RECENTLY, the case rate of tuberculosis has been increasing again. Addison’s disease (chronic primary adrenal insufficiency) caused by tuberculosis, however, is a rare disorder.1

We describe a patient with a history of tuberculosis who demonstrated sudden unconsciousness accompanied by hypotension, hypotension, and hypoglycemia during a routine preoperative examination. Further hormonal and radiologic examination revealed tuberculous Addison’s disease. The unconscious event may have been caused by abrupt onset of adrenal crisis.

**Case Report**

A 62-year-old man, 162 cm in height and weighing 54 kg, was admitted to the university hospital for lower maxillectomy and radical neck dissection for recurrent gingival cancer. He had a history of tuberculous pleuritis and periostitis approximately 40 yr ago, but there was no evidence indicating active tuberculosis. One year before admission to our institution, he underwent surgery for removal of gingival cancer and radiation therapy at another hospital. The perioperative clinical course was uneventful and laboratory findings were normal. Since then, he lived normally.

In the first preoperative evaluation on the admission day, he showed no clinical symptoms except for pain at the cancerous lesion of the gingiva. Blood pressure and heart rate were 124/76 mmHg and 70 beats/min, respectively. Serum sodium concentration was 124 mEq/l, but there was no other abnormal laboratory finding. Many examinations were performed during the next several days, all with no remarkable findings.

Five days after admission, 1 h after breakfast, he underwent a Gallium scan to examine bone metastasis of the cancer. One hour later, he underwent a bleeding-time examination. During the examination, he suddenly lost consciousness. He breathed spontaneously, but blood pressure could not be detected. His legs were immediately tilted upward, oxygen was administered by mask, and an intravenous infusion of lactated Ringer’s solution was started. A few minutes later, systolic arterial pressure was still at 60 mmHg. An infusion of dopamine 5 μg · kg⁻¹ · min⁻¹ was also started. Then, the blood glucose concentration during the resuscitation was found to be < 40 mg/dl. Twenty milliliters glucose, 50%, in saline was immediately injected intravenously, and intravenous fluids newly changed to 5% dextrose in lactated Ringer’s solution. Within a few minutes, his consciousness became clearer. Blood pressure was 100/80 mmHg and blood glucose concentration was 88 mg/dl. Serum sodium concentration was 118 mEq/l, potassium concentration was 4.9 mEq/l, and chloride concentration was 85 mEq/l. His condition stabilized, and his only complaint was one of general malaise.

Hormonal evaluation revealed that the plasma cortisol concentration was 12 μg/dl (normal range, 5 to 15 μg/dl). However, the corticotropin stimulation test showed almost no response (baseline cortisol concentration = 10 μg/dl, and peak cortisol concentration after administration of corticotropin = 11 μg/dl). In computed tomography and abdominal radiography studies, bilateral adrenal calcification was noted. Furthermore, hyperpigmentation on the hands and legs was also observed. He was thus diagnosed as having Addison’s disease and was treated daily with 10 mg oral hydrocortisone for the next 3 weeks. Serum sodium concentration increased to 135 mEq/l and blood glucose concentration increased to 70–80 mg/dl. No hypotension or other marked symptoms had been demonstrated since the supplemental therapy of cortisol was started.

Three weeks later, after the episode of unconsciousness, during steroid coverage (hydrocortisone 100 mg was intravenously administered three times, i.e., at induction of anesthesia, during surgery, and after surgery), the scheduled operation was performed during general anesthesia. The hemodynamic condition was stable throughout surgery and the postoperative course was uneventful.

**Discussion**

This report highlights the importance of considering Addison’s disease in the preoperative evaluation of patients with a history of tuberculosis. Tuberculosis formerly was a leading cause of Addison’s disease; however, it is now relatively rare in industrial countries.1
Addison’s disease usually proceeds insidiously and presents with a wide range of symptoms and signs. Therefore, the nonspecific nature of the symptoms frequently causes the diagnosis to be missed or delayed.¹ ³ ⁴

In this case, Addison’s disease was never suspected before the episode of unconsciousness, because the patient lived normally without any marked symptoms, such as hypoglycemia or hypotension. His previous perioperative courses were uneventful.

The positive findings for Addison’s disease in the patient were a history of tuberculosis, slight hyponatremia, and hyperpigmentation of the skin. Hyponatremia and hyperpigmentation of skin frequently occur in patients with Addison’s disease, who also demonstrate fatigue, weight loss, nausea, hypotension, and hypoglycemia.¹ In this case, however, hyperpigmentation of the skin was overlooked. The hyponatremia was also mild (at least for the control blood sample). Moreover, the provisional diagnosis of Addison’s disease was delayed until after the event described.

The episode of unconsciousness was accompanied by severe hypoglycemia, hypotension, and hyponatremia and was caused by acute adrenal crisis caused by unknown Addison’s disease. Addison’s disease can become a life-threatening adrenal crisis in any stressful situation.¹ The daily preoperative examinations after admission and those on the day of the unconscious event may have resulted in mental and physical stresses that caused the subsequent adrenal crisis.

This case suggests that during preoperative evaluation, we should consider the possibility of Addison’s disease when a patient has a history of tuberculosis and should not overlook nonspecific symptoms and signs of the underlying disease because it can be a life-threatening disorder.

References


Transient Hypotension as a Complication of Monitoring Transcervical Motor Evoked Potentials

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CENTRAL nervous system (CNS) injury is a potential complication of many surgical procedures. Intraoperative monitoring of CNS function frequently is used to warn of possible injury. Modalities include local or regional anesthesia with the patient awake, a wake-up test during general anesthesia, electroencephalography, electromyography, and various evoked potentials (EPs).¹ Evoked potentials involving spinal cord pathways are particularly useful during procedures on or near the spinal cord² and may allow localization of possible injury because of the topography of the monitored tracts. Intraoperative monitoring of motor EPs in humans was first reported in 1983³ and has since gained popularity as an improved method of detecting possible intraoperative spinal cord injury.⁴ ⁵ ⁶ Initial descriptions of the transcervical technique noted a lack of adverse effects,⁷ and it has more recently been described as “free of complication.”⁸ We report the first incidence of a significant complication of monitoring transcervical motor EPs.

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