Case Report

A clinical odyssey: ulcerative colitis, renal failure, obstructive uropathy, and *Streptococcus agalactiae* meningoencephalitis

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Abstract

Streptococcus agalactiae infections are primarily attributed to pregnancy and neonates. However, adults with underlying immune-compromised states can also be affected. We report a rare instance of GBS meningoencephalitis in a 40-year-old male with chronic kidney disease. The patient presented to us with obstructive uropathy with urinary tract infection. He developed generalised seizures during in-hospital care. Cerebrospinal fluid examination confirmed *Streptococcus agalactiae* infection. The immunocompromised state of the patient probably contributed to his illness (GBS meningoencephalitis). Early diagnosis by a qualitative multiplexed nucleic acid based in vitro diagnostic test (BioFire ® FilmArray ®) and early treatment helped achieve a successful outcome with no post infection sequelae. This case report demonstrates the importance of early accurate aetiological diagnosis and appropriate treatment to achieve an optimal outcome.

Keywords: *Streptococcus agalactiae, meningoencephalitis, renal insufficiency, colitis, inflammatory bowel diseases*

Introduction

Group B streptococcus (GBS), or *Streptococcus agalactiae* is a Gram-positive bacterium usually found in the female genital tract.[1,2] GBS is not only known to cause puerperal sepsis and neonatal meningitis but also affects non-pregnant adults, particularly those who are immunocompromised or have underlying comorbidities like hepatic or renal failure.[2,3] GBS meningoencephalitis is rare and accounts for only 0.3-4.3% of all cases of bacterial meningitis [1]. The annual incidence of bacterial meningitis is around 2.5 cases per 100,000 population and GBS is responsible only for around 15% of these cases.[2,4–6] Although GBS is a normal commensal in healthy individuals and usually present in the gastrointestinal and genital tracts, it may cause severe infections in individuals with underlying comorbidities such as diabetes mellitus, chronic kidney disease or any immunocompromised state. A few cases of GBS meningitis have been reported secondary to cerebrospinal fluid leakage or distant focal

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infection.[5,7,8] It is important to consider the risk of infection not only from fungal, viral and atypical microorganisms but also from GBS in immunocompromised patients. GBS can be particularly dangerous in immune compromised individuals, leading to serious complications.[2,4-6]

Case summary

A 40-year-old male from northern India presented to our centre in July 2023 with intermittent fever with chills over the previous two days, accompanied by reduced urine output and malaise. He was diagnosed with ulcerative colitis in 2016 and on regular follow up, was on amino salicylates for the past seven years and had been in clinical and endoscopic remission for the past 5 years. In 2018 he developed rapidly progressive renal failure secondary to bilateral obstructive uropathy (urolithiasis). He denied any history suggestive of long-term use of antibiotics, seizures or sepsis in the past. He remained asymptomatic until his present admission.

There was no history of altered sensorium, seizures, rashes or exposure to antibiotics or admission prior to admission at this centre. On examination, he had fever and tachycardia, but systemic examination revealed no localising signs for febrile illness. Haematological investigations indicated neutrophilic leukocytosis and high ESR and CRP. Urine examination revealed pyuria. There was no evidence of haematuria or proteinuria. Biochemical parameters suggested progressive worsening of azotemia. Blood and urine cultures did not reveal any bacterial growth. Abdominal ultrasound imaging revealed bilateral hydroureteronephrosis and a proximal ureteric calculus confirmed by CT imaging. The patient was started on ceftriaxone as per institution antibiotic policy. In view of worsening azotemia, the patient required three sessions of haemodialysis. He underwent bilateral Double J ureteral stenting. His condition improved clinically with improved renal functions, urine output and no further need for dialysis.

On the fifth day of hospital care, while recovering from febrile illness, the patient had loss of consciousness, and two episodes of generalised seizures followed by postictal confusion. No evidence of hypoglycemia, uremia or dyselectrolytemia was noted. Urgent CT imaging of the head revealed no abnormalities.

Urgent cerebrospinal fluid (CSF) examination was done which revealed cellular CSF with 30% lymphocytes and 70% neutrophils. CSF protein levels and globulins were significantly elevated. Microscopy and staining of CSF showed occasional inflammatory cells in a proteinaceous background and Gram-positive cocci in pairs. No acid-fast bacilli or cryptococci were observed.

The diagnosis of *Streptococcus agalactiae* associated meningoencephalitis was confirmed through CSF PCR panel test (CSF Biofire ® FilmArray ® Meningitis Encephalitis Panel). It has overall 94.2% sensitivity and 99.8% specificity (Table 1).

The patient was empirically initiated on ampicillin and exhibited improvement within 72 hours. After a two-week course of treatment with ampicillin at renal-adjusted doses, the patient displayed significant clinical improvement, was asymptomatic and discharged from the hospital. No post infection complications or sequelae were noted on follow up. The timeline of the case is as shown in **Figure 1.**

Table-1: Investigation table

PARAMETERS	VALUE	NORMAL RANGE
Haemoglobin	12.8	13-18 g/dl
WBC Differential count	6800 N-68%, L-28%	4000-11000 cells/cmm
Platelets	2.14	1.5-4 L/cmm
Urea/Creatinine	38/3.34	10-50/0.7-1.3 mg/dl
Total Protein Albumin Globulins	7.2 3.9 3.2	Protein : 6-8.3 g/dl Albumin: 3.5-5.5 g/dl Globulin: 2.0-3.5 g/dl
Sodium Potassium	136 4.67	Sodium: 136-145mEq/L Potassium: 5 mEq/L
Calcium Phosphorus	9 3.2	Calcium: 8.6-10.3 mg/dl Phosphorus: 2.8-4.5 mg/dl
Bilirubin	0.6	0.2-1mg/dl
AST ALT	20 18	AST: 15-37IU/L ALT: 46-116 IU/L
CRP	24mg/dl	
ESR	34mm/Hr	
Urine REME Cultures	Stary yellow Pus cells: 9-12 cells RBCs: 1-2 cells No growth	
USG Abdomen	Right Kidney: 11cm Left Kidney: 10.5cm Bilateral hydroureteronephrosis and proximal ureteric calculus	
NCCT KUB	Bilateral proximal ureteric calculi with associated with upstream hydroureteronephrosis	
NCCT Head	Normal	
Fundoscopy	Normal, No evidence of papilledema	
Blood culture /sensitivity	No growth observed	
Urine Culture Sensitivity	No growth observed	
CSF analysis **<u>CSF culture and sensitivity</u>**	Clear WBC: 102 cells (30% lymphocytes and 70 % neutrophils) RBC: 117cells Microscopy: Occasional inflammatory cells seen with proteinaceous background. Gram positive cocci seen in pairs. No acid alcohol fast bacilli seen. No cryptococci seen. Proteins: 136mg/dl Sugar: 87mg/dl (RBS: 120mg/dl) No growth observed	
CSF biofire ® FilmArray ® Meningitis Encephalitis Panel	*Streptococcus agalactiae*: POSITIVE	

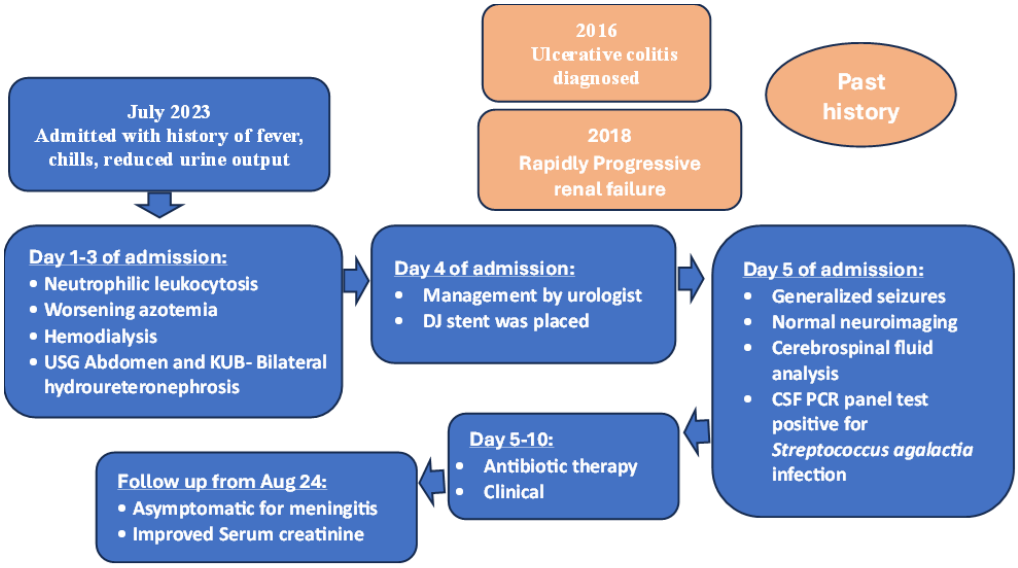

Figure 1: Timeline of illness

Discussion

The case presented here involves an adult male who developed Group B streptococcus meningoencephalitis which is an uncommon occurrence in an adult. GBS meningitis has a high mortality rate of up to 34%.[3,7–9]

GBS can colonise various parts of the body including the skin, lungs, urinary, gastrointestinal and genital tracts, even in healthy adults. Although a few case reports of GBS meningitis reveal a distant focal source of infection, the source of the GBS infection remained uncertain in our patient who initially presented with urinary tract infection (UTI) with obstructive uropathy. Urine cultures and microscopic examination did not show GBS. The patient responded well to the empirical antibiotic therapy. A study from eastern India revealed that only 1% of UTIs are caused by *S. agalactiae.*[10] While recovering, the patient developed generalised seizures in the absence of fever, headache or any meningeal signs which is an unusual feature of meningoencephalitis but can be attributed to the direct spread of the infection from a distant focus or associated bacteraemia. Importantly, immunocompromised individuals may not always exhibit typical signs of infection such as fever.[2,5,6] Thus, CSF examination is crucial to confirm or rule out bacterial meningoencephalitis while systematically ruling out other causes of seizures.

Investigation included the CSF BioFire® FilmArray® Meningitis Encephalitis Panel, which has an overall sensitivity of 94.2% and specificity of 99.8%.[12] Group B Streptococcus was identified. The CSF culture did not reveal any growth, most likely due to antibiotic use during hospitalisation. PCR tests in such situations are therefore valuable for early initiation of therapy. The panel used is a multiplexed nucleic acid-based in vitro diagnostic test intended for BioFire® FilmArray® systems. The BioFire ME Panel can simultaneously detect and identify nucleic acids from multiple bacteria, viruses, and yeast directly from non-centrifuged CSF obtained via lumbar puncture from individuals with signs and symptoms of meningitis and/or encephalitis. Only 0.2 ml of CSF is required for the test, and results were available within one

hour, allowing for the prompt initiation of empirical antibiotic therapy. These tests guide the institution of appropriate therapy.[12]

The patient had multiple comorbidities, including chronic kidney disease (CKD) and ulcerative colitis, which are known risk factors for GBS meningoencephalitis. CKD, in particular, can compromise the immune system and increase susceptibility to infections.[2,3,5] In a study by Bharati Kochar et al, it was found that patients with ulcerative colitis had a 1.63-fold higher incidence of meningoencephalitis when compared to a matched non-IBD (Inflammatory bowel disease) control group.[2] Treatment of IBD frequently involves systemic immunosuppression, which also contributes to increased risk of infection. Also, individuals with one or more coexisting medical conditions are shown to have a significantly elevated risk of developing meningoencephalitis.[2,4,6] Considering existing co-morbidities, our patient was at increased risk for GBS meningoencephalitis. In adults, GBS meningoencephalitis is typically associated with underlying conditions such as immunocompromised states, cerebrospinal fluid leakage, or endocarditis.[2,8] Thorough investigation of these conditions is therefore essential in adult GBS meningoencephalitis cases.

Treatment for GBS meningitis or encephalitis usually involves penicillin group of antibiotics as the first-line therapy for 2-4 weeks, with ceftriaxone as an alternativecontributes.

Conclusion

This case highlights a rare presentation of *Streptococcus agalactiae* meningoencephalitis in an adult with pre-existing chronic kidney disease, ulcerative colitis and obstructive uropathy. GBS meningitis is rare in adults. Early diagnosis and appropriate treatment were crucial in achieving a successful outcome in our case.

Declarations

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Authors' contributions: All the authors have contributed to collecting information on patient history, laboratory test results and preparation of manuscript

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