



Meningitis With Minimal Symptoms Caused By *Klebsiella Pneumoniae* in An Elderly Diabetic : Case Report And Literature Review

KEYWORDS

Meningitis, *Klebsiella pneumoniae*, diabetic.

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ABSTRACT Bacterial meningitis can have varying clinical presentations and cause considerable mortality and morbidity. *Klebsiella pneumoniae* is associated with meningitis in neonates and very rarely in adults. We here report an atypical case of meningitis due to this pathogen in a diabetic patient, wherein fever was present but classical signs and symptoms of bacterial meningitis were absent.

Introduction:

Acute bacterial meningitis is a common cause of mortality and morbidity in developing countries like India with *S. pneumoniae* and *N. meningitidis* being the commonest causes (1). The classical triad of fever, neck rigidity and altered sensorium is not present in the majority of cases but one study reported that 95% of the patients had at least two of fever, headache, neck rigidity and altered sensorium (2). *Klebsiella pneumoniae* is a Gram negative, non-motile capsulated bacterium, usually associated with neonatal meningitis, although recently it has also been associated with meningitis in adults(3). In fact, *K. pneumoniae* was the most common pathogen among adults with bacterial meningitis in a study at Taiwan(4). Gopal *et al* reported a case of meningitis caused by *K. pneumoniae* in a 34 year old diabetic patient with neurological symptoms in a study from South India. However, diabetes with meningitis having none of the typical features is rare(5). We report here one such case.

Case report:

A 55 year old female patient, resident of Bihar, East India, was admitted after she first presented to the General Medicine Outpatient department with the chief complaints of pain and weakness of both lower limbs for ten days. She reported a febrile illness about ten days before this presentation. She was treated for this by a local practitioner. The temperature settled within two days. No written record was available for this episode. Five days later she developed sudden onset weakness of lower limbs along with aching pain in both legs. She was unable to walk or stand up from a squatting position. There was no neck rigidity or history of vomiting. The weakness did not progress further after the onset. There was no bowel or bladder involvement. Her past medical history included Type II Diabetes for which she was taking Metformin 500 mg twice daily. On initial examination, she was conscious and oriented. Power was normal in the upper limbs. Mild weakness (Grade IV to IV+) was noted in hip flexors and knee extensors. Tone was not increased. Tendon jerk was diminished in both upper and lower limbs. It was non-elicitable in left knee and both ankles. Plantar reflex was absent on the right side. There was flexor response on the left side.

There was no sensory deficit of any modality. Notably, during the first 48 hours of admission she had two readings of high temperature of 99.6° and 100.2° F respectively.

Investigations revealed raised random and fasting blood sugar levels (160 mg/dl and 145 mg/dl respectively). Total Leucocyte count was 13,000 /mm³. Chest X-ray was normal. Nerve conduction study showed sensory-motor polyneuropathy with predominantly axonal pathology and reduced F wave response suggesting radicular pathology. On biochemical examination of the Cerebrospinal Fluid (CSF), protein was normal (30 mg/dl). Cell count was 20 cells/mm³. Culture of CSF on Blood agar, Chocolate agar and MacConkey agar without NaCl yielded Lactose fermenting, mucoid, non-hemolytic shiny colonies after 24 hours of aerobic incubation at 37 °C. The isolate revealed Gram negative short, stout bacilli, and was catalase positive, Oxidase negative using sterile commercial filter paper strips impregnated with 1% TMPPD (HiMedia labs, New Delhi, India). It was Indole negative, urease positive using Christensen's Urea agar slant, non-motile using Hanging drop and Semisolid agar stab techniques, utilised Citrate and produced gas but no H₂S in TSI slant. Based on these phenotypic traits, it was identified as *Klebsiella pneumoniae*. Antibiotic susceptibility test was carried out using the following antibiotic discs: Amoxiclav(30 µg), Levofloxacin (5 µg), Chloramphenicol(30 µg), Imipenem (10 µg), Ceftriaxone (30 µg) and Azithromycin(15 µg) by Kirby-Bauer disc diffusion test as per CLSI protocol (6). *P. aeruginosa* ATCC strain 27853 was kept as susceptible control. The bacterial isolate was susceptible to Amoxiclav, Azithromycin, Ceftriaxone, Chloramphenicol, Imipenem and Levofloxacin. The patient was treated with intravenous Ceftriaxone 2 gram twice daily for 10 days. She was also started on intravenous Dexamethasone 8 mg twice daily which was changed to oral Prednisolone 20 mg once daily on the fourth day. Ranitidine was given for gastric protection. Metformin dose was increased and Glimeperide was added for better blood sugar control. She was discharged on a tapering dose of Prednisolone after 10 days of intravenous antibiotics. She started showing improvement after the first few days of treatment. By the time of discharge, she could mobilize independently.

Discussion:

This case was a diagnostic challenge because of its atypical features. Lower motor neuron type of weakness without objective sensory loss or bladder and bowel involvement following a febrile illness suggested Guillain Barre Syndrome (GBS). However, the duration between fever and onset of weakness was too short. Moreover, the weakness was not typically ascending in nature as is the case with GBS. On nerve conduction studies, GBS typically shows a demyelinating pattern although predominantly axonal weakness may be seen (7). The nerve conduction abnormalities could also be explained by Diabetes but radicular involvement went against this. The latter raised the possibility of arachnoiditis as a possible diagnosis. Acute bacterial meningitis can lead to radiculopathy but this is usually a late complication (8). Magnetic Resonance Imaging (MRI) of the spine could not be done as the patient was unable to afford it. Intravenous immunoglobulin therapy for possible GBS was also not considered for the same reason. Presence of normal CSF protein could be due to partially treated acute bacterial meningitis.

Despite the availability of new antibiotics and the improvement in clinical care over decades, bacterial meningitis still remains an illness with a high morbidity and mortal-

ity(9). *Klebsiella pneumoniae* is usually a rare cause of meningitis in Europe and United States but a common etiological agent in countries like Taiwan and Korea(10). Underlying co-morbidities or predisposing factors for *K. pneumoniae* meningitis include diabetes, alcoholism and chronic liver disease(11). The rarity of occurrence of cases outside the continent of Asia raises the possibility of ethnicity or country of origin predisposing individuals to invasive disease(11). *K. pneumoniae* isolates with hypermucoviscosity are, in fact, capsulated and 'in-vivo', anti-opsonic, anti-phagocytic and are associated with severe disease(11). Isolates with K1 and K2 capsular antigen types are reportedly the most invasive pathogens(11). Absence or paucity of the classical manifestations of meningitis in this patient could be due to diabetes. Very few such reports are from India. One series mentions occurrence of *K. pneumoniae* meningitis as 4% of overall cases, isolation was more in neonates and very rare in adults(12). To the best of the authors' knowledge, this is the first reported case of possible meningitis caused by *K. pneumoniae* in an adult diabetic female patient from Eastern India, with atypical symptoms. This report highlights the importance of isolation of *K. pneumoniae* from CSF in adult diabetic patients and the fact that isolation should not be regarded as contamination or false positive if typical clinical features are absent.

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