

Intraoperative Hypokalemia Leading to Diagnosis of Adrenal Adenoma

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Primary hyperaldosteronism is a rare but potentially surgically curable form of hypertension. A case of hyperaldosteronism, which was accidentally discovered during an oral surgery procedure under general anaesthesia, is described. The patient had hypokalemic alkalosis accompanied with mild hypertension, with an aldosterone-producing adrenal adenoma subsequently confirmed and treated successfully by adrenalectomy. The anaesthetic course and some specific findings are discussed. (*Chang Gung Med J* 2006;29(4 Suppl):39-42)

Key words: primary hyperaldosteronism, hypokalemic alkalosis, hypertension, adrenal adenoma.

CASE REPORT

A 28-year-old male, weighing 47 kg and 158 cm in height at the time of presentation, was an out-patient and scheduled for excision of maxillary bony tissue and tooth extraction due to fibrous dysplasia. Preoperative complete blood cell count and chest X-ray were normal. His pre-anaesthesia blood pressure (BP) was 152/97 mmHg and heart rate was 57 beats/min. General anaesthesia was induced with fentanyl 150 µg and thiopental 300 mg intravenously. Tracheal intubation was facilitated with atracurium 30 mg. Anaesthesia was maintained with 1%-1.5% isoflurane and 40% nitrous oxide in oxygen. Intraoperative monitors included electrocardiogram (ECG), non-invasive blood pressure monitor, pulse oximeter and capnography. Anaesthesia was carried out smoothly until two hours after induction. Given the protracted nature of the surgery, a single arterial puncture was performed for blood-gas analysis. This analysis revealed: pH = 7.630; partial pressure of carbon dioxide (pCO₂) = 29.8 mmHg; partial pres-

sure of oxygen (pO₂) = 298.2 mmHg; bicarbonate (HCO₃) = 31.5 mEq/L; base excess (BE) = +11.1 mEq/L; Na⁺ = 142 mEq; K⁺ = 1.7 mEq; Cl⁻ = 96 mEq/L; Ca²⁺ = 0.85 mEq/L; sugar = 116 mg%; osmolarity = 280 mOsm/kg. As hypokalemia, metabolic alkalosis and hypertension were present concurrently, a presumptive diagnosis of hyperaldosteronism was made. The mechanical ventilator was reset for decreased frequency to correct hyperventilation and an intravenous infusion of potassium chloride was begun. The ECG was checked for the characteristic signs of hypokalemia but non-specific ST-T change was our impression. The surgery proceeded uneventfully and lasted for three hours in total. The patient regained consciousness within 15 minutes of the conclusion of the surgical procedure.

After full recovery from general anaesthesia in the post-surgery care unit, a more detailed past history was obtained. The patient stated that he had not been aware of any hypertension or associated problems except for the occasional headache, lower extremity weakness after vigorous exercise and a tin-

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Received: Sep. 10, 2004; Accepted: May 31, 2005

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gling sensation in the upper extremities, which had occurred intermittently for several years. After admission to a ward, his BP ranged between 143/82 to 168/106 mmHg. Serum cortisol concentration, sampled in the morning, was in the normal range, plasma renin level was low and aldosterone level was elevated. Serum creatinine and BUN levels were within normal limits. A low-density nodule in the right adrenal gland 1 cm in diameter was noted on computerized tomography. Results compatible with hyperaldosteronism were revealed by ^{131}I -6 beta-iodomethylnorcholesterol (or NP-59) scintigraphy during dexamethasone suppression. A right adrenalectomy was performed under general anaesthesia without any subsequent complications as of follow-up two months after the previous surgery. During surgery, an adenoma measuring 1 x 1 cm was identified over the lower pole of the right adrenal gland. Histological examination confirmed the diagnosis. The surgical procedure was uneventful and the patient recovered well postoperatively. At follow-up three weeks post surgery, his BP was normal and serum potassium level rose spontaneously to 4.37 mmol/L. Plasma renin and aldosterone had returned to normal levels, and there was no need for any subsequent medical treatment for this patient.

DISCUSSION

Hypokalemia is a common electrolyte disturbance during anesthesia. Intraoperative hypokalemia can be due to: (1) prolonged fasting prior to surgery; (2) hyperventilation of the lungs; (3) hypothermia;⁽¹⁾ (4) stress-induced translocation of potassium; and (5) glucose-solution challenge. However, the serum potassium level (as low as 1.7 mEq) in this case would not normally be expected. Furthermore, the hypokalemic state was resistant to aggressive intravenous potassium ion supplementation in the operating theater. The serum potassium level, rechecked after the patient had received a total of 40 mEq of potassium ion by rapid infusion in three hours, revealed only an increase of 0.3 mEq. This result was compatible with chronic potassium loss rather than acute change, and mild hypertension was also determined on arrival at the surgical suite. Hypertension concomitant with chronic hypokalemia may be due to essential hypertension, renal disease, adrenocortical dysfunction and/or pseudoaldostero-

nism. If cases of essential hypertension treated with diuretics consisting of potassium-depleting drugs are excluded, the most common cause of hypokalemia for hypertensive patients is primary aldosteronism.

In 1986, Helfant et al. characterized the ECG for various degrees of hypokalemia.⁽²⁾ In this instance, the signs of hypokalemia were not detected from Lead II ECG, which was monitored from the pre-induction evaluation, with the trace judged as representing a pattern of non-specific ST-T change. When the U wave combines with a flattened T wave, it may lead to the misinterpretation of the U wave as the T wave and the prolongation of the QT interval (actually a QU interval) is difficult to distinguish from ECG tracing. Hypokalemia also influences the amplitude of the QRS complexes. There is an inverse correlation for serum potassium concentration and the Sokolow-Lyon index: SV1+RV5 (or RV6).⁽³⁾ Since a Sokolow-Lyon index greater than 35 mm is one of the criteria for left ventricular hypertrophy, ECG readings may be unreliable for diagnosis of left ventricular hypertrophy for hypokalemia patients. However, left ventricular hypertrophy may develop in a primary hyperaldosteronism patient. This dysfunction may be more pronounced for primary hyperaldosteronism patients than for those with other forms of hypertension.⁽⁴⁾

On arrival at the surgical suite before induction, the patient's BP was 152/97 mmHg, followed by 145/90 and 148/89 mmHg at subsequent 3-min intervals. After induction, the BP soon dropped and was kept rather stable (between 100-80/60-40 mmHg) under anesthesia using isoflurane 1%-1.5% and 40% nitrous oxide in oxygen. There were no large swings in BP throughout the whole course of the anesthesia. The relatively low and stable blood pressure may have been due to hypervolemia induced by sodium retention and accompanied with blunted sympathetic tone and circulatory reflexes, which are associated with chronic hypokalemia.

Estimated prevalence of primary hyperaldosteronism ranges from 0.05%-2% of the hypertensive population,^(5,6) with recent evidence from Asia suggesting that it may be at least 5% in this region.⁽⁷⁾ The clinical features of primary hyperaldosteronism are not specific and some patients remain completely asymptomatic or have minimal symptoms. Others may have symptoms related to hypertension (e.g. headache), hypokalemia (e.g. polyuria and nocturia,

or muscle cramps) or both. Sometimes hyperaldosteronism is accidentally detected during a prenatal examination or health examination. For our patient, primary hyperaldosteronism was suspected because of the presence of hypertension and hypokalemia with metabolic alkalosis. At least six subtypes of primary hyperaldosteronism have been identified,⁽⁸⁾ with about two-thirds of all cases caused by an aldosterone-producing adenoma.⁽⁹⁾ Adrenalectomy cures the hypertension and hypokalemia for most patients who have aldosterone-producing adenomas.⁽¹⁰⁾ Pre-surgical administration of spironolactone (except in pregnancy) together with oral potassium supplementation may decrease the risks associated with anesthesia by correcting the hypokalemia and reversing hypertension,⁽¹⁰⁾ and minimizing the incidence of the postoperative complication of hypoadosteronism.

Although 7%-38% of patients with hyperaldosteronism have a low to normal serum-potassium level^(11,12) and very few patients have normal blood pressure,⁽¹³⁻¹⁵⁾ spontaneous hypokalemia in a hypertensive patient is a strong indicator for hyperaldosteronism. To the best of our knowledge, this may be the first confirmed case of hyperaldosteronism detected during an unrelated surgical procedure. We conclude by recommending vigilance for any abnormal laboratory data or clinical signs that may serve to assist in the diagnosis of undiscovered diseases during anesthesia.

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手術麻醉中非預期低血鉀導致醛固酮腺瘤的發現

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原發性皮質醛固酮分泌過多症是造成高血壓的原因之一，如果此症是因醛固酮腺瘤所引起，甚至可以外科手術切除腺瘤，而根治高血壓。根據以往西方文獻的統計，原發性皮質醛固酮分泌過多症，盛行率約占高血壓人口的 0.05~2 %，東方人這個比率或許更高。由於其症狀有時隱微不顯，一般不容易為病人覺察求醫，有患者甚至是在例行健康檢查或產前檢查中才被發現。本案例為可查文獻中第一例是在手術麻醉過程中，因有輕微高血壓，低血鉀及合併代謝性鹼血症等徵象，而被懷疑並於其後得到證實者。本文敘述此案例麻醉的經過及特別的發現，並探討麻醉應注意的事項。(長庚醫誌 2006;29(4 Suppl):39-42)

關鍵字：原發性皮質醛固酮分泌過多症，低血鉀併代謝性鹼血症，高血壓，醛固酮腺瘤。

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受文日期：民國93年9月10日；接受刊載：民國94年5月31日

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