PATIENT INFORMATION:
Swimmer’s Itch

What is Swimmer’s Itch?
Swimmer’s itch is a rash that appears as red, itchy bumps on the skin after swimming. It can occur hours or days after someone has been swimming in certain bodies of water, such as lakes. Swimmer’s itch is caused by the human body’s reaction to a parasite that lives in the water. The parasite does not survive in humans, and the rash will clear up within a couple of weeks.

Who is at Risk for Swimmer’s Itch?
Anyone who swims in non-chlorinated water that is home to large groups of birds (such as ducks or gulls) and a specific type of snail is at risk. The parasite lives in shallow shore areas with warmer water temperatures. Water known to be a source of the parasite will often have signs posted by public health officials to warn swimmers of the risk.

How to Prevent Swimmer’s Itch
Avoid water that is known to contain the parasite. If you don’t know if the water contains the parasite, rinse with clean water or towel off immediately after exiting the water.

How to Treat Swimmer’s Itch
Treatment is focused on relieving the discomfort caused by swimmer’s itch. It is very important not to scratch the rash to avoid an infection.1

♦ Cold compresses can be applied to affected areas.
♦ Colloidal oatmeal or Epsom salt baths can relieve discomfort. Use lukewarm (not hot) water in the bath.
♦ Baking soda and water can be mixed into a paste and applied to the itchy skin.
♦ Topical anti-itch creams or lotions (such as calamine) are available over-the-counter.

REFERENCES:


When to Contact Your Medical Provider
Talk to your medical provider if the rash skin does not improve or if it worsens after 3 days of treatment. If signs of infection (fever, pus, swelling) develop, contact your medical provider as soon as possible.

By Kaylyn DesRosier, PharmD Candidate
Nearly 73 million Americans have hypercholesterolemia (high cholesterol), a condition which can double the risk of heart disease. Less than one-third of people with high cholesterol have their low-density lipoprotein (LDL), or bad cholesterol, well managed. For years, the standard of therapy has been the statins, which include Lipitor® (atorvastatin) and Crestor® (rosuvastatin). Unfortunately, a disease called familial hypercholesterolemia results in highly elevated LDL and resistance to statin therapy for a small group of patients. Recently, a new class of drugs called the PCSK9 inhibitors were approved by the FDA to help treat this condition. Two drugs, Repatha™ (evolocumab) and Praluent® (alirocumab), are currently available.

Who may benefit from the PCSK9 inhibitors?

Both Praluent® and Repatha™ are used as add-on therapy to diet and maximally tolerated statin therapy in patients with clinical cardiovascular disease or heterozygous hypercholesterolemia, when LDL reduction has not been successful with previous treatments.

Who should not take a PCSK9 inhibitor?

Patients who have had severe hypersensitivity or anaphylactic allergic reactions to either Praluent® or Repatha™ should not take these medications.

Which conditions should I tell my doctor about before I start a PCSK9 inhibitor?

Prior to starting a PCSK9 inhibitor, tell your provider if you have a latex allergy, are pregnant or planning to become pregnant, or are breastfeeding. The needle cover of Repatha™ contains dry natural rubber, so an allergic reaction to latex may occur. Latex allergies are not a concern with Praluent®. Though significant drug interactions have not been identified with either drug, inform your doctor about all of your prescription and over-the-counter medications and supplements.

What are adverse events may occur while taking PCSK9 inhibitors?

Common side effects include upper respiratory infections, influenza, injection site reactions, muscle pain, urinary tract infections, and diarrhea.

How do I take my PCSK9 inhibitor?

- Both Praluent® and Repatha™ are administered by the patient as subcutaneous injections into the abdomen, thigh, or upper-arm.
- Each injection needs to warm up to room temperature for 30 minutes prior to administration.
- Rotate injection sites.
- Repatha™ is given either every two weeks (140 mg dose) or once a month (420 mg dose). The once-monthly 420 mg dose is given as three 140 mg injections within 30 minutes. Each injection is with a new syringe or auto-injector.
- Praluent® is given every two weeks and is available in 75 mg and 150 mg doses.

How do I store my PCSK9 inhibitor?

- Protect both medications from light exposure.
- Store both medications in the refrigerator at 36-46° F.
- Do NOT allow either medication to freeze or be exposed to temperatures above 77°F.
- Praluent® should not be exposed to room temperature (77°F) for more than 24 hours.
- Repatha™ may be kept at room temperature for up to 30 days.

How much do PCSK9 inhibitors cost?

Praluent® is estimated to cost $14,600 annually, or $560 per injection. Repatha™ has an annual cost of $14,100, or $542.31 per injection. Although copays are determined by individual insurance companies, both drugs have copay assistance programs.

The patient assistance program for Repatha™ allows eligible patients covered by non-government insurance programs to pay no more than $5 for each prescription refill.

Praluent® patient support allows eligible patients with commercial insurance to receive the first 6 months of therapy for a $0 copay. After the first 6 months, the copay becomes $10. Assistance for eligible patients without insurance provides 12 months of Praluent® free of charge.

By Matt Slagle, PharmD Candidate

References on Page 4

<table>
<thead>
<tr>
<th>Cholesterol Reductions in Heterozygous Familial Hypercholesterolemia with the PCSK9 Inhibitors</th>
<th>LDL Reduction (%)</th>
<th>Total Cholesterol Reduction (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Repatha™ (evolocumab)</td>
<td>61-71%</td>
<td>38-42%</td>
</tr>
<tr>
<td>Praluent® (alirocumab)</td>
<td>58%</td>
<td>36%</td>
</tr>
</tbody>
</table>
Savaysa™ (edoxaban) is a new anticoagulant agent approved in January 2015 by the FDA. Routine coagulation monitoring is not needed because edoxaban is a factor Xa (FXa) inhibitor. Edoxaban is indicated for stroke prevention in nonvalvular atrial fibrillation, deep vein thrombosis (DVT), and pulmonary embolism (PE). Edoxaban can also be used prophylactically for postoperative DVT prevention in patients undergoing total knee arthroplasty.

**Dosing:**

The typical adult dose of edoxaban is 60 mg by mouth daily for atrial fibrillation, DVT, and PE. Dosing for other different indications is described in Table 1.

Dose adjustments for creatinine clearance:
- >95 mL/min: do not use in patients with atrial fibrillation due to increased risk of ischemic stroke
- 15-50 mL/min: reduce dose to 30 mg daily
- <15 mL/min: not recommended

Patients with hepatic impairment:
- Not recommended for patients with Child-Pugh score B or C

Patients with low body weight (≤60 kg):
- Reduce dose to 30 mg daily

**Drug Interactions:**

Edoxaban is a p-glycoprotein substrate and can interact with other drugs that use the p-glycoprotein transport system. These drugs include azithromycin, clarithromycin, ketoconazole, and verapamil. In patients with a DVT or PE who are taking other drugs that are p-glycoprotein substrates, the edoxaban dose should be reduced to 30 mg daily.

Unlike rivaroxaban and apixaban, edoxaban does not interact with cytochrome P450 enzymes. This makes edoxaban a good alternative for patients on many medications. See Table 2 (page 4) for a pharmacokinetic comparison of anticoagulant agents.

**Adverse Effects:**

- Bleeding, including in mouth, pharynx, gastrointestinal tract, and skin
- Rash
- Abnormal liver function tests

**Edoxaban Compared to Warfarin:**

Edoxaban is comparable in efficacy and safety to warfarin, the oldest anticoagulant agent. Warfarin has its peak effect within four to five days. This time frame, along with its many drug interactions, make warfarin dosing difficult. For this reason, coagulation monitoring of INR is crucial for patient safety and efficacy.

Unlike warfarin, edoxaban has fewer drug interactions. Instead of 4-5 days, edoxaban reaches its peak effect in approximately one to two hours. Fewer interactions and a shorter time to peak effect makes monitoring less crucial with edoxaban. However, edoxaban’s half-life is much shorter than warfarin’s, so compliance is an important topic to discuss with patients before starting edoxaban. See Table 3 (page 4) for information on switching patients between edoxaban and warfarin.

Edoxaban was non-inferior to warfarin in a major study. The ENGAGE AF-TIMI 48 study focused on individuals with moderate-to-high risk atrial fibrillation. Primary endpoints included all-cause stroke events, systemic embolism, and mortality. This study also looked at safety related to increased bleeding risk.

In this randomized, double-blind, double-dummy trial, 21105 patients were given either warfarin adjusted to INR 2.0-3.0, edoxaban 60 mg daily, or edoxaban 30 mg daily. Stroke or systemic embolic events per year were decreased with edoxaban 60 mg compared to warfarin (1.18% vs. 1.50%). According to the study results, 197 people need to be treated with edoxaban 60 mg daily instead of warfarin to prevent one stroke or systemic embolic event per year. The incidence of major bleeds was also reduced in patients treated with edoxaban compared to patients treated with warfarin.

By Ka Zoua Moua, PharmD Candidate

**References and Tables on Page 4**

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**Table 1: Edoxaban Dosing**

<table>
<thead>
<tr>
<th>INDICATION</th>
<th>DOSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonvalvular Atrial Fibrillation Prophylaxis</td>
<td>60 mg by mouth daily</td>
</tr>
<tr>
<td>Deep Vein Thrombosis</td>
<td>60 mg by mouth daily starting 5-10 days after start of parenteral anticoagulant therapy</td>
</tr>
<tr>
<td>Pulmonary Embolism</td>
<td>60 mg by mouth daily starting 5-10 days after start of parenteral anticoagulant therapy</td>
</tr>
<tr>
<td>Postoperative DVT Prophylaxis (Arthroplasty of Knee)</td>
<td>30 mg by mouth daily starting 6-24 hours after surgery and continued for 11-14 days</td>
</tr>
</tbody>
</table>
Table 2: Anticoagulant Comparison

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Warfarin</th>
<th>Rivaroxaban</th>
<th>Apixaban</th>
<th>Edoxaban</th>
</tr>
</thead>
<tbody>
<tr>
<td>Target</td>
<td>VKORC1</td>
<td>FXa</td>
<td>FXa</td>
<td>FXa</td>
</tr>
<tr>
<td>Molecular weight, Da</td>
<td>308</td>
<td>436</td>
<td>460</td>
<td>548</td>
</tr>
<tr>
<td>Bioavailability, %</td>
<td>100</td>
<td>80</td>
<td>=50</td>
<td>=50</td>
</tr>
<tr>
<td>Dosing</td>
<td>Once daily</td>
<td>Once or twice daily</td>
<td>Twice daily</td>
<td>Once daily</td>
</tr>
<tr>
<td>Time-to-peak effect</td>
<td>4–5 d</td>
<td>2–3 h</td>
<td>1–2 h</td>
<td>1–2 h</td>
</tr>
<tr>
<td>Half-life, h</td>
<td>40</td>
<td>7–11</td>
<td>12</td>
<td>5–11</td>
</tr>
<tr>
<td>Renal clearance, %</td>
<td>None</td>
<td>33 (66)*</td>
<td>25</td>
<td>35</td>
</tr>
<tr>
<td>Monitoring</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Interactions</td>
<td>Multiple</td>
<td>3A4/P-gp</td>
<td>3A4/P-gp</td>
<td>P-gp</td>
</tr>
<tr>
<td>Antidote</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>

3A4 indicates cytochrome P450 3A4 enzyme; P-gp, P-glycoprotein; VKORC1, C1 subunit of the vitamin K epoxide reductase enzyme. *33% cleared as unchanged drug and 33% as inactive metabolites.

Table 3: Switching between Warfarin and Edoxaban

<table>
<thead>
<tr>
<th>Switching Direction</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Warfarin → Edoxaban</td>
<td>Discontinue warfarin. Start edoxaban when INR is ≤2.5</td>
</tr>
<tr>
<td>Edoxaban → Warfarin</td>
<td>Decrease edoxaban dose by 50% and start warfarin. Discontinue edoxaban when INR is stable (≥2). Monitor INR at least weekly once on warfarin therapy.</td>
</tr>
<tr>
<td>Edoxaban → Warfarin (With Parenteral Bridge)</td>
<td>Discontinue edoxaban and start warfarin and parenteral anticoagulant at next scheduled edoxaban dose. Discontinue parenteral anticoagulant once INR is stable (≥2)</td>
</tr>
</tbody>
</table>

PCSK9 Inhibitors References (from page 2)


REFERENCES:


The Mirena® (levonorgestrel) intrauterine device (IUD) is used for up to 5 years of contraception and is 99% effective. Mirena® is also FDA approved to treat menorrhagia.

Mirena® contains synthetic progestin that inhibits pregnancy by:
- Slowing the transport of the sperm
- Preventing the implantation of a fertilized egg

Do not use Mirena® if you are pregnant or if you have any of the following conditions:
- Pelvic Inflammatory Disease (PID) or history of PID
- Cervical or uterine cancer
- Breast cancer or other progestin sensitive cancer
- History of pelvic infections
- Hypersensitivity to levonorgestrel or any component of the IUD
- Liver cancer or disease

Precautions:
- If pregnancy occurs, IUD must be removed
- Bleeding patterns may change and become irregular or amenorrhea (absence of menstrual bleeding) may occur
- Expulsion of IUD may occur

Adverse Effects:
- Bacterial Vaginitis (13.6%)
- Vaginal Candidiasis (13.3%)
- Acne (12.3%)
- Headache (9.8%)
- Nausea/Vomiting (7.9%)
- Abdominal discomfort or pain (6.1%)
- Pelvic discomfort or pain (6.1%)
- Depression (5.4%)

After Mirena® is inserted, your provider will want to do an exam 4-6 weeks later and every year while the IUD is in place.

Mirena® was highly effective for up to 3 years in one study. The Mirena® IUD was associated with a low occurrence of side effects. Expulsion of the IUD within the first year after placement occurred in 3.5% of women in the study. Pelvic infections were not more common in the first 20 days after IUD insertion. In the 3-year study, only 6 pregnancies occurred in the 1751 women.

Mirena® can also be used to treat abnormal uterine bleeding. In 70 women, use of Mirena® decreased menstrual blood loss and improved quality of life.

By Mandy Major, PharmD Candidate

REFERENCES:
PATIENT INFORMATION:
Belsomra® (suvorexant): A Different Look at Treating Insomnia

What is Belsomra®?

Belsomra® (suvorexant) is a new medication that treats insomnia with fewer side effects than traditional sleep medications.

Suvorexant acts differently than other sleep medications. Suvorexant blocks hormones called orexins which play a role in waking you up and keeping you awake throughout the day.

Researchers believe that people with insomnia have higher levels of orexins in their brains. By blocking the orexins in the brain from working, suvorexant may help some people get to sleep and stay asleep.

How do I take suvorexant?

Some people might need more or less suvorexant depending on how well it works for them. Suvorexant comes in three different sized tablets, so you and your doctor can find which strength may work best for you.

Take suvorexant a half hour before you plan to go to bed at night. Suvorexant may take longer to work if you take it with food.

Suvorexant is intended to help you get a full night’s sleep, so you should plan on having at least seven hours of sleep before you take suvorexant.

Taking more than one dose of suvorexant before bed can increase the chances of side effects and make you very drowsy, so it is important to take it as prescribed by your doctor.

What are the side effects of suvorexant?

Suvorexant has fewer side effects than other available sleep medications. However, side effects can still happen. Normal side effects include diarrhea, dry mouth, and higher risk of flu or strep infection.

Serious side effects can happen with suvorexant. You should call your doctor right away if you:

- Feel sleepier than normal during the day
- Have problems concentrating
- Have episodes of sleep-walking, sleep-eating, sex while asleep, or sleep-talking

What are the warnings with suvorexant?

Suvorexant can interact with alcohol and some medications that also make you tired. When using suvorexant:

- Avoid drinking alcohol with suvorexant
- Have someone else drive if you have recently taken suvorexant
- Avoid taking other medications at night that make you drowsy, like pain medications, anti-anxiety medications, and some antidepressants

By Barry Bodle, PharmD Candidate

REFERENCES:


