World Journal of Surgery. 2012; 36:1571-1580

Traumatic aortic injuries associated with major visceral vascular injuries in major blunt trauma patients

Victor X. Mosquera, Milagros Marini, Ignacio Cao, Daniel Gulías, Javier Muñiz, José M. Herrera-Noreña and José J. Cuenca

Abstract

*Background***.** The objectives of this study were to report the clinical and radiological characteristics and outcomes of a series of acute traumatic aortic injuries (ATAIs) with associated injury to major aortic abdominal visceral branches (MAAVBs).

*Methods***.** From January 2000 to August 2011, 10 consecutive major blunt trauma patients with associated ATAI and injury to MAAVBs (group A) and 42 major blunt trauma patients presenting only an ATAI without MAAVB injuries (group B) were admitted to our institution.

*Results***.** Overall in-hospital mortality was 32.7%. In-hospital mortality in group A was 40% and in group B it was 31% ($p = 0.86$). Observed in-hospital mortality was slightly lower than the expected in-hospital mortality in both groups. Mean peak creatine phosphokinase was significantly higher in group A than in group B patients (23,008 ± 33,400 vs. 3,970 ± 3,495 IU/L; $p < 0.001$). Acute renal injury occurred in 50% of group A and in 26.2% of group B patients. Hemodiafiltration was required in 30% of group A and in 9.5% of group B patients. Median follow-up time was 64 months (range = 1–130 months). Group A survival was 60% at 1, 5 and 10 years. Group B survival was 69% at 1 year and 63.3% at 5 and 10 years ($p = 0.15$).

*Conclusions***.** Aortic injuries associated with MAAVB injuries in major trauma patients seem to present in a different clinical scenario. These patients present increased risk of rhabdomyolysis, visceral ischemia, and acute renal failure, as well as higher in-hospital mortality. A multidisciplinary approach combining endovascular and open surgical techniques for a staged treatment of these life-threatening aortic and MAAVB injuries is mandatory in this critical subset of trauma patients.

Introduction

Acute traumatic aortic injuries (ATAIs) associated with injuries to major aortic abdominal visceral branches (MAAVBs) are uncommon but highly lethal situations among major blunt trauma patients. In-hospital mortality in this critical subset of major trauma patients may vary between 50 and 100% [*1*, *2*], with exsanguinating hemorrhage being the most important cause of early death [*3*, *4*]. The vast majority of this subset of major trauma patients present in shock, severely physiologically compromised; sustain multiple injuries; and develop acute renal failure, an abdominal malperfusion syndrome, and/or coagulopathy [*4*, *5*]. Intra-abdominal vascular injuries are associated with extremely rapid rates of blood loss and pose a surgical challenge for exposure during celiotomy [*5*, *6*], given the posterior position of the MAAVBs.

The advent of different endovascular therapies for treating both ATAI [*7*, *8*] and injuries to visceral arteries [*9*–*11*] has enabled a revolution in the management of these catastrophic injuries among major trauma patients leading to a decrease in in-hospital mortality in most current series.

Clinical, anatomical, and radiological characteristics and both in-hospital and long-term survival from ATAI with associated injury to MAAVBs are poorly documented. All the studies published to date are separately focused on either visceral arterial injuries or ATAI management, with a lack of information about the management and outcomes of this critical subset of patients.

The objectives of this study were to describe the clinical profile and outcomes of a series of 10 consecutive patients with ATAI and associated injury to MAAVBs, and to compare such data with our last 10 years' experience managing ATAI in trauma patients.

Patients and methods

From January 2000 to August 2011, 54 major blunt trauma patients were admitted to our institution with ATAI. Two patients were excluded from the analysis because of deficient documentation of the time from injury to procedure or in extremis status on arrival. Among those major blunt trauma patients, 10 patients suffered an ATAI with associated injury to MAAVBs (group A), while the remaining 42 major trauma patients presented only an ATAI without MAAVB injuries (group B).

Data on 96 variables were recorded on a standardized form, including information on patient demographics, mechanism of injury, initial clinical presentation (blood pressure, respiratory rate, need of oral intubation at the site of the trauma or during transportation, Glasgow Coma Scale [GCS]), Injury Severity Score (ISS) [*12*], Abbreviated Injury Score (AIS) for each body area (head, chest, abdomen, extremities), Revised Trauma Score (RTS) [*13*], Trauma Injury Severity Score (TRISS) [*14*], associated injuries, and findings on diagnostic imaging tests.

For the purpose of the study, a major trauma patient was defined as a victim of trauma with an ISS [*12*] greater than 15 according to published literature [*12*, *15*]. An ISS score of greater than 50 predicts a mortality rate of over 50%, while a score greater than 70 predicts a mortality rate of nearly 100% [*12*]. The TRISS score directly predicts the expected death rate for blunt trauma [*14*].

Aortic injuries were classified according to their severity as type I (intimal tear), type II (intramural hematoma), type III (pseudoaneurysm), or type IV (rupture) [*16*]. The site of injury (aortic root-ascending aorta, aortic arch, aortic isthmus, mid and/or distal descending thoracic aorta, and abdominal aorta) and the type of definitive management (conservative, open repair, or endovascular repair) were also recorded.

Studied MAAVBs were left renal artery (LRA), right renal artery (RRA), superior mesenteric artery (SMA), celiac axis, and inferior mesenteric artery (IMA). Injuries to secondary branches were not considered for the purpose of the study. All abdominal vascular injuries were graded according to the American Association for the Surgery of Trauma Organ Injury Scale (AAST-OIS) [*17*].

The mechanism of blunt trauma was classified as motor vehicle crash (MVC), motorcycle crash (MCC), fall; auto versus pedestrian (AVP), crush under weight, and other.

Hypotension was defined as a systolic blood pressure <90 mmHg or the need of fluid and/or inotropic support to maintain a blood pressure ≥90 mmHg. An abnormal respiratory rate was defined as bradypnea <10 breaths per minute or tachypnea >30 breaths per minute. A GCS below 9 points was defined as the cutoff value for neurological bad prognosis on admission.

Acute renal injury (ARI) was defined using published criteria as changes in serum creatinine (increase in serum creatinine by twofold or decrease in glomerular filtration rate >50%) or changes in urine output (urine output <0.5 mL/kg/h during 12 h), or both [*18*]. Acute renal failure (ARF) was defined using published criteria as changes in serum creatinine [threefold increase in serum creatinine; serum creatinine ≥4.0 mg/dL (350 μmol/L) with an acute increase of at least 0.5 mg/dL (44 μmol/L), or a decrease in glomerular filtration rate >75%] or changes in urine output (urine output <0.3 mL/kg/h during 24 h or anuria during 12 h), or both [*18*].

Penetrating trauma was an exclusion criterion for the study.

The Institutional Review Board approved this study based on retrospective data retrieval, waiving the need for individual consent.

Management of ATAI

ATAI management included conservative treatment, thoracic endovascular aortic repair (TEVAR), or open surgical repair according to the clinical and radiological criteria of the trauma team that treated the patient. The criteria of patient management were modified with the incorporation of technological advances in both diagnostic and therapeutic fields, especially with the spread of thoracic aorta endografting [*19*].

Medical therapy included limitation of intravenous fluid infusion once the systolic arterial pressure exceeded 100 mmHg and use of β-blockers to reduce heart rate, cardiac contractility, and blood pressure to the lowest amounts that still maintained adequate end-organ perfusion [*20–22*]. Conservative management underwent a close radiological imaging surveillance protocol as we have previously published [*19*].

In our series, nine patients underwent open surgical repair of ATAI. Based on the time from injury to definitive aortic repair, seven patients underwent emergent (<24 h) open surgical repair and two patients underwent delayed (>24 h) open surgical repair. A Dacron graft was used in six patients and direct suturing of the injured aorta was performed in three patients. A left heart bypass was established in six patients, while in the remaining three cases surgery was performed by simple clamping technique (clamp and sew).

Eight patients underwent emergent (<24 h) TEVAR in our series. Endovascular stent placement procedures were performed in the operating room with patients receiving general anesthesia [*23*]. TEVAR was performed using the Talent® (Medtronic, World Medical Manufacturing Corp., Sunrise, FL, USA) thoracic stent graft in three patients, the Valiant® (Medtronic, World Medical Manufacturing Corp.) thoracic stent graft in four cases, and the Valiant Captivia® (Medtronic, World Medical Manufacturing Corp.) thoracic stent graft in one patient.

Emergent endovascular aortic repair was available at our institution only since January 2003 due to the need of an in-hospital stock of thoracic aortic endografts. Only three patients required open surgical repair since 2003 and those patients were not suitable candidates for an endovascular repair due to anatomic considerations.

Management of injuries to visceral arteries

The management of injuries to MAAVBs varied from conservative to endovascular or open surgical treatment. Conservative treatment included control of blood pressure and close clinical (urine output, creatine phosphokinase [CPK] levels, creatinine serum levels, metabolic acidosis) and imaging (focused assessment with sonography for trauma [FAST] and CT scan) surveillance. A conservative approach was selected for four cases. In one case, conservative management of a renal artery dissection was selected because the blood flow improved after TEVAR of an aortic transection originated at the isthmus level.

Early angiography and embolization or stenting was instituted at the discretion of the attending surgeon as a first-stage therapy of life-threatening bleeding, when uncontrolled bleeding persisted immediately postoperatively after an open repair, and as an adjunct to damage control. Endovascular management involved stenting of a LRA with a 7 × 18-mm RX Herculink Elite® stent (Abbott, Santa Clara, CA, USA) in one case and arterial embolization in three active bleeding cases. Proximal visceral artery embolization was performed with steel 0.035-in. Nester® coils (Cook Medical, Bloomington, IN, USA), whereas embolization of a distal visceral artery was accomplished with fibered platinum 0.018-in. microcoils, either VorteX™ microcoils (Boston Scientific, Natick, MA, USA) or Tornado® (Cook Medical). There was one unsuccessful attempt of celiac axis stenting, which was subsequently managed with a conservative approach thanks to the excellent collateral blood flow from the SMA.

Open surgical repair was required in three cases. Arteriorrhaphy with monofilament vascular suture was performed in one case of SMA injury and in one case of LRA injury. In the remaining case, a complete avulsion of RRA pedicle had occurred (type V renal OIS). Hence, ligation of the RRA was required followed by right nephrectomy.

Statistical analysis

Data are expressed as mean and standard deviation or median and range as appropriate. Proportions were compared with contingency tables by means of the χ^2 test with Yates' continuity correction or Fisher's exact test. The Mann–Whitney test was used to compare means. A p value of less than 0.05 was considered significant. Actuarial estimates of survival were accomplished with Kaplan–Meier methods. Differences in probability of survival between the groups were analyzed with the log rank (Mantel–Cox) test. The SPSS statistical program for Windows ver. 17.0 (SPSS, Chicago, IL, USA) was used to perform data analysis.

Results

Epidemiological and clinical characteristics of the ten major blunt trauma patients with associated ATAI and injury to MAAVBs (group A) are given in Table 1. Figures 1, 2, 3 show imaging tests in major trauma patients confirming the presence of ATAI with associated injury to MAAVBs.

Table 1. Epidemiological and clinical characteristics of major trauma patients with ATAI and associated major aortic abdominal visceral branch injury

Patient no.	Sex	Age (years)	Mechanism of trauma	Site of ATAI	Type of ATAI	Management of ATAI	Visceral arterial injury	Tyoe of MAAVBs	Management of visceral arterial injury	AAST-OIS
1	M	19	MCC	Mid-distal descending aorta	I	Conservative	LRA	Intimal tear	Conservative	III
2	M	32	Crush under weight	Abdominal aorta	II	Conservative	RRA	Thrombosis	Conservative	III (V considering abdominal aortic injury)
3	M	24	Crush under weight	Abdominal aorta	II	Conservative	LRA	Avulsion of renal vascular pedicle	Angioembolization with subsequent nephrectomy	III (V considering abdominal aortic injury)
4	M	72	MVC	Isthmus	III	TEVAR	LRA	Occlusion by aortic intimal flap	Endovascular	III
5	F	19	MVC	Mid-distal descending aorta	II	Conservative	SMA	Intimal tear	Open surgery	IV
6	F	60	MVC	Abdominal aorta	I	Conservative	Celiac axis	Dissection + distal thrombosis	Conservative	IV (V considering abdominal aortic injury)
7	M	34	MCC	Isthmus	IV	TEVAR	RRA	Occlusion by aortic intimal flap	Conservative	III
8	M	40	Crush under weight	Abdominal aorta	I	Conservative	LRA	Intimal tear with distal occlusion	Endovascular	III (V considering abdominal aortic injury)
9	M	44	MCC	Isthmus	IV	Conservative	RRA	Avulsion of renal vascular pedicle	Open surgery	III
10	F	60	AVP	Abdominal aorta	II	Conservative	LRA	Intimal tear	Open surgery	III (V considering abdominal aortic injury)

Patient no.	Hypotension (SBP < 90 mmHg)	ISS	TRISS	Peak CPK (IU/L)	Peak creatinine (mg/dL)	ARI	HDF	In-hospital death
1	Yes	42	14.3	5,040	0.90	No	No	Yes
2	Yes	35	8.5	3,541	1.8	Yes	No	No
3	Yes	45	32.9	45,557	4.7	Yes	Yes	Yes
4	Yes	66	99	67,161	3.0	Yes	Yes	Yes
5	Yes	66	98.7	3,244	0.90	No	No	No
6	Yes	66	94.2	2,979	0.91	No	No	No
7	Yes	50	15.3	5,784	1.2	No	No	No
8	Yes	42	14.3	1,165	1.5	No	No	No
9	Yes	59	77.1	93,000	5.4	Yes	Yes	Yes

M male, *F* female, *MCC* motorcycle crash, *MVC* motor vehicle crash, *AVP* auto versus pedestrian, *ATAI* acute traumatic aortic injury, *TEVAR* thoracic endovascular aortic repair, *MAAVBs* major aortic abdominal visceral branches, *LRA* left renal artery, *RRA* right renal artery, *SMA* superior mesenteric artery, *AAST-OIS* American Association for the Surgery of Trauma Organ Injury Scale, *ISS* Injury Severity Score, *TRISS* Trauma Injury Severity Score, *CPK* creatine phosphokinase, *ARI* acute renal injury, *HDF* hemodiafiltration

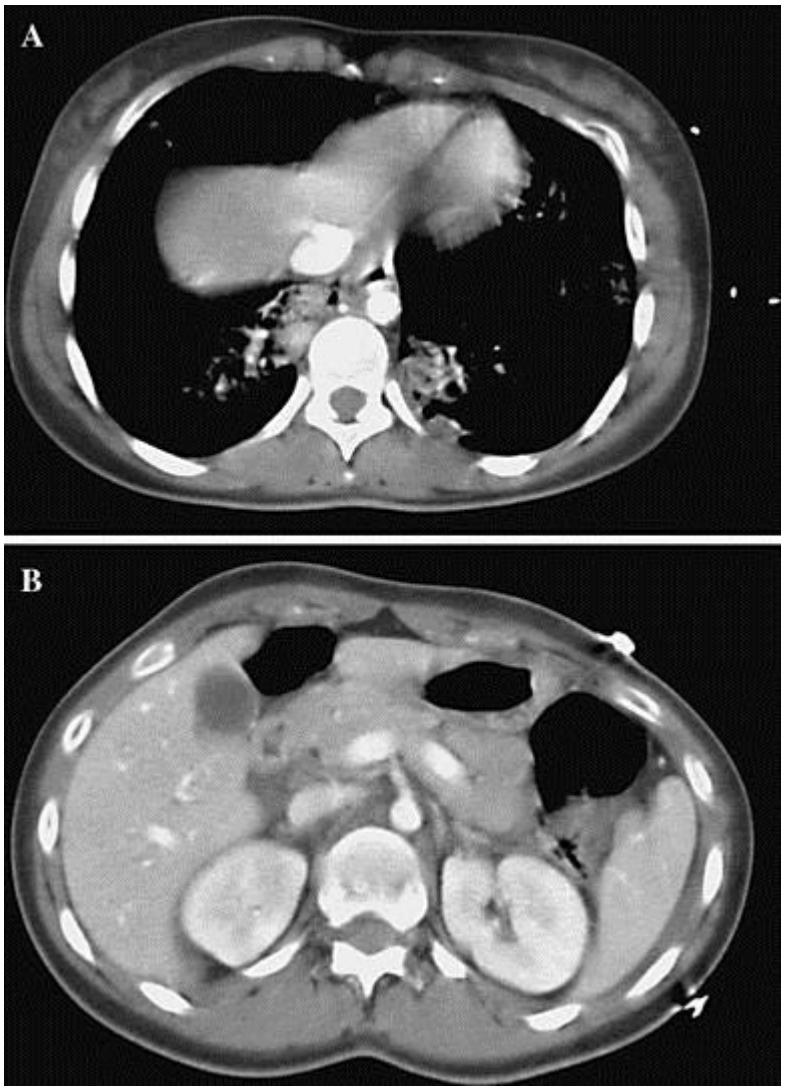

Fig. 1. On admission, multidetector CT (MDCT) images were taken of patient no. 5 of group A. **a** Thoracic MDCT axial slide shows a mid-descending aortic intramural hematoma. **b** Abdominal MDCT axial slide demonstrates a dissection of SMA, which underwent open surgical repair

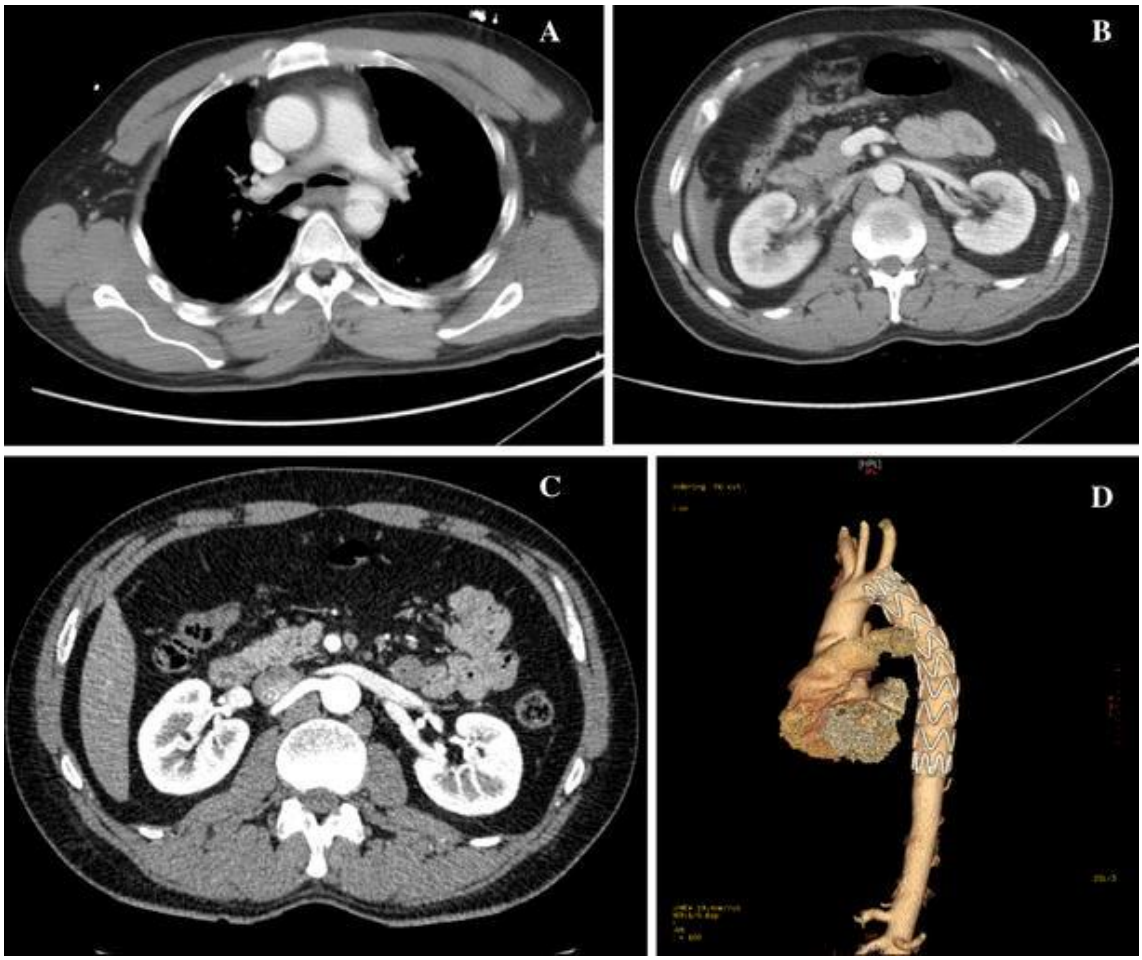

Fig. 2. On admission and 1 week later, control MDCTs were taken of patient no. 7 of group A. **a** Thoracic MDCT axial slide showing the aortic transection at the isthmus level. **b** Abdominal MDCT axial slide demonstrating a RRA dissection, which was conservatively managed because its blood flow improved after TEVAR. **c** One-week abdominal MDCT axial slide confirming improvement in RRA blood flow. **d** One-week three-dimensional thoracic MDCT reconstruction showing complete exclusion of the aortic injury after TEVAR

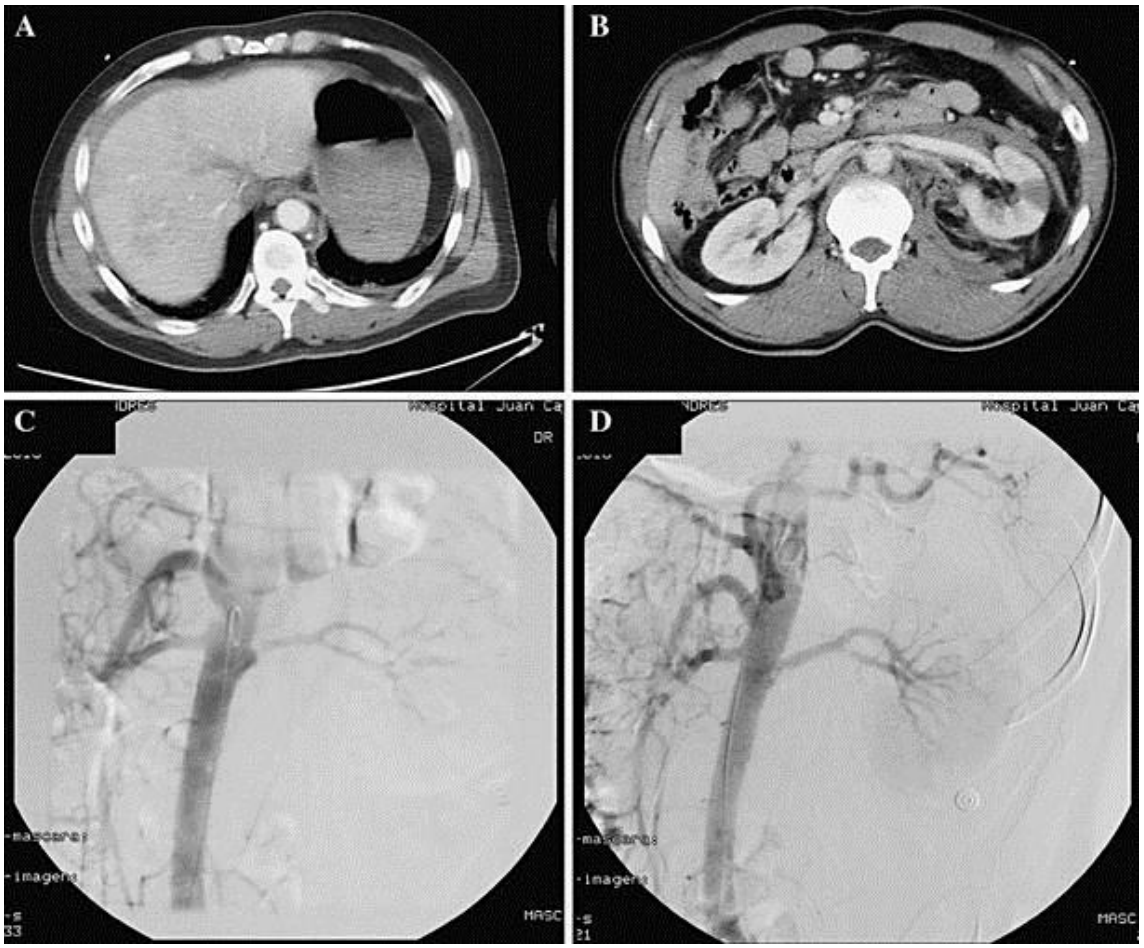

Fig. 3. On admission, MDCT and aortography images were taken of patient no. 8 of group A. **a** An intimal tear can be observed in the abdominal MDCT axial on admission. **b** Abdominal MDCT axial slide also shows a LRA perfusion defect due to an intimal flap. **c** Emergent abdominal aortography confirms the lack of perfusion in left kidney. **d** Control abdominal aortography after deployment of a 7-mm stent in the LRA. Notice the complete restoration of blood flow in the LRA

During the study period, 42 major blunt trauma patients with ATAI but without associated injury to MAAVBs were also admitted at our institution (group B). The differences in epidemiological and clinical characteristics between both groups of major trauma patients with ATAI are given in Table 2.

Table 2. Epidemiological and clinical characteristics in all patients and both trauma groups

	All patients (n = 52)	Group A (n = 10)	Group B (n = 42)	p Value
Male	43 (82.7%)	7 (70%)	36 (85.7%)	0.47
Age (mean ± SD)	45.1 ± 19.1	40.4 ± 18.5	46.2 ± 19.4	0.39
Age >55 years	19 (36.5%)	3 (30%)	16 (38.1%)	0.76
ISS (mean ± SD)	43.8 ± 15.7	51.9 ± 11.5	41.9 ± 16.1	0.036
RTS (mean ± SD)	5.5 ± 1.8	5.5 ± 1.9	5.5 ± 1.8	0.99
TRISS (mean ± SD)	50.3 ± 38	55.2 ± 41.1	49.1 ± 37.6	0.67
Head AIS >3	8 (15.4%)	2 (20%)	6 (14.3%)	0.97
Thorax AIS >3	35 (67.3%)	8 (80%)	37 (64.3%)	0.87
Extremity AIS >3	19 (36.5%)	5 (50%)	14 (33.3%)	0.53
Hypotension	38 (73.1%)	10 (100%)	28 (66.6%)	0.04
GCS < 9	22 (42.3%)	3 (30%)	19 (45.2%)	0.6
Need of ETI at site of accident or during transportation	38 (73.1%)	4 (40%)	34 (81%)	0.026

The p value of proportions analysis was obtained with the χ^2 test with Yates' correction or Fisher's exact test, while p values for means correspond to Mann–Whitney test
ISS Injury Severity Score, *AIS* Abbreviated Injury Score, *RTS* Revised Trauma Score, *TRISS* Trauma Injury Severity Score, *GCS* Glasgow Coma Scale, *ETI* endotracheal intubation

In patients with ATAI and associated MAAVBs (group A), the mechanism of trauma was MVC in 3 cases, MCC in 3, crush under weight in 3, and AVP in 1 case. In group B, the cause of trauma was MVC in 22 cases, MCC in 8, falls in 6, crush under weight in 3, and AVP in 3 cases (p = 0.21).

In group A, aortic injuries occurred at the abdominal aorta (5 cases, 50%), aortic isthmus (3 cases, 30%), and mid and distal descending aorta (2 cases, 20%). However, in group B, ATAI were located at isthmus level (21 cases, 50%) followed in frequency by mid and distal thoracic descending aorta (9 cases, 21.4%), aortic arch (6 cases, 14.2%), ascending aorta (3 cases, 7.2%), and abdominal aorta (3 cases, 7.2%) (p = 0.029). There were no differences in the types of aortic injury between both groups (Table 3).

Table 3. Types of ATAI in major blunt trauma patients with (group A) and without (group B) major aortic abdominal visceral branch injury

	All patients (n = 52)	Group A (n = 10)	Group B (n = 42)	χ^2
Type I	12 (23.1%)	3 (30%)	9 (21.4%)	
Type II	18 (34.6%)	4 (40%)	14 (33.3%)	
Type III	3 (5.8%)	1 (10%)	2 (4.8%)	0.5
Type IV	19 (36.5%)	2 (20%)	17 (40.5%)	

Overall expected mortality at admission was ≥50% according to an ISS score >50 in 19 patients (36.5%), whereas overall mean expected death rate calculated by TRISS score was 50.3 ± 38%. Although patients with combined aortic and MAAVB injuries presented a higher proportion of patients with an ISS >50, this difference did not reach statistical significance (50% vs. 33.3%, p = 0.53). Likewise, there were no statistically significant differences in the overall presence of severe (AIS > 3) associated nonvascular injuries and expected mortality calculated by ISS, RTS, and TRISS scores (Table 2).

Mean peak CPK was significantly higher in group A than in group B (23,008 ± 33,400 vs. 3,970 ± 3,495 IU/L; p < 0.001). Nine patients (90%) in group A had a peak CPK >2,000 IU/L, whereas 27 patients (64.3%) in group B presented a peak CPK >2,000 IU/L (p = 0.23).

ARI occurred in 5 patients of group A (50%), whereas 11 patients (26.2%) in group B developed it (p = 0.27). Three (30%) patients in group A and 4 patients (9.5%) in group B required hemodiafiltration (p = 0.23).

Overall in-hospital mortality was 32.7% (17 patients). In-hospital mortality in group A patients was 40% (4 patients), whereas in-hospital mortality was 31% (13 patients) in group B patients (p = 0.86). Observed in-hospital mortality was slightly lower than the expected in-hospital mortality estimated by TRISS in both groups (group A: observed 40% vs. estimated 55.2%, p = 0.65; group B: observed 30.9% vs. estimated 41.1%, p = 0.12).

Causes of death in group A were multisystem organ failure in two patients, acute respiratory distress syndrome in one, and hypovolemic shock in one patient. Causes of death in group B were hypovolemic shock in six patients, multisystem organ failure in three, acute respiratory distress syndrome in one, brain herniation in two, and septic shock in one case.

The management of ATAI in group A was conservative in eight cases and an emergent TEVAR in two patients. On the other hand, management of ATAI in group B was conservative in 28 cases (66.7%), emergent open repair in 7 cases (16.7%), delayed open repair in 2 cases (4.8%), and emergent TEVAR in 6 cases (14.3%).

There were no differences in ICU and in-hospital stay between the groups. The overall median ICU stay was 18 days (range = 0–105 days), the median ICU stay for group A was 12 days (range = 2–44 days), and the median ICU stay for group B was 18 days (range = 0–105 days) ($p = 0.53$). The overall median in-hospital stay was 23 days (range = 0–547 days), the median in-hospital stay for group A was 45 days (range = 2–113 days), and the median in-hospital stay for group B was 31 days (range = 0–547 days) ($p = 0.54$).

After hospital discharge, clinical and imaging follow-up was available for all patients at a median time of 64 months (range = 1–130 months). In group A, four patients had a more than 3-year follow-up.

Overall survival estimated by the Kaplan–Meier method, including early mortality, was 67.2% at 1 year and 62.2% at 5 and 10 years. Group A survival was 60% at 1, 5, and 10 years. There was no late mortality in group A patients beyond the first year after hospital discharge. Group B survival was 69% at 1 year and 63.3% at 5 and 10 years (Fig. 4). There were no statistically significant differences between group survivals ($p = 0.15$).

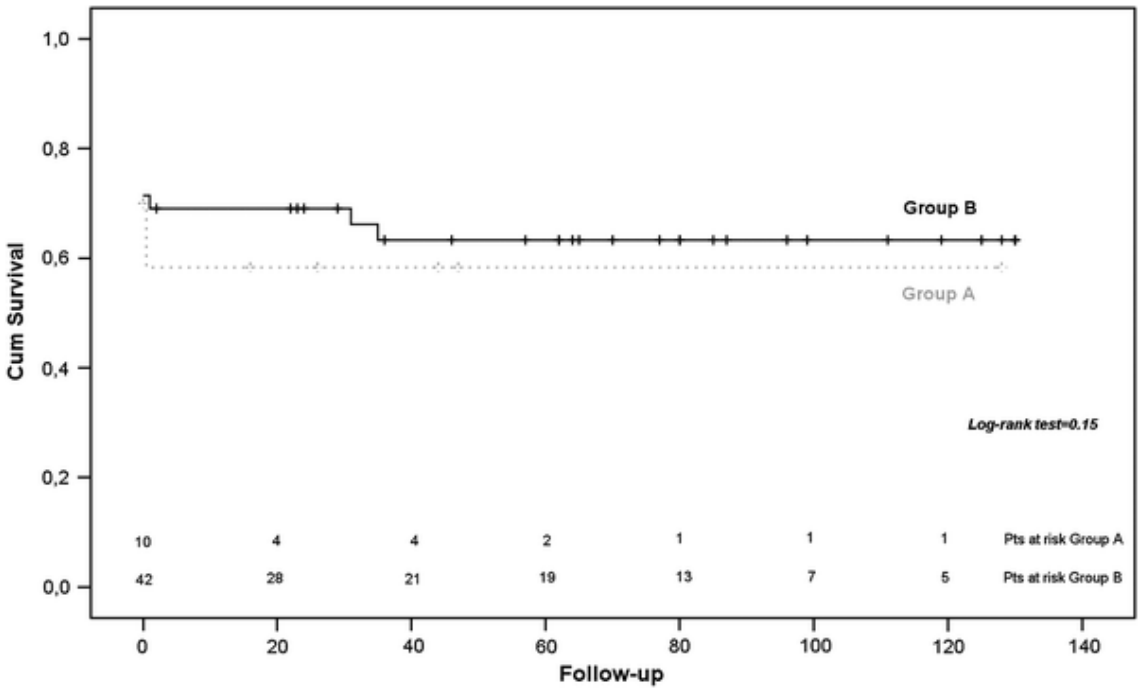

Fig. 4. Kaplan–Meier survival curves of ATAI patients with associated injury to major aortic abdominal visceral branch (MAAVB) (group A) and of ATAI patients without associated MAAVB (group B), including in-hospital mortality

Discussion

Our results suggest that ATAIs associated with MAAVB injuries in major blunt trauma patients affect the thoracic aorta more frequently than reported by other series, present a higher in-hospital mortality, and entail a different management than ATAI without MAAVBs.

Our experience is consistent with the literature to date, yet provides some new insights about major trauma patients with combined ATAI and MAAVBs. The association between aortic wall and visceral artery injuries typically appears in penetrating abdominal trauma and the affected segment of aorta is usually the abdominal aorta. However, this combination of vascular injuries is seldom reported in blunt

thoracoabdominal trauma. Asensio et al. [*24*] presented the largest published series of abdominal vascular injuries, including both penetrating and blunt trauma, with a total of 302 patients. In that series there were only 17 cases of abdominal aortic injury associated with other abdominal vascular injuries, most of them occurring in penetrating traumas. In-hospital mortality in those 17 patients reached 82.4% [*24*]. More recently, Teixeira et al. [*25*] reported an autopsy series of 102 major trauma victims with ATAI, among which there were only 4 patients (3.9%) with associated visceral vessel injuries. In our series, 19.2% of the 52 major blunt trauma patients with ATAI admitted to our institution in the last decade sustained an associated injury to MAAVBs. Moreover, it must be highlighted that in half of these patients with associated MAAVBs injury, the ATAI was located in the thoracic aorta, either isthmal or mid-descending thoracic aorta, differing remarkably from other series.

Major blunt trauma patients with associated ATAI and MAAVBs pose a great surgical challenge. The majority of these patients are in shock, physiologically compromised, have sustained multiple associated nonvascular injuries and significant blood losses, and their mortality rate remains extremely high. Few series have specifically focused on the management of both concomitant types of injury. In our series, all patients with ATAI and MAAVB injuries had, at the least, a severe nonvascular associated injury (AIS > 3). Additionally, major trauma patients with associated ATAI and MAAVB injuries had hemodynamic instability more frequently than patients without the combined vascular injuries. In fact, hypotension was significantly more frequent in patients with aortic and MAAVB injuries (100% vs. 66.6%, $p = 0.04$).

Major blunt trauma patients with concomitant aortic and MAAVB injuries also have a higher rate of morbidity and entail a more hazardous management at the ICU. We have found a significantly higher mean peak CPK in the group with combined ATAI and MAAVB injuries than in the group without associated MAAVB injury ($23{,}008 \pm 33{,}400$ vs. $3{,}970 \pm 3{,}495$ IU/L; $p < 0.001$). This finding may be explained by an increased tissue necrosis secondary to malperfusion syndrome and rhabdomyolysis in this critical subset of trauma patients. Furthermore, ATAI patients with associated MAAVB injuries had a higher rate of ARI (50% vs. 26.2%, $p = 0.27$), requiring hemodiafiltration more frequently.

In-hospital mortality in major trauma patients with associated ATAI and MAAVB injuries was slightly higher than in major trauma patients with ATAI without concomitant injuries to MAAVBs (40% vs. 31%). Nevertheless, in both groups of major trauma patients, observed in-hospital mortality tended to be lower than the expected in-hospital mortality estimated by TRISS.

These satisfactory outcomes in patients with ATAI, especially in the most critical subset of major trauma patients, are a result of a multidisciplinary approach that combines endovascular and open surgical techniques for a staged treatment of life-threatening aortic and visceral vascular injuries. The multidisciplinary approach for the management of the latter includes early embolization or stenting, early surgical intervention to control hemorrhage, damage control, utilization of early packing, and staged surgical procedures. Several series have reported the utilization of stenting and angioembolization as an adjunct to nonoperative management of blunt vascular visceral injuries and/or as a means of avoiding surgical intervention [*9–11*]. In some cases, we advocate for early endovascular management, either stenting or angioembolization of the visceral vascular injury, followed by an open surgical approach. According to our experience, nephrectomy may be required after renal angioembolization in renal Organ Injury Scale (OIS) grade IV–V cases as previously reported [*26*].

On the other hand, the introduction of TEVAR has revolutionized the definitive management of ATAI. TEVAR for ATAI was used initially in high-risk multiple injuries or elderly patients, but in many centers it has now become the initial procedure of choice, even in young or low-risk patients. TEVAR appears to lower mortality and morbidity rates in patients with ATAI [*8, 27, 28*]. Moreover, TEVAR has extended operative treatment to those patients not previously considered candidates for repair [*29*].

Nowadays, the Expert Opinion Committee of the Society of Thoracic Surgeons [*7*], the Clinical Practice Guidelines of the Society for Vascular Surgery [*16*], the American Association of Thoracic Surgeons [*8*], and the ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM Guidelines for the Diagnosis and Management of Patients with Thoracic Aortic Disease [*20*] suggest that ATAIs may be considered for treatment with endografts. Nevertheless, some cautionary measures must be taken due to the possibility of device- and procedure-related complications, especially in young patients, and the lack of information on the long-term durability of endografts [*7, 8, 16, 20*]. It must be also highlighted that in the last decade the management of ATAIs has improved thanks not only to the shift toward the use of endovascular repair techniques, but also to the advent of MDCT [*30*], the clinical management with aggressive blood pressure control and cardiac contractility [*20–22*], and the institution of delayed surgical treatment after the associated critical injuries have been stabilized [*31*].

As we have previously reported [*19*], in cases of multiple associated injuries, conservative management of the ATAI may be selected, especially in low-risk aortic injuries (small intimal tears or intramural hematoma without intimal tear; types I and II). However, up to 40% of ATAIs in which a

conservative approach is selected may develop a life-threatening aortic-related complication [*19*]. The initial type of aortic lesion (HR: 2.94, $p = 0.002$) and a TRISS score >50% (HR: 1.49, $p = 0.042$) on admission have been identified as risk factors for a worse long-term prognosis [*19*].

In spite of the small or nonexistent number of major trauma patients with associated ATAI and MAAVBs that arrive alive at the hospital, the number of patients in this series is small and the present study may lack sufficient statistical power to determine with confidence some clinically relevant differences. The management of these patients has been modified with the inclusion of technological advances in both diagnostic and therapeutic fields, especially with the spread of endovascular techniques. This study also has the limitations inherent in any retrospective series.

Conclusions

Associated aortic and MAAVB injuries in major trauma patients seem to present in a different clinical scenario. These patients present increased risk of rhabdomyolysis, visceral ischemia, and acute renal failure, as well as higher in-hospital mortality. A multidisciplinary approach combining endovascular and open surgical techniques for staged treatment of these life-threatening aortic and visceral vascular injuries is mandatory in this critical subset of trauma patients.

Conflict of Interest

The authors state that there are no potential conflicts of interest to be considered in this article.

References

1. Asensio JA, Forno W, Roldan G, Petrone P, Rojo E, Tillou A (2001) Abdominal vascular injuries: injuries to the aorta. Surg Clin N Am 81(6):1395–1416, xiii–xiv.
2. Asensio JA, Forno W, Roldan G, Petrone P, Rojo E, Ceballos J (2002) Visceral vascular injuries. Surg Clin N Am 82(1):1–20, xix.
3. Asensio JA, McDuffie L, Petrone P, Roldan G, Forno W, Gambaro E (2001) Reliable variables in the exsanguinated patient which indicate damage control and predict outcome. Am J Surg 182(6):743–751.
4. Fialka C, Sebok C, Kemetzhofer P, Kwasny O, Sterz F, Vecsei V (2004) Open-chest cardiopulmonary resuscitation after cardiac arrest in cases of blunt chest or abdominal trauma: a consecutive series of 38 cases. J Trauma 57(4):809–814.
5. Asensio JA, Soto SN, Forno W, Roldan G, Petrone P, Gambaro E (2001) Abdominal vascular injuries: the trauma surgeon's challenge. Surg Today 31(11):949–957.
6. Cox EF (1984) Blunt abdominal trauma. A 5-year analysis of 870 patients requiring celiotomy. Ann Surg 199(4):467–474.
7. Svensson LG, Kouchoukos NT, Miller DC, Bavaria JE, Coselli JS, Curi MA (2008) Expert consensus document on the treatment of descending thoracic aortic disease using endovascular stent-grafts. Ann Thorac Surg 85(1 Suppl):S1–S41.
8. Demetriades D, Velmahos GC, Scalea TM, Jurkovich GJ, Karmy-Jones R, Teixeira PG (2008) Operative repair or endovascular stent graft in blunt traumatic thoracic aortic injuries: results of an American Association for the Surgery of Trauma Multicenter Study. J Trauma 64(3):561–570 (discussion 570–571).
9. Hagiwara A, Murata A, Matsuda T, Matsuda H, Shimazaki S (2002) The efficacy and limitations of transarterial embolization for severe hepatic injury. J Trauma 52(6):1091–1096.
10. Chimpiri AR, Natarajan B (2009) Visceral arteriography in trauma. Semin Interv Radiol 26(3):207–214.
11. Sabe AA, Claridge JA, Rosenblum DI, Lie K, Malangoni MA (2009) The effects of splenic artery embolization on nonoperative management of blunt splenic injury: a 16-year experience. J Trauma 67(3):565–572 (discussion 571–572).
12. Baker SP, O'Neill B, Haddon W Jr, Long WB (1974) The injury severity score: a method for describing patients with multiple injuries and evaluating emergency care. J Trauma 14(3):187–196.
13. Champion HR, Sacco WJ, Copes WS, Gann DS, Gennarelli TA, Flanagan ME (1989) A revision of the Trauma Score. J Trauma 29(5):623–629.
14. Champion HR, Sacco WJ, Copes WS (1995) Injury severity scoring again. J Trauma 38(1):94–95.
15. Soreide K (2009) Epidemiology of major trauma. Br J Surg 96(7):697–698.
16. Lee WA, Matsumura JS, Mitchell RS, Farber MA, Greenberg RK, Azizzadeh A (2011) Endovascular repair of traumatic thoracic aortic injury: clinical practice guidelines of the Society for Vascular Surgery. J Vasc Surg 53(1):187–192.
17. Moore EE, Cogbill TH, Jurkovich GJ, McAninch JW, Champion HR, Gennarelli TA et al (1992) Organ injury scaling. III: chest wall, abdominal vascular, ureter, bladder, and urethra. J Trauma 33(3):337–339.
18. Bellomo R, Kellum J, Ronco C (2001) Acute renal failure: time for consensus. Intensive Care Med 27(11):1658–1685.

19. Mosquera VX, Marini M, Lopez-Perez JM, Muniz-Garcia J, Herrera JM, Cao I (2011) Role of conservative management in traumatic aortic injury: comparison of long-term results of conservative, surgical, and endovascular treatment. J Thorac Cardiovasc Surg 142(3):614–621.
20. Hiratzka LF, Bakris GL, Beckman JA, Bersin RM, Carr VF, Casey D Jr (2010) ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM Guidelines for the Diagnosis and Management of Patients with Thoracic Aortic Disease. J Am Coll Cardiol 55(14):e27–e129.
21. Pate JW, Fabian TC, Walker W (1995) Traumatic rupture of the aortic isthmus: an emergency? World J Surg 19(1):119–125, discussion 125–126.
22. Fabian TC, Davis KA, Gavant ML, Croce MA, Melton SM, Patton JH Jr (1998) Prospective study of blunt aortic injury: helical CT is diagnostic and antihypertensive therapy reduces rupture. Ann Surg 227(5):666–676 (discussion 676–677).
23. Mosquera VX, Herrera JM, Marini M, Estevez F, Cao I, Gulias D et al (2009) Mid-term results of thoracic endovascular aortic repair in surgical high-risk patients. Interact Cardiovasc Thorac Surg 9(1):61–65.
24. Asensio JA, Chahwan S, Hanpeter D, Demetriades D, Forno W, Gambaro E (2000) Operative management and outcome of 302 abdominal vascular injuries. Am J Surg 180(6):528–533
25. Teixeira PG, Inaba K, Barmparas G, Georgiou C, Toms C, Noguchi TT (2011) Blunt thoracic aortic injuries: an autopsy study. J Trauma 70(1):197–202.
26. Davis KA, Reed RL II, Santaniello J, Abodeely A, Esposito TJ, Poulakidas SJ (2006) Predictors of the need for nephrectomy after renal trauma. J Trauma 60(1):164–169 (discussion 169–170)
27. Akowuah E, Angelini G, Bryan AJ (2009) Open versus endovascular repair of traumatic aortic rupture: a systematic review. J Thorac Cardiovasc Surg 138(3):768–769.
28. Jonker FH, Giacovelli JK, Muhs BE, Sosa JA, Indes JE (2010) Trends and outcomes of endovascular and open treatment for traumatic thoracic aortic injury. J Vasc Surg 51(3):565–571.
29. Hong MS, Feezor RJ, Lee WA, Nelson PR (2011) The advent of thoracic endovascular aortic repair is associated with broadened treatment eligibility and decreased overall mortality in traumatic thoracic aortic injury. J Vasc Surg 53(1):36–42 (discussion 43).
30. Steenburg SD, Ravenel JG (2008) Acute traumatic thoracic aortic injuries: experience with 64-MDCT. Am J Roentgenol 191(5):1564–1569.
31. Demetriades D, Velmahos GC, Scalea TM, Jurkovich GJ, Karmy-Jones R, Teixeira PG, Hemmila MR, O'Connor JV, McKenney MO, Moore FO, London J, Singh MJ, Spaniolas K, Keel M, Sugrue M, Wahl WL, Hill J, Wall MJ, Moore EE, Lineen E, Margulies D, Malka V, Chan LS (2009) Blunt traumatic thoracic aortic injuries: early or delayed repair—results of an American Association for the Surgery of Trauma prospective study. J Trauma 66:967–973