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The association between obesity and lethal blood alcohol concentrations: A nationwide register-based study of medicolegal autopsy cases in Sweden

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ABSTRACT

Obesity is a global problem and in aspects of lethal ethanol intoxications virtually unexplored. The cause of death in ethanol intoxication is generally considered to be suppression of the respiratory function. Previous research indicates that respiratory function is more vulnerable in obese subjects than in those of normal weight. We hypothesized that lethal blood alcohol concentration (BAC) is lower in obese subjects compared to those of normal weight. We used the Swedish medicolegal autopsy register and identified all medicolegal autopsy cases in Sweden during the period from 1999 to 2013 ($N = 79{,}060$), and identified 1545 cases with ethanol intoxication identified as the primary cause of death. We studied the association between body mass index and lethal BAC using logistic regression models that we adjusted using several potential confounders such as age, sex, drugs, and extent of decomposition. We observed an association between obesity and lower lethal BACs. The estimated adjusted odds ratio of the association between obesity and a lethal BAC >3‰, using subjects of normal weight as reference, was 0.54, 95% confidence interval: 0.39–0.74. The result indicates that in obese subjects the lethal BAC is lower than in those of normal weight.

1. Introduction

The association between obesity and lethal concentrations of ethanol has to our knowledge not previously been systematically studied, despite an increasing prevalence of obesity [1], which also affects the practice of forensic medicine [2]. The prevalence of overweight and obesity has increased over the last few decades, and in 2013 the prevalence of these conditions world-wide was estimated to be around 37% and 38% in men and women, respectively [1]. Previous research recognizes that the lethal blood alcohol concentration (BAC) of ethanol is about 3–5‰ [3,4] and cases of lethal ethanol intoxication have been studied in various settings [3–6]. The mechanism in death due to ethanol intoxication is generally considered to be suppression of the respiratory center in the brain stem, with a concomitant lowered arterial oxygen saturation [3]. It has also been indicated that obesity might increase oxygen consumption, carbon dioxide production, and the respiratory workload [7]. Research into respiratory physiology in obesity has produced somewhat conflicting results, but it appears that obesity also lowers the functional residual capacity of the lungs [7]. The association between obesity and respiratory function has been studied in other areas of medicine such as obstructive sleep apnea [8] and in the setting of surgery and anesthesia [9]. It has also been proposed that when determining the cause of death in an obese individual without any signs of an obvious cause of death, respiratory depression should be considered [2]. Considering this background, we hypothesized that the lethal BAC would be lower in obese individuals than in those of normal weight.

2. Materials and methods

2.1. Study population

In Sweden, the National Board of Forensic Medicine is responsible for all medicolegal autopsies, which are performed

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at six departments across Sweden. We identified all cases ($N = 79,060$) of a medico-legal autopsy in Sweden from 1999 to 2013 using the autopsy register maintained by the Swedish National Board of Forensic Medicine. Ethanol was analyzed in 78.2% ($n = 61,855$) of these autopsies. In the autopsy register we identified 1545 cases in which the primary cause of death was listed as ethanol intoxication (International Classification of Disease code 980A [ICD-9] or T51.0 [ICD-10]) and ethanol had been analyzed in blood from the femoral vein. We excluded adolescents under 18 years of age ($n = 193$), of whom only one (0.5%) had a BAC >3‰. The study was approved by the regional ethics board in the south of Sweden (Dnr 2014/126).

2.2. Assessment of variables

We calculated BMI according to the formula weight [kg]/length $[m]^2$, and categorized the results into four groups: (i) underweight (<18.5 kg/m^2), (ii) normal weight (18.5 to <25 kg/m^2), (iii) overweight (25 to <30 kg/m^2), and (iv) obese (≥30 kg/m^2). We used the category of normal weight as reference in the analyses.

We included the BAC (‰) obtained by analyzing femoral blood to which potassium fluoride had been added to preserve the sample. The analyses of ethanol were all performed at the Swedish National Board of Forensic Medicine, Department of Forensic Toxicology, in Linköping, Sweden. Ethanol was analyzed using headspace gas chromatography with the reporting threshold set at 0.1‰ the process is described elsewhere [10–12]. In the statistical analyses we included the BAC both as a continuous variable and as a dichotomous variable with a cut-off value of 3‰. The category with the lower BACs was used as reference in the analyses.

We calculated the time (days) from death to autopsy and used this as a surrogate variable to adjust for degree of decomposition. In 1292 cases the date of death was certain and in another 152 cases the date of death was probable. In the remaining 101 cases the date of death was estimated to within one month at best and these were classified as having missing information. We classified the time from death to autopsy as a dichotomous variable, <10 days or ≥10 days. We used the cases in which the autopsy was performed within 10 days after death as reference.

We included sex as a dichotomous variable, using men as reference.

We categorized age at death into (i) 18–29 years, (ii) 30–39 years, (iii) 40–49 years, (iv) 50–59 years, (v) 60–69 years, and (vi) 70 years and over. In the analyses we used the category of 18–29 years as reference.

We categorized the detection of pharmaceuticals or illicit drugs in the toxicological analyses as either positive or negative. We used the positive cases as reference.

We categorized the year of the autopsy into (i) 1999–2003, (ii) 2004–2008, and (iii) 2009–2013, and we used the last category as reference.

2.3. Methods

We compared the mean BACs across BMI groups using analysis of variance (ANOVA). This approach allowed us to estimate whether the samples were drawn from different populations. However, this approach did not allow us to make conclusions about specific BMI groups. We also estimated the mean difference between the obese group and the normal weight group using the *T*-test. We report probability values (*P*-values) with 5% level of significance.

We further studied the association between BMI and the dichotomized BAC by fitting four consecutive logistic regression models in which we introduced potential confounders stepwise. In the first model (I) we studied the unadjusted association between BMI and a lethal BAC >3‰. In the second model (II) we introduced the potential confounding factors of age and sex, since these have previously been suggested to be associated with lethal concentrations of ethanol [3,4,13]. In the third model (III), we adjusted for time between death and autopsy to account for decomposition with potential postmortem production of ethanol [14]. We further adjusted this model for the presence or absence of pharmaceutical or illicit drugs in the toxicological analysis. Finally, in the fourth model (IV) we included the year of the autopsy to detect and adjust for possible temporal confounders.

We also performed sensitivity analyses to address potential confounding through diagnostic bias and/or the postmortem formation of ethanol. A diagnostic bias might result in ethanol intoxication at lower BACs being recorded as the primary cause of death in obese subjects. Therefore, we excluded all cases with an ethanol concentration below 3‰ and analyzed the association between BMI and a lethal BAC >5‰ in the remaining cases. To account for potential postmortem formation of ethanol, we analyzed the cases in which the time from death to autopsy was ≤3 days. We used unadjusted logistic regression models to estimate the associations.

We report the results of the logistic regression models using odds ratios (OR) and 95% confidence intervals (CI). The data was analyzed using IBM SPSS, version 22.0.0.0.

3. Results

3.1. Characteristics of the study population

The characteristics of the study population, stratified by BMI group, are shown in Table 1. In the obese group (≥30 kg/m^2) the mean BAC was 3.2‰, in those with normal BMI (18.5 to <25 kg/m^2) it was 3.5‰. Also, the proportion of individuals with a BAC >3‰ was lower in the group with obesity (58.2%) than in the group with normal BMI (71.6%). In an ANOVA test the mean BACs differed significantly ($P < 0.05$) across BMI groups. Female sex was more prevalent in the underweight (BMI ≤18.5) group (42%) and normal weight (27.8%) group than in the overweight (20.7%) and obese (20%) groups. The proportion of cases with a negative toxicological analysis for pharmaceutical or illicit drugs was higher in the underweight group (60.5%) than in the other BMI-groups (48.0–52.8%). There was a temporal relationship among cases with missing information about BMI, with more values missing in earlier years than in later years. In the group with missing information for BMI the mean ethanol concentration was 3.2‰.

3.2. Association between BMI and lethal BACs

In Table 2 we present the association between BMI and a lethal BAC >3‰ modeled using logistic regression. In the unadjusted logistic regression model (model I) obesity was negatively associated with a BAC >3‰ (OR 0.55; 95% CI: 0.41, 0.75), using the normal weight group as reference. Similarly, overweight indicated a negative association with a BAC >3‰ (OR 0.87; 95% CI: 0.66, 1.14). In model II, adjusting for age and sex had no relevant effect on the associations between BMI groups and a lethal BAC >3‰, leaving the results of model I virtually unaffected. Furthermore, in model II we observed no association between sex and a lethal BAC >3‰. No conclusive associations between age groups and a lethal BAC >3‰ was observed, but a tendency was found toward lower lethal BACs in the oldest (≥70 years) and youngest (18–29 years) age groups. In model III we adjusted for time from death to autopsy and for no detection of pharmaceuticals or illicit drugs, but the associations between BMI groups and lethal BAC >3‰ were not affected. We did observe, however, an association between negative screening for pharmaceuticals and