Bradycardia induced Polymorphic ventricular tachycardia during Living donor liver transplantation: A Case report

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Introduction

Serious arrhythmias have been documented in liver transplant recipients, especially at the time of caval clamping or post-reperfusion. We observed sudden onset ventricular bigemini followed by recurrent Polymorphic ventricular tachycardia in neohepatic phase in a liver transplant recipient.

Case Reports

A 44-year male patient with alcoholic liver disease, model for end-stage liver disease score 27 and hepatorenal syndrome was scheduled for living donor liver transplantation. Pre-operative cardiac assessment showed a prolonged QTc (470ms) on electrocardiogram with unremarkable echocardiography and negative dobutamine stress echocardiography for inducible ischemia.

Anaesthetic management

Anaesthetic induction with fentanyl, thiopentone sodium and rocuronium was uneventful with stable haemodynamics, 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 >65 mm Hg and stroke volume variance <13.

On reperfusion, sudden fall in systemic vascular resistance was noted. It 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 at this point of time.

Patient management after arrhythmias

We treated the bigemini with intermittent 100 mg IV lignocaine 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 with 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 625 ms. Amiodarone was immediately stopped. No antiemetic (5-hydroxytryptamine 3 [5-HT3] antagonist) was administered. Fluconazole was replaced with anidulafugin.

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 glycopyrrolate 0.2 mg. During weaning, heart rate increased above 75 bpm and arrhythmia disappeared.

Lignocaine infusion was tapered and stopped.

With a good graft function, requirement of vasopressors decreased and lactate as well as the other metabolic parameters normalized.

After successful spontaneous breathing trial, mechanical ventilation was weaned and trachea extubated. The heart rate remained above 90 beats/min.

Rest of the post-operative course was uneventful.

Discussion

Prevalence of QTc prolongation in cirrhotic ranges from 19.2% to 56%, but its association with increased mortality is controversial.[1]

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]

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].

Conclusions

- Routine measurement of serum magnesium levels and QTc interval is advisable.
- Anaesthesiologists should be aware of the risk of PVT during liver transplant surgery
- 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.


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