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Hemothorax Management After Endovascular Treatment For Thoracic Aortic Rupture

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WHAT THIS PAPER ADDS

This study has focused on the characteristics and management of hemothorax, occurring in a large cohort of patients with thoracic aortic rupture, treated with TEVAR. It may contribute further insights into the management of ruptured descending thoracic aorta and into optimizing the post-operative risk stratification after urgent TEVAR. Furthermore, it is hoped it will lead to larger studies to optimize current practice on the best management of hemothorax after TEVAR.

Objectives: The aim was to describe and analyze the management of hemothorax (HTX) and the occurrence of respiratory complications after endovascular repair of thoracic aortic rupture (TEVAR).

Methods: This was a multicenter study with retrospective analysis. Between November 2000 and December 2012, all patients with confirmed HTX due to rupture of the descending thoracic aorta treated with TEVAR were included. Respiratory function (acid base status, Pao_2, $Paco_2$, lactate, and respiratory index) was monitored throughout hospitalization. Primary endpoints were survival and post-operative respiratory complications.

Results: Fifty-six patients were treated. The mean age was 62 ± 21 years (range 18–92 years). Etiology included traumatic rupture ($n = 23$, 41%), atherosclerotic aneurysm ($n = 20$, 36%), Debakey type IIIa dissection ($n = 8$, 14%), and penetrating aortic ulcer ($n = 5$, 9%). The primary technical success of TEVAR was 100%. The in hospital mortality rate was 12.5% ($n = 7$). Hemothorax was drained in 21 (37.5%) cases. In hospital respiratory complications occurred in 23 (41%) patients who required a longer intensive care unit stay (days 2.3 ± 0.7 vs. 1.9 ± 0.8, $p = .017$), and hospitalization (26 ± 17 *vs*. 19 ± 17, $p = .021$). Those who developed post-operative respiratory complications had lower pre-operative PO_2 values (mmHg, 80 ± 24 vs. 91 ± 21, $p = .012$). Respiratory complications and in hospital mortality did not differ among aortic pathologies ($p = .269$ and $p = 1.0$, respectively), nor did in hospital mortality differ between patients with and without respiratory complications (13% vs. 12%; $p = .990$).

Conclusions: Thoracic aortic rupture still has a high mortality rate. Respiratory complications have not been eliminated by endovascular repair. HTX evacuation may have had a positive influence on the survival in these patients. Although traumatic and degenerative ruptures are two significantly different scenarios, survival and respiratory outcomes were similar and were not affected by the underlying aortic disease.

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INTRODUCTION

Rupture of the descending thoracic aorta has traditionally been treated by open graft replacement, with its associated mortality and complication rates which remain disturbingly high.[1–3] More recently, thoracic endovascular aortic repair (TEVAR) has proved to be an effective, less traumatic, alternative for the treatment of all thoracic aortic pathologies; indeed several papers have advocated TEVAR as the first choice for the emergency treatment of thoracic aortic catastrophes.[4–7] Rupture of the thoracic aorta is frequently associated with hemothorax (HTX); however, the

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management of HTX after TEVAR has not been debated until now, and there is still uncertainty on indication and timing of its treatment.[8–14] Hemothorax may lead to major complications such as compression of the esophagus and/or cardiovascular structures, respiratory insufficiency, and infection. All these complications may compromise the post-operative survival in an already critically ill subset of patients.[8] The purpose of this study was to analyze the management of HTX in patients with descending thoracic aortic rupture treated with TEVAR.

MATERIALS AND METHODS

Study population

The study was designed as a multicenter retrospective study. Between November 2000 and December 2012, all patients presenting with rupture of the descending thoracic aorta and HTX treated with TEVAR were identified and included in the analysis. Thoraco-abdominal lesions were excluded. Clinical and procedural data were collected prospectively and recorded in a dedicated database. All patients underwent pre-operative thoraco-abdominal computed tomography (CT) scans. Intervention was performed in the operating theatre, equipped to perform either open surgical or endovascular procedures. General anesthesia and antibiotic prophylaxis with a second generation cephalosporin was used in all patients. Four different thoracic endografts (EGs) were implanted: Excluder/TAG/C-TAG (W.L. Gore and Associates, Flagstaff, AZ, USA), Talent/Valiant/Captivia (Medtronic Vascular, Santa Rosa, CA, USA), TX-1/TX-2 (Cook, Bloomington, IN, USA), and Relay (Bolton Medical, Sunrise, FL, USA). Post-operatively, the patient was transferred to the intensive care unit (ICU). In all patients, respiratory function was monitored at admission and during hospitalization: acid base status, Pao_2, $Paco_2$, lactate, and the respiratory index (Pao_2/Fio_2) were evaluated.[15] Evacuation of the HTX was always discussed case by case by the operating team and the intensive care unit (ICU) physicians; generally, HTX was drained when the respiratory index (RI) was < 200 or when signs of respiratory or cardiovascular impairment were detected. The hemothorax was drained with a chest tube; video assisted thoracoscopy or open thoracotomy which was used selectively for complicated trapped HTX. Clinical and CT scan follow up were performed at 1, 6, and 12 months after the intervention, and annually thereafter.

Definition

Thoracic aortic rupture was defined as hemorrhage outside the boundaries of the aorta. Hemothorax was considered as any collection of blood in the pleural cavity, but isolated peri-aortic/mediastinal hematoma suggestive for a contained rupture was not counted as HTX. Comorbidities were defined according to the Society of Thoracic Surgeons Adult Database definitions.[16] On admission, hemorrhagic shock was defined as a combination of systolic blood pressure $<$ 80 mmHg after fluid resuscitation or need for α-amines, tachycardia ($>$ 110 bpm), anuria or urine output $<$ 15 mL/hour, unconsciousness, or circulatory arrest.[17] The HTX at its point of greater thickness in the baseline CT was classified: $<$ 2 cm, 2–3 cm, $>$ 3 cm. Delay was defined as the time interval between the diagnosis of aortic rupture/onset of symptoms and the start of the intervention. Traumatic aortic injury severity grading was classified accordingly to the clinical practice guidelines of the Society for Vascular Surgery (SVS).[18] The operative risk profile was estimated according to the EuroSCORE.[19] Operative outcomes were classified following the ad hoc committee on TEVAR reporting standards of the Society for Vascular Surgery/American Association for Vascular Surgery (SVS/AAVS).[20] Specifically, respiratory complications were defined as grade 1 if recovery was prompt with medical treatment, grade 2 for prolonged hospitalization or intravenous antibiotics, and grade 3 for prolonged intubation, tracheotomy, deterioration in pulmonary function, O_2 dependence, or fatal outcome. Primary endpoints were the evaluation of survival and respiratory complications.

Data analysis

Clinical data were recorded in Microsoft Excel (Microsoft Corp, Redmond, WA, USA). Results are presented as mean $\pm$ SD for continuous variables, and number (percentage) for categorical variables. Continuous variables were compared with the Mann–Whitney U test, and categorical variables by the Fisher exact test. The survival rate was estimated by means of the Kaplan–Meier method with the log-rank test. A p value $<$ .05 was considered significant. Statistical analysis was computed with SPSS, release 20.0, for Windows (IBM SPSS Inc., Chicago, IL, USA).

RESULTS

General data

During the study period a total of 500 TEVARs for descending thoracic aortic diseases were performed: 402 (81%) intact aortic lesions were treated; 42 (8%) were ruptured or traumatic lesions without HTX. Rupture with HTX was present in 56 (11%) cases: there were 40 (71%) males. Mean age was 62 $\pm$ 21 years (range 18–92 years). Traumatic ruptures ($n = 23$, 41%) were caused by blunt injuries in all cases; a grade IV injury was present in all cases. In particular, traumatic rupture involved the distal aortic arch below the left subclavian artery ($n = 9$, 39%) and the descending aorta ($n = 14$, 61%). Non-traumatic ruptures were caused by atherosclerotic aneurysm ($n = 20$, 36%), Debakey type IIIa dissection ($n = 8$, 14%), and penetrating aortic ulcer ($n = 5$, 9%). Overall demographic data, co-morbidities and risk factors are reported in Table 1.

Operative data

Emergent TEVAR was performed in 19 (34%) cases: all but three (5%) patients were treated within 3 hours of rupture with a median delay of 1.5 hours (range 0.5–24). The proximal landing zone was “zone 2” in 18 (32%) cases and “zones 3 or 4” in 38 (68%). Primary technical and primary

Table 1. Demographic and presentation data.

Variable[a]	All patients
Demographic data	
M : F	40 : 16
Age (mean, years)	62 ± 21
Risk factors (%)	
CAD	16 (29)
COPD	16 (27)
Obesity (BMI > 30)	15 (27)
Diabetes	13 (23)
AKI	8 (14)
Valve disease/LHF	7 (13)
Operative risk	
Shock (%)	19 (34)
EuroSCORE (mean, predicted mortality)	9 ± 4 (35 ± 23)

AKI = acute kidney injury; BMI = body mass index; CAD = coronary artery disease; COPD = chronic obstructive pulmonary disease; LHF = left heart failure.

[a] For numerical variables, mean ± SD; for categorical variables, number and percentages.

clinical success was obtained in all cases: open conversion was not required. Mean operation time was 107 ± 63 minutes (range 30—300; median 90 minutes) and the mean aortic coverage was 200 ± 9 mm (range 10—38; median 200 mm). Median blood loss was 200 mL (range 50—2,600 mL) and mean blood transfusion was 4 ± 4 units of packed red blood cells (range 0—27; median, 4). ICU stay was < 2 days in 13 (23%) cases, between 3 and 14 days in 24 (43%), and > 15 days in 19 (34%).

Hemothorax management

Left HTX was present in all cases and was bilateral in three (5%). Hemothorax thickness on pre-operative computed tomography angiography was < 2 cm in 13 (23%) cases, between 2 and 3 cm in 13 (23%), and > 3 cm in 30 (54%). Overall, HTX was drained in 21 (37.59%) cases: 1 of 13 (7%) for HTX < 2 cm, 6 of 13 (46%) for HTX between 2 and 3 cm, and 14 of 30 (47%) for HTX > 3 cm. All patients but one were drained within the first 3 post-operative days. A chest tube was used in 16 (28%), and surgical evacuation in six (11%). Co-morbidities and risk factors were similar in drained and retained HTX, but drained patients showed worst pre-operative levels of lactate (mEq/L, 3.1 ± 2.3 vs. 1.8 ± 1.0, $p = .028$), PCO_2 (mmHg, 41 ± 9 vs. 36 ± 5, $p = .037$), and post-operative RI (198 ± 106 *vs.* 311 ± 88, $p = .001$): in hospital mortality did not differ in these two groups (18% vs. 6%, $p = .197$). Although traumatic and non-traumatic ruptures had significantly different co-morbidities, risk factors and respiratory status (Table 2), the need of chest drainage (48% vs. 33%, $p = .838$) did not differ between the two groups.

Early outcomes

The aortic related mortality rate was 12.5% ($n = 7$): causes of death were shock ($n = 6$, 11%) and respiratory failure ($n = 1$, 2%). Mean hospitalization was 21 ± 16 days (range 2—83 days). TEVAR related complications are summarized in Table 3. In particular, respiratory complications occurred in

Table 2. Comparative data of aortic pathology ruptures.

Variable[a]	Traumatic ($n = 23$)	Non-traumatic ($n = 33$)	p
Demographic data (%)			
M : F	19 : 4	21 : 12	.145
Age (mean ± SD)	46 ± 20	74 ± 11	< .001
IHD	3 (13)	13 (39)	.039
Cardiac disease	0 (0)	7 (21)	.034
COPD	4 (17)	12 (36)	.145
Diabetes	1 (4)	11 (33)	.009
AKI	0 (0)	8 (24)	.016
Obesity (BMI > 30)	1 (4)	13 (39)	.004
Operative risk			
Shock (%)	13 (56.5)	6 (18)	.004
EuroSCORE (mean ± SD)	8 ± 2.5	10 ± 4	.038
Respiratory parameters (± SD)			
Acid base status	7.3 ± 0.1	7.4 ± 0.1	< .001
Po_2 (mmHg)	93 ± 23	82 ± 22	.076
Pco_2 (mmHg)	41 ± 9	37 ± 6	.050
Lactates (mEq/L)	3.3 ± 2.5	2 ± 1.4	.016
Hemothorax thickness (%)			
< 2 cm	2 (9)	11 (33)	.051
2—3 cm	3 (13)	11 (33)	.119
> 3 cm	18 (78)	11 (33)	< .001
Respiratory index (± SD)	208 ± 104	286 ± 105	.008

AKI = acute kidney injury; BMI = body mass index; CAD = coronary artery disease; COPD = chronic obstructive pulmonary disease; Hb = hemoglobin; ICU = intensive care unit; LHF = left heart failure; LOS = length of stay; SBP = systolic blood pressure.

[a] For numerical variables, mean ± SD; for categorical variables, number and percentages.

23 (41%) patients. Overall, no patients required definitive tracheotomy; briefly, those who developed post-operative respiratory complications were older (63 ± 21 vs. 62 ± 21, $p = .012$), had lower pre-operative PO_2 values (mmHg, 80 ± 24 vs. 91 ± 21, $p = .012$), and worse RI at the end of intervention (265 ± 110 vs. 311 ± 88, $p = .001$). No other significant differences were observed (Table 4). Respiratory complications led to longer mechanical ventilation (7 ± 9 vs. 2 ± 7 days, $p = .001$), ICU stay (2.3 ± 0.7 vs.

Table 3. Post-operative major complications.

Complication	n (%)	Treatment
Respiratory	23 (41)	
Grade 1	8 (14)	
Grade 2	8 (14)	
Grade 3	7 (12.5)	
Cardiovascular		
Atrial fibrillation	2 (3.5)	
Ischemic colitis	1 (2)	Hartman procedure
AVF malfunction	1 (2)	vBCT stent
Miscellaneous		
Paraplegia	1 (2)	
AKI	1 (2)	Temporary HD
Dysphagia	1 (2)	

AVF = arteriovenous fistula; AKI = acute kidney injury; vBCT = vein brachiocephalic trunk; HD = hemodialysis.

1.9 ± 0.8 days, $p = .017$), and hospitalization (296 ± 21 vs. 17 ± 12 days, $p = .009$). Post-operatively, traumatic and non-traumatic ruptures did not differ significantly for prolonged (> 10 days) ventilation time (9% vs. 12%, $p = 1.0$), respiratory complications (30% vs. 48%, $p = .269$), hospitalization (25 ± 21 vs. 19 ± 15 days, $p = .217$), and in hospital mortality (9% vs. 12%, $p = 1.0$). In hospital mortality did not differ between patients with and without respiratory complications (13% vs. 12%; $p = .990$).

Late outcomes

Forty-nine (87.5%) patients were discharged alive: 48 of 49 (86%) patients were available for follow up. Mean follow up was 39 ± 29 months (range 1–108; median 40 months). TEVAR related re-intervention was never required. Nine (19%) patients died during follow up (mean 24 months): aortic related mortality was not observed, and the predominant cause of death was acute myocardial infarction ($n = 4$). Estimated overall survival was 85 ± 4.8% at 3 months, 79 ± 5.6% at 1 year, and 61 ± 8.5% at 5 years.

DISCUSSION

Population based analysis and single center studies have reported a significant benefit of TEVAR versus open repair for peri-operative mortality, regardless of the indication for repair and type of pathology.[1,2,5–7] Nevertheless, peri-operative mortality after TEVAR for thoracic aortic rupture is still disturbingly high in the range of 8–30%.[1,5,6,22–25] The 12.5% in hospital mortality rate is good if compared with the 35.3% predicted pre-operatively, and it is consistent with the average 19.4% emerging from previous published series which have included all aortic pathologies as underlying causes of the thoracic aortic rupture.[1,5,6,8,22,23] By contrast, the long-term benefit of TEVAR has been associated with conflicting results. Goodney et al.[21] found that patients selected for TEVAR had worse long-term survival than patients selected for open repair. However, they studied an aneurysm cohort only, and postulated that TEVARs were offered to higher risk patients. The estimated 61% survival at 5 years in the present study is better than the 26% reported by Goodney et al., and compares favorably with other series (in the range 23–55%) which include different aortic catastrophes.[2,5,6,8,22–25] In an emergent setting, the rapidity and minimal invasiveness of TEVAR may be the preferred approach for patients who may not have tolerated a complex and risky operation.

On admission, HTX has frequently been noted after thoracic aortic rupture with an associated increased 30 day mortality.[1,8,9,14,22] Unfortunately, not all of the studies described the treatment and fate of HTX after TEVAR. In their multicenter study, Jonker et al.[8] reported a 41.4% rate of HTX on admission, and a six fold increased risk of in hospital mortality when it was present after rupture of a descending aneurysm; however, the management and outcomes of HTX were not described. Only Shu et al.[9] reported the treatment of HTX after TEVAR. They drained 22% (6/27) of the cases in order to improve respiratory function, but included a cohort of complicated dissections only and almost always used thoracocentesis to evacuate the pleural hematoma. This study is substantially different: the

Table 4. Intra-operative and post-operative details with reference to post-operative respiratory complication.

Variable[a]	All patients ($n = 56$)	No complication ($n = 33$)	Complicated ($n = 23$)	p
Demographic data				
M : F	40 : 16	25 : 8	15 : 8	.55
Age (yrs)	63 ± 21	62 ± 21	64 ± 21	.012
Risk factors				
CAD	16 (29)	8 (24)	8 (35)	.55
Valve disease/LHF	7 (13)	1 (3)	6 (26)	.015
COPD	16 (27)	7 (21)	9 (39)	.23
Diabetes	13 (23)	8 (24)	5 (22)	.99
AKI	8 (14)	5 (15)	3 (13)	.99
Obesity (BMI > 30)	15 (27)	8 (24)	7 (30)	.76
Aortic disease		.31		
Degenerative	33 (59)	17 (52)	16 (70)	
Trauma	23 (41)	16 (48)	7 (30)	
Operative risk				
Shock	19 (34)	10 (30)	9 (39)	.57
EuroSCORE (additive)	9 ± 4	9 ± 3	10 ± 4	.44
Preoperative parameters				
Acid base status	7.4 ± 0.1	7.3 ± 0.1	7.4 ± 0.1	.99
Po_2 (mmHg)	87 ± 23	91 ± 21	80 ± 24	.012
Pco_2 (mmHg)	38 ± 7	36 ± 5	40 ± 9	.13
Lactates (mEq/L)	2.4 ± 1.9	1.8 ± 1.3	3.1 ± 2.3	.53
Hemothorax thickness > 3 cm	29 (52)	20 (61)	9 (39)	.17
Respiratory index	265 ± 110	311 ± 88	198 ± 106	.001

AKI = acute kidney injury; BMI = body mass index; CAD = coronary artery disease; COPD = chronic obstructive pulmonary disease; Hb = hemoglobin; LHF = left heart failure; SBP = systolic blood pressure.

[a] For numerical variables, mean ± SD; for categorical variables, number and percentages.

percentage of pleural drainage is more than double and either chest tube or open surgery was used. These data may be explained by the fact that only patients with HTX on the baseline CT scan were included, and they underscore the different severity of the HTX in these cases. Nevertheless, this study agrees with that of Shu et al. that prompt drainage should be performed to improve respiratory function and survival. The aggressive approach may have been a contributing factor to explain the fact that the mortality rate of patients who needed drainage of their HTX was not different from those that were not drained, despite a more critical pre-operative respiratory condition. An important aspect to be analyzed is the meaning of HTX and the need for chest drainage in different settings. Eggebrecht et al.[22] showed that patients with complex aortic pathologies were at higher risk of a poor outcome. In this study, rupture of degenerative disease and traumatic rupture were significantly different populations, but the underlying pathology had no impact on post-operative outcomes. These data are supported by the study of Cambria et al.[6] on thoracic aortic catastrophes when outcomes were controlled for baseline co-morbidities and age, the effect of pathology was not significant. This is not because traumatic patients did worse, rather, the explanation lies in the different risk profile of the two groups: most of the traumatic ruptures occurred in younger patients, whereas patients with ruptures of degenerative disease were older and with multiple more severe co-morbidities. Hence, despite the less severe pre-operative condition, the latter group included more fragile patients who benefited from an equally aggressive drainage of HTX.

An interesting issue to be debated is how often there is rupture after acute type B dissection. This is not a rare event, and has been associated with a higher operative mortality than those with other types of complications.[26–29] The 14% in this study is less than the 23.1–60% reported in different types of studies, but it is similar to the 15.2% reported by Cambria et al.,[6] who also included all types of thoracic aortic ruptures.[26–29]

It has been demonstrated that when lysis of chest hematomas is incomplete, by the 7th day HTX may adhere to the lung and pleura, making it difficult to remove, and leading to respiratory complications or infection.[30] Therefore, clotted HTX should be considered for evacuation within a reasonable time; currently, it is still unclear whether, when, and how to perform chest drainage. A recent paper by Bradley et al.[31] showed that the volume of retained HTX was not predictive of post-operative respiratory complications. Shu et al.[9] drained the chest according to the amount of HTX specifically to improve respiratory function and obtained resolution in all cases. These experiences analyzed only specific populations such as non-aorta related trauma patients or dissections, did not use a specific timing algorithm or volume limit for HTX evacuation, or report the respiratory outcomes. All these data underscore the lack of uniformity in current practice for HTX management. This potentially also applies to this study, because 40% of the cases were drained. Nevertheless, the policy was to drain the chest promptly in the post-operative period, especially for the greater HTX thickness ($>$ 3 cm) and in patients with a respiratory index $<$ 200. Certainly, the compared groups are not similar, but such an aggressive approach in this subset of patients may have contributed to limiting the number of the most severe (SVS grade 2 or 3) respiratory complications and overall mortality. The incidence of respiratory complications after TEVAR for thoracic aortic rupture has been reported in the range of 15–39%.[2,4,5,22,25] A recent nationwide study on use and outcomes of TEVAR for ruptured thoracic aortic aneurysms reported that respiratory complications were alarmingly high at 63%, and even higher in the TEVAR group compared with conventional repair.[3] However, when pleural effusion or HTX were eliminated from the final analysis the respiratory complications were lower in the TEVAR group. None of these studies used a specific definition or analyzed the respiratory complication rate after TEVAR. The 41% reported in this study was higher than previously reported, but all types of respiratory complication should be included in accordance with the SVS guidelines.[20]

Limitations of the study

The multicenter design of this study has several limitations. The number of patients remains relatively small for adequate analysis and multivariate adjustment. Another important limitation was the retrospective design. Finally, owing to missing worldwide accepted guidelines, indications for the management of the retained HTX may have affected the results. However, due to the rarity of these events and its emergent nature, it will be very difficult to ever present a large and homogeneous study. Despite these limitations, there are no similar studies in the literature: the major critical aspect of the paper is the inability to present generalizable conclusions, but this cohort of patients with HTX due to thoracic aortic rupture treated with TEVAR is currently the largest available, and may lead to larger studies to outline the best management of this condition.

CONCLUSION

This study confirms that descending thoracic aortic rupture still carries a high mortality even in the TEVAR era; in addition, respiratory complications have not been eliminated by this less traumatic technique.

Prompt HTX evacuation may have contributed to limiting post-operative mortality in drained patients who presented with significantly worse pre-operative respiratory parameters. Although traumatic and degenerative ruptures are two significantly different scenarios, survival and respiratory outcomes were not affected by the underlying aortic disease.

CONFLICT OF INTEREST

None.

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