After Spinal Cord Injury Sexual Functions and Histological-Physiological Changes in the Penis

Serdar Toksöz1* and Taha Numan Yıkılmaz2

1Department of Urology, Samandag State Hospital, Turkey
2Department of Urology, Ankara Dr. Abdurrahman Yurtaslan Oncology Training and Research Hospital, Turkey

SEXUAL FUNCTIONS

The control of sexual function is made by central nervous system like many body functions. Normal hypothalamic-pituitary-gonadal axis are needed for erection health. Autonomic and somatic nervous system are in charge of the erection except the central nervous system. These nerves are responsible in achieving erection by stimulation of the bulbocavernosus and ishiocavernous muscle [1]. Somatic nervous structures (S2-4) with Onuf nucleus provide the sensation of the penis with these muscles. Nitric oxide (NO) synthesis occurs due to parasympathetic innervations comes from preganglionic neurons in the intermediolateral column in this level. Nitric oxide provides erection with cyclic guanosine monophosphate (cGMP). Erectile dysfunction may grow in the process of the beginning of these pathways or shortage [2]. There are 3 types of erection; Psychogenic, nocturnal and reflexes erection. While psychogenic erections happening with various alerts, the mechanism of nocturnal erection is not illuminated accurately. The reflex erection realize with touch and requires the normal functioning of parasympathetic structures and intermediolateral in the sacral cord. Sympathetic structures of the spinal cord (T10-L2) are involved in the psychogenic erection. The presence of bulbocavernosal reflex also indicate the presence of the sacral reflex [3].

SPINAL KORD INJURIES

Spinal cord injuries (SCI) was seen generally in young people (80%) and observed 10,000 patients each year in the United States [4]. Sexual dysfunction is a common complication in men with SCI [5]. A spinal cord injury (SCI) affects a male’s sexuality psychologically, physiologically and emotionally. There are 3 phases in SCI; spinal shock, reflex returns and adaptation phase. The reflex penile erection, ejaculatory function, bulbokavernozal and scrotal reflexes are lost in the first phase. In this phase, the cause of penis elongation and a bit erections are paralytic cavernous vasodilation depends on impacting vasoconstrictor fibers in SCI [6]. Reflexes activity in lower extremities or spasticity may occur in the second phase of SCI. The adaptation phase is the period of recovery of sexual function and often occurs in the first year of the SCI. However this recovery can not be fully realized. In one study, 52% of men coitus 2-3 times a week before the SCI but this rate has fallen to 30% after trauma [7]. A sexual relationship is indicated as 48% once in a week. Other researchers have suggested that the frequency of relationship decreased from 3-4 in a week to 1-2. The level of SCI, degree and trauma capacity of sexual function before injury are important parameters about sexual dysfunction. Returning 70-80% of sexual function was seen within 6 months in cervical or thorocal injury but 40% of patients function was returned at 6 months in lumbar spine damage [8]. Patients with lesions above level of T11 (UMN) and lower motor neurone (LMN) lesions, have reflex erections but patients with lesions in between levels of L2 and S2 spinal segments have psychogenic erections [5]. Reflex erection can be achieved 95% and 25% in complete upper motor neuron (UMN) and lower motor neurone (LMN) lesions, respectively. Comarr et al was found the ratio of 95% and 12% [9]. The cause of this difference occurs according to the level of the lesion can be explained by the amount of NO levels increased in corpus cavernosus at the UMN injuries. Reflex erection was observed in 95% patients of incomplete UMN lesions, the rate of reflex erection can be up to 50% in complete damage [5].

SCI-induced ED patients has significant effects on the physiology and histology of penile tissue. There are important clinical studies about SCI effects that evaluating the physiological changes on penis. Studies that showing histopathological changes are more experimental because of challenging the technically problems. In experimental studies, there are many models of creating denervation that made cavernous nerve damage with trauma. The result of studies done by creating cavernous nerve damage in patients with SCI will be significant because physiological and histological changes on the penis realized in the event of failure to achieve an erection. The study about SCI-induced ED patients revealed the histopathological effects of on
the penis, Shin et al. reported that apoptosis, collagen deposition, smooth muscle cell loss and fibrosis were increased in the corpus cavernous tissue specimens obtained by percutaneous biopsy from 10 SCI-induced ED patients. And these changes occur due to increasing TGF-1. The authors also suggested that the patients might benefit from early pharmacotherapy (PDE-5 inhibitors) to prevent this injury. Lee et al., studies when bilateral cavernous nerve injury in rats, apoptosis, fibrosis, TGF-beta 1 and HIF-1 alpha expression increased accordingly significantly due to denervation and they found NO decrease [11]. Jin et al., found that apoptosis, fibrosis and smooth muscle loss play an important role in the pathophysiology of neurogenic ED. They showed that apoptotic index peaked and then began to decline after neurological damage in 1-2 weeks and reported that apoptosis occurs significantly in cavernous endothelial cells more than other tissues [12]. Experimental and clinical studies related to treatment of the SCI-induced ED patients offer us an important contribution in this regard. A study was made by Andersen et al. in 2007, SCI-induced ED patients succeed their erections as 23% received no treatment, 60% PDE5, 7.5% intracavernosal injection, 3.5% turistile, 3% vacuum device methods [13]. PDE5 is preferred first-line therapy and better response is conserved in the upper motor neuron lesions due to reflex erection. Reflex erection is reduced with the decrease NO secretion in parasympathetic ends of the lower motor neuron lesions. Chronic use of PDE-5 has been shown in animal studies is inhibiting apoptosis development in the corporal sinusoids, protecting smooth muscle ratio, and reduce collagen deposition due to the stress of disease [14]. Especially thought to block the pathophysiological mechanism of fibrosis due to regular erections with daily use and increase blood flow. In this regard, there are many studies of investigating the effect of PDE5 therapy. As a summary of these studies, the meta-analysis of 49 studies by De Forge et al., that compared oral pharmacotherapy, intracavernous injections, topical and intra-urethral therapy, sacral neuromodulation and penile prostheses options. They demonstrated that PDE-5 is the gold standard treatment in SCI-induced ED patients [15], and the first therapy of SCI-induced neurological ED patients [16]. Thirty one SCI patients with a T6 or above lesion enrolled a study about the activity of 50 mg sildenafil and intrakavernosal treatment. Both sildenafil 50 mg and intrakavernosal treatment had similar effect independently lesion levels [17]. Denervation in SCI induced ED patients ruins the vazodilatation response, by the affect on NO/cGMP pathway, consist of permanent damage in the cavernous tissue and early intervention to prevent apoptosis in smooth muscle cells and endothelial cells or to inhibit cavernous tissue fibrosis is required to restore erectile function.

REFERENCES


