PHARMACOLOGY

γ Hydroxybutyrate, chemical formula $\text{COOHCH}_2\text{CH}_2\text{CHOH}$, is a normal constituent of mammalian metabolism and is found widely in the body. It is believed to be a neurotransmitter. It temporarily inhibits the release of dopamine in the brain and stimulates both growth hormone and prolactin release. It is both a metabolite and precursor of GABA. It is well absorbed orally, readily crosses the blood-brain barrier, and is subsequently metabolised to carbon dioxide. The pharmacokinetics are complex. The response to a low dose is unpredictable and elimination appears to be capacity limited, so the half life increases with larger doses. Its central nervous system depressant effects appear to be potentiated by other psychoactive drugs, particularly alcohol. No clinically effective antidote has been identified.

Since it was first manufactured, γ hydroxybutyrate has been used in various ways. It causes remarkable hypotonia, which has led to its use in France and Italy as an aid to childbirth. It causes a protein sparing effect which reduces the breakdown rate of body proteins. This effect, which is mediated by growth hormone, has led to its use among body builders. It is a potent hypnotic and sufficient doses will induce sleep within a matter of minutes, which has led to its use as an anaesthetic agent. It is also effective in decreasing the symptoms of alcohol and opiate withdrawal and has been used for the treatment of narcolepsy and in psychotherapy.

ABUSE

Most users ingest a capful of the liquid form of γ hydroxybutyrate (5–10 ml) and gain the desired effects within 15 to 60 minutes. In toxic quantities it may cause drowsiness, dizziness, hypothermia, nausea, and sometimes vomiting. Ataxia and incoordination are also commonly seen. Headache is sometimes reported. A moderate slowing of the heart rate is common and small changes in blood pressure may occur. Sufficiently large doses can cause

sedation, epileptic seizures, and loss of consciousness. Although recovery is usual within three hours, some patients have required intubation and mechanical ventilation.⁶ In each case described above γ hydroxybutyrate was taken with other substances, but it was the only agent common to each. The repeated pattern of sudden loss of consciousness followed by rapid recovery after a few hours would not be explained by any of the other substances taken, and offers compelling evidence that γ hydroxybutyrate was the cause.

The amount of γ hydroxybutyrate abuse in the United Kingdom has been reflected by the number of calls to the National Poisons Information Service (London) over the last two years, averaging one per week (personal communication: NPIS London). Physical dependence on γ hydroxybutyrate from chronic use is rare although it has been described.⁷

CONCLUSION

γ Hydroxybutyrate has become a fashionable drug on the dance and club scene in the United Kingdom. Accident and emergency staff need to be aware of its existence and its potential effects. The majority of people who have ingested it will recover spontaneously without long term sequelae, but its toxic effects may be dramatic while they last, particularly when it is taken with other drugs or alcohol.

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Pneumococcal pericarditis presenting as an out of hospital cardiopulmonary arrest

A R Wass, P Mann, J I Wilson

Accident and
Emergency
Department,
Pinderfields General
Hospital, Wakefield,
Yorkshire
A R Wass
P Mann
J I Wilson

Correspondence to:
Mr A R Wass, Accident and
Emergency Department,
Pinderfields General
Hospital, Aberford Road,
Wakefield WF1 4DG.

Accepted for publication
28 February 1997

Abstract

Serious complications of pneumococcal pneumonia have become uncommon with effective antibiotic treatment. Purulent pericarditis is a rare though well described complication of untreated pneumococcal sepsis. A case of untreated pneumococcal pneumonia complicated by purulent pericarditis is described. This presented as an out of hospital asystolic cardiopulmonary arrest.

(*J Accid Emerg Med* 1997;14:262–264)

Keywords: *Streptococcus pneumoniae*; untreated pneumonia; purulent pericarditis; out of hospital cardiopulmonary arrest

Case report

A 29 year old man presented with an out of hospital asystolic cardiopulmonary arrest. Though previously well, he had suffered for two weeks with a “flu-like” illness. Musculoskeletal chest pain had been diagnosed four days before presentation, when he developed pleuritic symptoms. He had complained of

progressive dyspnoea, orthopnoea, and ankle oedema in the 24 hours preceding presentation.

The patient then collapsed while seeking further medical attention. Effective bystander basic life support was started immediately. On arrival, the paramedic ambulance crew documented asystole and advanced life support was begun with intubation, ventilation, and administration of adrenaline and atropine through the endotracheal tube. Transfer to hospital was within 15 minutes of the collapse.

On arrival at hospital asystole persisted. Adrenaline given through a central vein resulted in the return of a pulse (rate 120) and blood pressure (90/60 mm Hg), but no spontaneous respiratory effort. Subsequent examination revealed a loud pericardial friction rub, gallop rhythm, elevated venous pressure, bilateral coarse respiratory crackles, ankle oedema to mid calf, and hepatomegaly. An electrocardiogram (ECG) showed tachycardia, widespread ST elevation, and non-specific T wave changes. A chest x ray showed cardiomegaly and bilateral pulmonary infiltrates (fig 1). On routine blood testing there was a neutrophil leucocytosis ($42 \times 10^9/\text{litre}$) and deranged renal function (creatinine $386 \mu\text{mol/litre}$, urea 28.6 mmol/litre , potassium 5.4 mmol/litre).

The patient was admitted to intensive care for ventilatory and inotropic support. Echocardiography showed moderate left ventricular function and a pericardial effusion, with right atrial collapse and early tamponade. Pericardiocentesis yielded over 100 ml of turbid aspirate without improving systolic function. Broad spectrum antibiotics were given. Aspirate microscopy showed frequent Gram positive diplococci, later confirmed as *Streptococcus pneumoniae*. Despite aggressive antibiotics and inotropic support the patient became anuric, hyperkalaemic (potassium 6.6 mmol/litre), and developed signs of disseminated intravascular coagulation (DIC). After transfer to the regional renal unit and successful resuscitation from a second cardiopulmonary arrest, reassessment confirmed DIC and systemic sepsis, with deteriorating renal and cardiac function.

Figure 1 Chest x ray showing cardiomegaly and bilateral pulmonary infiltrates.

In spite of maximum inotropic support, haemofiltration, and the insertion of an intra-aortic balloon pump, the patient suffered a third cardiac arrest from which he could not be resuscitated. A necropsy examination confirmed overwhelming pneumococcal sepsis arising from a left lower lobe pneumonia complicated by purulent pericarditis.

Discussion

Pneumococcal pneumonia is a common cause of morbidity and mortality in the community.¹ *Streptococcus pneumoniae* bacteraemia is a serious disease with an overall mortality of 19%.² Purulent pericarditis is a rare though well described life threatening complication of untreated pneumococcal infection. In a review of 113 cases of pneumococcal pericarditis, pneumonia (93.1%) or pneumonia with an accompanying empyema (66.6%) were the commonest underlying infections.³ Cases of direct spread from endocarditis or haematogenous spread from meningitis, subcutaneous abscesses,³ otitis media, mastoiditis, septic arthritis, and appendicitis⁴ have also been reported.

The overall incidence of purulent pericarditis has fallen by a factor of three since the advent of antibiotics in 1943.⁵ Before 1943 purulent pericarditis was predominantly a disease of children and young adults (median age 20 years), associated with severe bacterial infection.⁵ Sixty four per cent of cases resulted from direct extension of pleuropulmonary disease. Pneumococcus was the commonest organism, accounting for 51% of cases.⁵ Since 1943 purulent pericarditis has occurred in older patients (median age 49 years) who have underlying pericardial disease, chronic disease, malignancy, immunosuppression, or after thoracic surgery. Contiguous spread from pleuropulmonary disease currently accounts for 20% of cases, and overall just 9% result from pneumococcal infection.⁵ The reduced incidence of pneumococcal pericarditis relates to the susceptibility of the pneumococcus to antibiotics, the prevalent use of antibiotics in respiratory tract infections,⁴ and the increased use of pneumococcal vaccine.

Diagnosing pneumococcal pericarditis continues to present problems despite the advances of modern medicine,⁵⁻⁸ reflected by the fact that only 10–20% of cases are correctly diagnosed antemortem.^{3,5} The diagnosis is rarely considered if a focus of infection has already been identified within the chest.⁶ These patients are acutely unwell; however, the most reliable signs are pyrexia and tachycardia, which are also the least specific.⁶ Signs of right heart failure, pulsus paradoxus, or a pericardial rub are more specific though frequently absent.⁸

The definitive diagnosis of pneumococcal pericarditis is from culture of pericardial aspirate.⁹ Blood culture is positive in only 30% of patients⁶ but is helpful in these cases. Neutrophil leucocytosis in excess of $20 \times 10^9/\text{litre}$ is usual. The ECG may be diagnostic of pericarditis with generalised ST segment elevation without reciprocal changes (helping

to differentiate from acute myocardial ischaemia) and non-specific T wave changes.⁶ Low ECG voltages suggest a pericardial effusion.¹⁰ A chest x ray will identify pleuropulmonary disease and allow assessment of cardiac size. An expanding cardiac silhouette on serial chest x rays may indicate a developing effusion.⁶ Two dimensional echocardiography is non-invasive, identifies pericardial effusions, assesses for cardiac tamponade, and acts as a guide for pericardiocentesis.

Untreated pneumococcal pericarditis complicated by septicaemic shock is invariably fatal. Out of hospital asystolic cardiopulmonary arrest also has a poor prognosis, with an overall survival to discharge of about 2%.^{11 12} Published reports on the treatment of pneumococcal pericarditis are based upon case reports, small series, and review articles rather than on randomised trials. However, early diagnosis of purulent pneumococcal pericarditis with prompt aggressive treatment significantly reduces mortality.³ Cardiac tamponade is not unusual and can occur despite prolonged antibiotic treatment⁹ or even if the site of primary infection has been controlled. In this circumstance, pericardiocentesis is not only diagnostic but life saving too. Effective drainage of the pericardium (and empyema if present^{3 13}) in conjunction with high dose intravenous antibiotics offers the best results.³ The majority of published reports support open pericardial drainage, arguing that pericardiocentesis alone risks recurrent tamponade.^{3 4 7} However, there are isolated case reports of successful outcome from antibiotics accompanied by pericardial catheter drainage with repeated aspiration.^{14 15} Some argue that pericardectomy avoids constrictive pericarditis,^{7 8 13} while others regard this as controversial.⁴ The installation of antibiotics within the pericardial sac is unnecessary, as therapeutic levels are achieved with systemic treatment.⁷ General supportive measures are often required to overcome the complications of systemic sepsis and myocardial dysfunction resulting from an associated subepicardial myocarditis.⁴

CONCLUSIONS

Purulent pericarditis is a rare complication of untreated pneumococcal sepsis. It is an acute disease with a fulminant course which often remains undiagnosed during life. The potential for a good outcome depends upon awareness and a high index of suspicion, allowing early diagnosis and aggressive treatment.

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Conversion disorder presenting as a head injury

F E Poyner, P E Pritty

Accident and
Emergency
Department,
Derbyshire Royal
Infirmary, London
Road, Derby DE1 2QY
F E Poyner
P E Pritty

Correspondence to:
Dr F E Poyner, senior
registrar in A&E.

Accepted for publication
10 April 1997

Abstract

A case of a conversion disorder which presented as a head injury is described. This is a rare problem and by definition a diagnosis of exclusion.

(*J Accid Emerg Med* 1997;14:263-264)

Keywords: conversion disorder; head injury

Case report

A 35 year old female presented to the accident and emergency (A&E) department after a car

boot lid had fallen onto her occiput. There had been no loss of consciousness. When seen she had an intermittent stutter but there was no detectable neurological abnormality. She was discharged home with an appropriate advice sheet.

Two days later she consulted her general practitioner complaining of an unsteady gait and speech difficulties and was referred directly for a skull x ray. This showed no abnormality but the radiographer was concerned by