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Characteristics Of Neurogenic Shock Resulting From Spine Injury

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ABSTRACT

Neurogenic shock is a distributed shock characterized by loss of sympathetic tone due to acute spinal cord or central nervous system injury. Cervical spine injury, spinal anesthesia, Guillain-Barre syndrome, and transverse myelitis are among the causes of neurogenic shock. Neurogenic shock describes the sudden loss of autonomic tone due to Spinal Cord Injury (SCI). Disruption of the descending sympathetic pathway causes opposing vagal tone in vascular smooth muscle to decrease systemic vascular resistance and vasodilation. Hypotension resulting from neurogenic shock places patients at increased risk of spinal cord ischemia secondary to impaired autoregulation. Neurogenic shock occurs due to damage to the spinal cord above the 6th thoracic vertebra. There are no definitive diagnostic tests, but classically patients present with relative hypotension and bradycardia

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1. INTRODUCTION

Shock is a clinical syndrome that occurs due to hemodynamic and metabolic disorders characterized by failure of the circulatory system to maintain adequate perfusion to the body's vital organs. This arises due to serious events in the body's hemostasis, such as massive bleeding, trauma and severe burns (hypovolemic shock), extensive myocardial infarction or pulmonary embolism (cardiogenic shock), uncontrolled bacterial sepsis (septic shock), vasomotor tone inadequate (neurogenic shock) or due to an immune response (anaphylactic shock).

Neurogenic shock describes the sudden loss of autonomic tone due to Spinal Cord Injury (SCI). Disruption of the descending sympathetic pathway causes opposing vagal tone in vascular smooth muscle to decrease systemic vascular resistance and vasodilation. Hypotension resulting from neurogenic shock places patients at increased risk of spinal cord ischemia secondary to impaired autoregulation.

Neurogenic shock occurs most frequently after acute injury above T6, with a possible incidence of 29% in the cervical SCI population and 19% in thoracic SCI. Neurogenic shock is considered distributive in that it involves loss of vasomotor tone and instability due to imbalance in the autonomic nervous system. In addition to bradycardia and hypotension, many patients experience autonomic dysreflexia defined as a profound autonomic response to what is usually a mild stimulus such as bladder or bowel distension.3 Management of neurogenic shock, early diagnosis and treatment of the etiology should always be considered.

2. METHODS

The method used in this research uses Systematic Literature Review (SLR) to find out the most appropriate method for designing Enterprise Architecture in government agencies. This research applies three stages, namely: planning, implementation, and results analysis, which analyzes the results of the Literature Review that the author has carried out. The planning stage involves formulating the problem that you want to identify. The implementation stage involves the process of collecting papers to conduct a Literature Review. Analysis of the results, namely answering the existing problem formulation sourced from the Literature Review carried out.

3. RESULT AND DISCUSSION

Definition

Shock is a clinical syndrome that occurs due to hemodynamic and metabolic disorders characterrized by failure of the circulatory system to maintain adequate perfusion to the body's vital organs. This arises due to serious events in the body's hemostasis, such as massive bleeding, severe trauma and burns, extensive myocardial infarction or pulmonary embolism, sepsis due to uncontrolled bacteria, inadequate vasomotor tone (neurogenic shock) or due to an immune response.

Neurogenic shock is a distributed shock characterized by loss of sympathetic tone due to acute spinal cord or central nervous system injury. Sudden loss of autonomic tone causes a decrease in systemic vascular resistance and vasodilation. The clinical presentation of shock is generally hypotension, normal heart rate, or bradycardia.

Epidemiology and Etiology

There are various causes of neurogenic shock, including cervical spine injury, spinal anesthesia, Guillain-Barre syndrome, and transverse myelitis. While neurogenic shock is mostly associated with cervical spine injuries and high thoracic injuries, neurogenic shock occurring from transverse myelitis is very rare.

Neurogenic shock can occur from severe central nervous system damage (brain, cervical or thoracic spinal cord injury). In simpler terms: trauma causes a sudden loss of sympathetic stimulation to the blood vessels. This causes them to relax (vasodilation), resulting in a sudden drop in blood pressure (secondary to a decrease in peripheral vascular resistance). Neurogenic shock occurs due to damage to the spinal cord above the 6th thoracic vertebra. It is discovered in about half of people with spinal cord injury within the first 24 hours, and usually does not resolve for one to three weeks. After severe spinal cord injury (SCI), the acute loss of descending input to sympathetic preganglionic neurons causes neurogenic shock. , which is characterized by a decrease in peripheral vascular resistance and a relative predominance of vagal tone with bradycardia and decreased myocardial function.

Neurogenic shock occurs most frequently after acute injury above T6, with a possible incidence of 29% in the cervical SCI population and 19% in thoracic SCI. In the United States, traumatic spinal cord injuries affect between 8,000 and 10,000 people each year. The Trauma Audit and Research Network found 490 isolated spinal cords in its isolated spinal cord survey. Of these, only 19.3% of individuals suffered from neurogenic shock.

Pathophysiology

Several supratentorial regions, such as the insula cortex, medial prefrontal cortex, hypothalamus, and brainstem nuclei, are responsible for autonomic function. Didactically represented by the sympathetic and parasympathetic autonomic nervous systems, these nerves control vital signs, adapting them according to environmental demands. Parasympathetic fibers reach the transverse colon via the vagus nerve, innervating the heart, bronchi, digestive tract, and glands via long and short preganglionic fibers. The sympathetic autonomic nervous system, on the other hand, begins with the short preganglionic nerves up a chain of paravertebral ganglia descending parallel to the thoracic and lumbar spinal cord (T1-L2), down to the long postganglionic nerves. These nerves, in turn, innervate most of the viscera, including the smooth muscle of the heart and blood vessels.

The first lesion occurs immediately after trauma, followed by secondary lesions, triggered by primary mechanical injury, resulting in microvascular damage, edema, demyelination, ischemia, excitotoxicity, electrolyte changes, free radical production, inflammation, and late apoptosis. The location surrounding the injury site is considered the ischemic penumbra, with the possibility of functional restoration. melatonin, propofol, erythropoietin and thiopental can prevent lipid peroxidation immediately after injury in experimental models. Opioids are potentially lethal to injured cells, causing blockage of microcirculation and impairing functional recovery by acting as neurotransmitters at kappa receptors. In several models, including phase I studies in humans, the use of naloxone (an opioid antagonist) improved medullary functional recovery. After spinal cord trauma, in addition to the observed sensory and motor deficits, dysautonomia also results in neurogenic shock. Loss of sympathetic autonomic control over the parasympathetic nervous system is responsible for most of the symptoms that define neurogenic shock. Cardiovascular and respiratory disorders are the

most feared. Parameters such as systemic blood pressure, heart rate, glandular secretion, thermal control, bronchodilation and peripheral vascular resistance have been altered. During neurogenic shock, there is a predominance of the parasympathetic system over the sympathetic system, which can lead to the death of the patient. Hypotension as well as orthostatic hypotension improve within days or weeks, thanks to compensatory mechanisms that include: skeletal muscle activity, spasticity, increased muscle tone, awakening of sympathetic reflexes medullary (readjustment of catecholamine levels), and readjustment of the reninangiotensin-aldosterone system.

Clinical Manifestations

Neurogenic shock is a type of distributive shock, but should be a diagnosis of exclusion in the early phase of traumatic resuscitation after hemorrhagic shock has been ruled out. There are no definitive diagnostic tests, but classically patients present with relative hypotension and bradycardia. Bradycardia is often exacerbated by suction, defecation, turning, and hypoxia. Initially the skin is often warm and red. Hypothermia may develop due to profound vasodilation and heat loss. Often central venous pressure is low due to decreased systemic vascular resistance. There is some evidence to suggest that hypertension occurs within the first few minutes of SCI, followed by hypotension. Thus, patients with SCI should be closely monitored for the development of neurogenic shock even if none is present at presentation. A joint committee of the American Spinal Injury Association and International Spinal Cord Society proposed a series of definitions of common autonomic nervous system dysfunction (neurogenic shock, orthostatic hypotension, autonomic dysreflexia, temperature dysregulation, sweating). disorders) after adult SCI that must be assessed by a physician.

Diagnosis

In lesions below C3, patients present with symptoms of autonomic nervous system disorders, including possible cardiac arrest several minutes after injury. In the emergency room, the patient presented classically: flaccid paralysis, bradycardia and systolic arterial hypotension (SBp < 90 mm Hg) in the orthostatic position. Inability to empty the bladder is also an important clinical feature. In clinical practice, however, computed tomography is essential for diagnosis. According to data published in the Trauma Audit and Research Network database, neurogenic shock is correlated with cervical lesions in 19.3% of cases, thoracic spine lesions in 7%, and lumbar lesions in 3%.

Governance

Decreased systemic vascular resistance results in relative hypovolemia due to increased venous capacity, and administration of isotonic fluids is often necessary. However, hypotension due to neurogenic shock is often refractory to fluid resuscitation. However, hypotension in trauma patients cannot be assumed to be due to neurogenic shock initially, and could be a sign of hemorrhagic shock. Therefore, trauma victims with hypotension should be treated initially with crystalloid (0.9% sodium chloride, Ringer's lactate) or colloid (albumin, blood products) fluids and evaluated for ongoing blood loss. Patients should be adequately resuscitated from a hemodynamic perspective before undergoing spinal cord decompression surgery.

Hypotension should be treated promptly to avoid secondary ischemic SCI. Cervical SCI is frequently seen in patients who also have traumatic brain injury and hypotension is also not tolerated in the setting of traumatic brain injury. Mannitol should be avoided in case of shock in patients with suspected brain and spinal cord injury, as hypertonic saline is now recommended as a first-line osmotherapy agent in pediatric severe traumatic brain injury. If the hypotensive patient has normal chronotropy and inotropy, then agonists act as peripheral vasoconstrictors such as phenylephrine is indicated. Norepinephrine may also be considered, as it has agonist activity. Epinephrine and vasopressin infusions may be used in cases of refractory hypotension. Evidence for improvement in MAP goals (MAP >85–90 mm Hg over 7 days) in adults published by the Congress of Neurological Surgeons is weak. Of course, the definition of hypotension in children (systolic blood pressure < 70 mm Hg plus twice the age in years) is appropriate to consider when preventing and treating shock in general in children, but in the case of SCI, blood pressure may have to be maintained higher than the minimum acceptable blood pressure; blood pressure goals in pediatric patients with SCI are unknown.

If bradycardia is present, the patient may respond to infusion of atropine, glycopyrrolate, or vasoactives with chronotropic, vasoconstrictor, and inotropic properties such as dopamine or norepinephrine. Also, isoproterenol may be considered if a strictly chronotropic agent is required.2 Phenylephrine has the potential to cause reflex bradycardia, due to its lack of agonist activity, and

should be used with caution in patients with bradycardia as part of their neurogenic shock presentation. In rare cases, cardiac pacing has been successful but because the cause of bradycardia is neurochemical rather than electrophysiological, it may be prudent to use pharmacological treatment. If the patient Uncompensated vagal effects Loss of tone Sympathetic vasoconstriction Peripheral vasodilation Hypotension and bradycardia Neurogenic Shock shows some sensitivity to suction or positioning, one may consider administering atropine or glycopyrrolate before manipulation. Methylxanthines (theophylline, aminophylline) and propantheline have also been used for refractory bradycardia. Sinus bradycardia occurs most often in patients with severe cervical SCI, but patients can experience other dysrhythmias, including AV block, atrial fibrillation, or even cardiac arrest.

4. CONCLUSSION

Neurogenic shock is a distributed shock characterized by loss of sympathetic tone due to acute spinal cord or central nervous system injury. There are various causes of neurogenic shock, including cervical spine injury, spinal anesthesia, Guillain-Barre syndrome, and transverse myelitis. While neurogenic shock is mostly associated with cervical spine injuries and high thoracic injuries, neurogenic shock occurring from transverse myelitis is very rare. Trauma causes a sudden loss of sympathetic stimulation to the blood vessels. This causes them to relax (vasodilation), resulting in a sudden drop in blood pressure (secondary to a decrease in peripheral vascular resistance). Neurogenic shock occurs due to damage to the spinal cord above the 6th thoracic vertebra. There are no definitive diagnostic tests, but classically patients present with relative hypotension and bradycardia. Bradycardia is often exacerbated by suction, defecation, turning, and hypoxia. Initially the skin is often warm and red. Hypothermia may develop due to profound vasodilation and heat loss. Often central venous pressure is low due to decreased systemic vascular resistance.

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