Outbreak of Streptococcus pyogenes emm type 58 in a high dependency unit of a level-1 trauma center of India

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Background and Aims: Group A Streptococcus (GAS) can cause illnesses ranging from self-limited to severe, life-threatening, invasive infections. The objective of the following study was to investigate a suspected Streptococcus pyogenes outbreak in a high dependency unit (HDU) of our trauma center. Materials and Methods: All the isolates of beta hemolytic Streptococci were identified by standard microbiological methods, Vitek 2 system and latex agglutination tests. Antimicrobial susceptibility testing was performed as recommended by Clinical Laboratory Standards Institute. Exotoxin genes, including speA, speB, speC, speF, speM and speI were detected by polymerase chain reaction (PCR). The emm types of isolates of S. pyogenes were determined by sequencing the variable 5' end of emm gene after amplification by PCR. Results: In a 28 bedded poly-trauma ward with a four bedded HDU three out of four patients developed S. pyogenes emm type 58 infection. The strain was macrolide and tetracycline resistant and produced the Streptococcal pyrogenic exotoxins speB, speC, speF and smeZ. Surveillance sampling was done for investigation from patients, health-care workers and environmental samples. Conclusion: An outbreak of GAS infections was established caused by the uncommonly reported emm type 58. The outbreak was controlled by prompt treatment, intensive surveillance, feedback and training.

Keywords: Beta-hemolytic Streptococci, group A Streptococcus, outbreak, Streptococcus pyrogenic exotoxins emm types, trauma patients

Introduction

Streptococcus pyogenes (group A Streptococcus; GAS) causes a range of clinical syndromes from mild pharyngitis to severe, life threatening toxic shock syndrome. The M protein of GAS, encoded by the emm gene is a major virulence factor for the organism. The high variability of the amino-terminus of the emm gene makes emm typing an excellent epidemiological marker, recommended as the “Gold standard” by the Centers for Disease Control and Prevention (CDC). Invasive infections due to GAS have increased in the past two decades, presumably due to the emergence of virulent clones of a few emm types. In October, 2012, a cluster of cases of invasive GAS infections was noted in a high dependency unit (HDU) of a level-1 trauma care center of India. This initiated an intensive surveillance and search for additional cases/carriers of the pathogen and institution of prompt treatment, which could control the outbreak. This report elaborates the epidemiological investigation and control measures of the outbreak. Sequence analysis of emm gene and profiling of exotoxin production was done, to ascertain the similarity and virulence of the strains.

Materials and Methods

All the isolates of beta-hemolytic Streptococci (βHS) were identified by standard microbiological methods. The confirmation of identity was also done by the Vitek
2 (Biomerieux, France). Grouping of the Streptococci was performed by agglutination test (HiMedia Labs, Mumbai, India) according to manufacturer’s instructions. All the strains were stocked in stocking beads (Microbank™, Pro-Lab Diagnostics, Austin, Texas, USA) at −70°C till further analysis.

Exotoxin genes, including speA, speB, speC, speF, smeZ, ssa, speG, speH, speJ, speL, speM and speI were detected by polymerase chain reaction (PCR) as per published protocol.[6,7] Amplification of all the genes was performed with an initial 5 min denaturation at 94°C followed by 30 cycles of denaturation at 94°C for 30 s, 30 s of annealing at the appropriate temperature for each gene, as specified in Table 1 and 60 s of extension at 72°C with a final extension step at 72°C for 7 min. S. pyogenes ATCC strains 12351, 12344, 700294 and 51500 were used as controls.

The emm types of isolates of S. pyogenes were determined by sequencing the variable 5’ end of emm gene after amplification by PCR. For this, the DNA preparation of GAS strains was performed as described by the USA CDC. The amplification of emm gene and sequencing was performed as per the published protocol.[3] Amplification of the emm genes was performed by the “all M” primer having the following sequence:

- Forward primer: 5'-GGGTGACACCAAGAGGTGAGGAC-3'
- Reverse primer: 5'-GGGTGACACCAAGAGGTGAGGAC-3'

The emm gene-specific amplicon was used as a template to determine the sequence of hypervariable region of emm gene. 30 ng of PCR product was sequenced by using primer (5’-ATAAGGAGCTAAAAATGCTG-3’) with the dye terminator mix and subjected to automated sequence analysis on autosequencer as per manufacturer’s instructions. The cycling parameters were 96°C for 30 s, 50°C for 15 s and 60°C for 4 min. The emm gene sequence was subjected to homology search by Blast search analysis (http://www.ncbi.nlm.nih.gov/BLAST/Blast.cgi). A comparison of the nucleotide homology for the first 200 bases of the hypervariable region was conducted. Strains, which showed ≥95% homology with reference strain was designated the particular parental emm type. Types (based on variation in the type-specific region of emm gene) were designated according to the information available at CDC website (http://www.cdc.gov/ncidod/biotech/strep/doc.htm).

The sequences were submitted to GenBank and assigned sequence numbers.

The antimicrobial susceptibility testing of Streptococci was performed by the disk diffusion method on Mueller Hinton agar with 5% sheep blood according to the recommendations of the Clinical Laboratory Standards Institute (CLSI).[8,9] The following antibiotics were tested: Penicillin G, ampicillin, vancomycin, erythromycin, clindamycin, cefotaxime, ceftriaxone, linezolid, teicoplanin, ciprofloxacin, levofloxacin, tetracycline.

### Table 1: Primers and conditions to be used for PCR of virulence genes

| Gene | Primer direction | Primer sequence | Annealing temperature | Amplicon size |
|------|------------------|-----------------|-----------------------|---------------|
| speA | Forward          | 5’-CGGATCCATGTCAGGACCACCAGC-3’ | 55°C | 660 bp |
| speA | Reverse          | 5’-GGGATCCATGTCAGGACCACCAGC-3’ | 55°C | 170 bp |
| speB | Forward          | 5’-GGGATCCATGTCAGGACCACCAGC-3’ | 55°C | 627 bp |
| speB | Reverse          | 5’-GGGATCCATGTCAGGACCACCAGC-3’ | 55°C | 57°C |
| speC | Forward          | 5’-GGGATCCATGTCAGGACCACCAGC-3’ | 55°C | 1193 bp |
| speC | Reverse          | 5’-GGGATCCATGTCAGGACCACCAGC-3’ | 55°C | 723 bp |
| Ssa  | Forward          | 5’-GGGATCCATGTCAGGACCACCAGC-3’ | 55°C | 629 bp |
| Ssa  | Reverse          | 5’-GGGATCCATGTCAGGACCACCAGC-3’ | 55°C | 155 bp |
| smeZ | Forward          | 5’-GGGATCCATGTCAGGACCACCAGC-3’ | 55°C | 612 bp |
| smeZ | Reverse          | 5’-GGGATCCATGTCAGGACCACCAGC-3’ | 55°C | 678 bp |
| SpeG | Forward          | 5’-GGGATCCATGTCAGGACCACCAGC-3’ | 55°C | 535 bp |
| SpeG | Reverse          | 5’-GGGATCCATGTCAGGACCACCAGC-3’ | 55°C | 596 bp |
| SpeH | Forward          | 5’-GGGATCCATGTCAGGACCACCAGC-3’ | 55°C | 652 bp |
| speI | Forward          | 5’-GGGATCCATGTCAGGACCACCAGC-3’ | 55°C | 678 bp |
| speI | Reverse          | 5’-GGGATCCATGTCAGGACCACCAGC-3’ | 55°C | 678 bp |

PCR: Polymerase chain reaction
and chloramphenicol. The CLSI recommended inhibition zone sizes for βHS were used for interpretation. *Streptococcus pneumoniae* ATCC 49619 was used as control for antimicrobial susceptibility testing. The minimum inhibitory concentration (MIC) was also determined by E-test for all the above antimicrobials. The E-test was performed on 5% sheep blood agar according to manufacturer’s recommendations (Biomerieux Ltd., formerly AB Biodisk, Sweden).

PCR for *ermA*, *ermB* and *mefA* for detecting the macrolide resistance genes and *tetM* and *tetO* genes (for tetracycline resistance) was done by standard methods.[10,11]

The research work conducted in this investigation was part of an ongoing study on molecular epidemiology of Streptococci, which had received ethical clearance of the Institute’s ethical committee.

**Results**

The outbreak and its investigation occurred in a 28-bedded poly-trauma ward of a level-1 Trauma Center. The ward has a four-bedded HDU [Figure 1]. On 9th October, 2012, the tracheal aspirate and blood sample of a patient (A) in the high dependency cubicle grew a beta hemolytic *Streptococcus*. The clinicians were immediately informed. The patient was in clinical sepsis and was very toxic. She had a C5 burst fracture and was on a ventilator since July, 2012. The patient was immediately started on clindamycin and amoxicillin/clavulanic acid. The next day, the isolate was identified as GAS, showing inducible resistance to clindamycin (clindamycin MIC 0.094 μg/ml; D-test positive). The antimicrobial treatment was therefore changed to vancomycin along with amoxicillin/clavulanic acid. The tracheal aspirates and blood on 10th October also grew GAS. On the 11th October, a wound sample of another patient from the HDU (patient B) grew GAS. Since the patient had cervical spine fracture in June 2012 and was on the ventilator since then. The patient was immediately started on vancomycin. On the 12th October, the central line tip of a patient (C) sent for microbiological culture grew GAS. This patient was shifted to the ward from the ICU the previous day and the central line was removed since it was not clinically required.

Since it was unusual to get GAS from two patients in a cubicle (4 Bedded HDU), an intensive surveillance was initiated on the 11th October to trace the source of GAS and to ascertain if the same strain was causing the infection. Since a patient outside the cubicle (C) had also grown GAS on 12th October, the surveillance was enhanced to cover the entire ward. A total of 146 samples were taken. Of these, 48 were throat swabs of nursing staff in all the shifts, 4 were throat swabs of the house keeping staff, 16 were throat swabs/tracheal aspirates of other admitted patients, five were throat swabs of various attendants of the patients and 73 were environmental samples of various devices/surfaces in the wards.

Of these 146 samples, the ventilator tubing of patient B grew GAS. Apart from this, the tracheal aspirate and the tip of the suction tubing of another patient (patient D) grew GAS. This patient was a 2-year-old female, admitted to the ward since 1st August and was about to be discharged on the same day that surveillance samples were taken. Since GAS grew from her samples, she was kept in the ward and treated with amoxicillin/clavulanic acid. Her blood cultures were sterile and she was discharged after 2 days. A throat swab taken from this baby’s mother also grew GAS. She was also treated with amoxicillin/clavulanic acid. The repeat samples of
all the above patients after 5 days were sterile. However, on 21st October, the blood sample of patient B again grew GAS, which was immediately treated with vancomycin. None of the other samples from this patient or the surveillance samples taken this time grew GAS. From 21st October to 31st December, no further cases of infections due to GAS were seen in the ward. None of patients from whom GAS was isolated had a fatal outcome until 1-month follow-up.

PCR done for detection of exotoxins speA, speB, speC, speF, speG, speL, speH, speJ speM, smeZ and ssa revealed that all the isolates of GAS obtained from the various sources produced speB, speC, speG, speF and smeZ. However, none of them produced speA, speI, speM, speH, speL, speJ or ssa, suggesting that they were the same clone.

A sequence analysis of the emm gene was done for all the isolates recovered during this period. All the isolates belonged to GAS emm type 58 (GenBank accession numbers KC352715-KC352726).

All the isolates were sensitive to penicillin (MIC: 0.012-0.016 μg/ml), cefotaxime (MIC: 0.016-0.023 μg/ml), ceftiraxone (MIC: 0.016 μg/ml), ciprofloxacin (MIC: 0.38-0.75 μg/ml), vancomycin (MIC: 0.38 μg/ml) and linezolid (MIC: 0.19-0.38 μg/ml). They were resistant to erythromycin (MIC: ≥256 μg/ml) and had inducible clindamycin resistance (D-test positive, clindamycin MIC: 0.094-0.19). They were also resistant to tetracycline (MIC: 64 μg/ml). There was very little variation in the MICs of the isolates obtained from different sources. All the isolates were positive for ermA and tetM genes. They were negative for ermA, mefA and tetO genes.

The strain was very invasive, considering that it was isolated from blood samples of two patients. Moreover, in two patients, it was isolated from multiple sources [Table 2].

None of the health care workers grew GAS. Only one nursing staff had a beta-hemolytic Streptococcus from her throat, which was identified as Streptococcus dysgalactiae.

Since three of the four patients from whom GAS was recovered were admitted to the ward for a long time and none of the health care worker were found to carry the GAS strain, we can only speculate that the strain was introduced to the ward through the mother of patient D (since the same emm type was recovered from her throat) and spread through the ward due to suboptimal hand hygiene/infection control measures. We have an intensive, automated ongoing hospital acquired infection surveillance network and hand hygiene monitoring system.[12,13] The hand hygiene compliance for the months of September and October were respectively 64% and 73%. Since the isolate was obtained from suction tips and catheter tips, there was a definite lapse in infection control precautions in the ward. One of the reasons could be the involvement of patient’s attendants in tracheal/oral suctioning, without being properly trained and not being monitored by staff. Suboptimal disinfection/cleaning of devices, which is done by house-keeping staff may also be a factor contributing to cross-transmission of the pathogen.

The nursing staff was thoroughly sensitized about the need to augment the implementation of infection preventive measures. Training of new recruits is being continuously done on all aspects of hospital infection control.

**Discussion**

This is the first reported outbreak of M type 58 GAS. No study from India on invasive/non-invasive GAS has reported finding of emm 58.[14-16] In an ongoing study on molecular epidemiology of GAS at our Center, we have found that of the 126 invasive and non-invasive GAS strains from north and south India, there was only one emm 58 isolate (unpublished data). In this ongoing study, from January, 2007 to June, 2012, a total of 85 isolates of βHS were isolated from trauma patients. Of these, four were recovered from blood (only one of which was GAS, rest being GGS). All these four patients had a fatal outcome.

Emm 58 was not found in many large-scale studies from Nepal, China, Taiwan, USA and Serbia.[17-20] Studies from

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**Table 2: Sources of GAS and their clinical significance**

| Patient | Samples growing GAS                      | Clinical condition     | Treatment         | Outcome                  |
|---------|------------------------------------------|------------------------|-------------------|--------------------------|
| A       | Blood, wound aspärate, tracheal aspirate, throat | Patient in clinical sepsis | Vancomycin        | Recovered and discharged |
| B       | Blood, tracheal aspärate, throat         | Patient in clinical sepsis | Vancomycin        | Recovered and discharged |
| C       | Central line tip                         | Patient stable         | Amoxicillin/clavulinic acid | Recovered and discharged |
| D       | Tracheal aspirate, tip of suction tubing | Patient stable and was about to be discharged | Amoxicillin/clavulinic acid | Recovered and discharged |

GAS: Group A Streptococcus
Italy, Denmark, Argentina, Australia and Canada have found a very low prevalence (0.5-3%) of emm 58 amongst all GAS isolates. It is probably most prevalent in Japan, where it has been reported to account for 13-38% of the isolates in various studies.

Hospital acquisition of invasive GAS infections has often been reported in the literature. In a meta-analysis from 1992 to 2000 in Canada, fifteen outbreaks were identified; 9 (60%) of them involving only two cases. Hospital staff were infected in 1 of the 15 outbreaks, but colonized staff were identified in 6 (60%) of the 10 investigations in which staff were screened. GAS outbreaks have been identified in burns units, closed communities and long-term acute care hospitals. However, to the best of our knowledge, this is the first outbreak in a trauma care set-up, which houses highly susceptible, predominantly young adult population.

In most of the reported outbreaks of GAS, multiple emm types have been observed, suggesting multiple sources. However, in our study, a single emm type was isolated from all cases, suggesting a single source and cross-transmission. The isolate was a very virulent and invasive strain as it was isolated from blood culture and multiple sites of two patients both of them had a spinal injury. Three out of the four patients involved in this outbreak were admitted in the hospital for a prolonged time.

The Streptococcus pyrogenic exotoxins (SPEs) of GAS play an important role in its pathogenesis. The isolates in this outbreak produced speB, speC, speG, speF and smeZ. speC is carried on mobile elements and can be easily mobilized by lysogenic phages into non-toxicogenic strains, facilitating dissemination of toxigenicity.

The major limitation of our study was that we could not ascertain the actual source of the outbreak. Therefore, we could not undertake specific measures for source control. We could have been more aggressive in taking surveillance samples from naso-pharynx, scalp, per-vaginal and peri-anal etc., of the patients, as they have been implicated as possible sources of colonization and infection in earlier reported Streptococcal outbreaks. The doctors taking care of these patients are on daily rotation and they work in different shifts, which was a major hindrance in collecting surveillance samples from the doctors. However, we tried to take surveillance swabs from as many patients and their attendants and health care workers as possible. The best possible intervention in this situation of uncertainty was that all the colonized or infected patients, attendants and health care staff were treated with antibiotics along with enhanced hand hygiene and other hospital infection control practices.

Considering the high mortality of beta-hemolytic Streptococcal bacteremia, as observed in our set-up (unpublished data), we feel that we could control this outbreak through intensive surveillance, prompt treatment, attempted source tracing, molecular characterization, training and feedback.

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