Shock with Jugular Venous Distention

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Shock is a frequently encountered, life-threatening clinical syndrome that can be readily diagnosed at the bedside by observing hypotension and signs and symptoms of hypoperfusion (eg, mental status changes, weakness, cyanosis, dyspnea, and oliguria). There is a wide range of causes of shock, but few presentations include jugular venous distention (JVD). The common pathophysiology in the differential diagnosis of shock with JVD (**Table**) is inhibition of right ventricular outflow and/or right ventricular filling. This process translates into decreased right atrial emptying, as manifested by JVD. A systematic approach to the presentation of shock with JVD can prevent unhelpful diagnostic testing and loss of valuable time (**Figure 1**). An approach that includes a thorough history and physical examination, with echocardiography as an essential diagnostic modality, can differentiate among most underlying problems and help direct an appropriate management plan (**Figure 2**). This article provides an overview of conditions associated with the presentation of shock and JVD, with a focus on important features of the diagnosis and management of these conditions.

TAKE HOME POINTS

- Examining the neck for jugular venous distention (JVD) is critical for the evaluation of all patients who present with shock because, while shock has a broad differential diagnosis, there are relatively few presentations that include JVD.
- Echocardiography can be helpful in differentiating among specific etiologies for shock with JVD.
- In postoperative cardiothoracic surgery patients, shock with JVD should trigger a differential diagnosis that includes pericardial tamponade, pericardial clot, tension pneumothorax, and cardiac herniation.
- All acute inferior wall myocardial infarctions—especially those with hypotension and JVD—should be considered as involving the right ventricle and should prompt additional electrocardiograms with right-sided precordial leads (looking for ST elevation in leads V_3r and V_4r).

END-STAGE CONGESTIVE HEART FAILURE

The most common etiologies of heart failure include coronary artery disease, hypertension, valvular heart disease, cardiomyopathy, myocarditis, and congenital heart disease. Heart failure begins with impaired ventricular function that results in decreased cardiac output. Reduced output, in turn, activates neuroendocrine systems (renin-angiotensin system, arginine-vasopressin system, sympathetic nervous system) that produce systemic vasoconstriction, which results in increased afterload and further decreases cardiac performance. Decreasing cardiac output presents as shock, and progressive fluid retention due to neurohormonal activation causes an increase in right-sided pressure and JVD.[1]

Diagnosis

The presentation and hemodynamics of end-stage heart failure with systolic dysfunction are generally uniform, regardless of the initial pathology. Patients present with dyspnea, hypotension, pulmonary congestion, ventricular gallop, Cheyne-Stokes breathing, elevated jugular venous pressure, and peripheral vascular congestion. Chest radiography may confirm pulmonary vascular and alveolar congestion with findings of Kerley's B lines and prominence of pulmonary vessels, along with evidence of cardiomegaly with or without pleural effusion. Echocardiography is an excellent initial tool for evaluating these patients for structural and functional abnormalities. Measurement of brain natriuretic peptide levels has become an established diagnostic test for evaluating patients with suspected heart failure.[2]

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Table. Differential Diagnosis of Shock with Jugular Venous Distension

End-stage heart failure	Massive pulmonary embolism
Acute myocardial infarction	Right atrial or ventricular thrombus
Cardiac tamponade	Cardiac tumors
Pericardial thrombus	Cardiac herniation
Constrictive pericarditis	
Tension pneumothorax	

Treatment

Patients in advanced heart failure with New York Heart Association class III and IV symptoms should be treated with angiotensin-converting enzyme inhibitors, diuretics, aldosterone antagonists, and β-blockers. Digoxin continues to be helpful in some of these patients. When patients present with end-stage heart failure and are hypotensive, management is quite different. Parenteral inotropic agents may be initiated. However, inotropic agents, such as dopamine and dobutamine, may exacerbate myocardial ischemia by augmenting inotropy and increasing heart rate (all major determinants of myocardial oxygen consumption). Amrinone and milrinone are often reserved for use when other agents have proven ineffective or when patients have significant evidence of β-adrenergic blockade, but these phosphodiesterase inhibitors may induce hypotension or arrhythmias. Vasodilators should be used with extreme caution in the acute decompensated setting. The same may be said for initiation of β-blockers in the setting of initial presentation with severe volume overload and hypotension (so-called "wet-cold" pathophysiology). Nitroprusside can decrease filling pressures and can increase stroke volume by reducing afterload, but its use should be monitored closely because it can exacerbate hypotension. Intraaortic balloon pumping reduces systolic afterload and augments diastolic perfusion pressure, thus increasing cardiac output and improving coronary blood flow. It also is a useful bridging intervention that allows therapeutic measures, such as coronary revascularization or valvular surgery, to be undertaken.[1,3]

RIGHT VENTRICULAR MYOCARDIAL INFARCTION

Myocardial infarction (MI) is a major cause of cardiogenic shock. Related complications, such as acute mitral regurgitation, rupture of interventricular septum, and rupture of ventricular free wall, can produce severe hypotension. The present discussion mainly pertains to right-sided MI because of this condition's association with both shock and JVD. The principle cause of right ventricular MI is proximal occlusion of a dominant right coronary artery.[4]

Patients with right ventricular MI usually present with a triad of hypotension, elevated jugular venous pressure, and clear lung sounds. These patients may also complain of chest pain, nausea, dizziness, tachypnea, and tachycardia. Acute right-sided MI leads to decreased right ventricular compliance, reduced stroke volume, reduced preload for the left ventricle, and, as a result, reduced cardiac output. At the same time, acute right ventricular dilation may occur along with segmental wall motion abnormalities.[5]

Diagnostic Modalities

Clinical recognition of right-sided MI begins with obtaining an electrocardiogram (ECG) and observing ST-segment elevation in leads II, III, and aVF, with or without accompanying abnormal Q waves. All patients with acute inferior wall MI should have an initial ECG performed with the chest leads placed on the right chest in mirror image fashion to those normally placed on the left. ST-segment elevation in leads V_3r and V_4r is indicative of right ventricular MI.[6] If right coronary artery occlusion is proximal, it can produce associated findings such as PR-segment displacement, an indication of right atrial infarction, with or without symptomatic bradycardia, atrioventricular (AV) nodal block, or atrial fibrillation.[4] Echocardiographic evaluation usually reveals inferior wall motion abnormalities along with a dilated and akinetic right ventricle.

Treatment

The initial medical treatment of isolated anterior or inferior MI with borderline or stable hemodynamics (beyond acute revascularization or in noncandidates) might include anticoagulation, antiplatelet agents, diuresis, afterload reduction, and preload reduction using intravenous nitrates, morphine, β-blockers, and angiotensin-converting enzyme inhibitors.[7] In contrast, the initial treatment of inferior MI with hypotension mandates volume resuscitation. In this particular instance, preload reducers (eg, nitrates, vasodilators) should be avoided. Intravenous inotropes are recommended if volume infusion is unsuccessful in improving cardiac output.[4,8] Opening of the occluded coronary artery is crucial. Early reperfusion with thrombolytics/angioplasty/stenting/coronary artery bypass grafting leads to prompt hemodynamic improvement and subsequent recovery of right ventricle free wall contractility. Because it is also important to maintain AV synchrony, AV pacing for symptomatic bradycardia or advanced AV block may be indicated.

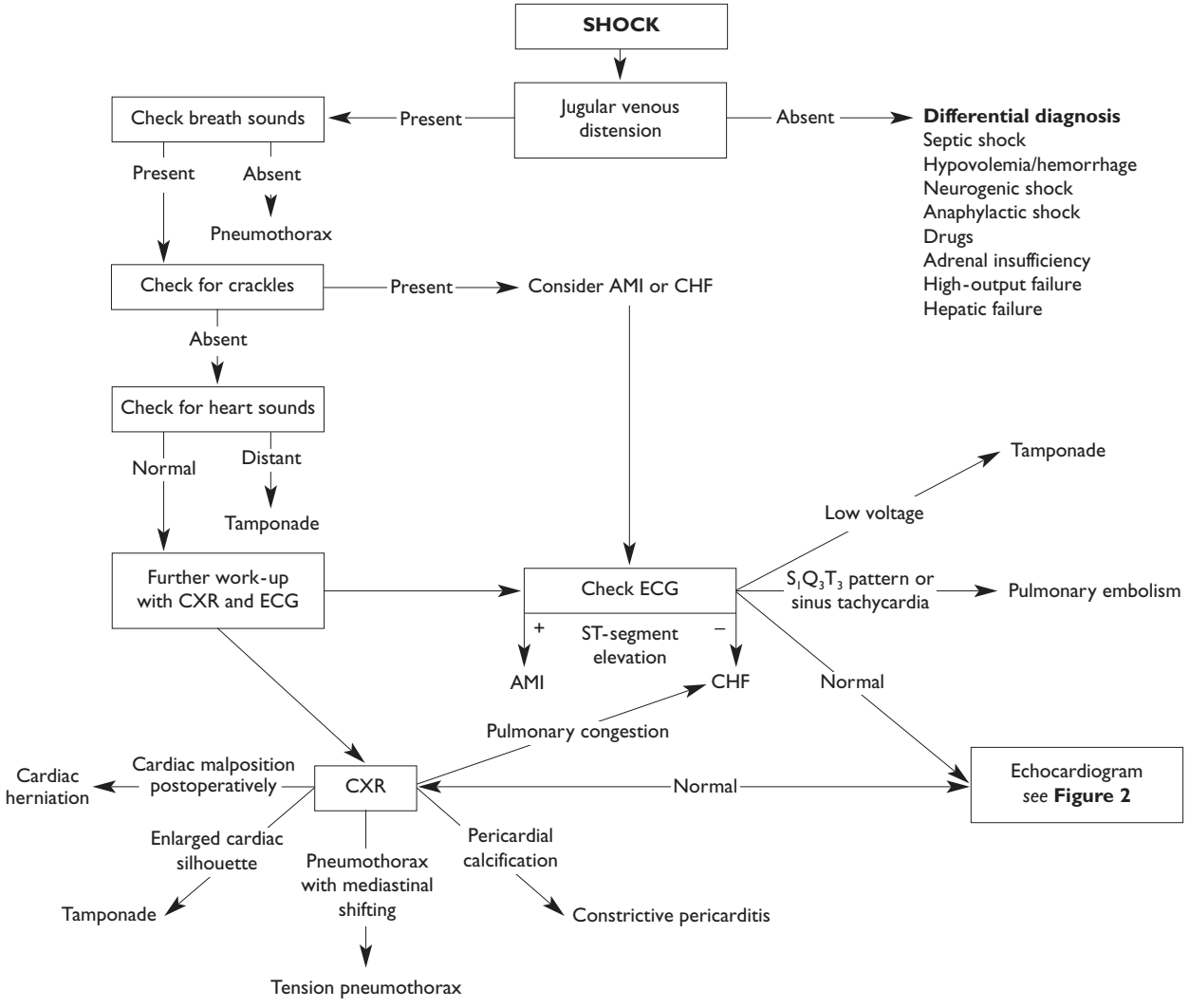

Figure 1. Algorithm for the diagnosis of shock. AMI = acute myocardial infarction; CHF = congestive heart failure; CXR = chest radiograph; ECG = electrocardiogram.

CARDIAC TAMPONADE

Tamponade is best thought of as a spectrum of pathophysiologic states rather than an "all-or-nothing" phenomenon. Tamponade can present with as little as 50 mL of fluid in the pericardial space or as much as 1 L or more of fluid, depending on the amount of time over which the fluid collects. Tamponade can be idiopathic or can be caused by various different underlying causes such as malignancy, trauma, thoracic surgery, radiation, drugs (eg, cyclosporine), uremia, connective tissue disease, infection, or complications of percutaneous coronary intervention. Early symptoms may resemble those of heart failure, including dyspnea and orthopnea. On physical examination, pulsus paradoxus, JVD with rapid x descent, and distant heart sounds can be appreciated.[9,10]

Pathophysiology

Physiologically, pericardial volume rises, causing a reduction in cardiac chamber volumes and a fall in diastolic compliance in all chambers. These events translate to restriction of cardiac inflow and outflow. Continued increases in pericardial pressure augment diastolic filling pressures to the point of decompensation. The hallmark of pure cardiac tamponade is progressive reduction of diastolic compliance with elevation and equalization of

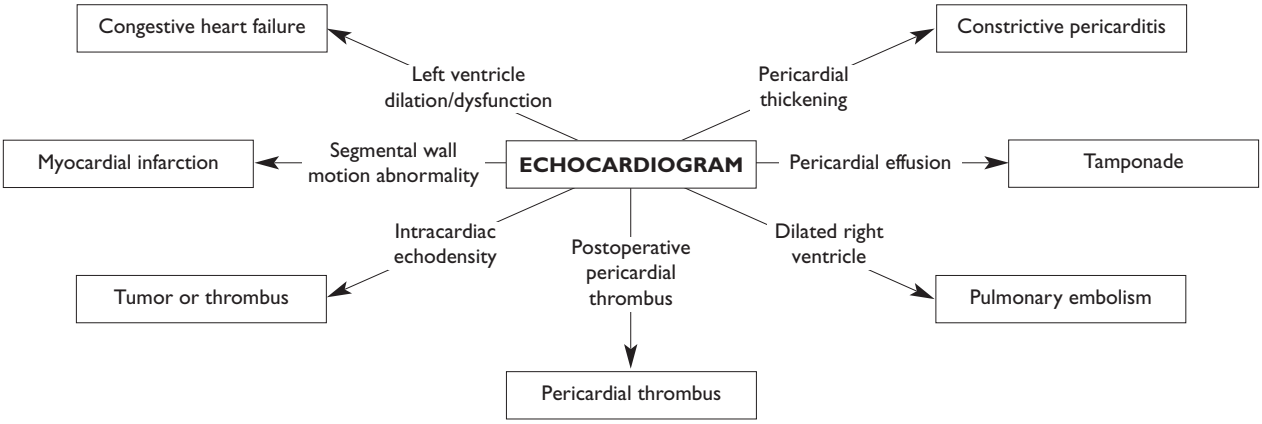

Figure 2. Algorithm depicting the utility of echocardiography in diagnosing the underlying cause in patients who present with shock and jugular venous distention.

the mean diastolic pressures in all 4 chambers. At this point, patients nearly always demonstrate pulsus paradoxus on physical examination.[9–12]

Diagnostic Modalities

The ECG in cardiac tamponade can reveal low voltage electrical alternans and changes characteristic of pericarditis. Without intervention, pulseless electrical activity may ensue. The echocardiogram may reveal decreased stroke volume with collapse of the right ventricular and right atrial free walls in late diastole. Collapse of chambers usually indicates decreasing cardiac output and impending tamponade.[12,13] In the presence of tamponade, cardiac Doppler ultrasonography reveals wide swings in flow across the tricuspid and mitral valves throughout the respiratory cycle. These flow variations are the central circulatory equivalent of pulsus paradoxus.

Treatment

Pericardial drainage is the treatment of choice when available, but medical supportive care, especially volume expansion, may be applied when pericardiocentesis is not immediately available. Temporary use of inotropic agents is of minimal benefit because the problem is primarily one of diastolic function, not systolic function. ECG- and/or echocardiographic-guided pericardiocentesis can be approached via a subxiphoid or apical route. The underlying cause should be treated to prevent recurrence of pericardial effusion. Subxiphoid pericardial window is the procedure of choice for malignant pericardial effusion or recurrent tamponade physiology. Prognosis depends on the underlying etiology.[13]

PERICARDIAL THROMBUS

Pericardial clot should always be considered in patients who are hypotensive after cardiac surgery, blunt trauma, or pericardiocentesis or who have a history of bleeding disorder.[14–16] Pericardial thrombus presents similarly to cardiac tamponade but may be indolent and can present up to 10 days postsurgery. Patients are usually tachycardic and hypotensive, with muffled heart sounds and significant JVD. Pulsus paradoxus may or may not be present. Although symptoms are vague, it is important to note the history and have a high clinical suspicion in postoperative or post-traumatic patients.[15]

Restriction of the heart by a localized mass in the pericardial space may cause atypical findings of compression of selected chambers by localized augmentation of intracardiac pressure, as in tamponade. Pericardial thrombus may act as an elastic form of constrictive pericarditis. A sharp early dip in right ventricular diastolic pressure is followed by a rapid early increase in diastolic pressure abruptly ending in a plateau phase ("square-root" sign).[14] Compression of any or all chambers has been noted. Tamponade after cardiac surgery is frequently due to pericardial effusion or pericardial clots, often located posteriorly. Transthoracic echocardiography (TTE) remains the initial diagnostic tool, but clots can very often be difficult to detect with TTE due to surgical wound dressings and mechanical ventilation.[14,15] Therefore, transesophageal echocardiography (TEE) may provide a better approach.[17]

Treatment

Treatment options in patients who have pericardial

thrombosis and who have any evidence of hemodynamic deterioration are confined to surgical exploration. Removal of the compressing clot results in rapid hemodynamic and symptomatic improvement.[14–18]

CONSTRICTIVE PERICARDITIS

Constrictive pericarditis is an uncommon disorder characterized by impaired diastolic filling, disassociated ventricular function, and systemic venous congestion. Most cases occur after cardiovascular surgery, radiation therapy, or tuberculosis infection. Usually patients present with orthopnea, venous congestion, cachexia, and dyspnea, but advanced cases can present with hypotension if cardiac output has deteriorated significantly due to ventricular underfilling. Physical examination demonstrates JVD along with other signs of central and peripheral congestion, including ascites and edema. Jugular venous pulsation is characterized by rapid x and y descent along with Kussmaul's sign (paradoxical increase of JVD with inspiration). Pericardial knock on cardiac auscultation is a pathognomonic sign of constrictive pericarditis.[19]

The pathophysiologic hallmark of constrictive pericarditis is loss of pericardial compliance. The pericardium is thickened, encasing the heart. This process leads to disassociation of intrathoracic and intracardiac pressures with respiration. Elevated end-diastolic right ventricular pressures restrict filling. Right ventricular pressure measurements disclose "dip and plateau" (square-root sign), indicating rapid early diastolic filling followed by lack of additional filling. There is also discordance between right ventricular and peak left ventricular systolic pressures during inspiration.[20]

Diagnostic Imaging

Echocardiogram is the diagnostic modality of choice for constrictive pericarditis. Pericardial thickening greater than 5 mm is usually indicative of constrictive pericarditis. Rapid early ventricular filling is represented on echocardiogram as a tall E wave on the mitral inflow Doppler signal. In addition, inflow velocities fall as much as 25% to 40%, and conversely, tricuspid velocity increases in inspiration. Abrupt intraventricular "septal bounce" in early diastole also indicates rapid early filling. The chest radiograph also has additional diagnostic value if pericardial calcification is present.

Treatment

Unrecognized cases of constrictive pericarditis can advance until patients have signs of hypoperfusion due to low cardiac output. Intravenous fluids can temporarily stabilize these patients. In contrast, stable patients respond well to β-blockers and diuretics for relief of symptoms of systemic congestion. Surgery is the most effective treatment choice with either partial or complete pericardial stripping.

TENSION PNEUMOTHORAX

Tension pneumothorax is a life-threatening condition that develops due to injury resulting in air leak into but not out of the pleural space, usually from some type of one-way valve mechanism. During inspiration, air escapes into pleural space, but during expiration, the communication between lung and pleural space may become occluded. Tension pneumothorax can develop following barotrauma that occurs during mechanical ventilation or during resuscitation, trauma, invasive procedures, or lung resection or can be caused by spontaneous pneumothorax, tumors, and infection. In tension pneumothorax, the pleural space usually contains 1.5 L to 2 L of air. Tension pneumothorax is heralded by a sudden deterioration in cardiopulmonary status caused by markedly increased intrapleural pressure that leads to displacement of mediastinum, depressed diaphragm, and compression of the heart and lungs.[21,22]

Patients may initially complain only of chest pain, and as their condition worsens, dyspnea and cyanosis may become apparent. On physical examination, patients have JVD, hypotension, and hyperresonant percussion of lungs over the affected hemithorax. Most notably, the trachea and heart are often shifted to the unaffected side.

Diagnostic Modalities

Chest radiography has a high diagnostic yield and is the diagnostic tool of choice in most cases of tension pneumothorax. Air in pleural cavity, diaphragm depression, and contralateral deviation of mediastinal structures are evidence of a tension pneumothorax. Computed tomography (CT) scanning is more sensitive and can be utilized (in noncritical patients) for diagnostic purposes when chest radiography is nonrevelatory, as is the case in limited ventral tension pneumothorax.[23] Several ECG findings have been described in tension pneumothorax, such as rightward axis deviation, diminution of precordial R wave, decreased QRS amplitude, and precordial T-wave inversion. These relatively nonspecific ECG changes have been attributed to either cardiac axis changes or direct pressure on coronary arteries from tension pneumothorax.[24]

Treatment

If there is suspicion of tension pneumothorax, management, including decompression, should be

approached urgently. Hemodynamically significant tension pneumothorax is treated with needle thoracostomy, which is performed by inserting a catheter needle in the second intercostal space in the midclavicular line. Needle thoracostomy always requires follow-up chest tube placement. If the condition does not resolve, open thoracostomy must be performed to determine whether the lung has a large air leak (eg, ruptured bronchus) requiring direct repair.[22,25]

MASSIVE PULMONARY EMBOLISM

Massive pulmonary embolism results when embolism size and underlying cardiopulmonary status interact to produce hemodynamic instability. Circulatory failure occurs in about 10% of patients with pulmonary thromboembolism. In acute pulmonary embolism, occlusion of 2 major pulmonary arteries is usually (but not always) required to create hemodynamic instability. However, elaboration of vasoactive hormones by even small thromboemboli may produce vasomotor instability. Pulmonary embolism often occurs in the setting of malignancy or recent history of surgery, although 50% of pulmonary embolism occurs in absence of such classic predisposing factors as immobilization and pregnancy.[26]

Massive pulmonary emboli lead to rapid onset of circulatory failure and signs of acute cor pulmonale. This critical state is often preceded by symptoms such as syncope, chest pain, dyspnea, and manifestations of acute right heart failure (JVD and accentuated pulmonary S_2). ECG may demonstrate various abnormalities including right axis shift, tachycardia, $S_1Q_3T_3$ pattern, right bundle branch block, or diffuse T-wave inversion.[27]

Diagnostic Imaging

Suspected pulmonary embolism may be approached with a variety of imaging studies such as echocardiography, ventilation/perfusion scan, spiral CT scan, or pulmonary angiography. However, when a patient presents with cardiovascular collapse due to massive pulmonary embolus, options are limited. The typical echocardiographic picture of a hemodynamically significant pulmonary embolus includes a dilated, hypokinetic right ventricle with an increased right ventricle-to-left ventricle-diameter ratio due to interventricular septal bulging into the left ventricle. Other findings include dilated pulmonary arteries, tricuspid regurgitation, and dilated inferior vena cava, all signs of acute rise in right ventricular pressure and volume. Hemodynamically important pulmonary embolus is unlikely in a patient with a normal echocardiogram.[26,27]

Treatment

Shock, which is not always present in this syndrome, confers a worse prognosis.[27] Therefore, patients with suspected massive pulmonary embolism often require aggressive resuscitation and stabilization. Administration of supplemental oxygen and volume expansion with crystalloid are the initial management for hypoxemia and hypotension, respectively. Fluid resuscitation should be monitored closely, since increased volume can worsen an already volume-overloaded right ventricle. Heparin should be instituted in all pulmonary embolism patients unless contraindicated, and it should be titrated to achieve rapid therapeutic anticoagulation to prevent recurrence.[28] Thrombolytic therapy should be considered for patients with shock caused by massive pulmonary embolism. Although thrombolytics seem appealing, only 1 study has demonstrated a mortality benefit.[27] Thrombolytics are not indicated in patients without right ventricular overload. For hemodynamically unstable patients with a contraindication to thrombolytic therapy who are refractory to medical treatment for over 1 hour or have ongoing cardiac arrest, surgical embolectomy should be considered.[26,27]

RIGHT ATRIAL THROMBUS

Right atrial thrombi are uncommon but are often clinically significant. Right atrial thrombi can be idiopathic or can be caused by many different processes, such as atrial fibrillation, hypercoagulable state (eg, malignancy, antiphospholipid antibody, protein C deficiency), cardiomyopathy, post-MI, central lines (including vascular access devices), Behçet's disease, pacemaker wire, and shunts.[29–33] The symptoms and physical findings associated with this syndrome depend on the size and location of the thrombus. Patients with right atrial thrombi may present with dyspnea, chest pain, or syncope. Pulmonary embolus is present in 98% of symptomatic cases, whereas hypotension and JVD are present in less than half of these symptomatic patients.[34] Right atrial thrombi are more likely to cause JVD rather than hemodynamic collapse. Regardless of their size, right atrial thrombi are associated with significant tricuspid regurgitation, leading to at least a minor elevation of jugular venous pressure. Thrombus obstructing right ventricular inflow at the tricuspid valve has been reported.[31–33] Both pulmonary embolism and right heart outflow tract obstruction are life-threatening complications of right atrial thrombus. Primary thrombi intrinsic to the heart are more likely to be nonmobile and less likely to be complicated by pulmonary embolism, whereas thrombi arising from systemic veins are more likely to be mobile and carry higher risk of pulmonary embolism.[35]

Diagnostic Imaging

Many diagnostic modalities for detecting right atrial thrombus have been studied. TTE remains the initial diagnostic choice because of its simplicity, versatility, and noninvasive approach.[33] However, TTE is likely to miss thrombi in the right atrial appendage, and this shortcoming accounts for TTE's low sensitivity. TEE is better than TTE at diagnosing right atrial thrombi.[35] One study indicated higher sensitivity of ultrafast CT scanning for visualization of right atrial thrombi compared with TTE.[36] A CT scan should be performed if there is a high clinical suspicion for thrombotic pathology despite negative findings on echocardiogram.[36,37] Digital subtraction angiography may also prove useful.

Treatment

Because of the infrequency of patients presenting with this condition, management of right atrial thrombi has been individualized. Therapeutic measures should be taken to prevent pulmonary embolism. If a large thrombus is the cause of shock, emergent surgery might be considered.[32,34,38] Rose et al[34] studied anticoagulation, surgery, and thrombolysis as primary approaches to treatment, and they concluded that thrombolytics are superior to surgical intervention. The combination of platelet glycoprotein inhibitors and heparin as a possible treatment option is currently under investigation.

CARDIAC TUMORS

In addition to embolized thrombus, cardiac tumors should be considered in the differential diagnosis of shock with JVD. Metastatic malignancies are more common than primary cardiac tumors. Lung cancer and melanoma are the most common causes of metastatic cardiac tumors, but renal cell tumors, breast tumors, and lymphomas are also well-documented secondary malignancies of the heart. Primary myxoma is the most common primary benign tumor of the heart, whereas sarcomas (angiosarcoma, rhabdomyosarcoma, fibrosarcoma, leiomyosarcoma) are the most common primary invasive malignancies of the heart.[39,40] Extrinsic compression by tumors, such as mediastinal lymphomas and bronchogenic carcinoma, have been reported to present as right heart failure, chest pain, and hypotension with symptoms suggestive of tamponade or pulmonary embolism.

Diagnostic Imaging

Cardiac tumors can result in cardiogenic shock by obstructing right atrial or ventricular outflow, thus leading to reduced cardiac output. These patients typically present with right heart failure accompanied by dyspnea, JVD, hepatomegaly, S_3, diastolic rumbles, and peripheral edema. Thirty percent of these patients may have had recent embolic events.[41,42] Imaging with TEE or magnetic resonance has proven effective in noninvasive detection of intracardiac masses.[43] Under emergent conditions, TTE can provide anatomic and hemodynamic information quickly, establishing the diagnosis and guiding treatment. Myxomas usually appear as intracavitary pedunculated (with stalk) or round masses on echocardiography (often arising in the area of the fossa ovalis), while metastatic malignancies are frequently associated with pericardial effusion.[39,40] Metastatic intracavitary lesions are more likely to be on the right side of heart and may cause right ventricular outflow tract obstruction.

Treatment

Surgery is indicated for cure of benign tumors because they do not commonly recur after resection. Malignant tumors (or multicentric benign tumors), on the other hand, have a poor prognosis since surgical approaches demand resection of significant portions of cardiac tissue. Palliative management should be directed at preventing embolic or arrhythmic phenomena associated with cardiac malignancy.[41]

CARDIAC HERNIATION

Cardiac herniation is a rare cause of shock and is usually a complication after pneumonectomy. Cardiac herniation has only been reported when the pericardium remains open after surgery. The heart protrudes through the pericardial opening and occupies removed lung space, which causes obstruction of outflow vessels, thereby "strangulating" the myocardium. Most cases occur within 72 hours of the pneumonectomy. Signs and symptoms include acute hypotension, JVD, chest pain, cyanosis, and sometimes superior vena cava syndrome.

Right-sided cardiac herniation can easily be confirmed by simple chest radiography demonstrating a characteristically abnormal cardiac silhouette. However, left-sided cardiac herniation may be difficult to diagnose without CT scan. Usually, these patients are unstable and may require surgical exploration. Treatment is surgical repair of the pericardium. Even with intervention, cardiac herniation is associated with high mortality.[44]

CONCLUSION

Cardiovascular collapse requires rapid assessment and treatment. Delineating the underlying cause of shock with JVD is essential for patient survival and maintaining hemodynamic stability. A thorough history and detailed physical examination should be undertaken during the

initial steps of the evaluation. In the absence of firmly established accompanying pulmonary congestion, these patients are generally acceptable candidates for fluid resuscitation while other data are being emergently gathered. The role of echocardiography is underutilized in critically ill patients. Emergent echocardiography should be considered as a first-line diagnostic modality in patients presenting in shock with JVD. **HP**

Test your knowledge and comprehension of this article with *Review Questions* on page 36.

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