Case Report

Acute Management of Cocaine-Associated Methaemoglobinaemia

Immo Weichert

Acute Medicine Unit, Royal Alexandra Hospital, Corsebar Road, Paisley PA2 9PN, UK

Correspondence should be addressed to Immo Weichert, immo.weichert.mail@gmail.com

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Methaemoglobinaemia is a potentially life-threatening complication of problem drug use. This is a case report of a 29-year-old man who presented himself cyanosed after a cocaine binge. It highlights the diagnosis and management of this condition from an acute medical perspective.

1. Case Presentation

A 29-year-old man presented himself to the emergency department after noticing that his lips and fingers had turned blue. He had been on a cocaine binge preceding his admission. He estimated that he had been taking at least 14 grams over a period of five days. He felt short of breath on moderate exertion but denied chest pain or any other systemic symptoms. He had been using cocaine regularly for at least ten years, always via the nasal passageways, never injected, and denied taking any other recreational drugs. He was not on any prescription or over-the-counter medications. His only past medical history was of alcohol dependence, though he had not drunk in excess for at least ten months. There was no significant family history.

He was alert and coherent, but slightly anxious. He was apyrexial, his noninvasive blood pressure was 135/96 mmHg, and his heart rate was 85 beats/min. He was tachypnoeic with a respiratory rate of 18/min. There was marked cyanosis of his fingers and lips, and he was, immediately upon arrival in the emergency department, commenced on high flow oxygen. His saturation measured by pulse oximetry (SO2) was 86% on 15 L of oxygen given via a nonrebreathing mask. Arterial blood gasses showed H+ 50.9 nmol/L, pCO2 5.8 kPa, pO2 72.3 kPa, lactate 0.7 mmol/L, bicarbonate 26.0 mmol/L, CO Hb 0.0%. His oxygen saturation on the blood gas (O2 Hb) was 66.6% and his methaemoglobin (Met Hb) level 32.9%. Chest X-Ray, ECG, full blood count, and routine biochemistry were unremarkable.

A diagnosis of methaemoglobinaemia was made, and he received one dose of methylene blue (1 mg/kg) intravenously over five minutes. He was transferred to the high dependency unit where he continued to receive high flow oxygen and was closely monitored.

His methaemoglobin levels, as well as his saturation, improved steadily over the next hours (Table 1). He made a full recovery and was discharged home the same evening after review by the local drug addiction liaison service with no further followup.

2. Discussion

Acquired methaemoglobinaemia is a well-recognized though still relatively rare complication of cocaine use. It is attributed to adulterants, cheaper substances that are being mixed with the pure cocaine base to increase the profits from selling the drug. Local anaesthetics are being used as they will produce a similar sensation when applied to mucosal surfaces as unadulterated cocaine [1]. Benzocaine is often implied in acquired methaemoglobinaemia, though its use as adulterant does vary between countries. Benzocaine is the most frequently found cutting agent in the UK [2] while a recent French study failed to show its significant use there [3].

Methaemoglobinaemia is caused by oxidation of the iron molecule in the heme group to the ferric state (Fe 3+). This renders it unable to carry oxygen and causes a functional anaemia. Blood normally can contain about 1% of methaemoglobin. Substances that put high oxidative stresses onto haemoglobin will increase the level above normal. Well-recognized causes for this are local anaesthetics (especially
failure should be treated after obtaining advice from the
National Poisons Information Service or similar and in a
dependency or intensive care environment.
Healthy and asymptomatic patients with low levels
of methaemoglobin may only need administration of high flow
oxygen and observation. As long as the offending agent has
been removed, Met Hb levels will return to normal within
36 hours [9]. Methylene blue (methylthioninium chloride)
is the antidote of choice if methaemoglobin levels have
reached 30% or if there is symptomatic hypoxaemia or
ischaemia [10]. It leads to the reduction of methaemoglobin
both via the NADPH-methaemoglobin reductase as well as
through its own intermediary, leucomethylene blue [11].
The initial dose is 1 mg/kg bodyweight given intravenously
over five minutes. Effects will be measurable within 30
minutes to one hour. In severe toxicity, this may have
to be repeated. Failure to respond to methylene blue
suggests G6PD deficiency where it can also cause profound
haemolysis. Repeated or high doses can lead to a para-
doxal increase in methaemoglobin [12]. Its effects during
pregnancy are unsure, and potential risks of teratotoxicity
need to be considered [13]. It can cause blue discolouration
of the urine as well as of the sclera [14]. Other rare but
serious side effects are related to its MAO-inhibitor action
[15], and it has been implied in the serotonin syndrome
[16]. The latter is a potentially life-threatening condition
carried by excessive serotonergic activity in the nervous
system. Features include mental status changes, autonomic
instability, and neuromuscular hyperactivity [17]. Patients
who have been on selective serotonin reuptake inhibitor
antidepressants (SSRIs) or clomipramine and are treated
with methylene blue should be observed for CNS e-
fects during

<table>
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<th>00:29</th>
<th>02:23</th>
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<tbody>
<tr>
<td>Met Hb in %</td>
<td>32.9</td>
<td>16.9</td>
<td>1.9</td>
<td>0.2</td>
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<tr>
<td>O₂ Hb in %</td>
<td>66.6</td>
<td>82.4</td>
<td>93.1</td>
<td>98</td>
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<tr>
<td>SO₂ in %</td>
<td>86</td>
<td>89</td>
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<tr>
<td>Time</td>
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prilocaine, benzoic acid), antibiotics (dapsone, trimethoprim,
sulfonamides), aniline dyes, and metoclopramide, as well
as nitrates. Blood that contains significant amounts of
methaemoglobin has a characteristic dark reddish-brown
colour. Above concentrations of 1.5 g/dL (Met Hb 10–15%),
this will cause cyanosis even in the absence of elevated
deoxyhaemoglobin levels. At relatively low levels this will
lead to a cyanotic but asymptomatic patient. Typically,
the cyanosis will not improve after increasing the oxygen
supply. Symptoms in otherwise healthy individuals will start
at methaemoglobin levels of 30% or more. These will be
related to oxygen deficiency in organs with high demands,
that is, the cardiovascular and central nervous system.
Patients will experience headaches, lightheadedness, anxiety,
dyspnoea, palpitations, and somnolence. Significant toxicity
will occur at methaemoglobin levels of 50% and over. This
will include cardiac arrhythmias, delirium, seizures, coma
as well as a profound metabolic acidosis. Death will occur
at concentrations above 60% [4], though in patients with
comorbidities this can occur at much lower levels.

Methaemoglobin interferes with traditional (not multi-
wavelength) pulse oximetry. In patients with relatively low
methaemoglobin levels this will give falsely low oxygen
saturations. Paradoxically, it will also lead to falsely elevated
oxygen saturations in the presence of significant concentra-
tions [5]. Arterial blood gasses will often show normal
PaO₂ levels but falsely low O₂ saturations. This difference
between the oxygen saturation from the blood gas analysis
and the saturation, as measured by the saturation probe, is
commonly referred to as the “saturation gap.” It suggests
the presence of a haemoglobin derivate that is not transporting
oxygen. In the presence of cyanosis, this is highly suggestive
of methaemoglobinemia [6]. In severe cases there will be
a significant acidosis. Lactate levels, urea, and electrolytes,
as well as a creatinine kinase and 12 lead ECG, can help
to assess the degree of tissue hypoperfusion and end-
organ damage. These investigations will also point towards
other cocaine-related toxicities. A pregnancy test should be
performed in women of childbearing age. A urine screen
for drug metabolites can help in unclear cases. Sometimes
a blood film will show Heinz bodies in the erythrocytes,
inclusions of denatured haemoglobin [7]. Good history
taking is paramount to identify the possible offending
agent and to distinguish primary from secondary causes
of methaemoglobinemia. In case of internal concealment
of cocaine (“body packers”), oral purgation and sometimes
surgical removal may be necessary [8]. Cases with severe
toxicity, decreased level of consciousness, or multiple organ
failure should be treated after obtaining advice from the

Conflict of Interests
No conflicts of interest have been declared.

References
“Benzocaine-adultered street cocaine in association with me-
themoglobinemia,” Clinical Chemistry, vol. 38, no. 4, pp. 596–
[2] UK Border Agency, “Further benzocaine seizure at Felixs-
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purity and perceived quality of street cocaine in France,”

No conflicts of interest have been declared.


