UDC 61

THE IMPACT OF SPINAL BRAIN INJURY ON THE OCCURRENCE OF CARDIAC RHYTHM DISORDERS

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Abstract: Traumatic spinal cord injury, which results in spinal cord injury, is the cause of a number of cardiovascular disorders, including extrasystoles and myocardial fibrillation. It is especially important to understand the correlation with damage to the cervical and high thoracic regions, because it is with such injuries that the risk of episodes of bradycardia, atrioventricular block (AV-blockade) and cardiac arrest is greatest due to constant parasympathetic activity. Such patients should be cared for in intensive care units to protect against negative consequences.

Keywords: Spinal cord injury, arrhythmia, autonomic innervation of the heart, ECG, atrial fibrillation.

Introduction: Traumatic spinal cord injury (TSCI) results from damage to the bony, ligamentous, and/or neurological structures of the spine and can cause significant morbidity and mortality. The overall global incidence is 10.5 cases per 100,000 population [1]. The majority of spinal cord injuries (60%) affect young, healthy males between the ages of 15 and 35 years, with the most common injury occurring in the cervical spine [2]. The incidence of cardiac arrhythmias among individuals with spinal cord injury (SCI), particularly those with thoracic injuries, remains unknown. If clinicians could stratify patients into high and low risk groups for cardiac arrhythmias and death following SCI, lives could be saved.

Without tonic influence from the autonomic nervous system, the human heart

contracts at a rate of approximately 100 beats per minute, which is the spontaneous rate of generation of pacemaker cells in the sinoatrial node. During rest, the normal heart rate (HR) is much lower, reflecting the inhibitory effect of parasympathetic tone. In contrast, sympathetic control of the heart originates from the upper thoracic spinal cord (Th1–Th5) [3].

The autonomic nervous system modulates cardiac electrophysiology, and autonomic dysfunction increases the risk of ventricular arrhythmias. In addition, cardiovascular morbidity and mortality are high among individuals with spinal cord injury due to a relatively sedentary lifestyle and a higher prevalence of other cardiovascular risk factors, including obesity and diabetes [4].

It has been hypothesized that patients with cervical and high thoracic SCI have impaired sympathetic innervation of the heart, indicating a higher risk of episodes of bradycardia, atrioventricular block (AV block), and cardiac arrest due to persistent parasympathetic activity [5].

Individuals with SCI, particularly at the level of the sixth thoracic vertebra (T6) or above, often have dysregulation of sympathetic vasoconstriction of the peripheral vasculature and splanchnic circulation, as well as impaired control of heart rate and cardiac output. In addition, impaired descending sympathetic control results in alterations in circulating plasma catecholamine levels, which can have profound effects on cardiovascular function. Although individuals with lesions below T6 often have normal resting blood pressure, there is evidence of increased resting heart rate and inadequate cardiovascular responses to autonomic provocations, such as head-up tilt and cold-face tests [6].

There is a study using a rat model of complete spinal cord injury at the 2nd/3rd thoracic (T2/3) and T9 levels to investigate cardiac rhythm disturbances resulting from SCI. Four weeks after SCI, rats were implanted with a radiotelemetry device for electrocardiogram and blood pressure monitoring. During 24-h recordings, heart rate variability in rats with T2/3 but not T9 injury exhibited a significant reduction in the time domain, and a decrease in power at low frequency but increased power at high frequency in the frequency domain which indicates reduced sympathetic and

increased parasympathetic outflow to the heart.

Pharmacological blockade of the sympathetic or parasympathetic branches confirmed the imbalance in autonomic control of the heart. Activation of the sympathovagal influence during induction of autonomic dysreflexia by colorectal distension caused various serious arrhythmias in rats with T2/3 injury. Meanwhile, intravenous infusion of the β1-adrenoceptor agonist, dobutamine, caused a higher frequency of arrhythmias in rats with T2/3 injury than in untreated controls and in animals with T9 injury. Taken together, the results indicate that high-level SMT increases the susceptibility to the development of cardiac arrhythmias, probably through a disruption of autonomic homeostasis [7].

Complete loss of sympathetic innervation results in loss of supraspinal control and is termed “decentralization” of the sympathetic nervous system. The result is loss of distal reflexes and vasomotor tone, leading to low resting blood pressure caused by vasodilation and loss of cardiac inotropy, elements that can cause neurogenic shock.

Bradycardia is a consequence of parasympathetic vagal tone, which opposes the sinus node, reducing conduction in the AV node and bundle of His, resulting in episodes of AV block. Thus, some patients with cervical SCI may be prone to bradycardia and sinus arrest due to vagal overactivity during, for example, tracheal stimulation. In individuals with an intact spinal cord, tracheal stimulation results in tachycardia and hypertension, a response that depends on sympathetic efferent pathways under supraspinal control.

Supraventricular tachycardia caused by atrial fibrillation with 12-lead ECG confirmation was observed in patients with cervical SCI, which peaked within the first 2 weeks. This was not observed in patients with thoracic SCI [5]. A higher incidence of nonspecific ST-segment elevation has also been reported in individuals with SCI. These individuals also had an increased risk of developing bundle branch block and intraventricular conduction delay compared with the intact population [5, 8].

Atrial fibrillation is a complex condition caused by several underlying mechanisms, including dysregulation of the autonomic nervous system. Studies have

shown that the risk of developing it after long-term follow-up was higher in the SCI group, especially in patients with thoracic spine injury, lumbosacral spinal cord injury, and multilevel spinal cord injury [9].

Individuals with SCI from the rostral to thoracic vertebra T5, where sympathetic fibers exit the spinal cord and innervate the immune system, have clinically significant systemic inflammation and an increased risk of infection. Recent studies suggest that epidural lumbosacral spinal cord stimulation, delivered at the lumbosacral level using targeted configurations that promote cardiovascular stability, can safely and effectively normalize blood pressure in individuals with chronic SCI [10].

Conclusions: It is appropriate for patients with acute SCI to be admitted to the intensive care unit, especially if the patient has high-grade SCI or demonstrates hemodynamic instability. Several studies have shown that intensive care unit monitoring and aggressive medical management improve outcomes in patients with acute SCI. In addition, such patients may develop multiple (cardiovascular, respiratory, and urinary) complications, trophic skin changes, and heterotopic ossification. It is also worth noting that traumatic spinal cord injury has been reported to cause bradycardia, which has caused cardiac arrest in 16% of patients with cervical segment injuries. Therefore, an interdisciplinary approach based on the intensive care unit is necessary to minimize the various problems [11].

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