

Cognitive Behavioral Group Therapy in Panic Disorder Patients: The Efficacy of CBGT versus Drug Treatment

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The aim of our study was to evaluate the effectiveness of Cognitive Behavioral Group Therapy (CBGT) in the treatment of Panic Disorder (PD) and to compare the treatment outcome of CBGT versus Paroxetine pharmacotherapy. Fifty seven patients referred to our anxiety disorder clinic for the treatment of PD were randomly allocated to receive either CBGT or Paroxetine. Follow up was done by a masked rater after four and twelve weeks of treatment in order to compare the efficacy of CBGT versus Paroxetine. CBGT and Paroxetine were both effective in the short term treatment of PD. Assessments at weeks four and twelve of treatment showed no statistically significant differences between the two groups in terms of treatment outcome. Treatment with CBGT alone for the acute phase of PD appears to be equally efficacious to treatment with Paroxetine alone. Our study shows that CBGT produced beneficial results, for it was associated with a reduction in the number and frequency of panic attacks and with an improved feeling of well-being.

Keywords Panic Disorder; Cognitive Behavioral Group Therapy; Treatment outcome.

INTRODUCTION

Panic Disorder (PD) is a frequent psychiatric illness with a lifetime prevalence of 3.5% and a 12-month prevalence of 2–3% (19). It is a potentially debilitating condition and is associated with frequent visits to the primary health care provider and to the Emergency Department as well as decreased work productivity especially during the period before referral to psychiatric treatment and during the initial stages of psychiatric intervention (15). In addition, PD is associated with an increased risk of suicidal behavior and comorbid psychiatric diagnoses such as depression and alcohol abuse (31).

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The currently recommended treatment approaches for PD include the use of antidepressant pharmacotherapy and/or Cognitive Behavioral Therapy (CBT) (3–5). CBT for the treatment of PD represents an important alternative to pharmacotherapy because 1) a subset of patients are either unable to tolerate drug treatment or suffer from unwanted side-effects, 2) many patients may refuse to take medication or may be pharmacotherapy nonresponders or partial responders (26), 3) patients who receive only drug therapy may relapse once the drug is stopped whereas CBT is thought to have a long-term protective effect for this disorder (23), and 4) CBT can be used together with pharmacotherapy in order to reduce withdrawal effects during medication taper (16,29) and to improve treatment response (3–5).

According to Barlow and Lechman (6), in the early stages of brief psychosocial treatments for PD, investigators focused on the use of in vivo exposure for treating phobic situational avoidance associated with agoraphobia, (1,3). Although exposure based therapy was found to be effective, a significant percentage of patients did not have a significant response to treatment. In the late 1980s, Barlow went on to develop “panic control treatment” (PCT) aimed at unexpected panic attacks

which involved both exposure therapy and cognitive restructuring, and these treatments were shown to be effective in a number of clinical trials (5,11,14).

In parallel work, the theory of cognitive therapy for depression was applied to the treatment of panic attacks. According to the view of cognitive behavioral therapy (CBT), patients who suffer from PD misinterpret mild pathological physical or psychological symptoms in a way that causes a heightened sense of alarm (8,10). For example, in PD patients, palpitations may trigger fear of an impending heart attack. This creates a viscous cycle of apprehension regarding imminent physical disaster which in turn leads to an escalation of somatic symptoms with further misinterpretations of the symptoms as catastrophic (11). Clark describes two further maladaptive cognitive processes which serve to perpetuate panic attacks. First, PD patients show a heightened awareness to bodily symptoms, and once these symptoms are identified, they are taken as further evidence of a serious physical or emotional disorder. Second, he proposes that patients engage in avoidance behavior which prevents them from understanding that their fears are unrealistic (11–13). The moderate view of CBT for the treatment of PD, therefore, is based on the idea that reducing a patient's tendency to interpret bodily or mental sensations in a catastrophic fashion will lead to a reduction in panic symptoms.

Multiple clinical trials have shown that CBT is a specific and highly effective treatment for PD (7–8,11–13,28). In a prospective trial, Sokol (28) found that cognitive behavioral therapy (i.e., aimed at modifying the patient's misunderstanding of bodily symptoms as catastrophic) was highly effective in reducing the frequency of panic attacks, and these results were maintained over a one year follow up period. Beck (8), in a crossover study of cognitive behavioral therapy for PD, showed that the patients who received cognitive therapy achieved significantly greater reductions in panic symptoms after eight weeks of treatment than did the group that received brief supportive psychotherapy. Interestingly, several investigators have shown that CBT is efficacious in preventing both physiological withdrawal symptoms and relapse of panic symptoms during the slow taper and discontinuation of benzodiazepines (25,29).

Telch (30) combined elements of PCT and CBT to treat agoraphobic PD patients in a group setting with positive results. More recently, Otto (26) in a clinical case series of PD pharmacotherapy nonresponders, demonstrated that the patients responded well to a standard program of brief group cognitive behavioral therapy. Indeed, a group setting is well suited to the treatment of PD because patients are able to become role models for each other as they learn to develop control over panic symptoms. In addition, cognitive behavioral group therapy (CBGT) has practical advantages in a busy clinic setting, for two therapists can provide treatment for a group of about 8–12 patients. This study was designed to examine the effectiveness of CBGT during the

acute treatment phase (initial three months) of Panic Disorder in a randomized, controlled fashion. Specifically, our goal was to compare the use of CBGT versus Paroxetine during the first three months of treatment with respect to several outcome parameters including frequency of panic attacks, level of anticipatory anxiety, and improvement in general functioning.

SAMPLE

57 PD patients were enrolled in our study, and 50 patients completed the trial. The group of study completers consisted of 29 women and 21 men, and the average age was 44.9 ± 12.7 years. All the completers in the sample met DSM-IV criteria for PD ($N = 31$) or Panic Disorder with agoraphobia ($N = 19$). The exclusion criteria were: 1) age less than 18, 2) comorbid psychiatric diagnosis and substance abuse (including benzodiazepines and alcohol), 3) psychological treatment in the past year, and 4) lack of ability to sign informed consent. The enrolled patients did not have any other Axis I diagnoses. The mean number of panic attacks per week before entering the study was 4.4 ± 3.2 in the Paroxetine group and 4.3 ± 3.6 in the CBGT group. Patients enrolled in the study had not received pharmacotherapy for PD in the two months preceding entry into the trial. All of the patients were in good health with the exception of 12 patients who had chronic, stable medical conditions such as ischemic heart disease ($N = 5$), hypertension ($N = 4$), non-insulin dependent diabetes mellitus ($N = 1$), and hypothyroidism ($N = 2$).

STUDY DESIGN

The study was conducted at the Sheba Medical Center which is a large, tertiary care medical center in Israel. The patients were referred to the Anxiety Disorder Clinic in the Department of Psychiatry either through the Emergency Room or by their primary care provider. All patients completed a semi-structured psychiatric interview performed by a senior psychiatrist (PND). This interview was based on DSM-IV-R psychiatric diagnoses. Patients who were appropriate for the study according to our inclusion and exclusion criteria signed informed consent for participating in the study. The study was approved by the hospital's ethical review board and the Ministry of Health.

After the initial screening interview, the patients were invited for an additional baseline diagnostic evaluation which was performed in two consecutive visits. At the end of this procedure, the patients were randomized to receive either pharmacological treatment with Paroxetine ($N = 33$) or Cognitive Behavioral Group Therapy ($N = 24$). Note that due to an unexpected staff shortage, an additional group of 8

patients who had been randomized receive CBGT was not able to participate in the study. Paroxetine was chosen versus other SSRIs because of its availability within the regional HMO system. (The possible options were Paroxetine or Fluoxetine, and due to its shorter half-life, Paroxetine was chosen). The pharmacotherapy condition involved monthly visits. Paroxetine was started at 10 mg per day and was increased up to 40 mg per day according to the patient's response. Paroxetine was continued throughout the 12 week duration of the study. The CBGT patients were formed into three groups of eight patients, and the groups met weekly for eight weeks.

Efforts were made to ensure that the study patients did not take benzodiazepines on their own to control panic symptoms. Patients made a verbal agreement with the investigators to take only the study medication and not to take benzodiazepines during the study. We also contacted each patient's general practitioner and requested that no benzodiazepines be prescribed during the study protocol. One psychiatrist (PND) assumed responsibility for dispensing all study medication in order to further control for uniformity of treatment according to the study protocol. Random urine drug screens for benzodiazepines were not performed.

The CBGT treatment program used at the Israeli site was largely modeled after the moderate view of CBT for PD as described by Beck and Clark. Patients were taught a variety of techniques aimed at increasing their ability to control panic symptoms. Each treatment session was two hours long, and the outline of material covered in each session was based on material presented by Bourne in his workbook on the step-by-step treatment of panic disorder (9).

Material covered in a sequential manner through the eight sessions included: 1) psychoeducation regarding the causes of PD, 2) coping strategies for controlling panic attacks (such as challenging catastrophic misinterpretations bodily sensations and counteracting panic at an early stage), 3) identification and reframing of negative cognitive schemata (which Bourne refers to as negative "self-talk"), 4) discussion of the role of physical exercise and nutrition in the treatment of PD, 5) relaxation techniques including abdominal breathing, progressive muscle relaxation, and

visualisation, and 6) assertiveness training (based on the idea that an effective communication style can reduce tension and frustration and improve interpersonal relationships). The patients were given homework assignments after each session. The group was designed as a closed group for only panic disorder patients with or without agoraphobia. The group met weekly for eight weeks. The principal therapist for the CBGT group was MGO, and AG was the co-therapist. Both therapists were psychiatric nurses. A board certified, senior psychiatrist was in charge of treating the patients in the medication group. A psychiatric nurse (EN) who was blind to treatment administered the psychiatric rating scales at baseline and at weeks four and twelve of the study.

INSTRUMENTS

The psychiatric rating scales that were used in this study were designed to evaluate symptoms of anxiety and depression as well as level of functioning. The instruments were administered at baseline, and at weeks 4 and 12 and included the following: the Hamilton Rating Scale for Anxiety (HRSA) (17), the Visual Analog Scale (VAS) (32), the Panic Self Questionnaire (PSQ) (22), and the Clinical Global Impression Scale (CGI-S, CGI-I) which rates both severity of symptoms and degree of improvement (18).

ANALYSIS

Statistical analysis was performed with *t*-test analysis, chi squares, and ANOVA with repeated measures. Levels of significance were set at 0.05 unless otherwise stated.

RESULTS

Demographic data and results with ANOVA with repeated measures are displayed in Table 1. Twenty seven out of 33 patients who were randomized to receive paroxetine completed the study, and 23/24 CBGT patients completed the study. Twenty seven out of 33 patients in the

Table 1 Demographics (*N* = 50)

	CBGT X ± SD (<i>N</i> = 23)	Pharmacotherapy X ± SD (<i>N</i> = 27)	<i>t</i>	CI	chi square	<i>p</i>
Age	44.8 ± 13.1	45.1 ± 11.9	−0.09	(−7.4, 6.8)		NS
Years of education	11.9 ± 2.4	12.6 ± 1.9	−1.1	(−1.9, 0.5)		NS
Disorder	9.8 ± 8.7	9.1 ± 6.4	0.3	(−3.6, 5.0)		NS
Gender (m/f)	10/13	11/16			0.04	NS
Family status (1/2/3)	5/15/3	7/20/0			3.7	NS
Diagnosis (PD/PDA)	13/10	18/9			0.54	NS

Table II

	Cognitive Behavioural Group Therapy			Pharmacotherapy		
	Baseline	Four weeks	Twelve weeks	Baseline	Four weeks	Twelve weeks
HRSA	21.4 ± 10.0	14.0 ± 10.0	9.9 ± 8.0	19.9 ± 9.7	15.1 ± 8.1	11.0 ± 6.9
CGI-severity	3.0 ± 1.1	2.74 ± 0.8*	2.6 ± 0.9	3.1 ± 0.8	3.2 ± 0.9*	2.7 ± 0.8
CGI-improvement	4.2 ± 1.4	2.4 ± 1.0	2.2 ± 0.8	4.4 ± 1.6	2.5 ± 0.7	2.5 ± 0.7
Visual Analog Scale	65.8 ± 21.0	47.1 ± 24.3	33 ± 19.1	60.3 ± 21.1	45.3 ± 25.1	34.4 ± 18.0
PSQ	4.3 ± 3.6	2.1 ± 2.5	1.5 ± 2	4.4 ± 3.2	3.0 ± 1.9	1.8 ± 1.3
HRSA	Group Effect (F 0.03, df 1,47, p ns), Time Effect (F 70.4, df 2,94, p < 0.00), Interaction (F 1.4, df 2,94, p ns)					
CGI-severity	Group Effect (F 1.0, df 1,48, p ns), Time Effect (F 10.5, df 2,96, p < 0.00), Interaction (F 3.4, df 2,96, p = 0.035*)					
CGI-improvement	Group Effect (F 1.1, df 1,48, p ns), Time Effect (F 11.3, df 2,96, p < 0.00), Interaction (F 1.7, df 2,96, p ns)					
Visual Analog Scale	Group Effect (F 0.1, df 1,48, p ns), Time Effect (F 91.1, df 2,96, p < 0.00), Interaction (F 1.2, df 2,96, p ns)					
PSQ	Group Effect (F 0.5, df 1,48, p ns), Time Effect (F 57.3, df 2,96, p < 0.00), Interaction (F 1.3, df 2,96, p ns)					

paroxetine group (81%) and 23/24 patients in the CGBT group (97%) responded well to treatment. The criteria for response in both groups was zero panic attacks per week. Six patients in the paroxetine group, four women and two men, dropped out of the study due to side effects of the drug. One male patient suffered from severe gastrointestinal side effects, whereas the female patients and the second male patient suffered from palpitations, restlessness, and insomnia. One patient from the CGBT group dropped out because of an unexpected trip abroad. There were no suicides attempts or completed suicides during the study. Also, there were no acute psychiatric hospitalizations of the subjects during the study. The improvement was statistically significant as measured by the rating scales at weeks four and twelve of treatment in both groups. With ANOVA with repeated measures, the improvement was statistically significant in both groups at week twelve on the PSQ (F = 57.3, p < 0.00, df = 2,96), the HRSA (F = 70.4, p < 0.00, df = 2,94), the VAS (F = 91.1, p < 0.00, df = 2,96), and on the CGI-S (F = 10.5, p < 0.00, df = 2,96), and the CGI-I (F = 17.3, p < 0.00, df = 2,96).

There were no significant differences between the PD and the PD with agoraphobia patients in terms of severity of anxiety symptoms as measured at baseline and at the visits for week four and twelve. There were no significant effects of age, sex, and duration of illness on the response to treatment. The response to treatment of the elderly patients (>60 years) was similar to that of the younger patients. Body weight was measured at baseline and at week 12 of treatment. No significant weight gain was seen among our patients who received paroxetine therapy.

DISCUSSION

Whereas CBT is a well accepted therapy for the treatment of PD, our study is one of the first, to our knowledge, to

examine the efficacy of CGBT for the treatment of PD in a randomized, controlled fashion. Our PD patients experienced an impressive overall improvement whether they were randomized to the CGBT group or to the Paroxetine group. At the end of week 12, patients in both groups reported a reduction in anxiety symptoms, a decreased frequency of panic attacks, and an increased general feeling of well being. The effect of CGBT administered without pharmacotherapy was equal to the effect of treatment with Paroxetine alone. This result is similar to Barlow's comparative CBT/pharmacotherapy study for panic where he found both treatments to have equal efficacy (4).

Our response rate of 81% for patients randomized to receive short-term Paroxetine treatment is consistent with the response rate reported in the related studies. In a double blind, placebo controlled study, Ballenger reported that 86% of PD patients became panic free following 10 weeks of 40 mg/day Paroxetine treatment (2). Additional short-term studies with Paroxetine have also attained very high recovery rates (20,24). Likewise, the high response rate among our CGBT patients is consistent with the high success rates reported for CBT in the treatment of panic disorder. In a crossover study of focused cognitive therapy, Beck *et al.* found that at eight weeks, 71% of the cognitive therapy patients were panic free, compared to 25% of the brief supportive psychotherapy group (8). Recent randomized, controlled short-term studies have also shown CBT to be very efficacious in the treatment of panic disorder (13,21).

The main strengths of this study are the parallel treatment design, the use of blinded raters, and the prospective nature of the study. The primary limitations of the study are the relatively small sample size and the lack of a placebo control group. Also, the relatively low baseline CGI severity scores in our patient population should be noted, for this may limit the generalizability of our findings to patients with mild to moderate symptoms. A control group would have strengthened the validity of this preliminary study, for based

on our current study design, we cannot rule out the influence of non-treatment effects on the study outcome such as the passage of time contributing to the treatment response. Furthermore, without a control group, the study is subject to possible unconscious, positive rater-bias, for even our blinded raters may have been predisposed to see improvement in all study subjects. However, according to the guidelines of the Israeli Helsinki committee, we were not permitted to offer our patients the possibility of placebo treatment in the presence of readily available standard treatment options (i.e., pharmacotherapy or cognitive therapy). Therefore, we chose to evaluate the effectiveness of CBGT by comparing it with Paroxetine which has proven efficacy in the treatment of Panic Disorder.

Our preliminary study demonstrates that CBGT represents a beneficial and cost-effective treatment strategy for the acute phase of Panic Disorder and compares favorably to Paroxetine treatment given alone. A follow-up study with a larger sample size is needed to confirm our results. In addition, future studies can address the question of the long-term effectiveness of CBGT for the maintenance treatment of Panic Disorder.

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