

Clinical Reports

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Starting with this issue, the *Clinical Workshop* section will be referred to as the *Clinical Reports* section more adequately to emphasize the merit and importance of articles concerned with the clinical basis of the practice of anesthesiology—Ed.

Hyperosmolar Hyperglycemic Nonketotic Coma, A Cause of Delayed Recovery from Anesthesia

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In 1957, Sament and Schwartz¹ reported a case of profound coma and hyperglycemia, but without acidosis. Since then, many such cases² have been published. A number of these reports were cases of patients who either had mild diabetes or were without known diabetes. The syndrome of nonketotic hyperosmolarity, dehydration, and hyperglycemic coma is frequently associated with diseases requiring surgical intervention.² Review of the literature reveals one previous account of a patient who developed this syndrome after anesthesia, with fatal results.⁶ The present report describes such a complication and discusses the diagnosis and management of the condition.

REPORT OF A CASE

An 82-year-old Caucasian woman was admitted to the hospital with complaints of claudication on walking and angina pectoris. Relevant past history included diabetes, infectious hepatitis, and glaucoma. The diabetes was controlled by 55 units NPH-50 insulin every morning. The urine was clear and negative for glucose and acetone. Electrolytes and blood chemistry values were normal except for a blood glucose of 240 mg/100 ml. The electrocardiogram showed left axis deviation, a left anterior hemi-block, and a widened

P wave. Roentgenogram of the chest was normal. The patient was scheduled for bilateral lumbar sympathectomy. Premedication included pilocarpine eye drops instilled in both eyes immediately before the administration of atropine, 0.4 mg, and diazepam, 10 mg im. The patient arrived in the operating room with an intravenous infusion of dextrose, 5 per cent in water, 1,000 ml, containing 10 units of regular insulin. After preoxygenation, anesthesia was induced with sodium thiopental, 300 mg, and succinylcholine, 60 mg, was given to facilitate tracheal intubation. Anesthesia was maintained with nitrous oxide, 2.5 l/min, and oxygen, 2.5 l/min. Supplemental doses of meperidine and pancuronium were added when necessary. During the three hours of anesthesia the patient received meperidine, 220 mg, pancuronium, 15 mg, whole blood, 1,000 ml, and dextrose, 5 per cent in water, 1,000 ml. At the conclusion of the operation for bilateral lumbar sympathectomy attempts to reverse the action of pancuronium with prostigmine, 5 mg, plus atropine, 2 mg, and that of meperidine with naloxone, 0.4 mg, were unsuccessful. The blood pressure was 160/100 mm Hg, pulse rate 90/min, and the electrocardiogram remained unchanged. The patient remained deeply comatose. Vital signs remained stable. Neuromuscular transmission was not monitored during or after the operation. The patient was moved to the recovery room, ventilation being mechanically controlled. In the recovery room her condition remained unchanged, with no sign of returning consciousness or spontaneous respiration. Rectal temperature was 36.2 C. The urine contained 4+ glucose but no acetone or diacetic acid. A tentative diagnosis of cerebrovascular accident was made. Blood was drawn for examination of electrolytes and blood gases. While these tests were being made, further treatment was carried out with regular insulin, 10 units iv, digoxin,

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0.75 mg, im, and a second dose of naloxone, 0.4 mg iv. The patient remained unconscious, without any respiratory effort. The laboratory values were: arterial blood pH, 7.41; PaCO_2 , 26 mm Hg; PaO_2 , 97 mm Hg; O_2 saturation, 99 per cent; chloride, 77 mEq/l; CO_2 , 20 mEq/l; potassium, 3.2 mEq/l; sodium 127 mEq/l; glucose 1,185 mg/100 ml; BUN 16 mg/100 ml. At this time the patient's condition was diagnosed as hyperosmolar, hyperglycemic nonketotic coma. The patient was transferred to the intensive care unit, where she made a complete recovery after being in coma for approximately 12 hours.

Treatment in the intensive care unit included administration of 0.45 per cent saline solution, 1,000 ml, followed by dextrose, 5 per cent in water. Regular insulin, 25 units, was administered intravenously by a single injection and repeated in 30 minutes. On the first postoperative day the patient received 65 units of regular insulin in divided doses as judged by urinalysis. Potassium chloride 60 mEq/l was added to the intravenous solutions of dextrose in half-physiologic saline solution and dextrose, 5 per cent in water. Blood glucose was 360 mg/100 ml and potassium 5 mEq/l. On the second postoperative day, 48 units NPH-50 insulin were administered, in addition to coverage by regular insulin as necessary. Blood glucose now was 175 mg/100 ml and potassium, 4.2 mEq/l. Further treatment consisted of the administration of digoxin, 0.25 mg, and antibiotics. The digoxin was repeated on the third postoperative day. The patient was monitored by electrocardiogram, central venous pressure readings, daily hematocrit determinations, and several chest roentgenograms.

DISCUSSION

The pathophysiology of the hyperosmolar syndrome has not been clearly established. Most investigators agree that the syndrome results from a decrease in intracellular water within the brain due to the osmotic effects of these predominantly extracellular (dextrose, etc.) substances.² The disturbance occurs equally frequently in male and female patients, most of the patients being middle-aged or elderly. Besides many of the reported cases being those of patients with mild diabetes, the condition is often found as a complication of steroid therapy,⁵ pancreatitis and a variety of other conditions.² Mortality from this disorder is high, approximately 60 per cent.

According to most investigators,³ a blood glucose concentration above 600 mg/100 ml in a comatose patient without significant ketosis is usually accompanied by serum

osmolality of our patient was calculated to be 331.9 (normal, 285–295). Several aspects of this disorder are of special interest to the anesthesiologist: During induced hypothermia a small amount of glucose, 65–75 g, iv, has resulted in blood sugar levels as high as 1040 mg/100 ml—this may be a cause of hyperosmolar coma.⁴ Many cases are diagnosed as “acute stroke,” as was our case. Most patients are kept without oral intake for 12 hours prior to operation, which may cause considerable dehydration, especially during the summer in rooms not efficiently air-conditioned. Diabetic patients should be given fluids intravenously several hours before operation is undertaken.

The anesthesiologist should be alert to the possibility of this syndrome when faced with a patient who remains comatose inappropriately for longer than usual periods following the termination of anesthesia. Failure of the patient to show a normal pattern of response to drug antagonists also suggests this diagnosis. Blood samples should then be obtained and analyzed for glucose, acetone, diacetic acid, and serum osmolality. These tests will indicate the diagnosis quite accurately, and specific therapy can be promptly instituted.

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