A high suspicion for intra-abdominal injury must always accompany the initial assessment of a patient involved in a motor vehicle collision, particularly if physical examination reveals the characteristic “seatbelt sign” (ecchymosis and/or abrasion of the lower part of the abdomen with or without similar markings across the side of the neck and the shoulder).\(^1\) Blunt trauma to this region can result in damage of variable severity to nearly any abdominal structure, visceral or musculoskeletal, depending on the dynamics of the impact. We present a case in which a functioning renal allograft lying in the right iliac fossa was severely injured by seatbelt compression, resulting in significant functional compromise and eventual loss. The patient later underwent successful retransplantation with a second living donor kidney.

**Case Report**

In December 2006, a 45-year-old man who had undergone living donor kidney transplantation in 1992 was a restrained driver who broadsided another vehicle. Although his condition was initially stable, he became hypotensive and received aggressive resuscitation with blood and crystalloids during transfer. On arrival at our level I trauma center, the patient had hematuria, abrasions over the right side of his chest, and an abdominal seatbelt sign demarcating an area of tenderness overlying his right-sided allograft. When focused abdominal sonography for trauma revealed free fluid, he was taken emergently to the operating room for exploratory surgery.

During laparotomy, he was found to have massive hemoperitoneum. Injuries included tears of the splenic capsule and the serosa of the transverse colon, and a grade IV injury of his renal allograft, with partial avulsion of the upper pole extending into the hilum and active bleeding resulting in retroperitoneal hematoma. Splenectomy was performed, and after evacuation of the hematoma, allograft bleeding was controlled with hemostatic sutures, topical application of procoagulants, and packing. His abdomen was closed in a temporary fashion, and he was taken to the intensive care unit for continued resuscitation and completion of his trauma assessment. Computed tomography of the abdomen obtained immediately postoperatively showed only segmental perfusion of the injured allograft in the right iliac fossa (Figure 1).

Initially the patient responded well, with stable hemodynamics and urine output greater than 100 mL/h. Sixteen hours later, the patient had worsening tachycardia, hypotension, and oliguria, and underwent exploratory surgery again for presumed hemorrhage. However, no
some are more susceptible to injury than others. The propensity of an organ or structure to be damaged by blunt impact, particularly as delivered by an overlying seatbelt, is related to its anatomic location and attachments. Especially vulnerable are structures that are either fixed, thereby unable to reconcile shear forces, or superficially located, with limited surrounding soft tissue to provide a protective cushion. For these reasons, a heterotopic renal allograft lying in the iliac fossa is at significant risk of injury from seatbelt compression.

Surprisingly, blunt trauma involving transplanted kidneys is infrequently described, and among the reported cases, only a handful are related to seatbelt injury. A review of the literature by Akabane et al indicated that reported cases of blunt allograft trauma shared preservation of the graft, either with surgical or conservative management. Our review including more recent reports confirms this finding, with the exception being described by Mohammed et al. In that case, the allograft lost function over several months after blunt impact, necessitating the patient’s return to hemodialysis. Our patient may be the only reported case of seatbelt-related allograft injury with significant functional compromise and eventual loss, although it is also possible that

significant bleeding was seen; in fact, the allograft was found to be hemostatic with intact vasculature. The patient was returned to the intensive care unit and required no additional surgical procedures during this admission. A subsequent nuclear medicine renogram showed partial perfusion and suboptimal function of the allograft, with delayed excretion of radio-isotope into the urinary bladder, but no urine leak (Figure 2).

During this hospitalization, the patient’s serum level of creatinine initially stabilized at 3.7 mg/dL (to convert to micromoles per liter, multiply by 88.4). Previous creatinine levels were 2.5 mg/dL at baseline and 3.0 mg/dL on presentation. However, the creatinine level increased to 5.3 mg/dL 2 weeks after the accident. Computed tomography of the abdomen showed a large perinephric fluid collection; this was drained percutaneously with some improvement in renal function. The fluid was found to be a seroma. The patient’s urine output remained greater than 1 L daily. At discharge to a rehabilitation facility 1 month after the accident, his creatinine level was 3.6 mg/dL. During the months following, his serum level of creatinine gradually increased to a peak of 6.7 mg/dL, and he returned to our center for possible retransplantation. The patient underwent a second living donor transplantation 6 months after the trauma event. His postoperative recovery was uneventful and he was discharged home on postoperative day 5, with a serum creatinine level of 0.9 mg/dL (Figure 3).

Discussion

Although the benefit of seatbelt use in providing passive occupant restraint during motor vehicle accidents is widely accepted, their use is not without risk. The patterns of injuries resulting directly from use of seatbelts have been widely investigated and described. Nearly every major abdominal structure is reported to have been injured by blunt abdominal trauma, although
such an adverse outcome after allograft injury by this specific mechanism is both seldom encountered and underreported.

Management of acutely injured kidney transplant recipients is not altogether different from their counterparts who do not have a transplanted kidney, because the priority is always to save the patient. Selective nonoperative management, now becoming the preferred treatment for most blunt trauma to native kidneys, should still be the guiding principle in addressing allograft trauma, if the patient is clinically stable. For instance, angiographic embolization, which is commonly used for injuries to the native kidney, was recently reported to be successful in preserving function of a renal allograft following seatbelt injury, thus avoiding transplant nephrectomy.

If surgery is unavoidable, the most appropriate procedure depends on the severity of injury. Previously, Urquhart et al proposed that no graft-directed operation besides nephrectomy is feasible or beneficial in trauma to an allograft, as efforts to repair the damage are often unsuccessful. More recent cases have described use of various surgical approaches short of nephrectomy to preserve allograft function. According to the organ injury scale, our patient sustained a grade IV injury, with the laceration extending through the renal cortex, medulla, and collecting system (see Table). Grade IV injuries are arguably the most controversial in terms of management, as lesser injuries are typically well-managed conservatively and greater injuries usually necessitate intervention. Admittedly the situation is different when addressing a damaged renal allograft rather than a damaged native kidney: the transplanted graft is solely responsible for obviating hemodialysis, unlike the shared function of paired native kidneys. It is difficult to speculate whether our patient’s allograft injury alone would have directed the same course of action, because he required exploratory surgery for hemorrhage from his splenic injury. In retrospect, repairing the damaged allograft, rather than performing nephrectomy, allowed some degree of renal function to exist for several months until retransplantation.

We expected allograft function to return with medical and expectant management, because its vascular supply was intact. Despite this, the allograft eventually proved unable to withstand the multiple injuries to which it was subjected. Already compromised by chronic allograft nephropathy, the kidney was subjected not only to direct compression by the seatbelt causing parenchymal damage, but also to early prolonged hypotension, as well as generous amounts of intravenous contrast material from the initial computed tomography, which together resulted in acute tubular necrosis and functional compromise. Blunt trauma to a renal allograft has been reported to precipitate acute rejection. Our patient, however, had appropriate immunosuppression levels throughout his

Figure 3  Serum levels of creatinine from before trauma admission to the time of retransplantation.

![Serum levels of creatinine](image-url)
hospitalization, and we never suspected involvement of an acute rejection process. Rather, it was the cumulative effect of these events that accelerated progression of the patient’s underlying chronic nephropathy and led to allograft failure.

Conclusion

As more people undergo kidney transplantation, and advances in immunosuppression allow improved allograft survival, it is inevitable that an increasing number of patients with kidney transplants will be treated by trauma centers for injuries directly or indirectly involving their allograft. It is important to remember that measures taken to preserve allograft function should never compromise efforts to resuscitate the patient. As such, losing the allograft should not be regarded as failure, because the patient can still be a candidate for retransplantation and can have a successful outcome.

References