CASE REPORTS

Intravascular haemolysis complicating treated non-ketotic hyperglycaemic diabetic coma

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Summary

A patient with non-ketotic hyperglycaemic diabetic coma is reported with the complications of cerebral oedema, oliguric renal failure, transient cataracts, possible acute pancreatitis and intravascular haemolysis. The last association has not been reported before, and it may have been due to rapid infusion of hypotonic saline producing haemolysis locally at the infusion site.

Case report

A 65-year-old Caucasian woman was admitted to hospital semi-comatose. There had been increasing thirst, polyuria and anorexia for 2 weeks, and for 2 days weakness and drowsiness, but no other relevant symptoms. 1 year previously she had had a pulmonary embolus, been found to have ischaemic heart disease and taken digitalis, diuretics and potassium supplements since.

On examination she was obese, very dehydrated, apyrexial and extremely drowsy. Respiratory depth was normal and the breath did not smell of ketones. The heart rate was 160 beats/min and the blood pressure 80/60 mmHg. Tenderness and guarding were present in the epigastrium and right hypochondrium. The central nervous system was normal apart from the depressed level of consciousness. The lenses were white and opaque.

Chest X-ray showed cardiomegaly but normal lung fields, and supine and erect abdominal X-rays were normal. The urine was pale and routine analysis showed $>2\%$ glycosuria with a trace of ketones. Haemoglobin 17.2 g/100 ml, PCV 52%, blood sugar 1120 mg/100 ml, urea 125 mg/100 ml, Na^+ , K^+ and Cl^- normal with HCO_3^- 23 mEq/l. The calculated serum osmolality was 384 mmol/l.

A diagnosis of non-ketotic hyperglycaemic diabetic coma with abdominal pain of undetermined cause was made and treatment started with intravenous fluids, ampicillin and insulin. The blood sugar fell rapidly on the second day and the improvement in

her level of consciousness was reversed for about 6 hr, following which it steadily returned to normal.

Despite adequate rehydration the urinary output fell to 200 ml on the second day, when the urine became red and then black and there was gross haemoglobinuria with no haematuria. Oxyhaemoglobin and methaemalbumin were present in the plasma, and on the third day the serum bilirubin was 3.4 mg/100 ml. After treatment for acute renal failure, a diuresis started on the fifth day when the blood urea had reached 245 mg/100 ml. Urine microscopy and specific gravity, creatinine clearance and blood urea had fallen to normal before the patient's discharge.

Because of the abdominal tenderness the serum amylase was estimated on the second day and found to be 1070 IU/l, raising the possibility of acute pancreatitis. It fell to less than 150 IU/l on the fifth day.

The patient made a full recovery and was discharged with the diabetes controlled by diet alone. The cataracts cleared by the second day.

Discussion

Non-ketotic hyperglycaemic diabetic coma is now a much documented entity and has recently been reviewed (*Postgraduate Medical Journal*, 1971).

Cerebral oedema occurring during treatment with insulin is well-known and its possible cause has been discussed (Clements, Prockop & Winegrad, 1968; Maccario & Messis, 1969).

Oliguric renal failure in this condition has been reported (Halmos, Nelson & Lowry, 1966) and is thought in most cases to be due to hypotension, dehydration and sometimes tubular necrosis, but in our case it is possible that haemolysis was a contributing factor.

An association between non-ketotic diabetic coma and acute pancreatitis has been found previously (Halmos *et al.*, 1966; Vinik, Seftel & Joffe, 1970).

Serum amylase levels in patients in this type of coma without clinical evidence of pancreatitis have seldom been reported and have usually been normal. The level reported in the present patient was raised but not enough to be diagnostic of acute pancreatitis. Although acute renal failure does produce raised levels it is unlikely to have been the cause here, because the amylase level fell to normal long before the blood urea began to fall.

The intravascular haemolysis lasted for a few days only and subsequent screening for abnormal haemolytic mechanisms, such as cold agglutinins, Coombs' test, osmotic fragility and haemoglobinopathy, was negative. The fact that the platelet count never fell below normal made microangiopathic haemolysis unlikely.

A more likely cause of the haemolysis became evident when considering the intravenous fluid used. In the first 6 hr the patient received 1l of 0.9% and 2l of 0.18% saline. This was followed by a further 4l of 0.18% saline in the next 24 hr. The large volume given was needed and the hypotonic solution used as it appeared appropriate. However, because of the rapidity of infusion this could have made the area around the tip of the cannula hypotonic and caused

haemolysis. One-fifth 'normal', or 0.18%, saline has been used for correcting dehydration in babies for a long time, but is seldom used now as haemolysis sometimes occurred.

We have no definite proof that the hypotonic saline infusion caused the haemolysis in our patient, but it seems likely. If large volumes of fluid are needed rapidly intravenously, 0.45% saline or infusions of 0.9% saline and 5% dextrose alternately are suitable.

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Villous adenoma in a blind duodenal loop

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Summary

A case of a villous adenoma in a blind duodenal loop is described. The difficulties in diagnosis are illustrated and the literature on duodenal villous tumours is reviewed.

Introduction

Since the first report in 1861 by Holmes, it has become apparent that villous adenomata are rare in the gastrointestinal tract, apart from the colon and rectum. In a review of ninety-three instances of villous tumours in parts of the intestinal tract other than colon and rectum (Bremer, Battaile & Bulle, 1968), eighty-two were in the stomach, five were in the jejunum and ileum and only six in the duodenum.

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A further five cases of duodenal villous tumours have been reported (Boyer & Hellfrich, 1966; Dwyer & O'Brien, 1970; Hancock, 1970; Makkar, Taek Song & Cogbill, 1969; Schorsch & Gutierrez, 1965), none have been reported in the blind duodenal loop of a polygastrectomy. The present case of villous adenoma in this situation therefore appears to be unique, in addition it presented special diagnostic difficulties and raises an interesting question in oncology.

Case report

In October 1970, a 71-year-old woman was admitted with tiredness of 7 years duration, more marked in recent months. She had been taking no medication during this period. On examination she was clinically anaemic, but there was no other