Shock in Emergency Department: Evaluation and Initial Management

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Abstract

Introduction: Circularity failure of adequate oxygen delivery for cells and tissues defines shock. Patient with shock could be hypertensive, normotensive, or hypotensive. Early, the effects of shock could be overturned, however, inadequate delayed management may lead to irreversible multiorgan failure and death.

Aim of Work: In this review, we will discuss the last available evidence about the best initial management on patient with suspected undifferentiated shock.

Methodology: We conducted a systematic search on PubMed search engine and Google Scholar search engine for all studies examining initial management of shock in adults.

Conclusions: The first step in adequate management is prompt assessment and securing of the airway, breathing, and circulation. Following initial assessment, the efforts should be directed toward a thorough diagnostic evaluation by laboratory and imaging studies. When initial assessment and/or laboratory evaluation suspect the presence of shock, hemodynamic support with intravenous fluids (IVFs) should be initiated, followed by vasopressors if IVFs fail.

Keywords: Shock in Adults; Management of Shock; Assessment of Shock; IV Fluids in ER

Introduction

Oxygen is essential element for fulfilling cellular and tissular metabolic needs. Circularity failure of adequate oxygen delivery for cells and tissues defines shock. Shock is a life-threatening state of tissue hypoxia regardless of the reasons. Cellular hypoxia could results from

reduced oxygen delivery (hypoperfusion), inadequate oxygen utilization, or failure of fulfilling increased oxygen need, or a combination of these. These events occur mostly when the circulatory failure results in hypotension. However, it is essential to bear in mind that patient with shock could be hypertensive, normotensive, or hypotensive. With rapid adequate response, the effects of shock could be overturned, however, inadequate delayed management may lead to irreversible multiorgan failure (MOF) and death. Efforts for preventing MOF is the mainstay of shock management.

Shock is classified to four main types: distributive, cardiogenic, hypovolemic, and obstructive. Each of these category could be caused by many conditions. However, a distinctive line may not always present between these types and many patients with circulatory failure may have a combination of more than one form of shock. This is usually called multifactorial shock.

Septic shock is the most commonly encountered type of shock among patients admitted to the intensive care unit (ICU), followed by cardiogenic and hypovolemic shock; patients with obstructive shock are rarely seen [1,2]. The percentage of each type of shock in emergency department is affected by the population served by that department [3,4]. A higher percentage of hemorrhagic shock is seen in busy urban trauma centers.

Methods

We conducted a thorough search on PubMed search engine (http://www.ncbi.nlm.nih.gov/) and Google Scholar search engine (https://scholar.google.com) for all studies examining initial management of shock in adults. All relevant available full articles were reviewed and included. The terms used in the search were: Shock in adults, management of shock, assessment of shock, IV fluids in ER.

Clinical presentation

Patient suspected to be in shock may present with many clinical finding. The clinical findings vary according to the etiology and the time of presentation whether in pre-shock, shock, or presented with end-organ failure. Undifferentiated shock may present with tachycardia; tachypnea; hypotension; oliguria; disturbed conscious level; Cool, clammy, cyanotic extremities; metabolic acidosis; increased lactate acid (lactatemia). It is crucial to remember that patients in the early stages of shock can be normotensive or hypertensive, such that hypotension does not have to be present for the diagnosis.

Tachycardia occurs as an early compensatory mechanism. It is worth mentioning that younger patients develop severe and persistent tachycardia before becoming hypotensive late in the course of shock compared with older patients. This feature may misguide the physician in this age group. Similar to tachycardia, tachypnea is also an early compensatory mechanism in patients with shock. It specifically denotes metabolic acidosis development. Despite being common among hospitalized [5], the presence of tachypnea is a useful tool to identify patients at risk of clinical deterioration [6].

Oliguria can be explained by shunting of renal blood flow to other vital organs or by a direct injury to the kidney.

Cool and clammy skin is due to compensatory peripheral vasoconstriction that aims to redirects blood to main vital organ. A cyanotic, mottled appearance is a worrying feature that may appear in advanced shock. However, the appearance of cool, clammy or cyanotic skin may also be due to underlying peripheral arterial vascular disease. Importantly, warm, hyperemic skin does not ensure the absence of shock because such an appearance may be present in patients with early distributive or terminal shock.

Detection of a high anion gap metabolic acidosis should always raise suspicion for the presence of shock. Importantly, the presence of a metabolic acidosis in states of shock is not specific and can also be due to acute kidney injury or toxin ingestion.

Elevated level of serum lactate is associated with poorer outcomes whether in conjunction with metabolic acidosis or by itself, with shock or with other conditions [7].

Most of these clinical manifestations are neither sensitive nor specific for the diagnosis of shock. However, they may provide clues to the cause of shock and narrow the differential diagnosis so that empiric management can be initiated.

Initial management

The initial management of undifferentiated shock include initial approach, initial assessment, initial diagnostic methods, and hemodynamic support. When possible, a multidisciplinary, team-based approach is preferred as it allows the simultaneous evaluation and administration of therapy.

Initial assessment

The first step in adequate management is prompt assessment and securing of the airway, breathing, and circulation. The airway should be stabilized and breathing is insured with oxygen. Patients with respiratory distress and/or marked hemodynamic instability should be intubated as a rule. The only exception is those with suspected tension pneumothorax, where the prompt drainage of air from the pleural space may quickly reverse shock and avoid intubation. Rapid sequence intubation is the preferred approach, typically with etomidate or ketamine, and rapidly acting neuromuscular blocker such as succinylcholine or rocuronium. Propofol, midazolam or any agent that may worsen hypotension should be avoided.

Circulation should be secured with adequate intravenous access so that patients can be immediately treated with intravenous fluids to restore adequate tissue perfusion. It is crucial that resuscitative efforts not be delayed for a detailed clinical assessment, nor should clinicians be conservative in terms of fluid resuscitation in patients with a history of heart failure or kidney injury. It is evidenced that liberal fluid resuscitation appeared to be life-saving in patients with septic shock and intermediate serum lactate levels [8].

For initial management, peripheral venous access or intraosseous access is sufficient in many patients with undifferentiated shock and hypotension. Vasoactive medications can be administered safely for hours to days in peripheral intravenous access, obviating the need for central venous catheterization in a number of patients [9]. Central venous access should be obtained in case of failure of obtaining peripheral access or in patients need large volumes or prolonged infusion, or in those who need prolonged infusions of vasopressors.

Patients should be assessed for the need for an immediate or early intervention (surgical intervention for example). If the patients become hemodynamically unstable during the evaluation and early treatment period, rapid redirection of the approach to the lifesaving management is essential.

Brief history is essential to direct the initial approach and adequate management of life-threatening conditions. This could be obtained from prehospital providers, hospital staff, family members, and the patient. The presence of hypotension, inspiratory stridor, oral and facial edema, hives, recent exposure to common allergens strongly suggests shock due to anaphylaxis. Prompt intramuscular injection of epinephrine is lifesaving. Patients on mechanical ventilation may also have a sudden elevation in peak inspiratory pressures. The typical adult dose is 0.3 mg injected into the mid-outer thigh and repeated every 5 to 15 minutes as needed.

Shock due to tension pneumothorax should be suspected in case of tachypnea, unilateral pleuritic chest pain and diminished breath sounds, distended neck veins, tracheal deviation away from the affected side, and risk factors for tension pneumothorax such a history of trauma, recent procedure, or in mechanically ventilated patients. Once strongly suspected, chest radiograph is not required and needle decompression or an emergent tube thoracostomy should be used. Ultrasound guidance is preferable for both diagnosis and tube placement.

Patients with dyspnea, tachycardia, hypotension, elevated jugular venous pressure, distant heart sounds, pulsus paradoxus, and known risk factors as trauma should be suspected to have pericardial tamponade. Point-of-care (POC) ultrasonography or bedside echocardiog-

raphy is preferred before attempt for pericardiocentesis. Ultrasonography also guides needle or catheter placement and examines the response to drainage of fluid from the pericardial sac. An emergency thoracotomy may be performed in rare cases with no to catheter drainage or in those who develop a cardiac arrest during resuscitation. It is worth mentioning that pericardiocentesis should not be attempted in patients with a pericardial effusion due to aortic dissection or myocardial rupture, as it may worsen bleeding. Such patients require emergent surgical intervention.

Shock is suspected to be due hemorrhage should be distinguished whether due traumatic or non-traumatic hemorrhage. A history of blunt or penetrating trauma more benefit from focused assessment with sonography for trauma (FAST) which is a rapid multiorgan bedside ultrasonography to identify blood in the abdomen. Positive results indicates the need for urgent surgical exploration to identify and control the source of bleeding. When there is no history of trauma and yet hemorrhagic shock is suspected, ruptured aorta should be considered. Patient with rupture of aorta usually presents with hypotension, abdominal, chest or back pain, known history of aneurysm or dissection. These patients may be too unstable to safely obtain a contrast-enhanced computed tomography (CT). Transesophageal echocardiography (for thoracic aorta) and abdominal ultrasound (for abdominal aorta), to detect perioaortic hematoma or aneurysmal disorder, are diagnostic choices prior to management.

For patients with the manifestations of upper or lower gastrointestinal bleeding, endoscopic intervention, embolization, or surgery could be attempted. Shock due to hemorrhage usually requires large volumes of blood products; vasopressors are avoided. Physician must insure adequate peripheral access and/or a large-bore, single-lumen central line in patient with suspected hemorrhage. A type and crossmatch, a complete blood count, and coagulation studies should be obtained.

Initial evaluation may indicate the presence of arrhythmia resulting in shock. Tachyarrhythmia can be cardioverted by atropine or infusions of vasoactive agents, temporary or permanent pacemaker placement is adequate for bradyarrhythmias. Arrhythmias may be the primary cause of shock or contribute to it; immediate management is potentially lifesaving. Nevertheless, arrhythmias can be secondary to shock-related metabolic disorders as acidosis or due to the underlying cause of shock as sepsis [10], pulmonary embolism, or myocardial infarction. Thus, their presence should prompt additional investigations.

Fever, hypotension, and a suspected septic source suggest septic shock. These patients benefit from the early administration of intravenous antibiotics and intravenous fluid resuscitation. The choice of antibiotic is determined by the suspected source. If the source is unknown and *Pseudomonas* is unlikely, combining vancomycin with a third- or fourth-generation cephalosporin, a beta-lactam/beta-lactamase inhibitor, or a carbapenem is adequate initial management. If *Pseudomonas* is likely, vancomycin should be combined with two antipseudomonal agents such as fluoroquinolone, aminoglycoside, piperacillin-tazobactam, cefepime, or ceftazidime. The presence of leukocytosis particularly with bandemia, as well as laboratory and imaging findings suggestive of a source, support the presence of sepsis as a cause of shock. Blood and other appropriate body fluid cultures should be obtained, preferably prior to the administration of antibiotics, in addition to imaging when necessary to facilitate timely source control. For follow up, serial vital signs, and serum lactate measures, can be used is septic shock patients.

Cardiogenic shock should be suspected in patient with hypotension associated with anterior crushing chest pain, respiratory distress, and ECG changes consistent with ST elevation myocardial infarction (STEMI). Elevated troponin or creatine phosphokinase levels and pulmonary edema on chest radiography further the suspicion of the diagnosis. Interventions include the administration of antiplatelet agents or heparin, coronary revascularization procedures, and/or an intraaortic balloon pump. Non-STEMI patients may additionally benefit from the administration of glycoprotein IIb/IIIa inhibitors. Cardiogenic shock may result from acute aortic or mitral valve insufficiency. Suggestive symptoms of aortic insufficiency include chest pain, hypotension, and new low-pitched early diastolic murmur. These patients should undergo POC ultrasonography or echocardiography prior to surgical intervention. Additional laboratory or imaging studies aimed at discovering the etiology may be required. Patients with acute respiratory distress and new systolic murmur following an acute myocar-

dial infarction (MI) should preferably undergo urgent echocardiography to look for mitral valve insufficiency or ventricular septal defect, which also typically needs urgent surgical intervention. Aortic dissection often present with tearing chest or back pain. Unlike patient with descending thoracic aortic dissection which may present with hypertension, patients with ascending aortic dissection are likely to present with hypotension and shock due to acute aortic insufficiency, pericardial tamponade, or myocardial infarction. Ascending aortic dissection is a cardiac surgical emergency and immediate consultation with a cardiac surgeon should be obtained.

Shocked patient with acute dyspnea and hypoxemia associated with hypotension is strongly suspected of having a pulmonary embolism (PE). Administration of systemic thrombolytic therapy could be lifesaving. Elevated D-dimer, troponin, and natriuretic peptide levels support the diagnosis; chest radiography is usually normal. Computed tomographic (CT) pulmonary angiography is the preferred diagnostic option. However, when CT is inadequate, a presumptive diagnosis may be obtained by POC cardiac ultrasonography or echocardiography; positive finding include right ventricle enlargement and/or thrombus. These findings justify the administration of a thrombolytic agent in absence of contraindications.

A history of glucocorticoid deficiency or withdrawal in patients with hypotension and volume depletion is suggestive an adrenal crisis. Initial management should include fluid resuscitation and dexamethasone 4 mg intravenously. The selection of dexamethasone is based on the ability to interpret serum cortisol measurements as part of the evaluation.

Patient may present with shock following some insect or animal bites, these patients require antivenom to reverse shock in addition to standard resuscitation.

Initial diagnostic evaluation

Following initial assessment, the efforts should be directed toward a thorough diagnostic evaluation. This include laboratory and imaging studies. Bedside telemetry and/or electrocardiogram (ECG) may suggest the etiology and should be performed in all patients with undifferentiated hypotension and shock. Arrhythmia and ST segment changes consistent with ischemia or pericarditis could be seen. A low-voltage ECG may be suggestive of a pericardial effusion. The classic signs of pulmonary embolism (S1, Q3, T3) or right ventricular strain may also be evident.

Laboratory tests should be performed early in patient with undifferentiated shock. An elevated serum lactate (> 2 mmol/L) is an early indicator of shock and could be very useful parameter especially in normotensive or hypertensive patients. Other important laboratory test include: Complete blood count and differential (CBC), atrial blood gases (ABG), cardiac enzyme, renal and liver function test, Coagulation studies and D-dimer level.

Complete blood count may suggest the type of shock. Anemia in the setting of bleeding supports hemorrhagic shock, and concurrent thrombocytopenia may suggest an etiology for hemorrhage. An elevated eosinophil count may suggest an anaphylaxis shock.

Although a leukocytosis may suggest septic shock, it is not specific for the diagnosis and may simply indicate a stress response. Decreased white blood cell count especially if associated with bandemia are more worrisome for sepsis in the setting of undifferentiated shock [11].

Lactate levels in shock are reflects the level of tissue perfusion and are due to increased production from anaerobic metabolism, aerobic metabolism, and decreased clearance by the liver, kidneys, and skeletal muscle [7,12]. However, although elevated lactate is a sensitive tool for the diagnosis of shock (normal level rules out the diagnosis), it is not specific and can also be found in conditions including metformin toxicity, diabetic ketoacidosis, and alcoholism.

Lactate level higher than 4 mmol/L are especially associated with increased mortality independent of organ dysfunction or hypotension. Increased level of lactate due to other conditions is also associated higher mortality [13]. Elevated cardiac enzyme such as troponin-I

or -T levels, brain natriuretic peptide, may indicate cardiogenic shock from ischemia but can also be due to demand ischemia or to pulmonary embolism (PE).

Imaging modalities could be helpful in suggesting the etiology of shock. A portable chest radiograph is typically performed in most patients with suspected shock to detect common causes as pneumonia (septic shock).

Point-of-care (POC) ultrasonography are commonly used in patient with undifferentiated shock and hypotension [14-17]. This includes rapid ultrasound in shock (RUSH), focused cardiac ultrasound (FOCUS), or abdominal and cardiac evaluation with sonography in shock (ACES). They are used as portable, bedside diagnostic tools. RUSH and ACES are multiorgan ultrasonography that examine the heart first, followed by ultrasound of the chest and abdomen and major blood vessels. Focused cardiac ultrasound (FOCUS) examines the heart only. The idea of these techniques in patients with undifferentiated shock are similar to focused assessment with sonography for trauma (FAST) used in trauma patients. POC ultrasonography is portable and inexpensive with no adverse effect. Its major advantage is the rapid examination of multiple organs, particularly the heart allowing to narrow the differential diagnosis and identify a potential etiology for shock. Empiric diagnoses can be obtained within minutes when compared with standard imaging modalities as shown by observational studies. Ultrasonography is more sensitive than portable chest radiography for the detection of pneumothorax, with high sensitivity (86 to 100 percent) and specificity (92 to 100 percent) [18-21]. However, POC has limited sensitivity for many other etiologies of shock when compared with definitive imaging modalities performed by fully-trained providers. This could be partially explained by the lack of standards regarding the training, performance, and indications for bedside ultrasonography. A good example of such limitation is evidenced in detection of pericardial effusion, while POC ultrasonography is considered sensitive and specific, comprehensive echocardiography with additional views may be essential for definitive diagnosis when effusions are complex, loculated, or small [22,23]. Additionally, many abnormalities cannot be readily detected using limited bedside views an regional wall motion abnormalities, valvular dysfunction, ventricular septal wall perforation, ruptured aortic aneurysms, and aortic dissection. One meta-analysis of nine studies has compared FOCUS-assisted clinical assessment with clinical assessment alone and reported that while FOCUS examination of the left ventricle and mitral valve was more sensitive than clinical assessment alone, it has similar specificity [24].

There is no data supporting the benefits of pulmonary arterial catheterization (PAC) on important outcomes. Hence, the routine insertion of Swan-Ganz catheters has largely declined [25-27]. However, hemodynamic measurements obtained by PAC is particularly helpful when the diagnosis or the type of shock remains undetermined or mixed. Other types of patient that may benefit from PAC include those with unknown volume status despite adequate fluid resuscitation, those with severe cardiogenic shock, or those suspected to have severe underlying pulmonary artery hypertension or cardiac tamponade.

The major hemodynamic indices measured on PAC are cardiac output, systemic vascular resistance, pulmonary artery occlusion pressure, right atrial pressure, and mixed venous oxyhemoglobin saturation (SvO_2). These measurements are most useful diagnostically but can also be used to guide fluid resuscitation, titrate vasopressors, and assess the hemodynamic effects of changes in mechanical ventilator settings [28].

Hemodynamic support

There is no consensus about the clinical cut-off that warrants hemodynamic support in patients with undifferentiated shock due to the fact that shock can be present when patients are hypotensive, hypertensive, or normotensive. As a rule of thumb, when initial assessment and/or laboratory evaluation suspect the presence of shock, hemodynamic support with intravenous fluids (IVFs) should be initiated, followed by vasopressors if IVFs fail to restore adequate tissue perfusion. While the optimal end-organ perfusion pressure is unclear, in general, experts suggest maintaining the mean arterial pressure greater than 65 to 70 mmHg, since higher targets do not appear to be associated with a mortality benefit and may be associated with increased risk of cardiac arrhythmias [29]. The total volume infused is determined by the etiology of shock. Patients with obstructive shock from pulmonary embolism or cardiogenic shock from LV myocardial

infarction usually require small volumes of IVF (500 - 1000 mL), while those with RV infarction or sepsis often need 2 to 5L, and those with hemorrhagic shock frequently require volumes > 3 to 5L. While the optimal choice of fluid is unknown, most patients with septic shock are treated with crystalloids as Ringer's lactate or normal saline, and those with hemorrhagic shock should be preferentially treated with blood products.

Vasopressors are frequently required in the treatment of patients with undifferentiated shock to restore adequate tissue perfusion. It is worth emphasizing that vasopressors may cause harm in patients with hemorrhagic or hypovolemic shock. Hence, vasopressors should only be used as an additional form of hemodynamic support when aggressive resuscitation has failed to restore adequate tissue perfusion, or as a last resort for patients in extremis. The best initial vasopressor and the optimal target mean arterial pressure are unknown [30]. However, intravenous norepinephrine (Levophed) is the most commonly used agent. When tachyarrhythmia preclude the use of drugs with excessive beta-adrenergic activity, intravenous administration of phenylephrine (Neo-synephrine) is the usual alternative. Dobutamine is the most commonly used inotropic agent in patients who have cardiogenic shock. Dobutamine is often administered in combination with norepinephrine to compensate the fall in peripheral vascular resistance that occurs when low doses of dobutamine are used. Vasopressor support should be titrated according to the response to limit side effects. In general, the target is 65 or higher mean arterial pressure according to individualized care. Although targeting higher mean arterial pressures in patients with chronic hypertension resulted in increased arrhythmia, this complication was offset by a reduced need for renal replacement therapy [31].

Conclusion

Circularity failure of adequate oxygen delivery for cells and tissues defines shock. Patient with shock could be hypertensive, normotensive, or hypotensive. Early, the effects of shock could be overturned, however, inadequate delayed management may lead to irreversible multiorgan failure (MOF) and death. The clinical findings vary according to the etiology and the time of presentation whether in pre-shock, shock, or presented with end-organ failure. Undifferentiated shock may present with tachycardia; tachypnea; hypotension. The first step in adequate management is prompt assessment and securing of the airway, breathing, and circulation. Following initial assessment, the efforts should be directed toward a thorough diagnostic evaluation by laboratory and imaging studies. When initial assessment and/or laboratory evaluation suspect the presence of shock, hemodynamic support with intravenous fluids (IVFs) should be initiated, followed by vasopressors if IVFs fail.

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