BJCR
Brazilian Journal of Case Reports

PCAA
Publicações Científicas de Acesso Aberto

Case Report

Crack Lung – A Case Report

Débora Lopes [1,*], Cátia Gorgulho [1], Joana Ribeiro [1], André Neto Real [1], Nuno Catorze [1]

[1] Intensive Care Unit, Local Health Unit of Médio Tejo, Abrantes, Portugal.

* Correspondence: debora18_lopes@hotmail.com.

Abstract: Cocaine is an alkaloid extracted from the leaves of Erythroxylon coca and was first isolated in 1859. Cocaine induces both acute and chronic toxicity, affecting virtually every organ system. A 32-year-old male patient, with a history of post-traumatic epilepsy and substance abuse involving cannabinoids and cocaine, presented to the Emergency Department (ED) following self-limited seizures. Arterial blood gas analysis revealed hypoxemic respiratory failure, while blood tests showed no significant abnormalities. A thoracic computed tomography (CT) scan demonstrated extensive consolidations in most of the lower, middle, and upper pulmonary lobes. Transthoracic ultrasound suggested signs of non-compacted cardiomyopathy, and pulmonary ultrasound revealed a B-line pattern bilaterally. The patient was admitted to the Intensive Care Unit (ICU), due to respiratory insufficiency requiring invasive ventilation. Over time, his respiratory failure progressively improved, allowing extubation on the eighth day of hospitalization. He was discharged after two weeks and referred for follow-up consultations in Neurology, Psychiatry, and Cardiology. A follow-up CT scan performed three weeks after discharge showed resolution of the pulmonary consolidations and ground-glass opacities. Furthermore, a cardiac magnetic resonance imaging (MRI) study conducted five weeks post-discharge ruled out non-compacted cardiomyopathy. Cocaine-induced lung alterations are diverse and often nonspecific, making their diagnosis reliant on thorough clinical and radiological correlation. In this case, pulmonary parenchymal changes observed on the CT scan and severe respiratory failure raised suspicion for crack lung. Other potential diagnoses, such as cardiogenic and neurogenic pulmonary edema, were excluded.

Keywords: Cocaine; Crack Lung; Hemorrhagic alveolitis.

Citation: Lopes D, Gorgulho C, Ribeiro J, Real AN, Catorze N. Crack Lung – A Case Report. Brazilian Journal of Case Reports. 2025 Jan-Dec;05(1):bjcr49.

https://doi.org/10.52600/2763-583X.bjcr.2025.5.1.bjcr49

Received: 29 September 2024
Accepted: 19 December 2024
Published: 24 December 2024

1. Introduction

Cocaine is an alkaloid derived from the leaves of Erythroxylon coca, a shrub native to Central and South America, Western India, and Indonesia [1]. It was first isolated in 1859 and initially utilized as a local anesthetic in eye surgery. Its anesthetic and vasoconstrictive properties made it particularly valuable in nasal and throat surgeries due to its ability to reduce bleeding [1]. The effects of cocaine are primarily mediated through three distinct mechanisms. First, it acts as a sympathomimetic agent by inhibiting the presynaptic reuptake of neurotransmitters such as serotonin and catecholamines (dopamine, norepinephrine, and epinephrine). This increases the availability of these neurotransmitters at adrenergic receptors (alpha1, alpha2, beta1, and beta2). Cocaine's euphoric effects result from serotonin reuptake inhibition, while its addictive properties are linked to dopamine reuptake inhibition in the mesolimbic-frontal cortex. Second, cocaine exhibits anesthetic properties by blocking sodium channels, thereby impeding neuronal conduction and reducing the permeability of excitable membranes to sodium. These effects extend to cardiac sodium channels, and in cases of severe overdose, they may present as QRS com-

plex prolongation on an electrocardiogram (ECG) and clinically manifest as negative inotropic effects [2]. Third, cocaine increases the concentration of excitatory amino acids, such as glutamate and aspartate, in the nucleus accumbens, thereby stimulating the central nervous system (CNS) [3].

Cocaine exerts acute and chronic toxic effects on multiple organ systems, largely due to its hemodynamic impact. Acute cardiovascular toxicity is characterized by arterial vasoconstriction and increased thrombus formation, with coronary vasoconstriction being a major contributor to episodes of cardiac ischemia. At high doses, cocaine's negative inotropic effects can result in acute left ventricular dysfunction and heart failure (HF). Additionally, it can lead to supraventricular and ventricular arrhythmias, as well as, albeit rarely, aortic dissection or rupture. Chronic cocaine use accelerates atherogenesis and promotes left ventricular hypertrophy, heightening the risk of cardiac ischemia and potentially leading to dilated cardiomyopathy [4,5].

In the respiratory system, inhaled cocaine may cause pneumothorax, pneumomediastinum, pneumopericardium, and subcutaneous emphysema due to barotrauma induced by the Valsalva maneuver. Vasoconstriction and thrombosis also predispose individuals to pulmonary infarction. Acute pulmonary toxicity, such as crack lung or acute eosinophilic pneumonia, can occur. Repeated cocaine use is associated with histopathological changes, including foreign body granulomatosis (leading to pulmonary hypertension), bronchiectasis, recurrent alveolar hemorrhage, and severe pulmonary fibrosis [6,7].

Cocaine also has profound CNS effects, causing complications such as psychomotor agitation (with or without hyperthermia), seizures, coma, headaches, intracranial hemorrhage, and focal neurological deficits [8]. In the gastrointestinal system, increased sympathetic tone elevates gastric acid production, while vasoconstriction causes local ischemia, leading to gastric and duodenal ulcers. Cocaine use has also been linked to ischemic colitis and intestinal infarction. Though less common, splenic and renal infarction can also occur [9-11].

Musculoskeletal complications, such as rhabdomyolysis, may develop, accompanied by elevated creatine phosphokinase (CK), myoglobin levels, and electrolyte disturbances like hyperkalemia and hypocalcemia [12]. Ocular effects, including mydriasis due to sympathetic activation, may result in angle-closure glaucoma or retinal vasospasm, potentially causing vision loss [13]. Levamisole, an immunomodulator commonly found as a cocaine adulterant, may cause agranulocytosis, leukoencephalopathy, and cutaneous vasculitis, leading to skin necrosis [14]. Movement disorders, such as akathisia, dyskinesias, choreoathetosis, repetitive stereotyped behaviors, and Tourette syndrome exacerbations, may also occur. Chronic use has been associated with selective cognitive impairments affecting attention, memory, and executive functions. Lastly, cocaine is strongly linked to psychiatric comorbidities, including suicidal ideation, suicide attempts, and psychotic symptoms [15].

Through this case report, we aim to alert healthcare professionals to the diverse spectrum of injuries—both common and rare—associated with cocaine use and highlight the importance of considering this factor when evaluating patients with a history of cocaine consumption.

2. Case Report

A 32-year-old male, employed in a commercial store and engaged in bodybuilding as a hobby, presented with a history of post-traumatic epilepsy for nine months of age. The patient reported substance use, including daily consumption of approximately six cannabinoid cigarettes and inhaled or smoked cocaine. Additionally, he self-administered multiple supplements and medications, such as acetate (100 mg/mL, 10 mL intramuscularly), testosterone (300 mg/mL, 10 mL intravenously), stanozolol (10 mg), yohimbine (10 mg), levothyroxine (125 mg), clenbuterol (40 mg), oxandrolone (10 mg), and various die-

tary supplements (artichoke, chromium, creatine, glutamine, Vitamin C/L-carnitine, Vitamin C/D3/Omega 3). A significant family history of sudden death at a young age was noted.

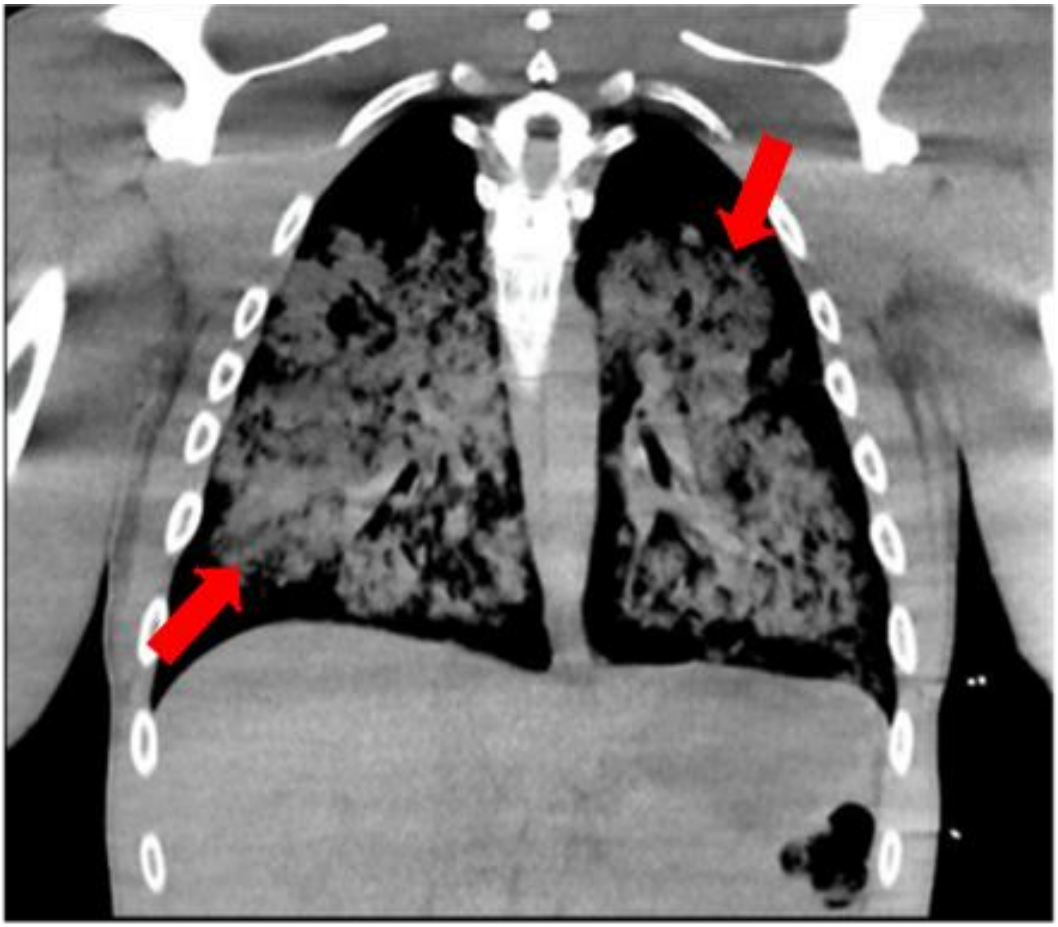
The patient was admitted to the Emergency Department (ED) following multiple self-limited seizure episodes, without a history of traumatic brain injury (TBI). Upon admission, the patient was presented with a Glasgow Coma Scale (GCS) score of 7 (E1V1M5), a respiratory rate exceeding 40 breaths per minute, and oxygen saturation ($SatO_2$) of 70% despite high-flow oxygen supplementation. Pulmonary auscultation revealed bilateral breath sounds with rhonchi scattered across both hemithoraxes. He exhibited a hypertensive profile (blood pressure: 178/68 mmHg), tachycardia (heart rate: 145 bpm), and a regular cardiac rhythm. The patient was febrile, with a recorded temperature of 38.5°C. Anicteric, without peripheral edema, and with an unremarkable abdominal examination. Some abnormalities were seen on blood analysis results, as seen in table 1.

Table 1. Abnormalities in blood test results.

Blood Analysis	**Results**	**Reference Values**
Leukocytes	16,29 10^9/L	4,0 – 10,0
Urea	43 mg/dL	17 - 43
Creatinine	1,3 mg/dL	0,8 – 1,2
Potassium	5,6 mmol/L	3,5 – 5,1
Aspartate aminotransferase	109 IU/L	0 – 50
Alanine aminotransferase	111 IU/L	0 - 50
C-Reative Protein	0,74 mg/dL	<0,50
Procalcitonine	0,66 ng/mL	0,30-1,2
Lactate dehidrogenase	548 IU/L	0 – 248
Creatine kinase	4145IU/L	0- 171
Creatine kinase - MB	46,0 U/L	<24,0
Myoglobin	578,4 ng/mL	<105,7
Troponine I	98,7 pg/mL	<2,3
Type B natriuretic peptide	69 pg/mL	

Arterial blood gas analysis showed severe hypoxemic respiratory failure accompanied by respiratory alkalosis and hyperlactatemia. A toxicological screen of the urine was positive for cannabinoids only. In the Emergency Department (ED), the patient required orotracheal intubation followed by invasive mechanical ventilation. A chest computed tomography (CT) scan revealed extensive ground-glass opacities and consolidations, involving almost entirely the lower lung lobes as well as the most dependent segments of the upper and middle lobes (Figure 1).

Figure 1. Chest CT scan at admission, showing ground glass areas and extensive consolidations of practically all parts of lower lobes and part of the most dependent upper lobes segments and middle lobe. No pleural or pericardial effusions. No other relevant alterations.

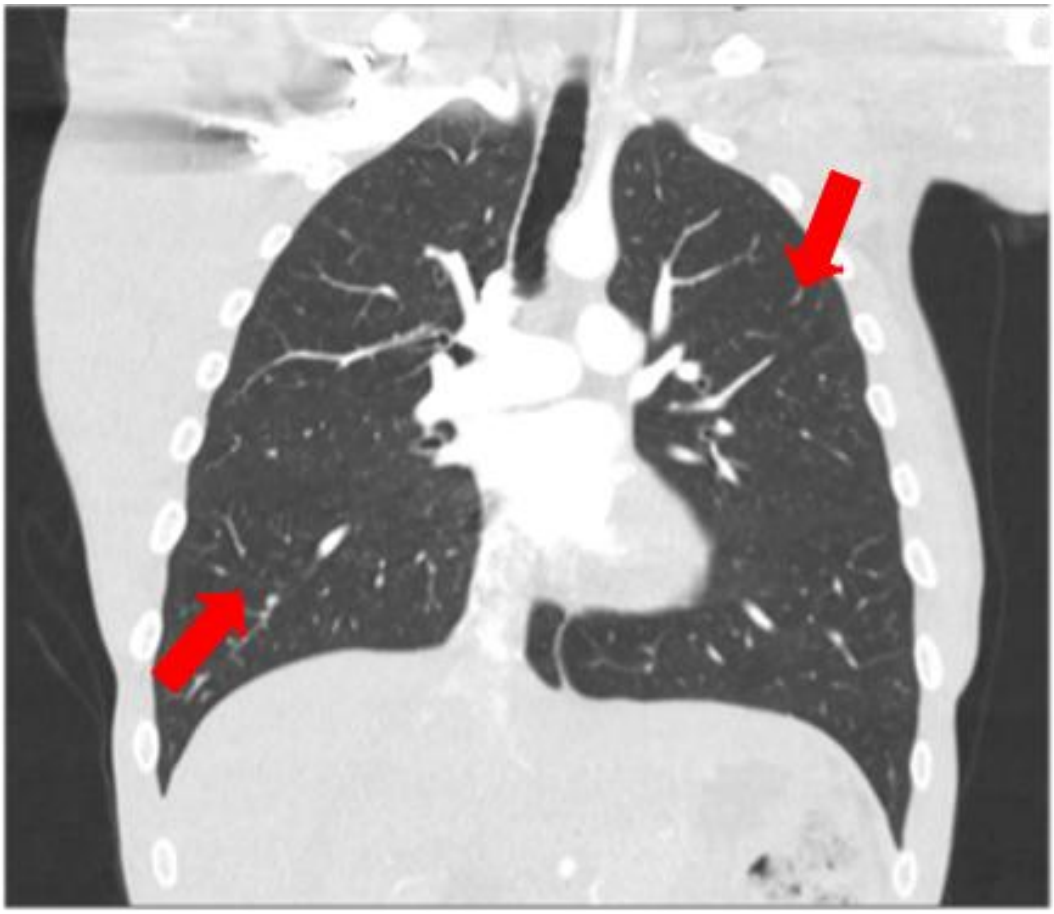


A cranial computed tomography (CT) scan was also performed, revealing only inflammatory changes, including mastoid opacification and pansinusitis, with no other abnormalities. Upon admission to the Intensive Care Unit (ICU), the patient had a Richmond Agitation-Sedation Scale (RASS) score of -5 while on continuous neuromuscular blockade. Despite invasive mechanical ventilation, the respiratory failure worsened, with P/F ratios falling below 100 and reduced lung compliance. A 17-hour period of prone positioning, combined with corticosteroid therapy using methylprednisolone for five days, resulted in improved oxygenation. Initial microbiological testing yielded no positive results.

Given the unexplained etiology of the respiratory failure, a transthoracic ultrasound was performed, showing signs suggestive of non-compacted cardiomyopathy, while pulmonary ultrasound indicated a congestive lung pattern.

Seventy-two hours post-intubation, the patient exhibited clinical deterioration with fever, elevated inflammatory markers, and the isolation of *Citrobacter koseri* from bronchial secretions. A diagnosis of ventilator-associated pneumonia (VAP) was made, prompting initiation of antibiotic therapy with piperacillin/tazobactam, which was later de-escalated to amoxicillin/clavulanic acid based on antibiotic sensitivity testing (AST). With comprehensive supportive management, the patient's oxygenation progressively improved, allowing for a gradual weaning from mechanical ventilation and planned extubation on the eighth day of ICU admission. Three weeks post-discharge, a follow-up lung CT scan revealed marked radiological improvement, with resolution of nearly all consolidations and ground-glass opacities in both lower lobes and the posterior segments of the upper lobes (Figure 2).

Figure 2. Chest control CT scan, performed 3 weeks after discharge, showing marked radiological improvement with resolution of almost all consolidation and ground glass areas of both lower lobes and in the posterior segments of both upper lobes.



Five weeks post-discharge, the patient underwent cardiac Magnetic Resonance Imaging (MRI), which ruled out non-compacted cardiomyopathy initially suggested by the transthoracic echocardiogram findings. Following discharge, the patient was referred for follow-up consultations in Cardiology, Neurology, and Psychiatry. Effective management of cocaine dependence is crucial to prevent future episodes of intoxication and to improve the patient's long-term prognosis.

3. Discussion

Initially, several diagnostic hypotheses were proposed to explain the patient's clinical condition. The respiratory failure was attributed to pulmonary edema of unclear etiology, with cardiogenic origin being the first hypothesis. An initial echocardiogram suggested non-compacted cardiomyopathy based on Jenni et al.'s criteria, leading us to consider this as a possible diagnosis. It is a rare disorder caused by incomplete embryonic compaction of the ventricular myocardium, resulting in a distinct myocardial structure with a compacted outer layer and a non-compacted inner layer retaining embryonic features. Although most cases are hereditary, typically autosomal dominant or X-linked, there may also be acquired forms. Patients may remain asymptomatic or present with HF symptoms, such as dyspnea on exertion, fatigue, and peripheral edema, as seen in this patient. However, the normal troponin I and pro-BNP levels on admission, along with the normal cardiac MRI findings after discharge, allowed us to exclude this hypothesis [16].

Another possible cardiogenic cause of pulmonary edema was stress cardiomyopathy (Takotsubo syndrome) [17]. This condition can result from catecholamine toxicity, microvascular dysfunction, or multivessel spasm. Cocaine use and seizures could both act as

triggers for this condition. The diagnostic criteria most commonly used for Takotsubo cardiomyopathy are those established by the Mayo Clinic, which include: 1. Transient segmental wall motion abnormalities typical of the syndrome. 2. Absence of obstructive coronary artery disease or acute plaque rupture. 3. New electrocardiographic changes or modest elevation of cardiac biomarkers (e.g., troponin). 4. Exclusion of pheochromocytoma or myocarditis. In this case, the absence of classic segmental changes, such as transient apical ballooning, along with normal electrocardiogram findings, troponin I, and pro-BNP values, excluded this diagnostic possibility [17].

Alternatively, the pulmonary edema could have been non-cardiogenic, with neurogenic pulmonary edema being a potential explanation. Neurogenic pulmonary edema typically occurs in response to neurological insults such as traumatic brain injury, intracranial surgery, status epilepticus, intracerebral or subarachnoid hemorrhage, or even electroconvulsive therapy [18]. Sympathetic hyperactivity leads to blood redistribution from systemic to pulmonary circulation, increasing pulmonary intravascular pressure. This, combined with heightened vascular permeability, contributes to pulmonary edema formation. Symptoms usually develop rapidly and resolve within 48 to 72 hours, with the outcome depending on the primary neurological insult. In this case, the onset of seizures could have triggered pulmonary edema, potentially induced by cocaine use. While we could not entirely rule out this diagnosis, the imaging findings on the chest CT scan were more consistent with cocaine-induced lung injury [18].

Cocaine-induced lung injuries are characterized on CT scans by findings such as ground-glass opacities, consolidations, paraseptal emphysema, and centrilobular nodules [19]. In our case, the ground-glass areas and extensive consolidations observed on the patient's chest CT scan at admission align with findings reported in other cases of crack lung [20, 21]. Although blood analysis and toxicological tests are limited in their ability to confirm or exclude cocaine intoxication, imaging studies provide crucial diagnostic information in these cases. The patient's history of prior cocaine use, the acute onset of dyspnea progressing to severe respiratory failure, and the characteristic CT scan findings strongly support the diagnosis of cocaine-induced lung injury.

The radiological improvement seen on follow-up imaging three weeks after discharge, with resolution of the consolidations and most ground-glass opacities, further supports this diagnosis [20]. Supportive care, combined with cessation of cocaine use, remains the cornerstone of management for these patients. The primary therapeutic goal is to ensure adequate oxygenation while allowing the acute lung injury to resolve [22]. Equally important is the follow-up care after hospital discharge. Referring patients to addiction rehabilitation programs is critical to preventing recurrence and improving their long-term prognosis.

4. Conclusion

The lung alterations caused by cocaine use are nonspecific and should be interpreted in the context of the patient's history, after excluding other potential causes. In patients presenting with respiratory failure and a history of cocaine consumption, crack lung should be considered as a possible diagnosis. This condition is characterized by acute and severe lung injury, which can rapidly progress to respiratory failure. Other thoracic complications related to cocaine use, with high prevalence, include barotrauma, talcosis, and bullous emphysema. Organizing pneumonia has also been observed, along with rarer but important complications such as pulmonary infarction, septic embolism, cardiogenic pulmonary edema, and eosinophilic pneumonia. These conditions must be kept in mind when forming a differential diagnosis for patients with respiratory symptoms and a history of cocaine use.

Funding: Not applicable.

Research Ethics Committee Approval: We declare that the patient approved the study by signing an informed consent form and the study followed the ethical guidelines established by the Declaration of Helsinki.

Acknowledgments: None.

Conflicts of Interest: The authors declare no conflicts of interest.

References

1. Stolberg VB. The use of coca: prehistory, history, and ethnography. J Ethn Subst Abuse. 2011 Apr;10(2):126-46. doi:10.1080/15332640.2011.573310.
2. Tella SR, Schindler CW, Goldberg SR. Cardiovascular effects of cocaine in conscious rats: relative significance of central sympathetic stimulation and peripheral neuronal monoamine uptake and release mechanisms. J Pharmacol Exp Ther. 1992 Aug 1;262(2):602-10.
3. Smith JA, Mo Q, Guo H, Kunko PM, Robinson SE. Cocaine increases extraneuronal levels of aspartate and glutamate in the nucleus accumbens. Brain Res. 1995 Jun 19;683(2):264-9. doi:10.1016/0006-8993(95)00383-2.
4. Lange RA, Cigarroa RG, Yancy CW Jr, Willard JE, Popma JJ, Sills MN, et al. Cocaine-induced coronary-artery vasoconstriction. N Engl J Med. 1989 Dec 7;321(23):1557-62. doi:10.1056/NEJM198912073212301.
5. Kolodgie FD, Wilson PS, Cornhill JF, Herderick EE, Mergner WJ, Virmani R. Increased prevalence of aortic fatty streaks in cholesterol-fed rabbits administered intravenous cocaine: the role of vascular endothelium. Toxicol Pathol. 1993 Sep-Oct;21(5):425-35. doi:10.1177/019262339302100501.
6. Maeder M, Ullmer E. Pneumomediastinum and bilateral pneumothorax as a complication of cocaine smoking. Respiration. 2003 Jul-Aug;70(4):407. doi:10.1159/000072905.
7. Ettinger NA, Albin RJ. A review of the respiratory effects of smoking cocaine. Am J Med. 1989 Dec;87(6):664-8. doi:10.1016/s0002-9343(89)80401-2.
8. Smollin CG, Hoffman RS. Cocaine. In: Goldfrank's Toxicologic Emergencies. 11th ed. Nelson LS, Howland MA, Lewin NA, Smith SW, Goldfrank LS, Hoffman RS, editors. New York: McGraw Hill; 2019. p. 1124.
9. Lee HS, LaMaute HR, Pizzi WF, Picard DL, Luks FI. Acute gastroduodenal perforations associated with use of crack. Ann Surg. 1990 Jan;211(1):15-7. doi:10.1097/00000658-199001000-00003.
10. Novielli KD, Chambers CV. Splenic infarction after cocaine use. Ann Intern Med. 1991 Feb 1;114(3):251-2. doi:10.7326/0003-4819-114-3-251.
11. Edmondson DA, Towne JB, Foley DW, Abu-Hajir M, Kochar MS. Cocaine-induced renal artery dissection and thrombosis leading to renal infarction. WMJ. 2004;103(7):66-9.
12. Flaque-Coma J. Cocaine and rhabdomyolysis: report of a case and review of the literature. Bol Asoc Med P R. 1990 Sep;82(9):423-4.
13. Libman RB, Masters SR, de Paola A, Mohr JP. Transient monocular blindness associated with cocaine abuse. Neurology. 1993 Jan;43(1):228-9. doi:10.1212/wnl.43.1_part_1.228-a.
14. Midthun KM, Nelson LS, Logan BK. Levamisole—a toxic adulterant in illicit drug preparations: a review. Ther Drug Monit. 2021 Apr;43(2):221-8. doi:10.1097/FTD.000000000000085.
15. Oliva F, Mangiapane C, Nibbio G, Berchialla P, Colombi N, Vigna-Taglianti FD. Prevalence of cocaine use and cocaine use disorder among adult patients with attention-deficit/hyperactivity disorder: a systematic review and meta-analysis. J Psychiatr Res. 2021 Nov;143:587-98. doi:10.1016/j.jpsychires.2020.11.021.
16. Weiford B, Subbarao V, Mulhern K. Noncompaction of the ventricular myocardium. Circulation. 2004 Jun 22;109(24):2965-71. doi:10.1161/01.CIR.0000132478.60674.D0.
17. Prasad A, Lerman A, Rihal CS. Apical ballooning syndrome (Tako-Tsubo or stress cardiomyopathy): a mimic of acute myocardial infarction. Am Heart J. 2008 Mar;155(3):408-17. doi:10.1016/j.ahj.2007.11.008.
18. Baumann A, Audibert G, McDonnell J, Mertes PM. Neurogenic pulmonary edema. Acta Anaesthesiol Scand. 2007 Apr;51(4):447-55. doi:10.1111/j.1399-6576.2007.01276.x.
19. Almeida RR, Zanetti G, Souza AS Jr, et al. Cocaine-induced pulmonary changes: HRCT findings. Braz J Pneumol. 2015 Jul-Aug;41(4):323. doi:10.1590/S1806-37132015000000025.
20. Giacomi FD, Srivali N. Cocaine use and crack lung syndrome. QJM. 2019 Feb;112(2):125-6. doi:10.1093/qjmed/hcy255.
21. Greenberg A, Stammers K, Moonsie I, Jose RJ. All puffed out—a case of crack lung. Clin Med. 2017 Apr;17(2):186-7. doi:10.7861/clinmedicine.17-2-186.
22. Gorelick DA. Testing for substances of abuse. In: American Psychiatric Association Publishing Textbook of Substance Use Disorder Treatment. 6th ed. Brady KT, Levin FR, Galanter M, Kleber HD, editors. Washington (DC): American Psychiatric Association; 2021.