Tigecycline (Tygacil): A Novel First-in-Class, Broad-Spectrum Intravenous Antibiotic For the Treatment of Serious Bacterial Infections

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INTRODUCTION

Antibiotic resistance has become a widespread problem. Many of the prominent bacterial infections identified today are rapidly becoming resistant to commonly prescribed antibiotics that are used to treat them. Life-threatening infections continue to emerge, and global concerns only continue to grow as the arsenal of effective drugs used to combat infections dwindles by the day.

It is well known that appropriate antibiotic therapy, when administered promptly and effectively, plays a vital role in the clinical management of critically ill patients with bacterial infections. The outcomes, however, become compromised as the pathogens responsible for causing the infections demonstrate some form of antibiotic resistance. As many as 70% of bacteria responsible for hospital-acquired (nosocomial) infections are resistant to at least one common antibiotic used in the past to treat it, and many strains now exhibit multidrug resistance (MDR). It is for this reason that a demand now exists for new and improved antimicrobial agents.

Over the last 20 years, there has been a shortage of new antibiotics in the pipeline, and the approval of tigecycline (Tygacil, GAR-936, Wyeth) represents an important advance in expanding the range of antibiotics available to combat infections. Tigecycline gained the approval of the U.S. Food and Drug Administration (FDA) as a first-in-class antibiotic on June 15, 2005. It is indicated as single-agent, intravenous (IV) therapy for serious bacterial infections, such as complicated intra-abdominal and skin and soft tissue infections.

The drug’s mechanism of action involves the inhibition of bacterial protein translation through its action on the 30S ribosomal subunit. In vitro studies have shown that tigecycline is highly active against gram-positive and gram-negative aerobes, atypical pathogens, and anaerobic bacteria. Tigecycline provides clinicians with a novel, broad-spectrum alternative that can be used empirically when symptoms first present themselves and when the identity of the specific offending bacterium is not yet known.

Empirical treatment of bacterial infections, regardless of their severity, remains one of the most daunting medical challenges that health practitioners face. Selecting a therapy is complicated by characteristics specific to each patient (e.g., age, chronic illness, drug allergy, and polypharmacy) as well as by other significant clinical considerations.

Patients who require routine medical care are often unintentionally exposed to MDR pathogens because of more frequent contact with sick people in the health care system. In turn, this increased contact can result in infections that require treatment with multiple antibiotics. These two points can be perceived as major risk factors for antibiotic failure.

It has been determined that the primary cause of hospitalization in the elderly is the presence of bacterial infections, which alone result in a 30% mortality rate in these patients despite the initiation of pharmacological therapy.

The Centers for Disease Control and Prevention (CDC) has stated that people infected with drug-resistant organisms are more likely to have longer hospital stays and need treatment with multiple drugs.

The use of antibiotic combinations to fight infections, along with extended hospital stays, places a great economic burden on our health care system. The consequences of antibiotic resistance will cost the U.S. between $4 billion and $5 billion annually. These figures will continue to increase until efforts are made to overcome this problem.

Research data suggest that the development and approval of new antibacterial agents have decreased by about 56% over the last 20 years. This in turn generates a sense of urgency in drug companies to look for new classes of antibiotics that can address the increasing resistance patterns emerging among the many common pathogens present today.

CHEMISTRY AND PHARMACOLOGY

Tigecycline is a glyclcycline antibacterial agent used for IV infusion. It is chemically designated as C_{29}H_{39}N_5O_8 with a molecular weight of 588.65. This orange lyophilized powder or cake is available in single-dose, 5-ml vials containing 50 mg of the powder. Figure 1 illustrates the agent’s chemical structure.

Tigecycline is the first member of a new class of glyclcyclines. It is a semisynthetic tert-butyl-glycylamido derivative of minocycline, possessing potent activity against tetracycline-resistant pathogens that are refractory by both efflux and ribosomal protection mechanisms. This product is free of excipients and preservatives.
MECHANISM OF ACTION

Tigecycline was designed to circumvent key bacterial resistance mechanisms that have affected previous antibacterial drug use, including ribosomal protection, macrolide or tetracycline efflux pumps, target-site modifications, beta-lactamases (including extended-spectrum beta-lactamases), and DNA gyrase mutations. It is generally bacteriostatic and blocks the entry of amino-acyl transfer RNA (t-RNA) molecules into the A site of the 30S ribosomal subunit, thus preventing protein synthesis (Figure 2). Tigecycline provides an expanded broad spectrum of in vitro activity against the following (Table 1):

- gram-positive pathogens, such as methicillin-resistant Staphylococcus aureus (MRSA) and Staphylococcus epidermidis (MRSE), and vancomycin-resistant enterococci (VRE) (both Enterococcus faecalis and Enterococcus faecium)
- gram-negative pathogens, such as Acinetobacter baumannii and Stenotrophomonas maltophilia
- anaerobic pathogens

As a class, glycyclines bind to the ribosome with an affinity that is five times greater than that of the tetracyclines. In addition, tigecycline binds to other sites of the ribosome in a manner never seen before, interfering with the mechanism of ribosomal protection proteins, and it is not expelled by macrolide or tetracycline efflux pumps.

PHARMACOKINETICS

Tigecycline’s distribution goes beyond the plasma volume and takes place extensively in the tissues, as seen by its steady-state volume of distribution (average, between 500 and 700 liters). It exhibits moderate plasma protein binding (range, 71%–89%). Linear dose proportionality is observed between tigecycline peak plasma concentrations and area-under-the-curve (AUC) values noted over the dose range of 12.5 to 300 mg. The agent is available only as an intravenous formulation because of its limited oral bioavailability. Evaluation of human liver hepatocytes following in vitro administration of tigecycline reveals only trace amounts of metabolite formation, suggesting that tigecycline is not extensively metabolized. After 14C-tigecycline is given, about 59% of the dose is eliminated via biliary and fecal excretion, and 33% is excreted in the urine. Unchanged tigecycline excreted in the urine makes up about 22% of the total dose administered.

By and large, biliary excretion of unchanged tigecycline and its metabolites is the primary route of elimination. Secondary routes include glucuronidation and renal excretion. Tigecycline’s mean half-life after a single 100-mg dose is 27.1 hours. Multiple doses of 50 mg every 12 hours yield a mean half-life of 42.4 hours.

CLINICAL TRIALS

Complicated Intra-abdominal Infections

Tigecycline 301 and 306 Studies

Babinchak et al. conducted two phase 3, randomized, double-blind studies comparing the safety and efficacy of tigecycline with that of imipenem/cilastin (Primaxin, Merck) in hospitalized adults. These patients were candidates for laparoscopy or had undergone a laparoscopic procedure or percutaneous drainage of an intra-abdominal abscess, and they also had probable or known complicated intra-abdominal infections (cIAIs) (Figure 3).

The 301 study took place in 96 centers within 17 countries, including the U.S., Canada, Europe, Latin America, India, and Asia. The worldwide 306 study took place in 94 centers within 27 countries in Europe, South Africa, and Asia.

A total of 1,759 patients were initially screened for enrollment; 1,658 were randomized to the clinical trials. Tigecycline 301 and 306 Studies

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A total of 1,759 patients were initially screened for enrollment; 1,658 were randomized to the clinical trials.
The primary efficacy endpoint was the observed clinical response within the microbiological modified intent-to-treat (m-mITT) and microbiologically evaluable populations at the test-of-cure visit (12 to 42 days after therapy). For the microbiologically evaluable and m-mITT populations, tigecycline demonstrated comparable efficacy and was statistically non-inferior to imipenem/cilastin.

Clinical cure rates for the microbiologically evaluable patients were 86.1% with tigecycline and 86.2% with imipenem/cilastin. Corresponding rates for the m-mITT patients were 80.2% with tigecycline and 81.5% with imipenem/cilastin.

Of the many observed diagnoses, complicated appendicitis was the most common disorder. The cure rate with tigecycline was 88.2%, and the rate with imipenem/cilastin was 89.3%.

For both treatment groups, the most common adverse drug events (ADEs) related to the gastrointestinal (GI) tract (nausea, vomiting, and diarrhea) were experienced by 44% of patients receiving tigecycline and by 39% of patients receiving imipenem/cilastin.** 14,15

** Complicated Skin and Skin Structure Infections
Tigecycline 300 and 305 Studies** 16-18
Ellis-Grosse et al. conducted two

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Table 1  *In vitro Activity of Tigecycline against Clinical Microbial Isolates*  

| Gram-positives | Yes | Staphylococcus aureus (MSSA)**; Staphylococcus epidermidis (MSSE); Staphylococcus haemolyticus; Streptococcus agalactiae; Streptococcus pyogenes; Streptococcus anginosus grp. (includes *S. anginosus, S. intermedius, and S. constellatus)**; Enterococcus faecalis (vancomycin-susceptible isolates only)**; Enterococcus faecium; Enterococcus avium; Enterococcus casseliflavus; Enterococcus gallinarum; Listeria monocytogenes |
| Gram-negatives | Yes | Citrobacter freundii; Enterobacter aerogenes; Enterobacter cloacae; Escherichia coli**; Klebsiella oxytoca; Klebsiella pneumoniae; Aeromonas hydrophila; Citrobacter koseri; Pasteurella multocida; Serratia marcescens |
| Anaerobes | Yes | Bacteroides fragilis**; Bacteroides uniformis**; Bacteroides vulgatus**; Bacteroides thetaiotaomicron**; Bacteroides distasonis**; Bacteroides avitus**; Prevotella spp.; Clostridium perfringens**; Peptostreptococcus micros**; Peptostreptococcus spp.; Porphyromonas spp. |
| Resistant gram-positives | Yes | Staphylococcus aureus (MRSA)**; Staphylococcus epidermidis (MRSE); Enterococcus faecalis (VRE); Enterococcus faecium (VRE) |
| Resistant gram-negatives | Yes | Acinetobacter baumannii; Stenotrophomonas maltophilia |
| Pseudomonas aeruginosa | No | |

* † Clinical efficacy has been demonstrated for susceptible isolates in complicated skin and skin structure infections* and in complicated intra-abdominal infections. † The clinical significance of *in vitro* activity is unknown.

Data from Wyeth and www.tygacil.com. 4,10

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domly assigned; 1,642 were included in the modified intent-to-treat (m-ITT) population; 1,601 patients made up the clinical modified (c-mITT) population; 1,382 patients were clinically evaluable; and 1,025 were microbiologically evaluable.

Patients were randomly assigned to receive either tigecycline and placebo or IV imipenem/cilastin for a period up to 14 days. The patients who were given tigecycline received an initial IV loading dose of 100 mg infused over 30 minutes, followed by a maintenance dose of 50 mg every 12 hours. Six hours after each infusion, the patients were given 100 ml of normal saline placebo to maintain blinding in the study. Patients receiving imipenem/cilastin were given 100 mg infused over 30 minutes, followed by a maintenance dose of 50 mg every 12 hours in 250 ml of normal saline, every 12 hours.

To be eligible for enrollment, the men and women had to be 18 years of age or older; had to have a cIAI requiring surgery; and had to have appendicitis, cholecystitis, diverticulitis, gastric/duodenal perforation, intra-abdominal abscess, intestinal perforation, or peritonitis. Patients were not eligible if they had any other concomitant health condition that prohibited the true evaluation of a clinical response or that rendered the completion of the planned course of therapy highly unlikely.

The worldwide study took place in 65 centers in 21 countries (in the U.S., Canada, Argentina, Chile, Guatemala, Mexico, Peru, and India). The worldwide study took place in 65 centers in 21 countries (in Europe, Asia, Australia, and South Africa).

A total of 1,153 patients were initially screened for study enrollment; 1,129 were randomly assigned; 1,116 were included in the m-ITT population; 1,057 made up the c-mITT population; 833 were clinically evaluable; and 540 were microbiologically evaluable.

Patients were assigned to receive either tigecycline and placebo or the IV antibiotic combination of vancomycin (generic) plus aztreonam (Azactam, Elan) in hospitalized patients with complicated skin and skin structure infections (cSSSIs) (Figure 4). The North America/South America study took place in 53 centers in eight countries (in the U.S., Canada, Argentina, Chile, Guatemala, Mexico, Peru, and India). The North America/South America study took place in 53 centers in eight countries (in Europe, Asia, Australia, and South Africa).
followed by 2 g of aztreonam in 100 ml of normal saline over one hour.

To be considered eligible for enrollment, patients had to be hospitalized and 18 years of age or older with a cSSSI characterized by a general wound infection with cellulitis, a major abscess, and an infected ulcer or a burn. The patients also had to have symptoms such as fever, wound discharge, erythema, swelling, pain, and a white blood cell count above 10,000 cells/mm³. Patients who satisfied all required criteria were ultimately enrolled if they needed IV antibiotic therapy for more than five days.

The primary efficacy endpoint was the clinical response within the clinically evaluable and mITT populations at the test-of-cure visit (approximately 12–92 days after the last administered dose). To establish the non-inferiority of tigecycline versus vancomycin/aztreonam, the investigators used a two-sided 95% confidence interval (CI) to determine the difference in efficacy. Non-inferiority was presumed when the lower limit of the two-sided 95% CI was –15% or greater. For the clinically evaluable and mITT populations, tigecycline demonstrated comparable efficacy and was statistically non-inferior to vancomycin/aztreonam.

Clinical cure rates for the mITT patients were 79.7% with tigecycline and 81.9% with vancomycin/aztreonam. Corresponding clinical cure rates for the clinically evaluable patients were 86.5% with tigecycline and 88.6% with vancomycin/aztreonam.

Of the many observed diagnoses, soft-tissue infections were the most common. The clinical cure rates were 86.3% with tigecycline and 87.3% with vancomycin/aztreonam.

GI-related ADEs (nausea, vomiting, diarrhea, anorexia, and dyspepsia) were most frequently reported for both treatment groups; these rates, however, were considerably higher with tigecycline (46% of patients) than with vancomycin/aztreonam (21% of patients).16–18

ADVERSE DRUG REACTIONS

The side-effect profile of tigecycline is similar to that of its comparators, namely the antibiotics used in the four published phase 3 clinical trials (vancomycin/aztreonam and imipenem/cilastin). Within the phase 3 studies, 1,415 patients were treated with tigecycline. Treatment-emergent ADEs led to the discontinuation of tigecycline in 5% of patients versus 4.7% for all comparator agents combined.

When all of the ADEs were assessed in conjunction with the use of tigecycline, the digestive system appeared to be the most commonly reported body system affected. ADEs included nausea (incidence, 29.5%), vomiting (incidence, 19.7%), and diarrhea (incidence, 12.7%). Additional notable adverse reactions included fever (7.1%), thrombocytopenia (6.1%), headache (5.9%), and hypertension (4.9%).

Table 2 summarizes the treatment-emergent ADEs from each phase 3 trial. Discontinuation of tigecycline was most often associated with nausea (1.3%) and vomiting (1%). Nausea and vomiting were usually mild to moderate in severity, and their onset usually occurred during the first few days of therapy. In each phase 3 trial, death occurred in 2.3% of patients taking tigecycline and in 1.6% of patients using comparator drugs. This difference was not statistically significant, because its relationship to treatment could not be adequately established. Careful consideration of all treatment groups collectively suggests that mortality is more closely associated with higher morbidity or more severe infection at the baseline evaluation.2

Fewer ADEs (range, ≥0.2% to <2%) reported in patients receiving tigecycline included, but were not limited to, pain and inflammation at the injection site, Bradycardia, tachycardia, anorexia, dry mouth, jaundice, hypoglycemia, somnolence, and taste perversion.
DOSAGE AND ADMINISTRATION

Adults

The recommended dosing regimen for non-elderly adults receiving tigecycline should include an initial loading dose of 100 mg, followed by a maintenance dose of 50 mg every 12 hours via an IV infusion, administered over a 30- to 60-minute span every 12 hours. Treatment of patients with cSSSIs or with cIAIs should last for five to 14 days, with the exact duration of therapy determined by the site and severity of the infection, the patient’s clinical improvement, and the progress in bacterial eradication.2

Geriatric Patients

It is not necessary to employ any dosing adjustments for tigecycline simply on the basis of age, sex, or race.6 The phase 3 clinical trials included a total of 388 subjects 65 years of age or older; following treatment, no significant differences were noted between these and younger subjects. However, the likelihood that these older patients were more vulnerable to experiencing ADEs cannot be ruled out, because they can and often do demonstrate greater sensitivities to various drug regimens.2

Patients with Hepatic Impairment

Tigecycline should be used with caution in patients with severe hepatic impairment, as indicated by a Child Pugh score of C. Dosing for these patients should start with an initial loading dose of 100 mg, followed by a reduced maintenance dose of 25 mg every 12 hours. These patients should be treated cautiously and monitored regularly for their response to treatment.

No dosing adjustments are needed for patients with mild-to-moderate impairment, as indicated by Child Pugh scores of A and B.

Patients with Renal Impairment

The dose does not have to be adjusted for patients with renal impairment or for those undergoing hemodialysis. A single-dose study showed that tigecycline is not removed by hemodialysis, an important point to remember if an overdose is suspected.2

Children and Pregnant or Lactating Patients

The use of tigecycline in patients younger than 18 years of age has not been evaluated; thus, safety and effectiveness data have not yet been established for these patients. As a result, this agent is not recommended for this age group.

Tigecycline is categorized as a pregnancy category D drug. No adequate or well-controlled studies have been conducted in pregnant women, and it is not clear whether tigecycline is excreted in human milk. Therefore, the risks and benefits of tigecycline should be considered before it is prescribed during pregnancy or lactation.

Preparation and Handling

Tigecycline is supplied in a single-dose, 5-ml vial containing 50 mg of lyophilized powder for reconstitution.2 The vials should be reconstituted with 5.3 ml of 0.9% normal saline or 5% dextrose to attain a desired concentration of 10 mg/ml.

After the drug is reconstituted, 5 ml of tigecycline solution is equivalent to 50 mg of the drug; it should appear yellow

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Treatment-Emergent Adverse Drug Events Reported from Phase 3 Clinical Trials of Tigecycline</th>
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<td>Tigecycline (N = 1,415) vs Comparator* (N = 1,382)</td>
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<tr>
<td>Digestive System</td>
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<tr>
<td>Nausea</td>
<td>29.5% vs 15.8%</td>
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<tr>
<td>Vomiting</td>
<td>19.7% vs 10.08%</td>
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<tr>
<td>Diarrhea</td>
<td>12.7% vs 10.8%</td>
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<tr>
<td>Body as a Whole</td>
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<tr>
<td>Infection</td>
<td>8.3% vs 5.4%</td>
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<tr>
<td>Fever</td>
<td>7.1% vs 9.8%</td>
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<tr>
<td>Abdominal pain</td>
<td></td>
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<tr>
<td>Headache</td>
<td>3.7% vs 2.9%</td>
</tr>
<tr>
<td>Pain</td>
<td>3.7% vs 2.9%</td>
</tr>
<tr>
<td>Abscess</td>
<td>3.2% vs 2.6%</td>
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<tr>
<td>Cardiovascular System</td>
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<tr>
<td>Hypertension</td>
<td>4.9% vs 5.6%</td>
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<tr>
<td>Hemic and Lymphatic System</td>
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<tr>
<td>Thrombocytopenia</td>
<td>6.1% vs 6.2%</td>
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<tr>
<td>Anemia</td>
<td>4.2% vs 4.8%</td>
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<tr>
<td>Leukocytosis</td>
<td>3.7% vs 2.5%</td>
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<tr>
<td>Metabolic and Nutritional</td>
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</tr>
<tr>
<td>SGPT increased†</td>
<td>5.6% vs 4.7%</td>
</tr>
<tr>
<td>SGOT increased†</td>
<td>4.5% vs 3.0%</td>
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<tr>
<td>Lactic dehydrogenase increased</td>
<td>4.0% vs 3.5%</td>
</tr>
<tr>
<td>Alkaline phosphatase increased</td>
<td>3.5% vs 2.6%</td>
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<tr>
<td>Healing abnormal</td>
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<tr>
<td>Peripheral edema</td>
<td>3.3% vs 3.3%</td>
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<tr>
<td>Amylase increased</td>
<td>3.1% vs 1.4%</td>
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<tr>
<td>Nervous System</td>
<td></td>
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<tr>
<td>Dizziness</td>
<td>3.5% vs 2.7%</td>
</tr>
<tr>
<td>Respiratory System</td>
<td></td>
</tr>
<tr>
<td>Cough increased</td>
<td>3.7% vs 3.8%</td>
</tr>
<tr>
<td>Other</td>
<td></td>
</tr>
<tr>
<td>Local reaction to procedure</td>
<td>9.0% vs 9.1%</td>
</tr>
</tbody>
</table>

SGOT = serum glutamic oxaloacetic transaminase (aspartate transaminase); SGPT = serum glutamate pyruvate transaminase (alanine transaminase).
† These events were more frequently reported on therapy for comparators and after therapy for tigecycline.

to orange in color. When the drug is being reconstituted, the vial should be swirled in an attempt to agitate the contents and to promote total dissolution of the tigecycline powder. After the powder dissolves, 5 ml of the resulting solution is withdrawn and added to a 100-ml IV bag for administration for IV infusion. To achieve a 100-mg dose, two vials must be reconstituted.

Tigecycline should be stored at room temperature (15° to 30°C) prior to reconstitution. After it is reconstituted, the resulting solution must be immediately transferred from its original vial. After it is injected into an IV bag, tigecycline can be stored at room temperature for up to six hours or placed in the refrigerator (2° to 8°C), where it can be maintained for up to 24 hours.

DRUG INTERACTIONS

As with any antibacterial drug, the concurrent use of tigecycline with oral contraceptives may render the oral contraceptives less effective. If patients are taking warfarin (Coumadin, Bristol-Myers Squibb), prothrombin times or other anticoagulation tests must be monitored because tigecycline can decrease the clearance of warfarin by 23% to 40%; in turn, it increases the peak plasma concentration of warfarin by 38% to 43%.³

Tigecycline does not inhibit metabolism mediated by the cytochrome (CYP) P450 isoforms 1A2, 2C8, 2C9, 2C19, 2D6, or 3A4.² As a result, it is understood that tigecycline does not impede or modify the breakdown of drugs metabolized by these enzymes. In turn, these enzymes do not affect the clearance of tigecycline because it is not extensively metabolized.

Tigecycline is compatible with dobutamine, dopamine HCl, lactated Ringer's solution, lidocaine HCl, potassium chloride, ranitidine, and theophylline. Amphotericin B, chlorpromazine, methylprednisolone, and voriconazole should not be used simultaneously with tigecycline.²,¹⁰

CONTRAINDICATIONS AND PRECAUTIONS

Tigecycline is absolutely contraindicated in patients with a known hypersensitivity to it. Because glycylcycline antibiotics as a class are similar in structure to the tetracycline antibiotics, they may exhibit comparable adverse effects. Therefore, tigecycline should be administered with caution to patients with a known hypersensitivity to the tetracycline class of antibiotics.

The use of tigecycline during pregnancy may cause fetal harm as it crosses the placenta, resulting in decreased fetal weight. It should not be used during tooth development because it may cause permanent discoloration of the teeth. It should be used with caution in patients with cIAIs secondary to clinically apparent intestinal perforations, because sepsis or septic shock can result.²

Tigecycline should be restricted for patients with proven or suspected bacterial infections, because misuse of the drug can lead to the development of drug-resistant bacteria.

Treatment with tigecycline should be continued as long as the patient tolerates it. If any adverse reactions become intolerable or if the patient does not improve with tigecycline therapy, the regimen should be discontinued and alternatives should be considered.

COST

The average wholesale price (AWP) of a 10-day supply of 50 mg/vial of tigecycline is $1,194.16 (10 days is the normal course of therapy for cSSSIs). Currently, the product is available only in the injectable form. The acquisition cost may vary according to the health care system and to promote total dissolution of the tigecycline class of antibiotics. The introduction of tigecycline provides new hope, because physicians now have an additional agent to use for complicated bacterial infections.

The addition of this drug to the ever-dwindling supply of antibiotics may prove to be vital, as the bacterial infections driven by the “superbugs” such as MRSA and VRE become more prevalent. Only time will tell whether tigecycline has the power to persevere in this age of growing antibiotic resistance.

REFERENCES


