Special aspects:

- Early and Late onset of Autonomic Dysreflexia
- The “silent” Autonomic Dysreflexia
- Autonomic Dysreflexia impact on cardiovascular system

AE Kyriakides

Autonomic nervous system regulates circulation by affecting the force and frequency of cardiac contraction and/or by affecting vasodilation or vasoconstriction in response to feedback from sympathetic and parasympathetic afferents. Spinal cord injury (SCI) above the T6 level results to ‘decentralization’ of the sympathetic nervous system and patient remains with no supraspinal sympathetic control of cardiovascular functions.

Early autonomic dysreflexia

Low resting blood pressure, orthostatic hypotension, and loss of diurnal fluctuation are blood pressure abnormalities commonly presented during the acute post-injury phase. In addition episodes of autonomic dysreflexia (AD) have also been reported during this period suggesting that the sympathetic reflex activity of the cord may not be completely abolished during spinal shock.

AD during the period of “spinal shock” was considered impossible. Silver has first reported at early seventies and reviewed thirty years later (2000) a group of patients who developed AD within 7–31 days post injury.

Krassioukov A, later has also reported three cervical complete tetraplegics who developed AD in acute phase. The earliest episode occurred on the 4th post injury day and no patient had a history of cardiovascular disease. Although pathophysiology of early AD is not clear, the differential diagnosis of AD during acute phase of SCI should be kept in mind as the recommended acute management is directed toward maintenance of the mean arterial blood pressure above a critical threshold in order to maintain spinal cord perfusion.

Pathophysiology. ‘Negative side of neuroplasticity’

It is important to focus on pathophysiology in order to better understand

the mechanisms of AD either in acute or chronic stages of SCI.

Disruption of the descending cardiovascular pathways results in sympathetic hypoactivity, alteration in morphology of sympathetic preganglionic neurons, plastic changes within the spinal circuits, and development of peripheral alpha-adrenoreceptor hyperresponsiveness.

Studies reveal that immediately after SCI, plasma catecholamines are low and remain low as long as there is no stimulation below the level of SCI. These findings support the concept of sympathetic hypoactivity below the level of high-SCI when not being subjected to afferent stimuli. These low plasma catecholamine levels are supposed to be responsible for the peripheral vascular alpha-adrenoceptors hyperresponsiveness possibly through an upregulatory or denervation supersensitivity mechanism. However, whether this hyperresponsiveness is a consequence of an increased number of receptors, abnormalities of postreceptor coupling mechanisms, or reduced presynaptic reuptake was not yet determined. The observation that clearance of catecholamines does not appear to be affected in quadriplegics would exclude the last explanation.

Other studies have proposed that the enhanced pressor response to noradrenaline in SCI patients was a consequence of lack of baroreceptor-mediated sympathoinhibition because patients with chronic autonomic failure secondary to other causes have not demonstrated such an exaggerated response. If loss of baroreceptor control was the principle mechanism we could probably explain why AD has reported to present even in acute phase.

Little is known about the alteration in morphology of sympathetic preganglionic neurons. Research in animals has demonstrated reversible atrophy of sympathetic preganglionic neurons probably as a consequence of partial deafferentation from the loss of descending projections from medullary neurons. Parallel animal studies have revealed sprouting of dorsal root afferents and spinal neurons that initially lose synaptic inputs, to have them replaced with inappropriate synapses. This negative side of neuroplasticity has been related with the development and severity of AD in the chronic phase of SCI, as it needs time to occur.

Nevertheless, reorganization and plastic changes of spinal cord pathways combined with loss of baroreceptor control as well as peripheral alpha-adrenoceptor hyperresponsiveness are likely more related to episodes of AD developed either in acute or chronic stages of SCI

Asymptomatic ('silent') AD

Literature has described asymptomatic paroxysmal hypertension during bladder contractions and voiding or during a routine bowel program.

Asymptomatic ('silent') AD was also reported during a vibrostimulation for sperm retrieval. The majority of SCI patients didn't report symptoms characteristic of AD and remained unaware of their arrhythmias and ECG abnormalities.

In a up-to-date study blood pressure and symptoms of AD were continuously monitored during urodynamic studies (UD) and systolic blood pressure elevations >20 mmHg was considered an AD reaction. AD patients were divided into a symptomatic group and a silent group and several parameters were compared. Patients with more symptomatic AD tended to have significant BP elevation and more rapid BP increments, and this was negatively correlated with age. BP monitoring during UD and other invasive procedures is strongly recommended.

The impact of AD episodes on cardiovascular system.

Consequently individuals with cervical or high thoracic SCI face two major opposite effects on blood pressure (BP). On one hand, they experience severe hypotension during both the acute and chronic stages. On the other hand life-threatening episodes of AD represent another aspect of disordered cardiovascular control. These repetitive BP fluctuations increase the possibility of shear injury to the blood vessel endothelium that may predispose to serious cardiovascular complications.

If the episode of AD is difficult to control and there is persistent coronary artery constriction, asymptomatic myocardial ischemia can occur. Few cases of asymptomatic myocardial ischemia following AD have been reported in the literature. Exceptionally interesting case of silent myocardial ischemia with no previous cardiovascular history presented by Ho. This case had also absence of coronary artery disease in a follow up angiography, suggesting that his myocardial ischemia have been caused by significant cardiac vasculature constriction during the AD episode.

Although none of published cases of cardiovascular complications secondary to AD resulted in fatality the impact of AD should not be underestimated, as cardiovascular disease is still the most common cause of death in chronic SCI when considering underlying and contributing causes together. Documentation and early recognition of

AD should be included as part of the standard neurological assessment and management of individuals with SCI.

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