Definition, Pathomechanism of the Thoracic Compartment Syndrome

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4.1 Introduction

Compartment syndrome can occur in any anatomical compartment when intracompartmental pressure exceeds the perfusion pressure of the tissue or organ within that compartment [1]. The more common abdominal compartment syndrome is extensively described, and various principles of management are well established. Abdominal compartment syndrome manifests clinically with increased peak airway pressures, hypotension due to decreased venous return to the heart via a compressed inferior vena cava, increased bladder pressure, and renal impairment. Thoracic compartment syndrome (TCS) is simply this same pathophysiology in the mediastinum, pericardium, or pleural space(s). TCS can result from massive resuscitation or secondary to injuries such as tension pneumothorax or pericardial tamponade.

Tension pneumothorax clearly illustrates the basic pathophysiology underlying TCS: increased intrathoracic pressure compromises venous inflow into the right heart, subsequently decreasing blood flow through the pulmonary vasculature into the left heart, resulting in cardiopulmonary collapse.

TCS due to sternotomy or thoracotomy closure is among the most elusive of the TCSs, but when present has profound effects on clinical course and patient survival. While similar in terms of venous inflow compromise to abdominal compartment syndrome, TCS due to chest closure is a rare clinical entity, and its management is not well defined. This type of TCS is most prevalent in cardiothoracic and pediatric surgery, however still relatively rare, with approximately 200 cases reported in the

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adult cardiothoracic literature. The overall incidence of delayed sternal or thoracotomy closure, a primary risk factor for thoracic compartment syndrome, is estimated at 1.5–2.8% in the adult cardiothoracic surgery population [2].

In cardiothoracic surgery, TCS typically presents with increasing airway pressures and tamponade physiology on attempted chest closure. This phenomenon is not unique to non-traumatic cardiothoracic surgery and may develop in attempted chest closure for traumatic injury as well, particularly in cases where myocardial and pulmonary edema are present. Additionally, TCS may not develop at the time of operation, but can present hours to days postoperatively [2].

4.2 Definition of Thoracic Compartment Syndrome

Thoracic compartment syndrome (TCS) is defined as any process by which increased pressure in the mediastinum, pericardium, or pleural space(s) compromises tissue and organ perfusion within that space. Multiple spaces within the thorax may be affected simultaneously. It is similar to abdominal compartment syndrome (ACS) in that increased compartmental pressure compromises blood flow through a large venous conduit, i.e., the inferior vena cava is compressed in ACS, and both the inferior and superior vena cavae are compressed in TCS, leading in both cases to severely decreased blood flow.

In general, traumatic TCS is precipitated by tension pneumothorax, pericardial tamponade, or myocardial edema, illustrated in Fig. 4.1; the hemodynamic effects of these processes are quite similar. In fact, the hemodynamic changes seen in pericardial tamponade and in myocardial edema are essentially the same, but differ in etiology and the thoracic compartments involved: in pericardial tamponade the pericardial compartment is primarily involved, while in myocardial edema there is typically more generalized elevation of the mediastinal, pericardial, and hemi-thoracic compartments. There are a few significant differences that delineate tension pneumothorax from pericardial tamponade and myocardial edema. One significant difference is that the pulmonary artery pressure is elevated in tension pneumothorax, while in

Hemodynamic parameter (normal values)	Heart rate (80 BPM)	Cardiac output (4–6 L/min)	Cardiac index (2.5–4 L/min)	Central venous and right atrial pressure (5–7 mmHg)	Systemic vascular resistance (800–1100)	Pulmonary artery (15 mmHg)	Left atrial wedge (7–10 mmHg)
Tension pneumothorax	↑	↓	↓	↑↑	↑	↑↑	↑↓
Pericardial tamponade	↑	↓	↓	↑↑	↑	↓	↑
Myocardial edema	↑	↓	↓	↑↑	↑	↑↑	↑

Fig. 4.1 Pathophysiologic alterations in thoracic compartment syndrome

pericardial tamponade and myocardial edema it is decreased. This occurs because in tamponade and myocardial edema the entire heart is externally compressed and the right ventricular ejection fraction is low, whereas in tension pneumothorax, compression of the lung induces marked pulmonary hypertension and compromises compliance of the pulmonary vasculature. A second difference is that in pericardial tamponade and myocardial edema, the left atrial wedge pressure is elevated, while in tension pneumothorax it can be elevated or decreased. This is due again to direct external compression of the heart seen in pericardial tamponade and myocardial edema, whereas in tension pneumothorax there is poor left heart filling due to decreased pulmonary vascular flow in addition to external compression.

4.3 Pathophysiology of TCS

In its simplest form, TCS is compressive compromise of venous return. The pathophysiology of tension pneumothorax arises primarily from this loss of venous inflow into the chest. In tension pneumothorax, increased hemi-thoracic pressure shifts the heart and mediastinum to the contralateral side, which compresses the inferior and superior vena cavae, compromising right atrial and ventricular filling and therefore end diastolic volumes. Collapse of the ipsilateral lung and compression of the contralateral lung increase pulmonary vascular resistance, impeding right ventricular outflow. This combination of pressure effects—inferior and superior vena cavae compression, increased pulmonary vascular resistance, and decreased right atrial and ventricular volumes—results in profoundly decreased right ventricular output. Compromised right cardiac output results in impaired pulmonary vascular filling and flow, which manifests in the left heart as markedly decreased left atrial filling volume. The increased intrathoracic pressure also adversely affects left atrial volume (Fig. 4.2) [3].

In pericardial tamponade (and in myocardial edema), venous return is also compromised by compression of the superior and inferior vena cavae, but in addition there is twofold compression of both ventricles and subsequent impaired myocardial perfusion. Vena cava compression significantly decreases right heart output by restricting blood volume flow into the right atrium. External compression of the right ventricle further limits the volume of blood that can be delivered into the pulmonary vasculature, which in turn decreases left atrial filling volumes and therefore left ventricular output, with concomitant external compression of both chambers by pericardial fluid. The resultant decreased cardiac output leads to decreased blood pressure, coronary perfusion, and myocardial ischemia [3].

Myocardial edema due to acute ventricular dilatation, often coupled with traumatic pulmonary edema, can cause elevated compartmental pressures in the pericardium, mediastinum, and involved hemithorax [4]. In this setting, attempts to close the chest can induce tamponade physiology. Pulmonary edema increases mediastinal pressure, and, coupled with increased cardiac size due to edematous ventricular dilatation causes what is essentially pericardial and mediastinal loss of domain. This was first described in 1975 by Rahi et al. as “tight mediastinum” following prolonged cardiothoracic surgeries [5]. Attempts to return the heart to the pericardium

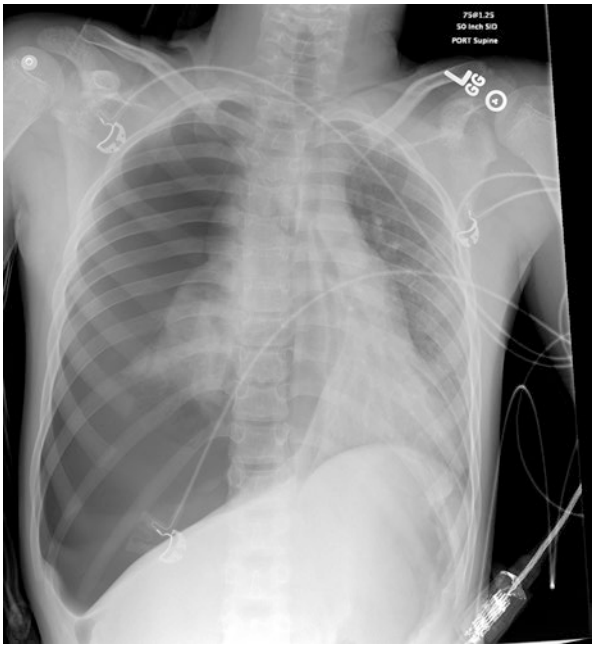

Fig. 4.2 Right-sided tension pneumothorax: The image demonstrates marked mediastinal shift away from the affected right hemithorax, including the superior vena cava (SVC), an inferior vena cava (IVC), total collapse of the ipsilateral lung, and compression of the contralateral lung

and to reduce the mediastinum cause decreased venous inflow from the superior and inferior vena cavae, which reduces diastolic volumes, compromising cardiac output, and precipitating hemodynamic collapse [4, 6].

The primary causes of prolonged open thoracotomy or sternotomy in cardiothoracic surgery are coagulopathy and uncontrolled bleeding, followed by cardiomegaly, decreased pulmonary compliance, and edema [4]. These processes are certainly present in chest injury, and case reports of traumatic TCS are consistent with the major causes of TCS described in the cardiothoracic literature [4, 6]. An additional factor seen in trauma that contributes to TCS and failure to close the chest is the massive resuscitation typically required in severe chest injury. The relative paucity of traumatic TCS case reports attests to the injury severity sustained by these patients, with their high mortality likely precluding even an attempt at delayed thoracic closure [4].

While management of traumatic TCS is evolving, several principles of abdominal compartment syndrome management do generalize to TCS. Stabilization and improvement of cardiac output, normalizing cardiac filling pressures and improving lung function prior to an attempt at thoracic closure are critical, as well as stabilizing other failing organ systems. Once this has been achieved, minimizing edema and volume overload in the massively resuscitated patient with aggressive diuresis or renal replacement therapy is critical [2]. Detailed descriptions of the technical aspects of thoracic closure in the face of TCS are beyond the scope of this chapter, but commonly used methods are skin flap closure over the sternotomy without fixation and synthetic prosthesis interposition if TCS prevents re-approximation of the sternum or thoracotomy [2].

4.4 Conclusion

TCS in trauma is a life-threatening process with a spectrum of etiologies, including tension pneumothorax, pericardial tamponade, myocardial, and pulmonary edema. Compression of venous return is the essential pathophysiology underlying all of the TCS causes listed above. TCS in trauma is associated with high injury severity and overall mortality. Current management involves emergent tube thoracostomy for tension pneumothorax, pericardial decompression via pericardiotomy, and thoracic skin closure without fixation of the sternum with or without prosthetic for tamponade on attempted chest closure, induced by myocardial and pulmonary edema. Ultimately, as with all compartment syndromes, TCS will persist until the underlying pathophysiology is treated.

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