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Plasma glucose, lactate, sodium, and potassium levels in children hospitalized with acute alcohol intoxication

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Received 26 October 2009; received in revised form 8 July 2010; accepted 8 July 2010

Abstract

The aim of our research was to study prevalence of changes in plasma levels of lactate, potassium, glucose, and sodium in relation to alcohol concentration in children hospitalized with acute alcohol intoxication (AAI). Data from 194 under 18-year-old children hospitalized to the two only children’s hospital in Estonia over a 2-year period were analyzed. The pediatrician on call filled in a special form on the clinical symptoms of AAI; a blood sample was drawn for biochemical tests, and a urine sample taken to exclude narcotic intoxication. The most common finding was hyperlactinemia occurring in 66% of the patients (n = 128) followed by hypokalemia (<3.5 mmol/L) in 50% (n = 97), and glucose above of reference value (>6.1 mmol/L) in 40.2% of the children (n = 78). Hypernatremia was present in five children. In conclusion, hyperlactinemia, hypokalemia, and glucose levels above of reference value are common biochemical findings in children hospitalized with acute AAI.

Keywords: Acute alcohol intoxication; Hyperlactinemia; Hypokalemia; High glucose level; Hypernatremia

Introduction

The age of onset of alcohol consumption is becoming lower in several countries, including Estonia (Marchi et al., 2003; Paton, 1999; Schöberl et al., 2008). Studies showed that the average age at initial alcohol consumption was 11.3 years in girls and 10.7 years in boys, and, on average, 85.9% of 13–16-year-old adolescents in Estonia had consumed alcohol in their lifetime (Markina and Sahverdov-Zarkovski, 2007). According to many other similar studies in other countries, increasing alcohol consumption among under 18-year-old children is a growing problem (Madu and Matla, 2003; Meyer et al., 2007; Sutherland and Willner, 1998; Woolfenden et al., 2002).

As the proportion of alcohol consumption has gradually increased, medical problems caused by acute alcohol intoxication (AAI) are becoming more severe. Although there are lots of studies related to different aspects of AAI in children, only few of them have looked at the biochemical changes during acute AAI (Bradford, 1984; Lamminpää, 1995; Marchi et al., 2003; Roy et al., 2003). However, there are studies performed in healthy adult volunteers (Lionte et al., 2004) and from case-report series (Albers and van der Lely, 2004) and showing that lactic acidosis, hypernatremia, hypokalemia, and hypoglycemia or hyperglycemia do occur. Hypoglycemia has been found to be a severe problem in young children with acute AAI (Lamminpää, 1995). The clinical significance of hypoglycemia has been underlined especially in small children as this may rapidly deteriorate their general condition (Bradford, 1984; Lamminpää and Vilska, 1990). Hypokalemia has been considered to be an important factor for patients with alcohol withdrawal syndrome or delirium where hypokalemia may be a life-threatening factor (Stasiukyniene, 2002). Interestingly, very little attention has been paid to changes in potassium level testing in AAI in adults or children (Lamminpää and Vilska, 1990). Hyperaldosteronism is one potential reason to getting hypokalemia, what was observed to lead to hypokalemia and hypernatremia with AAI (Hirschl et al., 1994). Vomiting and diarrhea can change the volume of water in the body from acute intoxication of alcohol. Thereby the hypovolemia in one’s turns the rising of sodium level (Perkin et al., 2007).
The aim of the study was to investigate the prevalence of changes in plasma levels of glucose, lactate, potassium, and sodium in children hospitalized with AAI, and the impact of serum alcohol concentration on these changes.

Materials and methods

All children under 18 years old who were hospitalized with AAI over a 2-year period (December 2005–December 2007) in the two main children’s hospitals in Estonia, Children’s Clinic of Tartu University Hospital and Tallinn Children’s Hospital, were included into the study. On hospitalization, an anonymous encoded form on medical assessment was filled in by the pediatrician on call to affirm drunkenness of the child.

The medical staff assessed the general condition of the child, the level of consciousness, memory about last few hours, medical history, balance on walking, body temperature, rate of heart and blood pressure. Two venous blood samples were drawn from each studied child—serum for the measurement of alcohol, plasma for the measurement of lactate, potassium, glucose, and sodium concentrations. Biochemical tests were performed immediately after the collection of samples. Urine sample was also collected to exclude the use of narcotic substances. Results were divided into three groups based on the alcohol concentration in the blood:

- Group A: 20–150 mg/dL;
- Group B: 151–250 mg/dL;
- Group C: >250 mg/dL.

Reference values of the local laboratories were used to determine the abnormalities in biochemical results. Thus, hypokalemia was defined as plasma potassium level <3.5 mmol/L, hypoglycemia as plasma glucose levels <3.3 mmol/L, and hyponatremia as plasma sodium level <132 mmol/L. Above the upper reference value were hyperkalemia (>5.1 mmol/L), hypernatremia (>145 mmol/L), and hyperlactatemia (≥2.4 mmol/L). Reference values are shown in Table 1.

In Tartu University Hospital, glucose was determined using the glucose oxidase method Cobas Integra 400plus (Roche), lactate with enzymatic colorimetry Cobas Integra 400plus (Roche), and potassium and sodium with the ISE direct method AVL 988-4 (Roche). In Tallinn Children’s Hospital, glucose and lactate were determined with enzymatic colorimetry Cobas Integra 400plus (Roche), and potassium and sodium potentiometrically with ABL 700 Radiometer (Radiometer Analytical), and potassium and sodium potentiometrically with ABL 700 Radiometer (Radiometer Analytical). Serum ethanol concentration was determined using the enzymatic method TDxFLx (Abbott Diagnostics) in both hospitals. Both laboratories participate in the quality control program Labquality.

Children were divided into two age groups: 10.0–13.9 year olds and 14.0–17.9 year olds to simplifying the statistical analysis for multiplicity of the 13 and 14 year olds in the study.

Statistical analysis was performed using statistical program Statistica 8.0. Pearson correlation coefficient was used to assess bivariate relationships and Student’s t-test for comparing the results between two groups. P value <.05 was considered statistically significant.

The study was approved by the Ethics Review Committee on Human Research of the University of Tartu, and procedures were followed in accordance with the Helsinki Declaration of 1975, as revised in 1983.

Results

Subjects’ characteristics and symptoms

From December 2005 to December 2007, 226 children with suspected AAI were hospitalized to the two hospitals. Twelve children were excluded because their serum alcohol concentration was below 20 mg/dL, and they were without symptoms and were considered as not having used alcohol. Narcotic intoxication was confirmed with a rapid urine test in two children and those two were also excluded. Hence, 212 alcohol-intoxicated children were eligible for the study. There were 91 children from Tartu University Children’s Clinic and 123 from Tallinn Children’s Hospital. Data of 194 children remained for statistical analysis, as no tests had been done in 14 children because of the refusal of testing or mistakes in blood drawing, and in four children some biochemical tests had not been conducted. Hospitalized children were aged 10.0–17.9 years, with the mean age being 14.2 years. There were 94 (48.5%) children in the age group of 10.0–13.9 years and 100 (51.5%) children in the age group of 14.0–17.9 years. In total, there were 119 boys forming a male:female ratio of 1.6:1. The gender ratio was very similar in both age groups: 59 boys and 35 girls in the younger age group and 60 boys and 40 girls in the older age group.

The main observed symptoms in the children of different alcohol concentration groups are given in Table 2. Children’s symptoms occurred mildly in Group A. Intensity of symptoms was growing with alcohol concentration in serum. Disorientation, somnolence, and aggressiveness were more notable. Children often were in bad condition with alcohol concentration >250 mg/dL in serum; they were confused, imbalanced, or in coma. Coma was measured from 5 to 13 points by Glasgow Coma Scale.
Temperature as well as rate of heart and blood pressure were similar in all the three alcohol concentration groups. Mean temperature was 35.8°C (minimum 33; maximum 37.6), heart rate was 84.4 bpm (minimum 47; maximum 140), systolic blood pressure was 112 mm Hg (minimum 76; maximum 153), and diastolic blood pressure was 62 mm Hg (minimum 30; maximum 110).

**Biochemical test results**

The venous blood samples were drawn about 10—15 min after hospitalization. The mean time between the ingestion of alcohol and collection of samples was 3 h and 15 min (range 0.5—9 h). A summary of the serum ethanol and plasma glucose, lactate, potassium, and sodium concentrations of all subjects (n = 194) is given in Table 1.

**Lactate**

Hyperlactinemia (>2.4 mmol/L) occurred in 66% of the children. There was a statistically significant but relatively weak negative correlation (r = −0.31; P = .0001) between lactate concentration and age, that is, lactate values of older children were more within reference range, whereas increased lactate values were more common in younger children. This correlation remained significant after correcting for blood alcohol concentration.

**Potassium**

The mean serum potassium concentration (3.6 mmol/L) was very close to the lower reference limit of 3.5 mmol/L, and half of the children (50%) were hypokalemic (<3.5 mmol/L). There was a statistically significant but relatively weak positive correlation between plasma potassium concentration and age (r = 0.3; P < .0001), that is, the younger children had more hypokalemia (64%) than older children. This correlation remained significant after correcting for blood alcohol concentration.

Five children (2.6%) had hypokalemia in the critical value, for example, under 2.8 mmol/L. Critical value reporting was originally highlighted by Lundberg who defined critical value as a result suggesting that the patient was in imminent danger unless appropriate therapy was promptly initiated (Dighe et al., 2006).

**Glucose**

In the overall group, there was a statistically significant but relatively weak negative correlation between the glucose concentration and age (r = −0.21; P = .003), that is, glucose levels decreased with increasing age (Fig. 1). This correlation remained significant after correcting for blood alcohol concentration (Figs. 2 and 3).

In the older age group, plasma glucose levels tended to be within reference range, whereas in the younger age group there was a tendency toward increased glucose levels. As seen in the scatter plot, the glucose level above reference value (6.1 mmol/L) was evident in 78 (40.2%) of the children. In 29 (15%) children, the glucose level was equal or higher than 7 mmol/L. In older children, glucose values were more stable within reference values, except for very high values, more than 9 mmol/L that would have necessitated further studies in respect to glucose metabolism. None of our subjects were hypoglycemic, that is, had plasma glucose <3.3 mmol/L.

**Sodium**

In general, plasma sodium concentration was within reference range, although in five children it had relatively high values (above 150.0 mmol/L).

**Severity of alcohol intoxication**

Girls in younger age group of Group A of alcohol (n = 3) had significantly higher serum alcohol concentration than boys (n = 15; 140 mg/dL, P = .02), whereas opposite tendency was seen in older age group (117 vs. 126 mg/dL); however, the latter difference was not statistically significant.

The mean plasma lactate concentration was above the reference value (<2.4 mmol/L) in each alcohol concentration group, except in the girls in Group A. Plasma potassium concentration did not differ between the different alcohol concentration’s groups, but in the younger age children group there was a statistically significant but relatively weak negative correlation between the ethanol concentration and plasma potassium concentration values (r = −0.23; P = .028). The plasma glucose concentrations were slightly lower in girls in each alcohol concentration group compared with boys, but the differences were not statistically
significant. The mean plasma sodium concentration tended to be slightly higher in Group C (alcohol concentration > 250 mg/dL; see Table 3). The plasma sodium concentration correlation to alcohol concentration was statistically significant but relatively weak positive correlation ($r = 0.21; P = .023$). There was a strong correlation ($P = .006$) between the alcohol level above 150 mg/dL and sodium concentrations.

Fig. 1. The relationship between plasma glucose concentration and age ($r = -0.21; P = .003$) in alcohol-intoxicated children. log decage:logGL: $y = 1.075 - 0.1862 \times x; \quad r = -0.2155; \quad P = .0025; \quad r^2 = 0.0465$.

Fig. 2. The relationship between plasma potassium concentration and age ($r = -0.21; P = .003$) in alcohol-intoxicated children. log decage:logK: $y = 0.3691 + 0.3395 \times x; \quad r = 0.2920; \quad P = .00004; \quad r^2 = 0.0853$. 
Consumption of alcohol among children is a growing problem in many countries, as in Estonia. Our study involved a relatively big number of children: 214 drunken children in two hospitals during 2 years. Similar studies have shown much smaller groups of drunken children (Marchi et al., 2003; Woolfenden et al., 2002). The ratio of girls and boys was similar to other studies (Marchi et al., 2003; McIntosh et al., 2004), where generally more boys were involved. The mean age of drunken children in our study was similar to that in other studies, except that we had significantly less 16-year-old children.

We did not compare our results with other reports of effects of acute intoxication in young adults. Most of these studies have been done in the controlled situation in volunteers with mean alcohol concentration in the blood up to 80–150 mg/dL (Cameli et al., 2009; Davies and Bowen, 1999). The mean measured blood serum concentration of alcohol in our study subjects was 203 mg/dL, and therefore, we did not consider these two situations comparable. Most frequently, children in our study were hospitalized with alcohol concentration between 150 and 250 mg/dL. However, it is quite likely that most children with mild AAI will not go to the hospital because their general condition is not so much affected.

Alcohol consumption causes many changes in plasma glucose, lactate, sodium, and potassium concentrations and is a complex process. For normal ranges, we have used local laboratory reference values that are based on recommended international references (Burtis et al., 2006; Heil et al., 2004). We did not use the control group as most of our patients were generally healthy without major previous illnesses.

Hyperlactinemia was a common finding in our children, which was a feature similar to the other studies (McDonald et al., 1994; Umpierrez et al., 2000). One cause for the lactic acidosis is tissue hypoxia because of mild hypothermia or ethanol-induced central nervous system depression and

Table 3
Number of patients and average plasma glucose, lactate, sodium, and potassium concentrations in children as a function of serum BEC group

<table>
<thead>
<tr>
<th></th>
<th>Group A (serum BEC 20–150 mg/dL)</th>
<th>Group B (serum BEC 151–250 mg/dL)</th>
<th>Group C (serum BEC &gt;250 mg/dL)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Girls</td>
<td>Boys</td>
<td>Total</td>
</tr>
<tr>
<td>Number</td>
<td>13</td>
<td>25</td>
<td>38</td>
</tr>
<tr>
<td>Glucose (mmol/L)</td>
<td>5.58</td>
<td>6.08</td>
<td>5.91</td>
</tr>
<tr>
<td>Lactate (mmol/L)</td>
<td>2.14</td>
<td>2.78</td>
<td>2.56</td>
</tr>
<tr>
<td>Sodium (mmol/L)</td>
<td>143.4</td>
<td>141.9</td>
<td>142.4</td>
</tr>
<tr>
<td>Potassium (mmol/L)</td>
<td>3.61</td>
<td>3.67</td>
<td>3.65</td>
</tr>
</tbody>
</table>

BEC = blood ethanol concentration.
altered blood vessel tone (Lien and Mader, 1999). The serum lactate concentration above 5 mmol/L is considered to be dangerous because such lactate levels have been associated with increased 3- and 30-day mortality levels in intensive care patients with AAI (Stacpoole et al., 1994). In our study, there were three children with lactate levels above 5 mmol/L. Thus, serum lactate level should be measured in every child with AAI.

The other possible reason for lactic acidosis is enhanced anaerobic glucose metabolism, which leads to an increased lactate production.

According to the plasma potassium reference values (3.5–5.1 mmol/L), there was a tendency toward hypokalemia. The mean plasma potassium level was near the lower reference value –3.6 mmol/L, the lower quartile being 3.3 mmol/L and the upper quartile 3.9 mmol/L. Critically low potassium values, that is, <2.8 mmol/L, were present in 2.6% of children (n = 5) and required correction with a potassium infusion. Continuous monitoring of these five or hypokalemic patients was also necessary as they were disoriented, somnolent, and their mean body temperature tended to be lower (35.4°C) than in other children with normal potassium levels (mean temperature 35.8°C). No arrhythmias were reported in any children.

This might result from ethanol-induced lactic acidosis and development of high glucose level because these two factors may decrease the cellular level of potassium and its increased loss with urine and vomiting.

Many studies have found that hypoglycemia is a common problem in AAI (Hart and Frier, 1998; Kerr et al., 1990; Wilson et al., 1981). A large proportion of scientific articles support the idea that the reason for hypoglycemia resulting in AAI is the inhibition of glyconeogenesis and glycogenolysis by alcohol (Marshall et al., 2008; Siler et al., 1998). However, other opposing articles also exist (Mokada et al., 2004). In our study, we have also seen more a tendency toward to higher glucose level rather than hypoglycemia. Alcohol may increase glucose levels through inhibiting the basal insulin secretion although glucose-stimulated insulin secretion has been found not to be impaired from increasing cortisol secretion and enhancing glycogenolysis from lactate (Shin et al., 2002). Increased cortisol level in the stress situation is one of the most important factors in the mechanism of increasing glucose level.

There was also a tendency toward hypernatremia. However, in most cases, it was mild and only in five children the plasma sodium levels exceeded 150 mmol/L. The most likely cause for hypernatremia is not the primary sodium excess but the water deficit and hyperaldosteronism, which commonly occur in AAI.

Despite the good statistical significance of our findings, the correlation coefficients were relatively low (r = −0.3 lactate concentration and age; r = 0.3 potassium concentration and age; r = −0.21 glucose concentration and age; r = −0.23 ethanol and potassium concentrations; r = 0.21 ethanol and sodium concentrations).

Future research

We are planning to study those children also with regard to hormonal changes, particularly hypothalamic—pituitary—adrenal axis.

In conclusion, intoxication from alcohol is the main cause of changing of biochemical tests and it may depend on the ethanol concentration in the blood. The biochemical changing up to the critical value can occur in all blood ethanol concentrations but is particularly important with ethanol concentration above 150 mg/dL. Hypokalemia is relatively common in children hospitalized with A1 and is more pronounced in younger children, whereas hypoglycemia, at least in our study, was rare. We recommend that plasma potassium, sodium, glucose, and lactate levels should be measured in all children hospitalized with AAI.

Acknowledgments

The study was funded by Estonian Science Foundation Grant No. 6592 and by Basic funding grant of Tartu University (PARPA). Acknowledgments to the Tartu University Children’s Clinic and Tallinn Children’s Hospital for the cooperation in collecting data.

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