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SHORT REPORTS

Psittacosis and Disseminated Intravascular Coagulation

Psittacosis most commonly presents as mild pneumonia or pyrexia of undertermined origin. The following patient had severe pneumonia, myocarditis, pericardial effusion, and disseminated intravascular coagulation—a hitherto unreported complication.

Case Report

A 36-year-old man had felt ill for 10 days, and had dyspnoea, fever, and slight cough with yellow sputum. He was confused, grey, and unwell, being dehydrated with a temperature of 39·7°C, pulse of 140/minute, and blood pressure of 90/70 mm Hg. He was dyspnoeic at rest and had signs of inflammation in the left lung. A chest *x*-ray film showed extensive opacities in the left lower lobe.

During the next 36 hours he deteriorated. An arterial blood sample showed pH 7·39, Po_2 20 mm Hg, and Pco_2 36 mm Hg. He was intubated and treated with positive pressure ventilation with an oxygen concentration (FIO_2) of 100%. After one and a half hours of ventilation, his arterial blood gases were pH 7·30, Po_2 25 mm Hg, and Pco_2 33 mm Hg. He was given intravenous chloramphenicol, fucidin, tetracycline, and intramuscular streptomycin. On the sixth day he developed surgical emphysema on the right side of his neck, though there was no evidence of a pneumothorax. Chest *x*-ray films showed that his heart shadow was becoming larger and more globular in shape, indicative of pericardial effusion. There was never evidence of cardiac failure. Despite a blood transfusion on the fourth day, his haemoglobulin had dropped to 9·0 g/100 ml on the sixth and his platelet count was 47 000/mm³. Chloramphenicol was discontinued. On the seventh day the platelet count was only 40 000/mm³. The only clinical evidence of bleeding was skin bruising at the site of venous and arterial punctures. Full coagulation studies were performed (see table), and led to a diagnosis of disseminated intravascular coagulation; heparin (500 units intravenously hourly) was started and continued until the 15th day. Streptomycin and tetracycline were discontinued and doxycycline begun.

Coagulation Studies

Days after Admission	Platelets /mm³	Prothrombin Time (Seconds)	Kaolin-Cephalin Time (Seconds)	Fibrinogen Titre	F.D.P. µg/ml
5	Reduced	0	0	0	0
6	47 000	0	0	0	0
7	40 000	3 mins.	6 mins.	No clot	320
8	79 000	23	45	1:2	320
9	47 000	22	37	1:8	320
10	43 000	28	59	1:8	320
15	140 000	15	36	1:32	40
27	172 000	17	48	0	40

F.D.P. = Fibrinogen degradation products.

By the eighth day his condition and the results of the coagulation studies had improved. On the tenth day he developed multiple ventricular ectopic beats and a short run of ventricular tachycardia. Intravenous lignocaine and oral practolol abolished the ventricular arrhythmias. He then developed difficulty in speaking and was unable to swallow. A tracheostomy was performed and he was ventilated. He maintained his good progress and was discharged 30 days after admission. Antibody titre to psittacosis was 1/16 on the third and seventh day and 1/256 on the thirtieth day. When seen in follow-up one month later the patient was fit and well, apart from slight generalized weakness. He mentioned that he kept tropical fish and that his only contact with birds was in the pet shop from which he bought his fish food.

Discussion

Psittacosis usually produces a mild respiratory infection, though occasionally it may cause severe respiratory failure. The mortality is mainly from complications and approaches 5%.[1] The considerable hypoxia in this patient necessitated mechanical ventilation. Psittacosis may also involve the cardiovascular system,[2 3] and may damage the liver, producing jaundice. Here it resulted in disseminated intravascular coagulation (D.I.C.), which is characterized by the deficiency of fibrinogen and other clotting factors and thrombocytopenia. Several viruses may cause D.I.C., which may be transient, fatal, or pass unnoticed. The platelet count is almost invariably depressed, but, as here, there is no correlation between the absolute level of circulating platelets and the haemorrhagic tendency.

In treating D.I.C. the first step is to treat its cause—for example, septic shock.[4] D.I.C. may be reversed with intravenous heparin; in D.I.C. with infectious mononucleosis a suitable dose may be 12 000 units over 24 hours.[5]

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Severe Peripheral Neuropathy after Mandrax Overdose

Poisoning with methaqualone and diphenhydramine (which are available in this country as Mandrax tablets containing methaqualone 250 mg and diphenhydramine hydrochloride 25 mg) is quite common and the clinical features are well known.[1 2] This case illustrates an unusual manifestation of Mandrax poisoning causing severe peripheral motor and sensory neuropathy; no similar complication has apparently been described.

Case Report

A 73-year-old man complained of numbness, pain, and weakness in the legs and feet. He had been treated for Mandrax overdose six months ago when depressed, having taken 28 tablets in the evening. A few hours later the police found him wandering in the street confused. After gastric lavage at the casualty department he was transferred to the medical department, and treated supportively, recovering consciousness seven days later. The first thing he felt on recovery was that "my legs have gone." Later he developed numbness below the knees, together with burning pain and tingling in the legs and feet, and considerable weakness.

The legs showed definite wasting of the anterior compartments as well as of the calves. There was no dorsiflexion of toes or ankles and there was a bilateral foot drop. Both knee jerks were depressed, the ankle reflexes were present, and there was no plantar response. Vibration sense was lost below the iliac crests and joint position sense was virtually absent in the toes and grossly defective at the ankles. Below both knees sensation to touch, pin prick, hot and cold was lost. The results of extensive laboratory and radiological investigations were normal. An electromyogram showed findings characteristic of a peripheral neuropathy.

Comment

These findings leave no doubt that this patient had peripheral neuropathy and the time relation to the overdose would implicate Mandrax as a causative agent. The antihistamine component of Mandrax does not appear to contribute appreciably to its toxicity[3] and