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Complete Atrioventricular Block and Torsade de Pointes Occurring in an Adult During Liver Transplantation

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Perioperative arrhythmias frequently occur during cardiac surgery and may also occur during non-cardiac surgery, particularly in patients with structural heart disease. Perioperative arrhythmias represents a major cause of morbidity in either cardiac or non-cardiac surgical procedures. Complete atrioventricular block (CAVB) and torsade de pointes (torsades) are critical arrhythmias, although their incidence during non-cardiac surgery is rare in patients with no underlying structural heart disease. We present a rare case of life-threatening arrhythmias of CAVB and torsades that occurred during cadaveric orthotopic liver transplantation in a 59-year-old man with no structural heart disease. Rapid recognition of the arrhythmias and prompt treatment using a transcutaneous pacemaker and cardioversion successfully saved the patient's life. The sinus rhythm was resumed during pacemaker removal postoperatively. The possible etiology and management of CAVB and torsades during liver transplantation are discussed herein.

Key words: atrioventricular block; liver transplantation; torsade de pointes

INTRODUCTION

Perioperative arrhythmias such as atrial fibrillation are common during non-cardiac surgery. Incidences of other types of arrhythmias, including bradycardia, heart block, supraventricular tachycardia, and ventricular tachycardia are also common.[1] Usually, the predisposing factors for perioperative arrhythmias are temporary insults such as hypoxemia, electrolyte imbalance, acidosis, cardiac ischemia, and catecholamine excess.[2]

Liver transplantation has improved the prognosis of patients with end-stage liver disease or hepatocellular carcinoma.[3] However, although liver transplantation is a life-saving procedure, and despite recent advances in instrumentation and medication, concerns regarding complications remain.

Life-threatening arrhythmias are rare in patients during liver transplantation.[4-8] In 1998, Lustik presented the first report of torsade de pointes (torsades) during liver transplantation in a 57-year-old woman with end-stage liver disease.[4] In 2008, Nisli reported the first case of transient complete atrioventricular block (CAVB) in a 30-month-old boy with bile duct paucity-associated liver cirrhosis during a living donor liver transplantation.[8] Herein, we report a case of CAVB and torsades during cadaveric orthotopic liver transplantation in an adult with no structural heart disease.

CASE REPORT

Following computed tomography of the abdomen because of an incidence of trauma in October 2005, a 59-year-old man with a history of hepatitis B virus infection was diagnosed as having liver cirrhosis and one incidental hepatic tumor. Angiography showed typical features of hepatocellular carcinoma (HCC) and liver biopsy confirmed HCC. The patient subsequently underwent open radiofrequency ablation treatment. During regular follow up in November 2007, three small recurrent nodules of HCC were found in segments 8, 6, and 6; subsequently, transarterial chemoembolization (TACE) was performed twice. After TACE, the patient's condition progressed to decompensated liver disease with bleeding esophageal varices and regenerative nodules. He was listed for liver transplantation according to the Milan criteria.

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In October 2008, the patient was scheduled to undergo cadaveric orthotopic liver transplantation. No cardiovascular abnormalities were observed during the preoperative examination; his electrocardiogram (ECG) showed a sinus rhythm of 80 beats/min and a prolonged QT interval of 0.53 seconds (Fig. 1A). Physical examination revealed a distended abdomen with shifting dullness and anemic conjunctiva. Laboratory investigation revealed the following findings: hemoglobin level, 10.2 g/dl (normal 13-18 g/dl); platelet count, 19,000 (normal range, 150,000-400,000/ul); total bilirubin, 1.5 mg/dl (normal range, 0.2-1.2 mg/dl); albumin, 2.8 g/dl (normal range, 3.8-5.3 g/dl); and prothrombin time, 17.5 seconds (normal range, 10–14 s); and normal values of electrolytes such as Na^+, K^+, Mg^+, and Ca^{++}. In the operating room, the general anesthesia process was uneventful. Histidine-tryptophan-ketoglutarate (HTK) solution was used for preservation of the liver during surgery. During liver transplantation, general oozing was noted, and stable hemodynamics were maintained by the aggressive administration of fluid and a large transfusion of blood. After about 6 h of surgery and portal vein anastomosis, we performed reperfusion by maintaining a body temperature of 35.2 ^oC. Subsequent to the procedure, bradycardia with an arterial pressure of 66/38 mmHg was noted. Rhythm strip demonstrated complete atrioventricular block with a ventricular response rate of 48 beats/min (Fig. 1B). Additionally, laboratory investigations showed normal values of Na^+, K^+, Ca^{++}, and glucose and a hematocrit of 25%. Arterial blood gas analysis showed the following values: pH, 7.28; pCO_2, 37.9; HCO_3, 20.8; BE, -6.0; and O_2 saturation, 100%. Transesophageal echocardiography (TEE) showed a normal cardiac wall motion and an estimated left ventricular ejection fraction of 55%. A 1-mg bolus of atropine and dopamine were administered intravenously, but the patient's condition did not improve. A transcutaneous pacemaker (TCP) was placed (Fig. 1C), and arterial pressure increased to 90/62 mmHg. However, pulseless polymorphic ventricular tachycardia, consistent with torsades, was observed after 5 min (Fig. 1D). Electrolyte analyses were normal, except that the level of magnesium ion was 0.32 mmol/l (normal range, 0.45-0.64 mmol/l). Two grams of magnesium sulfate were administered intravenously, and advanced cardiac life support with cardioversion was performed twice. Cardiac rhythm returned to a level consistent with that of complete atrioventricular block, and a TCP was again used to maintain regular rhythm until completion of surgery. When we momentarily shut down the TCP after the operation, a normal sinus rhythm was observed. The patient was transferred to the intensive care unit. Twelve hours after surgery, the TCP was removed and a sinus rhythm of 116 beats/min was noted using a rhythm strip (Fig. 1E).

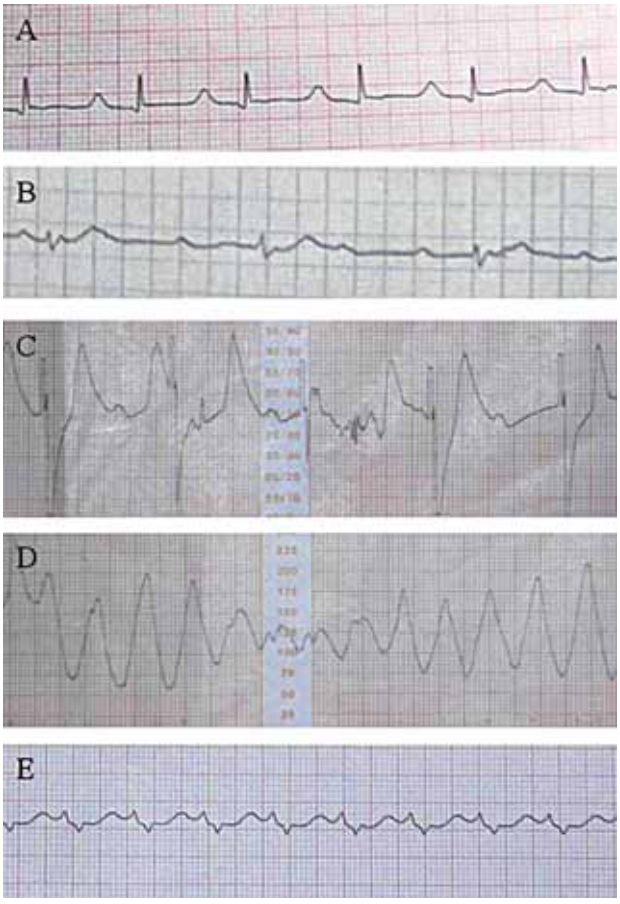

Fig. 1 (A)Preoperative ECG showing a sinus rhythm with 80 beats/min and prolonged QT interval of 0.53 seconds. (B)Complete atrioventricular block with ventricular response of 48 beats/min after portal vein reperfusion in perioperative period. (C)Pace rhythm after placement of TCP. (D)Pulseless polymorphic ventricular tachycardia, consistent with torsades de pointes was shown. (E)Sinus rhythm with rate of 116 beats/min was obtained after removal of TCP.

DISCUSSION

Perioperative arrhythmias are common and frequently cause morbidity and mortality in patients during cardiac as well as non-cardiac surgery.[1] Prevention, rapid recognition, and prompt treatment of life-threatening arrhythmias during operations are major challenges for surgeons. CAVB is defined as the complete dissociation between atrial and ventricular contractions and results in

hemodynamic collapse; this can be either a congenital or an acquired disease. CAVB rarely occurs during non-cardiac surgery in patients with no structural heart disease. When it does occur in non-cardiac surgery, transient CAVB is most commonly secondary to ischemic heart disease, hypoxia, and electrolyte abnormality.[2] Thus far, three cases of CAVB during renal transplantation have been reported.[9-11]

Only one case of CAVB during liver transplantation, which was in a 30-month-old boy, has been previously reported; in this instance, physicians were unable to determine the exact etiology of CAVB. Hypoperfusion, hypoxia of the heart, depressed baroreflex function, or anesthetic agents may contribute to CAVB.[8] In our patient, neither electrolyte abnormality nor ischemic change of ECG had been reported preoperatively. CAVB occurred just after portal vein reperfusion, and acute acidosis (pH, 7.28) was noted simultaneously.

Ischemic heart disease during surgery is less likely to occur because TEE has been increasingly used to detect the heart's normal wall motion in recent times. Thus, ischemic-reperfusion syndrome was considered to be the major cause of CAVB in our patient. Ischemic-reperfusion syndrome following revascularization of the liver graft during liver transplantation was described by Aggarwal in 1987.[12] This syndrome comprises severe cardiovascular dysfunction, bradyarrhythmia, decreased mean arterial pressure, and systemic vascular resistance. Ko and colleague reported that the incidence of post-reperfusion syndrome during the reperfusion period in living donor liver transplantation was greater when HTK was used compared with cases in which University of Wisconsin (UW) solution was used.[13] We used HTK solution in our patient, and it may have induced obvious post-reperfusion syndrome. Therefore, careful hemodynamic management is advised when using HTK solution.

Torsades is a form of polymorphic ventricular tachycardia (VT) occurring in patients with a prolonged QT interval.[14] Characteristic features of torsades are a prominent U wave, shifting electrical axis, and variable RR interval.[15] Other risk factors for torsades include occurrence of the acute phase of acute myocardial infarction; drugs (anti-arrhythmic agents of classes 1A and III and/or tricyclic antidepressants); electrolyte disturbances (hypokalaemia, hypomagnesemia, or hypocalcemia); metabolic disease (hypothyroidism, anorexia nervosa, or starvation); bradycardia (sinoatrial disease or AV block); and toxins (heavy metals or insecticides).[16] Following CAVB after reperfusion during liver transplantation, our patient with preoperative prolonged QT interval suffered polymorphic VT, which was consistent with torsades.

Risk factors for torsades in surgical patients include preoperative prolonged QT interval, perioperative CAVB, and hypomagnesemia. The prolonged QT interval in our patient may have been secondary to the effects of cirrhosis, since we knew of no other causes that would produce a prolonged QT interval. In patients with cirrhosis, the QT interval is prolonged in 46% of the candidates for liver transplantation. In these cases, the prolonged QT interval worsens in parallel with the severity of the disease, suggesting that cirrhosis is an important prognostic factor.[17] The QT interval often decreases to the normal range after liver transplantation in these patients.[18] Additionally, a major blood transfusion may result in hypomagnesemia following torsades.[19] Our patient received massive blood transfusions during his operation, resulting in hypomagnesemia. Thus, electrolyte monitoring and correction are important during a major operation, particularly those that result in massive blood loss.

Treatment of torsades includes defibrillation, discontinuation of predisposing drugs, administration of magnesium sulfate and isoproterenol, and temporary pacing.[20] Our patient was treated successfully with an injection of magnesium sulfate and cardioversion, following the placement of a pacemaker for heart block.

In summary, life-threatening arrhythmia during liver transplantation is a rare and challenging complication. Our report suggests the importance of recognizing reperfusion injury and prolonged QT interval as risk factors, and avoiding electrolyte abnormality in the cirrhotic patient presenting for liver transplantation. Further, in management of CAVB and torsades, transcutaneous pacemaker, cardioversion, and electrolyte correction are crucial for a successful outcome.

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