ISSN : 2350-0743

International Journal of Recent Advances in Multidisciplinary Research

Vol. 12, Issue 08, pp.11552-11556, August, 2025

RESEARCH ARTICLE

MANAGEMENT OF PSYCHIATRIC MANIFESTATIONS IN A PATIENT WITH BORDERLINE PERSONALITY DISORDER AND HEREDITARY AMYLOIDOSIS: A CASE REPORT

Parinda Parikh M.D.[1], Eunsaem Lee M.D.[2], Namitha Maria Mathew M.D.[3], Rithika Narravula B.S.[4], Dilinuer Wubuli M.D.[5], Isa Emre Gultekin.[6], Arushi Chandra-Kaushik B.A.[7] Chana Simanowitz FNP [8], Amanda Mineo BSN R.N.[9] and Mina Oza M.D[10]

[1]Department of Psychiatry, Weill Cornell Medical College, White Plains, USA; [2]Hangil Hospital, Jeonju, South Korea; [3]Amala Institute of Medical Sciences, Thrissur, Kerala, India; [4]University of Pittsburgh, PA, USA; [5]Department of Neurology, Toronto Western Hospital, Ontario, Canada; [6]Ankara University Medical School, Ankara, Turkey; [7]NYU Steinhardt School of Culture, Education, and Human Development, New York, USA; [8]2ND ARC Associates, White Plains, USA; [9]2ND ARC Associates, White Plains, USA; [10]2ND ARC Associates, White Plains, USA

ARTICLE INFO

Article History

Received 24th May, 2025
Received in revised form
27th June, 2025
Accepted 20th July, 2025
Published online 25th August, 2025

Keywords:

Borderline personality disorder, Hereditary amyloidosis, Apolipoprotein A-1, Familial Amyloidosis.

Corresponding author:
MAKOUNDOU Alaric

ABSTRACT

Background: Hereditary amyloidosis is a rare systemic disorder with variable organ involvement, including the liver, kidney and nervous system. Recent evidence suggests that central nervous system involvement in amyloidosis may exacerbate psychiatric symptoms via vascular and structural brain changes. **Case presentation:** We describe a 35-year-old woman with this condition who developed chronic affective instability, suicidality, and anxiety which were aggravated after the liver transplantation she underwent five years prior. Her psychiatric profile led to suspect Borderline personality disorder, raising challenges in diagnosis and management. She had a background of early childhood sexual trauma, emotional neglect, and social dysfunction, which likely contributed to her emotional dysregulation. In circumstances like this, individualized psychotherapeutic interventions are essential to alleviate stress associated with somatic and social factors. Furthermore, due to the potential risk of hepatic and renal impairment, pharmacologic treatment was administered with caution. **Conclusion:** This case represents the first reported treatment of a patient with co-occurring borderline personality disorder and amyloidosis. Reports describing the clinical overlap between the neuropsychiatric manifestations of systemic disease and personality pathology are exceedingly rare, making this observation noteworthy. Further detailed research is warranted to elucidate the underlying mechanisms and optimize management strategies for such complex presentations.

Citation: Parinda Parikh M.D., Eunsaem Lee M.D., Namitha Maria Mathew M.D., Rithika Narravula B.S., Dilinuer Wubuli M.D., Isa Emre Gultekin., Arushi Chandra-Kaushik B.A., Chana Simanowitz FNP, Amanda Mineo BSN R.N., Mina Oza M.D. 2025. "Management of Psychiatric Manifestations in a Patient with Borderline personality disorder and Hereditary Amyloidosis: A Case Report.". *International Journal of Recent Advances in Multidisciplinary Research,* 12, (08), 11552-11556.

INTRODUCTION

Borderline Personality Disorder is a severe and multifaceted psychiatric illness characterized by pervasive emotional instability, impulsive behavior, unstable interpersonal relationships, and identity disturbance (1). Individuals with Borderline Personality Disorder often experience intense fear of abandonment, chronic feelings of emptiness, and recurrent self-harm or suicidal behaviors (1). Epidemiological studies estimate its lifetime prevalence at approximately 1-3% of the general population, affecting approximately 10% of psychiatric outpatients and up to 20% of inpatients (2, 3). Borderline Personality Disorder is frequently accompanied by comorbid conditions such as major depressive episodes, generalized anxiety disorder, and dissociative symptoms. This significantly complicates both its clinical course and management (4-6). Amyloidosis is a group of disorders that is characterized by the extracellular deposition of insoluble, misfolded protein fibrils called amyloid. These fibrils disrupt normal tissue architecture, leading to progressive multiorgan failure. The condition can be classified broadly as either acquired or hereditary (7). Hereditary Amyloidosis, known as Familial Amyloidosis, is an autosomal dominant condition resulting from mutations in genes coding for amyloidogenic proteins. The most common form is the transthyretin (TTR) amyloidosis. However, several non-TTR variants do exist but are much rarer (8). The incidence of Amyloidosis is approximately 1 case per 100,000 person-years in Western countries. In the United States, there are approximately 1275 to 3200 new cases per year (9). Despite the severity of the disease, little is known about the psychiatric manifestations

and their treatment in patients with Hereditary Amyloidosis. Most common features are mood instability, fatigue, and cognitive impairment, which can result either from direct central nervous system (CNS) involvement or as secondary consequences of coping with a chronic multisystem illness (10). Comprehensive treatment should encompass both psychological symptoms and neurobiological factors, such as hepatic and renal involvement. This case describes a patient with Hereditary Amyloidosis who was later diagnosed with Borderline Personality Disorder, followed by a notable worsening and broadening of psychiatric symptoms, including depressive episodes, heightened anxiety, and psychosomatic distress. This presentation highlights the need to recognize how systemic illness may intersect with personality pathology, influencing both diagnostic clarity and integrated management strategies for optimal patient care.

CASE PRESENTATION

A 35-year-old woman presented with affective instability, suicidal ideation. She had a known diagnosis of Hereditary Apolipoprotein A-I Amyloidosis and had undergone liver transplantation at age 30. Four years post her liver transplantation, she became increasingly emotionally unstable and was subsequently hospitalized four times in a psychiatric ward due to suicidal ideation, wherein she was diagnosed with Borderline Personality Disorder. According to the Diagnostic and Statistical Manual of Mental Disorders (DSM-5 TR), she met the diagnostic criteria for Borderline Personality Disorder, a pervasive pattern of emotional instability, feelings of emptiness, fear of rejection, and suicidal ideation. Although the patient's mother was emotionally neglectful and unresponsive toward her, there were no developmental delays. She experienced significant difficulty maintaining peer relationships. She was sexually abused by her relatives in early elementary school. Subsequently, she demonstrated poor concentration and disruptive behavior in the classroom. She exhibited significant mood lability, persistent depressive symptoms, feelings of worthlessness, and elevated levels of anxiety. She was highly sensitive to criticism and displayed fear of rejection. She also expressed uncertainty regarding her gender identity and sexual orientation. She is maintained on immunosuppressive therapy with antidepressant and anxiolytic medications alongside psychotherapy. Her current pharmacological regimen includes sertraline 150mg, clonazepam 0.5mg, buspirone 15mg, gabapentin 100mg, topiramate 100mg, melatonin 5mg, and tacrolimus 6mg. Following hospital discharge, she has continued individualized behavioral psychotherapy and trauma-focused therapy in the outpatient clinic.

DISCUSSION

Borderline Personality Disorder is believed to arise from an interplay between genetic vulnerability and early life trauma, such as childhood abuse or neglect, which significantly increases the risk for developing the disorder (11). These environmental and genetic factors contribute to neurotransmitter dysregulation, including decreased serotonergic activity, which is linked to impulsivity, aggression, increased dopaminergic and noradrenergic activity, which may underlie emotional reactivity and stress sensitivity (12). This imbalance is accompanied by structural and functional changes in key brain areas, notably increased amygdala activity, reduced volume, and hypoactivation of the prefrontal cortex, which is associated with poor conflict monitoring and regulation of emotional responses (13). These neurobiological alterations culminate in the core features of Borderline Personality Disorder, including affective instability, impulsive behaviors, distorted self-image, and unstable interpersonal relationships (3). Amyloidosis encompasses a diverse group of disorders characterized by the extracellular deposition of insoluble protein or peptide aggregates arranged in a β-pleated sheet conformation, resulting in the formation of amyloid fibrils (14). More than 26 precursor proteins have been identified as capable of forming amyloid deposits (15). These deposits may be acquired or inherited in origin and can occur either locally or as part of a systemic process. Hereditary Amyloidosis arises from germline mutations that increase the amyloidogenic potential of specific proteins under certain physiological conditions. Known genetic variants of transthyretin, apolipoprotein A-I (apoA-I), apolipoprotein A-II, fibrinogen Aα-chain, gelsolin, and lysozyme have been implicated in hereditary forms of the disease (16). ApoA-I is a 28-kDa plasma protein synthesized in the liver and small intestine, and it is the main protein of high-density lipoprotein (HDL) (17). ApoA-I Amyloidosis may occur in a non-hereditary form, characterized by wild-type protein deposition in atherosclerotic plaques or in a hereditary form involving systemic deposition of variant proteins (17). A study of 253 carriers of the Leu75Pro variant found that 62% developed Amyloidosis while 38% remained asymptomatic (18). Renal (11%), cardiac (6%), and hepatic (5%) involvement occurs, respectively (19). Neurological involvement commonly presents as hoarse voice/dysphonia, polyneuropathy, and dermatologic lesions (15, 20-26). And there is a case report with Amyloidosis who presented with auditory perseveration (30). Although the pathophysiology of CNS manifestations remains unclear, some studies suggest that amyloid can accumulate as intracellular and extracellular aggregates, triggering secondary pathogenic cascades (10, 27). Leptomeningeal and cortical small- to medium-sized arteries and arterioles are most frequently affected by amyloid deposition. In advanced cerebral amyloid angiopathy, affected vessels exhibit acellular wall thickening with a smudgy appearance on haematoxylin and eosin-stained sections (27). On the other hand, there is a paucity of literature addressing the psychiatric manifestations in Amyloidosis. Several studies involving patients with Amyloidosis have identified a high prevalence of mood disturbances, with approximately 30% of patients exhibiting symptoms of anxiety and depression (31-34). More recent findings suggest an even greater burden, with 48.6% of patients reporting clinically significant levels of either depression or anxiety (34). Additionally, small vessel pathology has been implicated in the development of neuropsychiatric symptoms such as depression, apathy, and emotional lability (35). The psychiatric manifestations observed in Amyloidosis show considerable overlap with the clinical features of Borderline Personality Disorder, including affective instability, heightened anxiety, and anergia. Therefore, greater attention should be given to these shared features during the diagnostic process. Given the complex and heterogeneous nature of Borderline Personality Disorder, a comprehensive and individualized approach to treatment is essential. General therapeutic strategies include establishing clear boundaries, maintaining effective communication, and addressing maladaptive behaviors (36). Although there remains some controversy regarding optimal treatment modalities, psychotherapy is widely regarded as the first-line

intervention for borderline personality disorder. Among the available approaches, dialectical behavior therapy (DBT) and mentalization-based therapy (MBT) have demonstrated the strongest evidence for efficacy (37). DBT, a form of cognitive behavioral therapy (CBT), has been shown to reduce self-injurious behaviors, suicidal attempts, and the frequency of psychiatric hospitalizations (38). In patients with comorbid medical conditions such as Amyloidosis, psychopharmacologic interventions require careful consideration due to potential organ involvement and drug metabolism concerns. In such cases, non-pharmacologic, evidence-based psychotherapies are generally preferred. Nonetheless, psychotropic medications may be used to alleviate specific emotional and behavioral symptoms. While no pharmacologic agents are currently approved by the U.S. Food and Drug Administration (FDA) for the treatment of Borderline Personality Disorder, selective serotonin reuptake inhibitors (SSRIs) and atypical antipsychotics such as quetiapine are commonly prescribed off-label (39). Given the potential for hepatic and renal comorbidities in patients with Amyloidosis, clinicians must exercise caution with respect to drug selection, dosage, and route of administration.

CONCLUSION

This case highlights the complex interplay between a rare systemic condition, Hereditary Amyloidosis, and prominent psychiatric manifestations in Borderline Personality Disorder. The presence of early psychosocial adversity, including trauma and neglect, likely contributed to emotional dysregulation, while CNS involvement may have contributed to symptom severity, although the causal relationship remains uncertain. Given the overlapping features between systemic disease and personality pathology, careful diagnostic evaluation is essential. Furthermore, hepatic and renal comorbidities in Amyloidosis necessitate caution in psychopharmacologic management. This case underscores the importance of multidisciplinary collaboration and supports the use of individualized, non-pharmacologic interventions such as evidence-based psychotherapy in medically complex patients presenting with psychiatric symptoms. Further research is warranted to better understand the complexities of the neuropsychiatric impact of Amyloidosis and guide more individualized treatment approaches in patients with comorbid psychiatric and medical conditions.

Declarations

ACKNOWLEDGEMENT

The authors would like to thank colleagues for their support and guidance in the preparation of this case report.

Source of Funding: None

Conflict of Interest: None

Glossary of Abbreviations

Central nervous system (CNS)
Cognitive behavioral therapy (CBT)
Diagnostic and Statistical Manual of Mental Disorders (DSM-5 TR)
Dialectical behavior therapy (DBT)
High-density lipoprotein (HDL)
Mentalization-based therapy (MBT)
Food and Drug Administration (FDA)
Selective serotonin reuptake inhibitors (SSRIs)
Transthyretin (TTR)

REFERENCES

1. Lieb, K., Zanarini, M. C., Schmahl, C., Linehan, M. M., & Bohus, M. (2004). Borderline personality disorder. *Lancet (London, England)*, *364*(9432), 453–461.*https://doi.org/ 10.1016/S0140-6736(04)16770-6*
2. Lenzenweger, M. F., Lane, M. C., Loranger, A. W., & Kessler, R. C. (2007). DSM-IV personality disorders in the National Comorbidity Survey Replication. *Biological psychiatry*, *62*(6), 553–564. *https://doi.org/10.1016 /j. biopsych. 2006.09.019*
3. Skodol, A. E., Gunderson, J. G., Pfohl, B., Widiger, T. A., Livesley, W. J.et al.(2002). The borderline diagnosis I: psychopathology, comorbidity, and personality structure. *Biological psychiatry*, *51*(12), 936–950.*https://doi.org /10.1016/s0006-3223(02)01324-0*
4. Grambal, A., Prasko, J., Kamaradova, D., Latalova, K., Holubova, M. et al. (2016). Quality of life in borderline patients comorbid with anxiety spectrum disorders - a cross-sectional study. *Patient preference and adherence*, *10*, 1421–1433.*https://doi.org/10.2147/PPA.S108777*
5. Tomasetti, C., Autullo, G., Ballerini, A., de Bartolomeis, A., Dell'Osso, B.et al. (2024). Treating depression in patients with Borderline personality disorder: clinical clues on the use of antidepressants. *Annals of general psychiatry*, *23*(1), 21.*https: //doi.org/10.1186/s12991-024-00507-z*
6. Liu, Y., Chen, C., Zhou, Y., Zhang, N., & Liu, S. (2024). Twenty years of research on Borderline personality disorder: a scientometric analysis of hotspots, bursts, and research trends. *Frontiers in psychiatry*, *15*, 1361535.*https://doi.org/10.3389/fpsyt.2024.1361535*
7. Merlini, G., & Bellotti, V. (2003). Molecular mechanisms of amyloidosis. *The New England journal of medicine*, *349*(6), 583–596.*https://doi.org/10.1056/NEJMra023144*
8. Planté-Bordeneuve, V., & Said, G. (2011). Familial amyloid polyneuropathy. *The Lancet. Neurology*, *10*(12), 1086–1097.*https://doi.org/10.1016/S1474-4422(11)70246-0*
9. Bustamante, J. G. (2023, July 31). Amyloidosis. StatPearls (Internet). https://www.ncbi. nlm.nih.gov/ books/ NBK470285/
10. Poli, L., Labella, B., Cotti Piccinelli, S., Caria, F., Risi, B. et al. (2023). Hereditary transthyretin amyloidosis: a comprehensive review with a focus on peripheral neuropathy. *Frontiers in Neurology*, *14*, 1242815.*https://doi.org/ 10.3389/fneur.2023.1242815*
11. Zanarini M. C. (2000). Childhood experiences associated with the development of Borderline personality disorder. The Psychiatric Clinics of North America, 23(1), 89–101.https://doi.org/10.1016/s0193-953x(05)70145-3
12. Gurvits, I. G., Koenigsberg, H. W., & Siever, L. J. (2000). Neurotransmitter dysfunction in patients with Borderline personality disorder. The Psychiatric Clinics of North America, 23(1), 27–vi.https://doi.org/10.1016/s0193-953x(05)70141-6
13. Soloff, P., Nutche, J., Goradia, D., & Diwadkar, V. (2008). Structural brain abnormalities in Borderline personality disorder: a voxel-based morphometry study. Psychiatry

research, 164(3), 223–236.https://doi.org/ 10.1016/ j.pscychresns.2008.02.003
14. Obici, L., Franceschini, G., Calabresi, L., Giorgetti, S., Stoppini, M. et al. (2006). Structure, function, and amyloidogenic propensity of apolipoprotein A-I. *Amyloid: the international journal of experimental and clinical investigation: the official journal of the International Society of Amyloidosis*, *13*(4), 191–205. https://doi.org /10.1080/13506120600960288
15. Eriksson, M., Schönland, S., Yumlu, S., Hegenbart, U., von Hutten, H., Gioev et al. (2009). Hereditary apolipoprotein AI-associated amyloidosis in surgical pathology specimens: identification of three novel mutations in the APOA1 gene. *The Journal of molecular diagnostics: JMD*, *11*(3), 257–262. https://doi.org/ 10.2353/ jmoldx.2009.080161
16. Benson, M. D., & Kincaid, J. C. (2007). The molecular biology and clinical features of amyloid neuropathy. *Muscle & nerve*, *36*(4), 411–423. https://doi.org/ 10.1002 /mus.20821
17. Sorci-Thomas, M. G., & Thomas, M. J. (2002). The effects of altered apolipoprotein A-I structure on plasma HDL concentration. *Trends in cardiovascular medicine*, *12*(3), 121–128. https://doi.org/10.1016/s1050-1738(01)00163-3
18. Gregorini, G., Izzi, C., Ravani, P., Obici, L., Dallera, N., Del Barba, A., Negrinelli, A., Tardanico, et al. (2015). Tubulointerstitial nephritis is a dominant feature of hereditary apolipoprotein A-I amyloidosis. *Kidney International*, *87*(6), 1223–1229. https://doi.org/ 10.1038/ ki.2014.389
19. Cohen, O. C., Blakeney, I. J., Law, S., Ravichandran, S., Gilbertson, J., Rowczenio, D., … Wechalekar, A. D. (2022). The experience of hereditary apolipoprotein A-I amyloidosis at the UK National Amyloidosis Centre. *Amyloid*, *29*(4), 237–244. https://doi.org/ 10.1080/ 1350 6129.2022.2070741
20. Rowczenio, D., Dogan, A., Theis, J. D., Vrana, J. A., Lachmann, H. J., Wechalekar, A. D., Gilbertson, J. A., et al. (2011). Amyloidogenicity and clinical phenotype associated with five novel mutations in apolipoprotein A-I. *The American journal of pathology*, *179*(4), 1978–1987. https://doi.org/10.1016/j.ajpath.2011.06.024
21. Hamidi Asl, K., Liepnieks, J. J., Nakamura, M., Parker, F., & Benson, M. D. (1999). A novel apolipoprotein A-1 variant, Arg173Pro, is associated with cardiac and cutaneous amyloidosis. *Biochemical and biophysical research communications*, *257*(2), 584–588. https://doi.org/ 10. 1006/ bbrc.1999.0518
22. de Sousa, M. M., Vital, C., Ostler, D., Fernandes, R., Pouget-Abadie, J., Carles, D., & Saraiva, M. J. (2000). Apolipoprotein AI and transthyretin as components of amyloid fibrils in a kindred with apoAI Leu178His amyloidosis. *The American journal of pathology*, *156*(6), 1911–1917. https://doi.org/10.1016/S0002-9440(10) 65064-X
23. Hazenberg, A. J., Dikkers, F. G., Hawkins, P. N., Bijzet, J., Rowczenio, D., Gilbertson, J., et al. (2009). Laryngeal presentation of systemic apolipoprotein A-I-derived amyloidosis. *The Laryngoscope*, *119*(3), 608–615. https://doi.org/10.1002/lary.20106
24. Nichols, W. C., Gregg, R. E., Brewer, H. B., Jr, & Benson, M. D. (1990). A mutation in apolipoprotein A-I in the Iowa type of familial amyloidotic polyneuropathy. *Genomics*, *8*(2), 318–323. https://doi.org/10.1016/0888-7543(90) 90288-6
25. Obici, L., Bellotti, V., Mangione, P., Stoppini, M., Arbustini, E., Verga, L., Zorzoli, I., et al. (1999). The new apolipoprotein A-I variant leu(174) --> Ser causes hereditary cardiac amyloidosis, and the amyloid fibrils are constituted by the 93-residue N-terminal polypeptide. *The American journal of pathology*, *155*(3), 695–702. https://doi.org/10.1016/S0002-9440(10)65167-X
26. Hamidi Asl, L., Liepnieks, J. J., Hamidi Asl, K., Uemichi, T., Moulin, G., Desjoyaux, E, et al. (1999). Hereditary amyloid cardiomyopathy caused by a variant apolipoprotein A1. *The American journal of pathology*, *154*(1), 221–227. https://doi.org/10.1016/S0002-9440(10)65268-6
27. Revesz, T., Holton, J. L., Lashley, T., Plant, G., Frangione, B., Rostagno, A., & Ghiso, J, et al. (2009). Genetics and molecular pathogenesis of sporadic and hereditary cerebral amyloid angiopathies. *Acta neuropathologica*, *118*(1), 115–130. https://doi.org/10.1007/s00401-009-0501-8
28. (PDF) Neurobiology of Alzheimer's disease. (n.d.). https://www.researchgate.net/publication/26800518_Neuro biology_of_Alzheimer's_disease
29. Cohen, O. C., Blakeney, I. J., Law, S., Ravichandran, S., Gilbertson, J., Rowczenio, D., … Wechalekar, A. D. (2022). The experience of hereditary apolipoprotein A-I amyloidosis at the UK National Amyloidosis Centre. *Amyloid*, *29*(4), 237–244.https://doi.org/10.1080/13506129.2022.2070741
30. Martins-Correia, J., Sousa, L. Palinacousis in amyloidosis: exploring the hallucinatory phenomenon in brain pathology—a case report. *J Med Case Reports* 18, 345 (2024).https://doi.org/10.1186/s13256-024-04575-3
31. Stephen Lo, Janet Shu, Margot Phillips, Fangui Sun, John L Berk, Vaishali Sanchorawala; Symptoms of Depression and Anxiety Assessed By the SF-36 Questionnaire in Patients with AL Amyloidosis. *Blood* 2015; 126 (23): 3299. doi: https://doi.org/10.1 182/blood. V126.23.3299.3299
32. Shu, J., Lo, S., Phillips, M., Sun, F., Seldin, D. C., Berenbaum, I., Berk, J. L., & Sanchorawala, V. (2016). Depression and anxiety in patients with AL amyloidosis as assessed by the SF-36 questionnaire: experience in 1226 patients. *Amyloid: the international journal of experimental and clinical investigation: the official journal of the International Society of Amyloidosis*, *23*(3), 188–193. https://doi.org/10.1080/13506129.2016.1208081
33. Lopes, A., Fonseca, I., Sousa, A., Rodrigues, C., Branco, M., Coelho, T, et al. (2018). Psychopathological dimensions in subjects with hereditary ATTR V30M amyloidosis and their relation with life events due to the disease. *Amyloid: the international journal of experimental and clinical investigation: the official journal of the International Society of Amyloidosis*, *25*(1), 26–36. https://doi.org/10.1080/13506129.2018.1428795
34. Smorti, M., Ponti, L., Soffio, F., Argirò, A., Perfetto, F., Zampieri, M., Mazzoni, C, et al. (2023). Prevalence of anxiety and depression symptoms in a sample of outpatients with ATTR cardiac amyloidosis. *Frontiers in Psychology, 13*, 1066224.https://doi.org/ 10.3389/ fpsyg.2022.1066224
35. Markus, H. S., & de Leeuw, F. E. (2023). Cerebral small vessel disease: Recent advances and future directions. *International journal of stroke: official journal of the International Stroke Society*, *18*(1), 4–14. https://doi.org/ 10.1177/17474930221144911

36. JA;, M.-M. M. J. (n.d.). Borderline personality disorder. American family physician.https://pubmed.ncbi.nlm.nih.gov/35166488/
37. Storebø, O. J., Stoffers-Winterling, J. M., Völlm, B. A., Kongerslev, M. T., Mattivi, J. T., Jørgensen, et al. (2020). Psychological therapies for people with borderline personality disorder. *The Cochrane database of systematic reviews*, *5*(5), CD012955. https://doi.org/10.1002/ 14651858.CD012955.pub2
38. Cristea, I. A., Gentili, C., Cotet, C. D., Palomba, D., Barbui, C., & Cuijpers, P. (2017). Efficacy of Psychotherapies for Borderline Personality Disorder: A Systematic Review and Meta-analysis. *JAMA psychiatry*, *74*(4), 319–328. https://doi.org/10.1001/ jamapsychiatry. 2016.4287
39. Stoffers J;Völlm BA;Rücker G;Timmer A;Huband N;Lieb K; (n.d.). Pharmacological interventions for Borderline Personality Disorder. The Cochrane database of systematic reviews. https://pubmed.ncbi.nlm.nih.gov/20556762/

****