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Original Article

Severity and Outcome Analysis of Abdominal Vascular Injuries at a Tertiary Care Service Hospital

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Abstract

Introduction: Abdominal vascular injuries (AVIs) remain the most common cause of morbidity and mortality following penetrating and blunt abdominal trauma. This study was carried out at a tertiary care service hospital to review the institutional experience by analyzing the severity and outcome determinants of these injuries.

Materials and Methods: It was a retrospective observational study over 5 years from January 2012 to December 2016. The data available from the hospital records were analyzed to determine the mortality based on shock, on the number abdominal vessels injured along with other significant associated injuries. The primary outcome measure was survival.

Results: Of 192 vascular trauma patients, abdominal vascular injury was found in 17 (8.85%) cases accounting for 25 injured vessels. Two or more abdominal vessels were injured in four patients. AVI followed blunt abdominal trauma in 85% ofcases. External iliac arteries and inferior vena cava were the most commonly injured artery and vein, respectively. Arteries and veins were injured almost in equal proportions. Surgical interventions carried out were fog arty thrombectomy, vascular repair, autologous or prosthetic interposition graft, or vessel ligation. Five patients died within 24 h of injury as a direct consequence of AVI, whereas 3 patients died later due to associated injuries or other causes.

Conclusion: AVIs are highly lethal and major impact is seen within initial 24 h. In our setup, blunt trauma is responsible for majority of these potentially fatal injuries. Multiple vessel injury and serious associated injuries are responsible for poor outcome. Early recognition, balanced resuscitation, and damage control principle can possibly contribute to better survival.

Key words: Abdominal vascular injuries, Blunt, Penetrating abdominal trauma

INTRODUCTION

Abdominal vascular injuries (AVIs) involving major vessels are uncommon in everyday practice, but whenever encountered, these are highly lethal vascular events. Predictably, exsanguinating hemorrhage is the most important cause of early mortality in such cases. Intra-AVIs are associated with rapid blood loss and pose significant challenges of vascular exposure and control during laparotomy, given the posterior position of the majority of major abdominal vascular structures. Some of these patients may present in Emergency Department (ED) with cardiopulmonary arrest and may necessitate resuscitative ED thoracotomy, aortic cross-clamping, and open cardiopulmonary resuscitation en route to operating room (OR). A thorough knowledge of intra-abdominal vascular anatomy and a familiarity with the techniques of proximal and distal control combined with selective application of primary repair, bypass, or ligation as indicated is extremely essential for the successful management of these injuries.

The main aim of this study was to analyze the mechanism of injury, injury severity, and outcome of patients with

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intra-AVI seen following abdominal trauma, to study the effects of resuscitation and damage control surgery (DCS) on the outcome of these injuries, and to review our institutional protocol and experience with these injuries.

MATERIALS AND METHODS

It was a retrospective observational study of 17 consecutive cases of AVIs as a result of either isolated blunt or penetrating abdominal trauma or in a setting of polytrauma, who presented at our Trauma Center over 5 years from January 2012 to December 2016. Data were collected from hospital medical records for patient's age, gender, mechanism of injury, mode of injury, injured vessels, associated injuries, presence of shock at presentation in ED (systolic blood pressure [SBP] ≤90 mmHg), Glasgow coma scale, associated injuries, injury severity score, and mortality. Focused assessment sonography in trauma (FAST) was performed on all cases as primary survey protocol. Contrast-enhanced computed tomography (CECT) abdomen or CT angiography was performed in hemodynamically stable patients.

All the patients who presented with polytrauma with abdominal trauma or isolated blunt or penetrating abdominal trauma along with injuries to named intraabdominal vessels as identified either on CECT scan of abdomen and pelvis, CT angiogram, or during surgical exploration were included in the study. Those cases who died in ED during resuscitation or were found to have mesenteric tears as source of bleed were excluded from the study.

Decisions concerning the restoration of intravascular volume after injury were made using a standard algorithm according to the patient's initial hemodynamic status, response to crystalloid administration, and reassessment of hemodynamic stability using patient's vital parameters, level of consciousness, and urine output.[1] Patients who presented with Class I or Class II shock were infused with crystalloids. Transfusion of packed red blood cells (RBCs) was done in patients who responded only transiently to crystalloid administration. For refractory cases, the massive transfusion protocol was activated with the transfusion ratio of packed RBCs, fresh frozen plasma (FFP), and platelet as 1:1:1.

DCS was performed in the presence of unachievable hemostasis because of refractory coagulopathy, injuries amenable to packing, limited access to a major venous injury, an anticipated need for a time-consuming procedure, and bowel edema.

All statistical calculations, including Chi-square analysis and unpaired *t*-tests, were performed using SSPS version 17, and the data were reported as mean standard deviation. Specifically, we studied the mode of injury, shock as initial presentation, surgical procedure employed to stop bleed or to repair vessel and mortality. Follow-up data including length of intensive care unit stay, duration of ventilatory support, and if the patient died, cause, date, and time of death were recorded.

RESULTS

During the study period of 5 years from 2012 to 2016 from hospital records of the patients, 192 patients had presented to our center with vascular trauma including 17 (8.85%) patients who had sustained a major abdominal vascular injury. In these 17 patients, 25 named vessels were injured with varying degrees of severity. Two or more than two abdominal vessels were injured in 4 patients. During the same period, 681 abdominal trauma patients were admitted in our center, majority (78.35%) with blunt abdominal trauma, responsible for causing AVI in 2.80% of (15/534) cases, whereas penetrating abdominal trauma caused AVI in 1.36% (2/147) of the cases. Predominant mechanism of injury was road traffic accidents seen in 11 cases [Table 1]. The mode of injury was blunt trauma in fifteen (85%) of our patients as compared to penetrating trauma that was seen in the remaining 15%. Sixteen patients were brought within 24 h of injury and the average ED disposition time was 90 min. Injury to either artery or vein was observed in almost equal numbers, the most common arterial, and venous vessels to sustain injuries being external iliac artery [Figure 1] and the inferior vena cava (IVC) in 6 and 4 patients, respectively [Figures 2 and 3]. There were three renal pedicle injuries or avulsions [Table 2]. Single vessel injuries were seen in 13 cases and the remaining 4 cases had injuries to multiple vessels.

FAST was positive in 15 patients. Six patients, who were hemodynamically stable, were evaluated with CECT abdomen pre-operatively and revealed evidence of contrast leak from the injured vessel(s) in 4 cases and evidence of free fluid or blood in the intraperitoneal or retroperitoneal space including pelvis.

All the patients underwent exploratory laparotomy. Seven of the 11 venous injuries were repaired and four were ligated. Fogarty balloon thrombectomy was done in five of the 10 arterial injuries, ligation in 3 and end-to-end and prosthetic graft repair was carried out in one each. All three injured or avulsed renal pedicles were ligated to achieve control of hemorrhage followed by ipsilateral nephrectomy. Perihepatic or pelvic packing was done in 5 cases for associated hepatic or pelvic injuries.

Immediate mortality as a direct consequence of vascular injuries leading to hemorrhagic shock was observed to be 28.5%. Three of them died in OR during surgery and another two died post-operatively within 24 h of injury. Vessel wise immediate, early, and late outcomes are summarized in Table 3. Another three cases died later consequent to coagulopathy, acute renal failure, associated injuries, and sepsis. The overall 30 days survival was 52.9%.

DISCUSSION

AVIs are among the most lethal injuries encountered by modern-day trauma surgeons. The incidence of abdominal vascular injury varies depending on the injury setting. The incidence of abdominal vessel injury in patients with blunt trauma is estimated at approximately 5–10%. A similar incidence of 10.3% is reported in patients with penetrating stab wounds to the abdomen. In our study, the incidence was lower for both blunt (2.80%) as well as for penetrating (1.36%) abdominal trauma, whereas 75% of the patients with AVI sustained blunt trauma resulting in 7 deaths of 8 ($P = 0.338$). Patients with gunshot wounds (GSWs) to the abdomen are likely to have major vessel injury in 20–25% of cases.[2] We had one case of GSW who died of hemorrhagic shock.

Most of the initial experience in vascular trauma has emerged from the battlefields. During World War I, DeBakey and Simeone reported a 2% incidence of AVIs in 2471 patients.[3] Hughes reported seven (2.3%) iliac artery injuries in 304 patients.[4] Rich *et al.* reported 29 patients (2.9%) during the Vietnam conflict.[5] Jawas *et al.* reported 36 (10%) cases of major vascular injuries among 361 war-wounded admissions during second gulf war.[6] Over the past few decades now, these injuries are being reported with greater frequency from the civilian arena. These serious injuries are associated with extremely rapid rates of blood loss and pose challenges of exposure during laparotomy in view of the posterior position of the major abdominal vascular structures.[7] Patients also present in a hyperfibrinolytic state, which exacerbates the coagulopathy associated with the lethal triad of trauma.[8]

In 1984, Feliciano reported a 15% incidence of AVI seen in their trauma center.[9] In our study, 17 patients or 8.85% of 192 vascular injuries were AVIs. During this 5 years' study period, contrary to the existing data in favor of predominance of penetrating injuries by various authors,[10-12] we observed that majority of the injuries were following blunt abdominal trauma. Most of the 17 patients studied were found to have a single vessel injury, whereas four of them had injuries to multiple vessels. Although three of these four patient died, the mortality was not statistically significant ($P = 0.121$). Despite advances in prehospital emergency care trying to ensure these patients arrive with signs of life at trauma centers, few series have appeared before 1980 in the literature describing their management after reaching the hospitals.

The main consequence of these injuries in the vast majority of cases is hemorrhagic shock from intra-abdominal hemorrhage often leading to metabolic acidosis accompanied with coagulopathy and hypothermia, the so-called lethal triad of trauma.[8,13] Metabolic acidosis in trauma patients is the result of lactate overproduction, most often from hypoxia and hypovolemia. Acidosis adds to the overall lethality of preexisting injury primarily by depression of myocardial contractility and by impairment of coagulation. Furthermore, moderate-to-severe hypothermia (below 34°C) inhibits platelet function and slows coagulation factor activation. This self-perpetuating cycle is responsible for 80% of deaths in patients with major vascular injury and must be rapidly corrected to

Table 1: Mode of injury

Mode of injury	No injured
Road traffic accidents	11
Fall from height	3
Crush injury	1
Stab/empalement	1
Gunshot wounds	1
Total	17

Table 2: Injured vessels

Injured vessel	No injured
IVC	4
PV	1
SMV	1
Splenic vein	1
Renal pedicle	3
Common iliac artery	1
Common iliac vein	1
External iliac artery	6
External iliac vein	3
Internal iliac artery	3
Internal iliac vein	1
Total injured vessels	25

IVC: Inferior vena cava, SMV: Superior mesenteric vein, PV: Portal vein

Table 3: Outcome analysis

Injured vessel	Immediate outcome	Early deaths	Late deaths
IVC	3 Survived; 1 died	1	1
PV, SMV, Splenic V	One patient; died	-	-
Renal vessels	3 Survived	-	1
Common iliacs	2 Survived; 1 died	-	1
External iliacs	5 Survived	-	-
Internal iliacs	3 Survived	1	-
25 Vessels	16 Survived, 3 died	2	3

IVC: Inferior vena cava, PV: Portal vein, SMV: Superior mesenteric vein

prevent a dismal outcome. Patients also present in a hyperfibrinolytic state, which exacerbates the coagulopathy associated with the lethal triad of trauma.[8,14] In our series, 12 patients presented with shock resulting in mortality in 7 of them ($P = 0.146$). The remaining eleven patients who were not in shock underwent a detailed pre-operative evaluation to establish the nature and extent of the injuries.

Since 1980, there have been several series dealing exclusively with AVIs. Wiencek and Wilson reported one of the largest series consisting of 254 injuries over 5 years.[15] Asensio *et al*. published one of the largest series in the year 2000 consisting of 302 patients with 504 injured vessels managed in an urban trauma center.[16] In this series, 275 patients (91%) had retroperitoneal hematomas, the vast majority being located in zone I. Exposure for zone I supramesocolic injuries includes medial rotation of the left-sided viscera. This exposes the aorta from its entrance into the abdominal cavity through the aortic hiatus and includes exposure of the origin of the celiac axis, superior mesenteric artery (SMA), and the left renal vascular pedicle.[11,17] An alternative approach is the extended Kocher maneuver that exposes the suprarenal abdominal aorta between the celiac axis and the SMA but does not expose the supraceliac aorta at the hiatus.[10,18] Maneuvers used to expose injuries in zone I inframesocolic include reflecting the transverse colon and mesocolon cephalad, eviscerating the small bowel to the right, and transecting the ligament of treitz along with the loose tissue along the left side of the abdominal aorta until the left renal vein is located. This exposes the infrarenal aorta. To expose the suprarenal and infrarenal vena cava, the avascular line of toldt of the right colon is transected along with a Kocher maneuver sweeping the pancreas and duodenum to the left and incising the retroperitoneal tissues that cover the IVC.[11,19]

Hemodynamically stable patients with blunt trauma and suspected AVIs may benefit from triple-contrast abdominal CT scanning which helps localize a hematoma and evaluate solid organ injuries.[20,21] Stable patients with posterior wounds and most patients with anterior stab wounds should be evaluated with FAST examination to exclude hemoperitoneum, and those with equivocal abdominal signs with stab wounds may undergo laparoscopy to confirm peritoneal penetration. Patients with peritoneal signs or positive FAST and CT findings suggestive of ongoing bleed require exploration. Hemodynamically unstable patients with positive clinical findings and positive FAST also require immediate surgery for control of hemorrhage. If time permits, chest and pelvic radiography should also be performed to exclude bleeding into the chest or pelvic cavities. Many patients arrive with severe physiological compromise secondary to massive blood losses and associated injuries.[18,22] Angiography with or without embolization may be considered in stable patients, particularly in patients with blunt trauma [Figure 4].

Hemodynamically unstable patients with penetrating trauma including GSW of the abdomen should be transported to the OR as soon as possible after ensuring a secure airway and adequate ventilation, and no imaging studies are necessary. Patients with GSWs to the abdomen require laparotomy for evaluation and treatment, although some trauma surgeons prefer selective non-operative evaluation of abdominal GSWs in stable patients.[2] The assessment of hemodynamically unstable patients with blunt trauma to the abdomen may include FAST or diagnostic peritoneal lavage to confirm hemoperitoneum as well as portable chest radiography only if expeditious transport to the OR is not getting delayed.

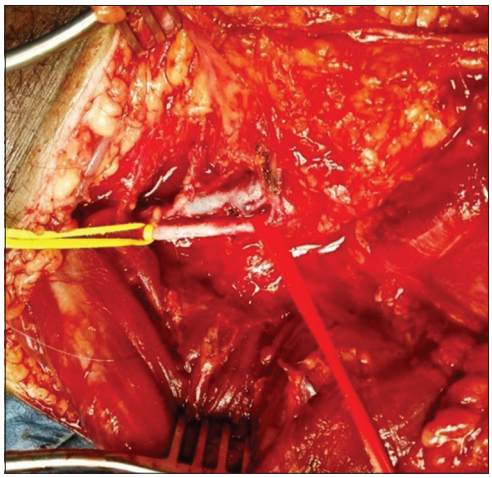

Figure 1: Injury to both left external iliac vessels

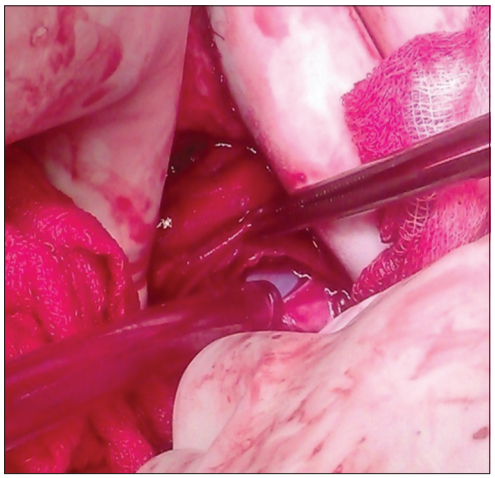

Figure 2: Inferior vena cava injury

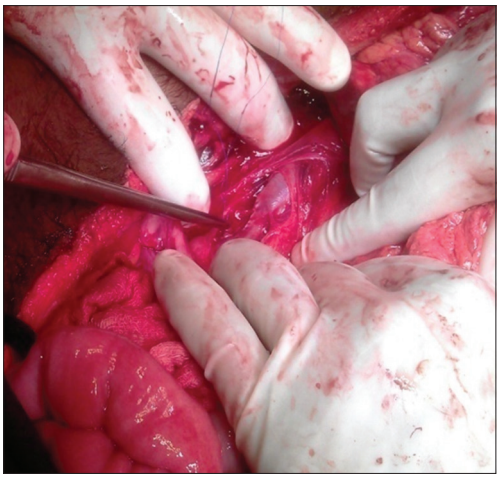

Figure 3: Inferior vena cava injury repaired

Figure 4: Post-nephrectomy right renal artery stump leak-embolized successfully

In a hypothermic traumatic shock swine model, Ding *et al.* demonstrated that temporary intravascular shunts may improve survival in SMA injuries compared with repair by primary vascular anastomosis.[14] Relative to pigs in the primary vascular anastomosis group, the animals treated with temporary shunting required less resuscitation fluid, retained higher SMA flow rates, normalized lactate levels faster, suffered less severe intestine histopathology, and had greater early survival.

Initial resuscitation of a patient with AVIs depends on his or her condition at arrival in the ED. As a possibility of intra-abdominal venous injury exists, lower extremity venous access is not recommended. Patients may require aggressive resuscitation involving the correction of acidosis, active rewarming, and massive blood transfusion for patients presenting with a shock index (heart rate/SBP) of >0.9.[23] FFP, platelets, cryoprecipitate, or recombinant factor VIIa may be required on an individual basis to correct coagulopathy induced by massive transfusion. A planned reoperation 24–28 h after the initial procedure is done complete a damage control sequence that has proven to be an invaluable technique in the management of severe injuries.[19,24,25] In our series, 35% of patients underwent damage control consisting of vessel ligation, bowel stapling, and packing. Similarly, 12% of the patients required prosthetic abdominal wall closure to deal with the sequelae of their ischemic reperfusion injury, massive volume replacement, and to prevent and to deal with the abdominal compartment syndrome.

Injuries to major abdominal vessels are uncommon but highly lethal vascular crises. Predictably, exsanguinating hemorrhage is the most important cause of early death [17,22,26] In our study, 28% of patients died of hemorrhagic shock within 24 h and half of them in OR during damage control resuscitation. Our overall mortality was 47% somewhat higher than the range reported in the literature of 32–46%.[27-29] Exsanguination accounted for 84% of mortality and the vast majority of deaths (136 of 162 patients) succumbed to the operative or perioperative period.[17] Corresponding figures in our series were 5 of 8 deaths. The most commonly injured arteries were the external iliacs, and the most commonly injured vein was the IVC. Injury to the IVC and portal vein had the poorest chance of survival. Despite many recent advances in shock management and damage control, AVIs remain extremely lethal.[15,26,30] In a study by Paul *et al.*, one hundred forty-four patients with one vessel injured had a mortality rate of 18.7%, whereas those with more than one vessel injured had a mortality rate of 48.7% ($P < 0.001$). A total of 46% of 117 patients in shock died compared with 9.6% of 104 patients not in shock ($P < 0.001$).[31] Similar results were observed in our study also with a mortality of 44% with single vessel injury and 75% with two or more than two vessel injuries ($P = 0.121$) [Figure 1]. Seven of 12 patients in shock (58%) died within 24 h, whereas one (10%) patient died out of 10 who were not in shock at presentation ($P = 0.146$). Other significant non-vascular injuries associated with or contributing to mortality were pelvic fracture, thoracoabdominal injuries, severe head injuries, and long bone fractures [Table 4].

Essential to the successful management of these injuries is a thorough knowledge of intra-abdominal vascular anatomy and a familiarity with the techniques of proximal and distal control combined with selective application of primary repair, bypass, or ligation as indicated. Exposure of the abdominal vasculature is difficult due to its protected location, as well as the presence of large retroperitoneal hematomas, which make approaches to these vessels quite difficult. Damage control principles should be observed in most of these patients to obtain desired outcome.

Table 4: Mortality predictors

Associated injuries	No injured	Deaths	*P* value
Pelvic fracture	6	4	0.164
Long bone fractures	4	3	0.121
Thoracoabdominal trauma	3	2	0.658
Severe head trauma	4	3	0.041

CONCLUSION

The surgical exposure and the often-associated intra-abdominal, pelvic, and other associated injuries may challenge the skills and judgment of even the most experienced surgeons. Rapid transportation to a trauma center, early recognition of the injuries, early surgical interventions, excellent knowledge of the anatomy, and good surgical judgment are critical for survival in such injuries. There have been some significant advances in the management of AVIs during the past decade. The introduction of the policy of scoop and run and early surgical control of the bleeding have now become the standard of care and have improved the survival in vascular injuries. The concept of damage control has gained popularity and acceptance, and many non-salvageable patients with vascular injuries have been saved. The recognition of abdominal compartment syndrome and the use of temporary abdominal wall closure with a prosthetic material are also an important step in improving the outcome. Endovascular technology has revolutionized the management of selected patients with specific vascular or solid organ injuries and false aneurysms. Finally, research in new powerful hemostatic agents is promising and may have a major impact in the management of AVI in the future.

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