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Does acute alcohol intoxication cause transaminase elevations in children and adolescents?

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ABSTRACT

Several long-term effects of alcohol abuse in children and adolescents are well described. Alcohol abuse has severe effects on neurodevelopmental outcome, such as learning disabilities, memory deficits, and decreased cognitive performance. Additionally, chronic alcohol intake is associated with chronic liver disease. However, the effects of acute alcohol intoxication on liver function in children and adolescents are not well characterized. The aim of this study was to determine if a single event of acute alcohol intoxication has short-term effects on liver function and metabolism. All children and adolescents admitted to the Department of Pediatrics and Adolescent Medicine between 2004 and 2011 with the diagnosis "acute alcohol intoxication" were included in this retrospective analysis. Clinical records were evaluated for age, gender, alcohol consumption, blood alcohol concentration, symptoms, and therapy. Blood values of the liver parameters, CK, creatinine, LDH, AP, and the values of the blood gas analysis were analyzed. During the 8-year study period, 249 children and adolescents with the diagnosis "acute alcohol intoxication" were admitted, 132 (53%) girls and 117 (47%) boys. The mean age was 15.3 ± 1.2 years and the mean blood alcohol concentration was 0.201 ± 0.049%. Girls consumed significantly less alcohol than boys (64 g vs. 90 g), but reached the same blood alcohol concentration (girls: 0.199 ± 0.049%; boys: 0.204 ± 0.049%). The mean values of liver parameters were in normal ranges, but AST was increased in 9.1%, ALT in 3.9%, and γGT in 1.4%. In contrast, the mean value of AST/ALT ratio was increased and the ratio was elevated in 92.6% of all patients. Data of the present study showed significant differences in the AST/ALT ratio ($p < 0.01$) in comparison to a control group. Data of the present study indicate that there might be an effect of acute alcohol intoxication on transaminase levels. The AST/ALT ratio seems to reflect the damage in hepatocytes after intensive alcohol consumption. The present study indicates a sex-specific difference in alcohol metabolism and effects between girls and boys: girls need less alcohol than boys to achieve the same blood alcohol levels than boys, and are more prone to loss of consciousness.

Introduction

Alcohol is one of the most frequently abused psychoactive drugs consumed by children and adolescents (Tõnisson, Tillmann, Kuudeberg, Lepik, & Väli, 2013). Binge drinking and other forms of alcohol abuse are a developing problem in young men and women (Meyer-Heim, Stocker, Kobler, & Lips, 2003; Tõnisson et al., 2013). The National Institute on Alcohol Abuse and Alcoholism (NIAAA) defined binge drinking as a pattern of drinking that increases blood alcohol concentration to 0.065% (80 mg/dL) or above (NIAAA, 2004). In children and adolescents, binge drinking is additionally characterized by the amount of alcoholic drinks/per age: 9–13 years: >3 drinks; 14–15 years: >4 drinks in boys and >3 drinks in girls; 16–17 years: >5 drinks in boys and >3 drinks in girls (Donovan, 2009).

Several long-term effects of alcohol abuse in children and adolescents are well known. Alcohol consumption at a young age is

Abbreviations: AST, aspartate aminotransferases; ALT, alanine aminotransferases; γGT, gamma-glutamyl transpeptidase; LDH, lactate dehydrogenase; AP, alkaline phosphatase; BE, base access; TBW, total body water.

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described as a risk factor for later alcohol addiction, drug abuse, and criminal behavior later in life (Grant & Dawson, 1997; Schöberl, Nickel, Schmutzer, Siekmeyer, & Kiess, 2008). Drinking alcohol before the age of 14 is associated with a four-fold higher risk for developing alcohol addiction later in life (Grant & Dawson, 1997). Furthermore, alcohol has severe effects on neurodevelopmental problems such as learning disabilities, memory deficits, and decreased cognitive performance (De Bellis et al., 2000, 2005; Medina et al., 2008). Additionally, chronic alcohol intake is associated with liver diseases later in life (Torruellas, French, & Medici, 2014; Yue et al., 2006). Chronic alcohol consumption is the major cause of liver disease in the Western countries (Torruellas et al., 2014). The first impact of chronic alcohol abuse on the liver is the development of steatosis (fatty liver) that might result in steatohepatitis, liver fibrosis, liver cirrhosis, and hepatocellular carcinoma (Massey & Arteel, 2012; Torruellas et al., 2014). In general, alcohol-associated liver diseases are one of the leading causes for disability, morbidity, and mortality worldwide (World Health Organization [WHO], 2014). Approximately 3.3 million deaths per year are the consequence of alcohol abuse (WHO, 2014).

In contrast to effects of chronic alcohol consumption, the effects of one-time alcohol intoxication on liver function in children and adolescents have not been investigated thus far (Li, Chen, Yu, Zhang, & Xu, 2004; Massey & Arteel, 2012; Yue et al., 2006). Several authors hypothesize that there might be parallels between one-time and chronic alcohol consumption and their effects on liver cells (Donohue et al., 2012; Li et al., 2004; Massey & Arteel, 2012). Only animal models in mice (Li et al., 2004) refer to an effect of alcohol on liver cells. Therefore, the aim of this study was to determine if a single event of alcohol intoxication has an effect on liver function parameters in children and adolescents.

Methods

Study design

This retrospective analysis over an 8-year period was conducted at the Department of Pediatrics and Adolescent Medicine of the Medical University of Vienna. The study was approved by the local Ethics-Committee, Medical University Vienna (EC Nr: 1091/2013).

Patients, inclusion, and exclusion criteria

All clinical records of children and adolescents, aged 9–18 years, hospitalized between 2004 and 2011 at the Department of Pediatrics and Adolescent Medicine were screened for the diagnosis "acute alcohol intoxication." Patients with poly-intoxication, diabetes mellitus type I and type II, liver and renal diseases, and medications that might influence the toxicity of alcohol were excluded.

The aim of the study was to evaluate the effect of acute alcohol intoxication on liver enzymes: aspartate aminotransferases (AST), alanine aminotransferases (ALT), gamma-glutamyl transpeptidase (γGT), and AST/ALT ratio. In the literature, limited data for standard levels of the AST/ALT ratio in children and adolescents are available (Giannini et al., 1999). Therefore, we implemented a control group, to compare the ratio with the study group. All children and adolescents of the control group were patients in the outpatient clinic of the Medical University of Vienna seen for reasons other than liver and muscle diseases (e.g., hypothyroidism, hemophilia, food allergies).

Data collection

After hospital admission with acute alcohol intoxication, the following blood parameters were determined: AST, ALT, γGT, AST/ALT ratio (De Ritis ratio), CK (creatine kinase), creatinine, and lactate dehydrogenase (LDH); blood alcohol concentration in per mille (‰; conversion factor: blood alcohol concentration of 1‰ is equivalent to 0.1% blood alcohol concentration); blood gas analysis: pH, pCO_2, standard bicarbonate, base excess, glucose, lactate, and electrolytes. For the blood gas analysis the following device was used: Radiometer 725®, Diamond Diagnostics, USA (sensor technology).

The following reference values were used: AST and ALT: <35 U/L in girls and <50 U/L in boys; γGT: <38 U/L in girls and <52 U/L in boys (Vajro et al., 2012); AST/ALT ratio: <1.0 (Giannini et al., 1999); AP: age 9–12: >332 U/L (girls) and >362 U/L (boys), age 12–15: >162 U/L (girls) and >390 U/L (boys), age 15–17: >119 U/L (girls) and >171 U/L (boys) and age >17: >105 U/L (girls and boys); creatinine <1.0 mg/dL, LDH: <250 U/L; and CK: 150 U/L in boys and 170 U/L in girls (Soldin, Murthy, Agarwalla, Ojeifo, & Chea, 1999; Van Hoof et al., 1990). The ranges of the blood gas parameters were: pH: 7.35–7.4.3; pCO_2: 37–50 mm Hg; standard bicarbonate: 22–31 mmol/L; base excess: −3/+3; glucose: 74–109 mg/dL; lactate: 0.5–2.2 mg/dL; electrolytes: sodium (Na): 136–145 mmol/L and potassium 3.5–5.1 mmol/L (Lamminpää & Vilska, 1990).

The alcohol consumption in grams (g) was calculated with the following formula: g (ethanol) = g/L (blood alcohol concentration) × total body water [TBW] (volume of distribution) (Karow & Lang-Roth, 2012). To predict the volume of distribution in children and adolescents, the modified Widmark equation was calculated by using their height and weight (Donovan, 2009); for boys: TBW (Volume of distribution) = −25.87 + (0.23 × Height) + (0.37 × Weight) and for girls: TBW = −14.77 + (0.18 × Height) + (0.25 × Weight) (Donovan, 2009).

In addition, weight, height, body mass index (BMI) (Himes & Dietz, 1994), age (years), sex, symptoms, therapy, and consumption history were evaluated. Data were collected from the patients' database system AKIM® (Siemens Austria) and from the case record database.

Statistics

The Spearman correlation coefficient (r_s) was used for bivariate correlation analysis of individual laboratory parameters. Descriptive statistics were expressed as frequency distribution, mean, and standard deviation. The two-sample *t* test was used to compare the mean and standard deviations of the two independent groups: girls versus boys according to quantity of alcohol (g), age (years), and blood alcohol concentration (‰). The Mann–Whitney *U* test was used to evaluate a correlation between symptoms and increase of blood alcohol concentration.

Data were analyzed with the software Statistical Package for Social Science (SPSS) for Windows (version 21.0). A p value of <0.05 was considered statistically significant.

Results

Patients' demographic data

During the 8-year study period between the years 2004 and 2011, 269 children and adolescents with the diagnosis "acute alcohol intoxication" were admitted to the Medical University of Vienna. Twenty patients had to be excluded from the analysis: 0.7% (2/269) suffered from type 1 diabetes and 7% (18/269) consumed additional drugs. A subgroup analysis showed that 39% (7/18) of