Case Report

Rare oxygen, a rare way to diagnose Conn’s syndrome

Wendela L. Greven and Thomas van Bemmel

Department of Internal Medicine, Gelre Ziekenhuizen, location Lucas, Apeldoorn, The Netherlands

Abstract

Background. Symptoms of mountain sickness are due to hypoxia of the brain. The pathogenesis is complex, but acid–base disturbances certainly play a role. When arterial oxygen levels drop, hyperventilation is induced, resulting in a respiratory alkalosis. However, this alkalosis inhibits the hyperventilation necessary for maintaining oxygen pressure. We present a case of a patient with symptoms of mountain sickness at relatively low altitudes, who appeared to have Conn’s syndrome (primary hyperaldosteronism).

Case. A 61-year-old male with hypokalaemic hypertension presented with symptoms of mountain sickness at relatively low altitudes. Hyperaldosteronism was suspected and laboratory results showed a non-suppressible aldosterone concentration and a mild metabolic alkalosis. A CT scan of the abdomen revealed an adenoma in the left adrenal gland.

Treatment of aldosterone blockade by eplerone normalized blood pressure and the symptoms of mountain sickness at low altitudes disappeared completely.

Discussion. We suggest that in our patient with hyperaldosteronism, the pre-existing metabolic alkalosis inhibited the central respiratory centre after relatively mild hyperventilation. Therefore, mountain sickness in our patient could occur at a relatively low altitude.

Keywords: Conn’s syndrome; hyperaldosteronism; mountain sickness

Introduction

Symptoms of mountain sickness include headache, light-headedness, breathlessness, fatigue, insomnia, anorexia and nausea. These symptoms are thought to result from hypoxia of the brain [1]. Although the pathogenesis is not fully elucidated, acid–base disturbances certainly play a role. Hypoxia at higher altitudes induces hyperventilation, resulting in respiratory alkalosis [1]. This alkalosis inhibits the central respiratory centre, causing hyperventilation instead of hyperventilation. As a consequence, hypoxemia induces hypoxia of the brain, resulting in symptoms of mountain sickness.

Therefore, if acid–base disturbances are important in the pathogenesis of acute mountain sickness, patients with pre-existing acid–base changes could be more prone to develop acute mountain sickness. We present a patient with remarkable complaints of acute mountain sickness at relatively low altitudes, who appeared to have a mild metabolic alkalosis as part of Conn’s syndrome.

Case report

A 61-year-old male attended our outpatient clinic for hypertension. His medical history revealed hypertension for 3 years that was treated by his general practitioner with ACE inhibition and a thiazid diuretic. He had one remarkable complaint. When skiing with his family, he suffered from headache and nausea that faded away after a couple of days. None of his family members had these symptoms. Moreover, the patient did not have symptoms when going on summer holidays (not in mountainous regions).

Initial laboratory results showed low serum potassium (2.3 mmol/L), a normal serum sodium (142 mmol/L) and a mild respiratory compensated metabolic alkalosis [pH 7.45, HCO₃⁻ 32 mmol/L, pCO₂ 6.2 kPa (46.5 mmHg) and pO₂ 8.9 kPa (66.8 mmHg)]. Since primary hyperaldosteronism was suspected, plasma aldosterone concentration (PAC) and plasma renin activity (PRA) were measured. After replacing the ACE inhibitor with a calcium blocker, the PAC measured in upright position was 660 nmol/L and the PRA 0.4 ng/L/h. Also, urinary aldosterone was measured after 2 days of salt loading. A high and non-suppressible level of aldosterone was found (104 nmol/24 h). Furthermore, a CT scan of the abdomen showed an adenoma in the left adrenal gland.

Secondary hypertension due to Conn’s syndrome was concluded. Our patient was referred for the laparoscopic removal of the adenoma in the adrenal gland. Meanwhile, treatment of aldosterone blockade by eplerone 50 mg normalized his blood pressure. Remarkably, on his next skiing holiday he did not suffer from headaches or nausea.
Discussion

In the last decade, Conn’s syndrome (primary hyperaldosteronism) as a cause of hypertension has been diagnosed more frequently [2]. High aldosterone levels cause sodium retention, resulting in volume expansion and hypertension. Moreover, potassium and hydrogen are lost, causing hypokalaemia and a mild metabolic alkalosis.

Our patient presented with symptoms of acute mountain sickness at a height of 2000–2500 m (6561–8202 ft, the altitude of skiing area in the European Alpes). This is not an altitude that normally causes symptoms of mountain sickness [3]. Symptoms of acute mountain sickness include headache, light-headedness, breathlessness, fatigue, insomnia, anorexia and nausea [1]. The pathogenesis is not fully elucidated, but hyperventilation due to hypoxaemia likely plays a major role [1]. Pathophysiologically this could be explained as follows: when ascending a mountain, the oxygen level of the surrounding air drops. Then hypoxaemia stimulates the peripheral chemoreceptors, causing hyperventilation. This hyperventilation prevents the decrease in oxygen pressure in the blood. However, as a result of hyperventilation CO₂ pressure will also decrease. Hence, arterial pH will increase slightly, resulting in an inhibitory response of the respiratory centre in the brain. This will inhibit the hyperventilation that was necessary to maintain oxygen pressure [2]. Finally, this complex mechanism might result in symptoms of acute mountain sickness by hypoxia of the brain. Fortunately, because of renal hydrogen retention, resulting in a metabolic compensation of the respiratory alkalosis, this central inhibitory response fades away in a few days [1]. This enables people to acclimatize and live at high altitudes. The cardinal rule in treatment for acute mountain sickness is to descend to a lower altitude and, if possible, to combine this with oxygen administration [1,3]. In order to prevent high-altitude sickness, the carbonic anhydrase inhibitor acetazolamide or dexamethasone is frequently used. Acetazolamide increases the renal excretion of bicarbonate resulting in a metabolic acidosis that in turn stimulates ventilation [1]. The mode of action of dexamethasone is unknown.

In our patient with Conn’s syndrome a mild metabolic alkalosis was already present due to hyperaldosteronism. Therefore, the delicate acid–base equilibrium could more easily be disturbed. Hence, a smaller decrease in CO₂ pressure would raise the pH just enough to cause inhibition of the central respiratory centre. Thus, at lower altitudes, brain hypoxia might develop, causing symptoms of mountain sickness. This explanation is supported by the fact that after treatment with eplerone the complaints of our patient disappeared completely.

This case, describing a patient with Conn’s syndrome and symptoms of acute mountain sickness, is exemplary for unravelling the pathophysiological changes in acute mountain sickness with basic physiological principles.

Conflict of interest statement. None declared.

References

1. West JB. The physiologic basis of high-altitude diseases. Ann Intern Med 2004; 16;141: 789–800
2. Young WF. Primary aldosteronism: renaissance of a syndrome. Clin Endocrinol 2007; 66: 607–618
3. Barry PW, Pollard AJ. Altitude illness. BMJ 2003; 26; 326: 915–919

Received for publication: 6.1.08
Accepted in revised form: 23.9.08