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Characteristics Of Neurogenic Shock

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ABSTRACT
Shock is a circulatory disorder which is defined as a condition of inadequate oxygen transport to tissues or perfusion caused by hemodynamic disorders. One type of shock, namely neurogenic shock, which is a form of distributive shock, is a critical condition resulting from dysregulation of the autonomic nervous system after spinal cord injury, especially in the cervical and upper thoracic vertebrae above T6, as a result of a traumatic event.. Worldwide , neurogenic shock occurs between fifteen and fifty-two events per million people annually with a higher incidence of cervical spine injury (SCI) (29%) compared to thoracic injury (19%). This causes hypotension and bradycardia, classic signs of neurogenic shock. There are no universally established hemodynamic parameters for neurogenic shock. However, most studies adopted the criteria of systolic blood pressure below 90 mmHg and heart rate below 80 bpm. Patients with neurogenic shock may also manifest as pink, warm skin due to dilation of subcutaneous blood vessels. Treatment of patients with neurogenic shock requires vasopressor drugs such as noradrenaline, phenylephrine, or metaraminol, and positive inotropic dopamine is recommended to treat vascular tone and prevent secondary injury.

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INTRODUCTION

Shock is a circulatory disorder which is defined as a condition of inadequate oxygen transport to tissues or perfusion caused by hemodynamic disorders. These hemodynamic disturbances can include a decrease in systemic vascular resistance, especially in the arteries, reduced venous blood return, decreased ventricular filling and very small cardiac output. One type of shock, namely neurogenic shock, which is a form of distributive shock, occurs due to failure of the vasomotor center due to a sudden decrease in blood vessel tone throughout the body. The result of changes in systemic vascular resistance is caused by injury to the nervous system (such as: head trauma, spinal injury, or general anesthesia that is too deep).[1,2]

The epidemiology of neurogenic shock is difficult to assess because it remains unknown how hemorrhagic shock and other injuries impact the hemodynamic effects of spinal cord injury. [3] Worldwide, neurogenic shock occurs between fifteen and fifty-two events per million people annually. Only about five percent of patients are young, and the majority are men between fifteen and thirty-five years old. The incidence of neurogenic

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shock is higher in cervical spine injury (SCI) (29%) compared with thoracic injury (19%). Nervous function disorders that tend to appear and that appear frequently are tetraplegia (53%) and paraplegia (42%). [3]

Spinal cord injury is most commonly associated as a causative factor in neurogenic shock. Neurogenic shock occurs when there is spinal cord injury and can cause hypotension due to loss of sympathetic tone. The disease presents with arterial hypotension without skin vasoconstriction (the skin does not cool) and tachycardia. Management of neurogenic shock can be carried out starting from the moment the incident occurs, if the patient experiences trauma, to avoid further injury the cervical level must be immobilized, ensuring an adequate airway, ensuring ventilation, maximizing circulation and the patient is immediately transferred to the hospital. [1,4]

METHOD

In recent years, the importance of effective instructional design in online learning environments has gained significant attention. Studies by Anderson and Dron (2011) and Garrison (2016) emphasize the necessity of incorporating social, cognitive, and teaching presence to enhance student engagement and learning outcomes. Additionally, the Community of Inquiry (CoI) framework, as highlighted by Akyol and Garrison (2011), provides a comprehensive model for assessing the effectiveness of online educational experiences. Recent meta-analyses, such as those conducted by Bernard et al. (2014), support the efficacy of well-structured online courses, noting improvements in student satisfaction and performance compared to traditional face-to-face settings. This body of literature underscores the critical role of evidence-based instructional strategies in the development of successful online learning programs.

RESULT AND DISCUSSION

Definition

Neurogenic shock is a critical condition resulting from dysregulation of the autonomic nervous system following spinal cord injury, particularly to the cervical and upper thoracic vertebrae above T6, as a result of a traumatic event. Dysregulation arises due to a lack of sympathetic and parasympathetic responses that cannot be resisted. Although rare, other potential causes of neurogenic shock include spinal anesthesia, Guillain-Barre syndrome, toxins affecting the autonomic nervous system, transverse myelitis, and various neuropathies involving the cervical and upper thoracic spinal cord. [5]

Epidemiology

The incidence of neurogenic shock is higher in cervical spine injury (SCI) (29%) compared with thoracic injury (19%). Nervous function disorders that tend to appear and that appear frequently are tetraplegia (53%) and paraplegia (42%). Spinal cord injury is most commonly associated as a causative factor in neurogenic shock. [3] Neurogenic shock occurs due to damage to the spinal cord above the level of the 6th thoracic vertebra. It is discovered in about half of people with spinal cord injuries within the first 24 hours, and usually does not go away for one to three weeks. [6]

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Approximately 8,000 to 10,000 people suffer traumatic spinal cord injuries each year in the United States. Data obtained from the Trauma and Tissue Audit following a comprehensive analysis of 490 isolated spinal cord injury cases found that only 19.3% of cases involved neurogenic shock. However, a retrospective study at a higher volume level 1 trauma center found that 31% of cases (19 of 62 patients) with upper cervical spine injuries (levels C1–C5) experienced neurogenic shock. [5]

There are no universally established hemodynamic parameters for neurogenic shock. However, most studies adopted the criteria of systolic blood pressure below 90 mmHg and heart rate below 80 bpm . Evaluating the epidemiology of neurogenic shock is a challenge, because it remains unclear how hemorrhagic shock and other injuries influence the hemodynamic consequences of spinal cord injury. [5]

Etiology

Neurogenic shock is a critical condition resulting from dysregulation of the autonomic nervous system following spinal cord injury, particularly to the cervical and upper thoracic vertebrae above T6, as a result of a traumatic event. Dysregulation arises from a lack of sympathetic impulses and an unopposed parasympathetic response. Although rare, other potential causes of neurogenic shock include spinal anesthesia, Guillain-Barre syndrome, toxins affecting the autonomic nervous system, transverse myelitis, and various neuropathies involving the upper cervical and thoracic spinal cord. [5]

Pathophysiology

Neurogenic shock is a clinical condition that manifests after spinal cord injury affecting the cervical and upper thoracic spinal cord levels. This condition triggers hemodynamic changes due to disruption of the descending sympathetic tract, usually due to fractures or dislocations of the vertebrae in that spinal area. The primary onset of spinal cord injury usually occurs within minutes of the initial injury, resulting in direct damage to the axons and nerve membranes in the intermediolateral nucleus, lateral gray matter, and anterior roots. This damage causes disruption of sympathetic impulses. [5]

Decreased sympathetic impulses cause dilation of blood vessels in the lower extremities. This, in turn, results in decreased cardiac filling which ultimately leads to hypotension and shock. In addition, reduced sympathetic control of the heart leads to a situation where unopposed vagal cardiac influences lead to bradycardia. [5]

Secondary spinal cord injuries occur within hours to days after the initial trauma. This phase of injury results from vascular disturbances, electrolyte shifts, and the development of edema, leading to the gradual onset of central hemorrhagic necrosis within the gray matter at the injury site. Several studies have shown that hypotension due to neurogenic shock worsens the severity of spinal cord injury. [5]

Neurogenic shock is caused by several complex processes at the cellular level, including N-methyl-D-aspartate (NMDA) accumulation and excitotoxicity, disturbance of electrolyte homeostasis, mitochondrial damage, and reperfusion injury. These mechanisms collectively contribute to controlled and uncontrolled apoptosis, resulting in the complex series of events that characterize neurogenic shock. [5.7]

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Diagnosis

Shock is said to be a state of inadequate tissue perfusion. Neurogenic shock occurs when the spinal cord is injured and sympathetic innervation to the heart along with vasomotor tone is lost, with parasympathetic innervation being dominated by an intact vagus nerve. This causes hypotension and bradycardia, classic signs of neurogenic shock. Preganglionic sympathetic neurons originating from the hypothalamus, pons, and medulla are located in the intermediolateral column cells of the spinal cord between the first thoracic vertebra (T1) and the second lumbar vertebra (L2). [8]

Theoretically, any SCI in or above it can cause sympathetic disturbances. Because sympathetic innervation of the heart occurs only from T1 to T5, it is often said that neurogenic shock can only occur when the lesion is above the mid-thoracic level (T6). Animal studies in cats, dogs, and mice show inconsistencies in the description of neurogenic shock, the occurrence of which ranges from minutes to hours post-transection. Some animal studies have also shown that immediately after spinal cord transection tachycardia occurs, possibly caused by the release of large amounts of catecholamines. [8]

In humans, the highest levels of bradycardia have been shown to occur approximately 4 days post-injury. However, this study was carried out in a specialist spinal cord injury unit and therefore did not take into account baseline changes in blood pressure either before hospital admission or in the emergency department. Systolic blood pressure below 90 mmHg in the supine position, which is not caused by low intravascular volume due to hemorrhage or dehydration, is characteristic of neurogenic shock. Severe arterial hypotension usually requires vasopressors. Due to its complex nature, there are no standard criteria for defining neurogenic shock. This is due in part to the changing nature of blood pressure and condition of trauma patients and the possibility of other etiologies of shock occurring simultaneously. [8]

In previous research, Guly and colleagues highlighted how difficult it is to identify patients with neurogenic shock in cases with a mixed picture of shock, with aspects such as pain, anxiety, and bleeding clouding a clear picture. Because of this problem, the blood pressure (BP) and heart rate (HR) used in previous studies were quite variable (figure 1). [8]

Table 1 A summary of the criteria used to define neurogenic shock from a selection of papers

Paper	sBP (mm Hg)	HR (bpm)
Bernhard *et al*[9]	<70	<60
Grigorean *et al*[10]	<90	–
Guly *et al*[7]	<100	<80
Lehmann *et al*[8]	<90	–
Levi *et al*[11]	<90	–
Ley *et al*[12]	≤90	≤90
Moerman *et al*[13]	<80	<60
Zipnick *et al*[14]	<100	<80

The systolic BP (sBP) in mm Hg and HR in bpm were recorded if discussed in the paper.

Figure 1. Various studies use standard blood pressure as a criterion for defining neurogenic shock

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Identification of neurogenic shock is important. Patients with neurogenic shock may also manifest as pink, warm skin due to dilation of subcutaneous blood vessels . [5] Often treatment for neurogenic shock is necessary before a definitive diagnosis of SCI is made. One of the characteristics of neurogenic shock is partial resistance to fluids, which can be confused with volume loss. This can cause the patient to become overhydrated, causing pulmonary or spinal cord edema. [8]

Governance

Neurogenic shock is a hemodynamic disturbance that can occur after spinal cord injury, findings from Summers et al. demonstrated that the etiology of clinical neurogenic shock is actually a spectrum of hemodynamic states that all result in hypotension but require different management strategies. Management protocols that include more extensive hemodynamic data collection have been shown to improve patient outcomes. Therapies that target specific circulatory mechanisms involved in the cause of shock should be carefully selected compared with general management approaches. However, the collection of detailed hemodynamic information (including cardiac output) in the early emergency stages of the disease process and before aggressive therapy is initiated is very difficult. [1]

Neurogenic shock usually occurs in patients who experience SCI above T6 which will cause vasodilation, bradycardia and hypotension and severe bradycardia or asystole can occur when treatments that stimulate the vagal reflex such as laryngoscopy or suction are performed. Treatment of patients with neurogenic shock requires vasopressor drugs such as noradrenaline, phenylephrine, or metaraminol, and positive inotropic dopamine is recommended to treat vascular tone and prevent secondary injury. It is recommended to monitor blood pressure using an arterial line and central venous catheter to administer medication as well as monitor central venous pressure. [9]

Management of blood pressure in patients with neurogenic shock after spinal cord injury was reported in research conducted by Dakson et al. One of the results of this study found that patients with mean arterial pressure (MAP) > 85 mmHg who could maintain it consistently for 5 days would have a significantly better rate of neurological recovery. Similar results were also reported in research by Hawryluk et al., (2015). This study, with a retrospective design involving 100 patients, aimed to determine the correlation between MAP and neurological recovery in spinal cord injury patients. One of the results of this study found that a higher MAP value on average in the first 2-3 days of treatment would correlate with better neurological recovery. [1]

In the acute stage after spinal cord injury, cardiovascular complications require immediate medical attention to prevent neurological disorders and morbidity. In particular, sympathetic nervous system disorders that commonly occur in patients with severe spinal cord injuries at T6 or higher can cause autonomic dysreflexia including hypotension (both supine and orthostatic) and cardiac arrhythmias (usually bradycardia). Administration of vasopressors (eg Dopamine) with sympathetic, chronotropic and inotropic cardiac support is recommended in patients with neurogenic shock with spinal cord injury, unless contraindicated. Following dopamine, the recommended second-line agent is norepinephrine as it also provides a combination of alpha and beta-adrenergic support.

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Vasoactive agents that induce reflex bradycardia, such as phenylephrine, should be avoided in injuries above T6 because the body may not be able to appropriately mount a sympathetic response to the bradycardia. [1]

Research shows that hypotension results in poor spinal cord perfusion and contributes to secondary injury and worse neurologic outcomes. There is prospective and retrospective evidence that augmentation of mean arterial pressure (MAP) in the acute phase can improve neurologic outcomes. The American Association of Neurological Surgeons (AANS) currently recommends that MAP be maintained above 85-90 mmHg for 7 days post-injury. The above-mentioned studies have also employed shorter durations of 5 days and demonstrated that higher MAP values best correlate with neurological recovery in the first 2-3 days post-injury. Discontinuation of vasopressor therapy within seven days, and clinical management of blood pressure focused entirely on vasoconstriction likely has major implications for the site of inflammation and the potential for subsequent neurologic recovery in the acute phase. [1]

The use of vasopressor therapy in the acute phase with intravenous vasopressors with or without inotropes is generally effective but not for long-term therapy in patients whose recovery tends to be slower. The use of peudoephedrine (PSE) in neurogenic shock is theoretically attractive because it is an a- and b-adrenergic agonist that can be administered orally, and is cheap. In Woo et al.'s study, evaluating the administration of PSE. Success was observed in 31 (82%) of 38 study patients. In this group of patients, the median time to discontinue vasopressor use was 8 days. Two patients (5%) failed to discontinue vasopressor therapy, whereas five patients (13%) had an inconclusive response to PSE. Daily PSE doses vary widely, ranging from 30–720 mg. The greatest limitation of this adjuvant therapy is the need for PSE therapy to be continued for several weeks in most patients. [1]

A recent case report from Kurozumi et al., reported the use of Indigo Carmin in a patient with neurogenic shock due to SCI who had undergone decompression and fixation. Indigo carmine is a dye that is clinically used for organ identification in urological and mammary gland surgery. The chemical structure of indigo carmine is similar to serotonin, which can create an increase in blood pressure in this case. In the case of indigo carmin successfully treated catecholamine refractory neurogenic shock. Thus, indigo carmine may be considered as an option for increasing blood pressure in patients with spinal cord injuries. However, further research cases are still needed to evaluate its effects and prove its usefulness in spinal cord injuries. [1]

Differential Diagnosis

Shock is a common endpoint of many medical conditions. It has been divided into four main types based on the underlying cause: hypovolemic, distributive, cardiogenic, and obstructive.

a. Hypovolemic Shock

Hypovolemic shock is the most common type of shock and is caused by insufficient circulating volume. The main cause is bleeding (internal and/or external), or loss of fluid from the circulation. Vomiting and diarrhea are the most common causes in children. Other

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causes include burns, environmental exposure and excessive urine loss due to diabetic ketoacidosis and diabetes insipidus. [6]

Patients with upper cervical SCI may experience difficulty breathing. The patient needs to be given oxygen 4-6 liters/minute and have an AGD examination. If signs of shock occur in SCI cases, it is necessary to differentiate whether the shock is hypovolemic or neurogenic shock. In hypovolemic shock there are signs of tachycardia and cold extremities along with hypotension, whereas in neurogenic shock there are signs of bradycardia and warm extremities accompanying hypotension. The two types of shock can also be differentiated by giving isotonic fluids such as 0.9% Nacl, or two liters of Ringer's lactate. If no improvement is found, then neurogenic shock should be suspected. Excessive fluid correction in neurogenic shock is dangerous because it can cause pulmonary edema. [10]

b. Cardiogenic

This type of shock is caused by the heart's failure to pump effectively. This can be caused by damage to the heart muscle, most commonly due to a large myocardial infarction. Other causes of cardiogenic shock include dysrhythmias, cardiomyopathy/myocarditis, congestive heart failure (CHF), or heart valve problems. [6]

c. Obstructive shock

Obstructive shock occurs due to obstruction of blood flow outside the heart. Several conditions can cause this form of shock, namely: [6]

- Cardiac tamponade where fluid in the pericardium prevents blood from entering the heart (venous return).
- Constrictive pericarditis, in which the pericardium shrinks and hardens, has a similar picture.
- Tension pneumothorax through increased intrathoracic pressure, obstructs blood flow to the heart (venous return).
- Pulmonary embolism is the result of thromboembolic events in the pulmonary blood vessels and inhibits the return of blood to the heart.
- Aortic stenosis restricts circulation by blocking the ventricular outflow tract

d. Distributive Shock

Distributive shock is caused by impaired oxygen utilization and energy production by cells. Examples of this form of shock are: [6]

- Septic shock is the most common cause of distributive shock. Caused by severe systemic infections that cause vasodilation which causes hypotension. Septic shock can be caused by Gram-negative and positive bacteria. Septic shock can be defined as “hypotension due to sepsis (systolic blood pressure <90 mmHg or a decrease of 40 mmHg from baseline) despite adequate fluid resuscitation accompanied by perfusion abnormalities that may include, but are not limited to, lactic acidosis, oliguria, or acute changes in mental status. Patients receiving inotropic or vasopressor agents may have normal blood pressure at the time perfusion abnormalities are identified.
- Anaphylactic Shock

Anaphylactic shock is a severe, life-threatening, generalized or systemic hypersensitivity reaction. Characterized by rapidly developing and life-threatening airway

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and/or breathing and circulation problems, usually associated with skin and mucosal changes. [6]

Hypovolemic	Hypotension, tachycardia Weak thready pulse Cool, pale, moist skin U/O decreased	Decreased CO **Increased** SVR
Cardiogenic	Hypotension, tachycardia Weak thready pulse Cool, pale, moist skin U/O < 30 ml/hr Crackles, tachypnea	Decreased CO **Increased** SVR
Neurogenic	Hypotension, BRADYCARDIA **WARM DRY SKIN**	Decreased CO **Venous & arterial vasodilation, loss sympathetic tone**
Anaphylactic	Hypotension, tachycardia Cough, dyspnea Pruritus, urticaria Restlessness, decreased LOC	Decreased CO **Decreased** SVR
Septic	Hypotension, Tachycardia Full bounding pulse, tachypnea **Pink, warm, flushed skin** Decreased U/O, fever	**Decreased** CO, **Decreased** SVR

Figure 2. Differences between each type of shock

Prognosis

The prognosis in cases of neurogenic shock is influenced by various factors, with 2 important elements, including: [5]

a. Spinal cord injury: Based on the American Spinal Injury Association (ASIA) scale, the severity of spinal cord injury significantly influences the overall prognosis.
b. Treatment response: An individual's response to treatment and management strategies significantly influences the expected outcome

In addition to the factors mentioned above, a person's prognosis in cases of neurogenic shock can further be influenced by several important factors, as explained below. [5]

a. Neurological deficits: The presence and severity of neurological deficits on initial examination can play an important role in shaping the overall prognosis.
b. Age: Age is a determining factor, as younger patients often show better prognostic potential for recovery and adaptation.
c. Concomitant organ injuries: Concomitant injuries affecting other organs can affect overall recovery and prognosis.
d. Glasgow Coma Scale (GCS) score: The GCS score, which assesses consciousness and neurological function, contributes to understanding a patient's recovery potential.

Individuals who have experienced neurogenic shock have a unique prognosis determined by these factors. [5]

CONCLUSSION

Neurogenic shock can cause complications, including prolonged and severe hypotension, which may require vasopressor therapy. This hypotension may persist for a long period of time, several weeks after the initial injury. Individuals with neurogenic shock may experience additional complications, such as autonomic dysreflexia, in the long term. [5] Autonomic dysreflexia occurs due to disruption of normal control of the sympathetic spinal cord due to

central nervous system disorders. This phenomenon was first observed by Anthony Bowlby in 1890 and later described by Guttmann and Whitteridge in 1947. Autonomic dysreflexia arises as an excessive and unbalanced sympathetic response to stimuli below the level of spinal cord injury. Factors that contribute to this condition include distension, stimulation, or manipulation of the bladder or bowel. [5] Patients with spinal cord injuries have an increased risk of developing deep vein thrombosis (DVT). Studies have shown that the incidence of preoperative DVT among patients with vertebral fractures is 13.10%. Additionally, advanced age is correlated with increased susceptibility to DVT. Other factors that contribute to DVT include venous stasis in the extremities due to muscle paralysis, venodilation, and hypercoagulable states.[5] Patients may experience disturbances in electrolyte balance, including the potential development of conditions such as hypokalemia. This electrolyte imbalance can be caused by hypotension and hypovolemia, which can increase plasma aldosterone levels. Gastrointestinal dysfunction, particularly paralytic ileus, may be associated with spinal cord injury. This event results from mesenteric vascular dysfunction that accompanies spinal cord injury. [5]

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