Autonomic Dysfunction in Spinal Cord Injury

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Autonomic dysfunction following spinal cord injury, which includes autonomic dysreflexia, orthostatic hypotension, body temperature dysregulation, bladder dysfunction, and bowel dysfunction, strongly influences the quality of life in spinal cord injury patients. Although understanding the autonomic dysfunction in spinal cord injury patients will improve their health and quality of life, few medical professionals are familiar with the dysfunction and its management. Given the sharp rise in the number of elderly patients with spinal cord injury in recent years, we consider that health professionals should be able to provide proper information on the management of autonomic dysfunctions to patients with spinal cord injury and their caregivers. This paper describes an overview of the autonomic dysfunction commonly found in people with spinal cord injury.

Key words: spinal cord injury, autonomic dysfunction, autonomic dysreflexia, orthostatic hypotension, body temperature dysregulation

Introduction

Impaired autonomic functions following spinal cord injury strongly influence the patients' quality of life. Although understanding the autonomic dysfunction in persons with spinal cord injury is crucial to improving the health and quality of life in these individuals, few medical professionals are familiar with the dysfunction. This paper briefly summarizes the main points of spinal cord injury and autonomic nervous system, and then provides an overview of the autonomic dysfunction commonly found in individuals with spinal cord injury.

Spinal cord injury

Spinal cord injury, which involves damage to the nerves within the spinal canal, often causes permanent changes in sensory, motor and autonomic functions below the site of the injury. Physical trauma, such as motor vehicle accidents, catastrophic falls and sports injuries, fractures or dislocates the vertebrae. The fractured or dislocated vertebrae irreversibly damage the corresponding level of the spinal cord, impeding neural communication between the brain and the body. The level of the injury and the severity of the damage to the nervous tissue determine how body functions remain intact or become disabled.

Recent advances in regenerative medicine research offer hope to spinal cord injury patients, their family, and health professionals. In particular, regenerative medicine using induced pluripotent stem (iPS) cells is anticipated to repair spinal cord injuries in the near future1). Given that the development of regenerative therapy for spinal cord injury still requires time, appropriate treatments, rehabilitation, and daily management can
contribute significantly to improving the quality of life in spinal cord injury patients.

**Epidemiology of spinal cord injury in Japan**

Even though Japan does not have a national database of spinal cord injury, there are an estimated 5,000 new spinal cord injury patients each year, and the total is probably more than 100,000 patients. A survey carried out from 1990 to 1992 concluded that the annual incidence of traumatic spinal cord injury was 40.2/1,000,000\(^2\).

However, a recent study showed a higher incidence of spinal cord injury especially in the elderly population\(^3\) (Figure-1). The epidemiological survey conducted in Tokushima Prefecture in 2011 and 2012 revealed that the annual incidence of traumatic spinal cord injury in elderly people (≥65 years old) was 215.7/1,000,000 in 2011 and 230.4/1,000,000 in 2012, whereas the incidence in individuals aged from 16 to 64 was 106.4/1,000,000 in 2011 and 90.8/1,000,000\(^3\).

Although other surveys indicate that there could be some regional differences in Japan\(^4\)-\(^6\), medical personnel should expect to witness a marked increase in elderly patients with spinal cord injury in the ensuing years.

**Autonomic nervous system**

The autonomic nervous system regulates various physiologic processes, such as the heart rate, digestion, respiratory rate, and urination without conscious control. The autonomic nervous system plays an essential role in maintaining homeostasis, i.e., the dynamic equilibrium of the biological systems to maintain relative constancy of the internal environment in spite of external environmental changes.

The autonomic nervous system is divided into two major divisions; the sympathetic nervous system and the parasympathetic nervous system. The two systems act like an accelerator and a brake on a car.

The sympathetic nervous system is activated in response to stress and danger and increases the metabolic activity and rate by releasing epinephrine (Figure-2A). On the other hand, the parasympathetic nervous system is activated during rest, sleep, and digestion and restores blood pressure and the resting heartbeat (Figure-2B). These two systems have opposite effects on the body organs, which serves to balance each other in order to maintain homeostasis.

**Autonomic dysreflexia in spinal cord injury**

Autonomic dysfunctions are common consequences of spinal cord injury. Spinal cord injury disrupts the descending spinal voluntary motor and involuntary autonomic pathways, resulting in dysfunctions of the cardiovascular system, sudomotor system, bladder, bowel and sexual organs.

The levels and the severity of injury to these pathways produce a variety of autonomic dysfunctions depending on the altered supraspinal control of the sympathetic and parasympathetic nervous systems\(^7\).

Common autonomic dysfunction in spinal cord injury includes the following:
- Autonomic dysreflexia
- Orthostatic hypotension
- Body temperature dysregulation
- Bladder dysfunction
- Bowel dysfunction

**Autonomic dysreflexia**

People with spinal cord injury above T5–T6 occasionally suffer from an acute syndrome of massive and uncontrollable sympathetic discharge, which is known as autonomic dysreflexia. Prompt and correct medical intervention is required
because it may lead to a potentially life-threatening hypertension, which must be recognized immediately; however, few health professionals are familiar with this condition.

Symptoms of the autonomic dysreflexia include the following:
- High blood pressure (>20 mmHg above baseline)
- Pounding headache
- Flushed face
- Sweating above the level of spinal injury
- Nasal stuffiness
- Nausea
- Slow pulse (lower than 60 beats per minute)
- Goose flesh below the level of spinal injury

Autonomic dysreflexia can occur as a result of peripheral or visceral stimulation below the level of the injury. The stimulation induces a widespread activation of the sympathetic nervous system, causing a rise in blood pressure. Normally, nerve receptors in the heart and blood vessels detect this rise in blood pressure. Then, the brain sends a message to the heart, allowing the heartbeat to slow down and the blood vessels to dilate. However, the spinal cord lesion blocks the descending inhibitory signals to below the level of the injury, failing to lower the blood pressure. (Figure-3)

Table-1 shows possible causes of autonomic dysreflexia in spinal cord injury.

If autonomic dysreflexia is suspected, treatment should be initiated immediately. Table-2 summarizes the treatment of autonomic dysreflexia. Since bladder dilatation and bowel distention are common causes of autonomic dysreflexia, medical professionals should investigate these conditions initially.

Medications are generally used only if the offending stimulus cannot be identified and removed, or when the symptoms persist even after removal of
the suspected cause. Oral nifedipine administration is a suitable antihypertensive medication for this situation.

Prevention of autonomic dysreflexia is the best approach not only for patients with spinal cord injury, but also for health professionals. Therefore, patients with spinal cord injury should be educated on bladder and bowel management techniques. A brochure on autonomic dysreflexia written in Japanese published by Beppu National Rehabilitation Center For Persons With Severe Disabilities will help the patients (http://www.rehab.go.jp/beppu/book/pdf/livinghome_no25.pdf).

**Orthostatic hypotension**

Another blood pressure disturbance caused by the autonomic dysfunction is orthostatic hypotension. Orthostatic hypotension is a sudden and significant fall in blood pressure when a person assumes a standing position. The Consensus Committee of the American Autonomic Society and the American Academy of Neurology defines orthostatic hypotension as a reduction of systolic blood pressure of $>20$ mmHg or a decrease in diastolic blood pressure of $>10$ mmHg within 3 minutes of standing. Symptoms of orthostatic hypotension include dizziness, lightheadedness, blurred vision, nausea, paleness, and temporary loss of consciousness.

Spinal cord injury patients with prolonged bed rest often experience orthostatic hypotension even while sitting on a wheelchair for a few minutes. A lesion of the baroreflex loop because of spinal cord injury fails to activate the sympathetic nervous system, narrowing the blood vessels to adjust the blood pressure. The loss of lower extremity muscle pump function due to a spinal cord injury can also cause orthostatic hypotension. Excessive venous pooling in the lower extremities occurring from the lack of the skeletal muscle pumping decreases venous return to the upper body.

Initial treatment of orthostatic hypotension is to have the patient lie down in a bed or to lean against the back of a wheelchair. Abdominal binders or compression garments can be helpful for patients with severe orthostatic hypotension. Gradually increasing the frequency of a sitting posture should improve the blood pressure adjustment. Hence, health professionals should encourage the patients

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**Table 1** Possible causes of autonomic dysreflexia in spinal cord injury

<table>
<thead>
<tr>
<th>Category</th>
<th>Noxious stimulus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bladder</td>
<td>Infection, Distension</td>
</tr>
<tr>
<td>Urinary tract</td>
<td>Urethral distension, Instrumentation, Calculus</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Distension, Instrumentation, Infection or inflammation, Ulceration, Reflux</td>
</tr>
<tr>
<td>Anorectal</td>
<td>Distension, Instrumentation, Hemorrhoids, Anal fissure</td>
</tr>
<tr>
<td>Dermatologic</td>
<td>Pressure sore, Ingressed ossification</td>
</tr>
<tr>
<td>Skeletal</td>
<td>Heterotopic ossification, Fracture, Joint dislocation</td>
</tr>
<tr>
<td>Reproductive</td>
<td>Labour and delivery, Menstruation, Testicular torsion, Ejaculation, Intercourse</td>
</tr>
<tr>
<td>Hematologic</td>
<td>Deep vein thrombosis, Pulmonary embolism</td>
</tr>
<tr>
<td>Central nervous system</td>
<td>Syringomyelitis</td>
</tr>
<tr>
<td>Medications</td>
<td>Nasal decongestants, Sympathomimetics, Misoprostol</td>
</tr>
</tbody>
</table>

**Table 2** Treatment steps for autonomic dysreflexia in spinal cord injury

<table>
<thead>
<tr>
<th>Intervention</th>
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<tbody>
<tr>
<td>1. Sit the patient upright</td>
</tr>
<tr>
<td>2. Loosen any tight clothing or constrictive devices</td>
</tr>
<tr>
<td>3. Monitor the blood pressure every 2 to 5 minutes during the episode</td>
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<tr>
<td>4. If no indwelling catheter is present, perform an intermittent catheterization</td>
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<td>5. In an indwelling catheter is present, hek it for obstructions and irrigate the catheter</td>
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<tr>
<td>6. If symptoms are still present and systolic blood pressure is $150$ mmHg or greater, treat the blood pressure pharmacologically</td>
</tr>
<tr>
<td>7. If symptoms are still present and systolic blood pressure is less than $150$ mmHg, manually disimpact the bowel</td>
</tr>
<tr>
<td>8. If symptoms persist, search for other precipitants (see Table-1)</td>
</tr>
<tr>
<td>9. Consider admission or referral if symptoms persist or no precipitant is found</td>
</tr>
</tbody>
</table>
to stay active and to avoid lying down on a bed for a long time.

**Body temperature dysregulation**

In recent years, heat stroke has become a social concern in Japan. According to the Fire and Disaster Management Agency, more than 55,000 people with heat stroke were transported to emergency hospitals from May 2015 to September 2015 ([http://www.fdma.go.jp/neuter/topics/houdou/h27/10/271016_houdou_1.pdf](http://www.fdma.go.jp/neuter/topics/houdou/h27/10/271016_houdou_1.pdf)). Although elderly persons have increased risk for developing heat stroke, patients with spinal cord injury also carry a high risk for hyperthermia because of body temperature dysregulation.

Most people with complete spinal cord injuries do not sweat below the level of the injury. Furthermore, many quadriplegics cannot even sweat above the level of the injury. With the loss of the ability to sweat, patients with high spinal cord injury are unable to lower their body temperature in a hot environment. Therefore, when patients with high spinal cord injury stay in temperature over 25°C with high humidity even for a short period of time, the body core temperature begins to rise immediately, and quickly develop hyperthermia. The symptoms of hyperthermia include nausea, headache, nasal congestion, tiredness, low blood pressure and reduced concentration.

One of the best treatments for spinal cord injury patients with hyperthermia is to wrap a cold wet towel around the neck. Health professionals should advise people with spinal cord injury to avoid hot environments and to turn on an air conditioner or an electric fan without any hesitation.

It should be noted that people with spinal cord injury are more prone to developing hyperthermia during exercise than people without spinal cord injury. Since Tokyo was elected as the host city for the 2020 Olympic and Paralympic games, the number of people with spinal cord injury who exercise regularly have increased. Medical personnel should provide education on how to exercise and prepare for competitions in the heat.

**Bladder dysfunction**

Spinal cord injury at any level almost always affects bladder functions, because the nerves controlling the bladder originate in the sacral spinal cord (S2–S4). The kidneys and ureters still work properly even after the spinal cord injury and keep producing urine and carrying the urine to the bladder.

Normally, when the stored urine expands the bladder wall, the stretch receptors send a message through parasympathetic sensory nerves and the spinal cord to the brain. Then, the brain sending a message back down the spinal cord and parasympathetic motor neurons to the bladder allows the detrusor muscle in the bladder wall to contract and the sphincter muscle to relax for voiding. (Figure 4) As a result, urine passes down the urethra to exit the body.

Injury to the spinal cord interrupting the neural communication between the bladder and the brain impairs the normal bladder functions which involve the autonomic action of coordinating the relaxation of the sphincter muscle and contracting the detrusor muscle, leading to a difficulty in proper urination.

Following a spinal cord injury, the bladder is usually affected in one of two ways: spastic (reflex) bladder and flaccid bladder. Spastic (reflex) bladder occurs when the stretch receptors are triggered from filling the bladder with urine. Since
Spinal cord injury blocks ascending messages to the brain, a micturition reflex is triggered at the sacral spinal cord to empty the bladder. Spastic bladder usually occurs when the injury is above the T12 level. Flaccid bladder occurs when the signals from the stretch receptor fail to contract the detrusor muscles even when the bladder is full. This can cause the bladder to become over-stretched. Prolonged fullness of the bladder forces the urine to flow back into the kidneys through the ureters, which is called "vesicoureteral reflux."

In both types of impairment, bladder management to prevent urinary tract infection is important. As residual urine in the bladder mostly causes urinary tract infection, bladder management begins with complete emptying of the bladder. Intermittent catheterization (ICP) is the preferred method for spinal cord injury patients to completely empty the bladder.

**Bowel dysfunction**

Spinal cord injury often disrupts the bowel function, resulting in complications ranging from constipation to bowel accidents. Bowel dysfunction following spinal cord injury is increasingly recognized as a major life-style problem, reducing the quality of life for spinal cord injury patients.

When the rectum walls are expanded with stools, stretch receptors in the rectal walls send messages to the brain through the spinal cord, causing an urge to defecate. The urge to defecate informs the person to go to the bathroom, and triggers the opening of the anal sphincter to push out the stool (Figure 5).

Bowel dysfunction mostly depends on the level of the injury, and just like bladder dysfunction, it can be categorized into two patterns: upper motor neuron bowel and lower motor neuron bowel. The upper motor neuron bowel resulting from a spinal cord lesion above the sacral level damages the urge to defecate. The anal sphincter will remain tight even when the rectum becomes full, and will open on a reflex basis. The lower motor neuron bowel, which results from a lesion to the sacral spinal cord and nerve roots, causes the loss of defecation reflex and the anal sphincter muscle remains open. As a result of these bowel dysfunctions, defecation may occur at any time and places in complete disregard of the patients’ wishes.

Bowel management is necessary for patients with spinal cord injury to stay healthy and to minimize

**Conclusion**

Proper management of autonomic dysfunctions in spinal cord injury patients will improve their health and quality of life. Given the sharp rise in the number of elderly patients with spinal cord injury in recent years, health professionals should be informed and prepared to provide proper information on the management of autonomic dysfunctions to spinal cord injury patients and their caregivers.

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**References**