Obstructive shock

DR Sepideh Hejazi
Assistant professor of pulmonology
Mashhad university of medical scince

Shock

Shock is a life-threatening condition of circulatory failure, causing inadequate oxygen delivery to meet cellular metabolic needs and oxygen consumption requirements, producing cellular and tissue hypoxia.

Shock

The effects of shock are initially reversible, but rapidly become irreversible, resulting in multiorgan failure (MOF) and death.

When a patient presents with undifferentiated shock, it is important that the clinician immediately initiate therapy while rapidly identifying the etiology so that definitive therapy can be administered to reverse shock and prevent MOF and death.

Obstructive shock

Obstructive shock is mostly due to **extracardiac** causes of cardiac pump failure and often associated with poor right ventricular output.

Obstructive shock

The causes of obstructive shock can be divided into the following two categories,

pulmonary vascular

mechanical

Obstructive	**Pulmonary vascular**	▪ Hemodynamically significant pulmonary embolus, severe pulmonary hypertension, severe or acute obstruction of the pulmonic or tricuspid valve, venous air embolus
	Mechanical	▪ Tension pneumothorax or hemothorax (eg, trauma, iatrogenic), pericardial tamponade, constrictive pericarditis, restrictive cardiomyopathy, severe dynamic hyperinflation (eg, elevated intrinsic PEEP), left or right ventricular outflow tract obstruction, abdominal compartment syndrome, aorto-caval compression (eg, positioning, surgical retraction)

Mechanical

Patients in this category present clinically as hypovolemic shock because their primary physiologic disturbance is decreased preload, rather than pump failure (eg, reduced venous return to the right atrium or inadequate right ventricle filling).

Mechanical

Mechanical causes of obstructive shock include the following:

- Tension pneumothorax
- Pericardial tamponade
- Constrictive pericarditis
- Restrictive cardiomyopathy

Mechanical

Pneumothorax

Pneumothorax occurs often in both blunt and penetrating trauma, and presenting symptoms and signs may be delayed

Patients may manifest tachypnea, ipsilateral chest pain, hypoxia, unilateral diminished or absent breath sounds, subcutaneous air, or unilateral hyperresonance to percussion, depending on underlying injuries and the extent of the pneumothorax.

Pneumothorax

If the clinician suspects a tension pneumothorax and the patient is hypotensive, a needle thoracostomy can be performed, as a temporizing measure, using a long, large angiocatheter or needle inserted above the rib at the second intercostal space in the mid-clavicular line, or at the fifth intercostal space in the mid-axillary line. a 4.5 cm (2 inch) needle is a reasonable first choice.

Depending on the patient's body habitus, needles as long as 8 cm may be required for effective decompression.

Pulmonary vascular

Most cases of obstructive shock are due to right ventricular failure from hemodynamically

significant pulmonary embolism (PE) or

severe pulmonary hypertension (PH).

the right ventricle fails because it is unable to generate enough pressure to overcome the high pulmonary vascular resistance associated with PE or PH.

Pulmonary vascular

While hemodynamic collapse in the setting of PE is traditionally attributed to mechanical obstruction, pulmonary vasoconstriction mediated by vasoactive mediators such as serotonin and thromboxane also contribute to the observed pathophysiology .

Patients with severe stenosis or with acute obstruction of the pulmonary or tricuspid valve may also fall into this category.

HEMODYNAMICALLY UNSTABLE PATIENTS

PE is stratified into massive, submassive, and low-risk based upon the presence or absence of hypotension and right ventricular dysfunction or dilation.

This stratification is associated with mortality risk .

Pulmonary embolism

In the small percentage of patients with hemodynamic instability, either at presentation or during the course of their illness, the symptoms range from mild hypotension to overt obstructive shock.

Hemodynamically unstable

When patients with suspected PE present with hypotension, initial support should focus upon restoring perfusion with intravenous fluid resuscitation and vasopressor support, as well as oxygenation and, if necessary, stabilizing the airway with intubation and mechanical ventilation.

Initial therapies

Respiratory support

Supplemental oxygen should be administered to target an oxygen saturation ≥90 percent.

Severe hypoxemia, hemodynamic collapse, or respiratory failure should prompt consideration of intubation and mechanical ventilation.

Initial therapies

Importantly, patients with coexistent right ventricle failure are prone to hypotension following intubation.

in this population high plateau pressures should be avoided.

Initial therapies

Hemodynamic support

The precise threshold that warrants hemodynamic support depends upon the patient's baseline blood pressure and whether there is clinical evidence of hypoperfusion (eg, change in mental status, diminished urine output).

In general, small volumes of intravenous fluid (IVF), usually 500 to 1000 mL of normal saline, followed by vasopressor therapy should perfusion fail to respond to IVF.

Initial therapies

Intravenous fluid

IVF is first-line therapy for patients with hypotension. However, in patients with right ventricular (RV) dysfunction, aggressive fluid resuscitation is not beneficial, and may be harmful .

small volumes of IVF increase the cardiac index in patients with PE, while excessive amounts of IVF result in RV overstretch (ie, RV overload), RV ischemia, and worsening RV failure.

Vasopressors

Intravenous vasopressors are administered when adequate perfusion is not restored with IVF. The optimal vasopressor for patients with shock due to acute PE is unknown, but norepinephrine is generally preferred

Norepinephrine – Norepinephrine is the most frequently utilized agent in this population because it is effective and less likely to cause tachycardia Other alternatives include dopamine and epinephrine, but tachycardia, which can exacerbate hypotension, can occur with these agents .

Hemodynamically unstable

For most patients who become hemodynamically stable following resuscitation and in whom the clinical suspicion for PE is high, prefer immediate anticoagulation with unfractionated heparin and prompt imaging for definitive diagnosis (usually computed tomographic pulmonary angiography [CTPA]).

Hemodynamically unstable

For patients with a high clinical suspicion for PE who are hemodynamically unstable (ie, systolic blood pressure <90 mmHg for >15 minutes, hypotension requiring vasopressors, or clear evidence of shock), When portable perfusion scanning or CTPA is not available, bedside echocardiography to obtain a presumptive diagnosis of PE (right ventricle enlargement/hypokinesis, regional wall motion abnormalities that spare the right ventricular apex [McConnell's sign], or visualization of clot) prior to the empiric administration of systemic thrombolytic therapy (ie, reperfusion therapy).

Hemodynamically unstable

If bedside echocardiography is delayed or unavailable, the use of thrombolytic therapy as a life-saving measure should be individualized; if not used, the patient should receive empiric anticoagulation.

suggest a similar approach for select patients with known PE whose course becomes complicated by hypotension during anticoagulation in whom the suspicion for recurrent PE despite anticoagulation is high.

Pulmonary embolism response teams

The decision to administer thrombolysis is strongly influenced by additional clinical factors.

For example, while a patient with proven PE-induced shock who is unconscious requiring very high doses of pressors is a candidate for immediate intravenous thrombolytic therapy, a patient who has low blood pressure for 20 minutes but who is awake, alert, and comfortable, with low oxygenation requirement might be considered for anticoagulation alone.

Hemodynamically unstable

For patients with suspected PE who remain hemodynamically unstable and the clinical suspicion is low or moderate, the approach to empiric anticoagulation should be the same as for patients who are hemodynamically stable; empiric thrombolysis is not justified in this population.

Wells criteria and modified Wells criteria: Clinical assessment for pulmonary embolism

▪ Clinical symptoms of DVT (leg swelling, pain with palpation)	3.0
▪ Other diagnosis less likely than pulmonary embolism	3.0
▪ Heart rate >100	1.5
▪ Immobilization (≥3 days) or surgery in the previous four weeks	1.5
▪ Previous DVT/PE	1.5
▪ Hemoptysis	1.0
▪ Malignancy	1.0
Probability	**Score**
Traditional clinical probability assessment (Wells criteria)	
High	>6.0
Moderate	2.0 to 6.0
Low	<2.0
Simplified clinical probability assessment (Modified Wells criteria)	
PE likely	>4.0
PE unlikely	≤4.0

DVT: deep vein thrombosis; PE: pulmonary embolism.

Empiric anticoagulation

The administration of empiric anticoagulation depends upon the risk of bleeding, clinical suspicion for PE and the expected timing of diagnostic tests .

There is no optimal prediction tool for assessing bleeding risk in patients with PE.

propose use of the Wells score to assess the risk of PE, careful clinical judgment is acceptable.

Empiric anticoagulation

Low risk for bleeding

Patients without risk factors for bleeding have a three-month bleeding risk of <2 percent; in such patients, empiric anticoagulation may be considered in the following patient groups:

- A high clinical suspicion for PE (eg, Wells score >6)
- A moderate clinical suspicion for PE (eg, Wells score 2 to 6), in whom the diagnostic evaluation is expected to take longer than four hours
- A low clinical suspicion for PE (eg, Wells score <2), if the diagnostic evaluation is expected to take longer than 24 hours

Risk factors for bleeding with anticoagulant therapy and estimated risk of major bleeding in low, moderate, and high ri categories

Risk factors*
Age >65 years
Age >75 years
Previous bleeding
Cancer
Metastatic cancer
Renal failure
Liver failure
Thrombocytopenia
Previous stroke
Diabetes
Anemia
Antiplatelet therapy
Poor anticoagulant control
Comorbidity and reduced functional capacity
Recent surgery¶
Frequent falls
Alcohol abuse

Estimated absolute risk of major bleeding (%)			
Categorization of risk of bleeding[Δ]	**Low risk[◊] (0 risk factors)**	**Moderate risk[◊] (1 risk factor)**	**High risk[◊] (≥2 risk factors)**
Anticoagulation 0 to 3 months[§]			
Baseline risk (%)	0.6	1.2	4.8
Increased risk (%)	1	2	8
Total risk (%)	1.6[§]	3.2	12.8[¥]

Unacceptably high risk for bleeding

For patients with **absolute contraindications** to anticoagulant therapy (eg, recent surgery, hemorrhagic stroke, active bleeding) or those assessed by their clinician to be at an unacceptably **high** risk of bleeding (eg, aortic dissection, intracranial or spinal cord tumors), empiric anticoagulation should **not** be administered.

The diagnostic evaluation should be expedited so that alternate therapies (eg, inferior vena cava filter, embolectomy) can be initiated if PE is confirmed.

Empiric anticoagulation

Moderate risk for bleeding

Patients with one or more risk factors for bleeding have a moderate (>3 percent) to high (>13 percent) risk of bleeding. In such patients, empiric anticoagulant therapy may be administered on a case-by-case basis according to the assessed risk-benefit ratio and the values and preferences of the patient.

As an example, might empirically anticoagulate a patient with moderate risk of bleeding if they have a high clinical suspicion for PE, severe respiratory compromise, or an expected delay for the insertion of a vena caval filter.

Anticoagulation

menstruation, epistaxis, and the presence of minor hemoptysis are not contraindications to anticoagulation but should be monitored during anticoagulant therapy.

Anticoagulation

The optimal agent for empiric anticoagulation depends upon the presence or absence of hemodynamic instability, the anticipated need for procedures or thrombolysis, and the presence of risk factors and comorbidities .

Anticoagulation

low molecular weight heparin (LMW heparin) may be chosen for patients with hemodynamically stable PE who do not have renal insufficiency in whom rapid onset of anticoagulation needs to be guaranteed (ie, therapeutic levels are achieved with four hours).

Anticoagulation

unfractionated heparin may be preferred by most experts in patients who are hemodynamically unstable in anticipation of a potential need for thrombolysis or embolectomy, LMW heparin is not contraindicated in this setting.

Direct thrombin and factor Xa inhibitors should **not** be used in hemodynamically unstable patients.

Hemodynamically unstable patients

In patients with PE who are hemodynamically unstable or who become unstable due to recurrence despite anticoagulation, suggest more aggressive therapies (ie reperfusion therapies) than anticoagulation.

Thrombolytic therapy is indicated in most patients, provided there is no contraindication

Embolectomy is appropriate for those in whom thrombolysis is either contraindicated or unsuccessful (surgical or catheter-based)

Reperfusion therapy

Thrombolytic therapy

Systemic thrombolytic therapy is a widely accepted treatment for patients with PE who present with, or whose course is complicated by, hemodynamic instability.

Reperfusion therapy

For those in whom systemic thrombolysis is unsuccessful, the optimal therapy is unknown.

Options include repeat systemic thrombolysis, catheter-directed thrombolysis, or catheter or surgical embolectomy, the choice of which is dependent upon available resources and local expertise.

Embolectomy

Embolectomy is indicated in patients with hemodynamically unstable PE in whom thrombolytic therapy is contraindicated. It is also a therapeutic option in those who fail thrombolysis.


THANKs FOR
ATTENTION