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Delafloxacin: A New Fluoroquinolone for Acute Skin and Soft Tissue Infections

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Skin and soft tissue infections (SSTIs) are commonly diagnosed in the outpatient or inpatient setting in patients of all different ages and backgrounds. SSTIs are first categorized as either purulent (abscess, carbuncle, furuncle) or non-purulent (cellulitis, erysipelas, necrotizing fasciitis) infections, then further classified as complicated or uncomplicated. Uncomplicated infections caused by *Staphylococcus aureus* or *Streptococcus pyrogens* are often simple abscesses, impetigo lesions, furuncles, and cellulitis, all of which involve superficial layers of the skin. Complicated infections, however, involve the deeper layers of the skin and soft tissue structures which often requiring the use of medical and surgical interventions. To help differentiate these complicated infections from simple infections the Food and Drug Administration (FDA) in 2010 proposed a new classification, acute bacterial skin and skin structure infections (ABSSSIs), to describe these complicated infections. To meet this new classification the infection must include cellulitis/erysipelas, wound infections, and major skin abscesses, all of which present with edema, erythema, or induration with a minimum surface area of ≥75 cm^2.[1-3]

The amount of individuals developing a complicated SSTI, whether community- or hospital- acquired, is steadily increasing. From 1993 to 2005, the incidence of complicated SSTIs resulting in emergency room visits increased 3-fold, and there was a 29% increase in SSTI diagnoses in hospital admissions from 2000 to 2004.[4,5] Amongst the causative pathogens responsible for these infections, *S. aureus* is one of the most common, exhibiting a high prevalence of methicillin-resistant *S. aureus* (MRSA) isolates. The resulting clinical and financial complications has caused increased hospitalizations and hospital days, poor mortality outcomes, and financial burden from cost of treatment for both hospitals and patients.[6-8] MRSA infections are associated with poorer clinical outcomes when compared to other infections, and the rate of treatment failure is higher than infections caused by methicillin-susceptible *S. aureus* (MSSA) infections.[9]

Moreover, with antibiotic resistance on the rise, there is a potential risk for decreased therapeutic options. From 1998 to 2004, pathogens isolated from an SSTI infection showing to be resistant to at least one antibiotic increased from 17% to 35%.[10] The number of *S. aureus* isolates from complicated SSTIs resistant to methicillin is now at 60% of all pathogens, in which 80% were of the community-acquired phenotype.[11] Treatment choice varies among patients according to the type of ABSSSI as well as the patient's clinical presentation. Depending on the severity, there are many acceptable options for oral and IV broad/narrow spectrum antibiotics to fit patient characteristics. Antibiotic coverage should usually include one with effectiveness against gram-positive organisms, with or without MRSA coverage.[1]

Delafloxacin (Baxdela©) is the first fluoroquinolone with MRSA coverage that received FDA approval for the treatment of ABSSSIs. The purpose of this article is to define and evaluate delafloxacin and its place in clinical practice.

Pharmacology

Mechanism of Action

Delafloxacin is a new fluoroquinolone antibiotic with bactericidal activity by inhibiting bacterial enzymes topoisomerase II (DNA gyrase) and topoisomerase IV. Both enzymes are responsible for bacterial DNA replication, transcription, repair, and recombination. Blocking DNA replication results in cell death for bacterial cells in growing and stationary phases.[3] Delafloxacin is shown to have higher bactericidal potency in comparison to the other fluoroquinolones due to three structural differences: 1) lack of a strong base at the C7 position, 2) addition of a chlorine atom (electron-withdrawing group) in the C8 position to stabilize the molecule, and 3) addition of an aromatic ring to the N1 position to increase molecular surface area.[3,12,13] These substituents allow for greater affinity to both DNA gyrase and topoisomerase IV. Also, development of resistance to delafloxacin would require many bacterial mutations that affect both drug targets. Low MICs are achieved (**Table 1**) with delafloxacin due to these structure changes.[14,15]

Pathogen coverage

Delafloxacin exhibits broad spectrum pathogen coverage for the pathogens most common in ABSSSI infections (see **Table 1** for susceptible pathogens and related MIC data). This new medi-

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cation seemed to also be effective against other pathogens when analyzed from *in vitro* studies, such as *streptococcus dysgalactiae*, *Enterobacter aerogenes*, *Haemophilus parainfluenzae*, *klebsiella oxytoca*, and *proteus mirabilis*. Approximately 90% of these pathogens exhibited MICs less than or equal to the breakpoint of delafloxacin against bacterial isolates, but the clinical significance of this data is unknown until further studied in clinical trials.[15] Notably absent is susceptibility data regarding *streptococcus pneumoniae*, delafloxacin should be avoided in respiratory infections until this bacteria susceptibility is determined.

Pharmacokinetics

The oral bioavailability of delafloxacin 450 mg is 58.8%. Oral administration of 450 mg and an IV infusion of 300 mg was shown to have similar AUC of 22.7 and 21.8, respectively (see Table 2). The volume of distribution at steady state is 30 to 48 L, with plasma protein binding of 84% (primarily binds to albumin). The half-life for oral administration varies between 4.2 to 8.5 hours after multiple doses and approximately 3.7 hours for a single IV dose, whereas the values ranged from 4.2 to 8.5 hours for multiple doses of oral delafloxacin.[15]

The primary metabolic pathway is glucuronidation via UGT1A1, UGT1A3, and UGT2B15. Delafloxacin has no significant active metabolites. Renal clearance of delafloxacin accounts for 35-45% of total drug and metabolite clearance. After a single intravenous dose, 65% of delafloxacin was excreted in urine unchanged and 28% was excreted in feces unchanged. For the oral dose of delafloxacin, 50% is excreted unchanged in urine and 48% is excreted unchanged in feces.[15]

Delafloxacin exhibits drug interactions with chelation agents, such as antacids (containing aluminum or magnesium), sucralfate, metal cations (iron), and multivitamins (containing iron or zinc). Co-administration with any of these agents can decrease the systemic absorption of delafloxacin, therefore should be taken 2 hours before or 6 hours after administration of chelation agents.[15]

Table 1 | MICs for Delafloxacin Susceptible Pathogens

Pathogens	MIC (mcg/mL)[a]		
	S	I	R
Gram-Positive Bacteria			
Staphylococcus aureus (MSSA and MRSA)	≤0.25	0.5	≥1
Staphylococcus haemolyticus	≤0.25	0.5	≥1
Streptococcus pyrogens	≤0.06	—	—
Streptococcus agalactiae	≤0.06	0.12	≥0.25
Streptococcus anginosus *Streptococcus constellantus* *Steptococcus intermedius*	≤0.06	—	—
Enterococcus faecalis	≤0.12	0.25	≥0.5
Gram-Negative Bacteria			
Escherichia coli *Klebsiella pneumoniae* *Enterobacter cloacae*	≤0.25	0.5	≥1
Pseudomonas aeruginosa	≤0.5	1	≥2

a: MIC values are from *in vtiro* studies
S = Susceptible; **I** = Intermediate; **R** = Resistant
MIC = mean inhibitory concentration; **MSSA** = methicillin-sensitive *staphylococcus aureas*; **MRSA** = methicillin-resistant *staphylococcus aureas*

Clinical Trials

Delafloxacin was approved based on two phase 3 studies comparing delafloxacin with vancomycin plus aztreonam in the treatment of ABSSSIs. This section includes a review of a phase 2 and phase 3 trial. The second phase 3 study has not been published as of writing this manuscript; however, a summary of findings will be discussed in this section as well as in Table 3.[16]

Table 2 | Pharmacokinetics of Delafloxacin

Parameters	450 mg Tablet (single dose)	450 mg Tablet (SS) Q12H	300 mg IV (single dose)	300 mg IV (SS) Q12H
Absorption				
T_{max} (h)	0.5-4.0	0.5-6.0	1.0-1.2	1.0-1.0
C_{max} (mcg/mL)	7.17	7.45	8.94	9.29
AUC (mcg·h/mL)	22.7	30.8	21.8	23.4
Distribution				
Protein binding	84%			
V_d	30-48 L			
Metabolism				
Glucuronidation	1% of total parent drug: UGT1A1, UGT1A3, UGT2B15			
Elimination				
CL (L/h)	—	—	5.9	6.7
Renal	~50%	—	~65%	—
Feces	~48%	—	~28%	—

AUC = area under the curve; **CL** = clearance; **C_{max}** = maximum concentration; **h** = hour; **IV** = intravenous' **L** = liter; **mcg** = microgram; **mg** = milligram; **mL** = milliliter; **Q12H** = every 12 hours; **SS** = steady state; **T_{max}** = time to maximum concentration; **UGT** = UDP Glucuronosyltransferase; **V_d**= volume of distribution;

Table 3 | Summary of Clinical Trials for Delafloxacin

Trial	Interventions	Primary Endpoint	Outcome Results	Outcome Difference; (95% CI)
Phase 2 Kingsley et al.[17]	• DLX 300 mg IV Q12H (n=81) • LZD 600 mg IV Q12H (n=77) • VAN 15 mg/kg IV[a] (n=98)	Investigator assessment of clinical cure at follow-up (5-14 days of treatment)[b]	DLX vs LZD: • cure rate 70.4% vs 64.9% DLX vs VAN: • cure rate 70.4% vs 54.1%	DLX vs LZD: • –5.4% (-20.0%, 9.1%) DLX vs VAN: • -16.3% (-30.3%, -2.3%)
PROCEED[18]	• DLX 300 mg IV Q12H (n=331) • VAN 15 mg/kg IV[a] + AZT 2 g IV Q12H (n=329)	Objective cure response at 48 to 72 h after treatment initiation[c] (5-14 days of treatment)	DLX vs VAN/AZT: 78.2% vs 80.9%	-2.6% (-8.79%, 3.57%)
Phase 3 O'Riordan et al.[16]	• DLX 300 mg IV Q12H x 3 days, then DLX 450 mg PO Q12H (n=423) • VAN 15 mg/kg IV[a] + AZT 2 g IV Q12H (n=427)	Objective cure response at 48 to 72 h after treatment initiation[c] (5-14 days of treatment)	DLX vs VAN/AZT: 83.7% vs 80.6%	3.1% (-2.0%, 8.3%)

a: Vancomycin levels were monitored in blood samples drawn from all patients on day 2 or 3 and on day 6+1; dosing was adjusted based on achieving a target trough of 15 –20 mg/mL.
b: clinical response categorazied based on ABSSSI signs and symptoms: cure (complete resolution), improved (near resolution, no additional antibiotics required), failure (additional non-study antibiotics required), or intermediate (assessment was incomplete). Signs and symptoms evaluated: fever ≥38 °C, lymphangitis, white blood cell count ≥15000 cells/mm3 or serum CRP level >5.0 mg/L.
c: Defined as ≥20 % reduction in erythema of lesion without evidence of clinical failure
95% CI = 95% confidence interval; **DLX** = delafloxacin; **g** = gram; **IV** = intravenous; **kg** = kilogram; **LZD** = linezolid; **mg** = milligram; **PO** = by mouth; **Q12H** = every 12 hours; **VAN** = vancomycin

Phase 2 Trial

The purpose of the first phase 2, multicenter, stratified, randomized, double-blind clinical trial for delafloxacin was to evaluate the efficacy, safety, and tolerability of IV delafloxacin in comparison to vancomycin and linezolid for the treatment of ABSSSIs.[17] Patients were randomized to receive IV dosing of either delafloxacin 300 mg (every 12 ± 1 hour), linezolid 600 mg (every 12 ± 1 hour), or vancomycin 15 mg/kg (dose varied for a goal trough between 15 to 20 mcg/mL) for 5 to 14 days, per investigator judgment. The comparator medications were chosen based off of FDA and Infectious Disease Society of America (IDSA) recommendations, in which both are used to treat suspected infections caused by MRSA. Additional coverage for gram-negative pathogens using aztreonam was added for diagnosed/presumptive gram-negative infection.[17]

Inclusion criteria for this study were age ≥18 years, a diagnosis of ABSSSI (cellulitis/erysipelas, wound infection, major cutaneous abscess or burn infection) with ≥75 cm^2 of erythema/induration determined by planimetry plus lymph node enlargement or one of the following: fever ≥38ºC, lymphangitis, white blood cell count ≥15000 cells/mm^3, or CRP >5 mg/L. Exclusion criteria consisted of patients with hypersensitivities/allergies to the study medications; other skin conditions at infection site; severely inadequate arterial blood supply to limb containing ABSSSI; severely immunocompromised patients; hypertension (≥180 mmHg systolic or ≥110 mmHg diastolic); use of effective systemic antibiotic therapy for >24 h within 14 days before study enrollment unless objective evidence of documented clinical progression; and use of more than one dose of an antibiotic potentially effective against the ABSSSI under study within 24 hours before study entry.[17]

The primary efficacy endpoint of the study was the investigator assessment of clinical cure response rate at the follow-up visit (day 14 ± 1 and ≥12 h after the final study drug dose) in the intention-to-treat (ITT) population, which was defined as all randomized patients. Cure rate was defined as the percentage of cures only in each treatment group. In addition, secondary efficacy endpoints were analyzed, including objective assessments measuring the total of erythema and induration at 12-hour intervals through day 5 at follow-up. Cessation of erythema/induration expansion and resolution of fever was considered clinical success at 48 to 72 hours after start of treatment. Safety assessments were also performed using non-directed questions for adverse event reporting, physical examinations, vital sign measurements, 12-lead electrocardiograms, and clinical laboratory tests.[17]

A total of 256 patients were randomly assigned to one of three study medications in a 1:1:1 ratio. Baseline characteristics were similar across all treatment groups. Of the 175 patients with identified pathogens known to cause ABSSSI, 90.9% isolates were *S. aureus* (67.2% were confirmed MRSA infections). At follow-up (day 14 ±1 and ≥12 hours after final drug dose was given), there was no difference in the primary efficacy endpoint (clinical cure rate) between delafloxacin 300 mg IV every 12 hours and linezolid 600 mg IV every 12 hours, 70.4% and 64.9% respectively (mean difference: −5.4%; 95% CI, −20.0% to 9.1%; P=0.496). The primary efficacy endpoint for vancomycin 15 mg/kg IV was 54.1%, a lower clinical cure rate than delafloxacin (mean difference compared to delafloxacin = −16.3%; 95% CI, −30.3% to −2.3%; P= 0.031). This cure rate may be driven partially by better outcomes observed in obese patients in delafloxacin arm. A post hoc analysis on obese patients showed a higher cure rate in the delafloxacin group versus the vancomycin group (78.8% versus 48.8%; mean

difference: −30.0%; 95% CI, −50.7% to −9.3%; P=0.009). In the trial, vancomycin was dosed correctly as demonstrated in the reported trough levels and free-drug AUC:MIC. A high percentage of patients on vancomycin were classified as cured or improved with an AUC:MIC ratio of ≥200, suggesting sufficient vancomycin exposure.[17]

Cessation of spread of erythema/induration and the proportion of patients who had ≥20% reduction in erythema at 48 to 72 hours for each treatment group was not statistically significant. However, the proportion of patients with ≥20% reduction in induration was higher for those in the vancomycin group versus the delafloxacin group (P = 0.089). In addition, cure rates were comparable among delafloxacin, linezolid, and vancomycin groups for MRSA infections (65.5%, 61.8%, and 65.6%, respectively). The use of prior antibiotics did not show to have a clinical impact on efficacy outcomes. Safety data showed that nausea, diarrhea, and vomiting was more common in delafloxacin group, with incidences of 21.8%, 15.4%, and 12.8%, respectively. Pruritus was more common in vancomycin arm (20.8%) than in the delafloxacin arm (7.7%).[17]

Phase 3 Trials

The objective of the phase 3 trial by Pullman et al. was to evaluate the clinical efficacy and safety of delafloxacin monotherapy compared with vancomycin plus aztreonam for the treatment of ABBSSI. The study was a multicenter, stratified, randomized, double-blind, non-inferiority trial where patients were to receive either IV delafloxacin 300 mg every 12 hours or vancomycin 15 mg/kg (goal trough of 15 to 20 mcg/mL) plus aztreonam 2 g every 12 hours. Duration of therapy was 5 to 14 days, depending on investigator's clinical assessment of the patients' signs and symptoms. Aztreonam was discontinued if cultures were negative for gram-negative pathogens. However, it is unknown if vancomycin was discontinued if cultures were negative for gram-positive (including MRSA) pathogens.[18]

Inclusion criteria were age ≥18 years and diagnosed ABSSSI (cellulitis/erysipelas, wound infection, major cutaneous abscess, or burn infection) with erythema ≥75 cm² and at least two systemic signs of infection. Exclusion criteria for the study were receipt of systemic antibiotics 14 days prior to enrollment unless one of the following was documented: patient received at least 48 hours of antibiotic therapy for a diagnosis of ABSSSI and clinical progression was documented, the patient completed a treatment course within 7 days for an infection other than ABSSSI with antibiotic that has no activity against bacterial pathogens that cause ABSSSI, and patients who received one dose of a single, potentially effective short-acting antibiotic for the treatment of the ABSSSI under study in the 14 days before study entry. The exhaustive list of inclusion and exclusion criteria are available in the trial's supplementary articles. Patients were seen on the day of screening, every day while on treatment, at follow-up (day 14 ± 1), and late follow-up (day 21 to 28). Adverse event data and use of post-treatment medications was obtained via telephone follow-up on day 30 after the last dose of study drug was given.[18]

The primary efficacy endpoint defined by the FDA was objective response at 48 to 72 hours after treatment initiation, defined as proportion of patients achieving ≥20% reduction in erythema of lesion without evidence of clinical failure. An additional European Medicine Agency (EMA)-defined primary efficacy measure was an investigator assessment evaluating clinical cure, or no additional signs/symptoms, during the follow-up visit in the ITT population, which included all patients randomly assigned to a treatment. A secondary endpoint was success of therapy at the follow-up visit, defined as cure or no rescue antibiotic needed. Safety outcomes were also assessed by documenting all reported adverse reactions, providing physical examinations, obtaining vital signs, 12-lead ECGs at baseline (and if clinically indicated thereafter), and clinical laboratory tests. Treatment-emergent AEs (TEAEs) were also documented through telephone follow-up, defined as adverse effects that occurred or worsened after first dose administration.[18]

Table 4 | Common AEs with Delafloxacin[15,16,18]

Adverse Event	Incidence[a] (N=741)
Nausea	8%
Diarrhea	8%
Headache	3%
Transaminase Elevation[b]	3%
Vomiting	2%

a: Incidence value for adverse events that occurred in ≥2% of the pooled adult phase 3 clinical trial population.
b: Includes increased ALT and AST.
AE = adverse events

Patients were randomized to receive either delafloxacin (n=331) or vancomycin plus aztreonam (n=329), with 660 patients in the ITT analysis and 650 in the safety analysis. Baseline characteristics were similar among both treatment groups. For the FDA-defined primary outcome, delafloxacin was non-inferior to vancomycin plus aztreonam at 78.2% versus 80.9%, respectively (difference = -2.7%; 95% CI, -8.78% to 3.57%). The investigator-assessed cure/success rates for both groups were similar for all four infection types. In addition, similar to the phase 2 study (Kingsley et al.), cure rates at late follow-up were higher among obese patients in the delafloxacin group (71.7%) compared to the vancomycin plus aztreonam group (57.4%) (difference = 14.3%; 95% CI, 1.34% to 26.9%). At follow-up, unlike the phase II trial, the cure rates were not significantly different between the two groups for obese patients (difference = 12%; 95% CI, -1.54% to 25.08%).[18]

TEAEs were reported for both groups, in which most were mild and unrelated to the study antibiotic. In the delafloxacin group, gastrointestinal TEAEs were amongst the most reported, specifically nausea and diarrhea. There were no cases of *C.difficile*, tendonitis, tendon rupture, peripheral neuropathy, myopathy, QT interval prolongation, or phototoxicity related to delafloxacin treatment. Vital signs, physical examinations, and ECGs were unremarkable in both groups.[18]

As mentioned previously O'Riordan et al. conducted a phase 3 trial that has yet to be published however some information is available which we will discuss now. This trial was multicenter, randomized, double-blinded study to evaluate the clinical efficacy of delafloxacin for both IV and oral formulations. Similar to the previous phase 3 trial, the inclusion criteria consisted of patients diagnosed with an ABSSSI, such as cellulitis/erysipelas, wound infection, major cutaneous abscess, or burn infection, with erythema ≥75 cm² and at least two systemic signs of infection. Patients were randomized 1:1 to receive either delafloxacin 300 mg IV for three days and then switched over to 450 mg oral tablet (n = 423) or vancomycin 15 mg/kg with aztreonam (n = 427) for 5 to 14 days. The primary efficacy endpoint was response at 48 to 72

hours. Response at 48 to 72 hours was not significantly different between delafloxacin (83.7%) and vancomycin plus aztreonam (80.6%) (difference = 3.1%; 95% CI, -2.0% to 8.3%). The most common adverse events experienced with delafloxacin treatment were mild nausea and diarrhea. Please see **Table 4** for the pooled adverse event data from both phase 3 trials.[15,16,18]

Adverse Events and Precautions

The most common adverse reactions reported with delafloxacin and ≥2% incidence include nausea, vomiting, diarrhea, headache, and elevations in transaminase (see **Table 4**).[15,16,18] In addition, fluoroquinolones are associated with Black Box Warnings, including tendonitis, tendon rupture, peripheral neuropathy, central nervous system effects (hallucinations, anxiety, depression, insomnia, severe headaches, and confusion), and exacerbations of myasthenia gravis. Other serious adverse reactions associated with fluoroquinolone use include *Clostridium difficile*-associated diarrhea and the development of drug-resistant bacteria. Although these events were not reported with the use of delafloxacin, it would be prudent to consider the adverse event risk similar to other fluoroquinolones until more clinical data is available. Delafloxacin carries the same Black Box Warnings as other fluoroquinolones. Contraindications include known hypersensitivity to delafloxacin or any other fluoroquinolone antibiotic, or any of the components of delafloxacin.[15]

Dosing and Administration

Delafloxacin is indicated for the treatment of ABSSSIs that are caused by susceptible bacteria and should only be used if infection is suspected to be caused by bacteria. Administer delafloxacin 300 mg via IV infusion over 60 minutes every 12 hours, or 450 mg via oral route every 12 hours for 5 to 14 days. Renal adjustment is based on estimated glomerular filtration rate (eGFR). For eGFR of 30 to 89 mL/min/1.73m^2: no dosage adjustments (oral and IV); eGFR 15 to 29 mL/min/1.73m^2: no dosage adjustments (oral), 200 mg every 12 hours (IV); End State Renal Disease (ERSD) <15 mL/min/1.73m^2, including hemodialysis: use not recommended. In renal impairment, closely monitor serum creatinine levels in patients with severe renal impairment receiving IV delafloxacin (eGFR 15 to 29 mL/min/1.73 m^2).[15,19] If serum creatinine begins to increase, consider changing to oral dosage form due to the seen accumulation of sulfobutylether-β-cyclodextrin (SBECD), a vehicle located in the intravenous formulation.[15]

In addition, oral delafloxacin can be taken with or without food. If there is a missed dose, it should be taken as soon as possible up to 8 hours prior to the next scheduled dose. Skip the dose if it is less than 8 hours before the next dose. Delafloxacin can be taken 2 hours before or 6 hours after antacids containing magnesium/aluminum, sucralfate, metal cations (iron), multivitamin preparations containing zinc or iron, or with didanosine buffered tablets for oral suspension or the pediatric powder for oral solution. For IV delafloxacin, do not administer with solutions containing multivalent cations through the same IV line.[15]

Cost

Information regarding wholesale prices of delafloxacin IV solution are not readily available to the public. A supply of 14 Delafloxacin tablets (Baxdela®) are sold at an average retail price of $1000.00. The manufacturer, Melinta Therapeutics™, offers patient assistance programs for patients with commercial insurance. Patients with commercial prescription insurance are eligible for a copay between $4-75 per prescription. Patients with public or private third party payer, or any federal or state healthcare program are not eligible for the manufacturer savings.[15,20]

Conclusions

Delafloxacin is a new fluoroquinolone that appears to be an acceptable antibiotic choice for the treatment of ABSSSIs based on the trials. This medication has shown to be non-inferior to other broad spectrum antibiotics (vancomycin plus aztreonam), and has exhibited pathogen coverage for MRSA. With emerging concerns for antibiotic resistance, delafloxacin provides an additional option for MRSA infections that are resistant to other broad spectrum antibiotics. Delafloxacin is generally well-tolerated by patients, exhibiting mild GI side effects, and can be used in patients with moderate renal impairment. Since this medication is available in both IV and oral formulations, it provides an easy transition from inpatient to outpatient antibiotic therapy. In addition, secondary trial endpoints indicate delafloxacin may have higher cure rates in obese patients than those treated with vancomycin. Altogether, the studies found that delafloxacin was safe and statistically non-inferior to vancomycin plus aztreonam in treating ABSSSIs, providing an additional option for treatment of ABSSSIs.

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