SHOCK MANAGMENT

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- Describe the presentation of Neurogenic shock
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NEUROGENIC SHOCK

INTRODUCTION

- Patients with TSCI (traumatic spinal cord injury) require intensive medical care and continuous monitoring of vital signs, cardiac rhythm, arterial oxygenation, and neurologic signs .
- The causes of TSCI in the United States are:

➢ Motor vehicle accidents: 48 percent

➢ Falls: 16 percent

➢ Violence (especially gunshot wounds): 12 percent

➢ Sports accidents: 10 percent

- The primary assessment of a patient with trauma in the field follows the ABCD prioritization scheme:

Airway, Breathing, Circulation, Disability (neurologic status).

Cardiovascular complications

- Neurogenic shock is a devastating consequence of spinal cord injury (SCI).
- It manifests as hypotension, bradyarrhythmia, and temperature dysregulation due to peripheral vasodilatation following an injury to the spinal cord.

ETHIOLOGY

- Sudden loss of sympathetic tone, with preserved parasympathetic function, leading to autonomic instability.
- Neurogenic shock is mostly associated with cervical and high thoracic spine injury.
- Neurogenic shock should be differentiated from hypovolemic shock; the latter is often associated with tachycardia.
- Other causes of neurogenic shock include spinal anesthesia, Guillain-Barre syndrome, autonomic nervous system toxins, transverse myelitis, and other neuropathies.

- Neurogenic shock is not to be confused with a spinal shock which is the flaccidity of muscles and loss of reflexes seen following spinal cord injury.
- An adequate blood pressure is believed to be critical in maintaining adequate perfusion to the injured spinal cord and thereby limiting secondary ischemic injury.

- Guidelines currently recommend maintaining mean arterial pressures of at least 85 to 90 mmHg .
- Patients with multiple injuries often receive large amounts of IV fluids for various reasons. Excess fluids cause further cord swelling and increased damage.

History and Physical

- Neurogenic shock can be a difficult diagnosis to make and requires meticulous investigation.
- Neurogenic shock is most commonly associated with a blunt cervical spine injury.
- Neurogenic shock should be considered only after a hemorrhagic shock has been ruled out in a traumatic patient
- The presence of vertebral fracture or dislocation raises the concern for a neurogenic shock.
- Bradyarrhythmia, hypotension, flushed warm skin are the classic signs associated with neurogenic shock

Treatment / Management

- Initial management of neurogenic shock is focused on hemodynamic stabilization.
- Hypotension should be treated first to prevent secondary injury.
- The first-line treatment for hypotension is intravenous fluid resuscitation.
- If hypotension persists despite euvolemia, vasopressors and inotropes are the second lines.

Treatment / Management

- Phenylephrine is commonly used as it is a pure alpha-1 agonist that causes peripheral vasoconstriction to counteract the loss of sympathetic tone.
- Norepinephrine has both alpha and beta activity, aiding both hypotension and bradycardia.

Bradycardia

- Bradycardia may require external pacing or administration of atropine.
- This complication usually occurs in severe, high cervical (C1 through C5) lesions in the first two weeks after TSCI.

- Methylprednisolone and corticosteroids showed promise in animal models.
- However, this has not been displayed in clinical trials, and steroids raise the risk for complications such as infection and are not recommended by multiple societies.
- Surgical intervention may be required for decompression of spinal injury and improvement of neurogenic shock.
- Symptoms of neurogenic shock have been reported to persist for as long as 4 to 5 weeks.

Autonomic dysreflexia

- Autonomic dysreflexia is usually a later complication of TSCI (SCI above T6) requiring acute management .
- This phenomenon is characterized by episodic paroxysmal hypertension with headache, bradycardia, flushing, and sweating.
- Medications often used in this setting:

  nitrates, nifedipine, hydralazine IV, labetalol IV

Respiratory complications

- including respiratory failure, pulmonary edema, pneumonia, and pulmonary embolism, are the most frequent category of complications
- Weakness of the diaphragm and chest wall muscles leads to impaired clearance of secretions, ineffective cough, atelectasis, and hypoventilation.

- Signs of impending respiratory failure, such as increased respiratory rate, declining forced vital capacity, rising pCO2, or falling pO2, indicate urgent intubation and ventilation with positive pressure support
- Tracheostomy is performed within 7 to 10 days, unless extubation is imminent.
- With a goal of preventing atelectasis and pneumonia, chest physiotherapy should be instituted as soon as possible; patients may also need frequent airway suctioning.

Venous thromboembolism and pulmonary embolism

- DVT is a common complication of TSCI, occurring in 50 to 100 percent of untreated patients, with the greatest incidence between 72 hours and 14 days.
- All patients should receive prophylactic treatment.
- LMWH, IPC(Intermittent pneumatic compression)

INFLLAMATORY SHOCK

INTRODUCTION

- Systemic inflammatory response syndrome (SIRS) is a clinical syndrome that is characterized by a robust inflammatory response, usually induced by a major body insult that can be infectious or noninfectious.

Examples of noninfectious conditions that can be complicated by SIRS :

- Pancreatitis
- Burns
- Hypoperfusion caused by trauma
- Amniotic fluid embolism
- Air embolism
- Fat embolism

Pancratitis

- Acute oncet of epigastric pain and elevated serum amylase and lipase.
- With supportive care many patients recover without any complications.
- 20 percent have severe acute pancratitis with organ failure.

Systemic complications

- Organ failure:

➢ Activating cytokine casacade manifests clinically as a SISRS

➢ ARDS

➢ Renal failure

BURNS

- Multisystem organ dysfunction(MODS):

➢ Affects severe burned patients with or without infection

➢ Severely burned patients develop MODS at two different distinct points: early (due to hypoperfusion and under resuscition) or late (due to sepsis).

➢ Increased risk with burn wounds>20% TBSA

➢ The burn wound or lungs are the most likely sites for an infection

Specific organ failure

- ATN and AKI:
  - Immediate fluid resusciation
  - Correcting electrolyte abnormalities
  - Correcting metabolic acidosis
  - Dialysis

- Pulmonary failure(ARDS)
- Cardiac failure
- Hepatic failure
- Hematologic failure(DIC)
- CNS failure
- GI failure

MANAGEMENT

- Immediate debridement
- Weaning from the ventilator and ambulated as soon as possible
- Early enteral nutrition

Amniotic fluid embolism

- A catastrophic condition occuring during labor or within 30 minutes postpartum.
- May be due to a vasospastic, inflammatory ,and/or immune reaction.
- Sudden cardiovascular collapse, hypoxemia and DIC during labor.

management

- Initial strategies should focus on standard basic and advanced cardiac life support maneuvers to treat shock and cardiopulmonary compromise so that adequate tissue perfusion for both mother and fetus can be ensured
- This typically involves manual chest percussions, emergency airway management with supplemental oxygen and intubation, and the establishment of intravenous (IV) access, for fluid resuscitation and arrhythmia management.
- Vasopressors should be administered to those whose shock is refractory to resuscitative measures

Air embolism

- Oncommon but catastrophic
- Entry of the air in to the vasculature
- Most common causes: surgery, trauma,vascular intervention, barotrauma from machanical ventilation,diving
- Clinical signs: sudden onset respiratory distress or neurological event
- Life-threatening cases: acute onset of RV failure,sudden loss of consciosnesshemodynamic collapse,cardiac arrest.

Management

- Airway stability, high flow oxygen, volume resuscitation, vasopressors
- For venus air embolism :Placing the patient in the left lateral decubitus head down position or trndelenburg
- For arterial air embolism: Placing the patient in the supine position
- Hyperbaric oxygen therapy for evidence of hemodynamic compromise.

THE END

THANK YOU FOR YOUR ATTENTION