Severe Fatal Group B Streptococcal Sepsis in an Adult


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Abstract

Objective: Group B Streptococcus (GBS) sepsis is largely a disease seen in neonates and infants. It is uncommon in adults except during pregnancy and puerperium. We hereby present an adult diabetic patient with severe fatal GBS sepsis with a view to increasing clinical awareness of this bacterial infection among adult patients. Clinical Presentation: A 56-year-old male from Saudi Arabia presented initially with features of pyelonephritis. He discharged himself against medical advice after 1 day but returned to the hospital 24 h later, with symptoms of headache, neck stiffness, confusion and left flank tenderness. Clinical and laboratory evaluation, including analysis of cerebrospinal fluid, confirmed sepsis (urosepsis, bacteraemia and meningitis) with GBS spp. Intervention: The patient was managed with intravenous ceftriaxone, vancomycin and later penicillin G and supportive ICU care. The patient required intubation initially. He expired from marked irreversible anoxic brain damage resulting from the sepsis. Conclusion: The case is presented to help in increasing the awareness of clinicians to GBS as a potential cause of morbidity and mortality in adults, particularly in those with chronic comorbid states.

Introduction

Group B streptococcal (GBS) sepsis is largely a disease seen in neonates and infants [1]. It is uncommon in adults except during pregnancy and puerperium [2]. A rising incidence of GBS sepsis has been increasingly recognised among non-pregnant adult patients [3]. It has also been sporadically reported among immunocompetent adult patients where it has a predominantly similar presentation as in a neonate, i.e., as soft tissue...
infection, meningitis, pyelonephritis and bacteraemia [3–5]. Less common presentations include endocarditis, myocarditis, abscesses, osteomyelitis etc. [2, 6, 7]. We hereby present an adult diabetic patient with severe fatal GBS sepsis (urosepsis, bacteraemia and meningitis), with a view to increasing clinical awareness of the occurrence of this bacterial infection in adult patients.

**Case Report**

Our patient A. is a 56-year-old male from Saudi Arabia, a known diabetic, who for the preceding 4 years was on erratic glibenclamide therapy. He presented with a 5-day history of lethargy, fatigue, dysuria and loin pain. He was found to have low-grade pyrexia (37.8°C), costovertebral angle tenderness and pyuria (200 white cells per high power field). He was admitted as a case of pyelonephritis and managed with i.v. cefuroxime 750 mg every 8 h. A day later, after three doses of cefuroxime, the patient discharged himself against medical advice, refusing both in- and outpatient therapy. Twenty-four hours later, he re-presented to the hospital with symptoms of headache, neck stiffness, confusion, slurring of speech and inability to walk. On examination he was found to be apyrexial (36.3°C), confused, drowsy, with nuchal rigidity, left flank abdominal tenderness and questionable left-sided facial weakness. His pupils were equal and symmetrical, but reacted sluggishly to light; there was no papilloedema or other overt cranial nerve palsy. He was admitted to the medical ICU as a case of probable meningoencephalitis. He had initial routine studies and an initial plain brain CT prior to lumbar puncture (LP), and was immediately given i.v. ceftriaxone 2 g stat, which was subsequently continued at 12-hourly intervals.

The initial results revealed the following: full blood count: leucocytes 20,700/mm³ (neutrophils 90%, with toxic granulations); Hb 13.6 g/dl; platelets 370,000/mm³ and ESR 92 mm/h. His random blood sugar was 18.8 mmol/l (NR < 7.8 mmol/l). Urine analysis showed 129 white cells per high power field. Liver function tests revealed total and direct bilirubin of 18.9 and 11.7 μmol/l, respectively (total bilirubin NR: 5–17 μmol/l), and raised alanine aminotransferase of 58 units/l (NR: 0–35 units/l). The following investigations were either negative or normal: chest radiograph, serum urea, electrolytes and creatinine; arterial blood gases; 12-lead electrocardiogram; serum cardiac enzymes (CPK, AST, LDH); coagulation profile (prothrombin and partial thromboplastin times) and plain and, subsequently, contrast-enhanced brain CT scan. LP done within 3 h of admission revealed cerebrospinal fluid (CSF) under pressure, with cloudy to purulent macroscopic appearance. CSF analysis showed 600,000 white cells/mm³ (polymorphs 40%, lymphocytes 60%, protein 1.5 g/l (NR < 0.45 g/l), glucose 1.8 mmol/l (NR 2.5–4.0 mmol/l), simultaneous random blood sugar 13.4 mmol/l. Gram stain revealed many gram-positive cocci while Ziehl-Neelsen staining was negative. Subsequently his CSF, blood and urine cultures grew GBS with sensitivity to penicillin G, ampicillin, ceftriaxone and vancomycin. The minimum inhibitory concentrations (MICs) for the CSF isolate showed: penicillin G = 0.125 μg/ml, ceftriaxone = 0.19 μg/ml, vancomycin = 1.5 μg/ml.

In view of progressive deterioration in consciousness and respiratory instability, i.v. vancomycin given every 12 h was added to ceftriaxone, and the patient was intubated 18 h post admission. He received other supplementary therapies including insulin, prophylactic heparin and ranitidine. While in the medical ICU, urinary bladder catheterisation was attempted but failed initially due to moderately severe urethral stricture. This necessitated bougienage prior to subsequent catheterisation. A repeat LP done 4 days later, while on vancomycin and ceftriaxone, showed FSC white cells 650/mm³ (polymorphs 60%, lymphocytes 40%), persistent but scant gram-positive cocci. The culture later grew GBS. CSF fungal element and India ink studies were negative. Serology for HIV, hepatitis B and C and *Brucella* spp. were negative. The patient remained on vancomycin and ceftriaxone for 4 days, when vancomycin was replaced with i.v. penicillin G 4 million units every 4 h for 2 weeks together with ceftriaxone. He completed a total of 4 weeks of ceftriaxone therapy, i.e., for 10 more days after completion of penicillin G therapy. In the 1st week he sustained a near cardiac arrest and was resuscitated. However, he had sustained a marked irreversible anoxic brain damage resulting from the sepsis. The brain damage was confirmed by electroencephalographic studies. He had tracheostomy following extubation in the 4th week. He expired after 2 months of hospital stay.
Discussion

Although GBS is commonly thought of as a maternal and paediatric pathogen, infections among non-pregnant females and male adults have been reported [3–5]. The predisposing factors for GBS infection in this patient are urethral stricture and obstructive uropathy, and poorly controlled diabetes mellitus [5]. In one study, the genito-urinary tract was found to be a major source of infection [8]. Our patient exemplified all the three major presentations of GBS infection – urosepsis, bacteraemia and meningitis. His initial presentation was with features of pyelonephritis due in part to urinary stasis from stricture. Pyelonephritis is a common manifestation of GBS infection in adults [8]. The subsequent dissemination of the infection in our patient is perhaps in part due to his refusal to continue medical treatment. He had presented ab initio with lethargy, fatigue and had marked CSF abnormalities. It is not inconceivable that he was actually incubating meningitis during the first admission when he was treated with cefuroxime, an agent known to be less than optimal in treating meningitis even due to susceptible bacteria [9]. Infection at multiple sites is not uncommon in GBS sepsis, and concomitant bacteraemia among patients with GBS meningitis is seen in over 80% of cases [5, 10].

Vancomycin and ceftriaxone started during the second hospitalisation should have effectively covered the GBS infection. But vancomycin alone is not appropriate because it is inhibitory in vitro and its CSF concentrations may not exceed the MIC of the organism if a high inoculum of GBS is present. In this context vancomycin should always be used in combination with a bactericidal agent. A 14-day course of penicillin G is considered the antibiotic regimen of choice for proven GBS infection [8]. However, some physicians preferred ampicillin over penicillin because higher levels may be achieved with the former. Studies have also revealed accelerated killing of GBS with the addition of gentamicin to either of the antibiotics [11]. Despite the poor permeation of gentamicin into CSF, it has been suggested that it should be used in combination with either penicillin or ampicillin for the initial therapy of GBS meningitis [11]. The penicillin G MIC for the CSF GBS isolate (= 0.125 μg/ml) in our patient fell within the range found by Bayer et al. [12] (0.04–0.16 μg/ml) and is as expected many times higher than the MICs for group A Streptococcus.

A poor prognosis has been associated with advanced age and the occurrence of neurologic and extraneurologic complications, particularly among chronically ill hospitalised patients [13]. Although our patient is not advanced in age, he had severe infection with neurologic and extraneurologic manifestations, and respiratory instability necessitating intubation. This presentation and the refusal of antibiotic coverage for a day may have led to the poor prognosis and ultimate fatality in our patient. Mortality in GBS sepsis and meningitis is reported at 15–18% [5, 14] although GBS sepsis in adults has been associated with a mortality of as high as 38% [15]. In conclusion, although the importance of GBS infection in neonates and infants is well known, its importance as a cause of invasive infection in adults is underestimated. This case is presented to increase the awareness of clinicians to GBS as a cause of severe morbidity and mortality in adults, particularly in those with chronic comorbid states.

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References


