

## Central Neurogenic Hyperventilation Secondary to a Critic Thyroid Status after Aortoortic Bypass

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**Keywords:** *lactic acidosis, metabolic acidosis, myxedema coma, central neurogenic hyperventilation*

**Running Head:** *Central Neurogenic Hyperventilation and Critic Thyroid Status*

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Central neurogenic hyperventilation (CNH) was defined for the first time by Plum and Swanson in 1959 as a syndrome with decrease in PaCO<sub>2</sub>, increase in PaO<sub>2</sub> and respiratory alkalosis with no other clinical or radiological feature of pulmonary disorder.

The main causes of hyperventilation are: pulmonary embolism, severe asthma attack, pneumothorax, myocardial ischemia, congestive heart failure, hyperthyroidism, pheochromocytoma, alcohol and drugs, severe metabolic acidosis, fever, acute pain, ischemia and traumatism. Even if most of CNH are caused by CNS tumours, especially lymphomas, invasion of brain tissue may produce tissue acidosis and develop hyperventilation.

In our literature research we found 6 cases of postsurgical CNH: 3 of them were after endoscopic ventriculostomy, 1 after anaplastic astrocytoma resection and 2 after general anesthesia in non neurosurgical procedures.

We report a case of a 63-year-old man scheduled for elective aortoortic bypass graft surgery due to a 70 mm aortic aneurysm. The patient suffered from hypertension and dyslipidemia and he was in treatment with ARBs and statins. There had been no reports of previous cerebrovascular diseases.

During the procedure, a hemorrhagic shock took place and the patient was transfused with: 8 red blood cell units, 1500cc plasma and 4 grams of fibrinogen. He arrived in the intensive care unit hemodynamically stable and breathing on a venturi mask. The arterial gasometry values at the moment were: pH 7.20, pO<sub>2</sub> 90, pCO<sub>2</sub> 45, HCO<sub>3</sub> 15, BE -7, lactate 8.5, SpO<sub>2</sub> 96%.

Acidosis was resolved within the next 24 hours. During the early postoperative phase, an acute renal failure took place with creatinine values of 2,8 and uremia of 120. He also suffered a paralytic ileus that was easily solved with IV prokinetics. Lactate levels stayed increased between 2,5-3, and hemoglobine was 10gr/dl.

Four days after surgery, the patient started to feel sleepy, hypothermic and developed an important cognitive impairment. His breathing rate was 50 bpm and he didn't answer to easy orders. The arterial gasometry values with 4 lpm nasal cannula support were:

pH 7.60, pO<sub>2</sub> 80 mm Hg; pCO<sub>2</sub> 16 mm Hg; HCO<sub>3</sub> 18 mmol/L; BE -10 mmol/L; Lactate 4, SpO<sub>2</sub> 95%.

A cranial CT-scan and MRI were performed along with an echocardiogram, chest X-ray, D-dimer and biochemical analysis. The results were normal with the exception of: creatinine 1.5, urea 47, TSH 0.001 microUI/mL (normal values 0,270- 4,200) and T4 0,1 ng/dl (normal values 0,93-1,70).

400 mg IV Hydrocortisone was administered every 24 hours in order to avoid an acute adrenal crisis. In addition, a bolus of 300 microgr IV levothyroxine was given, followed by daily doses of 100 microgr to treat hypothyroidism.

A normal cognitive level, breathing pattern and lactate values were achieved within the next 24 hours.

Hyperlactatemia physiopathology as a cause of CNH is not well known. It is suspected that bicarbonate produced as a result of high lactic acid levels could cross blood brain barrier, resulting in metabolic alkalosis and increasing CO<sub>2</sub> levels. As a result, the chemoreceptors responsible for the development of hyperventilation are activated.

The start of a critically ill patient hypothyroidism in just 4 days is not common. Our patient did not need support of mechanical ventilation, benzodiazepines administration or other drug that could affect the thyroid axis. As analgesic drug we used IV fentanyl, this may have decreased the severity of CNH as the publications of Adachi YU et al have suggested (8), it could be a good palliative treatment in CNH secondary to advanced tumours.

As a conclusion, we report Hypothyroidism Coma in critically ill patient showing up as CNH, as an important postoperative complication in abdominal aortic aneurysm surgery,

This could possibly be the first CNH case reported in this context at the time.

It is important to consider this option in our differential diagnosis when we confront a postsurgical patient after major surgery with polytransfusion and organ failure.

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