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SEVERE METHEMOGLOBINEMIA AND HEMOLYTIC ANEMIA AFTER INGESTION OF BENZOCAINE-COATED COCAINE PACKETS: A CASE REPORT

META-HEMOGLOBINEMIA GRAVE E ANEMIA HEMOLÍTICA APÓS INGESTÃO DE INVÓLUCROS DE COCAÍNA REVESTIDOS COM BENZOCAÍNA: RELATO DE CASO

METAHEMOGLOBINEMIA GRAVE Y ANEMIA HEMOLÍTICA TRAS LA INGESTA DE PAQUETES DE COCAÍNA RECUBIERTOS CON BENZOCAÍNA: REPORTE DE CASO

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ABSTRACT

Introduction: Acquired methemoglobinemia is a potentially fatal condition characterized by hemoglobin oxidation and consequent impairment of oxygen transport. Benzocaine is a local anesthetic with well-recognized oxidative potential, usually associated with methemoglobinemia after topical mucosal exposure, although unusual routes of exposure may also lead to severe and initially difficult-to-recognize presentations.

Objective: To report a case of severe methemoglobinemia and hemolytic anemia after intentional ingestion of multiple benzocaine-coated cocaine packets in the context of internal drug transport.

Case report: A previously healthy 17-year-old male was brought to the emergency department after ingesting multiple benzocaine-coated cocaine packets. He presented with pallor, marked jaundice, peripheral cyanosis, and peripheral oxygen saturation between 70% and 75%, despite supplemental oxygen and preserved ventilation. Arterial blood gas analysis showed elevated arterial oxygen partial pressure, whereas co-oximetry confirmed a methemoglobin level of 43.5%. The patient received two intravenous doses of methylene blue associated with ascorbic acid, with an initial decrease in methemoglobin levels; however, he developed laboratory rebound and progressive hemolytic anemia, requiring packed red blood cell transfusion. Abdominal computed tomography showed multiple intraluminal foreign bodies in the colon, sigmoid colon, and rectum, with no signs of perforation, pneumoperitoneum, or obstruction, and conservative management was adopted with progressive elimination of the packets.

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Conclusion: Methemoglobinemia should be considered in patients with cyanosis, oxygen-refractory desaturation, and preserved arterial oxygen partial pressure. In cases involving ingestion of packets containing illicit drugs, adulterants or coating substances such as benzocaine may act as persistent sources of oxidative toxicity, favoring methemoglobinemia rebound and hemolysis.

Keywords: Methemoglobinemia. Benzocaine. Hemolytic Anemia. Methylene Blue. Cocaine. Case Report.

RESUMO

Introdução: A meta-hemoglobinemia adquirida é uma condição potencialmente fatal caracterizada pela oxidação da hemoglobina e consequente comprometimento do transporte de oxigênio. A benzocaína é um anestésico local com reconhecido potencial oxidativo, geralmente associado à meta-hemoglobinemia após exposição tópica em mucosas, embora vias incomuns de exposição também possam levar a apresentações graves e inicialmente difíceis de reconhecer.

Objetivo: Relatar um caso de meta-hemoglobinemia grave e anemia hemolítica após ingestão intencional de múltiplos invólucros de cocaína revestidos com benzocaína no contexto de transporte interno de drogas.

Relato de caso: Um paciente do sexo masculino, 17 anos, previamente hígido, foi encaminhado ao serviço de emergência após ingerir múltiplos invólucros de cocaína revestidos com benzocaína. Apresentava palidez, icterícia acentuada, cianose periférica e saturação periférica de oxigênio entre 70% e 75%, apesar de oxigenoterapia suplementar e ventilação preservada. A gasometria arterial demonstrou pressão parcial arterial de oxigênio elevada, enquanto a co-oximetria confirmou nível de meta-hemoglobina de 43,5%. O paciente recebeu duas doses intravenosas de azul de metileno associadas ao ácido ascórbico, com redução inicial dos níveis de meta-hemoglobina; entretanto, evoluiu com rebote laboratorial e anemia hemolítica progressiva, necessitando transfusão de concentrado de hemácias. A tomografia computadorizada abdominal evidenciou múltiplos corpos estranhos intraluminais no cólon, sigmoide e reto, sem sinais de perfuração, pneumoperitônio ou obstrução, sendo adotado manejo conservador com eliminação progressiva dos invólucros.

Conclusão: A meta-hemoglobinemia deve ser considerada em pacientes com cianose, dessaturação refratária ao oxigênio e pressão parcial arterial de oxigênio preservada. Em casos envolvendo ingestão de invólucros contendo drogas ilícitas, adulterantes ou substâncias de revestimento, como a benzocaína, podem atuar como fontes persistentes de toxicidade oxidativa, favorecendo rebote de meta-hemoglobinemia e hemólise.

Palavras-chave: Meta-hemoglobinemia. Benzocaína. Anemia Hemolítica. Azul de Metileno. Cocaína. Relato de Caso.

RESUMEN

Introducción: La metahemoglobinemia adquirida es una condición potencialmente fatal caracterizada por la oxidación de la hemoglobina y el consecuente deterioro del transporte de oxígeno. La benzocaína es un anestésico local con reconocido potencial oxidativo, generalmente asociado a metahemoglobinemia tras exposición tópica en mucosas, aunque vías inusuales de exposición también pueden conducir a presentaciones graves y difíciles de reconocer inicialmente.

Objetivo: Reportar un caso de metahemoglobinemia grave y anemia hemolítica después de la ingestión intencional de múltiples paquetes de cocaína recubiertos con benzocaína en el contexto de transporte interno de drogas.

Reporte de caso: Un paciente masculino de 17 años, previamente sano, fue llevado al servicio de urgencias tras ingerir múltiples paquetes de cocaína recubiertos con benzocaína. Presentaba palidez, ictericia marcada, cianosis periférica y saturación periférica de oxígeno entre 70% y 75%, a pesar de oxigenoterapia suplementaria y ventilación preservada. La gasometría arterial mostró presión parcial arterial de oxígeno elevada, mientras que la cooximetría confirmó un nivel de metahemoglobina de 43,5%. El paciente recibió dos dosis intravenosas de azul de metileno asociadas con ácido ascórbico, con disminución inicial de los niveles de metahemoglobina; sin embargo, evolucionó con rebote laboratorial y anemia hemolítica progresiva, requiriendo transfusión de concentrado de eritrocitos. La tomografía computarizada abdominal mostró múltiples cuerpos extraños intraluminales en el colon, sigmoides y recto, sin signos de perforación, neumoperitoneo u obstrucción, adoptándose manejo conservador con eliminación progresiva de los paquetes.

Conclusión: La metahemoglobinemia debe considerarse en pacientes con cianosis, desaturación refractaria al oxígeno y presión parcial arterial de oxígeno preservada. En casos que involucran ingestión de paquetes con drogas ilícitas, adulterantes o sustancias de recubrimiento como la benzocaína pueden actuar como fuentes persistentes de toxicidad oxidativa, favoreciendo el rebote de la metahemoglobinemia y la hemólisis.

Palabras clave: Metahemoglobinemia. Benzocaína. Anemia Hemolítica. Azul de Metileno. Cocaína. Reporte de Caso.

1 INTRODUCTION

Methemoglobinemia is a potentially severe hematologic condition characterized by oxidation of the iron within hemoglobin from the ferrous to the ferric state, forming methemoglobin, a fraction unable to transport oxygen adequately.[1] This alteration reduces the functional availability of hemoglobin and impairs oxygen delivery to tissues, potentially causing functional hypoxia even when ventilation and arterial oxygen partial pressure are preserved.[1] Under physiological conditions, small amounts of methemoglobin are continuously produced, but erythrocyte reducing systems maintain low levels through compensatory enzymatic mechanisms.[1]

Acquired forms of methemoglobinemia are more frequent than congenital forms and usually result from exposure to drugs, local anesthetics, or chemical substances with oxidative potential.[2] Reported agents include benzocaine, lidocaine, prilocaine, dapsone, nitrates, nitrites, sulfonamides, and aniline compounds.[2] Clinical severity depends on the methemoglobin concentration, the rate of onset, the presence of anemia, the patient's cardiopulmonary reserve, and the persistence of exposure to the oxidizing agent.[2]

Benzocaine is an ester-type local anesthetic recognized as a relevant cause of acquired methemoglobinemia, especially when used on mucosal surfaces or in high doses.[3] Its metabolism may generate oxidizing compounds capable of overwhelming erythrocyte reducing capacity and converting functional hemoglobin into methemoglobin.[3] Although most reports involve topical application during endoscopic, dental, or otorhinolaryngological procedures, unusual exposures may also result in severe presentations that are initially difficult to diagnose.[3]

The clinical diagnosis of methemoglobinemia requires attention to the dissociation between peripheral oxygen saturation and arterial oxygen partial pressure.[4] Patients may present with cyanosis and persistently low peripheral oxygen saturation, with little or no response to supplemental oxygen, despite arterial blood gas analysis showing normal or elevated arterial oxygen partial pressure.[4] This finding, often referred to as the "saturation gap," is one of the main warning signs for dyshemoglobinemias and should prompt co-oximetry.[4]

Co-oximetry is essential for diagnostic confirmation because it directly quantifies the methemoglobin fraction and differentiates it from other causes of hypoxia or low peripheral oxygen saturation.[5] Conventional pulse oximetry may be misleading in this context, as it does not adequately discriminate between different forms of dysfunctional hemoglobin.[5] Therefore, isolated interpretation of peripheral oxygen saturation may lead to diagnostic delay, inappropriate escalation of ventilatory support, and undertreatment of oxidative toxicity.[5]

Treatment of symptomatic or severe methemoglobinemia is based on interruption of exposure to the oxidizing agent, clinical support, and administration of methylene blue when indicated.[6] Methylene blue promotes methemoglobin reduction through a nicotinamide adenine dinucleotide phosphate-dependent pathway, progressively restoring the functional capacity of hemoglobin.[6] However, its use requires caution in patients with glucose-6-phosphate dehydrogenase deficiency and in situations of persistent oxidative exposure, in which partial response, rebound, or associated hemolysis may occur.[6]

The association between methemoglobinemia and hemolysis increases clinical severity because it combines qualitative hemoglobin dysfunction with quantitative reduction of circulating erythrocyte mass.[7] Hemolysis may result from oxidative stress itself, chemical-induced erythrocyte instability, or, in some cases, adverse effects related to treatment.[7] Findings such as hemoglobin decline, hyperbilirubinemia, elevated lactate dehydrogenase, polychromasia, spherocytes, and schistocytes should prompt investigation and serial monitoring.[7]

Ingestion of packets containing illicit drugs in the context of internal transport is a high-risk clinical situation due to the possibility of systemic intoxication, intestinal obstruction, perforation, and need for surgical intervention.[8] Computed tomography is a fundamental examination for locating intraluminal foreign bodies, assessing their distribution throughout the gastrointestinal tract, and identifying signs of abdominal complications.[8] In clinically stable patients without signs of perforation, obstruction, or packet rupture-related intoxication, conservative management with in-hospital observation and facilitated intestinal elimination may be appropriate.[8]

The case described is clinically relevant because it combines severe methemoglobinemia, significant hemolysis, and ingestion of multiple packets containing benzocaine-coated cocaine.[9] In this setting, toxicological risk is not limited to the main illicit drug, since substances used as adulterants, diluents, or coatings may produce their own potentially fatal manifestations.[9] Persistence of packets in the gastrointestinal tract may function as a continuous source of oxidative exposure, favoring incomplete response to initial treatment and rebound methemoglobinemia.[9]

2 OBJECTIVES

The main objective of this report is to describe a case of severe acquired methemoglobinemia associated with intentional ingestion of multiple packets containing benzocaine-coated cocaine, with a clinical course complicated by relevant hemolysis, marked hyperbilirubinemia, transfusion requirement, and admission to the intensive care unit.

As secondary objectives, this report aims to discuss the main clinical elements that allowed recognition of methemoglobinemia, especially the dissociation between peripheral oxygen saturation and arterial oxygen partial pressure, as well as the importance of co-oximetry for diagnostic confirmation. It also aims to analyze the patient's laboratory course, response to methylene blue, methemoglobinemia rebound, association with hemolytic anemia, and the role of computed tomography in identifying intraluminal foreign bodies. Finally, the report seeks to highlight the need for a multidisciplinary approach in cases of oxidizing substance toxicity associated with internal transport of illicit drugs.

3 METHODS

This is a clinical case report based on retrospective analysis of data obtained during hospital care in an emergency department, followed by management in an intensive care unit and internal medicine ward. Clinical data, medical chart evolution, laboratory tests, arterial blood gas analyses, co-oximetry results, transfusion records, and abdominal computed tomography images were reviewed.

A narrative literature review was also performed to contextualize the case, focusing on acquired methemoglobinemia, benzocaine toxicity, therapeutic use of methylene blue, hemolysis associated with oxidative stress, glucose-6-phosphate dehydrogenase deficiency, packet ingestion for internal drug transport, and the role of computed tomography in these patients.

Information was organized chronologically, prioritizing clinical presentation, diagnostic reasoning, therapeutic approaches, radiological findings, laboratory course, and hospital outcome. Identifiable data were omitted to preserve patient confidentiality, including name, medical record number, specific dates, exact site of care, and any visual element that could allow individual recognition.

4 CASE REPORT

A 17-year-old Bolivian male, previously healthy and with no known relevant medical history, was brought to the emergency department by law enforcement officers after intentional ingestion of multiple packets containing benzocaine-coated cocaine in the context of internal illicit drug transport. According to information obtained on admission, ingestion had occurred approximately three days before hospital arrival, and the patient had already spontaneously eliminated a substantial portion of the ingested material before the initial medical evaluation. The patient was conscious, alert, oriented, and communicative, in fair general condition, without respiratory complaints proportional to the severity of the observed

desaturation. However, marked mucocutaneous pallor, pronounced jaundice, and evident peripheral cyanosis were noted.

On initial assessment, the patient was eupneic, without accessory muscle use, retractions, or clinical signs of acute respiratory failure. Nevertheless, peripheral oxygen saturation remained persistently low, ranging from 70% to 75%, even after administration of supplemental oxygen at 5 L/min through a nasal cannula. Lung auscultation showed no adventitious sounds, ventilatory asymmetry, or signs of bronchospasm. Cardiovascular evaluation showed no hemodynamic instability, signs of shock, congestion, or clinical findings suggesting a primary cardiopulmonary cause for the apparent hypoxemia. The abdominal examination was unremarkable, with no significant distension, tenderness, signs of peritoneal irritation, or clinical evidence of intestinal obstruction.

Given the marked discrepancy between low peripheral oxygen saturation, preserved ventilation, absence of relevant cardiopulmonary findings, and poor response to oxygen therapy, acquired methemoglobinemia was suspected. The simultaneous presence of peripheral cyanosis, jaundice, and pallor reinforced suspicion of a hematologic or toxicological process, especially in a patient with recent exposure to material possibly coated with an oxidizing substance. Point-of-care ultrasonography of the lungs, heart, and abdomen showed no significant abnormalities that could explain the presentation. Arterial blood gas analysis with co-oximetry, complete blood count, renal function, electrolytes, liver enzymes, bilirubin levels, hemolysis markers, and complementary tests to assess systemic severity were then requested.

Initial arterial blood gas analysis showed pH 7.60, carbon dioxide partial pressure of 27 mmHg, oxygen partial pressure of 122 mmHg, and bicarbonate of 26.5 mmol/L. Co-oximetry confirmed severe methemoglobinemia, with a methemoglobin concentration of 43.5%. This finding confirmed the mechanism of refractory desaturation, since arterial oxygen partial pressure was preserved while peripheral oximetry remained critically low. Given laboratory confirmation and clinical severity, intravenous therapy with 200 mg of 2% methylene blue was immediately initiated.

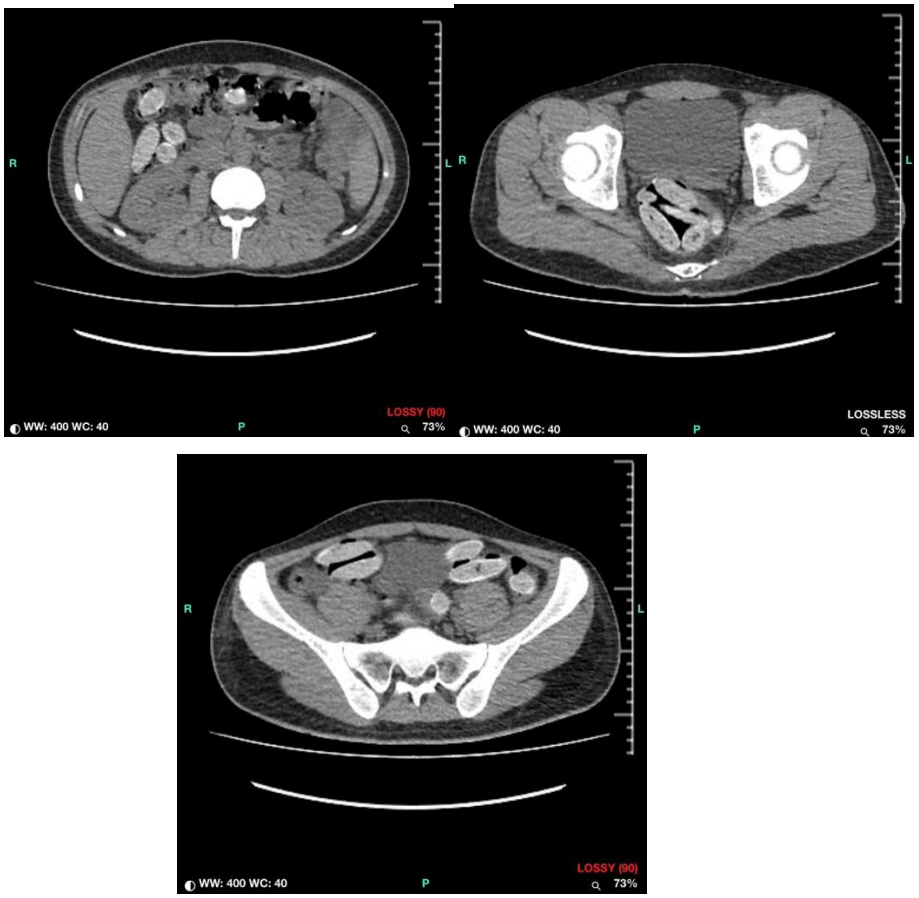
One hour after the first dose, peripheral oxygen saturation remained below 80% despite oxygen support. Considering the persistence of functional hypoxemia and the severity of initial methemoglobinemia, a second intravenous dose of 200 mg of methylene blue was administered. A 500 mg dose of ascorbic acid was also added, aiming to support control of oxidative stress and reduce the risk of progression of erythrocyte injury. One hour after the second dose, repeat arterial blood gas analysis with co-oximetry showed pH 7.48, carbon dioxide partial pressure of 38 mmHg, oxygen partial pressure of 121 mmHg, and

reduction of methemoglobin to 14.4%. The initial laboratory response was considered favorable, although the patient remained under close observation because of the risk of recurrence while foreign bodies remained in the gastrointestinal tract.

Initial laboratory tests showed hemoglobin of 10.8 g/dL and hematocrit of 30%, already suggesting anemia on admission. Hematologic analysis showed mild macrocytosis, polychromasia, and numerous spherocytes and schistocytes, compatible with active hemolysis. The white blood cell count showed leukocytosis of 17,000/µL, without left shift. Electrolytes and renal function were preserved, with no initial evidence of acute kidney injury. Laboratory testing showed elevated transaminases and lactate dehydrogenase, as well as marked hyperbilirubinemia, with total bilirubin of 20.33 mg/dL, direct bilirubin of 13.85 mg/dL, and indirect bilirubin of 6.48 mg/dL. Serologies for hepatitis C virus and hepatitis B surface antigen were negative.

Figure 1

Abdominal computed tomography showing multiple intraluminal foreign bodies with a packet-like appearance, distributed mainly in colonic segments, including the colon, sigmoid colon, and rectum, without evident signs of perforation, pneumoperitoneum, or intestinal obstruction



Abdominal computed tomography was performed to locate the remaining foreign bodies and evaluate possible complications related to packet ingestion. The examination identified multiple intraluminal foreign bodies in the colon, sigmoid colon, and rectum, with no signs of perforation, pneumoperitoneum, or intestinal obstruction. The general surgery team evaluated the patient and, given clinical stability, absence of acute abdomen, and distal location of the packets, recommended conservative management. The patient was kept fasting and measures to accelerate intestinal elimination were initiated, including oral mannitol, bisacodyl, and lactulose. This approach was associated with continuous clinical monitoring, serial laboratory follow-up, and surveillance for signs of rupture, systemic intoxication, obstruction, or need for operative management.

During observation in the emergency department while awaiting an intensive care unit bed, the patient remained on high-flow oxygen therapy through a Hudson mask. Despite initial improvement in methemoglobin levels after methylene blue, serial tests showed worsening hemolytic anemia, with hemoglobin decreasing to 8.6 g/dL. Rebound methemoglobinemia was also documented, with a later increase in methemoglobin to 34%. This pattern was interpreted as a probable consequence of continuous benzocaine absorption from the remaining packets in the gastrointestinal tract. In view of progressive anemia in the setting of active hemolysis and functional hemoglobin impairment, two units of packed red blood cells were transfused.

Figure 2

Packets eliminated by the patient during conservative management, compatible with foreign bodies previously ingested for internal transport, to be used only after complete anonymization and institutional authorization

After transfer to the intensive care unit, the patient was kept fasting, with continued laxative therapy and high-flow oxygen support through a Hudson mask. Light sedation was used for comfort and improved tolerance of supportive measures. Over the following two days, numerous intact packets were eliminated through the feces. After each elimination, the patient was clinically reassessed for abdominal pain, distension, vomiting, altered level of consciousness, worsening cyanosis, or signs of systemic intoxication. No signs of perforation, intestinal obstruction, or acute packet rupture were documented during hospitalization.

Daily laboratory monitoring showed progressive improvement after reduction of the intestinal exposure burden. Cyanosis gradually decreased, mucocutaneous color improved, hemoglobin levels stabilized, methemoglobin levels progressively declined, and bilirubin values decreased. Glucose-6-phosphate dehydrogenase testing was normal, making enzymatic deficiency less likely as a primary factor for severe hemolysis or inadequate response to methylene blue. The clinical course reinforced the hypothesis that hemolysis and rebound methemoglobinemia were mainly related to persistent oxidative exposure associated with benzocaine present in the ingested packets.

After four days in the intensive care unit, the patient showed significant clinical improvement, without relevant cyanosis, respiratory failure, hemodynamic instability, or significant abdominal complaints. With progressive elimination of the packets, hematologic stabilization, and improvement of laboratory parameters, he was transferred to the internal medicine ward. He remained under observation for two additional days, maintaining favorable evolution without new complications. He was discharged after complete clinical and laboratory recovery, without need for surgical intervention and without further documented adverse events.

5 LITERATURE REVIEW

Acquired methemoglobinemia remains a rare but highly relevant condition in emergency settings because its presentation may mimic severe hypoxemia without corresponding to primary ventilatory failure.[10] Early recognition depends on clinical suspicion in the presence of persistent cyanosis, low peripheral oxygen saturation, and preserved or elevated arterial oxygen partial pressure.[10] In retrospective series, oxidizing drugs and local anesthetics are among the main identifiable causes, although the diversity of involved agents makes rigid diagnostic protocols difficult.[10]

The central pathophysiology involves conversion of heme iron from the ferrous to the ferric state, preventing adequate oxygen binding and reducing effective tissue delivery.[11] In addition, the presence of methemoglobin increases the affinity of remaining hemoglobin

chains for oxygen, impairing its release into peripheral tissues.[11] For this reason, patients may show clinical signs of functional hypoxia even when the partial pressure of oxygen dissolved in plasma appears adequate.[11]

Benzocaine is one of the local anesthetics most frequently associated with acquired methemoglobinemia, mainly because of its ability to generate oxidizing metabolites.[12] Clinical reports describe methemoglobinemia after topical use on mucosal surfaces, anesthetic sprays, lozenges, oral preparations, and inadvertent or excessive exposure.[12] The relevance of the present case lies in the fact that exposure did not occur through usual therapeutic use, but through prolonged gastrointestinal contact with packets coated with benzocaine.[12]

The literature also describes cases of methemoglobinemia related to cocaine adulterated or mixed with benzocaine, reinforcing the importance of considering contaminants and adulterants during toxicological evaluation.[13] Benzocaine may be used as an adulterant or associated agent in illicit preparations because of its local anesthetic properties and physical appearance compatible with powdered substances.[13] Therefore, in patients exposed to cocaine or drug packets, methemoglobinemia may result not only from the main drug but also from substances added during preparation, dilution, or coating.[13]

The so-called “saturation gap” is one of the most important semiological elements in the evaluation of these patients.[14] Conventional pulse oximetry tends to show persistently reduced values, often in intermediate ranges, even after increased oxygen delivery.[14] Arterial blood gas analysis, on the other hand, may demonstrate normal or elevated oxygen partial pressure, creating a dissociation that should direct diagnostic reasoning toward dyshemoglobinemias.[14]

Co-oximetry is indispensable for diagnostic confirmation because it directly quantifies different hemoglobin fractions, including oxyhemoglobin, carboxyhemoglobin, and methemoglobin.[15] Without co-oximetry, interpretation of arterial blood gas results may be misleading, because oxygen partial pressure does not represent the functional capacity of hemoglobin to transport oxygen.[15] This point is especially relevant in emergencies, where low saturation may induce aggressive respiratory interventions before the true cause of the presentation is identified.[15]

Methylene blue is considered the main pharmacological treatment for symptomatic or severe methemoglobinemia.[16] Its mechanism depends on reduction of methylene blue to leucomethylene blue, which acts as an electron donor and favors conversion of methemoglobin back into functional hemoglobin.[16] The response is usually rapid when intoxication is not sustained by persistent exposure and when there is no significant metabolic limitation of the reducing pathway.[16]

Despite its efficacy, methylene blue requires caution in certain clinical circumstances.[17] Patients with glucose-6-phosphate dehydrogenase deficiency may have lower production of nicotinamide adenine dinucleotide phosphate, impairing therapeutic response and increasing the risk of hemolysis.[17] In addition, repeated doses or continuous oxidative exposure may contribute to erythrocyte instability, especially when the patient already has anemia or laboratory signs of hemolysis.[17]

Hemolysis associated with methemoglobinemia is particularly important because it reduces available erythrocyte mass while the remaining hemoglobin is functionally impaired.[18] In reports involving exposure to local anesthetics, hemolysis may occur due to direct oxidative stress, individual susceptibility, enzymatic deficiency, or the combined effect of intoxication and treatment.[18] In the reported case, the presence of spherocytes, schistocytes, polychromasia, elevated lactate dehydrogenase, hyperbilirubinemia, and progressive hemoglobin decline supported the diagnosis of clinically relevant hemolytic anemia.[18]

Glucose-6-phosphate dehydrogenase testing is useful in the interpretation of severe cases, especially when hemolysis is present or repeated methylene blue doses are required.[19] A normal result does not completely exclude hemolysis due to intense oxidative stress, but it reduces the likelihood of enzymatic deficiency as the primary cause of hematologic deterioration.[19] In situations where deficiency is known or strongly suspected, alternatives such as ascorbic acid, transfusional support, and intensive supportive care may become more important.[19]

Ascorbic acid may act as a complementary reducing agent, although its action is generally slower and less predictable than that of methylene blue in severe acute presentations.[20] Its use is usually considered in selected cases, especially when there is a relative contraindication to methylene blue, risk of hemolysis, or need for additional antioxidant support.[20] In the present case, its administration associated with the second treatment cycle had a plausible pathophysiological rationale, considering the severity of oxidative exposure and concomitant hemolytic evolution.[20]

Rebound methemoglobinemia is a coherent finding when exposure to the oxidizing agent remains active after the first therapeutic intervention.[21] In exposures involving extended-release drugs, compounds with longer half-lives, or persistent intraluminal sources, the initial decrease in methemoglobin may be followed by renewed laboratory elevation.[21] In the reported case, persistence of multiple packets within the gastrointestinal tract probably functioned as a continuous benzocaine reservoir, explaining recurrence after the initial response to methylene blue.[21]

Internal drug transport through packet ingestion, often described as “body packing,” represents a high-risk condition due to the possibility of rupture, systemic intoxication, intestinal obstruction, or perforation.[22] Clinical evaluation should consider the type of drug involved, the presumed number of packets, material integrity, abdominal symptoms, systemic signs of intoxication, and the possibility of surgical complications.[22] In patients without abdominal symptoms and without evidence of rupture, conservative management may be appropriate if performed in a monitored setting.[22]

Computed tomography has a central role in the evaluation of these patients because it allows identification of intraluminal foreign bodies and assessment of signs of complications.[23] Compared with plain radiographs, computed tomography offers greater sensitivity for packet localization and better characterization of obstruction, perforation, pneumoperitoneum, or other relevant abdominal abnormalities.[23] In the reported case, identification of packets in the colon, sigmoid colon, and rectum, without signs of immediate mechanical complication, supported the decision for conservative follow-up by the surgical team.[23]

The choice between conservative management and invasive approach must be individualized.[24] Signs of acute abdomen, persistent obstruction, perforation, clinical deterioration, severe systemic intoxication due to rupture, or failure of packet progression may change the strategy and justify surgical intervention.[24] In the present case, abdominal stability, distal location of the foreign bodies, and progressive elimination of the packets favored maintenance of conservative management.[24]

The most singular aspect of this report is the combination of severe hematologic toxicity and a persistent intestinal source of an oxidizing substance.[25] Unlike isolated topical benzocaine exposures, ingestion of multiple coated packets may prolong absorption, hinder definitive resolution, and require serial laboratory surveillance until complete elimination of the material.[25] Thus, the case reinforces that patients with ingestion of illicit drugs should be evaluated not only for toxicity of the main substance, but also for the effects of adulterants, diluents, and coatings capable of producing specific clinical manifestations.[25]

6 CONCLUSION

This report describes an unusual and severe case of acquired methemoglobinemia associated with intentional ingestion of multiple packets containing benzocaine-coated cocaine. The clinical presentation was marked by peripheral cyanosis, persistently low peripheral oxygen saturation, inadequate response to oxygen therapy, and preserved arterial oxygen partial pressure, configuring a highly suggestive pattern of dyshemoglobinemia.

Diagnostic confirmation by co-oximetry was essential to guide management and allowed immediate initiation of treatment with methylene blue. The initial reduction in methemoglobin demonstrated an adequate therapeutic response, but the subsequent rebound indicated probable continuous benzocaine absorption while packets remained in the gastrointestinal tract.

The clinical course was aggravated by hemolytic anemia, marked hyperbilirubinemia, and the need for packed red blood cell transfusion. These findings reinforce that severe methemoglobinemia should not be interpreted only as a functional hemoglobin disorder, but also as a possible marker of systemic oxidative injury with relevant hematologic repercussions.

Computed tomography was decisive for identifying intraluminal foreign bodies and ruling out signs of perforation, pneumoperitoneum, or intestinal obstruction. The absence of immediate abdominal complications allowed conservative management, with progressive packet elimination and complete clinical and laboratory recovery.

This case highlights the need for a multidisciplinary approach in patients who ingest packets containing illicit substances, especially when exposure to oxidizing agents such as benzocaine is present. Early recognition of the “saturation gap,” prompt co-oximetry, timely treatment of methemoglobinemia, surveillance for hemolysis, and careful radiological assessment are essential to reduce morbidity and prevent fatal outcomes.

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