Obstructive Shock

4

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4.1 Introduction

4.1.1 Definition of Obstructive Shock

Obstructive shock is a form of shock associated with mechanical obstruction of blood flow to the heart, specifically left ventricle (Fig. 4.1) [1].

The most distinctive feature of obstructive shock is that detecting the cause of obstruction is essential for the proper management and the response to the treatment is very immediate. This section deals with following three conditions of obstructive shock: tension pneumothorax, pulmonary thromboembolism, and cardiac tamponade.

4.1.2 Common Pathophysiology of Obstructive Shock

The common pathophysiology of obstructive shock is a reduction in the left ventricular (LV) preload. Increased intrathoracic pressure impairs venous return in tension pneumothorax. Increased right ventricular (RV) afterload impairs blood flow from right to left heart in pulmonary embolism. Decreased cardiac compliance impairs diastolic filling of heart in cardiac tamponade. Decreased LV preload leads to the relative increase in both LV contractility and heart rate, but eventually the stroke volume and cardiac output (CO) decrease. Because of the obstruction of the blood flow, distended jugular vein and inferior vena cava (IVC) can be commonly observed in physical examinations or bedside sonography.

4.1.3 Initial Approach and Diagnosis of Obstructive Shock

Except for hypotension, the most common symptoms and signs of obstructive shock are dyspnea and jugular venous distension (Fig. 4.2). Also, venous congestion due to obstruction can be visualized by bedside sonography as distended hepatic inferior vena cava (IVC) without significant inspiratory collapse (Fig. 4.2).

Careful medical history and physical examination in unstable patients provide a valuable information to distinguish the cause of the shock. In addition, bedside ultrasonography is one of the most useful tools to identify causes of shock. The presence, location, and volume of pneumothorax can be diagnosed by the ultrasonography and RV strain on the ultrasonography can be a very important finding in suspicion of massive pulmonary embolism. In particular, the ultrasonography is the diagnostic tool of choice in pericardial tamponade and it can also guide the pericardiocentesis.

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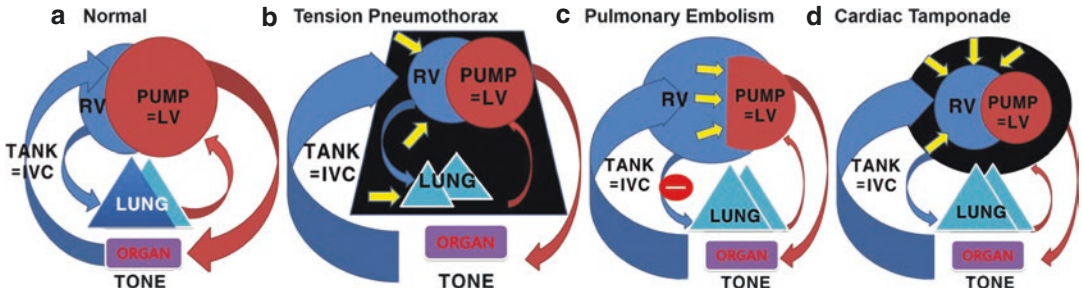

Fig. 4.1 Three types of obstructive shock. (**a**) Diagram describing the normal circulation. (**b**) Tension pneumothorax (Black) alters venous return, which results in decreased cardiac output and venous congestion. (**c**) Pulmonary embolism (Red prohibition sign) blocks the blood flow from right heart to left heart, which results in distension of right ventricle and diastolic septal flatening. (**d**) Cardiac tamponade (Black) compresses cardiac chambers, which results in decreased cardiac output and venous congestion. *RV* right ventricle, *LV* left ventricle, *IVC* inferior vena cava

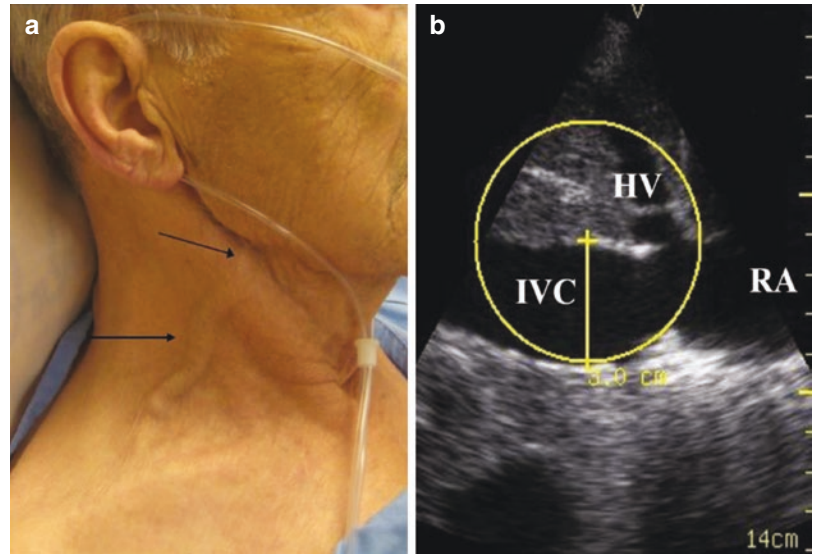

Fig. 4.2 (**a**) Jugular venous distension and (**b**) distended hepatic inferior vena cava. *IVC* inferior vena cava, *HV* hepatic vein, *RA* right atrium

4.1.4 General Management of Obstructive Shock

Airway management is required in patients with decreased mentality or with profound shock. As described above, most patients with obstructive shock also suffer from hypoxemia. Therefore, supplement oxygen should be delivered in patients with hypoxemia. Ventilatory support may be required in respiratory-distressed patients. Volume resuscitation is the first step for the circulatory support. In patients with obstructive shock, venous system is similar to those with volume overload. Jugular vein and IVC are distended and central venous pressure and pulmonary artery occlusion pressure are elevated. However, their cardiac output still can respond to volume resuscitation because cardiac filling pressure is increased in obstructive shock. Of course, immediate identification and management of the obstructive lesion are essential for the outcome. If blood pressure is not recovered rapidly, vasopressors can be used empirically.

4.2 Tension Pneumothorax

4.2.1 Definition of Tension Pneumothorax

A tension pneumothorax is considered to be present when a pneumothorax leads to significant hemodynamic compromise [2].

4.2.2 Epidemiology of Tension Pneumothorax

The incidence of tension pneumothorax varies widely among the studies including trauma patients, performed in emergency departments, or in intensive care units [2]. Tension pneumothorax was diagnosed in 5.4% of major trauma patients in one study [3].

4.2.3 Pathophysiology of Tension Pneumothorax

Causes of tension pneumothorax include trauma, obstructive lung diseases (asthma, chronic obstructive pulmonary diseases), and excessive positive pressure ventilation. Once the lungs are injured leaked air is accumulated in the pleural cavity and cannot escape. Accumulated air increases intrathoracic pressure and finally obstructs venous return to the heart, which results in the obstructive shock. Ventilation-perfusion mismatch and decreased vital capacity due to collapsed lung contribute to hypoxemia and respiratory distress [2].

4.2.4 Initial Approach and Diagnosis of Tension Pneumothorax

Most patients with pneumothorax have acute pleuritic chest pain and increasing volume of pneumothorax causes dyspnea, hypoxemia, tachycardia, and hypotension. Common physical findings are tracheal deviation toward the contralateral side of tension pneumothorax, hyperresonance and diminished lung sounds on the affected side, subcutaneous emphysema, and neck vein engorgement. Persistent shock may result in the bradycardia and pulseless electrical activity arrest.

Diagnostic modalities for pneumothorax are chest radiography (Fig. 4.3), chest CT, and ultrasonography (Fig. 4.4). However, confirmative evaluation should not delay treatment for unstable patients suspected of tension pneumothorax.

4.2.5 Management of Tension Pneumothorax

The definite treatment of tension pneumothorax is the decompression of accumulated air in the pleural space. Immediate needle decompression is the treatment of choice in the emergent situation. A 14-gauge needle or larger should be placed over the superior margin of the third rib in the midclavicular line (Fig. 4.5). A rush of air with clinical improvement of vital signs confirms the diagnosis. If there is no immediate improvement, do not hesitate to place a second needle in the next interspace. The chest tube should be placed subsequently.

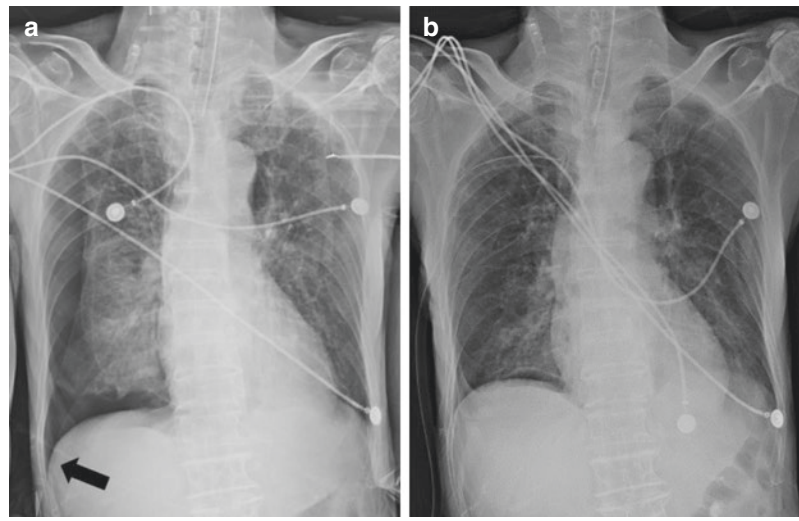

Fig. 4.3 (**a**) Pneumothorax in the right pleural cavity with deep sulcus sign (arrow). (**b**) Pneumothorax disappeared after tube thoracotomy

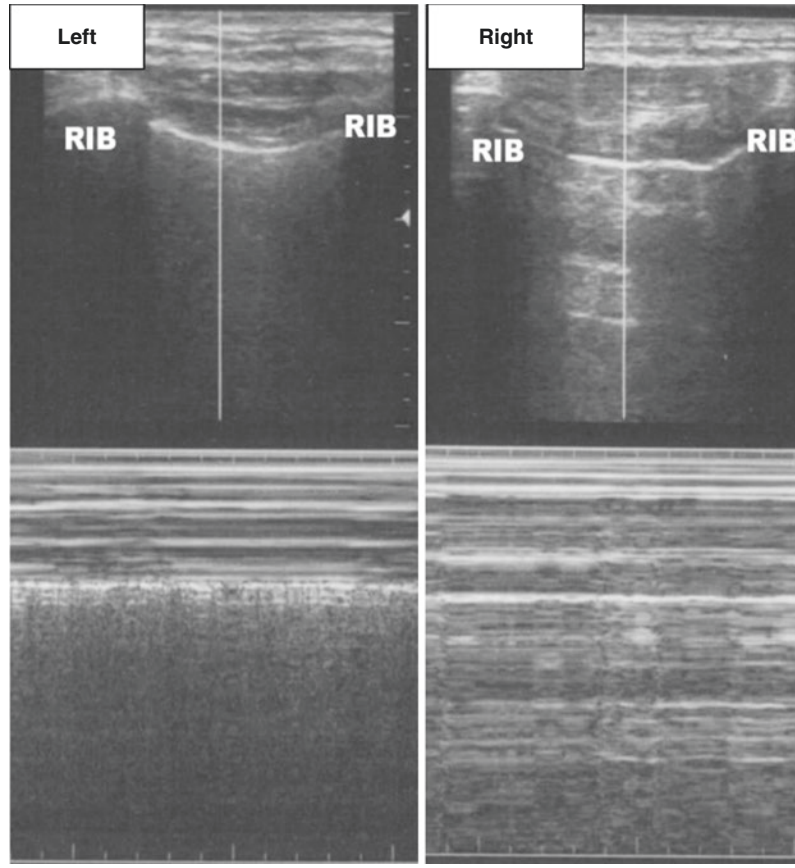

Fig. 4.4 M-mode image of bilateral lung. Left image shows normal seashore sign and right image shows stratosphere (bar code) sign indicating the presence of pneumothorax

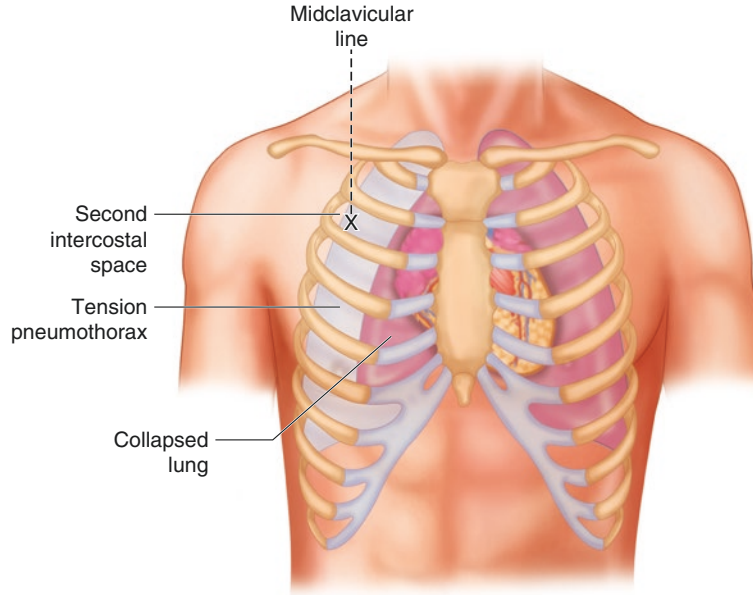

Fig. 4.5 Illustration of needle thoracotomy

4.3 Pulmonary Thromboembolism

4.3.1 Definition of Pulmonary Thromboembolism

Pulmonary thromboembolism is defined as a blockage of a pulmonary artery by the thrombus traveled mainly from veins of lower extremities [4]. Therefore, the term venous thromboembolism is used for both deep vein thrombosis and pulmonary thromboembolism.

4.3.2 Epidemiology of Pulmonary Thromboembolism

Venous thromboembolism occurs about ten million cases very year and this burden accounts for the third leading vascular disease following acute myocardial infarction and stroke [5].

4.3.3 Pathophysiology of Pulmonary Thromboembolism

Most common cause of pulmonary embolism is the thrombus from the deep veins of the lower extremities. Therefore, deep vein thrombosis and pulmonary thromboembolism compose of single disease entity, venous thromboembolism. Acute embolism of pulmonary artery leads to ventilation perfusion mismatch, which results in hypoxemia and dyspnea. If the thrombus obstructs a substantial portion of pulmonary artery, the increase in pulmonary vascular resistance impedes RV outflow and reduces LV preload and CO [4].

4.3.4 Initial Approach and Diagnosis of Pulmonary Thromboembolism

The symptoms and signs of pulmonary thromboembolism are usually nonspecific, making it difficult to diagnose. Therefore, pulmonary thromboembolism should be considered whenever unexplained symptoms including dyspnea, syncope, hypotension, and hypoxemia are present. D-dimer test has high negative predictive value to diagnose pulmonary thromboembolism and is widely used to exclude pulmonary thromboembolism. Because false-positive elevation of D-dimer is common in various conditions such as sepsis and malignancy, its positive predictive value is very low and further confirmatory diagnostic test is required. Wells' criteria are a clinical decision rule to provide the risk stratification for pulmonary embolism (Table 4.1) and pulmonary embolism rule-out criteria rule can be used (Table 4.2) [6, 7].

In patients with massive pulmonary thromboembolism and acute cor pulmonale, S1Q3T3 pattern can be observed in electrocardiography (ECG) (Fig. 4.6). However, this ECG changes are neither sensitive nor specific for pulmonary thromboembolism.

Table 4.1 Wells' score for pulmonary thromboembolism

Criteria	Points
Clinical signs and symptoms of deep vein thrombosis	3
Pulmonary embolism is most likely diagnosis	3
Tachycardia over 100 beats/min	1.5
Immobilization or surgery in previous 4 weeks	1.5
Prior diagnosis of deep vein thrombosis or pulmonary thromboembolism	1.5
Hemoptysis	1
Active malignancy	1

Low risk <2 points, intermediate risk 2–6 points, high risk >6 points
Pulmonary thromboembolism unlikely 0–4 points, likely >4 points

Table 4.2 Pulmonary embolism rule-out criteria rule (all nine factors must be present to exclude pulmonary embolism)

Clinical low probability (<15% probability of pulmonary embolism based on gestalt assessment)
Age <50 years
Pulse <100 beats/min during entire stay in ED
Pulse oximetry >94% at near sea level (>92% at altitudes near 5000 feet above sea level)
No hemoptysis
No prior venous thromboembolism history
No surgery or trauma requiring endotracheal or epidural anesthesia within the last 4 weeks

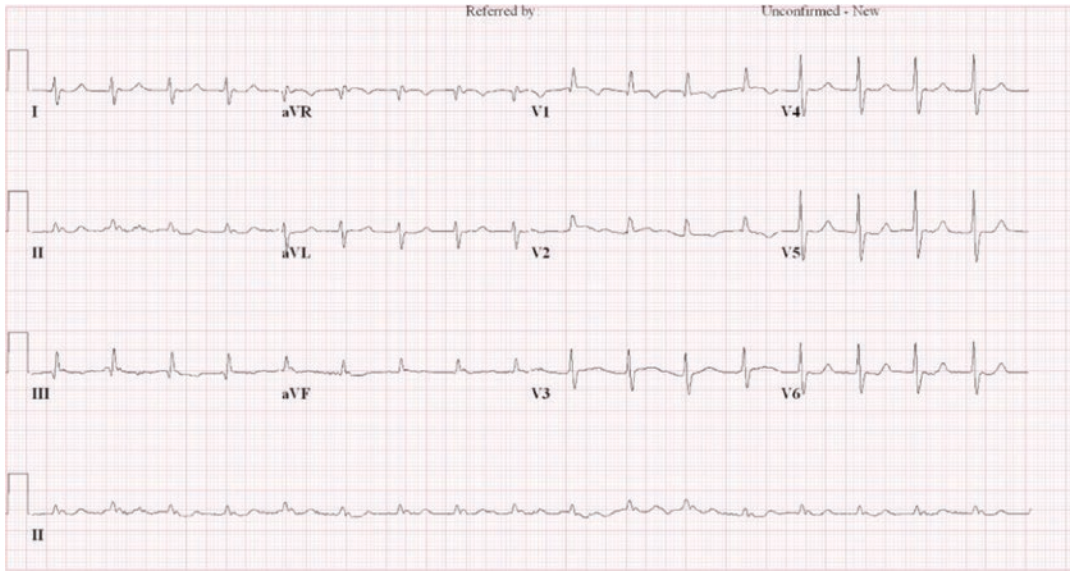

Fig. 4.6 Deep S wave in lead I, Q wave, and inverted T wave in lead III are classic findings of acute cor pulmonale

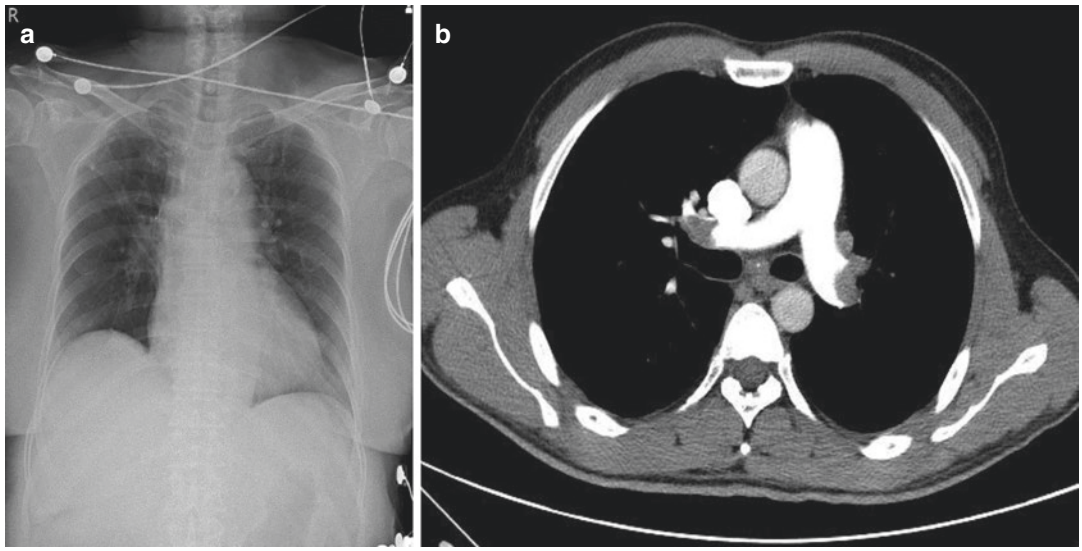

Fig. 4.7 (**a**) Chest radiography does not give any diagnostic information in pulmonary thromboembolism in most cases. (**b**) Bilateral main pulmonary arteries are occluded by thromboembolism in chest CT scan with contrast

In most cases, chest radiography does not give any diagnostic information in pulmonary thromboembolism (Fig. 4.7). For the confirmative diagnosis, chest CT scan with contrast or conventional angiography should be performed.

When pulmonary embolism is suspicious, the presence of deep vein thrombosis also should be excluded. Therefore, CT angiography protocol in many institutions includes the lower extremity venography by obtaining delayed phase of image.

Bedside ultrasonography can be helpful especially in patients with hemodynamic instability. Pulmonary thromboembolism can be directly visualized in pulmonary artery or right ventricle. Also, RV dilation and flattening of

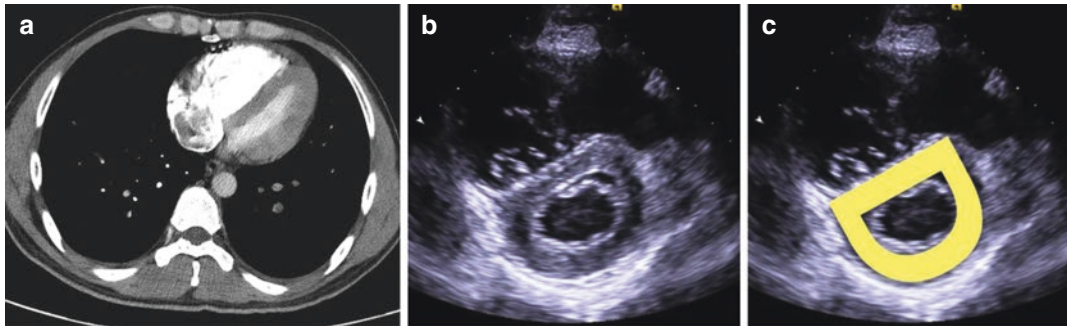

Fig. 4.8 (**a**) Chest CT shows RV dilation in massive pulmonary thromboembolism. Note that right ventricle is larger than left ventricle. (**b**, **c**) Typical D-shape left ventricle in massive pulmonary embolism is observed in parasternal short-axis view. Right ventricle is also larger than left ventricle indicating right ventricular dilation

interventricular septum (D-shape LV) can be seen especially in massive pulmonary thromboembolism with obstructive shock (Fig. 4.8). These findings are not sensitive enough to exclude pulmonary thromboembolism, but very specific to pulmonary thromboembolism if there is no history of chronic lung disease and pulmonary hypertension.

In addition, bedside ultrasound can detect proximal venous thrombosis using Doppler images and compression technique. Normal vein can be easily compressed by ultrasound probe. However, vein filled with thrombosis cannot be compressed by the probe.

Although CT angiography is more widely used nowadays, ventilation-perfusion lung scan can be helpful to diagnose the pulmonary embolism especially in patients with renal failure; in that circumstance the avoidance of radiocontrast dye is warranted.

4.3.5 Management of Pulmonary Thromboembolism

The primary treatment for pulmonary embolism is anticoagulant therapy, but since anticoagulant therapy does not lysate the thrombus immediately, thrombolytic therapy should be considered for severe cases of pulmonary embolism. The hemorrhagic complication rate of thrombolysis in pulmonary thromboembolism is higher than those in myocardial infarction and ischemic stroke because the burden of thrombosis is larger and consumptive coagulopathy is already present. Therefore, thrombolytic therapy is indicated in patients with high risk of death from pulmonary thromboembolism. In patients with life-threatening pulmonary embolism, thrombolysis should always be considered even in patients with relative contraindications. Meta-analysis which has evaluated the role of thrombolysis in pulmonary embolism showed that thrombolysis improves the all-cause mortality (1.39% vs. 2.92%, p value = 0.03) in intermediate-risk patients who were hemodynamically stable with RV dysfunction [8]. However, the rate of major bleeding events was significantly higher in thrombolysis group (7.74% vs. 2.25%, p value <0.001) in that study. One randomized clinical trial reported that thrombolysis could prevent hemodynamic decompensation but increased the risk of major hemorrhage and stroke in patients with intermediate-risk pulmonary embolism [9]. Because the hemorrhagic complication especially intracranial hemorrhage following thrombolysis therapy is usually catastrophic, the clinical benefit of thrombolysis in patients without hemodynamic instability is still controversial.

Two indications of thrombolysis in current guideline are acute massive pulmonary thromboembolism (high-risk pulmonary embolism, systolic blood pressure <90 mmHg, or a decrease in systolic arterial pressure of at least 40 mmHg for at least 15 min) and acute

sub-massive pulmonary thromboembolism (intermediate-high risk, normal blood pressure with RV dysfunction) [10]. Thrombolysis regimen is the intravenous infusion of 100 mg of tissue-type plasminogen activator (tPA) during 2 h in the absence of contraindications. In cases who cannot tolerate anticoagulation therapy, inferior vena cava filter can be used to prevent further occlusion of pulmonary artery. Lastly, venoarterial extracorporeal membrane oxygenator (VA ECMO) can be considered in profound obstructive shock or cardiac arrest from pulmonary thromboembolism.

4.4 Cardiac Tamponade

4.4.1 Definition of Cardiac Tamponade

Cardiac tamponade is defined as an acute circulatory failure due to the compression of the cardiac chambers by the pericardial effusion.

4.4.2 Epidemiology of Cardiac Tamponade

The incidence of cardiac tamponade is not well documented because most epidemiological studies have examined patients suffering from a pericardial effusion without focusing on tamponade [11].

4.4.3 Pathophysiology of Cardiac Tamponade

Causes of cardiac tamponade include metastatic malignancy, pericarditis, uremia, and tuberculosis. As fluid accumulates within the pericardial sac, intrapericardial pressure increases. There are relatively small changes in the intrapericardial pressure in the early stage because of the distensibility of parietal pericardium. However, if the fluid continues to accumulate beyond the limits, the intrapericardial pressure increases rapidly. If the intrapericardial pressure rises over the normal RV filling pressure, ventricular filling is restricted and results in the decrease in the RV end-diastolic volume. The decreased RV end-diastolic volume compromises CO which results in obstructive shock.

4.4.4 Initial Approach and Diagnosis of Cardiac Tamponade

Early symptoms include dyspnea at rest and with exertion. Common physical findings are tachycardia, narrow pulse pressure (reflecting decreased stroke volume), and neck vein engorgement. Inspiratory decrease in systolic blood pressure may be observed and this is called pulsus paradoxus. The classic Beck triad includes jugular venous distension, hypotension, and muffled hear sounds.

Chest radiography may reveal an enlarged heart silhouette and epicardial fat-pad sign (Fig. 4.9).

ECG usually shows low-voltage QRS complexes and sometimes electrical alternans (beat-to-beat variation in the amplitude) (Fig. 4.10).

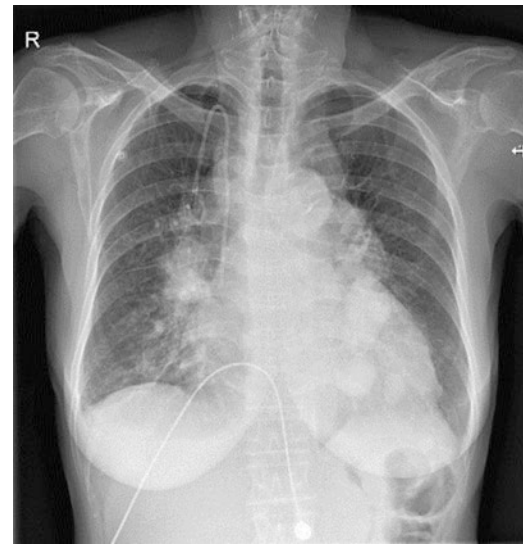

Fig. 4.9 Chest radiography of patient with malignant pericardial effusion

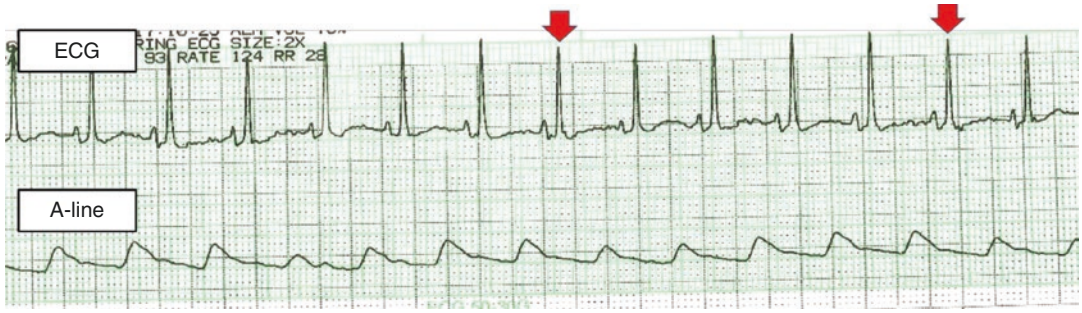

Fig. 4.10 Electrical alternans (beat-to-beat variation amplitude) is observed in patients with pericardial tamponade. Note that arterial wave also shows variation of size

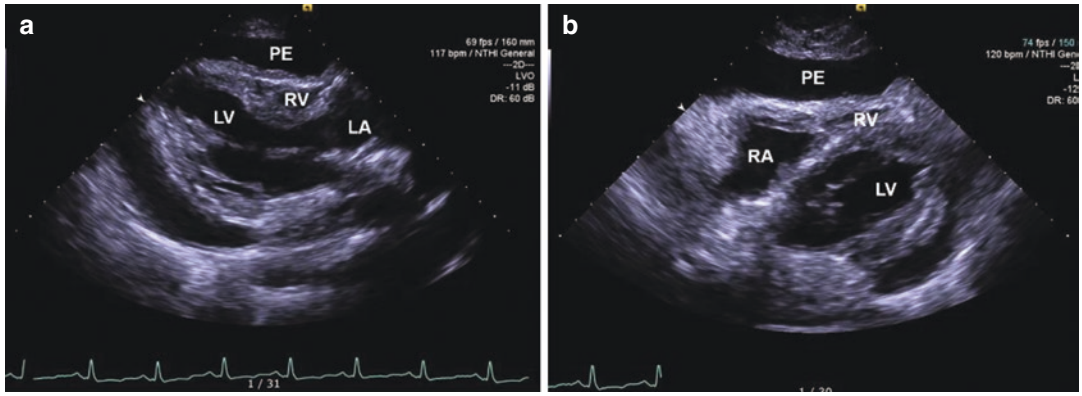

Fig. 4.11 Diastolic right ventricular collapse can be observed (**a**) in parasternal long-axis view and (**b**) in subxiphoid four-chamber view. *PE* pericardial effusion, *RV* right ventricle, *LV* left ventricle, *LA* left atrium

Echocardiography is the diagnostic test of choice. In addition to a large pericardial fluid volume, typical echocardiographic findings described in cardiac tamponade are right atrial compression, RV diastolic collapse, and dilated IVC with lack of inspiratory collapse (Fig. 4.11).

4.4.5 Management of Cardiac Tamponade

The primary treatment for cardiac tamponade is pericardiocentesis. If cardiac tamponade is suspected and the patient is not in cardiac arrest, expert consultation should take place. If cardiac arrest is ongoing or impending and cardiac tamponade is suspected, emergency pericardiocentesis should be performed. Pericardiocentesis is optimally performed using echocardiographic guidance to avoid cardiac perforation and coronary artery laceration (Fig. 4.12).

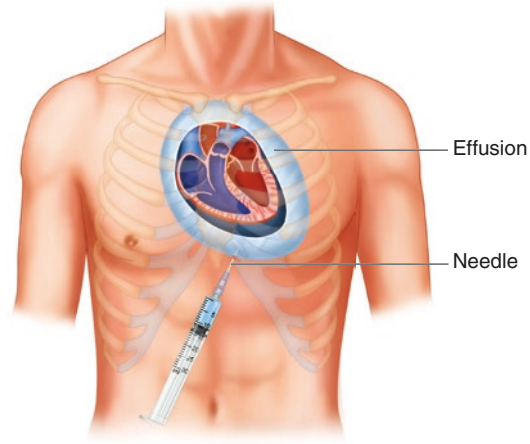

Fig. 4.12 Emergent pericardiocentesis for pericardial tamponade

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