Clinical Report

Anaesthetic management of a parturient with myocardial infarction related to cocaine use

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Cocaine abuse is common among parturients with an incidence of 11.8 to 20%. Myocardial infarction is a rare and lethal event during pregnancy with an incidence of 1 in 10,000 pregnancies. We present the anaesthetic management of a parturient of 36 wk gestation who suffered a myocardial infarction nine hours before delivery which was temporally related to "crack" cocaine use. The patient's cardiovascular system became unstable following cocaine use, and she required mechanical ventilatory support and pharmacologic stabilization guided by invasive haemodynamic monitoring. This patient survived a non-Q wave myocardial infarction, but the prognosis of peripartum myocardial infarction remains poor with a mortality rate of 30–40% which is increased if the infarction occurs in the third trimester or postpartum period. The optimal mode and timing of delivery after myocardial infarction is unresolved. The association between cocaine use and myocardial infarction was first described in 1982, and cocaine remains unique among local anaesthetics in its ability to compromise the cardiovascular system through both sympathomimetic effects and vasoconstrictive effects on coronary arteries. Because of the prevalence

of substance abuse, cocaine use should be considered in the differential diagnosis of sudden cardiovascular compromise in parturients.

L'abus de cocaïne est fréquent chez les femmes enceintes avec une incidence de 11,8 à 20%. L'infarctus myocardique est un événement rare mais mortel pendant la grossesse. Son incidence est de 1 sur 10,000 grossesses. Nous présentons la technique d'anesthésie d'une parturiente de 36 semaines de gestation qui a présenté un infarctus du myocarde neuf heures avant l'accouchement. Celui-ci a été mis en rapport chronologique avec l'utilisation d'un « crack » de cocaïne. L'état hémodynamique de la patiente devient instable après la prise de cocaïne, et nécessite un support ventilatoire ainsi qu'une stabilisation pharmacologique. Cette patiente a survécu à un infarctus qui ne présente pas d'onde Q. Le pronostic d'infarctus du myocarde de la période du péri-partum reste cependant sombre avec une mortalité de 30 à 40% ; la mortalité s'accroît si l'infarctus survient pendant le 3^e trimestre de la grossesse ou dans le post-partum. Le mode optimal ainsi que le moment de l'accouchement après l'infarctus du myocarde fut d'abord décrites en 1982. La cocaïne reste le seul anesthésique local capable de compromettre le système cardio-vasculaire par des effets sympathicomimétiques et coronaroconstricteurs. À cause de l'abus répandu de cette substance, l'usage de cocaïne devrait être envisagé dans le diagnostic différentiel de la décompensation cardiovasculaire brutale chez les femmes enceintes.

Key words

ADDICTION: cocaine;
ANAESTHESIA: obstetrical;
ANAESTHETICS, LOCAL: cocaine;
PREGNANCY: delivery;
COMPLICATIONS: myocardial infarction, hypertension.

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The use of cocaine, especially in its freebase "crack" form, is rapidly growing in popularity. In the United States, about 15% of the entire population have tried cocaine at least once.¹ Cocaine use is equally common in parturients with an estimated incidence of 11.8–20%.^{2,3} These figures have been further subdivided by socio-economic class with a 6.3% incidence in parturients with private insurance

and a 26.9% incidence in parturients with Medicaid or no insurance.³ Cocaine use is associated with numerous maternal-fetal complications including increased incidences of abruption, preterm labour, fetal distress requiring Caesarean section, infants with low birth weights, and congenital malformations.^{1,2,4,5} Cocaine's detrimental effects on the cardiovascular system are well known, and the first report of myocardial infarction temporally related to cocaine use appeared in 1982.⁶ Myocardial infarction (MI) is a rare event during pregnancy with an estimated incidence of 1 in 10,000 pregnancies,⁷ although ECG changes suggestive of ischaemia occur in up to 37% of parturients undergoing elective Caesarean section.⁸ The importance and cause of these changes are controversial.⁹ We present a case of MI occurring at 36 wk gestation temporally related to cocaine abuse.

Case report

A 33-yr-old gravida 2, para 1 woman at 36 wk gestation presented in severe respiratory distress requiring immediate tracheal intubation. The only obtainable history was hypertension for five years with noncompliance to treatment with alpha-methyldopa and the smoking of "crack" cocaine within the past hour. Her initial vital signs were: blood pressure 240/130 mmHg, pulse 140 bpm, and oral temperature 38°C. Physical examination in the emergency room demonstrated bilateral papilloedema, no jugular venous distention, diffuse rales, and a S4 gallop. Chest x-ray demonstrated bilateral, fluffy infiltrates. Precordial ECG demonstrated left ventricular hypertrophy (LVH) with 4 mm of ST elevation in leads V₂ and V₃. Arterial blood gas (ABG) analysis before intubation revealed pH 7.11, PCO₂ 50 mmHg, PO₂ 46 mmHg breathing room air. Urine drug screen was positive for cocaine and cocaine metabolites. The patient was placed in our high risk unit on labour and delivery. Her vital signs upon admission there were: blood pressure 190/90 mmHg, pulse 98 bpm, oral temperature 36.5°C, and assisted ventilation via an endotracheal tube. Her cervix was examined and found to be 3–4 cm dilated. The uterus was contracting every 3–5 min. Fetal heart tracing revealed a tachycardia at 180 bpm with poor variability. The urine contained protein at a concentration of 100 mg · dL⁻¹. Serum uric acid and liver enzymes were within normal values. A pulmonary artery catheter and radial arterial line were placed and initial measurements were: central venous pressure (CVP) 20 cm H₂O, pulmonary artery pressure (PAP) 54/28 mmHg, pulmonary capillary wedge pressure (PCWP) 30 mmHg, and cardiac output (CO) 8.4 L · min⁻¹. Repeat chest x-ray demonstrated bilateral infiltrates. Repeat ECG demonstrated LVH and 4 mm of ST elevation in leads V₂ and V₃. Blood gas analysis was pH 7.26, PCO₂ 46 mmHg, PO₂ 73 mmHg breathing 100% inspired oxygen. Due to the fetal

tracing, labor was induced with pitocin at 6 mU · min⁻¹. The patient was treated with labetalol 15 mg, furosemide 20 mg, and a nitroglycerin infusion at 1 µg · kg⁻¹ · min⁻¹. Vecuronium 10 mg was given and controlled ventilation instituted with 10 cm positive end expiratory pressure (PEEP) at 100% inspired oxygen. A total of midazolam 20 mg and fentanyl 1150 µg was used for sedation. The patient's cardiovascular system rapidly improved to: blood pressure 160/85 mmHg, pulse 90 bpm, CVP 8 cm H₂O, PAP 30/14 mmHg, PCWP 12 mmHg, CO 9 L · min⁻¹. The patient's ABG analysis improved to pH 7.35, PCO₂ 37 mmHg, PO₂ 164 mmHg breathing 50% inspired oxygen with 5 cm PEEP. Bedside two dimensional transthoracic echocardiography demonstrated concentric left ventricular hypertrophy. Left ventricular wall motion was normal with the exception of severe hypokinesis in the basal septal area. The patient's labour progressed and after four hours, a 2185 g female infant was delivered with outlet forceps. The neonatologist elected not to score Apgars due to the administration of fentanyl, midazolam, and muscle relaxants. The infant's arterial and venous blood gas analyses were pH 7.27, PCO₂ 48 mmHg, PO₂ 52 mmHg and pH 7.29, PCO₂ 44 mmHg, PO₂ 57 mmHg, respectively. The patient was monitored for two hours post-partum and then transferred to radiology. A CT scan of the head revealed no subarachnoid haemorrhage or mass lesion. The patient was then transferred to the ICU. Her ICU stay was unremarkable with the exception of two short episodes of ventricular tachycardia that did not require treatment. The trachea was extubated after 48 hr. The ECG evolved to LVH with biphasic T waves in leads V₂ and V₃. Creatine kinase values peaked at 427 iU · L⁻¹ with 5% MB fraction. Lactic dehydrogenase isoenzymes demonstrated reversed L₁ and L₂ peaks. The remainder of the patient's hospital course was unremarkable, and a modified Bruce protocol treadmill test three weeks after admission was negative. The patient refused coronary angiography and was discharged on metoprolol, diltiazem, and enalapril.

Discussion

There have been 82 reported cases of peripartum MI to date with the greatest incidence occurring in the third trimester in women older than 35 yr. The most common cause is atherosclerotic disease. Among the published case reports, approximately 30% had coronary artery examination by either angiography or postmortem exam. Forty per cent of these patients had extensive coronary atherosclerosis and 30% had a coronary thrombus without severe atherosclerotic disease.¹⁰ Other less common causes of myocardial infarction include anomalous origins of coronary arteries, inflammatory diseases, vasospastic phenomena, and spontaneous coronary artery dissection.¹¹ Peripartum MI has a high mortality rate of 30–40%.⁷

Unlike our patient, the majority of parturients reported suffered anterior transmural infarcts.^{12,13} The timing of the MI effects the mortality: third trimester infarction carries a 45% mortality rate compared with 23% in the first and second trimesters.¹¹ Postpartum MI is an especially lethal event and usually occurs in young pre-eclamptic parturients.¹⁴ In general, fetal outcome correlates with maternal outcome. Twenty three of the 75 reported fetuses died, and 65% of these deaths occurred at the same time as maternal death.¹⁰

Our patient had a history of cocaine abuse and her MI was temporally related to smoking "crack" cocaine. Cocaine is an ester local anaesthetic with unique sympathomimetic actions. Proposed mechanisms for these actions include blockade of catecholamine storage or binding, hyperresponsivity of effector cells, release of norepinephrine, central stimulation of the sympatho-adrenal axis, inhibition of catecholamine degradation, enhancement of calcium flux across cell membranes, and direct effects on vascular smooth muscle.¹⁵⁻¹⁷ Severe cardiopulmonary and neurological complications related to the use of cocaine have been reported including: myocardial infarction,¹⁸ dysrhythmias,¹⁹ rupture of the ascending aorta,²⁰ cerebral vascular accidents,²¹ seizures,²² and subarachnoid haemorrhage.²³ There have been 58 reports of MI temporally related to cocaine use since 1982.^{6,17,24} The important characteristics to observe are that MI occurs most frequently in young (<33-yr-old) males, habitual users, patients with no previous cardiac history, and intranasal cocaine users. Cocaine may precipitate MI by either increased oxygen demand secondary to tachycardia and elevated blood pressure²⁵ or decreased supply secondary to coronary artery spasm,^{26,27} coronary thrombus, or the acceleration of atherosclerotic disease.¹⁷ Studies during cardiac catheterization have demonstrated the predilection of cocaine's vasoconstrictive actions for diseased coronary arteries with greater than double the reduction in cross-sectional area in the lumens of atherosclerotic segments as compared to normal segments.²⁸ Numerous studies have examined cocaine's procoagulant effects. Suggested mechanisms include: increased platelet response to arachidonic acid, increased thromboxane production, and increased platelet aggregation.¹⁷

The normal cardiovascular changes seen during pregnancy may add to the stress on the parturient's heart. Blood and plasma volume increase by 30-40% with peak increases in the third trimester. Cardiac output increases 30-40% during pregnancy by an increase in both heart rate and stroke volume. The greatest increase occurs immediately after delivery when the cardiac output is 80% above prelabour values. This is attributed to auto-transfusion from uterine contraction.²⁹

The mode and timing of delivery after myocardial infarction remain controversial. Cohen and Hankins have reviewed the advantages and disadvantages of vaginal versus Caesarean delivery. Hankins in his large review observed a 23% mortality rate in Caesarean deliveries compared with 14% in vaginal deliveries. A mortality rate of 50% in women delivering within two weeks of infarction was also noted compared with no deaths when delivery was more than two weeks after infarction. Unfortunately, none of these groups were controlled or matched.⁷ Cohen, likewise, was unable to find convincing support for one mode over the other.³⁰

In conclusion, the anaesthetic management of a parturient sustaining a non-transmural MI temporally related to smoking "crack" cocaine nine hours before vaginal delivery is presented. This is the first reported incident but may become more common due to the prevalence of cocaine abuse.

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