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SHORT REPORTS

A new danger associated with airgun pellet injuries

The removal of airgun pellets from patients is a common problem in accident and emergency departments. Locating the pellet can be difficult, even with radiographic control. We report a case in which a new type of pellet was encountered, part of which was radiolucent. This type of pellet could lead to difficulties in localisation and removal.

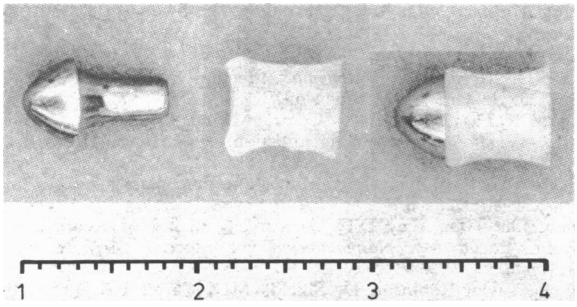
Case report

A 17 year old boy attended the accident and emergency department having been shot in the right side of the neck by an air rifle two hours previously. The pellet had not passed through clothing. On examination there was an entry wound of 5×5 mm on the skin overlying the right sterno-mastoid muscle, 6 cm above the clavicle. A small lump was palpable 5 cm posterior to the entry wound. Radiographs in two planes showed the pellet 5 cm posterior to the entry wound and 1 cm in depth.

A small incision was made over the pellet under image-intensifier control and the pellet removed from the superficial fibres of the trapezius. The wound was sutured. At this stage there was no evidence of further radio-opaque foreign bodies. The entry wound was explored and a small polyethylene cylinder, part of the original missile, removed from a depth of 1 cm. The entry wound was not sutured.

Comment

In this case the pellet was of two parts, a stainless steel head, and a polyethylene sleeve fitting over the tail (figure). On entry into soft tissue the sleeve had become detached from the head and had been left in the track of the missile, some distance from where the metallic head had come to rest. The danger with this pellet is that it splits into two components in the body and one of the components is radiolucent, making localisation by *x* ray examination impossible.



Hunting pellet from airgun showing stainless steel head and polyethylene sleeve: (left) components (right) assembled.

This type of hunting pellet (manufactured by Prometheus, Milbro) is designed for "exceptional penetration."[1] It has been on the market for 18 months, and we are told by the manufacturers that they have recently stopped marketing the pellet for a trial period of one year as they are concerned with its potential danger. The pellets, however, are likely to be on the market for some time until stocks have been used up.

The presence of this type of pellet is shown by its profile on radiographs: a rounded head with a narrow tail. Radiographs of all pellets in soft tissues should show the profile adequately, and if this type of profile is seen the presence of the polyethylene cylinder in the track of the missile should be suspected.

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Severe metabolic acidosis after ingestion of butanone

Misuse of organic solvents is an increasing problem.[1] We report a case of self-poisoning by ingestion of butanone, an organic solvent found in some household and commercial glues.

Case report

A 47 year old housewife born in south India was brought to the casualty department having been found deeply unconscious by her husband. The only relevant history was that she had developed chickenpox one week previously. On examination she was deeply unconscious and unresponsive to painful stimuli. She was hyperventilating, and her breath smelt of a popular glue. Peripheral circulation appeared normal, but blood pressure was 95/70 mm Hg and pulse 120 beats/min. A chickenpox rash was evident with related scratch marks. Blood-gas analysis shortly after her arrival showed oxygen pressure 11·3 kPa (85 mm Hg), carbon dioxide pressure 3·2 kPa (24 mm Hg), bicarbonate concentration 8·5 mmol(mEq)/l, anion gap 30·3 mmol(mEq)/l, and pH 7·19. Plasma electrolyte concentrations were sodium 138 mmol (mEq)/l, potassium 3·8 mmol(mEq)/l, and chloride 103 mmol(mEq)/l; urea concentration was 6·1 mmol/l (37 mg/100 ml), blood glucose concentration 15·7 mmol/l (283 mg/100 ml), haemoglobin concentration 13·4 g/dl, and white cell count $15·5 \times 10^9$/l. A plasma screen for salicylates and paracetamol yielded negative results.

She was given a slow infusion of 150 mmol (12·6 g) 8·4% sodium bicarbonate, after which there was obvious clinical improvement: the hyperventilation became less pronounced, blood pressure rose, and pulse rate slowed. Blood-gas tensions improved correspondingly: oxygen pressure was 10·4

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.

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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 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 autoantibodies have been detected since.

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.

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.

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