Unrecognized “crack” cocaine abuse in pregnancy

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Summary
We report a case of “crack” cocaine abuse in a pregnant patient associated with haematuria, proteinuria, haemolytic anaemia, renal impairment, thrombocytopenia and pulmonary oedema. The case illustrates the problems for clinicians where unrecognized cocaine abuse interferes with the diagnosis and management of a complicated pregnancy. In addition, we discuss the principles for the safe conduct of anaesthesia in the pregnant cocaine abuser. (Br. J. Anaesth. 1996;77:553–555)

Key words

“Crack” cocaine abuse in pregnancy is well recognized in the USA. In the UK the level of awareness to this increasingly common problem needs to be improved. This case illustrates the difficulties in differentiating the effects of cocaine abuse from the more usual complications of pregnancy. Cocaine has complex effects on many systems. It was not apparent until post-partum that the patient continued to abuse crack cocaine even while an inpatient. In retrospect the relationship between drug abuse and the course of her illness became clear.

Crack cocaine abuse increases maternal morbidity, fetal morbidity and death. It is likely that maternal crack cocaine abuse will be an increasing problem in the UK and at present the clinical effects may not be fully recognized.

Case report
A 32-yr old, gravida 5, para 3, Afro-Caribbean woman presented to her general practitioner for antenatal assessment at 10 weeks’ gestation. Her haemoglobin concentration was 10.1 g dl$^{-1}$ and the blood film showed macrocytosis. Cervical and high vaginal swabs were infected with Gonococcus and Trichomonas. Her infection was treated and she attended again at 19 weeks’ gestation when urinalysis showed moderate haematuria and mild proteinuria. Arterial pressure was 120/70 mm Hg and she had bilateral pitting oedema of the ankles. Cardiovascular and respiratory system examinations were otherwise unremarkable. Abdominal examination was consistent with a 31-week pregnancy with normal fetal heart sounds and movements. Urinalysis showed excessive blood and moderate protein. The full blood count result a week later showed haemoglobin 6.8 g dl$^{-1}$, mean cell volume 110 fl and platelets $63 \times 10^9$ litre$^{-1}$, and therefore hospital admission was arranged.

In hospital, physical examination revealed an apical ejection systolic murmur. Deep tendon reflexes were normal. Her blood film showed a reticulocyte count of 4.9%. Serum creatinine concentration was 99 mmol litre$^{-1}$, which is high for a pregnant patient. Urea and electrolyte concentrations, liver function tests, uric acid and clotting screen were within normal limits. Autoimmune profile, lupus and platelet antibodies and haemosiderin were within normal limits but haptoglobin concentrations were low at 0.2 g litre$^{-1}$. The 24-h urine volume was 2125 ml and protein 1.32 g litre$^{-1}$. Creatinine clearance was impaired at 90 ml min$^{-1}$. Free haemoglobin concentration was detected in urine. She was treated with folic acid 10 mg orally twice daily and transfused with 2 u. of packed red blood cells.

A bone marrow aspirate was performed and showed some megakaryocytes and a microangiopathic picture. Later the same day she became hypertensive with an arterial pressure of 145/95 mm Hg which decreased with bed rest alone. The patient admitted also to a history of smoking crack cocaine for the past year. At this stage the differential diagnoses being considered were pre-eclampsia, haematological illness related to renal disease or pregnancy, such as haemolytic–uraemic syndrome, or renal disease, probably secondary to drug abuse.

Additional blood transfusions were given over the next 2 days; haemoglobin concentration was then 9.3 g dl$^{-1}$. On day 4 of admission she left the ward for the afternoon and on her return she had a dry cough, left-sided pleuritic chest pain, shivering, temperature of 37.5°C, heart rate 110 beat min$^{-1}$, arterial pressure 140/80 mm Hg, ventilatory frequency 34 bpm and were pale, arterial pressure was 125/70 mm Hg and she had bilateral pitting oedema of the ankles.

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crackles on auscultation of the chest. Blood cultures were obtained, oxygen was given by face mask and antibiotic therapy with Augmentin was started. The next morning the patient had another episode of breathlessness thought to be caused by fluid overload. ECG was normal but a chest x-ray showed cardiomegaly and pulmonary oedema. Frusamide 20 mg i.v. produced improvement. However, an echocardiogram showed tachycardia, hyperdynamic left ventricle, ejection fraction of 62%, small pericardial effusion and mild tricuspid regurgitation.

There was an additional episode of breathlessness the next morning and a decision to expedite delivery was made. It was felt that she would not tolerate vaginal delivery and a Caesarean section was planned. The preoperative anaesthetic assessment noted the history of crack cocaine abuse, hepatitis B positive, high risk for HIV infection and the need for appropriate precautions. Despite the uncertainty of the diagnosis there was some comfort in the patient having a normal arterial pressure, no clinical evidence of heart failure, haemoglobin 9.7 g dl⁻¹, and normal urea and creatinine concentrations, and clotting screen. As the platelet count was 60 × 10⁹ litre⁻¹, arrangements were made for a platelet transfusion of 12 u. before surgery.

The patient was premedicated with ranitidine 50 mg i.v. and sodium citrate 0.3 mol litre⁻¹ (30 ml) orally before induction of anaesthesia. After local anaesthesia of the skin, cannulae were inserted via the right brachial artery and right internal jugular vein for monitoring of arterial pressure and central venous pressure. Her lungs were preoxygenated for 5 min and this was followed by rapid sequence i.v. induction with etomidate 20 mg, sufamethomycin 100 mg and alfentanil 1 mg, and application of cricoid pressure. Anaesthesia was maintained with 1% isoflurane and 50% nitrous oxide in oxygen. Fourteen minutes later a live male neonate, weighing 2.07 kg with Apgar scores of 6 and 8, was delivered. Synthetic oxytocin (Syntocinon) 7.5 u., fentanyl 100 mcg, vecuronium 6 mg and Augmentin 1.2 g were given i.v. The patient remained haemodynamically stable throughout and her trachea was extubated at the end of the procedure. The patient was monitored for a further 24 h in the intensive care unit. Pain was managed by intermittent i.v. boluses of morphine for 24 h and oral analgesia thereafter. The only problem was some oozing from the abdominal wound.

After operation a more discerning drug history was obtained. The patient had been smoking crack cocaine on hospital premises immediately before each episode of dyspnoea and this was thought to precipitate the presumed episodes of acute pulmonary oedema. Urine toxicology confirmed the presence of cocaine metabolites in urine.

The patient was discharged home 9 days after operation. A renal biopsy before discharge showed glomerular immune complex deposits (M dominant) on immunohistology and evidence of intraglomerular intravascular coagulation with some features of haemolytic-uraemic syndrome on electron microscopy. These findings are consistent with the mixed renal disease found in drug abuse. Additional blood investigations showed a haemoglobin concentration of 9.8 g dl⁻¹, platelets 120 × 10⁹ litre⁻¹ and serum creatinine concentration 130 mmol litre⁻¹. Follow-up of her suspected drug-induced haemolytic-uraemic syndrome by a renal physician was planned.

Discussion

Cocaine is an alkaloid derived from the shrub *Erythroxylon coca* and is available as a hydrochloride or free base crack cocaine. Cocaine hydrochloride is usually “snorted” achieving peak plasma concentrations in less than 15 min. Crack cocaine is smoked and produces euphoria in less than 1 min with a duration of only 5–10 min⁵. The effect of cocaine as a local anaesthetic is a result of its action on sodium channels preventing nerve depolarization. The systemic effects of cocaine are caused by block of reuptake of noradrenaline and dopamine from synapses in the central and sympathetic nervous systems thereby potentiating the effects of these neurotransmitters. The euphoric effect of cocaine is caused by excess neurotransmitters at the synapses of the limbic system. Sympathetic nervous system effects include vasoconstriction, tachycardia, hypertension and arrhythmias. Chronic abuse causes tachyphylaxis resulting from depletion of presynaptic neurotransmitters⁷.

Our patient had features in common with other reports⁸–¹¹, including a poor socioeconomic background and infrequent attendance for antenatal care. The paroxysmal dyspnoea was related to pulmonary oedema associated with acute intoxication after smoking crack cocaine. This has been described previously⁶–¹² in the non-obstetric population and may reflect transient left ventricular dysfunction or a direct pulmonary microcirculatory effect.

Cocaine abuse can be confused with pre-eclampsia¹³ in its presentation. In this case hypertension was not a prominent feature although the patient did have proteinuria, ankle oedema and thrombocytopenia. Haemolysis has been reported rarely and only as haemolytic-uraemic syndrome associated with cocaine abuse¹². Thrombocytopenia is well described¹³. Many cardiovascular complications are reported¹⁴ but we can find no evidence of pericardial effusion associated with cocaine abuse.

The routine questioning of patients about recreational drug use should become standard in our preoperative assessment. Where cocaine is implicated, non-emergency surgery should be delayed until signs of acute cocaine intoxication have disappeared. This may not be possible in obstetric patients where acute cocaine intoxication may cause fetal distress or placental abruption⁷–⁵. Propranolol has been used for the control of acute hypertension in cocaine intoxication but may increase hypertension and coronary vasoconstriction because of unopposed α adrenoceptor stimulation¹⁵. For this reason labetalol¹⁶, which antagonizes α and β adrenergic receptors, and esmolol¹⁷, the short-acting β selective antagonist, may be more appropriate agents to use. Hydralazine controls hypertension but does not restore uterine blood flow¹⁸. Arrhythmias are best treated with the β adrenoceptor antagonists labetalol or esmolol. Cocaine lowers the seizure threshold and therefore lignocaine is contraindicated. Nifedipine has been shown to be protective for cardiovascular side effects in animal models of cocaine toxicity only.
if given before cocaine. Calcium antagonists have also been associated with increased mortality during cocaine intoxication in animals but there is no evidence in humans. Diazepam is the drug of choice for controlling seizures in acute cocaine intoxication but barbiturates may be necessary. Other systemic complications of acute cocaine intoxication are best managed by waiting for the acute cocaine intoxication to wear off.

Regional anaesthesia is possible in cocaine abusing obstetric patients but is associated with several pitfalls. Regional anaesthesia requires a relaxed cooperative patient which may not be the case in acute intoxication. The cocaine abusing patient may suffer vasoconstriction and have a reduced circulating blood volume which would make extradural anaesthesia preferable to spinal anaesthesia. The interaction of cocaine with other local anaesthetics makes the calculation of a safe maximum dose difficult. Thrombocytopenia is common in the chronic cocaine abusing patient and may be a contraindication to regional anaesthesia.

Where it is not possible to delay surgery in the acutely cocaine intoxicated patient, selected balanced general anaesthesia seems to be without risk. No specific induction agent is recommended but thiopentone and etomidate have been suggested as suitable for induction of anaesthesia. Ketamine increases the risk of cocaine-associated arrhythmias. Alfentanil has been used to prevent the hypercardiac response to laryngoscopy and intubation in suitable for induction of anaesthesia. Ketamine is the drug of choice in the context of acute cocaine intoxication because of neurotransmitter depletion from nerve terminals. Alfentanil has been used to prevent the hypercardiac response to laryngoscopy and intubation in patients undergoing general anaesthesia for Caesarean section. For hypertensive disorders of pregnancy, many agents have been used to attenuate the cardiovascular response to laryngoscopy, including labetolol, alfentanil and fentanyl. These agents appear to be safe in the context of acute cocaine intoxication.

Halothane increases the chance of arrhythmias with cocaine. Isoflurane anaesthesia preserved blood flow to most major organs but also increased the incidence of arrhythmias in a swine model of acute cocaine toxicity. It is theoretically possible that indirectly acting vasoconstrictors such as ephedrine may be less effective in chronic cocaine abusers because of neurotransmitter depletion from nerve terminals. More dangerous is the interaction between cocaine and direct-acting sympathomimetics causing tachycardia, hypertension, left ventricular failure and pulmonary oedema.

In conclusion, we have reported a case in which crack cocaine abuse was associated with major complications during pregnancy. In the light of evidence of increasing drug abuse, we encourage anaesthetists to be aware of the pathophysiological effects of acute cocaine intoxication and the multiple organ involvement observed in chronic abuse.

References