An unusual case of hyponatraemia in diabetic ketoacidosis

P J Twomey, J Cordle, D R Pledger, Y Miao

CASE REPORT

Choleserolaemia when lipoprotein X is formed as a result of primary
and triglyceride concentrations and rarely hypercholester-

osis. Because hypertriglyceridaemia and hyper-

proteinemia are not rare in clinical practice, sodium
methods that do not contain a dilution step are preferable.4

Our case has two unusual features. First, the pseudohypo-
natraemia is not explained by the triglyceride and total
protein concentrations and disappeared when the glucose
returned to the euglycaemic range. Second, the older Katz
equation5 proved to be the most accurate when the indirect
method was used to estimate the euglycaemic sodium
concentration. This raises several questions: (1) does DKA
have an addition effect on either sodium metabolism or
sodium measurement in addition to the effect of hypergly-
caeemia alone as recently assessed by Hillier? There are
theoretical analytical reasons to believe that low bicarbonate
concentrations and acidosis may result in a relatively lower
indirect sodium than expected when compared with a direct
measurement.6 (2) It would be expected that the Katz
equation would work best with the direct sodium because
this equation was empirically modelled. (3) Are these
equations valid using modern technology—the Hillier paper
used the non-routine flame photometry methodology. (4)
The calibration of direct ISEs may only be valid in “normal”
sera. (5) Such population based equations may not perform
satisfactorily in each individual situation.

Ideally, physicians should know the sodium method used
by their laboratory. However, it is unlikely that they will
always know or appreciate associated issues, so that it may
sometimes be necessary for the chemical pathologist to
facilitate optimal patient care, especially when significant
hypertriglyceridaemia or hyperproteinemia exists. It is
important to elucidate the cause(s) of hyponatraemia to
ensure the appropriate interpretation of results and that
the appropriate therapeutic action is taken.7

Abbreviations: DKA, diabetic ketoacidosis; ISE, ion specific electrode

after excluding all known effects, the cause may remain unexplained. In addition, this case raises the question of the validity of derived equations using modern technology and we suggest that further research is carried out to answer this question in patients with DKA and hyperosmolar non-ketotic hyperglycaemia.

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<table>
<thead>
<tr>
<th>Analyte</th>
<th>Presenting concentrations (0 hours)</th>
<th>Intermediate concentrations (3 hours)</th>
<th>Glycaemic stable concentrations (17 hours)</th>
<th>Katz predicted glycaemic stable concentration (mmol/l)</th>
<th>Hillier predicted glycaemic stable concentration (mmol/l)</th>
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</thead>
<tbody>
<tr>
<td>Glucose</td>
<td>47.7 mmol/l</td>
<td>7.8 mmol/l</td>
<td>147</td>
<td>164</td>
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<td>Direct sodium</td>
<td>135 mmol/l</td>
<td>137 mmol/l</td>
<td>139</td>
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<tr>
<td>Indirect sodium</td>
<td>127 mmol/l</td>
<td>136 mmol/l</td>
<td>3.6 mmol/l</td>
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<tr>
<td>Direct potassium</td>
<td>6.9 mmol/l</td>
<td>4.5 mmol/l</td>
<td>3.7 mmol/l</td>
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<tr>
<td>Indirect potassium</td>
<td>6.8 mmol/l</td>
<td>4.4 mmol/l</td>
<td>3.7 mmol/l</td>
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<tr>
<td>Total protein</td>
<td>96 g/l</td>
<td>62 g/l</td>
<td>53 g/l</td>
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<tr>
<td>Triglycerides</td>
<td>2.08 mmol/l</td>
<td>1.97 mmol/l</td>
<td>1.3 mmol/l</td>
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<td>Cholesterol</td>
<td>5.32 mmol/l</td>
<td>3.63 mmol/l</td>
<td>2.3 mmol/l</td>
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<tr>
<td>Osmolality</td>
<td>336 mOsmol/l</td>
<td>314 mOsmol/l</td>
<td>285 mOsmol/l</td>
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<td>Actual sodium gap</td>
<td>8 mmol/l</td>
<td>3 mmol/l</td>
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<td>Pseudohyponatraemia effect</td>
<td>2 mmol/l</td>
<td>–2 mmol/l</td>
<td>–3 mmol/l</td>
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<td>Adjusted difference</td>
<td>6 mmol/l</td>
<td>5 mmol/l</td>
<td>1 mmol/l</td>
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</table>

**REFERENCES**