Cardiotoxic Effects of Raw Opium

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Abstract

While opioid drug toxicity and side effects of long-term opioid use during medical care are well studied, there is little information regarding effects of ingestion of raw opium. Characterization of the effects to a particular alkaloid is difficult since raw opium contains a number of alkaloids. Here, we present a case of poisoning due to ingestion of raw opium leading to severe myocardial suppression.

Keywords: Left ventricular dysfunction, myocardial suppression, raw opium

INTRODUCTION

Raw opium is widely cultivated in certain parts of the world and can be drunk, swallowed or smoked. In overdose, its alkaloids and their derivatives, called opiates, are well known to cause respiratory and central nervous system depression, which may lead to death. We present a case of raw opium poisoning where the patient suffered from severe myocardial suppression but successfully recovered with appropriate treatment.

CASE REPORT

A 13-year-old female with no significant medical history was brought to the emergency room (ER) with the complaints of loss of consciousness and unresponsiveness since 8-10 h. The patient was all right when she went to bed after dinner but her parents found her unresponsive and lying oddly on the bed when they woke up during the night. They also found a puddle of vomitus next to her on the floor. The patient was initially taken to a local clinic where the clinician diagnosed it as a case of organophosphorus poisoning. She was given atropine, pralidoxime and other supportive treatment. However, due to the patient's deteriorating condition and her hemodynamic instability, she was referred to a higher center. The travel time to our center from the clinic was about 3-4 h. On arrival in the ER, the patient was unarousable, had dilated pupils, flaccid limbs, bilateral plantar response as extensor, shallow breathing, elevated heart rate of 150/min, and severe hypotension (blood pressure = 60/40 mmHg). The patient was intubated and put on ventilatory and vasopressor support. Her electrocardiogram revealed sinus tachycardia. Her

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two-dimensional echocardiography (ECHO) revealed global left ventricular (LV) hypokinesia with a LV ejection fraction (LVEF) of 10%. Cardiac enzymes were found to be elevated (troponin I = 0.322 ng/ml, normal = <0.014 ng/ml; creatine phosphokinase-MB = 34.71 ng/ml, normal = 1.39-6.22 ng/ml). Supportive treatment was started, which included empiric antibiotics, steroids, digoxin, diuretics, antiepileptics, low-molecular-weight heparin, and correction of electrolyte abnormalities. Fluids were titrated keeping her poor LV function in mind. The patient was able to maintain her oxygen saturation and blood pressure on support and had adequate urine output. A definite diagnosis could not be made. After 24 h, the patient showed signs of awakening. She started stabilizing over the next few days, and her LV function improved steadily [Table 1]. She was extubated on the 4th day. Following recovery, she admitted consumption of raw opium (about a pebble-sized dose) after her dinner that night. She was shifted to ward on the 5th day and discharged with stable vitals on the 8th day. On follow-up after 7 days, the patient was found to have improved LV function with her LVEF being 56% with normal cardiac parameters.

DISCUSSION

Opium gum is a sticky dark brown substance with a strong odor that is obtained from the unripe seedpods of the

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Table 1: Serial echocardiography findings									
Day	LVID (ED)	LVID (ES)	LVEF (%)	Interventricular septal thickness	Posterior wall thickness	Remarks	Other findings		
Admission (day 1)	53 mm	42 mm	10	6 mm	4 mm	Severe LV systolic dysfunction with LV dilatation	Dilated LV; Diastolic dysfunction (Grade III); Fair RV contractility; Trivial MR/TR		
Day 3	-	-	15	-	-	Only screening done	Same as above		
Day 6	36 mm	22 mm	35-40	11 mm	7 mm	Moderate LV systolic dysfunction with recovering LV dimensions	LV Normal sized; Diastolic dysfunction (Grade I); Good RV contractility; Trivial MR/TR		
Day 8	34 mm	20 mm	45	10 mm	7 mm	Mild LV systolic dysfunction with normal LV dimensions and thickness	LV normal sized; Diastolic dysfunction (Grade I); Good RV contractility; Trivial MR/TR		

LV: Left ventricular; RV: Right ventricular; LVID: Left ventricular internal diameter; ED: End-diastolic; ES: End-systolic; LVEF: Left ventricular ejection fraction; MR: Mitral regurgitation; TR: Tricuspid regurgitation

opium poppy (Papaver somniferum), a plant of the family Papaveraceae. It is widely cultivated in our region (the state of Rajasthan and the nearby state of Madhya Pradesh).^[1] Raw opium can be drunk, swallowed or smoked. It has a bitter taste and eating it is not enjoyable. Despite this, it has been taken orally in many countries of the world including India.^[2] The pharmacologically active principles of opium reside in its alkaloids, the most important of which is morphine. Opiates exert their main effects on the brain and spinal cord. They alleviate anxiety, induce relaxation and sedation. They also impart a state of euphoria and enhanced mood. Hence, they are addictive drugs. Their principal therapeutic use is as analgesics.^[3] Opium toxicity should be suspected when the clinical triad of central nervous system depression, respiratory depression, and miosis is present. Naloxone as antidote is indicated for significant central nervous system and/or respiratory depression.^[4]

A few case studies have suggested that there is a relationship between opium addiction and coronary artery disease (CAD) and myocardial infarction. A case–control study by Masoomi *et al.* suggested that opium consumption is an independent risk factor for the development of CAD.^[5] Another case–control study by Masoumi *et al.* concluded that opium users have a higher risk of severe CAD as compared to nonusers.^[6] One case–control study by Khosoosi Niaki *et al.* proposed that opium addiction is a strong risk factor for the development of myocardial infarction.^[7] However, acute myocardial suppression following raw opium ingestion has not been well documented.

In our case, the patient had a social background of opium cultivation as her father was an opium cultivator. Clinical signs and symptoms of opium toxicity were confounded by pretreatment with pralidoxime and atropine. However, the previous physician's findings of a comatose state with the inability to arouse the patient on deep pain stimuli, difficulty in breathing, and pinpoint pupils were supportive of opium poisoning. The profound LV systolic dysfunction with dilated cardiac chambers and tachycardia created a diagnostic dilemma. These findings can represent acute myocarditis as well as acute onset dilated cardiomyopathy. Both of these can present as cardiogenic shock.^[8] The ECHO findings can be similar, namely, both may have marked ventricular dilatation with LV systolic dysfunction.^[9,10] In our case, the absence of prior symptomatic history and the ECHO findings favor a diagnosis of acute onset myocardial suppression induced by opium.

Apart from opium toxicity, in view of the clinical background, other poisonings such as celphos (aluminum phosphide) as well as scorpion bite were also being considered in the differential diagnosis.^[11,12] Naloxone was not given in view of risk of precipitation of pulmonary edema with a background of poor LV function.^[13] Further, since opioid poisoning and overdose cause death primarily by respiratory depression, naloxone is not considered necessary if airway and breathing assistance are already in place.^[4] After the patient recovered, we were able to obtain a more accurate history, and the patient admitted to ingesting raw opium that same night.

Limitations

We were unable to send serum toxicity profile including opioid levels hence a correlation between opioid levels and severity of myocardial suppression could not be commented. Moreover, such cases with a reversible toxicity can be good candidates for venoarterial extracorporeal membrane oxygenation at centers where it is available.^[14]

CONCLUSION

Oral ingestion of a large amount of raw opium can lead to severe myocardial suppression which resolves with adequate supportive treatment.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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