Trauma Case Reports 34 (2021) 100498

Contents lists available at ScienceDirect

Trauma Case Reports

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

Case Report

Prolonged mechanical rib separation is a key element to prevent thoracic compartment syndrome in penetrating chest trauma: A case report

Emily J. MacKay [a,1], Ng T. Niu [b,2], Jeremy W. Cannon [b,3], Lewis J. Kaplan [b,4], José L. Pascual [b,*]

[a] Department of Anesthesiology & Critical Care, Perelman School of Medicine at the University of Pennsylvania, United States of America
[b] Division of Traumatology, Surgical Critical Care & Emergency Surgery, Department of Surgery, Perelman School of Medicine at the University of Pennsylvania, United States of America

ARTICLE INFO

Keywords:
Cardiac trauma
Novel

ABSTRACT

Penetrating cardiac injury in trauma patients is highly morbid. Most cases do not survive long enough to manifest the severe physiologic consequences of massive blood product resuscitation, namely, thoracic compartment syndrome and right ventricular (RV) failure. This case exhibits a thoracic compartment syndrome and RV failure so severe that the open chest management required mechanical separation of a clamshell thoracotomy. The resuscitation and the techniques utilized to maintain an open chest will be described.

Introduction

Mortality for penetrating cardiac injury is high (range 27–42%) [1,2]. In many instances, these patients do not survive long enough to manifest thoracic compartment syndrome. The case presented illustrates the hemodynamic benefit of mechanical rib separation following a penetrating cardiac trauma and massive resuscitation.

* Corresponding author at: Division of Traumatology, Surgical Critical Care and Emergency Surgery, Penn Presbyterian Medical Center, 51 N. 39th Street, Medical Office Building 1st Floor, Suite 120, Philadelphia, PA 19104, United States of America.
E-mail addresses: Emily.mackay@pennmedicine.upenn.edu (E.J. MacKay), Ng.Niu@uphs.upenn.edu (N.T. Niu), Jeremy.Cannon@uphs.upenn.edu (J.W. Cannon), Lewis.Kaplan@uphs.upenn.edu (L.J. Kaplan), Jose.Pascual@uphs.upenn.edu, pascual@pennmedicine.upenn.edu (J.L. Pascual).
[1] Department of Anesthesiology & Critical Care, Hospital of the University of Pennsylvania, 310 Blockley Hall, 423 Guardian Drive, Philadelphia, PA 19104, United States of America.
[2] Division of Traumatology, Surgical Critical Care and Emergency Surgery, Penn Presbyterian Medical Center, 51 N. 39th Street, United States of America.
[3] Division of Traumatology, Surgical Critical Care and Emergency Surgery, Penn Presbyterian Medical Center, 51 N. 39th Street, Medical Office Building 1st Floor, Suite 120, Philadelphia, PA 19104, United States of America.
[4] Division of Traumatology, Surgical Critical Care and Emergency Surgery, Veterans Affairs Medical Center of Philadelphia, 3900 Woodland Avenue, Surgical Business Office, 5th Floor, Philadelphia, PA 19104, United States of America.

https://doi.org/10.1016/j.tcr.2021.100498
Accepted 14 June 2021
Available online 24 June 2021

Case

Consent was obtained for the write up of this this case-report. A 17-year-old male presented with gunshot wounds to the left chest and was actively receiving chest compressions upon arrival to the trauma bay. An anterolateral ED thoracotomy was immediately performed with evacuation of pericardial tamponade and cross clamp of the descending aorta. After conversion to a clamshell thoracotomy, a hole in the apex of the left ventricle was stapled closed with the patient regaining a perfusing rhythm. An expeditious transfer to the operating room followed where a laparotomy was conducted in addition to re-exploration of the chest. Procedures included 1) Revision of the single cardiac defect at the apex of the left ventricle with pledgeted sutures; 2) suture-ligation of both internal mammary arteries; 3) wedge resection of the left lower lobe of the lung; 4) splenectomy; 5) wedge resection of the greater curvature of the stomach; 6) primary repair of diaphragmatic defect. Both cavities were left open in damage control fashion, with the chest opening covered with an esmarch dressing and the abdomen with an Abthera Vac (ABThera™ KCI, San Antonio, TX). Operative resuscitation consisted of 17 units of pRBCs, 6 units of FFP, 2 units of platelets, 875 mL of cell saver, two liters of crystalloid, tranexamic acid supplemented with norepinephrine, phenylephrine and vasopressin drips. The patient required one round of open cardiac massage with prompt return of circulation.

In the surgical ICU, hemostatic damage control resuscitation continued in a 1:1:1 (FFP:platelet:RBC) ratio. Laboratory values on arrival to the ICU revealed a pH of 7.14, lactate >20 mg/dL, Hgb 11.2 mg/dL, platelets 10^3/μL, aPTT 89.3 s., INR 1.6 and fibrinogen 129 mg/dL. While ongoing product resuscitation continued, the patient required escalating doses of epinephrine (14 μg/min), norepinephrine (8 μg/min) and vasopressin (0.04 units/min). An hTEE (ImaCor Inc.® Garden City, NY) demonstrated an underfilled, hyperdynamic left ventricle (LV) (EF = 70%), severe dilatation of the right ventricle (RV) with severely depressed RV function but no pericardial effusion. Two hours after ICU arrival, the chest was reopened at bedside for continued hemodynamic decompensation consistent with tamponade physiology but exploration did not reveal a large clot burden. The damage control resuscitation continued in a 1:1:1 fashion.

Four hours after the initial bedside thoracotomy, the chest was reopened for persistent shock. Blood work immediately prior to reopening included: pH of 7.11, lactate of 12.2 mg/dL, Hgb 5.3 mg/dL, platelets 96 10^3/μL, aPTT 51.3 s, INR 1.2, and fibrinogen of 169 mg/dL. At this point the patient was receiving epinephrine (20 μg/min) norepinephrine (14 μg/min) and vasopressin (0.06 units/min). The resuscitation total up until this point consisted of 62 units of pRBCs, 61 units of FFP, 11 units of platelets, 15 units of cryoprecipitate, three liters of crystalloid, Factor VIIa, Vitamin K, DDAVP, sodium bicarbonate and calcium gluconate. Despite this ongoing resuscitation, the patient remained in a shock state, with evidence of end-organ damage by a precipitous urine output drop (<10 mL/h) – necessitating continuous renal replacement therapy (CRRT) after a failed diuretic challenge.

On examination of the chest, after washout of minimal clot, the heart was seen to be extremely swollen and extruding through the clamshell incision. It was decided to leave the partially opened Finochietto (Codman® Lees Summit, MI) retractor covered in an Ioban (3M™ St. Paul, MN) dressing to prevent recoil of the chest incision onto the heart itself (Figs. 1, 2). Following this second bedside opening of the chest, there was a marked steady improvement in hemodynamics over the course of several hours. The inotropic and vasopressor requirement markedly diminished and 2 h after the second bedside thoracotomy washout epinephrine was at 2 μg/min and both norepinephrine and vasopressin were stopped. With the Finochietto in place, the hTEE revealed a volume-resuscitated LV with hyperdynamic function (EF: 75%), a persistently severely dilated RV but improved (mild-to-moderately decreased) RV function. Transfusion requirements steadily decreased. First labs after the placement of the Finochietto revealed a pH of 7.27, a lactate of 11.6 mg/dL, Hgb of 12.3 mg/dL, platelets 125 10^3/μL, aPTT of 58.9 s, INR 1.5, fibrinogen 183 mg/dL. The 24-hour resuscitation totals (including OR) consisted of 97 units of pRBCs, 77 units of FFP, 15 units of platelets, 15 units of cryoprecipitate, 875 mL of cell saver and

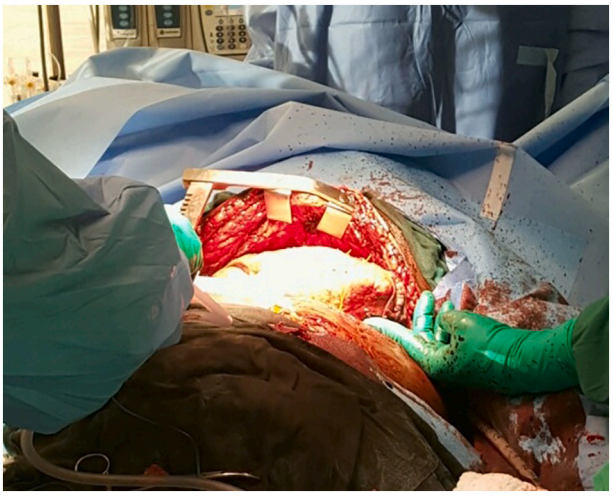

Fig. 1. Finochietto stenting of clamshell thoracotomy.
Placement of the Finochietto retractor following the second chest washout in the surgical ICU.

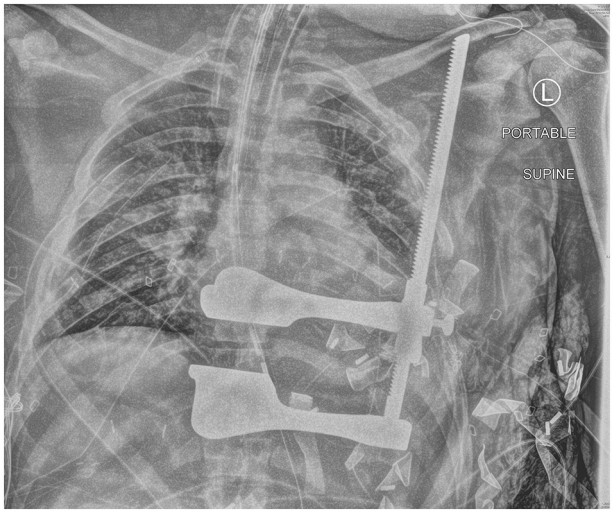

Fig. 2. Finochietto retractor on chest radiograph.
Chest radiograph with the Finochietto retractor in place.

four liters of crystalloid.

The following morning, a transesophageal echocardiogram (TEE) (Philips, Andover, MA) confirmed severe RV dilatation and severe RV failure without pericardial effusion (Fig. 3, Video 1). At this time the patient was not on any inotropic or vasopressor support, CRRT had been initiated and a pulmonary arterial catheter placed. The patient returned to the operating room for planned cavity washout of the abdomen and the chest. The Finochietto was replaced with a more permanent, orthopedic plate (Smith & Nephew Inc., Andover, MA) to stent the sternum (Fig. 4). Intraoperatively, the patient remained hemodynamically stable, without need of any inotropic or vasopressor support and no intraoperative transfusion. The 24-hour resuscitation total for postoperative day one was one unit of RBC and four units of FFP.

The patient returned to the operating room daily for four days for cavity washouts and bilateral lower extremity fasciotomies for secondary extremity compartment syndrome. The RV remained severely dilated with moderately depressed function. Attempts to optimize the RV function consisted of inhaled epoprostenol at 50 ng/kg/min, avoidance of hypercarbia, low dose epinephrine (2 μg/min), milrinone (0.125 μg/kg/min) and liberal diuresis to correct volume overload. Despite this, on postoperative day four, the left pupil was fixed and dilated; brain herniation confirmed by head CT. Brain death was declared by standard criteria, and care was

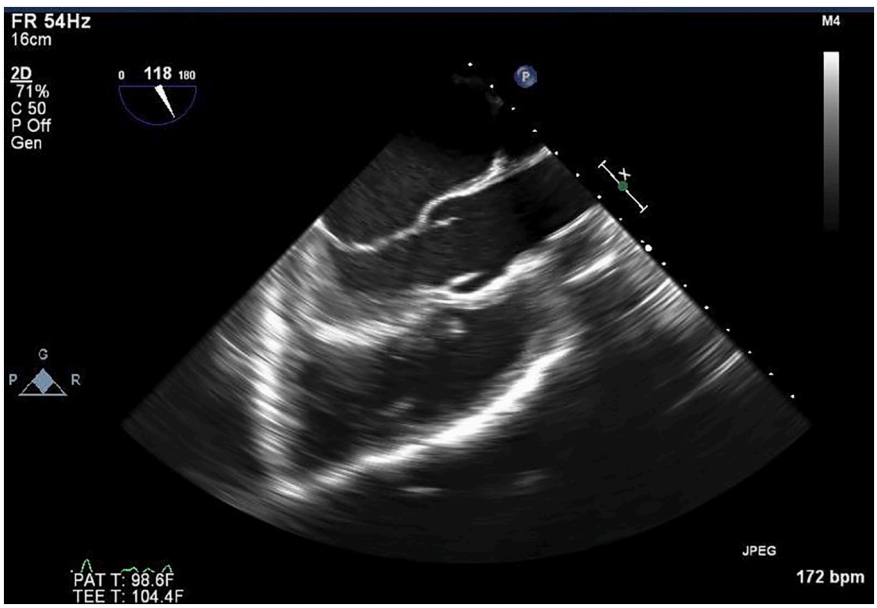

Fig. 3. Postoperative day one TEE.
The TEE view is a midesophageal long axis view with demonstrating severe, right ventricular (RV) dilation.

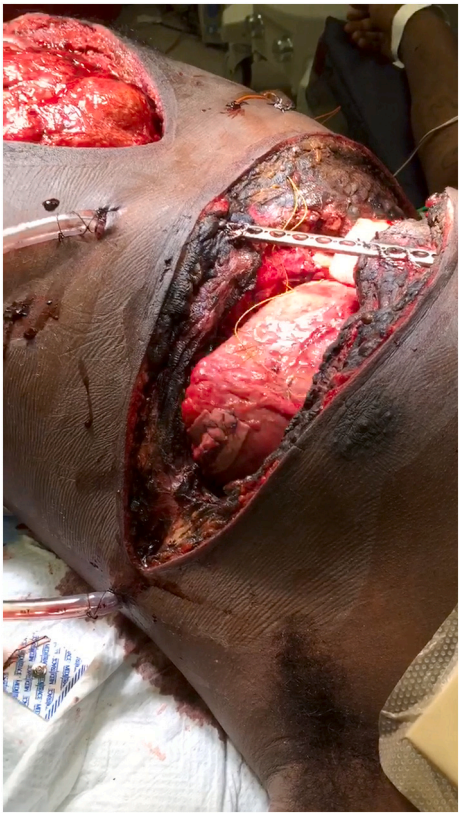

Fig. 4. Orthopedic plate stenting clamshell thoracotomy.
Orthopedic plate screwed to the cephalad and caudad portions of the sternum in order to maintain mechanical rib and sternal separation.

withdrawn at the request of the family. In total, the patient lived for 96 h with a clamshell thoracotomy, mechanically stented open.

Discussion

The survival benefits of damage-control surgery in trauma patients have been established [3]. Damage-control resuscitation; defined as a transfusion ratio of 1:1:1 [4] or 1:1:2 [5] administration of fresh frozen plasma (FFP) to platelets to packed red blood cells (pRBCs), while actively limiting crystalloid has further improved survival in trauma exsanguination [6]. However, nearly unavoidably in the exsanguinating patient requiring massive product resuscitation, even while limiting crystalloid, the RV is forced to contend with a constant state of volume overload. The physiologic consequences and the management of RV failure following cardiac surgery are increasingly better understood [7,8]. Yet, how to manage the physiologic consequences and reach hemodynamic homeostasis in RV failure is underreported and elusive following chest trauma. Also, when massive bleeding occurs with need for multiple vasopressors it is sometimes difficult to gauge when hemorrhage is less important than vasoplegia in contributing to shock and when reduction (not persistence) in blood product resuscitation is necessary. The case presented is a typical illustration of aggressively resuscitating a severe injury resulting in traumatic, shock and cardiac arrest. Where early, rapid maneuvers initially rescued the patient, the RV failure that ensued during that aggressive resuscitation was managed in an unconventional fashion for over 72 h with complete stabilization of hemodynamic compromise. Some would consider the resuscitation to have been excessive leading to RV failure while others would consider RV inevitable with HB levels never rising above 11 (in the first 48 h) indicating only a modest over-transfusion. Furthermore, the addition of steroids may have been useful in the setting of persistent vasoplegia despite ongoing transfusion resuscitation to address possible adrenal exhaustion. Finally, a Swan-Ganz catheter was placed on the second day with continuous imaging of the heart via TEE prior, but earlier placement of a PAC catheter may have better guided resuscitation. Despite hemodynamic recovery, the ultimate cerebral herniation likely occurred secondary to cerebral edema from overwhelming right heart volume overload.

Thoracic compartment syndrome and pericardial tamponade managed by delayed, *midline* sternal closure is well described in the cardiothoracic surgery literature [9]. The reports regarding delayed closure of a *clamshell* thoracotomy exists in bilateral lung transplants reported in the cardiothoracic literature. Force et al. investigated outcomes in bilateral lung transplants requiring delayed sternal closure [10]. The technique described utilized three sternal wires to stent the ribs open [10].

Thoracic compartment syndrome in the trauma patient was first described by Kaplan et al. in 1996 [11]. The case was successfully managed by leaving the left thoracotomy incision open and chest packed [11]. Vargo et al., investigated outcomes in abbreviated

thoracotomy with delayed sternal closure and demonstrated a survival benefit [12]. The temporary chest closure technique described was to cover the thoracotomy incision with either a Silastic sheet, or to close the skin only (with or without gauze packing) [12]. Lang et al. investigated outcomes after ED thoracotomy also utilizing skin closure only (with or without packing) [13]. Yet in all three reports, the natural closing recoil of the chest cavity remained unaltered and direct contact with divided bone and myocardium was not required.

There are two notable differences between the afore-mentioned reports and the case presented. The first difference is that, to our knowledge, this is the first description of a delayed closure technique after a clamshell thoracotomy. The second difference is the amount of blood products administered in this case for the first 24 h; far exceeded the amount reported by Vargo and Lang [12,13].

To our knowledge there is no other report describing the utility of prolonged, mechanical separation of a *clamshell* thoracotomy in trauma case. Chronologically, the hemodynamic turnaround clearly occurred after leaving in place of an incisional separating device; the Finochietto retractor. Additionally, the subsequent utilization of an orthopedic metal bar screwed to the sternum was a novel plan, devised between cardiac and trauma surgeons and to our knowledge, not found in any other published report.

Conclusion

Techniques for thoracic damage-control surgery and resuscitation for penetrating cardiac injuries continue to improve. Beyond the initial, massive resuscitation in the first 24 to 36 h, early de-escalation of massive volume resuscitation is a principle component in damage control resuscitation. In hindsight, earlier mechanical separation of the clamshell thoracotomy may have been helpful in controlling the ongoing coagulopathic bleeding, allowing for earlier de-escalation of massive volume resuscitation. In the event of extensive, subsequent swelling of the heart, temporary open chest management with prolonged active separation of the sternum may be required to restore sufficient chest cavity domain. The technique described herein, as well as other similar methods require further exploration.

Supplementary data to this article can be found online at https://doi.org/10.1016/j.tcr.2021.100498.

Attestation

All authors approved the final manuscript.

CRediT authorship contribution statement

All authors contributed to the write up of this manuscript.

Declaration of competing interest

All authors report no conflicts of interest.

Acknowledgment

The authors gratefully acknowledge the support of Janet McMaster for her role in obtaining permission for the write up of this case report.

References

[1] R.F. Buckman Jr., Penetrating cardiac wounds: prospective study of factors influencing initial resuscitation, J. Trauma 34 (5) (1993) 717–725, discussion 725-7.
[2] B.C. Morse, et al., Penetrating cardiac injures: a 36-year perspective at an urban, level I trauma center, J. Trauma Acute Care Surg. 81 (4) (2016) 623–631.
[3] J.W. Johnson, et al., Evolution in damage control for exsanguinating penetrating abdominal injury, J. Trauma 51 (2) (2001) 261–269, discussion 269–71.
[4] J.B. Holcomb, et al., The PRospective Observational Multicenter Major Trauma Transfusion (PROMMTT) study, J. Trauma Acute Care Surg. 75 (1 Suppl 1) (2013) S1–S2.
[5] J.B. Holcomb, et al., Transfusion of plasma, platelets, and red blood cells in a 1:1:1 vs a 1:1:2 ratio and mortality in patients with severe trauma: the PROPPR randomized clinical trial, JAMA 313 (5) (2015) 471–482.
[6] J.W. Cannon, et al., Damage control resuscitation in patients with severe traumatic hemorrhage: a practice management guideline from the Eastern Association for the Surgery of Trauma, J. Trauma Acute Care Surg. 82 (3) (2017) 605–617.
[7] G.J. Vlahakes, Right ventricular failure after cardiac surgery, Cardiol. Clin. 30 (2) (2012) 283–289.
[8] S. Itagaki, L. Hosseinian, R. Varghese, Right ventricular failure after cardiac surgery: management strategies, Semin. Thorac. Cardiovasc. Surg. 24 (3) (2012) 188–194.
[9] U. Boeken, et al., Open chest management after cardiac operations: outcome and timing of delayed sternal closure, Eur. J. Cardiothorac. Surg. 40 (5) (2011) 1146–1150.
[10] S.D. Force, et al., Outcomes of delayed chest closure after bilateral lung transplantation, Ann. Thorac. Surg. 81 (6) (2006) 2020–2024, discussion 2024–5.
[11] L.J. Kaplan, S.Z. Trooskin, T.A. Santora, Thoracic compartment syndrome, J. Trauma 40 (2) (1996) 291–293.
[12] D.J. Vargo, F.D. Battistella, Abbreviated thoracotomy and temporary chest closure: an application of damage control after thoracic trauma, Arch. Surg. 136 (1) (2001) 21–24.
[13] J.L. Lang, et al., Does temporary chest wall closure with or without chest packing improve survival for trauma patients in shock after emergent thoracotomy? J. Trauma 70 (3) (2011) 705–709.