

Case Report

Can Heroin Poisoning Lead to Stomach Necrosis? A Case Report



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ABSTRACT

Background: According to the last report of the United Nations Office on Drugs and Crime (UNODC), opiate use (including heroin) is 1.2% of the world population. In Iran, about 2 million people are drug addicts. Heroin reduces gastric motility and prolongs gastric emptying time and causes gastric dilation which can be a reason for gastric ischemia. Gastric ischemia is an uncommon condition due to the rich gastric blood perfusion and collateral arteries. As some studies show, gastric dilation can be the cause of gastric necrosis.

Methods: A 22-year-old woman presented to our hospital with severe abdominal pain and several episodes of vomiting. The patient declared that she had a history of addiction to methamphetamine and heroin. Abdominal examination revealed a soft and non-distended abdomen with generalized tenderness, mostly in the hypogastric region without rebound tenderness. Abdominal radiograph revealed that the stomach was highly distended. Abdominal CT without contrast confirmed severe gastric dilatation. In endoscopy, multiple necrotic lesions were seen throughout the stomach and mostly in the proximal part. Our finding in the laparotomy was gastric necrosis in the proximal part, which resulted in a total gastrectomy. Esophagojejunostomy was performed with roux en y reconstruction after total gastrectomy.

Conclusion: Opioids can increase the risk of gastrointestinal (GI) dysfunction and can increase the risk of infection in the GI tract. In our case, heroin abuse caused gastric dilation and massive gastric necrosis.

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1. Introduction

Heroin is an addictive semisynthetic opioid and is a white powder with a bitter taste but it is often mixed with other substances [1]. Mortality rates of heroin overdose are higher in users who use other substances and alcohol and occur mostly in long-term users [2]. In Iran, about 2 million people use drugs daily, which is about 2.7% of the population [3]. Studies show that the lifetime rate of opiate use, mostly opium, was between 1.2 and 8.6% in different parts of the country [4]. Heroin can be injected intravenously or subcutaneously, ingested, snorted, or smoked and like other narcotics, reduces the brain's responsiveness to hypoxia as a result of respiratory depression. Heroin can also affect baroreceptor and causes bradycardia. It stimulates histamine release as well as reduces peripheral vascular resistance and causes mild vasodilation of the cutaneous blood vessels [5, 6]. It also reduces gastric motility and prolongs gastric emptying time and can cause gastric dilation which can be a reason for gastric ischemia [7]. Gastric ischemia is an uncommon condition due to the rich gastric blood perfusion and collateral arteries. Several types of gastric ischemia exist, such as gastric infarction, gastric necrosis, moribund stomach, stress ulceration, and chronic ischemic gastritis. As some studies show, gastric dilation can be the cause of gastric necrosis [8, 9]. In this article, we introduce a 22 years old addicted woman with gastric necrosis due to heroin overdose.

2. Case Presentation

A 22-year-old woman was admitted to our emergency department due to severe abdominal pain with several episodes of vomiting. She reported no medical or pharmacotherapy history but had a history of addiction to methamphetamine and heroin in her social history. The patient declared that she had injected large amounts of heroin intravenously in the hours before the onset of her symptoms.

The patient was oriented to time, place, and person and her physical examination showed a Glasgow Coma scale score of 15/15. In her vital signs, blood pressure decreased to 80/60 mmHg, tachycardic pulse rate of 120 beats per minute, respiratory rate of 22 breaths per minute, and oxygen saturation of 95% in room air were assessed. In the abdominal examination, mild distension and mild generalized tenderness were observed.

Fluid resuscitation and serum therapy were started for the patient. Moreover, a nasogastric tube (NG tube) was inserted for the patient for gastric decompression and some gastric secretion was removed. The patient was then admitted to the Intensive Care Unit (ICU) and the patient's urine output was properly evaluated by inserting a Foley catheter.

Table 1 presents laboratory findings. In her laboratory findings coagulation tests, liver function tests, amylase, and lipase were in the normal range. Venous blood gas analysis indicated metabolic acidosis and the urine toxicology test was positive for morphine. Her abdominal radiograph on the day of hospitalization revealed a much-distended stomach (Figure 1). Abdominal CT without contrast confirmed severe gastric dilatation (Figure 2). In the laboratory findings, the day after the patient was admitted to the hospital, creatinine was back to the normal range. Moreover, on physical examination, the patient's general condition improved significantly but her abdominal tenderness persisted. Therefore, an endoscopy was performed on the patient. In endoscopy, multiple necrotic lesions were seen throughout the stomach and mostly in the proximal part (Figure 3). Due to the patient's endoscopic results, her persisted tenderness and increased drainage of the NG tube up to 2400 cc of biliary secretion made the patient a laparotomy candidate. During surgery, the patient's abdomen was opened with a midline incision. Our finding in the surgery was gastric necrosis in the proximal part, which resulted in a total gastrectomy. Esophagojejunostomy was performed with roux en y reconstruction after total gastrectomy.

Blood Index	Case	Blood Index	Case
WBC	15000	pH	7.21
PLT	270000	HCO ₃	15
Hb	14	Base excess	-8
BUN	60	CR	3.5

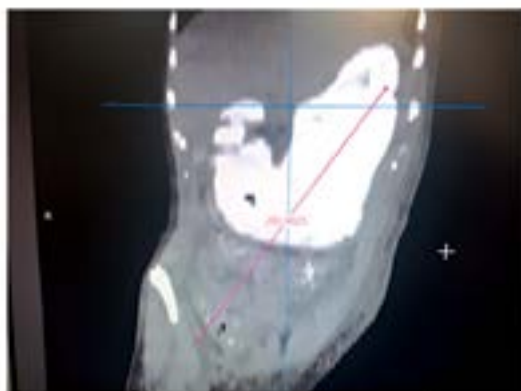
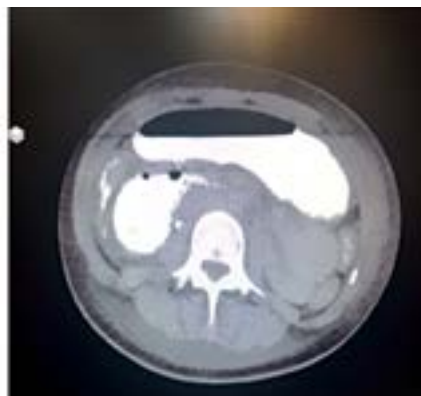


Figure 1. Dilated stomach in CT scan



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The patient was transferred to the ICU after surgery. On the second day of the post-operation date, she tolerated liquids and then a soft diet. Therefore, the patient was discharged on the seventh day after the operation with good general condition. She was doing well during follow-up visits in the outpatient department two weeks and two months after her discharge.

3. Discussion

Diacetylmorphine, known as heroin, is derived from the opium poppy plant. It is two or four times as potent as morphine and is synthetically derived from the morphine alkaloid in opium [10].

Heroin overdose is a critical cause of death among heroin users. It causes more than 100 000 deaths every year all

around the world [11]. Heroin overdose mostly occurs at home with others and other drugs [1]. Heroin can be used subcutaneously, intranasally, intramuscularly, and intravenously. During oral abuse of heroin, it undergoes first-pass metabolism to morphine via deacetylation. Unlike intravenous administration, oral ingestion does not cause a rapid onset of effects and is less desirable to users. 6-monoacetylmorphine and morphine activate opioid receptors [12]. It reaches its peak serum level in less than one minute when it is used intravenously, three to five minutes, intranasally and intramuscularly and five to ten minutes subcutaneously [13]. Heroin is a highly lipophilic substance and rapidly crosses the blood-brain barrier. Heroin is the agonist of opioid receptors, such as μ , κ , and δ [14]. It can affect μ receptors, μ_1 and μ_2 . Its analgesic effect is based on activating μ_1 and the respiratory depression and



Figure 2. Operative findings revealed gastric necrosis

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Figure 3. Stomach after total gastrectomy

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euphoria are based on μ_2 . Activation of μ_2 receptors also causes miosis, reduced gastrointestinal (GI) motility, and physiologic dependence. It causes some degree of analgesia by activating the Kappa receptor. Delta receptors are more involved in spinal analgesia [15, 16]. It also reduces peripheral vascular resistance (resulting in mild hypotension), and causes mild vasodilation of the cutaneous blood vessels (resulting in flushing). In case of very severe overdose, visceral blood is reduced due to severe hypotension. On the other hand, the mechanism of reduction in motility of the digestive system, it causes very severe distension of the stomach. In the case of severe distension of the stomach and simultaneous drop in blood pressure, mucosal blood supply is disturbed and can cause necrosis. As we mentioned, heroin reduces GI motility, so it can cause constipation [17, 18].

Opioids can increase the risk of GI dysfunction and can increase the risk of infection in the GI tract [7]. In our case, heroin abuse caused gastric dilation and massive gastric necrosis in a 22-year-old addicted woman. As it has been proved, gastric dilation is an event that can lead to gastric necrosis but no proven pathogenesis exists. It has several other etiologies, such as anorexia nervosa, trauma, diabetes, and electrolyte disturbances, which were all negative in our case [8, 9].

4. Conclusion

One of the severe complications of heroin poisoning can be gastric necrosis with a possible mechanism of hypotension and severe distension that leads to mucosal blood supply disturbance.

Ethical Considerations

Compliance with ethical guidelines

This article has no intervention on humans or animals by any of the authors. In addition, information about the human case study group is anonymous in this study.

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Authors' contributions

All authors contributed to the preparation of this article

Conflict of interest

The authors declared no conflicts of interest.

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