Body fluid abnormalities in severe hyperglycemia in patients on chronic dialysis: review of published reports

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Abstract

Reports of dialysis-associated hyperglycemia (DH) were compared to reports of diabetic ketoacidosis (DKA) and nonketotic hyperglycemia (NKH) in patients with preserved renal function. Average serum values in DH (491 observations), DKA (1036 observations), and NKH (403 observations) were as follows, respectively: glucose, 772, 649, and 961 mg/dl; sodium, 127, 134, and 149, mmol/l; and tonicity, 298, 304, and 355 mOsm/kg. Assuming that euglycemic (serum glucose, 90 mg/dl) values were the same (sodium, 140 mmol/l; tonicity, 285 mOsm/kg) for all three states, the hyperglycemic rise in the average serum tonicity value per 100-mg/dl rise in serum glucose concentration was 1.9 mOsm/kg in DH, 3.5 mOsm/kg in DKA, and 8.1 mOsm/kg in NKH. Neurological manifestations in DH patients were caused by coexisting conditions (ketoacidosis, sepsis, and neurological disease) in most instances, and by severe hypertonicity (>320 mOsm/kg), with clearing after insulin administration, in a few instances. In 148 episodes of DH corrected with insulin only, the mean increase in serum sodium per 100-mg/dl decrease in serum glucose (Δ[Na]/Δ[Glu]) was −1.61 mmol/l. In agreement with theoretical predictions, Δ[Na]/Δ[Glu] was numerically smaller in patients with edema than in those with euvolemia. The average hyperglycemic increase in extracellular volume, calculated from changes in serum sodium concentration during correction of DH using insulin alone, was 0.013 l/l per 100-mg/dl increase in serum glucose concentration. A small number of DH patients presented with pulmonary edema rectified by insulin alone. DH causes modest hypertonicity, with few patients having neurological manifestations caused usually by other coexisting conditions. In contrast to DKA or NKH, which usually presents with hypovolemia, DH causes hypervolemia manifested occasionally by pulmonary edema. Insulin is adequate treatment for DH.

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1. Introduction

Dialysis-associated hyperglycemia (DH) causes potentially different solute and fluid disturbances from hyperglycemia developing in patients with preserved renal function because it is not complicated by large osmotic diuresis. Hyperglycemia should cause hypertonic states in both patients on dialysis and those with preserved renal function. However, unlike severe hyperglycemia in preserved renal function, which routinely causes clinically significant extracellular (EC) volume deficits, DH causes EC expansion. The baseline status of EC volume is theoretically a major determinant of the degree of both hypertonicity and Ec volume expansion in DH, with edematous patients predicted to have greater hypertonicity and greater EC hypervolemia than euvolemic or...
hypovolemic subjects with the same degree of hyperglycemia (Tzamaloukas et al., in press).

In this report, we analyzed published reports on DH. We addressed three questions: (a) Do laboratory findings conform with theoretical predictions of solute and fluid abnormalities in DH? (b) Are specific predictions of the magnitude of body solute and fluid abnormalities in DH translated into clinical manifestations? (c) Do abnormalities in tonicity and EC volume differ between DH and hyperglycemia developing in patients with intact renal function?

The level of evidence of reports on severe hyperglycemia tends to be low because such reports are routinely retrospective and observational. With this proviso in mind, we analyzed published reports of severe DH and compared their findings on disturbances in body solute and fluids to those of major published studies in patients with preserved renal function and either diabetic ketoacidosis (DKA) or non-ketotic hyperglycemia (NKH). Based on this analysis, we developed a treatment plan for DH.

2. Tonicity in hyperglycemic syndromes

2.1. Tonicity and related clinical manifestations upon presentation with hyperglycemia

Osmolarity and tonicity are related but not synonymous. Serum osmolality is determined by the sum of all solutes in the serum, while tonicity, or effective osmolality, is the part of total osmolality contributed by EC solutes having difficulty crossing cell membranes and therefore causing (when their EC concentration changes) steady-state fluid shifts between the EC compartment and the intracellular (IC) compartment. A high urea concentration is routinely encountered in DH (Tzamaloukas et al., 2004) and contributes to total osmolality, but does not contribute to tonicity, because urea is distributed in total body water (McCurdy, 1970). The parameter of interest in this study is tonicity because it is the source of changes in cell volume and of resulting neurological manifestations in hyperglycemia (Pollock & Arieff, 1980). Tonicity was estimated from the formula: Tonicity(mOsm/kg)=2×[serum sodium](mmol/l)+[serum glucose]/18, where serum glucose is expressed in milligrams per deciliter (McCurdy, 1970).

Tables 1–3 show serum levels of glucose, sodium, and tonicity in DH, DKA, and NKH, respectively. DH cases from patients on peritoneal dialysis and hemodialysis are analyzed together in Table 1 because there are no differences in serum sodium concentration and tonicity when the degree of hyperglycemia is comparable between the two dialysis modalities (Rohrscheib et al., 2005).

Weighed mean serum glucose values at hyperglycemia differed between the three hyperglycemic syndromes presented in Tables 1–3. This finding complicated the comparison of the degree of hypertonicity. Fig. 1 shows estimates of increases in serum tonicity, normalized to those of increases in serum glucose, when baseline (at a serum glucose of 90 mg/dl) values for serum sodium (140 mmol/l) and tonicity (285 mOsm/kg) were the same in the three hyperglycemic syndromes. This assumption is reasonable because the average euglycemic serum sodium concentration in dialysis patients is normal regardless of their volume status (Ramdeen, Tzamaloukas, Malhotra, Leger, & Murata, 1998; Tzamaloukas & Avasthi, 1986, 1988; Tzamaloukas et al., 1995). Compared to the prediction of Katz (1973), the increase in tonicity per 100-mg/dl increase in serum glucose concentration was slightly lower in DH, larger in DKA, and greatly larger in NKH.

Another way to compare tonicity changes secondary to different degrees of hyperglycemia is to estimate serum sodium values at euglycemia using Katz’s Δ[Na]/Δ[Glu] value of −1.6 mg/dl per 100 mg/dl (Al-Kudsi et al., 1982). The calculated “corrected” serum sodium values were 137 mmol/l in DH, 143 mmol/l in DKA, and 163 mmol/l in NKH. Thus, for the same degree of hyperglycemia, hypertonicity is lowest in DH and highest in NKH. The differences reflect, almost certainly, differences in external fluid and solute balance between the three hyperglycemic syndromes (Tzamaloukas, 1982; Tzamaloukas et al., in press). In the case of NKH, the large value of the increase in tonicity, in comparison to both predicted increase and the increase in the two other major hyperglycemic syndromes, also reflects, in all probability, bias in selecting symptomatic cases of NKH for publication.

The main clinical manifestations of hypertonicity are neurological and result from a decrease in brain cell volume (Pollock & Arieff, 1982). Hypertonic neurological symptoms are usually absent in DH or NKH when serum sodium concentration is low and tonicity is <320 mOsm/kg (Daugirdas et al., 1989; Elisaf, Papagalanis, & Siamopoulos, 1993; Popli et al., 1990; Siamopoulos, Elisaf, & Pappas, 1992). The majority of patients with severe DH had only modest hypertonicity and no symptoms (Al-Kudsi et al., 1982; Krediet, Struijk, & Arisz, 1986; Tzamaloukas & Avasthi, 1986). Small numbers of patients with DH and severe hypertonicity developed neurological manifestations (coma and seizures) corrected by insulin. Hypertonicity in these patients was caused by either the use of hypertonic dextrose-enriched dialysate—mostly in peritoneal dialysis (Boyer, Gill, & Epstein, 1967; Gault, Ferguson, Sidhu, & Corbin, 1971; Handa & Kushner, 1968; Tzamaloukas, Levinstone, & Gardner, 1982; Tzamaloukas et al., 2004; Whang, 1967), with one case developing in a hemodialysis patient (Potter, 1966)—or pancreatitis (Ender, Howard, & Rosenberg, 1987). In addition to hypertonicity, neurological manifestations occurring in patients with DH may result from either DKA (Bohn & Daneman, 2002; Finberg, 1996; Tzamaloukas et al., 2004) or an underlying disease, such as sepsis or organic neurological syndrome (Tzamaloukas et al., 2004). The occurrence of neurological manifestations in DH
patients in the absence of severe hypertonicity should, therefore, intensify the search for DKA and underlying infectious or neurological diseases.

Neurological manifestations with modest elevations in tonicity and no other cause are rare in DH (Tzamaloukas & Avasthi, 1986; Tzamaloukas et al., 2004; Whang, 1967) or NKH. Maccario (1968) reported six patients with NKH and neurological manifestations that abated after correction of hyperglycemia. These patients had the following presenting serum values: glucose, 43±13 mmol/l (778±241 mg/dl); sodium, 129±5 mmol/l; tonicity, 302±9 mOsm/kg. The highest tonicity was 316 mOsm/kg. These findings suggest

<table>
<thead>
<tr>
<th>Reference</th>
<th>Glucose (mg/dl)</th>
<th>Glucose (mmol/l)</th>
<th>Sodium (mmol/l)</th>
<th>Tonicity (mOsm/kg)</th>
<th>N</th>
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<tbody>
<tr>
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<td>3400</td>
<td>189</td>
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<td>Whang, 1967</td>
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<td>62</td>
<td>125</td>
<td>316</td>
<td>1</td>
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<tr>
<td>Handa &amp; Kushner, 1968</td>
<td>1870</td>
<td>104</td>
<td>138</td>
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<td>Gault et al., 1971</td>
<td>977±652</td>
<td>54±36</td>
<td>143±4</td>
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<tr>
<td>Al-Kudsi et al., 1982</td>
<td>1174±248</td>
<td>65±14</td>
<td>125±5</td>
<td>316±13</td>
<td>12</td>
</tr>
<tr>
<td>Kaldany et al., 1982</td>
<td>871±211</td>
<td>48±12</td>
<td>124±3</td>
<td>297±12</td>
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<tr>
<td>Tzamaloukas et al., 1982</td>
<td>994±275</td>
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<td>124±4</td>
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<tr>
<td>Montoliu &amp; Revert, 1985</td>
<td>1170±18</td>
<td>65±1</td>
<td>114±1</td>
<td>293±1</td>
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<tr>
<td>Krediet et al., 1986</td>
<td>617±165</td>
<td>34±9</td>
<td>127±5</td>
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<td>Ender et al., 1987</td>
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<td>116</td>
<td>332</td>
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</tr>
<tr>
<td>Tzamaloukas &amp; Avasthi, 1988a</td>
<td>932±263</td>
<td>52±15</td>
<td>124±6</td>
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<tr>
<td>Tzamaloukas &amp; Avasthi, 1988b</td>
<td>716±218</td>
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<td>126±7</td>
<td>291±11</td>
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<td>Elia et al., 1993</td>
<td>920</td>
<td>51</td>
<td>133</td>
<td>317</td>
<td>1</td>
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<tr>
<td>Tzamaloukas et al., 2004</td>
<td>913±197</td>
<td>51±11</td>
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<td>Rohrscheib et al., 2005c</td>
<td>997±86</td>
<td>55±5</td>
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<td>305±8</td>
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<tr>
<td>Rohrscheib et al., 2005d</td>
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<td>34</td>
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<tr>
<td>Rohrscheib et al., 2005e</td>
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<td>39±14</td>
<td>130±7</td>
<td>300±11</td>
<td>75</td>
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<tr>
<td>Rohrscheib et al., 2005f</td>
<td>682±220</td>
<td>38±12</td>
<td>129±5</td>
<td>296±10</td>
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</tr>
<tr>
<td>Mean</td>
<td>772±247</td>
<td>43±14</td>
<td>127±6</td>
<td>298b</td>
<td>491</td>
</tr>
</tbody>
</table>

a Patients with ketoacidosis.

b Patients with NKH.

c Peritoneal dialysis patients with ketoacidosis.

d Hemodialysis patients with ketoacidosis.

e Peritoneal dialysis patients with NKH.

f Hemodialysis patients with NKH.

Missing data from one study did not allow the calculation of weighed standard deviation.
that the relationship between hyperglycemia and neurological manifestations, particularly changes in brain solute and brain function caused by hyperglycemia, will require further study.

2.2. Change in tonicity during treatment of DH

The use of insulin as the sole treatment for DH allows the testing of formulas predicting \( \frac{\Delta [Na]}{\Delta [Glu]} \) that were derived from the principle of body fluid distribution (Tzamaloukas et al., in press). The relation \( \Delta [Na]/\Delta [Glu] \) in studies of hyperglycemia in patients with preserved renal function (Hillier, Abbott, & Barrett, 1999; McNair, Madsbad, Christiansen, Christensen, & Transbol, 1982; Nanzi, 1981; Strand, Garcia, & Costales, 1987) was influenced by both the principle of body fluid distribution and unknown amounts of osmotic diuresis and fluid intake. Therefore, these studies are not appropriate for testing standard formulas predicting \( \Delta [Na]/\Delta [Glu] \) (Kashyap, Oster, & Singer, 1999; Kurtz & Nguyen, 2005; Oster & Singer, 1999; Tzamaloukas, 1982, 1988).

Table 3 shows studies reporting changes in serum sodium and glucose concentrations during the treatment of DH with insulin. Tonicity decreased with correction of hyperglycemia in all studies. The weighed average \( \Delta [Na]/\Delta [Glu] \) value was almost identical to Katz’s (1973) predicted value of \(-1.6 \text{ mmol/l per 100 mg/dl}\). However, the range of \( \Delta [Na]/\Delta [Glu] \) values was wide.

We tested whether the variation in \( \Delta [Na]/\Delta [Glu] \) values in DH studies is consistent with theoretical predictions. One prediction is that edematous states are associated with low numerical values of the ratio \( \Delta [Na]/\Delta [Glu] \) (Tzamaloukas et al., in press). The mean value of \( \Delta [Na]/\Delta [Glu] \) was \(-1.04\pm0.23 \text{ mmol/l per 100 mg/dl}\) in four patients with severe edema (Tzamaloukas et al., 1982, 2004) and was \(-1.65\pm0.08 \text{ mmol/l per 100 mg/dl}\) in five patients at or close to their dry weights (Tzamaloukas et al., 1982) \((P<.001)\). EC volume evaluations were not reported in other studies.

To test the prediction that the ratio \( \Delta [Na]/\Delta [Glu] \) decreases numerically as hyperglycemia progresses (Robin et al., 1979), we analyzed the relationship between \( \Delta [Glu] \) and \( \Delta [Na]/\Delta [Glu] \) in the studies reported in Table 4. These studies exhibited a range of \( \Delta [Glu] \) values between \(-414 \text{ and } -1209 \text{ mg/dl}\), with a midpoint value of \(-811.5 \text{ mg/dl}\) between the two extremes. By Wilcoxon signed rank test, the \( \Delta [Na]/\Delta [Glu] \) values of patients with \( \Delta [Glu]<811.5 \text{ mg/dl} \) \((n=103)\) and those with \( \Delta [Glu]>811.5 \text{ mg/dl} \) \((n=45)\) did not
differ, although the average value of patients with Δ[Glu]< 811.5 mg/dl was larger (−1.66±0.28 vs. −1.49±0.32 mmol/l per 100 mg/dl). Whether much larger Δ[Glu] values produce Δ[Na]/Δ[Glu] values that verify theoretical predictions is currently unknown.

The potential sources of error in studies reporting initial and final serum glucose and sodium measurements during the treatment of DH with insulin are worth noticing. Changes in the external balance of water and sodium could have pronounced effects on the ratio Δ[Na]/Δ[Glu] (Kurtz & Nguyen, 2005; Tzamaloukas, 1982). Only one study reported no fluid intake or urine output and no change in body weight during treatment (Tzamaloukas et al., 1982). Whether patients in the other studies had received fluid or had some urine output during treatment is not known.

A second potential error is inherent in the formula Δ[Na]/Δ[Glu]. The numerator of this formula is a small number that can cause a substantial error if it is offset by even 1 mmol/l, particularly when Δ[Glu] is also relatively small. To test for this type of error, one study compared change in serum chloride (Δ[Cl]) to change in serum sodium (Δ[Na]), and the formula Δ[Cl]/Δ[Glu] to the formula Δ[Na]/Δ[Glu] in DH treated with insulin. The comparison was based on a concept stating that the determinants of changes in serum sodium and chloride concentrations secondary to DH are exactly the same (Kapner & Tzamaloukas, 1991). The differences between the values Δ[Na] and Δ[Cl] and between the values Δ[Na]/Δ[Glu] and Δ[Cl]/Δ[Glu] were not significant (Tzamaloukas et al., 2004).

The factors affecting changes in tonicity during insulin treatment of DH require further study. Nevertheless, it appears that Katz’s (1973) Δ[Na]/Δ[Glu] ratio of −1.6 mmol/l per 100 mg/dl represents a useful approximation of the average change in tonicity in DH, but not in individual values.

Hypertonicity causes thirst and fluid consumption (Fitzsimons, 1961). Intense thirst was reported by approximately 20% of patients with severe DH (Tzamaloukas et al., 2004). An association between large fluid gains and DH has been documented (Ifudu, Dulin, & Friedman, 1994; Ramdeen et al., 1998; Tzamaloukas & Avasthi, 1986; Tzamaloukas et al., 1995). Normal tonicity, seen in approximately 15% of patients presenting with DH, provides a strong but insensitive indirect indication of fluid consumption. The development of hypotonicity during treatment with only insulin, encountered in approximately 50% of the cases (Tzamaloukas et al., 2004), is a more sensitive indirect indicator of fluid consumption during the development of DH. Determination of the exact frequency of thirst in patients with DH will require a prospective study.

3. EC volume abnormalities in hyperglycemia

3.1. EC volume in DH

Table 5 shows fractional increases in EC volume at the peak of hyperglycemia [(ΔV/ΔV1)/Δ[Glu]] calculated from the change in serum sodium concentration during the correction of hyperglycemia with insulin infusion only in the studies shown in Table 4. There is consistency between the different studies. In a hypothetical euvoletic patient with 14 l of euglycemic EC volume who develops a 66.7-mmol/l (1200 mg/dl) increase in serum glucose concentration, the calculated increase in EC volume using the observed average (ΔV/ΔV1)/Δ[Glu] value of 0.013 l/l per 100 mg/dl from Table 5 would be 2.2 l. The corresponding value computed from a theoretical model (Tzamaloukas et al., in press) was 2.3 l.

Theoretically, the slope of the relationship (ΔV/ΔV1)/Δ[Glu] declines slowly as serum glucose increases and different states of euglycemic EC volume produce different slopes of the relationship (ΔV/ΔV1)/Δ[Glu] (Tzamaloukas et al., in press). In agreement with the second prediction, the values (ΔV/ΔV1)/Δ[Glu] calculated in the four patients with severe edema were lower than those calculated in the five patients at dry weight (0.008±0.002 vs. 0.014±0.001 l/l per 100 mg/dl, respectively; P<0.001).

The fractional change in EC volume during the development or correction of DH can be calculated using the changes in serum chloride level (Seldin & Tarail, 1950) and in sodium concentration. Even though the two estimates of fractional change in EC volume correlate, the fractional change computed from serum chloride concentration changes is greater by approximately 20–30% than the one computed from serum sodium concentration changes (Tzamaloukas et al., 2004). This finding is consistent with previous reports of a lower apparent volume of distribution for chloride than for sodium in hypertonic EC expansion (Tzamaloukas, 1983; Wolf & McDowell, 1954).

3.2. EC volume changes in hyperglycemia and clinical manifestations

Fig. 2 shows calculated average changes in EC volume following the same rise in serum glucose concentration
The fractional increase in EC volume from euglycemic EC volume to hyperglycemic EC volume ($\Delta V/V_i$) was calculated from the change in serum sodium concentration during correction of hyperglycemia using the formula: $\Delta V/V_i = \frac{[Na]_1}{[Na]_2} - 1$, where $[Na]_1$ and $[Na]_2$ are, respectively, the serum sodium concentrations at euglycemia (after correction of hyperglycemia with insulin only) and hyperglycemia (Feig & McCurdy, 1997).

<table>
<thead>
<tr>
<th>Reference</th>
<th>Δ[Glu] (mg/dl)</th>
<th>$\Delta V/V_i$</th>
<th>Δ[Glu] per $V_i$ per 100 mg/dl</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gaut et al., 1971</td>
<td>−645</td>
<td>0.109</td>
<td>0.017</td>
</tr>
<tr>
<td>Kaldany et al., 1982</td>
<td>−724±224</td>
<td>0.096±0.029</td>
<td>0.014±0.004</td>
</tr>
<tr>
<td>Tzamaloukas et al., 1982</td>
<td>−792±249</td>
<td>0.093±0.043</td>
<td>0.012±0.003</td>
</tr>
<tr>
<td>Tzamaloukas &amp; Avasthi, 1988</td>
<td>−702±214</td>
<td>0.083±0.027</td>
<td>0.012±0.004</td>
</tr>
<tr>
<td>Tzamaloukas &amp; Avasthi, 1988</td>
<td>−522±126</td>
<td>0.072±0.015</td>
<td>0.014±0.002</td>
</tr>
<tr>
<td>Tzamaloukas et al., 2004</td>
<td>−730±229</td>
<td>0.092±0.036</td>
<td>0.013±0.002</td>
</tr>
<tr>
<td>Mean&lt;sup&gt;4&lt;/sup&gt;</td>
<td>−629±210</td>
<td>0.082±0.031</td>
<td>0.013±0.003</td>
</tr>
</tbody>
</table>

<sup>a</sup> DKA.  
<sup>b</sup> NKH.  
<sup>c</sup> Weighed for the number of observations in each study.

The development of pulmonary edema in DH can also be affected by a preexisting heart disease (Tzamaloukas et al., 1982). However, patients without heart disease may also develop pulmonary edema in DH (D’Elia et al., 1979). Other factors that may contribute to the development of this complication include (a) altered pulmonary capillary permeability in DKA (Brun-Buisson, Bonnett, Bergeret, Lemaire, & Rapin, 1985; Powner, Snyder, & Grenvik, 1975; Russel, Follansbee, & Mathay, 1981; Sprung, Rackow, & Fein, 1980) and (b) myocardial dysfunction due to impaired glucose utilization caused by insulin deficiency (Opie, 1969). Whether other factors, such as the rapidity of the development of hyperglycemia, also affect cardiovascular manifestations of DH is not known.
3.3. Principles of treatment

DH is unique among hyperglycemic syndromes because its pathogenesis may involve only abnormalities caused by the principle of body water distribution. Thirst can lead to net water gain in DH, but large losses of solute and water through osmotic diuresis do not develop. The treatment of DH, therefore, is simpler than that of hyperglycemia in patients with preserved renal function. The only treatment needed is insulin infusion with monitoring of the clinical signs of volume status and serum glucose and electrolytes. However, abnormalities in both tonicity and EC volume resulting from causes other than DH may be present and may require clinical attention.

Hypertonicity is usually modest in DH and causes neurological manifestations only in rare cases when it is greatly elevated from coexisting conditions causing external water loss. Other conditions (DKA and infections) are almost always present in DH patients with modest hypertonicity and neurological manifestations. Although infusion of insulin is usually the only measure needed to correct tonicity abnormalities in DH, small numbers of patients may require additional measures. Tonicity and serum sodium concentration at presentation can be used to guide the need for and the prescribed amount of water administration to these patients.

There are three possible combinations of tonicity and serum sodium concentration in DH:

(a) Tonicity is high, and serum sodium is high or even normal.

This combination, which is common in NKH (Table 3) but rare in DH (Table 1), is a sign of a large water deficit and signals the need for water administration. Katz’s \(\Delta[\text{Na}] / \Delta[\text{Glu}]\) formula can be used to estimate serum sodium concentration after correction of hyperglycemia. Total water deficit can then be calculated from the difference between this estimate and the desired (normal) serum sodium concentration, and from an estimate of total body water based on body weight (Adrogue & Madias, 2000).

(b) Tonicity at presentation is normal or low, and serum sodium concentration is low.

This combination is also unusual and indicates relative water excess, which can be removed by dialysis during or after correction of DH with insulin.

(c) Tonicity at presentation is high, and serum sodium concentration is low.

This is the usual combination in DH. Katz’s formula can provide an estimate of serum sodium level after the correction of DH with insulin. Water deficit or excess is small in this case and does not usually require special treatment.

The second part of the evaluation of patients with DH is the measurement of total body sodium. This evaluation is performed by history taking and physical examination. Most patients have no clinical signs of volume depletion or circulatory overload and require only insulin therapy. A very small number of patients with concomitant extrarenal fluid losses may develop clinical signs of hypovolemia at presentation with DH or during its treatment with insulin. Modest boluses of saline (e.g., 250 ml), with clinical evaluation of volume status after each bolus, are indicated in this case. Finally, a small number of patients with DH may present with pulmonary edema. There is evidence that insulin therapy alone is sufficient treatment for this syndrome. Whether insulin administration should be the only treatment in an individual case, or more drastic measures (emergency dialysis) are needed, is a judgment call. Emergency dialysis or ultrafiltration should be considered in patients with pronounced edema, severe hyperglycemia, and severe pulmonary edema.

Acknowledgment

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References


