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Test Your Knowledge

Respiratory Clinics

VENTILATORY MANAGEMENT IN A PATIENT WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE: A NEED FOR CAUTION

R Khajotia *MBBS (Bom), MD (Bom), MD (Vienna), FAMA (Vienna), FAMS (Vienna).* International Medical University, Seremban, Malaysia

Address for correspondence: Dr. Rumi Khajotia, Associate Professor in Internal Medicine and Pulmonology, International Medical University Clinical School, Seremban, Malaysia. Email: rumi@imu.edu.my

Khajotia R. Ventilatory management in a patient with chronic obstructive pulmonary disease: a need for caution. Malaysian Family Physician. 2008;3(3):166-167

A 67 year old obese male patient, chronic smoker, is admitted to the Intensive Care Unit (ICU) with complaints of acute shortness of breath which has progressively increased over the past 10 days. He is a known case of paroxysmal breathlessness for the past 17 years and has been on treatment from his family physician with oral and inhaled bronchodilators. Since the patient's condition progressively worsened in spite of home treatment, his family physician referred him to the hospital. On examination, the patient is drowsy and cyanosed with a very poor respiratory drive. His pulse rate is 86/min, regular and bounding. His BP is 156/92 mmHg. His chest examination reveals diminished air entry bilaterally over the chest, with prolonged expiration and scattered rhonchi. He is immediately transferred to the ICU and put on a volume-cycled ventilator (respirator) with assist-control (AC) mode, a high respiratory rate and FiO_2 of 80%. Four hours later, the patient is found to have developed cardiac arrhythmias. His ECG shows ventricular premature contractions (VPC's) with occasional supraventricular premature beats. The arterial blood gases (ABG) done on admission and 4 hours after ventilatory management, are as follows:

	On admission	After 4 hours of ventilation	(Normal values)
pH	7.256	7.49	7.38 – 7.42
PaO2	45 mmHg	79 mmHg	80 – 100 mmHg
PaCO2	88 mmHg	47 mmHg	38 – 42 mmHg
HCO3	42 mmHg	41 mmHg	22 – 26 mmol/L
SaO2	81%	95%	95 – 100%

Question

1. What do the serial arterial blood gases (ABG) indicate?
2. What is the most likely cause behind the serial changes in the ABG readings and the cardiac arrhythmias?
3. What are the complications that may occur as a result of severe metabolic alkalosis?
4. What are the learning issues in this case report?

Answer

1. On admission, the ABG shows a moderate hypoxia with *compensated respiratory acidosis.* However, following 4 hours of ventilatory management, the ABG shows predominant metabolic alkalosis.
2. The patient is a known case of *chronic* obstructive pulmonary disease (predominantly a blue-bloater), and therefore presented on admission with hypoxia, carbon dioxide retention, raised bicarbonate (HCO_3) levels (as a consequence of metabolic compensation) and respiratory failure. However, ventilatory management with an unduly *high respiratory (ventilatory) rate* led to a rapid washout of carbon dioxide from the blood within the spate of 4 hours, leaving no time for metabolic compensation to occur, resulting in the development of *"post hypercapnoeic metabolic alkalosis"* and the consequent cardiac arrhythmias.
3. Severe metabolic alkalosis causes diffuse arteriolar constriction with reduction in tissue perfusion. By decreasing cerebral blood flow, alkalosis may lead to tetany, seizures and obtundation. Metabolic alkalosis also decreases coronary blood flow and predisposes patients to *refractory arrhythmias.* Metabolic alkalosis causes hypoventilation which may impair weaning from mechanical ventilation. It is almost always associated with hypokalemia, which can cause neuromuscular weakness and ventricular premature contractions.
4. This case report incorporates a very valuable learning issue. From here we learn that while ventilating a patient with chronic obstructive pulmonary disease, unduly high respiratory rates should be avoided. This is because the patient has chronically raised $PaCO_2$ levels along with

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raised HCO_3 levels, as a result of metabolic compensation. When an overzealous effort is made to rapidly bring down the carbon dioxide levels by increasing the ventilatory rate, the $PaCO_2$ reduces rapidly leaving very little time for metabolic compensation to set in. Consequently, the patient develops severe post-hypercapnoeic metabolic alkalosis with potential complications. In conclusion, it must be emphasized that the $PaCO_2$ levels in patients with chronic obstructive pulmonary disease should be gradually reduced, giving adequate time for metabolic compensation to set in, thereby preventing a severe metabolic alkalosis from occurring, along with its consequent complications.

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RECOMMENDED FURTHER READING:

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High consumption of sugar sweetened soft drinks and fructose is associated with an increased risk of gout in men

Choi HK, Curhan G. Soft drinks, fructose consumption, and the risk of gout in men: prospective cohort study. *BMJ*. 2008;336(7639):309-12.
In this cohort study, 46 393 men with no history of gout at baseline, provided information on intake of soft drinks and fructose through validated food frequency questionnaires. During the 12 years of follow-up 755 confirmed incident cases of gout were reported. Increased intake of sugar sweetened soft drinks and fructose (from fruits and fruit juices) at baseline was associated with an increasing risk of gout.

U.S. Preventive Services Task Force. Screening for Prostate Cancer: U.S. Preventive Services Task Force Recommendation Statement. *Ann Intern Med.* 2008;149(3):185-91.
Recommendations: Current evidence is insufficient to assess the balance of benefits and harms of screening for prostate cancer in men younger than age 75 years (I statement). Do not screen for prostate cancer in men age 75 years or older (Grade D recommendation)