At the first sign of failure, count on VIMPAT—a first-in-class AED for the adjunctive treatment of partial-onset seizures.

**FORGING POWER IN EPILEPSY.**

- Patients achieved greater reduction in seizure frequency
- Proven efficacy with the broadest range of AEDs
- Power that was generally well tolerated
- The first novel mechanism of action in 10 years

**AVAILABLE IN TABLETS, ORAL SOLUTION, AND IV INJECTION TO MEET MORE PATIENTS’ NEEDS.**

VIMPAT tablets and oral solution are indicated as adjunctive therapy in the treatment of partial-onset seizures in patients with epilepsy who are 17 years and older. VIMPAT injection is indicated as short-term replacement when oral administration is not feasible in these patients.

**Important Safety Information**

**Warnings and Precautions**

AEDs increase the risk of suicidal behavior and ideation. Patients taking VIMPAT should be monitored for the emergence or worsening of depression, suicidal thoughts or behavior, and/or any unusual changes in mood or behavior. Patients should be advised that VIMPAT may cause dizziness, ataxia, and syncope. Caution is advised for patients with known cardiac conduction problems, who are taking drugs known to induce PR interval prolongation, or with severe cardiac disease. In patients with seizure disorders, VIMPAT should be gradually withdrawn to minimize the potential of increased seizure frequency. Multiorgan hypersensitivity reactions have been reported with antiepileptic drugs. If this reaction is suspected, treatment with VIMPAT should be discontinued.

VIMPAT oral solution contains aspartame, a source of phenylalanine. A 200 mg dose of VIMPAT oral solution (equivalent to 20 mL) contains 0.32 mg of phenylalanine.

Dosage adjustments are recommended for patients with mild or moderate hepatic impairment or severe renal impairment. Use in severe hepatic impairment patients is not recommended.

**Common Adverse Reactions**

The most common adverse reactions occurring in ≥10 percent of VIMPAT-treated patients, and greater than placebo, were dizziness, headache, nausea, and diplopia.

Please see adjacent pages for Brief Summary of full Prescribing Information.

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References:
1. Data on file, UCB, Inc.

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The Epilepsy Company is a trademark of the UCB Group of Companies.
Dizziness and Ataxia

Patients should be advised that VIMPAT may cause dizziness and ataxia. Accordingly, they should be advised not to drive a car or to operate other complex machinery until they are familiar with the effects of VIMPAT on their ability to perform such activities.

In patients with partial-onset seizures taking 1 to 3 concomitant AEDs, dizziness was experienced by 25% of patients randomized to the recommended doses (200 to 400 mg/day) of VIMPAT (compared with 8% of placebo patients) and was the adverse event most frequently leading to discontinuation (3%). Ataxia was experienced by 6% of patients randomized to the recommended doses (200 to 400 mg/day) of VIMPAT (compared to 2% of placebo patients). The onset of dizziness and ataxia was most commonly observed during titration. There was a substantial increase in these adverse events at doses higher than 400 mg/day. [see Adverse Reactions/Table 2 (6.1)]

Cardiac Rhythm and Conduction Abnormalities

PR interval prolongation

Dose-dependent prolongations in PR interval with VIMPAT have been observed in clinical studies in patients and in healthy volunteers [see Clinical Pharmacology (12.2) in Full Prescribing Information]. In clinical trials in patients with partial-onset epilepsy, asymptomatic first-degree atrioventricular (AV) block was observed as an adverse reaction in 0.4% (4/944) of patients randomized to receive VIMPAT and 0% (0/364) of patients randomized to receive placebo. In clinical trials in patients with diabetic neuropathy, asymptomatic first-degree AV block was observed as an adverse reaction in 0.5% (5/1023) of patients receiving VIMPAT and 0% (0/291) of patients receiving placebo. When VIMPAT is given with other drugs that prolong the PR interval, further PR prolongation is possible.

VIMPAT should be used with caution in patients with known conduction problems (e.g. marked first-degree AV block, second-degree or higher AV block and sick sinus syndrome without pacemaker), or with severe cardiac disease such as myocardial ischemia or heart failure. In such patients, obtaining an ECG before beginning VIMPAT, and after VIMPAT is titrated to steady-state, is recommended.

Atrial fibrillation and Atrial flutter

In the short-term investigational trials of VIMPAT in epilepsy patients, there were no cases of atrial fibrillation or flutter. In patients with diabetic neuropathy, 0.5% of patients treated with VIMPAT experienced an adverse reaction of atrial fibrillation or atrial flutter, compared to 0% of placebo-treated patients. VIMPAT administration may predispose to atrial arrhythmias (atrial fibrillation or flutter), especially in patients with diabetic neuropathy and/or cardiovascular disease. Patients should be made aware of the symptoms of atrial fibrillation and flutter (e.g., palpitations, rapid pulse, shortness of breath) and told to contact their physician should any of these symptoms occur.

Syncope

In the short-term controlled trials of VIMPAT in epilepsy patients with no significant system illnesses, there was no increase in syncope compared to placebo. In the short-term controlled trials of VIMPAT in patients with diabetic neuropathy, 1.2% of patients who were treated with VIMPAT reported an adverse reaction of syncope or loss of consciousness, compared to 0% of placebo-treated patients with diabetic neuropathy. Most of the cases of syncope were observed in patients receiving doses above 400 mg/day. The cause of syncope was not determined in most cases. However, several were associated with either changes in orthostatic blood pressure, atrial flutter/fibrillation (and associated tachycardia), or bradycardia.

Withdrawal of Antiepileptic Drugs (AEDs)

As with all AEDs, VIMPAT should be withdrawn gradually (over a minimum of 1 week) to minimize the potential of increased seizure frequency in patients with seizure disorders.

Multigorgan Hypersensitivity Reactions

One case of symptomatic hepatitis and nephritis was observed among 4011 subjects exposed to VIMPAT during clinical development. The event occurred in a healthy volunteer, 10 days after stopping VIMPAT treatment. The subject was not taking any concomitant medication and potential known viral etiologies for hepatitis were ruled out. The subject fully recovered within a month, without specific treatment. The case was consistent with a delayed multigorgan hypersensitivity reaction. Additional potential cases included 2 with rash and elevated liver enzymes and 1 with myocarditis and hepatitis of uncertain etiology.

Multigorgan hypersensitivity reactions (also known as Drug Reaction with Eosinophilia and Systemic Symptoms, or DRESS) have been reported with other anticonvulsants and typically, although not exclusively, present with fever and rash associated with other organ system involvement, that may or may not include eosinophilia, hepatitis, nephritis, lymphadenopathy, and/or myocarditis. Because this disorder is variable in its expression, other organ system signs and symptoms not noted here may occur. If this reaction is suspected, VIMPAT should be discontinued and alternative treatment started.
Phenylketonurics

VIMPAT oral solution contains aspartame, a source of phenylalanine. A 200 mg dose of VIMPAT oral solution (equivalent to 20 mL) contains 0.32 mg of phenylalanine.

ADVERSE REACTIONS

Because clinical trials are conducted under widely varying conditions, adverse reaction rates observed in the clinical trials of a drug cannot be directly compared to rates in the clinical trials of another drug and may not reflect the rates observed in practice.

In all controlled and uncontrolled trials in patients with partial-onset seizures, 1327 patients have received VIMPAT of whom 1000 have been treated for longer than 6 months and 852 for longer than 12 months.

Clinical Trials Experience

Controlled Trials

Adverse reactions leading to discontinuation

In controlled clinical trials, the rate of discontinuation as a result of an adverse event was 8% and 17% in patients randomized to receive VIMPAT at the recommended doses of 200 and 400 mg/day, respectively, 29% at 600 mg/day, and 5% in patients randomized to receive placebo. The adverse events most commonly (>1% in the VIMPAT total group and greater than placebo) leading to discontinuation were dizziness, ataxia, vomiting, diplopia, nausea, vertigo, and vision blurred.

Most common adverse reactions

Table 2 gives the incidence of treatment-emergent adverse events that occurred in ≥2% of adult patients with partial-onset seizures in the VIMPAT total group and for which the incidence was greater than placebo. The majority of adverse events in the VIMPAT patients were reported with a maximum intensity of ‘mild’ or ‘moderate’.

Table 2: Treatment-Emergent Adverse Event Incidence in Double-Blind, Placebo-Controlled Partial-Onset Seizure Trials (Events ≥2% of Patients in VIMPAT Total and More Frequent Than in the Placebo Group)

<table>
<thead>
<tr>
<th>System Organ Class/ Preferred Term</th>
<th>Placebo N=364 %</th>
<th>VIMPAT 200 mg/day N=270 %</th>
<th>VIMPAT 400 mg/day N=471 %</th>
<th>VIMPAT 600 mg/day N=203 %</th>
<th>VIMPAT TOTAL N=944 %</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ear and labyrinth disorder</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vertigo</td>
<td>1</td>
<td>5</td>
<td>3</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td><strong>Eye disorders</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diplopia</td>
<td>2</td>
<td>6</td>
<td>10</td>
<td>16</td>
<td>11</td>
</tr>
<tr>
<td>Vision blurred</td>
<td>3</td>
<td>2</td>
<td>9</td>
<td>16</td>
<td>8</td>
</tr>
<tr>
<td><strong>Gastrointestinal disorders</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nausea</td>
<td>4</td>
<td>7</td>
<td>11</td>
<td>17</td>
<td>11</td>
</tr>
<tr>
<td>Vomiting</td>
<td>3</td>
<td>6</td>
<td>9</td>
<td>16</td>
<td>9</td>
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<tr>
<td>Diarrhea</td>
<td>3</td>
<td>3</td>
<td>5</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td><strong>General disorders and administration site conditions</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fatigue</td>
<td>6</td>
<td>7</td>
<td>7</td>
<td>15</td>
<td>9</td>
</tr>
<tr>
<td>Gait disturbance</td>
<td>&lt;1</td>
<td>&lt;1</td>
<td>2</td>
<td>4</td>
<td>2</td>
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<tr>
<td>Asthenia</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td><strong>Injury, poisoning and procedural complications</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Contusion</td>
<td>3</td>
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<td>4</td>
<td>2</td>
<td>3</td>
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<tr>
<td>Skin laceration</td>
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<td>3</td>
<td>3</td>
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<tr>
<td><strong>Nervous system disorders</strong></td>
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<tr>
<td>Dizziness</td>
<td>8</td>
<td>16</td>
<td>30</td>
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<td>31</td>
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<tr>
<td>Headache</td>
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<td>11</td>
<td>14</td>
<td>12</td>
<td>13</td>
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<td>Ataxia</td>
<td>2</td>
<td>4</td>
<td>7</td>
<td>15</td>
<td>8</td>
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<tr>
<td>Somnolence</td>
<td>5</td>
<td>5</td>
<td>8</td>
<td>8</td>
<td>7</td>
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<tr>
<td>Tremor</td>
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<td>6</td>
<td>12</td>
<td>7</td>
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<tr>
<td>Nystagmus</td>
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<td>2</td>
<td>5</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>Balance disorder</td>
<td>0</td>
<td>1</td>
<td>5</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>Memory impairment</td>
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<td>1</td>
<td>2</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td><strong>Psychiatric disorders</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td><strong>Skin and subcutaneous disorders</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pruritus</td>
<td>1</td>
<td>3</td>
<td>2</td>
<td>3</td>
<td>2</td>
</tr>
</tbody>
</table>

Laboratory abnormalities

Abnormalities in liver function tests have been observed in controlled trials with VIMPAT in adult patients with partial-onset seizures who were taking 1 to 3 concomitant anti-epileptic drugs. Elevations of ALT to ≥3× ULN occurred in 0.7% (7/935) of VIMPAT patients and 0% (0/356) of placebo patients. One case of hepatitis with transaminases ≥20× ULN was observed in one healthy subject 10 days after VIMPAT treatment completion, along with nephritis (proteinuria and urine casts). Serologic studies were negative for viral hepatitis. Transaminases returned to normal within one month without specific treatment. At the time of this event, bilirubin was normal. The hepatitis/nephritis was interpreted as a delayed hypersensitivity reaction to VIMPAT.

Other Adverse Reactions in Patients with Partial-Onset Seizures

The following is a list of treatment-emergent adverse events reported by patients treated with VIMPAT in all clinical trials in patients with partial-onset seizures, including controlled trials and long-term open-label extension trials. Events addressed in other tables or sections are not listed here. Events included in this list from the controlled trials occurred more frequently on drug than on placebo and were based on consideration of VIMPAT pharmacology, frequency above that expected in the population, seriousness, and likelihood of a relationship to VIMPAT. Events are further classified within system organ class.

**Blood and lymphatic system disorders:** neutropenia, anemia

**Cardiac disorders:** palpitations

**Ear and labyrinth disorders:** tinnitus

**Gastrointestinal disorders:** constipation, dyspepsia, dry mouth, oral hypoesthesia

**General disorders and administration site conditions:** irritability, pyrexia, feeling drunk

**Injury, poisoning, and procedural complications:** fall

**Musculoskeletal and connective tissue disorders:** muscle spasms

**Nervous system disorders:** paresthesia, cognitive disorder, hypoesthesia, dysarthria, disturbance in attention, cerebellar syndrome

**Psychiatric disorders:** confusion, mood altered, depressed mood

**Intravenous Adverse Reactions**

Adverse reactions with intravenous administration generally appeared similar to those observed with the oral formulation, although intravenous administration was associated with local adverse events such as injection site pain or discomfort (2.5%), irritation (1%), and erythema (0.5%). One case of profound bradycardia (26 bpm; BP 100/60 mmHg) was observed in a patient during a 15 minute infusion of 150mg VIMPAT. This patient was on a beta-blocker. Infusion was discontinued and the patient experienced a rapid recovery.

**Comparison of Gender and Race**

The overall adverse event rate was similar in male and female patients. Although there were few non-Caucasian patients, no differences in the incidences of adverse events compared to Caucasian patients were observed.

**Postmarketing Experience**

The following adverse reactions have been identified during postapproval use of VIMPAT. Because these reactions are reported voluntarily from a population of uncertain size, it is not always possible to reliably estimate their frequency or establish a causal relationship to drug exposure.

**Cardiac disorders:** Bradycardia

**Skin and subcutaneous tissue disorders:** Rash

**DRUG INTERACTIONS**

Drug-drug interaction studies in healthy subjects showed no pharmacokinetic interactions between VIMPAT and carbamazepine, valproate, digoxin, metformin, omeprazole, or an oral contraceptive containing ethinylestradiol and levonorgestrel. There was no evidence for any relevant drug-drug interaction of VIMPAT with common AEDs in the placebo-controlled clinical trials in patients with partial-onset seizures [see Clinical Pharmacology (12.3) in Full Prescribing Information]. The lack of pharmacokinetic interaction does not rule out the possibility of pharmacodynamic interactions, particularly among drugs that affect the heart conduction system.

**USE IN SPECIFIC POPULATIONS**

**Pregnancy**

**Pregnancy Category C**

Lacosamide produced developmental toxicity (increased embryofetal and perinatal mortality, growth deficit) in rats following administration during pregnancy. Developmental neurotoxicity was observed in rats following administration during a period of postnatal development corresponding to the third trimester of human pregnancy. These effects were observed at doses associated with clinically relevant plasma exposures.
Lacosamide has been shown in vitro to interfere with the activity of collapsin response mediator protein-2 (CRMP-2), a protein involved in neuronal differentiation and control of axonal outgrowth. Potential adverse effects on CNS development can not be ruled out.

There are no adequate and well-controlled studies in pregnant women. VIMPAT should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Oral administration of lacosamide to pregnant rats (20, 75, or 200 mg/kg/day) and rabbits (6.25, 12.5, or 25 mg/kg/day) during the period of organogenesis did not produce any teratogenic effects. However, the maximum doses evaluated were limited by maternal toxicity in both species and embryofetal death in rats. These doses were associated with maternal plasma lacosamide exposures (area under the plasma-time concentration curve; [AUC]) ~2 and 1 times (rat and rabbit, respectively) that in humans at the maximum recommended human dose (MRHD) of 400 mg/day.

When lacosamide (25, 70, or 200 mg/kg/day) was orally administered to rats throughout gestation, parturition, and lactation, increased perinatal mortality and decreased body weights were observed in the offspring at the highest dose. The no-effect dose for pre- and post-natal developmental toxicity in rats (70 mg/kg/day) was associated with a maternal plasma lacosamide AUC approximately equal to that in humans at the MRHD.

Oral administration of lacosamide (30, 90, or 180 mg/kg/day) to rats during the neonatal and juvenile periods of postnatal development resulted in decreased brain weights and long-term neurobehavioral changes (altered open field performance, deficits in learning and memory). The early postnatal period in rats is generally thought to correspond to late pregnancy in humans in terms of brain development. The no-effect dose for developmental neurotoxicity in rats was associated with a plasma lacosamide AUC approximately 0.5 times that in humans at the MRHD.

**Pregnancy Registry**

UCB, Inc. has established the UCB AED Pregnancy Registry to advance scientific knowledge about safety and outcomes in pregnant women being treated with VIMPAT. To ensure broad program access and reach, either a healthcare provider or the patient can initiate enrollment in the UCB AED Pregnancy Registry by calling 1-888-537-7734 (toll free).

Physicians are also advised to recommend that pregnant patients taking VIMPAT enroll in the North American Antiepileptic Drug Pregnancy Registry. This can be done by calling the toll free number 1-888-233-2334, and must be done by patients themselves. Information on the registry can also be found at the website http://www.aedpregnancyregistry.org/.

**Labor and Delivery**

The effects of VIMPAT on labor and delivery in pregnant women are unknown. In a pre- and post-natal study in rats, there was a tendency for prolonged gestation in all lacosamide treated groups at plasma exposures (AUC) at or below the plasma AUC in human patients administered recommended doses of 400 mg/day.

**Nursing Mothers**

Studies in lactating rats have shown that lacosamide and/or its metabolites are excreted in milk. It is not known whether VIMPAT is excreted in human milk. Because many drugs are excreted into human milk, a decision should be made whether to discontinue nursing or to discontinue VIMPAT, taking into account the importance of the drug to the mother.

**Pediatric Use**

The safety and effectiveness of VIMPAT in pediatric patients <17 years have not been established.

Lacosamide has been shown in vitro to interfere with the activity of CRMP-2, a protein involved in neuronal differentiation and control of axonal outgrowth. Potential adverse effects on CNS development can not be ruled out. Administration of lacosamide to rats during the neonatal and juvenile periods of postnatal development resulted in decreased brain weights and long-term neurobehavioral changes (altered open field performance, deficits in learning and memory). The no-effect dose for developmental neurotoxicity in rats was associated with a plasma lacosamide exposure (AUC) approximately 0.5 times the human plasma AUC at the maximum recommended human dose of 400 mg/day.

**Geriatric Use**

There were insufficient numbers of elderly patients enrolled in partial-onset seizure trials (n=18) to adequately assess the effectiveness of VIMPAT in this population. In healthy subjects, the dose and body weight normalized pharmacokinetic parameters AUC and Cmax were approximately 20% higher in elderly subjects compared to young subjects. The slightly higher lacosamide plasma concentrations in elderly subjects are possibly caused by differences in total body weight (lean body weight) and age-associated decreased renal clearance. No VIMPAT dose adjustment based on age is considered necessary. Caution should be exercised for dose titration in elderly patients.

**Patients with Renal Impairment**

A maximum dose of 300 mg/day is recommended for patients with severe renal impairment (ClCR<30 mL/min) and in patients with endstage renal disease. VIMPAT is effectively removed from plasma by hemodialysis. Following a 4-hour hemodialysis treatment, AUC of VIMPAT is reduced by approximately 50%. Therefore, dosage supplementation of up to 50% following hemodialysis should be considered. In all renal impaired patients, the dose titration should be performed with caution. [see Dosage and Administration (2.2) and Clinical Pharmacology (12.3) in Full Prescribing Information]

**Patients with Hepatic Impairment**

Patients with mild to moderate hepatic impairment should be observed closely during dose titration. A maximum dose of 300 mg/day is recommended for patients with mild to moderate hepatic impairment. The pharmacokinetics of lacosamide has not been evaluated in severe hepatic impairment. VIMPAT use is not recommended in patients with severe hepatic impairment. [see Dosage and Administration (2.3) and Clinical Pharmacology (12.3) in Full Prescribing Information] Patients with co-existing hepatic and renal impairment should be monitored closely during dose titration.

**DRUG ABUSE AND DEPENDENCE**

**Controlled Substance**

VIMPAT is a Schedule V controlled substance.

**Abuse**

In a human abuse potential study, single doses of 200 mg and 800 mg lacosamide produced euphoria-type subjective responses that differentiated statistically from placebo; at 800 mg, these euphoria-type responses were statistically indistinguishable from those produced by alprazolam, a Schedule IV drug. The duration of the euphoria-type responses following lacosamide was less than that following alprazolam. A high rate of euphoria was also reported as an adverse event in the human abuse potential study following single doses of 800 mg lacosamide (15% [5/34] compared to placebo [0%]) and in two pharmacokinetic studies following single and multiple doses of 300-800 mg lacosamide (ranging from 6% [2/33] to 25% [3/12]) compared to placebo (0%). However, the rate of euphoria reported as an adverse event in the VIMPAT development program at therapeutic doses was less than 1%.

**Dependence**

Abrupt termination of lacosamide in clinical trials with diabetic neuropathic pain patients produced no signs or symptoms that are associated with a withdrawal syndrome indicative of physical dependence. However, psychological dependence cannot be excluded due to the ability of lacosamide to produce euphoria-type adverse events in humans.

**OVERDOSAGE**

**Signs, Symptoms, and Laboratory Findings of Acute Overdose in Humans**

There is limited clinical experience with VIMPAT overdose in humans. The highest reported accidental overdose of VIMPAT during clinical development was 1200 mg/day which was non-fatal. The types of adverse events experienced by patients exposed to supratherapeutic doses during the trials were not clinically different from those of patients administered recommended doses of VIMPAT.

There have been a single case of intentional overdose by a patient who self-administered 12 grams VIMPAT along with large doses of zonisamide, topiramate, and gabapentin. The patient presented in a coma and was hospitalized. An EEG revealed epileptic waveforms. The patient recovered 2 days later.

**Treatment or Management of Overdose**

There is no specific antidote for overdose with VIMPAT. Standard decontamination procedures should be followed. General supportive care of the patient is indicated including monitoring of vital signs and observation of the clinical status of patient. A Certified Poison Control Center should be contacted for up to date information on the management of overdose with VIMPAT.

Standard hemodialysis procedures result in significant clearance of VIMPAT (reduction of systemic exposure by 50% in 4 hours). Hemodialysis has not been performed in the few known cases of overdose, but may be indicated based on the patient's clinical state or in patients with significant renal impairment.

**PATIENT COUNSELING INFORMATION**

See FDA-approved Medication Guide and Patient Counseling Information section in the Full Prescribing Information.

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