Clinical Reports

Psychosis in mycoplasma infection

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Summary: This report describes a patient with psychosis due to a *Mycoplasma pneumoniae* infection. Although he received specific treatment for this infection, the diagnosis was only confirmed after clinical recovery. The neuropsychiatric complications of mycoplasma infection are discussed.

Introduction

In the assessment of psychosis, it is important to distinguish between organic and functional psychiatric illness. Some organic disorders, however, may present without neurological signs or impairment of level of consciousness, and the symptomatology may suggest virtually any form of functional psychiatric illness. Organic brain syndrome due to an infectious agent usually presents as a rapidly developing illness with headache, prostration and features of central nervous system involvement. Pyrexia is variable and may be absent. Presentation as a psychiatric illness was first recognized in syphilis and later in early epidemics of encephalitis lethargica. Viruses such as herpes simplex and zoster virus, mumps virus, measles virus and the human immunodeficiency virus are the usual infective agents.¹ We report a case of *Mycoplasma pneumoniae* infection, in which serological tests for this organism were persistently negative throughout the clinical course of the illness, but became positive after recovery.

Case report

A 35 year old Eurasian male scientist was brought, under police restraint, to casualty and was compulsorily admitted. He was irritable, aggressive and grandiose. His mother reported that he had complained of headaches, intermittent fever and dry cough during the preceding 10 days. He had consulted his general practitioner and was treated with erythromycin for 3 days prior to his admission. He had no past personal or family history of psychiatric illness and denied any psycho-active drug or alcohol abuse.

He had sustained superficial lacerations caused by

glass in his attempt to fly through a window, but otherwise his physical examination was normal. He was afebrile and remained so during his admission. His level of consciousness was unimpaired and he had no evidence of meningism or focal neurological abnormalities.

On examination of his mental state, he showed social disinhibition and inappropriate sexual behaviour. He had pressure of speech and his mood was characterized by irritability and elation. His stream of thought was disordered. There were, however, no Schneiderian first rank symptoms whose presence might have lent weight to a diagnosis of schizophrenia. On cognitive testing he was oriented in time, place and person. Attention and concentration were unimpaired on digit recall and serial seven testing, despite his distractibility. He had patchy recall of the previous 24 hours but formal testing of short term memory by the Babcock sentence was unimpaired. His intelligence was assessed as above average. The patient had no insight into his condition.

On admission his haemoglobin was 11 g/dl and white cell count was $7.2 \times 10^9/l$. Nine days later his haemoglobin has fallen to 9.8 g/dl. His blood film showed marked auto-agglutination, and his reticulocyte count was 4%. Cold agglutinins were detected. The direct antiglobulin test was negative. Routine biochemistry and thyroid testing were normal. Urine screening for psycho-active drugs was negative. His chest X-ray showed patchy right upper lobe consolidation. Extensive investigations in search of an infective agent were negative. The electroencephalogram was abnormal, showing generalized slow wave forms indicative of an encephalitic process. A computerized tomographic scan of the brain was normal. Examination of the cerebrospinal fluid was normal.

Over the week following admission, his psychotic

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symptoms fluctuated markedly in response to small doses of chlorpromazine. He was treated with 100 mg of chlorpromazine nightly, and became asymptomatic. When this drug was withdrawn on the fourth day, his hypomanic symptoms reappeared. For the next week he received 50 mg of chlorpromazine nightly. Twelve days after admission he was able to discontinue psychotropic medication and remain well.

The atypical course of his illness, the history of a preceding upper respiratory tract illness and his abnormal electroencephalogram, led us to search for an organic cause. He was discharged from hospital with a normal mental state and on no psychotropic medication 15 days after admission with a clinical diagnosis of psychosis following a presumed *M. pneumoniae* infection. Erythromycin had been commenced in hospital and continued as an outpatient.

Six weeks after discharge, IgM antibody to *M. pneumoniae* at a titre of 1 in 320 was detected, having been negative on days 1, 7, 10 and 30. Four months later he remained well. His chest X-ray and his haemoglobin were normal.

Discussion

Mycoplasma pneumoniae accounts for 2.4–14% of community acquired pneumonias in the UK.² Most individuals infected with *M. pneumoniae* develop mild upper respiratory tract infection but only 3–10% have clinically apparent pneumonia.³

Over the last 30 years there have been many reports of neuropsychiatric complications of *M. pneumoniae* infection. These have comprised meningoencephalitis, meningitis, polyradiculopathy and psychosis.⁴ The

incidence of these associations is said to be between 0.1–7% depending on the severity of the illness.⁵

Psychosis is least commonly reported; only eight cases have been described to date.^{6–8} In those cases for which information is available, psychosis was preceded by malaise and pulmonary symptoms sufficient to require hospital admission. Interestingly, psychosis was the presenting feature in our patient whose chest complaints were trivial despite radiological changes. Most cases described had normal cerebrospinal fluid and an abnormal electroencephalogram as in this case. Approximately half the patients with *M. pneumoniae* infection develop cold agglutinins but significant haemolysis is rare.³ It is possible that the fall in haemoglobin in our patient may have been due to haemolysis. Although the finding of cold agglutinins is not specific for *M. pneumoniae*, the strong association known to exist was a major diagnostic indicator in this case.

The mechanism by which *M. pneumoniae* affects the nervous system is unknown. Recently, the organism has been grown from the cerebrospinal fluid but it has never been isolated from the brain.⁹ Controversy exists as to whether it is direct invasion by the organism, or an autoimmune response which causes the neuropsychiatric sequelae.¹⁰ The effect of treatment on extra pulmonary complications is not known.

The atypical clinical course of this man's psychotic illness led us to suspect an organic brain syndrome. The diagnosis was only substantiated after recovery, when serological tests became positive. The need for repeated assays is emphasized, as the diagnosis of organic brain syndrome carries a different prognosis and social significance from that of psychiatric illness.

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