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Research Article

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Cigarette Smoking Erectile Dysfunction and its Extended Psychobiology

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Abstract

Increasing cigarette smoking has been observed in males as a sign of being macho and manly. However, the reality is contrastingly unfortunate as tobacco smoking has an adverse influence on male sexual health causing mild-to-severe grades of impotence or erectile dysfunction (ED) and leading to adversarial psychological consequences, together these are termed here as the tobacco-penis syndrome (TPS). This brief review details the epidemiology, pathophysiology, and molecular basis of ED in smokers. It also discusses the consequential psychological aspects beyond the TPS. The article aims to generate awareness among men with elaborated scientific pieces of evidence in a concise manner.

Keywords: Male Sexual Health, Impotence, Erectile Dysfunction, Smoking, Tobacco-Penis Syndrome, Psychological Disorders

Background

Impotence or Erectile dysfunction (ED) is defined as the inability to achieve and maintain a penile erection sufficient enough to have successful penetrative penovaginal sexual intercourse. The global prevalence of ED is about 15% and it is estimated that in three decades (1995 – 2025) the number will be close to double, which is 320 million due to several causes [1]. Smoking affects male virility and pushes them into a negative biopsychosocial balance, such as a preponderance of developing life-threatening cardiovascular diseases (CVD), loss of self and social esteem, increasing sexual performance anxiety, depression, and autonomic stress. About 75% of men irrespective of their median age group suffer from ED in their lifetime [2]. A whopping percentage (32%) of smokers develop ED in their life around the world [3]. Although smoking is a potential cause of ED, however, other causative factors such as metabolic illnesses that cause microvasculopathy (dyslipidemia, uncontrolled type 2 diabetes, and hypertension), alcoholism, obesity, lack of exercise, drugs such as beta-blockers, and mental illnesses (e.g., manic-depressive, anxiety disorders, etc.) also are some of the known key causes of developing ED [4- 6]. Therefore, all these causative factors can be interrelated to cause profound ED or TPS in male sexual health.

Objective

- Detailing the pathophysiology of TPS, and
- Generating awareness among smokers.

Physiology of Erection

A pair of dorsal cavernosa and a ventral spongiosum (containing urethra up to the penile tip) are the main constituents of the penile tissue and decide on its state, shape, and size. Caverns are the containers of the blood pool while spongiosum gives its soft consistency making the penis a unique organ. These caverns, when filled with blood called cavernosal vasodilatation due to the lowering of intracavernosal pressure, pressure-dependent turgidity called an erection happens. Leakage of blood from the caverns causes non-sustenance or loss of erection while non-filling hinders achieving an erection. The pudendal artery primarily pumps blood into the penis through penile arteries supplying the cavernous arterioles and its minute sinuses lined with endothelial tissue and causing an erection to happen when these are dilated. On contrary, cavernous vasoconstriction mediated by norepinephrine due to sympathetic drive (e.g., performance anxiety state) in the body keeps the penile erectile tissues in a flaccid state. Venous outflow occurs through post-cavernous venules that finally form the emissary veins that in turn empty into the cavernous vein, then goes to the deep dorsal vein, and finally into the superficial dorsal vein. During the erection, the veins are compressed (called veno-occlusion) preventing leakage of blood to maintain the penile turgor until ejaculation and orgasm happen. The lumbar segments of the spinal cord are activated by the impulses from the afferents from the penis and the descending tracts to mediate erection in response to various types of sexually exciting stimuli [7]. The efferent parasympathetic fibers (pelvic splanchnic nerves), called 'nervi erigentes' (NE) release acetylcholine and vasoactive intestinal polypeptide as its co-transmitter causing an erection [7]. The NE also contains noradrenergic-noncholinergic fibers, which contain an enormous amount of nitric oxide synthase (NOS) enzyme that catalyzes the formation of NO from an amino acid L-arginine in the blood [7]. NO, in turn, activates soluble guanyl cyclase that increases the produc-

tion of cyclic guanosine monophosphate (cGMP), which is a potent vasodilator [7]. Among several phosphodiesterases (PDE), the PDE-5 is found in abundance in corpora cavernosa that destroys the cGMP. Therefore, PDE inhibitors, such as sildenafil, tadalafil, vardenafil, and avanafil are the mainstay of ED treatment [7]. Fig. 1. Shows the cross-sectional structure of the penis where individual structures of one side are labeled which implies similarity on the other side.

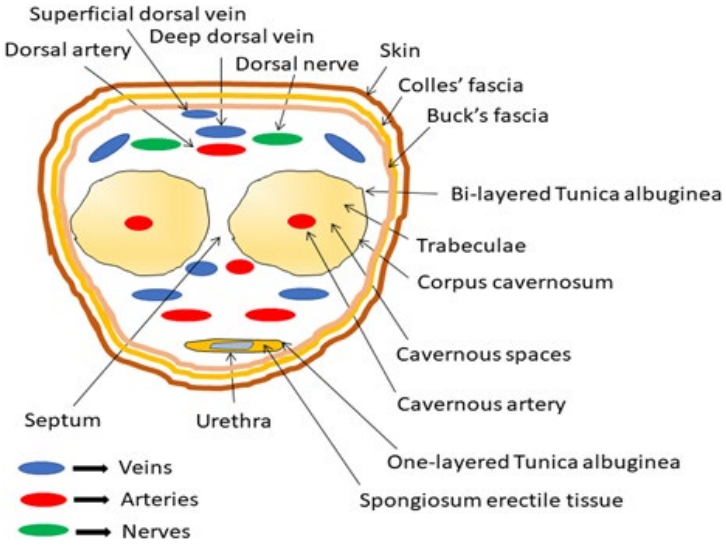

Figure 1: Cross-Section of The Penis

Fig. 1 shows the cross-section of the penile tissue and the associated structures. Beneath the skin, there are two types of fascia called the Colles' and Buck's fasciae, respectively from outside-inwards. These fasciae maintain the texture of the penis. Sidewise, there is a pair of corpus cavernosum, separated by a septum, and one corpus spongiosum, which are the key erectile tissue of the penis, covered by the bi-layered and one-layered tunica albuginea, respectively. The cavernous arteriole passes through the cavernous space while the urethra passes through the spongiosum. During the erection, the cavernous space is filled up with blood received from the cavernous artery. The venous outflow of blood happens through dorsal veins helping in achieving flaccidity. Alongside the dorsal vein, there lies the dorsal artery and dorsal nerve having abundant eNOS and nNOS helping to achieve the erection.

The Tobacco-Penis Syndrome (TPS)

Tobacco smoking and impotence (TPS) have been correlated for decades in various studies. Smoking can even cause permanent ED in a vulnerable population having potential comorbidities such as hypertension, diabetes, dyslipidemia, etc. as mentioned above. In this section, the pathophysiology of TPS is explained.

In the penile tissue, the endothelium of the arterioles and the sinuses, and smooth muscle cells mainly suffer from the impact of tobacco smoking. The NO plays the key deciding role in erection. Tobacco reduces the production of NO in the smooth muscle arterioles either by damaging the endothelial cells, downregulating the endothelial NO synthase (eNOS) production, or lowering the activity of neuronal NO synthase (nNOS) in the cavernous nerve endings as well as in the dorsal penile nerve in the penile smooth muscle. As a result of it, smoking causes diffuse vasculopathy at both the functional and structural levels, and in many instances, these are permanent changes [8], whereas endothelial-dependent vasodilatation does not happen in some heavy smokers. Some studies have shown that dysregulated vascular endothelial growth factors, migration of monocyte-like cells across the endothelium, loss of smooth muscles in the medial layer of the arterioles, and replacement by fibrous tissues or calcium deposits reduce the malleability of the vessels, and hence these are unable to dilate with response to eNOS [8]. Upon the release, NO diffuses into the neighboring smooth muscle cells of the corpus cavernosum and binds to soluble guanylyl cyclase that catalyzes the conversion of guanosine triphosphate into 3' 5' – cGMP, which in turn, activates the protein kinase-G (cGMP-dependent protein kinase I) that brings down the concentration of cytosolic calcium ions in the smooth muscle and endothelial cells. As a result of it, smooth muscle and arterioles are relaxed and it causes an erection to happen [8]. Nicotine in tobacco is a tertiary amine (composed of a pyridine ring), which induces endothelial dysfunction, which depends on the number of cigarettes consumed per day (dose) dose and years of smoking (duration). It increases the blood level of asymmetric dimethylarginine that inhibits eNOS [9] and as a result, smooth muscle and vascular endothelial cells remain in the contracted state leading to an irreversible ED or TPS. Cigarette smokes contain a high amount of superoxide radicals (O-) and reactive oxygen species. These are the potential destroyers of endothelial cells due to eNO shortage [8, 9]. Often permanent damage to the cavernous smooth muscle cells and the vascular endothelial lining by the above-mentioned mechanisms causes TPS.

The TPS Psychology

Nicotine dependence due to a long-term smoking habit and the

number of cigarettes smoked cause several psychiatric disorders [10]. This is one side of the coin. On the other side, mentally sick people show 3 times more preponderance of nicotine dependence (habitual smokers) and have 25% less chance of quitting when compared to the healthy population [11].

The TPS psychology is quite bizarre. Performance anxiety coupled with very low self-esteem versus a stupendous effort to cover up the issue leads to several behavioral issues commonly observed in several mental illnesses, such as schizophrenia, and other psychotic disorders [12]. As a result to it, often they are on hyping their superior sexuality which is untrue to hide the actual inability to perform penovaginal penetrative sexual intercourse (intrapersonal conflict due to functional impairment). Such thoughts, in them, are recurrent to keep them emotionally happy going with their lives, although within them they know what is the fact and what fiction they are spreading around. Many suffer from narcissistic personality disorders, called sexual narcissism [13]. This type of behavior is supported by a study that shows that smokers often use nicotine to reinforce social adjustment as they usually have a seriously downgraded coping strategy, and minimize the symptoms of mental illness that they possess [14]. Nicotine modulates the brain-derived neurotrophic factors, which is a type of endorphin that caters to the reward system of the brain, and therefore, nicotine deprivation in them causes serious withdrawal symptoms [15]. Fig. 2 gives the overall psychobiological consequences of tobacco smoking.

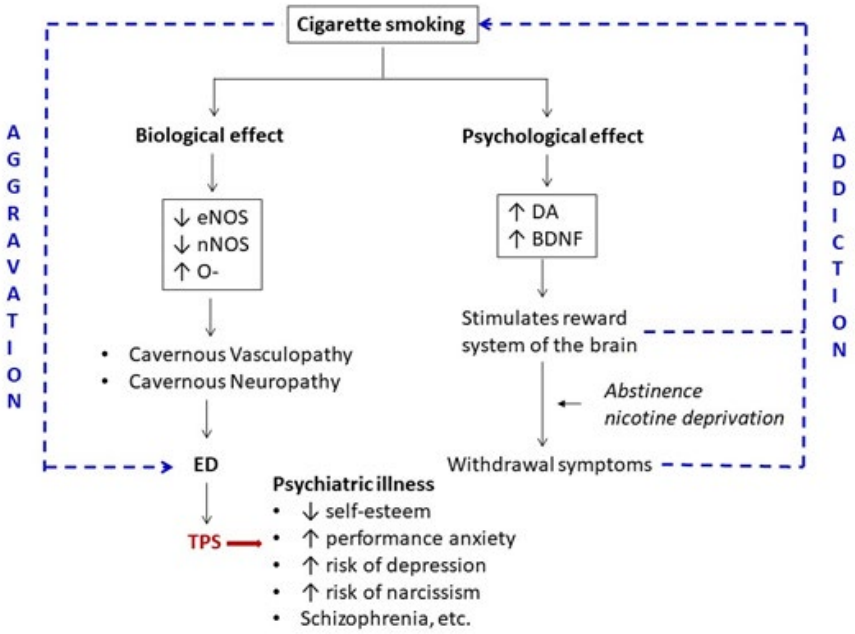

Figure 2: The Psychobiological Consequences of Tobacco Smoking

Fig. 2 describes the psychobiological effects of tobacco smoking in males. Smoking cigarette has biological and psychological effects simultaneously. Biologically it causes ED. Psychologically, it causes addiction over time by pleasuring the reward system of the brain by modulating dopamine (DA) and an endorphin called brain-derived neurotrophic factor (BDNF). Non-availability of nicotine causes severe withdrawal symptoms that push them more and more to crave smoking. More and more nicotine aggravates the ED into TPS, which in turn gives several mental illnesses as a cumulative consequence of ED and smoking.

Scope of Prevention

The duration (years of smoking) and dose (number of cigarettes smoked/day) are the major deciders of the onset, progression, and grade of ED, and then finally leading to irreversible TPS. Smoking causes as well as flares up various metabolic syndromes, which also contribute to developing TPS. Tobacco smoke affects the brain's reward system and releases dopamine or DA, a neurohormone for pleasure feelings [16]. A more elaborated description of the brain's involvement in smoking predominantly points toward the mesolimbic pathway where the dopaminergic neurons are abundant. These neurons project toward the nucleus accumbens and prefrontal cortex through the ventral tegmental area, which constitutes the key pathophysiology of tobacco addiction [16]. It is manifested by a strong uncontrollable craving for tobacco at times, and if not available, it gives serious side effects such as low self-esteem, inability to focus, nervousness, tremor, impulsive outbursts, and so forth, which are immensely encountered if the duration and dose of smoking are on the higher range. Such side effects produce an uncontrollable urge to continue smoking to get rid of those [17]. Hence, quitting smoking for chronic heavy smokers is not so easy and practically speaking, there is no preventive approach that assists in quitting smoking unless the person tries hard to quit nicotine by him/herself.

Conclusions

Smoking tobacco is not a sign of manliness or being macho as it is hyped. Rather smoking adversely affects sexual potency. It also affects other organs of the body causing several metabolic diseases. Quitting tobacco is not an easy task if the dose and duration

are high and lengthy, respectively. Our brain does not want to be deprived of dopamine-induced pleasure and elevated mood in the subcortical areas that it gets through tobacco smoking.

Various grades of ED occur over time following smoking and finally terminate into an irreversible TPS hampering one's psychological well-being. The overwhelming exhibition of being macho and the claim of being sexually hyper potent by some smokers is nothing but an effort to hide the gloomy side of their state of TPS. A larger study can be pursued to correlate the pretension of sexual hyper performance among smokers and their actual state of potency to assist them in coming out of financial loss (cigarettes burn a lot of cash) and several incapacitating mental illnesses, such as obsessive-compulsive disorders, bipolar disorders, delusional disorders, and so forth.

Associated comorbidities such as uncontrolled diabetes, hypertension, alcoholism, anxiety, and other behavioral disorders aggregated to a home-bound sedentary lifestyle increase the risk of developing TPS in vulnerable smokers.

Competing Interest

The author affirms no competing interest associated with this study.

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