Acute pancreatitis is listed by Joslin et al. as a lesion of possible aetiological significance in the pathogenesis of diabetes mellitus. Diabetic coma, however, is an unusual manifestation of acute pancreatitis. Toffler and Spiro describe 3 cases of coma due to acute pancreatitis in which the blood-sugar level was raised—in 2 of these cases the patients had previously been free of diabetes. Hughes found 57 cases of diabetic acidosis with acute pancreatitis reported in the world literature and added a case of his own. Forty of these occurrences were in known diabetic patients. Andrews, Nagler and Taylor, and Treece et al. describe cases of acute pancreatitis presenting as diabetic coma. Davidson has described the first case of diabetic coma without ketoacidosis in a patient with acute pancreatitis.

Case Report

History. A 60-year-old widow was admitted in a comatose state to the medical ward. She had been comatose for 5 days before admission. There was no family history of diabetes and the patient was not a diabetic (this history was obtained from relatives of the patient).

Examination. The patient was comatose and did not respond to painful stimuli. She was cyanosed, dehydrated, and there was no clinical evidence of the presence of jaundice or anaemia. Her pulse rate was 120/minute, temperature 100°F, respiratory rate 38/minute and her blood pressure was 60/30 mmHg. Crepitations were present at both lung bases. The liver was enlarged by one finger. Muscular tone and tendon jerks were normal and the plantar reflexes were flexor.

Laboratory reports. Her urine contained sugar (+ + + +) and acetone. Blood sugar estimations ranged from 262 mg. to 960 mg./100 ml. The blood urea ranged from 205 to 300 mg./100 ml. and her serum CO₂ content ranged from 12 to 23.5 mEq./l; the remaining serum-electrolyte levels were within normal limits. Her serum-amylase level was normal.

Treatment. Intravenous fluid therapy was instituted and large doses of soluble insulin were administered intravenously and intramuscularly. Pressor agents were used to maintain her blood pressure and antibiotics were given to treat her chest infection.

In spite of vigorous therapy the glycosuria and hyperglycaemia remained constant features, she could not be weaned off pressor drugs, and she remained comatose, finally dying several days after admission.

Autopsy findings. The peritoneal cavity contained bile-stained fluid. The pancreas was grossly swollen and had a mottled discoulouration, being white and chalky in some areas and haemorrhagic in others. The fat of the omentum and the fat around the pancreas was swollen and flecked with small white foci. Histological sections of the pancreas showed the histological features of an acute haemorrhagic pancreatic necrosis. The organ was necrotic with haemorrhages, congestion, thromboses in the vessels and a polymorphonuclear leukocytic infiltration. The fat in the organ appeared to have undergone fat necrosis, having a diffuse ground-glass basophilic appearance. The islets of Langerhans showed no hyalinization. A cholesterol stone was present in the neck of the gallbladder.

Discussion

Tully and Lowenthal list the following characteristics of the diabetic coma of acute pancreatitis:

1. The elevated blood sugar drops more rapidly than that of uncomplicated coma.
2. The serum amylase is raised.
3. Dehydration is extreme in the severe cases. These patients cannot be adequately hydrated without plasma and/or whole blood as well as fluid and electrolytes.
4. There are brief periods of increased insulin production or reduction of glucagon, which leads to episodes of hypoglycaemia.
5. Severe prostration is present.
6. There is failure to improve despite adequate therapy.
7. Abdominal examination may show tenderness and spasm in the epigastrum. If the patient is unconscious when seen for the first time, guarding and spasm may still be elicited.
8. Abdominal pain with those characteristics of pancreatic distribution is present.
9. Sweating occurs at some stage of the disease.

The case described shows many of the features described by Tully and Lowenthal. The serum amylase in this patient was normal. This may be owing to the test being done more than 72 hours after onset of the disease, the critical time for a raised serum amylase in acute pancreatitis, or to the severe degree of pancreatic necrosis that was present.

Acute pancreatitis may sometimes be the underlying cause of resistant diabetic coma. Hughes reviews 11 cases of death in resistant diabetic coma in which the main finding at autopsy was acute pancreatitis.

Marked glycosuria is a bad prognostic sign in acute pancreatitis because it is indicative of a severe degree of damage to pancreatic tissue. Mild glycosuria may appear in acute pancreatitis; Bell found glycosuria to be present in 35% of his cases of acute pancreatitis; Schumacker reviewed 700 cases of acute pancreatitis and found glycosuria in only 11%.

Hyperglycaemia is a frequent finding in acute pancreatitis. In Fallis' series hyperglycaemia was present in 47% of cases of acute pancreatitis; Schumacker reports 50% of his cases with blood-sugar levels of 200 mg./100 ml. or more.

In general it is agreed that acute pancreatitis may be followed by diabetes mellitus of short duration but permanent diabetes rarely follows acute pancreatitis—only 2% of cases of acute pancreatitis will develop diabetes as a permanent disease.

Permanent diabetes will develop in 14% of subjects with chronic pancreatitis without calcification of the pancreas, and in about 45% of those with calcification.

The majority of cases of acute pancreatitis presenting as diabetic coma, which have been described in the literature, have been misdiagnosed as simple diabetic coma; the pancreatitis was diagnosed at autopsy only. The condition carries a high mortality. In non-diabetics the mortality of cases with acute pancreatitis and acidosis is 83%, and in addition the condition is rare in non-diabetics.
SUMMARY

A case of acute pancreatitis presenting in diabetic coma in a non-diabetic patient is described. This is an unusual presentation of acute pancreatitis and it carries a high mortality.

I wish to thank Dr. M. M. Suzman, under whose care this patient was admitted, and the Medical Superintendent of the Johannesburg General Hospital for permission to publish. I also wish to thank Dr. C. Abrahams for the pathological report.

New Appliance

ANAESTHETIC EQUIPMENT FOR THE FIRST 6 MONTHS OF LIFE

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General anaesthesia for the newborn and infants in the first 6 months of life is potentially more dangerous and difficult to conduct than in the older age groups. It is generally agreed that many of the hazards are inherent in the equipment currently in use.

Although the available equipment is suitable for older infants, it does not comply correctly with the physiological and anatomical requirements of small infants.

Disadvantages of Existing Equipment

Besides being rather cumbersome and difficult to maintain in position, the chief drawbacks are dead-space and resistance to expiration arising from excessive tubing through which the expired gases have to travel.

To overcome dead-space, high flow rates of gases (these are dry) are used—but the lungs are then subjected to increased pressure from the incoming gases.

Another disadvantage of high flow rates is the cost of the anaesthetic due to wastage. This is proportionally high, depending on the cost of the anaesthetic agents.

The equipment, designed by myself, conforms to the ventilatory requirements of the very young and overcomes the objections raised above.

Principles of New Device

Dead-space and resistance have been curtailed to a bare minimum, namely 1.5 ml. The re-breathing tubing seen between thumb and forefinger (A in Fig. 1), is only a few inches in length and its lumen, although considerably reduced, will offer no resistance to the expired gases if re-breathing is permitted. This tubing must not be confused with the tubing from the anaesthetic machine which delivers the gases into the bag mount (B in Fig. 1).

The 350-400 ml. re-breathing bag, the smallest previously available, is replaced by a miniature rubber bag which, in reality, is a balloon with a capacity of approximately 100 ml. It is now comparatively easy to acquire the ‘feel’ of the patient, namely, the resistance of the thoracic cage and assess the correct amount of ventilation.

The equipment, made of plastic (acrylic—Perspex), is extremely light and simple to handle. It is so compact and small as to fit literally into the palm of the hand.

Since dead-space and resistance have been minimized, it is now possible to administer effective and safe anaesthesia with considerably reduced minute-volumes.

It is also possible to reduce the cost of the anaesthetic. Indeed, a saving of as much as R1.50 an hour can be effected when agents such as nitrous oxide, oxygen and halothane are administered.

Fig. 1. See text.

A great deal of the improvements can be ascribed to the device (Fig. 2A, 2B) which is used with a face-mask (Fig. 1), or with an endotracheal tube (Fig. 3).

Features

The device embodies the following features:

1. Its over-all dead-space is less than ¼ ml. and re-breathing during spontaneous respiration can be prevented with small gas flow rates.

2. The fresh gases enter the vertical limb via the horizontal limb and are evenly dispersed (Fig. 2B). There is therefore less hindrance to expiration from the incoming gases than with other devices (Fig. 2C).

3. The mechanism for the escape of gases from the circuit is housed in the upper portion of the vertical limb. It consists of a tap (Fig. 2B) which can be screwed in and

REFERENCES