Non-accidental salt poisoning

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INTRODUCTION

Deliberate salt poisoning is a serious cause of hypernatraemia in children and represents a diagnostic challenge for the treating physician. The most important aspect is to actually consider this diagnosis, given its rarity and the severe medical and social consequences associated with it, since parents and carers suddenly become suspects to be confronted with the possibility of having deliberately harmed their child. Wrongfully accusing carers may have serious repercussions and the paediatrician has to have good evidence before raising the diagnosis.[1] Moreover, salt poisoning is exceedingly rare. While the true incidence is unknown, as the diagnosis is likely missed in some cases, the annual incidence of recognised non-accidental salt poisoning in the UK in one study was approximately 1 in 10 000 000 children aged under 16 years.[2] Thus, most paediatricians will never encounter such a case in their professional life. Yet, considering this diagnosis is key to preventing the potentially fatal consequences. Here, we will review clinical and especially diagnostic aspects of salt poisoning. Due to its rarity, evidence-based guidelines are difficult to establish. Thus, the initial diagnosis has to rely mainly on our understanding of physiology and is ideally subsequently confirmed by forensic investigations.

HYPERNATRAEMIA AND SALT POISONING

A previous expert consensus statement made recommendations for the approach to the patient with suspected salt poisoning, emphasising the importance of weight measurements and paired plasma/urine biochemistries with calculation of the fractional excretion of sodium (FENa) to distinguish from the much more common hypernatraemic dehydration.[3] The emphasis on FENa, rather than absolute urine sodium concentrations is to account for the approximately 20-fold variability in urine concentration (50–1000 mOsm/kg), which makes absolute solute concentrations difficult to interpret.[4] Urine sodium concentrations as high as 152 mmol/L have been reported in hypernatraemic dehydration.[5] This is similar to those reported in salt poisoning, although most cases reported had concentrations above 200 and even as high as 374 mmol/L.[5]

PHYSIOLOGIC PRINCIPLES OF THE DIAGNOSIS

Plasma sodium concentration is measured in mmol/l, making it immediately clear that changes in concentration can be caused either by a change in the numerator (sodium) or the denominator (water volume). Thus, hypernatraemia can be caused either by an excess of salt (salt poisoning) or a deficiency in water (hypernatraemic dehydration). The kidneys regulate renal salt excretion in response to plasma volume: if plasma volume is expanded, salt excretion is increased and vice versa. Salt poisoning increases plasma volume due to the increased osmotic pressure, moving water from the intracellular to the extracellular space and to the consequent thirst and increased water intake leading to an increase in weight, provided the subject has access to water and has not lost excessive fluid, such as from vomiting or diarrhoea. Thus, salt poisoning is expected to be associated with increased salt excretion and, assuming no extra losses, with stable or increased weight (depending on fluid intake). Conversely, hypernatraemic dehydration is associated with volume loss and thus expected to be associated with a low FENa and decreased weight. However, these indices are not infallible and have to be interpreted with caution, as we will review here and illustrate with two case scenarios, which are based on our own experience.

CLINICAL SYMPTOMS OF SALT POISONING ARE SIMILAR TO DEHYDRATION

Clinical symptoms described in both accidental[6] and non-accidental[7] salt poisoning are primarily vomiting and diarrhoea, thirst and in more severe cases, seizures, irritability, drowsiness or coma. This is essentially identical to hypernatraemic dehydration and with vomiting and diarrhoea being the leading symptoms, it is not surprising that a diagnosis of salt poisoning may be missed, as the treating physician instinctively assumes an erroneous aetiology of dehydration.

Key learning points

- ▸ Salt poisoning is rare, but should be considered, if there is hypernatraemia without clinical evidence of severe dehydration.
- ▸ Patients at highest risk are those without access to free water.
- ▸ A history of previous unexplained episodes of hypernatraemia should raise suspicion of salt poisoning.
- ▸ A history of vomiting and diarrhoea does not exclude the diagnosis.
- ▸ Calculating the free water deficit (the minimal expected weight loss in hypernatraemic dehydration) and comparing it with the observed weight loss is helpful to assess the possibility of salt poisoning. If no recent weight is available, the weight after normalisation of plasma sodium should be used for comparison.
- ▸ Fractional excretion of sodium (FENa) is a key investigation, but if not available, clinical parameters, such as signs of dehydration and weight might be the only indicators. Moreover, FENa is difficult to interpret in patients with abnormal or unstable glomerular filtration rate.
- ▸ Once suspected, securing all administered substances is critical to prove the diagnosis.
- ▸ A high sodium concentration in a gastric aspirate can further help to prove the diagnosis of salt poisoning.

CHARACTERISTICS OF PATIENTS

Patients at highest risk for non-accidental salt poisoning are those without free access to water, that is, infants and disabled children. Otherwise, the thirst elicited by the rise in plasma sodium would quickly normalise it. Nevertheless, there are reports of able children aged 6 years, who developed hypernatraemia from being force-fed table salt.[8] [9] Presumably, these children were denied water by the perpetrators. While the Binghamton hospital disaster[6] and similar catastrophes[10] have raised doubts on the notion that infants would refuse to drink salty solutions, several case reports of deliberate salt poisoning concern patients receiving tube feeding.[7]

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It is important to realise that most patients with subsequently diagnosed non-accidental salt poisoning had multiple previous presentations with hypernatraemia, suggesting that the perpetrator had performed the poisoning repeatedly.[5] In other cases, there had been evidence of either concurrent or previous physical abuse.[3] Thus, a history of previous episodes of hypernatraemia or of physical abuse should be the most important red flag to raise suspicion of salt poisoning and prompt careful investigations.

INTERPRETING FENA

In steady state, renal excretion of sodium reflects intake to maintain equal sodium balance.[4] Assuming a glomerular filtration rate (GFR) of 100 mL/min and a plasma sodium concentration of 140 mmol/L, an average adult (1.73 m^2 body surface area) filters approximately 20 mol of sodium per day, equivalent to roughly 1.2 kg of salt. The estimated daily salt intake in adults ranges between 2 and 10 g/day,[11] which equates to approximately 0.2%–1.0% of the filtered load. Thus, a FENa <1% is expected in healthy subjects with normal salt intake and this is in line with reported FENa values in healthy children.[12] Consequently, the expectation in hypernatraemic dehydration is that FENa is <1%, whereas it is expected to be well above that in salt poisoning. Again, this fits with reports of FENa in dehydrated infants, which is typically <1%,[13] whereas it is substantially higher (2%–21%) in the few reported values from children with salt poisoning.[3 5] Thus, as highlighted in the Royal College of Paediatrics and Child Health guidelines,[3] FENa is an important tool in clarifying the aetiology of hypernatraemia. However, the key problem is that the expected values are based on normal kidney function. If GFR drops by 50%, only half of the amount of sodium is filtered and the same amount of sodium excreted now represents double the fractional excretion. In patients with chronic kidney disease, the expected values for FENa can be extrapolated from the degree of GFR impairment, but in acute kidney injury, for instance, in severe dehydration, when plasma creatinine has not reached steady state, expected values for FENa cannot be calculated. The most extreme scenario is of course the anuric patient, where a FENa simply cannot be obtained. One could argue that such a hypothetical case is extremely unlikely to occur, but a patient with end-stage kidney disease (ESKD) has, of course, the same risk as any other child to suffer from salt poisoning and we indeed experienced this scenario (case 1).

INTERPRETING CHANGES IN WEIGHT

Changes in patient weight are another important tool to delineate the aetiology of hypernatraemia with the simplified expectation, detailed above, that weight is decreased in hypernatraemic dehydration, whereas it is stable or increased in salt poisoning. Yet, there are several problems also with the interpretation of weight changes. Some of them are simply practical: a weight may not have been obtained at presentation. Or a recent previous weight may not be available to calculate the change. In this case, the weight after rehydration should be used to estimate the degree of dehydration.[3]

The key problem, however, is that salt is an effective emetic and vomiting and diarrhoea are common presenting symptoms in cases of salt poisoning and may cause weight loss.[3 7] Thus, the simple expectation that weight should be stable or increased in salt poisoning does not hold true on closer inspection and changes in weight have to be interpreted more carefully. Key is to calculate the expected change in weight, if hypernatraemia was due to water loss alone and compare it with the observed change. If the observed change in weight is less than the expected, than salt poisoning should be suspected. The expected change in weight is based on the calculation of the free water deficit, with the following formula:

Formula 1: calculation of free water deficit

$$\text{Weight (kg)} \times \text{total body water ratio (0.7 in an infant; 0.65 in an older child)} \times (\text{measured plasma Na} - 145\ [\text{upper limit of normal}]) / \text{upper limit of normal for plasma Na (145)}$$

For examples, please see tables 1 and 2.

Table 1 Laboratory values at presentation in case 1

Age (years)	2
Δ weight (g)*	+300
Expected Δ weight (g) with dehydration†	−1103
Plasma Na (mmol/L)	165
Creatinine (μmol/L)	361

Urine values were unavailable, as the patient was anuric.
*The weight change is in comparison to a weight obtained 2 days prior.
†The change in weight expected with hypernatraemic dehydration was calculated according to formula 1: weight (12.3 kg)×total body water ratio (0.65)×(observed plasma Na (165) – upper limit of normal for plasma Na (145))/upper limit of normal for plasma Na (145).

This is a very conservative calculation, as the calculated value refers to the theoretical concept of deficit of pure water. Since in clinical reality the fluids lost in vomiting and diarrhoea also contain sodium, even more fluid and thus weight would have had to be lost to account for the high plasma sodium concentration.

While sodium principally distributes to the extracellular fluid space, total body water should be used for the calculation, as intracellular water would shift to the extracellular space to dissipate an osmotic gradient between the fluid compartments after addition of salt.[14]

BIOCHEMICAL CHARACTERISTICS

Further hints to a possible diagnosis of salt poisoning can be contained in the biochemistries. In dehydration one would usually expect a slight elevation in plasma creatinine and especially urea levels, consistent with hypovolaemia.[15] In our case scenario 2, both were in the low normal range instead, arguing against significant hypovolaemia. Moreover, analysis of urine osmolality and electrolytes revealed that almost all of the urine osmolality (702 mOsm/kg) was constituted from sodium (321 mmol/kg) and accompanying anion. This is consistent with the high FENa (see below) and reflects the kidneys' attempt at excreting salt rather than conserving water.

FORENSIC ASPECTS

Once salt poisoning is suspected, it is absolutely critical to immediately involve the local child protection team to help protect the child from potential further abuse. Involvement of the police is also urgent to help gather evidence. Obtaining a gastric sample for sodium analysis should be considered and is especially easy to get in children with a gastric tube. Current feed preparation, as well as the ingredients used to make up the feed should be secured as soon as possible for forensic analysis.

CASE SCENARIOS

Case 1

A girl aged 2 years with ESKD secondary to left renal agenesis and small dysplastic right kidney presented for routine follow-up to the dialysis clinic. The mother reported that she had awoken the night before screaming and was irritable.

Her past medical history was relevant for having commenced peritoneal dialysis in the first month of life. She was developing well and gaining weight, but was dependent on tube feeding and had over

Table 2 Laboratory values in case 2

	Case 2	
Age (years)	0.1	0.2
Δ weight (g)*	−270	−470
Expected Δ weight (g) with dehydration†	−792	−745
Plasma Na (mmol/L)	183	177
Cl (mmol/L)	152	148
Urea (mmol/L)	3.8	3.2
Creatinine (μmol/L)	24	29
Osmolality (mOsm/kg)	361	N/A
Urine Na (mmol/L)	321	222
Creatinine (mmol/L)	N/A	<1
Osmolality (mOsm/kg)	702	512
FENa (%)	N/A	>3.6‡

Shown are key laboratory values for the episode (age 0.1 year), where salt poisoning was first suspected followed by the subsequent episode.
*The weight change at the presentation at age 0.2 years is compared with the weight the child had once plasma Na had normalised.
†The change in weight expected with hypernatraemic dehydration was calculated according to formula 1:
Presentation at age 0.1 years: weight (4.32 kg)×total body water ratio (0.7)×(observed plasma Na (183)−upper limit of normal for plasma Na (145))/upper limit of normal for plasma Na (145)=0.792 kg
Presentation at age 0.2 years: 4.83×0.7×(177−145)/145=0.745 kg
In both instances, the patient presented with weight loss, but the loss was less than expected, if the hypernatraemia had been due to dehydration.
‡The exact FENa could not be calculated, as the urine creatinine was measured at <1.0 mmol/L, so the real value for FENa could have been substantially higher than 3.6%.
N/A, data not available.

time developed an aversion to taking anything orally. The only enteral intake she received was a milk feed administered via gastrostomy tube. The mother prepared this feed freshly every day with a prescribed mixture of three components. The feed had commenced at 21:00 the preceding day and 5 hours later the child had woken up.

On examination, she was unsettled, with no evidence of dehydration. Her weight was 12.3 kg, increased by 300 g from a weight obtained 2 days earlier. Blood pressure could not be measured due to her discomfort.

Routine laboratory values obtained in clinic showed marked hypernatraemia (table 1). Review of previous laboratory values revealed two further episodes of hypernatraemia, 3 months (154 mmol/L) and 8 days earlier (150 mmol/L) that had not been investigated further.

The patient was admitted for observation and peritoneal dialysis. Plasma sodium concentration normalised over the following 48 hours.

The remaining feed from the day, as well as the containers with the respective ingredients were secured. Forensic analysis of the milk feed revealed a sodium concentration of 713 mmol/L (expected 14.8 mmol/L) and identified excess salt in one of the ingredients.

Case 2

A boy aged 7 weeks was brought to Accident and Emergency with a 4-day history of vomiting and diarrhoea. Examination revealed a modest weight loss (270 g) from a previous weight (4.275 kg) obtained 5 days before. He had previously presented to his general practitioner on several occasions with similar symptoms.

Blood tests in A&E revealed hypernatraemia (183 mmol/L), presumed to reflect hypernatraemic dehydration. He was given intravenous 0.9% saline and admitted to the ward. There, he was noted to have normal skin turgor and good peripheral perfusion. Biochemistries confirmed hypernatraemia (table 2), which normalised over the following 48 hours with intravenous fluids and recommencement of enteral feeding. There were no further episodes of diarrhoea and vomiting in the ward. His urinary sodium, obtained at admission, later returned markedly elevated at 321 mmol/L. No concomitant urinary creatinine measurement had been obtained, thus FENa could not be calculated. Urine osmolality was 702 mOsm/kg, indicating that sodium and accompanying anion constituted almost all of the osmotically active substances in the urine. Based on these measurements, suspicion of salt poisoning was raised, but vigorously denied. Social services were involved and he was discharged with weekly monitoring of plasma sodium.

He re-presented 1 month later following a reported 4-hour episode of vomiting. Again, there were no clinical features of dehydration. His weight was 4.82 kg, which was later compared with a weight of 5.29 kg, when plasma sodium had normalised.

On this occasion, comprehensive biochemistries were obtained and the FENa was elevated (table 2). Subsequent paired samples continued to demonstrate persistently high FENa (4.8%–6.2%) with otherwise normal renal function. His plasma sodium slowly normalised over the following 3 days. Forensic investigations later discovered excess salt in a jar with milk powder used for preparation of his milk feed.

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