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Antidepressants and Sexual Health – How to Improve Patients' Quality of Life?

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Abstract

Introduction: Depression is one of the most common psychiatric disorders, with its prevalence continuously increasing. Consequently, more individuals are using antidepressants and experiencing various adverse effects. One of the important, yet often overlooked, side effect of depression pharmacotherapy is sexual dysfunction. This article aims to review sexual dysfunction associated with antidepressant therapy, explore available management strategies, and highlight emerging therapeutic options.

Materials and methods: A literature review was performed using the PubMed and Scopus databases, covering publications from 2020 to 2024 focused on antidepressant-induced sexual dysfunction. Open-access publications in English-language were included, comprising original research articles, systematic reviews, meta-analyses, and narrative reviews.

Results: The most frequently reported antidepressant-related sexual dysfunctions include decreased libido, delayed ejaculation, anorgasmia, and erectile dysfunction. These effects are most often observed in patients treated with selective serotonin reuptake inhibitors (SSRIs) and serotonin-norepinephrine reuptake inhibitors (SNRIs). A lower, though still relevant, risk is associated with tricyclic antidepressants (TCAs) and monoamine oxidase inhibitors (MAOIs). Medications such as vortioxetine, agomelatine, vilazodone, and bupropion offer promising therapeutic alternatives with a reduced risk of sexual side effects. Moreover, growing evidence supports the effectiveness of adjunctive and non-pharmacological interventions.

Conclusions: Sexual dysfunctions significantly impair patients' quality of life and often lead to treatment discontinuation and recurrence of depressive symptoms. Despite their prevalence, many patients do not report these symptoms due to embarrassment, which underscores the need for a conscious, empathetic approach from doctors. Active monitoring and implementing effective strategies to minimise these side effects without compromising antidepressant efficacy are essential.

Keywords: SSRI, Antidepressants, Sexual dysfunction, Alternative treatment

Streszczenie

Wstęp: Depresja stanowi jedno z najpowszechniejszych zaburzeń psychicznych, a jej częstość występowania stale wzrasta. W rezultacie coraz więcej osób stosuje leki przeciwdepresyjne i doświadcza różnych działań niepożądanych związanych z ich przyjmowaniem. Ważnym, a jednocześnie często pomijanym skutkiem ubocznym farmakoterapii depresji są dysfunkcje seksualne. Celem niniejszej pracy jest omówienie dysfunkcji seksualnych występujących podczas terapii lekami przeciwdepresyjnymi oraz dostępnych strategii radzenia sobie z nimi, a także przedstawienie najnowszych opcji terapeutycznych.

Materiały i metody: Przeprowadzono przegląd literatury w bazach danych PubMed i Scopus, obejmujący publikacje z lat 2020–2024 dotyczące dysfunkcji seksualnych związanych z terapią przeciwdepresyjną. Uwzględniono ogólnodostępne artykuły w języku angielskim, w tym prace oryginalne, przeglądy systematyczne, metaanalizy i prace przeglądowe.

Wyniki: Dysfunkcje seksualne najczęściej zgłaszane przez pacjentów to zmniejszone libido, opóźniony wytrysk, anorgazmia

i zaburzenia erekcji. Najczęściej występują one u pacjentów stosujących inhibitory wychwytu zwrotnego serotoniny (SSRI), a także inhibitory wychwytu zwrotnego serotoniny i noradrenaliny (SNRI). Mniejsze, choć nadal istotne ryzyko, wiąże się ze stosowaniem trójpierścieniowych leków przeciwdepresyjnych (TCA) czy inhibitorów monoaminooksydazy (IMAO). Leki takie jak wortioksetyna, agomelatyna, wilazodon i bupropion, stanowią obiecującą alternatywę terapeutyczną z niższym ryzykiem zaburzeń seksualnych. Dodatkowo coraz więcej danych wskazuje na skuteczność terapii wspomagających oraz metod niefarmakologicznych.

Wnioski: Zaburzenia seksualne istotnie pogarszają jakość życia pacjentów, często prowadząc do przerwania terapii i nawrotu objawów depresji. Pomimo ich powszechności wielu pacjentów nie zgłasza objawów z powodu wstydu, co wymaga świadomego, empatycznego podejścia lekarzy. Kluczowe jest ich aktywne monitorowanie oraz wdrażanie skutecznych strategii minimalizujących objawy bez utraty skuteczności przeciwdepresyjnej.

Słowa kluczowe: SSRI, Leki przeciwdepresyjne, Dysfunkcja seksualna, Alternatywne leczenie

Introduction

Sexual dysfunctions represent a significant clinical issue in patients with depression, arising from both the disease itself and the pharmacological treatment used. The scale of this phenomenon is considerable, with estimates indicating that from 50% to 70% of patients with depression experience sexual disorders (1). The prevalence of these disorders is strongly associated with the type of antidepressants used. Selective serotonin reuptake inhibitors (SSRIs) are the most commonly prescribed first-line treatments for depression and are associated with the highest incidence of sexual side effects. Nevertheless, SSRIs are not the only therapeutic option. In cases of adverse effects, inadequate treatment response, or other indications, alternative classes of antidepressants are used, such as serotonin-norepinephrine reuptake inhibitors (SNRIs), tricyclic antidepressants (TCAs), or monoamine oxidase inhibitors (MAOIs) (1–3). Patients with depression constitute a significant proportion of the psychiatric patient population, and the search for new, effective treatment methods remains a key research priority. Despite numerous studies and attempts to introduce innovative approaches, pharmacological treatment, alongside psychotherapy, continues to be the foundation of therapy. Pharmacotherapy, despite its efficacy, is not without limitations and is associated with the risk of adverse effects. While severe adverse effects are rare, their risk increases in specific patient populations. This particularly applies to patients receiving tricyclic antidepressants (TCAs), where an increased risk of QT interval prolongation and cardiovascular events is observed. High-risk groups include elderly individuals, particularly females, patients with electrolyte disturbances, and those with pre-existing cardiac conditions such as arrhythmias, coronary artery disease, or left ventricular dysfunction (4-6). Another potentially serious but rare complication is the increased risk of internal bleeding in patients receiving SSRIs. This mechanism is linked to a reduction in intraplatelet serotonin levels, which are critical for normal platelet aggregation and hemostasis. This leads to platelet dysfunction, manifesting as prolonged bleeding time and an increased risk of clinically significant hemorrhages, particularly gastrointestinal bleeding and intracranial hemorrhage. This risk is notably elevated in patients concurrently receiving nonsteroidal anti-inflammatory drugs (NSAIDs), anticoagulants, and antiplatelet agents, especially during the initial phase of SSRI treatment (7,8). Despite the potentially serious, yet rare, aforementioned adverse effects, less severe adverse reactions are much more commonly reported as a consequence of pharmacotherapy for depression, though impacting patients' quality of life. These include weight gain, chronic fatigue, headaches, cognitive impairment, and, importantly from a quality-of-life perspective, sexual dysfunctions (9-11). Difficulties with sexual function, commonly referred to as antidepressant-induced sexual dysfunctions (AISD), can occur at all stages of the sexual response cycle, affecting both the arousal and sexual satisfaction phases. The primary neurochemical mechanism associated with AISD is serotonergic modulation, particularly the overactivation of serotonin receptors (especially 5-HT2 and 5-HT1A subtypes), which negatively impacts sexual desire, arousal, and orgasm (12,13). In the female population, issues during the sexual arousal phase are more frequently reported, manifesting as decreased vaginal lubrication and reduced genital sensation (13,14). Among men, common manifestations include erectile dysfunction and delayed ejaculation (1). Across both sexes, diminished libido and difficulty achieving orgasm are commonly observed. This issue can affect up to 40% of patients taking SSRIs (1,15,16). These disturbances significantly reduce patients' quality of life and may lead to strained interpersonal relationships, which further impacting the course of depression treatment (1,15). This article aims to review sexual dysfunction in individuals undergoing antidepressant treatment, as well as current

and future approaches to their management.

Material and Methods

A comprehensive literature search was conducted in the PubMed and Scopus databases to identify relevant studies on clinical trials and therapeutic developments in the treatment of sexual dysfunctions induced by antidepressant therapy. The search covered publications from January 2020 to December 2024, using keywords such as "SSRI," "SNRI," "antidepressants," "depression," "TCA," "sexual dysfunction," "side effects," "MAO inhibitors," and "alternative treatment."

Inclusion criteria encompassed original research articles, systematic reviews, review articles, and meta-analyses published in English that provided clinical or preclinical data. Opinion essays, articles in languages other than English, and studies with insufficient methodological details or unavailable full texts were excluded.

Selective Serotonin Reuptake Inhibitors

SSRIs are used in the treatment of various conditions, including depression, anxiety, post-traumatic stress disorder, and obsessive-compulsive disorder (17). Their mechanism of action involves inhibiting serotonin reuptake by blocking the serotonin transporter (SERT). SSRIs have minimal effects on other neurotransmitters, which contributes to a lower incidence of side effects (19,20). SSRIs are the first-line pharmacological treatment for depression and are the most commonly prescribed due to their high efficacy and favorable tolerability (3). Currently used SSRIs include sertraline, paroxetine, citalopram, escitalopram, fluvoxamine, and fluoxetine (3). Despite their good tolerability, these drugs are associated with side effects. The most common adverse effects of SSRIs include nausea, gastrointestinal disturbances, dizziness, headaches, fatigue, sleep disturbances (primarily insomnia), sexual dysfunction, and weight gain (17,20,21). It is important to emphasise that both insomnia and hypersomnia are significantly associated with the presence of depression (22). The initial phase of pharmacotherapy with SSRIs may lead to a temporary worsening of sleep disturbances. However, with continued treatment and the remission of depressive symptoms, many patients experience a gradual normalisation of sleep architecture. In the long term, the use of SSRIs may contribute to improved sleep quality, particularly in individuals for whom sleep disturbances constitute a key component of the clinical presentation of depression (23).

SSRIs could have a negative impact on patients' sexual health (3). In a study investigating the side effects of three SSRIs - sertraline, escitalopram, and fluoxetine - a significant proportion of participants reported sexual dysfunction as an adverse effect. The most commonly reported symptoms included decreased libido, anorgasmia, reduced sexual satisfaction, delayed ejaculation, and erectile dysfunction (20). According to studies, approximately 50% of patients treated with paroxetine experience decreased libido, premature ejaculation, and anorgasmia. In the case of fluoxetine, these disorders occur in about 30% of treated individuals (16). These symptoms may arise during SSRI therapy and persist even after treatment discontinuation. Sexual dysfunction as an adverse effect can also occur post-treatment, a condition known as post-SSRI sexual dysfunction (PSSD) (24). SSRIs may affect sperm morphology, count, motility, and the integrity of genetic material, while typically having no significant effect on ejaculate volume (25). A study assessing the effects of sertraline and fluoxetine on human sperm demonstrated that individuals taking these medications may experience impaired fertility. Both drugs can negatively affect sperm function, leading to reduced sperm motility. Fluoxetine induces this harmful effect even at low, therapeutic doses. Sertraline has minimal impact on sperm function at low doses. However, it causes significant sperm damage at concentrations exceeding therapeutic levels (26). The adverse effects of antidepressants on semen quality typically emerge around three months after starting treatment, which corresponds to the duration of spermatogenesis - a process that takes approximately 72 days (25). The case described by Elnazer and Baldwin involves a 30-year-old man who had been taking citalopram for three years to treat depression and anxiety. During the treatment there were observed abnormalities in semen parameters. Four months after discontinuing the medication, improvements were noted in sperm concentration, motility, and morphology (27). In another study, two cases were described of patients treated with SSRIs for depression who developed fertility problems. The first patient, treated with citalopram, was diagnosed with oligozoospermia. After discontinuation of the drug, semen parameters returned to normal within one month. The second patient had been taking sertraline for five years; his semen showed normal volume but a very low sperm count (20,000) with no motile sperm. Three months after discontinuation of sertraline, semen analysis revealed normal parameters, with a total of 40 million motile sperm (28). In general, it is assumed that semen parameters return to baseline within approximately three months after stopping antidepressant treatment. This timeframe corresponds to a full cycle of spermatogenesis, which is necessary to produce healthy and functional sperm. (25). However, these three case reports are far too limited to draw firm conclusions. Although they provide valuable insights into the potential reversibility of SSRI-induced semen abnormalities, further large-scale studies are needed to better understand the prevalence,

severity, and mechanisms of these adverse effects. A prospective cohort study could be designed involving a larger population of men treated with different SSRIs, assessing semen parameters, sperm DNA integrity, hormone levels, and sexual function at baseline, after 12 weeks of treatment (one spermatogenesis cycle), and following therapy discontinuation. Such a study would help provide clearer correlations between specific antidepressants, treatment duration, and the extent of reproductive impairment, ultimately supporting more individualised therapeutic decisions. This is particularly important as symptoms impacting sexual function may lead to discontinuation of the treatment, poor treatment adherence, overall health decline in patients, and worsening of depressive symptoms (20,29).

Serotonin-Norepinephrine Reuptake Inhibitors

SNRIs are medications that exert their effect by blocking serotonin (5-HT) and norepinephrine (NE) transport proteins, thereby inhibiting the reuptake of these neurotransmitters. This class of drugs includes venlafaxine, duloxetine, milnacipran, desvenlafaxine, and levomilnacipran (30,31). Along with SSRIs, SNRIs are among the most commonly prescribed medications for patients suffering from severe depression (32). However, similarly to SSRIs, SNRI use is associated with a high risk of sexual dysfunction. The most commonly reported issues by patients include erectile dysfunction, decreased libido, genital numbness, orgasm inhibition, and delayed ejaculation. However, there is still a lack of studies comparing the risk associated with specific drugs within this class (1,24,32). It is believed that duloxetine carries a lower risk of sexual dysfunction compared to venlafaxine (1). Duloxetine, like other SNRIs, inhibits the reuptake of serotonin (5-HT) and norepinephrine (NE), which may adversely affect sexual function by disrupting the balance of neurotransmitters in pathways responsible for sexual arousal (33). On the other hand, there is a study suggesting that women treated with duloxetine for stress urinary incontinence experienced an improvement in sexual activity. However, this effect is most likely attributable to the simultaneous alleviation of incontinence symptoms and the reduction of anxiety and depressive symptoms, which often accompany this condition. The study was conducted on 40 patients, which may limit its scientific validity (20).

Tricyclic Antidepressants

Tricyclic antidepressants (TCAs) are considered a second-line treatment for depression. Despite their proven high efficacy in treating severe or treatment-resistant depression, SSRIs are more commonly preferred due to their lower risk of adverse effects and higher safety margin in overdose cases (34). The therapeutic effect of TCAs is based on inhibiting the reuptake of serotonin and norepinephrine in the central nervous system (35). Currently used medications in this class include amitriptyline, desipramine, imipramine, clomipramine, doxepin, nortriptyline, and protriptyline (36). The use of these drugs is associated with a moderate risk of sexual dysfunction; however, there is a limited number of studies assessing this risk. Among TCAs, only clomipramine has been linked to a high risk of sexual dysfunction, comparable to that of SSRIs (1). The use of this medication is associated with a significant risk of ejaculation disorders, including retrograde ejaculation, delayed ejaculation, and anejaculation, as well as a high risk of erectile dysfunction (37). Additionally, decreased libido is among the adverse effects associated with clomipramine use (38).

Monoamine Oxidase Inhibitors

Due to numerous side effects, dietary restrictions, and safety concerns, Monoamine Oxidase Inhibitors (MAOIs) are used primarily when other antidepressants have proven ineffective (39). These medications work by inhibiting the monoamine oxidase (MAO) enzyme, which exists in two isoforms: MAO-A and MAO-B. MAO plays a key role in neurotransmitter inactivation, with MAO-A targeting norepinephrine, serotonin, and dopamine, while MAO-B primarily affects dopamine. As a result, MAOI therapy leads to increased synaptic availability of these neurotransmitters (40,41). In the treatment of depression, non-selective MAOIs such as tranylcypromine, phenelzine, and isocarboxazid, as well as selective inhibitors like moclobemide (MAO-A inhibitor) and selegiline (MAO-B inhibitor), are used (39,41,42). Sexual dysfunction is also among the adverse effects associated with MAOI use, affecting approximately 40% of both male and female patients taking these medications (42). Among non-selective MAOIs, phenelzine and isocarboxazid are more frequently linked to sexual dysfunction, whereas tranylcypromine is rarely associated with such issues (43). The use of moclobemide is associated with a low risk of sexual dysfunction (comparable to placebo), although the data supporting this are limited (1).

Trazodone

Trazodone is a triazolopyridine compound with a dual mechanism of action. It inhibits SERT and acts as an antagonist of 5-HT2A and 5-HT2C serotonin type 2 (5-HT2) receptors (44). Additionally, it blocks histaminergic and alpha-1-adrenergic receptors while exhibiting minimal anticholinergic effects (45). Trazodone is used in the treatment of depression both as monotherapy and in combination with other antidepressants (46). In addition

to its antidepressant effects, trazodone also exhibits anxiolytic and sedative properties. It is used in individuals suffering from insomnia and excessive agitation (47). The antagonistic action on 5-HT2 receptors reduces the risk of side effects such as insomnia, sexual dysfunction, and anxiety (44). The most common adverse effects of trazodone include excessive drowsiness, xerostomia, as well as headaches and dizziness. A rare but characteristic side effect is priapism (45). Priapism is a prolonged erection lasting more than four hours, unrelated to sexual stimulation or orgasm. It is most likely caused by the blockade of alpha-1-adrenergic receptors (48,49). The use of an extended-release formulation and caution in patients with anatomical penile deformities or sickle cell anemia can help to reduce the risk of adverse effects leading to sexual dysfunction (45).

Management of Antidepressant-Induced Sexual Dysfunction

The diagnostic process for sexual dysfunction during antidepressant therapy should begin with an evaluation of other potential factors unrelated to antidepressant pharmacotherapy that may contribute to its occurrence and intensification. A comprehensive medical history and assessment of the patient's sexual functioning are crucial, ideally obtained before treatment initiation and during therapy. First of all , alternative etiologies, including emotional and relational factors, must be excluded. Common causes of sexual dysfunction include chronic psychosocial stressors (e.g., interpersonal conflicts, occupational difficulties, financial hardship), relationship dysfunctions (e.g., poor sexual communication, mismatch in sexual needs, lack of intimacy or trust), sexual myths or anxieties, and past traumatic sexual experiences (50). Equally important is an assessment of the patient's somatic condition. Conditions such as diabetes, hypertension, obesity, atherosclerosis, endocrine disorders (e.g., hypothyroidism, hyperthyroidism, hypogonadism, and hyperprolactinemia), and peripheral neuropathies can significantly impair sexual function. Therefore, when clinically indicated, diagnostic work-up should include a targeted physical examination and laboratory tests, such as blood levels of glucose, lipids, sex hormones, prolactin, and thyroid-stimulating hormone (TSH)(51-57). Sexual dysfunction may also arise directly from depression itself, independently of pharmacologic treatment. Additionally, the presence of comorbid psychiatric conditions, such as anxiety disorders, obsessive-compulsive disorder, or post-traumatic stress disorder, should be considered, as they may further worsen sexual dysfunction symptoms (58).

The pharmacological history of the patient also needs to be carefully analysed, particularly regarding other medications that may induce or worsen sexual dysfunction. Among the most frequently implicated are cardiovascular drugs such as beta-blockers and thiazide diuretics. Beta-blockers (especially first- and second-generation) may impair sexual function by inhibiting sympathetic nervous system activity, reducing genital blood flow, and affecting the central nervous system, potentially leading to decreased libido, mood disturbances, and erectile dysfunction in men. Thiazide diuretics contribute to sexual dysfunction through electrolyte imbalances, reduced intravascular volume, and endothelial dysfunction, thereby impairing nitric oxide (NO) production - a key mediator of vasodilation. This may aggravate erectile difficulties in men and contribute to vaginal dryness and difficulty achieving orgasm in women (52). Statins, widely used to treat hypercholesterolemia, may also impact sexual functioning. By lowering cholesterol levels, a precursor for steroid hormones, they may reduce testosterone levels, contributing to diminished libido and erectile dysfunction (59). Some drugs, such as antipsychotics (e.g., risperidone, haloperidol) and prokinetics (e.g., metoclopramide, domperidone), act as dopamine receptor antagonists and inhibit the dopaminergic suppression of prolactin release. As a result, they can cause hyperprolactinemia, which secondarily reduces sex hormone levels and may lead to decreased libido, erectile dysfunction, and anorgasmia (56). Other pharmacological agents with confirmed effects on sexual function include pregabalin and lithium. Pregabalin, used primarily for neuropathic pain treatment, may cause reduced libido, erectile dysfunction, and orgasmic disorders. These effects are likely related to the drug's inhibition of voltage-gated calcium channels, which reduces the release of neurotransmitters essential for sexual arousal. Additionally, pregabalin increases extracellular levels of gamma-aminobutyric acid (GABA), which may suppress NO and vasoactive intestinal peptide (VIP) activity, further impairing sexual function (60). Lithium, commonly prescribed for bipolar disorder, may induce sexual dysfunction by impairing the NO-dependent endothelial pathway, disrupting smooth muscle relaxation in the corpus cavernosum, and thereby contributing to erectile dysfunction. It may also adversely affect all phases of the sexual response cycle, reducing desire, impairing arousal, and delaying or inhibiting orgasm (61). A thorough history should also include an evaluation of psychoactive substance use, particularly alcohol and opioids. Both substances can directly suppress the hypothalamic-pituitary-gonadal (HPG) axis, leading to reduced gonadotropin and testosterone levels. Alcohol may also damage testicular tissue, impairing hormone production. These mechanisms contribute to decreased libido, erectile dysfunction, and anorgasmia (62).

Only after excluding alternative causes of sexual

dysfunction should the relationship between symptoms and antidepressant pharmacotherapy be considered. Importantly, the emergence of sexual dysfunction following the initiation of treatment does not, by itself, confirm causality.

The primary goal of managing sexual dysfunction associated with antidepressant therapy is to minimise adverse effects on sexual health while maintaining the effectiveness of depression treatment. An individualised approach that considers the patient's specific needs is essential to improving quality of life and achieving therapeutic success. Proposed management strategies for AISD include pharmacotherapy modifications, adjunctive treatments, and psychotherapeutic support (1,50).

Watchful Waiting

In some cases, AISD may be temporary and resolve spontaneously as the body adapts to pharmacotherapy. This is particularly true for SSRIs, where symptoms such as decreased libido, delayed ejaculation, or erectile dysfunction often emerge early in treatment but may gradually decrease over time (3). This improvement is thought to be related to compensatory changes in the receptor system, including desensitisation of 5-HT1A receptors and restoration of neurotransmitter balance (24). A "watchful waiting" strategy may be appropriate, especially in patients who respond well to antidepressant treatment and whose sexual side effects are mild and do not significantly impair quality of life. In such cases, it may be reasonable to postpone active intervention while carefully monitoring the patient's condition and symptom progression. However, this approach requires regular assessment of adverse effects and open communication with the patient - both to evaluate the level of discomfort and to determine readiness to initiate alternative treatment strategies if no improvement occurs (50). In approximately 6-12% of patients, symptoms disappear completely or significantly improve within 4-6 months, despite no modification of antidepressant treatment (1).

Dose Reduction

Reducing the dose of an antidepressant can be considered in cases of treatment-emergent sexual dysfunction, especially when the current dose exceeds the minimal therapeutic dose. Evidence suggests that the risk of sexual dysfunction may be dose-dependent. However, there is significant individual variability - some patients experience sexual side effects even at low doses, while others tolerate higher doses without such adverse effects. These differences may result from pharmacodynamic and genetic factors (63). The decision to reduce the dose should be made carefully and individually, taking into account factors such as the patient's medical history, duration of remission, and the risk of depressive relapse. Regular monitoring of both the patient's mental state and the severity of sexual symptoms following dose adjustment is essential. It is important to note that dose reduction does not yield clinical benefit in all patients - some patients may experience a worsening of depressive symptoms, necessitating a return to the previous treatment regimen or a change in therapeutic approach (50). Moreover, rapid dose reduction carries a risk of withdrawal syndrome. Therefore, the dose should be reduced gradually, ensuring it is not lower than the minimal effective dose (64).

Drug Holiday

A short-term break from medication before anticipated sexual activity is one potential strategy for managing persistent sexual dysfunction. The benefits of a "weekend drug holiday" have been observed in two studies involving patients treated with SSRIs, excluding fluoxetine due to its long half-life (65,66). In women, improvements were noted in areas such as arousal, orgasm, lubrication, and overall sexual satisfaction, with no reports of serious adverse effects (65). In men, medication breaks significantly enhanced erection, ejaculation, and sexual satisfaction without negatively impacting mental health (66). However, this approach carries the risk of symptom recurrence and may cause anxiety due to the need to plan sexual activity. Additionally, it is not suitable for medications with a long duration of action (50).

Alternative Antidepressants

Several studies highlight the benefits of switching to an antidepressant with a lower risk of sexual dysfunction. One such option is vortioxetine, which works by inhibiting serotonin reuptake and modulating serotonin receptor activity. In patients experiencing sexual dysfunction due to the usage of SSRIs such as sertraline, citalopram, and paroxetine, switching to vortioxetine resulted in greater improvement in sexual function compared to switching to escitalopram, while maintaining antidepressant efficacy (67). Another study reported beneficial effects of transitioning from various classes of antidepressants to vortioxetine, noting improvements in sexual desire, orgasm, and arousal (68). Huang et al. identified agomelatine as an effective treatment option with a low risk of sexual dysfunction. Its mechanism of action involves agonistic effects on melatonin receptors MT1 and MT2, along with antagonism of the 5-HT2C serotonin receptor (69). Vilazodone is another promising alternative. In comparison with sertraline, it demonstrates similar efficacy in treating depression while having a minimal impact on sexual function. Its mechanism combines SSRI properties with partial agonism of the 5-HT1A receptor (70). A recent network meta-analysis of thirty randomised

controlled trials found that vortioxetine, agomelatine, and vilazodone are viable alternatives for male patients experiencing AISD, particularly in cases of ejaculatory disorders and reduced libido (37). Another antidepressant considered in such cases is bupropion, which acts by inhibiting the reuptake of dopamine and norepinephrine. In addition of not causing sexual dysfunction, it has also been suggested to have a beneficial effect on sexual function in women with hypoactive sexual desire disorder (1,71).

Adjunctive Treatment

Another strategy for managing AISD is the addition of an adjunctive agent to therapy. This approach may benefit individuals for whom a particular antidepressant is highly effective in treating depression while helping to reduce unwanted sexual side effects. However, despite the evaluation of various substances, clinical evidence supporting their efficacy remains limited (50). Phosphodiesterase type 5 (PDE-5) inhibitors, including tadalafil, sildenafil, and vardenafil, appear to have the most well-documented efficacy in this indication. These medications work by preventing the breakdown of cyclic guanosine monophosphate (cGMP), leading to vasodilation and increased blood flow to the penis during sexual stimulation. They are used to treat erectile dysfunction in men, including cases induced by antidepressant therapy (50,72,73). Herbal therapy also provides a promising alternative. One potentially beneficial substance is yohimbine, which is extracted from the bark of Pausinystalia yohimbe. Yohimbine is an α2-adrenergic receptor antagonist that increases norepinephrine levels, leading to vasodilation and improved penile blood flow. Additionally, it influences central adrenergic and serotonergic receptors, regulating mechanisms related to libido and erection. However, yohimbine use requires caution due to potential adverse effects such as insomnia, tachycardia, and increased blood pressure, as well as the risk of drug interactions (74,75). Researchers from the Medical University of Vienna recently conducted a clinical study assessing pharmacokinetic interactions between clomipramine and yohimbine. The combination therapy was found to have a favorable safety profile and good tolerability, although increased plasma exposure to yohimbine was observed due to its metabolism being inhibited by clomipramine, likely involving cytochrome P450 2D6. These findings highlight the need for further research and suggest the potential for complementary mechanisms of action between both medications in the future (38). In recent years, saffron (Crocus sativus) has also been investigated for its potential benefits in treating sexual dysfunction, including antidepressant-induced dysfunction. Its complex mechanism of action involves neurotransmitter modulation, antioxidant properties, and improved blood flow. Evidence suggests saffron's effectiveness in alleviating fluoxetine-induced sexual dysfunction, with reported improvements in erection and sexual satisfaction in men, as well as enhanced arousal, lubrication, and reduced pain in women (76,77). Recently, a herbal supplement containing saffron, cinnamon, ginger, Tribulus terrestris, and milk thistle showed positive results in women experiencing sexual dysfunction associated with sertraline therapy (78). A novel and promising approach involves the use of probiotics. A study conducted among women with depression treated with SSRIs found that adding a probiotic containing bacterial strains such as Lactobacillus acidophilus, Lactobacillus reuteri, Bifidobacterium bifidum, and Lactobacillus fermentum led to improved sexual function, increased sexual satisfaction, and a reduction in depressive symptoms compared to monotherapy alone. The beneficial effect of these strains may result from their influence on the gut microbiota and modulation of the microbiome-gut-brain axis. Depression is associated not only with monoamine deficiency but also with chronic low-grade inflammation, which selected probiotic strains have the potential to reduce. In addition, they may enhance the integrity of the intestinal barrier and stimulate the production of neurotransmitters, which may contribute to mood improvement and indirectly exert a positive effect on sexual functioning (29).

Non-Pharmacological Treatment

Psychoeducation plays a crucial role in depression treatment and serves as an essential support in managing sexual dysfunction. Informing patients about the possibility of such side effects, educating them on coping strategies, and involving their partners in the treatment process significantly increase adherence to therapy while reducing anxiety and feelings of isolation (50). Sexological consultation and general psychological approaches, considered among the least invasive methods, can provide effective support for patients struggling with sexual dysfunction. One of the most commonly used forms of psychotherapy is cognitive-behavioral therapy (CBT), which in the treatment of sexual dysfunction focuses on altering negative thoughts and beliefs about sexuality while teaching emotional coping techniques. Another beneficial approach is couples therapy, aimed at improving communication between partners and collaboratively finding solutions to existing difficulties (79,80). Physical exercise is also considered to be helpful to alleviate sexual dysfunction. Regular aerobic training may improve erectile function in men, while engaging in physical activity just before sexual intercourse has been shown to enhance desire in women (81,82).

Recently, a study was conducted to evaluate the effects of acupressure on sexual function in women with depression who were taking SSRIs. This technique involves applying pressure to specific points on the body using the hands to relieve physical and psychological discomfort. The results showed significant improvements in orgasm, desire, and sexual satisfaction among women who underwent acupressure, suggesting that this method could serve as an effective complementary approach in managing such dysfunctions (83).

Conclusions

Sexual dysfunction is a common side effect of depression treatment, making it essential to address this issue during medical consultations. Many patients refrain from reporting such problems due to embarrassment, highlighting the need for a proactive and empathetic approach by healthcare providers in initiating discussions on this topic.

Personalised treatment, including switching to antidepressants with a more selective mechanism of action - such as vortioxetine, agomelatine, vilazodone, or bupropion - may reduce the risk of sexual dysfunction. Additionally, promising results have emerged from studies on adjunctive treatments, including natural compounds and probiotics; however, further research is needed to confirm their effectiveness.

It is crucial to recognise that side effects, particularly sexual dysfunction, often lead to patients discontinuing their medication, which in turn worsens depressive symptoms. Therefore, optimising therapy and educating patients about potential adverse effects and strategies for managing them should be a key component of psychiatric care.

Conflict of interest

The authors have declared no conflict of interest.

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