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Case report: Could *hikikomori* be a specific presentation of psychotic decompensation in people with autism spectrum disorder?

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Summary

The phenomenon of "hikikomori" is characterized by spending most of the day in one room, visibly avoiding social situations and interpersonal relationships, and leaving one's room only at night so as not to be noticed by others, and spending a lot of time surfing the Internet or playing video games. Although first described in Japan, cases have been described from around the world. Studies on *hikikomori* available in the literature indicate a wide range of disorders diagnosed in this group of patients such as anxiety disorders, personality disorders, mood disorders, and psychotic disorders. However, it is most often observed with ASD (autism spectrum disorder). The aim of this study is to consider *hikikomori* as a specific presentation of psychotic decompensation in people with ASD. In the case of a 10-year-old boy with ASD, sulpiride brought an improvement in social withdrawal, which, together with the symptoms presented by him, may indicate the possibility of psychotic decompensation in the form of *hikikomori*. Due to the lack of therapeutic guidelines for the management of people with *hikikomori*, this may be a valuable guideline for the implementation of appropriate pharmacotherapy by clinicians, in which attention should also be paid to frequently occurring other psychiatric disorders.

Key words: *hikikomori,* autism spectrum disorders, psychotic decompensation

Introduction

The term *hikikomori,* or rather *shakaiteki hikikomori*, refers to the Japanese term for severe social withdrawal. The concept consists of the adjective *shakaiteki* meaning

'social' and the two verbs *hiku* and *komoru.* The words that make up the second part of this concept are assigned the following meanings: *hiku* – 'to withdraw', 'to give up', 'to leave'; *komoru* – 'to be in the castle and protect oneself', 'to remain in the temple and pray', 'to enter and hide', 'to be inside and not to go out' [1].

The use of the term *hikikomori* to refer to the process of acute social withdrawal syndrome dates back to the mid-1980s and was first used in an academic context by Norihiko Kitao [2]. In the scientific literature, however, the definition of this phenomenon was outlined and popularized in the 1990s by the Japanese psychiatrist Tamaki Saitou in his book *Shakaiteki hikikomori: Owaranai shishunki* [3]. Saitou, based on his clinical experience, defined hikikomori as "a condition that became a problem in the late 1920s, consisting of being confined to one's own home and not participating in society for six months or more, but not associated with any other mental problem that would cause it." According to his estimates, about one million Japanese children and adolescents were affected [4].

Although *hikikomori* has not yet been recognized as an independent diagnostic unit within the ICD-11 and DSM-5 classifications, attempts are made in the literature to define its diagnostic criteria. Exemplary diagnostic criteria were proposed in 2003 by the Japanese Ministry of Health, Labor and Social Welfare [5]. Then, in 2010, researchers Teo and Gaw [6] proposed specific criteria for diagnosing *hikikomori* for the DSM-5 classification, which included:

- Spending most of the day, almost every day, only in one room (usually in one's bedroom);
- Explicit and persistent avoidance of situations (e.g., attending school, working) and social relationships (e.g., friendships, contact with family members);
- The social withdrawal and avoidance interferes significantly with the person's normal routine, occupational (or academic) functioning, or social activities or relationships;
- The person perceives the withdrawal as ego-syntonic;
- In individuals under age 18 years, the duration is at least 6 months;
- The social withdrawal and avoidance are not better accounted for by another mental disorder, such as social phobia (e.g., avoidance of social situations because of fear of embarrassment), major depressive disorder (e.g., avoidance of social situations as a reflection of neurovegetative symptoms), schizophrenia (e.g., isolation due to negative symptoms of psychosis), or avoidant personality disorder (e.g., isolation due to fears of criticism or rejection).

A person with *hikikomori* had to meet each of the six proposed criteria. In their work, the researchers emphasized that an important feature of this disorder is prolonged social withdrawal. This phenomenon was characterized by spending most of the day and almost every day in one room, usually in one's bedroom, visibly avoiding social situations and interpersonal relationships, and leaving one's room only at night so as not to be noticed by others, and spending a lot of time surfing the Internet, reading or playing video games.

Studies on *hikikomori* available in the literature indicate a wide range of disorders diagnosed in this group of patients. Anxiety disorders (22%), personality disorders (18%), mood disorders (14%), and psychotic disorders (8%), among others, are observed. However, it is most often observed with autism spectrum disorders (27%) [7]. It also has to do with Internet abuse, although whether *hikikomori* causes Internet abuse or Internet addiction causes *hikikomori* remains debatable [8]. In a 2011 study of *hikikomori* people attending mental health centers in Japan, 33.3% had schizophrenia, mood disorders, or anxiety disorders, 32% had autism spectrum disorders or intellectual disabilities, and 34.7% had personality disorders or adjustment disorders. A third of these people needed drug treatment. In each of these groups, family problems were the most common factor in *hikikomori*. This was also compared with a group that did not seek help from mental health centers, but showed symptoms of *hikikomori*, and it turned out that their mental condition was weaker according to GAF (The Global Assessment of Functioning) [9].

Decompensation means the disintegration of a certain structure or system that previously functioned with the help of compensation systems. In psychology, decompensation is understood as the loss of proper defense mechanisms in response to, for example, stress. In the course of, for example, personality disorders or autism spectrum disorders, patients may compensate in the direction of, for example, persecutory delusions and delusions in order to defend themselves against a reality that is difficult to accept. Of course, the symptoms of psychotic decompensation may present a prodrome of psychosis, e.g., schizophrenic, but they may also occur as an independent diagnostic phenomenon. The results of a 2022 meta-analysis indicate that the co-occurrence of psychosis in people with ASD (autism spectrum disorder) is 9.4% ($N = 63{,}657$; – 95% CI = 7.52; –11.72), which is higher than in the general population [10]. However, whether and what autistic symptoms can lead to the development of psychosis is still being studied. The best-proven predictors of conversion to psychosis include genetic risk, a history of substance abuse and the degree of social deficit [11]. From a genetic perspective, it is worth noting several genes and chromosomal regions whose abnormalities may be present in both ASD and psychosis (e.g., deletion in chromosomal region 22q11.2 [12]), genes involved in synaptogenesis and synaptic maturation (CNVs) [13], and oxytocin genes [14, 15]. It is important to mention that genes are a marker of susceptibility rather than a reliable predictor of the development of ASD or schizophrenia, and it is only by interacting with other factors (e.g., environmental) that any of these disorders can develop. Preliminary results have also been published indicating that people at high risk of psychosis and ASD who have developed psychosis have a unique pattern of heightened neural response when focusing attention on both visual stimuli and images during EEG testing (electroencephalography) [16].

Given the common clinical features of psychosis and ASD, it is social and cognitive deficits that may promote the development of psychotic experience in individuals with ASD [17, 18]. A huge cohort study ($N = 5{,}359$; 12 years of follow-up) confirmed the hypothesis that children with ASD or high autistic traits scores would be more likely to report psychotic experiences. A diagnosis of ASD increased the likelihood of psychotic experiences by almost 3-fold, and an increase in the mean

autistic trait score of 1 *SD* was associated with a 17% increase in the chances of psychotic experiences. Some of these effects depended on the IQ of the subjects. Factors that particularly predisposed to psychotic experiences were disorders in semantic-pragmatic skills, articulation, repetitive stereotypical behaviors, and social inhibition [19]. However, among many adolescents with ASD who have psychotic experiences, a large percentage will never have a full psychotic episode. A 2021 meta-analysis found that people with ASD have features of prodromal psychosis. They are more common in men, but women may have more severe symptoms. However, the occurrence of ASD is not associated with conversion to full psychosis, but only with a psychotic experience [20].

A 2015 study concluded that no model can currently be seen as the "best fit" to explain the co-occurrence of ASD and psychosis, and it is important that future studies include strong evidence for the existence of different subgroups of the population within both disorders. When studying these subgroups, it is important to consider the evidence at multiple levels, both phenotypic and genotypic, because, while this is certainly not the case for all populations of SSDs (schizophrenia spectrum disorders), there is evidence to suggest that ASD may provide a pathway to SSD for certain subgroups, including those with early childhood onset schizophrenia, with McDD (multiple-complex developmental disorder) and deficit schizophrenia. This is related to the increased vulnerability model, which assumes that it is not the common propensity that makes individuals vulnerable to the development of both ASD and SSD, but the actual development of ASD itself (or the high level of ASD traits), that increases the susceptibility to SSD [21].

In addition to psychotic decompensation, *hikikomori* may be associated with, for example, the prodromal phase of psychosis or the negative symptoms of schizophrenia. Symptoms that link the two are social withdrawal, deterioration of social role-related functions, impaired hygiene, loss of drive, distrust, irritability, depressive mood, sleep disturbance, and loss of concentration. However, behavioral strangeness and other negative symptoms in addition to social isolation, such as cognitive deficits, are not necessarily present in *hikikomori* as opposed to schizophrenia. Sensory deprivation resulting from social withdrawal may exacerbate psychotic symptoms in *hikikomori* as well, blurring the line between the two diagnoses. In the absence of overt psychotic symptoms suggestive of an acute episode of psychosis, environmental modification may help to differentiate between *hikikomori*, psychosis and Internet abuse. The chronological development of symptoms may be another indication of which condition appeared first and "triggered" the other [22]. In a study published in 2012, a group of researchers asked psychiatrists what psychiatric disorder might be the one underlying *hikikomori* and 30% of them said it was schizophrenia [23]. It is important to remember that the negative symptoms are specific to psychosis and, above all, should suggest this diagnosis.

Furthermore, the literature points out that depressive disorders should be ruled out before considering a diagnosis of *hikikomori*. This is particularly important in view of the fact that in addition to the psychotic form of decompensation, mood decompensation – e.g., depressive decompensation – is also observed in the course of

ASD. Some researchers claim that the lack of interest in leaving the house, as well as withdrawal from social relationships, are the main features that connect hikikomori and depressive disorders and that they result from apathy, anergy or anhedonia. It has been concluded that people with *hikikomori* avoid social situations not because of anxiety or fear, but because of apathy [24, 25]. Kato et al. [24] describe that epidemiological studies conducted during the COVID-19 pandemic have shown that depression and modern depression (MTD) are also associated with *hikikomori*. MTD is a condition characterized by situation-dependent depressive symptoms and a visible tendency to social withdrawal. Lack of motivation to fulfill social roles and low self-esteem were common features of MTD and *hikikomori*. In a study comparing patients with depressive disorders and *hikikomori*, the tendency to MTD was higher in patients with *hikikomori*. The cross-lagged effects model implemented by Kato et al. [24] suggested a complex relationship between *hikikomori* and MTD, showed intercorrelation and supported the hypothesis that MTD is a transitional state to *hikikomori* and suggested that *hikikomori* may also be a transitional state and lead to MTD [26]. Furthermore, in people with autism spectrum disorder, the course of depressive disorders may be atypical and difficult to recognize due to cognitive, social and communication impairments characteristic of these individuals, and autistic features may mask depressive symptoms [27]. Therefore, the prevalence of depression in individuals with ASD in the literature is variable and may be underestimated. According to the meta-analysis by Hollocks et al. [28] 37% of people with ASD meet criteria for a depressive episode during their lifetime.

The aim of this study is to consider *hikikomori* as a specific presentation of psychotic decompensation in people with autism spectrum disorders and the pharmacological and therapeutic management of this type of patients based on the case of a patient diagnosed with psychotic decompensation in a person with ASD. The patient's details were anonymized and parental consent was obtained for the case report.

Case report

A 10-year-old patient was consulted in a mental health outpatient clinic for "anxiety" combined with nocturnal sleep disturbances, withdrawal from peer and family relationships, and episodes of aggressive behavior in response to frustration – essentially limited computer use. According to the parents, the patient was observed to be averse to social exposure – he went to school and then returned home directly. He did not maintain peer relationships. At home, he spent time with his family, although he preferred to stay in his room, he devoted a lot of time to computer-related activities – he played online games, and according to his mother, he also had friends via instant messaging. When parents tried to limit the boy's use of the Internet or computer, the patient became nervous and showed aggressive behavior towards the environment.

In the study of the patient's mental state, mood and psychomotor drive were balanced, affect were blunted, contact with him was excessively specific, he answered questions briefly, in the subject line of questions. He presented a limited understanding

of metaphor, and in the tests of the mystery box and pictorial metaphors, he presented incorrect answers. He negated the productive symptoms. He negated the suicidal thoughts and tendencies. With the initial diagnosis of pervasive developmental disorders, a follow-up at the mental health outpatient clinic was recommended and a referral was made to psychotherapy (which was not started) and to the neurology clinic.

Over the next 3 years, the patient made several visits to the mental health outpatient clinic, during which the focus was on the issue of his aggressive behavior, which gradually escalated over that time. Psychological and neurological diagnostics were also carried out, and he was diagnosed with ASD. Attempts have been made to introduce aripiprazole and risperidone due to the increase in aggressive behavior, however, the profile of side effects caused parents to discontinue the drugs and not agree to their re-introduction.

After 4 years from the first visit, the parents again reported the patient to the clinic, however, this time they reported a sudden change in the patient's functioning and behavior within a period of about 7 months – withdrawal from social and family functioning. The patient refused to attend school. He was spending days locked in his room and refused to leave the room even for meals. According to his parents, he even limited his use of the computer to some extent, spending most of his time in bed. He stopped self-care in terms of personal hygiene and cleanliness of the environment. When trying to motivate him to leave the room or undertake other activities, the patient presented aggressive behavior.

During the examination, the patient was well-oriented to place, time and self, in a neutral mood, with a decreased psychomotor activity. In contact, he answered questions briefly, with single words, reluctantly. When moving on to topics that interested him, he developed his statements more, presenting disturbances in the structure of thinking (derailment, loosening of association, sometimes paralogical thinking and ambisentence – the patient not presenting insight into syncretism of his statements). He reported the occurrence of a void in his head. He negated hallucinations and did not introduce delusional content. He refused to give a reason for his withdrawal from life and functioning, claiming that he did not understand why "everyone cannot simply give him peace of mind." He negated the suicidal thoughts and tendencies.

The patient was diagnosed with psychotic decompensation in a person with autism spectrum disorder. Due to the observed clinical picture and the lack of parental consent for the use of aripiprazole, it was decided to initiate sulpiride treatment in the patient, initially at a dose of 200 mg per day. The implementation of pharmacotherapy brought a partial improvement in the parents' relationship, the patient became more active at home and began to take care of hygiene. However, an increase in irritability was observed, the patient became verbally aggressive towards parents and siblings, which resolved when the dose of sulpiride was increased to 400 mg per day. After about half a year of pharmacotherapy, the boy became more active, began to leave the house and undertook sporadic peer activities.

Overview

Psychotic decompensation, which can occur in autism spectrum disorders, is characterized by symptoms such as disturbances in thinking (e.g., delusions, disorganization in the form of thought), perception (e.g., hallucinations), self-experience (e.g., the experience that one's feelings, impulses, thoughts, or behavior are under the control of an external force), cognition (e.g., impaired attention, verbal memory and social cognition), volition (e.g., loss of motivation), affect (e.g., blunted emotional expression), and behavior (e.g., behavior that appears bizarre or purposeless; unpredictable or inappropriate emotional responses that interfere with the organization of behavior). Psychomotor disturbances, including catatonia, may be present. Persistent delusions, persistent hallucinations, thought disorder, and experiences of influence, passivity, or control are considered core symptoms [21].

Strip et al. [22] in their study indicated symptoms linking *hikikomori* and schizophrenia. According to the researchers, these were social withdrawal, deterioration of functions related to social role, deterioration of hygiene, decreased drive, distrust, irritability, depressed mood, sleep disorders, and concentration disorders. However, bizarre behavior and other negative symptoms, in addition to the aforementioned social isolation, such as cognitive deficits, do not necessarily occur in *hikikomori*, unlike schizophrenia.

The diagnosis of depressive decompensation in autism spectrum disorders includes the presence of 5 out of 10 symptoms for most of the day, almost every day and for at least 2 weeks. To be diagnosed, at least one of the symptoms belonging to the affective cluster must be present, i.e., depressive mood (in the pediatric population this may manifest as increased irritability) or anhedonia [29]. Comparing the criteria for the diagnosis of a depressive episode with those proposed by Teo and Gaw [6] for *hikikomori*, we cannot see the typical social withdrawal present for at least 6 months. In the *hikikomori* criteria, on the other hand, the typically depressed mood is not included. However, criteria such as decreased activity, psychomotor slowing, sleep disturbance, and decreased energy may be associated with *hikikomori*-typical social withdrawal.

The table below shows the symptoms comparing schizophrenia, *hikikomori* and depressive episode

Table 1. **Correlation between Hikikomori, schizophrenia and episode depressive episode**

Symptoms	*Hikikomori*	Schizophrenia	Depressive episode
Anti-sociality	+	+	-
Decreased psychomotor activity	+	+	+
Focus on the inner world	+	+	-
Impaired attention and concentration	+	+	+
Disorganization in the form of thought	-	+	-
Alogia	-	+	-

table continued on the next page

Thought echo, sending/distracting thoughts	-	+	-
Sleep disorders	+	+	+
Blunted emotional expression	+	+	-
Psychomotor disturbances such as catatonia	-	+	-/+
Persistent delusions	-	+	-/+
Hallucinations	-	+	-
Inappropiate/unpredicatble/bizarre behaviors	-	+	-
Depressive mood	-	-/+	+

In the case of the boy, it is possible to observe the fulfilment of the diagnostic criteria proposed by Teo and Gaw [6] diagnosing *hikikomori*, i.e., spending most of the day, almost every day, only in one room, which was the child's bedroom, and spending this time using the computer and playing online games. The patient had a clear withdrawal from peer relationships, as well as a great reluctance to any social interactions, which significantly affected the boy's proper functioning. The boy did not see his withdrawal as abnormal, any attempt to activate the boy or limit access to the computer resulted in frustration leading to episodes of aggression. The patient met virtually all of the proposed diagnostic criteria for the diagnosis of *hikikomori*.

However, with the passage of time, the negative symptoms worsened. Previously, despite social withdrawal, the patient had completed his compulsory education and attended school regularly. Suddenly, he refused to attend school, and spent the whole day in his room, mostly in bed. Hygiene negligence was observed. He has noticeably reduced his use of the computer, where he used to spend most of his day in this way. The severity of these symptoms may also result from the occurrence of, among others, persecutory delusions and delusions of reference. In addition, the observed disturbances in the structure of thinking and ambisentence may suggest the diagnosis of psychotic decompensation in a person with ASD.

Due to the presented clinical picture, in which the patient did not present unambiguously psychotic symptoms, and negative symptoms and disturbances in the structure of thinking dominated, it was decided to include sulpiride in doses of 200–400 mg. Sulpiride is used in both schizophrenia and depression. Sulpiride is a benzamine derivative and the dose is adjusted depending on the symptoms. At lower doses, 200–400 mg, sulpiride preferentially blocks presynaptic D2 receptors, resulting in increased dopaminergic transmission. In the doses used, this drug has a weak antipsychotic effect, but it has a strong activating effect [30]. The use of the above-mentioned pharmacotherapy resulted in a gradual disappearance of the ailments. In addition, the quality of contact with the patient improved, the patient spoke in a logical way, and there were no visible disorders of the thinking structure in his statements.

The treatment of *hikikomori* is not clearly described. The available literature is much more focused on describing its epidemiology and symptoms [31]. As in other psychiatric disorders, the combination of pharmacological treatment and psycho-

therapy is valuable. The vast majority of studies on both larger groups of patients and individual cases of *hikikomori* describe psychotherapeutic management [31]. It concerns, for example, psychoeducation of families of people presenting with *hikikomori* symptoms, which is very important, especially since people who isolate themselves are often aggressive towards their roommates when trying to "drag" them out of the house [24]. In addition, parental involvement in treatment is effective in reducing anxiety in adolescents [32]. The CRAFT (Community Reinforcement and Family Training) program, which was originally developed on the basis of cognitive-behavioral therapy for family members of substance addicts, is most often used for family psychoeducation [33]. Other therapeutic interventions include home visits, social skills training, physical activity, and support groups [31]. However, the quality of the evidence for the operation of these procedures is low due to the high potential for statistical bias and the lack of randomized trials [34].

A 2014 study found that 78% of people with *hikikomori* want to undergo any kind of treatment, and most of them prefer psychotherapy to drug treatment [35]. Interestingly, patients declared that they preferred in-person psychotherapy rather than online [35], which somewhat contrasts with the study by Imai et al. [36], which proved that *hikikomori* patients rarely attend in-person appointments, and that the regularity of visits can be increased by "support from clinical staff". That is why home visits to patients are an increasingly common method, as they help the patient to reintegrate into society in a safe and gradual way. It is essential here to prepare the involved environment to reach the person with *hikikomori*, to maintain constant communication with him, and to expand the scope of activities and relationships, which supports them in finding their own way to participate in society [34]. Japanese psychiatrists are also more likely to choose psychotherapy over medication and outpatient treatment than inpatient treatment [37].

As far as pharmacological treatment is concerned, there are few reported cases in the literature and almost all of them involve the use of SSRIs (selective serotonin reuptake inhibitors) with varying degrees of effectiveness. One example is the case of a 35-year-old patient from Brazil who had hardly left his home for 14 months, including 3 months of depressive symptoms. Upon admission to the clinic, the patient took methylphenidate (54 mg/day) and desvenlafaxine (100 mg/day), which was changed to sertraline (200 mg/day). After a month, there was a reduction in depressive symptoms, but the symptoms of *hikikomori* did not decrease, so it was decided to include cognitive-behavioral therapy and psychoeducation of the patient's mother, which after four months resulted in the withdrawal of *hikikomori* symptoms – the patient began to leave the house, look for a job and engage in social life. This improvement continued after a few months [38]. Another case involving SSRIs is the use of paroxetine and the resolution of *hikikomori* symptoms in a person with concomitant OCD (obsessive-compulsive disorder) who had not left home for 10 years [39]. In patients with social anxiety and *hikikomori*, the combination of cognitive-behavioral psychotherapy with group therapy and pharmacological treatment (one of three drugs: paroxetine at a maximum dose of 40 mg/day, fluvoxamine at a maximum dose of 150 mg/day, or milnacipran at a maximum dose of 100 mg/day) resulted in an improvement defined

by the start of work or studies in only 37% of the subjects, and this after an average of two years [40]. However, the rate of improvement in this study was significantly better than in another *hikikomori* study, in which only five (17%) of the 29 *hikikomori* patients entered work (including part-time or with social care support) [41]. SSRIs (50 mg of sertraline) were also used in a 25-year-old man from Spain who had been at home for 4 years. In combination with psychotherapy and ad hoc lorazepam, they resulted in improvement [42]. The only case in the literature of a person with *hikikomori* being given drugs other than antidepressants concerns a 15-year-old boy from Turkey, who was hygienically neglected and agitated at the first psychiatric consultation, was therefore prescribed 0.5 mg of risperidone, and after only a few days there was an improvement. This was the case most similar to the one we described in terms of the patient's symptoms and treatment (both sulpiride and risperidone are antagonists of dopaminergic D2 and D3 receptors, although sulpiride is more selective here). It is worth noting that the boy in Turkey was ruled out with ASD and schizophrenia [43].

As can be seen from the given examples, there is still a lack of a concerted pharmacological management in *hikikomori*, so the treatment is somewhat "intuitive", and the implementation of treatment should also take into account the frequent co-occurrence of *hikikomori* with other mental disorders and, as can be seen in the case we have described, the possibility of psychotic decompensation in autism spectrum disorders.

Discussion

Assuming the hypothesis that *hikikomori* is in fact a specific form of, for example, psychotic decompensation in the course of autism spectrum disorders, the question arises as to why this phenomenon is so widespread in Japan (1.2%), and in other countries rather isolated cases are described. Of course, an instinctive explanation may be that the concept of *hikikomori* itself originated in Japan and is only beginning to penetrate the scientific community in other countries. Again, however, this raises the question of why this phenomenon has been described and conceptualized as a new psychopathological syndrome in Japan. The source of such a situation can be found, for example, in the cultural background, but in the sense of "particular" rather than "universal" as defined by Vogeley & Roepstorff in a 2009 publication [44]. Social cognition, understood as a set of mechanisms enabling proper communication and functioning in a group, although in its essential part common to all people (e.g., in the field of facial expressions), is subject to strong cultural modeling. The principles of functioning in groups, the moral code or the work ethos expected and imposed by the environment internalized during development have a very strong impact on the ways of e.g., emotional expression, views and even individual aspirations and expectations. Of course, this type of phenomenon within a given population will lead to a kind of natural selection and evolutionary "adjustment" of people to the fulfilment of these expectations. Thus, according to this conceptualization, cultural pressure is realized both at the individual level (through the adoption of general beliefs) and at the level of entire sociocultural groups, in which new mechanisms of evolutionary selection appear, unknown to other groups. Of course, for this type of phenomenon to occur,

a sufficiently long period of time is needed in which a given society is relatively separated from other populations in which cultural dogmas differ significantly. Interestingly, a society shaped in this way will obviously also change the cultural conditions that are its product, leading to the further perpetuation of certain patterns [11, 44]. An example of this type of phenomenon can be found, among others, in the study of Kobayashi et al. [45], where it was pointed out that depending on the sociocultural background, significant differences in the neural correlates of the theory of the mental mechanism are observed.

In the case of Japanese culture/society, there is a broad category of publications in literature called *nihonjinron*, focusing on its differences from other cultures. These include, among others, differences in the level of intensity of emotional expressions (in Japan, excessive expressions are perceived as rude) or greater directness in conversation on issues such as relationships. Importantly, cultural differences in the scope of these functions are also reflected, for example, in functional magnetic resonance imaging studies, where differences in the range of structures to be activated in the Japanese population compared to the American-Caucasian population were observed in tasks based on theory of mind. Hypothetically, this type of cultural conditioning could promote a higher prevalence of autistic traits in the population. And in fact, this is reflected, for example, in the increased prevalence of ASD in the Japanese population, which in the 2021 literature review of Sasayama et al. [46] was as high as 3% of the population. Meanwhile, in a 2022 review by Salari et al. [47], the worldwide prevalence of ASD was around 0.6% (1% in America and 0.5% in Europe). Given the nearly six-fold higher proportion of people with ASD in the Japanese population, it is safe to assume that the proportion of people with the so-called broad autism phenotype (BAP) will also be significantly higher than in the European population (where it is as high as 25% in some studies) [48]. Such a high prevalence of severe (even subclinical) ASD features in the population will predispose to a higher incidence of psychotic decompensation symptoms.

Clinical presentation based on social withdrawal and confinement at home may be a consequence of the axial symptoms of autism, where difficulties in communication and in establishing and maintaining social relationships lead to the occurrence of persecutory delusions or delusions about the environment, and thus fear of social exposure. In fact, in 2020, the AQ-J (The Autism-Spectrum Quotient, Japanese version) questionnaire was conducted in people with *hikikomori*. They found that compared to people without *hikikomori*, they had a higher severity of autistic traits, especially when it came to poorer social skills, poorer communication and poorer imagination, as well as lack of support and teasing from peers [49]. Another study examined autistic traits and *hikikomori* propensity. It turned out that the main autistic trait predisposing to *hikikomori* is difficulty in social interactions mainly due to a lack of support from other people, which can lead to failure in the workplace or school and consequently social withdrawal. It is rarely associated with internal stressors [50].

Recapitulation

In the case of a 10-year-old boy with ASD, sulpiride brought an improvement in social withdrawal, which, together with the symptoms presented by him, may indicate the possibility of psychotic decompensation in the form of *hikikomori*. Due to the lack of therapeutic guidelines for the management of people with *hikikomori* and the lack of randomized trials in the literature with an adequate number of studies on this topic, this may be a valuable guideline for the implementation of appropriate pharmacotherapy by clinicians, in which attention should also be paid to frequently occurring other psychiatric disorders. However, this is a case report and needs to be confirmed in further studies in larger groups of patients.

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