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Profound spontaneous hypoglycaemia in congestive heart failure

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Summary

Eleven cases of spontaneous hypoglycaemia in congestive heart failure in adults are reported. There were 5 males and 6 females, aged from 15 to 65 years (mean, 44 years). Blood sugar ranged from 2 to 42 mg/100 ml (mean 21 mg/100 ml). Six patients were in coma on admittance, 1 was confused, and 4 were conscious. The underlying condition was rheumatic valvular heart disease (3), chronic obstructive lung disease (4), coronary heart disease (3) and cardiomyopathy (1). Five of the 11 patients died. The mechanism of hypoglycaemia is discussed and thought to be a combination of factors such as liver dysfunction, low calorie intake, malabsorption, and increased glucose utilization by ischaemic tissues, including the heart. It is recommended that in patients with congestive heart failure presenting with coma or confusion, blood sugar should be checked for possible hypoglycaemia.

Key words: Hypoglycaemia – heart failure, congestive

Introduction

The clinical syndrome of spontaneous hypoglycaemia in congestive heart failure has been reported in neonates and infants^{2,3,7} but seldom in adults.^{1,8} On the basis of our experience with 11 cases of spontaneous hypoglycaemia in congestive heart failure in adults, it is suggested that this syndrome occurs more frequently than usually believed in adults. To our knowledge, profound hypoglycaemia associated with congestive heart failure, with blood sugar levels as low as < 2 mg per 100 ml, has not previously been reported.

Patients

During a 16-months' period 5 patients presented at Pahlavi University Nemazee Hospital with congestive heart failure and hypoglycaemia; over the same period, we reviewed the records of all patients for whom a diagnosis of congestive heart failure had been made and 6 other such cases were found who had been admitted for congestive heart failure and hypoglycaemia.

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Results

The clinical features of the patients are summarized in Table I. There were 5 males and 6 females. Their age ranged from 15 to 65 years with a mean age of 44 years.

Table I. Summary of cases of congestive heart failure and hypoglycaemia

Patient No.	Age	Sex	Underlying condition	Hypoglycaemic signs and symptoms	Blood sugar (mg/100 ml)
1	45	Male	Chronic obstructive lung disease, cor pulmonale	Coma	<2
2	65	Male	Chronic obstructive lung disease, cor pulmonale	Coma	<2
3	45	Female	Rheumatic heart disease, mitral stenosis	Coma	2
4	32	Male	Chronic obstructive lung disease, cor pulmonale	Coma	9
5	63	Female	Coronary heart disease, hypertension, diabetes mellitus	Coma	21
6	45	Male	Coronary heart disease	Confusion	24
7	29	Female	Rheumatic heart disease, mitral stenosis, mitral insufficiency	Conscious	38
8	50	Female	Cardiomyopathy	Weakness	36
9	15	Male	Rheumatic heart disease, mitral stenosis, mitral insufficiency	Conscious	36
10	60	Female	Coronary heart disease	Conscious	42
11	40	Female	Chronic obstructive lung disease, cor pulmonale	Coma	20

Three of the patients had rheumatic heart disease, 4 had chronic obstructive lung disease, 3 had coronary heart disease, and 1 had cardiomyopathy. Patient No. 5 was diabetic and had coronary artery disease and hypertension. She had previously been on an oral hypoglycaemic agent, but because of hypoglycaemia the drug was discontinued. However, the attack of hypoglycaemia was repeated whilst on diet only and her hypoglycaemia always coincided with congestive heart failure. None of the other 10 patients were taking any of the other known agents likely to cause hypoglycaemia. All 11 patients had symptoms and signs of right ventricular failure, including neck vein engorgement, enlarged tender liver, ascites (Patients No. 1, 2, 4, 8, 9 and 11) and oedema of the legs. However, only 7 of them had left ventricular failure (congestive rales, ventricular diastolic gallop). Six patients were in coma,

1 was confused, and 4 were conscious on admittance. Blood sugar was between less than 2 mg to 42 mg/100 ml (Somogyi-Nelson Method), with a mean of 21 mg/100 ml. Those blood sugar levels which were very low (such as less than 2 mg and 2 mg/100 ml) were rechecked from the same sample to be sure that they were not due to laboratory error.

The liver function tests were abnormal in 6 patients (including elevated SGOT, SGPT, bilirubin, alkaline phosphatase, and reversed A/G ratio), and was normal in 1. The tests were not performed in the other 4 patients.

Except in Patient No. 5, all the other 10 patients improved with intravenous hypertonic dextrose and they came out of coma. Five patients (Nos. 2, 4, 5, 9 and 11) died. Patient No. 5 did not improve and died 24 hours after admission because of advanced congestive heart failure, hypoglycaemia and respiratory failure. The other 4 patients (Nos. 2, 4, 9 and 11) completely improved after being given i.v. glucose; however, they died from 24 hours to 3 weeks after admission. In other words, they did not die because of hypoglycaemia but of advanced congestive heart failure and respiratory failure. Surprisingly, Patient No. 1 with blood sugar of less than 2 mg/100 ml and Patient No. 3 with blood sugar of 2 mg/100 ml did not die. Patient No. 2 with blood sugar of less than 2 mg/100 ml improved and came out of coma, but died 3 weeks later due to congestive heart failure and respiratory failure.

Discussion

Spontaneous hypoglycaemia associated with congestive heart failure is rarely reported in adults,^{1,8} though more cases have been reported in neonates and infants.^{2,3,7} The underlying cardiac disease in the latter has been reported to be congenital heart disease, mostly hypoplastic left heart syndrome.³ In a few infants the only abnormality was cardiomegaly on X-ray.^{2,7} The underlying conditions in our cases were 3 rheumatic valvular heart disease, 4 chronic obstructive lung disease, 3 coronary heart disease, and 1 cardiomyopathy.

It is not yet certain what is the mechanism of hypoglycaemia in congestive heart failure. It seems to us, however, that more than one factor is playing a role, such as low food intake, malabsorption due to passive congestion, oedema of the gastrointestinal tract, liver dysfunction (hepatic hypoglycaemia),^{1,4-6,8} and increased glucose utilization by ischemic tissues, including the oversized heart.

The above factors can all be seen in advanced heart failure; probably decreased blood flow to the muscles and liver in advanced congestive heart failure leads to inadequate delivery of substrates for adequate gluconeogenesis in the liver. In 4 neonates reported with congestive heart failure and hypoglycaemia,³ liver biopsy revealed reduced hepatic glycogen; however, glucose-6-phosphatase and phosphorylase activity were normal. Cardiac enlargement and congestive heart failure are reported to be associated with hypoglycaemia in infants^{2,7} and after raising the blood glucose in these cases symptoms and signs of heart failure disappeared.

Regarding the level of blood sugar in such cases, the lowest blood sugar which has been reported in adults is 7 mg/100 ml.¹ In our group, there were 3 cases in whom levels were either 2 mg/100 ml or less. The mechanism of very low blood

sugar in our cases may be due to the fact that our patients were referred very late and all of them had symptoms and signs of advanced congestive heart failure, long-standing passive liver congestion, possible cardiac cirrhosis and malnutrition due to low calorie intake as the result of low appetite. As regards the aetiology of hypoglycaemia rather than congestive heart failure, in none of our patients was there any clinical evidence of thyroid, adrenal or pituitary insufficiency. Four of the 5 patients who died unfortunately did not come to autopsy to see if they had hyperplasia or Islet-cell tumors, so these possibilities are not excluded. However, there was not any clinical evidence for the above conditions, except advanced congestive heart failure, respiratory failure and hepatic dysfunction. In the fifth patient (No. 11), an autopsy was performed and revealed pulmonary tuberculosis, fibrosis of the lungs, right ventricular hypertrophy and right atrial enlargement, and chronic passive congestion of the liver. There was not any evidence of valvular heart disease, no coronary heart disease, and no evidence of tuberculosis in the other organs, including the adrenal gland, and no insulinoma.

In conclusion, it is suggested that in any patient presenting with congestive heart failure and confusion or coma, blood should be drawn for checking glucose for possible hypoglycaemia, in which case the patients should be treated with hypertonic glucose and then glucose infusion. Also, in diabetic patients developing congestive heart failure, insulin, and/or oral hypoglycaemic agents should be reduced or discontinued, to prevent hypoglycaemia.

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