

Life-threatening asthma after heroin inhalation. A case report and a review of the literature

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Abstract. Heroin addiction may increase the risk of pulmonary involvement. We describe the case of a 23 year-old woman who was admitted to our unit for severe asthma attack non responsive to beta-2-agonists and acute respiratory failure, soon after heroin inhalation. The patient was successfully treated with non invasive positive pressure ventilation. Opiate inhalation can be an asthma trigger and should be considered in the care of patients with poorly controlled asthma and life-threatening asthmatic attacks. (www.actabiomedica.it)

Key words: Asthma, heroin, respiratory failure

Introduction

Heroin is a semi-synthetic opioid drug synthesized from morphine, a derivative of the opium poppy. Heroin addiction increases the risk of pulmonary involvement and it may cause a severe bronchospasm after inhaled/smoked use (1). A temporal relationship between inhalation of heroin and a status asthmaticus of remarkable severity has been described (1-8). Most of patients with heroin-induced status asthmaticus underwent intubation (1-8) and in some cases death occurred (3-4).

We report a case of a young woman with a life-threatening asthmatic attack after heroin inhalation, successfully treated with non invasive ventilation, and a review of the related literature.

Case report

A 23 year-old Caucasian woman was admitted to our unit for acute respiratory failure due to severe asthma attack, which did not respond to the repeated inhalations of beta-2-agonists. The severe bronchospasm followed the intake of heroin by inhalation, according to the "Chasing the dragon" technique, which implies that heroin is heated on foil and heroin vapour is inhaled. The patient reported that it was not the first time she used the "Chasing the dragon" technique to intake heroin.

The patient's clinical history was: poorly controlled atopic asthma (since the age of 9); virus induced thrombocytopenia at the age of 12; amenorrhea due to pituitary prolactin-secreting adenoma at the age of 20, heroine and cocaine abuse and tobacco smoking (2 pack/years) since the age of 16. At the moment of the hospitalization she was breathless and agitated with the following vital signs: temperature 38°C, blood pressure 110/70 mmHg, pulse rate 140 bpm. ABG analysis while breathing room air yielded the following: PO₂ 43.5 mmHg, PCO₂ 40.9 mmHg, pH 7.38. At the thoracic examination diffuse inspiratory and expiratory wheezes with prolonged expiratory phase were present. Chest radiograph demonstrated pul-

Abbreviations

ABG : Arterial Blood Gas

WBC: White Blood Cell

RBC: Red Blood Cell

LDH: Lactate Dehydrogenase.

BNP: B-type Natriuretic Peptide

CK-MB: Creatine Kinase-MB

NPPV = Non Invasive Positive Pressure Ventilation.

CPAP = Continuous Positive Airway Pressure.

PSV = Pressure Support Ventilation

FIO₂ = Inspired Oxygen Fraction

monary congestion and hyperinflation (Fig. 1). Blood chemistry showed leukocytosis with neutrophilia (WBC count was 17.23×10^3 with 72.4% neutrophils), erythrocytosis (RBC count was 5.83×10^6 /L with 17.5 g/dL haemoglobin level). Additionally, LDH, troponin, CK-MB and BNP levels were: 611 U/L, 0.43 ng/mL, 6.7 ng/mL and 225 pg/mL, respectively. ECG did not show any alteration. The urine toxicology screen was positive for opiates (1275 ng/mL) and methadone (900 ng/mL).

The patient was treated with systemic steroids (160 mg/day) and aerosol therapy with salbutamol 2.5 mg plus beclometasone 1.6 mg, 6 times per day and aerosol therapy with ipratropium 4 mg, 3 times per day. Oxygen therapy was delivered by means of a non-rebreather mask with an oxygen reservoir bag at a flow of 10 L/min, which ensured a 96% oxygen saturation. The patient received continuous pulseoximetry monitoring.

After 15 hours from the admission, her clinical conditions worsened and she showed acidosis and hypercapnia (ABG analysis showed: PO_2 74.4 mmHg, PCO_2 53.6 mmHg, pH 7.35): due to it, the patient immediately received NPPV. Initially, the mask was gently placed over the patient's face and held in position by a nurse for a few minutes until the patient was comfortable and in full synchrony with the ventilator (CARAT I, Hoffrichter GmbH-Germany), which generated a CPAP of 0 cmH₂O and a PVS of 10



Figure 1. Chest Radiograph

cmH₂O. The face mask was then secured by head straps, avoiding a tight fit. Afterwards, CPAP of 5 cmH₂O was applied and PSV was increased up to 15 cmH₂O in order to obtain an exhaled tidal volume of 400 mL corresponding to 7 mL/kg, a respiratory rate less than 25 bpm and patient's comfort. The FIO_2 was titrated to achieve an oxygen saturation greater than 90%. Contemporarily, the patient started with aminophylline, continuously i.v. administered (720 mg/day).

After 12 h of continuously administered NPPV, the patient had periods of rest (15 to 60 min) off the mask, while receiving supplemental oxygen, fluids or dietary liquid supplements. Nebulized bronchodilators were delivered in line with the ventilator. The patient was regularly examined for abdominal distention, for her ability to clear secretions or to protect the airways.

After 120 h from the NPPV beginning, the patient was weaned, showing an improvement in the respiratory failure, by titrating periods off NPPV to patient's tolerance and objective finding. At the moment of the weaning, ABG analysis while breathing FIO_2 0.28 yielded the following: pO_2 65.1 mmHg, pCO_2 45 mmHg, pH 7.44. Moreover, cardiac markers were within normal values (BNP 14 pg/mL, CK-MB 0.6 ng/mL, myoglobin 13 ng/mL and troponin I 0 ng/mL). The patient was also able to perform a spirometry, which showed a severe obstructive ventilatory defect (FEV_1 : 0.76 L, 23% of predicted value) (Fig. 2a). After 9 day from the hospitalization, ABG analysis while breathing FIO_2 0.28 yielded the following: pO_2 87.5 mmHg, pCO_2 47.1 mmHg, pH 7.39. The spirometry parameters were significantly increased (FEV_1 1.79, 56% of predicted value) (Fig. 2b). After 14 days from the hospitalization the ABG analysis was normalised (ABG while breathing room air yielded the following: pO_2 83.8 mmHg, pCO_2 44.8 mmHg, pH 7.47). The spirometry was normal as well (FEV_1 3.48, 108% of predicted value) (Fig. 2c) and the patient was discharged.

Discussion

This case further confirms that inhaling heroin may cause a life threatening asthma attack. Our patient presented acute respiratory failure and brocho-

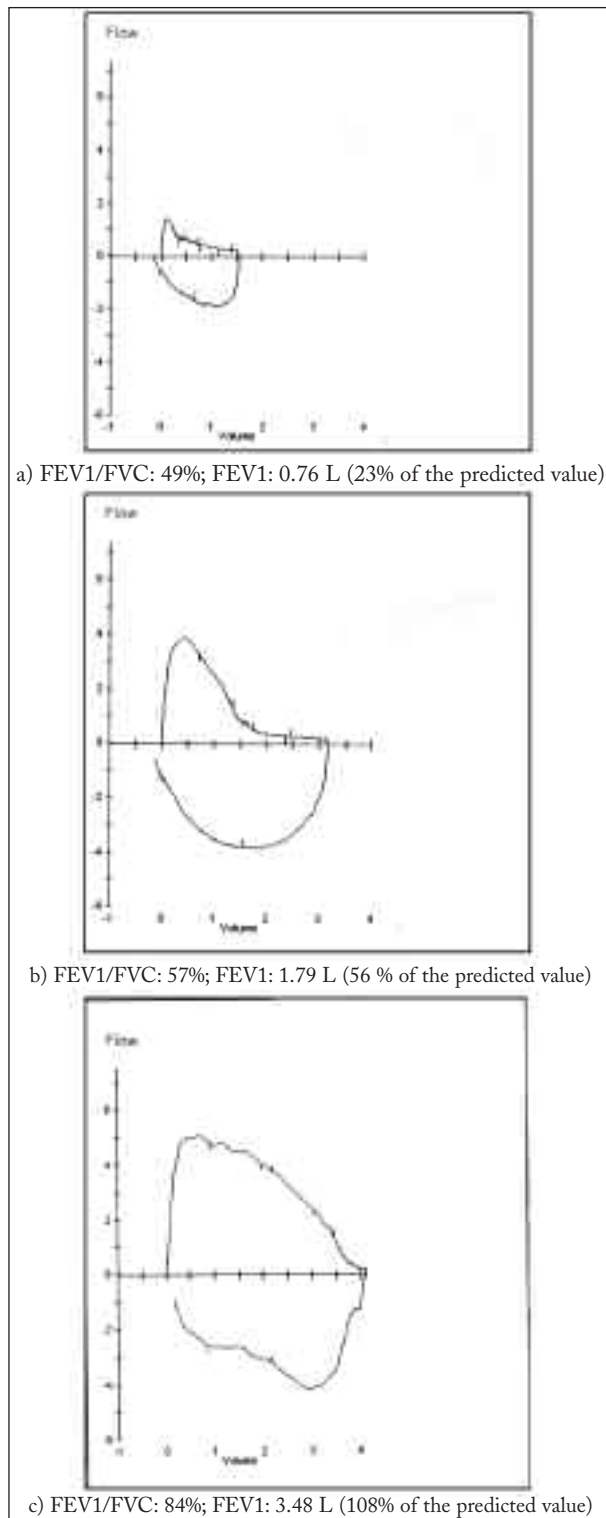


Figure 2. Flow/Volume curve at 5 (upper panel), 9 (middle panel) and 14 days (lower panel) of a patient with a life-threatening asthma attack after heroin inhalation

spasm non responsive to beta-2-agonist therapy after heroin inhalation. She also showed leucocytosis with neutrophilia, which was referred to a concomitant respiratory tract infection as well as a myocardial distress without ecg alterations. When she was able to perform a spirometry, that is after 5 days from the admission, an extremely severe obstructive ventilatory pattern was observed. The patient was successfully treated with non invasive mechanical ventilation combined to anti-asthma drug therapy.

In the last years, inhaled heroin use is increasing, because it is considered to be safer than the injecting use, although "Chasing the dragon" is not without risks. Fatal heroin overdoses (9) and lethal leucoencephalopathy (8, 10) resulting from non-injecting routes of administration have been reported. Chronic heroin smoking seems to be also related to an impaired lung function and higher prevalence of dyspnoea (11). Furthermore, heroin insufflation is a common asthma trigger (1) and up to now 13 cases of life-threatening asthma triggered by inhalation of heroin have been reported (1-8), including 8 pts. who were intubated (1-3) and 2 pts. who died (3-4).

It is unclear whether asthma is a prerequisite of heroin induced-asthma. Several patients showed asthma (2-5), but in other patients a history of asthma has not been reported (2, 6-8). Where detailed histories were available, no patients reported that heroin inhalation acted as a trigger from its first use; rather, it developed as a trigger following a period of several months of use (1). In a case series of 5 patients with status asthmaticus after heroin inhalation, a blood eosinophilia was found (5). This finding was not confirmed in our case and was not reported by another case series (3). Notably, in veteran heroin addicts a blood eosinophilia may occur and it is related to the intravenous drug use (12).

The mechanisms involved in heroin induced status asthmaticus are multifactorial. Chest tightness, wheeze, and rhinitis were reported in temporal relationship to morphine dust exposure in two pharmaceutical employees (13-14) and to heroin exposure in a drug dealer (6). A bronchoprovocation test, performed on the dealer, as well as a skin-prick test with extracts of pure heroine were positive (6). In one of the pharmaceutical workers, both inhalational and nasal

challenge with morphine resulted in a decline in the patient's pulmonary function, associated with dyspnea, wheeze, sneezing, and rhinorrhea. Nasal lavage after each challenge demonstrated an influx of basophils and eosinophils in an allergic pattern (6). These data suggest that uncontaminated morphine and heroin may produce bronchospasm, possibly through an allergic mechanism.

Pulmonary mast cell degranulation may be another possible mechanism for heroine induced bronchospasm. In fact, intradermal and intravenous morphine and synthetic opioid administration stimulate histamine release from mast cells through a direct pharmacologic mechanism, independent of an opiate receptor (15-16). Histamine release occurs in one fifth of patients receiving postoperative intravenous analgesia with morphine or heroin (17). Another possible mechanism is the inhibition of cholinesterase receptors, which has been demonstrated in animal species, but its clinical significance in humans is unknown (18). Moreover another possibility is that heroin powder, its contaminants, and/or cutting agents may serve as nonspecific airway irritants (1).

In contrast with the non receptor-mediated mechanisms above mentioned, potential receptor-mediated effects of opiate might be expected to reduce bronchospasm by reducing airway cholinergic tone. An opiate receptor has been localized in the rat airway (19), and a μ -opioid agonist inhibits cholinergic neurotransmission through a mechanism involving the opiate receptor in isolated human airways (20). Both central and peripheral cholinergic mechanisms appear to be involved in the expression of opiate withdrawal (21). Perhaps bronchospasm results from centrally mediated increased vagal tone during opiate withdrawal, and it manifests such as wheezing in patients with asthma. Alternatively, if adaptation to receptor-mediated effects occurs in the airways in opiate dependence, then withdrawal might result in a peripheral rebound in cholinergic tone, again resulting in bronchospasm in those with underlying asthma. While these mechanisms are speculative, the clinical observation underscores the importance of adequately treating opiate withdrawal in the setting of a severe asthma exacerbation.

In summary, a status asthmaticus with sudden onset of symptoms can be triggered by inhaled he-

roin. In patients with poorly controlled asthma and life-threatening asthmatic attacks, the heroin inhalation should be taken into consideration as a trigger factor.

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