Heroin inhalation and asthma

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Abstract

Opiate addiction is an increasing social problem, and there has been a change from taking opiates intravenously to inhaling them in many areas of Britain. Three patients with asthma who required mechanical ventilation soon after heroin inhalation were described. Two subsequently died of acute severe asthma. The patients were reluctant to admit to their addiction and persisted inhaling heroin despite medical advice and counselling.

Opiate inhalation can provoke life threatening asthmatic attacks and should be considered in patients at risk of abusing drugs who have poorly controlled asthma.

Introduction

The adverse effects of intravenous narcotic abuse are well recognised.[1] Inhalation of heroin vapour ("chasing the dragon") is now common and is considered by some to be fairly innocuous.[2] We report on three patients treated in our unit for severe asthmatic attacks shortly after inhaling heroin, which resulted in fatal respiratory arrest in two cases.

Case 1

A 19 year old woman was admitted after collapsing. She had suffered from asthma until the age of 7, and this had recurred six months after she started to inhale heroin. She had hay fever, smoked 10 cigarettes a day, and had inhaled heroin regularly for two and a half years. Although she was using a salbutamol inhaler and had been in hospital two months previously, her asthma was poorly controlled. Her companions reported that shortly after inhaling heroin she began to wheeze and within one hour had stopped breathing. On admission she had apnoea and asystole. After resuscitation she remained unconscious with fixed, dilated pupils and generalised convulsions. She had severe bronchospasm, which responded to treatment with nebulised salbutamol 10 mg every four hours and intravenous amniophylline 500 mg and hydrocortisone 200 mg every six hours. After ventilation for seven hours she was allowed to breathe spontaneously, and normal blood gas tensions were maintained with 40% oxygen. She died of hypoxic brain damage 61 hours after admission. Necropsy showed patchy inflammation, thickened bronchial walls, and mucus plugging throughout her lungs.

Case 2

A 23 year old heroin addict was admitted with severe asthma. He had atopic allergy on skin testing but had first developed asthma four years after starting to inhale heroin regularly, which he had done for six years; he had injected the drug only once. He smoked 20 cigarettes a day, took 60 ml of methadone mixture, and used a terbutaline inhaler. In the preceding year he had been admitted twice with asthma and had been discharged only two months previously. Each time he had defaulted from follow up, but on discharge his peak expiratory flow rate value had been normal.

Less than four hours after the start of the attack, which began shortly after he inhaled heroin, he had a respiratory arrest. He was intubated and ventilated; his inspiratory pressures were 50-55 cm H_2O (4·9-5·4 kPa), and they returned to normal over the next five days, permitting extubation. Ten days later he was discharged taking a reducing dose of oral corticosteroids and using steroid and terbutaline inhalers. His peak expiratory flow rate was 500 l/minute (predicted value 450 l/minute). He admitted that he commonly developed wheezing within minutes or hours after inhaling heroin. He was counselled, and follow up at a drug dependency clinic was arranged, but he defaulted from the medical follow up.

Three months later he collapsed at home. Despite having been wheezy the day before he had refused to seek medical help. On the way to hospital he developed ventricular fibrillation and was intubated and defibrillated twice by the ambulance crew. On arrival he had asystole with fixed, dilated pupils; severe bronchospasm made hand ventilation with an Ambu bag difficult, and despite attempted resuscitation he could not be revived.

Case 3

A 17 year old woman who had been admitted with asthma nine times over 18 months and had twice required ventilation was admitted with a further attack. She did not have atopic allergy but had suffered from asthma since the age of 3½. She smoked 20 cigarettes a day and had inhaled heroin for one and a half years. She was being treated with oral prednisolone 10 mg daily, an oral theophylline preparation, and salbutamol and ipratropium bromide inhalers. She strenuously denied inhaling heroin during the previous six months, but relatives and friends confirmed that she still smoked the drug. Within one and a half hours after the onset of wheezing her peak expiratory flow rate was unrecordable. She had marked acidosis (pH 7·29) and hypercapnia (carbon dioxide tension 7 kPa). She did not respond to bronchodilation and at three hours was exhausted, requiring ventilation. Her arterial carbon dioxide tension was 8 kPa and peak inspiration pressure 40 cm H_2O (3·9 kPa). Twenty four hours later she developed symptoms of narcotic withdrawal with hypertension, a tachycardia, sweating, and dilated pupils. She was weaned from the ventilator at 48 hours and was discharged once her peak expiratory flow rate returned to 400 l/minute. She defaulted from attending the outpatient department and the drug dependency centre and was subsequently admitted with a further attack of asthma after inhaling heroin.

Discussion

These cases show that inhaling heroin can provoke acute severe asthma. Two patients had respiratory arrest within a short time after the onset of wheezing, and the third required ventilation within three hours afterwards. Of 70 cases of asthmatic attacks in our unit in the past 18 months, in only nine was ventilation required. The three opiate addicts accounted for nine admissions and all required ventilation. They all had asthma that was poorly controlled and were frequently admitted to various hospitals, but they responded to standard treatment, achieving good values for peak expiratory flow rate before they were discharged. One patient persistently denied smoking heroin, but the evidence of witnesses and her withdrawal symptoms during treatment confirmed her drug abuse.

The respiratory effects of substance abuse have been reviewed.[1] The main problems are infection and

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pulmonary oedema after intravenous use. The effects on the airways are less, being confined to users of marihuana or hashish who have short term bronchodilatation,[3] although specific airways conductance may fall with long term use.[4] The only report of asthma after opiate inhalation described three less severe cases managed in a casualty department, in which all three patients recovered without ventilation.[5] Several studies have failed to show an increase in the incidence of asthma in opiate addicts, although in one study 28% of addicts with asthma had an attack within hours or days after using heroin.[6] Most of the patients were intravenous drug abusers, and the incidence of asthma in addicts inhaling heroin is unknown.

The mechanisms provoking bronchoconstriction are probably multifactorial. Opiates are powerful releasers of histamine, which can produce bronchoconstriction and local oedema, especially in subjects with atopic allergy or asthma.[7] Contamination with other drugs or talcs is probably less important when opiates are inhaled rather than injected. Inhaling freebase cocaine—that is, its alkaline derivative—can provoke reductions in the calibre of large and small airways greater than those from smoking tobacco alone.[8] Central respiratory depression may also be important in depressing the normal compensatory respiratory responses to an asthmatic attack and so hasten the onset of hypoxaemia and respiratory acidosis, thereby predisposing to early respiratory arrest. Early treatment with intravenous naloxone should help to reverse such central respiratory depression.

It is worrying that we failed to prevent recurrent and more severe attacks of asthma in our three patients despite recognition of their problem and appropriate counselling. The patients' reluctance to admit to their opiate addiction compounds the problem. The development of bronchospasm soon after inhaling heroin is a serious event and should be recognised as such.

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3 Tashkin DP, Shapiro BJ, Frank IM. Acute effects of marihuana on airways dynamics in spontaneous and experimentally-induced bronchial asthma. In: Craud MCB, Szara S, eds. *The pharmacology of marihuana*. New York: Raven Press, 1976:785-801.
4 Tashkin DP, Calvarese BM, Simmons MS, Shapiro BJ. Respiratory status of seventy-four habitual marihuana smokers. *Chest* 1980;**78**:699-706.
5 Oliver RM. Bronchospasm and heroin inhalation. *Lancet* 1986;i:915.
6 Ghodse AH, Myles JS. Asthma in opiate addicts. *J Psychosom Res* 1987;**31**:41-4.
7 Jaffe JH, Martin WR. In: Gilman AG, Goodman LS, Gilman H, eds. *The pharmacological basis of therapeutics*. New York: MacMillan, 1980.
8 Tashkin DP, Simmons MS, Coulson AH, Clark VA, Gong H. Respiratory effects of cocaine "freebasing" among habitual users of marihuana with and without tobacco. *Chest* 1987;**92**:638-44.
9 British Thoracic Association. Death from asthma in two regions of England. *Br Med J* 1982;**285**:1251-5.

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Diabetes in patients with hypertension receiving pharmacological treatment

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We looked at the records of all patients attending four health centres who had coexisting hypertension and diabetes to find which of these conditions had been diagnosed first.

Methods and results

About 75-90% of patients with type II diabetes in our area of western Sweden are treated at health centres. We looked at the records of all patients attending our four health centres who had coexisting diabetes and arterial hypertension. The blood pressure of all diabetic patients had been recorded at least once a year, and the urine of all hypertensive patients had been examined for glucose: fasting blood glucose concentrations had been measured at least once a year in all hypertensive patients attending the health centre of Trollhättan, in a systematic sample of hypertensive patients in Floda, and when clinically indicated at the health centres in Dalsjöfors and Svenljunga.

Results

The table shows that of the 226 patients with coexisting arterial hypertension and diabetes, 183 had had arterial hypertension before diabetes was diagnosed, while only 22 had had diabetes before hypertension was recognised; in all four health centres there was a significant overrepresentation of patients with hypertension developing diabetes compared with the opposite ($p<0.01$ for Floda, $p<0.001$ for the three other health centres and for the total series, χ^2 test). All the 183 hypertensive patients whose hypertension had preceded diabetes had been taking antihypertensive drugs at the time of onset of their diabetes; all but four had been taking a diuretic or a β blocker, or both. The mean time between starting pharmacological treatment for hypertension and developing diabetes (about eight years) was similar for patients of the four health centres.

Patients at four health centres in western Sweden with coexisting arterial hypertension and diabetes according to which condition developed first

	No who developed hypertension before diabetes	No who had hypertension and diabetes diagnosed at same time	No who developed diabetes before hypertension
Floda	23	4	2
Dalsjöfors	51	6	4
Svenljunga	55	7	10
Trollhättan	54	6	6
Total	183	23	22

Comment

As hypertension is more common than diabetes it might be expected that a diabetic person would develop arterial hypertension. We found, however, that it was more common for a hypertensive patient to develop diabetes than for a diabetic to develop hypertension, and all the hypertensive patients were taking antihypertensive drugs at the time of onset of diabetes. This supports our previous finding that women who were taking antihypertensive drugs (diuretics or β blockers, or both) had an increased risk of developing diabetes.[1,2] Too few women in either of our studies were taking other antihypertensive drugs to allow us to drawn conclusions about the other drugs.

This is further evidence for a connection between antihypertensive drugs and risk of developing diabetes and is one more reason to consider non-pharmacological treatment for hypertension initially.

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