Primum Non Nocere: Organ Donation After Electrocution and Transplantation of Electricity-Damaged Livers

Report of 2 Cases

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ABSTRACT

Liver transplantation remains the treatment of choice for patients with end-stage liver disease. However, allograft availability continues to be a problem, and extending the criteria for organ acceptance is key. Deceased donors after electrical accidents, as well as electricity-traumatized allografts, are not common but should be considered suitable. This study describes 2 cases of heart-beating organ donors with electrical injury to the liver. In 1 case, the electric shock was the cause of death; in the second case, the injury was caused by defibrillation at organ procurement. Both allografts had sustained sizeable electrical injury, and both resulted in excellent early posttransplant outcomes. These cases demonstrate that electrocution is not a contraindication to donation and that electricity-traumatized allografts may remain transplantable after careful assessment. Education of all staff in the management of such donors can optimize utility of such allografts.

ORGAN DONATION AFTER BRAIN DEATH (DBD) caused by electric shock is extremely uncommon, with only a few cases reported worldwide [1]. Using traditional wisdom, transplant surgeons have been reluctant to use organs from such donors. Electric shock means injury associated with exposure to electricity [2]. Electrocution is a fatal electric shock and accounts for >2 in 1 million deaths per year in the United States, the majority occurring within the workplace [3]. The present study describes 2 cases of liver transplantations using DBD allografts with electrical injury to the right lobe.

CASE 1: ELECTRIC INJURY TO THE LIVER DURING A FATAL ELECTRICAL INCIDENT

The donor was a previously fit 18-year-old female who was electrocuted after she accidentally fell onto a railway track. Computed tomography (CT) scanning demonstrated loss of cerebral gray matter in keeping with hypoxic brain injury, plus subarachnoid hemorrhage. Abdominal CT imaging revealed right anterior abdominal wall contusions, with subcutaneous and intramuscular gas locules under an overlying skin defect and heterogeneous opacification of the liver. Alkaline phosphatase (ALP) and γ-glutamyl transferase levels remained normal and in the range of 47 to 101 IU/L and 10 to 28 IU/L, respectively. Serum bilirubin levels also remained within normal limits. Aspartate aminotransferase (AST) levels were 374 IU/L at initial presentation, peaked at 991 IU/L after 4 hours, and declined to 914 IU/L on the day of the procurement. The AST trend mimicked creatine kinase (CK) levels: CK levels were 14,472 IU/L upon admission, peaked at 38,644 IU/L, and then decreased to 22,412 IU/L; it is therefore difficult to determine which fraction of AST was reflective of hepatic injury rather than systemic tissue destruction and extensive rhabdomyolysis, as indicated by the raised CK levels. Alanine aminotransferase (ALT) is not routinely measured at this trauma center. Renal function remained normal.

The patient was declared brain dead 1 day postadmission. After appropriate consent and assessment, the patient's liver, kidneys, and pancreas were allocated for transplantation. The lungs were declined for transplantation on the grounds of the thoracic injuries, and the heart was accepted for valves.

The donor had third-degree burns to the right side of neck and face and from the mid-sternum to the right upper abdominal quadrant. Laparotomy revealed a small amount of free fluid in the peritoneal cavity, which was bile-tinged around the gallbladder, with no macroscopic evidence of enteric or fecal contamination. The liver appeared to have a 5 × 5 cm well-demarcated area on segment

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V and on the gallbladder at the liver diaphragmatic surface, representing an electrical injury between the entry point (overlying skin) and gallbladder, which, in lieu of its bilious content, served as low-resistance conductor. During the warm phase, the injured portion of segment V was mauve, petechial, clearly demarcated from the surrounding parenchyma, and stiff on compression. This demarcation, surrounded by a pale halo, remained after cold perfusion and postimplantation (Fig 1A and B). The gallbladder and the extrahepatic bile duct appeared to be macroscopically intact. The kidneys and pancreas were grossly normal and procured for transplantation.

According to the National Health Service Blood and Transplant Clinical Guidelines and Policies, all hepatic donors aged <40 years are considered for graft splitting until proven otherwise based on

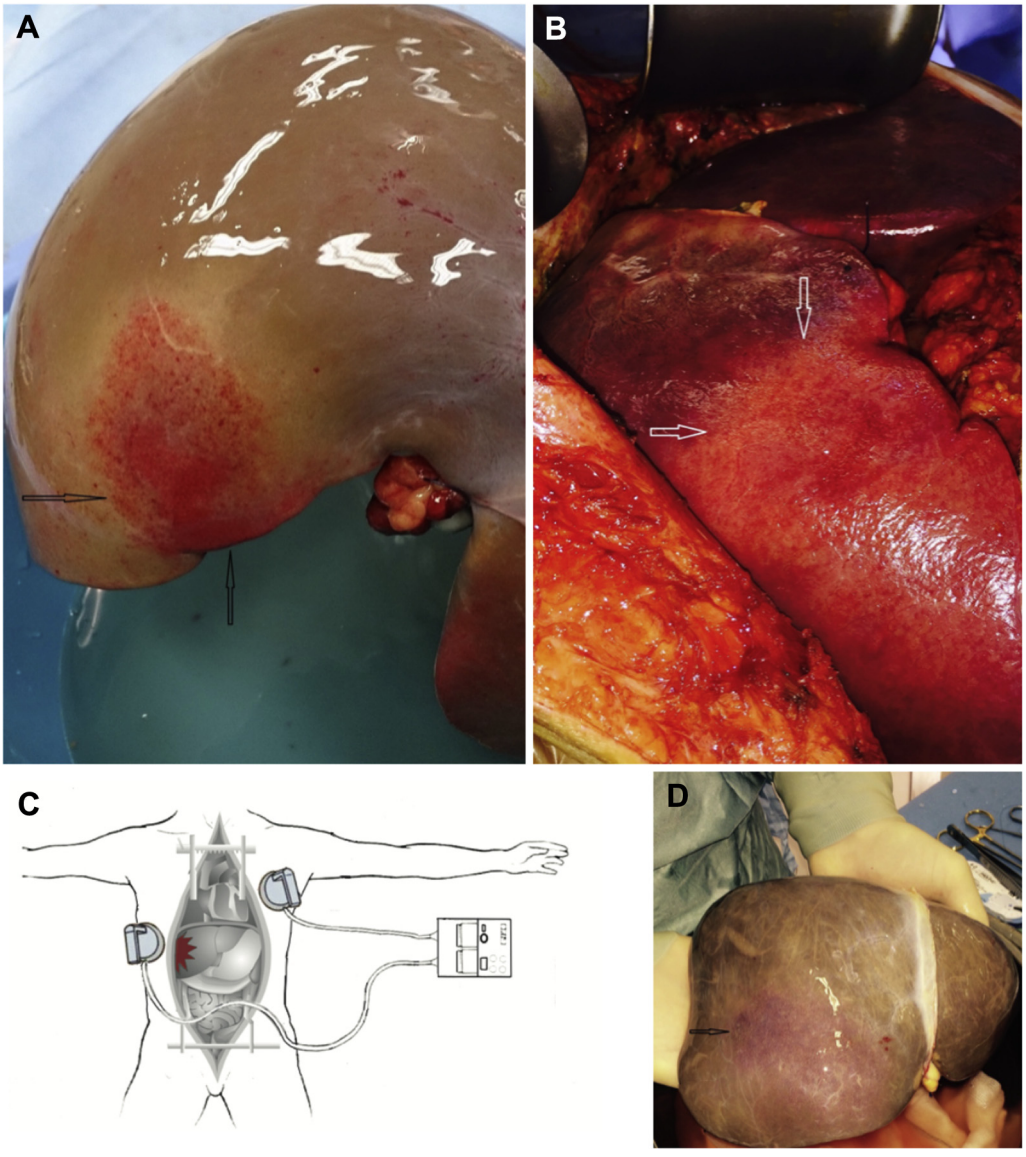

Fig 1. (**A**) Liver graft procured from a donor who sustained a fatal electrical injury after having fallen on railway tracks (case 1). There were third-degree burns on the skin overlying the liver. The electric current arced through the closest tissue of least resistance (ie, the gallbladder), resulting in electrical injury of the interposed segment V. The arrows point at the site of electrical injury; the injured area had a purple discoloration during the warm phase, which turned into petechial violet and became confluent as the juxtaposed parenchyma was becoming thinner. The gallbladder appeared macroscopically intact, even though the surrounding reactive fluid was bile-tinged, suggesting gallbladder microperforation. Note the pale halo surrounding the affected area. (**B**) Postperfusion appearances of the same graft. The affected liver assumed a bright orange hue postreperfusion, clearly distinct from the surrounding parenchyma (margins of the affected area are noted by the arrows). (**C**) Schematic representation of the positioning of the external paddles during defibrillation applied during procurement surgery (case 2). (**D**) Erythematous demarcation in segments V and VI, indicating electrical injury secondary to defibrillation. At that stage of retrieval, left coronary and falciform ligaments had already been mobilized.

donor history and organ inspection. Typically, graft splitting is ex vivo and performed at the recipient's hospital. Due to the sustained electrical injury, the donor's liver was no longer considered splittable and was allocated for whole organ transplantation. The recipient was a 57-year-old patient who had a second re-transplant for cholangiopathy and recurrent cholangitis. Initial indication had been autoimmune hepatitis, followed by urgent re-transplantation for hepatic artery thrombosis. The recipient's implantation and posttransplant course were uneventful. Routine day 1 and day 5 liver ultrasounds were unremarkable, and the results of liver function tests showed prompt normalization of graft function.

In view of the electrical injury pattern, the donor gallbladder and bile duct margin were sent for pathologic review, with the latter revealing acute inflammation of the gallbladder. The patient made an excellent recovery with normal graft function 4 months' posttransplant. The recipient had serial liver ultrasounds performed on days 1, 5, 20, and 85 posttransplant, which showed homogeneous liver parenchyma and no duct dilation. At 136 days after the transplant procedure, the patient remains well with normal results on liver function tests. Given the normal laboratory profile and the lack of abnormalities on the repeat ultrasounds, no additional cross-sectional imaging has been pursued thus far.

Apart from the liver, the donor's kidneys and pancreas were also transplanted; 1 kidney was implanted, with no posttransplant complications and a latest creatinine measurement of 125 μmol/L. The other kidney was implanted in a simultaneous kidney–pancreas procedure; the pancreas exhibited partial venous thrombosis that was managed conservatively with intravenous heparin. The recipient of the simultaneous kidney–pancreas transplant now has normal amylase and sugar levels and normal renal function.

CASE 2: ELECTRICAL INJURY TO THE LIVER DURING ORGAN PROCUREMENT

The donor was a 32-year-old male subject with a hypoxic brain injury. After confirmation of brain death and appropriate assessments, the heart, lungs, liver, pancreas, and kidneys were offered and accepted for transplantation.

During the organ procurement and at warm phase inspection, the liver had an optimal appearance and was characterized as potentially splittable. Intraoperatively, the patient went into ventricular fibrillation. Due to lack of internal defibrillation paddles, the electric shocks were delivered through external pads. The anesthesiologist applied the right external pad on the right lower thorax/upper abdomen, therefore including the liver in the arc (Fig 1C). The donor received 3 cycles up to 200 J. After transient sinus recovery, ventricular fibrillation relapsed, necessitating 3 additional electroshocks (all 200 J). The changes on the surface of the right liver lobe became evident during the cold phase, whereas the liver appearances had been unremarkable at the onset of the procurement surgery and throughout the warm phase. The procured organ was examined at the back-table at the transplant center by the same attending surgeon who examined and implanted the liver described in case 1. Upon cold perfusion, an ill-defined, 6 × 5 cm, patchy, mauve mark was noted on segments V/VI, which was interpreted as an electric shock–related injury (Fig 1D). Because the electrical injury happened during procurement, it was not possible to assess post–electrical injury serum AST levels.

The recipient was a 63-year-old female subject with primary sclerosing cholangitis. AST and bilirubin peak levels were 573 IU/L and 166 μmol/L, respectively (day 1). The patient had an uneventful postoperative recovery and was discharged from the hospital on posttransplant day 14 with normal liver function. At her follow-up appointment on postoperative day 70, all liver function test results remained normal (namely, AST was 26 IU/L, total bilirubin was 10 μmol/L, ALP was 61 IU/L, and γ-glutamyl transferase was 17 IU/L). Graft ultrasounds performed on days 1, 5, and 8 demonstrated uniform reflectivity and no evidence of biliary dilation. Similar to the first case, given the normal laboratory values and the repeatedly unremarkable ultrasound appearances, no further imaging was deemed necessary at that stage.

In addition to the liver, the donor's heart and kidneys were also transplanted. The heart recipient had a good recovery. One kidney was transplanted into a 37-year-old woman who had been on hemodialysis for 5 years. She had an uneventful postoperative recovery and has continued to do very well; the patient's latest creatinine measurement was 75 μmol/L.

DISCUSSION

Fatal electrical injuries are uncommon. Even though transient loss of consciousness and other neurologic sequelae are common during electric shock trauma, they are usually benign; however, direct or indirect cerebral electricity insult may result in brain death. The first case described here is such an example. Such donors have been considered with suspicion, due to the unpredictable impact of electrical injury on the organs of interest. The selection and assessment of organs affected by the electrocution are dictated by the electricity arc between entry and exit points. Electrocution is classified into low and high voltage (>1000 V), with the latter associated with the highest mortality [4]. Electric shocks may further be categorized according to the current type (direct or alternating). Most electrocutions are from alternating currents, whereas direct current–related fatalities typically occur secondary to lighting strikes [5]. In alternating current injuries, the involuntary prolonged spasms of the muscles adjacent to the entry point of the electricity tend to prolong contact duration and the amount of current passing through the body [6].

Electrical injury is caused by a combination of thermal and nonthermal mechanisms [7,8]: current generates heat according to Joule's law and results in irreversible [5] macromolecular denaturation and extensive coagulative necrosis [7]. Electroporation [6] destroys membrane integrity and alters membrane potential, resulting in irreversible cellular damage. The current triggers mechanical energy–related injury, secondary to violent muscle contractions. The outcome of electric shock injury depends on amperage, current type, frequency, voltage, contact duration, contact surface area, tissue resistance, and current pathway. At typical transmission frequencies (ie, 50–60 Hz), alternating current is suggested to be twice as dangerous per unit of applied voltage.

The electrical current's track through the body depends mainly on the relative resistance of the potential exit points [9], which in turn determine which organs are affected and to what extent. The current follows the shortest arc bridging entry to exit. Bones, adipose tissue, and tendons are known to have the highest resistance, whereas tissues with high

water content (eg, muscle, mucous membranes, blood, nerves) have the least. Dry, nonbroken skin has intermediate resistance [10]. Passage through the thorax has an estimated 60% mortality, whereas passing through the low extremities is lethal in 20% of cases [10–12]. Death after electrical shock is usually due to fatal arrhythmias or respiratory paralysis, with ventricular fibrillation being the main cause of electric shock death [11,13]. Electrocution may result in brain death after catastrophic brain hemorrhage or secondary to traumatic brain injury, severe cerebral edema, or hypoxic brain damage secondary to respiratory paralysis or cardiac output loss [14,15].

With the first donor, the cause of brain death was hypoxic brain injury; subarachnoid hemorrhage, even though present, would not be the cause of death because there was no evidence of mass effect. The hypoxic brain injury was most likely due to anoxia secondary to the transient cessation of respiratory function during the electrocution and the apnea which followed until resuscitation was undertaken by the bystanders and the emergency ambulance crew. In both cases, current passed through the liver, causing coagulative necrosis that gave the affected tissue a darker purplish coloration. In case 1, the injured liver was adjacent to the gallbladder, which attracted the electric arc, thus sparing the rest of the surrounding liver in contrast to case 2, in whom the current traversed through the liver until it reached the inferior vena cava (thus causing less well-demarcated heat and electroporation injury of an unpredictable extent).

Little is known about the laboratory profile changes after direct electrical injury to the liver. However, an electrical current prefers the route of least resistance, particularly through liquid mediums (blood and bile), rather than mediums of higher resistance (hepatocytes and connective tissue). The amount of energy released and the ensuing hepatic injury would be proportional primarily to the surface area, contact duration, current intensity, and voltage.

The laboratory profile after electrical injury to the liver would probably follow the respective biomarkers' behavior in blunt abdominal trauma. In a prospective study of 122 patients with blunt abdominal trauma, no patient with normal ALT levels had hepatic injury [16]. Almost all patients with liver injury (31 of 32) had elevated ALT findings, the level of which correlated with the severity of the insult. Likewise, a retrospective study of 99 patients with blunt abdominal trauma found that ALT levels >2 was associated with major hepatic injury; the same applied to patients with simultaneously elevated levels of ALT >2 times and AST >2 times [17]. Patients with normal ALT and AST levels were unlikely to have major liver damage. This difference was not seen with serum bilirubin and ALP levels.

Perhaps contrary to blunt abdominal trauma, electrical injury is commonly associated with extensive violent muscle contractions and resulting rhabdomyolysis, which dominates the laboratory picture, because abnormal serum AST and ALT levels in the setting of rhabdomyolysis occur in 95% and 75% of cases, respectively. AST levels decrease in parallel with CK levels for the first 6 days of hospitalization of such patients, indicating that skeletal muscle is a significant source of AST elevation in these occasions [18]. Thus, hepatocyte injury after direct exposure to electricity would be expected to be represented with high (>2) serum AST and ALT levels. Because a degree of rhabdomyolysis is to be expected during electric trauma, ALT is the preferred hepatic injury biomarker due to its specificity for liver injury, particularly in the presence of high CK levels. Biliary injury, even if it is significant, is not expected to be reflected immediately in the laboratory profile because cholestatic enzymes require a lag phase of induction and a period of impaired clearance to reach abnormal levels; these findings are therefore of questionable value in the acute setting. In our first case, cholestatic enzyme levels remained normal even though the electric current had evidently traversed the gallbladder, whereas serum AST levels were raised significantly and in parallel to CK levels.

Although posttransplantation AST/ALT levels are a widely accepted indicator of graft injury, the donor's transaminase levels are not considered absolute risk factors [19,20]. Grafts from donors with AST/ALT levels several times higher than normal often function well, especially when they are falling before procurement. It is the trend rather than the absolute transaminase concentration that drives the surgeon's final decision regarding suitability of organs. At a cellular level, electric hepatocyte injury is secondary to direct thermal damage and electroporation. Because AST/ALT levels reflect hepatocyte injury, and even high absolute transaminase levels do not necessarily exclude organ donation (especially if they are declining), there is no reason why the same should not apply in electric hepatic trauma. In such cases, especially if electric trauma to the organs of interest is suspected, the donor should undergo cross-sectional imaging before procurement.

The presentation of electrical injury to the liver is polymorphic and might not be immediately evident [21]. Initial hepatic necrosis can evolve into scarring or abscess formation. Microvascular thrombosis due to activation of the coagulation cascade triggered by electricity passage might translate into immediately apparent hepatic necrosis and/or biliary ischemia, resulting in ischemic cholangiopathy or biliary stricture and prestricture dilation at the injured areas, which might remain asymptomatic or manifest as cholestatic or biliary septic episodes. Hepatic parenchymal necrosis should be immediately apparent upon graft reperfusion. In such cases, the ischemic area should be closely monitored for potential transformation to abscess and ischemic cholangiopathy. Imaging with ultrasound and magnetic resonance imaging scans will identify any subsequent problems.

One case series on DBD caused by electric shock has been reported [1]. Of 3 pediatric donors who had suffered brain death due to anoxia by electric shock, 2 had altered liver enzyme levels before donation; however, all procured livers and kidneys were successfully transplanted. Hearts and lungs were not offered because of a lack of compatible recipients on the waiting list. The transplanted organs showed normal viability and function.

Due to current trends in organ donation and the need for more grafts, the drive to utilize all suitable donors should be maintained. Knowledge of the injury mechanics, along with appropriate assessment, can help the surgeon assess the suitability of donors experiencing electric shock injury. In addition, management of donors before procurement should be as multitrauma/burn patients to optimize organs for donation and protect them from further insult; this approach includes cardiac monitoring for cardiac arrest due to electrolyte disturbances and renal protection from extensive rhabdomyolysis or hypoperfusion injury.

Abdominal injuries after electric shock are rare, with the large and small bowel more commonly involved, followed by the stomach, pancreas, liver, gallbladder, and kidneys [22–25]. Injuries to the bowel may exclude donation if soiling is extensive. A proportion of electrocuted patients may also have traumatic visceral injuries from electric shock–triggered collisions or falls [14]. Finally, electrical injury to the allograft can occur iatrogenically after defibrillation or cardioversion during organ procurement surgery, as in the second case. If the heart is readily accessible, it is advisable to use internal cardiac paddles; if sternotomy and pericardiotomy have not been performed at that stage, care should be taken for the pads to be placed in such a way to ensure that the liver is insulated from the arc (ie, right paddle should be placed at the upper right hemithorax and the left paddle at the lower posterior-lateral chest, encasing the heart into the arc). If the right paddle is placed at the level or below the diaphragm, the shock is at risk of being delivered through the liver.

CONCLUSIONS

The reported cases described here demonstrate that allografts from electric shock injury donors can be transplanted with good outcomes. Knowledge of electrical injury pathophysiology and education of those involved in the management of such donors prior to organ donation and of the respective procurement surgeons, can facilitate optimal use of such organs.

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