fortune Journals

Review Article

JOURNAL OF PSYCHIATRY AND PSYCHIATRIC DISORDERS

ISSN: 2572-519X

Transition from Attention-Deficit/Hyperactivity Disorder (ADHD) to Borderline Conditions with Self-Harm, Affective Dependency, and Refractory Depression: Role of Dopamine Dysregulation, Secondary Gains, and Integrated Therapeutic Strategies

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Abstract

Patients—often female—with a prior diagnosis of ADHD evolve, in adolescence or adulthood, into borderline personality conditions, characterized by affective instability, self- harm, intense emotional dependency, refractory depressive episodes, and recurrent psychoactive substance use. The central hypothesis is the existence of a dopaminergic dependence syndrome, associated with temperamental and constitutional vulnerabilities, with an overlap between DSM Axis II aspects and neurochemical imbalances. This article proposes an integrated approach, involving typical antipsychotics (such as haloperidol), lithium, and intensive psychiatric psychotherapy, advocating Henri Ey's organic-dynamic model as an explanatory paradigm.

Keywords: ADHD; Borderline; Dopamine; Self-harm; Haloperidol; Personality; Organo- dynamism.

Introduction

The evolution of patients with childhood ADHD into borderline spectrum conditions in adulthood is a frequently observed phenomenon in clinical practice. The most common pattern involves young females who, after a trajectory marked by impulsivity and attention deficit, develop affective dependency, self-harm, substance abuse, and difficult-to-treat depressions [1-4]. Population studies indicate a high comorbidity rate between ADHD and borderline personality disorder (BPD), as well as between ADHD, BPD, and bipolar mood disorder (BMD); or between the latter (ADHD, BPD, BMD) and substance abuse disorders, instinctive disorders, eating disorders, impulse control disorders, anxiety disorder, depression; all with significant impact on chronicity and refractoriness [2, 5]. We prefer to speak of "thymopathy" rather than "bipolar disorder" because many of these patients have emotional, affective, and energetic dysregulation that does not necessarily pass through what we know as depression or mood exaltation (bipolarity). Therefore, the term "bipolar," in our view, does not adequately apply here.

Neurobiological Hypotheses and Dopaminergic Thymopathies

There is evidence that the common dopaminergic basis of ADHD and impulsive borderline conditions is involved in the chronification of these symptoms [6]. Robust evidence of "dis- dopaminergia" in ADHD,

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Received: July 10, 2025
Accepted: July 16, 2025
Published: July 19, 2025

Marcelo Caixeta., J Psychiatry Psychiatric Disord 2025
DOI:10.26502/jppd.2572-519X0252

BPD; there is even an overlap of a "dopamine dependence syndrome" (hypo-sensitive receptors) with ADHD. Patients would need a higher than normal dose of dopamine to "feel satisfied." On the other hand, it is a common observation in psychiatric clinics that the use of dopaminergic or, especially, noradrenergic medications can, paradoxically, worsen the affective (thymopathic) picture, producing irritability, insomnia, aggression, and mixed states [7-9]. Such side effects suggest the presence of a complex dopaminergic syndrome, in which the same neurochemical substrate that improves apathy can trigger affective disorganization [10] in those patients who, previously only hyperactive/hypoprosexic, now also have a severe anxious-depressive-self-mutilating- suicidal-impulsive thymopathy associated. Such patients tend to worsen with antidepressants, especially noradrenergic-dopaminergic, catecholaminergic ones. They become more irritable, dysphoric, anxious, impulsive, suicidal, with akathisia symptoms (especially associated with lithium and neuroleptics). Anticonvulsants can be useful for anxiety, especially valproate, but can, in the medium term, worsen depression—perhaps because they lower folic acid, important for serotonin production. Lithium alone also does not produce depressive/suicidal improvements in the short term; it needs to act much longer. In the short term, especially when suicidality or self-harm is intense, the use of a neuroleptic, in this case haloperidol, is necessary. It is necessary, however, to know how to use it, as it can induce akathisia and worsen the "noradrenergic" picture, with high suicidality.

These patients frequently present a disinhibited and hyperactive temperament in childhood, which transforms into anxious restlessness and, subsequently, into depressive and self- destructive symptoms [11]. This is a constitutive model, based on temperamental pathology, as described by Henri Ey in his concept of organo-dynamism, where organic and psychic factors interact continuously and pathologically [12]. Evolutionarily, a child's energy goes into play; in adults, it goes into worrying, hence the emergence of anxiety, and from that, at very high levels, into depression.

Psychodynamics, Secondary Gains, Drug Addiction, and Affective Dependency

The persistence of pejorative clinical symptoms after pharmacological improvement can be explained by mechanisms of secondary gain: the patient learns that by appearing fragile, they receive more attention and care from others [13]. Furthermore, drug addiction often arises as an attempt at self-medication: alcohol, cannabis, benzodiazepines, or stimulants are used to "regulate" mood or combat existential boredom [14]. Even after symptomatic control with drugs, substance dependence can persist as a second autonomous pathology, hindering remission and promoting relapses [15]. It becomes a "second nature," a "second pathology," requiring psychotherapy that the psychiatrist usually does not provide. The clinical picture is complex; psychodynamic factors mix with psychobiological factors in a very intricate way. For example, "bad psychobiological energy" in these cases can arise from three different sources: (1) the energy of the constitution itself/ADHD; (2) the anxious energy of thymopathy that manifests in adolescence, under the influence of "adolescent hormonopathy." (3) energy caused iatrogenically by akathisia, noradrenergia. This energy, in turn—following Ey's organodynamic model [12]—fuels purely psychodynamic/psychosocial factors. And psychodynamic/psychosocial/psychofamilial factors, when not properly treated, do not allow psychobiological improvement to stabilize (for example, one of our patients, very affectively avid—because of her hyperactive/borderline constitution—wanted to stay in the hospital, wanted the hospital psychiatrist to "adopt" her, and thus exaggerated symptoms, engaged in metassimulations, "did not want to get better." If the psychiatrist had not been so attentive to her psychodynamics, they would have kept medicating nonexistent, exaggerated, or modified symptoms due to the patient's psychopathology. Complex psychodynamics that end up interfering with psychobiological treatment. Another example is the patient who is psychobiologically well but resorts to old addictions, just for hedonism, just because they "have nothing to do," just because they want adventure, adrenaline, just because they want to escape their "boring normal life," and this again worsens their Axis I (according to the DSM system, the axis of clinically, psychiatrically manifest illness). So, without treating (psychotherapeutically, or with haloperidol) the underlying pathological constitution, hyperactivity, temperamental pathology, one cannot adequately treat Axis I (manifest illness).

An example is the patient who, because of her avid, voracious, oral constitution, requires a lot of affection from her mother, but the mother cannot provide it because she works, because she has another daughter, because she has to be somewhat "tough" with her daughter, since the father is lenient with her in terms of school or tasks to do. Such constitutional symptoms may worsen due to the anxiety/depression of adolescent hormonopathic thymopathy, but even if these biological changes are treated, the patient may not "want to get better." She may want to remain "a little sick," to attract the attention of her psychiatrist, her mother, because, during the illness, she received more attention. Or she may become more sadistic with her mother, because now the doctor has "replaced her mother," and so she can be sadistic with her mother, thus testing whether her mother "really likes me," having that pleasure of being hostile to her mother and her mother still forgiving and loving her. Such psychodynamic movements can worsen psychobiology, just as psychobiology can provide basic energy for these movements.

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Psychodynamic Implications and Failures of the Fragmented Approach

The traditional approach, where the psychiatrist merely prescribes and refers the patient for psychotherapy with another professional, proves ineffective in these cases [12, 16]. This is because the core of the suffering lies precisely in the interaction between biological and relational layers. The psychologist often lacks knowledge of the neurochemical or clinical bases of impulsivity and refractory depression, while the psychiatrist, focused on prescription, neglects the affective history, traumas, and family and psychosocial dynamics [17].

Integrated Pharmacotherapy: Haloperidol, Lithium, and Psychiatric Psychotherapy

In these cases, we observe a significant response to the use of haloperidol (higher doses in cases of intense suicidality, lower doses for maintenance), with an ataractic, antidepressant, and impulsivity-stabilizing effect [18, 19]. Lithium also proves effective, especially in cases with a bipolar type II pattern or mood instability-affective dependence-drug addiction (addictive behaviors). Unfortunately, lithium does not usually act quickly in these cases, sometimes taking 3 to 6 months to produce a stabilizing effect. Until then, these patients will require a neuroleptic strategy to emerge from suicidal or self-mutilating risk.

The ideal treatment requires:

*Mood and thought stabilization with haloperidol and lithium.

*Gradual reduction of psychoactive substances.

*Intensive psychiatric psychotherapy (as per, among others, our "M. Caixeta 2023 – Psychotherapeutic Psychiatry, Sparta Publishing House"), [19], aimed at reconstructing ego autonomy and elaborating pathological affective bonds [20].

(*) Specific Pharmacological Data

Haloperidol is a typical antipsychotic (first generation) that primarily acts as an antagonist of D_2 dopaminergic receptors. It is widely used in the treatment of psychoses, agitation, delirium, and also in impulsive conditions and refractory personality disorders—as you have explored in your articles.

Below, I highlight the main clinical and psychodynamic aspects of haloperidol, relevant this work:

*Class: Butyrophenone antipsychotic.

*Mechanism: D_2 antagonism in the mesolimbic and nigrostriatal system; extrapyramidal side effects come from the latter.

*Half-life: 12–36 hours (varies oral vs IM decanoate).

Main Clinical Indications

*Schizophrenia

*Delirium and acute agitation

*Tic disorders (such as Tourette's syndrome)

*Substance-induced psychoses

*Acute mania

*Impulsivity and self-aggression in personality disorders (increasing off-label use)

Uses in Personality Disorders (such as Borderline)

Its ataractic effect (calming and reducing internal tension) is notable even at low doses, without excessive sedation. Borderline patients frequently report:

*Reduction of impulsive-affective mixed states

*Containment of self-harm

*Lower reactivity to rejection

*More structured sleep without emotional "blunting"

Uses in Depression

There are also reports of a paradoxical antidepressant effect, especially in patients with dopaminergic instability or post-stimulant syndrome.

Adverse Effects

*Extrapyramidal symptoms (dystonia, akathisia, parkinsonism)

*Tardive dyskinesia (chronic use, especially in the elderly)

*Hyperprolactinemia

*Sedation, hypotension, risk of neuroleptic malignant syndrome

Psychodynamic Basis:

We have articulated the use of haloperidol within the psychodynamic model of "containment of drives" and ego reorganization in patients with borderline or impulsive structures (See Vassilis Kapsambelis – The Medications of Narcissism). It allows the patient to enter psychotherapy with less acting-out, facilitating symbolization and the therapeutic bond. The concept of "psychiatric ataraxia" brings haloperidol closer to the idea of an emotional regulator, almost a stabilizer of ego functions, and not just an antipsychotic. "Haloperidol, even in sub-antipsychotic doses, acts as a modulator of impulsivity and affective reactivity, allowing greater psychotherapeutic engagement. Its ataractic effect facilitates the containment of self-destructive acts and the symbolization of internal conflicts in borderline patients refractory to conventional management [18, 19, 20]."

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Future Directions [20,21,22]

Haloperidol, whether used in depression or in personality disorders, presents a serious problem, namely akathisia. Very high doses — generally above 250 mg/day

— can improve this somewhat, as the medication then enters a range of extrapyramidal protection. These doses are used in our hospital service for cases with high suicidality, agitated psychotic suicidal depression, etc.

As soon as the patient improves, the doses are gradually lowered. Maintenance can be done with 1 mg – 2 mg per day. Even so, there are patients who do not tolerate akathisia, so we use dosing on alternate days, e.g., 1 day on, 3 days off; 1 day on, 5 days off. There are studies showing that withdrawal of haloperidol for a few days can be beneficial for depression in some cases.

In our service, we have started a protocol with the use of droperidol in such cases, as it is very similar to haloperidol and has significantly less akathisia. The half-life of droperidol is short, but this may not greatly affect the treatment of depression, since, as mentioned above, periods of haloperidol withdrawal do not impair depression treatment. The more sedative effect of droperidol also allows it to be used at night, when it can also act as a hypnotic.

Conclusions

Hyperactive patients who evolve into severe borderline conditions, with self-harm, refractory depression, and drug addiction, constitute an important and often neglected clinical subtype. Their condition involves constitutional failures, maladaptive psychodynamic patterns, and dopaminergic alterations. Henri Ey's model, the clinic of personality, and the integration between psychopharmacology and psychotherapy prove fundamental. We advocate that haloperidol, lithium, and the therapeutic bond be the initial pillars of this management.

References

1. Nanking G. Dopamine dysfunction in borderline personality disorder: a hypothesis. J Neural Transm Suppl (2004): 663-672.
2. Joyce PR, Stephenson J, Kennedy M, et al. HTR1A and DAT1 gene variants increase the risk of BPD. Front Genet 4 (2013): 313.
3. Zanarini MC, Frankenburg FR, Reich DB, et al. Persistence of BPD symptoms: 16-year follow-up. Am J Psychiatry (2010): 973-982.
4. Berlin HA, Rolls ET, Iversen SD. Orbitofrontal cortex in BPD and impulsivity. Am J Psychiatry 162 (2005): 2360-2362.
5. Gunderson JG. Borderline personality disorder: A review. JAMA Psychiatry 70 (2011): 180-189.
6. Colle L, Hilviu D, Rossi R, et al. Self-harming and agency in BPD. Front Psychiatry 11 (2020): 449.
7. Paris J. Prescribing in BPD – clinical guidelines. Prescriber 20 (2009): 1257-1262.
8. Stevenson A, Patel K. Drug treatment for BPD. Cochrane Database Syst Rev (2021).
9. Crosby JZ, Bartemes K. Efficacy of psychotropics on self-injury. Transl Psychiatry 12 (2022): 172.
10. Leibenluft E, Gardner DL, Cowdry RW. The borderline self-mutilator. J Pers Disord 1 (1987): 317-324.
11. Stoffers JM, Rücker G, Timmer A, Huband N. Psychological therapies for BPD. Cochrane Database Syst Rev (2012).
12. Ey H. Études Psychiatriques I: Historique – Sémiologie. Paris: Éditions Desclée (1973).
13. Caixeta M. Psiquiatria Cognitiva. São Paulo: Editora Sparta (2023).
14. Caixeta M. Transtornos Psiquiátricos do Desenvolvimento. São Paulo: Editora Sparta (2021).
15. Caixeta M, Caixeta L. Manual Prático da Dependência de Drogas. São Paulo: Editora R & D Publicações (2019).
16. Caixeta M. Psiquiatria da Criança. São Paulo: Editora Sparta (2022).
17. Caixeta M. Psiquiatria Infantil. São Paulo: Editora Sparta (2022).
18. Caixeta M, Caixeta L. Psiquiatria Clínica. São Paulo: Editora Lemos (2020).
19. Caixeta M. Psiquiatria Psicoterápica. São Paulo: Editora Sparta (2024).
20. Caixeta M. Haloperidol em transtornos de personalidade refratários. OSF Preprints (2024).
21. Caixeta M. O Efeito Ataráxico do Haloperidol em Quadros Impulsivos e Transtornos de Personalidade Refratários. Zenodo (2024).
22. Caixeta M. Haloperidol em Depressões Refratárias: Hipóteses Clínicas e Psicodinâmicas Integradas. OSF Preprints (2024).

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