Atrial fibrillation during anhepatic phase liver transplantation: Now what?

Madam,

We report the occurrence of supraventricular tachycardia (SVT) and atrial fibrillation (AF) during the anhepatic phase of living donor liver transplant (LDLT) and describe its management.

After written informed consent, a 40 year old male with alcoholic liver disease underwent LDLT. He had no comorbidities. Preoperative echocardiography was normal. Intraoperatively, Flotrac™ monitor was used with radial artery

cannulation for measurement of blood pressure, systemic vascular resistance (SVR), and cardiac output. His baseline SVR was 619 dynes/s/cm^5, stroke volume variance 9%, and mean arterial pressure (MAP) 70 mm Hg. Target SVV was <12% and MAP 65 mm Hg. Thromboelastography was used to guide blood product administration.

After incision, 13 L of ascites was drained and intravenous albumin was administered to avoid post paracentesis circulatory disturbance. The dissection phase was complicated by massive blood loss managed with transfusion of 13 units of packed red blood cells (PRBCs) with a target hematocrit of 24%. Thromboelastogram-guided correction of coagulopathy was done with 10 units of fresh frozen plasma (FFP), 1 single donor platelet concentrate (SDPC), and 6 cryoprecipitates. Hypocalcemia was corrected with 10% calcium chloride infusion. Heart rate increased to 120 bpm. To maintain MAP >65 mmHg, noradrenaline and vasopressin infusions were increased from 3 to 45 μg/min and 0.8 to 3.2 units/h, respectively. Oliguria and metabolic acidosis on arterial blood gas (ABG) analysis (pH 7.21, base excess −13.7 mmol/L, lactate 3.4 mmol/L, pCO_2 32.3 mmHg, pO_2 189 mmHg) were observed. An infusion of sodium bicarbonate was started.

In the anhepatic phase, three PRBCs were transfused along with sodium bicarbonate infusion. A portocaval shunt was created. However, the patient continued to have unstable hemodynamics with SVR below 400 dynes/s/cm^5, SVV <8%, CO 10 L/min, and pulse rate of 120 bpm. Noradrenaline was stepped up to 45 μg/min and vasopressin to 4 U/h. The anhepatic phase lasted for 3.5 h. Arterial lactate levels increased from 3.4 to 5.7 mmol/L and base excess −10 mmol/L. But pH, serum bicarbonate, and serum ionized calcium levels were maintained within the normal range by sodium bicarbonate and calcium chloride infusions. Three hours into anhepatic phase, there was sudden onset of SVT which responded to 6 mg of intravenous adenosine. ABG showed normal pH, increased lactate (6.2 mmol/L), and base excess −8.6 mmol/L. Serum electrolytes were normal (Na 140, K 4.2, ionized Ca 1.1). Five minutes later, SVT recurred, which was unresponsive to sequential 6 and 12 mg of adenosine. In view of further increase in hemodynamic instability, cardioversion was attempted with 50 J biphasic shock. The rhythm now changed to AF with a ventricular rate of around 110/min. Acidosis, hypo/hyperkalemia, hypocalcemia, hypoglycemia, and hypothermia were ruled out. In anticipation of exacerbation of hemodynamic instability and arrhythmias on reperfusion in the setting of new-onset intraoperative AF, repeat cardiversion was attempted with 200 J biphasic shock and no response was noted. Subsequently, chemical cardioversion was done with amiodorone 150 mg over 10 min, followed by amiodarone infusion (1 mg/min for 6 h). Vasopressin was increased to 4.5 U/h. The rhythm remained irregularly irregular, but ventricular rate control was achieved to 80 bpm. With ongoing AF, graft was reperfused after prophylactic bolus of 500 μg phenylephrine. Transient fall in MAP to 40 mmHg was noted which increased to 80 mmHg. Adrenaline was avoided to decrease possibility of aggravation of tachyarrythmias. The patient continued to be in AF throughout with a ventricular rate of 80 bpm. CVP remained <10 mm Hg throughout the anhepatic period. Following reperfusion, the requirement of vasopressors did not change. One hour into the neo-hepatic phase, improvement in lactate levels (3.9), SVR (900), and urine output (100 mL/h) was observed. Vasopressor requirement decreased marginally with noradrenaline of 35 μg/min and vasopressin 3 U/h. After surgery, the patient was monitored, electively ventilated, and managed in transplant intensive care unit (ICU). A transthoracic echocardiogram was performed, which showed good contractility and normal filling of the atria, with no evidence of segmental wall motion abnormalities or clot. Over the next 8 h, vasopressors' requirement decreased further and was discontinued. After successful spontaneous breathing trial, trachea was extubated. Amiodarone infusion was continued and rhythm reverted to sinus 10 h after surgery. Further recovery was uneventful with ICU discharge to ward after 72 h.

AF is the most common supraventricular arrhythmia which increases morbidity and mortality.[1-4] Factors attributed to development of AF during LT include alcoholism, hypomagnesemia, preexisting cardiac conditions, massive blood loss, and acid base and electrolyte imbalances.[5] Although AF may be present preoperatively, it has also been described following reperfusion; in this patient, there was new-onset AF immediately prior to reperfusion

Although our recipient was a chronic alcoholic, there was no evidence of either preexisting cardiac comorbidities (such as ischemic heart disease, cardiomyopathy, or preexisting AF) or any other immediate precipitating factor such as dyselectrolytemia. Massive blood loss and high dose of vasopressors may have led to a pro-arrhythmogenic state. A further increase in vasopressor support during the anhepatic phase probably aggravated the situation. Even though electrical and chemical cardioversion was unsuccessful, rate control was achieved with amiodarone. In view of high vasopressor requirement, we avoided beta blockers or calcium channel blockers. During reperfusion, a conscious attempt was made to avoid arrhythmogenic drugs such as adrenaline as far as possible. In retrospect, regular intraoperative use of magnesium supplementation in the setting of massive blood loss and use of phenylephrine rather than noradrenaline to maintain SVR may have been beneficial.

In conclusion, new-onset SVT or AF may occur in anhepatic phase of LDLT especially when accompanied with massive blood loss and high inotropic requirement.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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