Delayed papillary muscle rupture is well documented.¹ The forces exerted on the heart from dynamic chest trauma are variable and depend on the elasticity of the thorax and the intra-abdominal pressure. A further variation depends on the points in the respiratory and cardiac cycles at which the insult occurs. The most vulnerable points are at maximal inspiration, and during isovolumic systole when all the valves are closed and the cavities hold the maximum amount of blood. The pathophysiology of the muscle rupture is not clear but it has been suggested that the intramural blood flow redistribution caused by local oedema, fibre rupture, and haematoma produces sufficient endocardial ischaemia to cause papillary muscle necrosis. Papillary muscles, being projections from the ventricular wall, are particularly prone to ischaemia in this way as they cannot benefit from a collateral supply. The subsequent proteolytic process reaches a maximum

over 24 hours that accounts for the delayed presentation.

In conclusion this case illustrates the need for the possibility of cardiac trauma to be considered in patients who sustain a blunt chest injury. This consideration should be a dynamic one, and the diagnosis should be reconsidered after any change in the patient's condition even where cardiac trauma was previously excluded. Although TTE may be useful in some cases, it has limited sensitivity compared with TOE and consideration should be given to more widespread application of the latter in ICUs.³

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Fatal flecainide intoxication

E Brazil, G G Bodiwala, D C Bouch

Abstract

Flecainide acetate is a potent class 1C antiarrhythmic agent used mainly for the treatment of supraventricular arrhythmias. Acute overdose of this drug is rare but frequently fatal. The clinical course of a patient that ingested a large quantity of flecainide as a suicide attempt is described and current therapeutic strategies discussed.

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Keywords: flecainide; toxicity

Case report

A previously healthy 36 year old man presented to the accident and emergency department having taken a deliberate overdose of approximately 100, 100 mg flecainide acetate tablets six hours previously. On arrival his clinical observations showed a systolic blood pressure of 140 mm Hg and a Glasgow coma scale (GCS) score of 15/15. His electrocardiography (ECG) monitoring strip showed a polymorphic ventricular tachycardia at a heart rate of 140 beats/min (fig 1).

Ten minutes after his arrival the patient had an episode of pulseless ventricular tachycardia which was treated successfully with a single unsynchronised shock at 200 joules. Subsequently his observations were stable with a systolic blood pressure of 170 mm Hg and GCS score of 15/15. ECG monitoring displayed sinus rhythm at a rate of 75 beats/min.

Analysis of 12 lead ECG showed the QRS duration to be prolonged at 0.2 sec (fig 2). Urea and electrolyte measurements were within normal limits. Arterial pH was 7.374.

Advice from a regional poisons information unit recommended gastric lavage, administration of activated charcoal, and infusion of sodium bicarbonate to raise the arterial pH to 7.5. The patient refused gastric lavage and activated charcoal but allowed treatment with hypertonic sodium bicarbonate. He agreed to come into hospital and was therefore admitted to the coronary care unit for monitoring and treatment with sodium bicarbonate 1.26%. While in coronary care the patient's ECG monitoring continued to demonstrate sinus rhythm at approximately 75 heartbeats/min with prolonged QRS duration.

Ten hours after admission the patient's condition deteriorated with an episode of hypotension secondary to ventricular tachycardia. This reverted spontaneously before treatment was instituted and the systolic blood pressure returned to 120 mm Hg. Electrolyte analysis was again normal and arterial pH was 7.404. Two hours later the patient deteriorated to a pulseless electrical activity cardiopulmonary arrest. Full resuscitation following Advanced Life Support guidelines was performed but was unsuccessful. The patient was pronounced dead 12.5 hours after admission. At postmortem examination a quantity of granular material was identified within the deceased's stomach indicating that absorption was still continuing up to the time of death. Serum flecainide concentration was 3.32 mg/l at postmortem examination. The usual therapeutic plasma concentration is 0.2–1 mg/l.

Accident and Emergency Department, Leicester Royal Infirmary, Leicester LE1 5WW
E Brazil
G G Bodiwala

Department of Pathology, Leicester Royal Infirmary
D C Bouch

Correspondence to:
Mr Brazil, Specialist Registrar in Accident and Emergency Medicine.

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Figure 1 ECG monitoring strip.

Figure 2 Twelve lead ECG.

Discussion

Flecainide acetate is a Vaughn-Williams class 1C antiarrhythmic agent. There are few reports regarding the management of flecainide toxicity. Acute overdose of flecainide acetate is uncommon but frequently fatal.^{1,2} Flecainide is a sodium channel blocking agent used mainly for the treatment of supraventricular arrhythmias. Absorption from the gastrointestinal tract is reasonably prompt with peak concentrations occurring after 3–4 hours, with a long plasma half life of 11 hours. The plasma pharmacokinetics are independent of dose in the therapeutic range. There is however, little information regarding plasma half life in toxic doses, but reduced hepatic and renal blood flow due to hypotension would reduce flecainide elimination and prolong duration of toxicity. One study has documented an elimination half life of 21.8 hours after an overdose of 3800 mg of flecainide.³ Plasma protein binding is low at 40%.

Adverse cardiac effects include moderate negative inotropic action and depression of all major conductive pathways.³ With increasing concentration, flecainide's action on conduction pathways is manifested on ECG as an increased PR interval and QRS duration.^{4,5} Toxicity is suggested when a 50% increase in QRS duration (0.18 sec) or a 30% prolongation in PR interval (0.26 sec) occurs.³

The primary treatment of acute flecainide overdose should be directed at reducing gastrointestinal absorption by gastric emptying and administration of activated charcoal.⁶ The fact that there was still evidence of drug absorption from the stomach of our patient 16 hours after ingestion would support this. There is no specific antidote to flecainide. Because of flecainide's large volume of distribution haemodialysis and haemoperfusion have been shown to be ineffective.⁷ A report regarding the management of two cases of acute flecainide overdose recommended the prophylactic use of either an intravenous or extrathoracic pacemaker.⁶

Reports of uncontrolled clinical studies have suggested a therapeutic role for hypertonic sodium bicarbonate as a treatment for flecainide intoxication.⁸ In a canine model one investigator found that administration of hypertonic sodium salts reversed the effect of class 1 antiarrhythmic drugs and suggested its potential use for the treatment of cardiotoxicity caused by sodium channel blocking drugs.⁹ Another study using canine Purkinje fibres confirmed the efficacy of sodium salts to treat flecainide toxicity and suggested that this was due to displacement of the sodium blocking drug caused both by the increased sodium concentration and increased alkalinisation.¹⁰ Despite an infusion of 1.26% sodium bicarbonate the arterial pH of our patient did not reach the recommended level of 7.5. Dobutamine has been reported to have successfully treated one patient with polymorphic ventricular tachycardia and profound hypotension secondary to an overdose of flecainide.¹¹

Because hypotension can develop rapidly after flecainide overdose, hepatic and renal blood flow are reduced. Measures to maintain vital organ perfusion will enhance flecainide clearance and redistribution to body tissues. One method to provide the haemodynamic support necessary to allow flecainide clearance and redistribution to occur is peripheral cardiopulmonary bypass support. In this procedure blood is withdrawn from the inferior vena cava and right atrium, oxygenated, and then pumped into the femoral artery.¹² Yasui *et al* showed that this technique allowed flecainide clearance and redistribution to continue in one patient whom had taken a flecainide overdose reducing the plasma half life to six hours.²

The sudden deterioration of the patient, in this case 16 hours after drug ingestion, having been apparently stable for 10 hours is alarming. The only evidence of ongoing toxicity at this stage was prolonged QRS complexes on ECG monitoring. There was however, evidence of ongoing absorption of the drug at postmortem examination which may have

caused the sudden catastrophic collapse. A similar deterioration to ventricular fibrillation has been reported 70 hours after ingestion.⁶ The plasma flecainide concentration at 73 hours in this patient was 0.7 mg/l having been 6.5 mg/l three hours after ingestion. This patient however, responded to a single defibrillatory shock. A second patient reported by the same author deteriorated to an electromechanical dissociation cardiac arrest 6.5 hours after flecainide overdose despite recording a blood pressure of 140/80 mm Hg before the arrest. Resuscitation was unsuccessful in this case.

Acute overdose of flecainide acetate is rare but produces serious cardiac compromise. The primary treatment aim is to prevent drug absorption by gastric lavage and administration of activated charcoal. Secondary measures to prevent cardiac conduction disturbance include the use of a prophylactic pacemaker and inotropic drugs. Attempts to displace the sodium channel blocking drug by administration of hypertonic sodium bicarbonate require frequent electrolyte measurements. Invasive techniques of haemodynamic support may allow clearance and redistribution to occur. We would recommend that all patients with flecainide overdose be admitted to intensive care for invasive monitoring even if they appear clinically

stable as they are at risk of subsequent deterioration. Despite these measures, however, mortality remains high.

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A case of streptococcal myositis (misdiagnosed as hamstring injury)

Norbert Kang, Dimitrios Antonopoulos, Atul Khanna

Abstract

Streptococcal myositis is a very rare bacterial infection of muscle with a high mortality. Diagnosis is difficult because of the paucity of clinical signs and symptoms at the onset. However, presentation of the disease appears to have changed over the last 50 years. A case of streptococcal myositis is presented (misdiagnosed as hamstring injury), which more closely reflects the current presentation of the disease. Some of the features that may help emergency clinicians to recognise the onset of the condition are highlighted.

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Keywords: necrotising fasciitis; myonecrosis; streptococcal myositis

Streptococcal myositis is an acute infection of muscle by invasive group A β haemolytic streptococcus causing myonecrosis without abscess formation. It differs from the more frequent and relatively benign streptococcal pyomyositis, which is characterised by the formation of abscesses in muscle and which has a good prognosis.

The standard surgical texts are misleading: "Streptococcal myositis resembles acute clostridial gas gangrene and was not described until *World War II*. After an incubation period of 3 to 4 days there is swelling, edema, and purulent wound exudate. These signs are followed by pain which rapidly becomes severe. Gas is present and the infected muscle changes from pale and soft to bright red, striped with purple and finally purple and gangrenous. The seropurulent discharge has a sour odour".¹

The principle source of this description is MacLennan who collected a series of eight cases of streptococcal myositis in soldiers with battle wounds during *World War II*.² This description differs from more recent reports including our own experience of the condition.

Case report

A 33 year old labourer presented with 24 hours of increasing pain in the medial aspect of his right thigh. There was no history of recent trauma. Slight erythema, tenderness, and swelling were noted. The pain was worse with movement especially extension and abduction. Sensation and pulses were intact throughout the lower limb. He was afebrile. A diagnosis of

RAFT Institute of Plastic Surgery, Mount Vernon Hospital, Northwood, Middlesex N Kang

Department of Plastic Surgery, St Andrew's Hospital, Patras, Greece
D Antonopoulos

Department of Plastic Surgery, Wordlesey Hospital, Stourbridge, West Midlands
A Khanna

Correspondence to:
Mr Norbert Venantius Kang,
6 Elgin Drive, Northwood,
Middlesex HA6 2YR.

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