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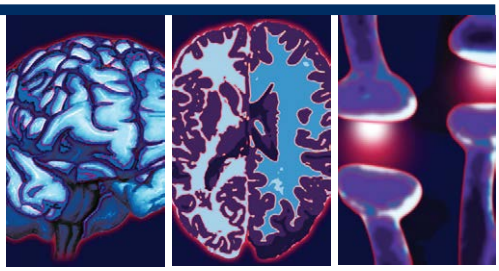

Delusional Depression: A Disorder of the Drive

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ABSTRACT

The distinction between melancholic and non-melancholic depression is an important way of classifying depressive disorders. Phenotypically and geneotypically, melancholia seems to be an entity of its own, which finds its extreme exacerbation in depressive delusion. The delusional depression is clinically easily overlooked and difficult to treat. This paper aims to contribute to the understanding of delusional depression by developing the symptomatology from a disorder of the drive. It is shown how the predisposed personality, primarily the typus melancholicus about increasing loss of energy and drive gets into inhibition, emotional numbness and fear and how fear turns the life-historically understandable worries about poverty, illness, guilt and hopelessness into delusional convictions. From the understanding of disease development, more effective therapeutic strategies, in particular on pharmacotherapy and psychoeducation, can be derived. A clinical case illustrates the investigation of depressive delusions.

Keywords

Anxiety, Delusional depression, Psychotic depression, Disorder of drive, Melancholia, Symptom development, Therapy

Introduction

Karl Jaspers and Kurt Schneider created the conditions for the current classification of mental disorders at the beginning of the 20th century and oriented their nosology towards the distinction between psychotic/endogen versus neurotic/reactive [1]. Psychotic symptoms are regarded as an expression of somatic diseases, while neuroses are attributed to misdevelopments in life history. Depending on the etiology, the focus of treatment is either on biological or on psychotherapeutic methods. This view was transferred to depressive disorders. The so-called endogenous, cyclothymic, melancholic, vital, autonomous depressions were classified as psychoses and were contrasted with reactive, neurotic, and personality-related depressions. While DSM-II [2] is still oriented towards the distinction between endogenous and neurotic depression, DSM-III [3] attempts to abandon etiological hypotheses in favor of improved reliability.

Akiskal and McKinney characterized depressive syndromes in 1975 by their uniform psychobiological course and thereby established the prerequisite for the dimensional classification of depressive syndromes according to severity [4]. With DSM-III from 1980 endogenous depression became subtypes of major depression with melancholic or psychotic features. ICD-10 [5] classifies melancholic symptoms with mild and moderate severity as somatic syndrome from 1991. The severe depressive disorder does not have this distinction, but is described without or with psychotic features. The occurrence of psychotic symptoms is linked to the severity of depression in both DSM-IV [6] and ICD-10. This connection is abandoned in DSM-V [7] with regard to bipolar disorder [8].

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However, there is still evidence that psychotic depression in relation to non-psychotic depression is distinguished by particular severity and frequency of episodes as well as increased suicidality [9-11]. The fact that melancholic depression is the prerequisite for the development of psychotic symptoms is indicated by both the large overlap of symptoms and the more severe depressiveness in psychotic depression [12,13].

Gordon Parker and his group have been calling for a return to the distinction between melancholic and non-melancholic depression since the 1990s. In their opinion, melancholia is a special psychopathological and biological entity that requires its own classification and differentiation from the major depression [14]. As in classical German psychiatry, psychotic symptoms are primarily identical with depressive delusions and are seen as an exacerbation of the melancholic syndrome [12,13,15].

DSM-IV and DSM-V require, as a precondition for the diagnosis of the melancholic subtype, the fulfillment of the criteria of major depression with the main characteristics "depressive mood" as well as "loss of joy and interest". In case of high symptom overlap, the melancholic state is then mainly specified by "the loss of affective resonance to normally joyful stimuli" and by "the particular quality of depressive mood". DSM-IV specifies the particular quality as sad or empty, distinguishing the type of sadness "from the normal sadness about the loss of a loved person" [6]. DSM-V tries to define the melancholic mood more precisely, as "deep discouragement, despair and moroseness or as the so-called feeling of numbness" [7]. Discouragement and moroseness are rather dysthymic emotions, which do not grasp the melancholic state. Despair is significant in the context of melancholia, but is not sufficiently defined [16]. Only the "feeling of numbness" points to a central aspect of melancholic psychopathology by bringing the feeling for the absence of affects into focus. It remains unclear, however, how depressive delusions can arise from an emotion that is extremely numbness and emptiness. It also remains unclear how the themes of depressive delusions are selected. The delusions are essentially limited to the themes of "guilt and sin", "illness" and "impoverishment". Kurt Schneider speaks of man's primal fears, of worries about the soul, the body and the necessities of life [17].

ICD-10 names the delusions of guilt and sin, impoverishment, hypochondria and nihilism, but also delusions of relationship and persecution as well as defamatory and accusatory auditory hallucinations. In DSM-V, psychotic symptoms, delusions and hallucinations are found as mood-congruent, in harmony with personal insufficiency, guilt, illness, death, nihilism, and deserved punishment. In DSM-V, the mood-incongruent psychotic symptoms include more and more symptoms from the schizophrenic spectrum with thought dispersal, thought insertion and the delusions of external influence, and therefore also show more and more transitions to schizophrenia [18-20].

In addition to the uncertainties in diagnostics, treatment remains still a problem. The recommendation of the combination of antidepressants and neuroleptics has only recently become established in most Guidelines [21-24]. There are no comparative studies for the combination of different antidepressants and neuroleptics and there are very different views on the use of benzodiazepines. The remission of unipolar psychotic depression occurs with medication at 50% within 2-3 months, at 75-81% within 6-12 months and at 94% within 24 months. A whole number of patients are ill 10 years and longer [9]. It remains unclear whether remission will return to the pre-morbid functional level. It remains unclear whether remission will restore the premorbid function level [25-27].

The following study we firstly follow the hypothesis that melancholia is its own entity, which can be understood as a disorder of the drive and can thus be distinguished from non-melancholic depression. Secondly, it shows how the depressive delusions develop from the interplay of melancholic inhibition and personality, and that depressive delusions are exacerbation of the melancholic syndrome. Thirdly, understanding symptom development can contribute to a significant improvement in therapeutic strategies. A case history illustrates these steps of investigation.

Melancholia and Drive

The current classification of mental disorders is based on the description of symptoms and their categorical order. In contrast to this view, there has always been the historically significant idea of an underlying basic disturbance, which tries to connect the individual symptoms to a meaningful unity and thereby to guide the understanding of the illness [28]. With regard to melancholic

syndromes and their current classification as affective disorder, there has always been evidence of the importance of the drive. In his book "Melancholia und Depression" Michael Schmidt-Degenhard explored the question of what distinguishes melancholia in contrast to depression. He worked out a line of tradition that emphasizes the importance of psychomotor inhibition in melancholia [29].

As early as 1867 Griesinger reported on the importance of inhibition in the development of melancholic syndrome [30]. There is a growing recognition that a central symptom of melancholia is not so much the sad mood but rather the depressive inhibition. In 1874 v. Krafft-Ebing intensified the examination of his patients' experience of illness and found that the melancholic patient was less desperate of sadness than of psychological anaesthesia, of being unable to feel any more [31]. In 1880 Schäfer classifies melancholia as inhibition psychoses and sees their psychopathological basis in mental paralysis. For the first time he differentiates between depressive mood and psychomotor inhibition [32].

With development of the concept of manic-depressive insanity in Kraepelin, the concept of depression is expanded in the following years, while melancholia disappears visibly. In the 5th edition of his textbook of 1896 Kraepelin restricted melancholia to the depressions of age and involution [33]. In the 8th edition of 1913 he integrated melancholia into manic-depressive insanity and abandoned it as his own diagnosis [34]. Following this tradition, Schneider replaced the term melancholia with the term endogenous or cyclothymic depression. Schneider interprets endogenous depression as change in feelings, as disturbance of vitality [35,36], but as an experienced clinician he knows that the syndrome includes a particular physical heaviness and alienation, which in extreme cases can become a "depressio sine depressione" [37].

In the following phenomenologically oriented psychiatrists take up the concept of melancholia as an own, monopolar course of disease and use it synonymously with endogenous depression or depressive psychosis. For Strauss in 1928, the cardinal symptom is the vital inhibition, which in his opinion describes the boundary between psychology and biology [38]. In the same year Gebsattel worked out in the most differentiated way the "endogenous inhibition" that "brings the personally formed urge, to become who you are to a standstill". He contrasts the will of the sick with the inability, the painful feeling of inhibition [39]. From the "basal inhibition of becoming", Gebsattel derives all further symptoms, the inhibition of thinking, will and feeling, but also transitions into compulsions and delusions. In 1937, in a work on melancholic depersonalization, he describes the depressive splitting: An inhibited, emptied ego chases after the actual ego, which was only remembered from healthy times, without being able to reach it. Gebsattel explains the change between psychomotor inhibition and agitation by how the patient behaves towards basal inhibition. Either he submits and complains e.g. lying in bed or he revolts and enters into an agitated state [40]. In 1961, Tellenbach focuses on the transition to melancholia and asks about the preconditions in personality. His investigations also end with inhibition, the inability to act, the increasingly empty circles of thoughts and agitation [41].

In 1996, Parker and Hadzi-Pavlovic named psychomotor inhibition as the most important symptom for distinguishing between melancholic and non-melancholic depression [42]. In their opinion, diagnostic differentiation cannot be made on the basis of depressive experiencing, but only on the basis of well observable psychomotor inhibition. In 2010, Parker and a number of internationally acknowledged researchers in the American Journal of Psychiatry demand that DSM-V should classify melancholia as an independent affective disorder [14]. In addition to psychomotor disturbance with inhibition and agitation, other symptoms mentioned are joylessness, lack of affective resonance and influence ability as well as cognitive and vegetative abnormalities. Psychotic symptoms are not described as obligatory, but as frequent and, as in classical German psychiatry, are limited to the delusional ideas of guilt and sin, impoverishment, illness and hopelessness.

The melancholic phenotype also seems to be a genotypic important research marker. In contrast to non-melancholic depression: 1. Hypercortisolemia is observed in the dexamethasone inhibition test. 2. Linear relationship exists between psychomotor inhibition and reduced response to dexamethasone suppression. 3 Typical sleep changes can be found with reduced REM latency, prolonged REM phases and reduced deep sleep. 4. There is a better pharmacological response to tricyclic antidepressants and ECT than to SSRI. 5. There is little response to

placebo, psychotherapy or social intervention.

Following the idea of a basic disturbance, the individual symptoms of melancholia can be interpreted as a disturbance of the drive and inhibition of the will. Psychomotor reactivity, which expresses mental processes, proves to be slowed down, bound, rigid and without resonance. The depressive affect is experienced in the body, as heaviness up to stiffness, as emptiness and numbness. Thinking is slow, viscous, inhibited, without intentionality. Thinking sticks broodingly to arbitrary contents, or circles around the frightening topics. Reduced concentration, perception and retentiveness become pseudo-dementia in the extreme. The inhibition is so deep that experience of time slows down, that experiencing is uncoupled from the usual every-day, loses itself in the past and is painfully experienced as depersonalization, derealization and desynchronisation [43]. The perspective of the future is lost in numbness and emptiness and thus becomes hopeless. Everywhere is inhibition, wanting without being able, which leads into the vicious circle of tension and agitated exhaustion. The concept of inhibition refers to the inner split, the increasing loss of energy and the struggle of the will against it.

Development of Delusional Depression

Tellenbach described the personality of the melancholic patient in 1961 as typus melancholicus [41]. On this conceptual basis, four dimensions of personality had been identified and operationalized: Love of order, conscientiousness, hypernomia/heteronomy and intolerance of ambiguity [44-46]. In the meantime, this is probably the best researched personality variant in depression [47]. Mundt et al. [48] found the typus melancholicus in 51% of endogenous depressives and increased personality traits of the typus in 25%. Tellenbach described the development of the disease as follows: The melancholic personality actively encloses itself within the narrow limits of its order. Everything is taken exactly and heavily and even the slightest guilt is carefully avoided. In this constant tension, the risk of depressive illness is created. Tellenbach calls this “includence”. The danger becomes virulent in the “remanence” when the melancholic person increasingly lags behind the demands on himself. In the increasing distance between will and ability, action loses the perspective of an orderly future and comes under the requirement to want everything at once and at the same time. The doubt, the to and fro of thinking becomes despair. Desperation describes the transition to melancholia, the beginning of the vicious circle of inability to make decisions and brooding, tension of will and inhibition, loss of energy and agitation [49].

The increasing psychophysical exhaustion leads to a paler and duller feeling, to depersonalization. Phenomenological authors such as Strauss, Minkowski, Binswanger, Tellenbach and Kraus have worked out depersonalization as specific change of the affect in melancholia since the middle of the 20th century [50]. Studies by Michal et al. [51] and Mula et al. [52] were able to establish a close correlation between depersonalization, the severity of depression and anxiety. Psychomotor inhibition is closely correlated with inhibition of affects, depersonalization and emptiness. As it corresponds to the personality of the typus melancholicus, the patient can often still show socially desirable behavior under exertion of will, can smile, react friendly or even funny, although he acts only from the memory of a comparable situation and a feeling that can no longer be felt. The investigator himself lacks affective resonance as orientation in the patient. When asked how the patient feels, he often answers with descriptions of situations and experiences, adhering to the concrete and representational. It is difficult for the investigator to develop an emotional perception of melancholic suffering. In the worst case, the severity of depression is overlooked due to socially desirable behavior or histrionic aggravation or schizophrenic parathymia are assumed. Knowledge of melancholia and its expression in tension and struggle against inhibition, in exhaustion and resignation is a necessary prerequisite for the empathic relationship with the sick person [53].

Since the melancholic affect is experienced as emptiness and absence of affects, and delusions are traditionally interpreted as disturbances of thought-content, the question of what depressive delusions are remains open. In order to identify delusions in melancholia, the delusional themes must be explicitly asked in the patient’s experience. The delusions are not bizarre, i.e. culturally inappropriate or completely unrealistic, but are generally about human concerns and fears. The delusional character can only be recognized by the degree of conviction, uncorrectability and the resulting relevance for action. In his early works on depressive delusions, Janzarik focused above all on the connection between personality

and choice of themes as well as between fear and delusions [54-56].

Fear arises in the face of a will that is losing its usual drive, which is visibly lagging behind its important goals, achieving them less and less, losing the perspective of its future and becoming increasingly hopeless. The fear emerging from melancholic inhibition sharpens the most significant themes and worries of the personality and gives them delusional character.

The delusional fear of suffering from an incurable physical disease is understandable from depressive experiences of physical heaviness and lassitude, vegetative changes, zoenaesthesia and depersonalization, which are linked with biographical experiences of somatic illnesses. The patient familiar with depression, who can attribute the physical symptoms to the depressive disorder, develops the delusions that he is hopelessly ill and will never recover.

The fear of failing with one's own existence, of no longer being able to care for oneself and one's family and of getting into ruin, which arises from the dwindling drive and is therefore understandable, leads against the background of biographical vulnerability for this subject, into delusions of impoverishment.

People who are primarily distinguished by their interpersonal relationships and responsibilities are increasingly concerned about their depressive inhibition and the fact that they increasingly owe something to their most important people. The fear of not fulfilling one's obligations, of failing one's spouse, of burdening or damaging children, or of having harmed them in the past, leads to delusions of guilt. If the reference point of the highest values is God, failure becomes delusions of sin. The exacerbation of fears that the situation will never improve and that all hope is in vain leads to nihilistic convictions and suicidality.

Therapy

Treatment of delusional depression is little researched compared to other psychiatric disorders. There is a consensus that the initial focus of treatment is on pharmacotherapy [57]. Until 2005 there was no evidence that combination of antidepressants and neuroleptics is superior to monotherapy with antidepressants [58]. The practice guidelines of the APA have nevertheless given recommendation for the combination treatment since 2000 [8]. Only a maximum of 5% of American psychiatrists had complied with this recommendation by 2007 [59]. The S3 guideline on unipolar depression has suggested a combination treatment since 2009, but due to general clinical experience [22]. In 2012, a new review of the studies concludes that the combination of antidepressants and neuroleptics is preferable to monotherapy with antidepressants [60]. In 2015 these results are confirmed [61].

ECT treatment is considered to be at least equally effective in most therapy guidelines as pharmacological first-line treatment. The literature on the relative efficiency of ECT compared with pharmacotherapy is limited by the lack of prospective, controlled trials [8,62]. Response rates were 82% for ECT, 77% for the combination of tricyclic antidepressant and antipsychotic and 51% for antidepressant monotherapy [63]. ECT shows higher remission rates of 92%-95% in psychotic depression versus 55%-83% in nonpsychotic depression [64,65]. There is also an indication for ECT Treatment, when an immediate symptom remission is required, e.g. in medical comorbidities or suicidality [66]. Brain stimulation therapies need to be carefully reassessed to prevent neuron apoptosis and synaptic plasticity defects with carefully reassessed dose and time interval for treatment and with appropriate dietary interventions [67]. This is of particular importance, as inflammatory processes and apoptosis are increasingly being blamed as possible causes of depressive disorders [68,69]. From this perspective, too, nutritional neuroscience is an emerging discipline with increasing influence on the field of mental disorders [70,71].

A research group at Cornell University, New York (STOP-PD, Study of Pharmacotherapy of Psychotic Depression) has been investigating the question of the importance of anxiety in the treatment of psychotic depression [72]. It has been shown so far that the presence of high levels of anxiety leads to significantly worse therapy results and increased suicidality.

Benzodiazepines are not generally recommended for delusional depression, but are given only for a short time in cases of severe agitation, anxiety, suicidality and depressive stupor or mutism [73,74]. However, there are indications that benzodiazepines and non-benzodiazepine hypnotics in treatment of major depression positively influence both the depressive symptoms and the response to antidepressant treatment [75].

These pharmacological findings are in accordance with the clinical significance of anxiety in melancholic depression, especially in the development of delusions. The therapy strategy derived from this insight should therefore consider the regular use of anxiolytically efficient benzodiazepines in the diagnosis of delusional depression. In my clinical experience, the high-dose treatment with benzodiazepines can lead to a rapid alleviation of depressive delusions and the associated suicidality, sometimes even to complete remission of delusional beliefs. If, on the other hand, fear and delusions do not subside, the melancholic symptoms do not improve.

Symptoms such as tension and agitation, brooding and especially sleep disorders are well observable parameters in dose-finding and dose-management with benzodiazepines. The fading away of these symptoms also indicates the remission of the delusional experience. The combination of antidepressants and antipsychotics must be carried out under strict observation of effects and side effects. Antidepressants which aim to improve the drive level can lead to a further energy deficit and increase depressiveness through overstimulation. The use of neuroleptics, which is primarily focused on reducing delusional beliefs and haunting brooding, must be handled carefully. In affective disorders an increased sensitivity for the occurrence of extrapyramidal motor side effects is described [76,77]. The Parkinson-like symptoms can increase psychomotor inhibition and akathisia can contribute to further energy loss and aggravation of suicidality through agonizing restlessness.

These seriously ill patients require inpatient care. The focus is initially on the administration of medication and help with the basic needs of life, on daily structuring and relief, on help with getting up, eating and body care. In psychoeducation, melancholic depression is conveyed as a loss of energy and drive, from which the understandable but dysfunctional tension of will and restlessness is derived, which leads into a downward-spiral. Delusions are directly addressed and explained as a legitimate but reality-distorting concern about material existence, physical health as well as the well-being of close people and one's own soul.

The biological effects of benzodiazepines and neuroleptics on anxiety and frightening beliefs as well as antidepressants on energy deficiency are explained, as is the importance of accepting the symptoms. The aim of these measures is to bring energy-consuming tension, restlessness, anxiety, brooding over delusional themes and sleep disorders to an end. The exhaustion hidden under restlessness thereby becomes visible. The initial loss of sleep can thus be transformed into an increased need for sleep. When, after a period of increased recovery and regeneration sleep returns to normal and psychomotor inhibition and numbness slowly subsides, the reduction of benzodiazepines and increased activation can begin. The creeping out of benzodiazepines towards the end of the treatment makes it possible to check whether the remission is stable or whether it leads to a renewed impairment of drive and mood.

Too early activation disrupts recovery as well as forced, problem-oriented psychotherapy. The clarification of the frightening delusional topics in everyday reality, the professional and economic situation, a possible physical illness or the culpably experienced overload of close people, represents an additional contribution to relieving the patient.

Case History

■ Introduction

The 65-year-old woman Mrs A. is brought to the clinic by her daughter with suicidal ideas. This is currently the sixth episode of a depressive disorder. Mrs. A. is the owner and managing director of a large nursery which is to be closed due to Mrs. A.'s age and declining profitability. Daughter and son work in the company. Gardening is her life's work; it is extremely difficult for her to give it up. This year's particularly cold winter is stressful, as the heating system keeps failing. The repair is no longer worthwhile, which is why Mrs. A. takes care of the coal fires herself, in the early morning hours. She cannot accept the freezing of the flowers and the loss of yield. The competition with an employee who recently resigned and went into business for himself in a nearby store is also a burden. Mrs. A. experiences this as treason and lack of loyalty.

For several months now, Mrs. A. has been experiencing signs of restlessness, sleep disturbances, exhaustion and increasing concerns about the company. She worries about how she can cope with the work, about giving up the nursery, the competition, the possible financial loss. An external psychiatrist diagnoses a severe depressive episode without psychotic symptoms and gives Mrs. A. venlafaxine 112.5

mg. As a result, restlessness, sleep disturbances and brooding worsen extremely. She only sleeps a few more hours and wakes up in the following nights with panic attacks and suicidal thoughts.

■ Personality

Mrs. A. fulfills all the criteria of the typus melancholicus. She is locked into the rigid order of family and work, perfectionism, conscientiousness and responsibility for the employees entrusted to her. She lives for her nursery and hardly takes a holiday. In personal contact, she loves clarity and unambiguity, and finds it difficult to bear dissenting opinions and disloyalty. She is fully identified with her company, which she has been running for over 40 years and can hardly imagine quitting. The low financial return, the competition with the former employee, the feeling of responsibility for the company, the failure of the heating system, all this, leads to growing tension and unrest. Mrs. A. is increasingly lagging behind her own expectations, developing greater restlessness, brooding, sleep disturbances and exhaustion. Sleep becomes superficial, dreamy, finally reduced to 2-4 hours with early awakening around 3.00 AM. Mrs. A. no longer regenerates, does not get out of bed in the morning, loses appetite and weight, and develops heaviness and rigidity throughout the body. She can no longer concentrate, can no longer remember anything, and is afraid of suffering from dementia. Finally, she can no longer interrupt her pondering, so that her doubts, about how the work can still be done become autonomous and desperate. She is inhibited, agitated, and ambivalent and can no longer decide what to do next.

■ Depersonalization

Upon admission to the clinic, Mrs. A. is severely inhibited psychomotorically, mimically rigid, without affective responsiveness. The feeling of numbness must be assumed and is confirmed by her on request. Depersonalization is an expression that the loss of drive has reached and emptied the feelings. Mrs. A. tries to be friendly during the inpatient admission, but the facial expression seems frozen, almost grimacing in an effort to smile. The tension of the will against psychomotor inhibition is obvious. She reports that she has lost the ability to rejoice or cry. Due to the emptiness of affect, there is also no emotional resonance in the investigator. With depersonalization, a derealization of the environment has also occurred, which Mrs. A. experiences as if in fog, pale and colorless. The food no longer tastes good; she has to force herself to eat in order not to lose any more weight.

■ Anxiety and Delusion

The increasing exhaustion of the drive brings Mrs. A. into an increasing paralysis of the will. She can hardly cope with her everyday life anymore, from which she is separated in a state of affective depersonalization and derealization. Thinking is determined by her most important themes, by her fear of failing, overburdening the children, bringing them to ruin and failing with her own existence. The fear of financial ruin becomes overwhelming in the face of severe inhibition and a hopeless future and turns into delusions of impoverishment. The focus of financial worries in exploration generates an increase of tension and restlessness, of rigidity in facial expression and posture, which blocks thinking and shows prolonged response latencies. The fears and worries described are understandable due to the severe inhibition. The degree of conviction of the contents must be actively explored. Mrs. A. is absolutely certain that the financial ruin has occurred. Thinking in pondering circles the delusional themes incessantly, disturb sleep and rest. The increasing loss of energy, no hope of improvement and the certainty of ruin become the ground of suicidality. Finally, Mrs. A. is convinced that her death would be a relief to everyone. She doesn't want to come to the clinic as an inpatient because she is stuck with working and dying at the same time.

■ Therapy

Her inpatient treatment at our clinic lasts for a period of 8 weeks. After diagnosis of a recurrent depressive disorder, currently severe depressive episode with psychotic symptoms (ICD-10: F33.3), Mrs. A. receives lorazepam 4 × 0.5 mg as a permanent medication. Zopiclon 7.5 mg and pipamperone up to 60 mg are given to regulate night sleep. As an anti-psychotic medication she receives risperidone 2 × 0.25 mg. The antidepressant medication with venlafaxine is initially reduced to 75 mg due to overstimulation and adjusted to a controlled level after the agitation has subsided. Below a dose of 187.5 mg, the serum level from the sum of venlafaxine and desmethylvenlafaxine is 355.9 micrograms/liter and thus in the upper therapeutic range (range: 100-400 micrograms/liter).

Under the medication with benzodiazepines, the fears and thus the certainty of financial ruin quickly subside, but still occasionally appear

in the course of the first week in pondering. Through the dosage of pipamperone up to 60 Mrs. A. gets more and more sleep. Sleeping time increases to more than 10 hours and early awakening is reduced. Mrs. A. reacts aversively to the onset of sedation and fears becoming addicted to lorazepam. She is afraid of losing control and being treated endlessly with benzodiazepines in the clinic. She wants to get well quickly and return to the nursery. The children are called in for family counseling and confirm that temporary workers in the company are organized and finances are balanced. Mrs. A. needs daily education for melancholia as severe psychophysical exhaustion and acceptance of her need for sleep and regeneration. The medication is explained as a measure to reduce her symptoms that inhibit regeneration. Fear, excessive concern, brooding, sleep disturbance must subside so that the underlying exhaustion can come to light. Psychomotor inhibition, numbness and depersonalization slowly improve in the course of her treatment. Due to the rapid self-activation, Mrs. A. experiences a resurgence of restlessness and early awakening and has to switch from pipamperone to quetiapine up to 75 mg. Below this medication, relaxation occurs, sleep disturbances and restlessness recede. From the fourth week of treatment, sleep duration, psychomotor inhibition and numbness return to normal. Her feelings of sorrow and later joy return. Lorazepam could be reduced by 0.25 mg on an experimental basis. Only mild vegetative withdrawal symptoms occur, with no effect on drive and affect. Lorazepam can therefore be sneaked out quickly and without any disadvantage. If the symptoms are largely reduced, in particular the delusional symptoms, risperidone can also be discontinued under quetiapine protection. Mrs. A. is finally discharged home with slight residual symptoms in the form of increased irritability, exhaustibility and reduced resilience. She will be treated in our outpatient clinic during her further recovery.

Conclusions

In the present paper, melancholic depression was described as a disorder of the drive and thus distinguished from non-melancholic depression. This view of a drive disorder unites the individual symptoms of melancholia into a meaningful unit. Depressive delusions arise from an extreme exacerbation of melancholic inhibition. The starting point for this development is usually the personality of the typus melancholicus. The inner tension of the depressively predisposed person leads to increasing loss of drive and energy. It is a vicious circle of tension, restlessness, brooding and sleep disturbances, which merge into psychomotor inhibition, loss of affective resonance and depersonalization, which is experienced in the extreme as a feeling of numbness. Fear and hopelessness resulting from "the will without ability" turn the themes of guilt, impoverishment, hypochondria and hopelessness into secure and uncorrectable convictions. The topics correspond to human worries and primal fears, are rooted in the personality and its life story, are not bizarre and are therefore easily overlooked. The delusion criteria of subjective certainty and uncorrectability, as well as the resulting actions, must be actively questioned and supplemented by an anamnesis with strangers.

Understanding these interrelations creates an adequate understanding for the patient, for his suffering and his endangerment. This is particularly important because the lack of affect and resonance makes it difficult to interact empathetically with the melancholic person. Therapy is not primarily based on diagnosis, but is orientated to the underlying drive disorder and the individual symptoms that inhibit recovery. Benzodiazepines and neuroleptics are initially aimed at the treatment of anxiety and delusions, while antidepressants focus on the underlying energy deficit. The dosage of medication requires a close monitoring of effects and side effects and must be corrected and adjusted over and over again. Activation with antidepressants that is too rapid and too strong can lead to strong increase in drive and anxiety and thus to an escalation of depressiveness and suicidality. In principle, recovery requires the establishment of a balance between calming and activating measures, primarily biological, but also psychoeducative and psychotherapeutic. Pharmacotherapy is accompanied by constant explanations of treatment steps and development of symptoms. Knowledge of the underlying drive disorder, symptom development and treatment options enables understanding, solidarity and competence in dealing with patients suffering from delusional depression. This experience gives the patient support and hope in his suffering and is an effective antidote to hopelessness and suicidality.

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