

Effectiveness of Cannabidiol Oil for Pediatric Anxiety and Insomnia as Part of Posttraumatic Stress Disorder: A Case Report

[Scott Shannon](#), MD, ABIHM

Assistant Clinical Professor of Psychiatry at the University of Colorado School of Medicine in Fort Collins. E-mail:

scottshannon@cowisp.net.

[Janet Opila-Lehman](#), ND

Naturopathic Physician at the Wholeness Center in Fort Collins, CO. E-mail: j.opila.lehman@gmail.com.

Copyright © 2016 The Permanente Journal

Abstract

Introduction

Anxiety and sleep disorders are often the result of posttraumatic stress disorder and can contribute to an impaired ability to focus and to demonstration of oppositional behaviors.

Case Presentation

These symptoms were present in our patient, a ten-year-old girl who was sexually abused and had minimal parental supervision as a young child under the age of five. Pharmaceutical medications provided partial relief, but results were not long-lasting, and there were major side effects. A trial of cannabidiol oil resulted in a maintained decrease in anxiety and a steady improvement in the quality and quantity of the patient's sleep.

Discussion

Cannabidiol oil, an increasingly popular treatment of anxiety and sleep issues, has been documented as being an effective alternative to pharmaceutical medications. This case study provides clinical data that support the use of cannabidiol oil as a safe treatment for reducing anxiety and improving sleep in a young girl with posttraumatic stress disorder.

INTRODUCTION

Cannabidiol (CBD) oil is a naturally occurring constituent of industrial hemp and marijuana, which are collectively called cannabis. CBD oil is 1 of at least 85 cannabinoid compounds found in cannabis and is popular for its medicinal benefits. After tetrahydrocannabinol (THC), CBD oil is the second-most-abundant component of cannabis. Other names for CBD oil include CBD-rich hemp oil, hemp-derived CBD oil, or CBD-rich cannabis oil. Considered to be generally safe, CBD has been used medicinally for decades. However, CBD is not medical marijuana and should be distinguished from high-CBD strains of medical marijuana, which do contain THC, such as “Charlotte’s Web.”

The most abundant compound in cannabis, THC is also a cannabinoid. The THC component induces the psychoactive effect, “high.” A cannabis plant has different amounts of CBD and THC depending on the strain and thus provides different recreational or medicinal effects. The cannabinoid profile of industrial hemp or medical marijuana is ideal for people looking for the medical benefits of CBD without the “high” of the THC.

The mechanism of action of CBD is multifold.^{1–3} Two cannabinoid receptors are known to exist in the human body: CB1 and CB2 receptors. The CB1 receptors are located mainly in the brain and modulate neurotransmitter release in a manner that prevents excessive neuronal activity (thus calming and decreasing anxiety), as well as reduces pain, reduces inflammation, regulates movement and posture control, and regulates sensory perception, memory, and cognitive function.^{4,2} An endogenous ligand, anandamide, which occurs naturally in our bodies, binds to the CB1 receptors through the G-protein coupling system. CBD has an indirect effect on the CB1 receptors by stopping the enzymatic breakdown of anandamide, allowing it to stay in the system longer and provide medical benefits.⁴ CBD has a mild effect on the CB2 receptors, which are located in the periphery in lymphoid tissue. CBD helps to mediate the release of cytokines from the immune cells in a manner that helps to reduce inflammation and pain.²

Other mechanisms of action of CBD include stimulation of vanilloid pain receptors (TRPV-1 receptor), which are known to mediate pain perception, inflammation, and body temperature.⁵ In addition, CBD may exert its anti-anxiety effect by activating adenosine receptors which play a significant role in cardiovascular function and cause a broad anti-inflammatory effect throughout the body.⁵ At high concentrations, CBD directly activates the 5-HT1A serotonin receptor, thereby conferring an antidepressant effect.⁶ Cannabidiol has been found to be an antagonist at the potentially new third cannabinoid receptor, GPR55, in the caudate nucleus and putamen, which if stimulated may contribute to osteoporosis.⁷

Since the 1940s, a considerable number of published articles have dealt with the chemistry, biochemistry, pharmacology, and clinical effects of CBD.⁸ The last decade has shown a notable increase in the scientific literature on CBD, owing to its identification for reducing nausea and vomiting, combating psychotic disorders, reducing inflammation, decreasing anxiety and depression, improving sleep, and increasing a sense of well-being.^{9–12} Findings presented at the 2015 International Cannabinoid Research Society at its 25th Annual Symposium reported the use of CBD as beneficial for kidney fibrosis and inflammation, metabolic syndrome, overweight and obesity, anorexia-cachexia syndrome, and modification of osteoarthritic and other musculoskeletal conditions.^{13–16}

Although studies have demonstrated the calming, anti-inflammatory, and relaxing effects of CBD, clinical data from actual cases is minimal. This case study offers evidence that CBD is effective as a safe alternative treatment to traditional psychiatric medications for reducing anxiety and insomnia.¹⁷

CASE PRESENTATION

A ten-year-old girl presented in January 2015 for a reevaluation of behaviors related to her diagnosis of posttraumatic stress disorder (PTSD) secondary to sexual abuse. Her chief issues included anxiety, insomnia, outbursts at school, suicidal ideation, and self-destructive behaviors. Her grandmother, who has permanent custody of the patient and her younger brother, accompanied her.

Our patient had been seen for an initial evaluation in January 2012 and received a diagnosis of PTSD secondary to sexual abuse on the basis of her history, clinical observations, and behaviors ([Table 1](#)). Her father had died 6 months earlier in a motor vehicle accident, and our patient's maternal grandparents became her permanent guardians. Before her father's death, our patient had no supervision from her father and very little supervision from her mother. An 11-year-old boy had molested her when she was 3 years old. Her medical history included her mother having methadone addiction, alcoholism, bipolar disorder, and depression. Her mother used marijuana her entire pregnancy with the girl. The patient presented in January 2012 as displaying aggressive, disobedient, impulsive, and sexually inappropriate behaviors. She also demonstrated low self-esteem and anxiety and had poor sleep (restless, interrupted, and unable to sleep alone).

Table 1

Timeline

Date	Presentation	Medications	Supplements	Other
January 31, 2012	New evaluation: 7.5-year-old girl. History of sexual abuse and neglect. Issues: Insomnia, sexual behaviors. Diagnosis: PTSD secondary to sexual abuse.	None	Melatonin, 1 mg/night	February 14, 2012, laboratory values: TSH, 2.46 mIU/L (reference range, 0.47–4.68 mIU/L); ferritin: 21 ng/mL (reference range, 10–150 ng/mL). February 16, 2012, laboratory values: Vitamin D ₃ : 39 ng/mL (reference range, 20–50 ng/mL)
February 20, 2012	Sleeping 2–3 hours/night. Started counseling; Cooperative and good behavior at counseling session. Anxious, traumatized.	Clonidine, 0.05 mg (half tablet) at bedtime	Inositol, 3 g 3 times/d; EPA fish oil, 500 mg/d	Eye movement desensitization and reprocessing therapy recommended
February 22, 2012	Did not do well with clonidine because of hallucinations, so she discontinued that treatment. Behavior still very rough; sleep poor.	Started imipramine therapy, 25 mg at bedtime		March 7, 2012: ECG was normal
August 8, 2012 ^a	Good summer. In play therapy. Overall better sleep and energy with imipramine therapy. Patient's 6-year-old brother also now in therapy.	Imipramine, 25 mg at bedtime		
January 21, 2015	Returned for evaluation and treatment after 3 years. Suicidal ideation; cut self on leg; defiant and stubborn. Had psychotherapy 3 years straight twice a month. Sleeps with brother; can't sleep alone.	Off all medications for past 18 months	Melatonin, 5 mg; St John's wort, 450 mg twice/d; magnesium, 300 mg/d; diphenhydramine, 25 mg/night	
February 16, 2015	Hard to manage. Has outbursts at school.		Magnesium and St John's wort: stopped treatment; EPA fish oil, 750 mg/d; diphenhydramine, 25 mg/night	February 11, 2015: Normal cortisol and DHEA levels
March 16, 2015	Better overall. Started animal-assisted therapy.		EPA fish oil, 750 mg/d; diphenhydramine, 25 mg/night	Started a regimen of CBD oil, 25 mg (1 capsule)/d at 6 pm
April 14, 2015	Sleeping better with CBD treatment. Getting biofeedback. Has stomachaches. Mood is more at ease.		EPA fish oil, 750 mg/d; diphenhydramine, 25 mg/night	CBD oil, 25 mg (1 capsule)/d at 6 pm
May 26, 2015	"Ghosts" waking patient up at night.		EPA fish oil, 750 mg/d	CBD oil, 25 mg (1 capsule)/d at 6 pm
July 22, 2015	Sleeping better; able to sleep in own room 3–4 nights/wk.		EPA fish oil, 750 mg/d	CBD liquid, 12 mg (in 4 sublingual sprays)/night; 12 mg more (in 4 sublingual sprays) during the day as needed for anxiety, typically 3 or 4 times/wk
August 24, 2015	Sleeping well. Handling school well.		EPA fish oil, 750 mg/d	CBD oil, 25 mg (1 capsule)/night; CBD liquid, 6–12 mg (in 2–4 sublingual sprays) as needed for anxiety, typically 2 or 3 times/wk

[Open in a separate window](#)

^aThere were additional visits in 2012 with no substantial changes.

CBD = cannabidiol; DHEA = dehydroepiandrosterone; ECG = electrocardiogram; EPA = eicosapentaenoic acid; PTSD = posttraumatic stress disorder; TSH = thyroid stimulating hormone.

Workup during 2012 included laboratory studies, which ruled out a thyroid dysfunction and an iron or vitamin D deficiency. The patient was started on a regimen of 1 mg/night of melatonin, which helped her sleep duration. Three grams of inositol 3 times a day and 500 mg/d of eicosapentaenoic fish oil were also helpful in reducing her anxiety. A trial of clonidine was implemented, which resulted in hallucinations and thus was discontinued. The patient was switched to a regimen of 25 mg of imipramine at bedtime to decrease her anxiety, which appeared to be helpful. Counseling sessions were started. The patient continued psychotherapy for 3 years, but she was not seen again in our clinic until the return visit in January 2015, when she was not receiving any of her medications and supplements.

At the patient's return in January 2015, she demonstrated the same prominent symptoms as at her initial presentation. At that time, the initial treatment included the following supplements and medications to assist with her sleep and anxiety: melatonin, 5 mg/night; magnesium, 300 mg/d; and diphenhydramine (Benadryl), 25 mg/night. Our patient demonstrated slight gains but was still having outbursts at school and was reportedly difficult to manage at home. In addition, her underlying anxiety continued.

Cannabidiol oil was explored as a potential additional treatment to help her insomnia and anxiety, but we deferred for two months while we waited for a response from other interventions. The grandmother preferred reducing the pharmacologic load given her granddaughter's failure to respond long term to psychiatric medications.

In March 2015, CBD oil was recommended as a potential additional treatment to help her insomnia and anxiety, and her grandmother provided full informed consent. Our patient was administered the Sleep Disturbance Scale for Children¹⁸ and the Screen for Anxiety Related Disorders (SCARED)¹⁹ before taking the CBD oil and each month afterward for the next 5 months. Test scores on the Sleep Disturbance Scale for Children and Screen for Anxiety Related Disorders demonstrated an improvement ([Table 2](#)).

Table 2

Patient's clinical progress in sleep and anxiety

Date of visit	Sleep scale score ^a	SCARED score ^b
March 16, 2015	59	34
May 25, 2015	42	24
July 22, 2015	41	19
August 24, 2015	37	16
September 22, 2015	38	18

^aA score of more than 50 is considered indicative of a sleep disorder on the Sleep Disturbance Scale for Children.

^bA SCARED score over 25 indicates a high probability of a childhood anxiety disorder.

SCARED = Screen for Anxiety Related Disorders.

A trial of CBD supplements (25 mg) was then initiated at bedtime, and 6 mg to 12 mg of CBD sublingual spray was administered during the day as needed for anxiety. A gradual increase in sleep quality and quantity and a decrease in her anxiety were noted. After 5 months, the patient was sleeping in her own room most nights and handling the new school year with no difficulties. No side effects were observed from taking the CBD oil.

DISCUSSION

Studies repeatedly recognize the prevalence of an anxiety-provoked sleep disorder after a traumatic experience.²⁰ Our patient was definitely experiencing this phenomenon, which was aggravated by daily stressful activities.

The main finding from this case study is that CBD oil can be an effective compound to reduce anxiety and insomnia secondary to PTSD. A review of the literature suggests some benefits from the use of CBD because of its anxiolytic and sleep-inducing effects.⁹ Animal studies support use of this treatment and report that "CBD may block anxiety-induced [rapid eye movement] sleep alteration via its anxiolytic effect on the brain."²¹

The strength of this particular case is that our patient was receiving no pharmaceutical medications (other than nonprescription diphenhydramine) but only nutritional supplements and the CBD oil to control her symptoms. Her scores on the sleep scale and the anxiety scale consistently and steadily decreased during a period of 5 months (see [Table 2](#)). She was ultimately able to sleep through the night most nights in her own room, was less anxious at school and home, and displayed appropriate behaviors. The patient's grandmother (her caregiver) reported: "My granddaughter's behaviors are definitely better being on the CBD. Her anxiety is not gone, but it is not as intense and she is much easier to be around. She now sleeps in her own room most of the time, which has never happened before."

Further study will need to be conducted to determine the permanency of our patient's positive behaviors and how long she will need to continue taking the CBD oil. We do not have a reasonable foundation to recommend dosing from the scientific literature. However, in our experience, this supplement given 12 mg to 25 mg once daily appears to provide relief of key

symptoms with minimal side effects. Our patient did not voice any complaints or discomfort from the use of CBD. We routinely asked about headache, fatigue, and change in appetite or agitation in addition to conducting a routine psychiatric evaluation. Although CBD is considered generally safe,¹⁷ the long-term effects are yet to be studied.

The ultimate goal is to gradually taper her off the use of CBD oil and transition our patient into lifelong coping strategies such as yoga, meditation, and various other therapeutic activities.

Marijuana and Medicine

Scientific data indicate the potential therapeutic value of cannabinoid drugs, primarily [tetrahydrocannabinol], for pain relief, control of nausea and vomiting, and appetite stimulation; smoked marijuana, however, is a crude [tetrahydrocannabinol] delivery system that also delivers harmful substances.

— Joy JE, Watson SJ Jr, Benson JA Jr. *Marijuana and medicine: Assessing the science base*. Washington, DC: National Academies Press; 1999.

Acknowledgments

CannaVest Corp, San Diego, CA, which had no involvement in the case study or distribution of the product, provided the CBD oil that was administered to the patient. No financial support was provided.

Kathleen Loudon, ELS, of Loudon Health Communications provided editorial assistance.

Footnotes

^aGW Pharmaceuticals is the founder of the Cannabinoid Research Institute, directed by Philip Robson, MD. Further research articles listed.

Disclosure Statement

The author(s) have no conflicts of interest to disclose.

References

1. Campos AC, Moreira FA, Gomes FV, Del Bel EA, Guimarães FS. Multiple mechanisms involved in the large-spectrum therapeutic potential of cannabidiol in psychiatric disorders. *Philos Trans R Soc Lond B Biol Sci*. 2012 Dec 5;367(1607):3364–78. doi: 10.1098/rstb.2011.0389. [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
2. Mechanism of action [Internet] Cambridge, United Kingdom: GW Pharmaceuticals plc; c2014. [cited 2015 Aug]. Available from: www.gwpharm.com/mechanism-of-action.aspx. [[Google Scholar](#)]
3. McPartland JM, Guy G. The evolution of cannabis and coevolution with the cannabinoid receptor—a hypothesis. In: Guy GW, Whittle BA, Robson PJ, editors. *The medicinal uses of cannabis and cannabinoids*. 1st ed. London, United Kingdom: Pharmaceutical Press; 2004. pp. 71–102. [[Google Scholar](#)]
4. Leweke FM, Piomelli D, Pahlisch F, et al. Cannabidiol enhances anandamide signaling and alleviates psychotic symptoms of schizophrenia. *Transl Psychiatry*. 2012 Mar 20;2:e94. doi: 10.1038/tp.2012.15. [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
5. Lee MA. CBD: how it works. O’Shaughnessy’s [Internet] 2011. Autumn. [cited 2016 Apr 26]:14. Available from: www.os-extra.cannabisclinicians.org/wp-content/uploads/2012/07/CBDiary21.pdf.
6. Crippa JA, Derenusson GN, Ferrari TB, et al. Neural basis of anxiolytic effects of cannabidiol (CBD) in generalized social anxiety disorder: a preliminary report. *J Psychopharmacol*. 2011 Jan;25(1):121–30. doi: 10.1177/0269881110379283. [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
7. McHugh D, Tanner C, Mechoulam R, Pertwee RG, Ross RA. Inhibition of human neutrophil chemotaxis by endogenous cannabinoids and phytocannabinoids: evidence for a site distinct from CB1 and CB2. *Mol Pharmacol*. 2008 Feb;73(2):441–50. doi: 10.1124/mol.107.041863. [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
8. Zhornitsky S, Potvin S. Cannabidiol in humans—the quest for therapeutic targets. *Pharmaceuticals (Basel)* 2012 May 21;5(5):529–52. doi: 10.3390/ph5050529. [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
9. Zuardi AW. Cannabidiol: from an inactive cannabinoid to a drug with wide spectrum of action. *Rev Bras Psiquiatr*. 2008 Sep;30(3):271–80. doi: 10.1590/s1516-44462008000300015. [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
10. Burstein S. Cannabidiol (CBD) and its analogs: a review of their effects on inflammation. *Bioorg Med Chem*. 2015 Apr 1;23(7):1377–85. doi: 10.1016/j.bmc.2015.01.059. [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
11. Fernández-Ruiz J, Sagredo O, Pazos MR, et al. Cannabidiol for neurodegenerative disorders: important new clinical applications for this phytocannabinoid? *Br J Clin Pharmacol*. 2013 Feb;75(2):323–33. doi: 10.1111/j.1365-2125.2012.04341.x. [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
12. Zuardi AW, Crippa JA, Hallak JE, Moreira FA, Guimarães FS. Cannabidiol, a Cannabis sativa constituent, as an antipsychotic drug. *Braz J Med Biol Res*. 2006 Apr;39(4):421–9. doi: 10.1590/s0100-879x2006000400001. [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
13. Fingerle J. CB2 agonism protects from inflammation related kidney damage and fibrosis. *Proceedings of the 25th Anniversary Symposium of the International Cannabinoid Research Society*; 2015 Jun 28–Jul 3; Wolfville, Nova Scotia, Canada. [[Google Scholar](#)]

14. Purohit V. Role of cannabinoids in chronic pain. Proceedings of the 25th Anniversary Symposium of the International Cannabinoid Research Society; 2015 Jun 28–Jul 3; Wolfville, Nova Scotia, Canada. [[Google Scholar](#)]
15. Starowicz K. Role of endocannabinoid system in pathogenesis of osteoarthritic pain. Proceedings of the 25th Anniversary Symposium of the International Cannabinoid Research Society; 2015 Jun 28–Jul 3; Wolfville, Nova Scotia, Canada. [[Google Scholar](#)]
16. Liu A. Therapeutic efficacy of a peripherally restricted CB1R antagonist/AMPK activator in diet-induced obesity/metabolic syndrome. Proceedings of the 25th Anniversary Symposium of the International Cannabinoid Research Society; 2015 Jun 28–Jul 3; Wolfville, Nova Scotia, Canada. [[Google Scholar](#)]
17. Bergamaschi MM, Queiroz RH, Zuardi AW, Crippa JA. Safety and side effects of cannabidiol, a Cannabis sativa constituent. *Curr Drug Saf*. 2011 Sep 1;6(4):237–49. doi: 10.2174/157488611798280924. [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
18. Ferreira VR, Carvalho LB, Ruotolo F, de Moraes JF, Prado LB, Prado GF. Sleep disturbance scale for children: translation, cultural adaptation, and validation. *Sleep Med*. 2009 Apr;10(4):457–63. doi: 10.1016/j.sleep.2008.03.018. [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
19. Birmaher B, Khetarpal S, Cully M, Brent D, McKenzie S. Screen for child anxiety related disorders (SCARED) [Internet] Pittsburgh, PA: Western Psychiatric Institute and Clinic, University of Pittsburgh; 1995. Oct, [cited 2016 Apr 26]. Available from: www.pediatricbipolar.pitt.edu/content.asp?id=2333#3304. [[Google Scholar](#)]
20. Pace-Schott EF, Germain A, Milad MR. Sleep and REM sleep disturbance in the pathophysiology of PTSD: the role of extinction memory. *Biol Mood Anxiety Disord*. 2015 May 29;5:3. doi: 10.1186/s13587-015-0018-9. [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
21. Hsiao YT, Yi PL, Li CL, Chang FC. Effect of cannabidiol on sleep disruption induced by the repeated combination tests consisting of open field and elevated plus-maze in rats. *Neuropharmacology*. 2012 Jan;62(1):373–84. doi: 10.1016/j.neuropharm.2011.08.013. [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]