




Brief report on the development of hemorrhagic pericardial effusion after thoracic surgery for traumatic injuries

Grace Martin Niziolek ¹, Philip Dowzicky,¹ Sarah Joergensen,¹ Alea Zone,¹ Martha M Brinson,¹ Niels D Martin ², Mark J Seamon,¹ Shariq Raza,¹ Jay Yelon,¹ Jeremy Cannon ¹

¹Surgery, University of Pennsylvania Perelman School of Medicine, Philadelphia, Pennsylvania, USA

²Surgery, University of Pennsylvania, Philadelphia, Pennsylvania, USA

Correspondence to

Dr Grace Martin Niziolek; grace.niziolek@penmedicine.upenn.edu

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ABSTRACT

Introduction Hemorrhagic pericardial effusion (HPE) is a rare but life-threatening diagnosis that may occur after thoracic trauma. Previous reports have concentrated on delayed HPE in those who did not require initial surgical intervention for their traumatic injuries. In this report, we identify and characterize the phenomenon of HPE after emergent thoracic surgery for trauma.

Methods This is a retrospective review of patients who required emergent thoracic surgery for trauma at a level 1 trauma center from 2017 to 2021. Using the institutional trauma database, demographics, injury characteristics, and outcomes were compared between patients with HPE and those without HPE after thoracic surgery for trauma.

Results Ninety-one patients were identified who underwent emergent thoracic surgery for trauma. Most were young men who sustained a penetrating thoracic injury. Seven patients (7.7%) went on to develop HPE. Patients who developed HPE were younger (18 vs. 32 years, $p=0.034$), required bilateral anterolateral thoracotomy (85% vs. 7%, $p<0.001$), and were more likely to have pulmonary injuries (100% vs. 52.4%, $p<0.001$). Five patients with HPE survived to hospital discharge. The two patients with HPE who died were both coagulopathic and had HPE diagnosed within 4 days of injury. The median time to HPE diagnosis in survivors was 24 days with four of five HPE survivors on therapeutic anticoagulation at the time of diagnosis.

Conclusions HPE may occur after emergent thoracic surgery for trauma. Those at highest risk of HPE include younger patients with bilateral thoracotomy incisions and pulmonary injuries. Early HPE, clinical signs of tamponade, and/or coagulopathy in patients with HPE portend a worse prognosis. Surgeons and trauma team members caring for patients after emergent thoracic exploration for trauma should be aware of this potentially devastating complication and should consider postoperative echocardiography in high-risk patients.

has only previously been reported as a complication of non-operative management of penetrating thoracic injuries.³ Here we present seven patients who developed HPE after thoracic surgery for penetrating injuries, which has not previously been described. We hypothesized that patients with cardiac injuries and those requiring therapeutic anticoagulation postoperatively were at higher risk of developing HPE. Additionally, we sought to identify other variables that may place patients at risk of developing postoperative HPE, characterize postoperative outcomes for these patients, and clarify how best to recognize and manage this rare condition.

PATIENTS AND METHODS

For this retrospective case–control study, we queried our institutional trauma registry for all patients who required emergent thoracic surgical exploration for trauma between January 2017 and December 2021. Those who did not survive past 12 hours and those younger than 18 years were excluded from the study. Patients were included if they underwent emergency resuscitative thoracotomy even if they did not sustain thoracic trauma. HPE was defined as sanguinous pericardial effusions that were confirmed upon a drainage procedure. Patient demographics, Injury Severity Score (ISS), chest Abbreviated Injury Scale (AIS), hospital-free days, intensive care unit (ICU)-free days, ventilator-free days, massive transfusion, and in-hospital mortality were obtained from the trauma registry.⁴ Medical record review identified chest injuries sustained, operative details, administration and indication for therapeutic anticoagulation, development of HPE, days to HPE diagnosis, and tamponade physiology. Operative notes were reviewed for the type of chest incision performed at initial and subsequent operations, placement of pericardial drains, and pericardial closure prior to the diagnosis of HPE.

Demographics, injury characteristics, and outcomes were compared between patients who developed HPE and those who did not. Similar analysis was undertaken to assess for differences in survivors versus non-survivors of HPE. A subgroup analysis of patients who underwent bilateral anterolateral thoracotomy was also undertaken as this incision was associated with higher rates of HPE. Descriptive analysis included percentages

INTRODUCTION

Hemorrhagic pericardial effusion (HPE) after penetrating and blunt trauma is an uncommon but well-documented phenomenon. HPE is most often observed after penetrating thoracic trauma but has also been reported after severe blunt thoracic trauma as well as after cardiac catheterization.^{1–2} Delayed HPE is a less common phenomenon and

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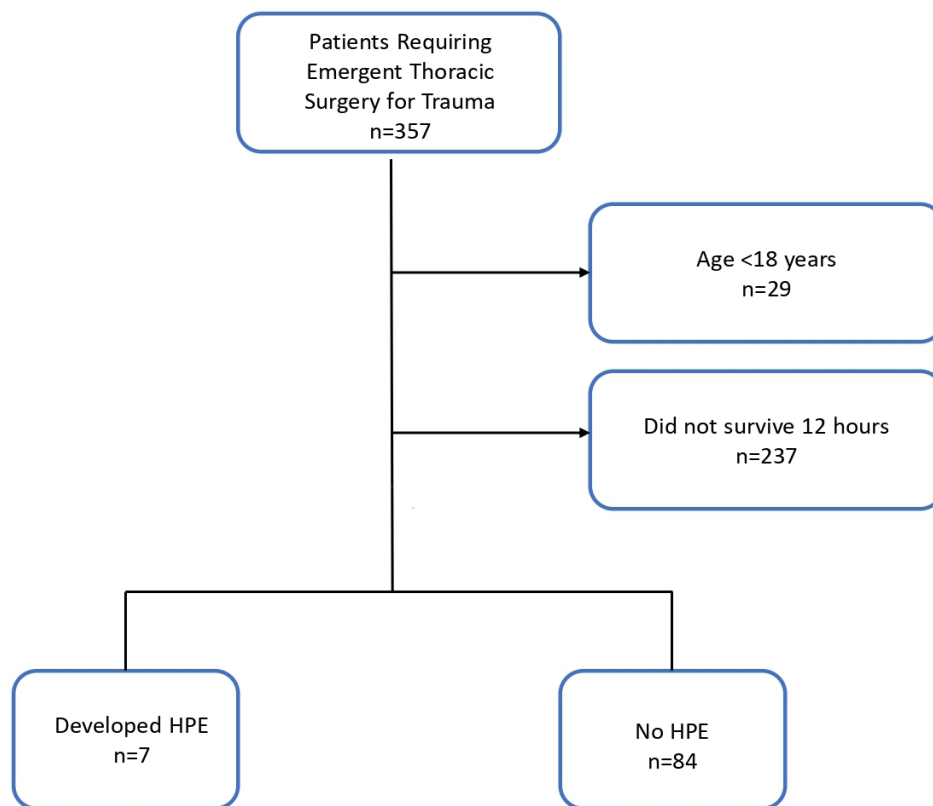


Figure 1 Flow diagram illustrating patients included in the final analysis. HPE, hemorrhagic pericardial effusion.

or medians with IQRs. Two-tailed Student's *t*-tests and χ^2 analyses were used to compare cases and controls. A *p* value of less than 0.05 was considered significant. All data analyses were performed using jamovi (The jamovi project (2021), V.1.6, <https://www.jamovi.org>, Sydney, Australia).

RESULTS

Trauma database review identified 357 patients who required emergent thoracic surgery during their index hospitalization. Of these patients, 91 survived past 12 hours and were included in the analysis (figure 1). Seven patients developed HPE after initial surgery for thoracic trauma. All seven patients were males who sustained gunshot wounds to the chest and were significantly younger compared with those who did not develop HPE (18 years vs. 32 years, $p=0.034$). There was no difference in modes of transport, ISS or chest AIS between the groups (table 1). Patients with HPE experienced more ventilator-free days (14 vs. 26 days, $p=0.013$) and fewer ICU-free days (9 vs. 22 days, $p=0.43$). There were no differences in mortality rates. Those who developed HPE were more likely to undergo bilateral anterolateral thoracotomy (BALT) (85.7% vs. 7.1%, $p<0.001$), more likely to have sustained pulmonary injuries (100% vs. 52.4%, $p=0.011$), and more likely to have required a second thoracic operation prior to their HPE diagnosis (85.7% vs. 4.8%, $p<0.001$). The majority of second thoracic operations were planned operations after initial damage control surgery. There were no differences in rates of cardiac injuries, pericardial closure, placement of pericardial drains, or therapeutic anticoagulation between HPE cases and controls. Patients who developed HPE were more likely to require either tractotomy (57.1% vs. 20.9%, $p=0.043$) or lobectomy (28.6% vs. 4.7%, $p=0.031$) for control of pulmonary hemorrhage. Prior to HPE

diagnosis, all patients experienced tachycardia (>90 beats/min), six patients (85.7%) had abnormal chest radiographs—five of which had widened cardiac silhouettes; five patients (71.4%) had a leukocytosis (>11), four patients (57.1%) were noted to have hypotension (<90 mm Hg systolic), and three patients (42.9%) experienced fevers >101.4 . Four patients had an echocardiogram performed at some point prior to their diagnosis. Of these patients, one patient had a moderate pericardial effusion noted, one had a trivial effusion noted, and two patients had no effusion noted. No patients were diagnosed with pericarditis prior to HPE diagnosis.

Two of the seven patients with HPE did not survive to discharge. There were no differences in demographics or injury characteristics between survivors and non-survivors with HPE (table 2). Both HPE non-survivors underwent BALT and required repeat thoracotomies for HPE. Neither of the patients had pericardial drains placed, likely because they did not undergo definitive thoracic closure. One death was attributed to severe coagulopathy and the other to erosion from a sternal spacer into the right ventricle (table 3). Both HPE non-survivors were diagnosed with HPE within 4 days of their initial injury. Of the five HPE survivors, one underwent pericardial closure and three were managed without pericardial drains placed at the time of their chest closure. Prior to the diagnosis of HPE, four patients (57.1% of all cases, 80% of survivors) required therapeutic anticoagulation. All four of these patients had been diagnosed with either a pulmonary embolism or deep vein thrombosis. Additionally, one patient had a concomitant diagnosis of a blunt cerebrovascular injury and another patient required anticoagulation while on extracorporeal membrane oxygenation. All four of these patients had been therapeutically anticoagulated for at least 3 days prior to being diagnosed with HPE. The only HPE survivor who did

Table 1 Demographics, outcomes, and management of injuries in patients requiring emergent thoracic surgery for trauma (n=91)

	HPE n=7	No HPE n=84	P value
Male (%)	100	86.9	0.307
Age (years; median, IQR)	18 (17.5–26.5)	32 (24–44)	0.034
Penetrating mechanism (%)	100	76.2	0.144
ISS (median, IQR)	25 (19–38.5)	26 (18–33)	0.536
Chest AIS (median, IQR)	4 (4–5)	4 (3–5)	0.655
Mode of transport			0.156
Fire rescue or ambulance (%)	14.3	42.0	
Police (%)	85.7	48.1	
Private vehicle (%)	0	10.0	
Hospital-free days (median, IQR)	1 (0–13)	15 (7–21)	0.089
ICU-free days (median, IQR)	9 (2–20)	22 (15.5–25)	0.043
Ventilator-free days (median, IQR)	14 (4–22)	26 (23–27)	0.013
Required massive transfusion (%)	85.7	50.0	0.069
Alive at discharge (%)	71.4	89.3	0.164
Initial chest incision			
Left anterolateral thoracotomy (%)	0.0	45.2	0.023
Right anterolateral thoracotomy (%)	0.0	11.9	0.283
Bilateral anterolateral thoracotomy (%)	85.7	7.1	<0.001
Median sternotomy (%)	28.6	36.9	0.750
Video-assisted thoracoscopic surgery (%)	0.0	1.1	0.772
Associated thoracic injuries			
Cardiac (%)	28.5	28.6	1
Pulmonary (%)	100	52.4	0.011
Great vessel (%)	28.5	7.1	0.054
Esophagus (%)	0.0	3.6	0.611
Tracheobronchial (%)	0.0	2.4	0.680
No injuries (%)	0.0	25.0	0.131
Therapeutic anticoagulation			
In-hospital	57.1%	31.0%	0.157
Discharged	57.1%	25.3%	0.093
Pericardium closed			0.146
Yes (%)	14.3	14.3	
No (%)	85.7	51.2	
Pericardium not opened (%)	0.0	34.5	
Pericardial drain placed			0.559
Yes (%)	28.6	26.2	
No (%)	71.4	38.0	
Pericardium not opened (%)	0.0	35.7	
Required chest reoperation			<0.001
Yes (%)	85.7	4.8	
No (%)	14.3	95.2	

Outcomes are expressed as either medians with SDs or percentage of total patients. Hospital and ICU-free days are out of 28 days.
AIS, Abbreviated Injury Scale HPE, hemorrhagic pericardial effusion; ICU, intensive care unit; ISS, Injury Severity Score.

not require therapeutic anticoagulation had been administered prophylactic anticoagulation prior to the HPE diagnosis.

Of those who developed HPE, three patients (42.9%) experienced clinical signs of pericardial tamponade, whereas another three patients had evidence of tamponade on echocardiography but did not develop tamponade physiology (table 4). All three patients with clinical tamponade physiology required emergent left anterolateral thoracotomy. Only one of these three patients survived to discharge. Those without clinical tamponade were

Table 2 Demographics and outcomes comparison of patients with hemorrhagic pericardial effusions, survivors vs. non-survivors (n=7)

	Survivors n=5	Non-survivors n=2	P value
Age (years, median, IQR)	18 (18–23)	23.5 (20–27)	0.879
Male (%)	100	100	1
Penetrating mechanism (%)	100	100	1
ISS (median, IQR)	25 (21–42)	26 (22–31)	0.626
Chest AIS (median, IQR)	4 (4–5)	4.5 (4–5)	0.623
Hospital-free days (median, IQR)	0 (0–1)	27.5 (27–28)	<0.001
ICU-free days (median, IQR)	4 (0–9)	25.5 (24.8–26.3)	0.004
Ventilator-free days (median, IQR)	7 (0–14)	26 (25–26)	0.048
Required massive transfusion (%)	80.0	100	0.495
Initial chest incision			
Bilateral anterolateral thoracotomy (%)	80.0	100	0.495
Median sternotomy (%)	40.0	0.0	0.290
Associated thoracic injuries			
Cardiac (%)	20.0	50.0	0.427
Pulmonary (%)	100	100	1
Great vessel (%)	40.0	0.0	0.290
Therapeutic anticoagulation	80.0%	0.0%	0.053
Pericardial drainage procedure			
Thoracotomy (%)	20.0	100	0.053
Subxiphoid window (%)	60.0	0.0	0.147
Pericardial drainage catheter (%)	20.0	0.0	0.495

Outcomes are expressed as either medians with SDs or percentage of total patients. Hospital and ICU-free days are out of 28 days.
AIS, Abbreviated Injury Scale; ICU, intensive care unit; ISS, Injury Severity Score.

subject to less invasive methods to relieve HPE, with three patients (75%) who received subxiphoid windows and one patient (25%) who underwent pericardiocentesis with interventional cardiology. Of the five patients who survived to discharge, three were prescribed short courses of direct oral anticoagulants for venous thromboembolic disease, one was prescribed a full-dose aspirin for a blunt cerebrovascular injury, and one patient was placed on prophylactic enoxaparin. Of the HPE survivors, two patients were discharged home and three patients were discharged to skilled nursing facilities.

Twelve patients required BALT, of which half (six patients) were diagnosed with HPE. All patients were male and sustained gunshot wounds resulting in thoracic trauma. Those who developed HPE after BALT had lower ISS (23 vs. 45, $p=0.046$) but similar chest AIS (4 vs. 4, $p=0.734$). No differences were noted in rates of specific types of injuries (pulmonary, cardiac, etc), therapeutic anticoagulation, pericardial closure, pericardial drainage, hospital-free or ICU-free days.

DISCUSSION

In this brief review, we report on seven patients who developed HPE after thoracic surgery for trauma. We compared demographics, injury characteristics, and outcomes of these cases with other controls who underwent thoracic surgery for trauma, most for penetrating thoracic trauma. Most notably, patients who developed HPE were younger and were more likely to have received BALT for severe pulmonary injuries compared with those without HPE.

We suspect that HPE likely occurs more frequently than is diagnosed as only three of seven patients had signs of clinical tamponade physiology during their hospitalization. Previous reports of HPE focus on those with blunt trauma or those with

Table 3 Initial management of patients who developed HPE after chest surgery for traumatic injuries; hospital courses of patients with HPE

Patient	Chest incision	Pericardial closure	Pericardial drain	Therapeutic anticoagulation	Reason for anticoagulation	Hospital day of HPE diagnosis	Reason for HPE	Alive at discharge
1	BALT	No	No	None		4	Sternal spacer erosion into RV	No
2	BALT	No	No	Heparin & argatroban drips	ECMO, DVTs	35	Unknown	Yes
3	BALT	No	No	None		21	Unknown	Yes
4	BALT, median sternotomy	Yes	Yes	Heparin drip	PE, DVT	25	Unknown	Yes
5	BALT	No	No	None		1	Coagulopathy	No
6	Median sternotomy	No	Yes	Enoxaparin	BCVI, DVT	16	Unknown	Yes
7	BALT	No	No	Enoxaparin, Plavix	PE	24	Unknown	Yes

BALT, bilateral anterolateral thoracotomy; BCVI, blunt cerebrovascular injury; DVT, deep venous thrombosis; ECMO, extracorporeal membrane oxygenation; HPE, hemorrhagic pericardial effusion; PE, pulmonary embolism; RV, right ventricle

delayed HPE after unexplored penetrating injuries.^{1,2} However, we concentrate on seven patients who developed HPE after thoracic surgery for penetrating injuries. Six out of seven patients required BALT—extensive incisions that are generally used for patients in extremis or with significant bilateral thoracic or transmediastinal injuries. As such, we suspect this likely represents a complication of unexpected trauma survivorship. In this study, high-risk patients appear to be those who are severely injured, most commonly with pulmonary or great vessel injuries, and/or require BALT for either resuscitation or hemorrhage control. Postoperatively, high-risk patients are those who require additional thoracic procedures as well as patients who require therapeutic anticoagulation or antiplatelet therapy. Six out of seven patients were either coagulopathic or therapeutically anticoagulated prior to their HPE diagnosis. In a case series of three patients who developed late HPE after blunt trauma, Gabram *et al* reported that two of three patients required therapeutic anticoagulation for venous thromboembolisms (VTEs). They observed that HPE occurred between 12 and 15 days after traumatic injury. They postulated that the combination of myocardial contusion or small torn pericardial vessels bled in a delayed fashion which may have been exacerbated or caused by the administration of therapeutic anticoagulation.¹ Similarly, four patients in our study required therapeutic anticoagulation for VTEs; however, the number of days to HPE diagnosis was longer with a range of 16–35 days after traumatic injury.

Table 4 Pericardial drainage procedure and mortality according to presence of tamponade physiology at diagnosis of HPE

A. Management of HPE			
Patient	Clinical tamponade	Pericardial drainage method	
1	Yes	Left anterolateral thoracotomy	
2	No	Subxiphoid window	
3	No	Subxiphoid window	
4	Yes	Left anterolateral thoracotomy	
5	Yes	Left anterolateral thoracotomy	
6	No	Pericardial drainage catheter (interventional cardiology)	
7	No	Subxiphoid window	
B. Mortality rates based on management of HPE			
Clinical tamponade	Pericardial drainage method	%	Alive at discharge
Yes (n=3)	Left anterolateral thoracotomy	100	33.30%
No (n=4)	Subxiphoid window	75.00	100%

HPE, hemorrhagic pericardial effusion.

Prior to HPE diagnosis, all patients were tachycardic, and six of seven patients had a widened cardiac silhouette on their chest radiograph. Hypotension was only present in four patients and may have been a result of tamponade physiology, though two patients were also in hemorrhagic shock at the time of their HPE diagnosis. Notably, those with clinical evidence of tamponade had the worst outcomes, with 66.7% mortality. Those without tamponade physiology were generally able to be treated with less invasive procedures, including percutaneous drainage. The method chosen to intervene on hemopericardium was likely the result of clinical stability. Those who underwent left anterolateral thoracotomy did so emergently and in one case, this was performed in the ICU. Although it is impossible to determine causation in this small study, progression to tamponade physiology appears to be associated with mortality.

The HPE survivors in this case-control study resemble post-pericardiotomy syndrome (PPS) as no definitive source for hemorrhage, such as missed cardiac injury or bleeding suture line was identified in these patients. Every HPE survivor underwent pericardiotomy via a large thoracic incision with the median time to HPE diagnosis 24 days after initial thoracic surgery. In one of the first studies to examine PPS risk factors, Miller *et al* found that younger age and lower body weight, among others, were associated with increased odds of developing PPS.⁵ Later, Imazio *et al* found that a pleural incision was an independent risk factor for development of PPS.⁶ Similarly, patients with HPE in this study were found to require BALT and were significantly younger compared with those who did not go on to develop HPE suggesting some commonality with PPS. Treatment for PPS is similar to that for pericarditis, and the European Society of Cardiology recommends colchicine added to aspirin or non-steroidal anti-inflammatory drug for acute PPS therapy.⁷ We cannot recommend any particular medication regimen either for prophylaxis or therapy as this is a small retrospective case series. However, the PPS guidelines also recommend routine follow-up with echocardiography every 6–12 months in those diagnosed with PPS to evaluate for progression to constrictive pericarditis. This may be the best takeaway from the PPS guidelines and should also be incorporated into the postoperative care of patients who develop HPE after thoracic surgery for trauma.

Our study has several limitations. This is a single-center, retrospective study and is underpowered to make any strong conclusions. Given the small study size, we are unable to perform multivariable analysis to identify independent predictors. Additionally, we do not report on post-discharge follow-up in either the patients with HPE or those without HPE. Future work should prospectively study patients at risk to understand the true incidence, risk factors, and outcomes.

CONCLUSIONS

HPEs may occur after thoracic surgery for traumatic injuries, and providers should be aware of this rare but potentially devastating complication. Patients at highest risk of developing HPE are those with large chest incisions, therapeutically anticoagulated, or coagulopathic. Once the patient is through the initial resuscitative period, HPE more resembles PPS as is observed in cardiac surgery patients. Providers should consider screening high-risk patients with an echocardiogram prior to discharge from the hospital. Patients diagnosed with HPE should be considered for longer-term follow-up echocardiography to evaluate for HPE recurrence or progression to constrictive pericarditis.

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ORCID iDs

Grace Martin Niziolek <http://orcid.org/0000-0003-1317-0511>

Niels D Martin <http://orcid.org/0000-0002-2157-0825>

Jeremy Cannon <http://orcid.org/0000-0002-2969-9316>

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