International Journal Dental and Medical Sciences Research
Volume 3, Issue 6, Nov-Dec 2021 pp 547-550 www.ijdmsrjournal.com ISSN: 2582-6018

Viral encephalitis with secondary Acinetobacter baumanii meningitis- a diagnostic challenge

Dr Kshitija Valvaikar[1], Dr Masood Mujawar[2]
1. MBBS, junior resident of medicine department, Goa medical college
2. MBBS, junior resident of medicine department, Goa medical college
Corresponding author: Dr Kshitija Valvaikar

Submitted: 10-12-2021 Revised: 22-12-2021 Accepted: 25-12-2021

I. INTRODUCTION

Meningitis refers to inflammation of the meninges whereas the term encephalitis involves inflammation of the brain parenchyma with evidence of meningeal involvement with signs and symptoms of focal or diffuse neurological deficit. Herpes simplex virus is the most common virus causing viral encephalitis The incidence of viral encephalitis is 3.5 to 7.5 per 100,000 people, with the highest incidence in the young and elderly(4)

HSV encephalitis usually involves frontal and temporal lobe hence is associated with behavioural, psychiatric disturbances, memory deficits and aphasia.

Acinetobacter, a gram-negative coccobacillus, is strictly aerobic and non-fermentative; and is well documented as a nosocomial pathogen. It is an opportunistic pathogen found on skin and respiratory and GI tract of humans. The organism exhibits diverse mechanisms of resistance, and this has lead to the emergence of strains that are resistant to all commercially available antibiotics.[1]

Acinetobacter baumannii is one of the ESCAPE organisms, which refers to a group of health care-associated organisms that have the potential for substantial antimicrobial resistance.It is important to note that carbapenem-resistant A. baumanii is one of the critical-priority pathogens on the World Health Organization priority list of antibiotic-resistant bacteria for effective drug development.

There is mounting evidence that A.baumannii can no longer be exclusively considered anosocomial pathogen, and is capable of causing profoundclinical disease in the absence of traditional nosocomialrisk factors.

Acinetobacter meningitisis usually reported in post neurosurgical patients and those with an External Ventricular Device (EVD) and is typically characterised by fever and change in consciousness. Meningeal signs, focal neurological signs, and seizure are found in a minority of patients. Increased CSF white blood cell counts (ranging from 100 to several thousand cells per μL) are typical. However,there are also reports of cases of true infection that have no CSF white blood cells. Typically, 75–100% of the white blood cells are polymorphonuclear cells.CSF protein is almost always raised, most typically being between 150 mg/L and 200 mg/L. CSF glucose may be normal or depressed.

All-cause mortality from Acinetobacter meningitis ranges from 15% to 71%. Highest mortality rates have been observed in neonates and in units where large numbers of carbapenem-resistant acinetobacter were observed.

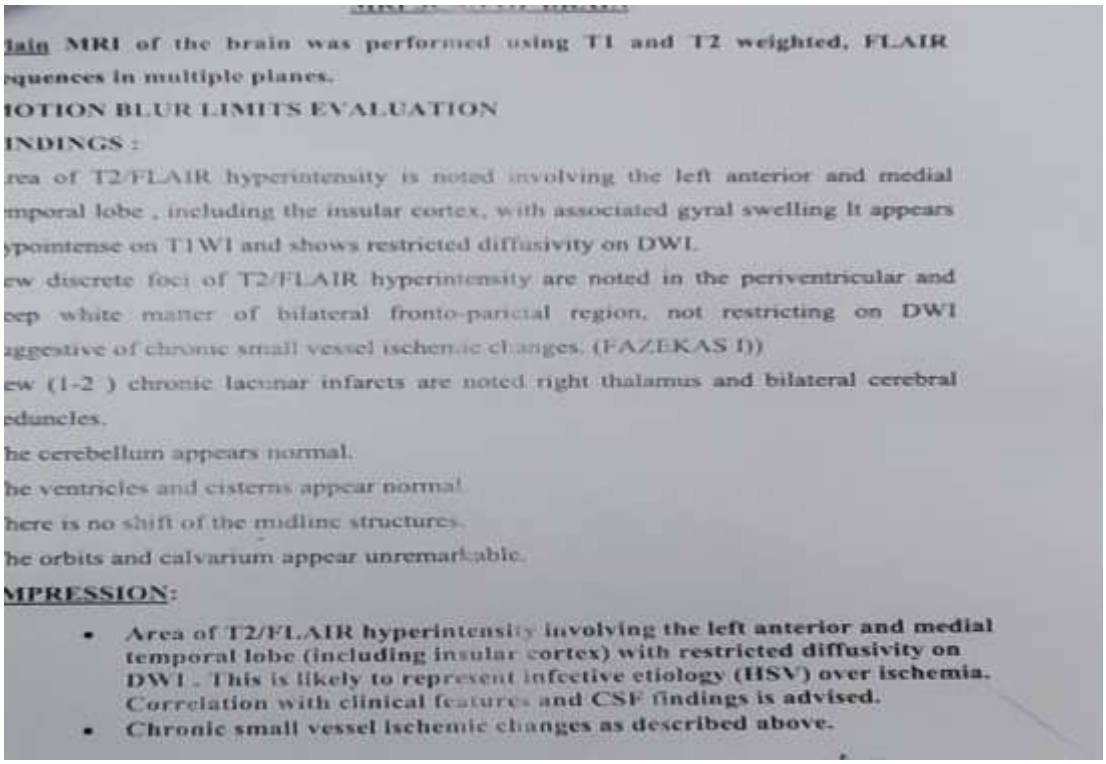
II. CASE REPORT

A 66 years old male, known case of Diabetes mellitus and Hypertension for the last 10 years on regular treatment, with no past history of neurosurgery or head injury and no past history of hospitalisation, presented to the emergency department with history of one episode of generalised tonic clonic convulsions, headache, vomiting and altered sensorium in the form of decreased verbal output since one day. There was no history of fever at admission. Initial examination revealed that the patient was afebrile, pulse rate was 80 beats/min, BP was 230/140mmHg, respiratory rate was 18breaths/min. The patient was conscious, confused and was not following oral commands and did not have any other neurological deficit. CT Brain at admission was normal. A provisional diagnosis of hypertensive encephalopathy was made and patient was started on nitroglycerine infusion and tablet amlodipine and injectable levetiracetam.

Even though the blood pressure was normalised, the patient's sensorium did not improve. The patient was found to have global aphasia,hence a repeat CT Brain was performed with the presumptive diagnosis of an ischaemic cerebrovascular accident in mind. However, only a lacunar infarct in the right thalamus was noted. The patient during the course of hospital stay was found

International Journal Dental and Medical Sciences Research
Volume 3, Issue 6, Nov-Dec 2021 pp 547-550 www.ijdmsrjournal.com ISSN: 2582-6018

to have high fever spikes. Blood investigations performed on admission showed a white blood cell count 15,300 cells/cmm (N-69% L-17%), haemoglobin 13.6 g/dL, platelet count 1.9 lakhs/cmm and random blood glucose 160mg/dL. Blood urea, creatinine were within normal limits. Serum electrolytes revealed hyponatremia with Sodium of 123 mmol/L. The patient subsequently had high fever spikes. Dengue NS1Ag and IgM was negative, ICT kit test for Malaria was negative. RTPCR for COVID 19 was negative. Blood, urine, RTA and bed sore cultures were sent and repeated which were unsuccessful in isolating any organism. As the clinical findings were not correlating with the findings on neuroimaging, MRI Brain was done. Area of T2/ FLAIR hyperintensity involving the left anterior and medial temporal lobe (including the insular cortex) with restricted diffusivity on DWI was noted suggestive of viral encephalitis (HSV) . Chronic small vessel ischaemic changes were also noted.



lain MRI of the brain was performed using T1 and T2 weighted, FLAIR
quences in multiple planes.
IOTION BLUR LIMITS EVALUATION
INDINGS :
rea of T2/FLAIR hyperintensity is noted involving the left anterior and medial
mporal lobe , including the insular cortex, with associated gyral swelling It appears
ypointense on T1WI and shows restricted diffusivity on DWI.
ew discrete foci of T2/FLAIR hyperintensity are noted in the periventricular and
eep white matter of bilateral fronto-parietal region, not restricting on DWI
ggestive of chronic small vessel ischemic changes. (FAZEKAS I)
ew (1-2) chronic lacunar infarcts are noted right thalamus and bilateral cerebral
eduncles.
he cerebellum appears normal.
he ventricles and cisterns appear normal
here is no shift of the midline structures.
he orbits and calvarium appear unremarkable.
MPRESSION:

- Area of T2/FLAIR hyperintensity involving the left anterior and medial temporal lobe (including insular cortex) with restricted diffusivity on DWI . This is likely to represent infective etiology (HSV) over ischemia. Correlation with clinical features and CSF findings is advised.
- Chronic small vessel ischemic changes as described above.

Neurophysician's opinion was sought and a lumbar puncture was performed as advised in view of subsequently noted nuchal rigidity. The patient was empirically started on Injectable antibiotics and Injectable Acyclovir considering the possibility of viral encephalitis. The CSF fluid was clear in appearance with microscopy showing lymphocytes 250/cmm. CSF biochemical analysis revealed mildly elevated proteins of 69mg/dL, low glucose of 99mg/dL for corresponding RBSL of 189mg/dLand chloride 109mmol/L and ADA was normal. Gram stain microscopy testing was negative. CSF CBNAAT was negative for Mycobacterium tuberculosis. Findings were consistent with viral encephalitis. CSF culture showed growth of Acinetobacter species sensitive to Piperacillin+Tazobactam, Cefoperazone+ Sulbactam, Imipenem, Meropenem, Amikacin, Colistin, Chloramphenicol, Nalidixic acid, Ciprofloxacin and Tigecycline. A repeat lumbar puncture done 4 days later was sent to a higher laboratory which again showed growth of Acinetobacter baumanii which was sensitive to Cefoperazone sulbactam, Gentamicin, Tigecycline, Minocycline, Colistin, Trimethoprim/Sulfamethoxazole and intermediately sensitive to Levofloxacin, Meropenem and Imipenem. CSF for HSV PCR was negative. Hence the clinical diagnosis of viral encephalitis with secondary Acinetobacter baumanii meningitis was made.

International Journal Dental and Medical Sciences Research
Volume 3, Issue 6, Nov-Dec 2021 pp 547-550 www.ijdmsrjournal.com ISSN: 2582-6018


GMC-139
PATIENT NAME: [redacted]
LAB ID: 43572
COLLECTION DATE: 29/09/2021
REPORTING DATE: 02/10/2021
REF BY: DR. GMC
SPECIMEN SOURCE: CSF

MICROBIOLOGY REPORT-BACTEC

Test Report Status: Final

CULTURE: Growth of Acinetobacter baumannii.

The patient was then started on Meropenem and Acyclovir was later discontinued. The patient was started on Colistin in view of persistent fever and after this became afebrile. However despite the full course of antibiotics, the patient continued to remain aphasic and had developed spasticity of all limbs. Henceinjectable Acyclovir was restarted and continued for its full course of 14 days with regular monitoring of renal parameters. The patient was given regular physiotherapy and occupational therapy. At the end of treatment, considerable clinical improvement was noted with improvement of aphasia and spasticity.

III. DISCUSSION

To our knowledge, this is the first reported case ofcommunity-acquired A. baumannii meningitis in India. A. baumannii is a well-established meningealpathogen in the neurosurgical settingbut has been infrequently described as acause of primary de novo meningitis. A. baumannii has been documentedas a cause of pseudomeningitis (Cunha et al., 1999) but ourpatient's CSF showed clear evidence of bacterialmeningitis. The negative Gram stain we attribute to themarked pleocytosis and heavily proteinaceous nature,which may have obscured the bacilli. Our case is inkeeping with the clinical scenario of the other cases,where fever and a disturbed level of consciousnesswerepredominant features. Furthermore, the antibioticsusceptibility profile was atypical for a nosocomial strain ofA. baumannii, a feature consistent with other reportedcases.A. baumannii is generally considered an opportunisticnosocomial pathogen and there is debate as to itsmechanisms of pathogenicity and virulence. The epidemiologicalprofile suggests that it is of low virulence anddisease is dependent on significant host immunologicalimpairment.

Comorbidities suchas chronic obstructive pulmonary disease, renal disease anddiabetes mellitus are predisposing factors for Acinetobacter infections. Interestingly,heavy smoking and excessive alcohol consumption werealso associated with increased risk of disease. Our patient's clinical presentation is unique and presented a diagnostic challenge. MRI and CSF studies markedly aidedus in our diagnosis.

Attempts to characterize the epidemiology of communitystrains of A. baumannii suggest that the community mayserve as a potential reservoir for both nosocomial andcommunity-acquired infections.As highlighted by a previous study, the more interesting questionis whether hospital strains that disseminate into thecommunity serve as a potential reservoir for community-acquiredinfections. A. baumannii is capable of prolonged survival in the environment, and, with increasing disseminationof the organism into the community via healthcare associatedinfection, it is feasible that A. baumannii willnow adapt to the hostile environment of a host with anormal immune response. It isforeseeable that this highly adaptable organism may soonevolve into a significant community pathogen.

Unlike the Acinetobacter strains found in nosocomial infections, the strain of Acinetobacter meningitis in the community-acquired cases did not show resistance to multiple antibiotics. The treatment of Acinetobacter meningitis is usually at

least three weeks. The response should be assessed clinically and with repeat CSF cultures.[3]Most adult patients with community-acquired Acinetobacter meningitis can be saved by timely therapy with appropriate antibiotics before deterioration of the systemic condition and impairment of consciousness.[4]

REFERENCES

[1]. Kamath SA. Shah SN. API TEXTBOOK OF MEDICINE. 11th ed. New Delhi: CBS: 2019. 179 p.
[2]. Lowman W, Kalk T, Menezes CN, John MA, Grobusch MP. A case of community-acquired Acinetobacter baumannii meningitis - has the threat moved beyond the hospital? J Med Microbiol. 2008 May;57(Pt 5):676-678. doi: 10.1099/jmm.0.47781-0. PMID: 18436607.
[3]. Zeina A Kanafani, Souha S Kanj.Acinetobacter infection: Treatment and prevention. UpToDate.
[4]. Chang WN, Lu CH, Huang CR, Chuang YC. Community-acquired Acinetobacter meningitis in adults. Infection. 2000 Nov-Dec;28(6):395-7. doi: 10.1007/s150100070013. PMID: 11139162.
[5]. Saema Said; Michael Kang, Viral Encephalitis; StatPearls [Internet]. Jan 2021