


Group g strep treatment guidelines

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Strep infections are commonly associated with skin and throat infections due to group A streptococcus (*S. pyogenes*). Nongroup streptococcus have also been involved in a mild to serious infection. Group B (*S. agalactiae*), group C and G streptococcus, and viridance group streptococcus (VGS) are known to colonize the human airways, gastrointestinal tract and diuretic tract. These bacteria are pathogenic under the right conditions. MICROBIOLOGY Traditionally streptococcus is classified using Lancefield group antigens and haemolysis on blood agar. The antigen group Lancefield does not correlate with the view. The classification of hemolysis is inaccurate. Molecular taxonomic studies have improved classification. Beta-hemolytic isolates under the lancefield A, C, F and G group are divided into large and small colony formation groups. Large groups of colonies have numerous virulence mechanisms and are labeled as piogen. A large colony of group C streptococcus is usually resistant to bacitracin. This method is used by many clinical laboratories in The Streptococcus Group A (GABHS) in many clinical laboratories. However, some groups of C Streptococci (GCS) are susceptible to bacitracin and can lead to misidentification if The Serological typing of Lancefield is not performed. Among the G group streptococcus (GGS), the sensitivity of bacitracin is reportedly higher than 67% (87). Trimethoprim/sulfamethoxazole (SXT) testing has been added to improve identification. Both GCS and GGS are receptive and GABHS are resilient. A serogrouping of the reagent is used for specific identification. The large colony of Lancefield GCS is variably classified into some of several possible species, namely *S. dysgalactiae*, *S. equisimilis*, *S. zooepidemicus* and *S. equi* (36). These species can be differentiated by microbiological and biochemical characteristics. All but *S. dysgalactiae* usually cause beta-hemolysis in the blood of the agar. *S. equisimilis* is the most common GCS cause infection in humans, but can also infect pets. Other species mainly infect animals. Most clinical laboratories do not bask GCS isolates. Small groups of colony formation are classified under the group Anginosus, formerly known as the group *S. milleri*, or group *S. intermedius*. While these small colonies of organisms may possess Lancefield groups A, C, F, G, and ungroupable antigen, they are commensals and rarely pathogenic in themselves. For example, the organisms of the Anginosus group with group A antigen may be differentiated from the *S. piogens* of their small colony of former and resistant to bacitracin. The Anginosus or *S. milleri* group is one of 5 VGS groups based on 16S rRNA sequences. Other groups of viridances classified by this method include the *Mitis* group, the *Salivarius* group, the *Bovis* group. Mutans (see table 1) (39). (39). was the term waste basket referring to streptococcus, which produce partial or no hemolysis on agar blood and were not grouped. VGS is established by excluding *S. pyogenes*, enterococci, pneumocococcons, *S. agalactiae* and large colonies of groups C and G (16), and has the following characteristics: vancomycin susceptibility, produces the enzyme aminopeptidase leucine, and does not produce pyrrolidoneyl arylaldamse (PYRR). Group B streptococcus (GBS) has only one species, i.e. *S. agalactiae*, but subclassified to 7 capsule serotypes, namely: Ia, Ib, II, III, IV, V and VI. Type III is most commonly associated with neonatal disease, while Ia and Ib with adult diseases (25.91). GABS is discussed elsewhere in this tutorial. This chapter discusses antimicrobial treatment of PYogen groups C and G, as well as VGS. He will not discuss the treatment of infections caused by *S. pyogenes*. *S. agalactiae* and *S. pneumoniae*. GROUP C And G STREPTOCOCCI Clinical manifestations Large colony forming GCS and GGS are part of normal human skin and oral flora (table I). Human infections due to GCS and GGS are much less common than GABHS and GBS. GCS and GGS have been linked to pharyngitis, although their exact role is unknown. Clinical manifestations do not differ clinically from GABA. Their association with rheumatic fever was investigated, but definitely not established (32), and their association with glomerulonephritis was anecdotal (36). Skin infection is the second most common. Other infections include puerperal and neonatal infections, bacteremia, endocarditis, meningitis, arthritis, osteomyelitis, pneumonia, toxic shock syndrome and rhabdomyolysis (8, 10, 11, 43, 48, 51, 75, 77, 79, 87, 89, 90). Infections with these organisms are common in patients with comorbidities such as chronic lung or heart disease, diabetes, malignancies (especially GGS), alcoholism and immunosuppressive therapy. GCS are common pathogens in animals, and many patients with this infection have a history of exposure to animals. In vitro Susceptibility penicillins, cephalosporins, carbapenems and vancomycin are the most active antimicrobial agent against Group C and G streptococcus (table 2-Table 4). Most strains are very sensitive to G penicillin with MIC zlt:0.05 zg/ml. strains with mic'gt:0.1 micrograms/ml are rarely found (56). A wide range of penicillins are also active. Rolston et al reported MIC90s 0.03 and 0.06 micrograms/ml for piperacillin and azlocillin respectively in 44 GCS and GGS isolates (62). Cephalotin and cefotaxim are very active with MIC90s 0.06 and 0.12 micrograms/ml respectively (62). Kansenshoyaku et al noted that all GGS and 99% of GCS were susceptible to 0.2 micrograms/ml of cephalotin (37). Other cephalosporins were less active, such as MIC90 for cephalixin was 3.13 micrograms/ml, and MIC90 for zlt/0.05 qgt:GCS and GGS, and MIC90 for cefaclor were 1.56 and 3.13 micrograms/ml respectively (37). Vancomycin is active with most isolates and has MIC90 zlt:0.5 mg/ml. Rolston et al reported that isolates with MIC 4.0 micrograms/ml isolates from cancer patients (69). Imipenem is active against both GCS and GGS, all isolates estimated by Muro et al were susceptible (47). The susceptibility of GCS and GGS to clindamycin and macrolimides is variable, and recent studies have reported some resistance (37, 43, 47, 62, 65). Kansenshoyaku et al found MIC90 for erythromycin 0.1 micrograms/ml for both GCS and GGS, but 16 of the 463 strains of GGS had MIC above 1.0 micrograms/ml (37). Kataja et al from Finland reported that 95% of the 21 macrolide-resistant GCS had the gene *mefA* or *mefE* drug efflux, and 94% of the 32 macrolide-resistant GGS had the *ermR* *emmatase* gene (38). Using guidelines from the National Committee on Clinical Laboratory Standards (NCCLS), Muro et al reported in vitro susceptibility to 113 GCS isolates and 35 GGS isolates received from 1992 to 1995. They were all susceptible to penicillin G, ampicillin, amoxicillin/claulanath, cefotaxim, imipenem, rifampin, and vancomycin (47). For GCS strains, the percentage of strains resistant to other antimicrobials were: erythromycin and azithromycin, 9%; Clindamycin, 8%; chloramphenicol, 2%; tetracycline, 17%; trimethoprite/sulfamethoxazole (SXT), 20% (47). For GGS strains, the percentage of resistance to other antimicrobials was: erythromycin, 32%; azithromycin, 16%; Clindamycin, 19%; chloramphenicol, 0%; tetracycline, 39%; and SXT, 23% (47). Tolerance, a condition in which bactericidal activity is more than 32 times greater than bacteriostatic activity, has been reported by numerous investigators (50, 56, 63). Saucis et al reported a tolerance for vancomycin in 54% of the 32 ISPS and GGS (97). The frequency and clinical importance of tolerance have not been established (43.87). The same team of researchers subsequently assessed the incorporation of meropenem, linzoid and hinustine/dalfopristin versus 130 clinical isolates GCS and GGS (98). MIC90 for meropenem, linesolide, hinukritinsin/dalfopristin, vancomycin and peniciline were 0.06, 2.0 and 0.25, 0.5 and 0.016 micrograms/ml respectively. Meropenem, linesolide, hinoustine/dalfopristin and penicillin were active against vancomycin-resistant or tolerant strains. Synergismicmism was demonstrated when aminoglycoside is added to penicillin, cefotaxim and vancomycin. Tailor et al found that all isolated trials have an expansion of homicide when penicillin is combined with gentamicin compared to penicillin alone (56). Rolston et al found that ISS isolates tolerant of penicillin were killed after adding gentamicin to penicillin or cefotaxim (63). Lam and Bayer compared the bactericidal interaction of penicillin, cefotaxima, or combined with gentamicin for 20 GGS isolates. Synergy was demonstrated in each combination of 80 to 90% isolates (44). Antimicrobial Therapy Reaction of INFECTIONS GCS or GGS to specific antimicrobials, as reported in the literature, is difficult to assess. Many patients in separate reports or demographic studies received several antibiotics with different doses, injection routes and duration of therapy. There are no controlled trials of the effectiveness of antimicrobials. Most patients registered with GCS and GGS infections received atenicillin or cephalosporin (often with aminoglycoside). A small number of patients were treated with other antimicrobials (vancomycin, erythromycin, clindamycin or chloramphenic). Based on in vitro data as well as recorded clinical experience, penicillin G is the preferred antibiotic (8, 10, 13, 43, 75, 87, 89). Alternative agents with relatively even activity include ampicillin, cefotaxim, imipenem and vancomycin. In vitro testing should be performed if clindamycin or macrolide are considered for therapy in light of recent reports of resistance to these agents. Less serious GCS or GGS infection (faringitis, cellulite) can be treated with similar therapies as for GABA; however, the activity in the macrolide or clindamycin test tube is not as consistent. Patients were willing to respond to therapy β-lactam antibiotic. Activity in the test tube of the new fluoroquinolones appears to be excellent for GCS and GGS, although the number of strains studied has been limited, i.e. 8 GCS, and 22 GGS (5). These new fluoroquinolones with elevated grams of positive activity such as levofloxacin, gatifloxacin, moxifloxacin, and hemifloxacin can be considered as an alternative because the MIC is consistently low (1.0 microgram/ml), but clinical data are lacking. Due to theoretical concerns about tolerance and the likelihood of synergy, many authorities have recommended combination therapy using penicillin plus gentamicin for serious infections such as sepsis and endocarditis (56, 75, 87). However, such recommendations were not substantiated by controlled studies. Watanakunachorn reported a relatively high mortality rate (40%) and a relatively high mortality rate (40%). for patients with endocarditis, despite the susceptibility of penicillin (90). This may be partly due to the high association with comorbidities of these patients. For serious infections such as endocarditis, bacteremia, and any septic condition penicillin 20 million units intravenously per day is recommended. Alternatives include 8g intravenously in separated doses. Vancomycin 2 g intravenously per day can be used for patients who cannot get beta-lactam agents. In addition, Linezolid or quinupristin/ dalfopristin can be used in beta-lactam patients, but clinical data Endocarditis therapy lasts 28 days, and in bacterimia or sepsis 14 days of therapy should be adequate. There is currently no consensus on the value of gentamicin. However, it is prudent to consider gentamicin initially to treat patients with severe infections prior to in vitro susceptibility results. If ONIC's ONIC is 0.1 micrograms/ml, the combination therapy should be used for a full course of therapy. VIRIDANS GROUPS OF STREPTOCOCCI Clinical manifestations of viridance streptococcus groups (VGS) have been recognized as low virulence. Transitional bacteria can occur after dental manipulation and often have no value in patients without predisposing conditions. It has been estimated that only 21% of positive blood cultures for VGS are clinically significant (81). VGS is the most common cause of valve endocarditis and late onset of prosthetic valve endocarditis (78, 83, 90). They have also been linked to serious scientist infections, bacteremia in neutropenic patients, neonatal sepsis and septicaemia/shock syndrome (also known as α Streptococcal Shock Syndrome) (4, 9, 24, 36). Places of colonization and VGS infection in humans are listed in Table 1. Some VGS produce dextran, which is associated with plaque formation and is closely related to infectious endocarditis in blood culture (78). Dextran production leads to the deposition of glycocalix, which promotes adherence and serves as a compliance factor. For example, *S. mutans*, *S. sanguis* (proposed nomenclature *S. sanguinis* (39.73). we will use *S. sanguis* in this chapter because of another recent proposal to keep the original name (42)), dextran-positive *S. mitis* (also known as *S. mitior*, *S. mitis* will be used in this chapter), and *S. bovis* are mouth-related to tooth disease and end. In their review of 229 cases with blood cultures positive for VGS, Dwyer et al reported that *S. mitis*, when isolated, should be considered as a clinically significant pathogen (23). *S. bovis* is a common gastrointestinal commensal and is reported to cause bacterioidia, endocarditis and meningitis (58.72). *S. bovis* biotype I bacteremia has been shown to be heavily associated with gastrointestinal malignancies while biotype II and *S. salivarius* are less likely to be associated (74). Anginosus group (also known as *S. milleri* or *S. intermedius* group) isolates have been associated with plying infections in oral, thoracic, abdominal and central nervous system sites (31). *S. anginosus* has been found mainly in the genitourinary tract and gastrointestinal tract, *S. constellatus* from the chest, and *S. intermedius* from the central nervous system, head and neck, and abdomen (35.92). Singh et al review 186 cases of *S. anginosus* infections and found 110 at least one abscess is identified. Among their 33 cases of bacteriology due to *S. milleri*, Salavert, et., noted that approximately 60% were intraabdomine by origin (76). Due to the frequent association of abscess formation, routine work in the anginosus bacteria group should include finding an abscess. The vitro susceptibility of VGS is supposed to be evenly susceptible to penicillin. However, Pfaller et al reported that 9.2% of VGS isolates from the scope Hospital Study Group were resistant to penicillin (53). In 1995, the American Heart Association guidelines for the treatment of endocarditis divided VGS into penicillin are susceptible, intermediate resistance, and high levels of resistance. Penicillin has been recommended for penicillin-susceptible VGS (94). An increase in the proportion of penicillin resistance has been reported even in serious infections, and this resistance is thought to be due to changes in penicillin protein binding (2, 29, 31, 57.59, 93). Alcaide, et al from Barcelona, Spain found that 33.6% of the 410 isolates were resistant to penicillin (41.5% *S. mitis*, 41.7% *S. sanguis*, 28.1% *S. salivarius*, and 14% *S. anginosus*) (2). They divided beta-lactam agents tested against penicillin-resistant strains into three groups. In the first group of imipenem, ceftriaxone, cefotaxim showed similar or higher activity of penicillin. Antibiotics in the second group showed lower activity than penicillin: ampicillin, amoxicillin/clavulanate, piperacillin, cefuroxima and cephopotoxim. The third group showed poor activity in the test tube: it includes the first generation of cephalosporins, ceftazidime, cefixime, cefaclor, cefbuten, and oxacillin. Traub et al from Germany reported their collection of 116 VGS isolates from patients and 162 isolates from healthy adults (85): all isolates were susceptible to vancomycin and teicoplanine; none of them had high-level resistance to gentamicin; they were all resistant to fusidic acid. Susceptibility to non-beta-lactam antibiotics did not correspond, for example, to ciprofloxacin - 59.7%; 89.2%; doxycycline, 65.8%; tetracycline, 56.8%; Clindamycin 87.8%; erythromycin, 59%; claritromycin, 74.9%; and SXT, rifampin, and chloramphenicol were more than 97%. All 12 *S. mitis* isolates were from patients and were resistant to penicillin. VGS susceptibility to penicillin G was 79.1%; ampicillin, 66.9%; piperacillin, 98.2%; cefoxitin, 76.6%; cefvoksim 96.8%; Cefotaxime, 98.6%; Ceftriaxone, 98.6%; Cephepin, 98.6%; imipenem 98,2%. Potgieter, et al from southern Africa studied 211 isolates from blood cultures showed that they were all consistently susceptible to cefotaxima, ceftriaxone and imipenem (57). The above reports indicate that imipenem, cefotaxim and ceftriaxone are acceptable alternatives for serious infections caused by strains, to the penicillin, but but Needed. In the case of intermediate resistance (MIC 0.25-2 microgram/ml penicillin) or high-level resistance (MIC 4 microgram/ml or more) synergies with aminoglycoside can still be achieved (15). Synergies should be considered after in vitro synergies have been confirmed. A high percentage of resistance to SXT, erythromycin and tetracycline was recorded (22.57). Unlike its group D colleague, enterocococca, *S. bovis* is very susceptible to penicillin. Doern et al, reported their experience with 352 VGS from 43 U.S. medical centers (Figure 1) (22). They found that 13.4% had a high resistance level (MIC zgt:4 micrograms/ml), 42.9% had intermediate resistance (MIC 0.25-2.0 micrograms/ml). Among the cephalosporins tested, ceftriaxone is the most active. When using a break point of 8 micrograms/ml for locsacin, less than 1% was observed, and less than 5% resistance was observed when using the break point of 2 micrograms/ml. In another U.S. study, blood cultures from 47 neutropenic patients who were on ciprofloxacin prevention were given VGS (most of them were *S. mitis* and *S. sanguis*). Penicillin resistance was found in 38%, ceftazidime resistance in 54%, and ciprofloxacin and ofloxacin resistance in 95% (46). Because of this there is increased resistance to quinolones, the use of quinolone especially those with a bad gram of positive coping activity for prevention in patients with neutropenic cancer should be carefully evaluated. Kennedy et al reported VGS isolation with an increase in MIC beta-lactams from the blood in 61 pediatric patients with malignancies, despite previous courses of empirical antibiotic therapy, which was either ceftazidime plus ampicin or piperacillin /tazobactam plus ampicin (40). Poor susceptibility to macrolide antibiotics has been demonstrated in 66 blood culture isolates from neutropenic cancer patients (1). Resistance of VGS to beta-lactams, SXT, clindamycin and macrolidam continues to grow even among non-iroptic patients (1, 3, 20, 41, 52, 53, 76, 86, 96). In a study conducted in the United Kingdom, resistance to beta-lactam and macrolides was found mainly in *S. mitis*, while higher rates of ciprofloxacin resistance were found in isolates identified as *S. bovis*, *S. mitis* and *S. mutans* (41). The increasing resistance of combos to VGS suggests that the use of beta-lactams and macrolides as preventive means for dental procedures, as well as empirical or preventive use in granulocytopeni patients should be reviewed (20, 53, 60, 95). Similarly, the choice of fluoroquinolones for prevention and treatment should be individualized. Resistance to penicillin in the Anginosus group is rare (34, 76, 85). However, et al reported a total resistance of 29% penicillin in VGS including Anginosus Group (57). VGS is still susceptible to vancomycin and some cephalosporins, but erythromycin, tetracycline, sulfamethoxazole and aminoglycoside (30, 31, 33, 55, 96) were reported. Synergy can usually be demonstrated with penicillin and aminoglycosides. Antimicrobial endocarditis Therapy Since the introduction of penicillin, the level of treatment of VGS exceeded 95%. Causes of failure include bacterial tolerance (when MBC is 32 times higher than MIC), inadequate levels of antimicrobials in vegetation (most likely due to insufficient dosing or poor drug penetration), and heart failure. In addition to maintaining the pump function, the main goal is to eradicate VGS infection. The AHA Guidelines (Table 5) for the treatment of native penicillin valve infection are susceptible to VGS (MIC zlt:0.1 zg/ml) including *s. bovis* consists of four-week therapy with a q a' single beta-lactam' agent or two-week therapy with a q combination of a q beta-lactam' agent' plus' aminoglycoside.francioli' et al' have recently reported a successful treatment of vgs' endocarditis' using q a' 2-week course of ceftriaxone 2g' plus q netilmicin' at q dose' of 4 mg/kg (27). although the guidelines are recommended with a four-week single-beta-lactam agent' for the elderly, we'd prefer to treat' with the combination of therapy and except in the patient with the ' impaired' renal' function or in the presence of the high level of amino-aminoglycoside resistance (mic/500 mcg/m). Gavalda et al. reported that in an experimental study of endocarditis, once a day intramuscular dosing of gentamicin is effective as multiple dosing as long as the total daily dose is 3 mg/kg (28). The overall rate of bacteriological

failures is extremely low (78). In patients who have been ill for more than 3 months before therapy, the relapse rate is higher and a longer period of therapy is recommended (54). Successfully oral penicillin plus intramuscular amine glycoside penicillin therapy for vGS has been reported (82). This form of therapy should only be tested in conditions where blood levels can be monitored and patient compliance can be guaranteed. For patients who are allergic to beta-lactam agents, vancomycin can be used. In young patients, the vancomycin gap may be faster, so it may be wise to know the vancomycin half-time period and adjust the pre-season interval. For patients with valve endocarditis due to strains with MIC between 0.1 and 0.5 micrograms/ml, AHA (table 5) recommends combining 4 weeks of penicillin treatment plus 2 weeks of amikoglycoside therapy. If a high level of resistance to aminoglycoside is present or synergies with aminoglycoside cannot be demonstrated, vancomycin should be used. Based on the Alcáide et al study, another option is to test susophtoxatima, ceftriaxone and imipenem. If MIC low, any of these agents can be considered. If MIC is high, vancomycin should be used. However, the official recommendation for the treatment of these high-quality sustainable organisms. Experimental studies using vancomycin plus gentamicin have shown that this combination is effective against strains resistant to penicillin (45). Patients infected with Anginosus streptococcus are at a higher risk of complications, so a higher dose of penicillin is recommended. Table 6 shows the 1997 Recommendation of the American Heart Association for antimicrobial regimens for patients undergoing dental, oral, respiratory or esophageal procedures to prevent infectious endocarditis (17). Non-heart infections are clinically, infections caused by the Anginosus group have responded well to penicillin and cephalosporins. With penicillin resistance on the rise, it is prudent to treat a serious infection with a combination of penicillin and aminoglycoside. Vancomycin or clindamycin can be used by penicillin allergic patients. Since an Anginosus group is often associated with an abscess, efforts should be made to rule out this possibility. When an abscess is present, drainage should be heavily considered. Failures in treatment were observed in patients with polymicrobial abscess and who were treated with metronidazolean anti-tionobob drugs. Due to the lack of anti-streptococcal activity in the aforementioned combination, the Anginosus group of streptococcus was isolated as the sole microbe in the liver abscess (55). Particular attention should be paid to neutropenic patients with VGS bacteria. Although it is not as common as a gram of negative and staphylococcal bacteriology, this problem is growing due to the regular use of antibiotic prophylaxis with fluoroquinolones, damage to the oral mucosa caused by chemotherapy, and the presence of neutropenia (6, 14, 61). Oral cavity is the most likely entry portal especially in those with damage to the mouth mucosa. Complications from VGS bacteremia include pulmonary infiltration, adult respiratory distress syndrome, hypotension and endocarditis (6). The related problem is septicæmia and shock syndrome due to VGS, which is associated with an 11% (24) mortality rate. Alternatives to high doses of penicillin include vancomycin. If another beta-lactam drug is used, imipenem, ceftriaxone, and cefotaxim have better activity than most other cephalosporins, including ceftazidime (2,57,88). Meningitis VGS rarely infects meningitis. In a report by the hospital for 1,000 beds in Barcelona, Cabellos et al reported 29 cases of streptococcal meningitis between 1977 and 1997 (12). Twenty out of 20 cases were VGS and an increase in MIC penicillin was observed. The experience of treating meningitis because of these organisms is limited. The antibiotic of choice should be the one that has the best penetration into the cerebrospinal fluid. Ceftriaxone, cefotaxim or high dose of penicillin should be Vancomycin can be used for penicillin allergic patients. Patients, fluid levels should be monitored to ensure an adequate level. Mixed Infections When VGS infection is suspected as part of a common mixed infection, the therapy should be directed against mixed infection. The doctor should make sure that the antimicrobial that is effective against VGS is part of the therapy. NUTRITIONALLY VARIANT STREPTOCOCCI (Abiotrophy defectives and A. adjacens) Nutritional variant of streptococcus is no longer classified by VGS (73). These streptococcus-looking organisms have previously been grouped with VGS due to some similarity to S. mitis, but the recent classification has moved them from a kind of streptococcus to abiotrophy (73). Unlike VGS, NVS require a pyridoxal or tiol supplement for growth. This group includes two types: A. defectivus and A. adjacens. Like VGS, these bacteria are commonly found in orofarinx, and have been linked to bacterioma, endocarditis, and eye infections including conjunctivitis, keratitis, endophthalmitis, and infectious crystalline keratopathy (71). The In vitro in vitro susceptibility test for this group is difficult to perform, and the results do not correlate well with the clinical outcome. In addition, infections due to these organisms are known to respond poorly to antibiotics (80). The nutritionally-nutritious version of streptococcus is generally less susceptible to penicillin than most other streptococcus, but many strains are tolerant (36.71). Most strains are reported to be susceptible to rifampina, clindamycin, erythromycin, chloramphenicol, and vancomycin (71). Susceptibility to tetracyclines, aminoglycosides and cephalosporins is a variable (36). The timing of the slain curves studies have shown that vancomycin and rifampin are synergistic (71). Antimicrobial therapy There are two groups of streptococcus that are harder to treat, tolerant organisms and nutritionally variant organisms. NVS organisms are also tolerant. Tolerance is defined as having MBC 32 times higher than MIC. In other words, bacterial growth can be inhibited but not killed until the concentration of antibiotics is increased by 32 times or more. Stein and others examined 30 cases of NVS endocarditis and found that relapse rates, bacteriological failure and mortality were higher than that of the viridan group. They believe that slow growth and the production of glycoacalix may have contributed to the lack of success (80). In addition, there is tolerance in penicillin and vancomycin (71). The current recommendation of the AHA is to treat the nutritionally-related version of streptococcus endocarditis similar to enterococcal infection (see table 5). However, even with 6 weeks of combined penicillin and gentamicin therapy, the failure rate is high (21). Experimental models of endocarditis have shown that vancomycin or vancomycin-gentamicin can be used as an alternative drug in patients when penicillin-aminoglycoside combination is ineffective or contraindicated (7). Clinical experience is still lacking. CAVEATS AND COMMENTS Recent changes in the susceptibility of streptococcus have changed the attitude of clinicians to this group of bacteria. Some penicillins and cephalosporins, which were previously considered exquisitely active, are no longer consistently effective. Beta-lactam antibiotics are either alone or combined for most patients with endocarditis infected with VGS, S. bovis, but alternative regimens are needed for special situations. Groups C and G streptococcus respond best to a combination of penicillin and aminoglycoside (26). The doctor should be aware of these new developments and be prepared to use MIC antimicrobials and a synergistic test for serious infections caused by streptococcus. LINKS 1. Alcáide F, Karratala J, Linares J, Gudíol F, Martín R. In vitro activities eight macrolide antibiotics and RP-59500 (Kvinupristin-Dalfopristin) against the viridance group of streptococcus isolated from the blood of patients with neutropen cancer. Antimrob Agents Chemother 1996; 40:2117-2120. (PubMed) 2. Alcáide F, Linares J, Pallares R, et al. In vitro activity 22 beta-lactam antibiotics against penicillin resistance and penicillin susceptible viridance group streptococcus are isolated from the blood. 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Equi, S. equisimilis, S. zoepidemicus) oropharyngeal flora, vaginal rectum skin S. canis is a zoonotic agent of pharyngitis skin infection bacteremia endocarditis osteomyelitis septic arthritis arthritis respiratory infection puerperal infection of neonatal sepsis toxic shock-like syndrome and genital endocarditis infections in neutropenic patients Mitis group S. mitis S. gordonii S. oralis S. sanguis S. parasanguis (S. pneumoniae) oropharinx and gastrointestinal tract Bacteremia, endocarditis, meningitis ARDS Anginosus or S. milleri group S. anginosus S. constellatus S. intermedius oropharynx, gastrointestinal tract, and vaginal flora, skin pios disease, brain, liver and appendic abscesses associated with polymicrobial infection endocarditis salivarius group S. salivarius S. thermophilus S. vestibularis language and gastrointestinal tract Frequent contaminants and rarely cause infection , such as bacteremia, meningitis Bovis group S. bovis S. equinus S. alactolyticus oropharynx, gastrointestinal tract, genital tract bacteria and endocarditis in patients with gastrointestinal malignancies, especially with the biotype I meningitis Mutans group s. mutans S. rattus S. cricetus s. downei S. macacae dental plaques and dental surfaces dental tooth decay endocarditis Nutritional variant of streptococcus renamed Abiotrophy A. adjacens A. defectivus oropharynx endocarditis, pancreatic sepsis abscess from media eye infections (conjunctivitis) Streptococcus Group C Streptococcus from various reports Antibiotic No. strains MIC90 mg/ml MIC Range Reference Penicillin G 17 0.15 0.04-0.15 56 125 0.05 0.01-0.06 0.05 37 125 0.05 <0.006-0.1 37 Ampicillin 125 0.1 <0.006-0.2 37 Cephalothin 125 0.2 0.05-0.39 37 Cephalexin 125 3.13 0.39-6.25 37 Cefaclor 125 1.56 0.39-6.25 37 Meropenem 48 0.06 <0.016-0.12 98 Erythromycin 125 0.1 0.0125-0.39 37 Ciprofloxacin 8 1 0.5-1.5 Clnafloxacin 8 0.5 0.13-0.5 5 Gatifloxacin 8 0.25 0.13-0.25 5 Levofloxacin 8 1 0.5-1.5 Moxifloxacin 8 0.13 0.06-0.13 5 Trovafloxacin 8 0.25 0.13-0.25 5 Vancomycin 48 0.5 0.06-1.0 98 Linezolid 48 2.0 0.5-2.0 98 Quinupristin-dalfopristin 48 .25 0.06-0.25 98 Table 3. Восприимчивость группы G Streptococci из различных доклов. Антибиотик MIC90 мг/мл MIC Диапазон Справочный антибиотик 0.017 .0025-.04 43 0.05 <0.0063-0.1 37 Ампициллин 0.05 <0.0063-0.2 37 Ампициллин 0.1 <0.0063-0.2 37 Цефалоспорины 0.12 0.06-0.12 64 Пиперациллин 0.06 0.03-1.0 19 Цефалотин 0.2 0.025-0.2 37 0.09 0.04-0.156 43 Цефотаксиме 0.027 0. 005-0.04 43 0.022 0.01-0.04 43 Ceftazidime 0.5 0.03-32.0 19 Cefoxitin 0.27 0.156-0.312 43 Cephalexin 3.13 0.1-6.25 37 Cefaclor 3.13 0.1-6.25 37 Cefpodoxime 0.12 not reported 68 Meropenem 0.06 <0.016-0.06 98 Vancomycin 1.13 0.312-2.5 44 0.64 0.312-1.25 43 .25 .25-0.5 64 2.0 0.25-4.0 69 0.5 0.12-0.5 98 Teicoplanin .06 <0.03-0.5 66 0.25 0.25-0.5 69 Linezolid 2.0 0.12-2.0 98 Quinupristin-dalfopristin 0.25 0.125-0.25 98 Erythromycin 0.06 <0.03-0.12 16 1.94 0.037-2.5 9 Clarithromycin .06 <0.03-0.12 16 Clindamycin 0.5 <0.03-0.5 16 1.1 0.06-2 9 Trimethoprim/sulfamethoxazole 0.12 0.25 15 Chloramphenicol 5.5 0.3-10 9 Ciprofloxacin 1.0 0.5-2.0 18 2.0 0.25-2.0 15 0.5 0.25-0.50 15 1.0 0.25-1 22 1.0 0.15-2.0 17 1.0 0.5-2.0 15 Levofloxacin 0.5 0.25-1.0 15 1.0 0.25-1 17 Sparfloxacin 1.0 0.25-1.0 15 0.5 0.12-2.0 17 Clnafloxacin 0.06 <0.03-0.12 15 0.25 0.06-0.25 22 Gatifloxacin 0.25 0.13-0.25 22 0.25 0.12-0.5 17 Moxifloxacin 0.13 0.06-0.13 22 Trovafloxacin 0.13 0.06-0.25 22 *Isolates from cancer patients Table 4. Восприимчивость группы С и группы G Стрептококки (49,62,84) Антибиотик No. of Strains MIC90 mg/mL MIC Range Reference Penicillin G 44 0.03 0.03-0.06 62 Cephalothin 44 0.06 0.03-0.5 62 Piperacillin 44 0.03 0.03-0.5 62 Azlocillin 44 0.06 0.03-0.25 62 Vancomycin 44 0.12 0.03-0.5 62 Erythromycin 44 1.0 0.03-1.0 62 20 0.5 0.12-1.0 49 Clarithromycin 20 0.25 0.06-1.0 49 Azithromycin 20 0.5 0.12-1.0 49 Quinupristin/dalfopristin 20 0.5 0.12-1.0 49 Gatifloxacin 10 0.125 0.125 84 Clnafloxacin 10 0.25 0.125-0.25 84 Ofloxacin 10 2 0.5-2.0 84 Levofloxacin 10 1 0.5-1.0 84 Ciprofloxacin 10 0.5 0.5 84 Sparfloxacin 10 2 0.125-2 84 Trovafloxacin 10 0.25 0.125-0.25 84 Table 5. Рекомендуемая терапия для VGS инфекций эндокардита из-за пенициллина восприимчивы viridans стрептококки и стрептококк bovis (минимальная концентрация ингибитора <0,1 мг/мл). Родной клапан Use any of the following: penicillin G 12-18 million units per day in a continuous drip or 6 divided dose plus gentamicin 3 mg/kg IV as one dose or 3 separated doses for 2 weeks. Penicillin G 12-18 million units per day in a continuous drip or 6 divided dose for 4 weeks. Ceftriaxone 2 g IV or CHAT daily for 4 weeks. Vancomycin 30 mg/kg does not exceed 2 g IV in 2 divided doses for 4 weeks. Infection of the prosthetic valve. Penicillin or vancomycin is like 2 and 3 for 6 weeks plus gentamicin at the same dose as higher for at least 2 weeks. Эндокардит из-за viridans стрептококков и стрептококков ковансон относительно устойчив к пенициллину G (минимальная концентрация ингибиторов >0.1 <0.5 мг l/l)*= 18= миллион= u/24= h= iv= either= continuously= or= in= six= equally= divided= doses= for= 4= weeks= plus= gentamicin= 3= mg/kg= iv= as= single= dose= or= 3= divided= doses= for= 2= weeks= vancomycin= 30= mg/kg= not= to= exceed= 2= g= iv= in= 2= divided= doses= for= 4= weeks= endocarditis= due= to= viridans= streptococci= with= (mic=>0,1мг/мл и 0,5 мг/мл) или питательно вариант стрептококков аквеозного кристаллического пенициллина G натрия, 18-30 миллион/ U/24 h IV либо непрерывно, либо в шести одинаково разделенных дозах или, ампициллин натрия 12 i/24 ч IV либо непрерывно, либо в шести разделенных дозах плюс гентамицин сульфат 1 мг/кг IM или IV каждые 8 ч в течение 4-6 недель» гидрохлорид Ванкомицина 30 мг/кг на 24 ч IV в двух равных дозах divided doses, exceed 2g/24 h if serum levels are not controlled plus gentamicin sulfate (similar dose, as stated above) for 4-6 weeks for patients with endocarditis of the prosthetic valve due to streptococcus Treat as a resistant streptococcus (MIC >g.5 mg/ml) for 6-8 weeks For patients with bacteriomy without endocarditis due to Viridians of the Streptococcus group and NVS. Penicillin G 12-18 million units IV continuously or in 6 divided doses for 2 weeks. Ceftriaxone 2 g IV or IM daily for 2 weeks Clindamycin 300 mg IV or PO q8h for a few weeks vancomycin 30 mg/kg to not exceed 2 g IV in 2 separated doses for 2 weeks. For patients with meningitis due to viridance group of streptococcus or NVS Ceftriaxone 2 g IV or IM daily or cefotaxim 2 g IV q6h for 2 weeks penicillin 18-30 million units IV in 6 separated doses within 2 weeks Vancomycin 30 mg/3 0 mg/kg does not exceed 2 g IV in 2 divided doses for 2 weeks For patients with mixed infection, where the viridance group of streptococcus or NVS is a beta-lactam/lactamase inhibitor combination in the recommended dose of Imipenem 500-750 mg every 6-8 hours IV. Above agents or clindamycin plus gentamicin. 4-week therapy is recommended for patients with 3 months in duration; 6-week therapy recommended patients with symptoms greater than 3 months in duration plus **Vancomycin therapy is recommended for patients allergic to beta-lactams; cephalosporins is not acceptable unless shown to be effective by susceptibility testing ***Clindamycin susceptibility should be checked. months= in= duration; 6-week= therapy= recommended= for= patients= with= symptoms= greater= than= 3= months= in= duration= plus= **vancomycin= therapy= is= recommended= for= patients= allergic= to= beta-lactams=; cephalosporins= is= not= acceptable= unless= shown= to= be= effective= by= susceptibility= testing= ***clindamycin= susceptibility= should= be= checked.=><3/3 months in duration; 6-week therapy recommended for patients with symptoms greater than 3 months in duration plus **Vancomycin therapy is recommended for patients allergic to beta-lactams; cephalosporins is not acceptable unless shown to be effective by susceptibility testing ***Clindamycin susceptibility should be checked. >г; симптомами<0,5 mg>г; mg>г; 6. Preventive schemes for dental, oral, respiratory or esophagus procedures (From the recommendations of the American Heart Association, 1997) (17) Situation agent Regimen Standard general prevention of Amoxicillin Adults: 2.0 g; Children 50 mg/kg orally 1h prior to the procedure Unable to take oral drugs Ampicillin Adults: 2.0 g IM or IV; Children 50 mg/kg IM or IV for 30 minutes prior to the procedure Allergy to penicillin Clindamycin