Unusual presentation of primary klebsiella meningitis: successful treatment with cefotaxime

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Summary

A man who presented with lumbar backache subsequently developed signs of meningitis. The causative organism was proved to be *Klebsiella pneumoniae*. Despite treatment with chloramphenicol and amikacin, the condition progressed until cefotaxime was added to the treatment regimen. The patient made a good recovery. This is the first report of the use of cefotaxime in klebsiella meningitis.

KEY WORDS: klebsiella pneumonia, primary klebsiella meningitis, cefotaxime.

Introduction

Meningitis caused by *Klebsiella pneumoniae* is a medical rarity. In a series of 3377 cases of meningitis, only 7 were attributed to the Friedlander bacillus (Jacob and Top, 1948), while Ransmeier and Major (1943) estimated that this organism was responsible for 3 out of 3714 cases of meningitis reported from the large cities of the United States.

Before the era of antibiotic therapy, mortality from this infection was virtually 100%, and the disease still poses formidable therapeutic problems (Soscia, Di Benedetto and Crocco, 1964). We report a man in whom non-specific backache preceded the onset of the classical signs and symptoms of bacterial meningitis by some 6 days. The responsible organism was proved to be *Klebsiella pneumoniae*, and the infection was eventually controlled with cefotaxime sodium (Claforan).

Case report

A previously healthy 49-year-old man was admitted to the orthopaedic service with a history of pain in the lumbar region. The symptom had developed gradually over 3 days until medical attention was sought. The nature of the pain was described as

aching, sometimes stabbing, and was occasionally radiated as a shooting pain up the spine. Examination at this time was within normal limits, and routine blood investigations, X-rays of the chest and spine and bone scan were normal.

Three days after admission, the patient developed a pyrexia and became drowsy and confused. He was transferred to a medical ward.

On examination he was drowsy but rousable, and disoriented for time and place. His temperature was 39°C, blood pressure 160/90 mmHg and pulse rate 100 beats per min and regular. There was marked nuchal rigidity and positive Kernig's and Brudzinski's signs. Papilloedema was not present, and the remainder of the neurological assessment was within normal limits. Heart, lungs, abdomen and ears, nose and throat were normal.

Abnormal laboratory findings were a leukocyte count of $16.5 \times 10^9/l$ and blood glucose of 11.9 mmol/l. The remainder, including serum electrolytes and urea, haemoglobin, liver function tests and serum enzymes, were normal. Electrocardiogram and X-rays of the chest, abdomen, skull and sinuses were normal. The first of a series of blood and urine cultures were negative. Lumbar puncture yielded turbid cerebrospinal fluid under raised pressure (280 mmH₂O); analysis showed protein 1.38 g/l, glucose 1.9 mmol/l, chloride 118 mmol/l, leukocytes $0.385 \times 10^9/l$, lymphocytes $0.19 \times 10^9/l$, red cells $0.02 \times 10^9/l$ and the presence of numerous Gram-negative bacilli. Culture of the fluid subsequently yielded *Klebsiella pneumoniae* sensitive to chloramphenicol, tobramycin, amikacin and cefotaxime. The same organism was demonstrated on the second blood culture; urine, stool and sputum cultures were negative on 4 occasions.

Before the results of the cultures and sensitivity tests were available, the patient was treated with penicillin G, 2 million units every 2 hr, and chloramphenicol, 1 g every 6 hr by intravenous injection. On receipt of the results 2 days later, amikacin, 500 mg

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twice daily, intravenously was substituted for the penicillin G.

Over the following 2 days, the patient's condition deteriorated; the temperature, which had initially normalized, became elevated once more, the patient was no longer rousable, but responded to noxious stimuli, and coarse crepitations and rhonchi were present in the left lung base. Renal and liver functions remained normal. Cefotaxime, 2 g every 4 hr intravenously was added to the treatment regimen.

Over the next 10 days, the patient's clinical condition improved. By the fourth day, he was afebrile and awake but disoriented; his chest was clear by day 7, and after 10 days he was alert and oriented. Lumbar puncture, 2 weeks after treatment with cefotaxime, yielded sterile, normal cerebrospinal fluid. Blood cultures were negative and routine blood investigations were normal at this time. All antibiotics were stopped one week later.

Discussion

The presentation of the infection is noteworthy. Generally, the clinical picture of klebsiella meningitis is similar to that of acute meningitis due to other bacteria, namely fever, headache, malaise and varying degrees of confusion with neck stiffness. The reason that our patient sought medical attention was lumbar back pain, a most misleading presentation. In retrospect, this suggested that osteomyelitis of lumbar vertebrae might be the primary source of the organism; no radiological evidence could be adduced in support of this, and bone scan was normal. The precise nature of the backache is still obscure.

K. pneumoniae is very rarely found as a primary invader of the central nervous system, the portal of entry usually being found in the ear, mastoid, sinuses, respiratory tract, wound infection or genitourinary or

respiratory tract, wound infection or genitourinary or gastrointestinal tracts. Of the 30 cases reviewed by Ransmeier and Major (1943), infections of the middle ear, mastoid and sinuses were found in more than half the adults. No such primary site was evident in our patient.

Hyperglycaemia was present in our patient and it is not infrequently found in klebsiella infections. Indeed, Ayvazian (1948) found that diabetes mellitus was present in 23% of the cases and furthermore, 43% of extrapulmonary klebsiella infections were associated with diabetes, as compared to 12% of pulmonary infections. It is possible that hyperglycaemia enhances capsule formation and thereby increases the virulence of the klebsiella bacillus (Hoogerheide, 1939). Once the meningitis had subsided, glucose tolerance was found to be normal in our patient.

Despite modern antibiotic therapy, klebsiella meningitis has a 50% or higher mortality (Soscia *et al.*, 1964). We believe the use of cefotaxime to have been instrumental in reversing the rapidly progressive downward course in this patient. To our knowledge, this is the first report of the use of cefotaxime in klebsiella meningitis.

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