PSITTACOSIS IN JOHANNESBURG

A CASE OF ATYPICAL PNEUMONIA

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Psittacosis is a specific infection of the psittacine or parrot family of birds including parrots, parakeets, cockatoos and budgerigars, and has long been known to be directly transmissible to man. Virus of the psittacine family infects many other non-psittacine species of birds, and canaries, pigeons, turkeys, domestic fowls, ducks, fulmar petrels and finches are known to contract the disease, and may infect man. The term 'ornithosis' is applied to non-psittacine infections. The evidence is that a vast avian reservoir of infection exists in domestic and wild birds.

Very few human cases are known to have been diagnosed in South Africa but the incidence of psittacosis is considerably higher than is realized and, with the increasing popularity of the budgerigar as a domestic pet, outbreaks of the disease can be expected.

The first cases of psittacosis were reported in 1880 and a pandemic originating in infected South American parrots occurred in Europe towards the end of the 19th century. In 1930 the virus responsible for the psittacosis-ornithosis group of infections was identified independently by several investigators. There is a high incidence of the disease in America and in the years 1952-55 the number of cases reported annually to the US Public Health Services varied from 135 to 495. 200 cases attributed to transmission from infected turkeys occurred in the US during 1954; otherwise infections have generally resulted from contact with infected psittacine birds.

The causative agent is one of a group of large filterable viruses of the psittacosis and lymphogranuloma inguinale group, antigenically related on the basis of complement-fixation tests.[1,2] Infected birds, especially newly imported birds, represent the main source of infection. Though usually ill, with listlessness, rough feathers, coryza and nasal discharge or bloody diarrhoea, they may be apparently healthy. Inhalation of dust from cages or aviaries contaminated by infected discharges, inhalation of feathers, and bites by infected birds, are the main source of infection. Hence it is thought that infection is commonly contracted during the cleaning of the bird's cage, as in the case reported here. Transmission from man to man sometimes occurs but is rare.[3-5] Clinical infection is considered to be rare in children and infants but that this may not be so is suggested by Berman *et al.*,[6] who discuss a number of cases occurring in infants in Israel in 1953 and 1954.

The Law

In 1930, after a serious outbreak of psittacosis in Britain with many deaths, a complete ban on the importation of psittacine birds into Britain was imposed. The ban was lifted in December 1951. Many outbreaks and some deaths from the disease have since been reported, and the embargo has been re-imposed. An outbreak of psittacosis, the first in 10 years, occurred in New York shortly after revocation of a law prohibiting the sale, breeding or importation of psittacine birds in New York state and New York city early in 1952.[4] In South Africa, Proclamation No. 211 of 19 May 1931, issued under sections 76 and 84 of the Public Health Act, prohibits the introduction into the Union by land or sea of all birds of the parrot family (psittacidae) and lovebirds and budgerigars (variously classified as Psittacidae or Psittaculidae) except where a permit therefor has first been issued by the Secretary for Public Health, subject to compliance with any conditions or requirements, such as a period in quarantine, stated in such permit. Psittacosis is a 'proclaimed disease' under the Stock Diseases Act 14 of 1911, and in practice permits are issued by the Veterinary Department. Permits are fairly freely issued but for restricted numbers and subject to veterinary certificates and quarantine of birds. Psittacosis, according to notifications, is by no means common in birds in this country.

CASE HISTORY

On 2 October 1956, during a polio epidemic, a European male aged 33 years, a fitter and turner, was admitted to the Johannesburg Fever Hospital with a provisional diagnosis of 'encephalitis', compaining of headache and low back pain for 3 days and cough for 1 day. He awakened on the morning of the commencement of his illness with a generalized throbbing headache felt particularly in the occipital and retro-orbital region and aggravated by moving and coughing. His eyes felt 'as if they would burst out', and he had aching lumbar pain (he had been diagnosed as having influenza), a sore throat, and an irritating non-productive cough which started on the day of admission to hospital, but no chest pain. He complained of difficulty in breathing. Questioning revealed anorexia of 2 days duration with nausea and constipation.

The patient lived in Johannesburg and had not been away for several months. He had been seen by one of us 7 years previously and had then been diagnosed as suffering from Bornholm disease.

On admission his temperature was 100°F and pulse rate 108 per minute. He appeared very ill, with marked irritability and congestion of the face and neck, conjunctival injection, and marked photophobia. The pharynx had a red granular appearance and the tongue was heavily coated. The cardiovascular system was apparently normal. Deep inspiration brought on paroxysms of coughing and occasional rhonchi were heard in the chest. The abdomen was slightly distended and there was tenderness in the right hypochondrium. The liver and spleen were not palpably enlarged. The tendon reflexes were depressed, but no paresis was found and no stiffness of the neck or back. The cranial nerves and fundi were normal.

During the 3 days after admission the temperature ranged between 100 and 103·4°F. He complained of very sore throat and severe headache. The paroxysmal cough persisted and he had one small epistaxis. Scanty pink macules resembling typhoid spots were observed in both flanks. Moist rales were heard in

the left axillary region. A portable X-ray on 6 October 1956 revealed a consolidation in the left mid-zone, and at this stage the patient coughed sputum streaked with bright red blood (Fig. 1).

Laboratory investigation was carried out on admission as follows: Blood: white-cell count 9,000 per c.mm. (polymorphs 85%, lymphocytes 11·5%, monocytes 3·5%) CSF: Clear; polymorphs 3 per c.mm., lymphocytes 4; protein 31 mg.%, sugar 65 mg.%. Blood culture in bile and broth, negative. Rickettsial

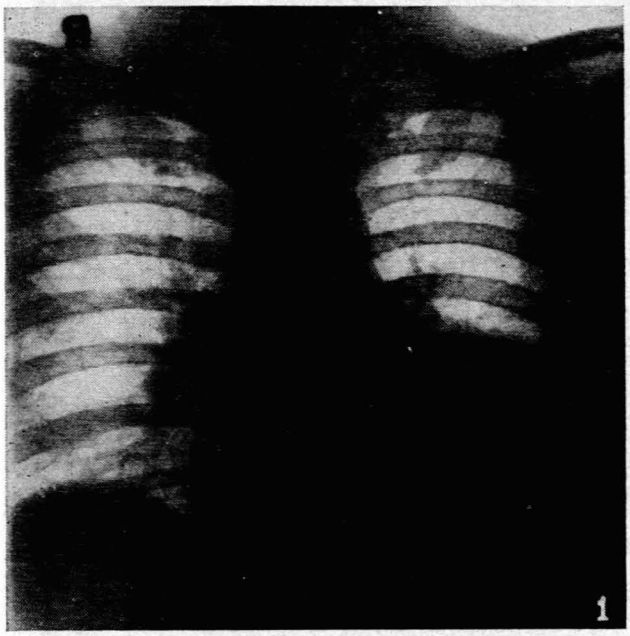

Fig. 1. X-ray of chest on 6 October 1956.

complement-fixation tests, negative. Cold agglutinins not present in the blood. Stool and urine cultures, negative for typhoid. Urine: No albumin, no sugar, bilirubin absent, urobilin +. Blood bilirubin: 0·4 mg.% on admission, 1·2 mg.% 6 days later.

A diagnosis of atypical pneumonia was made and investigation instituted to exclude psittacosis and other causes. On questioning, the patient said he had acquired a budgerigar on the day before his illness, the only contact with any other bird having been 2 weeks previously, when he had helped a friend clean out and build a ladder in the cage of his budgerigar.

The patient remained in hospital and was febrile for 8 days. Penicillin was administered from the 6th to the 11th day in dose of ½ million units 6-hourly and again in reduced dose from the 13th to the 17th day. After the temperature fell convalescence was uneventful.

A positive psittacosis-lymphogranuloma complement-fixation test was obtained in titre 1 : 10 from blood taken in the first week of illness. One week later the titre had risen to 1 : 100, and 10 days later to 1 : 200, indicating that the illness had in fact been psittacosis.

Enquiry revealed that the budgerigar inhabiting the cage worked on by the patient 2 weeks before his illness had never been well and that a canary had died in the same cage a short time before. Examination of the bird at the Poliomyelitis Research Foundation Laboratories, Johannesburg, revealed the presence of psittacosis virus.

DISCUSSION

Clinical Aspects of Psittacosis. Both the clinical manifestations and the severity of the disease are variable. The incubation period is usually 10 days, with extremes of 4-15 days. An abrupt onset is usual. In mild cases there is severe headache, with photophobia, malaise, fever, and pain in the back and limbs, the picture being that of 'pyrexia of unknown origin' or an influenza-like illness lasting 3 days to a week with uneventful convalescence. In severe cases signs and symptoms of lung involvement occur after a few days of intense headache, profound weakness, prostration and myalgia. Tachypnoea, chest pain (which may be pleural), and cough, either unproductive or with haemoptysis, are symptoms. There may be severe abdominal pain, nausea, vomiting or diarrhoea. Epistaxis and conjunctival injection or subconjunctival haemorrhage occur. There may be neck stiffness, and sore throat is common.

Diagnosis. The most generally applicable test is the demonstration of psittacosis antibody in the patient's serum. Two specimens are required, one in the first week and one about the 21st day. Chemotherapy may delay the appearance of antibodies for 3-6 weeks or even prevent their appearance. The finding of antibody in low titre in the first specimen suggests that the patient has had an infection with a virus of the psittacosis-lymphogranuloma group. A high titre of 1 : 30 or over in the first specimen is suggestive of the disease, and a 4-fold or greater rise in the second specimen is diagnostic. Meyer and Eddie[7] state that examination of repeated specimens for complement-fixing antibodies with a 2- to 4-fold increase in titre plus evidence of pulmonary involvement are sufficient to warrant the diagnosis of psittacosis; also that a titre of 1 : 16 obtained from a patient with clinical manifestations of psittacosis may be considered positive. It is also possible to isolate the virus from the patient's blood in the early days of infection and from the sputum when lung involvement has occurred. The isolation of virus from a bird contact, as in the case reported, gives further evidence of the nature of a suspected infection.

The Carrier State. Although the healthy carrier state is well known in birds, its occurrence in man is probably rare, although Eddie[8] reported a human carrier of psittacosis who harboured the virus for 10 years after a severe acute attack. Patients have spread the disease to nursing contacts and others.

Treatment. Case fatality has decreased from 50% to 2% with antibiotic therapy. Good results have been reported with the use of penicillin, which should be administered in large doses up to 2 million units per 24 hours. Chlortetracycline (Aureomycin) and oxytetracycline (Terramycin) have given equally good and possibly better results; chloramphenicol (Chloromycetin) is less active. Antibiotics are probably virustatic and not virucidal, hence the failure of chlortetracycline to clear up the carrier state in birds.[9]

Résumé. The clinical picture of psittacosis is usually that of an acute disease with an incubation of about 10 days. The symptoms are generally malaise, fever, severe headache and myalgia, and symptoms of nausea, vomiting and photophobia. Often no other abnormal signs are found and after a few days of pyrexia the patient recovers. In severe cases, towards the end of the first week there is evidence of pulmonary consolidation, a cough, usually unproductive, and chest pain which may or may not be pleural in nature, and the spleen may enlarge. The pulse is often relatively slow. Rose spots are noted in a few cases and a scarlatiniform rash has been described. Without chemotherapy the disease carries a high mortality. The symptoms of psittacosis are liable to be confused with those of influenza, 'Q' fever and other forms of atypical pneumonia, typhoid fever, and meningo-encephalitis, especially in cases characterized by

conjunctival injection and photophobia such as leptospiral meningo-encephalitis.

SUMMARY

1. A case of psittacosis is described, presenting with atypical pneumonia.

2. Diagnosis was established by the demonstration of complement-fixing antibodies in diagnostic titre for the psittacosis and lymphogranuloma venereum virus group and the isolation of virus from a budgerigar contact.

3. The clinical features of psittacosis are discussed.

4. Attention is drawn to an increasing incidence of the disease abroad and the suggestion is made that psittacosis is not very uncommon in South Africa.

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