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Autonomic dysreflexia in the practice of a urologist

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Autonomic dysreflexia (AD) is a potentially life-threatening condition that develops in patients with spinal cord injury (SCI) at or above the T6 segment. First of all this condition is characterized by uncontrolled arterial hypertension, which can lead to catastrophic complications and even death. The trigger for the development of AD is often urological complications, as well as diagnostic and therapeutic manipulations on the lower urinary tract. It is important for urologists to be aware of the AD syndrome, clinical features of AD, acute and chronic management, as well as prevention episodes of AD in patients with neurogenic lower urinary dysfunction. AD is defined as an increase of systolic blood pressure of 20 mmHg from baseline in response to various afferent stimuli originating below the level of spinal cord injury. AD is based on exaltation of spinal reflex activity with irradiation of impulses in the spinal cord under conditions of dennervation preganglionic sympathetic neurons located above the T6 segment and hyperactivity of peripheral α-adrenergic receptors. The main pathophysiological mechanism of AD is hypernoradrenalinemia, leading to vasoconstriction of the vessels of the skin, abdominal cavity, muscles below the level of neurological injury.

Keywords: autonomic dysreflexia; neurogenic bladder; spinal cord injury; neuromuscular bladder dysfunction.

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Автономная дисрефлексия в практике уролога

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Автономная дисрефлексия (АвтД) — потенциально жизнеугрожающее состояние, которое развивается у пациентов с позвоночно-спинномозговой травмой на уровне или выше сегмента Т6. Прежде всего это состояние проявляется неконтролируемой артериальной гипертензией, которая может привести к тяжелым осложнениям и даже летальному исходу. Причиной развития АвтД чаще всего становятся урологические осложнения, а также диагностические и лечебные манипуляции на нижних мочевыводящих путях. Практикующим урологам важно быть осведомленными о синдроме АвтД, его клинических проявлениях, диагностической и лечебной тактике, а также профилактике предотвращения эпизодов ее манифестации у пациентов с нейрогенной дисфункцией нижних мочевыводящих путей. АвтД определяется как повышение систолического артериального давления выше базового значения на 20 мм рт. ст. в ответ на различные афферентные стимулы, возникающие ниже уровня повреждения спинного мозга. В основе АвтД лежит экзальтация спинальной рефлекторной активности с иррадиацией импульсов в спинном мозге в условиях деннервации преганглионарных симпатических нейронов, расположенных выше сегмента Т6 спинного мозга и гиперактивности периферических α-адренорецепторов. Основной патофизиологический механизм АвтД — это гипернорадреналинемия, приводящая к вазоконстрикции сосудов кожи, брюшной полости, мышц нижних конечностей, расположенных ниже неврологического уровня повреждения.

Ключевые слова: автономная дисрефлексия; вегетативная дисрефлексия; нейрогенный мочевой пузырь; травма спинного мозга; позвоночно-спинномозговая травма; нервно-мышечная дисфункция мочевого пузыря.

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ЭКО•ВЕКТОР

Among the long-term consequences of spinal cord (SC) injury (SCI), along with locomotor and sensory disorders, a combination of autonomic and cardiovascular complications causes a loss of adequate central control over these systems [1, 2]. These complications include autonomic dysreflexia (AD). It occurs with SCI at the level of the sixth thoracic segment (T6) of the SC or above. AD is characterized by an overreaction of the autonomic nervous system with a significant increase in blood pressure in response to irritants that occur below the level of damaged SC. These irritants are most often associated with significant bladder or rectal overflow. If sensory disorders in chronic SCI are present, AD may be the only symptom of a urinary system problem, from an overflowing bladder to pain associated with renal colic [3].

Definition

AD is an increased reaction of the sympathetic part of the autonomic nervous system in patients with SC lesions at the level of the T6 segment or above. It is manifested by episodes of hypernoradrenalinemia due to irritating impulses below the neurological level of damage. The guidelines of the European Association of Urology defined AD as a sudden increase in systolic blood pressure > 20 mm Hg from the initial level [4, 5].

Prevalence

AD develops in 48%–90% of patients with neurogenic urination disorders who sustained SCI at the T6 segment and above [6]. AD most commonly occurs with cervical injury of the SC and occurs in >70% of patients with injuries within the C1–C8 segments. Moreover, the incidence of AD in case of damage to the SC at the level of T1–6 segments is approximately 30%.

Typically, AD symptoms develop 6–12 weeks after injury; in the early stages, compensation for hemodynamic disorders (period of spinal shock) is possible. Therefore, the prevalence of AD in chronic SCI increases. AD occurs in 38.7% of patients during the first 2 years after SCI; if the injury lasts >2 years, AD is recorded in 80% of the patients [7].

Pathogenesis

SC damage that occurs during SCI, above or at the level of the Th6 segment where the cardiac sympathetic center is located, deprives it of the preganglionic neurons of supraspinal centralized influence, which leads to denervation hyperreflexia, accompanied by hypernoradrenalinemia [1, 8]. Since in SCI both descending and ascending nerve pathways are interrupted, preganglionic neurons located below the injury level retain communication with intraspinal neurons and can be activated through spinal reflex pathways that are not damaged. Stimulation of visceral or somatic fibers below the neurological level of damage excites preganglionic cardiac neurons. The absence of an inhibitory response from the cerebral cortex and pontine urinary center under decentralized SC causes detrusor–sphincter dyssynergia and enhances spinal reflexes leading to AD.

The most common triggers for AD are overstretching of the bladder wall with its significant overflow, as well as overflow of the rectum, invasive interventions on the urinary tract (urodynamic examination, cystoscopy, bladder catheterization, etc.), and painful stimuli (such as injuries, bedsores, tight clothing). Sexual stimulation, ejaculation, urinary tract infection, bladder stones, rectal examination, and a session of remote shock wave lithotripsy can also cause AD [9]. Most authors believe that, in everyday life, the most common provoking factors of AD are bladder overflow (89% of the patients) and painful stimuli (75% of the patients) [10].

Clinical presentation

The clinical manifestations of AD can include sudden and persistent arterial hypertension (>20 mm Hg from baseline), headaches of varying intensities, dilated pupils, chills, piloerection accompanied by patchy skin hyperemia and profuse sweating above the level of the spinal injury, shortness of breath, increased spontaneous cramps of skeletal muscles, and changes in pulse rate [4]. In an investigation of the clinical manifestations of AD, Solinsky et al. [11] showed that during an episode of AD, 68% of patients have tachycardia with an average increase in heart rate of 20 beats per minute, while bradycardia is much less common and accounts for 0.3% of all registered episodes.

AD develops unexpectedly. If timely medical care is not provided, AD can have extremely serious consequences, such as subarachnoid hemorrhage, hypertensive encephalopathy, retinal detachment, cardiac arrhythmias, seizures, neurogenic pulmonary edema, renal failure (due to prolonged vasoconstriction of renal vessels), and lethal outcome.

Diagnostics

Despite the widespread prevalence of AD among patients with SCI, not all specialists who encounter this condition can recognize it promptly. The diagnostic criteria for AD are a sudden increase in systolic pressure by >20 mm Hg from the baseline and the clinical manifestations described above. Patients with a high level of SC damage, above the T6 segment, are characterized by arterial hypotension. Typically, their baseline systolic pressure is 15–20 mm Hg lower than that in the general population [9].

Treatment and prevention

In the case of AD, following a clear therapeutic algorithm is extremely important. The identification and

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elimination of exogenous/endogenous factors provoking AD appear to be key in the treatment and prevention of AS, and continuous monitoring of hemodynamic parameters (every 5 min) assesses the efficiency of the measures taken [13].

Since excessive bladder overflow is the most common cause of AD, the bladder should be emptied immediately, and invasive manipulation in the lower urinary tract should be discontinued. Rectal overflow is the second most common cause of visceral stimulation. To eliminate this factor, contents of the rectal ampulla should be evacuated. Irrigation of the anal area with lidocaine during diagnostic procedures contributed to a slight increase in blood pressure [16]. If the cause of AD is unknown, a thorough examination is required to rule out bedsores, burns, skin infections, abscesses of various localizations, and acute abdomen (appendicitis, acute intestinal obstruction, peritonitis, etc.).

The patient should be seated in a chair, or the head end of the bed should be raised to induce an orthostatic effect that naturally lowers blood pressure. If all these above manipulations are ineffective, pharmacological methods of correction are recommended [13, 14].

To date, no generally accepted pharmacological protocols have been established for the treatment of hypertensive crises during AD, and various approaches to resolving this condition are described. Most authors indicate that the use of 10 mg of nifedipine during an AD episode is more effective in achieving a rapid hypotensive effect [15, 17, 18]. The successful simultaneous use of angiotensin-converting enzyme inhibitors with dihydropyridine calcium channel blockers has been described. In this case, after sublingual administration of 25 mg of captopril and if it is insufficient for lowering blood pressure within 30 min of follow-up, 5 mg of nifedipine was given to the patient [19]. A dedicated study mentioned a clinical case of a successful intravenous infusion of a dihydropyridine calcium channel blocker, nicardipine, to stop AD [20].

The prevention of AD is aimed at reducing the frequency of episodes and its expanded clinical presentation. Botulinum toxin injections into the detrusor are still relatively limited in patients with damage to the cervical and upper thoracic spine because of the risk of AD development. However, data on a substantial decrease in the incidence of AD seizures after detrusor botulinization have been published. According to Jung et al. [23], botulinum toxin injections into the bladder wall of patients with severe SC damage reduced the mean systolic blood pressure. Persistent intake of anticholinergic drugs is a promising method for the prevention of AD caused by neurogenic dysfunction of the lower urinary tract. A recent study evaluated the effect of a selective M-cholinoblocker fesoterodine on the frequency and severity of AD [22]. Regular intake of alpha-1 blockers by patients with SCI causes a decrease in AD episodes. However, this is disputed by several researchers due to the risk of worsening arterial hypotension. The administration of nifedipine 30 min before the urodynamic study prevented effectively the onset of AD symptoms in patients with cervical spine injury. The constant intake of calcium channel blockers led to an increase in arterial or orthostatic hypotension [21].

Thus, AD can be classified as a dangerous complication of SCI. AD can be described as an uncontrolled sympathetic response to various painful stimuli and discomfort from the lower urinary tract, in particular to excessive bladder overflow, in patients with high-level SCI. AD is interpreted as a sudden increase in systolic blood pressure by >20 mm Hg from the baseline level. Treatment must be divided into non-drug and drug methods, relief of an acute condition, as well as preventive measures. The algorithm for AD prevention has not been established; however, among the promising methods, the constant intake of M-cholinoblockers and botulinum toxin injections into the detrusor are highlighted.

The most important aspect is providing information about the possible causes of AD, its clinical manifestations, and their prevention, not only for rehabilitologists, urologists, and general practitioners but also for the patients and those caring for patients with SCI.

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