Clinical Reports

Unrecognized bulimia nervosa: a potential cause of perioperative cardiac dysrhythmias

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Purpose: To report serious cardiac dysrhythmias in two patients whose bulimia nervosa was not revealed during preoperative screening.

Clinical Report: *Case #1:* A 25-yr-old woman with preoperative hypokalemia ($K^+ = 3.1 \text{ mEq}\cdot\text{L}^{-1}$) required anesthesia for removal of a wrist ganglion. She claimed the hypokalemia was of unknown etiology, and denied other medical problems. Shortly after induction of anesthesia with thiopental and isoflurane, the ECG revealed two runs of *torsades de pointes*. This was successfully treated by decreasing pulmonary ventilation, allowing $P_{ET}\text{CO}_2$ to increase from 32 to 45 mmHg.

Case #2: A 39-yr-old woman who denied any medical problems received propofol, rocuronium sevoflurane and N_2O during general anesthesia for breast augmentation. In the PACU, the patient complained of light-headedness, and the ECG revealed a heart rate of 44 bpm with P-R interval of 0.42 sec. Following 0.5 mg atropine, the heart rate increased but the P-R interval remained prolonged (0.36 sec) and the corrected Q-T interval was 0.51 sec. Treatment with 2.5 g MgSO_4 , 20 mEq KCl, and 9.4 mEq calcium gluconate *iv* normalized the Q-T interval, and decreased the P-R interval to 0.22 sec. Upon specific questioning, she admitted to a remote history of bulimia, but denied any bulimic behavior for the last 16 yr.

Conclusion: Two patients with histories of eating disorders failed to disclose this information during preoperative evaluation. Perioperative cardiac dysrhythmias developed in these patients, even though they claimed that eating behavior had returned to normal.

Objectif : Décrire la dysrythmie cardiaque sévère chez deux patients dont la boulimie (bulimia nervosa) n'avait pas été révélée pendant le dépistage préopératoire.

Résumé clinique : *Premier cas :* Une femme de 25 ans qui présentait de l'hypokaliémie préopératoire ($K^+ = 3,1 \text{ mEq}\cdot\text{L}^{-1}$) a eu besoin d'anesthésie pour l'excision d'un ganglion au poignet. Elle a prétendu que l'hypokaliémie était d'origine inconnue et a nié tout autre désordre médical. Peu après l'induction de l'anesthésie avec du thiopental et de l'isoflurane, l'ECG a indiqué deux séries de torsades de pointe. Le traitement efficace a consisté à diminuer la ventilation pulmonaire pour permettre à la $P_{ET}\text{CO}_2$ d'augmenter de 32 à 45 mmHg.

Deuxième cas : Une femme de 39 ans, sans problème médical avoué, a reçu du propofol, du rocuronium, du sévoflurane et du N_2O pendant l'anesthésie générale pour une mammoplastie d'augmentation. À la salle de réveil, la patiente s'est plainte d'étourdissement, et l'ECG a révélé une fréquence cardiaque de 44 bpm et un intervalle P-R de 0,42 s. Après l'administration de 0,5 mg d'atropine, la fréquence cardiaque a augmenté mais l'intervalle P-R est demeuré long (0,36 s); l'intervalle corrigé Q-T était de 0,51 s. Le traitement avec 2,5 g de MgSO_4 , 20 mEq de KCl, et 9,4 mEq de gluconate de calcium *iv* a normalisé l'intervalle Q-T et diminué l'intervalle P-R à 0,22 s. En réponse à des questions spécifiques, elle a reconnu une ancienne histoire de boulimie, mais a nié tout comportement boulimique depuis les 16 dernières années.

Conclusion : Deux patientes présentant des antécédents boulimiques n'ont pas dévoilé ce renseignement pendant l'évaluation préopératoire. Même si leur comportement face à la nourriture était, ont-elles dit, revenu à la normale, elles ont subi des dysrythmies cardiaques.

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BULIMIA nervosa is an eating disorder characterized by periodic binges of overeating along with self-induced vomiting, laxative, or diuretic abuse. More than 90% of cases occur in women, with peak incidence between 14 and 18 yr of age.¹ Bulimia may be associated with electrolyte abnormalities as well as protein-calorie malnutrition, both of which may predispose patients to cardiac dysrhythmias. *Torsades de pointes* ventricular tachycardia has been reported in patients undergoing medically supervised weight reduction with liquid protein diets.^{2,3} We report two cases of perioperative cardiac dysrhythmia in patients with a history of bulimia nervosa that was not revealed by routine preoperative evaluation.

Report of Two Cases

Case #1

A 25-yr-old woman required excision of a wrist ganglion. She was 145 cm tall and weighed 50 kg. Six weeks earlier, she was seen by an endocrinologist because of cold extremities and "weakness" which were attributed to hypokalemia; her serum potassium concentration was 2.4 mEq·l⁻¹. She was instructed to eat foods rich in potassium. Repeat determination on the day of surgery revealed that the potassium concentration had increased to 3.1 mEq·l⁻¹. Because she was young, otherwise healthy, and had no history of palpitations or other cardiac symptoms, we did not obtain a preoperative ECG. Her only medications were oral contraceptives and cefalexin; the latter because of recent dental restorations.

The patient refused regional anesthesia. General anesthesia was induced with 350 mg thiopental and deepened with inhaled isoflurane (up to 4% inspired) *via* facemask. Shortly after induction of anesthesia, the heart rate increased from 58 to 90 bpm. Her Q-T interval was 0.43 sec (Q-T_c = 0.54 sec), and the ECG monitor showed two three-beat runs of ventricular tachycardia with a typical *torsades de pointes* pattern (Figure 1). These resolved spontaneously and did not recur. To minimize the risk that hyperventilation-induced hypokalemia might provoke a recurrence of the dysrhythmia, we allowed the patient to breathe spontaneously, and P_{ET}CO₂ increased from 32 to 45 mmHg. The patient awakened from anesthesia and was taken to the PACU in stable condition. In the PACU, the heart rate gradually decreased from 69 to 42 bpm, but there were no dysrhythmias or heart block. Systolic blood pressure remained between 90 and 110 mmHg. Upon specific questioning in the PACU, the patient admitted to a history of bulimia nervosa, including self-induced vomiting, laxative, and

diuretic use. However, she denied any bulimic behavior during the three months prior to surgery.

Case #2

A 39-yr-old, 60 kg woman required general anesthesia for breast augmentation surgery. She denied having any medical problems. Preoperative hemoglobin and potassium concentrations were 13.3 g·dl⁻¹ and 4.6 mEq·l⁻¹, respectively. Following premedication with midazolam, anesthesia and relaxation were induced with 200 mg propofol and 50 mg rocuronium. Anesthesia was maintained with sevoflurane (approximately 1% inspired) and N₂O 50% in O₂. The patient also received 100 µg fentanyl and 10 mg metoclopramide intraoperatively. During the 75-min operation, the surgeon injected 140 ml of a solution containing 4.4 mg lidocaine, 0.6 mg bupivacaine, and 4 µg epinephrine per ml. The ECG revealed normal sinus rhythm at a rate of 50-60 bpm with normal intervals throughout. At the end of surgery, neuromuscular blockade was reversed with 2.5 mg neostigmine and 0.5 mg glycopyrrolate. Upon awakening, the trachea was extubated and she was taken to the PACU.

On arrival in the PACU, the patient's heart rate was 51 bpm with normal intervals observed on the ECG monitor. After approximately 30 min, the patient complained of nausea and received 4 mg ondansetron *iv*. Five minutes later, she required 50 µg fentanyl *iv* for pain control. Shortly thereafter, the patient complained of light headedness. At this time her blood pressure was 108/66 mmHg, and her heart rate was 44 bpm with a P-R interval of 0.42 sec (Figure 2a). Following 0.5 mg atropine, the heart rate increased to 86 bpm with a P-R interval of 0.21 sec (Figure 2b). Fifteen minutes later, the P-R interval again increased to 0.36 sec despite the fact that the heart rate was 84 bpm (Figure 2c).

In addition to the first-degree AV block, a 12 lead ECG (Figure 3) revealed flattening of the T waves and a prolonged Q-T interval of 0.50 sec (Q-T_c = 0.51 sec). Serum electrolyte concentrations included K⁺ = 3.6 mEq·l⁻¹ (normal 3.6-4.8), Ca⁺⁺ = 1.16 mmol·l⁻¹ (1.21-1.33) and Mg⁺⁺ = 1.4 mg·dl⁻¹ (1.8-3.0). On this basis, we administered 2.5 g MgSO₄, 9.4 mEq calcium gluconate, and 20 mEq KCL slowly *iv*. Repeat 12-lead ECG revealed that the heart rate was 67 with a PR interval of 0.22 sec, and a Q-T interval of 0.37 s (Q-T_c = 0.39 sec).

In view of the abnormal ECG and serum electrolytes we asked the patient whether she had a history of eating disorder. After some hesitation, she admitted that at 14 yr she weighed 80 kg and with the help of a Weight Watcher's® support group, she

FIGURE 1 Intraoperative ECG tracing (lead II) from patient #1 showing two 3-beat runs of polymorphic ventricular tachycardia (*torsades de pointes*).

A

B

C

FIGURE 2 PACU ECG tracings from patient #2

A: Initial bradycardia (HR=44 beats/min) and first-degree A-V block (P-R = 0.42 sec)

B: Immediately after 0.5 mg atropine *iv*.

C: Fifteen minutes after 0.5 mg atropine *iv*.

FIGURE 3 12-lead ECG from patient #2, 20 min after atropine, demonstrating first-degree A-V block (P-R = 0.40 sec) and Q-T prolongation (Q-Tc = 0.51 sec)

reduced her weight to 58 kg by age 21 yr. During the subsequent two years, she exhibited bulimic behavior, using diuretics and purgatives to reduce her weight to 50 kg. She claimed to have discontinued this behaviour spontaneously at age 23, and adamantly denied any subsequent diuretic or laxative use.

Discussion

Perioperative dysrhythmias in patients with eating disorders have been rarely recognized, poorly characterized, and without ECG documentation.⁴ We have observed and documented *torsade de pointes* and serious atrioventricular block in two patients with eating disorders. Both patients failed to inform us of their history of bulimia. Retrospectively, the need for dental restoration in the first patient may have been related to the dental deterioration associated with self-induced vomiting.⁵ While she denied bulimic behaviour during the three months before surgery, this patient's hypokalemia suggests either continued covert bulimic activity or incomplete repletion of her previous electrolyte abnormalities. The decision to proceed with surgery despite a serum potassium concentration of 3.1 mEq·l⁻¹ was based on the observations of Vitez *et al.*⁶ who suggested that the risk of cardiac dysrhythmia is slight in otherwise healthy

patients when serum K⁺ is between 3.0 and 3.5 mEq·l⁻¹.

Acute weight loss through the use of liquid protein diets is associated with prolonged Q-T intervals and *torsades de pointes* ventricular tachycardia, which can sometimes result in sudden death.² Both patients demonstrated Q-T prolongation, and the first demonstrated salvos of *torsades de pointes*. This dysrhythmia may have resulted from electrolyte abnormalities (possibly exacerbated by manual hyperventilation) or to bulimia-induced changes in the cardiac conduction system. We attributed the postoperative bradycardia to her being a physically fit, active young adult. However, bulimia may also be associated with decreased resting heart rate and exaggerated respiratory sinus arrhythmia, suggesting augmentation of vagal tone.⁷ This, in turn, increases the likelihood of early after-depolarizations which prolong the Q-T interval and set the stage for *torsades de pointes*.⁸

In retrospect, we probably should have administered MgSO₄ at the first sign of polymorphic ventricular ectopy and subsequently initiated a dilute KCl infusion. Class IA antiarrhythmic drugs such procainamide, which may further prolong repolarization, are contraindicated in this situation, while lidocaine is usually ineffective. Refractory cases benefit from augmentation of the heart rate by overdrive pacing. However, since

the dysrhythmia was self-limited, and did not recur in the perioperative period we felt that close observation was the most appropriate course of action.

The second patient had a more remote history of bulimia, and her preoperative electrolyte screen was normal. The minimal decrease in serum potassium⁹ and magnesium concentrations observed during recovery may have been related to the administration of 0.5 mg epinephrine *sc* by the surgeon. The large doses of local anesthetics administered *sc* may have affected cardiac conduction; high doses of bupivacaine, but not lidocaine, tend to prolong the P-R interval.¹⁰ However, the total dose of bupivacaine which our patient received, 84 mg, is well below the 3 mg·kg⁻¹ recommended safe dose for peripheral nerve blocks, and epidural anesthesia.¹¹ Absorption of local anesthetics from subcutaneous tissues is particularly slow, and the resulting plasma concentrations tend to be low: "Tumescent anesthesia" with large doses of lidocaine (up to 55 mg·kg⁻¹) with epinephrine 1:2,000,000 *sc* has been shown to be safe with peak plasma concentrations < 4 µg·ml⁻¹.¹²

The most intriguing aspect of this patient's electrocardiographic abnormality was that she had recurrent first degree A-V block within a few minutes of receiving sufficient atropine to increase her heart rate to 84 bpm. Most previous reports suggest that both the bradycardia and the conduction system abnormalities of bulimia nervosa respond to vagal blockade.¹³ In contrast, Tarlet *et al.*¹⁴ describe a patient with atropine-resistant bradycardia and heart block. These findings persisted even after electrolyte balance and nutrition were restored, suggesting a more permanent conduction system abnormality. Since our patient denied recent bulimia, had normal serum electrolyte concentrations preoperatively, and had persistent first-degree heart block, it is possible that her conduction system was similarly affected.

In summary, eating disorders such as bulimia nervosa may be associated with perioperative cardiac dysrhythmias; this susceptibility may persist after eating behaviour has returned to normal. Since patients frequently fail to mention a recent or remote history of eating disorders during their preoperative interview, it is important that anesthesiologists have a high index of suspicion, particularly in patients who seem especially concerned with body image, exercise, and "perfection."¹ An unexpectedly low heart rate or prolonged P-R or Q-T intervals prior to induction of anesthesia provides another clue. Under these circumstances, the risk of *torsades de pointes* can be reduced by avoiding hyperventilation and bradycardia. Should dysrhythmias occur, determination of serum potassium, calcium, and magnesium concentrations are essential in guiding therapy.

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