Possible central anticholinergic syndrome following transdermal scopolamine in an ambulatory surgery patient

John A. Norton, DO · Babak Khabiri, DO ·
Fernando L. Arbona, MD · Alan J. Kover, MD

Received: 19 April 2011 / Accepted: 14 September 2011 / Published online: 12 October 2011
© Canadian Anesthesiologists' Society 2011

To the Editor,

We share an interesting case wherein use of a scopolamine transdermal patch for postoperative nausea and vomiting (PONV) prophylaxis may have contributed to the delayed emergence of an ambulatory surgery patient. The patient gave written consent for publication of this article.

A 28-yr-old female (height 1.65 m, weight 90 kg) with a history of obsessive-compulsive disorder treated with citalopram presented for elective outpatient ankle arthroscopy. On a previous occasion, she had experienced severe PONV following a similar procedure performed under general anesthesia. A decision was made to administer a regional technique (sciatic and saphenous nerve blocks) combined with an intravenous general anesthetic using propofol. In the preoperative holding area, a transdermal scopolamine patch 1.5 mg was placed behind the patient's left ear for additional PONV prophylaxis.

Following application of routine monitors and block placement, the patient was administered propofol $200 \mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ *iv* which was titrated down to $150 \mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ during the procedure. Oxygen was administered via nasal cannula at $2 \text{ L}\cdot\text{min}^{-1}$ while ventilation was monitored via capnography. For additional PONV prophylaxis, dexamethasone 8 mg *iv* and later ondansetron 4 mg *iv* were administered intraoperatively.

After approximately 45 min of surgery, the patient's heart rate began to increase (from $80 \text{ beats}\cdot\text{min}^{-1}$ baseline to $110 \text{ beats}\cdot\text{min}^{-1}$), and fentanyl $100 \mu\text{g}$ *iv* was administered slowly for suspected pneumatic tourniquet pain.

Following completion of the one-hour procedure and deflation of the tourniquet, the patient's heart rate remained elevated. Administration of propofol was discontinued approximately 15 min prior to room departure, and she was moved to the recovery area with continued monitoring while breathing spontaneously—though unresponsive to either verbal or tactile stimulation.

Ten minutes after her arrival in the postanesthesia recovery unit, the patient remained unresponsive with an elevated heart rate of $140 \text{ beats}\cdot\text{min}^{-1}$ and blood pressure of $120/70 \text{ mmHg}$. The patient's oxygen saturation was 100% with nasal cannula oxygen, and on examination, her pupils revealed bilateral mydriasis, nearly unresponsive to light, and a disconjugate gaze. A gag reflex was also present. Over the next 30 min, blood gas, electrolyte, and blood glucose readings were ordered, with normal results, while her electrocardiogram showed sinus tachycardia. Consideration was given to the possibility of central anticholinergic syndrome, and the scopolamine patch was removed after being in place for approximately two hours.

Our hospital's internal medicine colleagues, who had been notified relatively early in the case, had begun preparations for further neurological evaluation, workup, and intensive care unit admission. A decision was made to administer physostigmine for possible diagnostic and therapeutic intervention. Over the next 30 min, physostigmine 1.5 mg *iv* was administered in 0.5 mg increments. Within minutes of administering the last 0.5 mg dose, the patient became responsive and followed commands. Her blood pressure remained stable while her heart rate decreased to her baseline. The patient was admitted to hospital overnight for observation, and she was discharged the next morning with complete resolution of the suspected anticholinergic signs and without further sequelae.

J. A. Norton, DO (✉) · B. Khabiri, DO ·
F. L. Arbona, MD · A. J. Kover, MD
The Ohio State University, Columbus, OH, USA
e-mail: john.norton@osumc.edu

Transdermal scopolamine is efficacious and administered frequently as part of a multi-modal pharmacologic prophylaxis regimen in patients considered at high risk for PONV.^{1,2} We suspected that a central anticholinergic syndrome probably contributed to this patient's delayed emergence in the postanesthesia care unit, and in this case, our suspicion was peaked after excluding more likely causes of sympathetic excess, including hypoxemia, hypercapnia, and metabolic or acid-base disturbances. Serotonin syndrome has some similar nonspecific presenting features (mental status changes, accelerated heart rate), though it usually occurs in patients on multiple serotonergic agents and with other clinical signs, including hyperreflexia, nausea/vomiting, agitation, and hyperthermia in severe cases.³ Other causes of delayed emergence were entertained, including a psychiatric somatoform (conversion) disorder, but these were unlikely given resolution of her symptoms with administration of physostigmine. Ultimately, an anticholinergic drug interaction between scopolamine and the patient's concurrent use of citalopram may have played a role in the patient's delayed emergence. Citalopram and other selective serotonin reuptake inhibitors (SSRIs) can have centrally acting anticholinergic effects.^{4,5} In addition to the SSRIs, numerous other medications may have similar anticholinergic side

effects which may be significant. In light of the need to treat ambulatory patients with a PONV history preemptively, this case reaffirms the need to consider prophylactic treatment options carefully, especially in patients where the potential for centrally acting anticholinergic drug interaction may exist.

References

1. *Kranke P, Morin AM, Roewer N, Wulf H, Eberhart LH.* The efficacy and safety of transdermal scopolamine for the prevention of postoperative nausea and vomiting: a quantitative systematic review. *Anesth Analg* 2002; 95: 133-43.
2. *Gan TJ, Sinha AC, Kovac AL, et al.* A randomized, double-blind, multicenter trial comparing transdermal scopolamine plus ondansetron to ondansetron alone for the prevention of postoperative nausea and vomiting in the outpatient setting. *Anesth Analg* 2009; 108: 1498-504.
3. *Sporer KA.* The serotonin syndrome. Implicated drugs, pathophysiology and management. *Drug Saf* 1995; 13: 94-104.
4. *Burrows GD, McIntyre IM, Judd FK, Norman TR.* Clinical effects of serotonin reuptake inhibitors in the treatment of depressive illness. *J Clin Psychiatry* 1988; 49(Suppl): 18-22.
5. *Milne RJ, Goa KL.* Citalopram. A review of its pharmacodynamic and pharmacokinetic properties, and therapeutic potential in depressive illness. *Drugs* 1991; 41: 450-77.