



Complications of Spinal Cord Injuries: Two Case Reports and a Review of the Literature

Spinal Cord Yaralanmalarında Komplikasyonlar: İki Olgu Sunumu ve Literatür Derlemesi

Complications of Spinal Cord Injuries

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Özet

Omurilik yaralanmaları etkilenen omurilik düzeyine bağlı olarak bireyde fiziksel kısıtlılığa yol açan ve birçok sistemi etkileyen bir sağlık problemidir. Etkilenen omurilik seviyesi ve bunun sonucunda gelişen disfonksiyonun derecesine bağlı olarak erken ve geç dönemde tedavinin yoğun bakım şartlarında sürdürülmesi gerekebilir. Bu hastalarda erken ve uygun tedavi ile sağ kalım artmaktadır. Yine hasta tedavi altındayken oluşabilecek komplikasyonlar açısından dikkatli olunmalı ve hasta konforu açısından bu komplikasyonlarla mücadele edilmelidir. Bizde yoğun bakımımızda servikal fraktürü olan ve kardiyovasküler ve solunumsal problemler ile karşılaştığımız iki vakayı sunmak istedik.

Anahtar Kelimeler

Spinal Kord Yaralanmaları; Pulmoner Komplikasyonlar; Kardiyovasküler Komplikasyonlar

Abstract

Spinal cord injuries are a health problem that affects many systems and leads to physical restrictions associated with the level at which the spinal cord is affected. It may be necessary to maintain early and late-stage treatment under intensive care conditions according to the degree of dysfunction that develops as a result of the level affected in the spinal cord. With the appropriate early treatment, survival increases in these patients. However, care must be taken with respect to complications that may develop while the patient is undergoing treatment and intervention must be made for these complications to provide patient comfort. In this paper, we present two cases of patients with cervical fractures who developed cardiovascular and respiratory problems while in the intensive care unit.

Keywords

Spinal Cord Injuries; Pulmonary Complications; Cardiovascular Complications

DOI: 10.4328/JCAM.4818

Received: 29.09.2016 Accepted: 26.10.2016 Printed: 01.03.2017

J Clin Anal Med 2017;8(2): 168-71

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Introduction

Spinal cord injuries are a health problem that affects many systems and leads to physical restrictions associated with the level at which the spinal cord is affected. It may be necessary to maintain early and late-stage treatment under intensive care conditions according to the degree of dysfunction that develops as a result of the level affected in the spinal cord. With the appropriate early treatment, survival increases in these patients. However, care must be taken with respect to complications that may develop while the patient is undergoing treatment and intervention must be made for these complications to provide patient comfort. In this paper, we present two cases of patients with cervical fractures who developed cardiovascular and respiratory problems while in the intensive care unit.

Case Report 1

A 34-year-old male was admitted to the Intensive Care Unit (ICU) from the Emergency Department because of a fall from height. In the physical examination, the patient was conscious, with a poor general status, temperature of 37°C, pulse of 40, blood pressure of 60/30, weak spontaneous respiration, and quadriplegia. On tomography, a C5-C6 fracture was determined and the patient was admitted with a trauma board and cervical collar. As respiration was poor, the patient was intubated and mechanical ventilation was applied. Fluid treatment was started as spinal shock was considered. Dopamine at a dosage of 10µg/kg/min was started because of hypotension. Intervention with atropine was made for spontaneous bradycardia. On the 3rd day of hospitalisation, the vital signs had improved and the patient was admitted for cervical stabilisation surgery. During the operation the patient was given one unit of erythrocyte suspension and postoperatively the patient was admitted to the ICU without any problems. From the 2nd day, as bradycardia and asystole developed during spontaneous and tracheal aspiration, intervention was made with atropine. While the procedures were being applied to the patient, hypertension attacks occurred, so intervention was made with nitrate. Cardiology consultation was requested and the recommendations were applied. On the 10th day, a tracheostomy was opened in the patient. Spontaneous respiration was attempted in the patient, who was conscious. From time to time, T-tube was attempted. As bradycardia and asystole continued, the Cardiology Department was again consulted and a temporary cardiac pacemaker was applied. Thereafter, no bradycardia or asystole was observed. The patient was monitored with spontaneous respiration or T-tube. As the mental status of the patient was thought to have been impaired, an evaluation was made by the Psychiatry Department and 50 mg quetiapine was added to the treatment. Complications that developed, such as pneumonia, were treated. On the 80th day of hospitalisation, the patient was exitus due to the development of sepsis.

Case Report 2

A 36-year-old male presented at the Emergency Department following a fall from height. On the cervical tomography examination, fractures were determined in the C4-C5-C6 vertebrae and clinically there was quadriplegia. Other findings were normal and the patient was conscious. With respect to respiratory follow-up, the patient was admitted to the ICU and monitored, initially with oxygen given by mask. On the 2nd day, the patient was operated on by the brain surgeon and cervical stabilisation was applied. Postoperatively, the patient was intubated and

transferred to ICU. On the same day the patient was extubated, a mask was applied, and respiratory physiotherapy was started. On the 5th day, due to respiratory problems and loss of consciousness, the patient was intubated and attached to a mechanical ventilator. Pneumonia developed and was treated. 'Weaning' was attempted. On the 20th day, during tracheal aspiration, asystole was observed and intervention was made with 0.5mg atropine. Sedation was applied to the patient. In the following days, as spontaneous bradycardia and asystole were observed, generally following tracheal aspiration, cardiology consultation was requested. On the 23rd day, a temporary pacemaker was applied and thereafter no bradycardia or asystole occurred. During follow-up, pneumonia and urinary tract infection developed and these were treated. On the 70th day, a percutaneous endoscopic gastrostomy (PEG) was applied. On day 110, ECO was applied for the etiology of a high fever which had developed. Following the determination of vegetation, the antibiotics were changed for the diagnosis of infective endocarditis. The general status of the patient deteriorated with low blood pressure values seen and septic shock was accepted. On the 3rd day of treatment for this, the patient was exitus due to septic shock.

Discussion

Spinal and spinal cord injuries are often encountered in intensive care units (ICU) and are difficult to treat. Causes in the etiology comprise traffic accidents in 38%, falls from height in 30%, assault in 14%, sports accidents in 9%, and other reasons in 4%. The mean age is 40.7 years and these injuries are seen more often in males [1]. In Turkey the mean age has been determined as 37.81 ± 13.65 years and the main cause of these injuries is vehicle accidents, at the high rate of 48% [2,3]. While high cervical lesions cause tetraplegia, lower lesions cause paraplegia. Tetraplegia and paraplegia have been reported to be present in half of these injuries.

Until proven otherwise, it should be considered that there may be spinal injury in every multi-trauma patient. Advanced life support must be provided for patients first, before the primary evaluation. The first task to be performed is to ensure that the airway is clear, and the respiratory and circulation parameters must be corrected. The possibility of a secondary life-threatening injury must be eliminated. Transport of patients with suspected spinal injuries must be made on a spinal board to provide sufficient cervical immobilisation [4].

According to the degree of dysfunction that develops as a result of the level of the spinal cord affected, it is necessary to maintain early and late-stage treatment in ICU [5].

In spinal injuries, the neurological effects that occur during trauma form the primary damage. However, the basic aim is to prevent secondary damage. Events occurring at the tissue and cellular level with impaired perfusion and oxygenation in the spinal cord create a chain, exacerbating the neurological effects [6]. Secondary damage may be increased for reasons such as hypotension, shock, reduced arterial oxygen, catecholamine expression, hypercoagulability, or hyperthermia. The aim must be to prevent and treat these causes [7].

Pulmonary complications in spinal injury cases are the most important and most common causes of morbidity and mortality [6-9]. When clearing the airways of patients with cervical spinal cord injuries, manoeuvres which will increase the damage must be avoided and the neck must be kept in a neutral position. In patients who develop respiratory failure because of the loss of

protective reflexes due to concomitant head trauma or coma, or a reduction in the diaphragm assistive respiratory muscles, intubation must be applied [6]. The respiratory functions are greatly affected in these patients and mechanical ventilation support is necessary. The extent of the effect on respiratory functions varies according to the level of the neurological deficit and whether or not it is a complete lesion [8].

In spinal injuries at the C1-C3 level, all the respiratory muscles are paralysed and mechanical ventilation is unavoidable. At the C3-C5 level, respiratory functions are affected to varying degrees and, with time, the respiratory functions may be corrected. In lesions below C5 when there is no chest trauma, the respiratory functions are not expected to be affected in the acute phase. However, in the subsequent period, with the increasing difficulty in clearing secretions and coughing that is associated with paralysis of the intercostal and abdominal muscles, an increase in pulmonary complications is expected [7].

Cardiovascular complications are a significant problem in the early and chronic stages, requiring early diagnosis and intervention. The degree of cardiovascular effect is closely related to the lesion location and level [10]. In cervical or upper thoracic injuries the supraspinal sympathetic control mechanism, which manages cardiovascular functions such as coronary blood flow, cardiac contractility, heart rate, and vascular tone is removed and the parasympathetic system becomes suppressed. As a result of this, cardiac arrhythmias occur, primarily bradycardia [11]. Hypotension develops resulting from arterial dilation and the pooling of blood in the periphery. This period, characterised by vasodilation, bradycardia, hypotension, reduced peripheral vascular resistance, and reduced pre-loading, may last for longer than five weeks and is known as neurogenic shock [6,7,10,11]. The aim of treatment is to maintain a blood pressure level that will provide oxygenation of the spinal cord and other organs. It has been reported that in the post-acute injury period, bradycardia is seen in 64%-77% of patients. It is seen most often in the first five weeks after injury and sometimes vasopressor treatment may be required. Occasionally heart rate may fall as low as 20 bpm and cardiac arrest may be seen at the considerably high rate of 15%. The aim in treatment is to maintain systolic blood pressure above 90 mmHg. If there is bradycardia together with hypotension, it is important to maintain heart rhythm and therefore anticholinergic drugs should be used. Sufficient oxygenation must be provided for the patient and vagal stimulus must be avoided. If heart rate does not increase to a sufficient level, atropine should be administered intravenously at a dose of 0.5 mg, not exceeding a total dose of 2 mg. If no response is obtained to the atropine, adrenaline should be used and a temporary pacemaker should be applied. The pacemaker can be applied by the transcutaneous or transvenous route [12,13]. Some studies have reported that there can be beneficial effects from the administration of 0.25-0.5 mcg/min isoproterenol infusion and theophylline 200mg bolus [14].

Another subject involving the cardiovascular system is autonomic dysfunctions. The primary pathology is that the injury results in interruption to the functions of the parasympathetic and sympathetic nerve functions, which are the two divisions of the autonomous nervous system. The events that occur are known as autonomic dysfunctions and these include orthostatic hypotension, autonomic dysreflexia, thermoregulation disorders, and hyperhidrosis.

Autonomic Dysreflexia: Although blood pressure values in those with cervical and upper thoracic region injuries are generally

lower than those of healthy individuals, the majority of these patients experience the event known as autonomic dysreflexia, which is characterised by hypertension attacks. As a result of disrupted control of the sympathetic neurons in the spinal cord of the central nervous system, the abnormal reflex that is formed against the stimulus in the level below the lesion is defined as sympathetic activity in the pathology [15]. A throbbing-type headache accompanying hypertension is the most commonly encountered complaint. Above the level of the lesion, redness and 'flushing', excessive sweating, and increased skin temperature on the face, neck, and arms, as well as nasal congestion, are seen. Below the level of the lesion, coldness, paleness, and piloerection as a result of sympathetic stimulation of hair follicles occur. Bradycardia develops with the parasympathetic effect and as a reflex response of high pressure baroreceptors [16].

The importance of untreated attacks is that they may cause intracranial bleeding, retinal bleeding, convulsion, myocardial infarct, and even death. In the treatment, first, the cause that has triggered the attack must be removed. Attempts to lower the blood pressure by putting the patient into a sitting position and loosening any tight clothing should be made. The preferred drugs for treatment are nifedipine and nitrates. When these are not effective, alternatives include captopril, terazosin, prazosin, and phenoxybenzamine. The blood pressure of the patient must be kept under close observation [17-19].

Orthostatic hypotension: This condition is defined as a fall in systolic blood pressure of > 20 mmHg or in diastolic pressure of >10mmHg when moving from a sitting to a standing position [20]. In the pathology, a disruption occurs in the descending cardiovascular pathway because of the damage in the spinal cord, sympathetic hypoactivity occurs, and because the parasympathetic system carried by the vagus nerve is intact, there is parasympathetic activity that cannot be fulfilled. The blood pressure reflex control is disrupted because of sympathetic hypoactivity and hypotension develops. Other factors in the etiology are changes in baroreceptor sensitivity, loss of pumping functions in skeletal muscles, decondition in the cardiovascular system, hyponatremia and low plasma volume, a fall in plasma catecholamine level, over-response in the peripheral alpha adrenergic receptors, morphological changes in sympathetic neurons, and renin-angiotensin-aldosterone activity [21-24]. While orthostatic hypotension may be clinically asymptomatic, symptoms such as dizziness, feeling faint, fatigue, clouded vision, and syncope may be seen. It is seen more often in tetraplegic patients than in paraplegics, with the rate in tetraplegics reported as high as 82%. It is not a finding generally seen in the acute stage and it may continue for years [23,25]. Pharmacological and non-pharmacological approaches can be attempted in treatment. The patient and their family must be informed about the subject, there must be sufficient water and salt intake, and the head should be kept raised during sleep. Elastic stockings, an abdominal cushion, the application of abdominal TENS, and passive exercises applied to the patient have been seen to be beneficial [23,26]. When these are not useful, midodrine, fludrocortisone, and ephedrine can be tried in the treatment [25].

Thermoregulation Disorder: In this disorder, efferents from the thermoregulation centre in the hypothalamus leave the spinal cord from the C7 level. The thermoregulation function is impaired following spinal cord injuries. The frequency increases in lesions at T6 and above. It becomes difficult to provide compat-

ibility with changes in the environmental temperature and the body temperature is seen to increase in hot environments and decrease in the cold. This is known as poikilothermia and, to preserve the body temperature, the environmental temperature must be of an appropriate heat. These patients may sometimes have a fever of 40°C without the presence of any infectious agent. This is known as 'quad fever' and is seen in the first weeks and months following injury. In the chronic period, hypothermia is a frequently reported finding. Shivering against the late during the quad fever period cold occurs late. It must be considered that the hypo- or hyperthermia attacks that can develop in these patients could result from the injury. In the treatment, the room temperatures and the patients' clothing must be adjusted accordingly [27,28].

Hyperhidrosis: Excessive sweating is a frequently seen finding after injuries of the spinal cord. In the pathology, the sweat glands have both adrenergic and cholinergic innervation and are under the control of the hypothalamus. The inhibitor impulses to the brain are interrupted and the sweat glands are continuously stimulated. There is generally anhidrosis below the level of the lesion. Just as in autonomic dysfunction, hyperhidrosis starts as a result of sympathetic hyperactivity in response to a disturbing stimulus originating below the level of the lesion. The trigger usually originates from the bladder or bowel. If excessive sweating disturbs the patient, impairs functions, or if there is a risk of pressure sores developing, treatment must be applied. In the treatment, the triggering agent must be removed. Anticholinergic drugs (transdermal scopolamine) and sympathetic blockage can be applied. Topical agents such as talcum powder, aluminium salts, or botox injections are other treatment methods [24-29].

Conclusions

Spinal cord injury and its complications cause important physical, psychosocial and economic problems. Treatment and rehabilitation of a spinal cord injured patient with a multidisciplinary team approach immediately after the injury is essential not only for prevention, early diagnosis and treatment of complications, but also for the patient reaching maximum functional capacity and independence.

Competing interests

The authors declare that they have no competing interests.

References

- Nowrouzi B, Assan-Lebbe A, Sharma B, Casole J, Nowrouzi- Kia B. Spinal Cord Injury Facts and Figures at a Glance, National Spinal Cord Injury Statistical Center, Birmingham, Alabama. *J Spinal Cord Med* 2015; 39(2): 243- 4.
- Cosar SN, Yemisci OU, Oztop P, Cetin N, Sarfakioglu B, Yalbuздag SA et al. Demographic characteristics after traumatic and non-traumatic spinal cord injury: a retrospective comparison study. *Spinal Cord* 2010;48(12):862-6.
- Karacan I, Koyuncu H, Pekel O, Sumbulođlu G. Traumatic spinal cord injuries in Turkey: a nation-wide epidemiological study. *Spinal Cord* 2000;38(11):697-701.
- Ropper AE, Neal MT, Theodore N. Acute management of traumatic cervical spinal cord injury. *Prac Neurol* 2015;15:266-72.
- Miko I, Gould R, Wolf S, Affi S: Acute spinal cord injury. *Int Anesthesiol Clin* 2009;47:37-54.
- Royster RA, Barboi C, Peruzzi WT. Critical care in the acute servical spinal cord injury. *Top Spinal Cord Inj Rehabil* 2004;9 (3):11-32.
- Stevens R, Bhardwaj A, Kirsch JR, Mirski MA. Critical care and perioperative management in traumatic spinal cord injury. *J Neurosurg Anesthesiol* 2013;15:215-29.
- Vazquez RG, Sedes PR, Farina MM, Marques AM, Velasco MEF. Respiratory Management in the Patient with Spinal Cord Injury. *Biomed Res Int* 2013;168757.
- Berney S, Bragge P, Granger C, Opdam H, Denehy L. The acute respiratory management of servical spinal cord injury in the first 6 weeks after injury: a systematic review. *Spinal Cord* 2011;49:17-29.
- Furlan JC, Fehlings MG. Cardiovascular complications after acute spinal cord injury: pathophysiology, diagnosis, and management. *Neurosurg Focus* 2008;25(5):13.

- Bilello JF, Davis JW, Cunningham MA, Groom TF, Lemaster D, Sue LP. Servical spinal cord injury and the need for cardiovascular intervention. *Arch Surg* 2003;138:1127-9.
- Partida E, Mironets E, Hou S, Tom VJ. Cardiovascular dysfunction following spinal cord injury. *Neural Regeneration Research* 2016;11(2):189.
- Oh YM, Eun JP. Cardiovascular dysfunction due to sympathetic hypoactivity after complete cervical spinal cord injury: a case report and literature review. *Medicine* 2015;94(12).
- Schulz-Stübner S. The use of small dose teophylline for treatment of bradycardia in patients with spinal cord injury. *Anesth Analg* 2005;101:1809-11.
- Grigorean VT, Sandu AM, Popescu M, Iacobini MA, Stoian R, Neascu C et al. Cardiac dysfunctions following spinal cord injury. *J Med Life* 2009;2:133-45.
- Campagnolo DI, Merli GC. Autonomic and cardiovascular complication of spinal cord injury in: *Spinal cord medicine* ed: Kirshblum S, Campagnolo DI, Delisa JA, Lippincott Williams, Philadelphia, 2002.p.123-34.
- Zheng MM, Phillips AA, Elliott SL, Krassioukov AV. Prazosin : a potential new management tool for iatrogenic autonomic dysreflexia in individuals with spinal cord injury? *Neural Regen Res* 2015;10(4):557-8.
- Blackmer J. Rehabilitation medicine,1:autonomic dysreflexia. *CMAJ* 2003;169:931-5.
- Esmail Z, Shalansky KF, Sunderji R, Anton H, Chambers K, Fish W. Evaluation of captopril for the managment of hypertension in otonomic disreflexia: Apilot study. *Arch Phys Med Rehabil* 2002;83:604-8.
- Hagen EM. Acute complications of spinal cord injuries. *World J Orthop* 2015;6(1):17-23.
- World Health Organization, The International Spinal Cord Society. *International Perspectives on Spinal Cord Injury*. World Health Organization; 2013.
- Popa C, Popa F, Grigorean VT, Onose G, Sandu AM, Popescu M et al. Vascular dysfunctions following spinal cord injury. *J Med Life* 2010;3:275-85.
- Claydon VE, Steeves JD, Krassioukov A. Orthostatic hypotension following spinal cord injury: understanding clinical pathophysiology. *Spinal cord* 2006; 44:341-51.
- CarvalhoAEM, DiasLP, LimaFP,PaulaJAR, LimaMO. Cardiovascular autonomic control in paraplegic and quadriplegic. *Clin Auton Res* 2016;26(2):117-26.
- Krassioukov A, Eng JJ, Warburton DE, Teasell R. A systematic review of the management of orthostatic hypotension after spinal cord injury. *Arch Phys Med Rehabil* 2009;90:876-85.
- Solinsky R, Svircev JN, James JJ, Burns SP, Bunnell AE. A retrospective review of safety using a nursing driven protocol for autonomic dysreflexia in patients with spinal cord injuries. *J Spinal Cord Med* 2016;14:1-7.
- Bazley FA, Pashai N, Kerr CL, Ali AH. The effects of local and general hypothermia on temperature profiles of the central nervous system followingspinal cord injury in rats. *Ther Hypothermia Temp Manag* 2014;4(3):115-24.
- Song YG, Won YH, Park SH, Ko MH, Seo JH. Changes in Body Temperature in Incomplete Spinal Cord Injury by Digital Infrared Thermographic Imaging. *Ann Rehabil Med* 2015;39(5):696-704.
- Gorman PH. Unilateral hyperhidrosis from a contralateral source in an individual with C4 complete tetraplegia. *J Spinal Cord Med* 2010;33(4): 428-30.

How to cite this article:

Karabacak P, Gülsoy KY, Osmanlioglu HÖ, Yavuz L. Complications of Spinal Cord Injuries: Two Case Reports and a Review of the Literature. *J Clin Anal Med* 2017;8(2): 168-71.