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Case Report
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A Pediatric Case of Cardiac Toxicity Associated With Illicit Fentanyl Ingestion

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Abstracts

Context: In North America, opioid-related deaths are on the rise. We report a case of pediatric cardiac toxicity likely related to illicit fentanyl ingestion.

Case Details: A 14-year-old male ingested half of an unknown illicit pill. Two hours post ingestion, the patient experienced loss of consciousness, hypotension, cyanosis and diaphoresis. Initial labs revealed elevations in lactate and high sensitivity troponin levels. Chest pain was reported 8 hours post hospital arrival. A chest X-ray revealed right-sided aspiration pneumonia. An electrocardiogram showed ST elevation over the anterior leads and T wave inversion over the inferior leads. An echocardiogram demonstrated borderline systolic function. Cardiac inflammation in the RCA and LAD distributions was evident on cardiac magnetic resonance imaging. Comprehensive urine drug screen was positive for fentanyl and its metabolites, cannabinoids, ondansetron, metoclopramide and ranitidine and was negative for xylazine. The management consists of the administration of non-invasive positive-pressure ventilation, naloxone, dopamine, nor epinephrine and ceftriaxone. 24 hours post admission, the patient was weaned off inotropes and discharged home five days post presentation. A repeat cardiac MRI performed 6 months post ingestion was normal.

Conclusion: There appears to be a relation between pediatric illicit fentanyl use and cardiac toxicity that warrants further analysis.

Keywords: Non-pharmaceutical fentanyl; Illicit; Overdose; Cardiac injury

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Introduction

In North America, there has been a dramatic increase in opioid related death [1,2]. Fentanyl in particular has been implicated in at least 655 deaths in Canada between 2009 and 2014 [3]. Opioids are known to cause respiratory depression, which in turn may lead to severe hypoxemia and cardiac arrest. Direct cardiac toxicity is not a common manifestation of opioid overdose. We are reportinga pediatric case of Non-Pharmaceutical Fentanyl (NPF) ingestion that was associated with manifestations of cardiac toxicity.

Case Presentation

A 14-year-old male was found unresponsive, hypotensive, cyanotic (50% oxygen saturation) and diaphoretic two hours after a reported ingestion of a half of a round, blue-green unidentified pill. There was evidence of hematemesis on the patient's pillow with report of continued emesis en route to the hospital. An initial chest X-ray revealed right-sided aspiration pneumonia. Initial laboratory investigations showed a lactate of 6.4 mmol/L (0.5-1 mmol/L), glucose of 13.0 mmol/L (4-7 mmol/L), a high sensitivity troponin of 206 ng/L (1-14 ng/L). Both blood acetaminophen and salicylate levels were undetectable. A comprehensive urine drug screen was positive for fentanyl and its metabolites, cannabinoids, ondansetron, metoclopramide and ranitidine and negative for xylazine. Chest pain was reported 8 hours post arrival to the hospital. An ECG showed ST elevation over the anterior leads and T wave inversion over the inferior leads (Figure 1) and an echocardiogram demonstrated borderline systolic function; both tests normalized on subsequent examinations. High sensitivity troponin peaked at 311ng/L (normal 1-14 ng/L) within 24 hours whereas lactate normalized within a few hours post presentation. Cardiac inflammation in the RCA and LAD distributions possibly secondary to vasospasm was evident on a cardiac MRI performed two days post hospital admission (Figure 2).

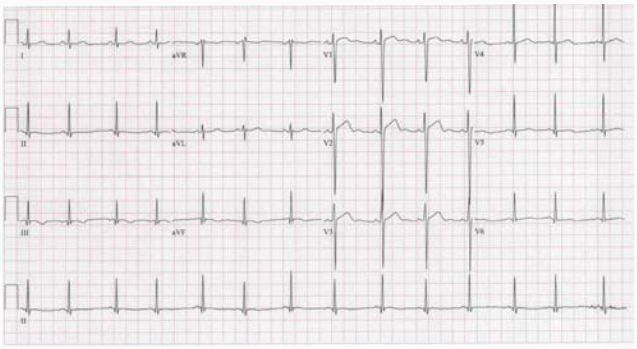

Figure 1: An electrocardiogram taken at the time of chest pain showing ST elevation over the anterior leads and T wave inversion over the inferior leads.

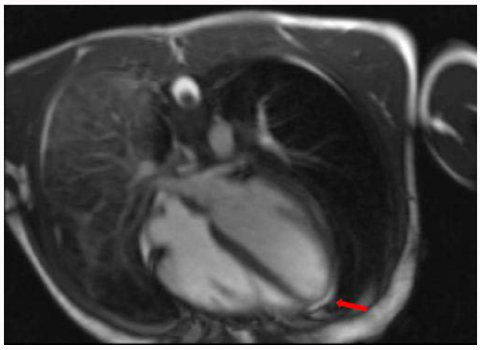

Figure 2: Cardiac MRI, Red arrow indicating an area of epicardial inflammation at the apex of the left ventricle.

Therapeutic management involved the administration of non-invasive positive-pressure ventilation, intravenous naloxone, dopamine, nor epinephrine and ceftriaxone. 24 hours post admission, the patient was successfully weaned off inotropes and switched to room air a few days later. The patient was discharged home after five days of hospitalization. A repeat cardiac MRI performed 6 months post presentation was normal. The patient achieved full recovery.

Discussion

It is well known that the mechanism of opioid-related deaths is associated with the binding of u-opioid receptors at certain sites in the central nervous system inducing respiratory depression that is potentially fatal [4]. Opioid exposures can also contribute to cardiovascular effects such as hypotension and QTc prolongation [5]. Morphine intake can induce histamine release that may result in hypotension whereas methadone ingestion is associated with prolongation of the QTc interval [5]. Cardiac injury following opioid overdose may also occur due to co-ingestions or adulterations of drugs. Xylazine, a centrally acting α-2 agonist that is commonly used as a veterinary tranquilizer was detected in heroin, cocaine and fentanyl abuse cases that resulted in mortality [6,8]. Xylazine has been reported to cause hypotension, diffuse T-wave inversion on ECG, and elevation of cardiac biomarkers [7]. A sympathomimetic agent like-cocaine may explain or contribute to cardiac toxicity. In this patient, a comprehensive urine drug screen was positive for fentanyl and its metabolite but negative for xylazine, cocaine and amphetamines. Though the patient did have detectable tetrahydrocannabinol, which has been implicated in cardiac toxicity, the patient's presentation was more consistent with recent fentanyl use. Other possible causes that could have contributed to the finding of cardiac toxicity include the presence of a cannabinoid effect, another unidentified cardiotoxic illicit substance or a takotsubo cardiomyopathy.

Conclusion

There may be a link between cardiac toxicity and illicit fentanyl use in the pediatric population that requires further investigation of future case reports.

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