Official Newsletter of the **Utah Poison Control Center**

TOXICOLOGY TODAY

2018 • VOLUME 20 • ISSUE 2

HEALTH
UNIVERSITY OF UTAH

Reversal Methods for NOACs

By B. Zane Horowitz, MD, FACMT

Introduction

In the last decade, the FDA has approved five novel oral anticoagulants (NOACs) that either directly inhibit thrombin (dabigatran) or inhibit factor Xa (rivaroxaban, apixaban, betrixaban, and edoxaban). Direct factor Xa inhibitors work by binding both free and clot-bound factor Xa, which ultimately prevents the formation of a thrombus.

Prescribed for similar situations, warfarin is a vitamin K antagonist that works by blocking the formation of clotting factors II, VII, IX, and X. It was approved by the FDA in 1954 and has been the mainstay of treatment to prevent thromboembolic events in susceptible individuals. Several limitations to the use of warfarin include its narrow therapeutic index, an increased risk of bleeding, numerous drug-drug and drug-food interactions, and the frequent laboratory monitoring it requires.[1] However, clinicians have years of experience using the medication, it is affordable, and an antidote (vitamin K) to reverse toxic effects is widely available.

TABLE 1: The Factor Xa Inhibitors

Generic Name	Brand Name
Rivaroxaban	Xarelto
Apixaban	Eliquis
Betrixaban	Bevyxxa
Edoxaban	Savaysa

Xa inhibitors all have the suffix "– xaban" in their names.

The advantages of the NOACs over warfarin include a more standardized dosing, little to no laboratory monitoring, and fewer of the same drug-drug and drug-food interactions that plague warfarin. In addition, they have a faster time to onset of therapeutic effect. For these reasons, NOACs have become widely prescribed, and as they gain market share, significant concerns have been raised regarding the ability to reverse the NOACs' anticoagulation effects in an emergency situation.

Bleeding is a major risk of the NOACs. The inability to assess the level of anticoagulation with standard coagulation laboratory values and, at least when they were first available, limited therapeutic options to treat major bleeding episodes.[1,2]

Bleeding was reported in 7% of 223 exposures to NOACs reported to eight poison centers between 2012 and 2014, including one subdural hematoma. Read on to learn more about the current therapeutic options to reverse effects of NOACs.

Preventing Absorption and Enhancing Elimination

Activated charcoal binds many xenobiotics, such as NOACs, and if administered soon after an ingestion, may prevent or minimize absorption. Activated charcoal has been demonstrated to enhance elimination of xenobiotics in a few instances. Scientists at Bristol-Myers Squibb studied the effect of activated charcoal on a single 20 mg dose of apixaban in healthy volunteers.[4] Activated charcoal administered at 2 hours and 6 hours decreased absorption of the drug. A decrease in half-life was noted in both activated charcoal groups. While an interesting study, it has limitations, and one should consider administering activated charcoal in an acute overdose if the ingestion was recent (1-2 hours prior), but it is unlikely to play a significant role in the treatment of a patient with active bleeding chronically on NOACs.

(cont. on pg. 3)

IN THIS ISSUE

Health Care Professionals, tell us what you think about the UPCC.

Survey Link:
http://utahcop.az1.qualtrics.com/jfe/form/SV_1GoBMY69hpcluWx

TOXINS IN THE NEWS

Brodifacoum in "Spice"

By B. Zane Horowitz, MD, FACMT

In March, several patients presented to emergency departments in the Chicago area with unexplained bleeding disorders. They had epistaxis, bleeding gums, hematuria, and GI bleeding. Some were seen multiple times for minor bleeding before any testing was done, but it was found that all had elevated INRs (some as high as 20) despite not being on warfarin. A subsequent investigation found that all these patients had used a form of "spice" a synthetic cannabinoid that goes by the trade names Blue Giant and Matrix among others.

Despite removal of the specific products from the smoke shop it was sold in, more cases showed up in other cities through the Midwest and mid-Atlantic states. All symptomatic patients have been traced back to various synthetic cannabinoids, and 3 patients died of bleeding-related complications. The compound brodifacoum, a rodenticide that was restricted in the US, was detected in several samples.

Some samples also contained a mixture of other rodenticides: difenacoum and bromodiolone. These compounds are known as "super-warfarins" and were designed to last for a very long time to overcome the resistance rodents had developed to the original coumadin-based rat poisons.

Despite speculation as to why and how these compounds were added to the potpourri mixtures sold as "spice," no rational explanation exists, as brodifacoum does not enhance the effect or duration of the synthetic cannabinoids and only leads to bleeding complications. A factory manufacturing error or intentional adulteration of these products are the best theories.

As of this printing, no cases have been detected in Utah or the Western United States. The CDC has sent advisory alerts to increase awareness of this problem, and emergency physicians should have a heightened suspicion in any patient with a bleeding problem and check an INR. The patient should be questioned as to their recreational drug use. If the INR is elevated and noncritical bleeding is occurring, reversal with fresh frozen plasma is advised.

For severe, life-threatening, or intracranial bleeding, rapid correction with a prothrombin complex concentrate (PCC), such as Kcentra, is advised. Please call the poison center to assist in management of these cases—or just to report their occurrences. A public health service will likely want to analyze the product, if possible, or send specialty toxicologic analysis on the patient's blood to find out if brodifacoum, or related compounds, were involved. All affected patients will likely need high doses of oral Vitamin K1 for several months and frequent repeat INR testing, because the duration of brodifacoum can last 9 months.

Rodenticide pellets

"Spice" *Credit: DEA*

OUTREACH EDUCATION

Older Adult Medication Safety Program

By Sherrie Pace, MS, MCHES®

Research indicates that 88% of older adults take at least one prescription drug, and 36% take five or more.[1] Therefore, this age group is prone to medication administration errors, misuse, and drug-drug and drug-food interactions.

Approximately 5% of poison exposure cases managed by Utah Poison Control Center (UPCC) involve individuals age 60 and older, and many of these cases involve medication. Furthermore, research shows that of children's visits to the emergency department for a poison exposure involving medication, 43% of the cases involved an uncle, aunt, or grandparent's medicine.[2]

The American Association of Poison Control Centers (AAPCC), in an effort to reduce poison exposures in older adults and in children who access their medications, developed the Older Adult Medication Safety (OAMS) program. The UPCC helped pilot the program in Utah during 2016 and 2017.

Data collected from Utah and other states helped inform and improve the program. The OAMS program is now a routine part of UPCC poison prevention education. The program focuses on five main topics:

1. Participate in your health by asking your health care professional questions and learning about your medications
2. Read and follow the instructions on the labels of medications you use, including over-the-counter drugs
3. Write down and keep track of your medicine schedule and use
4. Keep medicine up, away, and out of sight of children
5. Be prepared and know who to call for help if you experience a problem with your medicine

The UPCC Health Educators continue to provide the OAMS program in the metropolitan area of the state. The program will be coming soon to the UPCC website for individuals and organizations to access and use in their communities.

References

1. Qato DM, Wilder J, Schumm LP, Gillet V, Alexander GC. Changes in prescription and over-the-counter medication and dietary supplement use among older adults in the United States, 2005 vs 2011. *JAMA Intern Med.* 2016 Apr;176(4):473-82. doi: 10.1001/jamainternmed.2015.8581.
2. *Safe Kids Worldwide:* Keeping Families Safe Around Medicine (March 2014). https://www.safekids.org/med_report_2014. Accessed September 1, 2018.

(cont. from pg. 1)

NOACs

Hemodialysis may enhance the elimination of xenobiotics with small volumes of distribution that are of reasonable size and not highly protein bound. The direct factor Xa inhibitors available in the United States are all highly protein bound, but dabigatran has much lower protein binding and a relatively small volume of distribution. Stangier found that in a patient on dabigatran 150 mg who receives chronic dialysis, 62% of dabigatran was removed after 2 hours of dialysis and 68% after 4 hours of dialysis.[7] While dabigatran can be removed by dialysis, we must explore the risks and benefits of using this technique in an actual bleeding patient. In a patient with a life-threatening bleed, the risk of inserting a dialysis catheter may outweigh the benefits of removing some of the drug. In a non–life-threatening bleed, only supportive care until the drug is metabolized may be necessary. In a large intentional overdose, it is important to weigh these risks and benefits and consult with the poison center prior to initiating hemodialysis. A commercially available reversal agent for dabigatran, along with hemostatic agents, may be better therapeutic options.

Hemostatic Agents

Hemostatic agents contain clotting factors in various concentrations and are important considerations in patients on NOACs experiencing bleeding complications. Hemostatic agents include fresh frozen plasma (FFP), 3-factor prothrombin complex concentrate (PCC), 4-factor PCC, activated PCC (aPCC), or recombinant factor VIIa (rFVIIa) (see table 2). The main goal of using hemostatic agents in NOAC reversal is to overwhelm inhibition of direct thrombin or factor Xa.[5]

TABLE 2: Hemostatic Agents Composition[5]

Concentrate	Clotting Factors
FFP	II, VII, XI, and X
3-Factor PCC	Inactivated II, IX, and X
4-Factor PCC	Inactivated II, VII, IX, and X
aPCC	Inactivated II, IX, and X with activated factor VII
rFVIIa	Activated factor VII

FFP has several limitations. It has to be thawed prior to administration, and at most institutions, FFP may need to be obtained from the blood bank resulting in delayed administration. In addition, its lower concentrations of clotting factors result in a need for larger doses and volumes that may not be practical when treating a life-threatening bleed.[5] Four-factor PCC agents address many of the aforementioned weaknesses but are not as widely available.

Limited information about the use of hemostatic agents to reverse the effects of NOACs is available and is primarily limited to animal studies and healthy human volunteers. Variations in studies that assess different coagulation parameters make it difficult to identify preferred therapeutic options. The only hemostatic agent that reversed all anticoagulant effects of rivaroxaban and dabigatran was aPCC.[6] Using aPCC could put the patient at a higher risk of having a thrombotic event, in essence changing the anticoagulation state to that of a hypercoagulable state. Once the bleeding risk has resolved, restarting anticoagulation may be necessary to avoid thrombosis.

Reversal Agents

Two reversal agents have been recently approved for use, one for dabigitran (the only DTI) and one for the factor Xa inhibitors. Idarucizumab (Praxbind) was the first reversal agent approved by the FDA; this humanized monoclonal antibody fragment binds to and inactivates dabigatran.[8-11] Clinical trials show that a 1 gm infusion of idarucizumab normalized the anticoagulation effects of dabigatran for 30 minutes.[9] Higher doses of 2-4 gms of idarucizumab showed a reversal of dabigatran for greater than 12 hours.[9]

The largest study to date included 503 adults taking dabigatran therapeutically with either a life-threatening bleed or need for urgent surgery, in which a dose of 5 gms IV (given as two boluses 15 minutes apart) completely reversed the anticoagulation, based on both dilute thrombin time and ecarin clotting times, in 98% of the patients.[11] The current recommended dose of 5 gms IV includes an option to repeat once, if bleeding remains uncontrolled.[11]

Andexanet alfa (Andexxa), a recombinant modified human factor Xa protein, binds and inhibits the Xa inhibitors.[12] Phase II trials showed that andexanet alfa can decrease the effects of rivaroxaban in a dose-dependent manner.[9] In the higher doses study during the Phase II trials, it decreased the activity of rivaroxaban by as much as 81%.[9] An open label trial in patients with a major bleeding event as a result of factor Xa inhibitors showed that the median anti-factor Xa activity decreased by 89% after a loading dose of an andexanet alfa infusion in 26 patients taking rivaroxaban. The decrease was sustained from baseline of 86% at the end of the 2-hour infusion.

In 20 patients on apixaban, a 93% decrease was seen in the median anti-factor Xa activity at the end of the loading dose infusion and remained decreased by 92% at 2 hours.[12] The most serious adverse events related to using andexanet alfa in an open label trial were a failure to respond clinically with adequate hemostasis in 21% and thrombotic events in 18% of the patients.[12] In May 2018, the FDA issued an accelerated approval for andexanet alfa for use only in cases of bleeding with rivaroxaban and apixaban. However, it likely works for all 4 agents in this class.

A bolus dose of 400 to 800 mg is given no faster than 30 mgs per minute, followed by a 2-hour infusion of 4 to 8 mgs per minute. The higher dose is recommended for patients who have taken more than 10 mg rivoraxaban or 5 mg apixaban within the previous 8 hours. The lower dose is administered for patients presenting more than 8 hours after the last dose or who have taken 10 mg or less of rivaroxaban or 5 mg or less of apixaban. Because andexanet alfa is marketed in 100-mg vials, 4 to 8 vials are needed for the initial bolus dose and 5 to 10 vials for the subsequent infusion. Pharmacies should consider stocking 18 to 20 vials to treat acute, life-threatening bleeding due to a factor Xa inhibitor.

Recommendations

When addressing the bleeding concerns of a patient with a NOAC exposure, you should first assess how long the patient has been taking this medication. Patients taking a NOAC chronically are at more risk for bleeding compared to patients taking a single, massive dose.[2,3] In the case of an intentional overdose, the patient can be *(cont. on pg. 4)*

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NOACs

given activated charcoal if the ingestion occurred in the past 6 hours. Patients should be monitored for bleeding for a minimum of 24 hours in the hospital in the case of serious ingestions. Supportive care, including IV fluids, to maintain kidney function should be administered.

For serious bleeding associated with NOAC, if hemostatic agents are to be used, we recommend PCC or aPCC to reverse the effects of the NOAC. Local control and FFP may be considered for noncritical bleeding. Although theoretically capable of enhancing elimination, hemodialysis makes little sense because it possesses a risk of bleeding due to its own anticoagulation requirements, though you can consider it in patients who take large doses of dabigatran.

The preferred reversal agent for dabigitran is idarucizumab (Praxbind). For the factor Xa inhibitors, newly approved andexanet alfa shows promise as a reversal agent for life-threatening bleeding such as intracranial hemorrhage. We recommend that you continue to monitor drug approvals and cautions for these antidotes. While these antidotes show promise and are effective in treating NOAC overdoses and exposures, following evidence for beneficial and adverse outcomes as these drugs become more widely used and studied will help you provide the best care. Call the Utah Poison Control Center at 1-800-222-1222 for assistance on these cases, as the decision to use any of these agents can be difficult.

Acknowledgment to Christian Armstrong, PharmD, for contributing to this article.

References

1. Liotta EM, Levasseur-Franklin KE, Naidech AM. Reversal of the novel oral anticoagulants dabigatran, rivoraxaban, and apixaban. *Curr Opin Crit Care*. 2015 Apr;21(2):127-33.
2. Stevenson JW, Minns AB, Smollin C, et al. An observational case series of dabigatran and rivaroxaban exposures reported to a poison control system. *Am J Emerg Med*. 2014 Sep;32(9):1077-84.
3. Spiller HA, Mowry JB, Aleguas A Jr, et al. An observational study of the factor Xa inhibitors rivaroxaban and apixaban as reported to eight poison centers. *Ann Emerg Med*. 2015 Aug 19.
4. Wang X, Mondal S, Wang J, et al. Effect of activated charcoal on apixaban pharmacokinetics in healthy subjects. *Am J Cardiovasc Drugs*. 2014 Apr;14(2):147-54.
5. Babilonia K, Trujillo T. The role of prothrombin complex concentrates in reversal of target specific anticoagulants. *Thromb J*. 2014 Apr 17;12:8.
6. Marlu R, Hodaj E, Paris A, et al. Effect of non-specific reversal agents on anticoagulant activity of dabigatran and rivaroxaban: a randomised crossover ex vivo study in healthy volunteers. *Thromb Haemost*. 2012 Aug;108(2):217-24.
7. Stangier J, Rathgen K, Stähle H, Mazur D. Influence of renal impairment on the pharmacokinetics and pharmacodynamics of oral dabigatran etexilate: an open-label, parallel-group, single-centre study. *Clin Pharmacokinet*. 2010 Apr;49(4):259-68.
8. Majeed A, Schulman S. Bleeding and antidotes in new oral anticoagulants. *Best Pract Res Clin Haematol*. 2013 Jun;26(2):191-202.
9. Mo Y, Yam FK. Recent advances in the development of specific antidotes for target-specific oral anticoagulants. *Pharmacotherapy*. 2015 Feb;35(2):198-207.
10. Pollack CV Jr, Reilly PA, Eikelboom J, et al. Idarucizumab for dabigatran reversal. *New Eng J Med*. 2015, 6;373:511-20.
11. Pollack CV Jr, Reilly PA, van Ryn J, Idarucizmab for dabigatran reversal — full cohort analysis. *New Eng J Med*. 2017, 377:431-441.
12. Connolly SJ, Milling TJ, Eikelboom JW, et al. Andexanet alfa for acute major bleeding associated with factor Xa inhibitors. *New Eng J Med*. 2016, 375:1131-1141.

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Learn the Terminology of Cannabinoids

By Taylor Rhien, PharmD

Over the past few years, public attitude toward and access to marijuana has shifted. Many states have legalized medical marijuana, and 8 states currently allow recreational use. This trend has correlated with increased emergency department visits and calls to poison centers.[1] To assist patients, health care providers need to know the effects of marijuana as well as its terminology and the many forms it comes in.

A cannabinoid is any chemical that agonizes cannabinoid receptors in the body. The most well-known is Δ^9-tetrahydrocannabinol, or THC, which is primarily responsible for the psychoactive effects of marijuana. Over 60 other cannabinoids have been identified in the Cannabis plant, including cannabidiol, or CBD.[2] CBD has received much attention for numerous medicinal claims and is encountered with increasing frequency.

Synthetic cannabinoids emerged as drugs of abuse in the early 2000s. While they also interact with cannabinoid receptors, synthetics can differ substantially from natural cannabinoids and from one another in structure and clinical effects. They are generally referred to as a group by early brand names, such as "Spice" or "K2." These drugs are often labeled in ways to avoid legal repercussions. Recent sales of one synthetic cannabinoid under the guise of CBD oil in some Utah vape shops resulted in several hospitalizations across the Wasatch Front.[3] Synthetic cannabinoids don't act like typical marijuana and can produce acute psychosis, cardiac arrhythmias, and acute renal failure.

Marijuana is available today in many forms and strengths. Dried leaves of the plant can be smoked or ingested. Every possible food combination has been made from marijuana extracts, including brownies, cookies, gummy candies, and chocolate. Liquid preparations are commonly inhaled using electronic cigarette devices, ingested, or applied sublingually. Even topical formulations for application to the skin are available.

Additionally, the potency of modern marijuana products varies significantly. Since the 1980s, the average THC content of marijuana has risen from 3% to 12%.[4] THC concentrates, or "dabs," can be made by extracting THC with butane or other solvents achieving a THC content of 90% or more.[2] Common names for these butane hash oil (BHO) concentrates refer to their consistencies, such as "shatter," "wax," "budder," or "crumble." The highly concentrated THC can produce acute anxiety, panic attacks, and even coma.

The terminology of marijuana can be confusing. One medical database contains over 700 marijuana slang terms, and many of these refer to different forms, amounts, and methods of use.[5] Because every marijuana case is unique, health care providers are encouraged to consult with the Utah Poison Control Center on such cases.

About the Author

Taylor Rhien, PharmD, has been with the Utah Poison Control Center since 2013. He initially started while a student at the University of Utah College of Pharmacy and later began taking incoming poison exposure calls. He has been a full-time specialist in poison information since his graduation in 2016. His experience also includes work as a pharmacy intern at the University of Utah Hospital. Taylor is interested in learning about obscure and uncommon poisonings, and he also enjoys helping callers with the most routine types of exposures. He recently took his 10,000th call at the poison center.

References

1. Wang GS, Roosevelt G, Le Lait MC, et al. Association of unintentional pediatric exposures with decriminalization of marijuana in the United States. *Ann Emerg Med.* 2014; 63(6):684-9.
2. Hoffman RS, Howland MA, Lewin NA, Nelson LS, Goldfrank LR. *Goldfrank's Toxicologic Emergencies.* 10th ed. New York, NY: McGraw-Hill Education; 2015.
3. Utah Poison Control Center warns about "SPICE" product sold at local smoke shops [Press Release]. Salt Lake City, UT: Utah Poison Control Center; December 19, 2017 https://healthcare.utah.edu/publicaffairs/news/2017/12/CBD-oil.php. Accessed February 15, 2018.
4. Volkow ND, Compton WM, Weiss SR. Adverse health effects of marijuana use. *N Engl J Med.* 2014;371(9):879.
5. POISINDEX® System [Internet Database]. Cambridge, MA: IBM Watson Health; 2018. Accessed February 15, 2018.

http://poisoncontrol.utah.edu/newsletters/pdfs/annual-report-archive/2017.pdf

M-44 Sodium Cyanide

The United States Department of Agriculture, Animal and Plant Health Inspection Service, Wildlife Services would like to remind you that M-44 sodium cyanide devices are used in Utah. The ejector device contains a capsule of sodium cyanide that is placed in the ground and scented with lure. This device is used in specific situations to control coyotes to protect livestock and endangered species. This device is primarily used on private lands but may also be used on federal land in any county in the state. Areas where the M-44 sodium cyanide device is used should be marked with signs. While human exposure to this device is extremely unlikely, be aware that it contains 91% sodium cyanide, which is potentially lethal. A recent case in Idaho involved a dog and a child who were accidentally exposed. The dog was killed immediately and the child required emergency care. If someone is exposed to a device that has discharged into them, call 911 immediately. Contact the Utah Poison Control Center for assistance in management with antidotes. Pets exposed should be taken immediately to an emergency veterinary provider.

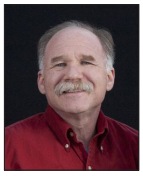
Close-up of a set M-44 device

Photo Credit: Guy Connolly, US Department of Agriculture National

Medical Toxicology Transitions

A Sincere Thank You

E. Martin Caravati, MD, MPH, retired from the University of Utah on June 30. Dr Caravati spent over 30 years at the University and has worked with the Utah Poison Control Center in several different capacities as associate medical director, medical director, and consultant since 1987. We are enormously grateful for his years of service and dedication to the UPCC.



B. Zane Horowitz, MD, completed his time as interim medical director on July 31. Dr Horowitz has graciously assisted the UPCC in his role as interim medical director for most of the last 7 years.

Welcome

Michael J. Moss, MD, joined the UPCC as medical director on August 1. Dr Moss is an emergency medicine physician who recently completed a 2-year medical toxicology fellowship at the Oregon Health & Science University. Press Release: https://healthcare.utah.edu/publicaffairs/news/2018/08/poison-control-director.php

Heartfelt Thank You!

Your support is critical to the success of the Utah Poison Control Center. Thank you for consulting us and informing us of your toxicology cases. Your contributions allow us to monitor trends in poisoning in Utah in a truly real-time manner. This past year, your consultations raised awareness about products sold as CBD oil that turned out to be a new synthetic cannabinoid.

Your ongoing consultations and reports are critical and allow UPCC to play a key role in surveillance of potential public health threats and monitoring of opioids and other drug poisoning problems.

Thank you to all who advocated tirelessly on our behalf before the Utah Legislature. The legislature awarded the UPCC an additional state appropriation that restored critical funding to maintain operations.

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Contact Us:
poison@hsc.utah.edu
Utah Poison Control Center
30 South 2000 East, Suite 4540
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The Utah Poison Control Center expresses its sincere thanks to the health care professionals, public health officials, and toxicology colleagues that work together to treat and prevent poisonings.

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