Insulinoma presenting as alcoholic stupor

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Summary: We report a case of prolonged loss of consciousness due to hypoglycaemia following ethanol abuse in a non-diabetic. The patient also reported symptoms compatible with hypoglycaemia following heavy manual work. Further investigations revealed a pancreatic insulinoma, which was successfully removed surgically. The patient remains asymptomatic 18 months later, despite occasional episodes of ethanol abuse. This case illustrates how heavy exercise and/or alcohol abuse can aggravate spontaneous hypoglycaemia.

Introduction

Severe hypoglycaemia is a known complication of excessive alcoholic intake in insulin-dependent diabetics.¹ Protracted hypoglycaemia in alcoholic diabetics can cause permanent brain damage^{2,3} and death, which is otherwise uncommon even after large doses of insulin.⁴ We are, however, unaware of any report of an insulinoma presenting with hypoglycaemia following an alcoholic binge.

Case report

A 29 year old bricklayer was found unrousable 36 hours after an alcoholic binge. A blood sample revealed a plasma glucose of 1.6 mmol/l, and he regained consciousness promptly following intravenous dextrose. He admitted to a high intake of alcohol and reported that over the previous 2–3 years he had had several episodes of prolonged sleep following alcoholic binges. He did not volunteer any other symptoms, but admitted to having had intense hunger, sweating and fainting sensations if his meals were delayed, particularly after heavy manual work. He had not suffered any episodes of sudden loss of consciousness or convulsions. On examination, he was not obese (height: 185 cm, weight: 79.6 kg), had a flushed face and sweaty, tremulous hands. Apart from vitiligo no other abnormality was found.

He was admitted to the Metabolic Ward for a prolonged fast. Plasma glucose was 2.2 mmol/l on admission and fell to 1.4 mmol/litre 6 hours after starting the fast. He felt dizzy and sweaty, but was fully conscious and his orientation was normal. Serum

insulin concentrations associated with the blood glucose concentrations mentioned above were 20 and 30 mU/l respectively, and were therefore inappropriately high.

A computed tomographic scan showed no abnormality in the pancreas. Venous blood samples from superior mesenteric, splenic and portal veins revealed an area of high insulin secretion (serum insulin >160 mU/l) near the neck of the pancreas. At laparotomy, a nodule 1 cm in diameter was located and removed from this area. Histology confirmed the diagnosis of insulinoma. The patient had to be maintained on a continuous glucose infusion before and during the operation. No further infusion was necessary post-operatively.

One month later, the patient underwent a 24 hour fast with no symptoms of hypoglycaemia. At the end of the fast, his plasma glucose was 5.3 mmol/l and serum insulin was 4.2 mU/l (normal fasting concentration: <10 mU/l). Over the past 18 months he has remained well, and despite occasional alcoholic binges, he has not had any episode of prolonged unconsciousness.

Discussion

This patient presented in coma, which in the first instance appeared to be the result of alcoholic stupor. However, the history of previous hypoglycaemia-like spells related to exercise, and the protracted coma with profuse sweating, together with the blood glucose concentrations of 1.6 mmol/l following his last binge confirmed the existence of spontaneous hypoglycaemia. Further investigations resulted in the diagnosis of an insulinoma. It should be mentioned

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that alcoholic binges resulted in this diagnosis, which would otherwise have been delayed in view of his mild symptoms. It is noteworthy that while he abstained from alcohol and strenuous physical work, he remained asymptomatic.

Alcohol inhibits gluconeogenesis in the liver,⁵ and by so doing impairs the major mechanism which aids recovery to a normoglycaemic state following hypoglycaemia. Alcohol therefore impairs the action of glucagon and adrenaline, which are released during hypoglycaemia to stimulate gluconeogenesis and restore normoglycaemia. This effect is distinct from the inhibitory action of ethanol on insulin-stimulated glucose metabolism *in vivo*⁶ and that of its more recently discovered metabolites, propanediol and butanediol, on insulin-stimulated glucose metabolism *in vitro*.⁷

Strenuous exercise alone has been reported to cause

hypoglycaemia,⁸ but in association with insulin hypersecretion and possibly alcohol intoxication, the risk of inducing hypoglycaemia must be considerably increased. These factors had probably contributed to these patients 'dizzy' spells at work.

In conclusion, ethanol can not only potentiate the hypoglycaemic effect of insulin⁹ but also prevent recovery from hypoglycaemia. Prolonged spells of loss of consciousness from hypoglycaemia in insulin-dependent diabetics must be scrutinized for concomitant alcoholism, whilst similar prolonged spells of loss of consciousness in alcoholics must be scrutinized for hypoglycaemia.

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