Injury, Int. J. Care Injured 44 (2013) 60–65

Contents lists available at SciVerse ScienceDirect

Injury

journal homepage: www.elsevier.com/locate/injury

Aortic injuries in crush trauma patients: Different mechanism, different management

Victor X. Mosquera [a,*], Milagros Marini [b], Javier Muñiz [c], José M. Lopez-Perez [d], Daniel Gulías [b], José J. Cuenca [a]

[a] *Department of Cardiac Surgery, Complejo Hospitalario Universitario de A Coruña, Spain*
[b] *Department of Interventional Radiology, Complejo Hospitalario Universitario de A Coruña, Spain*
[c] *Instituto Universitario de Ciencias de la Salud, Universidad de A Coruña, Spain*
[d] *Intensive Care Unit, Complejo Hospitalario Universitario de A Coruña, Spain*

ARTICLE INFO

Article history:
Accepted 22 September 2011

Keywords:
Emergency medicine
Aorta
Endovascular
Surgery

ABSTRACT

Background: The objective of this study is to report the clinical and radiological characteristics and early and long-term survival of a series of acute traumatic aortic injuries (ATAI) in crush trauma patients, and to compare such data with our last 30 years experience managing ATAI in deceleration non-crush trauma patients.
Methods: From January 1980 to December 2010, 5 consecutive ATAI in crush trauma and 69 in non-crush trauma patients were admitted at our institution. ISS, RTS and TRISS scores were similar in both groups.
Results: Overall in-hospital mortality was 24.3%. There was no in-hospital mortality in crush patients and 26.1% in non-crush patients ($p = 0.32$).

All aortic-related complications occurred in non-crush patients. Median follow-up was 129 months (range 3–350 months). Non-crush group survival was 76.8% at 1 year, 73.6% at 5 years, and 71.2%% at 10 years. There was no mortality during follow-up in the crush group. Mean (SD) peak creatine phosphokinase was significantly higher in crush group than in non-crush group: 7598 (3690) IU/L vs. 3645 (2506) IU/L; $p = 0.041$. Incidence of acute renal injury was higher in crush trauma patients (100% vs. 36.2%; $p = 0.018$). Low-severity injuries were more common in crush trauma patients (100% in crush patients vs. 43.5% in non-crush patients, $p = 0.04$).
Conclusions: Aortic injuries in crush thoracic trauma patients seem to present in a different clinical scenario from aortic injuries in high-speed thoracic trauma thus requiring distinct considerations. When planning the initial management of aortic injuries in crush trauma, the increased risk of rhabdomiolysis and subsequent acute renal failure, as well as a tendency to develop lower-risk aortic wall injuries, must be considered.

Blunt rupture of the thoracic aorta may occur in patients with multisystem trauma and has devastating consequences.[1] The most common mechanism of acute traumatic aortic injury (ATAI) is the shearing stress to the aorta during rapid deceleration and appears in motor vehicle injuries and falls.[2] However, there are other mechanisms such as compression of the aorta between sternum and thoracic spine (osseous pinch), and direct load causing aortic wall strain and medial tears.[2–4] The latter would explain how the aorta is injured in thoracic trauma due to crush.

Clinical and radiological characteristics and both in-hospital and long-term survival of ATAI in crush trauma is poorly documented. All the studies published to date are only focused on aortic injuries in deceleration non-crush trauma patients, there being a lack of information about ATAI in crush trauma patients. In fact, the different mechanism of ATAI and the special clinical features of patients with ATAI and crush syndrome may require a management distinct from the one in deceleration trauma ATAI.

The objective of this study is to report the clinical and radiological characteristics and early and long-term survival of a series of 5 consecutive ATAI in crush trauma patients, and to compare such data with our last 30 years experience managing ATAI in trauma patients.

Patients and methods

From January 1980 to December 2010, 80 patients were admitted to our institution with ATAI. Six patients were excluded from the analysis because of deficient documentation of the time

* Corresponding author at: Department of Cardiac Surgery, Complejo Hospitalario Universitario de A Coruña, As Xubias n°84, CP 15006, A Coruña, Spain. Tel.: +34 981178000x298814; mobile: +34 699054009; fax: +34 981178299.
E-mail address: vxmr@yahoo.es (V.X. Mosquera).

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doi:10.1016/j.injury.2011.09.022

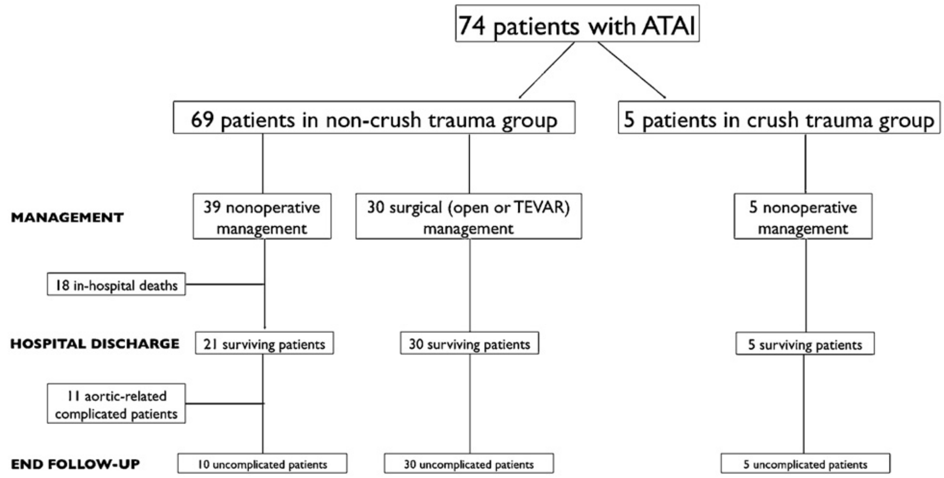

Fig. 1. Flow chart depicts the patient distribution at 3 different stages (management; hospital discharge; follow-up). ATAI: acute traumatic aortic injury. TEVAR: thoracic endovascular aortic repair.

from injury to procedure and/or in extremis status on arrival. Five patients suffered ATAI in a crush trauma, whilst the remaining 69 patients presented a more common deceleration non-crush trauma. Flow diagram in Fig. 1 depicts the flow of patients.

Data collection included age, gender, mechanism of injury, initial clinical presentation (blood pressure, Glasgow coma scale [GCS]), injury severity score (ISS),[5] abbreviated injury score (AIS) for each body area (head, chest, abdomen, and extremities), revised trauma score (RTS),[6] trauma injury severity score (TRISS),[7] method of diagnosis (computed tomography scan, angiography, and transesophageal echocardiogram), and peak serum creatine phosphokinase (CPK).

Aortic injuries were classified according to the classification proposed by Azizzadeh et al. in 2009 in type I (intimal tear), type II (intramural haematoma), type III (pseudoaneurysm), or type IV (rupture).[8,9] Types I and II were considered low-risk injuries, and types III and IV high-risk injuries.

Aortic injuries were also classified according to the site of aortic injury (aortic root-ascending aorta; aortic arch; aortic isthmus; mid and/or distal descending thoracic aorta), and the type of definitive management (conservative, open repair or endovascular repair).

An ISS score of more than 50 points predicts a mortality rate of over 50%, whilst a score of more than 70 points predicts a mortality rate of nearly 100%.[5] The TRISS score directly predicts the expected death rate for blunt trauma.[7]

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.[10] Acute renal failure (ARF) was defined using published criteria as changes in serum creatinine (increase in serum creatinine by threefold; 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.[10]

Patient management was conservative treatment, aortic endografting or open surgical repair according to the clinical and radiological criterion of the trauma team involved. 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.[11]

Institutional Review Board approval was obtained.

Statistical analysis

Data are expressed as mean and standard deviation or median and range, when appropriate. Proportions were compared with contingency tables by means of Chi-square with Yates' correction or Fisher's exact tests when appropriate, whilst the Student's *t*-test or Wilcoxon rank sum test were used to continuous variables. 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 analysed with the Log Rank (Mantel–Cox) test.

The SPSS statistical program (except 'program' in computers) for Windows version 17.0 (SPSS, Chicago, IL) was used to perform data analysis.

Results

Epidemiologic and clinical characteristics of the 5 crush trauma patients are described in Table 1. Fig. 2 shows imaging tests in crush trauma patients confirming the presence of ATAI. During the study period, 69 non-crush trauma patients with ATAI were also admitted to our institution. Differences in epidemiologic and clinical characteristics between crush trauma and non-crush trauma patients are shown in Table 2.

Overall expected mortality at admission was ≥50% according to an ISS score >50 points in 22.9% of patients, whilst overall mean (SD) expected death rate calculated by TRISS score was 34.96% (35.76%).

All thoracic or thoracoabdominal crush trauma were the result of occupational accidents and were caused by compression by heavy machinery, blocks of stones, trunks, etc. In the non-crush group, the cause of the trauma was a car accident in 44 cases, a motorbike accident in 11, falls in 8 and a pedestrian–vehicle accident in 6 cases.

There were no statistically significant differences in the overall presence of severe extrathoracic injuries and expected mortality calculated by ISS, RTS and TRISS scores (Table 2). However, no patient in the crush group presented severe head and neck injury (head & neck AIS > 3) vs. 10 patients (14.5%) in the non-crush group.

Mean (SD) peak CPK was significantly higher in the crush group than in the non-crush group (7598 (3690) IU/L vs. 3645 (2506) IU/L; $p = 0.041$). All patients in the crush group had a peak CPK > 2000 IU/