

Case Report

A Fatal Heroin Addict with Myocardial Lesion

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This is a histological report of a myocardial lesion of a 44-year-old white man who was found dead in a hotel with circumstances strongly suggestive of heroin intoxication. Based on autopsy findings and toxicologic analysis, the present case was an instance of straightforward heroin overdose in snorter. The most striking pathologic finding of the heart was a few patches of marked dark mottling appearance in the left ventricle and ventricular septum. Histological appearance of the lesions revealed marked congestion with intramyocardial extravasation of blood. Since the deceased had patent coronary arteries without evidence of atheroma, the lesions were thought to be the results of coronary artery spasm. There has also been substantial evidence in the previous reports to believe that the condition is secondary to heroin-induced coronary artery spasm. However, its actual underlying mechanism remains unclear.

Keywords: Heroin intoxication, Myocardial lesion, Sudden death

J Med Assoc Thai 2009; 92 (2): 279-83

Full text. e-Journal: <http://www.mat.or.th/journal>

Nasal inhalation (sniffing or snorting) is a common route for self-administration of many toxic substances, solid (e.g. cocaine, heroin) as well as volatile (e.g. butane, toluene). Even though some of the substances administered by intranasal route have been reported to induce acute myocardial infarction, cocaine-induced acute myocardial infarction is most common and well described in published reports⁽¹⁾, whereas heroin-induced myocardial lesion is rare.

The authors report a case of sudden death caused by intranasal heroin overdose with myocardial lesion.

Case Report

A 44-year-old Caucasian man was found dead in a supine position with his right face and right trunk on the bed in the room of the hotel where he stayed alone. There was light brownish fluid flowing from his mouth. Marked peripheral cyanosis was exhibited. There was some white powder scattered on his right arm and right side of his body. No paraphernalia was found. There were two packets containing some white powder on the table near the bed. His passport identi-

fied that he was American and his place of birth was New York. He came to Thailand a few days previously and just moved from another hotel. According to his family, the deceased had not been known to use drugs nor had a previous medical history.

At autopsy, the deceased revealed a muscular white man of middle age, who was 1.76 m tall and weighed 75 kg. External findings included some minimal light brownish fluid on the right angle of his mouth, marked peripheral cyanosis, and pupil sized about 5 mm in diameter both eyes. No recent or remote needle puncture marks were found in any sites. Some white powder was found on the lateral side of his right arm, right elbow, right ante-cubital fossa, and right dorsum of hand. Two recent minimal abrasion wounds were found on the left nostril internally and externally. No signs of trauma or injury were detected in other sites.

The most significant gross internal observation was abundant hemorrhagic pulmonary edema with a combined lung weight of 1,680 g. Microscopic examination of representative lung sections revealed marked congestion and edema with some areas of hemorrhage. No pulmonary disease or pneumonia was found. Copious blood tinged froth was found along the way of his lower respiratory tracts. The heart weighed 360 g. Its cross sections demonstrated no appearance of old myocardial infarction, but a few

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patches of marked dark mottling in some areas of the left ventricle and ventricular septum. Histologic examination of the heart revealed marked congestion with a few patches of intramyocardial extravasation of blood (Fig. 1). The coronary arteries and the aorta showed no atherosclerotic changes. The liver weighed 1,660 g and presented moderate congestion. Microscopic examination of the liver showed no portal lymphocytic infiltration or granulomas. Lymph nodes at the porta hepatis and adjacent to the common bile duct were normal in size. The remainder of the gross and microscopic anatomic studies was unremarkable.

Toxicology panel was positive for the presence of morphine in the deceased's blood, hair, bile and urine; but not for cocaine. The morphine

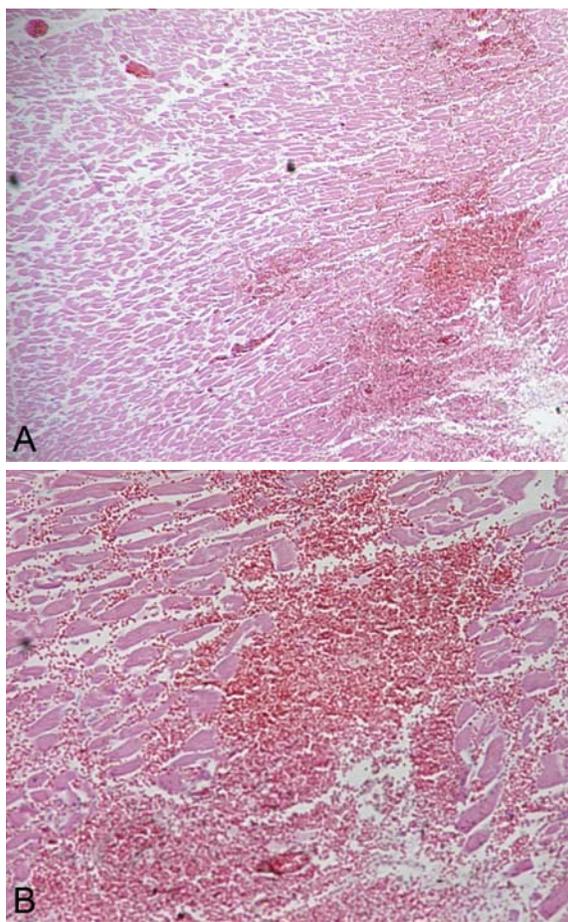


Fig. 1 Histological sections of myocardial lesions showing marked congestion with a few patches of intramyocardial extravasation of blood (hematoxylin and eosin stain): (A) original magnification X10 and (B) original magnification x100

blood concentration was 0.64 mg/ml. His blood and urine contained no ethanol, antidepressants, and benzodiazepines. Analysis of the white powder residue in both packets found at the scene, one weighed 0.02 g and the other 0.01 g, was positive for heroin hydrochloride. Heroin was also identified in swabs of the deceased's nasal mucosa.

The cause of death was attributed to acute accidental narcotism.

Discussion

Heroin (diacetylmorphine) was first synthesized by the Bayer Company in 1889 as a less addicting analgesic drug from morphine⁽²⁾. Since then its use has been abused for getting euphoric and content feeling. Once in the body, heroin is quickly metabolized to 6-monoacetylmorphine, which is then metabolized to morphine, with their half-lives 10 min and 20 min respectively. After crossing the blood-brain barrier, heroin is converted to morphine and binds rapidly to opioid receptors, thus feeling a surge of pleasurable sensation, a rush^(3,4).

Heroin is usually injected, sniffed/snorted, or smoked. Intravenous injection provides the greatest intensity and most rapid onset of euphoria (7-8 sec), while intramuscular injection produces a relatively slow onset of euphoria (5-8 min)^(3,4). When sniffed, its peak effects are usually felt within 10-15 min and smoked almost instantly⁽⁵⁾. However, these non-injecting routes are not as efficient and most of the drug is wasted. Until recently the street purity of heroin has increased over the years (up to 80% pure), although most street heroin is cut with other drugs or with substances such as sugar, starch, powdered milk, or quinine (only 25-50% pure)⁽⁶⁾. Because of this, snorting heroin is now an effective route of administration.

Since adverse effects of health such as acquired immunodeficiency syndrome (AIDS) and hepatitis has dramatically increased in heroin injectors, together with intensive public awareness, there have been a substantial increase in transitions in route of heroin use in the United States^(7,8). Other reasons for changing the route of administration include drug dependence, overdosing, dying suddenly, concerns about social stigmatization and self-image, social acceptance, preference for intranasal drug use^(9,10), and the lack of requirements for additional drug paraphernalia⁽⁵⁾. The spread of intranasal heroin use has therefore been found in both experienced drug users and naive drug users, especially in some cities, such as New York City^(9,10).

When heroin that is not heavily cut becomes available to users that get used to a heavily cut product, an overdose is possible unless the dose is adjusted. Therefore, the notion that heroin use via snorting is safe has recently been contradicted by various studies^(11,12). Furthermore, there was no significant difference between the blood morphine concentrations of non-injectors and injectors, and the cause of the deaths was respiratory depression^(11,12).

Regarding the cardiac complications associated with heroin poisoning, there have been some reports of arrhythmia⁽¹³⁾, acute cardiomyopathy⁽¹⁴⁾, haemoglobinemia⁽¹⁵⁾, and acute myocardial infarction in living heroin users, either with pre-existing coronary artery disease⁽¹⁶⁾ or with patent coronary artery⁽¹⁷⁾. Some studies have claimed that heroin might have a systemic effect⁽¹⁸⁾ or direct toxicity to the heart⁽¹⁹⁾. Different reasons have been proposed, including rhabdomyolysis with cardiac involvement, hypoxia, acidosis, vasoconstrictive agents released by muscle necrosis, or heroin-related hypersensitivity reactions⁽²⁰⁾. Heroin itself might also have a direct effect on the coronary arteries and induce acute coronary occlusion, either by provoking a local coronary spasm or inflammation⁽¹⁶⁾. Some studies have suggested that heroin acted directly on the vasomotor center to increase parasympathetic activity, reduce sympathetic tone, and stimulate the release of histamine from mast cells, thus resulting in bradycardia and hypotension both of which might have contributed to acute myocardial infarction^(21,22). Heroin-induced cerebral vasospasm after snorting purified heroin has also been reported^(23,24). Additionally, enhanced parasympathetic activity might also play a role in the initiation of coronary spasm⁽²⁵⁾. Therefore, there has been substantial evidence to believe that the acute myocardial infarction is secondary to heroin-induced coronary artery spasm. However, the actual mechanism underlying such a condition remains unclear.

The present case is a straightforward instance of fatal heroin overdose with acute myocardial lesion in snorter. Such a diagnosis was made by the results of toxicologic analysis as his blood morphine concentration was within the lethal range (> 0.5 mg/ml) and the autopsy finding, severe pulmonary edema, was consistent with the solely typical finding of death from heroin overdose. Since hair testing can demonstrate multiple deposits of the drug along the length of the hair shaft, the presence of the drug in hair will demonstrate its chronic use. Detection of morphine in the deceased's hair explained that he would have been a

regular drug user for some time, not a first episode of drug use. Positive heroin analysis in samples collected from nasal areas supported its acute poisoning as well as the route of administration. In addition, the analysis of white powder residue in both packets found at the scene was positive for heroin hydrochloride. Since the deceased had normal coronary arteries without evidence of atheroma, suggesting that coronary spasm is most likely a contributing factor of acute myocardial lesion.

Conclusion

Heroin-induced myocardial lesion is rare. There are few reports describing heroin induced such a lesion in the literature as discussed earlier. To two cases previously published, the author now adds another one. Based on the autopsy findings and toxicologic analysis, the case is a straightforward instance of heroin overdose in a snorter. Unlike the previous reports, both of which occurred in living individuals after heroin injections, the present lesion was found in a dead person following heroin snorting. To the author's knowledge, the present lesion is probably the first case reported in a heroin snorter. Although there has been substantial evidence, including the present case, to believe that heroin-induced acute myocardial lesion is secondary to coronary artery spasm, its underlying mechanism of increasing the vasospastic susceptibility of the coronaries is still not clear.

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การเสียชีวิตของผู้เสพเฮโรอีนร่วมกับพยาธิสภาพของหัวใจ

สุภาวรรณ เศรษฐบรรจง

ผู้พิมพ์รายงานพยาธิสภาพของหัวใจในผู้เสียชีวิตที่เป็นชายผิวขาวอายุ 44 ปี พร้อมด้วยสมภาวการณ์ที่ชี้แนะว่าจะเป็นผลจากพิษของเฮโรอีน จากผลการผ่าศพตรวจและผลการตรวจวิเคราะห์ทางพิษวิทยา ปรากฏว่ารายนี้เป็นตัวอย่างการเสียชีวิตที่เป็นผลโดยตรงจากการได้รับเฮโรอีนเข้าสู่ร่างกายทางการสูดเข้าทางจมูกเกินขนาด โดยพบลักษณะสีแดงคล้ำหลายหย่อมบริเวณผนังหัวใจห้องล่างซ้ายและผนังกล้ามเนื้อระหว่างห้องล่างของหัวใจซึ่งเป็นพยาธิสภาพที่เด่นมากของหัวใจ จากการตรวจเนื้อเยื่อบริเวณดังกล่าวโดยกล้องจุลทรรศน์ พบว่ามีลักษณะเลือดคั่งอย่างมากร่วมกับมีเลือดออกนอกหลอดเลือดเข้าไปแทรกอยู่ในกล้ามเนื้อหัวใจ และเนื่องจากหลอดเลือดแดงเลี้ยงหัวใจของผู้เสียชีวิตมีลักษณะปกติจึงเชื่อว่าพยาธิสภาพดังกล่าวเป็นผลจากการหดเกร็งของหลอดเลือดแดงเลี้ยงหัวใจ ทั้งนี้ยังมีหลักฐานอย่างเพียงพอในหลายรายงานที่เชื่อว่าพยาธิสภาพที่เกิดขึ้นใน หัวใจเป็นผลทุติยภูมิจากการหดเกร็งของหลอดเลือดแดงเลี้ยงหัวใจที่ถูกชักนำโดยเฮโรอีนด้วย อย่างไรก็ตามกลไกที่แท้จริงที่ทำให้เกิดภาวะดังกล่าวยังไม่เป็นที่ทราบชัดเจน
