American Journal of Emergency Medicine xxx (xxxx) xxx

Contents lists available at ScienceDirect

American Journal of Emergency Medicine

journal homepage: www.elsevier.com/locate/ajem

Identifying cardiogenic shock in the emergency department

Madison Daly [a], Brit Long [b], Alex Koyfman [c], Skyler Lentz [d,*]

[a] Division of Emergency Medicine, The University of Vermont Medical Center, United States of America
[b] SAUSHEC, Emergency Medicine, Brooke Army Medical Center, United States of America
[c] The University of Texas Southwestern Medical Center, Department of Emergency Medicine, United States of America
[d] Division of Emergency Medicine, Department of Surgery, The University of Vermont Larner College of Medicine, United States of America

ARTICLE INFO

Article history:
Received 4 July 2020
Received in revised form 16 September 2020
Accepted 17 September 2020
Available online xxxx

Keywords:
Cardiogenic shock
Cardiology
Myocardial infarction
Heart failure

ABSTRACT

Introduction: Cardiogenic shock is difficult to diagnose due to diverse presentations, overlap with other shock states (i.e. sepsis), poorly understood pathophysiology, complex and multifactorial causes, and varied hemodynamic parameters. Despite advances in interventions, mortality in patients with cardiogenic shock remains high. Emergency clinicians must be ready to recognize and start appropriate therapy for cardiogenic shock early.
Objective: This review will discuss the clinical evaluation and diagnosis of cardiogenic shock in the emergency department with a focus on the emergency clinician.
Discussion: The most common cause of cardiogenic shock is a myocardial infarction, though many causes exist. It is classically diagnosed by invasive hemodynamic measures, but the diagnosis can be made in the emergency department by clinical evaluation, diagnostic studies, and ultrasound. Early recognition and stabilization improve morbidity and mortality. This review will focus on identification of cardiogenic shock through clinical examination, laboratory studies, and point-of-care ultrasound.
Conclusions: The emergency clinician should use the clinical examination, laboratory studies, electrocardiogram, and point-of-care ultrasound to aid in the identification of cardiogenic shock. Cardiogenic shock has the potential for significant morbidity and mortality if not recognized early.

1. Introduction

In patients presenting to the emergency department with shock, cardiogenic shock (CS) is the primary cause in 14–15% of cases [1,2]. Although definitions vary, CS is a clinical diagnosis broadly defined as a state of low cardiac output with associated inadequate end-organ perfusion or tissue hypoperfusion secondary to cardiac dysfunction [3]. Commonly used criteria derived from the SHOCK trial consists of hypotension (systolic blood pressure (SBP) <90 mmHg or > 90 mmHg requiring vasopressor or inotrope use), evidence of end-organ hypoperfusion, and cardiac index (CI) <2.2 l/min/m^2 or pulmonary capillary wedge pressure (PCWP) ≥ 15 mmHg [3,4]. Although this definition is useful to standardize inclusion criteria for clinical trials, it is less useful for diagnosing CS in the emergency department (ED).

In the ED, CS can be challenging to identify because of the diverse presentations, overlap with other shock states (i.e. sepsis), poorly understood pathophysiology, complex and multifactorial causes, and varied hemodynamic parameters [5]. In the absence of invasive cardiac output (CO) and PCWP values, CS can be inferred using evidence of elevated filling pressures (i.e. pulmonary congestion or elevated jugular venous pressure (JVP)), clinical signs of hypoperfusion, SPB < 90 mmHg or need for vasopressors/inotropic support and a history or echocardiogram suggestive of cardiac failure. Mortality secondary to CS is high (~25–70%), but early recognition and intervention improve survival [5,6]. Emergency physicians can diagnose CS on admission and must maintain a high clinical suspicion when evaluating any critically ill patient with hemodynamic instability. This review will focus on recognition and evaluation of suspected CS using physical examination, laboratory assessment, and point-of-care ultrasound.

2. Methods

The authors searched PubMed and Google Scholar for articles using a combination of the keywords “cardiogenic shock”, “myocardial infarction” and “heart failure”. The search was conducted from the database's inception to August 2020. Authors evaluated case reports and series, retrospective and prospective studies, systematic reviews and meta-analyses, and other narrative reviews. Authors also reviewed guidelines and supporting citations of included articles. The literature search was restricted to studies published in English, with focus on the EM and critical care literature. Authors decided which studies to include for the review by consensus. When available, systematic reviews and

* Corresponding author at: 111 Colchester Ave, Attn: Surg-Emergency Medicine, Burlington, VT 05401, United States of America.
E-mail address: skylentz@gmail.com (S. Lentz).

https://doi.org/10.1016/j.ajem.2020.09.045
0735-6757/

meta-analyses were preferentially selected. These were followed sequentially by randomized controlled trials, prospective studies, retrospective studies, case reports, and other narrative reviews when alternate data were not available. A total of 72 articles were selected for inclusion in this narrative review. Of these, there were 3 systematic reviews and meta-analyses, 5 randomized controlled trials, 18 prospective studies, 22 retrospective studies, and 24 narrative reviews or expert consensus documents.

3. Discussion

3.1. Etiologies of cardiogenic shock

Most studies of CS focus on patients with CS secondary to myocardial infarctions (MIs) involving the left ventricle. Although MIs are the primary cause of CS (~70%), any cause of ventricular dysfunction and reduced CO or cardiac index (CO/body surface area) as a potential etiology must be considered [7]. It is important to obtain an electrocardiogram (ECG) as soon as CS is suspected; ST elevation in ≥2 contiguous leads suggests an acute MI (STEMI) and is an indication for urgent reperfusion [8]. Other causes include nonischemic right heart failure, myocarditis, takotsubo cardiomyopathy, hypertrophic cardiomyopathy, or valvular heart disease (Table 1). CS is also a challenging diagnosis, as it exists along a continuum rather than a static state, ranging from worsening heart failure to refractory shock with irreversible end organ damage (Fig. 1) [3]. CS becomes even more variable with the occurrence of secondary insults such as arrhythmias or progressive ischemia and acidosis [3]. It should be noted that in over 60% of cases, CS is not present on admission but later develops within 48 h of hospitalization as the patient progresses down the continuum of shock [9]. The occurrence of shock has a median time of onset of ~6 h post MI [10]. It is important to frequently reevaluate patient hemodynamics, symptoms, physical examination, and point-of-care ultrasound.

3.2. Mortality in cardiogenic shock

Although mortality secondary to CS is high [5], early recognition and intervention improve survival [6]. Using data which included the SHOCK trial registry, 30-day in-hospital mortality was 57%, based on 1217 patients diagnosed with CS secondary to left ventricle (LV) or right ventricle (RV) failure due to an acute MI [14]. Depending on risk factors, mortality ranges from 22% to 88% [14]. Risk factors associated with a higher mortality include shock on admission, increased age, previous coronary artery bypass grafting (CABG), inferior MI, older age, left main disease, creatinine >1.9 mg/dl, decreased SBP, anoxic brain injury, and clinical evidence of end-organ hypoperfusion [14,15]. In the GRACE trial, the rate of CS post STEMI decreased by 2.4% between 1999 and 2006, likely due to the increased use of percutaneous coronary intervention (PCI), an important form of early intervention for patients with MI complicated by CS [16]. Specifically, as the use of PCI increased by 37% and 18% in ST and non-ST elevation MI patients, respectively, the rate of CS decreased by 2.4% in ST elevation MI patients and 0.2% in non-ST elevation MI patients [16]. Even when stratifying patients based on risk factors, including anoxic brain injury, severely reduced ejection fraction (EF), end organ hypoperfusion, etc., PCI and CABG benefited both low and high-risk patients [14]. Early diagnosis and appropriate treatment remains, particularly in the case of myocardial ischemia, an important modifiable contributor to outcomes for patients with CS.

Additionally, the longer CS progresses, the more likely there will be a maladaptive inflammatory response secondary to an increase in cytokines like tumor necrosis factor (TNF)-alpha and interleukin (IL)-6, which inhibit cardiac activity [5,17,18]. There is also an increase of vasopressin and angiotensin II, which increases afterload, worsens CO, and increases water and salt retention, contributing to pulmonary edema [5]. Nitric oxide (NO) is increased through the activation of NO synthase, leading to vasodilation and myocardial depression [17]. These maladaptive responses lead to myocardial ischemia, worsening cardiac tissue damage, depressed CO, and a secondary distributive shock. It should be noted that some cases of CS are iatrogenic, when patients on the verge of CS are treated with aggressive diuretics, nitrates, beta blockers, angiotensin-converting enzyme (ACE) inhibitors, and morphine [19]. Therefore, as the first physician to evaluate patients, emergency physicians need to identify and treat CS in a time-sensitive and clinically appropriate manner.

3.3. History and Clinical Examination

The presenting complaint of patients with CS may include dyspnea, orthopnea, chest pain, fatigue, altered mental status, and/or lower extremity swelling. Physical examination may reveal signs of congestion including peripheral edema, JVD, crackles/rales on auscultation, and signs of hypoperfusion such as cool, poorly perfused extremities (Table 2). Although there are few resources that describe the frequency of examination findings for CS specifically, there have been several studies that evaluate examination findings associated with acute heart failure—a potential precursor of cardiogenic shock. A meta-analysis by Martinale et al. [20] provides insight on the diagnoses of acute heart failure in the ED using history and examination findings. Specifically, orthopnea (positive likelihood ratio (LR) 1.9), JVD (positive LR 2.8), hepatojugular reflex (positive LR 2.2), lower extremity edema (positive LR 1.9), and rales (positive LR 1.8) increase the likelihood that a dyspneic patient has heart failure. S3 has the highest positive LR at 4.0, but despite its high specificity for heart failure (97.7%), the sensitivity is low (12.7%) [20,21]. Careful auscultation should be performed to listen for a murmur; a new murmur suggests a structural or valvular abnormality that may be the cause or a contributor to CS [8]. Many patients have a sinus tachycardia to compensate for a reduced stroke volume [11]. In a small retrospective review of 30 patients in undifferentiated shock, those with CS (compared to patients with distributive and hypovolemic shock, respectively) were more likely to have JVD (80% compared to 0% and 20%, respectively), cold skin (57.1% compared to 14.3% and 28.5%, respectively), and pulmonary rales (75% compared 16.7% and 8.3%, respectively) [22]. In another prospective study with 68 patients, physicians used specific clinical examination findings to differentiate categories of shock. CS was categorized by SBP less than 90 mmHg, signs of poor perfusion (cold hands, poor capillary refill, and weak pulse), elevated JVP > 7 cmH_2O, S3 gallop, and crackles to 1/3 of the lungs. Of 68 patients, 11 met criteria for CS. In patients with echocardiographic evidence of low cardiac output, elevated JVP predicted CS with an accuracy of 80%, which was unchanged when adding the presence of crackles [23].

Table 1
Causes of cardiogenic shock [5,10,11]. 70% of cardiogenic shock cases are caused by acute myocardial infarctions [7]. Effects of acute MI with approximate percentages taken from the results of the SHOCK trial registry [12,13].

Acute Myocardial Infarction and Associated Complications (Myocardial Infarction Causes 70% of the Cases of Cardiogenic Shock)
Left Ventricular Failure (79%)
Acute Mitral Regurgitation (7%)
Ventricular Septal Defect (4%)
Isolated Right Ventricular Infarction (3%)
Tamponade or Cardiac Rupture (2%)
Other
Left ventricular outflow tract or filling obstruction
Right Ventricular Failure
Myocarditis
Myocardial depression secondary to septic shock
Cardiomyopathy
Myocardial contusion
Acute aortic insufficiency
Iatrogenic from medications or medication toxicity
Tachy- or bradyarrhythmia

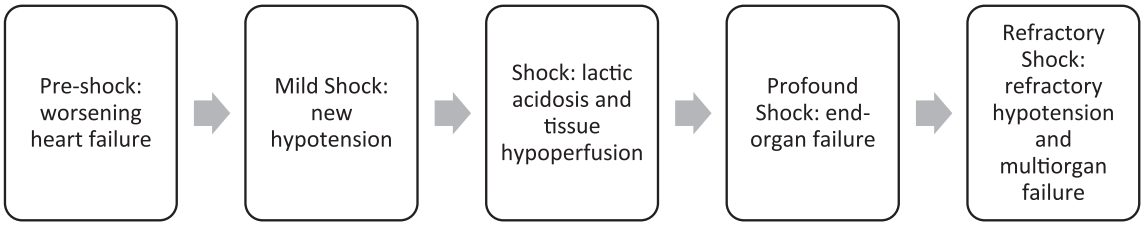

Fig. 1. A representation of the continuum of cardiogenic shock [3]. This spectrum may deviate with secondary insults (e.g. new arrhythmias).

Table 2
Physical exam components seen in acute heart failure and subsequent cardiogenic shock [8,10,22,24,25].

Signs of Congestion
Jugular Venous Distension
Jugular Venous Pressure (elevated >6–8 cmH_2O)
Pulmonary Rales or Crackles
Peripheral Edema
Cardiac Ascites
Hepatomegaly
Orthopnea
Abdominal Jugular Reflux
Signs of Impaired Perfusion and Hemodynamic Compromise
Cold Extremities
Delayed Capillary Refill
Hypotension
Narrowed Pulse Pressure
Tachycardia or Symptomatic Bradycardia
Tachypnea
Confusion/Altered Mental Status
Oliguria
Other
Ventricular S3 Gallop
Displaced PMI
New Murmur

JVD, at rest or induced by abdominal pressure, or an elevated JVP>7 cm H_2O identified patients with an increased PCWP ≥18 mmHg with a sensitivity and specificity of 81% and 80%, respectively [26]. JVP may be difficult to assess due to body habitus and positioning of the patient (the head of the bed should be placed at 45 degrees which can be difficult in patients with severe orthopnea) [27]. JVP is measured by calculating the highest pulsation point in centimeters above the sternal angle and then adding 5 (as the right atrium is 5 cm below the sternal angle), which correlates to a pressure in cm H_2O (Fig. 2). Elevated values are often considered greater than 6–8 cm H_20 [24]. Of note, elevated JVP is associated with increased risk of mortality, with a relative risk (RR) of 1.52 [24].

A SBP of <90 mmHg may not be seen in every case of cardiogenic shock. In a study using the SHOCK trial registry, 5.2% of CS patients did not have overt hypotension, though they did demonstrate evidence of peripheral hypoperfusion and low CO. [25] A low cardiac output leads to an adaptive catecholamine release in early CS, which increases systemic vascular resistance (SVR) and transiently maintains blood pressure, though generally with a narrow pulse pressure [28]. Normotension, despite a low cardiac output, can be explained by the equation, systemic mean arterial pressure = CO x SVR. In one retrospective study, those with impaired peripheral perfusion still had a high mortality rate of 43% despite a SBP > 90 mmHg, though this was less than the 66% mortality rate observed in those with impaired perfusion and a SBP < 90 mmHg [28]. The non-hypotensive or occult cardiogenic shock presents a challenge requiring a careful clinical examination to identify subtle findings of hypoperfusion (Table 2).

Patients with clinically significant pulmonary edema on imaging can present with wheezing or clear lung sounds rather than rales [29]. In one study, pulmonary congestion was not seen in 28% of cases of CS secondary to MI and LV failure [25]. Those without pulmonary vascular congestion are sometimes called "cold and dry"; they have cool extremities with a delayed capillary refill from a high SVR and low cardiac output but may not have an elevated PCWP to cause pulmonary edema [8]. It should be noted those with CS primarily from right ventricular failure may not have pulmonary edema. Rather, they may have

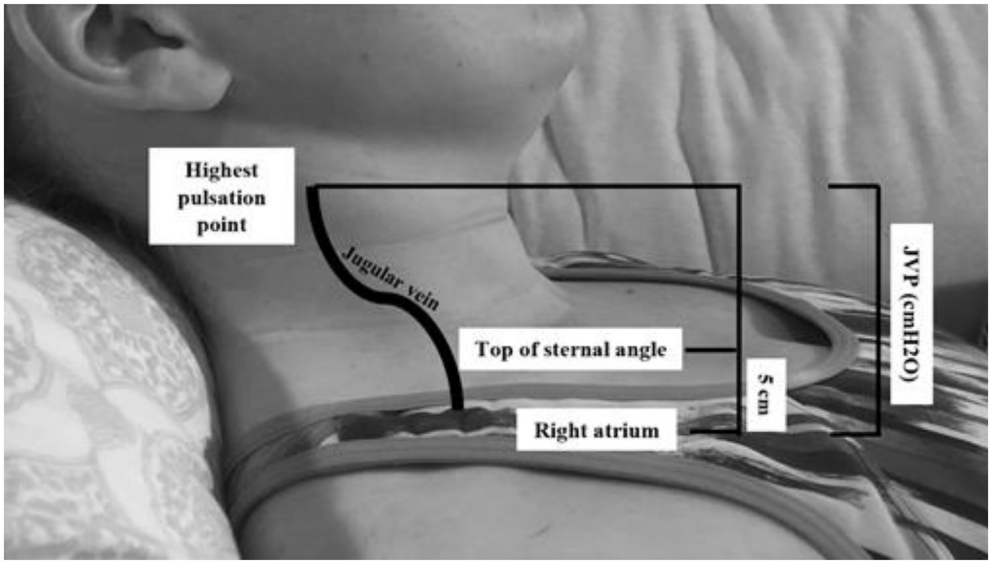

Fig. 2. Measuring Jugular Venous Pressure [24].

more pronounced JVP elevation, hepatomegaly, and peripheral edema [8]. They share the findings of poor peripheral perfusion and tissue hypoperfusion as seen in those with LV failure [8].

Though no single examination finding is definitive, a detailed evaluation for signs of congestion and peripheral hypoperfusion along with a careful review of vital signs may reveal early findings of CS.

3.4. Diagnostic Studies in Cardiogenic Shock

Although there is not a single test that can be used to diagnose CS, laboratory results and diagnostic studies are important for the evaluation and management of suspected CS in the ED as they contribute to the overall clinical picture and prognosis.

3.4.1. Laboratory studies

A basic metabolic panel, magnesium, complete blood cell count, lactic acid, troponin, NT-pro-BNP, and a hepatic panel should be obtained if CS is suspected. Laboratory studies may reveal metabolic acidosis, renal hypoperfusion with resulting acute kidney injury (AKI), leukocytosis or other inflammatory abnormalities, and possible evidence of cardiac ischemia with an elevated troponin [11,30]. Other laboratory abnormalities associated with CS include hypoalbuminemia, increased inflammatory cytokines, and diabetes-independent hyperglycemia [31-34].

NT-pro-BNP levels are generally elevated in CS, and although there is conflicting evidence, elevated levels are thought to be associated with an increased mortality [8,32,35,36]. An elevated NT-pro-BNP is not specific to heart failure nor CS. However, it may be useful in the evaluation of the dyspneic patient in the ED in the correct context. NT-pro-BNP displays variable sensitivity and specificity for acute heart failure based on the cutoff value; as expected a higher cutoff yields an increasing specificity at the cost of a decreasing sensitivity. A level ≥ 1000 pg/ml demonstrates a sensitivity of 84.4%, specificity of 65.5%, and a positive LR of 2.7. At ≥1500 pg/ml, sensitivity decreases to 75.5% while specificity increases to 72.9% with a positive LR 3.1. Alternatively, a low NT-pro-BNP suggests against heart failure as the cause of dyspnea. When the cut-off is lowered to ≥300 pg/ml, the negative LR is 0.09, suggesting the potential use of the test to rule out acute heart failure [20,21].

Lactate elevation is not specific to sepsis and can be seen in any shock state, including CS. Hyperlactatemia in shock results from increased production during a stressed/inflammatory state and from hypoxia-induced anaerobic glycolysis [18]. In a small study comparing 7 CS patients to 7 healthy volunteers, lactate levels were significantly

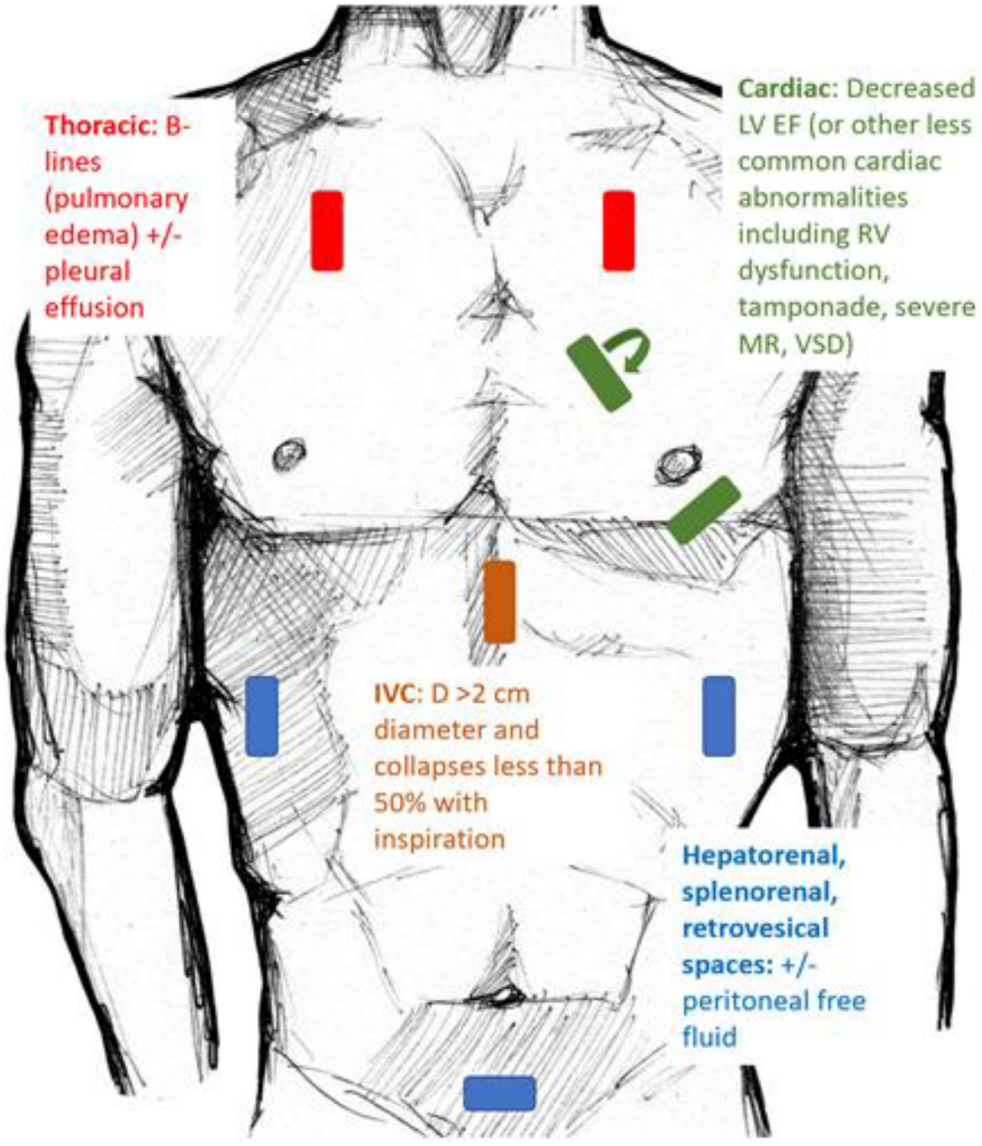

Fig. 3. RUSH bedside US exam for the evaluation of undifferentiated hypotension with associated findings suggestive of CS [45].

elevated in patients with CS. Using infusions with labeled lactate, they also showed there was no significant change in lactate clearance, and therefore elevated levels were likely due to increased production [18]. Elevated lactate is also an important prognostic factor [37]. In two observational studies including CS patients, elevated lactate levels >2 mmol/l were associated with increased mortality [7,34]. Specifically, an increase in blood lactate (per mmol/l) increased the risk of mortality with an adjusted odds ratio (OR) of 1.4 [7,38]. Lactate should be trended to assess for persistence or clearing in response to therapy [9].

As most cases of CS are secondary to acute coronary syndrome, troponin is an important test to obtain, in the appropriate clinical context, while evaluating a patient with undifferentiated hypotension. In a retrospective analysis of 700 patients who presented to the ED with hypotension, a troponin ≥0.1 ng/ml was independently predictive (OR 37.5 95% 95% CI 7.1–198.2) of a cardiac etiology [1]. Though associated with a cardiogenic etiology, an elevated troponin was also seen in 13.3% of non-cardiogenic causes of hypotension, and many of the cardiogenic causes of hypotension did not have an elevated troponin, limiting its sensitivity and specificity as a single test [1]. The troponin may have prognostic value. In a cohort of patients that presented with non-ST elevation acute coronary syndrome, the degree of troponin elevation was associated with an increased risk of CS (OR 1.87, 95% CI 1.61 to 2.18) and mortality [39].

CS results in venous hypertension with reduced renal blood flow and subsequent reduced glomerular filtration rate, leading to AKI secondary to acute tubular necrosis [40]. Absent of etiology, AKI is an indicator of shock severity and is associated with fluid retention, electrolyte abnormalities, acidosis, and poor outcomes [37]. In an observational study of 154 CS patients, 31% developed AKI based on a creatinine rise of ≥0.3 mg/dl or ≥ 50% increase from baseline. In the same study, AKI was independently associated with 90-day mortality (OR 12.2) [40].

Hepatic injury is also common in CS due to a combination of arterial hypoperfusion and venous congestion [37]. In an observational analysis using data from the CardShock registry, 58% of patients had abnormally elevated alanine transaminase (ALT) [41]. In the same study, >20% increase in ALT over 24 h was associated with increased mortality. Hypoxic hepatitis, defined as an increase of aminotransferase levels >20 times the upper limit of normal, was seen in 18% of CS patients from the IABP-SHOCK II trial. These patients had a 68% short term mortality rate, which was higher than those without hypoxic hepatitis [42].

3.4.2. Electrocardiogram

The patient should be placed on telemetry monitoring and an ECG should be obtained urgently to evaluate for signs of ischemia (e.g. ST segment elevations or depressions), a STEMI, or arrhythmia [8,11,29]. In a retrospective cohort study of admissions with acute-MI associated CS, 50.8% had arrhythmias on admission, with atrial fibrillation, ventricular fibrillation, and ventricular tachycardia being the most common [43]. In a meta-analysis that compared ECG findings with acute and chronic heart failure—but not specifically CS—there was a positive association with ischemic changes (positive LR 2.9), T wave inversions (positive LR 2.4), and atrial fibrillation (positive LR 2.2). While there was an association with ST depressions (positive LR 2.0 (95% CI 1.0–3.8)) and the diagnosis of acute heart failure, confidence intervals included 1.0 [20].

3.4.3. Chest Radiography

Chest x-rays can demonstrate a variety of findings in heart failure and CS. Findings, including Kerley B-lines, interstitial/alveolar/pulmonary edema, cephalization, and pleural effusions, are specific to heart failure in the dyspneic patient (89.2–98.9%), although they lack sensitivity (54.7–56.9%). Alternatively, cardiomegaly is relatively sensitive (74.7%) but not as specific (61.7%) [20]. A normal chest x-ray should not exclude heart failure or CS. In a retrospective secondary analysis of the ADHERE registry with over 85,000 patients, over 18% of patients with heart failure had no signs of congestion on chest x-ray [44].

3.4.4. Point-of-care echocardiogram for evaluating cardiogenic shock

In those with suspected CS an urgent echocardiogram is required [29]. The initial exam can be quickly performed as a point-of-care ultrasound by the emergency clinician. The RUSH ultrasound examination (Fig. 3) can assist in determining the specific etiology of a patient with undifferentiated shock by evaluating "the pump, the tank, and the pipes," or rather the heart, the inferior vena cava (IVC)/intra-abdominal and pleural compartments, and large vessels including aorta [45]. In CS, transthoracic echocardiogram classically demonstrates a hypodynamic, dilated LV, with poor LV contraction and associated inadequate motion of the anterior leaflet of the mitral valve during systole and diastole (i.e. poor contractility). Visual estimation, rather than quantitative measurements of the EF by emergency physicians through simply "eyeballing" LV function is an adequate assessment to detect a low EF in the acute setting. In a prospective study emergency physicians performing a limited echocardiogram correctly detected a low EF, when compared to a formal echocardiogram, with a sensitivity of 98% and a specificity of 86% [46]. Importantly, a reduced EF is not necessary to make the diagnosis of cardiogenic shock; even with decreased LV contractility, CS patients may not have a severe reduction in LVEF [19,47]. In fact, the mean EF in a cohort of CS patients is about 30%, which is reduced but higher than expected [4].

In CS, the IVC, which is an indirect measurement of effective intravascular volume, usually has a diameter of >2 cm diameter and

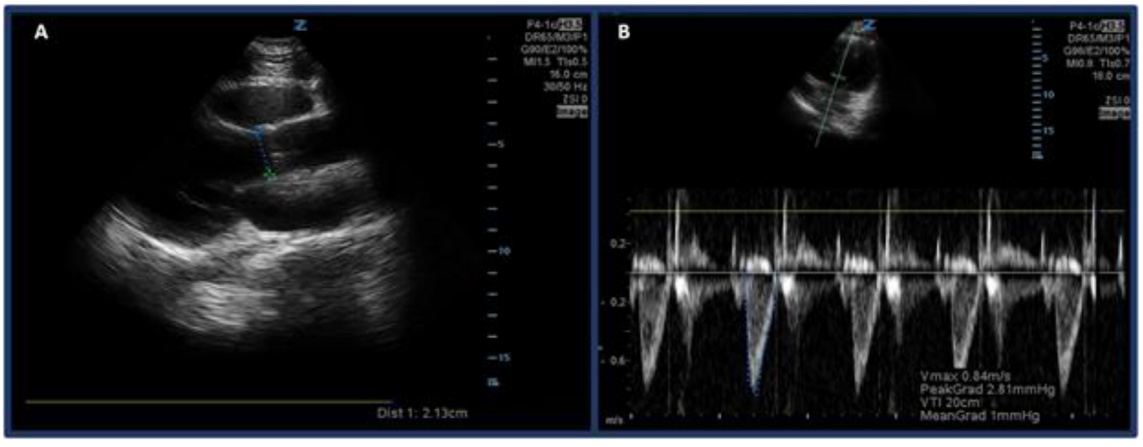

Fig. 4. A) Parasternal long axis view with LVOT diameter of 2.13 cm. B). Apical-5-chamber view using PW doppler to measure VTI of 20 cm, a normal VTI is 18–22 cm [55]. Using the eqs. SV=VTIxD^{2}x0.785 and CO=SV x HR, with a HR of 85, SV = 71 ml, and CO = 5 L/min. Using the eq. CI = CO/BSA, CI = 3.1 L/min/m^2 (a normal cardiac index).

collapses less than 50% with inspiration. These findings correlate with an elevated central venous pressure [48]. However, the IVC assessment may be inaccurate if the patient has already received vasodilators, diuretics, and/or is mechanically ventilated.

Thoracic windows are likely to show pulmonary edema identified by ≥3 B-lines (i.e. vertical, comet tail artifacts) in at least 2 areas of the bilateral chest, which are the result from fluid accumulation in the interstitium [49]. Lung ultrasound examining for pulmonary edema has a positive LR 7.4 and a higher sensitivity and specificity for pulmonary edema when compared to chest x-ray [20]. Along with pulmonary congestion, there may be pleural and peritoneal fluid on RUSH examination [29].

A meta-analysis found the RUSH protocol to be both sensitive and specific (0.89 and 0.97, respectively) in the diagnosis of CS [50]. Despite a high positive LR of 22.29, there was only a moderate negative LR of 0.17, suggesting the RUSH examination should not be used to exclude CS [50]. The RUSH examination should be used in the context of a careful history and physical examination rather than used alone to diagnose cardiogenic shock.

The echocardiogram can also be used to evaluate for evidence of right ventricular failure. This may include a RV > ⅔ the size of the LV in an apical view and flattening of the interventricular septum [51]. The RV systolic function can be assessed visually or by obtaining objective measurements. A tricuspid annular plane systolic excursion (TAPSE), assessing the maximal systolic excursion of the lateral tricuspid annulus using the M-mode in an apical 4 chamber view, evaluates RV systolic function [52]. A TAPSE of <17 mm suggests RV dysfunction; a low TAPSE is associated with a lower CI and decreased survival [53].

Rather than estimating cardiac function through "eyeballing," a means to non-invasively assess CO with ultrasound is to first determine the stroke volume (SV) using left ventricular outflow tract velocity time interval (LVOT VTI), or the velocities of blood flow at the aortic outflow tract, and LVOT diameter. Specifically, SV, or the amount of blood ejected through the left ventricle per beat, is estimated by the LVOT VTI × cross sectional area of the LVOT [VTI (cm) x D^2 x0.785 (cm^2)] [54]. The SV can be multiplied by the heart rate to estimate the cardiac output. The LVOT VTI can be measured serially to assess the response to treatment. To measure LVOT diameter, place the phased array probe in the parasternal long axis view and measure the distance of the LVOT just above the aortic valve while in mid-systole. VTI is measured in the apical-5-chamber view (Fig. 4). Using the pulsed-wave doppler mode, the doppler wave is placed just above the aortic valve and doppler waveforms are recorded. The axis should be aligned with the outflow tract to avoid over/under estimations. If available on the ultrasound machine, select the "LVOT VTI" measurement tool, and measure the waveform of one ejection period [52]. Normal LVOT VTI ranges from 18 to 22 cm, although possibly lower with patients with HR >95 bpm [55]. In patients with atrial fibrillation, VTI measurements will likely be an underestimate of true value, and therefore averaging 3–5 consecutive waveforms is suggested. In a retrospective study of patients with heart failure, a low LVOT VTI of <10 cm was associated with 12-month adverse outcomes including death and need for left-ventricular assist device (LVAD) implantation [54].

3.4.5. Central venous oxygen saturation

Invasive pulmonary arterial catheters are not routinely placed in the emergency department and should not be routinely utilized for ED management of cardiogenic shock. However, if a central line is present in the upper body, a central venous oxygen saturation ($S_{CV}O_2$) can easily be obtained by sampling and performing co-oximetry on venous blood (i.e. venous blood gas) from the distal superior vena cava. The $S_{CV}O_2$ is used as a surrogate of the mixed venous saturation (from the pulmonary artery) and represents the desaturated hemoglobin, and thus oxygen delivery and consumption, returning to the right side of the heart from the systemic tissue beds [56]. Cardiac output, hemoglobin, and arterial oxygen saturation are the major determinants of oxygen delivery [57]. The $S_{CV}O_2$ is generally reduced, due to a decrease in oxygen

Table 3
Cardiogenic Shock Diagnosis and Management Pearls and Pitfalls.

Problem	Pitfall	Pearl
Diagnosis		
Occult cardiogenic shock with normotension	Not performing a careful history and examination.	Up to 5.2% of patients with cardiogenic shock are normotensive. Physical examination should focus on signs of hypoperfusion (e.g. cool and poorly perfused extremities, altered mental status, oliguria, etc.) and congestion (e.g. pulmonary crackles, an elevated JVP > 6–8 cm, S3 etc.).
Misdiagnosis	Diagnosing septic shock, rather than cardiogenic shock because of an elevated lactate or hypotension.	An elevated lactate is not specific to septic shock; cardiogenic shock should be on the differential diagnosis. There is no single laboratory study to diagnose cardiogenic shock. Suggestive studies include an elevated troponin, NT-pro-BNP, elevated creatinine, and low $S_{CV}O2$ (e.g. < 60%).
	Not evaluating the ECG.	An ECG may reveal ischemia or arrhythmia. Myocardial infarction is the most common cause of cardiogenic shock.
Imaging	Relying on chest x-ray alone to diagnose cardiogenic shock	Chest x-ray can demonstrate a variety of findings. While findings are specific, they should not be used to exclude pulmonary congestion.
	Not performing a point-of-care ultrasound.	The classic ultrasound findings in cardiogenic shock include a reduced EF (mean EF of 30%), IVC > 2 cm, and/or signs of pulmonary edema with ≥3 B-lines in bilateral lungs (higher sensitivity and specificity than chest x-ray for pulmonary edema).
		Isolated RV dysfunction causing shock may be present.
		An estimated stroke volume can be measured by ultrasound using left ventricular outflow tract velocity time interval.
Management		
Hypotension	Starting an inotrope first, potentially worsening hypotension in those with an inappropriately low SVR.	Target MAP ≥65 mmHg. Start with norepinephrine to normalize MAP first then add an inotrope for ongoing signs of hypoperfusion.
	Misidentifying cardiogenic shock as sepsis and administering excess volume.	Use history, examination, and point-of-care ultrasound to identify cardiogenic shock.
Respiratory failure	Starting NIPPV in those who are preload dependent leading to hemodynamic instability.	Treat with oxygen or NIPPV. NIPPV works best in those who are not preload dependent (i.e. those with a pathologically elevated central venous pressure), have reduced LV function, and pulmonary congestion.
Refractory shock or structural abnormality	Failure to refer early to advanced centers for mechanical support.	Emergent consult with cardiothoracic surgery and interventional cardiology is recommended. When patients

Table 3 (*continued*)

Problem	Pitfall	Pearl
		are properly selected, mechanical support devices (e.g. axial flow pumps, intra-aortic balloon pump, veno-arterial extracorporeal membrane oxygenation, etc.) may improve outcomes.

delivery in severe anemia or low flow states such as CS [56]. Though variability exists, a $S_{CV}O_2$ of >70% is considered normal in healthy individuals [57]. In other words, 25–30% of the oxygen content is removed from hemoglobin as it passes through the global tissue beds. Though the $S_{CV}O_2$ may be low in any shock state, it has been demonstrated to be lower in those with cardiac failure. In an observational study of a critical care population the mean $S_{CV}O_2$ in those with cardiac failure was 60% compared to 70% overall [58]. In an ED population, the mean $S_{CV}O_2$ in decompensated CS was 32% in the severely decompensated group and 51% in the mildly decompensated group [59]. The authors suggest sampling $S_{CV}O_2$ may aid in detecting occult cardiogenic shock [59]. Therefore, a low $S_{CV}O2$ (e.g. < 60%) can support the diagnosis of cardiogenic shock and be used to trend the response to therapy [9].

3.5. Treatment

The focus of this review is on the identification of CS, but a few points on treatment are warranted (Table 3). The primary goal is stabilization of shock to maintain organ perfusion while searching for an underlying treatable cause (i.e. MI, arrhythmia, etc.). Norepinephrine is associated with less arrhythmias than dopamine [60] and is the vasopressor of choice for initial stabilization of shock [8,9,56,61]. Norepinephrine stimulates beta-1 adrenergic receptors to increase cardiac contractility and alpha-1 receptors to induce vasoconstriction and raise the blood pressure [62]. A goal MAP ≥65 mmHg or higher in those with chronic hypertension is recommended [9]. A MAP <65 mmHg in the first 24 h in those with CS is associated with an increased mortality (adjusted OR 2.0, 95% CI 1.4–3.0) when compared to a MAP ≥65 mmHg [63]. A recent randomized trial suggests improved outcomes in those with post-MI CS with norepinephrine as compared to epinephrine; both groups were allowed to use dobutamine [64]. Those with an SBP < 90 mmHg and examination evidence of low CO, such as cool extremities, and high SVR should be stabilized first on norepinephrine. If hypoperfusion persists, an inotrope such as dobutamine or milrinone may be added [6]. Dobutamine or milrinone, if started first, may worsen hypotension through their vasodilatory effects [62]; as many as 18% of patients with CS have an additional inflammatory mediated distributive, low SVR shock [17]. Dobutamine is rapidly titratable and the preferred, first line inotrope [9]. If bradycardia is present, epinephrine can be considered [6]. In those suspected to have hypovolemia, without signs of pulmonary edema, a small rapidly delivered 250–500 mL fluid bolus can be cautiously trialed [3,8,29]. If no improvement in hemodynamics is noted, fluids should be discontinued. If there is evidence of acute heart failure or CS, beta blockers and renin-angiotensin-aldosterone system antagonists should be avoided until hemodynamic stabilization has been achieved [6,29].

Up to 80% of patients with CS develop respiratory failure [65]. Hypoxemia should be managed, targeting a saturation of >90% with simple oxygen, non-invasive positive pressure (NIPPV), or intubation as needed [8]. Hypotension prior to intubation is associated with cardiac arrest and poor outcomes [66-68]. Stabilization and normalization of the blood pressure should be attempted before intubation. High flow nasal cannula (HFNC) generates low levels of positive end expiratory pressure (PEEP) and is being investigated in heart failure [69]. HFNC is an option if NIPPV is not tolerated [70]. NIPPV has demonstrated benefit in cardiogenic pulmonary edema [71]. In the hypotensive patient, NIPPV must be used with caution with a low starting pressure (i.e CPAP of 5–8 cm H_2O) due to a risk of worsening hypotension [70]. The mask should be removed immediately with any signs of hemodynamic deterioration. NIPPV will be most beneficial to patients who are not preload dependent (i.e. those with a pathologically elevated central venous pressure), have reduced LV function, and pulmonary congestion [65]. NIPPV in patients with cardiogenic shock from isolated right ventricular failure is not generally recommended as it may worsen RV afterload and decrease preload [51,70].

Along with stabilization, treatment of the underlying cause is mandatory. As MIs are the primary cause in 70% of cases of cardiogenic

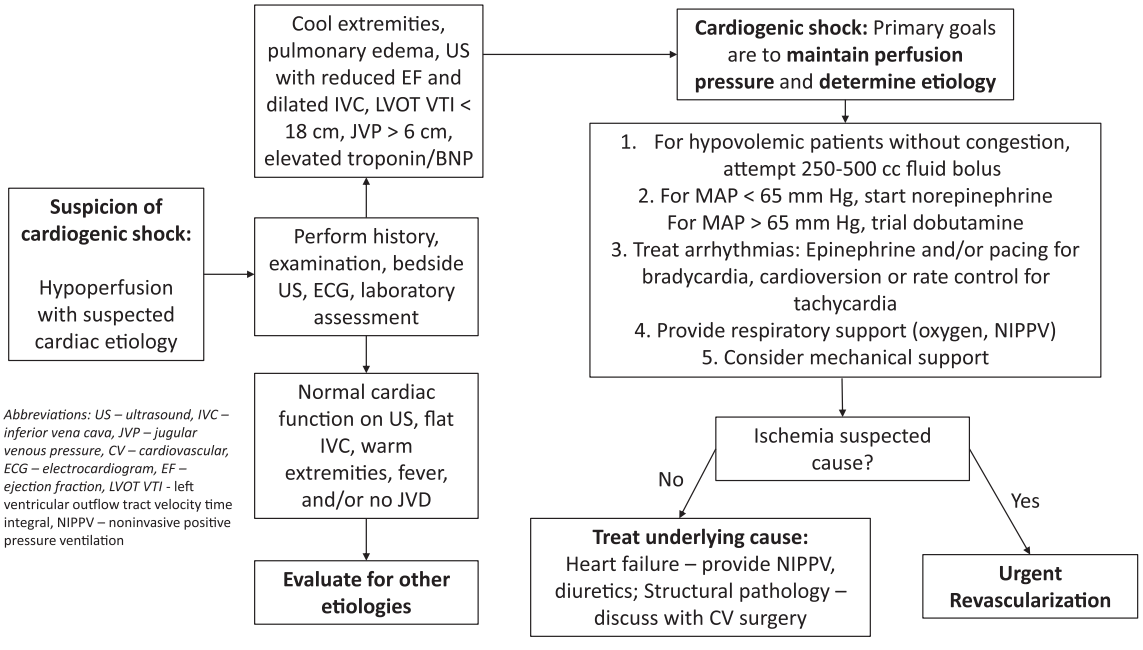

Fig. 5. Recommended evaluation of a patient with potential cardiogenic shock.

shock [7], urgent revascularization is recommended and improves long-term outcomes if MI is the underlying cause [8,72]. Tachy- or brady-arrhythmias should be treated if thought to be contributory. In the severest cases of shock, treatment with mechanical support devices (e.g. axial flow pumps, intra-aortic balloon pump, veno-arterial extra-corporeal membrane oxygenation, etc.,) may be considered in consultation with the cardiology team [8,27,29]. These devices may reduce the need for vasopressor and inotropic support and improve outcomes by reducing myocardial oxygen demand and thus ischemia [8]. Structural or valvular complication should be suspected with a new murmur or echocardiography findings of ventricular free wall rupture or VSD [8]. A cardiac surgery consultation should be obtained urgently if a structural complication is suspected.

3.6. Recommended evaluation pathway

As discussed, there is no single examination finding or laboratory test that can definitively diagnose CS. When there is a high suspicion of CS in the setting of hypotension or signs of hypoperfusion, we suggest using history, a detailed physical examination, point-of-care ultrasound, laboratory analysis, and ECG to aid in diagnosis (Fig. 5 and Table 3). An arterial catheter should be considered to monitor blood pressure and guide treatment [29]. Beyond a focused cardiac and pulmonary examination, physical examination should evaluate for JVD, urine output, and extremity perfusion. The RUSH examination and calculation of EF/CO/CI through LVOT VTI measurements are valuable adjuncts to the evaluation [45]. Using a comprehensive approach to evaluate for CS will create a better understanding of this heterogeneous disease and help guide management [29].

3.7. Limitations

This is a narrative review, and thus pooling of data from individual studies was not conducted. Much of the included literature consists of studies conducted in non-ED settings, and thus generalizing these studies to the ED setting is challenging. The majority of the studies consisted of small sample sizes, and most of the included resources consisted of retrospective studies, narrative reviews, guidelines, or expert consensus documents. Where appropriate, higher quality studies including an ED population of acute heart failure and acute coronary syndrome were included. Much of the included literature evaluated history and examination findings associated with heart failure. Few randomized controlled trials or prospective studies were available on the ED evaluation and management of cardiogenic shock.

4. Conclusions

Cardiogenic shock is difficult to diagnose in the ED, has a high mortality rate, and exists on a continuum. The emergency clinician should use a careful history, physical examination, laboratory studies, ECG, and point-of-care echocardiography to aid in the identification of CS. Early identification, stabilization, and treatment improve survival.

Declaration of Competing Interest

NONE.

Acknowledgements

MD, BL, AK, and SL conceived the idea for this manuscript and contributed substantially to the writing and editing of the review. This manuscript did not utilize any grants, and it has not been presented in abstract form. This clinical review has not been published, it is not under consideration for publication elsewhere, its publication is approved by all authors and tacitly or explicitly by the responsible authorities where the work was carried out, and that, if accepted, it will not be published elsewhere in the same form, in English or in any other language, including electronically without the written consent of the copyright-holder. This review does not reflect the views or opinions of the U.S. government, Department of Defense, U.S. Army, U.S. Air Force, or SAUSHEC EM Residency Program.

References

[1] Henning DJ, Kearney KE, Hall MK, Mahr C, Shapiro NI, Nichol G. Identification of hypotensive emergency department patients with cardiogenic etiologies. Shock. 2018; 49(2):131–6. https://doi.org/10.1097/SHK.0000000000000945.
[2] Gitz Holler J, Jensen HK, Henriksen DP, et al. Etiology of shock in the emergency department: a 12-year population-based cohort study. Shock. 2019;51(1):60–7. https://doi.org/10.1097/SHK.0000000000000816.
[3] Bellumkonda L, Gul B, Masri SC. Evolving concepts in diagnosis and Management of Cardiogenic Shock. Am J Cardiol. 2018;122(6):1104–10. https://doi.org/10.1016/j.amjcard.2018.05.040.
[4] Hochman JS, Sleeper LA, Webb JG, et al. Early revascularization in acute myocardial infarction complicated by cardiogenic SHOCK. SHOCK investigators. Should we emergently Revascularize occluded coronaries for cardiogenic shock. N Engl J Med. 1999;341(9):625–34. https://doi.org/10.1056/NEJM199908263410901.
[5] Jones TL, Nakamura K, McCabe JM. Cardiogenic shock: evolving definitions and future directions in management. Open Heart. 2019;6(1):e000960. https://doi.org/10.1136/openhrt-2018-000960.
[6] van Diepen S, Katz JN, Albert NM, et al. Contemporary Management of Cardiogenic Shock: A Scientific Statement From the American Heart Association. Circulation. 2017;136(16). https://doi.org/10.1161/CIR.0000000000000525.
[7] Harjola V-P, Lassus J, Sionis A, et al. Clinical picture and risk prediction of short-term mortality in cardiogenic shock. Eur J Heart Fail. 2015;17(5):501–9. https://doi.org/10.1002/ejhf.260.
[8] Cyrus Vahdatpour, David Collins, Sheldon Goldberg. Cardiogenic shock. J Am Heart Assoc. 2019;8(8):e011991. https://doi.org/10.1161/JAHA.119.011991.
[9] Levy B, Bastien O, Karim B, et al. Experts' recommendations for the management of adult patients with cardiogenic shock. Ann Intensive Care. 2015;5(1):52. https://doi.org/10.1186/s13613-015-0052-1.
[10] Khalid L, Dhakam SH. A review of cardiogenic shock in acute myocardial infarction. Curr Cardiol Rev. 2008;4(1):34–40. https://doi.org/10.2174/157340308783565456.
[11] Alpert JS, Becker RC. Cardiogenic shock: elements of etiology, diagnosis, and therapy. Clin Cardiol. 1993;16(3):182–90. https://doi.org/10.1002/clc.4960160305.
[12] Hochman JS, Boland J, Sleeper LA, et al. Current spectrum of cardiogenic shock and effect of early revascularization on mortality. Results of an international registry. SHOCK registry investigators. Circulation. 1995;91(3):873–81. https://doi.org/10.1161/01.cir.91.3.873.
[13] Hochman JS, Buller CE, Sleeper LA, et al. Cardiogenic shock complicating acute myocardial infarction–etiologies, management and outcome: a report from the SHOCK Trial Registry. SHould we emergently revascularize Occluded Coronaries for cardiogenic shocK? J Am Coll Cardiol. 2000;36(3 Suppl A):1063–70. https://doi.org/10.1016/s0735-1097(00)00879-2.
[14] Sleeper LA, Reynolds HR, White HD, Webb JG, Dzavík V, Hochman JS. A severity scoring system for risk assessment of patients with cardiogenic shock: a report from the SHOCK trial and registry. Am Heart J. 2010;160(3):443–50. https://doi.org/10.1016/j.ahj.2010.06.024.
[15] Sakamoto K, Matoba T, Mohri M, et al. Clinical characteristics and prognostic factors in acute coronary syndrome patients complicated with cardiogenic shock in Japan: analysis from the Japanese Circulation Society cardiovascular shock registry. Heart Vessels. 2019;34(8):1241–9. https://doi.org/10.1007/s00380-019-01354-9.
[16] Fox KAA, Steg PG, Eagle KA, et al. Decline in rates of death and heart failure in acute coronary syndromes, 1999-2006. JAMA. 2007;297(17):1892–900. https://doi.org/10.1001/jama.297.17.1892.
[17] Kohsaka S, Menon V, Lowe AM, et al. Systemic inflammatory response syndrome after acute myocardial infarction complicated by cardiogenic shock. Arch Intern Med. 2005;165(14):1643–50. https://doi.org/10.1001/archinte.165.14.1643.
[18] Debrunner M, Schuiki E, Minder E, et al. Proinflammatory cytokines in acute myocardial infarction with and without cardiogenic shock. Clin Res Cardiol. 2008;97(5):298–305. https://doi.org/10.1007/s00392-007-0626-5.
[19] Reynolds HR, Hochman JS. Cardiogenic shock: current concepts and improving outcomes. Circulation. 2008;117(5):686–97. https://doi.org/10.1161/CIRCULATIONAHA.106.613596.
[20] Martindale JL, Wakai A, Collins SP, et al. Diagnosing acute heart failure in the emergency department: a systematic review and meta-analysis. Acad Emerg Med. 2016; 23(3):223–42. https://doi.org/10.1111/acem.12878.
[21] Long B, Koyfman A, Gottlieb M. Diagnosis of acute heart failure in the emergency department: an evidence-based review. West J Emerg Med. 2019;20(6):875–84. https://doi.org/10.5811/westjem.2019.9.43732.
[22] Patch R, Cartin-Ceba R, Yilmaz M, Afessa B, Gajic O. Accuracy of the physical exam in the differentiation of shock syndromes. CHEST. 2007;132(4):561A. https://doi.org/10.1378/chest.132.4_MeetingAbstracts.561.
[23] Vazquez R, Gheorghe C, Kaufman D, Manthous CA. Accuracy of bedside physical examination in distinguishing categories of shock: a pilot study. J Hosp Med. 2010;5(8):471–4. https://doi.org/10.1002/jhm.695.

[24] Shah P, Cowger JA. Cardiogenic shock. Crit Care Clin. 2014;30(3):391–412. https://doi.org/10.1016/j.ccc.2014.03.001.
[25] Menon V, White H, LeJemtel T, Webb JG, Sleeper LA, Hochman JS. The clinical profile of patients with suspected cardiogenic shock due to predominant left ventricular failure: a report from the SHOCK Trial Registry. SHould we emergently revascularize Occluded Coronaries in cardiogenic shocK? J Am Coll Cardiol. 2000;36(3 Suppl A): 1071–6. https://doi.org/10.1016/s0735-1097(00)00874-3.
[26] Butman SM, Ewy GA, Standen JR, Kern KB, Hahn E. Bedside cardiovascular examination in patients with severe chronic heart failure: importance of rest or inducible jugular venous distension. J Am Coll Cardiol. 1993;22(4):968–74. https://doi.org/10.1016/0735-1097(93)90405-p.
[27] Klein T, Ramani GV. Assessment and management of cardiogenic shock in the emergency department. Cardiol Clin. 2012;30(4):651–64. https://doi.org/10.1016/j.ccl.2012.07.004.
[28] Menon V, Slater JN, White HD, Sleeper LA, Cocke T, Hochman JS. Acute myocardial infarction complicated by systemic hypoperfusion without hypotension: report of the SHOCK trial registry. Am J Med. 2000;108(5):374–80. https://doi.org/10.1016/s0002-9343(00)00310-7.
[29] Ponikowski P, Voors AA, Anker SD, et al. 2016 ESC guidelines for the diagnosis and treatment of acute and chronic heart failure: the task force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC)developed with the special contribution of the heart failure association (HFA) of the ESC. Eur Heart J. 2016;37(27):2129–200. https://doi.org/10.1093/eurheartj/ehw128.
[30] Seeto RK, Fenn B, Rockey DC. Ischemic hepatitis: clinical presentation and pathogenesis. Am J Med. 2000;109(2):109–13. https://doi.org/10.1016/S0002-9343(00)00461-7.
[31] Jäntti T, Tarvasmäki T, Harjola V-P, et al. Hypoalbuminemia is a frequent marker of increased mortality in cardiogenic shock. PLoS One. 2019;14(5). https://doi.org/10.1371/journal.pone.0217006.
[32] Jarai R, Fellner B, Haoula D, et al. Early assessment of outcome in cardiogenic shock: relevance of plasma N-terminal pro-B-type natriuretic peptide and interleukin-6 levels. Crit Care Med. 2009;37(6):1837–44. https://doi.org/10.1097/CCM.0b013e31819fe896.
[33] Kataja A, Tarvasmäki T, Lassus J, et al. The association of admission blood glucose level with the clinical picture and prognosis in cardiogenic shock - results from the CardShock study. Int J Cardiol. 2017;226:48–52. https://doi.org/10.1016/j.ijcard.2016.10.033.
[34] Abdin A, Pöss J, Fuernau G, et al. Prognostic impact of baseline glucose levels in acute myocardial infarction complicated by cardiogenic shock—a substudy of the IABP-SHOCK II-trial [corrected]. Clin Res Cardiol. 2018;107(6):517–23. https://doi.org/10.1007/s00392-018-1213-7.
[35] Tolppanen H, Rivas-Lasarte M, Lassus J, et al. Combined measurement of soluble ST2 and amino-terminal pro-B-type natriuretic peptide provides early assessment of severity in cardiogenic shock complicating acute coronary syndrome. Crit Care Med. 2017;45(7):e666–73. https://doi.org/10.1097/CCM.0000000000002336.
[36] Lemm H, Prondzinsky R, Geppert A, et al. BNP and NT-proBNP in patients with acute myocardial infarction complicated by cardiogenic shock: results from the IABP shock trial. Crit Care 2010;14(1):P146. doi:https://doi.org/10.1186/cc8378.
[37] O'Brien C, Beaubien-Souligny W, Amsallem M, Denault A, Haddad F. Cardiogenic shock: reflections at the crossroad between perfusion, tissue hypoxia, and mitochondrial function. Can J Cardiol. 2020;36(2):184–96. https://doi.org/10.1016/j.cjca.2019.11.020.
[38] Tehrani BN, Truesdell AG, Sherwood MW, et al. Standardized team-based Care for Cardiogenic Shock. J Am Coll Cardiol. 2019;73(13):1659–69. https://doi.org/10.1016/j.jacc.2018.12.084.
[39] Jolly SS, Shenkman H, Brieger D, et al. Quantitative troponin and death, cardiogenic shock, cardiac arrest and new heart failure in patients with non-ST-segment elevation acute coronary syndromes (NSTE ACS): insights from the global registry of acute coronary events. Heart. 2011;97(3):197–202. https://doi.org/10.1136/hrt.2010.195511.
[40] Tarvasmäki T, Haapio M, Mebazaa A, et al. Acute kidney injury in cardiogenic shock: definitions, incidence, haemodynamic alterations, and mortality. Eur J Heart Fail. 2018;20(3):572–81. https://doi.org/10.1002/ejhf.958.
[41] Jäntti T, Tarvasmäki T, Harjola V-P, et al. Frequency and prognostic significance of abnormal liver function tests in patients with cardiogenic shock. Am J Cardiol. 2017;120(7):1090–7. https://doi.org/10.1016/j.amjcard.2017.06.049.
[42] Jung C, Fuernau G, Eitel I, et al. Incidence, laboratory detection and prognostic relevance of hypoxic hepatitis in cardiogenic shock. Clin Res Cardiol. 2017;106(5): 341–9. https://doi.org/10.1007/s00392-016-1060-3.
[43] Vallabhajosyula S, Patlolla SH, Verghese D, et al. Burden of arrhythmias in acute myocardial infarction complicated by cardiogenic shock. Am J Cardiol. 2020;125 (12):1774–81. https://doi.org/10.1016/j.amjcard.2020.03.015.
[44] Collins SP, Lindsell CJ, Storrow AB, Abraham WT. ADHERE scientific advisory committee, investigators and study group. Prevalence of negative chest radiography results in the emergency department patient with decompensated heart failure. Ann Emerg Med. 2006;47(1):13–8. https://doi.org/10.1016/j.annemergmed.2005.04.003.
[45] Perera P, Mailhot T, Riley D, Mandavia D. The RUSH exam: rapid ultrasound in SHock in the evaluation of the critically lll. Emerg Med Clin North Am. 2010;28(1):29–56 vii https://doi.org/10.1016/j.emc.2009.09.010.
[46] Ünlüer EE, Karagöz A, Akoğlu H, Bayata S. Visual estimation of bedside echocardiographic ejection fraction by emergency physicians. West J Emerg Med. 2014;15 (2):221–6. https://doi.org/10.5811/westjem.2013.9.16185.
[47] Ramanathan K, Harkness SM, Nayar AC, et al. 887–2 Cardiogenic shock in patients with preserved left ventricular systolic function: Characteristics and insight into mechanisms. Journal of the American College of Cardiology. 2004;43(5, Supplement 1):A241. https://doi.org/10.1016/S0735-1097(04)91027-3.
[48] Kircher BJ, Himelman RB, Schiller NB. Noninvasive estimation of right atrial pressure from the inspiratory collapse of the inferior vena cava. Am J Cardiol. 1990;66(4): 493–6. https://doi.org/10.1016/0002-9149(90)90711-9.
[49] Wooten WM, Shaffer LET, Hamilton LA. Bedside ultrasound versus chest radiography for detection of pulmonary edema: a prospective cohort study. J Ultrasound Med. 2019;38(4):967–73. https://doi.org/10.1002/jum.14781.
[50] Keikha M, Salehi-Marzijarani M, Soldoozi Nejat R, Sheikh Motahar Vahedi H, Mirrezaie SM. Diagnostic Accuracy of Rapid Ultrasound in Shock (RUSH) Exam; A Systematic Review and Meta-analysis. Bull Emerg Trauma. 2018;6(4):271–8. https://doi.org/10.29252/beat-060402.
[51] Wilcox SR, Kabrhel C, Channick RN. Pulmonary hypertension and right ventricular failure in emergency medicine. Ann Emerg Med. 2015;66(6):619–28. https://doi.org/10.1016/j.annemergmed.2015.07.525.
[52] Millington SJ, Arntfield RT. Advanced point-of-care cardiac ultrasound examination: Doppler applications, Valvular assessment, and advanced right heart examination. Glob Heart. 2013;8(4):305–12. https://doi.org/10.1016/j.gheart.2013.11.003.
[53] Schneider M, Binder T. Echocardiographic evaluation of the right heart. Wien Klin Wochenschr. 2018;130(13):413–20. https://doi.org/10.1007/s00508-018-1330-3.
[54] Tan C, Rubenson D, Srivastava A, et al. Left ventricular outflow tract velocity time integral outperforms ejection fraction and Doppler-derived cardiac output for predicting outcomes in a select advanced heart failure cohort. Cardiovasc Ultrasound. 2017;15(1):18. https://doi.org/10.1186/s12947-017-0109-4.
[55] Blanco P, Aguiar FM, Blaivas M. Rapid ultrasound in shock (RUSH) velocity-time integral. J Ultrasound Med. 2015;34(9):1691–700. https://doi.org/10.7863/ultra.15.14.08059.
[56] Vincent J-L, De Backer D. Circulatory shock. Finfer SR, Vincent J-L, eds. N Engl J Med. 2013;369(18):1726–34. https://doi.org/10.1056/NEJMra1208943.
[57] Shepherd SJ, Pearse RM. Role of central and mixed venous oxygen saturation measurement in perioperative care. Anesthesiology. 2009;111(3):649–56. https://doi.org/10.1097/ALN.0b013e3181af59aa.
[58] Bracht H, Hänggi M, Jeker B, et al. Incidence of low central venous oxygen saturation during unplanned admissions in a multidisciplinary intensive care unit: an observational study. Crit Care. 2007;11(1):R2. https://doi.org/10.1186/cc5144.
[59] Ander DS, Jaggi M, Rivers E, et al. Undetected cardiogenic shock in patients with congestive heart failure presenting to the emergency department. Am J Cardiol. 1998;82 (7):888–91. https://doi.org/10.1016/S0002-9149(98)00497-4.
[60] De Backer D, Biston P, Devriendt J, et al. Comparison of dopamine and norepinephrine in the treatment of shock. New England Journal of Medicine. 2010;362(9): 779–89. https://doi.org/10.1056/NEJMoa0907118.
[61] Werdan K, Ruß M, Buerke M, Delle-Karth G, Geppert A, Schöndube FA. Cardiogenic shock due to myocardial infarction: diagnosis, Monitoring and Treatment. Dtsch Arztebl Int. 2012;109(19):343–51. https://doi.org/10.3238/arztebl.2012.0343.
[62] Jentzer JC, Coons JC, Link CB, Schmidhofer M. Pharmacotherapy update on the use of vasopressors and inotropes in the intensive care unit. J Cardiovasc Pharmacol Ther. 2015;20(3):249–60. https://doi.org/10.1177/1074248414559838.
[63] Burstein B, Tabi M, Barsness GW, Bell MR, Kashani K, Jentzer JC. Association between mean arterial pressure during the first 24 hours and hospital mortality in patients with cardiogenic shock. Crit Care. 2020;24. https://doi.org/10.1186/s13054-020-03217-6.
[64] Levy B, Clere-Jehl R, Legras A, et al. Epinephrine versus norepinephrine for cardiogenic shock after acute myocardial infarction. J Am Coll Cardiol. 2018;72(2): 173–82. https://doi.org/10.1016/j.jacc.2018.04.051.
[65] Alviar CL, Miller PE, McAreavey D, et al. Positive pressure ventilation in the cardiac intensive care unit. J Am Coll Cardiol. 2018;72(13):1532–53. https://doi.org/10.1016/j.jacc.2018.06.074.
[66] Jaber S, Amraoui J, Lefrant J-Y, et al. Clinical practice and risk factors for immediate complications of endotracheal intubation in the intensive care unit: a prospective, multiple-center study*. Crit Care Med. 2006;34(9):2355–61. https://doi.org/10.1097/01.CCM.0000233879.58720.87.
[67] Heffner AC, Swords DS, Neale MN, Jones AE. Incidence and factors associated with cardiac arrest complicating emergency airway management. Resuscitation. 2013; 84(11):1500–4. https://doi.org/10.1016/j.resuscitation.2013.07.022.
[68] Schwartz DE, Matthay MA, Cohen NH. Death and other complications of emergency airway Management in Critically ill Adults a Prospective Investigation of 297 tracheal intubations. Anesthes. 1995;82(2):367–76.
[69] Makdee O, Monsomboon A, Surabenjawong U, et al. High-Flow Nasal Cannula Versus Conventional Oxygen Therapy in Emergency Department Patients With Cardiogenic Pulmonary Edema: A Randomized Controlled Trial. Ann Emerg Med. 2017;70 (4). https://doi.org/10.1016/j.annemergmed.2017.03.028 465–472.e2.
[70] Masip J, Peacock WF, Price S, et al. Indications and practical approach to noninvasive ventilation in acute heart failure. Eur Heart J. 2018;39(1):17–25. https://doi.org/10.1093/eurheartj/ehx580.
[71] Berbenetz N, Wang Y, Brown J, et al. Non-invasive positive pressure ventilation (CPAP or bilevel NPPV) for cardiogenic pulmonary oedema. Cochrane Database Syst Rev. 2019;4. https://doi.org/10.1002/14651858.CD005351.pub4.
[72] Hochman JS, Sleeper LA, Webb JG, et al. Early revascularization and Long-term survival in cardiogenic shock complicating acute myocardial infarction. JAMA. 2006;295 (21):2511–5. https://doi.org/10.1001/jama.295.21.2511.