A bag full of sugar makes your sodium go down!

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Introduction

For many years granulated sugar and honey have been used to promote wound healing [1,2], especially in the tropical areas of the world. Already by the 17th century the value of sugar in wound healing was reported [3], but also in modern medicine there is still a widespread, though not evidence-based, use and even a revival of sugar and honey use in topical wound care. Both substances have been shown to have anti-microbial and granulation-inducing properties [4,5].

The use of sugar in the treatment of open wounds is not always safe, as we present in a case of acute renal failure during wound care with sugar.

Case

A 54-year-old male was admitted for correction of a large midline incisional hernia using the component separation technique according to Ramirez et al. [6]. The medical history was remarkable for obesity. Moreover he had a possible systemic rheumatic disease, which was never classified, for which he took low dose prednisone. The hernia developed after a Hartmann’s procedure, performed because of perforated diverticulitis 3 years before.

A few days after correction of the hernia a wound infection developed. The infection was treated by removing the skin stitches after which, due to the large subcutaneous dissection that had to be performed during the initial operation, a 15 × 20 × 6 cm skin wound developed. Contaminated haematoma as well as abundant skin and fat necrosis were removed.

The wound was filled with ~1 kg of granulated sugar. Three times a day a wound inspection was performed and additional sugar was added in order to keep the wound completely filled with sugar. No rinsing with water or other topical measures for wound care were performed. Five days later the patient became anuric and the serum creatinine rose to 6.33 mg/dl. In addition, severe hyponatraemia (119 mmol/l) was found. Blood glucose levels were normal. There were no signs of dehydration and we could not find any reason to suspect acute tubular necrosis. There were no clinical signs of cerebral oedema. When analysing the hyponatraemia a surprisingly high plasma osmolality (315 mosm/kg) was measured. As the calculated plasma osmolality (2 × [Na⁺]p + [glucose]p/18 + [BUN]p/2.8) was only 266 mosm/kg, a large osmol gap (49 mosm/kg) was present.

The presumptive diagnosis of acute hyperosmolar syndrome with acute renal failure and hyponatraemia due to absorption of sucrose was made. Sugar treatment was stopped immediately and haemodialysis was performed. There was a rapid decrease in plasma osmolality during haemodialysis (Figure 1). Because plasma osmolality remained elevated a second dialysis was performed which normalized plasma osmolality. After a few hours diuresis returned and after 1 week creatinine levels were normalized (0.88 mg/dl). Wound care was resumed with rinsing only and healing by secondary intention occurred after 2 months.

Discussion

We describe a patient who developed acute renal failure and hyperosmolar hyponatraemia due to absorption of sucrose from a large wound. Sucrose, which is a disaccharide of glucose and fructose, has a molecular weight of 342.3 kDa. In the gut, sucrose is hydrolyzed by disaccharidases and glucose and fructose are absorbed separately. After systemic administration, however, no hydrolysis occurs and sucrose is not metabolized. The only route of elimination is by glomerular filtration. The hyperosmolar state can...
induce so-called ‘osmotic nephrosis’ and lead to acute renal failure. A vicious circle with continuing absorption of sucrose and impaired elimination develops and hyperosmolality worsens. This has also been described after intravenous administration of mannitol [7].

Acute renal failure due to absorption of sucrose is rare, but it has been reported more than 15 years ago [8,9]. However, renal failure due to intravenous sucrose has been reported frequently [10–13]. In most cases sucrose has been used as an additive in immunoglobulin preparations. The exact mechanism by which sucrose causes renal failure is unknown, but osmotic swelling of tubular cells has been found, similar to the tubular damage that can be found after use of mannitol (osmotic nephrosis) [7,10,11]. In addition strong preglomerular vasoconstriction may contribute to the fall in glomerular filtration rate [14].

In most cases of hyponatraemia, hypotonicity caused by appropriate antidiuretic hormone secretion due to hypovolaemia or overhydration due to acute renal failure are plausible explanations for the hyponatraemia. The high serum osmolality with a large osmol gap present in this case led to the diagnosis and subsequent adequate treatment. When analysing hyponatraemia, measurement of osmolality and osmol gap is important to obtain a full picture of the derangement.

Conflict of interest statement. None declared.

References


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