An unusual case of ST elevation

Gordon K, MBChB, DipObst(SA), DA(SA), Registrar
Department of Anaesthetics, Critical Care, and Pain Management, Grey's Hospital
Wise R, MCBhB, Dip Obst(SA), DipPEC(SA), FCA(SA), CertCritCare(SA), MMed, Clinical Lecturer, University of KwaZulu-Natal
Department of Anaesthetics, Critical Care, and Pain Management, Grey's Hospital
Perioperative Research Group, Department of Anaesthetics, Nelson R Mandela School of Medicine, University of KwaZulu-Natal
Correspondence to: Robert Wise, e-mail: robertwise@webafrica.org.za
Keywords: coronary, artery, vasospasm, myocardial infarction, intraoperative

Abstract

This report presents an unusual case of ST elevation on induction of anaesthesia in a patient undergoing an elective, noncardiac surgical procedure. Through scrutiny of this case and its unanticipated events, together with the subsequent workup and management, the diagnosis of coronary artery vasospasm may be appreciated as an important entity of coronary artery disease. It serves to highlight coronary artery vasospasm as an underappreciated diagnosis, to allow the anaesthetist to anticipate it as a potential risk in certain populations, and to distinguish it from other coronary artery disease and to institute appropriate management.

Peer reviewed. (Submitted: 2013-06-05. Accepted: 2013-08-13.) **South Afr J Anaesth Analg 2013;19(5):270-273**

Introduction

Anaesthesiologists are frequently faced with patients suffering from perioperative cardiac events. Fortunately, intraoperative ST elevation is not that common. However, it is important to consider all possibilities when encountering it as incorrect assumptions may result in incorrect therapy. We present an interesting case of ST elevation that occurred soon after induction of anaesthesia.

Case study

A 61-year-old patient with no previous cardiovascular history was scheduled for an elective acromioplasty. Despite her arthropathy, she was an active patient with good effort tolerance. She had never experienced angina or palpitations, and had no family history of heart disease. She was a chronic smoker. She had undergone prior surgery in 2008 for a prolapsed bladder, and both the general anaesthetic and recovery were uncomplicated. The preoperative examination, haematological workup, electrocardiogram (ECG) and chest X-ray were all normal.

An uncomplicated interscalene brachial block was performed using a nerve stimulator on the awake patient (70 mg of plain bupivacaine in a volume of 20 ml). A prophylactic antibiotic was given prior to induction. Following preoxygenation, she was induced with an elective sequence induction, using propofol 140 mg, vecuronium 4 mg and fentanyl 200 µg. The intubation was uncomplicated and she was initiated on intermittent positive pressure ventilation and placed in a semi-sitting position for the surgery. The patient's anaesthetic was maintained with sevoflurane, oxygen and air.

Five minutes after her induction, the patient became acutely hypotensive. The mean arterial pressure dropped from a preoperative value of 85 mmHg to 55 mmHg. This hypotension was resistant to repeated boluses of ephedrine and phenylephrine. An initial compensatory tachycardia of 110 beats per minute (bpm) fell to 50 bpm, and 0.5 mg of atropine was given twice. The patient remained hypotensive and bradycardic, so 1 ml of 1:10 000 adrenaline was administered. There was a brief period of tachycardia, but both her heart rate and blood pressure subsequently normalised without any further inotropic support. However, significant ST elevation was noted on ECG monitoring. The elective surgery was postponed and a cardiologist consulted.

On review of the history and 12-lead ECG, the cardiologist advised urgent thrombolysis. Tenectaplase was ordered, but it was not readily on hand in theatre. Therefore, the patient was kept on the operating table and monitored. Serial ECGs showed complete normalisation and a return

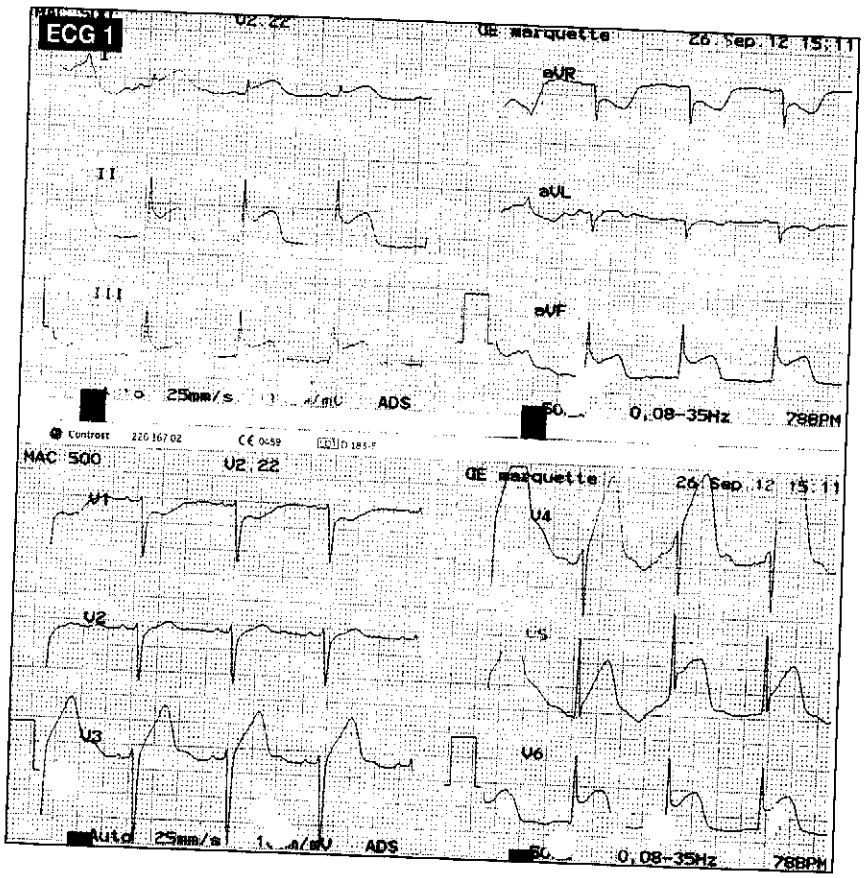
to the patient's preoperative state, with no evidence of any evolving myocardial infarction. A decision was made to omit thrombolysis and monitor her cardiovascular status in the high care unit (HCU) overnight. Angiogram facilities were not immediately available and the investigation was deferred until the following morning.

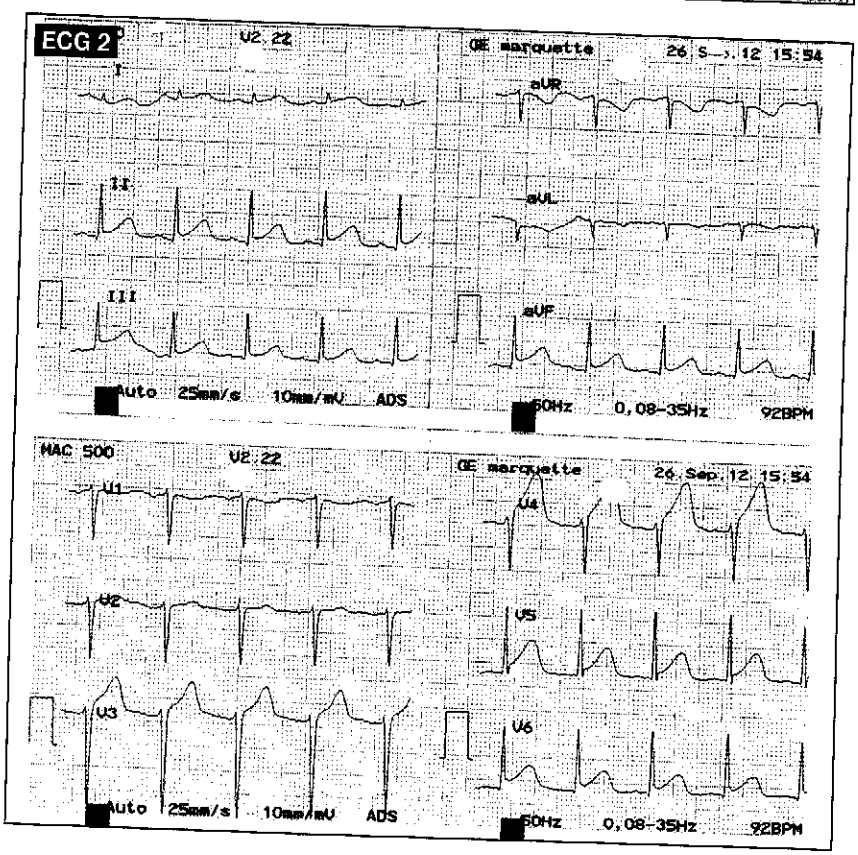
On admission to the HCU, she remained stable with no chest pain, nor any changes in her heart rate or blood pressure. Appropriate counselling of the patient and her family took place. She remained in the HCU for the next 48 hours, and was treated as an ST elevation myocardial infarction with enoxaparin, clopidogrel, aspirin and simvastatin.

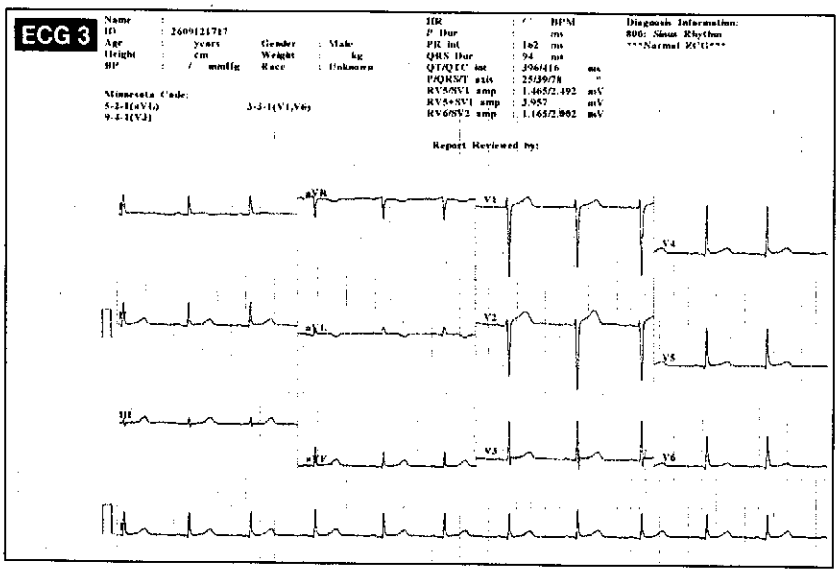
Investigations

Two-hourly ECGs and six-hourly serological cardiac enzyme levels were performed in the HCU. The initial ECG, performed on the table following the patient's period of instability, indicated significant ST elevation in leads II, III, AVF, V3, V4, V5 and V6, suggestive of inferiolateral infarction. Serial ECGs showed normalisation with intermittent ST elevation in the same leads, but no further indicators of evolving ischaemia as time progressed.

Cardiac markers taken two hours post peri-arrest state were elevated with a creatine kinase-MB of 51.3 U/l, and cardiac troponin I of 24 650 ng/l. These showed a downward trend over the next two days, suggestive of an acute episode of ischaemia, followed by fairly rapid resolution, which correlated with the ECGs. Further special investigations aimed to identify the precipitating factor for this episode of coronary artery insufficiency. Normal mast cell triptase and histamine levels drawn on admission to the HCU excluded an anaphylactoid reaction that may have accounted for the initial hypotension and subsequent inadequate epicardial perfusion. Serum chemistry showed a slightly low magnesium level 0.65 mmol/l. This may have been a contributing factor. Finally, a coronary artery angiogram was performed to exclude any physical obstruction to the vessels. It revealed normal epicardial coronary arteries with some apical hypokinesis, but ventricular tachycardia developed twice during the procedure. A diagnosis of coronary artery vasospasm was proposed.







Differential diagnosis

This incident was recognised as a case of acute myocardial ischemia, with a subsequent ST elevation myocardial infarction from an early stage. Coronary artery disease and obstruction of a vessel by an atherosclerotic plaque were foremost in our minds. However, a normal angiogram excluded this possibility, and the patient's rapid recovery suggested a dynamic obstruction to the coronary vessels, rather than a fixed stenosis, thus suggesting a type II myocardial infarction, rather than a type I, according to the American Heart Association classification.

Inadequate coronary perfusion then had to be evaluated. The potential for anaphylaxis was considered and excluded biochemically. Local anaesthetic toxicity, following the brachial block, was considered, although the regional technique was uncomplicated, had worked well and the episode of instability was too short-acting to be attributed to an intravascular dose of bupivacaine.

Consequently, a diagnosis of vasospasm was suggested. Hypercontractility of the coronary arteries was the likeliest cause in a patient who was a chronic smoker with low magnesium levels. Various precipitants experienced during the induction of anaesthesia may have triggered the vasospasm, namely the hypotension induced by the propofol induction, a Bezoldt-Jarisch reflex secondary to the positioning, the use of ephedrine, the adrenaline bolus, histamine release induced by the fentanyl, or even a brief period of hyperventilation.[1] A nicotine withdrawal may also have contributed.[2]

Treatment

On diagnosis of coronary artery vasospasm, the patient's anticoagulants were discontinued. It should be noted that the treatment of choice for an acute event of confirmed vasospasm is the administration of nitrates.[2] This management option was not considered in the acute setting with this index patient. Therapies to prevent future events of vasospasm include the cessation of smoking, statin therapy, antioxidants, calcium-channel blockers, aspirin and oestrogen in postmenopausal females.[3]

The patient was discharged from the HCU after 48 hours and was monitored by the cardiology department. She was educated about the diagnosis, initiated on a new dietary plan and advised to stop smoking. She returned to her preoperative level of functioning. Her acromioplasty was postponed for six months.

Discussion

Coronary artery vasospasm is an uncommon diagnosis in the developing world. Limited cardiology services to confirm this diagnosis, coupled with misdiagnosis of these cases as forms of fixed coronary artery obstruction, seem to account for the under-reporting of this condition.[2,4] It is a form of coronary artery disease that causes a sudden, transient narrowing of one or more of the coronary arteries. This differs to the chronic, progressive obliteration caused by atheromatous plaques described with traditional coronary artery disease. The temporary constriction of vessels is due to the hypercontractility of coronary vascular smooth muscle, which, in turn, is due to endothelial dysfunction with reduced endothelial nitric oxide activity,[3] increased oxidative stress and chronic low-grade inflammation. Just as the pathophysiology of the condition differs from the classic picture of stenotic coronary artery disease, so too does the patient profile. Smoking is the single greatest risk factor, whereas other conditions, such as hypertension and diabetes, have no significant risk at all.[3]

Coronary artery vasospasm, while under anaesthesia, is by no means a novel idea in the current literature. Indeed, several precipitating factors for vasospasm may be unique to a theatre setting, including sudden hypotension induced by certain induction agents or neuraxial anaesthesia, hyperventilation, the manipulation of visceral organs during surgery,[5] the deflation of tourniquets during orthopaedic procedures,[6] and the administration of a combination of vasopressors and histamine-releasing agents.[3] Yet it is a phenomenon that is infrequently reported, and as described by this case report, is a diagnosis that is settled upon when all else has been excluded.[1]

It is appreciated that coronary vasospam is an under-diagnosed entity, and new attention is being paid to the subtleties of this condition and its early recognition and

management. Patients who are at risk are not the typical overweight, middle-aged men who suffer from coronary artery stenosis, but rather younger patients, particularly of Eastern Asian origin, who are smokers.[3] However, a definitive diagnosis is difficult in a theatre setting as angiography reveals normal vessels in 70% of patients who experience vasospasm. Intracoronary injection of acetylcholine induces a short-lived vasospasm that is visible on imaging in the affected artery or arteries.[1,3] The degree of constriction may also be visualised. The invasive nature of the required test and expertise render it an on-table investigation that is far from ideal, but is currently the gold standard diagnostic tool.

The pathophysiology of coronary artery vasospasm entails that the implicated vessels have no fixed organic stenosis, but rather hypercontractility of the smooth muscle lining the vessel. Thus, the management of choice is vasodilators, and specifically nitrates and calcium-channel blockers, rather than the thrombolytics traditionally used in acute coronary syndrome.[2] The degree of constriction of the artery and subsequent ischaemia varies, resulting in a spectrum of disease. It stands to reason to relieve the obstruction as soon as the vasospasm has been identified.

Conclusion

In conclusion, coronary vasospasm is a difficult diagnosis to make intraoperatively. However, it should be considered in younger patients who smoke and who do not necessarily fit the typical high-risk profile for cardiac ischaemic events. The diagnosis is usually made in the coronary catheter laboratory. The management of coronary vasospam is nitrates and calcium-channel blockers.

The following should be borne in mind:

- Not all coronary artery disease is an atherosclerotic fixed obstruction. Vasospasm is due to dynamic hypercontractility and constriction.
- The perioperative setting provides numerous precipitants for at-risk patients to develop vasospasm. Anaesthetists should be sensitive to this possibility.
- Nitrates and calcium-channel blockers are the managing agents of choice.

Financial support and industry affiliation

Drs Gordon and Wise declare that they have no financial or personal relationships which may have inappropriately influenced them in writing this paper.

References

1. Wakabayashi K, Suzuki H. Cardiopulmonary arrest due to persistent coronary spasm in a young woman: are we properly diagnosing vasospastic angina? Int J Cardiol. 2011;148(3):56-59.
2. Sidi A, Dahleen L, Gaspardone A. Coronary vasospasm during anaesthesia induction: awareness, recognition, possible mechanisms, anaesthetic factors and treatment. J Clin Anaesth. 2008;20(1):64-69.
3. Yasue H, Nakagawa H, Itoh T, et al. Coronary artery spasm: clinical features, diagnosis, pathogenesis and treatment. J Cardiol. 2008;51(1):2-17.
4. Novas BG, Boada PS, Puig BR, Pelegri GD. Coronary artery vasospasm in general anaesthesia induction. Rev Esp Anestesiol Reanim. 2013;60(4):233-236.
5. Green MS, Gomes S. Intraoperative coronary artery vasospasm: a twist in the tale! Middle East J Anaesthesiol. 2011;21(2):299-304.
6. Ohmori H, Takahashi H, Yamakaye M, Namiki A. Intraoperative cardiac arrest due to coronary vasospasm after tourniquet release: a case report. Masui. 2006;55(4):460-463.