Case Report

Development of torsade de pointes caused by exacerbation of QT prolongation during clipping of cerebral artery aneurysm in a patient with subarachnoid haemorrhage

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We report the case of a 79-yr-old woman with subarachnoid haemorrhage (SAH) in whom torsade de pointes (TdP) caused by worsening the QT prolongation occurred during clipping of cerebral artery aneurysm. This patient shows a potential risk of occurrence of life-threatening tachyarrhythmia, TdP by prolonging the QT interval during surgery in patients with SAH even with no additional factors that predispose to TdP. Therefore, a proper monitoring of the QT interval is necessary as a predictor of TdP. When ventricular tachyarrhythmia occurs, recognition of TdP is important because antiarrhythmic drug therapy for TdP is different from that for ventricular tachycardia that is not TdP.

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Various ECG abnormalities are known to occur frequently after subarachnoid haemorrhage (SAH) and prolongation of the QT interval is one of them.1–4 Because the QT prolongation can cause a life-threatening tachyarrhythmia called torsade de pointes (TdP) that is an atypical form of ventricular tachycardia, SAH is widely accepted as a potential cause of TdP.1,3 DiPasquale and colleagues1 found prolonged QT interval and TdP in 43 and 6% of patients with SAH in the acute period after haemorrhage, respectively. In contrast, Sommargren and colleagues2 recently demonstrated that a prolonged QT interval was present in 166 (73%) of 227 patients with SAH in intensive care unit (ICU) but only one patient developed TdP. Moreover, although many patients with SAH undergo surgical clipping of ruptured aneurysms, intraoperative TdP has rarely been reported. Whether SAH alone is capable of causing TdP or not remains controversial. We report a case of TdP caused by exacerbation of QT prolongation during clipping of anterior communicating artery aneurysm in a patient with SAH in the absence of other predisposing factors.

Case report

A 79-yr-old, 140 cm tall, 40 kg woman had an acute onset of severe headache. On arrival in our hospital, the patient was lethargic (Hunt-Hess Grade III). She had been in good health and had been taking no medications until the morning of admission. There was no family history of sudden death. Three-dimensional computed tomography demonstrated SAH caused by a ruptured anterior communicating artery aneurysm, and an emergency craniotomy for aneurysm clipping was scheduled. A preoperative ECG showed a mildly prolonged QT interval with a corrected to heart rate (QTc interval of 0.48 s) and T wave inversion in leads V3-6. Echocardiography revealed no wall motion abnormalities and normal ejection fraction. Routine laboratory investigations and chest X-ray were normal.

The patient received atropine 0.01 mg kg\(^{-1}\) i.m. for premedication. When she was connected to a 5-lead ECG (II, V5) on arrival in the operating room, the heart rate (HR) was 92 beats min\(^{-1}\) and the QT interval appeared not to have changed compared with the preoperative ECG. General anaesthesia was induced with propofol 1 mg kg\(^{-1}\), fentanyl 2 \(\mu\)g kg\(^{-1}\) and vecuronium 0.1 mg kg\(^{-1}\), and maintained with sevoflurane 1–1.5% and nitrous oxide (60%) in oxygen. Fentanyl and vecuronium were added as needed. The patient was haemodynamically stable throughout induction. Three hours after the start of surgery, when the anterior cerebral artery was dissected to expose the aneurysm, the HR suddenly decreased from 98 to 40 beats min\(^{-1}\). The cause of this was unclear. The bradycardia lasted for \(\sim\)10 s and the HR...
returned to baseline spontaneously. Fifteen minutes later, bradycardia occurred again. It was followed by a premature ventricular contraction occurring on the T wave of the previous beat and then polymorphic ventricular tachyarrhythmia resembling ventricular fibrillation developed (Fig. 1). Because the arrhythmia was accompanied by circulatory collapse, DC cardioversion was performed immediately and restored normal sinus rhythm. To prevent recurrence of the ventricular tachyarrhythmia lidocaine 1 mg kg⁻¹ and nifekalant 0.3 mg kg⁻¹, a novel class III antiarrhythmic agent, were given, followed continuous infusions of these drugs at rates of 1.5 and 0.4 mg kg⁻¹ h⁻¹, respectively. At this time, arterial blood gases and serum electrolyte concentrations, including potassium, calcium and magnesium, were within normal limits. A 12-lead ECG showed no evidence of an acute myocardial ischaemic event but markedly prolonged QTc interval of 0.57 s. An echocardiography revealed no wall motion abnormalities. When we carefully reviewed the ECG tracing of the ventricular tachyarrhythmia, it proved to be TdP (Fig. 1). Nifekalant was immediately discontinued. A few minutes later TdP with circulatory collapse recurred. After DC cardioversion successfully converted it to sinus rhythm, 2 g of magnesium sulphate was administered, followed by a continuous infusion at a rate of 2 mg min⁻¹. The HR was maintained at ~100 beats min⁻¹ with a continuous infusion of isoproterenol at a rate of 0.005–0.02 μg kg⁻¹ min⁻¹. Body temperature maintained within normal limits. Surgery was completed without further incident. A temporary transvenous pacemaker was placed to maintain the HR after surgery, and then the patient was transferred to the ICU. Although the QTc interval increased further to 0.63 s the next day, there was no recurrence of TdP in the ICU. Thereafter, the QTc interval gradually returned to normal and was 0.44 s on the fourth postoperative day. Lidocaine and magnesium were discontinued on the fourth postoperative day, and then cardiac pacing was terminated on the sixth postoperative day.

Discussion

There are several causative factors that worsen QT prolongation and may lead to the development of TdP. These include congenital long QT syndrome, drugs, myocardial ischaemia, electrolytes imbalance and cerebrovascular disease.⁵,⁷ Congenital long QT syndrome was ruled out because of the patient’s medical history and normalization of the QT interval after surgery. No drugs that affected the QT interval had been administered. There was no evidence of an acute myocardial ischaemia. Serum concentrations of potassium, calcium and magnesium were normal during surgery. Machado and colleagues⁸ have reported that the aetiology of development of TdP in patients with SAH is multifactorial and SAH cannot cause TdP in the absence of other predisposing factors. However, we believe that SAH alone was the cause of TdP by producing worsening QT prolongation in this patient.

Nifekalant is a pure potassium channel blocking agent (class III antiarrhythmic agent) developed in Japan and has no significant negative inotropic activity. Because this drug is effective in treating lidocaine resistant tachyarrhythmias, it has, often in combination with lidocaine, recently been used in Japan to prevent life-threatening refractory tachyarrhythmias, particularly in haemodynamically unstable patients.⁹ Because we failed to diagnose the first tachyarrhythmia as TdP in this patient, lidocaine and nifekalant were given. However, nifekalant can aggravate TdP. Similarly, many antiarrhythmic drugs which are effective in treating ventricular tachyarrhythmias other than TdP, including quinidine, procainamide, disopyramide and amiodarone are contraindicated in TdP. With respect to drugs for prevention of recurrence of TdP, we successfully used magnesium and isoproterenol during surgery. According to 2005 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care,¹⁰ magnesium is recommended as the first line of therapy for the short-term prevention of TdP irrespective of serum magnesium level but is not recommended in treating ventricular tachyarrhythmias other than TdP. Moreover, isoproterenol is useful in treating TdP² but is harmful in ventricular tachyarrhythmias other than TdP because most of them are caused by myocardial ischaemia.⁶ Drug therapy for TdP is completely different from that for ventricular tachyarrhythmias other than TdP. Therefore, identification of TdP is important.

Generally, a QTc of more than 0.5 s is associated with a high-risk development of TdP, but there is no definitive threshold below which the risk of TdP is absent.¹,⁴,¹¹ This patient had a QTc of 0.48 s on the preoperative ECG, which we did not consider dangerously prolonged. But a QTc value was 0.57 s after the first tachyarrhythmic event and increased further to 0.63 s until the next day. This indicates that even if patients with SAH have normal QTc interval before surgery and there are no additional factors that predispose to the QT prolongation, the QT interval can increase within a relatively short period, which may in turn trigger life-threatening tachyarrhythmias such as TdP. Therefore, the QT interval should be monitored continuously as a predictor of TdP. However, there are some problems in measuring the QT interval.¹¹ It is defined as time from the beginning of the QRS complex to the end of the
T wave, but the latter may be difficult to determine. The HR is very variable during surgery but there are few studies regarding the best way to adjust for the HR. Various ECG artifacts during surgery may interfere with measuring the QT interval. In addition, no computerized QT interval monitors are currently available. We propose the following recommendations for the QT interval monitoring to identify patients at risk of TdP: selection from the preoperative 12-lead ECG of the ECG lead in which the T wave end is well defined, typically V3, V4 or II, and repeated manual measurement of the QT interval using conventional ECG recording systems or cursors on the ECG monitor screen. It is also important to detect QT-related arrhythmias that develop into TdP including sudden bradycardias, enhanced U waves, T wave alterations, polymorphic premature beats, couplets and non-sustained polymorphic ventricular tachycardia.

This patient shows a potential risk of intraoperative occurrence of TdP triggered by further worsening of the QT prolongation in patients with SAH, even in the absence of factors that predispose to TdP. Therefore, a proper monitoring of the QT interval is necessary as a predictor of TdP. When ventricular tachyarrhythmia occurs, recognition of TdP is important because antiarrhythmic drug therapy for TdP is different from that for ventricular tachyarrhythmias that are not TdP.

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