Archives of Psychiatry and Psychotherapy Online First Nr 31: 1–8

10.12740/APP/205141

A 33-year-old man with memory gaps: a case study of a patient with suspected Fahr's disease

Jakub Michał Kucharski, Marta Demkow – Jania, Anna Klimkiewicz

Abstract:

Memory disturbances are observed across various psychiatric disorders and can occasionally occur in healthy individuals. These disturbances can affect different memory components and can manifest with varying degrees of severity. In some cases, memory gaps are filled with untrue memories. Differential diagnosis of memory impairments requires consideration of both psychiatric conditions (e.g., dementia, ADHD) and neurological disorders (e.g. brain damage in the hippocampal regions). This paper presents the case of a 33-year-old man who sought treatment at a Psychiatric Outpatient Clinic due to low mood, anxiety attacks, and memory problems. Initially, he was diagnosed with mixed anxiety and depressive disorder. However, during the diagnostic process, the patient revealed other atypical symptoms, including episodic memory gaps, false memories of brain surgery, and thought content disturbances. The differential diagnosis included, among others, Fahr's disease. Numerous examinations and neuropsychological testing finally led to the appropriate diagnosis—personality disorder and panic disorder. This case highlights the importance of collaboration between psychiatrists and clinical psychologists in the diagnostic process. Additionally, it draws attention to the possibility of atypical clinical presentations of disorders.

personality disorders; Fahr's syndrome; memory disturbances

INTRODUCTION

Accurate diagnosis and the selection of the most appropriate treatment often require close collaboration between psychiatrists and clinical psychologists. Their complementary skills and diverse diagnostic tools enable a comprehensive assessment of the patient's condition, ensuring adequate care. This is particularly critical when the clinical situation is ambiguous, and the symptoms do not form typical psychopathological syndromes. Such was the case for the patient whose history is the subject of this paper.

Jakub Michał Kucharski[1], **Marta Demkow – Jania**[1], **Anna Klimkiewicz:** Department of Psychiatry, Medical University of Warsaw, Nowowiejska St. 27, 00-665 Warsaw, Poland.
Correspondence address: kuba.kucharski.97@gmail.com

The aim of presented case is to highlight how psychopathological symptoms, seemingly stemming from distinct domains, can result from decompensation within severe personality disorder. Moreover, it underscores the importance of teamwork between psychiatrists and clinical psychologists in the diagnostic process.

Memory impairment manifests as a feature in various clinical conditions for example: dementia, depression, anxiety disorders, head trauma, human immunodeficiency virus (HIV) infection, neurosyphilis, vitamin deficiencies, thyroid diseases, and the effects of pharmacological agents on cognitive functions [1,2]. A comprehensive assessment of symptoms necessitates a thorough psychiatric and physical examination (including a neurological evaluation), as well as laborato-

ry tests, neuroimaging, neuropsychological assessments, and, in some cases, genetic testing.

Currently, there are no definitive diagnostic standards for early-onset memory disorders. However, DSM-5 and ICD-11 present diagnostic criteria for Major Neurocognitive Disorder, which corresponds to dementia. According to DSM-5, the diagnosis requires substantial impairment to be present in one or more cognitive domains (usually more). The impairment must be sufficient to interfere with independence in everyday activities [7]. On the other hand, ICD-11 requires marked impairment in two or more cognitive domains. Additionally, cognitive decline must not be attributable to normal aging [8].

The diagnosis of personality disorders is likewise a complex process. According to dimensional-categorical approach, it requires an evaluation of the degree of functional impairment and an assessment of pathological personality traits.[3,4,5) Psychometric tools, such as the *Level of Personality Functioning Scale–Brief Form 2.0 or The Personality Inventory for DSM-5*, may be useful in establishing an accurate diagnosis.

DSM-5 criteria for personality disorder include an enduring pattern of inner experience and behavior that deviates markedly from the expectations of the individual's culture. This pattern must be manifested in two (or more) of the following areas: cognition, affectivity, interpersonal functioning, or impulse control. The pattern must be inflexible and pervasive across a broad range of personal and social situations; it must lead to clinically significant distress or impairment in social, occupational, or other important areas of functioning. Additionally, the pattern must be stable and of long duration, with its onset traceable back at least to adolescence or early adulthood [7]. ICD-11 criteria emphasize that personality disorder is characterized by problems in functioning of aspects of the self and/or interpersonal dysfunction. The disturbance must manifest in patterns of cognition, emotional experience, emotional expression, and behavior that are maladaptive, and it may be consistently evoked by particular types of circumstances and not others [8].

CASE REPORT

The sources of information about the patient used in this case study included: psychiatric examination results, psychological tests, biochemical tests, imaging studies, outpatient clinic records, hospital treatment records, diagnostic interviews with the patient and his family and electroencephalography results.

A 33-year-old patient, presented to the Psychiatric Outpatient Clinic due to a six-month history of low mood and diminished drive. A few days prior to his visit, he experienced his first-ever panic attack, which prompted him to seek help. The panic attack occurred after a stressful conversation with his partner regarding raising a mortgage for an apartment. The patient denied any other significant events which could contribute to his deteriorating mental condition.

During the outpatient assessment, the patient was oriented in person, place, and time. He spontaneously engaged in coherent verbal communication and exhibited calm, appropriate behavior. He reported intensified symptoms, including anxiety, anergia, anhedonia, and increased tearfulness. He reported a single panic attack accompanied by strong vegetative symptoms, such as heart palpitations and dyspnea. He also reported concentration difficulties, troubles falling asleep, and nightmares. His partner noted a gradual withdrawal from social life over the past two years. The patient denied suicidal thoughts, attempts, or self-harm but admitted to fleeting resignation thoughts. Additionally, he reported episodic memory difficulties, such as an inability to recall details about meeting his wife for the first time or their engagement. No psychotic symptoms or abnormalities of the form of thinking were observed. He denied any history of elevated mood.

The patient had a history of psychiatric treatment for school phobia during adolescence, with good response to fluoxetine. Since then, he had not been under psychiatric care or on psychotropic medication. He had never received psychotherapy. Approximately seven years earlier he reported surgery for a frontal lobe glioma but was unable to provide details or medical records related to the treatment. He denied other somatic conditions, including epilepsy, meningitis, or Lyme disease. Routine blood tests (com-

plete blood count, C-reactive protein, TSH, electrolytes, glucose, lipid profile, liver and kidney function parameters, folate and B12 levels) revealed no abnormalities. His physical and neurological examinations were also unremarkable.

The 33-year-old reported no alcohol consumption, occasional smoking, and no use of psychoactive substances. He had no criminal history. The patient had a high school education but stated he chose not to take his final exams. He worked as a part-time taxi driver and assisted his father in running a cleaning business. Married for two years and childless, he described his wife as supportive and their marriage as happy. He had positive memories of his upbringing and maintained close relationships with his parents and sister. He reported never experiencing any form of abuse. The family history revealed his mother had been treated for mixed anxiety and depressive disorder, his maternal uncle had a history of alcohol dependence, and his grandfather's sister had schizophrenia. Otherwise, the family psychiatric history was unremarkable.

At the Psychiatric Outpatient Clinic, the patient was diagnosed with mixed anxiety and depressive disorder. He was prescribed sertraline, titrated to 100 mg daily, trazodone CR at 50 mg for sleep, and alprazolam 0.25 mg as needed for panic attacks. Psychotherapy was recommended, and a follow-up appointment was scheduled for six weeks later. The patient was instructed to provide documentation related to his previous brain tumor treatment.

After six weeks of treatment, he reported slight improvement in mood and resolution of panic attacks. However, apathy and memory problems persisted. Additional history from his partner revealed that a few years earlier, the patient had taken out a significant loan but could not recall its purpose or how the money was spent. Initially, he claimed the funds were needed for brain surgery, but this was inconsistent with the timeline of the alleged operation. Recently, he had contacted various neurosurgical departments in an attempt to locate medical records, without success.

The patient was admitted to a neurology department for further diagnostics. Brain MRI (with and without contrast) revealed widened cerebrospinal fluid spaces over the frontal and, to a lesser extent, parietal lobes. Gradient-echo imaging showed signal voids in the globus pallidus consistent with degenerative changes. There was no evidence of prior intracranial surgery. When informed of these findings, the 33-year-old expressed surprise and was unable to explain his strong belief in the history of brain tumor and neurosurgical intervention. His family was equally shocked, recalling that he had regularly traveled weekly to a tertiary medical center seven years earlier. At the time, his relationship with his current wife was in its early stages, and she never asked about specifics of his treatment.

During the hospitalization EEG results were within normal limits. Lumbar puncture with cerebrospinal fluid analysis was performed. It included tests for Lyme disease (IgG and IgM), Alzheimer's disease (phospho-tau protein), and autoimmune encephalitis (antibodies against NMDA, AMPA1, AMPA2, DPPX, LGI1, CASPR2, and GABAb antigens). No abnormalities were shown. Given incomplete symptom remission, sertraline was replaced with duloxetine (up to 60 mg daily) and pregabalin (150 mg daily) was introduced. Neurological follow-up for memory disturbances was recommended. Dissociative disorders were considered, though no clear traumatic trigger was identified.

One week post-discharge, the 33-year-old returned to the outpatient clinic in significant distress. His behavior was withdrawn, and he communicated minimally, exhibiting severe anxiety and marked depressive symptoms. His condition appeared to worsen after his wife discovered additional loan agreements totaling approximately the patient's annual income, which he did not recall signing. Duloxetine was discontinued, and olanzapine at 10 mg daily was initiated.

A month later, the patient reported slight improvement in anxiety and sleep but complained of sedation and increased appetite. Attempts by his wife to discuss debts and the alleged brain tumor triggered panic attacks, alleviated by alprazolam 0.25 mg. Olanzapine was replaced with risperidone (4 mg daily). After several weeks, he experienced a delusional episode in which he believed his wife had installed surveillance cameras in their home. This prompted an emergency hospital admission to the Department of Psychiatry at the Medical University of Warsaw for further evaluation and treatment.

During the patient's stay in the psychiatric ward, no psychotic symptoms were observed, only significantly exacerbated depressive-anxiety symptoms, including panic and generalized anxiety, low mood and drive, apathy, anhedonia, sleep continuity disturbances, and difficulty concentrating. Basic blood tests were performed, which did not reveal any clinically significant abnormalities. Risperidone was discontinued and sertraline was introduced at a dose of 200 mg daily, alongside trazodone at a dose of up to 150 mg daily for sleep. Benzodiazepines were also used on an as-needed basis during the initial days of hospitalization.

During the hospitalization, there was a suspicion of Fahr's disease due to the ambiguous symptoms (depressive, anxiety, psychotic) and changes observed in imaging studies (degeneration in the globus pallidus). Fahr's disease is a rare neurological disorder characterized by the presence of calcifications in the basal ganglia, which can manifest with neuropsychiatric symptoms such as mood disturbances, cognitive dysfunctions, and behavioral changes.(6) A repeat head MRI was conducted, and after a radiological consultation, this diagnosis was excluded due to the mild degree of changes in the basal ganglia, which were described as discrete. The MRI also did not show any abnormalities in brain structures related to the processing of memory traces. A repeat EEG was ordered, and its results were within normal limits.

In the psychiatric ward, a series of neuropsychological tests were performed (Benton Visual Retention Test, Rey-Osterrieth Complex Figure Task, Rey Auditory Verbal Learning Test, Bender-Gestalt Test, DCS-II Test, 10-pair association learning test, Color Trails Test CTT-1 and CTT-2) to confirm or exclude any organic basis for the symptoms. However, the results did not suggest any intellectual decline of organic origin. The patient retained the ability to memorize and recall both auditory and visual information after a time delay. The quantitative results of all tests were within normal limits.

Subsequently, the WAIS-R (PL) test was administered, which indicated that the patient's overall intelligence level was above average on the full scale.

The next step was for the patient to complete the SCID-5-SPQ personality questionnaire, in which he provided responses consistent with what is known as the "Cluster C" pattern, suggesting significant traits of avoidant, dependent, and obsessive-compulsive personality disorders. In the psychologist's conclusions, it was noted that "the patient exhibits a panic fear of abandonment and has a tendency to fantasize in confrontational situations or to shield himself with memory lapses."

The 33-year-old then completed the MMPI-2(R) test. Based on the results and numerous conversations with the patient, the clinical psychologist described him as "presenting a neurotic profile, experiencing brief and intense anxiety states escalating to panic attacks, chronically feeling tense, nervous, and, secondarily, a sense of depression and hopelessness." The tests indicated that the patient's concentration, attention, and memory disturbances had no organic basis but were due to his "inability to develop appropriate coping strategies in difficult relational situations." This, in turn, was a consequence of maladaptive personality patterns, predominantly dependent and avoidant in nature. The symptoms revealed in the clinical picture were most likely of psychological, rather than organic, origin.

During hospitalization, numerous conversations were held with the patient, his wife, parents, and sister. In these discussions, the patient showed traits of dependent personality towards his wife and had a profound fear of separation. This anxiety seemed to have escalated over the past few years, possibly related to growing inequalities between the partners (in education, earnings, career, and social relations—on the patient's disadvantage). After many hours of sessions with the clinical psychologist, the patient finally admitted that although he did not recall the circumstances of taking the loans, he was almost certain that they were taken to balance the financial disparity between him and his wife. He did not admit to this because he was terrified of losing his partner. It seems that the patient indeed did not have access to these memories; however, the likely cause was not a brain disease, but rather the decompensation of his personality disorder. Its peak occurred when the patient experienced a quasi-psychotic episode, during which he expressed the belief that his wife had installed cameras in the house to monitor him.

What is more, the extended psychiatric interviewing revealed that the patient's relationship with his parents was not as close as he had reported in the beginning. It turned out that he received a pattern of inconsistent care from them. The parents were often "emotionally unavailable" and showed no concern for his needs, behaviors or everyday struggles. It seems that neglect from his parents might have led to the development of an anxious attachment style by the 33-year-old.

The patient's belief in a supposed brain tumor arose during his early acquaintance with his future wife, while courting her. The 33-year-old admitted that during this period, the care, compassion following treatment, and fear of illness were emotions that brought them closer, and the patient derived benefits from the supposed illness.

After several weeks of hospitalization, the patient was discharged with a diagnosis of mixed personality disorder, primarily characterized by avoidant and dependent traits, and panic disorder. Proper treatment with sertraline at a dose of 200 mg daily led to significantly reduced frequency of panic attacks. For the continuation of the treatment, he was referred to psychotherapy.

Discussion

According to the DSM-5, personality disorders are characterized by a persistent, pervasive, and inflexible pattern of behavior that leads to clinically significant distress or impairment in functioning [7]. The patient discussed in this case demonstrated traits from the so-called Cluster C, which includes avoidant, dependent, and obsessive-compulsive personality disorders. He experienced difficulties in separating from close individuals, fear of loneliness and rejection, hypersensitivity to social criticism, and insecurity in social interactions.

The latest International Classification of Diseases (ICD-11) suggests a shift away from specific personality disorder categories, proposing instead a classification based on the severity of the disorder and patterns of personality. Cluster C is reflected in patterns such as negative affectivity and anankastia [8].

The prevalence of avoidant personality disorder is estimated to be approximately 2.7%, dependent personality disorder around 1%, and obsessive-compulsive personality disorder about 2.5%.[9-12] Studies indicate that patients with anxiety-related personality disorders are significantly more likely to suffer from depressive and anxiety-spectrum disorders than those with other personality disorders or the general population [13]. They are also characterized by high levels of neuroticism [14].

The patient's primary reason for seeking psychiatric help was panic anxiety and low mood. Symptom exacerbation coincided with discussions with his wife about challenging life issues, such as their mortgage. Symptoms peaked when he was confronted with false or concealed events from his past (brain tumor, loans). His fears of losing his partner seemed to intensify alongside growing disparities between them in professional success, income, education, and social relationships. These dynamics likely triggered significant anxiety about losing his partner and her support and care.

An unusual symptom in this case was the patient's memory gaps, which he filled with false recollections. Research highlights the impact of anxiety-depressive disorders on neuropsychological functions, including memory and executive functions. A study by Airaksinen et al. (2005) showed that individuals with anxiety disorders exhibit significantly greater deficits in episodic memory and executive functions compared to the general population.[15) These abnormalities appear related to the encoding of memory traces rather than their retrieval. Hallford et al. (2022) and Spinhoven et al. (2009) demonstrated a tendency toward overgeneralization and reduced specificity in autobiographical memory in patients with depression and Cluster C disorders.[16,17) These findings offer a potential explanation for the patient's difficulties in recalling significant life events. Regarding unwanted memories, such as the concealed loans, psychological mechanisms like repression may have been involved.

An anxious attachment style also predisposes individuals to generate false memories (Hudson et al., 2018, 2023) [18,19]. Patients with this attachment style may misremember past events, as seen in this case. The untrue memory of a severe cancer diagnosis likely served an emotional regulation function and reinforced the patient's sense of identity, aligning with his current personal goals [18,20-22]. Moreover, his life circum-

stances, where close associates did not verify his claims, enabled such fabrications to persist.

Before being admitted to a psychiatric unit, the patient experienced a quasi-psychotic episode, reporting delusional thoughts about his wife installing cameras in their home to observe him. This episode was brief, isolated, and unaccompanied by other psychotic symptoms. It appears to have stemmed from psychological decompensation. Potentially, it could be a derealization phenomenon during a severe anxiety attack. The patient was fully critical of this episode and could not recall its details clearly, raising further questions about the nature of his symptoms while casting doubt on whether it was an acute psychotic episode.

Differential diagnosis ruled out schizophrenia. Although factors such as suspected psychosis, the patient's age, social withdrawal, and a family history of schizophrenia supported its consideration, no other symptoms—such as hallucinations, other delusions, abnormalities of the form of thinking, behavioral abnormalities — were observed. Furthermore, the symptoms did not persist for the requisite duration (one month in schizophrenia). In this case, the single, brief episode with delusional content did not meet the criteria [8].

The patient's nonspecific symptom profile prompted the clinical team to include rare conditions in the differential diagnosis, such as Fahr's syndrome. This syndrome can present with a wide spectrum of psychiatric symptoms, ranging from affective disorders and memory disturbances to psychotic symptoms [6,23]. Diagnostic criteria of Fahr's syndrome are shown in Table 1. [23,26,27]. The patient's MRI revealed degenerative changes in the basal ganglia, which could have corresponded to Fahr's syndrome. However, the changes were too mild to substantiate such a diagnosis. Moreover, extensive psychological testing did not provide evidence of organic underpinnings for the patient's cognitive dysfunction. These findings led to the exclusion of Fahr's syndrome. Nevertheless, it remains possible that the patient could develop a neurological condition in the future, warranting regular monitoring of the basal ganglia changes.

Table 1. Fahr's syndrome diagnostic criteria (adapted from Moskowitz et al. and Ellie et al.) (6,26,27)

Fahr's syndrome diagnostic criteria
Bilateral calcification of the basal ganglia visualized on neuroimaging. Other brain regions may also be observed
Progressive neurologic dysfunction, which generally includes a movement disorder and/or neuropsychiatric manifestations
Absence of biochemical abnormalities and somatic features suggestive of a mitochondrial or metabolic disease or other systemic disorder
Absence of an infectious, toxic, or traumatic cause
Family history consistent with autosomal dominant inheritance

Dissociative disorders were also considered at one stage of diagnosis. However, the patient's memory gaps did not resemble typical dissociative amnesia; elements of episodic memory were overly generalized rather than entirely erased and were sometimes filled with fabricated content. The memory deficits did not appear suddenly or resolve rapidly, as often observed in dissociative syndromes. They also did not emerge in response to a specific stressful event. Furthermore, individuals with conversion disorders often deny memory problems, while this patient was fully aware of and critical toward his deficits [7,24,25]. Table 2 sums up the disorders which were ruled out during the differential diagnosis.

The possibility of malingering was thoroughly investigated before a final diagnosis was made. Throughout the diagnostic process, the clinical team found no evidence of intentional fabrication or inconsistencies in the patient's accounts. He underwent psychological testing that included validity scales, none of which suggested simulation. Over six months, the patient was repeatedly evaluated by different professionals, and interviews with several of his close associates further minimized the likelihood of deliberate deception.

Table 2. Psychiatric disorders taken into account in the differential diagnosis.

Psychiatric disorder	Matching features	Why it was ruled out
Schizophrenia	Episode of delusions, age of onset	No other symptoms such as hallucinations, specific delusions, abnormalities of the form of thinking, behavioral abnormalities, negative symptoms
Fahr's syndrome	Wide range of neuropsychiatric manifestations	Too discrete changes in the basal ganglia
Dissociative amnesia	Memory gaps	Symptoms did not emerge in response to a specific stressful event
Dementia	Memory gaps	Memory disturbances were not typical for any type of dementia, no other signs of cognitive impairment, very early age of onset, no confirmation in laboratory tests (phospho-tau protein), neuroimaging, neuropsychological tests
Lyme disease	Wide range of neuropsychiatric manifestations	No evidence for infection, no non-psychiatric manifestations
Malingering	False memories	No evidence of intentional fabrication or inconsistencies in the patient's accounts

CONCLUSIONS

This paper presents the case of a patient with a nonspecific constellation of symptoms, which initially raised the possibility of extremely rare conditions such as Fahr's syndrome (estimated prevalence <1/1,000,000) [23]. Through comprehensive history-taking, corroboration with relatives, extensive laboratory and imaging studies, and significant contributions from clinical psychology, a final diagnosis was established: mixed personality disorder with predominant dependent and avoidant traits, and panic disorder.

While this clinical scenario was highly atypical, it may not necessarily be rare. Patients with personality disorders represent a large population that may present to healthcare facilities with diverse symptoms. This publication aims to encourage clinicians to utilize all available diagnostic tools, including not only psychiatric assessments and laboratory tests but also modern psychological testing and psychological interventions, which are the key components of a correct diagnosis in such atypical cases of mental and cognitive (memory) disorders.

REFERENCES

1. Griem J, Stone J, Carson A, Kopelman MD. Psychologic/functional forms of memory disorder. Handb Clin Neurol. 2016;139:407–17.
2. Sellal F, Weiss C. [Memory diseases]. Rev Prat. 2023;73(10):1081–1084.
3. Skodol AE, Oldham JM, American Psychiatric Association Publishing, The American Psychiatric Association Publishing textbook of personality disorders. Third edition. Washington, DC: American Psychiatric Association Publishing; 2021. 1
4. Skodol AE, Morey LC, Bender DS, Oldham JM. The Alternative DSM-5 Model for Personality Disorders: A Clinical Application. Am J Psychiatry. 2015;172(7):606–613.
5. Hopwood CJ, Kotov R, Krueger RF, Watson D, Widiger TA, Althoff RR, rt al. The time has come for dimensional personality disorder diagnosis. Personal Ment Health. 2018;12(1):82–86.
6. Amisha F, Munakomi S. Fahr Syndrome. W: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 [cytowane 16 November 2024].: http://www.ncbi.nlm.nih.gov/books/NBK560857/
7. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders [Internet]. DSM-5-TR. American Psychiatric Association Publishing; 2022 [17 November 2024]: https://psychiatryonline.org/doi/book/10.1176/appi.books.9780890425787
8. World Health Organization (WHO). International Classification of Diseases, Eleventh Revision (ICD-11). 2022.
9. Rizvi A, Torrico TJ. Obsessive-Compulsive Personality Disorder. W: StatPearls [Internet]. Treasure Island (FL): StatPearls

Publishing; 2024 [16 November 2024]: http://www.ncbi.nlm.nih.gov/books/NBK597372/

10. Hansen BJ, Thomas J, Torrico TJ. Dependent Personality Disorder. W: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 [7 November 2024]: http://www.ncbi.nlm.nih.gov/books/NBK606086/
11. Fariba KA, Torrico TJ, Sapra A. Avoidant Personality Disorder. W: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 [16 November 2024]: http://www.ncbi.nlm.nih.gov/books/NBK559325/
12. Skodol AE, Oldham JM, American Psychiatric Association Publishing, redaktorzy. The American Psychiatric Association Publishing textbook of personality disorders. Third edition. Washington, DC: American Psychiatric Association Publishing; 2021. 1 s.
13. Disney KL. Dependent personality disorder: a critical review. Clin Psychol Rev. December 2013;33(8):1184–1196.
14. Bienvenu JO, Brandes M. The interface of personality traits and anxiety disorders. Primary Psychiatry. 2005;12(3):35–39.
15. Airaksinen E, Larsson M, Forsell Y. Neuropsychological functions in anxiety disorders in population-based samples: evidence of episodic memory dysfunction. J Psychiatr Res. 2005;39(2):207–14.
16. Hallford DJ, Rusanov D, Yeow JJE, Barry TJ. Reduced specificity and increased overgenerality of autobiographical memory persist as cognitive vulnerabilities in remitted major depression: A meta-analysis. Clin Psychol Psychother. wrzesień 2022;29(5):1515–1529.
17. Spinhoven P, Bamelis L, Molendijk M, Haringsma R, Arntz A. Reduced specificity of autobiographical memory in Cluster C personality disorders and the role of depression, worry, and experiential avoidance. J Abnorm Psychol. 2009;118(3):520–530.
18. Hudson NW, Chopik WJ. Seeing you reminds me of things that never happened: Attachment anxiety predicts false memories when people can see the communicator. J Pers Soc Psychol. luty 2023;124(2):396–412.
19. Hudson NW, Fraley RC. Does attachment anxiety promote the encoding of false memories? An investigation of the processes linking adult attachment to memory errors. J Pers Soc Psychol. 2018;115(4):688–715.
20. Mayor C, Schneider M. Confabulations in a teenager with a right frontal hemispherotomy: Possible underlying mechanisms. Clin Neuropsychol. November 2023;37(8):1787–808.
21. Haque S, Juliana E, Khan R, Hasking P. Autobiographical memory and hierarchical search strategies in depressed and non-depressed participants. BMC Psychiatry. 18 November 2014;14:310.
22. Klumpp H, Amir N, Garfinkel SN. False memory and obsessive-compulsive symptoms. Depress Anxiety. 2009;26(5):396–402.
23. Saleem S, Aslam HM, Anwar M, Anwar S, Saleem M, Saleem A, i in. Fahr's syndrome: literature review of current evidence. Orphanet J Rare Dis. 8. 2013;8:156.
24. Boulet C, Lopez-Castroman J, Mouchabac S, Olié E, Courtet P, Thouvenot E, i in. Stress response in dissociation and conversion disorders: A systematic review. Neurosci Biobehav Rev. 2022;132:957–967.
25. Thomas-Antérion C. Dissociative amnesia: Disproportionate retrograde amnesia, stressful experiences and neurological circumstances. Rev Neurol (Paris). 2017;173(7–8):516–520.
26. Moskowitz MA, Winickoff RN, Heinz ER. Familial calcification of the basal ganglions: a metabolic and genetic study. N Engl J Med. 8 lipiec 1971;285(2):72–77.
27. Ellie E, Julien J, Ferrer X. Familial idiopathic striopallidodentate calcifications. Neurology. marzec 1989;39(3):381–385.