



## SCREENING AND LABORATORY DIAGNOSIS OF GROUP B STREPTOCOCCUS (GBS) IN PREGNANT WOMEN TO PREVENT NEONATAL GBS SEPSIS

\*Dr. Nandini Loganathan

Consultant Microbiologist & Infection Control Officer, Cloudnine Hospital, India

### ARTICLE INFO

#### Article History:

Received 27<sup>th</sup> March, 2021  
Received in revised form  
15<sup>th</sup> April, 2021  
Accepted 20<sup>th</sup> May, 2021  
Published online 26<sup>th</sup> June, 2021

#### Key Words:

GBS, Antibiotics,  
Laboratory Diagnosis,  
Neonates, Risk Factors.

### ABSTRACT

Group B Streptococcus (GBS) is a leading cause of invasive neonatal infections and a significant pathogen in immunocompromised adults. Screening to detect GBS colonization in pregnant women determines the need for antibiotic prophylaxis in that pregnancy. Universal screening for GBS and intrapartum antibiotic prophylaxis remains the main preventive strategy to reduce the neonatal mortality and morbidity secondary to GBS. Efficient determination of the GBS colonization status of pregnant women is crucial. This article presents the risk of GBS colonization and the various diagnostic methods for GBS species determination and the treatment.

Copyright © 2021. Nandini Loganathan. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Citation: Dr. Nandini Loganathan. "Screening and laboratory diagnosis of group B streptococcus (GBS) in pregnant women to prevent neonatal GBS sepsis.", 2021. *International Journal of Current Research*, 13, (06), 17792-17796.

## INTRODUCTION

GBS is one of the causes of invasive neonatal infections. Neonatal infections can be early or late onset. Group B Streptococcus (group B strep, GBS) emerged in the 1970s as the most common cause of sepsis in newborns. Early-onset disease declined by 80% with the use of intrapartum prophylaxis. GBS can cause serious illnesses like septicemia, pneumonia, and even meningitis (1). Group B strep (GBS) is a type of bacteria that is naturally found in the digestive and lower reproductive tracts of healthy human adults. About 1 in 4 pregnant women are colonized with GBS. Colonized with GBS does not mean that there is an infection or the person is unclean. Anyone can colonize GBS (2, 3). Unfortunately, babies can be infected by GBS before birth through several months of age due to their underdeveloped immune systems. Few babies who are exposed to GBS become infected, but GBS can cause babies to be miscarried, stillborn, born preterm or become very sick and sometimes even die after birth (2,3).

GBS can cause serious illnesses like septicemia, pneumonia, and even meningitis. Some GBS survivors experience handicaps such as blindness, deafness, mental challenges, and/or cerebral palsy.(3) Most GBS infections that develop at birth can be prevented if women who tested positive receive at least 4 hours of intravenous antibiotics just prior to delivery.(3, 4). Screening and laboratory diagnosis of the GBS colonization status of pregnant women is crucial to prevent neonatal GBS infections.

**GBS IN PREGNANT WOMEN:** Group B Streptococci (GBS) are Gram-positive cocci that form short chains in clinical specimens and longer chains in culture; features that make them indistinguishable on Gram stain from *Streptococcus pyogenes*. (21) *Streptococcus agalactiae* is the only Streptococcus species harboring the Lancefield group B cell-wall-specific polysaccharide antigen. On the basis of type-specific capsular polysaccharides GBS can be subdivided into 10 different serotypes (Ia, Ib, and II to IX). (1,14). Approximately 10%–30% of pregnant women are colonized with GBS in the vagina or rectum. (15,16, 17) GBS colonization during pregnancy can be transient, intermittent, or persistent (18,19,20). Although some women with GBS colonization during a pregnancy may be colonized during subsequent pregnancies, a substantial proportion may not be colonized (8).

\*Corresponding author: Nandini Loganathan,  
Cloudnine Hospital, India.

GBS colonization most commonly does not cause any symptoms. Some GBS colonized women can present with vaginal burning, vaginal irritation or abnormal discharge which may be mistaken for Candida and treated incorrectly. GBS can cause abortion, stillbirths, preterm labor, and premature rupture of membranes (2). At times GBS also causes significant maternal morbidity, including endometritis, chorioamnionitis, bacteremia, puerperal sepsis and postpartum wound infections. Occasionally can cause meningitis, septic thrombophlebitis, or other serious complications. (6,5). Although GBS seldom causes disease in healthy adults, it is responsible for serious infections in diabetics, elderly individuals, and immunocompromised patients (5). GBS can also cause urinary tract infections, which can be symptomatic or asymptomatic. GBS in the urine indicates heavy colonization and has a greater risk of transmission to the neonate if not treated. Hence a urine culture for GBS is recommended at the first prenatal visit. (3,2) GBS colonization in pregnant women is a major risk factor for the emergence of early neonatal GBS infection contributing to neonatal morbidity and mortality.

### NEONATAL GBS INFECTION

In neonates, GBS can present as early onset GBS (EOGBS) or late onset GBS (LOGBS) infection (1). EOGBS occurs within the first week of life usually within the first 24 hours. (3, 8) It is by vertical transmission of GBS from a colonized mother to the newborn, through either ascending infection from the genital tract or GBS transmission to the newborn during labor and birth. (7). EOGBS occurs mainly after the onset of labor or in connection with ruptured membranes, although infection of the fetus can happen through intact membranes also. Bacteremia without a focus is the most common clinical syndrome, followed by pneumonia and meningitis. Early onset GBS infection has more fatal outcomes among premature neonates (8,6,9). LOGBS presents after 7 days of age, about 7 to 90 days postpartum. (3, 8) LOGBS is most likely acquired after birth, from breast milk or from nosocomial or community sources. Prematurity is the main risk factor for developing LOGBS, and bacteremia without a focus of infection is the most common presentation. The mortality rate for LOGBS is lower, but meningitis and subsequent sequelae are more frequently associated with LOGBS (5, 1).

**PREVALENCE OF GBS COLONIZATION:** Prevalence of GBS colonization varies among various developed and developing countries. About 15-40% of pregnant women are estimated to be carriers of GBS in the United States of America (17). The transmission rate from the colonized mother to the newborn is estimated to be 40-73%. Among this, 1-2% develop early onset sepsis (11). Various other developed nations have shown GBS colonization rates as: United Kingdom (Oxford) 21.3% (42), Canada 19.5% (41), and Sweden 25.3% (43). The prevalence of GBS colonization from developing countries comparatively shows a lower rate: Lebanon 17.7% (44), Brazil 17.9% (45), from India (Vellore) 5.8% (46), and (Pondicherry) 2.3% (47); except for Zimbabwe which shows a higher rate of 60.3% (48). In a study by Santhanam et al., from CMC Vellore (India), showed that 7.6% of mothers were colonized with GBS. Approximately two-third of new born to the above mothers were colonized. No instances of invasive GBS cases reported, as intrapartum antibiotic prophylaxis was given. (49)

Study by Nancy et. al, a meta-analysis by collecting data from 34 (1981-2017) studies comprising 9552 cases showed the prevalence of GBS infection in pregnant Indian women (35-37 weeks of gestation) as 7.7 % (744/9552). The prevalence of GBS colonization was higher in younger women (18-25 years) as compared to older (30-35 years) women (6.7% vs 3.6%), The risk of preterm delivery and PROM was higher amongst GBS infected women as compared to uninfected women.(50)

**RISK FACTORS FOR NEONATAL GBS:** Recto-vaginal colonization with GBS in pregnant women is the primary risk factor for EOGBS infection. Other Maternal and obstetric factors like gestational age <37 completed weeks, longer duration of membrane rupture, intra-amniotic infection, young maternal age, black race, and low maternal levels of GBS-specific anticapsular antibody also contribute to the risk factors (8). A previous sibling with invasive GBS disease may indicate a low level of specific GBS antibodies in the mother, a risk factor that could persist in subsequent pregnancies. (11, 12) Black race, young maternal age and certain obstetric procedures such as the use of intrauterine fetal monitoring devices or performing five or more vaginal examinations during labour are other risk factors. (10, 8,13)

**GBS SCREENING:** Since 1996, the CDC in collaboration with several professional societies has published recommendations for GBS screening. In 2019, the stewardship of these guidelines was transferred to three professional organizations ACOG, APP and ASM. The guidelines for prophylaxis and treatment of GBS infection in pregnant women and newborns are curated by ACOG and AAP. The American Society for Microbiology (ASM) is responsible for maintaining and updating guidelines for standard laboratory practices related to detection and identification of GBS (22, 23). Universal antepartum screening for GBS and appropriate intrapartum antibiotic prophylaxis (IAP) still remains the main critical component for preventing early-onset GBS neonatal disease. Noteworthy changes in the most recent ACOG guidelines include the recommendation for antepartum screening for GBS at 36 0/7 to 37 6/7 weeks of gestation (22). This is a change from the recommendation of 35 0/7 weeks of gestation from the 2010 CDC guidelines (24). ACOG provides complete guidelines for GBS prophylaxis during pre-term delivery and women with unknown GBS status (22).

### SPECIMEN COLLECTION AND PROCESSING:

**Vaginal-rectal swabs:** A single swab instead of two swabs can be used to collect the vaginal and rectal samples. Vaginal-rectal swabs have shown to have high bacterial yield. (25-28). Without using a speculum, the specimen is first collected from the vagina near the introitus by inserting the swab about two centimeters and the same swab is inserted one centimeter into the anal sphincter to collect the rectal specimen. (26, 27, 29-31). Flocked swabs are recommended to collect the sample and immediately placed into Amies transport medium or equivalent and transported within 24 hours to the laboratory. If processing or transport of the sample is delayed, can be refrigerated in Amies transport medium at 4- 8°C or store specimens collected in Eswabs at room temperature (32, 33). Culturing specimens greater than 24 hours after collection may yield false-negative results and is strongly discouraged. If delayed beyond 24 hours, rejection and recollection of specimen requested.

**Urine Culture:** Routine screening for asymptomatic bacteriuria in pregnant women is recommended. Detection of GBS,  $10^4$  colony-forming units (CFU)/ml or greater is significant. (8)

**Early onset GBS disease:** Infants with signs of early-onset GBS disease, culture of blood, cerebrospinal fluid (CSF) and other samples depending on the clinical presentation to be collected and processed. (8)

**PROCESSING:** GBS screening specimens are incubated in selective enrichment broth [Todd Hewitt Broth with gentamicin (8 µg/ml) and nalidixic acid (15 µg/ml), Lim Broth, Carrot Broth or Granada Liquid Biphasic broth] prior to agar media plating or NAAT. (27,30, 34). Tryptic Soy Agar with 5% Sheep's Blood; Columbia Agar with 5% Sheep's Blood; Columbia Agar with 5% Sheep's Blood, with colistin and nalidixic acid; Brilliance GBS, ChromID StrepB, Chromagar, StrepBSelect are acceptable media for GBS culture and isolation (35). Standard microbiological methods to be used for plating, incubating and identification of Group B Streptococci. CAMP test, latex agglutination or MALDI can be used for the identification of the candidate isolates (36, 37, 38). Nucleic acid amplification testing (NAAT) from enrichment broth can also be used for identification of GBS. Latex agglutination directly from enrichment broth and direct-from-specimen immunoassays are unacceptable methods for GBS detection. Culture media and GBS isolation methods should detect both hemolytic and non-hemolytic strains. (38)

**SUSCEPTIBILITY TESTING:** *Streptococcus agalactiae* (GBS) remain predictably susceptible to penicillin and cefazolin. Therefore, routine AST is not required prior to administration of these agents. (39). Antimicrobial susceptibility testing is recommended on all GBS isolates from pregnant women with penicillin allergy. ACOG 2019 guidelines recommends intravenous penicillin as the preferred agent for intrapartum GBS prophylaxis for non-penicillin-allergic women. In case of non-severe penicillin allergies, first generation cephalosporins like cefazolin are recommended. (22). In case of severe penicillin allergy, Clindamycin is the recommended agent for GBS prophylaxis. AST for both Clindamycin and Erythromycin is recommended by Clinical and Laboratory Standards Institute (CLSI). Erythromycin is used as screening tool for possible inducible clindamycin resistance. Both Clindamycin and Erythromycin are tested, but only Clindamycin needs to be reported. (39)

GBS isolated from urine culture of penicillin-allergic pregnant women are also tested for clindamycin resistance. Though clindamycin being inappropriate for treatment of urinary tract infection, tested for use in intrapartum GBS prophylaxis. Significant count of GBS isolated in the urine culture is a mark of high level of GBS colonization. (22) Additional or alternative agent reporting may be considered at individual institutions, but is not broadly recommended. Laboratories must develop methods to ensure AST is performed for all at-risk patients.

**Recommended regimens for intrapartum antibiotic prophylaxis for prevention of early-onset group B streptococcal (GBS) disease. (40)**

) Penicillin is the drug of choice when not allergic to it.

- ) Penicillin G, 5 million units IV load, then 2.5-3 million units IV every 4 hours until delivery.
- ) OR
- ) Ampicillin 2 grams IV load, then 1 gram every 4 hours until delivery.
- ) When allergic to Penicillin, depending on the risk assessment of allergy other alternative drugs are chosen.
- ) LOW RISK – Cefazolin 2 gram IV load, followed by 1 gram IV every 8 hours until delivery.
- ) HIGH RISK – Check for the antibiotic susceptibility of the GBS

**Susceptible to Clindamycin-** Clindamycin 900 mg IV every 8 hours until delivery

**Resistant to Clindamycin – Vancomycin** is the drug of choice (weight based dosage of 20 mg/kg every 8 hours). Maximum single dose is 2 gram. Minimum infusion time is 1 hour, or 500 mg/30 min for a dose >1 g.

) **UNKNOWN RISK:** When there is no history of allergy to penicillin available, the following options can be followed:

#### Penicillin allergy testing

Administration of a cephalosporin

Administration of Clindamycin if susceptible isolate

Administration of Vanomycin if isolate is not susceptible to Clindamycin

**LIMITATIONS OF SCREENING:** Approximately about 50-60 % of women give birth at home in India according to the National Family Health Survey (NFHS). Hence the true incidence of GBS colonization and invasive GBS disease in the neonates is unknown. Culturing of blood from the ill neonates are not always done in the rural primary healthcare centers. Abortions, stillbirths, pre-term births not investigated also adds on the unrecognized GBS cases. (51). Factors contributing to sample processing, such as use of appropriate transport media, sample processing within 48 hours from the time of collection, systematic inoculation in selective enrichment broth further sub-cultured on selective differential media not followed could also add to the under-reported Group B Streptococcus. (10)

## CONCLUSION

Universal screening for GBS and intrapartum antibiotic prophylaxis remains the main preventive strategy to reduce the neonatal mortality and morbidity secondary to GBS. Implementation of universal screening strategy or clinical risk based strategy can be made depending on the prevalence of the risk factors for early onset GBS disease and the obstetric practice followed. The empirical choice for such infants should include an antibiotic to which group B streptococci are susceptible, such as a -lactam, cephalosporin, or vancomycin. When group B streptococci are identified in culture, penicillin G is the drug of choice, with ampicillin as an acceptable alternative therapy. Vancomycin should be added to recommended empirical therapy if there is evidence of meningitis or critical illness to expand coverage. Currently, there is no vaccine to protect newborns from GBS bacteria and disease. Vaccine for GBS in under the process of development and research which could be expected in the future.

**ABBREVIATIONS:**

GBS – Group B Streptococcus

EOGBS – Early onset group B Streptococcus

LOGBS – Late onset group B Streptococcus

CDC – Centre for Disease Control and Prevention

ACOG - American College of Obstetricians and Gynecologists

ASM - American Society for Microbiology

IAP - Intrapartum Antibiotic Prophylaxis

AST - Antimicrobial Susceptibility Testing

**REFERENCES**

- Rosa-Fraile M, Spellerberg B. 2017. Reliable detection of group B streptococcus in the clinical laboratory. *J Clin Microbiol* 55:2590–2598.
- CDC, International Group B Streptococcus awareness broucher, Aug 2017.
- Center for Disease Control and Prevention, 10 July 2017 “Protect Your Baby from Group B Strep.”
- Shet A, Ferrieri P. Neonatal and maternal group B streptococcal infections: A comprehensive review. *Indian J Med Res* 2004;120:141-50.
- Edwards MS, Baker CJ. 2005. Group B streptococcal infections in elderly adults. *Clin Infect Dis* 41:839 – 847.
- Schrag SJ, Zywicki S, Farley M et al. Group B streptococcal disease in the era of intrapartum antibiotic prophylaxis. *N Engl J Med* 2000;342: 15–20.
- Desa DJ, Trevenen CL. Intrauterine infections with group B beta-haemolytic streptococci. *Br J Obstet Gynaecol* 1984;91:237-9
- CDC, Morbidity and Mortality Weekly Report (MMWR). Prevention of Perinatal Group B Streptococcal Disease. Revised Guidelines from CDC, 2010 Recommendations and Reports November 19, 2010/59(RR10);1-32
- Phares CR, Lynfield R, Farley MM, Mohle-Boetani J, Harrison LH, Petit S, et al. Active Bacterial Core surveillance/Emerging Infections Program Network. Epidemiology of invasive group B streptococcal disease in the United States, 1999-2005. *JAMA* 2008;299:2056-65.
- Melin P. Neonatal group B streptococcal disease: from pathogenesis to preventive strategies. *Clin Microbiol Infect.* 2011 Sep;17(9):1294-303.
- Verani JR, Schrag SJ. Group B streptococcal disease in infants: progress in prevention and continued challenges. *Clin Perinatol* 2010; 37: 375–392.
- Faxelius G, Bremme K, Kvist-Christensen K, Christensen P, Ringertz S. Neonatal septicemia due to group B streptococci – perinatal risk factors and outcome of subsequent pregnancies. *J Perinat Med* 1988; 16: 423–430
- Van Dyke MK, Phares CR, Lynfield R et al. Evaluation of universal antenatal screening for group B streptococcus. *N Engl J Med* 2009; 360: 2626–2636.
- Baker CJ, Edwards MS Group B Streptococcal INFECTIONS. In: Remington JS, Klein JO, editors. Infectious diseases of the fetus and newborn infant Philadelphia; W.B, Saunders; 2001 p, 1091 -156.
- Regan JA, Klebanoff MA, Nugent RP. The epidemiology of group B streptococcal colonization in pregnancy. Vaginal Infections and Prematurity Study Group. *Obstet Gynecol* 1991;77:604-10.
- Yancey MK, Schuchat A, Brown LK, Ventura VL, Markenson GR. The accuracy of late antenatal screening cultures in predicting genital group B streptococcal colonization at delivery. *Obstet Gynecol* 1996;(88):811--5.
- Campbell JR, Hillier SL, Krohn MA, Ferrieri P, Zaleznik DF, Baker CJ. Group B streptococcal colonization and serotype-specific immunity in pregnant women at delivery. *Obstet Gynecol* 2000 Oct;96(4):498--503.
- Lewin EB, Amstey MS. Natural history of group B *Streptococcus* colonization and its therapy during pregnancy. *Am J Obstet Gynecol* 1981;139:512--5.
- Hoogkamp-Korstanje JA, Gerards LJ, Cats BP. Maternal carriage and neonatal acquisition of group B streptococci. *J Infect Dis* 1982;145:800--3.
- Hansen SM, Uldbjerg N, Kilian M, Sorensen UB. Dynamics of Streptococcus agalactiae colonization in women during and after pregnancy and in their infants. *J Clin Microbiol* 2004;42:83--9.
- Procop, G. W., & Koneman, E. W. (2016). *Koneman’s Color Atlas and Textbook of Diagnostic Microbiology* (Seventh, International edition). Lippincott Williams and Wilkins.
- Committee on Obstetric Practice. 2019. Prevention of early-onset group B streptococcal disease in newborns. *Obstet Gynecol* 134:e19–e40.
- Puopolo KM, Lynfield R, Cummings JJ. 2019. Management of infants at risk for group B streptococcal disease. *Pediatrics* 144.
- Verani JR, McGee L, Schrag SJ. 2010. Prevention of perinatal group B streptococcal disease revised guidelines from CDC, 2010. *Morb Mortal Wkly Rep* 59:1–31.
- Dunne WM, Holland-Staley CA. 1998. Comparison of NNA agar culture and selective broth culture for detection of group B streptococcal colonization in women. *J Clin Microbiol* 36:2298–300.
- El Aila NA, Tency I, Claeys G, Saerens B, et al. 2010. Comparison of different sampling techniques and of different culture methods for detection of group B streptococcus carriage in pregnant women. *BMC Infect Dis* 10.
- Philipson E, Palermينو D, Robinson A. 1995. Enhanced Antenatal Detection of Group B Streptococcus Colonization. *Obstet Gynecol* 85:437–439.
- Votava M, Tejkalová M, Drábková M, Unzeitig V, Braveny I. 2001. Use of GBS media for rapid detection of group B streptococci in vaginal and rectal swabs from women in labor. *Eur J Clin Microbiol Infect Dis* 20:120–2.
- Dillon HC, Gray E, Pass MA, Gray BM. 1982. Anorectal and vaginal carriage of group B streptococci during pregnancy. *J Infect Dis* 145:794–9.
- Platt MW, McLaughlin JC, Gilson GJ, Wellhoner MF, Nims LJ. 1995. Increased recovery of group B Streptococcus by the inclusion of rectal culturing and enrichment. *Diagn Microbiol Infect Dis* 21:65–8.
- Quinlan JD, Hill DA, Maxwell BD, Boone S, Hoover F, Lense JJ. 2000. The necessity of both anorectal and vaginal cultures for group B streptococcus screening during pregnancy. *J Fam Pract* 49:447–8.
- Trotman-Grant A, Raney T, Dien Bard J. 2012. Evaluation of optimal storage temperature, time, and transport medium for detection of group B streptococcus in StrepB carrot broth. *J Clin Microbiol* 50:2446–2449

33. Rosa-Fraile M, Camacho-Muñoz E, Rodríguez-Granger J, Liébana-Martos C. 2005. Specimen storage in transport medium and detection of group B streptococci by culture. *J Clin Microbiol* 43:928–30.
34. Altaie SS, Dryja D. 1994. Detection of Group B Streptococcus. Comparison of Solid and Liquid Culture Media With and Without Selective Antibiotics. *Diagn Microbiol Infect Dis* 18:141–144.
35. Verani JR, McGee L, Schrag SJ. 2010. Prevention of perinatal group B streptococcal disease revised guidelines from CDC, 2010. *Morb Mortal Wkly Rep* 59:1–31.
36. Christie K, Atkins N, Munch-Petersen E. 1944. A note on a lytic phenomenon shown by group B streptococci. *Aust J Exp Biol Med Sci* 22:197–200.
37. Lancefield RC. 1933. A Serological Differentiation of Human and Other Groups of Hemolytic Streptococci. *J Exp Med* 57:571–595.
38. Filkins L, Hauser J, Robinson-Dunn B, et al. 10 March 2020. Guidelines for the Detection and Identification of Group B Streptococcus. American Society for Microbiology.
39. CLSI. 2019. Performance Standards for Antimicrobial Susceptibility Testing. 29th ed. CLSI.
40. Prevention of group B streptococcal early-onset disease in newborns. ACOG Committee Opinion. American College of Obstetricians and Gynecologists. *Obstet Gynecol* 2020; 135:e51-72.
41. Davies HD, Adair C, McGeer A, Ma D, Robertson S, Mucenski M, et al. Antibodies to capsular polysaccharides of group B Streptococcus in pregnant Canadian women: relationship to colonization status and infection in the neonate. *J Infect Dis* 2001; 184: 285-91.
42. Jones N, Oliver K, Jones Y, Haines A, Crook D. Carriage of group B streptococcus in pregnant women from Oxford, UK. *J ClinPathol* 2006; 59: 363-6.
43. Hakansson S, Axemo P, Bremme K, Bryngelsson AL, Wallin MC, Ekstrom CM, et al. Group B streptococcal carriage in Sweden: a national study on risk factors for mother and infant colonisation. *Acta Obstet Gynecol Scand* 2008; 87: 50-8.
44. Seoud M, Nassar AH, Zalloua P, Boghossian N, Ezeddine J, Fakhoury H, et al. Prenatal and neonatal Group B Streptococcus screening and serotyping in Lebanon: incidence and implications. *Acta Obstet Gynecol Scand* 2010; 89: 399-403.
45. Zusman AS, Baltimore RS, Fonseca SN. Prevalence of maternal group B streptococcal colonization and related risk factors in a Brazilian population. *Braz J Infect Dis* 2006; 10: 242-6
46. Mani V, Jadhav M, Sivadasan K, Thangavelu CP, Rachel M, Prabha J. Maternal and neonatal colonization with group B Streptococcus and neonatal outcome. *Indian Pediatr* 1984; 21: 357-63.
47. Sharmila V, Joseph NM, ArunBabu T, Chaturvedula L, Sistla S. Genital tract group B streptococcal colonization in pregnant women: a South Indian perspective. *J Infect Dev Ctries* 2011; 5: 592-5.
48. Mavenyengwa RT, Afset JE, Schei B, Berg S, Caspersen T, Bergseng H, et al. Group B Streptococcus colonization during pregnancy and maternal-fetal transmission in Zimbabwe. *Acta Obstet Gynecol Scand* 2010; 89: 250-5.
49. Santhanam et al. Prevalence of group B Streptococcal colonization among pregnant women and neonates in a tertiary hospital in India. *J Turk Ger Gynecol Assoc* 2017; 18: 181-4.
50. Ashary, Nancy and Modi, Deepak, A Meta-Analysis for Association of Maternal Group B Streptococcus Colonization and Preterm Birth in Indian Population. *Lancet*: October 19, 2018.
51. Narava S, Rajaram G, Ramadevi A, Prakash GV, Mackenzie S. Prevention of perinatal group B streptococcal infections: A review with an Indian perspective. *Indian J Med Microbiol* 2014;32:6-12.

\*\*\*\*\*