Chapter #25

BIPOLAR AFFECTIVE DISORDER: THE PSYCHODYNAMIC APPROACH OF ETIOLOGY

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ABSTRACT
This chapter represents an attempt to review recent studies on the etiology of bipolar affective disorder from a psychodynamic perspective. The multifactorial etiology of bipolar disorder, although recognized and empirically demonstrated, continues to generate difficulties in understanding because the individual contribution of these factors is generally low, most of them being not specific to bipolar disorder. During the last years, more and more studies focused on investigating the impact that environmental factors have in triggering bipolar disorder. Among these, traumatic childhood events seem to influence the risk of developing bipolar disorder, but the way this occurs remains unclear and needs further examination. The psychodynamic approach of etiology of bipolar disorder does not exclude the impact of genetic and biological factors, but the emphasis is placed on the unique significance that these stressors have for each patient. Exploring, from a psychodynamic perspective, the relational patterns and the defensive and adaptive processes that the patient calls upon can facilitate the understanding of the etiology and related therapeutic implications.

Keywords: psychodynamic psychiatry, resistant depressive episodes, genetic factors, hypomania.

1. INTRODUCTION

The perspective that the present material wishes to emphasize is that currently, in clinical practice, there is a higher recurrence of depressive episodes in bipolar affective disorders, a longer duration of them, and a lower rate of complete and rapid recovery. Thus, currently, depressive episodes in bipolar affective disorders are a therapeutic challenge for both psychiatrists and psychotherapists, due to the high suicidal risk and the emotional "void" that must be processed and with which the "psi" specialist has the duty to empathize. Consequently, this article emphasizes both the pharmacological and the psychoanalytic perspective on the major depressive episode in bipolar affective disorders, more than the particularities of the manic episode are highlighted.

Affective disorders represent a group of illnesses with various etiologies and intensities, with a primary impairment of mood and, adjacently, of thinking and activity. They manifest as depressive, manic, hypomanic and mixed episodes, the evolution of these episodes being cyclical, with good remission. Bipolar affective disorder is a severe mental disorder characterized by dramatic mood swings, abnormal changes in a person's mental state, energy and ability to function. Depressive episodes are followed by manic episodes, the term bipolar reflecting precisely this continuous swing between two extremes, from the depressive pole to the manic one. In DSM-5, bipolar and related disorders, found in Chapter 3, constitute a separate chapter, placed between Schizophrenia Spectrum Disorders and

Other Psychotic Disorders (Chapter 2) and Depressive Disorders (Chapter 4) (American Psychiatric Association, 2013). This positioning suggests the recognition of the role of bipolar disorder as a bridge between the two diagnostic classes, in terms of symptomatology, family history and heritability. DSM-5 abandons the multi-axial formulation of the previous edition, the current formulation being a non-axial one. There is a frequent overlap between affective disorders and psychoses, both symptomatically and syndromologically. Situations of this kind can be encountered in schizophrenia (for example, in post-schizophrenic depression), in schizoaffective disorders, or in schizophreniform disorders associated with mood disorders. On the other hand, many cases of treatment-resistant depression may actually be cases of bipolar depression, mistakenly thought to be unipolar depression.

2. ETIOLOGY

As regards etiology, there is a consensus that the genesis of bipolar disorder is multifactorial, at the junction of biological factors with life events and vulnerability factors. In the last decade, the increased interest in this disorder contributed to the emergence of new evidence about the contribution of genetic factors and their interaction with environmental factors. However, although numerous genetic and environmental risk factors have been identified, their individual contribution is generally small, and most of these factors are not specific to bipolar disorder.

In 2017 University of Michigan published the results of a study carried out throughout 12 years on a sample of 1.100 people, to identify the causes of bipolar disorder (Michigan Medicine, 2017). Out of the 1.100 volunteers, more than 730 suffered from bipolar disorder, and the research team analyzed a myriad of data related to participants' genetics, emotions, life experiences, medical history, motivations, diet, temperament, sleep, and cognitive patterns throughout the study. The research program used 7 phenotypes, including the standard criteria used by doctors to diagnose and monitor bipolar disorder. These phenotypes included: cognitive changes (thinking, reasoning, and emotion processing), psychological dimensions (personality, temperament), motivated behaviors (substance use or abuse), life aspects (family, intimate relationships, and traumas), sleeping patterns and circadian rhythms and indicators related to change in symptoms or response to treatment over time. The results of this research show that there are no single factors - genetic changes, chemical imbalances or life events - that fundamentally contribute to the onset of bipolar disorder, the experiences of patients with this disorder being unique. And yet, they all share certain traits that have been grouped into the 7 categories of phenotypes, accompanied by the following results:

- migraines are 3.5 times more common among people with bipolar disorder than among people without this disorder. Eating and anxiety disorders, alcohol problems, and metabolic syndrome are also more common among people with bipolar disorder;
- people with bipolar disorder reported more frequently the presence of a childhood trauma, associated with changes in self-control and attention;
- persons with bipolar disorder had higher levels of saturated fat in their diet, and researches have shown the connection between the level of certain fat molecules in patients' blood and their emotional state or symptom severity;
- the study has highlighted a low level of key bacteria in the gastrointestinal tract and a lower microbial diversity in patients treated with antipsychotics;
- poor sleep plays a key role in bipolar disorder, connections being identified to the severity of depression and mania in female patients but not in male patients;

- persons with bipolar disorder - and especially men - who also have a strong neurotic tendency are more likely to show a more severe form of bipolar disorder;
- a number of cognitive abilities-memory, executive functioning, and motor skills-were lower among people with bipolar disorder;
- two genes - CACNA 1 (Calcium voltage-gated Channel subunit Alpha 1) and ANK3 (Ankyrin-3) – seem to play an important role in susceptibility to developing bipolar disorder. Moreover, many genetic variations have been identified as risk factors for bipolar disorder, and more recent researches have examined the role of these variations in the development of bipolar disorder; moreover, recent genome-wide association studies identified 15 genes linked to bipolar disorder and more than 60 associated genomic loci (Mullins et al., 2021; Stahl et al., 2019);
- stem cells sampled from participants have also proven useful in studying the cellular aspects of bipolar disorder. For example, neurons derived from the patients' stem cells were found to be more excitable, but they calmed down when they were exposed to lithium, a drug commonly used in the treatment of bipolar disorder;
- the key features of the verbal patterns of these patients can be used to estimate the patients' affective states and, at the same time, they can be used to anticipate the need for intervention to prevent manic or depressive episodes.

In 2018, a review of the relevant literature on etiology and risk factors in the occurrence of bipolar disorder. The authors' conclusion was that although various genetic and environmental factors have been identified, they are non-specific to bipolar disorder, being associated with other mental disorders. On the other hand, the analyzed articles emphasized the importance of studying the interaction between genetic factors and environmental factors and the authors examine these interactions separately, for each category of factors (Rowland & Marwaha, 2018).

Therefore, as regards genetic factors, their contribution to bipolar disorder has been identified and demonstrated, the concordance in the case of monozygotic twins being between 40-70% and the risk of developing this disorder throughout life in first-degree relatives being of 5-10% (Craddock & Jones, 1999). However, much more frequently, the relatives of bipolar patients suffer from unipolar depression, suggesting that genetic risk transcends diagnostic categories. Moreover, bipolar disorder does not follow a Mendelian pattern of transmission, the studies not identifying individual genes with a strong causal connection to this disorder (Badner et al., 2012). The risk of developing this disorder is only partly due to multiple single-nucleotide polymorphisms, which are otherwise widespread in the general population (Craddock & Sklar, 2013). Although various studies have attempted to identify the genes responsible for this disorder through associative studies or linkage analyses, the results have not led to the identification of a specific mode of genetic transmission.

2.1. Infections

It is presumed that infections (and especially intrauterine infections) interfere with fetal and postnatal neural development. Two analyzed studies revealed a 4-, respectively 5-fold higher risk of occurrence of bipolar disorder with psychotic symptoms in patients whose mothers were exposed to influenza during pregnancy (Okusaga et al., 2011; Parboosing, Bao, Shen, Schaefer, & Brown 2013). However, the hypothesis that gestational viral infections would increase susceptibility to bipolar disorder has not been confirmed. Exposure to viral infections in adulthood has been investigated by only a few studies, which analyzed the connection between seropositivity for Coronaviruses, influenza type A and type B viruses and affective disorders with or without psychotic features and suicide

attempts. Only influenza type B was correlated to a history of suicide and psychotic symptoms (Aldinger & Schulze, 2017).

A week immune system or immune dysfunctions could lead to infections frequently associated with bipolar disorder. Considering the important function as regulator for the immune system of the HLA-G (human leukocyte antigen G), it has shown that this protein is linked to the etiology of bipolar disorder (Sundaresh et al., 2018).

2.2. Smoking during Pregnancy

Few studies have highlighted an increased risk for bipolar disorder caused by smoking during pregnancy, but so far there are no systematic research protocols regarding the correlation between smoking during pregnancy and bipolar disorder (Ekblad, Gissler, Lehtonen, & Korkeila, 2010; Talati et al., 2013). Moreover, sometimes the mother who smoked during pregnancy is most likely due to confounding by familial background factors (Chudal, Brown, Gissler, Suominen, & Sourander, 2015).

2.3. Birth Complications

It is still unclear whether bipolar disorder is in any way influenced by birth complications. A single study suggests a 2.5 times higher risk of developing bipolar disorder in patients born by planned cesarean section (Chudal et al., 2014). Other studies analyzed a possible connection between the baby's weight at birth, the mother's age and bipolar disorder, but the hypothesis was not confirmed. Only one study found that premature birth could be associated with bipolar disorder (Nosarti et al., 2012). Till today, there are no other studies analyzing the potential impact of birth complications on the clinical evolution of bipolar disorder so that is difficult to decide for a clear association.

2.4. Climate and environmental factors

It is presumed that climate changes influence the regulation of affective states, particularly in the case of bipolar disorder (Rybakowski, 2021). Some connection between seasonal variations and bipolar symptoms have been highlighted through systematic reviews, the climate been a trigger for the onset of bipolar disorder or for seasonal peaks or patterns for different mood episodes associated with bipolar disorder (Geoffroy, Bellivier, Scott, & Etain, 2014). A higher correlation between seasonal frequency and manic episodes have been observed comparing with depressive episodes. Generally, the climax of mania occurs in spring and summer, with another third peak in the middle of winter, while depression is more common in winter and spring (Hochman, Valevski, Onn, Weizman, & Krivoy, 2016). Moreover, the data show that climatic factors such as the middle hours of the day, the average temperature, the number of sunshine hours - day-night alternation - are associated with relapses in bipolar disorder. It is the reduction of natural light that mainly triggers depressions. The correlation between sunlight and affective states is also supported by the therapeutic effect of phototherapy in mood disorders (Niemegeers, Dumont, Patteet, Neels, & Sabbe, 2013; Galima, Vogel, & Kowalski, 2020). Young argue, however, that this factor gradually loses its importance given the weakening of the circadian rhythm caused by electric lighting – the switch between manic and depressive states corresponding to high activity during lighting period (increased catecholaminergic expression) to low activity, depression (increased somatostatin and corticotrophin releasing factor) (Young & Dulcis, 2015). It is not clear if the vulnerability to climate and seasonal changes is linked to gender considering that there are studies that consider it greater for women and others for man (Hochman et al., 2016; Geoffroy, 2014).

2.5. Childhood Traumas

A life history that includes childhood traumas is common in patients with bipolar disorder. The prevalence of post-traumatic stress disorder (PTSD) in patients with bipolar disorder ranges from 16% to 39% (Otto et al., 2004). Broadly, traumas are obvious in nearly 50% of the patients with bipolar disorder and influence both the onset and clinical evolution of bipolar disorder (Garno, Goldberg, Ramirez, & Ritzler, 2005). The review of Aas (Aas et al., 2016) concluded that:

o childhood traumas influence the clinical evolution, generating a younger age at which the onset of bipolar disorder occurs. Traumas also increase the risk of a rapid cyclical evolution, the risk of psychotic symptoms, the risk of more lifetime episodes, of suicidal ideation and suicide attempts, as well as the risk of substance abuse;

o gender differences also seem to be significant. Women with bipolar disorder reported more frequently childhood traumas and had a more severe evolution of bipolar disorder (increased episode cyclicity, early onset, suicide attempts, and more frequent depressive episodes).

By contrast, Quarantini point out that bipolar patients with childhood traumas showed more manic than depressive symptoms compared to the control group (Quarantini et al., 2010). In terms of type of trauma, most researches focus on sexual and physical abuse, but there are indications that emotional abuse and parental neglect are the most prevalent of the types of traumas. A possible explanation would be the difficulty of detecting emotional abuse through assessment questionnaires. It is worth noticing that, in this context, to the traumatic experience of bipolar patients caused by childhood traumas, their own turbulent behaviour during manic episodes is added.

Childhood traumas are associated with lower resilience that links directly with decreased quality of life in bipolar disorder (Citak & Erten, 2021).

2.6. Life Events

Psychological stressors represent life events that affect the evolution of bipolar disorder, although their relationship to the onset of the disorder has been much less investigated, compared to the onset of unipolar depression. Various researchers have pointed out that certain life events influence the age of onset and the clinical evolution of bipolar disorder. According to their type, certain events trigger either mania or depression (Johnson, 2005; Alloy et al., 2005). The literature highlights that, in general, positive events and achievement of some goals are followed by manic episodes; negative events, not just the positive ones, can trigger both depression and mania (Johnson et al., 2008). Many factors have been studied: interpersonal problems (e.q. loss of a loved one, illness), social status, financial crises, work-related difficulties, failure, job-related issues (e.q. unemployment), number of exposures to events as they are a trigger or a risk factor of relapse and psychotic features. Gershon concluded that exposure to trauma is correlated with more severe interpersonal difficulties leading to more severe depressive episodes (Gershon, Johnson, & Miller, 2013).

Bipolar disorder is frequently associated with substance abuse, such as cannabis, opioids, cocaine, sedatives, and alcohol, although the causality is more difficult to demonstrate because of the relatively small number of prospective and longitudinal studies. However, more and more studies highlight cannabis as a risk factor and examine the relationship between the frequency of consumption, age of the consumer, and the onset or severity of bipolar disorder (Lalli, Brouillette, Kapczinski, & de Azevedo Cardoso, 2021). For opioids, alcohol, and drugs, prospective studies show that they increase the risk associated with bipolar disorder, without examining the specific differences (Preuss, Schaefer, Born, & Grunze, 2021).

2.7. Social Support

According to Greenberg, people with bipolar disorder show greater deficits in the area of social relationships, such as relationships with parents, family, partner and friends (Greenberg, Rosenblum, McInnis, & Muzik, 2014). The relapse rate is higher for people without a support system. Moreover, the presence of a partner at the onset of the disorder has a positive effect on the clinical evolution and especially on the recovery between episodes, while patients without partners are at a higher risk of psychotic manifestations. In addition to social support, family behavior significantly influences the evolution of bipolar disorder. Excessively expressed emotions and negative affective style increase the risk of relapse and may even influence the prevalence of suicidal ideation in young bipolar patients. Miklowitz points out that patients who were overly criticized by close relatives showed more severe symptoms in both depressive and manic episodes (Miklowitz, Wisniewski, Miyahara, Otto, & Sachs, 2005).

In conclusion, according to the authors, the studies prove that, of all the factors examined, childhood traumas contribute the most to the onset and evolution of bipolar disorder. Bipolar patients with a traumatic history will have a more rapid episode cyclicity, more frequent psychotic manifestations, a greater number of episodes throughout life, and a greater risk of suicide, suicidal ideation, and substance abuse. It is noteworthy that emotional abuse is generally ignored in the literature, even though it is assumed to have the highest prevalence among early adversities.

2.8. Parental Style

A recently published comparative study examines parenting style as an important risk factor associated with psychiatric disorders (Abbaspour, Bahreini, Akaberian, & Mirzaei, 2021). The study compares parental styles in 130 patients suffering from schizophrenia, bipolar disorder or depression using the Parental Bonding Instrument (PBI) to analyze the data. The results show that the optimal parental style (low control, increased care) is present in 43.05% of bipolar patients, in 47.7% of patients with severe depression and in 38.5% of patients with schizophrenia. However, 62.30% of all analyzed patients reported ineffective paternal styles and 51.53%, ineffective maternal styles. Moreover, maternal parenting styles were significantly different ($p=0.007$), while paternal styles did not show significant differences ($p= 0.848$). In the case of bipolar patients, they experienced an excessive control from both parents. Most patients were affected by ineffective parenting styles and, despite the existence of bio-psychosocial factors associated with these disorders, the crucial role of the parents, and especially of mothers, should not be ignored.

3. DIFFERENTIAL DIAGNOSIS

The differential diagnosis can be made with other mental disorders, with other medical conditions and with the effects of certain medicine substances. General medical conditions that can give symptoms similar to bipolar affective disorder are cardiac disorders, respiratory disorders (pneumonia), endocrine disorders, infectious or inflammatory diseases, malignant tumors, metabolic deficiencies, nutritional deficiencies, stroke, hydrocephalus, Alzheimer's dementia, Parkinson's disease, arthritis rheumatoid, tuberculosis. Pharmacological causes can be the administration of analgesics, antibacterial agents, antineoplastics, cardiac and antihypertensive medication (betablockers), antipsychotics, anti-inflammatory (corticosteroids), contraceptives.

Psychiatric disorders that can mimic the symptoms of bipolar affective disorder are depression or mania secondary to a somatic condition, dysthymic disorder, substance-induced mood disorder, recurrent depressive disorder, adjustment disorder with depressed mood, sadness, grief depression, dementia, attention deficit hyperactivity disorder (ADHD), schizophrenia with negative symptoms, the depressive or manic phase of schizoaffective disorder.

4. THE MANAGEMENT OF MAJOR DEPRESSIVE EPISODE IN BIPOLAR AFFECTIVE DISORDER

The management of major depressive episode in bipolar affective disorder has as main goals ensuring the safety of the patient: assessing the risk of suicide, assessing the need for hospitalization; verifying the diagnosis by reference to non-psychiatric differential diagnoses (medical causes or drugs) and trying to clarify the psychiatric diagnosis; collection of anamnestic data; paraclinical investigation to exclude other possible causes of secondary depression (hemogram, urine samples, thyroid function, liver function, calcium dosage, phosphate dosage, glucose, vitamin B12, folic acid); using a combined therapeutic approach that includes pharmacological agents and psychotherapy.

The evaluation is done considering several criteria: clinical form, intensity, comorbidities, somatic problems, suicidal risk, level of social functioning. The diagnosis is made according to ICD 10 and DSM 5.

Monitoring involves tracking the patient's general condition, psychiatric symptoms, as well as his somatic condition (clinical and paraclinical effects of the treatment), compliance level and social functioning.

The therapeutic approach involves psychological treatment (which can be achieved through cognitive-behavioral therapy, interpersonal therapy, psychoanalytically oriented psychotherapy, supportive psychotherapy, group therapy, family therapy, drug associations), lifestyle change (physical exercise, increasing the group of social support, adequate nutrition) and the pharmacological approach (with serotonin-norepinephrine reuptake inhibitor (SNRI), selective serotonin reuptake inhibitors (SSRIs), tricyclic antidepressants, heterocyclic antidepressants).

The treatment phases are:

a. acute phase (up to 12 weeks to achieve complete remission);

b. continuation phase (lasts from 4 to 6 months);

c. maintenance phase (long-term commitment to prophylactic treatment with an antidepressant, relapse prevention).

Choice of antidepressant medication is done according to the clinical image. It is considered that activating antidepressants can potentiate anxiety and insomnia, and by decreasing psychomotor inhibition they can increase the risk of suicide. When they are used, it is necessary to combine them with benzodiazepine-type anxiolytics (which also have a secondary antidepressant effect) and some sedative-type neuroleptics (Levomepromazine, flupentixol decanoate - Fluanxol). Suicidal risk requires strict supervision, and sleep disorders are corrected with neuroleptics.

In elderly patients, antidepressants without atropine action are preferred, so that they can be administered even in the presence of comorbidities (glaucoma, prostate adenoma, heart disease).

The choice of an antidepressant will consider therapeutic adequacy, tolerance and compliance, avoiding adverse reactions, as far as possible, using an effective dose in monotherapy, efficiency and effectiveness in the medium and long term.

For short periods, we are interested in rapid onset of action, resolution of symptoms, good tolerance, safety in administration (no lethal effect in suicide attempts)

For the long term, we are interested in: tolerability, the possibility of being titrated easily (blood level), prevention of symptoms appearing when the treatment is interrupted (absence of discontinuity syndrome), recovery as complete as possible, with a remission that leads to the return to the most previously achieved high level of functioning, avoiding relapses and recurrences, respecting the patient's dignity and quality of life, the financial possibility to procure the medicine in the long term.

Relapse is defined as: the exacerbation of depressive symptoms in a patient with an acute episode of the disease, after the initial suppression of symptoms and their reappearance within 6-9 months from the onset of the acute episode. In patients with bipolar depression, the relapse may take the form of a manic turn. In patients with unipolar depression, the frequency of relapses can reach up to 66% of cases, being most often a form of manifestation of therapeutic inadequacy. This is favored by: the obvious severity of the acute depressive episode, the heredocollateral history of unipolar depressive affective illness, a large number of previous depressive episodes. Relapse prevention is achieved through continuous treatment, over an average duration of 6-9 months.

5. THE PSYCHODYNAMIC PERSPECTIVE

Text Contemporary psychodynamic approaches also admit that affective disorders are strongly influenced by genetic and biological factors. From a psychodynamic perspective, affective disorders refer to people whose character structures are generated by depressive dynamics, including personalities characterized by the denial of depression and who are called manic, hypomanic and cyclothymic. But the fundamental themes that organize depressive and manic people are similar. From a psychoanalytic point of view, the manic mood is unconsciously sought as a form of release from the oppression of depression (Bollas, 2021). Early experiences of abuse, neglect or separation can lead to a neurobiological sensitivity that predisposes the individual to respond to stressors by developing a major depressive episode. Kendler, Neale, Kessler, Heath, and Eaves (1992) reported an increased risk of major depression in women separated from their mother or father during childhood or adolescence. In 2001, Kendler, Gardner, Neale, M. and Prescott also noticed gender differences regarding the depressogenic effect of stressful life events: men were more sensitive to the depressogenic effects of divorce, separation, and professional problems, while women reacted mainly to the depressogenic effects of relationships with those close to them. Losing a parent in childhood significantly increases the risk of major depression in adulthood. Parental divorce in early childhood has also been associated with increased risk for depression (Gilman, Kawachi, Fitzmaurice, G. & Buka 2003). But not only early losses are associated with greater vulnerability to depression, but also physical and sexual abuse. Various studies show that women with a history of abuse or neglect have a two times higher probability to have negative relationships and low self-esteem compared to women without such a history (Bernet & Stein, 1999; Bifulco, Brown, Moran, Ball, & Campbell, 1998; Brown, 1993) and abused or neglected women who have negative relationships and low self-esteem are ten times more likely to develop depression. Gabbard (2005) reviews a number of studies that have analyzed the influence of early trauma on depression and the permanent biological alterations that these traumas produce, such as: 18% lower left hippocampus volume in depressed women abused vs. depressed women non-abused in childhood; an increased level of corticotropin (CRF) that induces pituitary secretion of adrenocorticotropic hormone (ACTH), so early experiences of

neglect or abuse can activate the stress response and they can induce an increase in the activity in neurons containing CRF; increased pituitary, adrenal, and autonomic responses to stress, etc., the researchers concluding that hyperactivity of the hypothalamic–pituitary–adrenal axis and of autonomic nervous system associated with hypo-secretion of CRF are a persistent consequence of childhood abuse that may contribute to the diathesis for adult depression.

Although early stressors are an inherent component of the psychodynamic model that associates adult pathology with early trauma, the dynamic perspective also considers the significance of individual stressors. The clinician must always monitor the meaning that an apparently common stressor may have for the patient and to explore the meaning of all stressors, to identify the unique way in which each stressor affects the patient. Hammen (1995) notes that a considerable consensus has been reached that what matters less is the occurrence of a negative life event and more the personal interpretation of the meaning of the event and its importance in the context of its occurrence.

The most psychodynamic approaches suggest that in the case of depressive patients it is question of a fundamental narcissistic vulnerability and a fragile self-esteem, to which anger and aggressiveness are added (which result in guilt or self-denigration) and the search for a protective authoritarian figure that cannot be found and a punitive Superego completes the depressive picture. Freud (Mourning and Melancholia, 1917) links depression to early childhood losses and sees self-depreciation - omnipresent in depressive patients - as the result of an inward-directed rage against the background of identification with the lost object. Abraham (1924) develops this idea, suggesting that depressive adults experienced a severe decline in self-esteem in childhood, adult depression being triggered by a new loss or injury, which arouses intense negative feelings toward present and past figures who have hurt the patient through withdrawals, real or imaginary, of their love. Klein (1940) sees manic defences (omnipotence, denial, contempt, and idealization) as response to painful feelings caused by the object loss. An integral aspect of the manic defensive posture is often a desire to triumph over the parents and to reverse the parent-child relationship, a desire that can give rise to guilt and depression. In Klein's view, this mechanism is partly responsible for the depression that often occurs after success or failure. According to Gabbard (2005), Klein's formulation can help clinicians decipher the psychological significance of the coexistence of manic episodes with biological determinants. In dysphoric manic patients, the defensive function of mania is obvious: anxiety and depression pierce the manic episode, requiring the re-emergence of manic denial. In a much attenuated form, hypomanic defences are designed precisely as a defence against the threat of depressive affects. Unlike Freud and Klein, Bibring (1953) sees melancholic states as the result of tension between ideals and reality, any damage to self-esteem being a possible trigger for depression. For Bibring, the key to understanding depression was narcissistic vulnerability and the Superego had no key role in this process. Joffe and Sandler (1965), who studied the files of depressive children at the Hampstead Clinic in Great Britain, note that they become depressed when they feel that they lost something essential to their self-esteem and feel too helpless to prevent this loss, which becomes a kind of lost paradise, idealized, desired with intensity but unattainable. Jacobson (1971) develops Freud's formulation, suggesting that depressed patients actually behave as if they were insignificant, lost love objects, and eventually this bad internal object - or lost external love object - turns into a sadistic Superego. Arieti (1977) postulates a pre-existing ideology in severely depressed patients: the husband, the wife or an institution is often a dominant other in the psychological world of the depressed individual, who lives for this other, being convinced that life is worthless if he/she cannot get the desired answer from the partner.

In the phenomenology of depression, in the foreground is a loss of self-esteem, says Otto Fenichel (1945). I have lost everything; now the world is empty, the patient seems to feel, when the loss of self-esteem is mainly caused by a loss of external resources or I have lost everything because I deserve nothing, when, in the intra-systemic conflict between Ego and Superego, the Superego wins. Psychoanalysis attributes an important role to aggressiveness in the onset of depression in adults, with countless psychoanalysts considering aggressiveness to be one of the most important components - if not the most important - in the onset of depression. The depressive reaction seems to be always related to undischarged aggression. (Joffe & Sandler, 1965) or inhibited, aggressive drives are the core of depression. (Schultz-Hencke, 1940)

Adler (1908) gave the aggression drive a prominent role but, unlike Freud, for whom aggressiveness had its origin in the death drive, he derives aggressiveness from sexual and self-preservation drives. In the subsequent debates about the compulsive or non-compulsive nature of aggressiveness, Anna Freud would conclude that aggressiveness lacks the characteristics of a drive (the organ, the energy) and Kunz proved the supposed non-specificity of aggressiveness by the lack of an object reserved for it. *That is why we emphasize, like Kunz, that the enormous efficiency, the characteristic of being always ready of aggressiveness and destructiveness does not become sufficiently intelligible except under the premise of its reactive nature* (Thomä & Kächele, 2006). Kohut would criticize the theorization of aggressiveness as a primary drive, for him destructiveness representing a primitive product of disintegration, "the narcissistic rage" having a role of self-preservation, of maintaining some systems of the Self. Thomä and Kächele (2006) would also identify the considerable difference between aggressiveness and destructiveness:

The loud aggression, which is directed against people or objects that stand in the way of our satisfaction, quickly disappears after the goal is achieved. The narcissistic rage, on the other hand, is inexhaustible, it never ends. Conscious and unconscious phantasms have acquired their independence from events that give rise to rivalry aggressiveness, and now act permanently as inexhaustible forces of cold-blooded destruction.

Facing the threat of loss of self-esteem, the patient tries to retroactively cancel this loss, aggravating the situation, says Otto Fenichel (1945), by pathologically introjection of the loved object in an ambivalent way, which leads to feelings of absolute annihilation.

The sadistic-oral introjection of the object, whose love is desired as a narcissistic resource, is like a lighted match thrown over the dust of dammed narcissistic need.

Through this narcissistic identification (*the shadow of the object has therefore fallen over the Ego*), the object is completely replaced by the alteration of the Ego. After the introjection, the sadism is taken over by the Superego and attacks the Ego already altered by the introjection. Now the Superego treats the Ego as the patient unconsciously wishes to treat the object. Freud saw the melancholic's self-reproaches as accusations directed against the introjected object, and Abraham added that often, contrary to expectations, grievances appear from the introjected object and in the form of accusations that the real object has actually brought to the patient. Fenichel (1945) adds an important detail: yes, in depression, the Ego is helpless and cedes in front of the Superego, but in terms of the existence of a reproaching Superego, self-reproach is not the only way to try to attack an introjected object; in terms of the existence of an Ego who is reproached, self-reproach is both the flattery of the Superego and a plea for forgiveness, a way of getting into the Superego's good graces. But this attempt fails.

The immeasurable sadism, inherent in the oral instinctual tendencies and remobilized through regression, devoted itself to the Superego; all the rage with which the Ego unconsciously wished to attack its object is now unleashed against the Ego (Fenichel, 1945).

In other words, the Ego lays down the arms:

The melancholic's fear of death allows only one explanation: that the Ego surrenders, because it is hated and persecuted by the Superego instead of feeling loved (Fenichel, 1945).

For Freud, the genesis of a severe Superego corresponds to a gentle education or, on the contrary, it is in proportion to the aggressiveness that the child felt towards the parents and that he/she had to repress. Stiemerling (2002) distinguishes four important characteristics of the Superego of depressive persons:

- they are in conflict with the Ego, which they treat as an object;
- remained at a stage of infantile development;
- may be the genetic heir of the parental instance;
- the child probably had considerable aggressiveness against the authorities (parents) and had to give up satisfying those vengeful aggressive tendencies. He/she helps himself/herself out of this situation, recognizing this authority that becomes the Superego

Any psychodynamic pattern of depression will focus on the early traumatic experiences that determine the child to develop problematic Self and object representations, as well as the defence mechanisms that the child uses to control painful affects. Of these, some may contribute to the development of depression, while others may contribute to protection against depression. Therefore, turning against oneself through excessive self-criticism is an immature defence that contributes to the development of dysphoria, while intellectualization can positively influence the level of dysphoria. As regards the relational patterns of depressive people, psychodynamic approaches have identified the origins of dysthymic dynamics in early experiences of separation or loss of the object, as well as in other family circumstances, such as: parental neglect towards the child's needs, the absence of explanations that facilitate the child's understanding of traumatic events, a family atmosphere where mourning is discouraged, or a parent's depression. According to Gabbard (2005), the psychodynamic perspective on defences, as well as the identification of the unique meanings that defence mechanisms and object relations have for each depressed person can facilitate the understanding and treatment of depression.

6. THE REVERSE OF DEPRESSION: HYPOMANIA

People with hypomanic personality have a fundamentally depressive organization, counteracted by the defence of denial, and when this defence fails, depression emerges. The hypomanic individual is full of enthusiasm and energy, boastful, grandiose and funny. He/she makes big plans, can't slow down, and can be attracted to drugs, alcohol, or medication. For Akhtar (1992), the individual with hypomanic personality is overtly cheerful, highly sociable, idealizes others, workaholic, seductive and logical, and at the same time, secretly guilty of aggression towards others, unable to be lonely, empathically deficient, unable to love, corruptible and lacking a systematic approach in his/her cognitive style. McWilliams (1994) nuances this portrait, pointing out that most individuals with a hypomanic character, however, present a more attenuated version than that described by Akhtar, being capable of love and having an integer character: individuals with hypomanic psychology are known for their high energy, state of excitement, mobility, distractibility and sociability. They are often very good hosts, story-tellers, mimes, good at puns,

treasures to their friends who, nevertheless, complain, because, owing to their habit of turning any serious remark into a joke, it is difficult to you get too close to them emotionally.

Fenichel (1945) also noticed the oral organization of hypomanic people: they talk all the time, crunch, smoke, drink or chew gum, and they are often overweight. Their constant agitation, however, suggests considerable anxiety, despite their displayed enthusiasm, and their joy seems fragile, unstable. Bollas (2021) points out, however, that it is the anal organization that becomes obvious in the analysis of the manic-depressive, the analytical work contributing to the understanding of the fact that there is a subjacent sexualisation of the manic episode: The manic-depressive individual sucks the productions of the mind as if they were a mother's breast. The mind-sucking is violent and intense. Although some of the mental food is orally discharged through speech, it is as if a phantasm unconscious of the discharge of the residues through the anus is operating at the same time. The urethra is activated, creating an anal-urethral connection that generates a genital sensation. Thus, he/she fools the world through manic processes of elimination.

The manic self is terrified of attachment because losing the object could be devastating for it. Suicide attempts and blatant psychotic behaviour can suddenly invade the manic fortress if any loss becomes too painful to be denied (McWilliams, 2011). Moreover, on the background of the primitiveness of the defensive and adaptative processes involved, many hypomanic and cyclothymic individuals are prone to self-fragmentation, their precarious self-esteem being maintained only by avoiding pain and capturing the attention of others. Hypomaniac individuals are masters in attracting others and awakening their deep attachment, but without the reciprocity of an equally deep investment. Bollas (2021) also confirms this picture:

Manic-depressive individuals are like Zeus. They do not need sexual contact with the other to procreate - their children are born through the mouth, anus, urethra, and genitals. By inversion logic, this absence of need for another person means that all others need the manic-depressive. There is therefore an extreme urgency, almost at the limit of panic, to make the world recognize that it needs him/her before being hit by a catastrophe. As in the case of depressive persons, regarding the relational patterns of manic individuals, the clinician discovers repeated traumatic separations that the child could not process emotionally, the death of an important person whose grief could not be elaborated, divorces, repeated moves, criticism, physical or emotional abuse. But the extreme defences to which the manic resorts - denial, cleavage, turning against oneself, etc. - indicate, however, that these losses were somehow different from those of the depressive person, perhaps more severe or much less metabolized. Bollas (2021) describes the early environment of the manic-depressive person as veiled in an invisible inertia, people with this disorder being deprived of the experience of processing the ups and downs of life, of emotional oscillations of any kind: This does not happen in the family of the manic – depressive person, who prefers a quiet life and doesn't bother to help his/her children manage their drives. The manic-depressive individual was an unseen and uncelebrated child, whose family was generally inattentive to his/her inner world.

7. MOURNING AND MELANCHOLY. THE SIMILARITY BETWEEN THE TWO IN THE FACE OF LOSS

In presenting his early theory of mourning in "Mourning and Melancholy" (Freud, 1917), Freud begins by defining the similarities between the two responses to loss that he otherwise seeks to distinguish. Mourning and melancholia involve similar symptoms:

"deeply painful detachment, cessation of interest in the outside world, loss of the capacity to love, [and] inhibition of all activity." Moreover, both mourning – "normal" and melancholia – "pathological" can occur as "a reaction to the loss of a loved one or to the loss of an abstraction that has taken one's place, such as one's country, freedom, and ideal, and so on". Whether in response to literal death or symbolic loss, mourning is an experience of pain and a process by which the sufferer relinquishes emotional ties to the lost object. While drawing on prevailing assumptions about the mourning process, Freud suggested that this detachment of the libido occurs through a "reality testing."

Although he acknowledged a lack of complete knowledge of reality testing, Freud argued that the weeper destroys attachments, primarily through memory work: "Each of the memories and expectations in which the libido is linked to the object is raised and discharged, and the detachment of the libido is achieved as far as it is concerned. When this stage of mourning is completed, the Ego becomes free and uninhibited again."

The work of overcoming mourning, as Freud describes it, involves a process of obsessive recollection during which the survivor revives the existence of the lost other in the space of the psyche, replacing a real absence with an imaginary presence. This magical restoration of the lost object allows the bereaved to assess the value of the relationship and understand what he has lost through the loss of the other. But the prolongation of the existence of the lost object, at the heart of the effort to overcome the pain, does not persist indefinitely, because Freud argued that the sufferer, comparing the memories of the other with the actual reality, reaches an objective determination that the lost object no longer exists. With a very specific task to perform, the experience of Freudian loss then attempts to transform the memory to which the person is attached into a memory without a future. Mourning comes to a decisive and "spontaneous" end, according to Freud, when the survivor has detached his emotional connection to the lost object and attached his free libido to a new object, thus accepting consolation in the form of a substitute for what he been lost.

It is confusing that the term "depression" has been applied both to the state accompanying mourning and to what results from defenses against suffering. The path leading to confronting and grieving the loss is associated with a painful depressive feeling involving guilt, regret, remorse, and a desire to make amends. These feelings were thought by Klein in 1952 (Klein, 2011) to represent the depressive position and are very different from those observed in depressive illnesses. Although mixed states are common, severe depressive illness or melancholia results from the defense against loss and therefore against all those feelings associated with the depressive position. Freud's (1917) description of both mourning and melancholia gains further depth from his later formulation, which argues that all conflict has deeper roots in the conflict between the life and death instincts. Specifically, although attitudes toward this formulation vary, it appears to be particularly applicable to bereavement conflict. After a mourning, the life instinct seems to slowly recover and help the patient to let go of the attachment to the dead object and engage in feeding the life instinct. The death instinct is more difficult to formulate, but it can be thought of as an anti-life force, expressed as the conservative tendency to hold on to the lost object and thus favor the development of melancholy.

The central problem remains the ability to judge reality. In the case of real loss through death, Freud (1917) described how "each of the memories and expectancies, which demonstrate the attachment of the libido to the lost object, is subject to the verdict of reality that the object no longer exists" (Phillips, 1999, pp. 122-123). Here the judgment involves the question of loss of love, and the particular incident of "neglect or disappointment" must disappear by the application of the "proof of reality." The choice dictates whether the loss of love is faced and perceived in realistic proportions, requiring an appropriate amount of

guilt to be felt, leading to a loss of idealization - both of Self and of the object. Sometimes the patient's level of development propels him in the direction of change because the patient comes to believe that he is strong enough to survive threats of loss.

8. CONCLUSION/DISCUSSION

The studies on the etiology of bipolar affective disorder have highlighted various risk factors: from genetic, perinatal, neuro-anatomical and neuro-chemical influences to environmental, psychosocial or climatic factors. The multifactorial etiology of bipolar disorder, although recognized and empirically demonstrated, continues to generate difficulties because the individual contribution of these factors is generally small, most of which being not specific to bipolar disorder. Therefore, during the last years, more and more studies focused on investigating the impact that environmental factors have in triggering bipolar disorder. Among these, early traumas appear to be omnipresent in the case of patients with bipolar disorder, the relationship between childhood abuse and bipolar disorder severity being an additional argument for the increased causal relation in this case. And yet, although traumatic childhood events seem to influence the risk of developing bipolar disorder, the manner in which this occurs remains unclear and needs further examination. In the case of bipolar patients, traumatic childhood events seem to be closely correlated with increased affective instability or emotional disturbances. The psychodynamic approach of etiology of bipolar disorder does not exclude the impact of genetic and biological factors, but the emphasis is placed on the individual significance of stressors. The clinician must continuously monitor the meaning that an apparently common stressor may have for the patient and explore the meaning of all stressors to identify the unique way in which each stressor affects the patient. The exploration, from a psychodynamic perspective, of the relational patterns and the defensive and adaptative processes that the patient calls upon can facilitate the understanding of the etiology and related therapeutic implications.

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