Case report

The Emergency Medical Service of Paris (SAMU, France) was called to a 35-year-old woman complaining of dyspnea. Her past medical history included simple chronic asthma for more than 10 years and arterial hypertension. She had never required hospitalization for her pulmonary disease and was well managed with chronic therapy, including inhaled beta-agonists, inhaled corticosteroids and theophylline. A few minutes before the call, she instilled a topical ophthalmic beta-blocker (carteolol), which had been recently prescribed for her. An acute severe asthma attack immediately followed the instillation.

The advanced life support team arrived at her home 12 min after receipt of the call. The patient was in severe respiratory distress, with extreme agitation, sweating and cyanosis. She was unable to speak at all. Her peak expiratory flow rate (PEF) was impossible to measure. The pulmonary examination revealed no breath sounds. Her blood pressure was 160/80 mmHg, pulse 90/min and respiratory rate 40/min.

Peripheral intravenous (i.v.) access, cardiac monitoring and continuous pulse oximetry were instituted. The initial treatment included inhaled beta-agonists, systemic corticosteroids (prednisolone 60 mg), salbutamol 1 mg/h i.v. and oxygen (8 l/min). This initial management failed and the patient became somnolent with acute oxygen desaturation (SpO2 = 68 %). Rapid sequence intubation, using ketamine 2 mg/kg i.v. and succinylcholine 80 mg i.v., and mechanical ventilation were swiftly achieved. The patient was initially very difficult to ventilate. The highest peak inspiratory pressure was greater than 60 cm H2O. The treatment was modified, with administration of epinephrine 3 mg/h by continuous i.v. infusion, supplemented by 1 mg intratracheally every 5 min and salbutamol 6 mg/h continuous i.v. infusion, also supplemented by repeated intratracheal administration. Sedation was obtained with midazolam 10 mg/h and paralysis with vecuronium 6 mg/h, both by continuous i.v. infusion. Central venous access was performed for the infusion of adrenergic agents. One and one-half liters of Ringer’s solution was infused during her prehospital management. The patient was transferred by Mobile Intensive Care Unit to a Pulmonary Intensive Care Unit.

Abstract

Objective: We describe a patient with a prolonged and severe hypercapnia occurring during an episode of status asthmaticus induced by ophthalmic instillation of carteolol.

Setting: Prehospital Emergency Medical Service and Pulmonary Intensive Care Unit in a university hospital.

Patient: A 35-year-old female developed an acute asthma attack while at home, which required advanced life support.

Intervention: On hospital admission, arterial blood gases revealed a PaCO2 of 208 mmHg. Hypercapnia persisted with a PaCO2 of more than 190 mmHg for 10 h, with pH always less than 7.00. The patient was finally discharged after 26 days without sequelae.

Conclusion: This case illustrates the cerebral and cardiovascular tolerance of severe and prolonged hypercapnia associated with major acidosis.

Key words Near fatal asthma · Mechanical ventilation · Hypercapnia · Respiratory acidosis

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Prolonged severe hypercapnia complicating near fatal asthma in a 35-year-old woman
On arrival, her blood pressure was 140/110 mmHg, the heart rate 101 beats/min and the axillary temperature 36.0°C. The laboratory findings on admission were as follows: arterial blood gas (100% FIO₂): pHa 6.73, PaCO₂ 208 mmHg, PaO₂ 69 mmHg, SaO₂ 72%. The initial serum electrolytes and renal indices included: sodium 148 mEq/l, potassium 6.3 mEq/l, chloride 95 mEq/l, creatinine 135 μmol/l, BUN 8.2 mmol/l, and lactate 4.66 mmol/l. Pressure control ventilation was initiated using a Servo 900 C ventilator (Siemens-Elema, Stockholm, Sweden) at an inspiratory fraction of oxygen (FIO₂) of 1.0; an inspiratory/expiratory time ratio of 1/3 and a respiratory rate of 16 cycles/min. The respiratory status was characterized by major bronchospasm with intrinsic positive end-expiratory pressure (reading directly from the ventilation machine) at 20 cm H₂O and it was impossible to increase the minute ventilation to more than 5 l/min due to peak inspiratory pressure elevation (59 cm H₂O). The chest radiograph revealed no pneumothorax, no infiltrate and an elevation of the left diaphragm. The in-hospital treatment included continuation of neuromuscular blockade (pancuronium 4 mg/h), beta-agonist infusion (salbutamol 5 mg/h), epinephrine infusion (5 mg/h), bicarbonate administration (250 mEq) and mechanical ventilation. The administration of isoflurane (2%) was started 5 h after admission. The evolution of hypercapnia, PaO₂/FIO₂, pH and minute ventilation is represented in the Figure. The PaCO₂ persisted at over 190 mmHg for more than 10 h. The patient's acid-base balance and arterial gas exchange improved slowly and reached a pH of 7.45, PaCO₂ of 35 mmHg, PaO₂ of 89 mmHg at an FIO₂ of 0.3 at 72 h after admission. The initial plasma lactate (4.66 mmol/l) fell to 0.90 mmol/l after 7 hours. The patient was discharged from hospital on day 26 without sequelae.

Fig. 1 Evolution of arterial PaCO₂, pH, arterial partial pressure of oxygen/FIO₂ and minute ventilation during the first 24 hours of hospitalization