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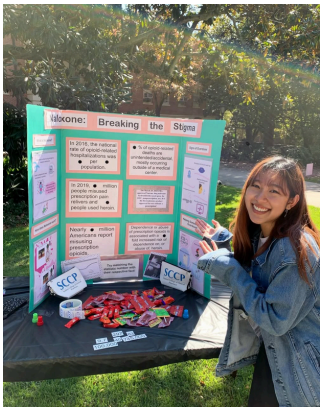
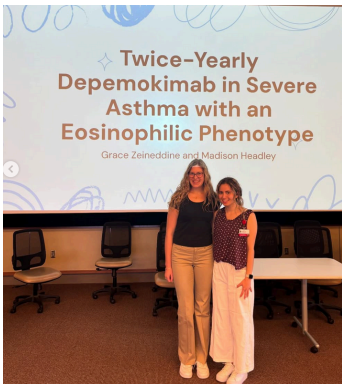
Student Clincal Digest

STUDENT COLLEGE OF CLINICAL PHARMACY





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Here's What Has Happened in SCCP This Year and What's to Come!

Welcome to our newsletter! We are thrilled to share with you the latest updates in our organization.

SCCP's mission is to encourage the professional development of pharmacy students interested in clinical pharamcy and have a positive impact in our community. We provide opportunities for professional growth, education, leadership, and advocacy in clinical pharmacy.

Before we begin, we would like to express our gratitude to our faculty advisors: Dr. Phillips and Dr. Stone. Their continued support and dedication have allowed our organization to continue to run smoothly and effectively. Additionally we would like to recognize our parent oraganization: American College of Clinical Pharmacy (ACCP).

This year has been filled with so much fun. Roza, Caroline, Angel, Logan and Emmali led the charge in educating the community on Naloxone during pharmtoberfest in October.

Our P3 and P4's competed in the ACCP clinical pharmacy challenege in the fall and our P1s and P2's competed in ACCP's clinical research challenge in the spring.

We conducted journal clubs and jeapordy/ crossword drug card reviews throughout the year to support our students' education.

Our guest speakers this semester included Dr. Yolanda Whitty, the clinical pharmacy manager at Wellstar Hospital and Dr. Salman Hasham a cardiology ambulatory care clinical pharmacist with Grady Health System. We even got to hear from our very own: Alyssa Utz! She once was UGA's SCCP president and now is an anticoagulation ambulatory care clinical pharmacist with Grady Health System.

Next year, we are excited to incorporate point of care (POC) events to further service our community with our new exec position POC Liason led by Simin.

Enjoy this semester's newsletter!

Grace Zeineddine

SCCP Vice President
Student Clinical Digest Co-Editor

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Prevalence of Prescription Medication Use That Can Exacerbate Heart Failure Among US Adults with Heart Failure

By: Isabel Campba, Pharm.D. Candidate 2026

According to the Journal of Cardiac Failure, in 2024, an approximate 6.7 million Americans over the age of 20 had heart failure, and prevalence is expected to rise by another 2 million adults over the next decade. An even more shocking statistic is that it accounted for almost 425,000 deaths in 2021, with mortality from heart failure being higher now than in 1999 despite evidence-based guideline-directed medical therapy (GDMT).[1] One factor that contributes to patient death is heart failure exacerbation, which can be worsened with the use of common medications like non-steroidal anti-inflammatory drugs (NSAIDs) and certain antihypertensives.[2] Despite having resources detailing which drugs exacerbate heart failure, some patients are still prescribed these medications; knowing how often this occurs can help prescribers avoid these patterns.

A study performed by A. Zheutlin et al. in 2024 examined how many exacerbating medications were prescribed to patients with heart failure. Using a national Center for Disease Control and Prevention (CDC) survey called the National Health and Nutrition Examination Survey (NHANES), the authors collected data from adults regarding their prescribed medications. Further sensitivity analysis was conducted on patients suspected to have reduced ejection fraction (HFrEF) on at least one or two GDMT agents, which included angiotensin-converting enzyme inhibitors (ACE-i), angiotensin receptor blockers (ARB), angiotensin receptor neprilysin inhibitors (ARNI), or mineralocorticoid receptor antagonists (MRA) at the time of survey. It was determined that out of the 687 adults who participated, 14.5% (95% CI 10.4%, 19.5%; n = 92) received medications that were known to exacerbate heart failure; the most commonly prescribed drugs that could cause an exacerbation were diltiazem, meloxicam, and ibuprofen.[3]

Subgroup analysis showed that there was no significant difference in the number of males and females that were prescribed exacerbating medications (aOR 0.66, 95% CI 0.23, 1.46; p = 0.31). Additionally, when examining the rates of prescribing for non-hispanic whites (15.1%; 95% CI 9.8%, 22.4%), non-hispanic blacks (14.2%; 95% CI 8.8%, 22.0%), hispanics (14.0%; 95% CI 7.4%, 24.7%), and other/multi-race patients (9.3%; 95% CI 0.6%, 61.5%), there was no significant difference in the amount of exacerbating prescriptions received by each subgroup (p > 0.05 for all).[3]

Sensitivity analysis for the suspected HFrEF group revealed that 15% (95% CI 9.8%, 21.4%) of patients on at least one GDMT medication were prescribed a medication that could exacerbate their heart failure, and 14.1% (95% CI 12.4%, 27.1%) of patients on at least two GDMT medications were prescribed an exacerbating medication. Lastly, 19% (95% CI 12.4%, 27.1%) of patients on a loop diuretic had a medication that exacerbated heart failure, and 8.3% (95% CI; 4.9%, 12.9%) of patients within the analysis were taking an NSAID.[3]

Overall, this study was well-balanced since the NHANES oversampled specific subgroups in order to improve generalizability. However, this study lacked data regarding a patient's over-the-counter medications. potentially leaving out frequently used medications that could also exacerbate heart failure, such as NSAIDs. Additionally, more specific data on the patient's comorbid conditions, specific ejection fraction status, and prescribing clinician characteristics were unavailable, limiting analysis regarding prescribing patterns.[3]

In conclusion, patients with heart failure can have unnecessary difficulties and hospitalizations due to exacerbations that could have been avoided with mindful prescribing patterns. Being familiar with drugs that can exacerbate heart failure can minimize these complications, improving patient outcomes and extending their lives.

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Characterization of Lamotrigine Disposition Changes During and After Pregnancy in Women with Epilepsy

By: Martavius Cladd, Pharm.D. Candidate 2026

Lamotrigine, commonly used in the treatment of epilepsy, is widely prescribed in women of childbearing age due to its favorable safety profile in pregnancy. However, pregnancy can induce pharmacokinetic changes that reduce lamotrigine plasma levels, which in turn can increase seizure risk.[1] Understanding how lamotrigine's clearance changes during gestation and postpartum is crucial for pharmacists managing therapy in pregnant patients.

Pregnancy markedly increases lamotrigine clearance. This increase can begin as early as 5 weeks gestation, rising by up to roughly triple the amount of the nonpregnant baseline by the third trimester. In the MONEAD study, approximately 91% of the 170 pregnant women experienced a 275% increase in clearance, while 9% showed minimal change, indicating significant variability between patients as it pertains to metabolism during pregnancy.[1] Without dose adjustments, declining lamotrigine plasma levels can lead to breakthrough seizures[2]. Dose increases of 20–25% are often needed throughout gestation to maintain therapeutic levels.[3]

After delivery, lamotrigine clearance returns rapidly to pre-pregnancy baseline, typically within 2–3 weeks.[1] Without appropriate dose reduction, supratherapeutic levels may result in toxicity, including nausea, dizziness, and ataxia.[4] Pharmacists should ensure empiric dose tapering is initiated immediately postpartum and completed within 21 days, with therapeutic drug monitoring (TDM) to confirm safe concentrations.[4]

The increase in lamotrigine clearance during pregnancy is largely driven by hormone mediated enzyme induction. Lamotrigine is metabolized primarily by the hepatic enzyme UGT1A4. During pregnancy, estradiol concentrations rise and upregulate UGT1A4 through estrogen receptor-α pathways, which in turn accelerates lamotrigine metabolism.[5] Additionally, enzyme-inducing antiseizure medications such as phenytoin, phenobarbital or carbamazepine can elevate lamotrigine clearance even outside of pregnancy. Likewise, estrogen-containing medications, such as combined oral contraceptives, increase lamotrigine clearance by about 20-50%6. Karanam et al. reported that women on enzyme-inducing drugs or hormonal therapies had 84% and 33% higher baseline lamotrigine clearance, respectively.[1] Due to the unpredictable compounded effects of these clearance changes observed during pregnancy, further support for individualized dose management is essential.

Regular TDM should be performed throughout pregnancy and postpartum.[2] The patient's therapeutic lamotrigine concentration preconception baseline should be established to guide later dose adjustments.[2] During pregnancy, monitor lamotrigine levels at least once per trimester (or monthly), adjusting doses to avoid concentrations falling below 65% of baseline.[3] During postpartum, reduce the dose incrementally back to the pre-pregnancy level within 2–3 weeks after birth, with TDM to avoid toxicity[4]. Implementation of contraceptive counseling explaining that estrogen-based contraceptives may reduce lamotrigine levels postpartum should be considered in follow-up plans.[6]

By anticipating pharmacokinetic changes and using TDM, pharmacists can guide therapy adjustments that maintain seizure control in pregnant patients while preventing toxicity in the postpartum period. This proactive management is vital to protect both maternal and fetal health, as well as the well-being of the newborn (by avoiding maternal sedation or other adverse effects). Pregnancy induces substantial increases in lamotrigine clearance via estrogen-mediated UGT1A4 hormonal enzyme induction, and these effects swiftly reverse after delivery. Dosing must be dynamically managed through close monitoring, evidence-based adjustments, and clear communication among the healthcare team to ensure optimal outcomes for women with epilepsy on lamotrigine during and after pregnancy.

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Early Initiation of Sodium-Glucose Cotransporter 2 Inhibitors to Treat Patients Post Acute Coronary Syndrome

By: Logan Hartzell, Pharm.D. Candidate 2027

The connection between cardiovascular health and type 2 diabetes mellitus (T2DM) is well established. When patients develop advanced diabetes, they often have pertinent cardiovascular risk factors, such as obesity, hypertension, and dyslipidemia.[1] Additionally, the biological mechanisms behind diabetes progression (microangiopathy, macroangiopathy) are independent risk factors increasing the risk of cardiovascular disease (CVD) in patients with diabetes.[1] Among patients with T2DM and established CVD, sodium-glucose cotransporter-2 inhibitors (SGLT2i), are antidiabetic drugs associated with a significant reduction in cardiovascular events. These drugs work by inhibiting the channel in the kidneys which allow for glucose reabsorption, causing glucose to be excreted in the urine and lowering serum blood glucose.

In previous trials studying both dapagliflozin and empagliflozin, both SGLT2i drugs demonstrated a statistically and clinically significant reduction in major adverse cardiovascular events (MACE).[2] However, when it comes to the interaction between diabetes and acute coronary syndrome (ACS), past trials tend to have excluded ACS patients from their studies. Further, patients who have undergone coronary artery bypass graft surgery (CABG), are underrepresented in other studies linking SGLT-2i use to cardiovascular outcomes.[2] Since there are multiple efficacy and safety concerns regarding the use of SGLT-2i in ACS patients, several studies have been proposed to identify whether these drugs are both safe and efficacious for use in ACS patients.

Following a myocardial infarction (MI), there are a spectrum of risks for developing a recurrent MI, heart failure, and life-threatening arrhythmias. The trajectory of risk begins once symptoms appear, as it is strongly correlated to total ischemic time.[3] The standard of care for an acute MI includes early reperfusion and invasive strategies, antiplatelet and antithrombotic therapy, early initiation of renin-angiotensin-aldosterone-system inhibitors (RAAS), beta-blockers, and statins. Although SGLT2 inhibitors are used as cardiorenal protective agents in T2DM, heart failure, and kidney disease, there is a potential mechanism for SGLT2 inhibition to reduce risk of acute MI.[3] This is not through any inhibition of cardiac thrombosis, but rather attenuation of neurohormonal activation, cardiomyocyte necrosis, and reperfusion injuries.[3]

Additionally, early initiation of SGLT2i therapy may also improve outcomes by augmenting endothelial function through vasodilation, myocardial metabolism, and preserving contractility and coronary blood flow.[3] The use of SGLT2-inhibitors is often discouraged during acute illness, limiting the available empiric data regarding use post-MI. However, safety outcomes that are available suggest that initiating an SGLT2i acutely following cardiac arrest was safe, with no serious adverse effects attributable to the SGLT2i.[2] This lack of clinical data underscores the importance of further study into the specific cardiovascular effects of SGLT2 inhibitor drugs.

Patients with complicated acute MI's are at high risk for both early and progressive heart failure and death. Despite the success of current therapies, there is an unmet need for further risk reduction, particularly in the early period following a cardiac event.[2] The potential mechanism of SGLT2i therapy may be of unique benefit in patients with acute MI. However, ongoing efforts are required to evaluate the safety and efficacy of these drugs within the days following an acute MI.[3]

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New Drug Study: Clinical Applications of Sodium-Glucose Cotransporter Inhibitors

By: Nicholas Holthaus, Pharm.D. Candidate 2027

Since entering the market, Sodium Glucose Cotransporter-2 inhibitors (SGLT2is) have been integral to the management and treatment of diabetes. After their introduction, a wide berth of alternative indications began to be explored. Although currently only approved for diabetes and heart failure, this study presents a meta-analysis of randomized controlled trials (RCTs) to evaluate the potential for SGLT2is to include indications for other disease states.

Obesity

Obesity is a common comorbidity found in patients with diabetes. The two disease states have an interconnected relationship making obesity a helpful target for improving diabetes control. A large-scale meta-analysis evaluated the relationship between SGLT2is, specifically canagliflozin, empagliflozin, sotagliflozin, and licogliflozin, and weight loss in patients with diabetes. In this study, the four agents led to an average weight loss equating to 2-3% of total body weight.[1] Another promising aspect of SGLT2i use in weight loss is the type of weight targeted. An alternative randomized controlled trial evaluated canagliflozin for its method of weight loss which was determined to be predominantly visceral fat while sparing lean muscle.[2]

Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD)

MASLD is another disease state that is closely linked with diabetes. The use of SGLT2is in this disease state can lead to not only reduced hepatosteatosis, but also a reduction in fatty liver content in patients with MASLD. One study even demonstrated a 22% reduction in fatty liver content by the end of treatment.[3] It was also determined that SGLT2is can have a synergistic effect with pioglitazone to improve control of MASLD.

Polycystic Ovarian Syndrome (PCOS)

Patients with PCOS often experience metabolic disturbances and impaired endogenous insulin function, sometimes even leading to insulin resistance (IR). The use of SGLT2is in women with PCOS showed many favorable outcomes including the improvement of hyperinsulinemia and hyperandrogenemia with the use of licogliflozin.[4] Additionally, another study determined the use of canagliflozin-metformin combination therapy resulted in improved menstruation frequency, weight control, hyperandrogenemia, and improvement of insulin resistance.[5]

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Anemia

As many as 1 in 4 patients with diabetes are affected by anemia. Although the mechanism is poorly understood, SGLT2is have a marked improvement in lab values relating to anemia, specifically hemoglobin and hematocrit.[6] Studies were conducted more specifically with each SGLT2i agent, but the overall consensus was that SGLT2i use in anemic diabetic patients was a promising opportunity that needs further evaluation.

Lipid Metabolism

Hyperlipidemia and other lipid related disease states are often important treatment targets. Complications exacerbating cardiac issues make lipids an important component of health management in diabetic patients. Studies conducted on the use of SGLT2is for the management of lipids in diabetic patients concluded that SGLT2i use resulted in an overall decrease in total triglycerides and an increase in LDL-C and HDL-C. Although LDL-C increase is often associated with poor clinical outcomes, the size of the LDL-C is more beneficial to patient outcomes so it is not a large point of concern, but warrants further investigation.[7]

Hyperuricemia

Hyperuricemia in combination with diabetes can be associated with cardiovascular disease, renal complications, and metabolic syndrome. A number of studies evaluated the use of SGLT2is in hyperuricemia and found benefit in the use of empagliflozin, dapagliflozin, ipragliflozin, luseogliflozin, and tofogliflozin in reducing uric acid levels in patients suffering with hyperuricemia.[8]

Obstructive Sleep Apnea Syndrome (OSAS)

Clinical studies of SGLT2i use in patients with diabetes and OSAS have shown promise in improving both disease states. Ertugliflozin showed a marked impact on OSAS showing 48% reduction.[9] The mechanism of action is thought to be through an increase in endogenous glucose production, which leads to a decrease in production of CO2 and, in turn, a decrease in the incidence of OSAS.

Syndrome of Inappropriate Antidiuresis (SIAD)

SIAD is a dysfunction of the kidneys that leads to a state of continuous hypotonic hyponatremia. Multiple studies of empagliflozin in SIAD found that use of SGLT2is leads to increased electrolyte-free water excretion via their mechanism of action and in turn increases the average level of plasma sodium measured.[10]

Obesity Management: Current and Emerging Therapies

By: Emmali Taghan, Pharm.D. Candidate 2027

Obesity, defined as a body mass index (BMI) of greater than or equal to 30, is a common chronic condition affecting many adults in the United States. Data from 2021-2023 shows that 40.3% of adults aged 20 and older in the U.S. had obesity.[1] Reduced calorie intake paired with increased physical activity are the foundations of weight-loss, as well as behavioral therapy.[2] However, these lifestyle modifications are difficult to sustain and weight loss from these lifestyle changes alone may be small. Pharmacological interventions paired with lifestyle changes can be useful to help achieve and sustain more significant weight loss.[3]

Incretin modifiers, originally developed for diabetes management, have become a popular therapeutic option for obesity management. These medications work by mimicking incretins, which are gastrointestinal hormones that regulate insulin secretion from the pancreas. Incretin hormones include glucagon-like peptide-1 (GLP-1) and gastric inhibitory peptide (GIP), both of which promote insulin release, delay gastric emptying, and suppress glucagon secretion.[4,5] The current incretin modifiers approved by the FDA for obesity management include liraglutide, semaglutide, and tirzepatide.[3]

Extensive clinical trials have demonstrated that liraglutide, semaglutide, and tirzepatide have positive clinical outcomes in patients who are overweight or obese. All three medications were shown to significantly increase weight loss compared to placebo.[3] Tirzepatide (15mg) showed the most weight loss at 20.9% in the SURMOUNT-1 trial, compared to 3.1% with placebo.[3] Semaglutide (2.4mg) showed the second-highest weight loss at 15%, compared to 2.4% with placebo in the STEP-1 trial. Liraglutide (3mg) showed weight loss of 8.4kg from a baseline weight of 106kg, compared to 2.8kg with placebo in the SCALE Obesity Trial.[3]

Some emerging incretin-based therapies are currently being evaluated for their efficacy in weight loss and their potential to offer better tolerability, reduced dosing frequency, and alternative routes of administration (e.g., oral). These medications include oral GLP-1 receptor agonists, amylin analogues, dual GLP-1/GIP agonists, glucagon/GLP-1 receptor agonists, and triple agonists (GIP, glucagon, and GLP-1).[3]

Many of these drugs are still in phase 2 or earlier of their clinical trials. Oral GLP-1 agonists have shown weight loss ranging from 5-15.1%, with only one drug (danuglipron) having substantial discontinuation rates (>50%). Amylin analogues show slightly lower percentages of weight loss, ranging from 4.2-10%. Dual GLP-1/glucagon show weight loss reductions ranging from 7.2-14.5% and no treatment-related discontinuations. Glucagon/GLP-1 agonists show weight reductions ranging from 6.2-14.9%; however, discontinuation rates range from 6.8-29%. Triple agonists show weight loss reduction ranges of 7.2-17.5% and discontinuation rates of 6-16%.[3]

The results of clinical trials evaluating incretin modifiers and their efficacy in weight loss are highly promising. Patients were shown to lose more weight with these medications than with lifestyle modifications alone. However, limitations with these medications are prevalent. The long-term outcomes of these drugs have not been studied extensively. The cost of these agents can also be a potential barrier to access and adherence. It is possible that the development of new therapies, such as amylin analogues, dual GLP-1/glucagon agonists, and triple agonists, can improve availability and affordability of these treatments.[3]

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Combining Aztreonam-avibactam to Form a Dynamic Duo Against Gram-Negative Pathogens

By: Grace Zeineddine, Pharm.D. Candidate 2026

In the antimicrobial world, carbapenems and other β-lactams play a crucial role in treating severe bacterial infections due to their broad-spectrum activity, especially against multidrug-resistant (MDR) pathogens.[1] There are gaps, however, in their coverage. According to CDC[2], there has been an increase in gram-negative hospital-acquired infections resistant to carbapenems. Carbapenem-resistant (CR) organisms include those resistant to *Enterobacterales* (CRE), *Pseudomonas aeruginosa* (CRPA), and *Acineobacter baumannii* (CRAB).[1] One of the biggest enzymes of concern leading to antimicrobial resistance in these bacteria is Metallo-β- lactamases (MBLs). They inactivate all β-lactams except aztreonam and one other.[1] Azetronam, however, is prone to ESBLs (extended-spectrum β-lactamases) and AmpC β- β-lactamases, which can make the drug ineffective against strains producing these enzymes.[1] Avibactim is a drug that can neutralize these two lactamases, which would restore aztreonam's activity if the two were used together.[1] Al Musawa et all explore this promising new drug combination of Azetronam (ATM) with Avibactim(AVI) that can even work on MBLs and CREs.

ATM is a monobactam antibiotic that works by binding penicillin-binding protein 3 (PBP-3) in gram-negative bacteria, disrupting cell wall synthesis and causing bacterial death.[1] AVI is a non-β-lactam β-lactamase inhibitor that blocks ESBLs and AmpC β-lactamases.[1]

Three clinical trials discussed in the article evaluated ATM-AVI's efficacy and safety. This includes the REVIST trial (phase 3), ASSEMBLE trial (phase 3), and Rejuvenate trial (Phase 2a). The REVISIT trial compared ATM-AVI +/- metronidazole against colistin in the treatment of complicated intra-abdominal infections (cIAI), hospital-acquired pneumonia (HAP), and ventilator-associated pneumonia (VAP)1. Results of the intent-to-treat analysis showed no significant difference in clinical cure with 68.4% with ATM-AVI vs 65.7% meropenem.[1] Only 10 patients had MBL-producing bacteria, limiting conclusions for this subgroup.[1]

In the ASSEMBLE trial, ATM-AVI was compared against the best available therapy (BAT) in MBL-producing gram-negative infections1. The clinical cure rate in MBL infections was 41.7% ATM-AVI vs. 0% with BAT1. This strengthens the case for ATM-AVI, however, the results are limited by a small sample size of 15.

The Rejuvenate trial studied ATM-AVI's pharmacokinetics and safety in 34 cIAI patients1. ATM-AVI was well tolerated, but elevated liver enzymes (AST/ALT) occurred in 26.5% of patients.[1] The second most common adverse effect was diarrhea 14.7% but was found not to be related to ATM-AVI1. No severe adverse event related to the medication occurred.

The European Medicines Agency (EMA) approved ATM-AVI in April of 2024 for cIAI, cUTI, HAP, and serious gram-negative infections with limited treatment options1. The FDA also approved it this past February (2/7/2025) in patients 18 years or older who have limited or no alternative options for the treatment of cIAI.[3]

ATM-AVI has its own limitations. It's effective against most MBL-producing bacteria, but studies suggest novel mechanisms of resistance with mutations in the β- lactamases.[1] Additionally, some E. coli isolates, especially a subset in the UK with PBP-3 mutation inserts showed reduced susceptibility.[1] Finally, while ATM-AVI targets MDR Enterobacterales, it has limited activity against *Pseudomonas aeruginosa.*[1]

In conclusion, more research is needed to fully understand ATM-AVI, including dose optimization, synergistic combinations, and real-world resistance patterns. Continued stewardship and surveillance will be essential. For now, however, it is a promising new weapon against MDR bacteria.

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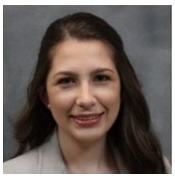

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