

Pseudomonas aeruginosa meningitis in an elderly - A case report

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ABSTRACT

Community-acquired meningitis caused by *Pseudomonas aeruginosa* is rare and has a very high mortality rate. We describe the case of a 74-year-old well-controlled diabetic female who spontaneously developed *Pseudomonas* meningitis causing altered sensorium, confusion, and irritability. Cerebrospinal fluid grew *P. aeruginosa* sensitive only to meropenem. Blood cultures were sterile. Noncontrast computed tomography of the brain showed age-related cortical atrophy. The patient responded well to 15 days of broad-spectrum antimicrobial therapy with intravenous meropenem and levofloxacin. This is the first reported case of spontaneous community-acquired *P. aeruginosa* meningitis in a patient with no history of head trauma, neurosurgery, or lumbar spinal puncture.

Key words: Fever, Headache, Irritability, Meningitis, Neck pain

Community-acquired meningitis caused by *Pseudomonas aeruginosa* is a rarity and has a very high mortality rate [1]. Meningeal involvement is almost always secondary to a surgical procedure or head trauma and occasionally bacteremia. The cerebrospinal fluid (CSF) profile of *P. aeruginosa* meningitis is similar that of pyogenic meningitis. Early recognition and treatment are of utmost importance to reduce the mortality and complications. We describe a rare case of a 74-year-old otherwise healthy female with *P. aeruginosa* meningitis who presented with atypical manifestations and ultimately had good outcome.

CASE HISTORY

A 74-years-old female was admitted in the emergency with the history of being found unconscious at home by her relatives. Thereafter, she was in altered sensorium, irritable, and could not recognize her relatives. She stayed alone at home and was apparently alright until the previous day night. There was no history of fever, headache, and convulsions. She had Type 2 diabetes mellitus (controlled with metformin) and hypothyroidism. There was no history of cardiac ailment or any addictions.

On admission, she was disoriented, afebrile with pulse rate of 98/min, blood pressure 140/80 mmHg, and respiratory rate 32/min. General survey did not show pallor, icterus, generalized lymphadenopathy, and cyanosis. Examination of the central nervous system revealed very irritable, extremely restless patient without any focal neurological deficit. Her Glasgow coma scale was E₂M₅V₂ (9/15). Her pupils were normal sized with sluggish reaction to light. Doll's eye reflex was normal. Plantar responses were bilaterally up going. There was terminal neck stiffness as evidenced by increase in irritability on complete neck flexion.

A provisional diagnosis of meningoencephalitis, cerebral malaria, or metabolic encephalopathy was made. After collecting blood and CSF samples for culture, treatment with intravenous (IV) ceftriaxone 2 g BD and vancomycin 1 g TID was initiated according to our hospital protocol for the treatment of acute bacterial meningitis in adults. Steroid (IV dexamethasone 8 mg QID) was given 30 min before antibiotics. In addition, as aseptic meningitis due to viral pathogens could not be excluded, acyclovir 500 mg TID IV was also administered.

Her laboratory tests showed hemoglobin 11.5 g/dl, total WBC count of 31,200/mm³ with 97% neutrophils, and 3% lymphocytes. Peripheral smear showed neutrophilic leukocytosis with shift to the left and toxic granules in neutrophils. Her blood urea was 48 mg/dl, serum creatinine 1.5 mg/dl, serum sodium 140 mmol/L, potassium 3.9 mmol/L, serum creatinine phosphokinase 235 U/L, C-reactive protein 25.6 mg/dl, total serum proteins 8.6 g/dl with albumin 4.2 g/dl, and globulin 4.4 g/dl. The liver function tests were within normal limits. Her Hemoglobin A1c (HbA1c) was 6.2%. She was nonreactive for HBV surface antigen, hepatitis C virus, and HIV 1 and 2 antibodies. Card test for malaria (paracheck) was negative while the peripheral smear did not show malarial parasite. Dengue virus NS1 antigen and serology were negative. Her chest radiograph on admission was normal. Her blood cultures were sterile on the 2nd and 5th day of incubation. Urine routine examination showed 4 to 5 pus cells/high power field, few red blood cells, and glycosuria. Culture was sterile. Examination of CSF revealed turbid CSF with 1280 leukocytes of which 85% were polymorphs and 15% lymphocytes. Its biochemical analysis showed 232 mg/dl of proteins, 78 mg/dl glucose (corresponding blood glucose 284 mg/dl), and adenosine deaminase 6 U/L. Gram-stain of CSF showed Gram-negative

Specimen : CSF		
Test : MS.Culture & Sensitivity		Method : CULTURE BY CONVENTIONAL & SENSITIVITY BY VITE
Smear : Gram Negative Bacilli present		
ORGANISM : PSEUDOMONAS AERUGINOSA		
REMARKS :		
DRUG	MIC(microgm/mL)	SENSITIVITY
Ampicillin	>=32	RESISTANCE
Amoxicillin/Clavulanic Acid	>=32	RESISTANCE
Amikacin	<=2	SENSITIVE
Ciprofloxacin	2	INTERMEDIATE SENSITIVE
Ceftriaxone	16	RESISTANCE
Cotrimoxazole	>=320	RESISTANCE
COLISTIN	<=0.5	SENSITIVE
Cefepime	16	INTERMEDIATE SENSITIVE
Cefoperazone/Sulbactam	16	SENSITIVE
Ceftazidime	>=64	RESISTANCE
Cefturoxime	<=1	SENSITIVE
Gentamicin	1	SENSITIVE
Imipenem	2	SENSITIVE
Meropenem	2	SENSITIVE
Nalidixic Acid	>=32	RESISTANCE
Piperacillin / Tazobactam	32	INTERMEDIATE SENSITIVE
Tigecycline	4	RESISTANCE

Figure 1: Culture and sensitivity pattern of cerebrospinal fluid

rods while culture grew *P. aeruginosa* sensitive to carbapenem, colistin, aminoglycosides, and intrinsically resistant to ceftriaxone and amoxicillin (Fig. 1). There were no fungal elements and acid-fast bacilli. Her noncontrast CT brain was apparently normal except for age-related cerebral atrophy (Fig. 2).

On the 2nd day of admission, there was further deterioration of her respiratory pattern and she was shifted to intensive care. Her arterial blood gas showed acute respiratory alkalosis probably due central neurogenic hyperventilation. Her chest radiograph remained normal. Her antibiotics were escalated to IV meropenem 2 g 8th hourly and levofloxacin 500 mg 12 hourly on the 2nd day. Supportive treatment with steroid, oxygen by face mask, proton-pump inhibitor, multivitamins, and oral nutrition through Ryle's tube was continued. Within 48 h of changing IV antibiotics, she became more alert, orientated, and started recognizing her relatives. Her clinical condition gradually improved. IV antibiotics were continued for 14 days. Repeat CSF examination done on the 15th day showed clear fluid with normal cell count and biochemistry. Transthoracic echocardiography done before discharge was normal. There was no evidence of infective endocarditis. She refused treatment after 15 days and was discharged against medical advice.

DISCUSSION

P. aeruginosa, a Gram-negative nonfermenting bacillus, is an important cause of sepsis, especially in patients with compromised immune mechanisms [1]. It is the most common pathogen isolated from patients who have been hospitalized longer than 1 week, and it is a frequent cause of wide range nosocomial infections because of its inherent antibiotic resistance. Reservoirs of infections are often the respiratory equipment.

P. aeruginosa is a very uncommon cause of bacterial meningitis in adults. In the Netherlands, its incidence is 1.6 cases per 100,000 adults per year [2]. Most of the cases are nosocomial and occur in patients who undergo neurosurgical procedure or head trauma. In a study by Juhi *et al.*, out of the 121 cases of postsurgical meningitis, 10 (9.9%) cases of nosocomial Pseudomonas meningitis were identified [3]. Previous studies by Erdem *et al.* [4] and Huang *et al.* [5] had also shown that *P. aeruginosa* was responsible

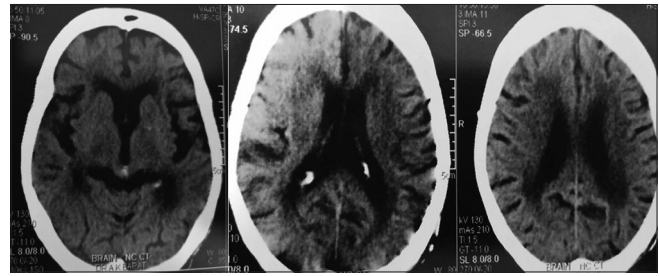


Figure 2: Noncontrast computerized tomography scan of brain showing diffuse cerebral atrophy

for 8.3–10.7% of meningitis in post-neurosurgical patients. In an Indian study, from National Institute of Mental Health and Neurosciences, Bangalore, Srinivas *et al.* found 15.6% cases of post-operative Pseudomonas meningitis out of 415 cases of meningitis over a period of seven years [6]. Incidence is higher in immunocompromised patients like those with malignancy, poorly controlled diabetes, HIV infection, etc., [7].

Although infection in healthy persons is extremely rare, there has been increasing recognition of *P. aeruginosa* is an important cause of community-acquired infections [1]. It has been linked to warm and moist environments such as hot tubs and swimming pools. One case of community-acquired pneumonia due to *P. aeruginosa* was directly linked to the use swimming pool [8]. A recent Dutch investigation showed that *P. aeruginosa* was detectable in 10 out of 14 swimming pools (71%) mainly on play accessories [1]. Yet, another case of spontaneous nosocomial *P. aeruginosa* meningitis was reported by Parr *et al.* in a 78-year-old female receiving adjuvant postsurgical chemotherapy for colon adenocarcinoma [9]. A case of 6-year-old boy developing nosocomial *P. aeruginosa* meningitis was reported by Nunn and Wellman (1956) following the use of a contaminated pump-oxygenator for pulmonary valvulotomy. Our patient did not have any obvious overt risk factor. Her diabetic state was well-controlled during the past 3 months as per her relatives and HbA1c level which was within the normal range. There was no history of recurrent infections to suggest immunosuppression. However, older adults have increased propensity to develop bacterial meningitis due to immunosenescence (age-related decline in immune function) [7].

Another peculiar feature of our case was the absence of fever. The clinical presentation of acute bacterial meningitis in older adults (defined as >60-year-old) may be different than that in younger adults, with fewer patients manifesting with the classic symptoms of fever, neck stiffness, and altered mental state than among younger adults [10]. Terminal neck stiffness, as in our case, in an elderly patient may be attributed to generalized increase in rigidity and cervical spondylosis. Thus, the diagnosis may be delayed due to atypical clinical presentation. This case thus highlights the diagnostic challenge associated with bacterial meningitis presenting in an older patient. The diagnosis in our case was made even more difficult owing to the absent febrile response. CSF (biochemistry and cell count) findings are indistinguishable from other forms of bacterial meningitis.

Community-acquired spontaneous meningitis caused by *P. aeruginosa* is usually a rapidly progressive and highly lethal disease in older adults and carries a very high morbidity and mortality rate to the tune of 61% [11]. However, owing to early diagnosis and energetic treatment, our patient survived without any sequel. CSF examination was normal after 15 days of treatment. Hence, prompt recognition and rapid diagnosis are vital as the prognosis worsens with treatment delay. Treatment is based on the sensitivity pattern and the recommended duration of treatment is 21 days with third-generation cephalosporin and aminoglycoside or/carbapenem.

CONCLUSION

P. aeruginosa meningitis, though is typically nosocomial, can also be community-acquired. Physicians should have a high index of suspicion to consider the diagnosis even when all of the classical signs and symptoms may not be present simultaneously in elderly. Lumbar puncture should, therefore, be utilized when diagnostic uncertainty remains. Choice of the antibiotic is decided by the local resistance pattern. Early diagnosis and treatment are warranted to reduce mortality.

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