Special Article

Erectile Dysfunction in People with Epilepsy: Similarities and Difference with the General Population

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Erectile dysfunction is a common problem in people with epilepsy(PWE). The etiology is multifactorial involving most epilepsy, antiepileptic drugs, and co-morbidity. It is an extensive influence on the quality of life and well-being of patients and their partners. Apart from epileptic disease and antiepileptic drugs (AEDs) use, the general medical history with the question of other possible risk factors and the specific sexual history play the most important roles in the guideline-based diagnostics. In addition, a thorough clinical examination and specific laboratory tests should be carried out. The appropriate treatment to be provided for epileptic patients with erectile dysfunction (ED) requires evaluation according to patient requirements, the epileptic condition and comorbidities and the medicines available for managing epilepsy. Various approaches might be helpful for treatment of ED in PWEs. Contemporary treatment algorithms for ED involve the use of non-pharmacological therapies; psychotherapy, changes of AEDs and pharmacological therapies such as phosphodiesterase type 5 (PDE5) inhibitors and intra-cavernosal injection therapy of vasoactive agents, as well as vacuum erection devices and penile prosthesis implants in PWEs. The success rates are high in most cases. Every neurologist, urologist, andrologist and other practitioners of sexual medicine should have detailed knowledge in this field. This state-of- the art article evaluates current and emerging therapeutic options for ED problems in PWEs.

Keywords: Erectile dysfunction, Sexual dysfunction, People with epilepsy

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Erectile dysfunction (ED) is the most common sexual problem in men. ED is defined as the consistent or recurrent inability to achieve and/or maintain penile erection sufficient to permit successful sexual intercourse, and it is a complex and heterogeneous disorder[1,2]. The incidence of ED is increasing globally, and it mainly affects men who are older than 40 years. ED occurs through multiple and complex mechanisms, including deterioration of the central or peripheral neural pathways, inadequate arterial supply to the penis, endothelial dysfunction, impaired smooth muscle tone, structural damage of the sinusoidal space of the erectile tissue, hormonal disorder, and psychological factors[3]. ED has also been found to be a symptom of serious illness such as cardiovascular disease, diabetes, metabolic syndromes, and all-cause mortality[4]. Although ED is not life threatening, it has a weighty effect on intimate relationships. It compromises the quality of life, and overall self-esteem for men[5].

Epilepsy is a chronic neurological condition characterized by recurrent epileptic seizures. They happen because of sudden, abnormal electrical activity in the brain. Seizures are divided into two main groups: focal seizures that happen in just one part of the brain and generalized that are a result of abnormal activity on both sides of the brain. Epilepsy has many possible causes, including illness, brain injury, and abnormal brain development, but sometimes the cause is unknown[6,7]. The epilepsy syndrome and localization influence the development of sexual dysfunction. Not only epilepsy but also antiepileptic drugs (AEDs) affect hormones and the neuroendocrine system, life style and can cause psychological problems. In addition, there are the physiological effects of epileptic discharges in brain regions mediating sexual behaviors[8]. The high rates of ED in PWE are likely, in some cases, to be a manifestation of hypothalamic dysfunction related to seizures and interictal discharges. This association between neurophysiological brain dysfunction and reproductive abnormalities is clearly relevant for physicians. Furthermore, as difficult as it may be to explore with patients in the closed office, sexual well-being is a high priority for almost everyone. The success rates are high in most cases. All neurologists, urologists, andrologists, and other practitioners of sexual medicine should have detailed knowledge in this field. This state-of-the art article evaluates current therapeutic options for ED problems in PWE.

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Prevalence of ED in the general population and PWEs

ED affects as many as 30 million men in the United States alone and 152 million men worldwide. It is projected that, by 2025, 322 million men worldwide will have ED[9].

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The prevalence of ED in the general population is 20 to 22%[10], which increases with age. The MMAS (Massachusetts Male Aging Study) demonstrated a 40% prevalence of ED in men aged 40 years, affecting up to 52% of men between the ages of 40 to 70 years that increased to almost 70% in men aged 70 years. In Asian populations the estimated prevalence of ED is 5 to 15% (40 to 49 years of age) and 39 to 49% (60 to 70 years of age)[11]. In Thai males, it is about 38% (40 to 70 years of age)[12]. PWE suffering from epilepsy had a significantly higher ED prevalence than the general adult population. In PWE, the ED prevalence data is controversial, with a wide prevalence range (i.e. from 23% up to 57%) depending on the methodology used and the type of epilepsy investigated[13-18]. However, most of the scientific reports on the prevalence and incidences of ED are based on questionnaire surveys and self-reported in the study population. In addition, different countries have cultural differences regarding sexuality that might influence the result[19], only half of the men who self-report ED are concerned about it. Western civilization has become more open-minded about discussing ED.

Pathophysiology of ED in general population and PWEs

In general, ED is frequently attributable to both psychogenic and physiologic factors. The physiological alterations in various neural, vascular, hormonal and endothelial functions. Physiology of ED is a normal neurovascular phenomenon that requires dilation of penile vasculature, relaxation of smooth muscle, increased intra-cavernosal blood flow and normal veno-occlusive function. Penile vascular disease is the most common cause of organic ED and may involve several pathophysiological mechanisms, including impaired arterial inflow, impaired smooth-muscle cavernosal relaxation, chronic ischemia-induced increased cavernosal smooth-muscle contraction, cavernosal fibrosis, veno-occlusive dysfunction and chronic or episodic hypoxaemia. Endothelial dysfunction appears to be the final common pathway for many cases of ED. Apart from age, the other main risk factors are those of vascular disease (smoking, diabetes mellitus, hypertension, abnormal lipid profile, obesity and lack of exercise), that damages endothelial function which can induce ED. Other factors include depression and endocrine disorders. Many neurological disorders including spinal cord injury, multiple sclerosis and cavernous nerve damage following major pelvic cancer surgery, commonly lead to ED. Endocrine disorders. Endocrine disorders, such as hypogonadism, hyper-prolactinaemia and thyroid disease play a significant role in ED physiology. Seizures can alter the release of hypothalamus and pituitary hormones. Temporal lobe epilepsy has an adverse effect on testicular endocrine. 40% of PWEs have low levels of testosterone. Testosterone regulates the cavernosal nerve structure and function, nitric oxide synthase expression and activity. Psychiatric comorbidity and endocrine abnormalities appear to be related to ED and seizure intractability to AED medication. Also antiepileptic therapy, and mainly enzyme-inducing drugs such as carbamazepine, phenytoin, and phenobabitone impaired not only hormonal release, but also the protein-binding sexual hormones, elevate Sex hormone binding globulin (SHBG) and reduce bioactive testosterone level in blood circulation, and may result in ED in PWEs[20-22]. In addition, epilepsy signifies anxiety and depression, low self-esteem and immaturity and it could lead to avoiding situations that call for affective sexual involvement[23,24].

Diagnosis and investigation

The initial evaluation of ED in PWEs should be done with awareness of multi-factorial etiologies. Several questionnaires have been developed to score the ED problem objectively. The short five-question form of the International Index of Erectile Function (IIEF), the IIEF-5 or Sexual Health Inventory for Men is useful for both diagnosis and assessment of the response to treatment. A full history and thorough clinical examination of the patient are needed to confirm that the patient is suffering from ED and/or another sexual dysfunction. It is important to determine whether ED is psychogenic or organic. The physician has to identify risk factors or comorbid disease. The medication history pays an important role. It is not plausible to consider antiepileptic drugs (AEDs) as the only cause of ED. Besides, the epilepsy itself, the AEDs and psychosocial components, other organic and medication induced factors might be causative[16,20].

Psychogenic ED is likely in younger men with no vascular risk factors who report an abrupt onset of ED and persistent early morning or nocturnal erections. Psychogenic ED can be caused by several problems, principally performance anxiety, but also guilt, depression, relationship problems, or fear and personal anxiety[20].

The physical examination of a man with ED will be directed, to a certain extent, by his history and should include assessment of the external genitalia, the endocrine and vascular systems, and the prostate gland in most patients[16].

The initial laboratory evaluation includes a full endocrine and metabolic status. The serum level of testosterone, estradiol, prolactin, and the thyroidal function should be evaluated. General investigations include serum concentrations of total testosterone (before 11 am), fasting glucose, fasting lipids and, in men over 50 years of age, prostate-specific antigen, luteinizing hormone, prolactin and high-density lipoprotein/low-density lipoprotein fractions of cholesterol[2,16].

Furthermore, special investigations, such as Colour Doppler imaging provides information about penile hemodynamic after maximal smooth-muscle relaxation has been induced with a vasoactive agent. Its aim is to distinguish arterial insufficiency and veno-occlusive dysfunction from other causes of erectile failure. Nocturnal penile tumescence and rigidity testing to evaluate the frequency, duration and rigidity of nocturnal erections is more of historical interest, and its contemporary use is largely limited to medicolegal assessment of erectile function[16].

Furthermore, a psychological screening for depression and anxiety disorders and a complete evaluation of sexual and relationship history is necessary to understand a patients complaints. Life stress should be considered as a contributing factor[2,16,19,25].

Therapy

Once the etiology of ED is determined, proper treatment strategies should follow. The goals of treatment of ED on PWEs are the same as with the general population. The successful treatment of ED has been shown to lead to a resolution of depression and the restoration of self-esteem, and thus improvement in the quality of life. The treatment options for men with ED are now varied and effective. Selecting from these various treatment options depends on several factors, such as the severity of ED, underlying causes and patient and partner choice. The results of the few studies that have been performed indicate that the only lifestyle modification may make a difference in ED incidence and its continuation or initiation of physical activity. Midlife changes in lifestyle other than physical activity may not have a beneficial effect on ED because it is simply too late[16].

In the following section, apart from conventional therapy; PDE-5 inhibitors, injection, vacuum pumps, and surgery, we review experimental and clinical studies investigating novel approaches that have significant pharmacotherapeutic potential in the management of erectile dysfunction.

Non-pharmacological therapy

Psychosexual therapy:

Psychosexual therapy for ED cannot be standardized because the source of anxiety varies among patients. Relationship difficulties, depression, guilt, problems with intimacy and lack of sexual experience may all increase anxiety and/or conflict, which may then manifest as ED. Psychosexual treatments range from simple sex education through improved partner communication to cognitive and behavioral therapy and are often combined with ED pharmacotherapy. A large proportion of patients have a combination of psychogenic and organic ED. Organic ED may be associated with progressively worsening performance anxiety, which further worsens erectile function. To treat the semen holistically, the physician and psychotherapist may need to collaborate and combine counselling with a physical therapy, such as an oral pharmacological agent[16].

Physical exercise:

Although, there is no scientific evidence that physical exercise can improve erection directly, it was reported that low levels of physical exercise can be associated with ejaculatory and erectile disorders. On the other hand, higher levels of physical exercise have been shown to improve erectile function in hypogonadal men undergoing testosterone replacement therapy[26]. Further scientific research is required to investigate whether there are specific benefits of acupuncture for men with ED before acupuncture can be accepted as evidence-based practice.

Pharmacotherapy

Current guidelines recommend a stepwise goal oriented decision making approach for the management of erectile dysfunction taking into account the patients' and partners' needs and preferences. The stepwise treatment algorithm recommends oral medications as first line therapy based on the ease of administration, reversibility and cost[26,27].

Oral pharmacological therapy:

1) PDE-5 inhibitors: The clinical efficacy and safety profile of PDE5i has been reported by numerous high-quality, well-designed, blinded, randomized controlled trials comparing PDE5i both to placebo and to other PDE5i drugs[28-30]. It works in a similar manner to increase nitric

Table 1. PDE-5 inhibitor (Modified from Hatzimouratidis, 2016)[32]

PDE-5 inhibitors	Dosage (mg)	Onset (minutes)	T1/2 (hrs.)	Adverse effect	PDE selectivity
Sildenafil (Viagra®)	25, 50, 100	15 to 60	4 to 5	Headache, Flushing, Dyspepsia	Low activity against PDE6, very low activity against PDE1
Vardenafil (Levitra®)	2.5, 5, 10, 20	15 to 30	4.8 to 6	Headache, Flushing, Rhinitis	Low activity against PDE6, very low activity against PDE1
Tadalafil (Cialis®)	2.5, 5, 10, 20	15	17.5 to 21	Headache, Dyspepsia, Backache	Low activity against PDE11, very low activity against PDE6
Avanafil (Stendra®)	50, 100, 200	33 to 52	5.36 to 10.66	Headache, Flushing, Backache, Fatigue	Highly selective for PDE5
Mirodenafil (Mvix®)	50, 100	40 to 90	1.32 to 3	Headache, Flushing, Backache, Dyspepsia	Comparable to sildenafil for PDE5
Udenafil(Zydena®)	100, 200	60 to 90	11 to 13	Flushing, Headache, Ocular hyperemia	Comparable to sildenafil for PDE5
Lodenafil(Helleva®)	160	80 to 120	2.2 to 4.4	Rhinitis, Headache, Flushing, Visual disorder	Low activity against PDE1 and PDE6

Table 2. Novel therapy option for erectile dysfunction (modified from Eric Chung, 2019)[53]

Therapeutic agents	Mechanism of action	Additional description	Status
Cellular-based therapy	1. Stem-cell therapy2. Platelet-rich plasma therapy	Methods to enhance the therapeutic effects with the use of growth factors, gene manipulationand matrixen	Phase I clinical trial
Gene therapy	1. Activator of nitrergic–neural system 2. Endothelial GFs promoters3. Modulator of ion channels in smooth muscle cells	Concurrent use of growth factors and vectordelivery system	Phase I & II clinical trial
Potassium channels	hMaxi-K (Ca2+ sensitive potassium channel), to relax corporal smooth muscle	Single intracavernous injection of a plasmid vector that expresses the *hSlo* gene, which encodes the αα-subunit of the Maxi-Kchannel related to cavernosal relaxation	Phase I clinical trial
Nitric oxide synthase (NOS) isoforms and drug delivery systems guanylate cyclase (GC)	1. Guanylate cyclase (GC) activators(e.g., BAY 60-2770) 2. RhoA/Rho-kinase inhibitor (e.g., Fasudil)	Drug delivery systems using oral disintegratingformulations and nanotechnology(e.g., nanoethosomes, transfersomes and penetrationenhancing lipid vesicles)	Preparing for clinical trial
Tissue engineering, Neural auto transplantation, Cavernous muscle cell auto transplantation	Tissue engineering using biomaterials, a cellularscaffolds and matrices Penile transplant using vascularizedcomposite allografts	Issues relating to cost, intensive programs, immunosuppression, psychological, safety andethical concerns	Phase I clinical trial
Melanocortin (MC) receptors	Peptide derives from pro-opiomelanocortin (POMC) protein	After injection into CNS structure, it induces erection	Phase IIa & IIb clinical trial
Endothelins (ETs)	Family of peptide potent vasoconstrictor, ETs has an importance role in the maintence of the flaccid state of the penis	It facilitated cavernosal smooth muscle relaxation, prolonged penile tumescence	Phase I clinical trial

GF = growth factor

oxide (NO) concentration and have relatively similar side effect profiles. It is important to note that PDE5i only works in combination with sexual stimulation, and despite the initial success in 65 to 70% of patients, 30 to 40% does not respond to PDE5i alone. One ED suggests that oral PDE5 inhibitors unless contraindicated, should be offered as a first line of therapy for erectile dysfunction[30,31]. Phosphodiesterase inhibitors (PDE5 inhibitors) are structurally similarly to cGMP and compete with cGMP at the catalytic site of PDE5. Their effectiveness correlates with their ability to block the degradation of cGMP in trabecular smooth muscle cells, which prolongs the low calcium state within the cells, resulting in relaxation and increased blood flow to the penis.

2) Dopamine receptor agonists: Apomorphine is a non-selective dopamine receptor agonist which stimulates both D_1 and D_2 receptor families, it has a higher affinity for D_2-like receptors that are thought to be the main site for the induction of erections in the paraventricular nucleus (PVN)[33]. It is therefore postulated to increase erectile responses by acting as a conditioner in the PVN, increasing the response to sexual stimuli resulting in enhanced erections induced in the periphery. It is degraded by the first-pass metabolism, so must be taken via injection, sublingually, or via a nasal spray formulation. In one of the largest clinical trials of apomorphine, a statistically significantly higher percentage of patients receiving apomorphine reported achieving and maintaining erections firm enough for intercourse versus the placebo group (48 to 53% vs. 35% for placebo, $p<0.001$). The efficacy of apomophine has also been affected by dose-limiting side effects (nausea and dizziness) and by a relatively short duration of effective functioning for 35 minutes[28, 33].

New drugs group; designed to act selectively on the D_4 receptor agonist (ABT 724, PD 168077, PIP 3EA), induces the release of oxytocin in brain areas that influence the activity of mesolimbic dopaminergic neurons mediating the appetitive and reinforcing effects of sexual activity. This is an exciting area of research which may lead to the development of more selective DA agonists capable of inducing penile erection but without concomitant emetic effects[28].

Topical therapy:

Prostaglandin E1 (PGE1), Topiglan (Macrochem Corp) is a combination of alprostadil gel 1% with 5% SEPA; an absorption enhancer. A study showed that its use resulted in an erection sufficient for penetration in 38.9% of the patients vs. 6.9% of the patients who received a placebo[34]. Alprox-TD, (Nexmed, Inc.) is a combination of alprostadil with NexAct; an absorption enhancer[28]. Improved erections were reported in 52% versus 20% in the placebo group. Adverse effects noted with both topical therapies were penile burning, genital pain, penile erythema and partner vaginal burning. Intraurethral alprostadil, often marketed as the Medicated Urethral System for Erection (MUSE), is a single-use pellet containing alprostadil suspended in polyethylene glycol administered using an applicator. Data from key clinical studies of intraurethral alprostadil show that it has a fast onset and a good safety profile, with no risk of penile priapism, fibrosis (as seen with intra-cavernosal injection) or other typical systemic effects observed with oral ED drugs[35]. The mechanism of action is that PGE1 modulates cAMP systems and activates a prostacyclin receptor on the corporal smooth muscle to increase the activity of adenylate cyclase and the accumulation of cAMP. An increase in cAMP leads to a cascade of intracellular changes that culminate in decreased intracellular calcium, and in turn, in the relaxation of the corporal smooth muscle cells and an erection[36].

Intra-cavernosal injection (ICI):

Alprostadil (Caverject® Impulse, Pfizer); prostaglandin E1 (PGE1), papaverine; a non-selective PDE5i, and phentolamine, which is a non-selective alpha-adrenergic antagonist that inhibits smooth muscle contraction. Intracavernosal vasoactive drug injection therapy is an effective alternative with minimal systemic side effects compared to oral ED therapy[37]. PGE1 can be used as a monotherapy or in combination with other vasoactive agents[32,35]. More recently aviptadil, a synthetic vasoactive intestinal polypeptide (VIP) that increases the activity of adenosine cyclase, was introduced and is available as a combination of aviptadil/phentolamine (Invicorp©). ICI injections are a moderately invasive therapeutic option and require a degree of manual dexterity, from the patient or partner, with education to learn the mechanics of self-injection. Discontinuation rates are typically greatest within 3 to 6 months of commencement and are usually due to factors such as pain, fibrosis, the lack of a sexual partner, loss of spontaneity and anxiety[38].

The principal side effects of an ICI of alprostadil are pain at the site of injection, which occurs in up to 30% of patients, and corporal fibrosis resulting in the development of penile nodules and curvature in 9 to 23.3% of mid- and long-term users. Priapism is a rare complication that can cause irreversible ischemic damage to the corporacavernosa with subsequent fibrotic damage and permanent loss of erectile function. Systemic side-effects are uncommon (approximate 1%), and include dizziness, tachycardia and hypotension, and result from leakage of the drug into the circulation[28].

Vacuum constriction devices

The vacuum constriction device involves application of a vacuum to the penis in a vacuum cylinder causing tumescence and rigidity, which is sustained using a constricting ring at the base of the penis. The penile physiological changes differ from a normal erection in that trabecular smooth-muscle relaxation does not occur, and blood is simply trapped in both the intracorporal and extra-corporal compartments of the penis distal by the constricting ring. Approximately 60 to 70% of men find the device straight forward. Satisfaction rates, both short and long term, vary considerably from as low as 27% to 68% short term, to as

high as 69% with 2 years follow-up. Adverse effect include petechiae; pain occurs at the site of the ring and ejaculatory changes, numbness and pivoting of the penis at the base. Contraindication of these devices is used in men who are taking warfarin and in men with an increased risk of intravascular thrombosis due to myeloproliferative diseases and sickle-cell anemia[28].

Penile vibratory stimulation (PVS)

PVS is a battery operated device that can provide excitation of afferent penile nerves at various regulated frequency and amplitudes. PVS should activate the ejaculatory reflex for patients with spinal cord injury above T10 seeking to collect retrogradely ejaculated semen in fertility treatment[26,39]. The Viberect® is a vibratory stimulation handheld device approved by the FDA for treatment of ED. It is clamp-shaped with two oscillating discs facing each other near the tips, and the glans penis is placed between the two oscillating discs to receive concurrent dorsal and ventral stimulation at adjustable frequencies and amplitudes.

The efficacy of this device, in a randomized controlled study by Fode et al (2013)[40] involving 68 men who underwent nerve-sparing radical prostatectomy, 30 men who received PVS to the frenulum daily for 6 weeks, using the Ferticare® vibrator, showed a trend towards better erections. After 1 year, 53% in the PVS group had an IIEF score 18 compared with 32% in the control group, although no statistical achievement was achieved.

Low intensity extracorporeal shock wave therapy (LIESWT)

Shockwaves induce a localized stress on cell membranes and this triggers the release of angiogenic factors, such as increased NO production through increased activity of endothelial NO synthase (eNOS) and neuronal NO synthase (nNOS), platelet-derived growth factor (PDGF) and vascular endothelial growth factor (VEGF). These shockwaves also cause membrane hyperpolarization, activation of the Ras signaling pathway, non-enzymatic synthesis of NO and induction of stress fibers and intercellular gaps[26,41,42].

Recent meta-analysis[41] of 14 studies showed that LIESWT could significantly improve the International Index of Erectile Function (IIEF) [mean difference: 2.00; 95% confidence interval (CI), 0.99 to 3.00; $p<0.0001$] and Erection Hardness Score (EHS) (risk difference: 0.16; 95% CI, 0.04 to 0.29; $p = 0.01$). In addition, the therapeutic efficacy was noted to last for at least 3 months.

Surgery

Surgical treatment composes of penile revascularization surgery and penile prosthetic implantation. It is usually reserved for patients in whom more conservative therapy has failed or for whom conservative therapy is contraindicated. Most of these patients will have significant arterial or venous disease, penile corpus cavernosum fibrosis or Peyronie disease, or will, by choice, prefer the prospect of a 'one-off' solution. While the outcome of surgical intervention may be more reliable in certain selected patients, the incidence of morbidity and complications is significantly greater than with medical treatment[28,37]. Penile prosthesis usually consists of a pair of rods which can be bent upright or downward depending on its use.

Alternative medicine

There are many plants that improve erectile function. Herbal products, such as yohimbine which is an indole alkaloid derived from the bark of the African yohimbe tree. It has been used to treat fatigue, depression, diabetes, and sexual dysfunction. The proposed mechanism of action is via the inhibition of central alpha-2-adrenergic receptors, decreasing central inhibition of arousal, and increasing penile nerve stimulation resulting in increased nitric oxide (NO). Common adverse effects include headache, sweating, agitation, hypertension and insomnia. Other plants that can arouse sexual activity, such as Korean red ginseng[43-46], *Tribulus terrestris*, Horny Goat Weed (Epimedium spp.), Tongkat Ali (*Eurycoma longifolia* Jack)[26], *Ginkgo biloba*[47,48], Saffron: *Crocus sativus* Linn[49,50], Red Kwao Krua (*Butea superba*)[51], and *Andrographis paniculata*[52].

Novel therapies

There are many options for new therapies that have been through a study process, such as gene therapy, potassium channels, nitric oxide synthase (NOS) isoforms and drug delivery systems guanylate cyclase (GC), growth factor targets, tissue engineering, neural auto transplantation, cavernous muscle cell auto transplantation, melanocortin receptors, and endothelins (shown in Table 2).

The past decade has seen an increase in our understanding of the physiology of penile erection, the pathophysiology of ED, and the development of new agents to manage ED. Failure to respond to PDE5-Is; a first line drug, in severe cases of ED has caused efforts to develop new treatment alternatives. Novel therapies for ED in the future will arise from an improved appreciation of erectile physiology and dysfunction. The development of new classes of drugs and therapeutic options should improve the therapeutic algorithm available to the clinician, for the successful therapy for both the patient and partner.

Conclusion

ED is a common compliant and is often associated with a reduced quality of life for sufferer and partner. ED is associated with a variety of risk factors. In PWEs, the causes of ED are more complicated than with the general population. Contemporary treatment algorithms for ED involve the use of non-pharmacological therapies; psychotherapy, changes of AEDs and pharmacological therapies such as phosphodiesterase type 5 (PDE5) inhibitors and intra-cavernosal injection therapy of vasoactive agents, as well as vacuum erection devices and penile prosthesis implants in PWEs,

for improving and/or restoring sexual function in most PWEs.

What is already known on this topic?

The authors knew of the incidence and prevalence of sexual dysfunction in general population and people with epilepsy. Many reported about the association between sexual dysfunction and epilepsy; risk factors can affect sexual problem.

What this study adds?

The review article has been useful to conclude the prevalence, etiology, diagnostic tools, and treatment of sexual dysfunction; focus in erectile dysfunction, in people with epilepsy.

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Potential conflicts of interest

The authors declare no conflicts of interest.

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ภาวะเสื่อมสมรรถภาพทางเพศในผู้ป่วยโรคลมชัก: ความเหมือนและความแตกต่างกับบุคคลทั่วไป

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ภาวะเสื่อมสมรรถภาพทางเพศเป็นปัญหาที่พบได้บ่อยในผู้ป่วยโรคลมชัก สาเหตุเกิดจากหลายปัจจัยได้แก่ จากโรคลมชักเอง ยารักษาโรคลมชักและโรคที่เกิดร่วม ภาวะนี้มีผลกระทบคุณภาพชีวิตของผู้ป่วยและคู่สมรส การตรวจวินิจฉัยนั้นนอกจากจะพิจารณาจากภาวะโรคลมชัก ยารักษาโรคลมชักแล้วยังต้องซักถามประวัติโรคร่วม ยารักษาโรคร่วม ปัจจัยเสี่ยงอื่น ๆ โดยเฉพาะประวัติเรื่องสัมพันธภาพทางเพศ ก็มีความสำคัญที่จะเป็นข้อมูลช่วยในการวินิจฉัย รวมถึงการตรวจร่างกาย การตรวจทางห้องปฏิบัติการ ส่วนแนวทางการรักษาต้องประเมินความต้องการของผู้ป่วย ภาวะโรคลมชัก โรคร่วมตลอดจนยารักษาโรคลมชัก นำมาพิจารณาทุกปัจจัยเพื่อช่วยในการรักษาภาวะเสื่อมสมรรถภาพทางเพศ การรักษาจะมีทั้งแบบไม่ใช้ยา ได้แก่ การพูดคุย การเปลี่ยนชนิดยารักษาโรคลมชัก และการรักษาด้วยยา ได้แก่ ยากลุ่มยับยั้งเอ็นไซม์ฟอสโฟไดเอสเทอเรส (PDE5 inhibitors) การฉีดยาขยายหลอดเลือดที่องคชาต (intracavernosal injection) การใช้อุปกรณ์สุญญากาศ (vacuum erection devices) และการผ่าตัดใส่องคชาตเทียม (penile prosthesis implants) ความสำเร็จในผลการรักษาจะสูงหากแพทย์ที่เกี่ยวข้อง ทั้งแพทย์ระบบประสาท แพทย์ระบบทางเดินปัสสาวะ แพทย์ที่รักษาบุรุษชายวัยทอง มีความรู้ความเข้าในเกี่ยวกับปัญหาเสื่อมสมรรถภาพทางเพศ บทความนี้จะช่วยแพทย์ผู้รักษาในการประเมินตัวเลือกการรักษาปัจจุบันและที่เกิดขึ้นใหม่สำหรับปัญหาภาวะเสื่อมสมรรถภาพทางเพศของผู้ป่วยโรคลมชัก