G38 Diabetic Ketoacidosis—A Silent Death

Zabiullah Ali, MD, Office of the Chief Medical Examiner, State of Maryland, 111 Penn Street, Baltimore, MD; Mary G. Ripple, MD, 68 Bluebird Road, Port Deposit, MD; and Barry S. Levine, PhD, and David R. Fowler, MD, Office of the Chief Medical Examiner, State of Maryland, 111 Penn Street, Baltimore, MD*

The goal of this presentation is to determine the percentage of cases investigated by the Office of the Chief Medical Examiner (OCME) with a known history of diabetes versus de novo detection of diabetes

Diabetic ketoacidosis may be the initial manifestation of type I diabetes or may result from increased insulin requirement in type I diabetic patients during the course of infection, trauma, myocardial infarction, or surgery. It is a life-threatening medical emergency with mortality rate just under 5% (1). Type II diabetic patients may develop ketoacidosis under severe stress such as sepsis or trauma.

Cases investigated by OCME over a 6-year period whose cause of death was diabetic ketoacidosis were identified. For each case, the initial investigation and follow-up investigation report were reviewed to determine whether a history of diabetes was included. In all cases investigated by OCME, a specimen, usually blood is analyzed routinely for volatile substances, including methanol, ethanol, acetone, and isopropanol by Gas chromatography at a limit of quantitation of 0.01 g/dL

A postmortem diagnosis of diabetic ketoacidosis is based on either some or all of the following: a history of diabetes, increased vitreous humor glucose, or increased blood acetone. From January 1996 to December 2001, 20,406 autopsies were performed, with 34.49% (n=7039) natural deaths. The total number of deaths secondary to diabetic ketoacidosis was 1.43% (n=101), with 85.1% (n=86) of them available for review. A total of 35.2% (n=31) of the decedents did not have a previous diagnosis of diabetes and were diagnosed for the first time at autopsy. The age of the deceased ranged from 10 years to 70 years with a male to female ratio of 62:24. The race was not significantly different with African American to Caucasian ratio of 46:40. In this study, a total of 57 cases (66.2%) were diagnosed based on vitreous acetone and/or vitreous glucose, and/or blood acetone. In 18 cases (20.9%), vitreous and blood acetone were used for diagnosis. The urine and blood acetone were used instead of vitreous acetone in 6 cases (6.8%) with or without vitreous glucose. The other cases were diagnosed either based on vitreous acetone alone (2 cases; 2.3%), blood acetone and vitreous glucose (1 case; 1.16%), decomposition fluid (1 case; 1.16%), and vitreous acetone and glucose (1 case; 2.1%). The variability of specimens tested depended on the availability of test material. There were 4 (4.6%) decomposed cases, in which urine and blood acetone were used in 2 cases, liver acetone in 1 case, and decomposition fluid and blood acetone in 1 case. The blood acetone level ranged from 0.01 g/dL to 0.117 g/dL (mean=0.035 g/dL). The vitreous acetone range was from 0.014 g/dL to 0.97 g/dL (mean=0.05g/dL). The level of the vitreous glucose ranged from 89 mg/dL to 1233 mg/dL (mean=597 mg/dL).

A positive acetone can indicate diabetic ketoacidosis, isopropanol ingestion, or malnutrition. Acetone, a ketone body, is produced in the liver from spontaneous decarboxylation of acetoacetate, which is produced as a result of incomplete breakdown of fatty acids. Once acetone has been detected, the Medical Examiner routinely requests a vitreous glucose concentration. An elevated vitreous glucose with an elevated vitreous acetone indicates diabetic ketoacidosis. It is recommended that the volatile toxicology analysis at a Medical Examiner's Office should not only include ethanol, but also acetone to screen for the diabetic ketoacidosis in cases of sudden deaths.

Diabetes Mellitus, Ketoacidosis, Acetone

 ** Presenting Author*