



## CASE REPORT

# Successful Use of Naloxone to Reverse Opioid-Induced Atrial Fibrillation

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## Introduction

A 25-year-old male presented to the Emergency Department in atrial fibrillation with a rapid ventricular response in the setting of acute heroin intoxication. A single IV dose of naloxone 0.4 mg immediately converted the patient to a sinus rhythm after three doses of metoprolol failed to slow the patient's heart rate. Given the safety profile of naloxone, this therapy can be considered in carefully selected cases of acute heroin intoxication complicated by acute atrial fibrillation with rapid ventricular response.

## Case Report

A 25-year-old male presented to the Emergency Department after being found lying in bed by paramedics. His father and friends reported that he had a history of alcoholism and likely used heroin that night. He was initially assessed by medics to be minimally responsive with pinpoint pupils and near-apnea. Two milligrams of intranasal naloxone as well as supplemental oxygen by nasal cannula were administered and the patient immediately became more alert with a prehospital HR recorded in the 150's bpm. He arrived in the Emergency Department awake and alert, complaining of palpitations. A chart review of his prior visits revealed a history of severe alcoholism, alcohol-induced seizures, thrombocytopenia, a prior ICU admission for alcohol withdrawal, and a history of methamphetamine-induced tremor (for which he was placed on metoprolol). The patient and his father denied history of any cardiac disease.

On arrival, vital signs were BP 140/86 mm Hg, HR 160 bpm irregular, RR 16/min, T 98.7 °F, SpO<sub>2</sub> 98% on room air. The patient was awake and alert and had an unremarkable physical exam except for an irregularly irregular tachycardia on heart exam. Specifically, there were no signs of exophthalmos or thyromegaly on the head and neck exam. The lungs were clear to auscultation without rales, the abdomen was non-tender, and there were no signs of cyanosis, clubbing, or edema in the extremities.

An ECG revealed atrial fibrillation with a rapid ventricular response. The patient was initially treated with metoprolol 5 mg IV every 10 minutes for 3 total doses without effect on HR or rhythm. The patient was then given naloxone 0.4 mg IV and almost immediately converted to a normal sinus rhythm with a HR ~100 bpm. An echocardiogram was obtained which showed normal chamber size, left ventricular function, and valvular function. A complete blood count, serum chemistries, and thyroid function were all normal with the exception of a platelet count of 59,000 cells/mcL.

## Discussion

The most common ECG abnormalities in acute heroin overdose are non-specific ST-T changes, sinus tachycardia, and left or right atrial enlargement [1]. Atrial fibrillation is a reported but uncommon dysrhythmia associated with acute heroin intoxication [2-5]. Although the precise mechanisms that result in this rhythm are not known, it is thought that the vagolytic and vagomimetic actions of heroin (or of its morphine

metabolites), combined with the effects of respiratory depression (i.e. hypoxemia, hypercarbia, acidosis) can result in the formation of tachydysrhythmias [2,3,6]. Furthermore, an association between prescription opioid use and atrial fibrillation has been suggested with the release of data from the REGARDS study [7]. In this study, the prevalence of atrial fibrillation was found to be higher in prescription opioid users than nonusers (12.5% vs. 7.6%,  $p < 0.001$ ). After adjustments for potential confounders, including adjustment for benzodiazepine and alcohol use, the odds ratio for prescription opioid use and atrial fibrillation remained statistically significant (OR 1.29 [95% CI, 1.16-1.51]).

Naloxone was administered in an attempt to convert atrial fibrillation after three doses of metoprolol had failed to slow the rhythm. Mixed opioid agonist-antagonists, such as naloxone or nalorphine, rapidly inhibit the vagotonic effects of opiates and have been used to convert atrial fibrillation to sinus rhythm in patients with acute heroin intoxication.

Labi first published the response of atrial fibrillation to nalorphine in a retrospective case series of six patients with heroin intoxication [2]. In this study, the exact time of conversion from atrial fibrillation to sinus rhythm was unable to be extracted as patients were not continuously monitored; however, the pharmacokinetics of nalorphine suggest that this change likely occurred soon after administration of this medication. Similarly, in a study by Duberstein, the use of nalorphine was associated with conversion from atrial fibrillation to sinus rhythm in six patients with acute heroin intoxication [3]. Although the authors of the second study felt that the relief of hypoxia led to conversion of arrhythmias, Glauser, et al. suggests an alternative explanation is that the antagonistic effect of nalorphine terminated these arrhythmias [1]. In our patient, supplemental oxygen was continuously

administered in the prehospital setting as well as during the initial Emergency Department stay yet failed to convert the patient to a sinus rhythm.

In summary, a 25-year-old male with acute heroin intoxication presented with atrial fibrillation with rapid ventricular response. A single IV dose of naloxone 0.4 mg immediately converted the patient to a sinus rhythm after three doses of metoprolol failed to slow the patient's heart rate. This therapy requires further animal and human testing before it can be routinely recommended, however the safety profile of naloxone suggests that it may be safely considered in selected cases of atrial fibrillation with rapid ventricular response in the setting of acute heroin intoxication.

## References

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