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Acute complications of spinal cord injuries

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Abstract

The aim of this paper is to give an overview of acute complications of spinal cord injury (SCI). Along with motor and sensory deficits, instabilities of the cardiovascular, thermoregulatory and broncho-pulmonary system are common after a SCI. Disturbances of the urinary and gastrointestinal systems are typical as well as sexual dysfunction. Frequent complications of cervical and high thoracic SCI are neurogenic shock, bradyarrhythmias, hypotension, ectopic beats, abnormal temperature

control and disturbance of sweating, vasodilatation and autonomic dysreflexia. Autonomic dysreflexia is an abrupt, uncontrolled sympathetic response, elicited by stimuli below the level of injury. The symptoms may be mild like skin rash or slight headache, but can cause severe hypertension, cerebral haemorrhage and death. All personnel caring for the patient should be able to recognize the symptoms and be able to intervene promptly. Disturbance of respiratory function are frequent in tetraplegia and a primary cause of both short and long-term morbidity and mortality is pulmonary complications. Due to physical inactivity and altered haemostasis, patients with SCI have a higher risk of venous thromboembolism and pressure ulcers. Spasticity and pain are frequent complications which need to be addressed. The psychological stress associated with SCI may lead to anxiety and depression. Knowledge of possible complications during the acute phase is important because they may be life threatening and/ or may lead to prolonged rehabilitation.

Key words: Spinal cord injuries; Autonomic dysreflexia; Cardiovascular disease; Orthostatic hypotension; Bradycardia; Thromboembolism; Respiratory insufficiency

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Core tip: The paper provides an overview of acute complications of spinal cord injury. Frequent complications in the acute phase of are bradyarrhythmias and hypotension. Other complications are instability of temperature (hypothermia and hyperthermia), pain, spasticity and autonomic dysreflexia (AD). AD is associated with an abrupt, uncontrolled sympathetic response, elicited by stimuli below the level of injury, and it can cause severe hypertension, cerebral haemorrhage and death. All personnel caring for the patient should be able to recognize the symptoms and intervene promptly. Knowledge of possible complications during the acute phase is important because they may be life-threatening and/or may lead to prolonged rehabilitation.

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INTRODUCTION

Traumatic spinal cord injury (SCI) may cause long-lasting dysfunction in many organ systems, and together with permanent change of function, lead to a higher morbidity together with a lower quality of life^[1,2]. The management of acute SCI has changed significantly during the past decades due to increased knowledge about the pathophysiology of SCI together with new diagnostic methods and treatment methods. The spinal cord is affected by both the immediate physical effects of trauma, and secondary pathologic processes. Especially ischemia and oedema may worsen the injury during the first few hours after an injury.

Knowledge of possible complications during the acute phase is important because they may be life-threatening and/or may lead to prolonged rehabilitation.

DEFINITION

Traumatic spinal cord injury is defined as an acute injury of the spinal cord which results in a varying degree of paralysis and/or sensory disorder^[3]. Injury to the cauda equina is usually included in the definition, while other isolated injuries to nerve roots are excluded^[4].

Based on pathophysiological changes the early acute phase is defined to be 2-48 h after the injury, the subacute phase from 2 d to 2 wk, and the intermediate phase from 2 wk to 6 mo^[5]. Based on timing of surgery studies have found that early decompression either < 24 h or < 72 h resulted in statistically better outcomes compared to delayed decompression^[6]. However, the clinically acute phase is usually defined as the first 4-5 wk after the injury.

ANATOMY

An acute traumatic SCI starts with an abrupt, injury to the spine leading to fractures or dislocations of vertebrae. Displaced bone fragments and disc material causes the immediate injury leading to irreversible damage of axons and broken neural cell membranes. Ruptured blood vessels may cause bleeding in the spinal cord, and thereby increase the damage during the subsequent hours. Several mechanisms contribute to the total injury of the spinal cord tissue.

Goals in the management of SCI-patients include minimizing the primary neurological damage, and preventing secondary cord injury due to hypoperfusion, ischemia, and apoptotic, biochemical and inflammatory changes^[7].

An acute injury above the sixth thoracic (Th6) vertebra disturbs the descending pathways to neurons of the sympathetic trunk (in the intermediolateral cell

column) from the first thoracic (Th1) to the second lumbar (L2) vertebrae. The consequences are abolished supraspinal control of the sympathetic nervous system, and lack of inhibition of the parasympathetic nervous system resulting in an increased sympathetic activity below the injury level. Along with motor and sensory deficits, instabilities of the cardiovascular, thermoregulatory and broncho-pulmonary system are common after a SCI. Disturbances of the urinary and gastrointestinal systems are typical as well as sexual dysfunction^[8,9]. Patients with injury below Th6 will have intact sympathetic and parasympathetic control of the heart and lungs. Thus, the responses from the organ systems will differ between patients with tetraplegia and patients with paraplegia^[10].

SURGERY

After a traumatic SCI, the number of complications during the acute phase hospitalization, depends on the timing of surgery, with less complications when surgery is performed soon after the injury^[11]. It's proposed that patients with traumatic SCI should be operated within 24 h following injury to reduce complications. If impossible to operate within 24 h, efforts should be made to perform surgery earlier than 72 h after a the injury^[11].

ACUTE COMPLICATIONS

Neurogenic shock

Neurogenic shock is due to severe hypotension and bradycardia in cervical injuries due to drop in blood pressure in relation to an acute SCI^[12,13]. Hypotension is defined as systolic blood pressure < 90 mmHg in supine position, and is due to low intravascular volume (e.g., blood loss, dehydration)^[8]. Because of an intact parasympathetic influence *via* the vagal nerve and a loss of sympathetic tone due to disruption in supraspinal control, neurogenic shock develops as a result of imbalance of the autonomic control^[12]. Depending on the severity of the SCI, prolonged and severe hypotension, requiring vasopressive therapy may last up to 5 wk after injury^[12].

In the Trauma Audit and Research Network database, the percentages of neurogenic shock was 19.3% in cervical injuries^[14]. In thoracic and lumbar injuries the reported incidence was 7.0% and 3.0%, respectively^[14].

Cardiovascular disease

Injuries to the autonomic nervous system are the cause of many of the cardiovascular complications following a SCI. Cardiovascular dysfunction in patients with cervical and high thoracic SCI may be life-threatening and may exacerbate the neurological impairment due to the spinal cord injury. Patients have higher morbidity and mortality as a result of the autonomic dysfunction^[15-17]. A Canadian study found that SCI is associated with an increased odds of heart disease (OR = 2.72) and stroke (OR = 3.72) compared to ablebody^[18].

In the acute phase many irregularities of the cardiac rhythm may occur; sinus bradycardia and bradyarrhythmias

(14%-77%)^[19] including escape rhythm, supraventricular ectopic beats (19%)^[19], ventricular ectopic beats (18%-27%)^[19,20], orthostatic hypotension (33%-74%)^[21,22], increased vasovagal reflex, vasodilatation and stasis^[20]. Sidorov *et al*^[23] found that orthostatic hypotension persisted during the first month following SCI in 74% of cervical and 20% of upper thoracic motor complete SCI patients. Following cervical injuries both sinus bradycardia and arterial hypotension frequently arise^[8,24-26]. Bradycardia is reported in 64% to 77% of cervical SCI^[20]. Studies have found a peak in incidence four days post-injury, then a gradual decline in incidence^[27]. Arterial hypotension is reported in 68% of patients with motor complete cervical SCI (ASIA A and B) who develop bradycardia. Of these will 35% require vasopressors, and 16% will have a cardiac arrest^[28]. In the acute phase arterial hypotension in the acute phase can be misunderstood as loss of volume. This may lead to over hydration in the acute phase.

Common autonomic disturbances after 4 to 5 wk post-injury are autonomic dysreflexia, orthostatic hypotension (also in sitting position), reduced cardiovascular reflexes (which regulate blood pressure, blood volume and body temperature) and the absence of cardiac pain^[20]. The prevalence of autonomic dysreflexia in patients with SCI with injury above Th6 is 48%-90%^[28,29]. Krassioukov *et al*^[30] found an incidence of early AD of 5.2% in a population of acute SCI, the earliest episode of AD occurred on the 4th post-injury day. Patients with cervical or thoracic injuries above Th4 may have disrupted the sympathetic afferent fibres including cardiac pain fibres; their sensation of ischemic cardiac pain may be changed (referred pain) or absent^[31].

Secondary cardiac changes in patients with tetraplegia, are loss of muscle mass in the left ventricle (due to physiological adaptation to reduced myocardial load^[32]) and pseudo infarction - a rise in Troponin with or without ECG changes^[25,33].

TEMPERATURE REGULATION

Abnormal temperature control is another well-known clinical phenomenon after SCI, especially in patients with cervical and high thoracic injuries. This is largely due to reduced sensory input to thermo-regulating centres and the loss of sympathetic control of temperature and sweat regulation below the level of injury^[34]. A number of temperature regulation disorders following SCI have been described. Some patients have poikilothermia-an inability to maintain a constant core temperature irrespective of the ambient temperature. Injuries above Th8 are often associated with fluctuating temperature, hypothermia and hyperthermia^[25].

SWEAT SECRETION

The sweat glands are largely sympathetically innervated in the upper part of the body from Th1-Th5, and in the lower part of the body from Th6-L2. Supraspinal control of sweat excretion is located in regions of the hypothalamus

and amygdala^[34]. Changes in sweat secretion often occur after SCI, and excessive sweating (hyperhidrosis), absence of sweating (anhidrosis) and diminished sweating (hypohidrosis) may all occur.

Excessive sweating is a common problem in persons with SCI^[35,36]. In most individuals, episodic hyperhidrosis is usually associated with other autonomic dysfunctions such as autonomic dysreflexia and orthostatic hypotension, or with post-traumatic syringomyelia. Most common symptoms are minimal/abolished sweating under the level of injury and profuse sweating over the level of injury. This is due to compensatory increase in sweat secretion above the level of injury due to the loss of sympathetic stimulation below the level of injury, which results in reduced sweat production^[37]. Sweating may also occur exclusively below the level of injury. This type of sweat is reflex sweating, and is usually a symptom of a massive autonomic response that occurs particularly with cervical and high thoracic injuries (above Th8-Th10).

Respiratory complications and dysphagia

Cervical injury has major effects on the pulmonary system, and respiratory difficulties are one of the major complications and a frequent cause of death, both in the acute and chronic phase after injury^[38]. Studies have found that 67% of acute SCI patients experience severe respiratory complications within the first days after the injury; atelectasis (36.4%), pneumonia (31.4%), and respiratory failure (22.6%)^[39].

In the acute phase 84% of patients with injuries above C4 and 60% of patients with injuries from C5 to C8, will experience respiratory problems^[40], and 75%-80% of tetraplegia above C4 and 60% of tetraplegia caudal to C4 will need invasive mechanical ventilation^[41]. Close surveillance of respiration is important. In addition a total of 65% of patients with injuries at levels from Th1 to Th12 may have severe respiratory complications^[42]. A 30%-50% reduction of vital capacity is described during the first week post injury in patients with injuries at C5-C6. It is recommended that vital capacity and arterial blood gases should be measured until the patient is stable^[41-43].

Thromboembolism

Individuals with SCI have a higher risk of coagulation disorders and venous stasis due to physical inactivity, altered haemostasis with reduced fibrinolytic activity and increased factor VIII activity^[44]. They are therefore predisposed to thromboembolism^[45,46]. During the first year post-injury, the incidences of deep vein thrombosis and pulmonary embolism are estimated to be 15% and 5%, respectively^[47]. The incidence is highest 2-3 wk after the injury, followed by a small peak three months after the injury^[48]. During the chronic phase, the incidence of clinically significant thromboembolism is less than 2%^[44].

Pressure ulcers

Pressure ulcers are a common complication following SCI. Good prevention requires identifying the individuals at risk for developing pressure ulcers^[49]. Pressure ulcer is the

most common long term complication in SCI. Meticulous surveillance in the acute phase and in the operating theatre to prevent pressure ulcers is vital^[50].

Heterotopic ossification

Heterotopic ossification (HO) is a frequent, irreversible complication after SCI^[51], and involves para-articular formation of mature lamellar bone in soft tissues^[52]. The incidence varies between 10% to 53% in different studies^[51,53]. The development of HO starts usually within the first 2-3 wk post injury below the level of injury^[51,53]. The most common joints affected are hip (70%-97%) and knee^[51,53]. Substantial HO, the patients present with a reduction in range of motion of the joint in 20%-30%^[53], whereas ankylosis develops in only 3% to 8%^[53].

BLADDER

SCI interrupts control of the bladder^[54]. Immediately after SCI, the bladder and sphincter are frequently hypotonic. In the chronic phase the bladder dysfunction is classified as either an upper or lower motor neuron syndrome.

Upper motor neuron syndrome (reflex bladder) involves loss of cortical inhibition of sacral reflex arcs due to disturbance of descending spinal tracts, leading to detrusor hyperactivity often in combination with detrusor sphincter dyssynergia^[55]. Inhibition of the stretch reflex by the pontine storage centre is abolished. A minor amount of stretch will give a contraction of the bladder wall, the external urethral sphincter lacks voluntary control, resulting in recurrent, spontaneous voiding.

Lower motor neuron syndrome is due to injury to the sacral (S2-S4) part of the autonomic nervous system resulting in a diminished motor stimulation of the bladder and reduced or absent contractility of the detrusor and subsequently an enlarged bladder^[56,57].

BOWEL

Between 27% and 62% of patients with SCI report having problems with their bowel, the most frequent symptoms are obstipation, distension and abdominal pain^[58]. Other symptoms are rectal bleeding, haemorrhoids, incontinence and autonomic dysreflexia^[58]. Spinal shock leads to loss of all activities, under the level of injury, including autonomic function and reflexes. During the first four weeks, 4.7% of patients experienced acute abdominal symptoms, while 4.2% reported acute gastro duodenal ulceration and haemorrhage^[58,59].

Spasticity

Patients with an acute complete SCI present with spinal shock associated with muscle paralysis, reduced muscle tone and absent tendon reflexes under the level of injury^[60]. Spasticity is usually established after 2-6 mo post injury with exaggerated tendon reflexes, increased muscle tone, and muscle spasms^[61,62]. Up to 70% of patients with SCI develop spasticity^[63].

Pain

In the acute phase the patients encounter a range of sensory experiences following the trauma. Acute pain commonly accompanies the injury and recedes as healing occurs. Chronic pain is a frequent, disabling complication of SCI. Up to 80% of patients with SCI are reported to suffer from pain^[64]. Patients with SCI may have nociceptive or neuropathic-type of pain or a combination of the two^[65].

In order to reduce the evolution of chronic pain, it is important to minimize the primary neurological damage, and prevent secondary injury due to hypoperfusion, ischemia, and apoptotic, biochemical and inflammatory changes of the cord^[66].

MUSCULOSKELETAL AND METABOLIC COMPLICATIONS

Musculoskeletal pain is common in chronic SCI^[67]. The muscles atrophy in response to reduced activity^[61]. Studies have found that all patients with complete SCI have some extent of deterioration of muscle, joints and ligaments^[61]. Therefore, the patients with SCI experience a period of "metabolic chaos", *i.e.*, an strong catabolic process, which is generated by the loss of physical pressure on muscle, joints and ligaments^[61]. This results in bone demineralization leading to hypercalciuria, renal urolithiasis and bladder stones, which may lead to renal failure^[61].

IMMUNOLOGICAL MEDIATED NEURO-INFLAMMATION

Excessive activity of matrix metalloproteinases (MMP) in the cord immediately after the injury lead to break of the blood-spinal cord barrier, entering of leukocytes into the injured cord, and disintegration of cells^[68]. Studies have shown that MMP-9 and MMP-2 both are important in the regulation of inflammation and neuropathic pain after peripheral nerve injury. They may also contribute to the SCI-induced pain^[68]. By blocking the effects of MMP early using pharmacologic agents, an improvement in long-term neurological recovery may be possible, together with reduced glial scarring and neuropathic pain^[68].

SEXUALITY

Immediately after a SCI, most patients are focused on the physical improvement. However, when they manage to accept their injury, dealing with sexuality is an important step in the physical and psychological rehabilitation process^[69].

ANXIETY AND DEPRESSION

Many patients with SCI experience psychological stress. Patients with a good mental health are usually capable of coping with stress, but the patients response is affected

by the cause and extent of injury, and the patients current life situation^[70]. Proper attention and care for each patient's way of dealing with their injury psychologically are important. To prevent or minimize the problems physical, pharmacological or psychological interventions should be available. Interventions will be pain relief, avoidance of sensory and/or sleep deprivation, providing a familiar atmosphere, as well as giving the patient careful explanations and reassurance^[70,71]. If possible the patient should have access to psychotherapy and pharmacological treatment during their rehabilitation^[70,71].

ASSOCIATED INJURIES

Many patients with SCI have associated injuries to other body parts and organ systems, which may affect negatively affect rehabilitation outcome. The most commonly associated injuries include extremity fractures (29.3%), loss of consciousness (28.2%), pneumothorax (17.8%), and traumatic brain injury affecting cognitive or emotional functioning (11.5%)^[72].

SPECIALIZED CARE OF PATIENTS WITH SCI

Patients with acute traumatic SCI should be managed at a trauma centre with SCI experience, particularly patients with concomitant injuries^[73]. The first European centre specializing in SCI was established in 1944 at Stoke Mandeville Hospital in England. The objective of specialized SCI centres is to advance the care for patients with SCI and thereby improve the neurological recovery. In a recent review Parent *et al*^[74] found that early transfer to a specialized SCI centre, lead to a reduced length of stay and decreased mortality.

FUTURE RECOMMENDATIONS

Frequent complications in the acute phase after SCI are arrhythmias, bradycardia, hypotension, pain and spasticity. Knowledge of possible complications during the acute phase is important because they may be life-threatening and/or may lead to prolonged rehabilitation. There is still a need for increased knowledge about the acute cardiovascular complications following SCI as well as temperature regulation, pain and spasticity.

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