Acute coronary syndrome associated with phencyclidine use

Hakeem Ayinde, Robert Solomon, Maria-Elise Sanchez, James Diggs, Prafulla Mehrotra

ABSTRACT

Introduction: Many studies have documented the deleterious effects of psychoactive substances like cocaine and amphetamines on the coronary vasculature. However, the impact of phencyclidine (PCP) on the arteries of the heart has largely gone unrecorded.

Case Report: We report a case of a 41-year-old female presented to our hospital with chest heaviness, shortness of breath, and nausea, which started at rest and lasted about 30 minutes. Her symptoms resolved on arrival to the emergency room. Electrocardiogram showed transient T wave inversions in V2 and V3 leads, and troponins peaked at 1.01 ng/ml 6 hours after arrival. She received standard therapy for non-ST elevation myocardial infarction. An urgent cardiac catheterization revealed severe vasospasm in 3 cm length of the proximal left anterior descending artery, and milder vasospasm in the mid-portion of the artery; spasm resolved after multiple doses of intracoronary nitroglycerin. We excluded the presence of common precipitants of coronary vasospasm. However, the patient admitted to phencyclidine use about two hours prior to the onset of symptoms, and a urine toxicology screen was positive only for the drug. Given the strong temporal relationship of symptoms to PCP use and absence of common precipitants of coronary vasospasm, we concluded that her coronary spasms were induced by PCP.

Conclusion: We describe a case of acute coronary syndrome in a low risk patient thought to be induced by PCP. Our case illustrates the need for physicians to be aware of PCP ingestion as a possible cause of coronary artery spasm when presented with a young adult patient suffering from acute coronary syndrome.

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CASE REPORT

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Keywords: Chest pain, Coronary vasospasm, Phencyclidine

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INTRODUCTION

Phencyclidine (PCP), a synthetic compound originally intended to be used as an anesthetic drug, has been abused throughout the United States for several decades. Its prevalent use has especially been noted in Washington DC where in 2012, up to 12% of male arrestees tested positive for the drug, compared to a 1% rate in other major cities in the country [1]. While PCP is principally noted for its

hallucinogenic effects, it has also been reported to cause such cardiovascular effects as tachycardia, hypertension, and rarely cardiac arrest [2]. Even though animal studies have implicated PCP as a cause of coronary vasospasm, this has not been documented in humans to the best of our knowledge.

We discuss the case of a young adult woman at low risk for coronary artery disease, who presented with acute coronary syndrome due to coronary vasospasm in the setting of acute PCP ingestion.

CASE REPORT

A 41-year-old African American woman presented to the emergency department complaining of chest heaviness, palpitations, shortness of breath, and nausea at rest, which lasted about 30 minutes. She had two similar episodes in the previous month, and these resolved without therapy. Her only cardiovascular risk factor was smoking of a one-fourth pack of cigarettes per day. Her current medications included only vitamins and iron. Symptoms had improved on arrival to the emergency room, and vital signs were within normal limits with a blood pressure of 105/58 mmHg and pulse rate of 85 beats/minute. Physical examination was normal. Electrocardiogram showed transient T wave inversion in both leads V2 and V3 (Figure 1). Laboratory studies revealed rising troponin levels that peaked at 1.01 ng/ml 6 hours after arrival. She was diagnosed with non-ST elevation MI and given standard therapy of aspirin, clopidogrel, and enoxaparin, and she was subsequently prepared for urgent coronary angiography.

Coronary angiography revealed severe vasospasm in 3 cm length of the proximal left anterior descending artery, as well as a milder vasospasm in the mid-portion of the vessel (Figure 2A). Three doses of 200 µg intracoronary nitroglycerin were needed to relieve the vasospasm, after which there were no areas of flow-limiting stenosis identifiable in the coronary arteries (Figure 2B). Further history revealed PCP ingestion about 2 hours prior to the onset of symptoms, and a urine drug screen was positive only for phencyclidine. The patient was discharged on sublingual nitroglycerin as needed, and advised to avoid PCP and other drugs of abuse in the future.

DISCUSSION

To the best of our knowledge, this is the first reported association between PCP ingestion and acute coronary syndrome. Our patient was a young adult woman at low risk for coronary artery disease who presented with chest pain at rest and was found to have severe coronary spasm that was reversed by nitroglycerin during angiography. Urine toxicology screen was positive for PCP, but negative for other stimulants such as cocaine or amphetamine. We considered and excluded other common triggers for

Figure 1: Initial electrocardiogram showing transient T wave inversions in V2 and V3

Figure 2: (A) Antero-posterior, cranial view of coronary angiogram showing severe spasm in proximal Left anterior descending (white arrows) and milder stenosis in the more distal portions artery (black arrows). (B) Resolution of spasm (arrows) after intracoronary nitroglycerin.

coronary spasm (Table 1) [3, 4]. She had a history of cigarette smoking but she did not smoke any cigarettes prior to onset of symptoms on the day she presented. We suggest that the coronary vasospasm may have been caused by PCP ingestion since her symptoms started only about 2 hours after smoking PCP.

PCP (1-(1-phenylcyclohexyl)piperidine hydrochloride) is a noncompetitive NMDAR (N-methyl-D-aspartate receptor) antagonist initially introduced in the 1950s as a dissociative anesthetic agent but withdrawn because of its prolonged unmanageable side effects [5]. It is now a commonly abused street drug in major US cities, especially Washington DC [1].

The major cardiovascular effects in cases of PCP toxicity are hypertension and tachycardia [2], probably due to the inhibition of neuronal catecholamine reuptake or potentiation of noradrenaline release [6].

Although undocumented in humans, animal studies have demonstrated the effects of PCP on coronary vasculature [7–10]. In vitro, PCP appeared to have a vasoconstrictive effect on the coronary arteries in pigs and dogs. It also caused a reversible reduction of coronary blood flow in guinea pigs [7–10]. While PCP induced a paradoxical increase in coronary blood flow in dogs, a simultaneous ECG recording showed that ischemic changes were present [10]. The effects of PCP on the coronaries were inhibited by detromethorphan, a direct

Table 1: Associated factors and precipitants of coronary vasospasm

Associated factors and precipitants of coronary vasospasm	
Pharmacologic agents	Physiologic factors
Catecholamines (epinephrine, dobutamine, etc.)	Genetic predisposition
Hypercholinergic crisis	Stress (physical, mental)
Histamine (Kounis syndrome)	Exposure to cold
Ergot derivatives (e.g., ergonovine)	Valsalva maneuver
5-Hydroxytryptamine antagonists (Ondansetron, Dolasetron)	Hyperventilation
Smoking	Coronary intervention procedures (balloon angioplasty)
Beta-adrenergic blocking agents	
Withdrawal from chronic nitroglycerin use	Disease States
Cocaine	Thyrotoxicosis
Amphetamines	Pheochromocytoma
Ecstasy	Neurofibromatosis
Heavy alcohol use	Magnesium deficiency (particularly in alcoholics)
Postpartum bromocriptine use	
5-Hydroxytryptamine agonists (Sumatriptan)	
Butane	

inhibitor of the PCP receptor [7]. This suggests that PCP may act directly on receptors in animal coronary arteries.

Recent data on PCP effects in humans is not extensive most likely because it is not a widely prevalent drug of abuse [1], and because its clinical use has been discontinued for many years. Additionally, many street drug abusers ingest multiple drugs, and thus there may be confounding effects of these drugs if their activity and pharmacokinetics are not well known. For example, up to 34% of male arrestees in major US cities tested positive for multiple drugs in 2012 [1].

The closest drug to PCP that is in clinical use is Ketamine (2-(2-chlorophenyl)-2-(methylamino) cyclohexan-1-one). Ketamine is a structural analogue of PCP with similar effects but less toxicity, and it is used as a dissociative sedative for brief procedures, particularly in children. The side effects of ketamine and PCP are similar at toxic doses, and in addition, the former has been associated with chest pain when used for analgesia or as a drug of abuse.

Our patient developed chest pain two hours after smoking PCP. We acknowledge that the temporal relationship does not prove PCP as the culprit. However, evidence from animal studies and reports on ketamine (a

structural analogue of PCP) use in humans support the suggestion of PCP as the precipitant of coronary spasm in our patient.

CONCLUSION

Although there is paucity of data in humans, phencyclidine (PCP) has been shown to cause vasoconstriction and reduction in coronary blood flow in animal models. Additional studies are needed to confirm the effects of the drug on the human coronary artery. Our case illustrates the need for physicians to be aware of PCP ingestion as a possible cause of coronary artery spasm in young adult patients presenting with acute coronary syndrome.

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Author Contributions

Hakeem Ayinde – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Robert Solomon – Acquisition of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Maria-Elise Sanchez – Acquisition of data, Analysis and interpretation of data, Drafting the article, Final approval of the version to be published

James Diggs – Acquisition of data, Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Prafulla Mehrotra – Substantial contributions to conception and design, Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Guarantor

The corresponding author is the guarantor of submission.

Conflict of Interest

Authors declare no conflict of interest.

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