

Clinical Reports

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Primary Aldosteronism with Uncommon Complications

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A case of primary aldosteronism with severe total-body potassium depletion and its uncommon manifestation is reported. Virtually all features of this disease, caused by an aldosterone-secreting adenoma (Conn's syndrome)¹⁻³ are attributable to aldosterone, which conserves sodium and promotes the excretion of potassium, leading to progressive depletion of total body potassium stores.

REPORT OF A CASE

In September 1974, a 28-year-old Caucasian man weighing 84 kg was admitted to Columbia-Presbyterian Medical Center for the first time, for evaluation of hypertension of eight years' duration. According to his history, in June 1974, after two weeks of thiazide therapy, he had collapsed and had been found to have severe hypokalemia and flaccid quadriplegia, which had been reversed following potassium supplementation. Evaluation revealed persistent hypokalemia, a right adrenal mass, high aldosterone levels, and decreased renin activity in plasma, pointing to a diagnosis of primary aldosteronism, confirmed by high aldosterone levels in the blood in the right adrenal vein. Treatment with an investigational aldosterone antagonist (Win

24540) was started, and on readmission for right adrenalectomy, in December 1974, blood pressure was in the range of 120-170/70-115 torr; the EKG was normal (a previous EKG had shown negative T waves and prominent U waves in leads I and V₂₋₆). Serum electrolytes (Na⁺, 135; K⁺, 4.4; Cl⁻, 100 mEq/l), as well as results of additional studies, were within normal limits. Following premedication (diazepam, 10 mg, im), infusion of 5 per cent dextrose in water was started and anesthesia induced with thiopental 2.5 per cent, iv, in increments to a total dose of 900 mg. Tonic muscular contractures of arms, shoulders, neck and anterior chest wall developed upon blood-pressure cuff inflation, first thought to be due to a light anesthetic state. Halothane, 0.5-1.5 per cent in oxygen, was started; however, localized contraction persisted despite increasing anesthetic depth. The possible diagnoses of myotonia dystrophica, malignant hyperpyrexia, electrolyte imbalance, and cerebrovascular accident were considered and anesthesia discontinued. Serum electrolytes, serum CPK levels, arterial blood gases, and temperature remained normal, and the patient recovered completely.

Two muscle biopsies, for histologic and physiologic studies of muscle responses to caffeine and halothane, did show abnormalities, although they were not characteristic of malignant hyperpyrexia. Threshold concentration of caffeine to elicit contraction and the maximum amplitude of caffeine-induced contractures were higher than normal, indicating that the capability of sarcoplasmic reticulum to release calcium was increased, resulting in tension greater than normal. Treatment with spirinolactone was initiated and surgical intervention deferred for further evaluation. Despite this conservative treatment, the patient remained persistently hypokalemic and hypertensive, and tired easily, and surgical treatment was reconsidered. Since the muscle biopsy studies were inconclusive, other possible causes of the muscle contractures during anesthesia were considered. Increased muscle excitability due to potassium derangement seemed most likely.

On readmission of the patient for operation in November 1975, serum K⁺ was 2.3 mEq/l and blood pressure in the range of 120-140/70-90 torr. To determine whether there was a marked potassium deficit and to approximate when total body potas-

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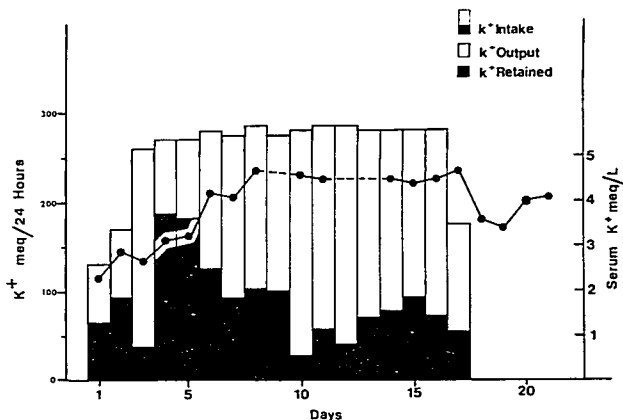


FIG. 1. Diagrammatic representation of potassium balance study during a course of oral K^+ supplementation with Na^+ restriction. Note the correction of serum potassium after the third day of oral K^+ supplementation and the rate of potassium retention after nine days.

sium deficit had been corrected, potassium balance was measured during oral supplementation.⁴ After nine days of this regimen, potassium plateaued at a lower level, indicating that the deficit was corrected (fig. 1). When the patient had retained approximately 1,300 mEq of potassium (indicating that his total intracellular potassium might have been depleted by 30–40 per cent, he was scheduled for operation.

Serum electrolytes on the day prior to operation were normal (Na^+ , 136; K^+ , 4.5; Cl^- , 100 mEq/l), as was the EKG. Blood pressure fluctuated between 130–200/60–120 torr; weight was 69.9 kg. Preparations for possible development of malignant hyperpyrexia were made. Following premedication (secobarbital, 100 mg, meperidine, 50 mg, and atropine, 0.4 mg, im), the patient arrived in the operating room awake and apprehensive. An intravenous infusion line, a central venous pressure catheter, and an arterial cannula were inserted. Droperidol, 2.5 mg, iv, was given. An epidural catheter was placed through a 16-gauge Tuohy needle at the L2–3 interspace and chloroprocaine, 3 per cent, 30 ml, injected; a sensory level of T4 was achieved. A temperature probe was inserted into the nasopharynx following topical anesthesia with tetracaine, 0.5 per cent, 5 ml. Analgesia for right adrenalectomy was maintained for four hours with chloroprocaine, 3 per cent, meperidine, and droperidol (total doses; 2,100, 200, and 12.25 mg, respectively). The procedure was well tolerated, the postoperative course uneventful, and the patient has been normotensive and normokalemic since.

DISCUSSION

During induction of anesthesia the altered balance of cortical and/or spinal inhibitory and facilitatory pathways' influence on spinal motoneurons (the former is inhibited while the latter is not altered) facilitates the increased muscle excitability due to potassium derangement.⁵⁻⁷ The extent of muscle excitability is, among other factors, a function of the difference between the threshold of excitation and the resting membrane potential which is, according to the Nernst equation E_K (transmembrane potential) = $-61.5 \log K_i/K_o$, determined mainly by the ratio of intracellular (K_i) and extracellular (K_o) potassium concentrations. In initial stages of potassium deficiency the value of K_o is reduced relatively more than that of K_i , increasing the negativity of the resting potential and thus bringing it further from the threshold potential, which conforms with a clinical picture of decreased muscle tone and strength. With time, K_i continues to decrease, bringing the resting membrane potential to a less negative value, thus partially compensating for the changes caused by the initial decrease in K_o . When

such a patient is started on potassium supplementation, the process is reversed and K_e levels can be normal before K_e deficit is corrected, resulting in a less negative resting membrane potential than at normal K_e/K_o , bringing it closer to the threshold of excitation, which can lead to increased muscle excitability. Our patient was probably in such a phase when anesthetized the first time.

This case illustrates the extensive K^+ deficit with uncommon clinical manifestation that a relatively large aldosterone-secreting adenoma can produce. It also illustrates that we lack readily accessible means to evaluate this deficit, except by measuring potassium balance, which is not always practical. Serum potassium level is apparently not a reliable criterion, since its normalization occurs before the K_e deficit is corrected, and EKG findings may also be equivocal. It would seem advisable to consider the nature and severity of the underlying cause of hypokalemia and its duration in order to evaluate properly the potassium derangement, which, if not corrected, can lead to well-known and dangerous complications (arrhythmia), as well as to some less-common complications, as presented here.

SUMMARY

In a patient who had primary aldosteronism and severe total-body potassium depletion muscular tonic contractures developed during induction of anesthesia. After correction of the potassium deficit, the patient underwent un-

eventful anesthesia and transabdominal right adrenalectomy. Neither serum potassium level nor EKG seems to provide a reliable index of correction of potassium deficit. Measurement of potassium balance provided a method of quantitating the potassium depletion and of determining when the potassium deficit had been corrected. Balance studies should be utilized preoperatively when long-term potassium loss is suspected to reduce complications secondary to hypokalemia.

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