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SPECIAL ARTICLE

Brazilian Psychiatric Association guidelines for the treatment of social anxiety disorder

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Social anxiety disorder (SAD), one of the most prevalent anxiety disorders, is not well recognized. In most cases, SAD follows an unremitted and chronic course, affecting individual functioning in relationships, education, and work. Due to the disorder's relevance in Brazil, guideline-based treatments adapted to the Brazilian social and economic context are needed. We conducted a systematic review assessing several treatment modalities for SAD. PubMed, Cochrane, SciELO, and ClinicalTrials.gov were searched using the Medical Subject Headings social anxiety disorder and social phobia. Of the 438 selected articles, 20 were selected. Selective serotonin reuptake inhibitors are considered a first-line treatment for SAD due to their large effect size and database of evidence. Monoamine oxidase inhibitors, benzodiazepines, and the anticonvulsants pregabalin and gabapentin are also effective. Divergent results have been found for the serotonin-norepinephrine reuptake inhibitor venlafaxine. Among psychological interventions, robust data support cognitive behavioral therapy (whether individual, group, or remote) as a first-line option. Psychodynamic psychotherapy, exposure and social skills therapy, self-help therapies (with or without support), cognitive bias modification, virtual reality exposure therapy, and mindfulness-based therapy are also effective techniques. Psychological interventions are better tolerated and there is evidence that they provide better long-term benefits than pharmacological agents. Access to treatment (considering the Brazilian socioeconomic context), treatment adherence, short and long-term response rates, and side effects must be considered when choosing the best treatment strategy.

Keywords: Social phobia; social anxiety disorder; phobic disorders

Introduction

Social anxiety disorder (SAD) is characterized by intense and disproportionate fear or anxiety associated with one or more social situations, such as social interactions, being observed while eating or drinking in public, or performing in front of others.[1,2] Patients expresses concern about potential negative evaluations from others regarding their behavior, performance, or for displaying anxiousness.[1,2] Significant social gatherings are either evaded or endured with intense fear or anxiety. The symptoms must be sufficiently intense to result in significant distress or impairment in personal, family, social, educational, occupational, or other critical domains, and they must persist for a minimum of several months.[1,2]

The prevalence of SAD ranges from 0.5% to 12% in adults.[2-4] In the United States, the median age at onset is 13 years.[2] A functional impact analysis of adults with SAD found that 29.9% have serious impairment, 38.8% have moderate impairment, and 31.3% have mild impairment.[3,5] Women diagnosed with SAD have a higher prevalence of social fears, co-occurring major depressive disorder, and other anxiety disorders. In contrast, men are more prone to fear dating and are more likely to have comorbid oppositional defiant disorder, conduct disorder, or

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antisocial personality disorder and are more likely to resort to alcohol and illicit drugs to alleviate their symptoms. In adolescents, SAD has been reported to increase the risk of active suicidal thoughts and suicide attempts.[2]

Brazilians face a range of challenges in diagnosing and treating mental disorders. Some examples include initial recognition of mental health issues (such as SAD, which is often diagnosed late), limited and unequal access to mental health services, the limited capacity of non-specialized healthcare services to identify and refer such patients, a lack of integration among services, cultural and social factors that result in stigma and delay treatment seeking, as well as socioeconomic problems that restrict access to specialized treatment and care.[6,7]

Several treatment guidelines for SAD have been developed worldwide, such as the Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines,[8] England's National Institute for Health and Care Excellence guideline on the recognition, assessment and treatment of SAD,[4] and the Canadian clinical practice guidelines for the management of anxiety, posttraumatic stress, and obsessive-compulsive disorder.[9] The Brazilian Medical Association's treatment guidelines were published in 2010,[10] but they have not yet been updated. Thus, the purpose of this article is to present an updated guideline for both pharmacological and non-pharmacological treatment of SAD.

Methods

Eligibility criteria

This systematic review included the following types of studies: clinical trials, metanalyses, and systematic reviews that included clinical trials. Non-systematic reviews or government documents or other guidelines could be used if they contained essential information for answering the study questions. Case reports, series of case reports, and editorials were excluded. There were no restrictions on publication date or language.

Participants

Studies that included men or women diagnosed with SAD (social phobia or social anxiety disorder) according to the DSM-IV, DSM-5, ICD-10, or ICD-11 and who were treated with pharmacological and/or psychotherapeutic interventions were eligible. Information sources: PubMed, Cochrane, SciELO, and ClinicalTrials.gov.

Selection criteria (screening)

The following PubMed Medical Subject Headings were used as search keywords: Social Anxiety Disorder OR Social Phobia AND Treatment. The selection process was performed independently by two reviewers (TMA and DCS) using the Rayyan (https://www.rayyan.ai) selection platform (http://www.rayyan.ai). Duplicates were identified through the software. Abstract screening yielded 4,860 results, of which 438 were selected.

Data collection process (eligibility)

The selected articles were assessed for eligibility by TMA and DCS. After full text reading, articles that did not met the inclusion criteria and presented a high risk of bias for all items were excluded. In this phase, 245 articles were excluded and 20 were selected.

Data items (outcomes)

The data were organized according to the PICO strategy (Patient/Population of interest, Intervention/Exposure, Control/Comparison and Outcome). The main outcomes assessed in most studies were the effectiveness of interventions for reducing SAD symptomatology according to the appropriate scales. Secondary outcomes varied according to study methodology. The effectiveness of interventions was assessed mainly through the odds ratio (OR), risk ratio, and standardized mean differences (SMD).

Other data

A series of secondary measures were assessed in these articles, including improved Clinical Global Impression scores, improved comorbidities (such as depression and other anxiety disorders), all-cause dropouts, dropouts due to side effects, and other measures.

Risk of bias assessment

We used the Robvis tool for systematic reviews and metanalyses and the RoB 2 tool for randomized trials. We considered the corresponding level of evidence (Level 1 for systematic reviews or meta-analysis and Level 2 for randomized controlled trials [RCT]) only if the risk of bias was low. When the risk of bias was high or inconclusive, we downgraded the level of evidence by at least one point in the 2011 Oxford Classification.

Synthesis and evidence

At this stage, all authors read the relevant articles in their entirety and then conducted a critical analysis of the evidence, extracted the results, and categorized the strength of the evidence. The levels of evidence and recommendation grades were assigned according to the 2011 Oxford Classification. For further information, see https://www.cebm.net/wp-content/uploads/2014/06/CEBM-Levels-of-Evidence-2.1.pdf

Results

Supplementary Table S1 provides data about the articles included in the guidelines, organized according to the PICO system. Supplementary Table S2 shows the risk of bias results for systematic reviews with or without meta-analyses for pharmacological therapies. Supplementary Table S3 shows the risk of bias results for randomized clinical trials on pharmacological therapies. Figure 1 summarizes the guideline recommendations.

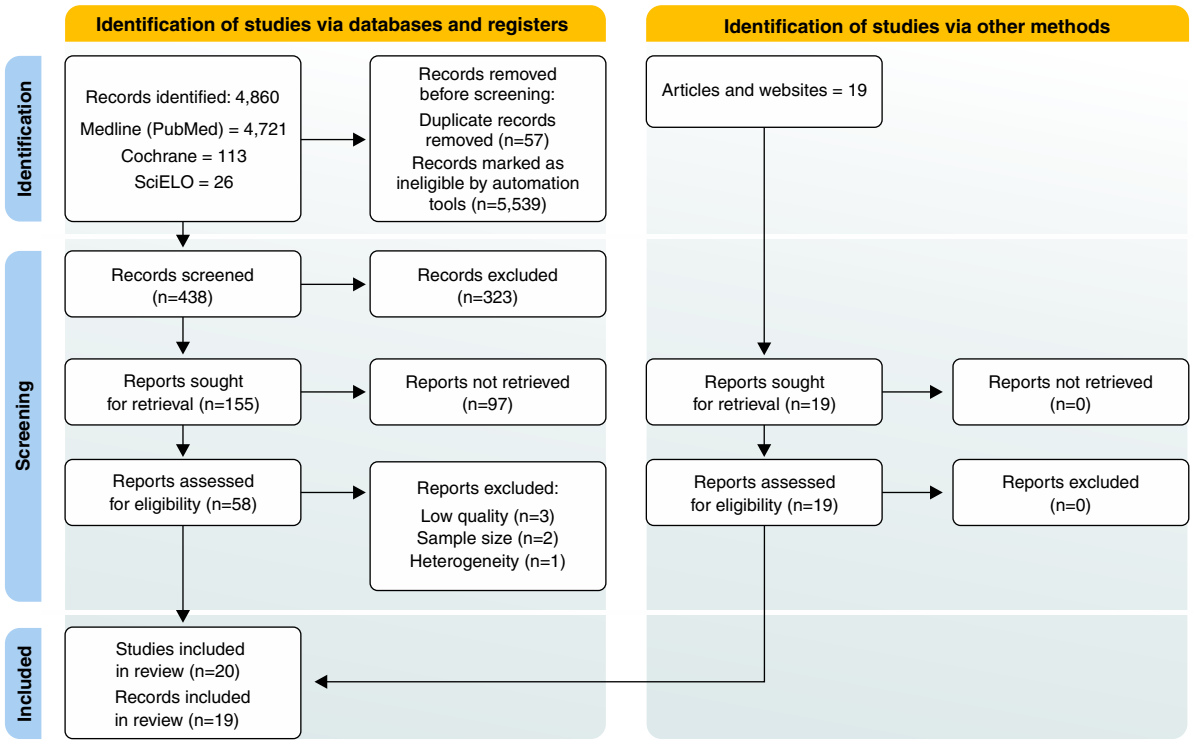

Figure 1 Flowchart. Page et al.[11]

Pharmacological treatment

Selective serotonin reuptake inhibitors (SSRIs)

SSRIs have been the most extensively tested drug class among individuals with SAD. Citalopram,[12] escitalopram,[13-16] fluoxetine,[12,15] fluvoxamine,[12,13,15-17] paroxetine,[12,13,15,16,18,19] and sertraline[12,13,15,16] have been tested and found efficacious for reducing symptoms according to measurement scales. However, in one study, escitalopram, fluvoxamine, paroxetine, and sertraline performed worse than placebo regarding dropout rates due to side effects.[16] Nevertheless, the level of evidence for this group is 1.

Serotonin-norepinephrine reuptake inhibitors

Venlafaxine is the only agent for which RCTs have fond positive results.[12,15,16,20] However, tolerance is not always satisfactory, and one study observed a higher dropout rate for venlafaxine than placebo (level of evidence: 2).[16]

Monoamine oxidase inhibitors (MAOIs)

Beside some evidence supporting their use in SAD,[12] the quality of the available data on irreversible MAOIs (such as phenelzine and tranylcypromine) and reversible inhibitors of MAO-A in SAD is rather low (level of evidence: 3 to 1).[16,20] Among reversible inhibitors of MAO-A, the main tested medications were moclobemide and brofaromine (level of evidence: 2 to 1).[12,15,16,20] Figure 2 suggests the order in which medications should be selected. Considering the side effects of this group, the age of relevant studies, and the fact that prescription requires strict dietary control, they should be a third-line treatment option.

Benzodiazepines

There is less evidence for alprazolam and clonazepam than for SSRIs and serotonin-norepinephrine reuptake inhibitors.[12] In one study, bromazepam and clonazepam had a better response than placebo.[16] Due to the risk of dependence and lack of studies on long-term treatment, the level of evidence was classified as 2.

Anticonvulsivants

Although antiepileptic drugs have been extensively reviewed for SAD,[15] only two have shown significant efficacy results.[15] Gabapentin and pregabalin are both ligands at the alpha-2 delta site on voltage-gated calcium channels, and both reduce the release of a range of excitatory neurotransmitters by binding to that site. Three RCTs have found positive treatment results for alpha-2 delta ligands.[15] The onset of anxiolytic effects is relatively rapid, occurring within the 1st week of treatment.[15] The dose response for anxiolytics has only been formally assessed for pregabalin, and efficacy was only evident at the maximum dose (600 mg/day).[15] This contrasts with its effects in other anxiety disorders, such as generalized anxiety disorder, in which a response occurred at much lower doses (150 mg/day).[15] There are no data on long-term treatment or relapse prevention for alpha-2 delta ligands (level of evidence: 3).[15] Valproate, topiramate, levetiracetam, and tiagabine have also been studied, and

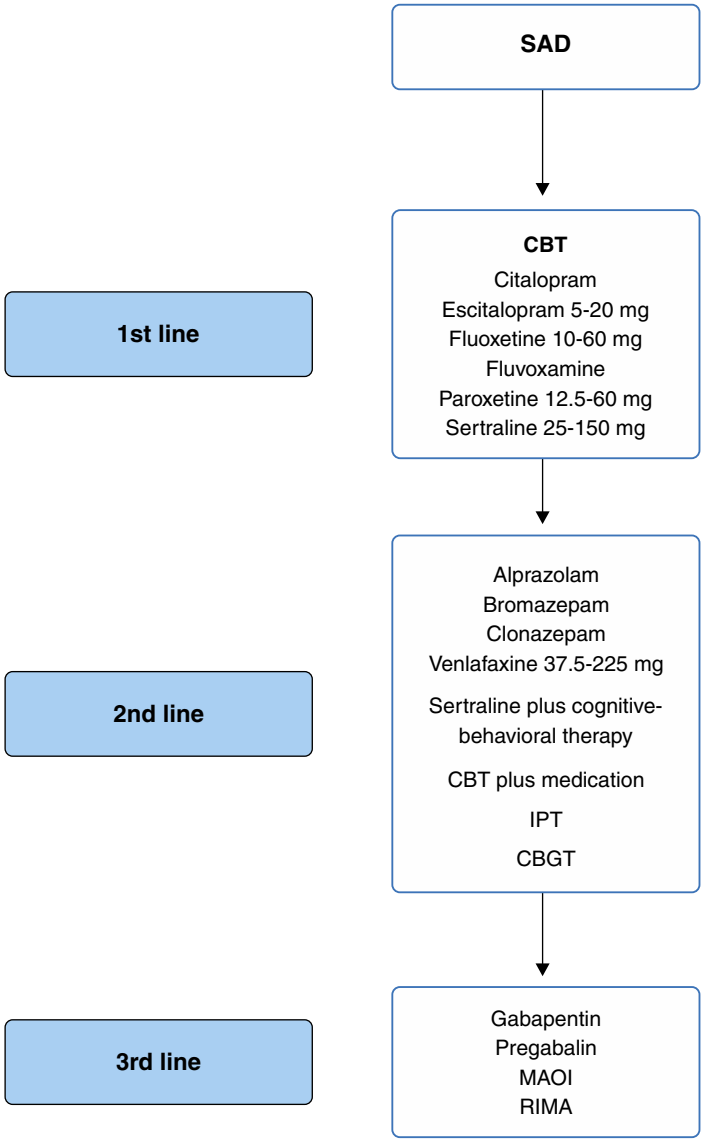

Figure 2 Guideline recommendations for the treatment of SAD. CBGT = cognitive behavioral group training; CBT = cognitive behavioral therapy; IPT = interpersonal therapy; MAOI = monoamine oxidase inhibitor; RIMA = reversible inhibitors of MAO-A; SAD = social anxiety disorder.

all were associated with reductions in relevant social phobia scales. However, the samples for all of these studies were small (ranging from 17-54 participants), and the magnitude of change in symptom ratings was within the range reported for placebo arms in other RCTs.[15,16]

Other medications

No current evidence supports the use of 5HT1A partial agonists (buspirone), olanzapine, quetiapine, beta-blockers, or norepinephrine reuptake inhibitors.

Duration of pharmacological treatment

The duration of SAD treatment can vary depending on the individual's needs and the psychiatrist's treatment plan. Some studies tested the medications for 6 to 28 weeks.[12,14,15,17,18,20-22] This guideline recommends 12 to 24 months of treatment.

Combined therapy (pharmacological and psychological)

Few studies have addressed the role of combined psychopharmacological interventions with psychotherapy. However, based on the overall findings of the present review, combined treatment is superior to isolated interventions.[12,13,15,22] In a meta-analysis aimed at identifying the best treatments for SAD, a combination of psychotherapy (cognitive behavioral therapy [CBT]/exposure) and psychopharmacology was superior to certain medication classes, including SSRIs (OR 0.83 [0.52, 1.33]), MAOIs (OR 0.46 [0.18, 1.18]), and benzodiazepines (OR 0.30 [0.09, 0.97]). Nevertheless, the sample size used in the combination analysis (230 participants) was small.[15]

Following the same trend, a meta-analysis on acute SAD treatment found that combined treatment was superior (SMD -1.30, 95%CI -1.73 to -0.88) to isolated interventions, such as CBT (SMD -1.19 [-1.56 to -0.81]), group CBT (SMD -0.92 [-1.33 to -0.51]), or psychopharmacology with SSRIs or serotonin-norepinephrine reuptake inhibitors (SMD -0.91 [-1.23 to -0.60]). Although the findings indicated a large effect size, the sample was very small (156 individuals).[12] This study found that combining group CBT with MAOIs or SSRIs (three studies), and combining psychodynamic therapy with benzodiazepines, SSRIs, or benzodiazepines (one study each) were favorable.[12]

Another meta-analysis with a more robust sample (1,020 patients) also found that combined treatment was superior (d = 2.15 [1.35-2.95]) to CBT alone (d = 1.10 [0.93-1.28]) or group CBT alone (d = 1.01 [0.72-1.29]).[12]

In an RCT with 102 participants, combined treatment with paroxetine and cognitive therapy resulted in significant improvement compared to placebo alone. It was also superior to medication alone and group cognitive therapy alone, but not significantly so.[19] However, in 12 months of follow-up, combined treatment did not remain superior to cognitive therapy alone; it was superior only to placebo and was accompanied by higher relapse rates.[19] A more recent RCT with 146 participants assessed combined treatment with psychotherapy and sertraline, comparing intervention groups with sertraline or placebo associated with group CBT or group psychodynamic therapy. It was found that combined treatment was superior to psychotherapy alone and that sertraline enhanced the development of social skills and improved specific SAD symptoms.[22]

Non-pharmacological treatment – psychotherapies

Several recent meta-analyses have aimed at identifying which psychological interventions are most satisfactory for SAD.[12,15,21,23-26] Among the evaluated psychotherapeutic approaches, CBT had better results than group CBT, psychodynamic therapy, or face-to-face therapies, and mindfulness had better effects than CBT.[23] However, it is important to emphasize that the authors, for the purpose of analysis, combined CBT and exposure in the same category. While exposure techniques can be part of a larger CBT treatment protocol, individual exposure is just one intervention in the CBT treatment protocol.

Another meta-analysis found that CBT had greater efficacy than group CBT, followed by exposure plus social skills training and a self-help group with support. Psychodynamic psychotherapy and mindfulness had smaller efficacy effects.[21,24] Another study compared CBT with a placebo psychotherapy group. Despite small to moderate effect sizes, the authors found that the OR of treatment response was 3.51 for CBT compared to placebo.[24] Similar results were found in previous meta-analyses involving placebo psychological comparison groups.[23]

In a more recent meta-analysis, group CBT was compared with waitlist, group psychotherapy, pharmacotherapy, and individual CBT. Group CBT had a larger effect than waitlist and a smaller effect than group psychotherapy or CBT, but it was not superior to medication alone. Group CBT helped relieve SAD symptoms but did not improve the general psychopathology outcome, which included symptoms of generalized anxiety and depression.[19,21,22]

Another important aspect to consider is the heterogeneity of the studies regarding treatment duration, the number of treatment sessions, and session duration, all of which have a considerable impact on the number of treatment hours, thus making it difficult to compare studies. The level of evidence was classified as 2.

Other important considerations include the persistence of treatment effect and the risk of post-treatment relapse. A meta-analysis of nine RCTs of CBT found significant effects in post-treatment (Cohen's d = 0.68 across all trials) that were maintained during follow-up, with no decrease in treatment effect size (0.76).[15] CBT was superior to medication during the maintenance phase because it helped protect against relapse.[15] Cognitive therapy was more effective during post-treatment and follow-up than paroxetine and was superior to combined treatment regarding Liebowitz Social Anxiety Scale results in 12 months of follow-up.[18]

CBT should be considered the best initial intervention for SAD due to its large effect sizes (SMD -1.19, 95%CI -1.56 to -0.81)[12] and its lower risk of adverse effects than pharmacotherapy. Among individuals who refused psychological interventions, SSRIs have shown more consistent evidence of benefit.[8] The effect of CBT was also greater than that of psychodynamic psychotherapy (SMD -0.56, 95%CI -1.03 to -0.11) interpersonal psychotherapy, MBI, or supportive therapy (SMD -0.82, 95%CI -1.41 to -0.24).[12] Other meta-analyses have also maintained the superiority of CBT (level of evidence: 1).[23,24,27]

There is strong evidence that CBT is an effective treatment for SAD, although a considerable number of patients do not benefit from it. Important heterogeneity was observed between studies in the meta-analyses, which differed regarding assessment instruments, treatment protocols, techniques, total treatment time (number of sessions), and session duration.[19,23,24,27] All of these aspects make it difficult to compare treatment outcomes across studies. Additional research with more standardized outcome measurements and treatment descriptions, as well as specific attention to longitudinal monitoring, is necessary to identify long-term treatment effects.

Based on the currently available evidence, CBT continues to be the psychological treatment of choice for SAD. However, considering the patient's preferences, the complexity of the condition, and the expertise of the professional responsible for the treatment, other psychotherapeutic approaches may be of benefit, despite less supportive evidence, such as psychodynamic psychotherapy and interpersonal psychotherapy. Moreover, since not all patients respond to CBT, other options should be considered.[12,13]

Duration of psychotherapeutic treatment

In clinical trials, CBT treatment can range from three to 28 weekly sessions, with an average duration of 12 weeks.

Each session varies from 60 minutes to 150 minutes depending on whether the intervention was CBT or group CBT. CBT for social anxiety often involves a structured program of sessions that focus on cognitive restructuring, exposure therapy, relaxation techniques, and social skills training.[19,23-25]

Other therapies

Mindfulness-based interventions (MBI)

MBI were tentatively included in this guideline since they comprise different intervention modalities and conceptual difficulties, as do relationship and self-help therapies. Because patients must engage in daily mindfulness exercises, the intervention's treatment effects are difficult to assess: they could be due to the intervention itself or simply to the passage of time. In a meta-analysis comparing MBI with waiting list, MBI had demonstrable effect on SAD symptoms (0.89 0.53-1.26), but its effect was smaller than active treatments, such as CBT or group CBT (-0.20, -0.42, -0.03). An additional analysis of the five single-arm trials found that MBIs had a medium effect on SAD symptoms (g = 0.48). Although MBIs may hold promise, further research is needed to determine whether they are effective.[13,28]

Cognitive bias modification

Cognitive bias modification, a novel experimental technique built on cognitive theories of SAD, aims to reduce negative cognitions and thereby diminish anxiety susceptibility and symptoms. Current findings broadly support cognitive theories of SAD that consider a bidirectional or mutually reinforcing relationship between symptoms and cognitions. However, the small therapeutic effect observed for cognitive bias modification indicates that more reliable and efficient interventions specifically tailored for SAD are needed.[25] Considering the available evidence, cognitive bias modification has limited efficacy for SAD treatment.[27]

Internet-delivered cognitive behavior therapy

Evidence regarding Internet-delivered CBT indicates that technology-assisted interventions for SAD hold potential.[27] An analysis of 21 trials showed significantly lower post-assessment SAD symptoms than passive control conditions (g = 0.84 and 0.82, respectively). Internet-delivered CBT had a small advantage over active control conditions (g = 0.38).[27]

Virtual reality exposure therapy (VRET)

It has been shown that VRET is an alternative means of developing social skills to reduce SAD symptoms. VRET enables patients to face feared social situations through immersion in simulated scenes, gradually mitigating difficulties, such as the costs involved in exposure to real situations, as well as the uncontrollability of real stimuli, which makes it difficult for therapists to control and grade the intensity of aversive stimuli.[27,29-31] As a treatment for SAD, VRET had significant effectiveness during post-intervention and longitudinal follow-up. However, its post-treatment results were similar to *in vivo* ET, although VRET's effect was lower than *in vivo* ET at later follow-up times. These interventions did not differ significantly regarding dropout rates. Despite the lack of significant differences between VRET and *in vivo* ET, the low cost and flexibility of VRET indicate that it holds promise for rehabilitation.[27,29-31]

Exercise

The concept of exercise as a treatment for mental disorders is still controversial. The few studies conducted specifically for SAD have methodological limitations. Furthermore, exercise data are sometimes presented for anxiety disorders at other times for anxiety alleviation. A meta-analysis concluded that exercise programs are a viable treatment option for anxiety. High-intensity exercise regimens have been found more effective than low-intensity regimens.[32] The results suggest that exercise programs may be useful in general practice. However, no significant outcome differences (rating scale scores) were found between patients diagnosed with anxiety disorders and patients with high anxiety. The authors concluded that they were limited by the small number of studies and the wide variation in exercise interventions.[32]

Discussion

This guideline has discussed evidence-based interventions for SAD. It is important to consider several aspects when evaluating patients, including differential diagnoses, psychiatric and medical history, comorbid conditions, and treatment adherence to formulate a correct diagnosis and implement an appropriate and effective treatment modality.

Regarding pharmacological interventions, antidepressants (mainly SSRIs) are the most studied agents in the literature, with robust data available. Several metanalyses have shown that paroxetine, fluoxetine, sertraline, fluvoxamine, and escitalopram are effective for SAD.[12-15,17,18,22]

Paroxetine appears to be more efficient than the other medications.[12,15,18,19] SSRIs tend to separate from placebo during the acute phase (after 4-6 weeks), and higher doses may be necessary to achieve remission.[20] There is little data available on long-term treatment due to the methodological and financial difficulties of conducting an appropriate RCT. However, SSRIs have been linked to lower relapse rates after 24 weeks of treatment.[12,15,18-20] Side effects are prevalent, mainly at the beginning of treatment, and tend to decrease after the 1st month. The most common side effects are nausea, insomnia, sexual dysfunction, irritability, headache, and diarrhea. Moreover, discontinuation symptoms may be relevant, mostly in patients who abruptly stop the medication and in agents with a short half-life (i.e., paroxetine).[12,15,18-20]

Venlafaxine is the only serotonin and noradrenaline reuptake inhibitor to have been assessed in RCTs, and there are conflicting results about its effectiveness. While some metanalyses=13,15,20] have supported

venlafaxine, reporting an expressive response and improved anxiety symptoms among patients with SAD, a systematic review and metanalysis[16] concluded that venlafaxine was ineffective for SAD. Its poor performance may be related to the number of dropouts (due to side effects) or to bias and methodological heterogeneity among the included RCTs. Frequent side effects related to venlafaxine and other serotonin-norepinephrine reuptake inhibitors include nausea, insomnia, irritability, diarrhea, increased blood pressure, sexual dysfunction, and autonomic symptoms.[16] The withdrawal symptoms of venlafaxine are among the most prevalent of any antidepressant.[16]

MAOIs are effective for SAD, with previous systematic reviews and metanalyses reporting large effect sizes.[12,13,15] One study found that the evidence was of low quality for phenelzine[19] and inconclusive for moclobemide.[16] However, due their poor tolerability, the involved dietary restrictions (i.e., the risk of hypertensive crisis if tyramine is ingested, which is associated with the blockage of monoamine oxidase), and their potentially harmful interactions with serotonin/noradrenaline agents, adherence may be limited.[33,34] Considering the side effects of this group of medications, the age of relevant studies, and the strict dietary control requirements, we classify them as a third-line treatment option.

Benzodiazepines, largely used in anxiety disorders,[35] are supported by most data. Systematic review and metanalyses[12,16,20] have found them to be effective for SAD, with large effect sizes and high response rates. Moreover, these drugs appear to decrease the incidence of relapse after the acute treatment phase.[12,16,20] However, the long-term side effect profile should be assessed due to the risk of tolerance, dependence, abstinence, and cognitive impairment from chronic use.[35,36]

The anticonvulsants pregabalin and gabapentin are associated with improved SAD symptoms, while in other studies levetiracetam, gabapentin, and pregabalin showed limited effectiveness[12,13,15,16] However, the data on these agents are limited and further studies are needed for more robust evidence. Prevalent side effects associated with anticonvulsants include sedation (especially at higher doses), dizziness, dry mouth, confusion, and blurred vision.[37]

According to the available evidence, buspirone, norepinephrine reuptake inhibitors, beta-blockers, and mirtazapine are not associated with improvements in SAD and should not be used to treat it.[16,20,38,39] Second-generation antipsychotics (olanzapine, risperidone, and quetiapine) have also been found ineffective for SAD.[16,20] Moreover, when prescribing antipsychotics it is important to consider the risk of side effects, such as metabolic syndrome, weight gain, sedation, and extrapyramidal symptoms.[38]

Regarding psychotherapy, robust data support CBT as a treatment for SAD. Clark and Wells' model appears to be the most effective method,[19,40] with individual CBT showing better results than group CBT.[12,24] Even so, group CBT is regarded as effective[23,24] and should be considered an option when available. To adapt to the new paradigm of appointments and personal relationships that began during the COVID-19 pandemic, Internet-delivered CBT can also able considered an effective treatment option.[27] Thus, we recommend training for physicians and psychologists in this technique, specifically for anxiety disorders.

MBI has become popular in the recent years, and its efficacy has been assessed for several mental health disorders. While Mayo-Wilson et al.[12] found no evidence in favor of MBI as a treatment for SAD, a later meta-analysis of 11 studies[28] found gains in pre-post effect and comorbid symptoms that continued during follow-up. Compared to evidence-based treatment (i.e., CBT), MBI was less effective, indicating that further studies are required for better characterization of its role in SAD treatment.

Some evidence has been found for other modalities, such as psychodynamic psychotherapy, exposure and social skills therapy, and self-help groups (with and without support).[12] Evidence suggests that VRET can help manage the core symptoms of SAD,[27,29-31] although compared to active control, VRET appeared less effective during follow-up. Because few studies have assessed combinations of medication and psychotherapy, the available evidence is limited.[13,15,19] Further research on the potential benefits of such combinations is needed, and practitioners can discuss the idea with patients. However, in our opinion, no strong evidence supports combined therapy for SAD.

In conclusion, SAD is a prevalent disorder that greatly affects quality of life and follows a chronic and unremitting course if untreated. The scope of this review was to offer evidence-based options for the pharmacological and non-pharmacological management of SAD. Based on the literature, SSRIs should be considered first-line agents. Moreover, benzodiazepines, MAOIs, and the anticonvulsants pregabalin and gabapentin should be regarded as effective alternatives. Although the results for venlafaxine are conflicting, it remains an option, especially for patients who do not respond to SSRIs. Regarding psychological interventions, robust evidence indicates that CBT (whether individual, group, or Internet-delivered) is a first-line treatment option. Psychodynamic psychotherapy, exposure and social skills therapy, self-help groups (with and without support), and VRET are also effective techniques. Additional data are needed regarding mindfulness, although it appears to be a promising intervention for SAD. Although psychological interventions have fewer side effects than pharmacological agents, there is less evidence about their long-term efficacy. Considering Brazil's context, especially the limited access to treatment (often provided in the primary care system), choosing the most appropriate treatment regarding response rate, adverse effect profile, and adherence is of vital importance. To reduce heterogeneity among studies, we recommended considering various aspects, such as the expertise of the physician who prescribes treatment and that of the professional who applies it (especially psychotherapy), access to and the possibility of completing therapy, and potential complications. When there is no treatment response or the selected treatment option cannot be applied, we advise following the other recommendations outlined in Figure 2.

Disclosure

The authors report no conflicts of interest.

Author contributions

LB: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Project administration, Resources, Software, Supervision, Validation, Visualization, Writing – original draft, Writing – review & editing.
TMA: Data curation, Formal analysis, Investigation, Methodology, Project administration, Resources, Software, Validation, Visualization, Writing – original draft, Writing – review & editing.
DCS: Data curation, Formal analysis, Investigation, Methodology, Project administration, Resources, Software, Validation, Visualization, Writing – original draft, Writing – review & editing.
ABP: Data curation, Formal analysis, Investigation, Validation, Visualization, Writing – original draft, Writing – review & editing.
AFP: Data curation, Formal analysis, Investigation, Validation, Visualization, Writing – original draft, Writing – review & editing.
LAVGA: Data curation, Formal analysis, Investigation, Validation, Visualization, Writing – original draft, Writing – review & editing.
DLV: Data curation, Formal analysis, Investigation, Validation, Visualization, Writing – original draft, Writing – review & editing.
FFL: Data curation, Formal analysis, Investigation, Validation, Visualization, Writing – original draft, Writing – review & editing.
LFMD: Supervision, Validation, Visualization, Writing – original draft, Writing – review & editing.
RLO: Validation, Visualization, Writing – original draft, Writing – review & editing.
QC: Supervision, Validation, Visualization, Writing – original draft, Writing – review & editing.
MS: Supervision, Validation, Visualization, Writing – original draft, Writing – review & editing.
AEN: Supervision, Validation, Visualization, Writing – original draft, Writing – review & editing.
AGS: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Project administration, Resources, Software, Supervision, Validation, Visualization, Writing – original draft, Writing – review & editing.
RRU: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Project administration, Resources, Software, Supervision, Validation, Visualization, Writing – original draft, Writing – review & editing.
All authors have read and approved of the final version to be published.

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