

Emergency Management of Circulatory Failure

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Abstract

Sepsis is a common presentation in the emergency department. Accurate triage, rapid recognition, early resuscitation, early antibiotics and eradication of the source of infection are the key components in delivering quality sepsis care. SIRS was removed from the sepsis clinical syndrome and is not a part of the definition of sepsis.

Keywords: *Emergency Management; Circulatory Failure; Sepsis*



A 68 years old male, with history of type II diabetes and hypertension, both on oral medications, is brought to the emergency department by his family, after he complained of worsening generalized abdominal pain.

On examination he is conscious and oriented, has abdominal pain that radiates to the lower and central back and is hypotensive on presentation. His skin is cool but dry.

This above is an example of a patient developing circulatory failure. The patient was eventually diagnosed with dissecting aortic aneurysm, which was managed by emergency vascular repair, followed by ICU admission until recovery.

What is a circulatory failure? Or 'shock' state?

By definition, circulatory failure is an acute state in which tissue perfusion is impaired, resulting in decreased delivery of oxygenated blood to body tissues and eventually ending in end-organ damage, system failure and eventually death if not treated adequately.

A state of shock, or circulatory failure, is a life-threatening condition. Emergency physicians should be well aware of its clinical presentation i.e. symptoms and signs suggestive of developing circulatory shock, should also know causes of circulatory failure, as well as management protocols and guidelines, in order to be able to identify and reverse this condition through delivering initial resuscitation management and defining further management according to the situation.

In the mentioned example, the patient was developing significant intra-abdominal bleeding. Rapid diagnosis and effective initial treatment in the ER eventually saved his life.

As shall be explained further in this article, the patient was going into a 'hypovolemic' shock with circulatory failure resulting from significant loss of intra-vascular volume.

Hypovolemic shock is one cause of circulatory failure, however, other types of shock result from different pathologies. Most recent classification of shock include the following types in addition to hypovolemic shock:

- Cardiogenic shock
- Distributive shock
- Obstructive shock.

All types of shock and circulatory failure will lead to tissue damage, end organ failure and death if not treated, it is important to understand that initial resuscitation and management is essentially similar, management of the underlying pathology will differ. Early recognition of type of shock and causative process is critical for effective management.

Despite the different causes and mechanisms, all types of shock, i.e. obstructive, hypovolemic, distributive and cardiogenic, will result in decreased tissue perfusion and subsequent hypoxia and end-organ damage, leading eventually to tissue death and multi-system failure if not adequately treated.

The common pathology in all types of shock or circulatory failure, is the transition in cellular metabolism from aerobic to anaerobic pathways, which results in accumulation of lactic acid in tissues and in the blood. This increase is used as biochemical a marker for diagnosis of shock.

Clinical presentation of shock

A patient in a state of shock and impending circulatory failure, will demonstrate one or more of the following symptoms and signs:

1. Altered level of consciousness, ranging from anxiety, agitation, delirium or coma.
2. Pulse rate alterations, mostly sinus tachycardia-i.e. pulse rate greater than 100 bpm- that excludes patients with concomitant heart block, or cardiac patients on beta-blockers.
3. Abnormal respiration, mainly rapid and shallow respiration-tachypnea.

4. Fall in Blood pressure. Compensatory mechanisms will maintain normal or near-normal blood pressure (systolic and mean pressure) in the initial stages of shock. It is only when these inherent compensatory mechanisms fail, that blood pressure will be affected resulting in hypotension.
5. Skin changes: cold and clammy-moist-skin, is a common feature in a patient in a shock state, except patients developing a distributive shock i.e. septic shock, where the patient will have warm skin due to peripheral vasodilation.

Returning to the example given above, the patient was developing a 'hypovolemic' shock. A hypovolemic shock can develop as a result of (a) hemorrhagic etiology, for example, gastrointestinal tract bleeding, massive hemoptysis, trauma or a dissecting aortic aneurysm. It can also develop secondary to non-hemorrhagic pathologies such as severe vomiting and diarrhea, burns, bowel obstruction and Pancreatitis. These etiologies result in significant dehydration and subsequently hypovolemic shock without bleeding.

We will mention briefly here other types of shock as they do not pertain to the mentioned example. Beginning with cardiogenic shock, in which blood volume and intravascular volume is not affected, but failure of the cardiac pump due to, for example, myocardial infarction, causes failure of tissue oxygenation with subsequent circulatory failure, and a similar end-point of end-organ damage and death.

Obstructive shock is another type of circulatory failure that results from conditions such as tension pneumothorax or cardiac tamponade. In which the myocardium is not affected, but restrictive conditions prevent efficient pumping of blood, i.e. obstructing blood flow. Leading to circulatory failure and hypoxemia. Rapid detection and knowledge of specific management for each is thus essential.

A 'distributive' shock develops as a complication of condition such septicemia and Anaphylaxis, both result in significant peripheral vasodilation followed by a fall of arterial blood pressure and the clinical picture of circulatory failure. It should be noted however, that patients with a distributive shock will have warm skin as opposed to the clammy, cold and mottled skin of other types of shock due to peripheral vasoconstriction.

The stages of shock

Development and progression of circulatory failure and shock passes through multiple stages, the rate by which the patient progresses through shock stages, along with patient characteristics, determine the attitude and type of intervention required by ER physician.

Management of a patient presenting to the ER during initial stages with intact compensatory mechanisms, will obviously be different than an unconscious patient with significantly deranged vital signs.

Pre-shock and compensatory mechanisms

The patient in the given example had a progressive intra-abdominal bleeding from a dissecting Aortic aneurysm. As a result to intravascular blood loss, compensatory mechanisms were triggered in an attempt to maintain oxygen supply and distribution to tissues.

These are innate homeostatic mechanisms, responsible for maintaining adequate tissue perfusion in response to ensuing pathology.

With progressive loss of intra-vascular volume, an increase in heart rate and peripheral vasoconstriction take place in order to direct blood to more vital organs such as the brain, liver and kidneys. Tachypnea rapid respiratory rate attempts to increase blood oxygen content and saturation. These responses contribute to the clinical picture and should be rapidly detected and interpreted by the emergency physician.

The term 'pre-shock', refers to the stage where an insulting pathology is ongoing, compensatory mechanisms are triggered in response and are maintaining oxygen delivery and tissue perfusion at an adequate level preventing hypoxemia.

It is only when these compensatory mechanisms fail or are overwhelmed by ensuing or progressing insult 'i.e. uncontrolled haemorrhage, that the stage of actual 'shock' develops. Accompanied as a result by a fall of blood pressure and altered consciousness, indicating circulatory failure.

With ongoing insult and failure of compensatory mechanisms, circulatory failure and hypoxemia will eventually lead to end-organ damage, manifesting clinically as renal and liver failure, altered consciousness level with delirium and subsequent coma, and eventually ending in death.

Management of shock and circulatory failure in the ER department

Considering the latest guidelines, literature and hands-on clinical experience, management of a shocked patient in the ER department should follow the following sequence:

- Initial stabilization and resuscitation
- Collecting history
- Physical examination
- Work-up and laboratory investigations
- Immediate management depending on type of shock.

During the first step, a quick survey of the patient is conducted and is aimed at determining the need for critical interventions such as endotracheal intubation and/or mechanical ventilation. Ensuring a patent and secure airway still represent the emergency physicians first priority during the initial stabilization stage.

Urgent airway management such as endotracheal intubation and assisted ventilation are often needed in conditions such as pulmonary edema, in which there is a failure of oxygenation of blood in the lungs. And in cases of airway obstruction as with patients developing anaphylactic shock with laryngeal oedema and subsequent airway obstruction.

After securing the airway and ensuring adequate breathing, the next step should be intravenous access, which in patients developing shock means insertion of a central venous line in order to deliver the required amount of fluids for resuscitation and circulatory support.

A 12-lead ECG can be obtained at this stage if myocardial infarction and 'cardiogenic shock' is suspected.

Brief history collection is often essential in determining the etiology and thus type of shock and eventually guide management, this of course should not delay primary management and resuscitation in any way. In the given example, the patient's age, medical history and clinical presentation with abdominal pain radiating to the back, were essential to achieving the diagnosis of a dissecting aneurysm and from there planning and implementing effective specific treatment, knowledge of causes and types of shock were critical in saving the patient's life.

Obtaining history often proves more difficult, for example in situations where a patient is received unconscious or cannot communicate his symptoms. In these situations, taking brief history from relatives or paramedics may be necessary.

Further examples for the importance of history taking in a patient with shock or pre-shock include complaints of chest pain, hematemesis, trauma or severe diarrhea. Which should lead the ER physician to identifying the possible type and cause of shock and guide effective ordering of investigations.

Physical examination

Quick and effective physical examination represents an essential initial step in identifying a patient in circulatory failure, as well as determining the cause, type and stage of shock and subsequently the plan of management. For example, a patient presenting with rapid pulse, hypotension, but has warm skin instead of cold and clammy, is unlikely to have a hypovolemic shock. He would instead be more likely to have a distributive type of shock either septicemia or anaphylaxis.

Laboratory investigations and initial work-up

During the initial management of a patient in a shock state or pre-shock and following conducting the initial physical examination and history, investigations are requested for diagnosis and determining the type of shock and causative factor(s). These may include a 12 lead ECG, blood tests for CBC, serum lipase, amylase, basic chemistry panel including liver and kidney function testing as well as arterial gases, cardiac enzymes, capillary blood glucose, will aid in the initial diagnosis and subsequently the management of the patient. For example, a shocked patient with elevated cardiac enzymes (Troponin and CK-MB), indicate a state of cardiogenic shock resulting from significant myocardial infarction, hence management with urgent resuscitation followed by angioplasty.

Radiological investigations are also essential in these situations, a chest X ray and/or echocardiography, are often helpful in determining the diagnosis of a shocked patient. As an example, a patient presenting with a clinical picture of circulatory failure and shock, who has a tension pneumothorax on chest X ray, will be efficiently managed by a chest tube insertion, significantly reducing morbidity and mortality.

Bed-side ultrasonography is increasingly being utilized by ER physicians, being a fast and effective diagnostic tool, a well-trained ER physician with access to bed-side ultrasound will be more likely to detect conditions such as intra-abdominal hemorrhage.

Management of shock in the ER

As discussed earlier, the primary cause for end-organ failure and death in a patient with shock is impaired tissue perfusion. Thus, management of shock is primarily focused towards treating hypoxemia, through improving oxygen delivery and utilization, restoration of adequate tissue perfusion and systemic blood pressure, improving cardiac output and treatment of the underlying pathology.

The ABCDE algorithm remains essential for every emergency physician to understand and implement despite advances in emergency medicine, so it is important to quickly explain it here.

ABCDE refers to the following steps in management of a patient in shock:

- (A), Airway: Maintaining patent and secure airway: The ER physician should determine the need for critical airway interventions such as endo-tracheal intubation, however, he should also bear in mind that these procedures carry risks and complications that should be weighed against the benefit depending on the situation. For example, intubation can worsen hypotension if sedatives are used. Moreover, the use of positive-pressure ventilation can lead to decreased 'preload'. Consequently, fluid resuscitation may be required prior to such interventions to prevent circulatory collapse.
- (B), Control of the work of breathing: Respiratory muscles consume a significant amount of oxygen particularly during rapid breathing 'tachypnea' which is a common feature of shock state, this may result in accumulation of lactic acid and acidosis if not controlled by mechanical ventilation and the use of sedatives, both increase patient survival in such settings.
- (C) Circulation support and optimization: This is achieved by crystalloid fluid infusions through peripheral or central lines. By doing so, cardiac pre-load is increased, followed by an increase in stroke volume and subsequently cardiac output, in turn leading to improved tissue perfusion and oxygen delivery. Isotonic crystalloid fluids should be initially used (i.e. 0.9% normal saline), the rate and volume should be titrated to achieve a CVP of 8 - 12 mm/Hg and a urine output of 0.5 ml/Kg/Hour (or 30 ml/hour). Total volume of crystalloid fluids required for resuscitation often range between 4 - 6 liters.

Colloids have been suggested in the past for use instead of crystalloids, however, they were not proven to have beneficial value superior to crystalloids.

- (D), Ensuring adequate oxygen delivery: This is done by two methods, first, decreasing tissue oxygen requirement, by providing analgesia and sedation and muscle relaxation, particularly respiratory muscles in the setting of tachypnea. Second, by improving and maintaining arterial oxygen saturation, via providing supplemental oxygen and maintaining a hemoglobin level of 10 g./dl. Or more blood transfusion in cases of hemorrhagic shock and significant blood loss. Success is measured by measuring serum lactate and central venous oxygen saturation, to ensure adequate oxygenation and prevention of cellular shift to anaerobic metabolism.
- (E), Achieving resuscitation 'end-points'. In other terms, the 'goals' of resuscitation. Which can be summarized in maximizing survival and minimizing morbidity. As a general standard that can be used for most cases, resuscitation should aim at achieving a MAP of 60 - 90 mm/Hg, CVP of 8 - 12 mm/Hg, a urine output of more than 0.5 ml/Kg/hour and a central venous Oxygen saturation of greater than 70%.

In the given example, the 68 years old male patient was developing a hypovolemic shock.

Compensatory mechanisms were failing which was evident by his low blood pressure and cool skin.

The patient was immediately managed by insertion of a central venous line, through which, initial fluid resuscitation was started using 0.9% saline and was kept at a rapid rate. His airway was patent on examination and he was breathing spontaneously, his Oxygen saturation was still above 95% and thus supplemental Oxygen was not started. Blood samples were collected for laboratory investigations, a 12-lead ECG was done. However, the patient's history and clinical picture led the ER physician to suspect a hemorrhagic etiology, namely dissecting aortic aneurysm (considering the patient's age, history of hypertension abdominal pain radiating to the back). This was confirmed by a bed-side ultrasonography and ECG changes.

Upon determining the diagnosis, type of shock and the etiology, the on-call surgery team was alerted, meanwhile, results of laboratory investigations further confirmed the diagnosis by showing a significant normocytic normochromic anemia due to blood loss, along with elevation of urea and creatinine.

A urinary catheter was inserted and showed initial oliguria, which was later corrected with crystalloid infusions which were titrated to maintain a urine output of 35 ml/minute. The patient's arterial pressure and pulse were monitored and were maintained at more than 70 mm/Hg and less than 100 bpm throughout. The patient was then transferred to the operating theatre where the surgery team eventually performed open repair of the descending aorta with endo-vascular stent and was discharged from the hospital after recovery in both the ICU and ward.

Note how success of managing this patient and eventually saving his life, relied on the skills and experience of the ER team, accurate investigations and systematic approach to management.

Initial central venous line insertion and fluid administration should not be delayed until the cause and type of shock is determined. Crystalloids (0.9% saline) should be started immediately at a rate of (30 ml/Kg body weight over 5 - 20 minutes) and should be maintained for through the initial 3 hours of resuscitation, monitored by the patient's urine output, blood pressure and pulse. This can change to blood transfusion if the patient is a 'slow-responder', or according to the physician's clinical judgement, the goal remains the same in all cases, which as mentioned earlier, is to improve oxygen delivery and perfusion.

Vasopressors

These agents are considered in cases where the patient is meeting end-points of resuscitation with crystalloids, but oxygen delivery is still inadequate with fluids alone.

In this case, Nor-epinephrine should be considered as first choice. Given at a dose of 0.1 - 2 ug/Kg/minute.

The use of vasopressin instead of norepinephrine, or the use of epinephrine, is no longer recommended in this setting.

Dopamine infusion can be used instead, at a rate of 2 - 20 ug/Kg/minute, as a second choice vasopressor especially in patients with cardiac arrhythmias, in which case nor-epinephrine might carry more risk than benefit.

After stabilization of circulation and achieving adequate oxygen supply and utilization, further management is directed towards detecting and treating the underlying pathology [1-20].

Conclusion

Circulatory failure and shock is a reversible cause of death encountered regularly in the ER. Causes, presentations and types of patients are variable and often complex and multi-factorial in nature and the physician's clinical judgement and experience is essential, however, a systematic approach to the diagnosis and treatment along with knowledge of guidelines and end-points, significantly reduces morbidity and mortality, it is essential for ER physicians to remain in touch with the latest evidence based approaches and developments and to have a methodical approach to critically ill patients despite the circumstances and situations, in the fast-paced world of emergency department, it may represent the difference between life and death.

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