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Spinal Cord Ischemia - From Diagnosis to Treatment

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Abstract

Purpose: The aim of the article is to outline the frequency of occurrence, course, diagnosis, possible treatment of spinal cord ischemia (SCI) on the basis of literature overview and to raise awareness of this rare yet devastating condition.

Views: SCI compared to cerebral stroke is a relatively rare disease. It is diagnosed 100 times less often than a cerebral stroke. The root causes of its origin, proper treatment and long-term prognosis are still inconclusive. Magnetic resonance imaging (MRI) with DWI is a main tool to confirm the SCI and rule out broad spectrum of possible alternative diagnoses. SCI is a medical condition, in which prompt recognition, accurate diagnostic steps, and reperfusion therapy are essential to ensure desirable functional outcome and reduce mortality and disability. Although there are no specific guidelines regarding treatment, administration of recombinant tissue plasminogen activator (rt-PA) might be an effective therapy for acute ischemic stroke preventing permanent spinal dysfunction. In surgical causes, close cooperation between neurologist and neurosurgeon is necessary to provide combined appropriate management promptly.

Conclusions: Due to the relative rarity of SCI, the multi-center studies of ischemia of a spinal cord and its treatment would be advisable in neurological practice to enhance current knowledge. Fast diagnosis is crucial for appropriate care and desirable long-term outcomes.

Keywords: ischemic myelopathy; spinal cord ischemia; spinal cord infraction

Introduction

Spinal cord ischemia or spinal cord infraction (SCI) compared to cerebral stroke is a relatively rare disease. The root causes of its origin, proper treatment and long-term prognosis are still inconclusive. SCI comprises 5,7% cases of acute myelopathy and 1-2% of all neurovascular events although the exact incidence and prevalence remain unclear. Recent studies have shown that myelopathy related to ischemic diseases accounts for 14-18% of patients with transverse myelitis, suggesting the underdiagnosis of SCI [1]. It is diagnosed 100 times less often than a cerebral stroke. SCI typically occurs

between 50 and 70 years old on average [2]. SCI mainly presents as anterior spinal artery syndrome or anterior spinal cord syndrome (ASCS) in up to 87.2% of the cases [3-5]. MRI with DWI (Diffusion-weighted contrast) should be taken into account for initial diagnosis of spinal cord ischemia. A combination of DWI with ADC maps is recommended to distinguish SCI from other differential disorders. There are no established rules for the treatment of this condition.

Rapid intravenous administration of rt-PA might be an effective treatment for acute ischemic stroke preventing permanent spinal dysfunction [6].

Spinal cord vasculature- neuroanatomy

The principal blood supply to the spinal cord is via a single anterior spinal artery (ASA) and two posterior spinal arteries (PSA) (fig.1). The ASA provides blood to the anterior two-thirds of the spinal cord, and the PSA delivers blood to the posterior one-third of the spinal cord and posterior horn [7]. The second source of blood supply constitute radicular arteries, which originate from segmental arteries and these in turn from ascending aorta. Segmental arteries give rise to radicular branches which penetrate spinal cord bilaterally via intervertebral foramen. Each of the radicular arteries supply functionally separate parts of spinal arteries (fig. 1). The first segment of spinal cord (C1-Th3) is supplied mainly with branches that originate from vertebral artery and carotid artery. The thoracic region (Th3-Th7) is supplied with branches of posterior intercostal arteries and superior intercostal artery. In the lumbosacral part (below Th8) the blood comes from artery of Adamkiewicz- the largest anterior radiculomedullary artery. The artery of Adamkiewicz (or arteria radiularis magna) typically arises from the left side of the aorta between T8 and L2 in 75% of people, however, it can also be present above T8 [7-9].

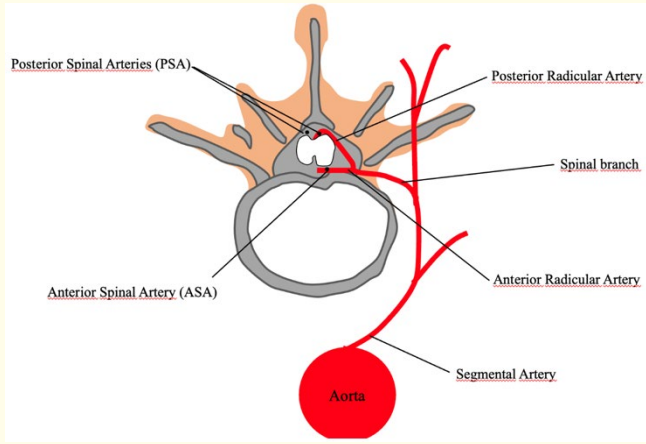

Figure 1: Illustration of spinal cord vasculature.

The mechanisms of spinal cord ischemia- pathophysiology

There exist two underlying major pathophysiological mechanisms for spinal cord infarction. Radicular artery territory infarcts are triggered by occlusion of the anterior or posterior artery, whereas central and transverse infarcts stem from systemic hypoperfusion. As for the main causes of SCI, aortic interventions and pathologies can be distinguished accounting for around two-thirds of the cases [3]. Few reported cases of spinal cord ischemia are due to the existence of collateral circulation which is created by vascular network. Ischemia is responsible for an inflammatory response and NMDA-mediated neuronal excitotoxicity [9, 10]. In various ischemic incidents, the presence of heat shock proteins might be witnessed [11, 12]. Spinal cord blood flow differs in particular areas of the cord [13]. Numerous research has revealed that due to the density of motoneurons, thoracolumbar segment is most vulnerable to hypoper-

fusion. Cervical cord remains the second most affected region in up to 25% of patients [5].

Etiology

SCI is most closely associated with aortic diseases most likely because of the vulnerability of the thoraco-lumbar spinal cord to hypoperfusion. A wide spectrum of other conditions may lead to spinal cord infraction: atherosclerosis, degenerative disease, systemic hypotension, cardiac embolism, coagulopathies, vasculitis, connective tissue disorders, thrombophilia [4, 14-16]. In 20-30 % the etiology remains unknown [3]. Ischemic myelopathy might result from mechanical compression exerted by an osteophyte on the anterior spinal artery, especially while abrupt moves, physical exertion, or injury [17, 18]. Important risk factors for spinal cord ischemia are: aortic surgeries, renal artery embolization, aortic counterpulsation [19].

Aortic disorders as a primary cause of SCI

The most frequent and primary cause of SCI is aortic atherosclerosis followed by other aortic conditions, such as aortic dissection and complications of surgery performed on aorta [3, 18] Atherosclerotic plaques are located in the area of region of branch to the spinal cord. This is turn may lead to impairment of blood flow or constitute an embolic material [19].

Aortic dissection (AD) when extends into the descending aorta resulting in insufficient perfusion of segmental arteries that supply the spinal cord [2]. Acute onset of paraplegia or paraparesis with a thoracic sensory level can be a dramatic presentation of dissection of the aorta. Dissection is usually preceded by rapid onset of severe pain in the chest or back.

Midthoracic or lower are thoracic cord are most affected because they are supplied by the intercostal arteries that frequently suffer owing to aortic dissection [12]. A larger deficit may occur if the dissection involves the artery of Adamkiewicz, which supplies the levels of T10 to L2 in most patients [20]. Aortic aneurysm and aortic dissection are life-threatening conditions and should be investigated carefully in the presence of spinal cord infarction.

Clinical presentation - Spinal cord injury syndromes depending on the area of ischemia

Given the vascular territory involvement, the clinical presentation of spinal cord infarction is with various degrees of dysfunctions [3].

Anterior spinal artery syndrome (ASAS)

The ASAS is the most frequent clinical presentation of SCIa [21, 22]. Symptoms typically include motor paralysis, the loss of pain and temperature sensation below the level of the lesion. The proprioception, vibratory sense, and fine touch are preserved. Other symptoms include back pain, or autonomic dysfunction such as hypotension, neurogenic bowel or bladder, and sexual dysfunction. Initially, due to the spinal shock, paralysis is flaccid. There have also been reported problems with controlling vesical and rectal sphincters [20, 23].

Posterior spinal artery syndrome PSAS

Posterior spinal artery syndrome (PSAS) occurrence is rare and then be a diagnostic challenge. It is characterized by ipsilateral loss of proprioception, fine touch, pressure, and vibration below the lesion. The tendon and cutaneous reflexes are abolished. The pain and sensory sensation are preserved excluding the part of cord segment which has suffered. In severe cases, large spinal cord lesions can also affect surrounding spinal tracts (lateral part of corticospinal tract) resulting in bilateral movement deficit [19, 24].

Central syndrome of spinal cord injury

Central cord syndrome in the vast majority of cases affect the cervical spinal cord, and it mainly occur after a fall with hyperextension of patient's neck [25]. Symptoms typically include paralysis or loss of fine control of movements in the arms and hands; however, movement of legs with varying degrees of loss of pain, temperature, light touch, and pressure sensation below the level of injury and

possibly urinary dysfunction remain intact.

Brown-Séquard syndrome

Hemodynamic disturbances and spondylosis might lead to the neurological condition called Brown-Séquard syndrome. The patient presents with weakness or paralysis and proprioceptive deficits on the side of the body ipsilateral to the lesion accompanied by loss of pain and temperature sensation on the opposite side [26, 27].

Diagnosis of spinal cord ischemia

The fast diagnosis is essential to prevent mortality and disability. Neurological examination is conducted initially, followed by neuroimaging [15]. MRI is a preferential method although in up to 24% of patients the image might be entirely normal [10, 28]. Therefore, in consecutive days MRI should be repeated to rule out spinal cord compression. MRI imaging usually include sagittal and axial T1 and T2-weighted sequences and diffusion-weighted imaging (DWI) [29]. DWI MRI is considered as a highly sensitive method for detecting SCI within 8 hours since the onset [12, 30]. A combination of DWI with ADC maps is recommended to distinguish SCI from myelitis and demyelinating disorders [6]. In the acute stage, ischemia presents as a restriction in diffusion-weighted imaging of the spinal cord, hyperintense signal on T2 and STIR, and isointense on T1 [31]. DWI-negative might be present in the hyperacute setting (less than 24 hours) and should not exclude the diagnosis especially when other symptoms are present. "Owl eyes", "pencil-like", "positive anterior cauda" hyperintensity may support, but are not pathognomonic for the diagnosis of SCI [28, 29]. Differential diagnosis includes broad spectrum of myelopathies such as compressive, infectious, or inflammatory. Hyperintensities on T2-weighted images are not typical of ischemia and can also be revealed in various conditions. In terms of multiple sclerosis, the lesions may occur in any part of the spinal cord but within the cervical enlargement plaques are most commonly found in the lateral columns. Additionally, in multiple sclerosis, hyperintensities might be revealed in T2-weighted MR images in brain as well. In vascular congestion caused by spinal malformations which is difficult to distinguish from SCI as well, frequently there is "flow-void phenomenon" on T2-weighted images [32]. Other autoimmune disorders which may cause cord signal hyperintensities comprise systemic lupus erythematosus or Sjogren's syndrome [14].

Treatment of spinal cord ischemia and outcome

Spinal cord ischemia is a medical condition, in which accurate treatment in necessary to achieve favorable functional outcome. Unlike cerebral stroke, in which guidelines for management are well-established, the management of acute SCI is still under discussion. If the cause is ischemic etiology, risk factors assessment such as diabetes mellitus, hypertension, hyperlipidemia should be carried out and accurate treatment should be implemented [19]. The treatment concepts have its origins in managing acute ischemic stroke and includes airway and ventilation management, fever and glycemic control, anticoagulation, antiplatelet, and thromboprophylaxis therapy [33]. The administration of intravenous rt-PA within an adequate time window (to 4,5 hours from the onset of the symptoms) could be useful before scheduled neurosurgical procedure. Only a few cases of using thrombolysis have been described in literature [17, 34-37]. It would be advisable to set up a registry of patients with SCI that have been treated with rt-PA in order to evaluate the effectiveness of the treatment and establish management standards. If there is a mechanical compression, the management requires immediate surgery. Thus, close cooperation between neurologist and neurosurgeon is essential. The effective preventive measure during aorta surgeries is cerebrospinal fluid (CSF) drainage which improve spinal cord perfusion pressure. Hnath et al. used preoperative lumbar CSF drainage for endovascular aortic repair showed no SCI in the 56 cases, compared with 5 episodes of SCI in 65 patients operated without drainage [38]. Importantly, appropriate rehabilitation must be followed so as to prevent complications which may occur due to immobilization. These actions should ensure returning to mobility and independence in daily life activities [10].

Conclusions

Despite being an uncommon disease, SCI might be a severe, life-threatening condition. Aortic disease is the most common cause of SCI. Knowledge of spinal cord vasculature is required to fully understand pathophysiology and symptomatology. In suspected SCI, fast diagnosis, therapy, rehabilitation is crucial for long-term outcomes. Currently, recommendations derive from few described cases

and guidelines regarding management of cerebral stroke. The use of rt-PA could have an impact on the beneficial effect of treatment and functional outcome. However further evaluation and multi-center studies of ischemia of a spinal cord and its treatment would be advisable in neurological practice. In surgical causes, close cooperation between neurologist and neurosurgeon is necessary to provide combined appropriate management promptly.

Conflict of interest

Absent.

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