kPa (78 mm Hg), carbon dioxide pressure 3.3 kPa (25 mm Hg), bicarbonate concentration 14.0 mmol/l, and pH 7.4. She was admitted to the intensive care unit and within 12 hours had regained consciousness. Renal and hepatic function remained normal throughout the illness.

When her home was searched a strong-smelling substance in a rum bottle was found. Toxicological analysis confirmed that this was not rum but butanone (methyl ethyl ketone). Gas-liquid chromatography showed butanone to be present in her plasma in high concentration (13.2 mmol/l (95 mg/100 ml)) and in her urine. The plasma lactate concentration in blood taken at the time of admission was also raised (14.3 mmol/l (129 mg/100 ml)).

She made a complete and uneventful recovery and was discharged from hospital after one week. She denied having swallowed anything unusual. We can only presume that she had sought solace from her chickenpox rash but that the rum bottle had inadvertently been filled with butanone.

Comment

Butanone is extensively used as an industrial solvent and is found in some commercial and household glues. There are no previous reports of self-poisoning by ingestion of it. The metabolic effects of butanone are unknown, and it is not clear whether the substantial degree of lactic acidosis in the present case was directly due to butanone or merely a consequence of the initial circulatory insufficiency (which itself was of uncertain aetiology). The considerable haemodynamic improvement seen when the acidosis was reversed is compatible with both suggestions.

We thank Professor R D Cohen for permission to report this case and Guy's poisons unit and the toxicology unit, St George's Hospital, London, for their help with butanone estimations.

¹ Black D. Misuse of solvents. *Health Trends* 1982;14:28-9.

(Accepted 16 September 1982)

The London Hospital, London E1 1BB

P G KOPELMAN, MD, MRCP, lecturer, department of metabolism and endocrinology

P Y KALFAYAN, MB, BCHIR, senior house officer, medical unit

Neurological deficit associated with *Mycoplasma pneumoniae* reversed by plasma exchange

A wide range of neurological disorders have been described in association with *Mycoplasma pneumoniae*.¹ Vascular, infective, and autoimmune causes have been postulated.^{2,3} We describe a case of transverse myelitis and psychosis associated with the appearance of antibodies to nervous tissue that partially responded to plasma exchange.

Case report

A 22 year old man with an eight-day history of sore throat and productive cough presented with increasing dyspnoea. A chest x-ray film showed patchy pneumonia. Within 48 hours he developed a flaccid paraplegia with sensory loss to the level of T3-4. Culture of cerebrospinal fluid grew no pathogens but *Mycoplasma pneumoniae* complement fixation test on the cerebrospinal fluid was positive at a titre of 1/128, with serum positive at 1/1024, suggesting a transverse myelitis associated with *M. pneumoniae* infection. He required intubation and over the next week sensory loss progressed to the level of C6; the serum titre of *M. pneumoniae* increasing to 1/4096. He remained alert and orientated. Cold agglutinins (anti-I, 1/32) were detected. The prothrombin time and partial thromboplastin time were abnormal with a positive result of screening test for coagulation inhibitors. Further investigation showed specific inhibiting activity to factors V and X, but without haemostatic defect: Strongly positive antibodies to myelin and nervous tissue were detectable by standard indirect immunofluorescence techniques using delipidated rat cerebellum (see table). Plasma viscosity was not increased.

Three plasma exchanges of 4 litres were performed over four days on an Aminco continuous flow cell separator. Replacement fluid consisted of 0.9% saline, plasma protein fraction, and fresh frozen plasma. After the second exchange the patient was able to maintain a normal PO_2 concentration without support and the sensory loss began to regress. Antineural cell and myelin antibodies were reduced over this period as were the clotting factor inhibitors and titre on *M. pneumoniae* complement fixation test (1/512).

Seven days after the last exchange the patient developed intermittent mental disturbance. He became confused and disorientated, experiencing visual hallucinations, restlessness, paranoia, and abnormal dreams. The transverse myelitis remained stable at T6, the plasma viscosity and *M. pneumoniae* complement fixation test titres were unchanged. The cerebrospinal fluid showed a raised protein concentration of 1.3 g/l. The electroencephalogram was diffusely abnormal and acute organic brain syndrome was diagnosed. Antineural cell antibodies increased and five more exchanges were carried out with a further pronounced reduction in antibody concentrations. The patient had no hallucinations after the first exchange and the abnormal dreams stopped after the second. He became lucid, alert, and orientated, with normal electroencephalogram. Concentrations of antibody in

Myelin and antineural cell antibodies in normal control and in patient before and after plasma exchanges, using indirect immunofluorescence on delipidated rat cerebellum*

	White matter		Granular layer (cell bodies)	Molecular layer (glial cells)
	Myelin	Glial cells		
Normal control	±	—	±	—
Patient:				
Before first exchange	+++	++	+	+
Before second exchange	++++	+++	++	++
Before third exchange	±	±	±	±

— = Negative. ± = Faint. + = Moderate. ++ = Strong. +++ = Very strong. ++++ = Intense staining.

*5 µm cryostat sections of cerebellum were delipidated by treatment for 30 minutes with dried acetone followed by three minutes in xylene. Immunofluorescence was carried out by standard techniques.

cerebrospinal fluid were not monitored during the period of exchange, though the raised protein concentration in cerebrospinal fluid returned to normal range. At this stage he remained clinically stable and no auto-antibodies have been detected since.

Comment

Neurological disease associated with *M. pneumoniae* infections (including psychosis and acute transverse myelitis^{2,4}) has been described in 2% of cases.¹ The aetiology is uncertain but an immunological basis has been thought more common in younger individuals.^{2,3} In our patient, culture of cerebrospinal fluid grew no pathogens and neurological symptoms progressed despite the normal viscosity and stable titres at *M. pneumoniae* complement fixation test. Immunofluorescence techniques using rat cerebellum demonstrated the presence of strong antibodies to myelin and neural cells and these antibodies fell sharply after plasma exchange, with rapid improvement in the neurological state. Later deterioration in mental state coincided with returning neural cell antibodies.

M. pneumoniae affects the immunogenicity of the erythrocyte I antigen proteins,⁵ leading to production of cold agglutinin. The organisms may produce similar changes that affect other systems during natural infection, leading to widespread autoantibody production.

The improvement after removal of the antibodies by plasma exchange in our patient suggests that autoantibody production following *M. pneumoniae* infection may be the basis of the neurological disorders.

¹ Noah ND. *Mycoplasma pneumoniae* infection in the United Kingdom 1967-73. *Br Med J* 1974;ii:544-6.

² Hodges GR, Fass RJ, Saslaw S. Central nervous system disease associated with *Mycoplasma pneumoniae* infection. *Arch Intern Med* 1972;130:277-82.

³ Clyde WA Jr. Neurological syndromes and mycoplasma infections. *Arch Neurol* 1980;37:65-6.

⁴ Westenfelder GO, Akey DT, Corwin SJ, Vick NA. Acute transverse myelitis due to *Mycoplasma pneumoniae* infection. *Arch Neurol* 1981;38:317-8.

⁵ Feizi T, Taylor-Robinson D, Shields MD, Carter RA. Production of cold agglutinins in rabbits immunized with human erythrocytes treated with *Mycoplasma pneumoniae*. *Nature* 1969;222:1253.

(Accepted 4 October 1982)

London Hospital, Whitechapel, London E1 1BB

F E COTTER, MRCP, registrar in haematology

D BAINBRIDGE, MB, BCHIR, senior lecturer in immunology

A C NEWLAND, MRCP, MRCPATH, senior lecturer in haematology