BRITISH COLUMBIA CENTRE ON SUBSTANCE USE

Medetomidine Webinar Q&A

This document contains questions and answers from the BCCSU's *Clinical Management of Medetomidine-related Presentations* webinar, held on May 11, 2026. This webinar is aimed at acute care clinicians providing care to people experiencing suspected medetomidine intoxication and withdrawal. There is a lack of published evidence on the management of medetomidine-related presentations, and the approaches to care provided within this document are based on clinical experience. This document is not a substitute for the professional judgment of a health care professional. Health care professionals must take into consideration the circumstances of the patient and all applicable laws, regulations, and standards, including those set by relevant governing bodies. The authors make no representation of warranty, and assume no liability, for the contents of this document.

Clinicians are encouraged to consult the 24/7 Addiction Medicine Clinician Support Line (788-945-7619) for clinical decision-making around medetomidine-related presentations and management approaches.

Q1: Can sedated patients who have been transported to the ED due to suspected medetomidine exposure be safely transferred to a community site for monitoring? What if that site does not have clinical monitoring capabilities or regulated providers?

A: Sedated patients at risk of medetomidine withdrawal should remain monitored in a clinical setting.

Q2: For sedated patients in the ED, what would signal the need for escalated level of care (e.g., ICU)?

A: Patients likely do not require a higher level of care if they are awake, not vomiting, and are tolerating oral clonidine; or, if they are sedated with well-controlled vital signs. Patients with reasonably controlled heart rate and blood pressure who are not vomiting can be treated aggressively with oral clonidine (0.3mg-0.5mg every 6 hours) to prevent needing an escalated level of care. Patients being administered anti-emetics require a level of care that can accommodate cardiac monitoring for QTc prolongation risk. Sedated patients with severe, non-responsive vomiting and persistent hypertension or tachycardia likely require an escalated level of care.

Q3: Can you see just one hallmark symptom of medetomidine withdrawal, or is it usually a triad of hypertension, tachycardia, and vomiting?

A: The triad is most common; however, other combinations are possible. There is significant variation in severity of withdrawal, considering individual pharmacokinetics, route of administration, other substances used, and other factors.

Q4: How do benzodiazepines factor into clinical presentations and management of medetomidine withdrawal?

A: A person can present with one or more withdrawal syndromes, including benzodiazepine withdrawal; and due to overlapping symptoms, it may be easy to overlook one of these withdrawal syndromes. However, each withdrawal syndrome requires appropriate, and sometimes distinct, treatment. In approaching treatment, consider how each medication will affect the patient's overall clinical syndrome. For patients with known benzodiazepine use, phenobarbital can be used to pre-emptively treat benzodiazepine withdrawal. Patients typically tolerate phenobarbital plus methadone/morphine and/or dexmedetomidine/clonidine well.

Q5: Does co-use of stimulants or stimulant withdrawal impact the clinical picture and clinical management of medetomidine intoxication or withdrawal at all?

A: Methamphetamine intoxication can complicate assessment of hypertension because a person can have methamphetamine-induced hypertension at the same time as medetomidine and opioid withdrawal. A stimulant "crash" can also contribute to hypoactive mental status seen in medetomidine withdrawal.

Q6: Is a previous episode of severe medetomidine withdrawal predictive of subsequent episodes? Is there anything that suggests risk of severe medetomidine withdrawal?

A: There is a tendency for patients who have previously experienced severe medetomidine withdrawal to be more likely to experience it again; however, this observation may be confounded by provider awareness of a patient's previous admission to the ED/ICU due to medetomidine withdrawal, which may result in rapid initiation of aggressive treatment. Patients who use a larger quantity of dope are often more at risk of severe medetomidine withdrawal; however, sometimes patients who use a small quantity of dope can need ICU-level care, whereas some who use a larger quantity may not.

Q7: What is the role of atropine in the management of medetomidine-related bradycardia?

A: If other vital signs are stable and heart rate >40bpm (many patients may be stable with a baseline heart rate ~40bpm), no intervention is likely necessary as bradycardia typically lasts ≤5 hours. Atropine is rarely recommended by addiction medicine for medetomidine withdrawal and is typically only warranted if there is indication of impending cardiac arrest.

Q8: What role does ondansetron (brand name Zofran) have, especially when considering use among tachycardic patients?

A: There has not been much observed clinical benefit with ondansetron for medetomidine-related nausea and vomiting. D2 antagonists (e.g., prochlorperazine, olanzapine) have been much more effective. However, there is generally no reason to avoid ondansetron solely based on tachycardia.

Q9: If a patient is hypotensive and experiencing medetomidine withdrawal, is there concern about treating with clonidine given it can further decrease blood pressure?

A: Discrepancy between heart rate and blood pressure trends can be seen when treating with clonidine; however, these are usually brief. The primary treatment challenge is usually with severe hypertension and bradycardia; tachycardia and hypotension together is less common with medetomidine withdrawal. In these rare situations, clinicians can consider other alpha agonists such as guanfacine. Guanfacine has a better safety profile than clonidine and is associated with less severe bradycardia and hypotension.

Q10: What is the recommended approach to alpha agonists for patients who cannot tolerate oral medication due to vomiting, particularly when trying to avoid dexmedetomidine, or in areas without access to dexmedetomidine?

A: The suggested approach is to use anti-emetics first: Administer IV olanzapine or prochlorperazine to allow for oral tolerance. After ~30 minutes of IV anti-emetics, administer a loading dose of clonidine (with dose based on patient's systolic blood pressure) and monitor for tolerance and effect. Transdermal alpha agonists have not been found helpful since the dose is usually quite low, and it takes ~12-24 hours to absorb the dose.

Q11: What do monitoring protocols look like in the ED and on the ward, for both medetomidine intoxication and withdrawal? How often are vital signs checked, and should the patient be on telemetry?

A: Telemetry isn't required unless a patient becomes severely hypertensive, tachycardic, or has QT prolongation. Vital signs are recommended every 4 hours for 24h, alongside COWS, POSS, and RASS. Also monitor for myocardial ischemia or changes in myocardial function.

Q12: How do you do a cross-titration from dexmedetomidine infusion to clonidine dosing?

A: This is typically approached on a case-by-case basis, considering timeframe and symptom control.

- Typically, if an individual has:
  - Been on dexmedetomidine for 24-48 hours *and*
  - They have been stabilized at a controlled dose for at least 24h *and*
  - Their systolic blood pressure is <160mmHg with a normal heart rate *and*
  - They are tolerating oral meds, talking, and eating,

  they would be well enough to down-titrate dexmedetomidine.
- Down-titration of dexmedetomidine infusion is very slow (to prevent rebound nausea and vomiting), at 0.2mcg/kg/hr every 1-2 hours.
- Oral clonidine is typically started at 0.2mg-0.3mg every 6 hours and up-titrated by 0.1mg each dose, as dexmedetomidine infusion is decreased, with a maximum clonidine dose of 0.4mg-0.6mg every 6 hours.
- If the patient begins vomiting, increase the dexmedetomidine infusion rate (consider administering a dexmedetomidine bolus).
  - If systolic blood pressure increases by more than ~10-15mmHg, pause the down-titration of dexmedetomidine infusion—typically for 2-6 hours or potentially longer, depending on magnitude of vital signs changes and symptoms—and re-stabilize.
  - Consider increasing clonidine or adding anti-emetics prior to resuming down-titration of dexmedetomidine infusion.

Q13: What is the suggested approach to initial dosing for clonidine outpatient management?

A: Suggestion is to start with 0.2mg-0.3mg oral clonidine (twice daily or three times daily, based on risk assessment and response to first dose), depending on the patient's history of withdrawal and blood pressure.

Q14: What are the expected complications if a patient returns to non-prescribed substance use whilst on an outpatient clonidine taper?

A: There is a theoretical risk of harm should someone continue taking clonidine as well as other unregulated drugs (i.e., from drug poisoning or related complications). Patients should be counseled to stop taking clonidine should they return to non-prescribed substance use; however, patients should also be advised of the risk of rebound hypertension if clonidine is abruptly discontinued.