Bradycardia induced polymorphic ventricular tachycardia during living donor liver transplantation

Sir,

We present a successful management of recurrent polymorphic ventricular tachycardia (PVT) in a 44-year-old liver transplant recipient with alcoholic liver disease, a model for end-stage liver disease score 27 and hepatorenal syndrome. Pre-operative electrocardiogram showed prolonged QTc (470 ms) with unremarkable transthoracic echocardiography and negative dobutamine stress echocardiography.

Anaesthetic induction with fentanyl, thiopentone sodium and rocuronium was uneventful with stable haemodynamics. Arterial blood gas analysis demonstrated normal blood gases and electrolytes. Anaesthesia was maintained with isoflurane, fentanyl and atracurium. In dissection phase, significant blood loss was managed with massive blood transfusion. Intravenous (IV) fluids with noradrenaline and vasopressin infusion were administered to maintain mean arterial pressure of >65 mm Hg and stroke volume variance of <13. On reperfusion, sudden fall in systemic vascular resistance responded to bolus 200 μg phenylephrine with increased noradrenaline (0.3 μg/kg/min) and vasopressin (2.4 units/h) support. Five hours later, abdominal closure was started with pulse of 60 bpm and blood pressure of 116/74 mm Hg on noradrenaline 0.2 μg/kg/min with vasopressin 1.8 unit/h. Sudden onset ventricular ectopics and bigeminy were observed, which was managed by intermittent 100 mg lignocaine IV and magnesium sulphate 2 g IV infusion. Adequate anaesthetic depth, normal blood gas and electrolytes were confirmed, and sinus rhythm was restored. On resumption of surgical stimulus, recurrent PVT 6–8 beats run at 170–180 bpm were noted with transient response to lignocaine, magnesium sulphate and defibrillation (200 J biphasic shock). Lignocaine infusion was started at 1.5 mg/kg/h. Loading dose of amiodarone (150 mg) was administered followed by infusion (1 mg/min). Sinus rhythm got restored, but QT interval increased with a corrected QTc of 625 ms. Amiodarone was immediately

stopped. No antiemetic (5-hydroxytryptamine 3 [5-HT3] antagonist) was administered. Fluconazole (known to cause QT prolongation) was replaced with anidulafungin. Echocardiography confirmed a good myocardial contractility with the absence of wall motion abnormalities, right ventricular outflow tract dilatation or thromboembolism. During post-operative period, ventricular premature contractions and 5–6 beat runs of PVT recurred, when heart rate fell below 58–60 beats/min. Heart rate was maintained more than 75 bpm with IV injection of glycopyrrolate 0.2 mg. During weaning, heart rate increased above 75 bpm and arrhythmia disappeared. Lignocaine infusion was tapered and stopped. Given good graft function, decreased requirement of vasopressors and normalising lactate, laboratory and metabolic parameters, the patient was weaned from mechanical ventilation. The heart rate remained above 90 beats/min. Rest of the post-operative course was uneventful.

Prevalence of QTc prolongation in cirrhotic ranges from 19.2% to 56%, but its association with increased mortality is controversial.[1] Bal and Thuluvath observed no survival differences in patients with and without prolonged QTc interval, but life-threatening ventricular arrhythmia is reported consequent to a prolonged QTc interval in cirrhosis during stress.[1-4] In our recipient, pre-operative prolonged QTc interval (470 ms) increased to peak in neohepatic phase (625 ms). Multiple blood transfusions could have led to hypomagnesaemia at the cellular levels, contributing to rhythm disorder.

Torsade de pointes is reported at varied stages (after anaesthetic induction, dissection phase, caval clamping in anhepatic or portal vein unclamping in neohepatic phase) of liver transplantation.[2,3] Amiodarone, 5-HT3 antagonists and sevoflurane may prove detrimental by aggravating QTc prolongation.[3] A list of known drugs to cause QTc prolongation is shown in Table 1.[5] Bradycardia as an important risk factor for PVT was identified only on spontaneous abolition of arrhythmias above a heart rate of 75 bpm.

In refractory PVT, to restore the sinus rhythm, administration of magnesium with isoprenaline (1–10 μg/min) to increase the heart rate up to 90–100 beats/min is suggested.[2] Routine measurement of serum magnesium levels and QTc interval is advisable. Anaesthesiologists should be aware of the risk of PVT during liver transplant surgery, and a high index of suspicion for prolonged QTc may change the management of ventricular arrhythmia with lignocaine, magnesium and by an increase in basal heart rate to prevent its recurrence.

Table 1: Drugs causing prolongation of QTc interval and reported to cause torsades de pointes

Antiarrhythmic drugs
Type 1A
Quinidine, procainamide and disopyramide
Type 1C (increase QT by prolonging QRS interval) encainide, flecainide
Type 3
Amiodarone, sotalol, d-sotalol, bretylium, ibutilide, dofetilide, amakalant, semantilide
Calcium channel blockers
Prenylamine, bepridil, terodiline
Psychiatric drugs
Thioridazine, chlorpromazine, haloperidol, droperidol, amitriptyline, nortriptyline, imipramine, desipramine, clomipramine, doxepin, lithium, chloral hydrate, pimozide
Anaesthetic agents
Sevoflurane
Antihistamines
Terfenadine, astemizole, diphenhydramine, hydroxyzine, ebastine, loratadine, mizolastine
Antimicrobial and antimalarial drugs
Erythromycin, clarithromycin, ketoconazole, fluconazole, pentamidine, quinine, chloroquine, halofantrine, amantadine, sparfloxacin
Serotonin agonists/antagonists
Ketanserin, cisapride, ondansetron, granisetron
Immunosuppressant
Tacrolimus
Other agents
Vasopressin, adenosine organophosphates, papaverine, cocaine

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Conflicts of interest

There are no conflicts of interest.

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