Coma and Hyperglycemia Following Drinking of Acetone

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SUMMARY

A patient with coma due to drinking of acetone is described. Recovery was followed by hyperglycemia and decreased carbohydrate tolerance persisting for four months. A similar course, although not so prolonged, has been recorded by other authors in poisoning due to inhalation of acetone vapor. Possible mechanisms by which acetone poisoning might affect carbohydrate metabolism are briefly discussed. DIABETES *15*:810-11, November, 1966.

Cases of acetone poisoning in man resulting from inhalation of vapors or from absorption through the skin have been reported. [1,2,3] It is the purpose herein to describe the course of a patient who ingested acetone for suicidal reasons.

CASE REPORT

A forty-two-year-old shoe factory worker was brought to the hospital one hour after he had drunk with suicidal intent approximately 200 ml. of pure acetone. His stomach had been aspirated one-half hour earlier at another hospital. On admission he was stuporous, and his breath smelled strongly of acetone. His cheeks were flushed and respiration was shallow. Temperature and blood pressure were normal, and the pulse rate was 108 per minute and regular. His throat was red and swollen and erosions were seen on the soft palate and at the entrance to the esophagus. The abdominal reflexes were absent, but the tendon reflexes were normal. No other abnormal findings were noted.

Lange's nitroprusside test for acetone and acetoacetate* in the urine was strongly positive on admission, and traces of albumin and a few hyaline casts and leukocytes were also found. Repeated examinations for sugar were negative.

The CO_2 combining power of the blood on admission was 14 mEq. per liter and normal the next day. Blood sugar determined about twenty hours after admission, and two hours after an infusion of 500 ml. of isotonic glucose had been given, was 181 mg. per 100 ml. (Somogyi-Nelson method). Fasting blood sugar levels determined on the fourth, seventh, eighth and ninth days after admission were: 134, 144, 107, and 139 mg. per 100 ml., respectively. Two hours after a meal on the ninth day the blood sugar was 181 mg. per 100 ml. Liver function tests were normal.

Shortly after admission, the patient lapsed into deep coma and his breathing became more shallow. An infusion of isotonic saline was given, alternating with 1/6 molar sodium lactate and with 5 per cent glucose. A total of 1,000 ml. saline, 500 ml. of sodium lactate and 1,000 ml. of glucose were administered during the first twenty-four hours. With the hope of promoting the excretion of acetone, a mercurial diuretic was given. Tetracycline was given prophylactically for infection. Nikethamide was injected intravenously with transient lightening of the coma. Excessive secretion of saliva and mucus necessitated intermittent suction.

Twelve hours after admission the patient regained full consciousness and complained of a severe sore throat. On the advice of our otolaryngologist the patient was given 10 mg. of prednisone daily and the antibiotic treatment was continued. Prednisone was reduced in dosage on the seventh day and withdrawn on the ninth day after admission. During the first week the patient's temperature rose to 38° C. on several occasions, but no signs of pneumonia were detected.

When the patient started walking, on the sixth day of admission, a marked disturbance of gait was noted. Hyperesthesia of the legs was present, and extension of the legs with the hips flexed resulted in considerable pain. He was discharged after thirteen days in hospital and failed to return for further examinations until two months later, when his gait had improved.

Four weeks after ingestion of acetone the patient noted polydypsia and polyuria. A postprandial blood sugar level was 325 mg. per 100 ml. An oral glucose tolerance test was performed two-and-one-half months after ingestion and the values obtained were within diabetic range (figure 1a). The patient was treated with mild dietary restriction for two months, and his fasting blood sugar returned to normal. A glucose tolerance test was repeated five months after the poisoning, and values at the upper limits of normal were observed (figure 1b). During the test the urine contained considerable amounts of sugar (figure 1b). No family history of diabetes was known. The patient refused further examinations.

DISCUSSION

Glycosuria was found in each of six cases of poisoning due to inhalation of acetone vapor in factory workers and in hospital patients with applied immobiliza-

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*Urine, nitroprusside, glacial acetic acid, overlay with ammonia water.

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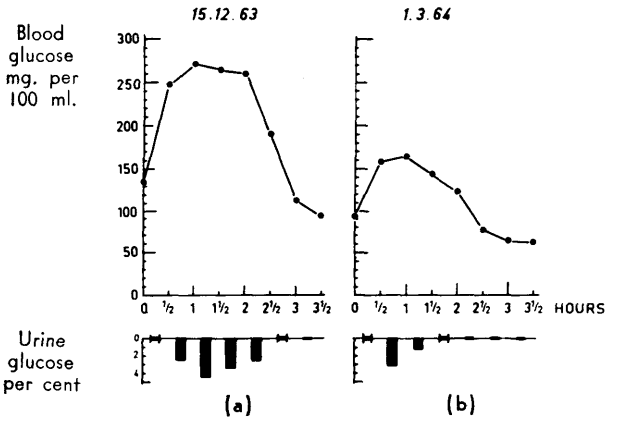

FIG. 1. Oral glucose tolerance tests two-and-one-half-months (a) and five months (b) after acetone poisoning, using capillary blood. Glucose load 100 gm., Hagedorn-Jensen's method of blood glucose determination.

tion casts set by acetone.[1,2,3] In all these subjects coma or marked drowsiness was present. In three, the fasting blood sugar was determined and found elevated (306, 170, 140 mg. per 100 ml., respectively). Acetoacetic acid (Gerhardt test) was found in the urine of two patients in whom it was looked for.[3]

The duration of hyperglycemia in the present patient was longer than that reported in others. In one, a physician who had no previous history of diabetes, hyperglycemia and glycosuria required insulin treatment for four days.[1] Acetonuria persisted for two weeks and was still present when the blood sugar had returned to normal. Glucose tolerance determined two weeks later was normal. In another, a seven-year-old boy, hyperglycemia persisted for three days and glycosuria for one day following the intoxication.[3] Glucose tolerance was slightly abnormal two weeks after the episode, and normal two months later. On both occasions sugar appeared in the urine after administration of glucose. It is improbable that the small dose of the prednisone was responsible for the persistence of hyperglycemia in the present patient.

The cause of the hyperglycemia in acetone intoxication is unknown. Experimental intoxication has readily elevated the blood sugar in animals.[2,4-7] A rise in blood concentration of acetoacetate and beta-hydroxybutyrate has been noted following acetone administration,[4] and there is experimental work in rats indicating that acetone might be metabolized in pathways leading to acetoacetate and three carbon compounds able to form hexose.[8,9] It is, however, difficult to explain on this basis the continuing hyperglycemia recorded in the above-mentioned patients. Furthermore, it has been observed that in comatose rats with acetone poisoning, the bulk of the acetone is excreted and only a small part is metabolized.[5]

Recently it has been shown that intravenous administration of acetoacetate and beta-hydroxybutyrate to animals results in an immediate increase in secretion of pancreatic insulin[10] with hypoglycemia.[11] In more chronic studies, repeated administration of these ketones resulted in a decrease in pancreatic insulin content[12] and lasting hyperglycemia.[13] Whether or not a similar mechanism may be involved in causing hyperglycemia in acetone poisoning remains to be proven.

The nature of the decreased renal threshold for glucose noted in the present patient and in the other case mentioned above[3] cannot be elucidated on the basis of the information available.

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