CASE REPORT

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Hypoglycemia as the Responsible Factor in a Truck Driver Accident Fatality

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ABSTRACT: A case of hypoglycemia causing a truck driver to lose control of his vehicle and his resulting traumatic asphyxial death is presented. The incident was considered suspicious in spite of poor visibility, because an experienced, healthy operator lost control in the early morning hours after a relatively short (4-h) drive. The autopsy revealed no evidence of natural disease and the driver had no alcohol or drugs in his system. Although this is apparently a rare occurrence, the diagnosis of underlying hypoglycemia as the causative factor in a traffic fatality can be reasonably suspected and accurately made if appropriate specimens are properly obtained and tested. Legislation mandating the retention of antemotem blood specimens on all trauma patients will permit more accurate assessment of most chemical etiologies of traffic accidents.

KEYWORDS: pathology and biology, hypoglycemia, motor vehicle accidents, "natural death at the wheel"

The role of natural disease as a precipitating factor in traffic accidents and resulting fatalities has been well documented [1,2]. Suggestions that subtle biochemical mechanisms, including hypoglycemia, can produce unconsciousness, or at least distraction, and thus cause crashes, have been offered but are difficult to prove. A recent case in which history, circumstances, and appropriate retention and testing of specimens occurred together confirmed such a diagnosis and is the subject of this report.

Case Report

A previously healthy 45-year-old male truck driver overshot the rather sharp, downhill turn of a northbound off-ramp from an interstate highway in eastern Connecticut (Figs. 1 and 2). This happened at 8:30 a.m. one mid-August morning, during which a moderately thick fog blanketed the roadway. He had left his home base in New Jersey some 4 h earlier. He was heading for southern Massachusetts, a trip he had made uneventfully many times

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FIG. 1—Overturned truck. Note turn (yaw) marks without brake (skid) marks through stop sign.

previously. A CB radio report overheard him saying at 7:30 a.m. that he wanted to get a cup of coffee. An hour later his tractor-trailer overturned, pinning him inside. It required some 15 min to extract him from the driver's cab. He survived briefly ("DOA with efforts") in a nearby Rhode Island hospital emergency room, during which glucose fluid therapy was initiated.

The autopsy was conducted 24 h after death, following body refrigeration, and showed a stocky male with anatomical findings of traumatic compression asphyxia, including abundant petechial hemorrhages of the face and upper chest and fractures of the ribs and spine; however, no hemothorax or other hemorrhage was observed. There was no significant cardiac or other natural disease; the pancreas was softened because of autolysis but otherwise unremarkable. The stomach was contracted and empty, with the urinary bladder moderately full; an odor of alcohol was not detected. Toxicologic studies were negative for ethyl alcohol, carbon monoxide (5.8% COHb), and other drugs including amphetamine, caffeine, narcotics, and hypnotics.

A refrigerated antemortem blood specimen was available; glucose analysis from a chemical laboratory using the Beckman glucose oxidase method [3] revealed less than 10 mg/dL glucose (the normal range is 18 to 180 mg/dL) in both antemortem blood and postmortem vitreous specimens. Repeat analyses at another laboratory revealed near zero concentrations. A postmortem blood specimen showed a glucose concentration of 786 mg/dL. Further postmortem chemical studies were not performed. Urinalysis showed +1 protein but was otherwise normal.

Inquiries to the trucking company and the State Police were successful in obtaining further information about the vehicle and cargo, as well as time logs of check-in and arrival during previous hauling trips. However, no adverse data, sick calls, or vehicular failure were ever documented. All systems of the truck, including the brakes, were subsequently checked and found to be functioning properly. The load, which was within legal weight limits, was nontoxic and nonflammable and consisted of sacks of plastic pellets.

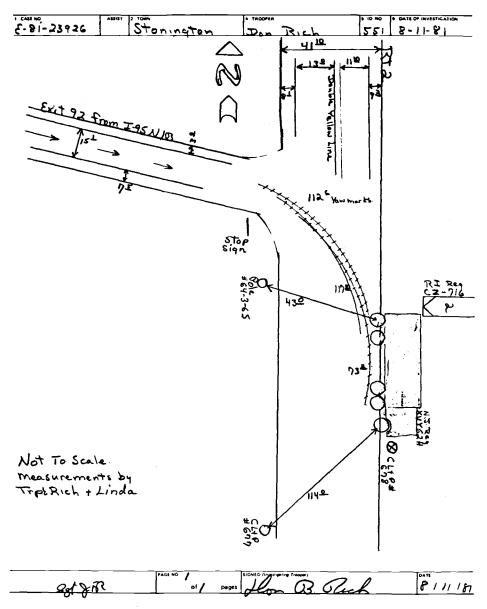


FIG. 2—Police diagram indicating exit route, measurements of roadway, and final resting place of vehicle.

The decedent's personal effects included three notes from doctors, including one from an optometrist (he apparently did not wear glasses; none were recovered), one from an orthopedist consulted for minor neck and back pains, and one that appeared to indicate therapy for rectal bleeding. Correspondence addressed to the wife requesting further medical information, along with several telephone calls, have to this time been unanswered.

Discussion

Questions have been raised about low glucose levels and their interpretation, with special reference to automobile drivers, and attempts have been made to relate low levels to the

possible causation of traffic accidents. Instances of hypoglycemia in alcohol intoxication, as well as in chronic alcoholism, have been documented in the hospital setting [4], but only inferred from behind the wheel of a vehicle.

A review of recent two-year statistics (1979-1980) from the Rhode Island Medical Examiner's Office, covering cases in which a postmortem vitreous chemical examination was performed on automobile fatalities, reveals that 7 out of 265 cases (3%) showed depressed glucose levels, that is, below 15 mg/dL. Of these, three were drivers of automobiles involved in fatal accidents, two being elderly without history or findings, and one a 30-year-old male with 0.31-g/dL blood alcohol. Only one of the incidents involved aberrant behavior, an abrupt U-turn on a heavily traveled road for no apparent reason by one of the elderly drivers. In any event, diabetes mellitus, islet cell tumor, or drug-induced hypoglycemia were not diagnosed in any of these fatalities, although intoxication could have been a contributory factor in the case of the 30-year-old man. Further, the postmortem intervals were prolonged in each case, so that a low vitreous glucose value could have resulted solely from postmortem glycolysis.

Since 1978 it has been a legal requirement for hospitals in Rhode Island to obtain admission blood specimens from all "trauma patients" who are received in an emergency room or admitted to a hospital, and to secure them for potential use by the medical examiner in the event the patient dies [5]. This occurred in the present case, where one of the blood specimens was in a gray top (sodium fluoride) tube, specifically obtained for glucose testing in the living patient. It is, of course, often crucial to retain and test such specimens after a long hospitalization, as it is the only way a positive value for alcohol, carbon monoxide, drugs, or any similar antemortem condition can be documented. Such legislation or regulation should be in effect in all jurisdictions, so that chemical factors and their involvement in traumatic conditions could be more completely diagnosed. (See a letter written by one of the authors concerning this in this issue of the *Journal*.)

Glycosylated hemoglobin analysis is a relatively new test that indicates the long-term effects of diabetes mellitus, especially hyperglycemia, without necessarily having to rely on a single glucose determination. An analysis for glycosylated hemoglobin was performed in this case, despite a prolonged interval (5 months) during which the blood was refrigerated but hemolyzed, which is known to interfere with some testing methods. The thiobarbituric acid method was used, in which hemolysis interferes but minimally, and the level obtained was 13.4 nmol of fructose equivalent per milligram of hemoglobin. (Controls in pregnant women are 12.3 ± 3.1 , as determined by high-performance liquid chromatography.³) Chronic hypoglycemia may also be identified by this test, but an acute process, as in the present case, unfortunately cannot be diagnosed.

It has been suggested that changes in carbohydrate metabolism can follow episodes of asphyxia, hypoxia, and other similar phenomena that increase the blood sugar, probably because of the release of epinephrine [6]. More recently, terminal cardiopulmonary resuscitation has been shown to elevate the peripheral blood glucose to over 500 mg/100 g in almost 10% of nondiabetic cases [7]. In the present situation, the short interval (minutes) between the accident and death, as well as the intravenous dextrose administration and the resuscitation measures employed probably accounts for the elevated glucose in the postmortem blood, in contrast with the depleted glucose in the antemortem blood and the postmortem vitreous humor.

The latter findings, at once incompatible with life at face value, probably indicate the glycolytic process that occurs over time, albeit slowed by chemical preservatives and refrigeration. On the other hand, the paucity of similar findings in other cases of this nature, the lack of a suitable explanation for the driving behavior in this instance, and the confirmation of low glucose in the antemortem blood specimen support the conclusion that the hypoglycemia antedated the accident, the compression asphyxia, and the resulting fatal outcome.

³J. Susa, Director, Pediatric Metabolic Research Laboratory, Rhode Island Hospital, personal communication, 1982.

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