Clinical Reports

Lymphocytic meningitis as the sole manifestation of Q fever

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Summary: A young man who presented with a 3 week history of fever and severe headache accompanied by mild leukocytosis, was found to have lymphocytic meningitis due to *Coxiella burnetii*. Thus, Q fever can present as lymphocytic (aseptic) meningitis responsive to tetracycline with no evidence of pulmonary involvement.

Introduction

The nervous system is often involved in rickettsial diseases. The pathogenesis is related to an invasion of vascular endothelium by rickettsia, causing a vasculitis which may also occur in the central nervous system.¹ Thus, a variety of neurological symptoms may be encountered, and sometimes they dominate the clinical picture.² *Coxiella burnetii* is different from most other rickettsia and it is distinctly unusual to diagnose Q fever lymphocytic meningitis in the absence of any pulmonary involvement.

Case report

A previously healthy 29 year old man was admitted because of continuous fever (around 38°C), nausea and severe bifrontal headaches of 3 weeks duration. A complete physical and neurological examination was normal, with no signs of meningeal irritation. The white blood cell count was $12-14.9 \times 10^9/l$ (77% neutrophils) but all other routine tests including sedimentation rate, urinalysis and liver chemistry tests were normal. Autoantibodies were not found. The electrocardiogram, chest and sinus radiographs as well as echocardiography, brain computed tomographic scan and electroencephalogram were normal. Lumbar puncture gave cerebrospinal fluid (CSF) which contained 120 cells per litre of which 90% were mononuclear, glucose 2.8 mmol/l (blood glucose 6.6 mmol/l) and protein 0.21 g/l. Direct

Gram stain, tests for tuberculosis and serial cultures and serology tests were all negative, except for a positive indirect immunofluorescent antibody test for Q fever in serum, but not in the cerebrospinal fluid (CSF). The test showed phase-II antibodies (1:800) as well as specific IgM antibodies, while phase-I antibodies were not found. The patient had no history of recent animal exposure, staying in rural areas or intake of untreated milk products. Doxycycline, 100 mg twice daily, was administered for 2 weeks, resulting in a rapid and complete disappearance of all symptoms and findings.

Discussion

The entity of rickettsial lymphocytic meningitis or encephalitis is well recognized.³ A variety of neurological symptoms and signs may be encountered in the course of rickettsial diseases, including marked headache, altered mentation and multifocal neurological signs.^{4,5} CSF abnormalities have been reported in about two-thirds of patients with Rocky Mountain spotted fever,² usually showing a mild to moderate mononuclear pleocytosis, mildly elevated protein and normal (or slightly decreased) glucose levels. This pattern is similar to the findings in our patient. However, in Q fever both meningoencephalitis and abnormal CSF are considerably less frequent.⁶ Though customarily included among the rickettsial diseases in most textbooks, *Coxiella burnetii*, the aetiological agent of Q fever, is different from most of the rickettsiae in several important characteristics. First, humans are usually infected by inhalation of contaminated aerosols with no need for a vector,

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Accepted: 18 January 1993

since *C. burnetti* is a highly resistant micro-organism; second, it enters the host cells by a passive mechanism unlike true rickettsiae and a rash almost never occurs in acute Q fever; third, patients lack cross-reacting antibodies to proteus X strains (Weil-Felix reaction).¹ Therefore, rickettsaemia often follows a proliferation of micro-organisms in the lungs and a self-limited febrile illness with or without pneumonia is the most common form of Q fever.^{1,6} Nevertheless, Q fever can present as an acute hepatitis without any pulmonary manifestations⁶ and our report demon-

strates that it can also present as isolated lymphocytic meningitis. A lumbar puncture is not usually performed on patients with Q fever. However, the common occurrence of severe headache noted in 65–90% of the patients,⁶ as well as the biology of the infection¹ suggest that lymphocytic meningitis in Q fever may occur much more frequently than realized, and in fact, *C. burnetti* has been isolated from the CSF of such patients.⁷ Serology for rickettsial diseases including *C. burnetti* should always be performed in unexplained lymphocytic meningitis.

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Postgrad Med J (1993) **69**, 637–640

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Pseudotumour cerebri associated with arteriovenous malformations

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Summary: The association of intracranial hypertension and arteriovenous malformations is described in two patients. Both patients had the typical clinical features of pseudotumour cerebri and were found to have intracranial arteriovenous malformations on arteriography. The mechanism of raised intracranial pressure in patients with arteriovenous malformations is discussed.

Introduction

The recognition of a syndrome of raised intracranial pressure without ventricular distension dates from 1897.¹ The number of different putative causes and associations has expanded over the years.^{2,3} It is known that pseudotumour cerebri may occur with no identifiable cause, or secondary

to various specific disorders.⁴ The common factor in producing the raised pressure is a disturbance of the normal dynamics of cerebrospinal fluid production and absorptions.^{5–7} By definition, the term pseudotumour cerebri excludes patients with raised intracranial pressure secondary to space occupying lesions producing a mass effect, or patients with ventricular distension.⁸ However, there is a small group of patients with pseudotumour cerebri who do have a focal vascular lesion, usually involving the venous sinuses, producing raised intracranial

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Accepted: 18 December 1992