KU DRUG INFORMATION CENTER School of Pharmacy

# New Drugs in Pharmacy Practice

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**EDUCATIONAL OBJECTIVES**

After completion of this activity, the participant will be able to:

1. Identify the FDA approved indication, dosage and administration, and mechanism of action for each of the new drugs.
2. Provide counseling points pertinent to each new product and recognize the appropriate monitoring parameters for each agent.
3. Understand where the new drugs may fit into current practice based on the characteristics of the products

**TARGET AUDIENCE**: Pharmacists
**ESTIMATED TIME TO COMPLETE:** 1.5-2 hours
**FEE**: Available for free at www.pharmacy.ku.edu/dic-services

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This activity has been approved by the Kansas State Board of Pharmacy for 1.5 contact hours (0.15 CEUs) under the course number 17-00001. The activity is available for CE credit through December 31, 2017.

## Exondys 51™ (eteplirsen)

### Introduction

Duchenne Muscular Dystrophy (DMD) is a rare genetic disorder that affects one in 3600 male infants globally.[1] DMD is caused by mutations in the dystrophin gene which leads to an absence or deficiency in the dystrophin protein responsible for keeping muscle cells intact.[1,2] The lack of dystrophin results in muscle degeneration and may progress to respiratory, orthopedic, and cardiac complications.[2] Exondys 51 (eteplirsen) was approved on September 19, 2016, through the accelerated approval process as the first drug to treat DMD correctable to exon 51 skipping which allows for production of functional dystrophin protein.[2,3]

### Indication

Eteplirsen is indicated to treat DMD in patients with a mutation of the DMD gene amenable to exon 51 skipping. A clinical benefit of eteplirsen has not been shown; therefore, confirmatory trials be may be necessary for continued approval of eteplirsen.[4]

### Mechanism of Action

Eteplirsen binds to exon 51 of dystrophin pre-mRNA which excludes the exon during mRNA processing. The exclusion or skipping of this exon during mRNA processing results in production of an internally truncated (or shortened) and functional dystrophin protein.[4]

### Dosage and Administration

Eteplirsen is available in two different single-dose vial concentrations: 100 mg/2 mL (50 mg/mL) solution and 500 mg/10 mL (50 mg/mL) solution. The recommended dose is 30 mg/kg administered as an intravenous infusion once weekly over 35 to 60 minutes. If a dose of eteplirsen is missed, it may be administered as soon as possible after the scheduled time. Eteplirsen does not contain preservatives and should be administered immediately after dilution and infusion of diluted eteplirsen should be completed within 4 hours after dilution. Eteplirsen is not recommended to be co-administered with other medications in the same intravenous access lines.[4] (*continued on page 2)*

## Inside this issue



## To Obtain CE Credit

- After reading the content of this newsletter in its entirety, go to www.pharmacy.ku.edu/dic-services to access the post test online.
- You will be awarded 1.5 hours of CE after achieving a 80% or higher pass rate on the post test.
- A certificate will be available for download and print after successfully passing the post test.
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## Highlights-Exondys 51

Manufacturer: Sarepta Pharmaceuticals

Approval Date: September 19, 2016

Pronunciation: e TEP lir sen

Exondys 51 was granted orphan status and approved through the accelerated pathway.

The product is an antisense oligonucleotide of the phosphorodiamidate morpholino oligomer subclass.

A clinical benefit has not been established; therefore, continued approval of Exondys 51 will depend on verification of a clinical benefit in confirmatory trials.

The physician must initiate a statement of medical necessity and other forms in order for the patient to obtain the medication.

*[Exondys 51, continued from page 1]*

### Efficacy and Safety

The safety and efficacy of eteplirsen were evaluated in two randomized, double-blind, placebo and historical controlled trials in patients with confirmed DMD correctable to exon 51 skipping.[3] Males (7 to 13 years of age) previously taking glucocorticoids for at least 24 weeks and were able to walk 200-400m on the 6 minute walking test (6MWT) were included in the studies. In Study 201, a total of 12 patients were randomized to receive weekly intravenous eteplirsen at 30mg/kg (n=4), eteplirsen at 50mg/kg (n=4) or placebo (n=4) for 24 weeks. The participants then transitioned into Study 202, the open label extension study, in which all 8 patients continued with weekly intravenous treatments at the same dose and placebo-treated patients were randomized to receive either 30 mg/kg or 50 mg/kg for 36 months.[3,5]

The primary endpoint of study 201 was dystrophin production based on muscle biopsies at week 12 for the 50 mg/kg cohort, at week 24 for the 30 mg/kg cohort and placebo, and at week 48 for all patients. The study also included an ambulatory functional endpoint assessed by a 6-Minute Walking Test (6MWT).[3] The dystrophin-positive fibers in the 50mg/kg group at 12 weeks did not have a significant difference compared to placebo. The dystrophin-positive fibers in the 30 mg/kg group at week 24 had a statistically significant change of 22.9% compared to the placebo group. At week 48, the 30 mg/kg and 50 mg/kg group had a statistically significant increase of 47.3% in dystrophin fibers and the four patients that were later randomized to 30 mg/kg or 50 mg/kg had an increase of 37.7%. The comparison of all the cohorts and the placebo/delayed cohorts did not show a significant difference in dystrophin fibers. Patients treated with eteplirsen had a 67.3 meter(m) advantage on the 6MWT at week 48 compared to the placebo/delayed group with the exclusion of two patients that had rapid progression of disease in the 30 mg/kg group. There were no treatment related adverse events reported in this study.[3]

In Study 202, the primary endpoint was 6MWT and the secondary endpoint was pulmonary function testing (PFT) compared to a historical control. At months 12 and 24, the comparison of the eteplirsen group to the historical control cohort did not show a statistically significant difference in 6MWT distances. At 36 months, the eteplirsen group had a significant difference in 6WMT decline compared to the historical control cohort with a difference of 151 m. Two of the 12 patients (16.7%) in the eteplirsen group lost ambulation compared to six of the 13 (46.2%) patients in the historical control cohort. The eteplirsen group showed a decrease in the decline of all three pulmonary function components (mean percentage of predicted maximum inspiratory pressure, mean percentage of predicted maximum expiratory pressure, and mean percentage of predicted forced vital capacity) compared to values found in natural history of patients with DMD.[5]

There were no reports of systemic reactions and no serious adverse events related to treatment. The most frequently reported adverse events while taking eteplirsen were headache, procedural pain related to biopsy and catheter placement, and proteinuria. There were no deaths reported.[5] Preliminary data from a third study showed a mean increase of 0.28% in dystrophin levels at 48 weeks of treatment in 12 patients treated with eteplirsen compared to pre-treatment levels.[4] The study is ongoing and will enroll more than 160 patients for evaluation up to 96 weeks in patients with DMD.[6] Note: Information regarding Study 201 and 202 may differ from the package insert as the FDA excluded clinical efficacy data they found to not be substantial evidence of efficacy.

### Adverse Effects

The most common adverse events reported were balance disorder and vomiting with an incidence greater than 35% and were higher in the treatment group compared to placebo.[4]

### Drug Interactions

Eteplirsen did not significantly inhibit the major CYP enzymes based on in vitro studies.

[Exondys 51, continued from page 2]

Eteplirsen did not significantly induce CYP2B6, CYP3A4, or CYP1A2. Additionally, eteplirsen was not a substrate for or inhibitor of any of the major human transporters (such as OAT1, BCRP, P-gp, etc). Eteplirsen is expected to have a low potential for drug-drug interactions.[4]

### Contraindications

None.[4]

### Special Populations

*Pregnancy:*

Currently, there are no studies in human or animals evaluating the use of eteplirsen during pregnancy.[4]

*Lactation*

There are no data in humans or animals that assessed eteplirsen and its effects on breast milk production, its presence in breast milk, or effects on the breastfed infant.[4]

*Pediatric*

Eteplirsen is indicated for use in pediatric patients with a confirmed mutation of DMD gene amenable to exon 51 skipping. In juvenile male rats, there were no effects on the male reproductive system, neurobehavioral development or immune function.[4]

*Geriatric*

There is no information in the geriatric population as eteplirsen was studied in male pediatric patients and the disease predominantly affects children and young adults.[4]

*Renal or Hepatic Impairment*

No studies have been conducted to assess the use of eteplirsen in renal or hepatic impairment.[4]

### Monitoring Parameters/Counseling Points

Use of a topical anesthetic cream at the infusion site may be considered prior to administration of eteplirsen.4 If there are signs of an allergic reaction such as hives, itching, shortness of breath, wheezing, swelling of the face, lips, tongue or throat, contact a healthcare provider. Some side effects of this medication include nausea, upset stomach, irritation or pain at the injection site, bruising, balance disorder, or symptoms of common cold. Ensure healthcare providers are aware of prescription, over-the counter-medications, vitamins, natural supplements taken concomitantly. Monitor for increased levels of skeletal muscular dystrophin to assess efficacy.[7]

*Hypertension affects approximately 72 million people. It is a risk factor for MI, heart failure, stroke and cardiovascular diseases.*

# Byvalson™ (nebivolol/valsartan)

### Introduction

Hypertension affects approximately 72 million people and is a risk factor for myocardial infarction, heart failure, stroke and cardiovascular disease.[8,9] Control of high blood pressure has been shown to greatly reduce the risk of these events.[10] Monotherapy is the standard initial treatment and may be increased if blood pressure goals are not met. Combining drugs from different classes may be more effective in controlling blood pressure than increasing the dose of one drug alone.[11] On June 6, 2016, the FDA approved Byvalson (nebivolol/valsartan), for the treatment of hypertension. Byvalson is the first and only fixed-dose combination of a beta blocker and angiotensin II receptor blocker available on the market.[12]

### Indication

Nebivolol/valsartan is approved to treat hypertension.[13]

### Mechanism of Action

Nebivolol is a beta-adrenergic receptor blocking agent that has beta-1 selectivity in extensive metabolizers and at doses $\leq$ 10 mg. At higher doses and in poor metabolizers, nebivolol inhibits both beta-1 and beta-2 adrenergic receptors. The blood pressure lowering effect of nebivolol has not been definitively established, but may be due to its ability to decrease heart rate, myocardial contractility, sympathetic activity, and peripheral vascular resistance, while suppressing renin activity, and causing vasodilation.[13]

Valsartan is an angiotensin II, type 1 receptor blocking agent that exhibits antihypertensive properties by inhibiting the negative feedback mechanism of angiotensin II on renin secretion. The inhibition of renin secretion blocks both vasoconstriction and aldosterone secretion which subsequently lowers blood pressure.[13] (*continued on page 4)*

### Highlights-Byvalson

Approval Date: June 6, 2016

Manufacturer: Allergan

Byvalson is the first and only beta blocker and angiotensin II receptor blocker fixed-combination therapy available on the market.

Byvalson may be substituted in patients already receiving 5mg nebivolol and 80mg valsartan.

Byvalson is available as a purple, capsule-shaped tablet which contains the Opadry® II Purple film-coat. The Opadry® II is made of polyvinyl alcohol-part hydrolyzed, titanium dioxide, talc, polyethylene glycol, iron oxide red, and ferrosoferric oxide/black iron oxide.

*[Byvalson, continued from page 3]*

### Dosage and administration

Nebivolol/valsartan is available as a 5 mg nebivolol/80 mg valsartan oral tablet. The recommended starting dose is one tablet 5mg/80mg nebivolol/valsartan once daily in patients not on 80 mg valsartan or up to and including 10 mg of nebivolol. The maximum therapeutic effect can appear within 2 to 4 weeks of initiation. Increasing the dose of nebivolol/valsartan does not result in further lowering the blood pressure. Nebivolol/valsartan can be used as monotherapy or in combination with other blood pressure lowering agents.[13]

### Efficacy and Safety

A Phase 3, randomized, double-blind, multi-center, placebo-controlled trial evaluated the safety and efficacy of nebivolol/valsartan in patients with type 1 or 2 hypertension. A total of 4118 patients (18 years of age or older) were randomly assigned to one of the following groups: nebivolol/valsartan 5/80mg (n=555), nebivolol/valsartan 5/160mg (n=555), nebivolol/valsartan 10/160mg (n=555), nebivolol 5mg (n=555), nebivolol 20mg (n=555), valsartan 80mg (n=555), valsartan 160mg (n=555), or placebo (n=277) daily for 4 weeks. Doses were doubled in the respective groups in weeks 5-8 and results were analyzed based on the final dose.[14]

The primary efficacy endpoint evaluated the difference in trough seated diastolic blood pressure at baseline to week 8. The key secondary efficacy endpoint evaluated the difference in seated systolic blood pressure at week 8. The following additional secondary efficacy endpoints were also assessed in the study: changes from baseline in mean trough seated diastolic and systolic blood pressure at week 4, changes in 24 hour ambulatory blood pressure monitoring (ABPM) values for diastolic and systolic blood pressure from baseline to week 8 in the fixed-dose combination of 20/320mg daily compared to nebivolol 40mg daily and 320 mg valsartan daily; diastolic blood pressure responder rates at week 8 at levels less than 90 mmHg and less than 80 mmHg, and systolic blood pressure responder rates (<140mmHg and <130mmHg) at week 8; changes from baseline in mean trough seated diastolic and systolic blood pressure at each visit, changes in pulse rate at week 8, and proportions of participants achieving treatment goal (blood pressure of 140/90 mmHg without type 2 diabetes or 130/80 mmHg with type 2 diabetes) at weeks 4 or 8.[14]

The fixed-dose 20/320mg group showed significantly greater reductions in diastolic blood pressure than the nebivolol 40mg and valsartan 320mg groups and the fixed-dose combinations had a greater reduction compared to the monotherapy groups. The result was found to be similar for the systolic blood pressure from baseline to week 8. At Week 4, the fixed dose 5/80mg and 5/160mg groups had a statistically significant reduction in diastolic and systolic blood pressure compared to the respective monotherapy groups (nebivolol 5 mg/day, valsartan 80 mg/day, valsartan 160mg). The fixed-dose combination 5/160mg compared to valsartan 160mg did not show a significant difference in systolic blood pressure. For the 24 hour ABPM substudy, the fixed-dose 20/320mg group showed significantly greater reductions in 24 hour diastolic and systolic blood pressure at week 8 than the valsartan 320mg group but not the nebivolol 40mg group. Blood pressure control rates at week 8 were significantly greater in the 20/320mg group compared to both nebivolol 40mg and valsartan 320mg. Patients in the fixed-dose groups and monotherapy groups had similar pulse rates at week 8. There was no significant difference in pulse rate observed in patients treated with fixed-dose combinations versus all corresponding nebivolol monotherapies or in the valsartan group compared to placebo. Proportion of patients achieving treatment goal was not reported in the journal article.[14] The most common treatment emergent adverse events were sinus bradycardia, bradycardia, nausea, fatigue, nasopharyngitis, upper respiratory tract infection, prolonged ECG QT, headache, and dizziness.[15] There were two deaths reported considered to be unrelated to the study drug in the single run-in phase. There were no deaths reported in the double-blind treatment phase.[14]

**Table 1. Post-marketing adverse events**

| | |
|---|---|
| **Nebivolol** | Atrioventricular block (both second and third degree), myocardial infarction, somnolence, syncope, vertigo<br>Raynaud's phenomenon, peripheral ischemia/claudication, thrombocytopenia, pruritus, psoriasis, various rashes and skin disorders, vomiting abnormal hepatic function (including increased AST, ALT and bilirubin), hypersensitivity (including urticaria, allergic vasculitis and rare reports of angioedema), acute renal failure, acute pulmonary edema, bronchospasm, erectile dysfunction |
| **Valsartan** | Hypersensitivity (angioedema). elevated liver enzymes, hepatitis, impaired renal function, renal failure, hyperkalemia, alopecia, bullous dermatitis, thrombocytopenia, vasculitis |

*[Byvalson, continued from page 4]*

## Adverse Events

The incidence of adverse events was equal in patients on nebivolol/valsartan and placebo. Serious adverse events observed with use of this drug were hypotension and hyperkalemia. Moderate adverse events reported included bradycardia, nausea, fatigue, nasopharyngitis, upper respiratory tract infection, prolonged ECG QT interval, headache, and dizziness.[13]

## Drug Interactions

There are currently no studies evaluating drug interactions with the combination nebivolol/valsartan; however, drug interactions with the individual components have been noted.[13]

Nebivolol: Concomitant use with CYP2D6 inhibitors and other beta-blocking agents should be avoided. Patients should be monitored while receiving catecholamine-depleting drugs such as reserpine or guanethidine, digitalis glycosides, and calcium channel blockers.[13]

Valsartan: Due to the potential increase in serum potassium levels with RAAS inhibitors, potassium sparing diuretics, potassium supplements, and potassium-containing salt substitutes, patients should be monitored during therapy. Concomitant use with non-steroidal anti-inflammatory agents may lead to possible acute renal failure or decrease in renal function. Patients treated with valsartan and ACE-inhibitors concomitantly should have their blood pressure, renal function and electrolytes monitored. Serum lithium levels should be closely monitored during concomitant use due to an increase in lithium concentrations and toxicity. Nebivolol/valsartan should not be administered in patients taking aliskiren who have diabetes or renal impairment with a GFR less than 60 mL/min.[13]

## Contraindications

Nebivolol/valsartan is contraindicated in patients who have severe bradycardia, heart block greater than first degree, cardiogenic shock, decompensated heart failure, sick sinus syndrome (unless a permanent pacemaker is in place), severe hepatic impairment (Child-Pugh score >B), diabetic patients currently taking aliskiren, and/or hypersensitivity to any component of the product.[13]

## Special Populations

*Pregnancy*

Use of nebivolol/valsartan should be avoided in pregnant women, especially those in the second and third trimesters. Animal studies have shown that valsartan causes fetal toxicity at doses considered toxic to the mother. As for nebivolol, animal studies have demonstrated fetal and perinatal death at doses equivalent to the maximum dose recommended in humans. The product should be discontinued if pregnancy is suspected. [13]

*Lactation*

It is unknown whether nebivolol/valsartan or its individual components is excreted into breastmilk of humans, has an effect on a breastfed infant, or \effect on milk production. Nebivolol/valsartan should not be used in breastfeeding women due to the potential for serious adverse reactions (such as bradycardia) in nursing infants and postnatal renal development. [13]

*Pediatric*

Use off nebivolol/valsartan has not been established in this population. [13]

*Geriatric*

Studies do not show a difference in clinical response between younger and elderly patients. However, elderly patients may experience greater sensitivity to the medication. [13]

*Renal Impairment*

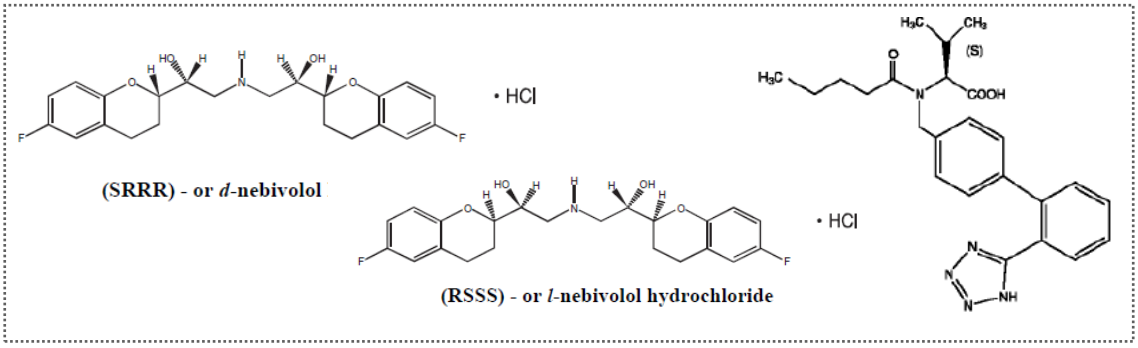
Patients with mild to moderate renal impairment do not require dose adjustments while using nebivolol/valsartan; but the safety and effectiveness in patients with moderate to severe renal impairment (creatinine clearance of less than or equal to 60 mL/min) has not been studied. This product is not recommended as initial treatment in patients with severe renal impairment.[13]

*Hepatic Impairment*

Nebivolol/valsartan has not been studied in patients with hepatic insufficiency. There are no dose adjustments required in patients with mild hepatic impairment. The use of nebivolol/valsartan is not recommended in moderate hepatic impairment or severe hepatic impairment.[13]

## Monitoring parameters/ counseling points

Nebivolol/valsartan should not be discontinued abruptly in patients with coronary artery disease as it can worsen angina or cause myocardial infarction and ventricular arrhythmias. Hypotension, which typically presents as lightheadedness, may occur during the first days of therapy. Patients should notify their physician if they experience syncope and discontinue using the medication until the physician has been consulted. Due to the risk of hyperkalemia while taking nebivolol/valsartan, patients should discuss taking salt substitutes containing potassium. Nebivolol/valsartan should not be taken by women who are pregnant or nursing.[13]



# Zinplava™ (bezlotoxumab)

**Highlights-Zinplava**

Approval Date: October 21, 2016

Pronuniciation: bez loe TOX ue mab

Manufacturer: Merck

Zinplava is a monoclonal antibody approved to decrease recurrence of CDI and is not indicated to treat CDI.

The product is available as a 1,000mg/40mL single dose vial and must be infused over 60 minutes.

Zinplava is available through specialty pharmacies and select pharmacies.

The most common reported adverse events included nausea, pyrexia and headache.

## Introduction

Clostridium difficile infection (CDI) is the most common cause of infectious diarrhea in hospitalized patients.[16] In 2011, there were approximately half a million clostridium difficile infections with 83,000 patients that had a recurrence and 29,000 patients died within 30 days of first diagnosis.[17,18] Because CDI has become a health concern, several novel therapies including fidaxomicin has been developed to address prevention of CDI with many others still in the development phase.[19,20] Zinplava (bezlotoxumab) is a monoclonal antibody approved by the FDA on October 21, 2016. Zinplava is the first monoclonal antibody on the market to prevent CDI recurrence in conjunction with standard treatment.[21]

## Indication

Bezlotoxumab is approved to decrease occurrence of CDI in adult patients (>18 years of age) and those with high risk for CDI recurrence on antibacterial therapy. Bezlotoxumab should be used an adjunctive agent and not for treatment of CDI.[22]

## Mechanism of Action

Bezlotoxumab is a human monoclonal antibody that binds to C. difficile toxin B which neutralizes the toxic effects of toxin B.[22]

## Dosage and Administration

Bezlotoxumab is available in a 1,000mg/40mL (25mg/mL) single-dose vial injection. The recommended dose is 10mg/kg infused over 60 minutes. Bezlotoxumab can be infused through a peripheral or central line, but should not be given as a bolus or IV push. Other products should not be co-administered with bezlotoxumab in the same line.[22]

## Efficacy and Safety

The safety and efficacy of bezlotoxumab were evaluated in two randomized, multicenter, double-blind, Phase 3, placebo-controlled trials (Trial 1 and Trial 2) in patients ≥18 years old with a confirmed CDI diagnosis. In Trial 1, a total of 807 patients were randomized to receive a single dose of bezlotoxumab IV infusion 10mg/kg in addition to CDI standard of care treatment (metronidazole, vancomycin, or fidaxomicin) (n=403) or placebo (n=404) in addition to standard of care for 10-14 days. In Trial 2, 407 study participants received bezlotoxumab and 399 received placebo. The endpoints evaluated were clinical cure of the presenting episode of CDI, recurrence of CDI and the sustained clinical response.[22]

The clinical cure rate was lower in the treatment group compared to placebo in Trial 1 but was higher in Trial 2. A sustained clinical response was higher in the bezlotoxumab group (60.1% and 66.8%) than the placebo group (55.2% and 52.1%) in Trial 1 and 2, respectively.[22]

Heart failure was a serious adverse event reported in 2.3% of patients in the treatment group and 1% of patients in placebo. Mortality rates were 7.1% and 7.6% of bezlotoxumab and placebo patients, respectively. The most common Infusion-related reactions were nausea, headache, fatigue, pyrexia, dizziness, dyspnea, and hypertension which resolved within 24 hours. There was one patient that discontinued bezlotoxumab to ventricular tachyarrhythmia. There were no anti-bezlotoxumab antibodies identified in both Trial 1 and 2.[22]

## Adverse Events

The most common adverse events reported were headache, nausea, and pyrexia.[22]

## Drug Interactions

Bezlotoxumab is removed through the catabolic pathways; therefore, metabolic drug-drug interactions may be unlikely.[22]

*(continued on page 7)*

*[Zinplava, continued from page 6]*

**Contraindications**

None.[22]

**Special Populations**

*Pregnancy*

The use of bezlotoxumab in pregnancy has not been studied in humans or in animals.[22]

*Lactation*

It is unknown whether bezlotoxumab is present in breast milk or has an effect on the infant or an effect on milk production.[22]

*Pediatric*

Bezlotoxumab has not studied in patients below 18 years of age.[22]

*Geriatric*

Clinical studies showed that there was no difference in safety and efficacy of bezlotoxumab in patients older than 65 compared to younger; therefore, dose adjustments are not recommended.[22]

*Renal Impairment*

There was no clinically significant difference of bezlotoxumab in patients that have normal renal function and in those with renal impairment.[22]

*Hepatic Impairment*

No clinical differences were seen in patients with hepatic impairment and those that have normal hepatic functions when exposed to bezlotoxumab.[22]

**Monitoring Parameters/ Counseling Points**

Bezlotoxumab should be used in conjunction with antibacterial therapies for the prevention of recurrent CDI. Advise patients that Bezlotoxumab is not an antibiotic and does not treat CDI. It is important patients continue antibacterial treatment regimens to completion as directed by healthcare providers.[22]

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# New Drugs in Pharmacy Practice Continuing Education Newsletter

The Drug Information Center at the KU School of Pharmacy offers a newsletter with monographs of recently FDA approved drugs. The newsletter will allow for pharmacists to learn about new products available on the market and where they fit into current practice. A new issue will be published every 3 months.

The newsletter has been approved by the Kansas State Board of Pharmacy as a CE activity. After reading the newsletter, the participant may receive 1.5 hours of CE (0.15 CEUs) upon scoring 80% or higher on the online post test through December 31, 2017. The CE certificate will be available to print for submission to the Kansas State Board of Pharmacy. You must submit the certificate to the KBOP within 30 days of completing the activity in order to receive CE credit. Note that this is not an ACPE accredited CE activity; therefore, the CE will not be included in the NABP CE monitor.

You may go to www.pharmacy.ku.edu/dic-services to access the post test online. First time users will have to register with your name and additional information. Your personal information entered on the website are required information that the DIC must have on file inn order to comply with the State Board of Pharmacy requirements as a CE provider. Your personal information will not be shared with third parties.