnesium concentrations rose from 0.92 ± 0.09 to 1.04 ± 0.10 mmol/L ($P < 0.01$). Serum corrected calcium concentrations responded to treatment with haemodialysis (2.54 ± 0.22 mmol/L).

When compared with the average of the three prior dialysis sessions, blood pressure was significantly elevated mid-dialysis ($156 \pm 17/87 \pm 22$ versus $126 \pm 22/71 \pm 11$ mmHg; $P = 0.02$) and post-dialysis ($158 \pm 31/80 \pm 16$ versus $134 \pm 22/73 \pm 11$ mmHg; $P < 0.001$).

Soft water for dialysis is generated in a multistep process involving passage through multiple filters, one of two water softeners running in parallel and then through the RO unit. Two weeks prior to this incident, a failure of the twin-stage RO caused activation of a high-conductivity alarm prompting immediate cessation of haemodialysis. On investigation, the casing of one of the two water softeners was found to have cracked, with leakage of ion exchange resin beads downstream, blocking the RO membranes. A temporary solution to provide soft water was implemented using the remaining water softener with daily manual regeneration and water softness checks. Water softness was confirmed prior to recommencement of haemodialysis for a reduced number of patients through use of the yes/no Palintest and dialysate biochemical analysis, with normal sodium (139.4 mmol/L) and bicarbonate (28.5 mmol/L) concentrations found. Replacement of the second water softener allowed increased capacity and automatic switching between water softeners when depleted. At this point, it was felt that there was no longer a need to continue daily water softness testing.

On the day preceding the development of hard water syndrome, remedial work was performed on the second water softener, but automatic switching between softeners was deactivated, leading to their depletion and exposure of the patients to hard water.

Dialysis was restarted after replacement of the water softener spreader manifolds in the unit to prevent recurrence. Water quality was confirmed with the Renal Association test prior to recommending dialysis.

DISCUSSION

This cause of RO unit malfunction has not been reported before and has prompted replacement of the water softener spreader manifolds throughout local dialysis units. While the frequency of water quality checks was within that recommended by the Renal Association guidelines [5], root cause analysis and peer review by renal technicians from other dialysis units were conducted to identify preventative strategies. These include that in-house renal technologists be present whenever external companies are conducting work on the unit and that water softness checks be performed daily after any such work.

Hard water syndrome was initially described in 12 patients dialysed against hard water in 1967, causing severe hypercalcaemia and hypermagnesaemia following water softener failure [1]. Other causes described in both home and unit haemodialysis settings include incorrect dialysate formulation [2], high calcium concentrations in the home water supply [3], failure to replenish the water softener and connecting the efflux port of the RO unit to the dialyser [4]. The symptoms of hard water syndrome include nausea, vomiting, weakness, hypertension and increased risk of arteriovenous fistula thrombosis. Headache and nausea are not infrequently encountered among the dialysis population, making early or mild cases of hard water syndrome hard to identify. Suspicion must therefore be raised should a larger proportion of patients experience these symptoms over the course of a single dialysis session.

Haemodialysis safety has progressed significantly since hard water syndrome was first described in 1967. Despite this, the largest reported cohort of patients arose from a tertiary centre 50 years later. Identifying the cause of the failure in the water supply is therefore relevant to all haemodialysis centres, given the significant morbidity associated with this syndrome.

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CONFLICT OF INTEREST STATEMENT

None declared. The results presented in this article have not been published previously in whole or part, except in abstract format.

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