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Case Report

Spinal cord injury without radiological abnormality (SCIWORA) in a young female and pharmacological treatment option: a case report with review of literature

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ABSTRACT

Acute spinal cord injury strikes labour active young and middle-aged population, especially men, and consequently usually results in difficult neurological sequel. Also disables normal quality of life and everyday functioning in these patients despite many available supportive measures. Spinal cord injury without radiological abnormality (SCIWORA) presents a great diagnostic challenge because radiological and computed tomography pictures are without visible pathology which would explain the new onset of the neurological deficit. For the first time we report a true spinal cord injury without radiological abnormality in the X-ray, computed tomography and magnetic resonance imaging in a young female manifested with moderate neurological deficit after the traffic accident. Although SCIWORA is very rare in adults, high level of suspicion in emergency department is advisable as the timely applied neuroprotective measures can prevent the onset of the neurological deficit. It is important to emphasize that emergency magnetic resonance imaging application is the diagnostic key. Albeit, the SCIWORA neurologic deficit can show up with a delay of four days after the spinal cord injury in a patient with a normal physical and radiological examination. Treatment of these patients is another challenge for every physician. In fact pharmacological treatment options are still in the experimental research phase. We can conclude that currently the neuroprotective measures of the acute spinal cord injury patient started in the emergency department regardless the radiological test findings represent the right and successful key treatment.

Keywords: Corticosteroids, SCIWORA, Spinal cord injury, MRI

Abbreviations: Spinal cord injury without radiological abnormality (SCIWORA); Multislice computed tomography (MSCT); Magnetic resonance imaging (MRI)

INTRODUCTION

Spinal cord injury without radiological abnormality (SCIWORA) is a clinical and radiological entity for acute traumatic myelopathy with or without visible neurological lesions on the magnetic resonance imaging (MRI) of the spinal cord.[1,2] Besides, the X-ray and multislice computed tomography (MSCT) results are without pathological substrate. As it is well-known the MRI is superior to MSCT for the spinal cord injury detection. In the SCIWORA patients MRI has not only diagnostic but also prognostic role.[3,4] Hence the SCIWORA entity occurs very often in the pediatric population till the age of 8. The reason is that during the growth morphological and biomechanical changes of the spine and its associated ligaments occur. This leads to

ossification of the spine and the result is loss of the spine elasticity and flexibility which prevents the SCIWORA phenomenon in the adult population.[5-8] In the adults the degenerative and spondylotic spine changes should not be considered as abnormal radiological finding so the correct term is Spinal Cord Injury Without Radiological Evidence of Trauma (SCIWORET).[9-11] In addition spinal cord injuries are frequent in the male adult population.[12] Hence this case report is unique because it demonstrates the real clinical picture of the true SCIWORA in a young female which had inherent neurological deficits with the normal finding of the MRI of the cervical and thoracic spinal cord after the injury.

CASE REPORT

A 30-years old Caucasian female injuried in the traffic accident as a car passenger during the truck attack on her passenger side, arrived in the emergency department. Upon her arrival she was appropriate immobilised, conscious, spontaneous respiratory sufficient and hemodynamic stable. Her initial vital signs were arterial blood pressure 127/82 mmHg, heart rate 83/min, respratory rate 14/min, pulse oximetry oxygen saturation 99% and Glasgow coma score 14. During primary survey she was conscious, in contact, but slightly disorientated in the time and space. She had amnesia for the short period before and after the accident. Her medical history did not include any serious illness and her drug therapy included antidepressive drug fluvoxamine, diazepam and antacid occasionally. During the survey she complained on the headache, and the pain in her neck and back. The physical examination inspection did not reveal any visible injuries. Right-sided weakness with palpable arterial pulsation was noticed. Immediately neurologist was called. The initial neurological examination revealed the right-sided hemiplegia with depressed miotatic reflexes and hemihypoesthesia for the all quality sensation with paresthesia in the right leg. Babinski reflex was negative. The patient was continent, but urinary catheter was inserted due to quantative and quality diuresis monitoring. According to consiliar neurologic opinion due to acute spinal cord injury suspicion symptomatic therapy with a single dose of methylprednisolone 1.5 gr intravenously was applied together with ranitidine as gastroprophylaxis. As well antiedematose therapy with 10% mannitol was applied during 3 days. Immediately emergent MSCT polytrauma protocol which encompasses MSCT of the head, neck, thorax, abdomen and pelvis was done. No neurological substrate was found which could explain the neurological deficit. Color Doppler of the carotid and vertebral arteries was done to exclude the artrey dissection. Since the test was limited by cervical spine pain and immmobilisation MSCT angiography of the head and neck was done. The result was normal. According to present neurological deficit and normal MSCT head and neck finding the SCIWORA was suspected. Next day, the MRI of the brain, cervical and thoracic spinal cord was done. The finding was also normal, without pathological substrate that could explain the existing neurological deficit (Fig. 1). On the third day control neurological examination found progressive recovery of the motoric function of the right arm and leg. Hemihypoesthesia was still present. During the hospitalisation further recovery of the right-sided motoric function and sensation was recorded. After seven days the patient was discharged from the hospital with the appropriate immobilised cervical spine with hard collar, in good shape, mobile, with good sphincter control and residual neurological deficit of the reduced right foot dorsal flexion, paresthesia of the right upper arm with weakness of the whole right arm. Two weeks after hospital discharge control X-ray functional recordings of the cervical spine were made. The recordings were normal. Consequently, the cervical immobilisation was removed. Regard the residual neurological deficit the somatosensory evoked potentials of the right median and tibial nerve were done. The result was also normal.

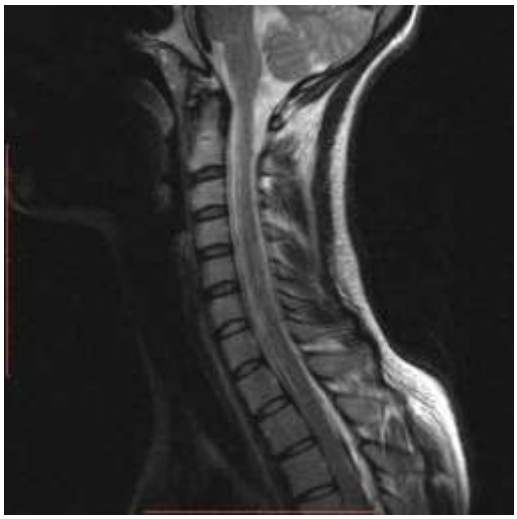

Figure 1: Magnetic resonance imaging of the cervical spinal cord without pathological substrate.

DISCUSSION

Spinal cord injury is a result of the mechanical trauma which causes local spinal cord deformity by compression, contusion or longitudinal distraction owing hyperextension, hyperflextion or rotation movement.[6,13] In our case the neurological deficit was the consequence of the cervical spinal cord contusion due hyperextension injury.[2] Radiological diagnostic could not find the pathological substrate. The neurological deficit after spinal cord injury is the result of the mechanical force effect and the initial spinal cord injury damage magnitude, so called primary spinal cord injury.[14] In every patient with the primary spinal cord injury the most important is to prevent and decrease the secondary spinal cord injury damage. This secondary injury results from the increased metabolism and oxygen requirements after the primary spinal cord injury.[15] If the oxygenation of the damaged neural tissue is inadequate, tissue ischemia occurs.[16] This ischemia leads to activation of the complex cascade of the biochemical and cell process which loss of

homeostasis is integral part of the secondary neural injury. This cascade leads to emergence of the oxygen free radicals, lipid peroxidation activation with the subsequent release of the posttraumatic inflammatory mediators, lowering the high energy phosphate reserves, and loss of the neuronal and microvascular membrane integrity.[14] The final result is cell death.[14] So the main goal in the spinal cord injury is to decrease and prevent the secondary spinal injury. Therefore, the cornerstone of the acute spinal cord injury management consists of applying the initial neuroprotective measures which make the proper, without delay, spinal cord immobilisation, airway and breathing adequate management and protection to enable sufficient oxygenation of the damaged area, and maintainig the arterial blood pressure.[17] According to the literature, SCIWORA management consists of the pharmacological treatment together with the external cervical spine immobilisation with the hard collar during 12 weeks.[18,1] Operative treatment is indicated if there is spinal cord compression or instability owing to secondary extraneural injury.[8]

Pharmacological treatment option

Thus, timely applied pharmacological treatment has neuroprotective mechanism by preventing the emergence and activation of the secondary inflammatory response.[12,14] This encourages functional recovery of the injured neural tissue. The main effect of the pharmacological treatment is to diminish the secondary inflammatory response by various mechanisms. Although, the methylprednisolone therapy was the main pharmacological therapy for the acute spinal cord injury, new guideliness according to the Official journal of the Congress of the neurological surgeons do not recommend its use in the acute spinal cord injury with the evidence level 1 or 2.[19] Methylprednisolone application is still considered experimental and the effect on the neurological deficit recovery after continuous infusion during 24 hours is negligible.[17] As well, enough evidence for the benefit of the 48 hours continuous methyprednisolone infusion was not found.[17] However, the research showed that patients who were on the high dose methyprednisolone therapy had greater incidence of the respiratory complications such as pneumonia, especially if they were initially without consciousness and on mechanical ventilation.[19] These patients also had 1.5 times higher incidence of the gastrointestinal hemorrhage despite adequate gastroprotection, 2 times higher incidence of wound infection, 3 times higher incidence of pulmonary embolism, and 2.5 times higher incidence of the thrombophlebitis.[19] Other complications were hyperglycemia, corticosteroid myopathy and neuropathy, sepsis, and there were rare cases of femoral head avascular necrosis.[19] They also had longer hospital stay.[19]

Another experimental neuroprotective drug is tirilazad mesylate, nonglucocorticoid 21-aminosteroid.[21] It has neuroprotective mechanism proved in the preclinical research. Cytoprotective neuronal effect achieves through antioxidant mechanism by increasing the neuronal membrane stability, diminishing genesis and removing oxygen free radicals, inhibiting lipid peroxidation and maintaining the level of the endogenous vitamin E.[22] The main place of these actions is neurovascular barrier with the preserving its permeability during and after injury, and in the end neuronal tissue edema.[22] But tirilazad is still considered experimental drug and further research is needed.[22]

GM1 gangliosides had a role in the syptomatic neuronal treatment. They are acid glycolipids which are the main component of the neuronal membrane.[21] The highest concentration of the GM-1 gangliosides is located in the synaptic junction.[21] The experimental animal models demonstrated their ability to stimulate neuronal cells growth with the consequence of the neuronal tissues regeneration. So far, medical evidence do not support the clinical benefit of their use in the treatment of the patients with the acute spinal cord injury.[19,23]

Implementing the opioid antagonist naloxon in the pharmacological treatment of the acute spinal cord injury presents a new perspective in this area. Moreover the experimental research has revealed that spinal cord injury leads to increase in the beta-endorphin secretion form the hypophyse.[24] The result is fall of the arterial blood pressure with the decreased spinal cord perfusion.[24] The naloxon acts by antagonizing beta-endorphin effects which improves arterial blood pressure and the spinal cord perfusion at the same time. There is a better physiological and neurological recovery.[24] Nevertheless, further research is still required although naloxon application in the acute spinal cord injury is very promising.

To mention, due to lack of evidence of neuroprotection, new guidelines do not recommend hypothermia application in the spinal cord injury.[25]

In our patient due right-sided hemiplegia with hemihypoesthesia, in accordance with consiliar neurologist and neurosurgeon the single bolus dose of methylprednisolone within 8 hours from injury was applied. Although it is not in the guidelines the antiedematose therapy with 20% mannitol for 3 days was also implemented. However, the patient's after treatment course had no complications.

SCIWORA incidence in the adult population makes 12% of the acute spinal cord injury cases.[26] Manifested neurologic deficit clinical picture is variant from mild, transient neurologic deficit caused by spinal cord commotion to persistent neurologic deficit caused by parenchymatous neural hemorrhage and neural tissue disruption.[1,27] Our case is unique because SCIWORA is rare in the adult population between 20 and 40 years of age, especially in female population.[28,29] The mechanism of the SCIWORA is segmental spine instability with transient movement owing spine and spinal cord extension in different avenues.[30] These avenues can be

hyperflexion, hyperextension or longitudinal distraction of the spinal cord. Rare, but also possible cause of SCIWORA is vascular damage of the spinal cord which usually brings to delayed neurological deficit initiation after the injury, especially in diabetics and smokers.[31] The consequence of the abnormal intersegmental movement and vascular damage is spinal cord ischemia on the microvascular level and edema emergence.[15] SCIWORA neurological deficit symptoms vary from the transient to the persistent. They manifest immediately after the injury or have delayed onset with a time period of latency from 30 minutes till 4 days.[32] Only 2% of adults with SCIWORA have delayed neurological deficit onset.[33] It is important to emphasize that all spinal cord levels are prone to SCIWORA injury. However, the cervical part is generally striked, in particular the maximum flexion point in the cervical 5 and 6 level, as it was in our patient.[6] Next is thoracic, and lumbosacral area. In our patient hyperextension injury of the cervical spine was the mechanism of the SCIWORA genesis.

Yet, parenchymatous damage of the spinal cord according to the MRI and initial neurologic deficit highly correlates with the patient prognosis. The first group with complete spinal cord injury and great hemorrhage have a detrimental outcome and rarely neurological deficit improvement.[34] The second group with MRI detectable minor spinal cord hemorrhage and edema have a neurological deficit recovery with time, but they rarely return to the normal neurological function. The third group with the neurological deficit due spinal cord contusion and normal MRI finding has the best prognosis and usually complete neurological deficit recovery.[34] Although, our patient belongs to the third group with the best prognosis of neurological deficit recovery, this was not her outcome. In the beginning she had quick and good neurological deficit recovery, but in the end it was incomplete. She had a residual neurological deficit of the decreased dorsal flexion of the right foot, paresthesia of the right upper arm and the weakness of the whole right arm. New guideliness also recommend in the patients with the major cervical spine injury flow screening of the vertebral arteries, as it was done in our case as well.[25,35]

CONCLUSION

To conclude, SCIWORA is associated with the worse neurological outcome, delayed neurological deficit onset, and high portion of the complete neurological injury. Owing to serious consequences after SCIWORA, every trauma patient requires high level of the suspicion and vigilance no matter neurological deficit is immediately present or not. According to guidelines the polytrauma patients, those with craniofacial injuries and comatose patients have a greater chance of misdiagnose the cervical spinal cord injury, so MRI of the cervical spinal cord should be done in the early phase without delaying and waiting for the neurological assessment.[36] Therefore, we can conclude that at this moment pharmacological treatment are lacking, hence SCIWORA treatment is based on the timley applied neuroprotective measures which prevent and diminish recurrent spinal cord injury and neurological deterioration yet in the emergency department.[18]

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