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. 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. 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. Amiodarone, 5-HT3 antagonists and sevoflurane may prove detrimental by aggravating QTc prolongation. A list of known drugs to cause QTc interval is shown in Table 1. 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. 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.

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**Conflicts of interest**
There are no conflicts of interest.

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**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, sematilide |
| Calcium channel blockers |
| Preynilamine, 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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Sir,

We read with great interest the case report on the management of post-dural puncture headache (PDPH) with mannitol and the letters in response to the same in the IJA. The authors have mentioned the postulated mechanism of action of mannitol - drawing fluid from neuronal glial cells, resulting in dehydration and reduction in brain volume, whereby it ‘refloats’, thus minimising the meningeal traction effects.

We would like to share our experience of PDPH in a parturient for labour epidural analgesia (LEA) managed with mannitol infusion successfully.

In our institute, we practice LEA regularly with 0.1% bupivacaine/ropivacaine with fentanyl 2 µg/ml as intermittent bolus doses of 10–15 ml one hourly. Recently, in one case, we encountered an inadvertent dural puncture twice at L3/4 and L2/3 intervertebral spaces at a distance of 2.7 cm (approximately) with the use of an 18-gauge Tuohy’s needle. The parturient was gravid 2, moderately built, presenting with vertex lie of the foetus with no other comorbidity.

On per vaginal examination, the cervix was 5 cm dilated with 3–5 mild contractions for every 15 min. It was a dilemma to decide to attempt a third prick, but considering the demand of labour analgesia and threat of PDPH for which epidural catheter could further facilitate the injection of saline in achieving seal effect on the dural rents, a third attempt of epidural at L4/5 level was made. Loss of resistance was appreciated, but the catheter could not be negotiated. By then, the parturient became uncooperative, labour progressed to cervical dilatation of 6–7 cm with increasing intensity/frequency of contractions and the procedure was abandoned and analgesia was provided with tramadol intravenously.

The child was delivered vaginally at around 12 noon uneventfully. Immediately, oral analgesics containing caffeine, triptans and crystalloid infusions (ringer lactate/normal saline) 2 L/day along with strict bed rest and watch on the symptoms of PDPH started. Antibiotics and dexamethasone 8 mg BID were administered. Oral hydration was encouraged. The need of epidural blood patch for the management of headache was explained to the parturient and relatives. Meningism developed the next day morning and she complained of headache at around 12 noon i.e., at 24 h of delivery. It was again a dilemma to decide about epidural autologous blood patch, considering the failed epidural trials on the previous day. We administered 100 ml of 20% mannitol as infusion over 30 min twice 12 hourly. On the 3rd day morning, there was a dramatic relief in her headache except local back pain. Subsequently, she was managed conservatively and was discharged on 8th day. On weekly telephonic follow-up, she was fine and relieved of all her symptoms.

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