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Social Anxiety Disorder

Nesrin Dilbaz, Aslı Enez and Serçin Yalçın Çavuş
Ankara Numune Research and Training Hospital
Turkey

1. Introduction

1.1 Epidemiology of social anxiety disorder (SAD)

SAD, also known as social phobia, is characterised by excessive fear of embarrassment or humiliation in social situations, which in turn leads to marked distress or avoidance of these situations and functional impairment as described in DSM-IV-TR.

It is a common disorder with early onset, significant comorbidity and functional impairment (Meron Ruscia et al., 2008).

SAD has been ranked as one of the top ten chronic disorders – mental or physical – in terms of its effects on objective outcomes, such as days of work lost and reduced health-related quality of life (Alonso, 2004). According to the National Comorbidity Survey, SAD is the most reported anxiety disorder and has a lifetime prevalence of 12% (Kessler et al., 2005), with considerable coexisting psychiatric disorders, such as depression, anxiety, and substance-related disorders. Lifetime prevalence of social anxiety disorder among Turkish university students was 23% (Dilbaz 2006). Co-occurring SAD and depression carry a substantial risk of suicide, which further complicates treatment (Beesdo et al., 2007; Thase, 2007). SAD symptoms normally emerge during early adolescence and continue throughout adulthood; they affect women more often than men (Fehm et al., 2005). In clinical samples the ratio of male and female changes in the favour of males. (Dilbaz and Güz 2002). Trials suggest that social anxiety even below the diagnostic threshold is clearly associated with adverse outcomes like elevated risk for comorbid disorders and associated with impairment in diverse areas of life.

Despite the growing understanding of this condition, information is lacking on key aspects of the disorder and many individuals, including doctors, psychiatrists and patients, unaware about this condition.

1.2 Children and adolescents

SAD, which so often begins in childhood, precedes other comorbid disorders and may be a direct or indirect risk factor for other disorders, such as depression and substance abuse. Epidemiologic findings show that in the pediatric primary healthcare setting anxiety disorders are very common ranging from 1% to 10% (Briggs-Gowan et al., 2000; Busch et al., 2002; Costello, 1989), but are unrecognized and under-treated (Chavira et al., 2004; Wren et al., 2003). In community settings, rates of SAD in youth range from 0.5% to 4% (Essau et al., 1999; Wittchen et al., 1998) and from 3% to 6.8% in primary care settings (Busch et al., 2002; Costello, 1989; Chavira et al., 2004). Recent research suggest that lifetime prevalence rates in adolescents in the US and Germany are between 5% and 15% (Heimberg et al., 2000;

Lewinsohn et al., 1993). Although the age of onset is usually in the early teens with a mean age of onset of 15.5 years (Schneider et al., 1993) children as young as 8 years old have been diagnosed with the disorder (Beidel & Turner, 1998).

Shyness, behavioral inhibition and selective mutism can be considered in spectrum of social anxiety disorder in childhood. Children who are rated by their parents as having a shy temperament in infancy or in early childhood had an approximately 2 or 3 times increased probability of having an anxiety disorder in adolescence (Prior et al, 2000). In a five year longitudinal study, researchers found that behavioral inhibition-which can be described as a tendency to demonstrate fearfulness or resistance when faced with an unfamiliar stimuli, in pre-school children appeared to be a predictor for social anxiety in middle school (Hirshfeld-Becker et al., 2007). Selective mutism can be considered an extreme form of social anxiety including features of shyness and behavioral inhibition, where the most prominent feature is the inhibition of speech in select situations. Comorbidity rates between selective mutism and social anxiety disorder range from 70–95% (Dummit et al., 1997; Black & Uhde, 1995) and characteristics such as shyness, anxiousness, withdrawal and seriousness are used to describe both selective mutism and social anxiety alike (Steinhausen & Juzi, 1996; Kumpulainen et al., 1998).

Studies have shown that in children aged 7–13 years with SAD, 60% had an additional psychiatric diagnosis, of whom 36% had an anxiety disorder as follows: generalized anxiety disorder 10%; attention deficit hyperactivity disorder 10%; specific phobia 10%; and selective mutism 8% (Biedel et al., 1999). Those individuals who develop comorbid disorders will also have an increased risk of suicidal ideation and suicide attempts.

In treatment of social anxiety disorders in childhood and adolescence, most practitioners advise the initial use of psychological interventions followed by pharmacotherapy when necessary. Specific treatments have employed cognitive-behavioral group therapy for SAD in adolescents, social effectiveness therapy for children (Beidel et al., 2000) and 'coping cat' child behavior therapy (Flannery-Schroeder et al., 2000). Overall, most clinical researchers now believe that CBT is the treatment of choice for youth with internalizing disorders including SAD (March et al., 2003).

There are very few pharmacotherapy trials in general and even fewer randomized, double-blind, placebo-controlled trials in childhood anxiety disorders. Studies with both open-label and double blind placebo control groups have shown promising results ranging from 36–100% success rates (Compton et al., 2001; Mancini et al., 1999). Selective serotonin reuptake inhibitors (SSRI) and serotonin norepinephrine reuptake inhibitors (SNRI) are defined as first line pharmacotherapy for social anxiety disorders in youth with a careful assessment of suicidal ideation before starting these antidepressants.

The development of comorbid mental disorders such as depression and substance abuse, children and adolescents with this illness are at risk for educational or occupational underachievement, and failure to achieve financial and emotional independence. The challenge of preventing the consequences of SAD lies in early diagnosis.

2. Organic etiology of SAD

Although the etiology of SAD is poorly understood, emerging evidence indicates multidimensional causes. It is a distinct psychiatric disorder with genetic underpinnings and is associated with neurobiological and environmental mechanisms.

Over the past two decades, numerous neurobiological methods have been used in studies of SAD including structural, functional and receptor brain imaging, pharmacological trials, candidate gene investigations and studies of psychophysiological, endocrine, biochemical and behavioral responses to stressful challenges. It has been hypothesized that affect regulation is compromised in individuals with SAD, either due to hyperactivity in emotion triggering areas like the amygdala and insula, or hypoactivity in modulatory regions like the anterior cingulate and prefrontal cortices. It also has been hypothesized that SAD patients would show exaggerated amygdala responses to angry or threatening faces in comparison to healthy control subjects and that amygdala hyperresponsivity is associated with enhanced fear conditionability in SAD (Furmark, 2009).

A range of neurotransmitters may be important in SAD including the monoamines, glutamate, GABA, and several neuropeptides, but to date, the serotonergic and dopaminergic transmission systems have received most of the attention.

Serotonin has been implicated in animal models of fear and anxiety (Graeff, 2002) and the therapeutic efficacy of SSRIs (Ipser et al., 2008) strongly suggests that serotonin has a crucial role in SAD. Allelic variation in serotonin-related genes modulate amygdala responsivity both in healthy volunteers (Hariri & Holmes, 2006) and in patients with SAD (Furmark et al., 2004). Lanzenberger et al. demonstrated a significantly lower serotonin-1A receptor binding potential in SAD patients relative to controls in the amygdala, anterior cingulate cortex, insula, and dorsal raphe nuclei in their PET study (Lanzenberger et al., 2007). Serotonergic involvement is also supported by neuroendocrine challenges studies (Tancer, 1993).

Dopamine is known to play a central role in motivation and reward-seeking behaviors and several lines of evidence point to a dysfunction of this transmitter system in SAD like patients with Parkinson's Disease, which is associated with dopamine hypofunction, appear to have enhanced risk for developing SAD (Richard et al., 1996). Abnormal central dopaminergic neurotransmission has also been reported in animal trials relevant to SAD, such as studies of social subordination in primates (Grant et al., 1998). Two independent SPECT studies also point directly to an altered dopamine system activity in SAD. Tiihonen et al. reported that the striatal dopamine reuptake site density was markedly lower in patients with SAD than controls, presumably reflecting a smaller number of dopaminergic synapses and neurons in the basal ganglia (Tiihonen et al., 1997). Schneier et al. also observed that striatal dopamine D2 receptor binding was significantly lower in subjects with SAD than in comparison subjects (Schneier et al., 2000).

Genetically-oriented studies of SAD and related constructs such as behavioral inhibition, neuroticism, introversion and harm-avoidance suggest that genetic factors play at least a moderate role in the etiology of excessive social anxiety (Stein et al., 1994). The short (s) allele of the promoter polymorphism of the human serotonin transporter gene (the 5-HTT-linked polymorphic region; 5-HTTLPR) has been associated with anxiety-related personality traits, increased fear conditionability, and life-stress-induced affective disorder (Serretti et al., 2006). Several other genotypes influence amygdala responsivity and could thus be considered in future studies of SAD, for example the tryptophan hydroxylase-2 gene (G-703T polymorphism) (Brown et al., 2005) and the catechol-O-methyltransferase gene (COMT Val158Met) (Smolka et al., 2005).

3. Diagnosis and assessment of SAD

The first challenge in the treatment of SAD is making a correct diagnosis. There is evidence that SAD is under-diagnosed and under-treated in primary care and specialist settings alike

(Katzelnick & Greist, 2001). Possible reasons for this may be lack of diagnostic awareness, lack of diagnostic threshold clarity or the presence of co-morbid disorders. Moreover, as consultation with a clinician may be perceived as social interaction, the nature of the disorder may cause patients to delay seeking help, and when they eventually do, it is often with physical complaints or psychiatric comorbidity.

During the initial evaluation it is important to find out severity of symptoms as well as the degree of avoidance and functional impairment present. Rating scales such as the Liebowitz social anxiety scale that is translated and validated to Turkish also, may be helpful in assessing both feared situations and avoidance and can be used to monitor the patient's treatment progress (Liebowitz 1987, Dilbaz and Guz). A range of other SAD scales are also available, like the Brief Social Phobia Scale (clinician rated) and the Social Phobia Inventory (patient rated) (Davidson et al., 1997; Conner et al., 2000).

4. Co-morbidity

Significant comorbidity of social anxiety and mood disorders have been consistently shown in the literature (Kessler et al., 1999; Lecrubier & Weiller, 1997; Pini et al. 1997). Social anxiety has also been linked to severity (Merikangas & Angst, 1995) and persistence (Alpert et al. 1997) of mood disorders. Also comorbid mood disorders have worsen social anxiety symptoms and result in greater impairment in patients with SAD (Erwin et al., 2002). A significant number of patients presenting with social anxiety will have a secondary anxiety disorder, particularly panic disorder with agoraphobia and generalized anxiety disorder (Mennin et al. 2000, Schneier et al., 1992). Comorbid depressive or anxiety disorders complicate SAD, the severity of symptoms and suicidality should be assessed and hospitalisation considered if indicated. In such cases, the treatment of choice should ideally target both the mood and the anxiety components.

Social phobia and avoidant personality disorder were introduced in the DSM classification system nearly 30 years ago. Since then it has been shown that these two disorders can be found highly comorbid in patients, in some trials as high as 89% (Schneier et al., 1991). Some researchers have interpreted this high rate of overlap to mean that social phobia and avoidant personality disorder reflect a spectrum of social anxiety (Tillfors & Ekselius, 2009).

Social anxiety disorder is associated with high rates of alcohol use disorders (Morris et al., 2005). Almost half of the patients with lifetime SAD meet criteria for lifetime prevalence of an alcohol use disorder (Grant et al., 2005) and it is a significantly high rate when compared to general population. Among the anxiety disorders, SAD shows a particularly problematic risk profile for comorbid alcohol use disorders, as SAD is associated with higher rates of alcohol use disorders relative to most other anxiety disorders (Kessler et al., 1997).

Recently, researchers have identified that individuals with SAD appear particularly vulnerable to marijuana-related problems too. Data from the National Comorbidity Study suggest that individuals with SAD are 7 times more likely to experience marijuana dependence relative to the general population (Agosti et al, 2002) and undergraduates with higher social anxiety appear to be particularly vulnerable to marijuana use problems (Buckner et al, 2007; Buckner et al., 2008a). In a study, adolescents with SAD were nearly 5 times more likely to develop marijuana dependence as young adults compared to adolescents without SAD (Buckner et al., 2008b).

5. Treatment of SAD

Current recommended treatment options for social anxiety disorder include pharmacotherapy and cognitive behavioural therapy (CBT). (Dilbaz 2005) Although several randomised controlled trials (RCTs) have failed to show efficacy for β -adrenoceptor antagonists in generalised SAD, it has been suggested that these agents may be useful in non-generalised SAD, patients with performance anxiety only. Efficacy in the treatment of generalised SAD has been demonstrated for a number of interventions, (Stein, 2003; Blanco et al., 2003) including SSRIs, high potency benzodiazepines (e.g. clonazepam), MAOIs (e.g. phenelzine), reversible inhibitors of monoamine oxidase A (MAO-A) [RIMAs, e.g. moclobemide], certain antiepileptics (e.g. gabapentin, pregabalin), serotonin-noradrenaline reuptake inhibitors (venlafaxine) and CBT (Zaider & Heimberg 2003).

5.1 Nonpharmacological treatments

These treatments include exposure, cognitive re-structuring, relaxation techniques and social skills training, often used in combination. Literature have concluded that cognitive behavioral therapy (CBT) is often effective for treating social anxiety disorder. The goal of cognitive behavioral therapy (CBT) is to provide techniques and practice to patients with social anxiety disorder, so they can learn to change how they think about and behave in situations that terrify them. CBT may be offered individually or as part of group therapy. There is continued interest in the question of whether group or individual treatment is more effective. Early studies suggested some superiority for group treatment (Wlazlo et al., 1990), and arguments were raised that the group setting would provide a richness of exposure experiences not easily replicated in individual treatment; by the late 1990s the weight of evidence suggested that there was no clear superiority between group and individual treatment (Gould et al., 1997).

Meta-analyses of the efficacy of CBT for SAD that have compared various types of CBT with each other and with control conditions have yielded the highest effect sizes for exposure-based interventions (Federof & Taylor, 2001; Hope et al., 1995). In exposure therapy, the type of CBT most often used and studied for social anxiety disorder, therapists gradually expose patients to the dreaded situation and suggest ways to manage fear. The exposure-based extinction of fear is now thought to involve new learning that actively inhibits the fear reaction to a given cue (Davis et al., 2006). In other variations of CBT – not as well studied as exposure therapy – patients learn and practice social skills and relaxation techniques.

5.2 Pharmacological treatments

SSRIs; evidence from RCTs supports that the efficacy and tolerability of almost all SSRIs (escitalopram (Stein et al., 2005), fluvoxamine, (Van Vliet et al., 1994; Stein et al., 1999) paroxetine, (Liebowitz et al., 2002; Baldwin et al., 1999; Stein et al., 1998) sertraline (Katzelnick et al., 1995; Liebowitz et al., 2003), fluoxetine (Black et al., 1992), citalopram (Bouwer & Stein, 1998) in the treatment of SAD, so SSRIs can be regarded as first-line treatment in SAD. These agents have the additional benefit of treating comorbid conditions commonly seen with SAD (Van Ameringen et al., 2004). Fluoxetine, fluvoxamine and sertraline have been the most studied SSRIs in socially phobic children and adolescents and have shown good efficacy and tolerability in this group (Robinson & Hood, 2007).

Venlafaxine; has shown promising results in open-label and controlled trials in the treatment of SAD. Venlafaxine is an effective treatment option for generalised social anxiety

disorder but has no superiority from paroxetine in clinical trials (Allgulander et al., 2004; Liebowitz et al., 2005). While venlafaxine is an effective treatment, this may be related to its serotonergic profile, and the authors are unimpressed with response to the specific noradrenergic agent reboxetine from both the literature and clinical experience. Venlafaxine is associated with significant side effects and discontinuation syndrome and described as second-line treatments of social anxiety in most settings (Robinson & Hood, 2007).

MAOIs; Phenelzine was one of the first established treatments for SAD with several early double-blind, placebo-controlled trials demonstrating efficacy in this disorder (Versiani et al., 1992; Gelernter et al., 1991). In light of their adverse effect profiles and the dietary restrictions associated with use of these agents, together with the availability of alternative treatments, MAOIs are currently not considered to be a first-line treatment for SAD.

RIMAs; reversible inhibitors of monoamine oxidase A, appears inferior to that of phenelzine in efficacy but the main advantages of moclobemide over phenelzine are superior tolerability and no concern about dietary restrictions at the standard dosage of 600 mg/day moclobemide. This may be a particularly important consideration in the long-term treatment of SAD (Stein et al., 2002).

Benzodizepines, despite the positive results, the adverse effect profile, the potential for dependence, the possibility of rebound anxiety, the possible negative consequences of state-dependent learning and their ineffectiveness in the treatment of depression shows that these agents should not be considered as first-line monotherapy for SAD (Jackson 1995; Keith et al., 2003).

Beta Blockers; Beta-blockers are effective at blocking peripheral autonomic symptoms such as tachycardia, tremor, sweating, blushing and dry mouth and thus have a potential role as anxiolytics. They are effectively used in the treatment of performance anxiety but there is substantial evidence that beta-blockers are not effective in social phobia (Liebowitz et al., 1992; Turner et al., 1994) and that better options are available. A limited role in performance anxiety is indicated.

Gabapentine, Pregabalin and Levetiracetam; Some anticonvulsants trials suggest that gabapentin, pregabalin and levetiracetam could be alternative agents for patients who are nonresponsive to SSRIs and SNRIs (Pande et al., 1999; Pande et al., 2004; Zhang et al., 2005). However these agents need further studies about the safety and efficacy in SAD.

Other pharmacological agents for treatment of SAD; *Tricyclic antidepressants* have been used but failed to be an effective treatment choice for social anxiety disorder (Emmanuel et al., 1997; Simpson et al., 1998). There are trials with *buspirone* in literature but these trials have both negative and positive outcomes in social anxiety disorder. So that buspirone may be a useful agent for augmentation in social anxiety disorder (Robinson & Hood, 2007). There is a little evidence for *bupropion* efficacy in social phobia (Emmanuel et al., 2000). There are trials that have found *mirtazapine* as an effective treatment option for social anxiety disorder (Mrakotsky et al., 2007; Mörtberg, 2006). However in a recent study mirtazapine showed no superiority to placebo for treatment of SAD (Shutters et al., 2010).

Atypical antipsychotics have been shown to have anxiolytic properties in the literature (Depping et al., 2010; Vulink et al., 2011). Olanzapine (Barnett et al., 2002) and quetiapine (Vaishavi et al., 2007) have been found effective as monotherapy in the treatment of SAD. In another study switchover to aripiprazole effectively improved social anxiety in patients with schizophrenia (Stern et al., 2009).

5.3 Inadequate response to pharmacotherapy and augmentation strategies

Criteria for remission of SAD proposed by Ballenger (Ballenger, 2001) include absence of core symptoms of SAD, no or minimal anxiety (e.g. in anticipation of social interaction) as rated by rating scales such as the Hamilton Rating Scale for Anxiety, no functional impairment (the Sheehan Disability Scale may be used to evaluate this), (Sheehan et al., 1996) and remission of course of action when first-line treatment with a co-morbid depression as reflected by the Hamilton Rating Scale for Depression. It should, however, be noted that the mean reduction in Liebowitz Social Anxiety Scale scores was <50% in a review of 19 double-blind, placebo-controlled trials involving patients with SAD. Switching to another SSRIs, venlafaxine or MAOIs has been suggested in non responders or patients with adverse event. Augmentation strategies that may prove useful in partial treatment responders include buspirone, clonazepam, gabapentin, bupropion or new generation antipsychotics, although empirical data are lacking (Barnett et al., 2002; Schutters et al., 2005; Pallanti et al., 1999).

In case of comorbidity moclobemide was found to be effective and well-tolerated in the SAD patients with anxiety disorders as well as SSRIs. In the presence of co-morbid alcohol abuse, MAOIs and benzodiazepines can complicate the treatment, SSRIs have usefulness in reducing the alcohol consumption (Naranjo & Knoke, 2001). CBT may be the treatment of choice during pregnancy and lactation.

6. Conclusion

SAD is a prevalent and disabling disorder that often remains undetected unless the clinician takes a careful history. Present consensus supports that SSRIs can currently be regarded as first-line treatment in SAD because of their proven efficacy, tolerability and ability to treat co-morbid conditions such as depression or other anxiety disorders (Stein, 2003; Blanco et al., 2003; Ballenger et al., 1998). There is a recent evidence venlafaxine XR may also be considered in the first-line. Second-line treatments include MAOIs (e.g. phenelzine) and RIMAs (e.g. moclobemide).

The combination of CBT and an SSRI is often espoused as best practice, unfortunately there is little hard evidence supporting this (despite considerable face validity). Future studies taking a good look at combination therapies of this type are encouraged and also future research should focus on complicated and treatment refractory SAD and treatment strategies in special populations (e.g. children and adolescents).

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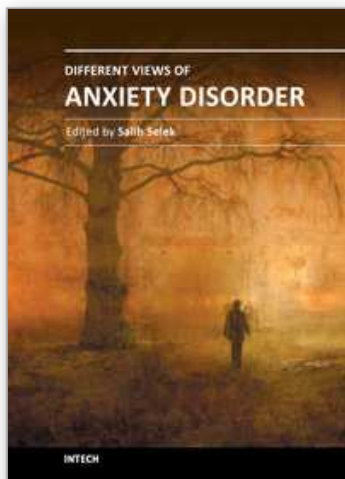
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