Severe Fatal Group B Streptococcal Sepsis in an Adult

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Introduction

Group B Streptococcal (GBS) sepsis is largely a disease of neonates and infantst[1], it is uncommon in adults, except in pregnancy and Purperium[2]. Its rising incidence has been increasingly recognised among immunocompromised adult patients. It has also been sporadically reported among immunocompetent adults patients where it has a predorminantly similar presentation as in neonates i.e., as soft tissue infections, meningitis, pyelonephritis and bacteraemia[3,4]. Less common presentations include endocarditis, niyocarditis, abscesses, osteoniyelitis etc.[2,5,6]. We present an adult diabetic patient, with severe fatal CBS sepsis (urosepsis, bacteraemia and meningitis), with a view to increasing clinical awareness of this bacterial infection among adult patients.

Case Report

A 56 year old Saudi male was a known diabetic for the preceeding 4 years who was on erratic glibenclamide therapy and also diagnosed as a case of cervical spondylosis few months prior to presentation. He presented with five days history of lethargy, fatigue, dysuria, loin pains and was found to have low grade pyrexia 37.8°C, loin tenderness and pyuria (200 white cells per high high power field). He was admitted as a case of pyelonephritis and managed with IV cefuroxime 750 mg eight hourly. A day later after three doses of cefuroxinie, patient discharged himself against medical advise refusing both in- and out-patient therapy. Twenty—four hours later. he represented to the hospital with 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. Pupils were equal and symmetrical, but reacted sluggishly to light; there was no papiloedema or other overt cranial nerve palsy. He was admitted to the medical ICU as a case of probable meningoencephalitis. He had initial routine investigations, an initial plain CT brain, prior to lumbar puncture-LP and was immediately given IV ceftriaxone 2 gm stat, which was subsequently maintained at 12 hourly interval.

Flis initial results revealed: full blood count; leucocytes-20, 700 per cmm (neutrophils-90%, with toxic granulations); Hb-13.6 gm per dL; platelets 370,000 per cmm and ESR 92 mm per hour. His random blood sugar was 18.8 mmol per litre. Urine analysis showed 129 while cells per high power field. Liver function tests revealed raised total and direct bilurubin-18.9 and 11.7 mic.mol per litre respectively and raised alanine aminotransferase 58 units per litre. The following intvestigations were either negative or normal:chest radiograph, serum urea, electrolytes and creatinine; arterial blood gases; 12 lead electrocardiogram; serum cardiac enzymes: coagulation profile (prothrombin and partial throni boplastin limes) and plain and subsequently, contrast enhanced brain CT scan. LP done within three hours of admission revealed cerebrospinal fluid-CSF under pressure, with cloudy to near pussy macroscopic appearance. CSF analysis showed 600,000 white cells per cmm (polymorphs 40%, lymphocytes 60%). protein-I.S gm per litre (NR<0.45 gm per litre), glucose 1.8 niniol per litre (NR: 2.5-4.0 rnmol per litre, simultaneous randoni blood sugar 13.4 mmol per lire). Gram stain revealed many gram positive cocci while Zielli-Neelsen staining was negative. Subsequently his CSF, blood and Urine cultures grew Group B Streptococcus with pan-sensitivity to penicillin-G ampicillin, ceftriaxone and vancomycin. The minimum inhibitory concenterations, MICs, for the CSF isolate showed; penicillin G 0.125 mic. gni per ml.. ceftriaxone 0.19 mic. gm per rnl., vancomvcin 1.5 mic. gm per ml.

In view of progressive deterioration in consciousness and respiratory instability, IV vancornycin 1 gm 12 hourly was added to ceftriaxone and patient intubated, eighteen hours post admission. He received other supplementary therapies including insulin, prophylactic heparin, ranitidine etc. While in the medical ICU urinary bladder catheterisation was attempted but initially failed due to moderately severe urethral stricture. This necessitated bouginage prior to subsequent catheterisation. A repeat LP done 4 days later, whilst on vancomycin and ceftriaxone, showed CSF white cells 650 per crnm (polymorph 60%. lymphocytes 40%), persistent but scant gram positive cocci and culture which later grew Group B Streptococcus. CSF fungal element and India ink studies were negative. Serology for HIV, Hepatitis B and C and Brucella spp. were negative. Patient remained on vancomycin and ceftriaxone for four days, when vancomvcin was replaced with IV penicillin G 4 million units every four hours for two weeks together with ceftriaxone. He completed a total of 4 weeks on ceftriaxone i.e., for 10 more days after completion of penicillin G course. In the first week he sustained a near arrest and was resuscitated but had sustained a marked anoxic brain damage, confirmed by electroencephalographic recordings. He had tracheostomy following extubation in the fourth week. He expired after two months of hospital stay.

Discussion

Although GBS is commonly thought of as a maternal and paediatric pathogen, infection among non-pregnant female and male adults have occassionally been reported[3,4]. The predisposing factors for GBS infection in the patient described above are urethral stricture and probably obstructive uropathy and poorly controlled diabetes mellitus. Immunosuppressive states, like diabetes mellitus and obstructive uropathy secondary to benign prostatic hypertrophy and chronic renal failure have previously been described as well known risk factors for GBS infection[4] and in one series genito-urinary tract was found to be a major source of infection[7].

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 GBS infection[7]. The subsequent dissemination of the infection is perhaps due in part to his refusal of medical intervention. He had presented ab initio with lethergy, fatigue and had extreme CSF cellular changes. It is not inconceivable that he has actually incubating meningitis during the first admission when he was treated with cefuroxime, an agent known to be less than optimum in treating meningitis even due to susceptible bacteria[8]. Multiple infected sites is not an uncommon presentation of GBS sepsis and concomitant bacteraemia among GBS meningitis patients was seen in over 80% of cases[4,9].

The initial vancomycin and ceftriaxone started during the second hospitalisaton would have effectively covered the GBS isolated. But a 14 day course of penicillin G is considered the antibiotic regimen of choice for proven GBS infection[7] and this is why it was introduced to replace vancornycin by fifth day when sensitivity was known. In all likelihood all the three isolates from CSF, blood and urine are the same as they share the same antibiogram. The penicillin G MIC for the CSF GBS isolate (~0. 125 mic. gm per ml) in our patient fell within the range found by Bayer et al., (0.04-0.16 mic. gm per ml) and is as expected many times higher than the MICs for Group A Streptococcus[10].

Poor prognosis has been associated with advanced age and the occurrence of neurologic and extra-neurologic complications, particularly among chronically ill hospitalised patients[11]. Although our patient is not advanced in age, he had severe infection with neurologic and extraneurologic manifestations and respiratory instability necessitating intubation at a point. This presentation and the refusal of antibiotic coverage for a day might have led to the poor prognosis and ultimate fatality in our patient. Otherwise mortality in both GBS sepsis and meningitis is similar, reported at 15-18%[4,12]; although CBS sepsis in adults has acaused mortality of as high as 38%

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 under estimated. The case is presented to help in increasing the awareness of clinicians to GBS as an important cause of morbidity and mortality in adults, particularly in those with chronic co-morbid states.

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