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CASE REPORT

Long QT syndrome: anaesthetic management at delivery

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SUMMARY. We describe the anaesthetic management of a spontaneous vaginal delivery at 39 weeks' gestation in a 22-year-old patient with congenital long QT syndrome. With a strong family history of sudden deaths, the patient had an initial QT interval corrected for rate (QTc) of >600 ms. Following a once-daily 50-mg dose of atenolol over the previous 11 months, her QTc remained prolonged at 560 ms. To minimise any increase in catecholamine levels and consequent risk of malignant ventricular arrhythmias, a combined spinal-epidural technique was selected using intrathecal diamorphine and levobupivacaine, with intravenous and oral magnesium and potassium supplementation. Levobupivacaine was substituted for routine racemic bupivacaine to decrease the risk of drug-induced cardiotoxicity. Delivery outcome was successful and uneventful. We outline the pathophysiology, risks and treatments of long QT syndrome, and discuss the analgesic management of this patient in labour with congenital long QT syndrome.

Keywords: Congenital; Long QT syndrome; Labour; Analgesia; Combined spinal epidural; Levobupivacaine

INTRODUCTION

Long QT syndrome (LQTS) represents a group of arrhythmogenic cardiovascular disorders originating from cardiac ion channel mutations. A prolonged QT interval escalating into episodes of torsades de pointes is the hallmark of this group. This ventricular tachydysrhythmia can either revert spontaneously to sinus rhythm, producing syncope or pseudo seizures in its wake, or deteriorate into ventricular fibrillation and sudden death. The majority of clinical manifestations are related to emotional and physical stress, but have also been known to occur during sleep. Although LQTS can be either familial or acquired, it now seems likely that patients with acquired LQTS may actually be carriers of a mutant silent gene responsible for familial LQTS.[1]

There are very few case reports of anaesthetic and analgesic management of patients with this condition. The problem is further magnified by confusing and contradictory advice on management in the literature.

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CASE REPORT

A 22-year-old G_3 P_0 Caucasian woman was referred, by the obstetric team to our anaesthetic clinic at 38 weeks' gestation to discuss analgesic options for her forthcoming delivery. She had been diagnosed with Romano Ward Syndrome, the commonest type of familial LQTS, 12 months earlier. There was a strong family history of LQTS; two of her female cousins had died suddenly, both aged 22 years, and an aunt had done so at 50 years. Both the cousins had Romano Ward Syndrome while the aunt had an acquired form of LQTS. The patient had no history of palpitations or sudden collapse.

In view of her family history and a QT interval corrected for rate (QTc) of >600 ms, she was informed and then advised at diagnosis by a consultant cardiologist to have an automatic implantable cardiovertor defibrillator (AICD), which she declined. She was placed on atenolol 50 mg once daily, but her compliance to this therapy was doubtful. She had already had gynaecological procedures at our hospital on two earlier occasions pre-diagnosis. On each occasion she had no documented cardiac risk history and had had an uneventful routine general anaesthetic with no electrocardiogram (ECG) abnormalities.

On presentation at the clinic, and subsequently on the labour ward, she appeared very anxious and nervous. She was of average build with a weight of 75 kg and height of 170 cm. She had a normal heart rhythm, pulse

rate and blood pressure. Airway assessment (Mallampatti 1) revealed nothing to suggest difficulty in gaining control of her airway if anaesthesia or resuscitation were to prove necessary. The lumbar vertebrae and the intervertebral spaces were well delineated, so epidural or spinal needle insertion was expected to be easy.

The QT interval on the ECG at the time of diagnosis of LQTS was 600 ms, which corrected for rate (QTc), was 635 ms (normal QTc <430 ms). Following treatment with atenolol for about a year the 12-lead ECG in the anaesthetic clinic revealed a QTc of 560 ms. This suggested that either the patient had not been compliant with atenolol, or she was in need of further treatment. Serum electrolyte concentrations were within normal range.

Her management plan was developed following discussion with the cardiologists and at a departmental meeting. She presented from home in early spontaneous labour to the labour ward within a week of her anaesthetic assessment. She had remained asymptomatic throughout her pregnancy. A very cautious approach was adopted. She was nursed in a room fully equipped with cardiac arrest trolley, defibrillator facilities and transvenous pacing wires and leads. Special care was taken to keep her away from sudden loud noises, especially those due to telephones, pagers and monitor alarms. Two large i.v. cannulae secured venous access, and Hartmann's solution and drugs were administered in an overall target rate of 125 mL/h.

Blood samples were regularly analysed for potassium, sodium, magnesium and calcium. On admission, her serum sodium was 142 mmol/L, potassium 3.8 mmol/L, magnesium 0.85 mmol/L and adjusted calcium 2.3 mmol/L. Serum potassium was maintained at high normal levels of 4.5–5 mmol/L. This was facilitated by one oral Sando K (12 mmol of potassium) and an intravenous potassium chloride supplement of 40 mmoL in 100 mL of 0.9M sodium chloride over two hours

On advice from our cardiology colleagues, a magnesium sulphate infusion was initiated at a rate of 3 g/h as a prophylaxis against torsades de pointes. Serum levels of magnesium were monitored to avoid toxicity and potential augmentation of neuromuscular blockade.

Continuous ECG (lead II) and intermittent 12-lead ECG monitored heart rate and rhythm. Blood pressure and temperature were monitored non-invasively in a bid to maintain them in the normal range. The fetal heart rate and pattern were also monitored. Since the cardiotocograph may be difficult to interpret when the parturient is on β blockers or when there is a possibility that the fetus may have inherited LQTS, the threshold for fetal scalp blood sampling was low.

On admission, analgesia was provided by inhaled Entonox (50% nitrous oxide in oxygen), followed by a single intramuscular injection of diamorphine 10 mg, whilst further assessment was made. Once labour was recognised as fully established, the prepared plan was put into action, and a standard combined spinal-epidural technique was used for analgesia at the L2–L3 vertebral level. One mL of 0.25% levobupivacaine with diamorphine 300 μg was injected into the subarachnoid space through the spinal needle. This resulted in rapid pain relief with no significant drop in blood pressure. The epidural catheter placement was then tested with 2 mL of 0.5% racemic bupivacaine with 1:200 000 epinephrine, which is part of our routine epidural management. After 20 min an epidural infusion of 0.1% levobupivacaine with alfentanil 10 μg/mL was started at the rate of 8 mL/h. This was later increased to 10 mL/h and then to 12 mL/h. This technique provided good analgesia for the next 9 h after which the patient began complaining of inadequate pain relief.

A review of the epidural catheter and its connections revealed that the catheter had been displaced. It was promptly re-sited in the same space and re-secured. A 5-mL loading dose of 0.25% levobupivacaine was administered and the epidural infusion restarted at 12 mL/h. This resulted in good analgesia within the next 15 min.

The patient had a normal spontaneous delivery about 12 h after admission. A healthy male baby, weighing 3.5 kg with Apgar scores of 8 at 1 min and 10 at 5 min was delivered. During this time the patient had good analgesia, stable blood pressure and no abnormal heart rhythms. The epidural catheter was removed promptly after delivery. She was subsequently nursed in the high dependency unit for 12 h where she was weaned off the magnesium infusion. Throughout the peripartum phase the recorded magnesium concentrations ranged from 2.0 to 2.4 mmol/L.

Postnatally, adequate analgesia was maintained by regular oral administration of diclofenac sodium 50 mg/8 h and paracetamol 1 g/6 h. Serum magnesium, calcium and potassium were monitored uneventfully for the next three days on the postnatal ward after which she was discharged home. At discharge, her serum sodium was 144 mmol/L, potassium 4.6 mmol/L, magnesium 1.6 mmol/L and adjusted calcium 2.2 mmol/L.

DISCUSSION

Long QT syndrome represents a group of cardiac ion channel pathologies that can be congenital or acquired.[1] Congenital LQTS is an important and previously underestimated cause of sudden death in young people.[2] It can be inherited as an autosomal dominant (Romano Ward syndrome) or autosomal recessive (Jervell and Lange-Nielson syndrome) condition. Drugs, electrolyte imbal-