REVIEW ARTICLE

The crack user patient – diagnosis and therapy in urgent care

O paciente usuário de crack *– diagnóstico e terapêutica na urgência*

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ABSTRACT

Crack has extremely harmful effects and is associated with the risk of sudden death. Brazil is one of the few countries where the use of cocaine and crack is increasing. About 1% of the Brazilian population frequently consumes cocaine and apparently half of this consumption is in the form of crack, which corresponds to 600 thousand users; the emergency care for these people should increase in the coming years. Acute cocaine intoxication is characterized primarily by manifestations of noradrenergic and dopaminergic hyperactivity, which affects several organs and systems. Drug use is an aggravating factor in any patient admitted to an emergency room. The doctor-patient relationship with drug users such as crack is usually difficult. The goal of treatment of acute intoxication by crack is based on general support, abstinence, and relapse prevention; it must be set up properly by a trained professional to prevent an even increase in morbidity and mortality resulting from its use.

Key words: Cocaine; Crack Cocaine; Cocaine-Related Disorders; Emergency Medicine; Therapeutics.

RESUMO

O crack é uma substância de efeitos extremamente danosos e se associa a riscos de morte súbita. O Brasil é dos poucos países no mundo onde o consumo de cocaína e crack está aumentando. Cerca de 1% da população brasileira faz algum consumo frequente de cocaína e aparentemente metade desse consumo é na forma de crack, o que corresponde a 600 mil usuários; e o atendimento de emergência dessas pessoas deve aumentar nos próximos anos. A intoxicação aguda por cocaína se caracteriza, fundamentalmente, por manifestações de hiperatividade noradrenérgica e dopaminérgica, que afeta vários órgãos e sistemas. O uso de drogas é fator agravante em qualquer paciente admitido em pronto-atendimento. A relação médico-paciente com usuários de drogas como crack é, usualmente, de difícil realização. O objetivo do tratamento da intoxicação aguda pelo crack baseia-se em suporte geral, abstinência e prevenção de recaídas; e deve ser instituído de forma adequada por profissional habilitado, para evitar aumento ainda maior da morbimortalidade decorrente do seu uso.

Palavras-chave: *Cocaína; Cocaína Crack; Transtornos Relacionados ao Uso de Cocaína; Medicina de Emergência; Terapêutica.*

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INTRODUCTION

In a recent report, the United Nations showed that Brazil is among the few countries in the world where the consumption of cocaine and crack is increasing.[1] The explana-

tion involves geographical reasons because Brazil borders producing regions such as Bolivia, which facilitates its distribution.[1] Crack has extremely harmful effects and is associated with severe adverse outcomes such as violence, crime[2,2,3] homicide,[4] health problems[5-8], increased mortality[3], neuropsychological alterations[9], overdosing[10], and intense psychosocial damages that are not always subjected to documentation.

The World Health Organization estimates that cocaine is used by more than 14 million people in the world.[11] Approximately 1% of the Brazilian population use cocaine frequently and apparently half of this consumption is in the form of crack, which corresponds to 600,000 crack users according to estimates of the Ministry of Health.[1]

A study at UNIFESP following up for more than 15 years the first 131 crack users identified in the early 1990s in the city of São Paulo showed that about 30% of them died in the first five years of consumption[1]; mostly by murder. If this study can be used to evaluate what happens in Brazil as a whole, the death of at least 180,000 crack users will happen in the coming years.[1]

Based on these data, it is observed that the demand for emergency treatment of crack users is increasing and will continue in this trend. This results in increased frequency of emergency visits of patients with acute intoxication and frequent complications from using this type of drug. This article proposes to review and update the diagnosis and clinical treatment to this type of patient, in addition to addressing complications that may occur during acute intoxication by crack.

METHODS

This literature review was divided into two stages: the first consisted of a search on the MedLine, LILACS, and SciELo, and PubMED sites. The descriptors used were: crack AND/OR cocaine AND/OR treatment AND/OR therapy AND/OR urgency AND/OR emergency AND/OR protocol. The selection was made through words and information of interest found in the titles and summaries of articles.

The second step was to search for relevant information in material produced by the Ministry of Health, Federal Council of Medicine, and articles published in UpToDate.com®.

Information related to therapies considered the most current is presented.

RESULTS

Pharmacokinetics and pharmacodynamics

Crack is a derivative of cocaine and acquired its named because of the sound it makes when consumed; it is also called cocaine stone.[11,12] In Brazil, cocaine is predominantly consumed intranasally ("snorted") while crack is smoked but can also be used intravenously and orally (less frequent).[13]

Crack spreads very rapidly from the lungs to the brain. Its effects are immediate (five seconds), very intense (it is said that they are 10 times more intense than intravenous cocaine) and fleeting (five minutes). Because of these characteristics (Table 1), the use becomes compulsive and frequent.[13]

Table 1 - Pharmacokinetic properties of *crack*[13]

Pharmacokinetic properties	Form/intensity
Cocaine concentration	30-80%
Route of administration	Inhaled/Smoked
Plasma percentage	70-80%
Speed of onset of effects	Very rapid
Maximum plasma concentrations	8-10 seg
Duration of effects	5-10 min
Triggering dependence	Intense

Source: LIZASOAIN I, MORO MA, LORENZO P. Cocaine: pharmacological aspects. Revision. Pharmacology Department. Medical School. Universidad Complutense de Madrid. ADICCIONES, 2002.

The bioavailability of crack varies between 10 and 20%. The plasma peak level of smoked cocaine (crack) occurs within 10 seconds after use; however, the individual variability is great.[13] The distribution volume varies between 1.5 and 2 U/kg. Cocaine is rapidly metabolized, typically by enzymatic hydrolysis to produce benzoylecgonine, ecgonine-methyl ester, and later ecgonine; 1 to 5% being excreted in the urine without alterations. The benzoylecgonine hydrolysis occurs in 45% of the administered dose, similar to the hydrolysis percentage of ecgonine-methyl ester. Neither of these metabolites has significant biological activity in humans.

Norcocaine nitroxide and other free radicals are potentially active metabolites produced in small amounts, usually clinically, and not pharmacologically significant.[13] Benzoylecgonine can be detected in urine three to four days after the last consumption; the concentration found will depend on the amount

of cocaine consumed and the sensitivity of the test used. The route of administration also influences the amount of plasma benzoylecgonine, which will be eliminated through urine. When cocaine is smoked, although the effects produced are much more intense and early, the amount absorbed is less, and therefore, the concentration of plasma benzoylecgonine is also less.[13]

Alcohol consumption by cocaine addicts is very common, being observed in up to 99% of users;[13] the reason for this simultaneous use is not yet fully understood. The potentiation of euphoria by the simultaneous intake of alcohol may underlie this association, although a decrease in undesirable effects, such as a migraine induced by cocaine, is reported. This combination implies risk and increased morbidity and mortality associated with cocaine. Epidemiological data indicate that the practice of this association increases the risk of sudden death from cocaine in up to 18 times.[13] Ethanol, in *in vitro* studies, inhibits the activity of methylesterase by decreasing the hydrolysis of benzoylecgonine. In the presence of ethanol, cocaine is transesterified by liver esterases to ethyl cocaine or cocaethylene, increasing the N-demethylation to norcocaine. The cocaethylene metabolite has pharmacological and toxic activity (cardiac and liver).[13]

Cocaine behaves as a sympathomimetic amine of indirect action. It mimics the actions of catecholamines without directly acting on adrenergic or dopamine receptors and only increases the availability of neurotransmitters in the synaptic clefts. Cocaine inhibits the type 1 reuptake process (noradrenaline and dopamine reuptake by the presynaptic membrane).[13] The crack's mechanism of action aid understanding the effects in the body, provides signs and symptoms for the diagnosis of acute intoxication, and anticipates possible complications.

Diagnostic criteria

The diagnostic criteria of acute cocaine intoxication were established by the Diagnostic and Statistical Classification of Mental Disorders (DSM-IV) (Table 2).

The expected effects of smoked cocaine (crack) are more fleeting than its intranasal use ("snorted"). Therefore, symptoms of abstinence are most commonly noted in possibly crack users (Table 3).

Table 2 - Diagnostic criteria for cocaine intoxication according to DSM-IV

A. Recent cocaine use.
B. Behavioral or psychological alterations ill-adapted and medically significant (euphoria or affective blunting; changes in sociability; hypervigilance; interpersonal sensitivity; anxiety; tension or anger; stereotyped behaviors; impaired judgment; impaired social or occupational functioning), which develop during or shortly after using cocaine.
C. At least two of the following symptoms that develop during or shortly after the use of cocaine:
(1) Tachycardia or bradycardia
(2) Dilated pupils
(3) High blood pressure or below normal
(4) Perspiration or chills
(5) Nausea or vomiting
(6) Evidence of weight loss
(7) Agitation or motor retardation
(8) Muscular weakness, respiratory depression, thoracic pain, or arrhythmias
(9) Confusion, dyskinesia convulsions, dystonia, or coma
D. The symptoms do not result from a general medical condition or can be better accounted for another mental disorder.

Source: DSM-IV.

Table 3 - Diagnostic criteria for cocaine abstinence according to DSM-IV

A. Cessation (or reduction) of heavy and prolonged use of cocaine.
B. Dysphoric mood and at least two of the following physiological alterations that develop from a few hours to a few days after the A criterion:
(1) Fatigue
(2) Vivid and unpleasant dreams
(3) Insomnia or hypersomnia
(4) Increased appetite
(5) Psychomotor retardation or agitation
C. The symptoms of criterion B cause clinically significant distress or harm in social, occupational, or other areas of important functioning.
D. The symptoms do not result from a general medical condition or can be better accounted for another mental disorder.

Source: DSM-IV.

Acute toxicity

Acute cocaine intoxication is characterized by primarily manifestations of noradrenergic and dopaminergic hyperactivity affecting various organs and systems.[13]

The most common manifestations separated by organic systems are as follows:

- **cardiovascular system –** palpitations, bradycardia or tachycardia (most common), arrhythmias (atrial fibrillation is the most common, ventricular tachycardia and ventricular fibrillation are the most common causes of sudden death due to co-

caine), hypertension and acute myocardial infarction. The concurrent use of cocaine and alcohol produces cocaethylene, which is an active metabolite and with more cardiac toxicity than cocaine. Its arrhythmogenic potential and sudden death induction is higher than that of cocaine.[13]

- **respiratory system** – tachypnea and irregular breathing. Crack is responsible for the widest range of acute complications related to lung and airways such as: acute pulmonary edema (usually of non-cardiogenic etiology by increasing alveolar-capillary permeability); exacerbation of bronchial asthma; pneumothorax; pneumomediastinum, and pneumopericardium.[13] The "crack lung" of possible ischemic etiology, is a hemorrhagic alveolitis syndrome that presents with a cough, dyspnea, and nonspecific chest pain. It can also be associated with hypoxia, productive cough with hemoptoico, and sometimes frank hemoptysis, fever, focal infiltrates, and bronchospasm.[13,14]
- **digestive system** – anorexia, nausea, vomiting, diarrhea, and the most serious, gastroduodenal ulcers with bleeding and perforation, ischemic colitis.[13]
- **hepatic** – cocaine has specific hepatotoxicity, being primarily metabolized by the plasma and hepatic cholinesterases, however, a small proportion of the administered dose follows the hepatic microsomal oxidative pathway that gives rise to reactive metabolites capable of being free radicals. Cocaethylene, the metabolite mixed of cocaine and alcohol, also has this property. The acute toxic hepatic lesions caused by cocaine are of cytolytic type, which leads to increased plasma hepatic enzymes.[13]
- **metabolism** – malignant hyperthermia suggests a misfit in the dopaminergic control of body temperature. Hyperthermia, stiffness, and shaking may arise. Rhabdomyolysis may arise after intravenous administration or crack use; even though rare, it is very serious.[13]
- **eyes** – mydriasis, conjunctival vasoconstriction, vertical nystagmus[13]
- **neurological** – headache (the most frequent complication); stroke (in some centers, it is the most common cause of stroke in young people); cerebral hemorrhage (usually subarachnoid); seizures (generalized tonic-clonic type). Crack is what is most associated with these acute complications.[13]
- **psychological** – anxiety comes as the euphoric symptoms disappear, mental confusion, irritability, visual and tactile hallucinations, perception alterations, paranoid reactions, and tonic-clonic seizures.

The use of crack cocaine can impair cognitive skills involved especially with the executive function and attention. This involvement alters the problem-solving ability, mental flexibility, and speed in processing information.[15] It can cause damage to mental functions, with damage to memory, attention, and concentration. In many cases, depending on genetic predisposition, the development of psychiatric symptoms occurs, psychotic and anxious, such as depression, delusions, and panic attacks; it can also cause bipolar disorder resulting from the rapid and intense euphoria mechanism, immediately after its use, which is rapidly replaced by depression when the user is in abstinence.[15]

Treatment of acute intoxication

Non-pharmacological clinical aspects

It is extremely difficult to establish a physician-patient relationship with drug users such as crack to obtain reliable information regarding the pattern of use and properly analyze the impact of drugs on the patient's life, due to the circumstance of the consultation (emergency) or the patient's mental state (agitated, aggressive, hostile).

Treatment should be interdisciplinary, directed to various affected areas: physical, psychological, social, legal, and quality of life.[16] The goal of treatment of acute intoxication is to provide general support, start abstinence, and prevent relapses.[16] The patient's entry into the addiction treatment network should be started. There are several important points in the initial approach to the patient with crack intoxication (Table 4)[12] that help ensure a successful treatment of intoxication and dependence.

Clinical and pharmacological aspects

General conduct and specific discharge criteria.

General conduct

The care of the intoxicated patient follows steps that are not always sequential. Although well defined, their implementation presents, thus far, many controversial aspects.[17]

Table 4 - Important points in the initial approach of a patient intoxicated with *Crack*[12]

Specific anamnesis
Ask about the use of illicit drugs without betraying any judgment of value, perhaps after asking about the use of cigarettes, alcohol, and any another drug that is relevant.
Ask about the pattern and amount consumed and any behaviors associated with drug use that could damage his own health and that of others (e.g., smoked or injected drugs, activities during intoxication, financial implications, capacity of caring for children, violence in relation to others).
Ask about the beginning and development of drug use in relation to other life events.
Ask about damage caused by use of drugs, more particularly: injuries and accidents; driving under the influence of drugs; interpersonal relationship problems; injectable drugs and the risks associated with them; legal/financial problems; risky sex while intoxicated, subsequent repentance reason.
Investigate the reasons one has to use drugs.
Investigate the dependence by asking about the development of tolerance, withdrawal symptoms, use of larger amounts or for periods longer than intended, continuation of use despite problems, difficulty to stop or reduce use, and drug cravings.
Ask about the needs of habitation (housing) and employment.
Ask if the patient is prepared to stop using the drug.
Ask about the menstrual cycle and inform women that the use of drugs can interfere with the menstrual cycle, sometimes giving the false impression that she could not get pregnant.
General management
Advise the person to completely stop using the drug and signal intention to help in this regard.
Clearly inform the patient about the evaluation of results from drug use and explain the link between the level of use, their health problems, and short and long term risks of continuing using at the same level.
Discuss the best ways to reduce or stop using drugs, if one is willing.
Provide very clearly, recommendations for stopping the harmful use of substances and willingness to help the patient accordingly.
Provide information and support to patients, their caregivers, and their families.
Reassess frequently if pregnant or breastfeeding women.
Advise the pregnant to stop using any drugs and support accordingly.
Examine newborns of drug users to verify presence or absence of abstinence symptoms (known as syndrome of neonatal abstinence).
Advise and support mothers who breastfeed to not use any drugs.
Advise and support mothers with substance use disorders to only breastfeed their infants during at least the first six months, unless contraindicated.
Provide, if available, social supporting services to mothers with harmful use of drugs and small children, including additional post-natal visits, parent training, and child care during consultations.
Refer patients to self-help groups and therapeutic or rehabilitation hostels.

Source: Modified from Medical General Guidelines for the Integral Assistance To Crack. Federal Council of Medicine. 2011.

- **initial clinical evaluation –** the aim is to verify whether the patient presents a disorder that poses an imminent risk of death. Therefore, the quick and rigorous physical examination is essential to assess the following situations:[17]
- **respiratory conditions –** life-threatening disturbances that require immediate attention including airway obstruction, apnea, severe bradypnea or tachypnea, pulmonary edema, and acute respiratory failure;
- **circulatory conditions –** significant alterations in blood pressure or heart rate, ventricular arrhythmias, congestive heart failure, shock and cardiac arrest;
- **neurological conditions –** convulsions, increased intracranial pressure, coma, fixed and dilated pupils, and severe psychomotor agitation.

When conditions permit, the assessment may be extended up to include other data such as skin and appendages, temperature, hydration status, etc.

- **stabilization –** consists in the implementation of measures to correct the disorders that represent an imminent risk of death and keep the patient under appropriate conditions to establish the final diagnosis and subsequent specific treatment.

From that point, it is expected that the diagnosis or strong suspicion of cocaine intoxication has been reached based on anamnesis, physical examination, or reports from escorts or rescuers.

Specific conduct

- **airways and breathing**[14] **–** supplemental oxygen should be administered if necessary (subjective

medical evaluation of breathing or capillary oxygen saturation is less than 95%).

Succinylcholine should not be administered if rapid sequence intubation is required. The plasma cholinesterase metabolizes succinylcholine and cocaine. Therefore, the co-administration can prolong the half-life of both drugs by increasing the half-life of cocaine, effects of intoxication, and duration of the neuromuscular blockade. Succinylcholine may aggravate hyperkalemia and cause life-threatening arrhythmias due to rhabdomyolysis and hyperthermia. A non-depolarizing neuromuscular blocker, such as rocuronium, should be used if the blockade is indicated. The anesthetic induction agents that are accepted for use in patients intoxicated with cocaine are: benzodiazepines, etomidate, and propofol.

- **cardiovascular complications**[14] – cardiac stimulation caused by cocaine is mediated by central route, i.e., via the sympathetic nervous system, therefore, the sedation with benzodiazepines with appropriate dosage and appropriate route of administration is generally sufficient to alleviate the cardiovascular symptoms. Diazepam should be administered at an initial dose of 10 mg intravenously, followed by 5 to 10 mg intravenously every three to five minutes. Phentolamine can be administered, an alpha-adrenergic antagonist, to counteract the effects caused by norepinephrine release when there is symptomatic and refractory hypertension induced by cocaine. It should be administered as an intravenous bolus. The usual dose is 5 to 10 mg every five to 15 minutes, depending on patient's response.

Beta-blockers should not be used in the treatment of cardiovascular complication induced by cocaine because they can cause deviation and concentration of the adrenergic stimulation to alpha-receptors and cause coronary vasoconstriction and ischemia in the distal vascular beds of different organs. In the rare cases in which beta-blockers need to be used, their administration should be preceded by an infusion of phentolamine to prevent excessive alpha stimulation.

Hypertensive emergencies can be seen in cases of cocaine intoxication, and the approach is the same as in any hypertensive crisis, that is, reduction of the systemic diastolic pressure to 100 to 105 mmHg in two to six hours after initiation of treatment, however, the initial reduction in systemic mean arterial pressure should not exceed 25% of the blood pressure before treatment.

Hypertension induced by cocaine should not be treated promptly, as it resolves after the metabolization of cocaine, which happens in the first hours of hospitalization. Treatment with benzodiazepines and phentolamine, as described, is sufficient in most cases.

The toxicity caused by cocaine, in general, results in hypertension, however, intoxication by a large dose of cocaine can result in systemic hypotension due to the blockade of transmembrane sodium channels. Hypotension should be treated with an infusion of 0.9% NaCl intravenously. Direct vasoconstricting agents, such as norepinephrine and phenylephrine, can be used by titrating the dosage according to the effect if the systemic arterial hypotension persists after rapid infusion of 2 to 3 L of 0.9% NaCl. In such cases, an electrocardiogram should be performed, and, with the widening of the QRS complex (sodium channels blockade signal), hypertonic sodium bicarbonate, 1 to 2 mEq/kg in bolus by caliber venous access should be administered. The response to treatment should be evaluated with a new electrocardiogram after infusion of sodium bicarbonate. Narrowing or normalization of QRS is expected.

- **psychomotor agitation**[14] – agitated patients are sedated with benzodiazepines as needed after confirmation that they are not hypoglycemic or hypoxic. Diazepam administration is suggested at an initial dose of 10 mg intravenously, followed by 5 to 10 mg intravenously every three to five minutes until the agitation is controlled. The patient should be monitored for respiratory depression and hypotension. Intramuscular lorazepam (not available in Brazil) can be used if the venous access is unavailable; however, the peak effect is delayed (10-20 minutes).

Patients with severe hyperthermia should be cooled rapidly, within 30 minutes or less, through immersion in cold water, the quickest method. The cooling by evaporation of water spray might be enough when hyperthermia is present.

- **specific thoracic pain syndrome**[14] – chest pain associated with cocaine use represents 40% of all consultations involving cocaine in emergency services in the United States. Approximately 6% of patients presenting chest pain associated with cocaine use have elevated myocardial necrosis markers.

Despite that acute coronary syndromes are important concerns, other differential diagnoses that may present chest pain associated with cocaine in

emergency situations should not be excluded, such as pneumothorax and crack lung.

The chest pain associated with the use of cocaine[18] is usually described as retrosternal (76%), with dyspnea (62%), of tightness or pressure (55%), and with diaphoresis (48%).

The evaluation of patients with cocaine-associated chest pain includes electrocardiography, chest radiography, and myocardial necrosis markers. The electrocardiographic has limited accuracy[19,20] in the evaluation of chest pain associated with cocaine use. The absence of electrocardiographic evidence of ischemia does not always correlate with the clinical frame or markers of myocardial necrosis. The sensitivity and specificity of the electrocardiogram are approximately 36% and 90%, respectively.[19,20]

The initial conduct in front of chest pain associated with cocaine use includes the administration of oxygen and reduction in sympathetic activity with the use of intravenous benzodiazepines. Benzodiazepines should be administered to anxious patients, agitated, with hypertension or tachycardia, in the form of 5 mg intravenously diazepam every three to five minutes or 1 mg Lorazepam every five to 10 minutes to achieve the desired sedation. Nitroglycerin should be administered to patients with chest pain associated with the use of cocaine and hypertension as follows: 0.4 mg sublingual every five minutes as needed for up to three doses. If additional administration of nitroglycerin is required, the intravenous infusion can be started with the dose titrated as desired.

There is no consensus regarding the best treatment regimen using benzodiazepines and nitrates to control chest pain associated with the use of cocaine; it is possible[21] that faster resolution of pain is achieved when combining the two agents.

Beta-blockers are contraindicated in patients who have used cocaine in the past 24 hours. Phentolamine, an alpha-adrenergic antagonist, can be used to reduce cocaine-induced coronary vasoconstriction. Phentolamine is given as an intravenous bolus at 1.0 to 2.5 mg every five to 15 minutes, as required. In a prospective observational study[22], in patients with no history of drug abuse, cocaine was intranasally administered at 2 mg/kg while performing a coronary catheterization. Cocaine increased the cardiac output, blood pressure, and coronary vascular resistance, and reduced the light of coronary arteries by 13%. The administration of phentolamine returned the vascular diameter to normal.

- **crack lung specific syndrome**[14] – Chest X-ray should be performed in all crack users. Eosinophilia can be observed in crack users, which may be associated with some immunological mechanism. Pulmonary alterations must be accompanied by special vigilance with regard to oxygenation, ventilation, and symptomatic care. The involvement of airways requires tracheal intubation (succinylcholine should not be used in rapid succession).
- **pediatric considerations**[14] – children may require treatment when presenting signs and symptoms of cocaine intoxication or complications from the exposure to cocaine when living with drug dependent adults. Unintentional exposure to cocaine can occur via inhalation of free base vapors (crack) or ingestion.

Passive exposure to cocaine is manifested by respiratory symptoms in children with or without fever.[14] Increased incidence of focal or generalized seizures my still occur in children under eight years old. Clinical findings in older children are similar to those in adults.

Discharge criteria [14]

Patients with symptoms of acute cocaine intoxication can be discharged after six to eight hours of returning to normality, with an appropriate referral for treatment of addiction.

The patient with chest pain should be observed for eight to 12 hours until at least two dosages of myocardial necrosis markers and serial electrocardiograms are conducted. The discharge is possible in the absence of pain and a normal electrocardiogram result.

Hospitalization should be pursued when the patient presents psychomotor agitation, hyperthermia, or other neurological complications from cocaine toxicity for monitoring and controlling sequels. The patient can be discharged after six to eight hours of observation and when symptoms are completely resolved, and he/she is awake, alert, walking without difficulty, and the clinical reassessment shows no findings to justify hospitalization.

CONCLUSION

The patient with severe complications of cocaine use should be admitted to the hospital or referred to another hospital with proper complexity.

The use of drugs, particularly cocaine or its derivatives (such as crack), is an aggravating factor in any patient admitted to emergency care, either by the inherent toxicity of the drug, or drug interactions and therapeutic peculiarities that the user and his clinical condition demand.

Treatment of acute intoxication and its complications should be instituted properly by a qualified professional in order to avoid further increase in mortality resulting from drug use.

Additional resources - in order to obtain emergency information for toxicologist doctors call the Toxicology Center at the João XXIII Hospital in Belo Horizonte – Phone: 0800 722 6100 or visit the International List of Poison Centers in the World Health Organization[14] site: www.who.int/gho/phe/chemical_safety/poisons_centres/en/index.html.

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