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AN OVERVIEW ON THE ANTIBIOTICS USED IN THE TREATMENT OF GONORRHEA

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ABSTRACT
Gonorrhea is the second most commonly reported disease in the United States: 350,062 cases of gonorrhea were reported in 2014. Sexually transmitted diseases caused by Weisseria gonorrhoeae are the cause of pelvic inflammatory disease in women, which can lead to serious reproductive problems including tubal birth defects. ectopic pregnancy, and chronic pelvic pain. Inhibition of sequelae and transmission of sexual partners is highly dependent on immediate detection and effective antimicrobial treatment. However, treatment has been compromised due to the lack of regular testing of antimicrobial susceptibility in clinical care and the emergence of antimicrobial resistance used to treat gonorrhea.

KEYWORDS: Sulfonamide, Cephalosporin, Tetracyclines, Spectinomycine Microlides.

INTRODUCTION
Sexually Transmitted Infections (STI) gonorrhea remains a major public health problem worldwide. This requires urgent international attention and resources because the global burden of infection is growing and Neisseria gonorrhoeae (gonococcus), the etiological agent of gonorrhea, is transforming into a superbug and may be incurable due to its resistance to all antimicrobial levels available for treatment of diseases. Gonorrhea has been successfully treated with antimicrobials for the past 70 to 80 years.[1] However, in the international arena, there is now a widespread spread of N species. gonorrhoeae that are resistant to many antibiotics previously and currently widely available for treatment (e.g., sulfonamides, penicillins, previous cephalosporins, tetracyclines, macrolides, and fluoroquinolones). The recent occurrence of failure to treat diarrhea with extended-spectrum cephalosporins (ESCs) cefixime and ceftriaxone and the emergence of gonococcal strains showing high clinical resistance to all ESCs combined with resistance to almost all other antimicrobial therapies available, has resulted in great concern. , as evidenced by the publication of medical and media books and the development of global, regional, and national action / response programs. Because of this fact, there are fears that gonorrhea may be incurable with antimicrobial monotherapy.[2] In response to these concerns, recommendations for dual-use antimicrobial therapy, namely, in particular ceftriaxone and azithromycin, have been introduced in the United States, the United Kingdom, and throughout Europe. Unfortunately, the risk of gonococcal isolate in ceftriaxone has declined worldwide, and azithromycin resistance is easily selected and is already widespread in many settings. Ideally, these two types of antimicrobials may not be long-term solutions and, moreover, are not affordable in many areas that lack resources. In addition, more expensive antimicrobials, such as high-quality ceftriaxone, are not even available in monotherapy low-dose settings.[3]

The forthcoming public health problem of antimicrobial gonococci cannot be underestimated, as treatments will be more expensive, and as treatment failure occurs, treatment costs will increase significantly due to serious complications that endanger the normal and reproductive health of infected people individuals considering the global nature and burden of gonorrhea, high levels and uncontrolled antimicrobial use, low control and antimicrobial resistance (AMR) and treatment failure, gradual revision of treatment guidelines in many local areas, and abnormal ability to gonococci develop and keeping AMR, the global gonococcal AMR problem is likely to worsen in the foreseeable future, and serious complications of gonorrhea will emerge as a silent epidemic.[4]

By understanding the evolution, emergence, and spread of AMR in N. gonorrhoeae, which includes its cellular

and phenotypic mechanisms, unexpectedly antimicrobial resistance to clinical use, future methods of genetic testing for AMR may allow for specific regional-specific therapies and antimicrobial design., and the design of an antimicrobial novel to avoid resistance can be done in a logical way. This review focuses on the history and evolution of antiretroviral therapy and the emerging resistance, genetic and phenotypic pathways for gonococcal antimicrobial pre-recommended and still recommended antibodies, including costs or biological benefits, as well as important actions and advances needed for diagnosis and treatment. Resistance to gonococcal strains and, ultimately, maintain gonorrhea as a treatable disease.[4]

Data Report on Gonorrihoea: — Rates of Reported Cases by Year, United States, 1941–2019.

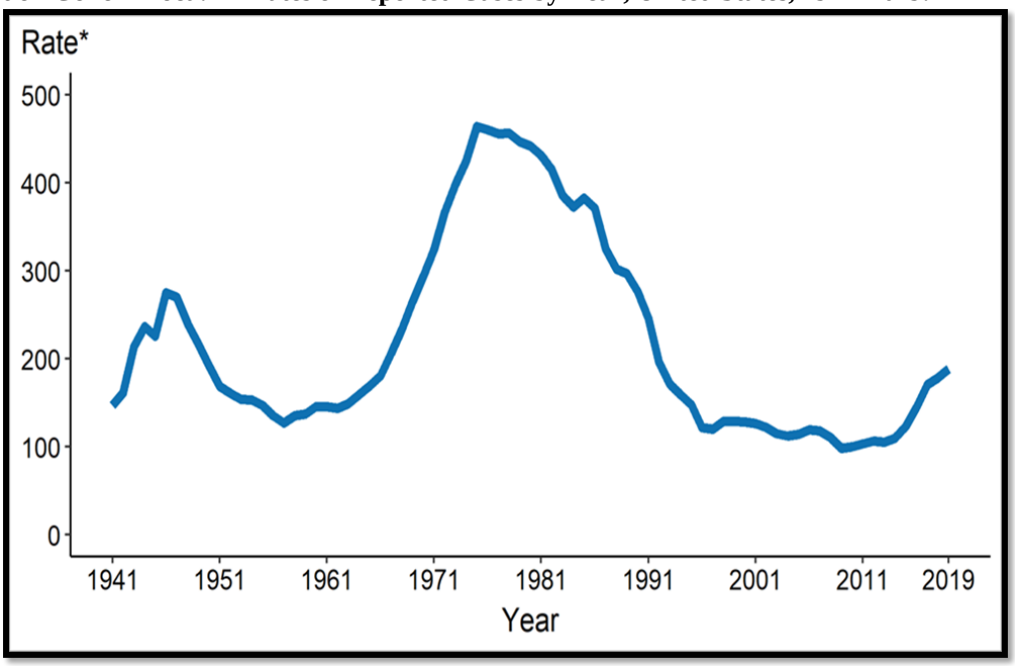

Figure 1: Diagnosis of Gonorrhea And Detection Of Antimicrobial Resistance In Neisseria Gonorrhoeae.

Diagnosis of Gonorrhea

The diagnostic guidelines for gonorrhea are described earlier. As mentioned above, gonorrhea usually has no symptoms, and when symptoms do occur, they are usually undiagnosed. Therefore, a proper laboratory diagnosis is essential for a certified diagnosis, trial, and medical examination. Diagnosis of gonorrhea is established with the discovery of N. gonorrhoeae or its genes in private or external models by microscopy of color smears, culture, or nucleic acid amplification tests (NAATs). Only proven and validated methods should be used. Ideally, AMR testing of gonococcal isolate should be an important part of laboratory diagnosis.[5]

Gonococci can be identified as intracellular diplococci in polymorphonuclear leukocyte by microscopy (magnification, × 1,000) Gram- or methylene blue-stained smears. This method is cheaper, provides faster results, and has higher sensitivity and diagnostic clarity in men with symptoms of urethral discharge. However, microscopy is not recommended as the only way to diagnose cervical, pharyngeal, or rectal gonorrhea, or asymptomatic patients, because side effects do not involve infection, due to low sensitivity. In addition, operating features are highly dependent on microscopist information. Importantly, this method does not provide any AMR data.[5]

The culture, the old "gold standard," offers high sensitivity and clarity of up to 100% (if proper animal-specific ratings are used) and is the only established method that allows complete AMR testing. However, the process is slow, and in order to achieve high sensitivity and precision, it is important to firmly establish the conditions for sample collection, transport, storage and cultural approach, as gonococci are highly sensitive to external factors.[5]

In settings with additional resources, NAATs quickly change the culture to detect gonococci. NAATs have many benefits, e.g., they get lifeless gonococci; be more sensitive than all other diagnostic methods, especially the pharyngeal and rectal specimens; and they do not want much about template collection (rare, self-assembled samples, such as urine [men] and soaps in the vagina [women], can be used effectively), transport, and storage. NAATs are also faster, allow for self-efficacy, and enable the discovery of several viruses simultaneously. However, NAATs are also negative, e.g., they do not allow AMR testing, the appropriate timing of clinical trials is still being discussed and commercial and internal NAATs show different sensitivity and specific specifications in their acquisition of N. gonorrhoeae. Types of Commensal Neisseria, which are common in

the pharynx and rectum but also, rarely, in the urogenital tract, have genetic homology and N. gonorrhoeae and may also respond to gonococcal NAATs, leading to false reports. The limited specificity of gonococcal NAATs results in lower predictor levels (PPV), especially in people with low birth weight. (with different target sequences) can be used to validate all NAAT-screening-positive samples. Importantly, in the arrangements used only by NAATs for the detection of gonococci, it is important that laboratories, epidemiologists, and physicians engage with and be aware of adequate local, national, and / or international testing for gonococcal antimicrobial surveillance (GASP). It is important to note that immunofluorescence tests, enzyme immunoassays tests, and point-of-care tests are detected to provide adequate antigen or antibody and, in particular, sensitivity sensitivity is not available for clinical diagnostic purposes.[6]

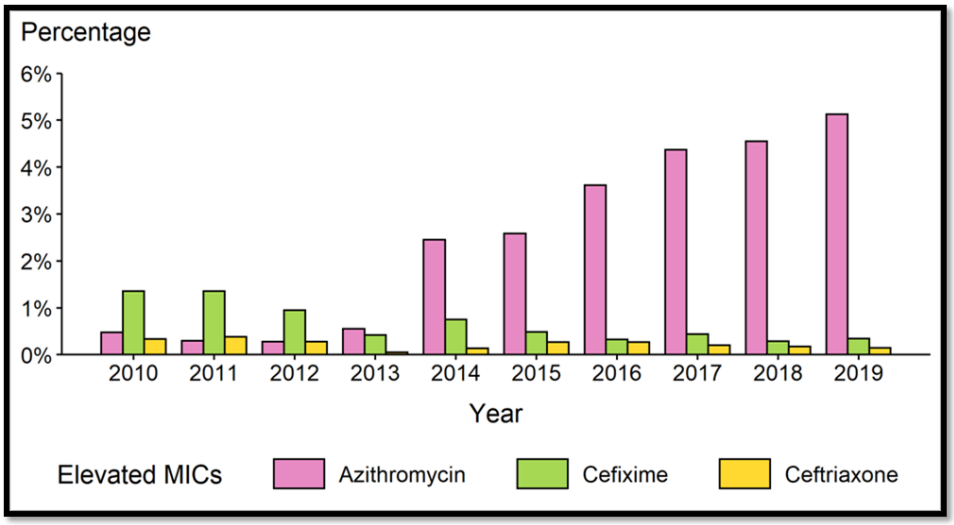

Figure-2:

Detection of Antimicrobial Resistance in N. gonorrhoeae

The agar dilution method determines antimicrobial MICs (µg / ml) and is a "gold standard" method. However, especially for testing small numbers of isolated areas, this method is effective and not suitable for routine AMR testing. Therefore, the quantitative Etest method of MIC detection, which is comparable to the agar purification method, is commonly used. In addition, AMR quality determination can be obtained by disc distribution of the disc. However, in order to adequately reproduce and interpret to accurately reflect the MIC values of a given antibiotic, these methods require extensive quality assurance and appropriate quality controls. Disk distribution methods are recommended to be used only if MIC determination does not occur, due to limited resources or other reasons, and any new, emerging, or abnormal AMR should be confirmed by MIC determination.[7]

In AMR testing, culture and phenotypic AMR testing remain important. However, in most low-dose settings, the diagnosis of gonorrhea depends on the syndromic treatment of patients, and no templates are taken. In many high-resource settings, NAATs have quickly replaced traditional ones in order to acquire N. gonorrhoeae. For an improved AMR focus, it is important to strengthen cultural capacity around the world. However, it is also necessary to improve the rapid genetic testing of AMR. These methods should be used appropriately in the care setting as well as for rapid, critical, and specialized genetic testing for gonococcal diagnosis. These methods can provide direct diagnostics and direct individualized treatments, ensure the effective use of antimicrobials and affect your control of both gut and AMR. Statistical modeling may assess the impact of NAAT-based AMR tests on the spread of resistance and clinical outcomes. Powerful transmission models can capture the effects of competing factors, such as the detection and further treatment of gonorrhea, an increased risk of re-infection, and a decrease or delay in the detection of AMR in gonorrhea and AMR-resistant strains. Unfortunately, no gonococcal NAATs are available. any decisions of AMR. However, testing of existing cells for the detection of one or more of the AMR-resistant genes involved in plasmid penicillin resistance, chromosomal mediated penicillin resistance, plasmid- and chromosomal mediated tetracycline, resistance and macrolide, fluoroquinolone resistance, ESC resistance, and multidrug resistance. Unfortunately, in many AMR decisions, the sensitivity and specificity of these AMR cell tests to determine AMR is often low. For example, in currently recommended antimicrobials, ESCs, correlates between highly defined resistance decisions, ESC MICs of gonococcal species, and very low therapeutic effect. Genetics is a major challenge in the development of AMR ESC genetic testing. Reviews that require constant review and new target sequences are less likely to be profitable for NAAT-producing companies in the short term. Other "specialized" cell tests detecting mutations involved in ESC resistance to

defined gonococcal XDR strains have also been performed recently.[8]

Table 1: List of Drug Use In Gonorrhea.

DRUG	APPROVED BY	DOSAGE FORM	DRUG DURATION	RISK FACTORS
Cifixime	DGCI	TABLET,CAPSUL	12 HOUR	NO RISK
Sulfonamides	FDA	TABLET	8 HOUR	INCREASE RISK OF CONGENITAL MALFORMATION
Penicillin	FDA	CAPSULE, TABLET	6 HOUR	SKIN RASH, SWELLING
Tetracycline	FDA	CAPSULE	6 HOUR	NOT USE IN PREGNANCY, DECREASE KIDNEY FUNCTION.
Spectinomycin	FDA	INJECTION	12 HOUR	LIVER AND KIDNEY DISEASE.
Quinolones	FDA	TABLET, CAPSULE, INJECTION	4 HOUR	LUNGS DISEASE
Macrolides	FDA	INJECTION, TABLET, CAPSULE	12 HOUR	KIDNEY DAMAGE, LIVER INFLAMMATION.
Cephalosporin's	FDA	TABLET	6-8 HOUR	LIVER INFLAMMATION

Treatment Regimens of Antibiotics

The Preantimicrobial Era

During the preantimicrobial period, treatment of gonorrhea was a healthy lifestyle, fresh air, proper nutrition, and rest, avoidance of alcohol and sex, and access to standardized treatment for various forms of balsamic, urethral irrigation, chemical compounds, hyperthermia.During the second half of the 19th century, gonorrhea was commonly treated with the type of Indonesian pepper (cube) and balsam extracted from a South American tree. magnesium hydroxide, or ammonium carbonate or incorporated into gelatin tablets. Antiphlogistic drugs, to avoid irritation, to keep the body cool (using salt), and to cleanse the urine can also be used until the symptoms of inflammation subside, and then cubebs or copaiba balsam three times a day. Soap and water enema, oral laudanum (opium tincture), and warm baths were used to reduce urine retention, and if necessary, catheterization was performed. Acute urethritis can also be treated by internal irrigation with warm potanganum permanganate diluted for a few weeks.[8]

In the late 1800s, searches for specific antimicrobial compounds were initiated, and numerous metallic compounds were investigated, e.g. During World War I, soldiers were given prophylactic packages that included condoms, calomel oil (mercuryl chloride), and Argyrol / Protargol (silver compounds), as well as postcoital treatment centers, including urethral irrigation centers, too. were used. Mercury compounds were later commonly used; for example, Mercurochrome-220 was used as an antiseptic for urinary tract. By mixing 1% mercurochrome in a 50% glucose solution, the injection becomes safer and more effective. Then, in addition to mercurochrome veins, a silver-protein complex or mercurochrome was inserted into the urethra, or seminal vesicles infused with potassium permanganate.[8]

Diathermy or hyperthermia was also used in the beginning of several settings. Initially, only swollen joints in patients with gonococcal arthritis were burned. However, when some arthritis reactions only responded to the addition of genital hyperthermia, the genitals also began treatment. A cold cabinet, with only the head outside, was used, and temperatures above 41 ° C were kept for 4 to 6 hours. Treatment, usually 5 or 6 treatments, given every third day, was needed. Up to 80 to 90% of cases of gonococcal arthritis can be cured. Pre-treatment with mercurochrome to hypertonic glucose was later shown to increase the effectiveness of hyperthermia. Finally, a few treatments for hyperthermia were usually needed with pelvic floor heating, i.e., implantation of approximately 2 hours of warmth in the rectum in men and the vagina in women (sometimes with the rectum), resulting in up to 44 degrees Celsius. ° C. Hyperthermia was considered the best treatment for gonococcal arthritis and, in general, also relieves any genital symptoms.[8]

The Antimicrobial Era

History of antimicrobials identified and presented with the emergence of resistance, including genetic resistance mutations, as well as changes in the recommended first-line antimicrobials.

Sulfonamides

In 1935, Gerhard Domagk discovered sulfanilamide. Sulfonamides were the first antimicrobials used to treat diarrhea; Sulfanilamide initially cured 80 to 90% of gonorrhea diseases.Sulfapyridine was first discovered in 1940 to 1941, and a 1-week course of sulfapyridine could cure most cases where sulfanilamide failed. Unfortunately, in 1944, many gonococcal species showed clinical resistance, and by the late 1940s,> 90% of gonococcal isolate was resistant to sulfonamides in vitro. Sulfonamides (e.g., sulfamethoxazole) continued to be used, especially when combined with trimethoprim and in low-dose settings, for decades.[9]

Penicillin

In 1928, Alexander Fleming mistakenly concluded that compounds produced by fungi could kill staphylococci and other organisms that cause numerous infectious diseases. He discovered that the fungus belonged to the genus Penicillium, and after being called "the fungus juice," the compound was named penicillin in early 1929. In 1930, Cecil Paine used an extracellular extract from the penicillin-producing penicillin Penicillium notatum to treat gonococcal. ophthalmia in a child. However, it was not until 1943 that this "miracle drug" was officially prescribed for the treatment of gonococcal urethritis, and penicillin marked the new era in the treatment of diarrhea and other infectious diseases. Penicillin soon replaced sulfonamide as the first treatment for gonorrhea. Penicillin cures over 95% of infections, using low doses of 45 mg. However, over time, MICs of penicillin against gonococcal species increased due to chromosomal resistance, and the prescribed doses increased gradually to obtain appropriate therapeutic levels. Penicillin (0.6 to 1.6 million units) was reported, and this resistance was confirmed by in vitro testing. A gradual increase in half the number of gonococcal strains and increased resistance to penicillin was observed over the next two decades. However, after the "epidemic" of gonorrhea in the United States and many other "sex-revolutionary" countries in the 1960's, penicillin levels needed to treat simple acne were greatly increased, and treatment failure was reported. In 1976 the two types of asm-lactamase-encoding plasmids caused high levels of penicillin, from Southeast Asia and sub-Saharan Africa, in certain gonococcal species from the United States and the United Kingdom strengthened fear that for decades-long-term use of penicillin may soon end. The rapid spread of these species worldwide caused great concern. However, when penicillin was discontinued as a first-line antiretroviral drug in the United States and several other countries about a decade later, the main reason was the emergence of chromosomal penicillin resistance. Outbreaks of chromosomal-resistant penicillin gonorrhea in Durham, NC were the first major contributors to the continued use of penicillin. Currently, penicillin-resistant forms of penicillin with plasmid and / or chromosomal are common worldwide.

Since 1989, penicillin has not been recommended for the treatment of gonorrhea due to the widespread prevalence of allergies; penicillin resistance remains common.In 2014, a total of 826 (16.2%) split to show penicillin resistance alone or combined with other resistance phenotypes.[10]

Tetracycline

The first tetracycline, chlortetracycline (aureomycin), was discovered in the soil bacteria in the allotment in 1945 by Benjamin Minge Duggar. Tetracyclines were used early in the treatment of epilepsy, especially in patients who were allergic to penicillin. However, tetracycline MICs against gonococcal species increase over time, due to chromosomal resistance levels. The emergence of tetM determinant (which causes high tetracycline resistance) in conjugative plasmid in the mid-1980s led to the release of tetracycline in medical guidelines in the United States and in many countries around the world. These gonococcal strains with high plasmid-mediated resistance to tetracycline were first reported in 1986 in the United States and soon thereafter in the Netherlands and are now widespread worldwide.[11]

Spectinomycin

In the early 1960's, spetinomycin was synthesized and marketed as a special treatment for gonorrhea. Spectinomycin is a closely related aminocyclitol to the aminoglycosides produced by Streptomyces spectabilis. Spectinomycin is naturally produced by many organisms, including cyanobacteria. After the emergence of plasmid-mediated penicillin resistance, spetinomycin was commonly used to treat these conditions. However, in 1967, spetinomycin resistance was reported with penicillin-susceptible gonococcal strain in the Netherlands, and in 1981, spetinomycin-resistant gonococcal isolate and plasmid-mediated high-level resistance to penicillin was reported in Philippines. 1981 in South Africa. In Korea, spetinomycin was introduced as a treatment for first-line gonorrhea in the US military. [20]However, after only 4 years, an 8.2% clinical failure rate was defined. In addition, in 1983, spetinomycin-resistant gonococcal isolate was reported from London, United Kingdom. Next, spetinomycin was discontinued as the first monotherapy for gonorrhea internationally. Currently, spetinomycin resistance, especially high-dose resistance, is very rare in gonococcal species worldwide. However, spetinomycin is currently not available and widely used in many countries, and it is feared that resistance will be selected as soon as spectinomycin is introduced with first-line treatment. In addition, spetinomycin is not very effective in treating pharyngeal gonorrhea, that is, its effectiveness is about 80%.[12]

Quinolones

Quinolone antimicrobials were discovered by George Lesher and his colleagues as a product of the production of chloroquine in the 1960s, and quinolone nalidixic acid was introduced to treat urinary tract infections in humans. Nalidixic acid is a precursor to all quinolones, and the following quinolones, with a broad spectrum known as fluoroquinolones. Fluoroquinolones ciprofloxacin and ofloxacin were previously recommended for the treatment of gonorrhea, and ciprofloxacin in particular was widely used in the treatment of diarrhea from the mid-late 1980s onwards. Initially, low doses, e.g., 250 mg, of ciprofloxacin were used, but clinical failure was reported in 1990. The recommended dose of ciprofloxacin was increased to 500 mg, but resistance increased and spread rapidly, initially in the West Asian Province. In some Asian countries in the Western Pacific, fluoroquinolones were discontinued as an effective treatment for first-line gonorrhea in the

mid-1990's. Ciprofloxacin-resistant gonococcal strains were then rapidly exported or emerged independently. In the United States, in 2000, fluoroquinolone-resistant strains originally imported from Asia were prevalent in Hawaii, and after that, the strains first spread to the West Coast and then to the rest of the United States, especially among MSM. In 2007, fluoroquinolones were discontinued from CDC-recommended treatments for gonorrhea, with no exception. Due to the high levels of fluoroquinolone, many Asian and European countries discontinued ciprofloxacin as a first-line treatment in the early 2000's. Currently, the prevalence of fluorooccal-resistant gonococcal strains is high worldwide.[14]

Macrolides

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Cephalosporins

The first compounds of cephalosporin were isolated from the fungal culture Cephalosporium acremonium, first discovered by Giuseppe Brotzu in 1948. The chemical modification of these and other similar compounds resulted in the first beneficial use of antimicrobial, namely, cefalotin, developed in 1964. Cephalosporins are commonly recommended by cephalosporins internationally for treatment of the disease following the depletion of fluoroquinolones ESC third generation ceftriaxone (injection) and cefixime (oral). No other injection or oral ESCs have obvious benefits over ceftriaxone and cefixime. However, other oral cephalosporins have been used when cefixime is not available, e.g., cefditoren and celdinir in Japan, cefuroxime in several European countries, cefpodoxime in the United States, and ceftibuten in Hong Kong.[17] Over the past two decades, gonococcal strains that are resistant to ESCs seem to have begun to emerge in Japan and spread around the world. In Japan, ceftriaxone was not approved for the treatment of gonorrhea from the 1990s to the early 2000s. As a result, many oral cephalosporins and dosage medications, including some very effective ones, were discontinued monotherapy, but when resistance was detected, cefodizime or spectinomycin was given. Many low-dose oral cephalosporins were commonly used, which may have caused subinhibitory cephalosporin concentration and, accordingly, may be selectively resistant to cephalosporin. In addition, when single-dose cefixime (the most potent oral oral ESC) used in Japan, it usually consisted of only 300 mg of cefixime, in contrast to the 400-mg dose used internationally. Thus, between 1995 and -2000, in Fukuoka, Japan, the MIC peaks of cefixime and ceftriaxone against gonococcal isolate reached 0.25 μg / ml and 0.064 μg / ml, respectively. In addition, between 1999 and 2002, in six hospitals in central Japan, concentrations of gonococcal isolates and in vitro resistance to cefixime (MICs ≥0.5 μg / ml) and ceftriaxone (MICs ≥0.5 μg / 0.9%) up to ≥0.5 μg / ml. %, respectively. This also translated into the failure of cefixime treatment. Similarly, from 1999 to 2001, eight treatment failures with cefixime (200 mg twice orally, 6 hours apart) were reported, and in 2002 to 2003, four treatment failures with an extended cefixime program (200 mg orally twice daily for three days). were written. In 2006, all oral ESCs were not included in treatment regimens in Japan, and since then, ceftriaxone (1 g intravenously), the most commonly used, cefodizime (1 g intravenously), and spectinomycin (2 g) g intramuscularly) has been recommended for the first time. -a powerful treatment for anogenital and pharyngeal gonorrhea. Over the past decade, problems that diminished the tendency or resistance of ESCs have spread around the world, and their existence has been documented worldwide. ESCs are not well known. Currently, cefixime treatment failure has been confirmed in Japan, several European countries, Canada, and South America, and a few ceftriaxone treatment failures for pharyngeal gonorrhea have been reported in Japan, other European countries and Australia.[16]

It is very worrying that the first forms of gonococcal XDR, which show high clinical resistance to all ESCs combined with resistance to almost all other antimicrobials available for treatment, have recently been discovered in Kyoto, Japan, Quimper, France, and Catalonia, Spain. All of these types of XDR have also been identified in high-risk, often transmitted, i.e., sex workers (CSWs) or MSMs. Since ceftriaxone is a last-line treatment for gonorrhea monotherapy, the emergence of XDR gonococci may trigger a period of

chronic gonorrhea using antimicrobial monotherapy. Fortunately so far, however, based on the intensified surveillance in Kyoto and Osaka (2010 to 2012) after the identification of the first version of XDR (H041), this type is not yet widespread in the local community, which may indicate a decrease in body weight.[15]

Standard Treatment Guidelines (Stg) For the Treatment of Neisseria Gonorrhoeae

Neisseria gonorrhoeae can be diagnosed by culture or nucleic acid amplification tests (NAATs), and by Gram stain in men with urethritis. In settings without available laboratory diagnostic support, diagnosis is often made clinically, based on the presence of symptoms such as vaginal and urethral discharge. The treatment of gonococcal infections is complicated by the rapidly changing antimicrobial susceptibility patterns of N. gonorrhoeae, raising concerns about the eventual development of untreatable gonococcal infections with serious sexual and reproductive health consequences.[21]

Rationale for the Guidelines

Since the publication of the World Health Organization (WHO) Guidelines for the management of sexually transmitted infections (STIs) in 2003, changes in the epidemiology of STIs and advancements in prevention, diagnosis and treatment necessitate changes in STI management. There is an urgent need to update treatment recommendations for gonococcal infections to respond to changing antimicrobial resistance (AMR) patterns of N. gonorrhoeae.[22] High-level resistance to previously recommended quinolones is widespread and decreased susceptibility to the extended-spectrum (third-generation) cephalosporins, another recommended first-line treatment in the 2003 guidelines, is increasing and several countries have reported treatment failures. These guidelines for the treatment of common infections caused by N. gonorrhoeae form one of several modules of guidelines for specific STIs. Other modules will focus on treatments for Chlamydia trachomatis (chlamydia), herpes simplex virus type 2 (HSV-2; genital herpes) and Treponema pallidum (syphilis). In addition, future work will provide guidance for syphilis screening and treatment of pregnant women, STI syndromic approach, clinical management, STI prevention, and treatments for other STIs. It is strongly recommended that countries take updated global guidance into account as they establish standardized national protocols, adapting this guidance to the local epidemiological situation and antimicrobial susceptibility data.[23]

These guidelines were developed following the methods outlined in the 2014 WHO handbook for guideline development. The Guideline Development Group (GDG) included international STI experts, clinicians, researchers and programme managers. The GDG prioritized questions and outcomes related to treatment of gonococcal infections to include in this update, and a methodologist and a team of systematic reviewers from McMaster University, the WHO Collaborating Centre for Evidence-Informed Policy, independently conducted systematic reviews of the effectiveness of different treatments for gonorrhoea. The evidence was assessed using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) approach and presented to the GDG. Conflicts of interest were managed according to WHO guidelines and declared before the recommendations were discussed and finalized. Research implications were also developed by the GDG.[24]

CONCLUSION

Alternative types of medications are also available when ceftriaxone can be used to treat urogenital or rectal gonorrhea. Although the medication will stop the infection, it will not repair any damage done by the infection. Antimicrobial resistance to gonorrhea increases anxiety, and effective treatment of gonorrhea becomes more difficult. Medical examination - follow-up testing to ensure that the infection has been successfully treated - is not required for genital herpes and rectum; however, if a person's symptoms persist for more than a few days after receiving treatment, he or she should return to a health care provider for a re-examination. A medical examination is required 7-14 days after the treatment of people who are being treated for a sore throat. Because re-infection is common, men and women with gonorrhea should be re-examined within three months after initial infection treatment, regardless of whether they believe their sexual partners have been successfully treated.

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