A 23 Year old Woman with a Closed Head Injury who Developed Rapidly Fluctuating Sodium Levels

CASE REPORT

A 23-year-old woman was admitted to the intensive care unit (ICU) with a closed head injury. Her progress had been uneventful until day 11 when a nosocomial pneumonia developed which was treated with intravenous Timentin® (ticarcillin sodium and potassium clavulanate) at a dose of 3.1 g in 100 mL of normal saline, 6-hourly. Later that day she had a large diuresis, raising the possibility of diabetes insipidus (DI). A venous blood sample was taken at 1600 hr. The plasma sodium concentration at this stage was 160 mmol/L, whereas earlier that day (i.e. 0600 hr) it had been 143 mmol/L (Table 1). As the results suggested the diagnosis of DI, a 5% dextrose infusion was commenced at a rate of 100 mL/hr. However, no further episodes of polyuria were observed and a repeat venous blood sample taken at 2100 hr revealed a plasma sodium of 123 mmol/L.

As the patient's neurological status had remained unchanged, the rapid change in the sodium values lead us to the most likely diagnosis. A venous biochemical analysis was performed at 0146 hr. The biochemical results throughout this period are shown in Table 1.

Name	Age	Sex	Time of Collection	Date
Ms. C. K.	23	F		25.03.00

	0600	**1600**	**2100**	**0146**	**hours**	
Sodium	143	160	123	138	mmol/L	(135 - 145)
Potassium	3.9	3.6	3.0	3.5	mmol/L	(3.2 - 4.5)
Chloride	109	113	88	105	mmol/L	(100 - 110)
Bicarbonate	20	19	23	23	mmol/L	(22 - 33)
Anion Gap	14	28	12	10	mEq/L	(4 - 13)
Urea	9.1	7.3	5.4	6.5	mmol/L	(3.0 - 8.0)
Creatinine	0.07	0.04	0.05	0.05	mmol/L	(0.05 - 0.10)
Glucose	8.3	6.1			mmol/L	(3.0 - 6.0)
Total Protein	85	66			g/L	(62 - 83)
Albumin	40	29	34		g/L	(33 - 47)
Lactate		1.1	1.0		mmol/L	(0.7 - 2.5)

Figure 1. Plasma biochemical profile performed on venous blood taken from the patient at 0600, 1600, 2100 and 0146 hours

Diagnosis: Factitious hypernatraemia and elevated anion gap due to ticarcillin contamination (1600 hr sample) and hyponatraemia due to dextrose contamination (2100 hr sample).

Factitious biochemical results are uncommon in intensive care practice due to the widespread use of arterial cannulation for blood sampling, reducing the incidence of contamination with intravenous infused fluid. In our patient the dramatic reduction in plasma sodium from 1600 to 2100 hr (i.e. after only 5 hours) raised the possibility of an erroneous result. The sample taken at 2100 hr was noted to have been collected from the arm receiving the dextrose infusion. Re-examination of the results from the sample taken at 1600 hr revealed a raised anion gap with a normal lactate level as well as hypernatraemia (Figure 1). It was noted that this sample had been collected from the same arm receiving the Timentin® infusion. The plasma sodium concentration measurement was then repeated on a venous specimen from an uncontaminated site at 0146 hr on 26.03.00 and was recorded as 138 mmol/L.

It was believed that the blood sample collected at 1600 hr was contaminated by intravenous fluid containing a high sodium concentration (15.6 mmol of sodium ticarcillin in 3.1g of timentin when diluted in 100 ml of normal saline yields a final sodium concentration of approximately 300 mmol/L). The increased anion gap was attributable to the presence of the unmeasured ticarcillin anion. Although true elevations in both sodium concentration and anion gap could result from an intravenous infusion of Timentin,[1] a dose of 3.1g of Timentin would only result in very small elevations in both anion gap and sodium concentrations (e.g. approximately 1-2 mEq/L) at steady state.[2] There were no other anion sources accounting for the raised anion gap.

The hyponatraemia in the blood sample taken at 2100 hr was thought to have been caused by contamination with 5% dextrose. Although no glucose measurements were available from this sample to substantiate this, we arrived at this conclusion by examining the changes in fluid balance and electrolytes. A decline in plasma sodium concentration of 37 mmol/L in 5 hours with only 500ml of 5% dextrose infused (containing a glucose concentration of 300 mmol/L) suggested that the plasma sodium concentration of 123 mmol/L was an artifactual measurement. For this to have been a true reduction in plasma sodium, a free water infusion of approximately 10 L or an increase of plasma glucose of approximately 100 mmol/L would have been required.[3,4] Similarly, a reduction in glucose concentration of 50 mmol/L or a loss of free water of 4 L would have been required to explain the change in plasma sodium from 123 (at 2100 hr) to 138 mmol/L at 0146 hr).[3,4] These changes were not possible with the volume and type of fluid administered. The alterations in total protein, albumin, urea and creatinine were also consistent with sample dilution. Had these been true changes in plasma sodium concentrations, neurological manifestations (which were absent) would also have developed.

This report highlights once again the importance of proper sampling of blood for biochemical analysis. Whilst artifactual sodium, potassium and calcium abnormalities have been described secondary to errors in sampling of blood, spurious anion gap elevation of this nature has not been reported. It is also important to emphasise that when laboratory data are incompatible with the clinical presentation, erroneous results due to sampling or collection errors should be considered.

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