PHARMACIST CE:

Pediatric QTc Prolongation: A Review of Risk Factors and Management Strategies

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CE FOR **PHARMACISTS**

COMPLETE ARTICLE AND CE EXAM AVAILABLE ONLINE: **WWW.PSWI.ORG**

Learning Objectives

- Describe the pharmacist's role in preventing pediatric QTc prolongation.
- Explain the changes in QTc interval over time that impact a LQTS patient's risk of experiencing a cardiac event.
- Recommend appropriate electrocardiogram monitoring for patients with ADHD.
- Identify the modifiable and non-modifiable risk factors for QTc prolongation.
- Select an appropriate dose of magnesium for a pediatric patient experiencing torsade de pointes.

The QT interval is a rate dependent interval calculated on an electrocardiogram (EKG) that reflects the time between ventricular depolarization and repolarization. In healthy adults, a normal QTc is typically less than 450 milliseconds (ms) for men and 460 ms for women while QTc greater than 500 ms has been associated with greater risk of ventricular arrhythmias.[1] The QT interval varies with age in pediatric patients, but QTc prolongation is generally defined as greater than 460 ms or an increase from baseline of 60 ms.[2-4] When the QT interval is prolonged, some myocardial cells become refractory which can result in spontaneous depolarization, known as early afterdepolarization. Early afterdepolarization can increase the risk of both ventricular dysrhythmias, such as torsade de pointes (TdP) or ventricular fibrillation, and sudden cardiac death.[1,5] There are many factors that can contribute to prolongation of the QT interval including genetic causes such as congenital long QT syndrome (LQTS), advanced age, history of heart disease, hypothyroidism, bradycardia, electrolyte derangements, and medications.[1,6-8] Many medications that cause prolongation of the QT interval interact with the delayed potassium rectifier channels in the cardiac myocyte. When the potassium ion channel is blocked, the action potential is prolonged, resulting in an increased QT interval.[7] Antagonism of the potassium ion channel and resulting QTc prolongation is more likely to occur when multiple QTc-prolonging medications are utilized concurrently.

TThe Credible Meds QT Drug List is a database identifying over 200 medications that carry risk of QTc prolongation and divides the medications into risk categories of known risk of TdP, conditional risk of TdP, and possible risk of TdP and also denotes drugs to avoid in LQTS.[6] Although this list risk stratifies QTc-prolonging medications, it is still essential that pharmacists critically evaluate the likelihood of QTc prolongation within the context of other patient risk factors in order to recommend medication therapy modifications if necessary. Table 1 reflects QTc-prolonging medications that are commonly utilized in pediatric patients. Pharmacists have an integral role in recognizing patients with non-modifiable risk factors for QTc prolongation, intervening on modifiable risks such as electrolyte abnormalities including hypokalemia, hypomagnesemia, and hypocalcemia, as well as recommending appropriate medication therapies in order to prevent QTc prolongation and reduce the risk of ventricular arrhythmias and sudden cardiac death in pediatric patients.

Congenital Long QT Syndrome

Congenital long QT syndrome is caused by mutations within genes involved in the function of myocyte sodium and potassium ion channels resulting in the delay of ventricular repolarization. Those with inheritable repolarization disorders are predisposed to malignant arrhythmias

that can precipitate syncope, sudden cardiac arrest, or sudden cardiac death.[9-13] Approximately 1 in 2,500 caucasians are afflicted with congenital LQTS, however this number is thought to be closer to 1 in 2,000 live births when considering patients with unconfirmed genotypes and normal QTc intervals.[11] Diagnosis is based on the measurement of the QTc and subsequent rule out of secondary causes such as medication therapies and electrolyte abnormalities. Parameters to diagnose QTc prolongation vary by age and sex. In patients between 1 and 15 years of age, QTc interval greater than 460 ms is considered prolonged.[10] The efficacy of EKG screening of infants and children for congenital LQTS is controversial. Some argue that it is cost-effective in preventing sudden infant death syndrome as well as sudden cardiac death in childhood, while others deem it unreliable and only necessary in patients with a family history of LQTS or in those presenting with clinical symptoms of cardiac dysfunction.[14]

LQTS is known to be an autosomal dominant disorder. While over 600 mutations have been found to affect 16 known LQTS genes, 3 of these genes make up 95% of genotype-positive LQTS and 75% of all diagnosed LQTS.10,12 LQT1 and LQT2 are related to gene mutations KCNQ1 and KCNH2, respectively, affecting potassium channel function. LQT3 is the result of the sodium channel gene mutation, SCN5A. Interestingly, literature has shown that each genotypic mutation has unique triggers that precipitate an event.[10,15-17] Sympathetic activation induced by exercise puts patients with LQT1 at increased risk of developing an arrhythmia, while patients with the LQT2 genotype experience cardiac events during emotional stress and waking from sudden noises during sleep. Patients with the LQT3 genotype can experience events during sleep or at rest without either emotional stress or stimulating arousal.

A variety of risk factors can contribute to a patient with LQTS experiencing a cardiac event. Risk increases in patients with a history of prior syncope or a QTc greater than 500 ms. Age and sex are also significant risk factors that impact pediatric patients. In a study conducted by Goldenberg et al, age related changes differed in patients with LQT1. Females had lower risk of cardiac events than males when less than 16 years of age (HR 0.58; p=0.005), but a higher risk between 16 and 40 years old (HR 3.35; p=0.007).[14,18] No difference in cardiac events was found between male or female LQT2 or LQT3 carriers less than 16 years of age, but LQT2 females had a significantly higher risk of developing their first cardiac event between 16 and 40 years old compared to male counterparts (HR 3.71; p=0.010). Results from Vink et al further support age-related differences of QTc intervals in children and adolescents with LQT1 and LQT2.[13] In males with LQT1, QTc interval was found to shorten until 20 years of age (p=0.02), while there were no changes seen in the female sample. Similar results were seen in males with LQT2, where QTc prolongation was evident until puberty (p=0.0001) at which time it began shortening. LQT2 female patients showed a similar trend based on age (p=0.003). However, there was no difference in risk of cardiac events between groups. A possible explanation for these age and sex-related differences is the changes in sex hormone levels during puberty leading to developmental changes in the QTc interval, though this theory has proven difficult to assess.[19]

Poor patient adherence is a well-documented risk factor for precipitating a cardiac event in patients with LQTS. In a single-center, prospective, observational study conducted by Ninomiya et al, it was found that non-adherence with medication treatment was a sole risk factor for frequent symptoms after diagnosis (p=0.02).[20] Non-adherence was defined as patients not taking their medicine for more than two days immediately before an LQTS-related cardiac event. Waddell-Smith et al most recently published a study in 2016 on long-term beta-blocker adherence in familial LQTS.[21] Adherence was assessed by calculating each patients' medication possession ratio (MPR). MPR is calculated by comparing the days' supply available to the patient based on refill history to a predetermined study period, typically 30 or 90 days. For example, if a patient receives a 30 day supply of medication with three refills and they are seven days late in picking up their third refill, then they only possessed an 83 day supply of medication during the 90 day period, giving them an MPR of 0.92. Adequate adherence in this study was defined as an MPR greater than or equal to 0.8, ideal being 1.0. Of the 68 patients included in the study, 51% (n=35) had suboptimal adherence with MPR less than 0.8, seven of which never filled a prescription. Patient rationale for non-adherence included disorganization, fear of adverse effects, and perceived low risk of having an event irrespective of cardiologist concerns. With the imminent risk of experiencing a cardiac event if not managed appropriately, pharmacists can play a critical role by assisting with patient compliance.

TABLE 1. QTc-Prolonging Medications Common to Pediatric Pharmacy[9]

QTc-Prolonging Medications Common to Pediatric Pharmacy	
Atomoxetine	Fluconazole
Amiodarone	Fluoxetine
Azithromycin	Haloperidol
Ciprofloxacin	Levofloxacin
Citalopram	Methadone
Diphenhydramine	Ondansetron
Erythromycin	Quetiapine
Escitalopram	Risperidone

Adapted from information obtained from CredibleMeds

Disease-States Associated with QTc Prolongation

Attention-deficit hyperactivity disorder (ADHD) is the most commonly diagnosed pediatric neurobehavioral disorder, occurring in 8% of children.[22] With 3.5 million children prescribed stimulants annually in the United States, it is likely that many pharmacists provide care to patients with ADHD on a regular basis.[23] In the recent past, concerns have been raised regarding the safety of ADHD medication therapies in children due to reports of sudden cardiac death. Stimulants were associated with 6-15% of all pediatric sudden cardiac deaths and it is estimated that a sudden cardiac death occurs for every 4.5 million methylphenidate prescriptions filled.[24] This is of particular concern as congenital heart disease is a risk factor for sudden cardiac death and ADHD is more common in children with congenital heart disease, occurring in two thirds of patients with hypoplastic left heart syndrome and in 50% of children with total anomalous pulmonary venous return.[4] This combined information prompted the United States Food and Drug Administration (FDA) to consider addition of a black box warning for possible cardiovascular risks associated with stimulant medications. However, at the recommendation of the FDA Pediatric Advisory Committee, this warning was not pursued. Similarly, the American Heart Association (AHA) released recommendations in 2008 addressing cardiovascular monitoring in children receiving medication treatment for ADHD.[4] These recommendations include obtaining an EKG prior to prescribing stimulants, atomoxetine, clonidine or guanfacine in order to detect patients with LQTS and were based on evidence of successful implementation of population screening in Japan, Italy and a variety of athletic associations in the United States. Additionally, the AHA recommends a repeat EKG at 12 years of age given the changes in QTc that can occur with puberty as previously discussed.

Since the release of the AHA recommendations, several studies have been published to evaluate the cardiovascular effects of many of the medication therapies for ADHD alone and in combination. A prospective, controlled study by Karpuz and colleagues of 285 children evaluated EKGs in children without ADHD, children with ADHD not receiving medication therapy, those treated with methylphenidate, those treated with risperidone and those receiving combination therapy with methylphenidate and risperidone.[25] Notably, this study excluded patients with any history or symptoms of cardiovascular disorders and evaluated patients after three months of medication therapy. QTc was found to be significantly longer in all treatment arms compared to untreated ADHD and healthy controls ($p<0.001$). However, there were no arrhythmias or ST changes associated with this finding. Similarly, Sayer and colleagues performed a randomized, parallel group trial comparing cardiovascular effects of guanfacine monotherapy, dexmethylphenidate extended-release monotherapy and combination therapy in ADHD patients between 7 and 14 years old.26 Again, patients with personal or family history of cardiovascular disorders were excluded. There were no significant changes in QTc in any group and the authors concluded that these ADHD medications do not have any adverse effects on cardiovascular health. In a systematic review by Stiefel and colleagues, there was weak and inconsistent evidence that methylphenidate, amphetamines and atomoxetine can cause EKG changes in pediatric patients with ADHD, but these changes were not considered clinically significant.[24] This prompted the AHA and American Academy of Pediatrics (AAP) to release a joint statement walking back the AHA's previous guidelines noting that it is reasonable, but not mandatory, to obtain an EKG in ADHD patients prior to initiating medication therapy.[27]

Another population at increased risk of ventricular arrhythmias associated with QTc prolongation are post-surgical pediatric congenital heart disease patients. Congenital heart defects associated with cardiomyopathy and ventricular surgery, particularly single ventricle physiology, are known to be arrhythmogenic and patients commonly require pacing and anti-arrhythmic medication management post-operatively.[28] Additionally, many therapies utilized in the post-operative period, such as epinephrine and steroids, can be pro-arrhythmic and electrolyte abnormalities are common. In this high risk population, it is essential that pharmacists consider the cumulative likelihood of QTc prolongation and ventricular arrhythmias associated with the patients' medication regimens, drug-drug interactions and current condition.

Only a limited number of QTc-prolonging medications have been specifically evaluated in critically ill pediatric patients. Two case reports describe adolescent males with sepsis developing prolonged QTc and arrhythmias with fluconazole and ciprofloxacin.[29,30] Fluconazole was associated with documented TdP while ciprofloxacin induced bradycardia. The patients had no other documented risk factors for QTc prolongation although it is possible there was end organ dysfunction associated with their sepsis which was not reported. In a small, single-centered, retrospective study investigating the safety of quetiapine for treatment of delirium in the pediatric intensive care unit, 19% (n=3) of EKGs obtained revealed QTc prolongation.[31] However, none of these abnormalities resulted in arrhythmias and QTc prolongation resolved spontaneously or with dose reduction of quetiapine. Although large studies have demonstrated that ondansetron alone is infrequently associated with QTc prolongation or arrhythmias,[3,32-34] these studies were performed in the general pediatric population and, often, excluded patients with congenital heart disease. In a retrospective cohort study of pediatric intensive care patients, 40% (n=57) of patients experienced QTc prolongation following ondansetron administration.[35] Electrolyte abnormalities, end organ dysfunction and concomitant use of other QTc-prolonging medications displayed a statistically significant association with the occurrence of QTc prolongation ($p<0.05$) and patients with three or more risk factors for QTc prolongation were significantly more likely to develop QTc greater than 500 ms ($p<0.05$). However, no patients in the study experienced arrhythmias or TdP.

The evidence related to methadone use in critically ill pediatric patients is mixed. In a retrospective, cohort study of 64 pediatric patients admitted to the

pediatric intensive care unit, 21 children (33%) developed QTc prolongation following methadone initiation.[36] Of note, 61% (n=39) of the study population had congenital heart disease putting them at higher risk of arrhythmia, but none of the instances of QTc prolongation resulted in TdP or any other type of arrhythmia. A limitation of the study was that the median time to EKG following methadone initiation was only 5 days. This may be an explanation for the differing results found by Schwinghammer et al in a retrospective, cohort study performed at University of California Davis Children's Hospital.[37] QTc prolongation was identified in 50.6% (n=45) of the study population and occurred more frequently in patients with cardiac disease and end organ dysfunction although these results were not statistically significant. Of interest, methadone is often used for prevention and management of iatrogenic withdrawal from opioids at the research institution. In evaluating this type of regimen on QTc prolongation, the authors found that QTc prolongation was more likely to occur with higher maximum methadone dose (OR 2.56; 95% CI 1.15-5.7), but that, in 48.9% (n=22) of patients with QTc prolongation, the longest measured QTc occurred during methadone tapering. Additionally, patients who received more days of methadone therapy were more likely to develop QTc prolongation (OR 1.03, 95% CI 1.01-1.05). This demonstrates that QTc prolongation associated with methadone is not necessarily dose-dependent, but related to cumulative methadone exposure likely due to known accumulation of the drug over time and that patients remain at risk of QTc prolongation even with decreasing doses of methadone. In each of these studies a significant proportion of patients were excluded from enrollment due to lack of QTc monitoring, this exhibits an opportunity for pharmacists to identify patients at risk of QTc prolongation and request obtaining EKGs appropriately.

Management of LQTS and Torsade de Pointes

For patients with familial LQTS, lifelong pharmacological management has been proven to reduce cardiac event rates. Beta-blockers are the treatment option of choice per the American College of Cardiology, American Heart Association and European Society of Cardiology co-authored guidelines.[12,38] They are recommended as a class 1 indication for all patients with a clinical diagnosis of LQTS and a class IIa indication for asymptomatic patients.[38] Beta-blockers are believed to diminish the adrenergic-mediated triggers that could otherwise precipitate an event. Studies have shown that beta-blockers reduce the rate of syncope in all patients with LQTS, however a significant reduction in cardiac event rates was evident in LQT1 and LQT2, but not LQT3 patients.[39] Also, patients who were symptomatic prior to starting beta-blocker therapy were 5.8 times more likely experience a recurrent cardiac event compared to asymptomatic patients. Unfortunately, randomized, prospective trials have not been conducted and, therefore, the impact of beta-blocker

use on mortality is yet to be established and there is not data available to conclude which beta-blocker is preferred over the others.

In the event of cardiac arrest or LQTS patients who are deemed high-risk, ICD therapy is a class 1a indication. For patients who are deemed high-risk and ICD therapy is contraindicated, a left cardiac sympathetic denervation is considered a class 2a indication.[10,12,38] Non-pharmacologic methods should be considered adjunct to medication therapy in order to further reduce risk of inciting a cardiac event in patients with LQTS. Patients should avoid adrenergic-type stimuli such as alarm clocks, loud doorbells, and ringtones.[10] Patient education is also critical to avoid use of QTc-prolonging medications (Table 1) and to check with a pharmacist or cardiologist before taking over-the-counter medications.[40] Immediate family should also be educated on how to properly respond to fainting episodes in order for the patient to receive prompt medical attention.

Torsade de pointes is a potentially fatal result of an untreated prolonged QT interval. It can cease spontaneously or further develop into ventricular fibrillation. Patients that are hemodynamically unstable, or that become unresponsive or pulseless, should be managed according to standard resuscitation algorithms. This generally involves electric cardioversion and intravenous (IV) magnesium. In stable patients, it is imperative to initiate therapy promptly as they can decompensate rapidly. Whether it is for a single episode of TdP or for prevention of recurrent TdP, IV magnesium is a proven first-line therapy for initial management.[12,40-44] Pediatric dosing ranges from 25 to 50 mg/kg, with a max of 2 grams. Management of TdP also involves amending any contributing factors such as correcting electrolyte derangements or discontinuing medications known to prolong the QT interval.[12] Overdrive pacing is an option for patients refractory to stabilization with magnesium whether it is done mechanically via reprogramming an already existing implantable cardioverter-defibrillator (ICD) or medically via the titration of an isoproterenol continuous infusion to achieve a heart rate of 100 beats per minute in order to increase sinus heart rate and decrease the QT interval.[12,40,41]

The Pharmacist's Role

Pharmacists have the opportunity to ensure that the risks associated with QTc prolongation in pediatric patients are adequately assessed, properly monitored and mitigated when possible. This concept is strongly supported in the adult literature. In a study conducted in the cardiac intensive care and intermediate care units at Indiana University Health Methodist Hospital, Tisdale and colleagues saw a 35% reduction in the odds of QTc prolongation with implementation of a best practice alert for pharmacists in the electronic health record.[46] The alert was designed to notify pharmacists when QTc-prolonging medications were prescribed for patients at moderate to high risk of QTc-prolongation based on a validated risk assessment tool previously developed by the authors.[46,47] Pharmacists were then prompted to either recommend alternative medication therapy, suggest increased monitoring of EKG and electrolytes or override the alert with no further action. Unfortunately, despite the tool being highly selective, 82% (n=382) of the best practice alerts were overridden with only 13% (n=51) resulting in increased monitoring and 17.9% (n=84) resulting in discontinuation of the prescribed medication. Although this research documents a pharmacist's positive impact on clinical outcomes, it highlights the significant risk of alert fatigue when implementing strategies to identify patients at risk of QTc prolongation. Development of a pharmacist-driven algorithm has been found to be an effective strategy to ensure adequate QTc monitoring and reduce the risk of QTc prolongation in adult patients. Implementation of such a strategy at Barnabas Health Behavioral Health Center, an acute care adult psychiatric hospital in New Jersey, not only increased EKGs obtained in patients at risk of QTc prolongation by 26%, but also reduced EKGs obtained unnecessarily by a similar amount.[48] Additionally, the authors saw a significant increase in pharmacist involvement in management of QTc prolongation from approximately 6 to nearly 98 interventions monthly. Similarly, Ng and colleagues found a 50% reduction in all QTc prolongation and a 10-fold reduction in incidence of QTc greater than 500 ms after implementation of a pharmacist-driven algorithm in the medical intensive care and intermediate care units at University of Southern California Medical Center.[49] These outcomes were achieved without any evidence of increased hospital length of stay or health-care costs. The results observed at these institutions demonstrate the ability of pharmacists to ensure cost-effective use of healthcare resources which is invaluable in this time of rising healthcare costs.

Predictably, the pediatric evidence available on this topic is lacking and lags behind that of our adult colleagues, but shows promise. In a study by Hutchins and colleagues in 2017, a QTc monitoring protocol was developed by a pediatric cardiologist and clinical pharmacist which suggested obtaining a baseline EKG for patients on 3 or more QTc prolonging medications and a follow up EKG for patients remaining on that regimen for 5 or more days.[50] The authors found that a follow up EKG was obtained in all patients that met criteria after the protocol was implemented compared to approximately 50% (n=11) of eligible patients during the pre-intervention period. Additionally, documented pharmacist interventions increased from 1.5 to 8 recommendations weekly with use of the protocol. Both of these findings were statistically significant ($p<0.05$). In distributing surveys to a variety of pediatric institutions, the authors discovered that only 11% (n=6) of respondents had policies or procedures related to medication-induced QTc prolongation monitoring in place and only half of institutions utilized any kind of alert in the electronic medical record for pharmacists, prescribers or nurses to review. This displays another opportunity for pharmacists to influence patient care and ensure safe medication monitoring practices at their institution through development, implementation and promotion of a policy or protocol addressing QTc prolongation and the risk of TdP.

It is not only hospitalized pediatric patients that could benefit from pharmacist involvement in QTc prolongation risk assessment. Many medication therapies

utilized for pediatric disease states treated in the outpatient setting, such as ADHD and depression, carry a risk of QTc prolongation. The AHA and AAP recommend that children prescribed medication therapy for treatment of ADHD be carefully assessed for heart conditions, including a thorough interview gathering the patient's past medical history and family history.[27] A family history of cardiomyopathy, LQTS, Wolff-Parkinson-White syndrome or Marfan syndrome warrant a referral to a pediatric cardiologist.[4] It would be prudent for pharmacists to include this evaluation when counseling patients and their families on new stimulant and atypical antipsychotic prescriptions. Additionally, outpatient pharmacists have the opportunity to identify patients at higher risk of TdP that may benefit from EKG monitoring such as patients with history of LQTS and those being treated with multiple QTc-prolonging medications.

Conclusion

Although occurring less frequently than in adults, pediatric patients remain at risk of QTc prolongation and appropriate monitoring is essential in identifying patients with LQTS in order to prevent severe cardiac events such as sudden cardiac death secondary to TdP. Pediatric patients receiving QTc-prolonging medications should be evaluated for other factors known to contribute to QTc prolongation such as cardiac conditions, electrolyte abnormalities, drug-drug interactions, end organ dysfunction affecting drug metabolism and additional medication therapies that may prolong QTc. Patients with LQTS require education on their significant risk of a cardiac events, the importance of medication compliance and how to manage their condition both pharmacologically and non-pharmacologically. Given the rarity of protocols related to QTc monitoring in pediatric institutions, pharmacists are also needed to establish these types of policies in order to promote institutional changes within the health system. These are all areas of opportunity for both inpatient and outpatient pharmacists to have a valuable impact on patient safety in preventing adverse outcomes.

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Assessment Questions

1. **True or False:** The United States Food and Drug Administration currently requires a "black box" warning for sudden cardiac death on all stimulant medications.
   a. True
   b. False

2. The American Heart Association suggests that it is reasonable to obtain an electrocardiogram prior to initiation of which ADHD medication therapy or therapies?
   a. Methylphenidate
   b. Guanfacine
   c. Clonidine
   d. Atomoxetine
   e. All of the above

3. According to the American Heart Association, a family history of which of the following disease states does NOT require evaluation by a pediatric cardiologist prior to initiating medication therapy for ADHD?
   a. LQTS
   b. Cardiomyopathy
   c. Congenital Heart Disease
   d. Wolff-Parkinson-White Syndrome

4. **True or False:** Pre-pubescent LQT1 females are at higher risk of experiencing a cardiac event than pre-pubescent LQT1 males.
   a. True
   b. False

5. A 17 year old female patient with LQTS weighing 60 kilograms arrives at the EDTC after experiencing an episode of syncope. Her EKG is showing signs of torsade de pointes. The physician would like to give a dose of magnesium to stabilize the patient. What dose do you recommend?
   a. 50 mg/kg of magnesium sulfate, max dose of 3000 mg
   b. 20 mg/kg of magnesium sulfate, max dose of 1500 mg
   c. 50 mg/kg of magnesium sulfate, max dose of 2000 mg
   d. 15 mg/kg of magnesium sulfate, max dose of 1000 mg

6. **True or False:** The CredibleMeds QT Drug List indicates which QTc-prolonging medications cannot be administered concurrently.
   a. True
   b. False

7. **True or False:** If a patient develops torsade de pointes, the recommended first line treatment is an isoproterenol continuous infusion.
   a. True
   b. False

8. **True or False:** The mechanism by which many medications prolong the QT interval is by blocking the delayed potassium rectifier channels in the cardiac myocyte.
   a. True
   b. False

9. Which of the following are risk factors for QTc prolongation?
   a. Congenital heart defects
   b. LQTS
   c. Tachycardia
   d. A & B

10. Did the activity meet the stated learning objectives? (if you answer no, please email sarahs@pswi.org to explain)
    a. Yes
    b. No

11. On a scale of 1 – 10 (1-no impact; 10-strong impact), please rate how this program will impact the medication therapy management outcomes or safety of your patients.

12. On a scale of 1 – 10 (1-did not enhance; 10-greatly enhanced), please rate how this program enhanced your competence in the clinical areas covered.

13. On a scale of 1 – 10 (1-did not help; 10-great help), please rate how this program helped to build your management and leadership skills.

14. How useful was the educational material?
    a. Very useful
    b. Somewhat useful
    c. Not useful

15. How effective were the learning methods used for this activity?
    a. Very effective
    b. Somewhat effective
    c. Not effective

16. Learning assessment questions were appropriate.
    a. Yes
    b. No

17. Were the authors free from bias?
    a. Yes
    b. No

18. If you answered "no" to question 17, please comment (email info@pswi.org).

19. Please indicate the amount of time it took you to read the article and complete the assessment questions.

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November/December 2019
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Activity Type: Knowledge-based
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Quiz Answer Form

circle one answer per question

1) a b
2) a b c d e
3) a b c d
4) a b
5) a b c d
6) a b
7) a b
8) a b
9) a b c d
10) a b
11)_____
12)_____
13)_____
14) a b c
15) a b c
16) a b
17) a b
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