Efficacy of Cognitive-Behavioral Therapy (CBT) on Panic Disorder: A Case Study

Tamrat Zelalem Teshome1*, Rajendran Natarajan2, Dawit Negassa3
1Lecturer in Clinical Psychology, Department of Psychology; Head, Counseling and Guidance Program - Haramaya University, Ethiopia
2Associate Professor in Clinical Psychology, Department of Psychology - Haramaya University, Ethiopia
3Assistant Professor in Special Needs and Inclusive Education - Haramaya University, Ethiopia

Abstract: Panic Disorder (PD) is among the types of Anxiety Disorders (AD); it is characterized by the presence of sudden anxiety attacks, followed by physical and affective symptoms, fear of having a new attack and avoidance of events or situations in which panic attacks have occurred. PD affects two to three times more women than it affects men, and may affect up to 3.5% of the world's population throughout life. The most common treatment modalities used to treat the continuum are the administration of psychotropic medications and the application of behavioral therapy. This particular case details on a female university student who suffered from severe panic attacks for a period of 1 year wherein numerous interventions, including psychotropic medications, had failed to ameliorate her suffering. The patient was treated using the Mastery of Your Anxiety and Panic (MAP) protocol. Prior to treatment, the patient reported that she experienced an average of seven panic attacks per week, her Beck Anxiety Inventory (BAI) score was 58, and her Beck Depression Inventory (BDI) was 23. At the completion of treatment, the patient reported that the number of panic attacks was reduced gradually in the following weeks. At 4-week, 6-week, and 16-week follow-up, the patient had not experience any symptoms of panic attacks.

Keywords: Cognitive-Behavioral Therapy, Panic Disorder.

THEORETICAL BASIS

This article aims at the application of Cognitive-Behavioral Therapy (CBT) for a patient with Panic Disorder (PD), focusing on the nature of the disorder, the interventions used in CBT, and the outcome with this treatment modality.

Panic Disorder (PD) is among the types of Anxiety Disorders (AD). ADs are the most common psychiatric disorder occurring more often in women than in men. Plethora of empirical literature reviews reported on the treatment of anxiety disorders includes the administration of psychological medications and psychotherapy (CBT).

PD is characterized by the presence of sudden anxiety attacks, followed by physical and affective symptoms, fear of having a new attack and avoidance of events or situations in which panic attacks have occurred [1]. The course of PD tends to be chronic in the majority of patients [2, 3] and PD is associated with a reduced quality of life and impaired psychosocial functioning [4, 5].

As compared to the other types of AD, the onset of PD is frequently later, occurring in the late twenties on average [6]. PD affects two to three times more women than men, and may affect up to 3.5% of the population throughout life [6]. Patients frequently report PD onset after a period of stress [7], and it is associated with a high social cost; patients with PD have reduced productivity and often use public health services, such as emergency rooms, medical visits and evaluations [8].

Numerous studies have confirmed the effectiveness of Drug Therapy (DT) for PD [9, 10]. Among the pharmacological options used in the treatment of PD are Selective Serotonin Reuptake Inhibitors (SSRI), Tricyclic Antidepressants (TAD), Monoamine Oxidase Inhibitors (MAOI), Serotonin and Noradrenaline Reuptake Inhibitors (SNRI), and Benzodiazepines [11]. However, many patients, although being under DT, remain symptomatic and have recurrence of symptoms.

Studies have shown that after 4 years on DT about 30% of patients are asymptomatic, 40-50% are better, but still symptomatic, and 20-30% remain the
same or worse [12]. It is also known that presence of residual symptoms is associated with increased risk of relapses [13]. Cognitive-Behavioral Therapy (CBT) for PD is a therapeutic alternative that has good short-and long-term response for both core panic symptoms as well as the residual and often persistent symptoms of anticipatory anxiety, phobic avoidance, and agoraphobia [9, 10, 11, 14].

Various studies have confirmed that CBT can change PD course not only for preventing relapses, but also because it prolongs the time interval between them [15]. Brief treatment (often in the range of 12 sessions) with CBT is associated with high (75%) panic free rates among patients. The efficacy demonstrated in studies suggests that CBT outcomes are better than long-term pharmacology: 87% of patients remain without attacks at 1 year, and 75-81% at 2 years after completion of brief CBT [16, 15]. The effect size found for therapeutic response ranges between 0.6-2.3, depending on the dimension being evaluated [5, 14]. CBT for PD is also associated with improvements in co-morbid conditions and quality of life [17, 1]. Heldt et al. in a study of 32 patients has demonstrated that reducing the symptoms of anticipatory anxiety and avoidance are more important for improving quality of life than changes in the frequency of panic attacks [5].

PSYCHOTROPIC APPROACH TO TREATMENT

The biological model of anxiety postulates that anxiety and panic are in response to a neurobiological defect in brain function, most probably neurotransmitter abnormalities or a metabolic imbalance [18]. The prescribed medical procedure for the treatment of anxiety involves the administration of a benzodiazepine, antidepressant, and buspirone [19]. The most commonly prescribed medications in this protocol are Xanax® (alprazolam), Valium® (diazepam), and Imipramine [20].

Keltner and Folks [19] report that the side effects of Alprazolam include chemical dependence, dizziness, headache, depression, and anxiety. If mixed with alcohol, it can also be fatal. Side effects for Imipramine include chemical dependence, sedation, confusion, and delirium. Side effects for Buspirone include dizziness, headache, depression, stimulation, excitement, and hyperventilation. It is ironic that many of these side effects are the very symptoms that prompt the patient’s initial presentation for care.

Anxiolytics have failed to establish their superiority over other medications in the treatment of anxiety and panic attacks [21]. The most significant issue surrounding the issue of benzodiazepines is the addictive side effect of this class of agent [19]. H. W. Clark and McClanahan [22] report that Benzodiazepine can produce chemical dependence in as most of the patients to whom they are administered. This addictive side effect can be so severe that medical detoxification, which requires hospitalization and longterm medical intervention, may be required to taper the patient off this class of medication [19].

Examination of the seminal Cross-National Collaborative Panic Study (CNCPs) [12], one of the largest efficacy studies to date, indicates that benzodiazepines did not produce significantly better results than the administration of the placebo. A follow-up study conducted by Marks et al. [21] reported that side effects of alprazolam were originally underreported and were more severe than originally reported. In fact, during follow-up, the alprazolam group had 3½ times more panic attacks than the placebo group.

According to Michelson and Marchione [20], imipramine is effective only when combined with behavioral therapy. However, it is encumbered by the numerous and severe side effects that had led many subjects to terminate treatment early. Uhlenhuth et al. [23] reported a 50% drop-out rate by Week 8 of treatment when imipramine was administered. Klerman et al. [24] reported that long term amelioration of symptoms through the use of imipramine has not been established, and many patients fail to respond at all to the medication. There is even a risk of antidepressant-induced mania or psychosis in some patients [25].

THE COGNITIVE-BEHAVIORAL MODEL AND TREATMENT

Model

According to CBT models, panic attacks arise from distorted interpretations of bodily symptoms [22]. Dizziness or heart palpitations may be interpreted, for example, as an impending heart attack or stroke. Such interpretations increase arousal and intensify bodily sensations, thus confirming a sense of impending “danger” and generating more catastrophic interpretations and more anxiety in a rapid spiral.

According to Barlow’s version of this model, an initial panic attack represents a “false alarm” where too much anxiety is signaled, often in response of life stress. Stimors reported by patients include negative life events such as a threatening loss or severe disease to the self or loved one, as well as separations or interpersonal difficulties [26]. Such “alarm reactions” are hypothesized to be more likely in biologically (genetic heritage) or psychologically (sensitivity to anxiety symptoms) vulnerable individuals. After the first attack, the person becomes apprehensive about new attacks, and develops fears of the physical sensations associated with autonomic arousal.

Repetition of attacks is hypothesized to make individuals increasingly more sensitive to internal stimuli and to situations in which the attack occurred,
and to heighten surveillance of any physical sensation. Combined with that is anticipatory anxiety, i.e., fear of having another attack and catastrophic interpretations of symptoms when they do occur [27, 15]. Such fear-conditioned behavior leads the individual to avoid somatic symptoms (for example: physical exercises) or places associated with previous attacks (agoraphobia) [26]. As a consequence, patients start having limitations in their everyday activities [15]. Accordingly, CBT is used to eliminate the hypervigilance to symptoms, correct distorted interpretations and beliefs, and eliminate the agoraphobia.

**Treatment**

Efficacy of cognitive-behavioral therapy in the treatment of panic attacks has been clearly established [22, 26, 20, 28]. The foremost behavioral treatment model was developed by Barlow and Craske [28] and includes cognitive restructuring, interoceptive exposure, and breathing and relaxation training.

**Cognitive restructuring**

Cognitive theory posits that attitudinal change is essential to long-term amelioration of symptoms. Holon, Evans, and DeRubeis [18] reported that cognitive-behavioral therapy produced greater change in attributional styles than pharmacotherapy. These researchers further reported that change in the patient’s attributional style can moderate relapse rates.

**Exposure therapy**

Exposure therapy, using a cognitive-behavioral approach, has been widely applied and evaluated in the treatment of anxiety disorders. In vivo exposure therapy is the treatment of choice for post-traumatic stress disorder [29]. Long-term follow-up reports (ranging from 4 to 9 years post treatment) indicate that two thirds of patients continued to experience an amelioration of symptoms when exposure therapy was originally used [30, 31]. Lelliott et al. [31] also reported that exposure therapy was more efficacious than antidepressant medication in the treatment of panic disorder.

**Relaxation training**

Klosko, Barlow, Tassinari, and Cerny [28] reported that patients treated with cognitive-behavioral therapy that included relaxation training had far superior outcome compared with those who received alprazolam. Specifically at 2-year follow-up, 85% of the patients who received the cognitive-behavioral treatment with relaxation training reported that they remained free of panic attacks, compared with 50% of the patients who received the alprazolam, and only 35% of the patients who received the placebo. When these reports were adjusted for the placebo effect, the cognitive behavioral therapy was twice as effective as the medication-only group.

**Elements of CBT**

CBT is typically a brief treatment, between 10 and 20 structured sessions, with clear objectives to be achieved. It aims at correcting catastrophic interpretations and conditioned fears of body sensations and avoidance. It is practical, task-based and the patients and therapists roles are active. CBT can be performed individually or in groups [32, 5]. Despite studies suggesting that CBT should be brief, recent data on therapy “dose” have suggested that a higher number of sessions, either in-person or telephone counseling sessions, is associated with better response to CBT in PD [5]. Nonetheless, positive outcomes have been reported for the application of very brief treatment (in the range of six sessions) offered in clinic and primary care settings [22, 33, 8].

CBT can be introduced at any stage of treatment, ranging from primary prevention to interventions to individuals refractory to other treatments [15]. It can be started concomitantly with medications [27, 5] and uses the following resources as techniques: psychoeducation, anxiety coping techniques (muscle relaxation & abdominal or diaphragmatic breathing), cognitive restructuring, interoceptive and gradual in vivo exposure [27, 32, 5].

**CBT sessions**

CBT sessions in PD are structured, following the basic model proposed by Beck [18]. Initial sessions are dedicated to patient assessment, psychoeducation, and training of techniques to cope with anxiety (muscle relaxation and abdominal breathing) if these techniques are used. As sessions progress, greater attention is given to cognitive, interoceptive, and in vivo exposure interventions. Final sessions are devoted to the consolidation of gains and to relapse prevention efforts. Relapse prevention is aided by full practice of interoceptive exposure in a wide variety of situations, ensuring that patients do not “leave well enough alone” and avoid full exposures in feared situations. Also, rehearsal of likely future difficulties (e.g., special events where anxiety may arise) and appropriate coping techniques, along with a review of the strategies that the patient found helpful in the past, provided additional elements of relapse prevention efforts [26, 32]. According to Wade et al., chronic stress was a predictor of worse outcome in patients with agoraphobia 12 weeks after CBT [21] as well as in a 2-year follow-up assessment [5]. Therefore, it is important to identify situations that predispose to a higher psychological vulnerability to prevent relapses and chronicity [5, 15].

**CASE PRESENTATION**

This case depicts the classic symptomatology of panic attacks, how these symptoms affected daily functioning of the patient, the treatment approach employed to alleviate the symptoms, and the patient’s response to treatment.
Identifying information has been modified in order to ensure the patient’s anonymity. The patient, Jalene (name changed), was female in her early 20s. At the time of treatment, she was a first year Computer Science student, with Semester Grade Point Average (SGPA) of 1.75. She was from a semi-urban area and low socio economic background. Her families were not supportive of her education. They rather wanted her to marry, form a family and raise children.

Prior to presenting to the Guidance and Counseling Service of Haramaya University, Patient was being treated by a Physician in a Higher Health Center, twice. Physician placed her on Alprazolam to which she had an adverse reaction. Yet, she used to consult a Physician was only when her suffering gets intense and unbearable.

**PRESENTING COMPLAINTS**

Patient reported that she developed physiological complains that included increased heart beat, chest pain, shortness of breath, dizziness, sudden perspiration, and numbness on palms while she was writing exams for the Ethiopian School Leaving Certificate Examination (ESLCE) to join university, in 2016. Since then, she developed the above complaints even in class rooms, cafeteria, and in other public areas. She felt that the panic attacks were becoming problematic at her classroom, and her teachers and classmates had commented on her condition. The patient stated that she experienced panic attacks for more than 1 year. She sought out help from religious and cultural places. She terminated these services, which she used to get in her living area, because she had to join her university study.

The salient criteria for a diagnosis of PD, without agoraphobia, according to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition - DSM-IV [34] are: a history of recurrent unexpected panic attacks (Criterion A), with at least one of the panic attacks followed by 1 month of (a) persistent concern, (b) worry about the consequences or implications of the panic attack, or (c) a significant change in behavior because of the panic attack. The presentation must not include agoraphobia (Criterion B), and the panic attacks are not due to substance use or general medical condition (Criterion C), or another psychiatric disorder (Criterion D). Patient condition met with these criteria.

**HISTORY**

Patient was born in a semi-urban district of West Ethiopia and spent her childhood years there. She is the last born and has one elder brother and one elder sister.

She reported that her mother had some sort of psychiatric illness, which she does not fully explain and had been receiving cultural and religious treatment for her disorder for many years. She noted that her father was both alcoholic and addicted to Khat (a psychoactive substance). She described situations where her father would come home intoxicated and report that he would beat her mother. Also, she reported that during these tirades, her father would beat her too shouting “you are not my daughter”. During these situations, her siblings would shirk away in fear for themselves.

**ASSESSMENT**

Contemporary assessment may include various modalities to glean salient information from the patient. For the purposes of this case, an in-depth clinical interview was supplemented by administrations of the Mini-Mental Status Examination (MMSE; Veterans Administration), Beck Depression Inventory (BDI) [35], and the Beck Anxiety Inventory (BAI) [18]. The was administered during the initial intake assessment. The BAI and BDI were administered pre-and post treatment and at 4-and 6-week follow-up. During the course of treatment, the patient recorded various aspects of panic attacks and negative thoughts on daily basis.

**MMSE**

The MMSE assesses domains that include orientation, recent recall memory, attention, language abilities, and visuo-spatial abilities. Given the fact that it gathers factual information, traditional psychometric data are not applicable.

**BAI**

The BAI is a 21-item self-report measure of the severity of anxiety in psychiatric populations. Internal consistency is very high (α = .92), and test-retest reliability is also high (r = .75). Validity of the BAI was established through correlation to other instruments that assess similar states of anxiety [18].

**BDI**

The BDI is the most widely used self-report inventory in measuring change in depressive symptoms [18]. The BDI is a 21-item self-report measure of depression. Each item is scored on a 0 to 3 Likert-type scale, with 63 as the maximum score that can be given. A score of 11-20 indicates a mild depression, 21-30 indicates a moderate state of depression, 31-40 indicates a severe state of depression, and a score in excess of 41 indicates an extreme state of depression. Item 9 assesses suicidal ideation and is a critical item regardless of the score assigned for the total administration.

**CASE CONCEPTUALIZATION**

The patient presented with classic symptomatology of PD. In the diagnostic stage of the treatment process, it was essential to understand the relationships among the problems, and the therapist approached the case from a two-level model of
evaluating the psychological problem [36], where an underlying psychological mechanism creates problems with the patient’s mood, behavior, or cognitions. The patient experienced difficulties with mood, behaviors, and cognitions. The underlying psychological mechanism was her fear of losing control and poor performance in her various roles, especially in her courses.

Persons’ [36] two-level psychological model matched with Patient’s presenting issues. For instance, her anxious mood stemmed from her fear of losing control of her emotions. When she was anxious she feared “going crazy” and she experienced intrusive negative thoughts. These thoughts were reminiscent of statements that her father had made during her childhood.

**TREATMENT PLAN**

Given the fact that Patient’s list of problems was behavioral in nature and the behavioral intervention program was the most appropriate approach. This decision is supported by the fact that behavioral and cognitive-behavioral interventions in the treatment of ADs have become the treatment of choice and have proven that they are more effective than psychopharmacological interventions [18].

Barlow and Craske’s [26] *Mastery of Your Anxiety and Panic (3rd ed.)* (MAP-3) was the protocol used in this case. This treatment protocol addresses the physiological components of anxiety and panic (i.e., anticipatory anxiety, cued versus uncued panic), hyperventilation, breathing retraining, deep muscle relaxation, cognitive restructuring, causal analysis, exposure training, and planning for the future. During the course of treatment, the patient had been given homework assignments (Auto Biography Work - ABW) that included tracking the panic attacks, negative cognitions, triggers, and self-administered relaxation exercises.

**Course of Treatment and Assessment of Progress**

Patient was treated for ten 50-minute sessions. The course of treatment also included two follow-up sessions, which were conducted at 4- and 6-week post treatment intervals.

**DIAGNOSTIC INTAKE INTERVIEW**

At intake, Patient’s BAI score was 58 and her BDI score was 23. These scores indicated that she experienced an “extreme” state of anxiety and a “moderate” state of depression. The patient had a history of suicidal ideation but never made any attempt to commit. The author conducted the diagnostic intake interview and assigned the diagnosis of PD without agoraphobia.

**TREATMENT SESSIONS**

Information was gathered during the first session that supplemented the initial diagnostic interview. This information indicated that this was her fifth attempt to seek treatment for panic attacks. However, she used to consult with General Physician and Clinical Nurses, not with Psychiatrists or Clinical Psychologist. The patient felt no better improvement even after the administration of Alprazolam for 7 days.

A functional analysis was conducted and elucidated the problem list, which included uncued panic attacks and hyperventilation. The first session included psychoeducation surrounding the nature of panic attacks, current treatment approaches, and expectations and roles of therapy. The patient was asked to track her panic attacks on a daily basis, and this procedure was followed for the entire course of treatment.

At the beginning of each session, between-session homework was reviewed. Information that was tracked for the panic attacks included physiological changes, intensity, and the frequency, location of the attack. Cognitions were also been tracked, it included the following components: the activating event, self-talk, emotional consequence, and the disputations of non-constructive talk.

The first three sessions focused on the functional analysis or causal analysis of Patient’s panic attacks. Her panic attacks were preceded by feelings of anger and frustration in a vast majority of the incidents. The data indicated that most of her panic attacks experienced in the morning, and the majority of these attacks occurred in classrooms and cafeteria, as a result she had to be absent from classes. Her most relaxed time was in the late evening at dorm. As she reported, she experienced physiological reactions including: palpitation, sweating, dizziness, and headache.

Patient reported that she had experienced at least seven episodes of panic attacks per week. Number of panic attacks persisted continuously until week 4 of treatment, when the number decreased gradually in the following weeks until the end of week 10.

At the initiation of treatment, her average weekly anxiety score was 4.0 on a 10-point scale. This score consistently decreased over the course of treatment to a level it came down to 1, by week 8. This level was maintained through the completion of the treatment. Week 3 focused on abdominal breathing exercises and tracking. Week 4 was the initiation of relaxation techniques. At the initiation of week 5, she stated that the deep muscle relaxation was so effective that “the relaxed feeling lasted all night and even into the class day the following morning.” This was the patient’s first exposure to relaxation techniques.
FOLLOW-UP

The patient was followed for a period of 6 weeks post-treatment. Formal follow-up assessment was conducted at 4-, 6-, and 16-week post-treatment. Each of the follow-up assessments included the administration of the BAI, BDI, and a functional analysis of panic attack and accompanying symptoms.

The decreased average anxiety score per week for the 4- and 6-week follow-up also was maintained. The patient’s BAI score at intake was a 58, which indicated a “severe” state of anxiety. At 4-week post treatment follow-up, the patient’s BAI score was gradually reduced to a score of 4, which is in the non distressed range. This low score was maintained at post-treatment follow-up with a score of 2.

The patient’s BDI score at intake was 23, which indicated moderate depressive symptoms. However, this score was reduced to a score of 3 at 4-week follow-up and to a score of 2 at 6-week post-treatment follow-up, and was maintained at 16-week follow-up.

Therefore, it can be implied from this case that, the efficacy of Cognitive and Behavioral Therapy plays a greater role in the improvement of physiological and psychological turmoil in a patients with anxiety and panic disorders.

RECOMMENDATIONS TO CLINICIANS

Clinicians are often reluctant to treat anxiety without “supplemental” anxiolytics. This case illustrates the power of behavioral interventions in a patient who had not responded to anxiolytics previously.

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