Citalopram-induced ventricular tachycardia under general anesthesia

Sir,

Anesthesia-attributable cardiac arrests are mostly related to medication and airway management problems.[1] We report recurrent episodes of pulseless ventricular tachycardia (VT) under a seemingly uncomplicated general anesthetic in a patient with no comorbidities.

A 65-year-old, 55 kg women with a possible cholangiocarcinoma was undergoing a diagnostic staging and right portal vein embolisation before definitive surgery. She had no comorbidities but was on oral citalopram 20 mg daily for a stress disorder with no reported adverse effects.

Within the 2 weeks leading to this event, she had placement of a stent in the right bile duct, an endoscopic retrograde cholangiopancreatogram, an insertion of a percutaneous transhepatic cholangiography (PTC) catheter, and a staging laparoscopy, under multiple episodes of uneventful general anesthesia (GA). The PTC losses were replaced daily with intravenous crystalloids and supplementary oral feeds. She was on prophylactic fluconazole and tazocin. Her preoperative Hb was 10.6 g/l, INR 0.98, Na 139 mmol/l, K 3.8 mmol/l, urea 1.8 mmol/l, creatinine 51 μmol/l, corrected calcium 2.23 mmol/l, and magnesium 0.74 mmol/l. She was self-ventilating on room air, peripheral oxygen saturation (SpO_2) 99%, body temperature 37°C, heart rate 70/min, blood pressure (BP) 140/88, and Glasgow Coma Scale 15/15.

She was prehydrated with 500 ml gelofusine, preoxygenated, and was induced with fentanyl 2 mcg/kg, propofol 1.5 mg/kg, and atracurium 0.6 mg/kg followed by

uncomplicated endotracheal intubation and mechanical ventilation. Anesthesia was maintained with oxygen, nitrous oxide, and sevoflurane. Following 10 min of the commencement of the interventional procedure and stable GA, a spontaneous onset multifocal arrhythmia was noted which quickly progressed to a pulseless VT. She had no palpable central pulses, and advanced life support treatment was initiated. An immediate single DC shock of biphasic 160J returned a sinus rhythm with good peripheral pulse. She was given metaraminol 1 mg intravenously, and systolic BP stabilized to 120–130 mmHg. Procedure was recommenced while defibrillator pads were in place. After a further 15 min, she developed a second episode of VT of spontaneous onset, and this was successfully cardioverted. She received 100% oxygen supplemented with desflurane during these episodes. The procedure was completed during concurrent postresuscitative care. No potential contributory anesthetic factors were identified in a stepwise review.

During the recovery phase, two further self-limiting episodes of VT were observed. She regained consciousness and was extubated. No further organ support was required. On examination, she was alert and oriented. Her pupils were equal and reacted to light. Her Glascow coma score was 15/15. She displayed normal breathing pattern with SpO2 of 100% while on 40%oxygen. Heart rate was stable and peripheries well perfused. The postevent 1-h and 24-h troponin was 6 ng/l (normal <16 ng/l) and an echocardiogram did not reveal any abnormality. Twenty-four hours later, her electrocardiogram (ECG) showed slightly prolonged QTc, 450–460 ms. No further rhythm abnormalities were observed on 24-h monitor. Her citalopram was stopped, and metoprolol 2.5 mg once daily was commenced. She was counselled, and her partial hepatectomy was carried out a month later under an uneventful GA supplemented with epidural analgesia.

Her spontaneous onset pulseless VT under GA had no obvious etiology. She was, however, taking regular citalopram, a medication known to predispose select individuals for prolonged QT syndrome associated arrhythmia. Her ECG showed no evidence of prolonged QT preoperatively. She also had been fully optimized for her anesthetic.

Higher doses of citalopram increase the risk of QTc prolongation and torsades de pointes[2] and may be cumulative in hepatic impairment. Acquired long QT syndrome is most commonly caused by medications,[3] and citalopram has a dose-effect relationship. VT *per se* has not been reported in association with citalopram although there is a significant 90-day risk of a hospital encounter for ventricular arrhythmia following its prescription.[4]

There were no anesthetic events such as hypoxia, hypercapnia, or hypotension that could have predisposed her to VT. Sevoflurane is an unlikely trigger for cardiac dysrhythmias. Our suspicion was that citalopram may have shifted her cardiac susceptibility toward that of dysrhythmia in the presence of anesthetic agents. Such an anesthetic interaction has not been previously reported.

Therefore, through a process of exclusion, we infer citalopram-induced cardiac sensitivity to inhaled anesthetic agents, was the most likely cause of her cardiac arrhythmia. We conclude that citalopram may enhance cardiac susceptibility to life-threatening arrhythmias under GA even in the absence of a prolonged QTc in the ECG preoperatively.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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