Elevated Postmortem Ethanol Concentrations in an Insulin-Dependent Diabetic

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Abstract

A 54-year-old woman (165 cm, 37 kg) was found dead in her home during a welfare check after not having been seen for at least three days. The body showed clear evidence of decomposition. Her head was in what appeared to be a pool of blood. The residence was clean, neat, and showed no evidence of violence. Insulin was found in the refrigerator, and syringes were in the kitchen cabinet. In agreement with these physical findings, her clinical history indicated that she suffered insulin-dependent diabetes mellitus. Autolytic changes were noticed at autopsy, and no obvious cause of death was demonstrated. The autopsy heart blood sample screened negative for cocaine and/or metabolite (benzoylecgonine), phenethylamines, opiates, and barbiturates by radioimmunoassay. The alcohol concentration was 0.51 g/dl in the blood, 0.04 g/dL in the brain, 0.08 g/dL in the liver, and 0.05 g/dL in the urine, and acetone levels were 42 mg/dL, 53 mg/dL, 14 mg/dL, and 19 mg/dL, respectively. Isopropanol was also present in all samples analyzed. The cause of death was ruled as metabolic acidosis due to diabetes mellitus. Possible bacterial postmortem production of ethanol is considered as an explanation for the increased concentration of ethanol found in the postmortem heart blood.

Introduction

Putrefaction is a highly variable process that depends not only upon environmental variables such as temperature and humidity, but also on the corpse itself (1). The process of decomposition can be affected by certain prior medical conditions of the decedent, such as diabetes (2). Bacteria from the intestinal flora migrate throughout the body during the first hours after death, depending upon ambient temperature (1). A wide number of species of bacteria and yeast can produce ethanol from glucose present in blood and tissues (3). Tissues and fluids with high glucose concentrations are more susceptible to postmortem production of ethanol, and urine and the more isolated tissues and fluids such as brain and vitreous humor are less susceptible (1). The probability of postmortem alcohol synthesis is also affected by the temperature of the location where the body remains, time between death and specimen collection, and the combination of both factors (4). Based on this information, the presence and reporting of alcohol concentrations in decomposed bodies presents a dilemma to the forensic toxicologist because it is not possible to know if or how much ethanol originated from ingestion and how much is from postmortem production. An atypical distribution of ethanol or its presence only in body fluids such as blood is normally considered an indication of postmortem production (3,5). In a retrospective study of autopsied medical examiner cases with mention of decomposition or putrefaction, Gilliland and Bost (5) classified the presence of ethanol as endogenously produced in 55 out of 286 cases. The mean blood ethanol concentration was 0.07 g/dL, and the highest blood ethanol concentration without history of having consumed any ethanol was 0.16 g/dL (5). The authors reported two additional cases with higher blood ethanol (0.24 g/dL and 0.27 g/dL), but they indicated that the absence of ethanol ingestion in these two cases was less reliable.

In general, in cases in which the presence of ethanol was due to postmortem production, ethanol was present in the blood and bile and accompanied by negative or significantly lower concentrations in the vitreous and urine (5). These results are in agreement with those previously found by Zumwalt and colleagues (3), in which the highest endogenous blood ethanol concentration reported was 0.22 g/dL with no ethanol detected in the urine.

Because glucose is the primary source of endogenous ethanol due to microbial production (3), it is possible that the ratios for the ethanol concentration among the tissues may differ when the decedent has a history of diabetes. Postmortem ethanol levels of an insulin-dependent diabetic are presented and discussed in this paper.

Methods

Samples

Sample organs from the decedent included the liver and brain. Also submitted were blood, urine, and stomach contents. The heart blood was preserved with potassium oxalate and sodium fluoride and refrigerated after being sampled. The
rest of the samples were frozen immediately after autopsy. The autopsy was performed within 24 h of the discovery of the body. Evidence from the scene suggested that the decedent had been dead approximately three days prior to being found. Brain and liver samples were diluted with deionized water (1+2) and ground to homogeneity.

Radioimmunoassay
A sample of the postmortem heart blood was screened for barbiturates, cocaine and/or benzoylecgonine, opiates, and methamphetamine (and/or related compounds) by RIA (radioimmunoassay) using DPC Coat-a-Count® in a Cobra® automated gamma-counter instrument.

Ethanol analysis
Samples of blood, liver, brain, and urine were analyzed in duplicate for occasionally consumed low-boiling-point alcohols (methanol, ethanol, propanol, and isopropanol) and ketones (acetone, methyl ethyl ketone).

For each sample, one analysis was performed with a Perkin-Elmer 8000 gas chromatograph with a flame-ionization detector (GC–FID), an HS-101 headspace sampler, and a 2.0-m x 6-mm i.d. glass column, 6.6% Carbowax 20M on Carbopak B. The second replica analysis was performed using a Perkin-Elmer AutoSystem XL GC–FID with TurboMatrix 110 headspace sampler and a capillary column (RTX BAC-1, 30-m, 0.32-mm i.d., 1.8-μm df).

The internal standard was 1-propranol. Fifty microliters each of the blood or the urine samples or tissue homogenates was diluted with 1.5 mL of a 2 g/dL 1-propanol solution in 20-mL capacity glass vials with aluminum caps fitted with rubber membranes.

For the Perkin-Elmer 8000 GC, chromatography conditions were as follows: the oven temperature was 80°C; the analysis time was 3.90 min; the injector temperature was 170°C; the detector temperature was 190°C; the injection time was 0.08 min; the equilibration time was 0.1 min; the pressurization time was 0.5 min; the thermostat temperature was 60°C; the thermostat time was 12 min; the needle temperature was 90°C; the withdrawal time was 0.2 min; the transfer line temperature was 60°C; the venting time was 0.3 min; and the flow rate was 30 to 40 mL/min.

For the Perkin Elmer Autosystem XL GC, chromatography conditions were as follows: the oven temperature was 40°C; the analysis time was 2.10 min; the injector temperature was 150°C; the detector temperature was 150°C; the injection time was 0.03 min; the pressurization time was 0.5 min; the thermostat temperature was 80°C; the thermostat time was 15 min; the needle temperature was 75°C; the withdrawal time was 0.2 min; the venting time was 0.3 min; and the flow rate was 2.0 mL/min.

Results
The postmortem heart blood sample analyzed was negative for barbiturates, cocaine and/or benzoylecgonine, opiates, and methamphetamine (and related compounds). The concentrations of ethanol and acetone for the different tissues analyzed are given in Table I. As shown in Table I, the heart blood ethanol concentration was 0.51 g/dL, and brain and liver ethanol concentrations were 0.04 g/100g and 0.08 g/100g, respectively. Urine ethanol concentration was 0.05 g/dL.

Clinical results from an independent laboratory, including vitreous, urea nitrogen, creatinine, and total protein values, are shown in Table II. The heart blood glucose concentration reported was 617 mg/dL, and the normal range is 70–110 mg/dL. The urea nitrogen concentration was 116 mg/dL, elevated compared to a normal range of 5–28 mg/dL. Vitreous creatinine concentration was 4.3 mg/dL, which was also higher than the 0.6–1.4 mg/dL normal range. As seen in Table II, the urea-nitrogen/creatinine ratio (27.0) was also elevated compared to the reference ratio range (5–22). The total protein was 1.9 g/dL, lower than the normal range of 6.0–8.5 g/dL.

The volume of vitreous humor available was insufficient for glucose analysis, so only postmortem heart blood glucose levels were reported (Table II).

Discussion
The heart blood ethanol concentration found in this case is consistent with concentrations reported in acute ethanol-intoxication fatality cases (6). The presence of an elevated concentration of acetone (> 15 mg/dL), however, suggests high ethanol concentration may at least be partially due to postmortem endogenous ethanol production. No vitreous humor was available for toxicological analysis and therefore further volatile analyses were performed on brain, liver, and urine.

<table>
<thead>
<tr>
<th>Table I. Postmortem Concentration of Ethanol and Acetone in Heart Blood, Liver, Urine, and Brain</th>
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<tbody>
<tr>
<td>Tissue</td>
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<tr>
<td>Heart blood</td>
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<td>Brain</td>
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<td>Liver</td>
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<td>Urine</td>
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<th>Table II. Postmortem Concentrations of Glucose in Postmortem Heart Blood and of Vitreous Humor Urea Nitrogen, Creatinine (and their Ratios), and Total Protein*</th>
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</thead>
<tbody>
<tr>
<td>Analysis</td>
</tr>
<tr>
<td>Heart blood glucose</td>
</tr>
<tr>
<td>Vitreous</td>
</tr>
<tr>
<td>Urea nitrogen</td>
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<tr>
<td>Creatinine</td>
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<td>Urea nitrogen/creatinine</td>
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<td>Total protein</td>
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* Normal ranges have been included for comparison.
Ethanol concentrations in the tissues were not consistent with acute ethanol intoxication. Ethanol concentration in the brain was 0.04 g/100 g. The accepted brain/blood ratio range is approximately 0.65–0.94 (6). Therefore, the brain ethanol concentration found in this case would be more consistent with blood concentrations between 0.04 and 0.06 g/dL. Similarly, the liver concentration in this case (0.08 g/100 g) would also be more consistent with a blood ethanol concentration of approximately 0.08–0.09 g/dL if a liver to blood ratio of 0.91 is applied (6). The ratio between ethanol blood concentration and ethanol concentration in the other tissues analyzed in this case is atypical for an exogenous source of ethanol (5,6) and points to endogenous postmortem production resulting from postmortem decomposition. The decreased levels of protein concentration found in the present case (Table II) are also an indication of postmortem decomposition.

Ethanol is either not produced or is produced in small concentrations in tissues and fluids such as urine, which under normal clinical conditions does not contain significant amounts of glucose, and more isolated tissues and fluids such as brain and vitreous humor (1). However, in the present case, the levels of ethanol in brain, liver, and urine are higher than those typically reported in the literature for endogenous production (5). A possible explanation is a high level of glucose due to diabetes mellitus. The glucose levels reported in this case were only measured in the heart blood. Unfortunately, the vitreous humor glucose concentration was not measured by the independent laboratory because of insufficient sample. The vitreous glucose concentration would have been of great interpretative value because of the low possibility of contamination of the vitreous humor by blood and bacteria (7).

As mentioned, glucose is necessary for microbial production of alcohol if bacteria are present due to postmortem changes such as decomposition. Ethanol was also present in the urine (Table I) and could also be an indicator of glucosuria, a condition typical of diabetes mellitus, this assuming no external source of alcohol.

The heart blood glucose level is elevated in this case, supporting a clinically elevated antemortem glucose concentration, even taking into account that postmortem blood glucose is not the most accurate indicator of the glucose level in the body at the time immediately prior to death.

Acetone was present in all tissues analyzed. Acetone is produced, antemortem, in smaller quantities than the other so-called ketone bodies (β-hydroxybutyrate and acetoacetate) (8). In addition, it is eliminated through expired air, and its blood levels are therefore decreased. Overproduction of ketone bodies can occur in cases of uncontrolled diabetes (8). The acetone concentration found in this case is consistent with acute diabetic ketoacidosis (6). Concentrations of the β-hydroxybutyrate and acetoacetate were not determined in the present case.

In conclusion, results presented in this paper support the view that postmortem blood ethanol levels might be greatly increased by postmortem production, especially among the diabetic population, and give evidence supporting the need for multiple blood and tissue samples for interpretative purposes.

References


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