**Nephroquiz**
(Section Editor: M. G. Zeier)

**Hard times with hard water**

**Case**

A 69-year-old man with known coronary artery disease, atrial fibrillation, peripheral vascular disease and chronic renal insufficiency was admitted to the hospital acutely ill. He had received an angiotensin-converting enzyme inhibitor, a β-blocker and phenprocoumon. He was dyspnoeic and in acute distress. He had distended neck veins and rales bilaterally. Furthermore, he had pitting oedema of the sole remaining lower extremity. After a brief physical examination and administration of oxygen, morphine and nitroglycerin, laboratory information was available. An electrocardiogram (ECG) was done (Figure 1, left panel). His haemoglobin was 11.9 g/dl, haematocrit 37 vol%, white count 9100, platelets 123 000 μl^3, INR was 8.1 and serum creatinine was 700 μmol/l. His electrolytes were calcium 1.78 mmol/l, phosphorus 2.1 mmol/l, pH was 7.13, with an HCO3^−_ of 9 mmol/l and a PaCO2 of 27 mmHg, indicating metabolic acidosis with a rather low respiratory compensation. We were in a quandary; dialysis was indicated for various reasons; however, the INR value prompted us to administer vitamin K and fresh frozen plasma over the next 24 h. Then dialysis was performed in our intensive care unit.

After the dialysis procedure, the patient, who had been disoriented to somnolent, was now comatose. His serum calcium value was 5 mmol/l. A second ECG was done (Figure 1, middle panel). A second haemodialysis was performed with a different dialysis machine and different concentrates. However, the serum calcium value did not fall below 5 mmol/l. An ionized value was 2.2 mmol/l. We were confronted with acute hypercalcaemia that did not respond to therapeutic interventions.

**Question**

What can possibly cause ‘malignant’ hypercalcaemia in dialysis patients?

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Fig. 1. ECGs obtained at different time points. Left panel: leads V1–V6 at admission. Middle panel: shortening of the ST interval and flattening of the T wave during hypercalcaemia. Right panel: normalization after calcium was returned to normal range.
Answers to the quiz on the previous page

Our patient had no reason to suffer from any known hypercalcaemic syndrome. Moreover, hypercalcaemia within hours to minutes directs the attention to a technical problem. None of us had ever seen the ‘hard water syndrome’ before. In a foregone era, hypercalcaemia and hypermagnesaemia occurred because of problems with ‘tap’ water used for dialysis [1]. Several dialysis unit epidemics drew the attention of nephrologists to water purification [2,3]. We retired the elderly reverse-osmosis (RO) machine and dialysis apparatus, even though we could find no fault with these machines. We substituted a new machine and a different RO machine. With the new machinery, the patient’s calcium values decreased and, after numerous treatments, he gradually recovered. A follow-up ECG was improved (Figure 1, right panel).

We measured calcium from the tap, from the RO machine and from the dialysis machine. We found that the tap gave hard water (no surprise in Berlin). The RO machine was excellent in reducing the concentrations to zero. Oddly, the dialysis machine, although receiving a calcium value of zero, returned a much higher calcium value. We reasoned that something must be wrong in the electrolyte distribution of the concentrates. The detection device of the machine determines a deviation of 5 mmol/l in terms of electrochemical deviance. This deviation had not occurred. We reasoned that the calcium aberration was not sufficient for the machine to find it. Our data (Figure 2) initially suggested that the dialysis machine and its mixing device must be at fault. The RO machine was innocent since the calcium concentration after the device was zero. We blamed the dialysis machine, but were unable to incriminate it further. The manufacturers of both devices sent technical representatives to our department immediately. However, they were unable to find fault with either of their products.

We then carefully examined the back (where no nephrologist is likely ever to look) of our dialysis and RO machines very carefully. We found that water enters the RO machine (Figure 3) after first passing through a filter (labelled Weichwasser Zulauf). The RO machine has two other taps, namely an efflux tap for high-calcium waste water (labelled Konzentrat) and an efflux tap for purified water (labelled Permeat) that is intended for the dialysis machine. The connectors on these taps were identical and they were not colour-coded (Figure 3). Thus, no protection in terms of a ‘false hook-up’ was possible in our machines.

Intensive care medicine requires technical support that is above and beyond routine care. This catastrophe would not have happened in our dialysis unit, where a central water supply is outfitted to avoid such problems. However, we serve five different intensive care units spread over 25 km². We are forced to rely on a separate RO machine to supply our vintage dialysis machines.

Fig. 3. Reverse osmosis machine with an inlet tap (Weichwasser Zulauf) and two outlet taps, namely an efflux tap for high-calcium waste water (Konzentrat left) and an efflux tap for purified water (Permeat right) that is intended for the dialysis machine. These two taps lie next to each other and have identical connectors. The German terms Konzentrat and Permeat are confusing. It would have been better if these had been labelled ‘to dialysis machine’ and ‘to drain’.
machines with purified water. Conceivably, hard water syndrome occurred in our patient because of an inadvertent hose reversal.

Our diagnosis: hard times with hard water.

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References


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