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Neurological and Psychiatric Symptoms Caused by Congenital Venous Anomaly: Clinical Case and Literature Review

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Summary. One of the most common vascular brain malformations is developmental venous anomaly (DVA). This anomaly is usually considered to be asymptomatic but there are some reports in the literature about DVA causing neurological symptoms due to neurovascular compression, obstructive hydrocephalus, venous infarction, or intracerebral hemorrhage. There are no publications on DVA causing psychiatric symptoms. This paper presents a clinical case of DVA in which the patient develops a mood disorder along with neurological symptoms that are typical for a parietal brain lesion. Along with this clinical case, a review of literature is presented which includes classification of vascular brain malformations, prevalence of DVA, clinical manifestations, and characteristics in imaging studies. The article also reviews the functions of the parietal lobe, discusses mood disorders possibly related to parietal lesions, and briefly introduces cyclothymic disorder.

Keywords: affective disorders, cyclothymia, developmental venous anomaly, parietal lobe.

INTRODUCTION

Developmental venous anomaly (DVA) is the most common malformation of the vascular system of the brain [1]. DVAs are usually asymptomatic, detected by accident, and considered to be alternative anatomical variants that pose no health risk [2]. The mechanism of DVA formation is not clear, but the currently accepted theory suggests that due to abnormal development, occlusion, or early insufficiency of capillaries or small transcerebral veins, DVA is formed as a compensatory venous drainage system of the cerebral parenchyma [2, 3]. It was once thought that DVA might be associated with a high risk of haemorrhages and neurological dysfunction, but current visual diagnostic capabilities suggest that the incidence of DVA in the population is much higher than previously thought and can be considered a benign brain abnormality [4, 5]. The following clinical case introduces a 64-year-old woman who was diagnosed with a venous angioma in the parietal lobe of the left hemisphere of the brain. This anomaly manifested as both neurological and psychiatric symptoms which began to appear at a similar time. This clinical case is particularly interesting because venous angiomas are rarely symptomatic, and reports of psychiatric symptoms associated with venous angiomas, especially in the parietal part of the brain, are rarely found in the available literature.

CLINICAL CASE

The 64-year-old primary school teacher lost her job in 2016. This event increased the patient's sensitivity and mood swings from mania to passivity. She began visiting psychiatrists since 2018, initially applying for "mania": she had a lot of activities, slept little at night, and had conflicts with her daughter who eventually convinced her to seek medical help. At that time, she was diagnosed with F41.2 (Mixed anxiety and depressive disorder) according to the ICD-10 (International Classification of Diseases-10). At the same time, a headache began to appear in

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the forehead, which bothered the patient at night. A year later, dizziness and difficulty walking appeared. At the same time, there was an episode of "bad mood". The psychiatrist's record stated that the patient changed jobs frequently and complained of anxiety, unexplained fear, lack of motivation, and speech problems. Treatment with sulpiride 200 mg/d, bromazepam 3 mg/d was administered and a consultation with a psychologist was recommended. During psychodiagnostics, the patient was anxious, tense, the pace of mental processes was slow. Narrowed social communication and unwillingness to be among people were observed. The Beck Depression Inventory was conducted, and self-examination data showed pronounced symptoms of depression and moderate symptoms of anxiety. The House-Tree-Person Test indicated pronounced anxiety, tension, immature personality, the need for care, demonstrativeness, sensitivity to the opinion of others, concentration on somatic problems, and projections of the need for emotion control. There were fewer signs of depression, but they were recorded. In May 2019, in addition to the previous treatment, quetiapine 25 mg/d was added, and according to the patient, it was suitable for her as it improved her sleep. In August, the patient experienced some manic symptoms, could not sleep at night, found it difficult to drive, and had racing thoughts. In October of the same year, the patient was diagnosed with F34.9 Persistent mood [affective] disorder, unspecified (ICD-10). The patient had elevated mood, was feeling active, had many plans for the future, and even established a firm. Sodium valproate 500 mg/d was added to the previous treatment. She was referred to a 3rd level psychiatrist for the diagnosis and treatment revision. The 3rd level psychiatrist diagnosed her with F34.0 (Cyclothymia, ICD-10). In November, the dose of quetiapine was adjusted to 12.5 mg/d. In March 2020, the patient contacted her family doctor for speech disorder and stuttering that worsened when the patient felt excited. The patient was referred to a 2nd level neurologist for a consultation. In June of the same year, because of the epidemiological situation, the patient was consulted by telephone due to deteriorating vision in both eyes – her vision worsened both looking far and near. She was referred to a 2nd level ophthalmologist for a consultation. In June, a computed tomography (CT) scan of the head was performed: it was carried out by the *BrightSpeed* scanner in 16 layers. In the left parietal lobe, a focal hyperdense area of ~0.5 centimeters of what initially looked like a cavernoma was observed. Magnetic resonance imaging (MRI) was recommended for further inspection. Subtentorial area did not show any changes in focal density. The structures of the central line were not deviated. The ventricular system was of a medium width. Subarachnoid convexital gaps and the base tanks were widened because of the involutional changes in the brain due to aging. Visible face cavities were airy. No destructive changes in the bones were observed. After the CT scan, a 0.5 cm cavernoma of the left parietal lobe was diagnosed, the patient was also diagnosed with a transient cerebral ischemic attack and a vascular headache. Treatment with Ultracode, pramiracetam and Tanakan was prescribed. In August 2020, the patient complained of deteriorating well-being, impaired speech, and treatment without any perceived effect. She was referred to a 2nd level neurologist for a consultation. During the consultation, it was objectively assessed that the patient was depressed, but her speech was not disturbed at the time of the examination. She did not have any sensory deficit. While standing in Romberg's pose, the patient was unsteady. No meningeal or other pathological symptoms were reported. An intense tremor of the patient's fingers and eyelids was observed. The patient seemed emotionally sensitive with unstable mood. The autonomic nervous system reactions were strongly expressed. The CT scan was re-evaluated, the diagnosis of Q28.9 (Congenital malformation of circulatory system, unspecified, ICD-10) was determined. Treatment with piracetam 800 mg and vinpocetine 5 mg was prescribed. Because of the lesions found in the CT scan, it was recommended to perform an MRI of the brain. In October, the patient complained of experiencing a feeling of aggression and a stuttering speech, especially when agitated or when talking fast. The psychiatrist added tiaprid 100 mg/d to the previous treatment. In December, because of the epidemiological situation, the patient was consulted by phone regarding her treatment. She was complaining about the desire to bite her nails. According to the patient, tiaprid helped alleviate her symptoms. The daily dose of tiaprid was increased to 150 mg/d. In January 2021, the patient was consulted by her psychiatrist by phone about complaints of a greatly impaired well-being, elevated mood and insomnia. In the same month, the patient contacted her family doctor because of headache and dizziness. An intense tremor of fingers and eyelids was observed. It was decided to extend the treatment with piracetam and vinpocetine. The patient's health only worsened. In February of the same year, the patient was consulted by a 3rd level neurologist because of localized pain in the forehead, dizziness, unstable balance, dimmed eyesight, and speech impairment when talking fast. The patient said she was feeling better mentally and had stopped taking the medication prescribed by the psychiatrist at the end of 2020. She was given the diagnosis of I67.8 (Other specified cerebrovascular diseases, ICD-10). In February, she was also examined by a neuro-ophthalmologist; background retinopathy and retinal vascular lesions were detected. No congestion was observed. In a new CT scan with contrast, venous angioma was detected in the left subcortical area. There were no other structural changes. Insufficient circulation in the vertebrobasilar pool was observed. The focal area seen in the left parietal lobe was specified to be a venous angioma. Vasodilators – vinpocetine 20-30 mg/d or pentoxifylline 400-800 mg/d, pramiracetam 600 mg/d, and Hjertemagnyl 150 mg every 2nd evening – were prescribed. Due to the persistent hypersensitivity, feelings of tension and anxiety, a psychiatrist's consultation was recommended. In March 2021, the patient said that her well-being improved and her stuttering decreased.

DISCUSSION AND LITERATURE REVIEW

Classification of vascular cerebral malformations and the prevalence of DVAs

Vascular malformations of the brain are detected in 0.1-4.0% of the general population. 4 subtypes of vascular malformations are distinguished: 1) arteriovenous malformations; 2) cavernous malformations (CM); 3) developmental venous anomalies (DVA); 4) capillary teleangiectasia [6].

DVA is the most common among these sub-types (in 1978, a study of 4,069 corpses was performed; it was found that the incidence of DVAs in the brain was 2.6% of all autopsies) [7].

Clinical manifestation of DVA and its characteristics in imaging studies

Venous angiomas of the brain consist of radially oriented enlarged medullar veins that drain into the transparenchymal venous stem [8]. Venous angiomas are histologically seen as a composition of thickened and hyalinized veins with insertions of normal neural parenchyma [5]. Angiographically, DVAs are seen as deep medullar veins during the early or middle venous phase with one large draining vein. In a CT scan with contrast, DVAs may look like wide draining veins. In MRI, DVA has a flow void in both T1 and T2 weighted images. The administration of contrast improves the visibility of the draining vein, and it is often possible to visualize medullary veins [8]. During conventional angiography or MRI with contrast, DVAs can be seen as a "head of a jellyfish" [9]. DVA is usually non-symptomatic, but several cases of neurovascular compression, obstructive hydrocephalus, venous infarction, and intracerebral bleeding that are caused by DVAs are found in the literature [10]. In cases where DVA is associated with bleeding, the most common cause is actually CM rather than the DVA itself, which is also one of the most common malformations found in combination with DVAs (13-18% of cases). Usually, CM and not DVA is what most often causes hemorrhages and related symptoms [7, 9]. Clinical manifestations directly related to DVA are debatable, although in some symptomatic cases the pathogenetic mechanism might be mechanical, e. g., symptoms may be caused by compression of the DVA component, which causes obstructive hydrocephalus, but symptoms such as headaches do not have a clear association with DVAs. Some authors argue that based on intraoperative findings, DVA can be found in every case of CM, even if the DVA was not visualized by performing a preoperative MRI. It is believed that due to the presence of DVA, blood products leak into the surrounding parenchyma, microhemorrhages occur, causing angiogenic response, and in this way CM might develop [9]. Although symptoms in patients with DVAs are usually associated with other comorbid pathologies, the fact that isolated cases of DVAs can sometimes cause a variety of problems from minor symptoms to severe bleeding should not be underestimated [11]. Cases of DVAs causing psychiatric symptoms are rarely found in the literature.

Functions of the parietal lobe of the brain

The parietal lobe is known for its important role in processing sensory information from different parts of the body, it is related to the perception of information, decision-making, digit recognition, integration, perception of speech and spatial position [12]. The patient described in this article experienced neurological symptoms that are consistent with parietal region damage, in this case, a speech disorder and an impaired coordination of movements. The question arises whether the parietal area can be related to psychiatric (affective) symptoms in this patient. More recently, it has been discovered that the parietal lobe also plays an important role at every stage of emotion processing, including the determination of the emotional significance in a stimulus, the formation of emotional states, and the regulation of emotions [13, 14]. Changes in the structure and function of the somatosensory cortex that is located in the parietal lobe have been identified in a number of mental illnesses, including anxiety disorders, major depression, schizophrenia, bipolar disorder, post-traumatic stress disorder, specific phobias, obesity, and obsessive-compulsive disorder. A characteristic finding in the presence of mood disorders is a change in the volume of gray matter of the somatosensory cortex. For example, in bipolar disorder, a decrease in the volume of gray matter in the somatosensory cortex of the left hemisphere is typically observed [14].

Mood disorders appearing after a parietal lobe damage

As mentioned earlier, there is a lack of data on DVAs causing psychiatric symptoms because of the parietal lobe damage, but it is possible to consider other cases where parietal lobe damage manifests with symptoms similar to those of our patient.

One of the cases describes a 75-year-old patient who experienced anxiety with manic episodes after an acute infarction in the right parietal lobe. The woman was hospitalized for a sudden mental impairment, left-hand hemiparesis, and dysarthria. One day before the admission, she experienced disorientation and weakness in her left hand; after 20 hours, inappropriate language, agitation, and dysarthria appeared; at the same time the characteristic features of mania started developing. The patient's speech was fast-paced, the woman seemed to be in a euphoric mood. There was a decrease in the need for sleep. She had hypertension and diabetes, but the patient had no history of mental health problems and there was no family history of mental illnesses. At the time of admission, an initial brain MRI with diffusion-weighted images was performed, which revealed an acute infarction of the right parietal lobe with a slight lesion of the posterior temporal area [15].

In another case, a 70-year-old patient was hospitalized for rehabilitation after suffering a brain infarction, which resulted in a mild right hemiplegia and a higher brain dysfunction. Previous MRI imaging showed low signal on T1-weighted image and high signal on T2-weighted image in the left temporal and parietal lobes, leading to the diagnosis of brain infarction. Head CT revealed an area of low absorption in the left temporal and parietal lobes but showed no atrophy of the cortex. On the 26th day after hospitalization, the patient suddenly started experiencing manic symptoms: an elevation of mood, a tense speech, hyperactivity, insomnia, and excitement; these symptoms caused problems in his daily life at the hospital. On the 29th day after hospitalization, the patient was referred to a psychiatric hospital on an outpatient basis. He was diagnosed with an organic manic disorder and was hospitalized. The patient was given lithium carbonate (400 mg/d) and risperidone (2 mg/d). Since the mania lasted more than 1 week, he was diagnosed with secondary mania. His manic condition gradually improved, and he was transferred to the previous hospital. He managed to carry out rehabilitation without any problems and without exacerbation of mania. The patient was discharged on the 139th day after re-admission [16].

Cyclothymia

Cyclothymia is attributed to the mood spectrum disorders and it usually occurs at an early age. Cyclothymia is characterized by episodes consisting of symptoms of hypomania and depression that do not meet all the criteria for bipolar or major depressive disorder [17]. This disorder is characterized by persistent, spontaneous, and reactive mood swings that are associated with anxiety and impulsive behavior [18]. Hypomania is characterized by increased self-confidence, greatness, decreased need for sleep, increased talking, rapid change of thoughts, distraction, excessive activity directed to a certain goal, psychomotor agitation, and irresponsible behavior to satisfy one's needs. Hypomania is similar to mania, but although the symptoms are the same, the duration of their manifestation varies, for example, mania can last from several weeks to months, while hypomania lasts only for several days or less. Also, mania can sometimes manifest with psychotic symptoms that are accompanied by delusions or hallucinations. Hypomania does not cause psychosis or greater problems in social or working life [19].

CONCLUSIONS

In the clinical case described, the patient experienced psychiatric affective symptoms quite late, at the age of 58, which is not typical for mood disorders. Bipolar spectrum disorders, including cyclothymia, typically occur at a young age [20]. Although it was initially thought that the symptoms were of a psychiatric origin, it was decided to conduct a CT scan of the brain when neurological symptoms started to appear, thus finding that there was a developmental venous anomaly in the patient's brain. Interestingly, the DVA usually does not cause any symptoms, making this case a rare occurrence. This case emphasizes the fact that psychiatric symptomatology can sometimes be of organic origin. Special attention should be given if the patient has atypical characteristics of a particular psychiatric illness – in this case, the atypical age of the patient with cyclothymia was unusual.

References

1. Idiculla PS, Gurala D, Philipose J, Rajdev K, Patibandla P. Cerebral cavernous malformations, developmental venous anomaly, and its coexistence: a review. Eur Neurol 2020; 83(4): 360–8. https://doi.org/10.1159/000508748
2. Pereira VM, Geibprasert S, Krings T, et al. Pathomechanisms of symptomatic developmental venous anomalies. Stroke 2008; 39(12): 3201–15. https://doi.org/10.1161/STROKEAHA.108.521799
3. Lee C, Pennington MA, Kenney CM 3rd. MR evaluation of developmental venous anomalies: medullary venous anatomy of venous angiomas. AJNR Am J Neuroradiol 1996; 17(1): 61–70.
4. Garner TB, Del Curling O Jr, Kelly DL Jr, Laster DW. The natural history of intracranial venous angiomas. J Neurosurg 1991; 75(5): 715–22. https://doi.org/10.3171/jns.1991.75.5.0715
5. Töpper R, Jürgens E, Reul J, Thron A. Clinical significance of intracranial developmental venous anomalies. J Neurol Neurosurg Psychiatry 1999; 67(2): 234–8. https://doi.org/10.1136/jnnp.67.2.234
6. Singer RJ, Ogilvy CS, Rordorf G. Vascular malformations of the central nervous system [cited 25 Jan 2022]. Available from: https://www.uptodate.com/contents/vascular-malformations-of-the-central-nervous-system
7. Aoki R, Srivatanakul K. Developmental venous anomaly: benign or not benign. Neurol Med Chir (Tokyo) 2016; 56(9): 534–43. https://doi.org/10.2176/nmc.ra.2016-0030
8. Truwit CL. Venous angioma of the brain: history, significance, and imaging findings. AJR Am J Roentgenol 1992; 159(6): 1299–307. https://doi.org/10.2214/ajr.159.6.1442406
9. Akers A, Al-Shahi Salman R, A Awad I, et al. Synopsis of guidelines for the clinical management of cerebral cavernous malformations: consensus recommendations based on systematic literature review by the Angioma Alliance Scientific Advisory Board Clinical Experts Panel. Neurosurgery 2017; 80(5): 665–80. https://doi.org/10.1093/neuros/nyx091
10. Mullaguri N, Battineni A, Krishnaiah B, Migdady I, Newey CR. Developmental venous anomaly presenting with spontaneous intracerebral hemorrhage, acute ischemic stroke, and seizure. Cureus 2019; 11(8): e5412. https://doi.org/10.7759/cureus.5412
11. Kurt G, Aslan A, Kara E, Erol G, Şahin MB, Uçar M. Different aspects on clinical presentation of developmental venous anomalies: are they as benign as known? A single center experience. Clin Neurol Neurosurg 2021; 201: 106443. https://doi.org/10.1016/j.clineuro.2020.106443
12. Bisley JW, Goldberg ME. Attention, intention, and priority in the parietal lobe. Annu Rev Neurosci 2010; 33: 1–21. https://doi.org/10.1146/annurev-neuro-060909-152823
13. Zhang FF, Peng W, Sweeney JA, Jia ZY, Gong QY. Brain structure alterations in depression: psychoradiological evi-

dence. CNS Neurosci Ther 2018; 24(11): 994–1003. https://doi.org/10.1111/cns.12835
14. Kropf E, Syan SK, Minuzzi L, Frey BN. From anatomy to function: the role of the somatosensory cortex in emotional regulation. Braz J Psychiatry 2019; 41(3): 261–9. https://doi.org/10.1590/1516-4446-2018-0183
15. Kang SY, Paik JW, Sohn YH. Restlessness with manic episodes due to right parietal infarction. J Mov Disord 2010; 3(1): 22–4. https://doi.org/10.14802/jmd.10007
16. Arai H, Matsumoto S, Sekiyama R, Fukuoka T. Secondary mania after cerebral infarction in the recovery phase: case report. Prog Rehabil Med 2018; 3: 20180021. https://doi.org/10.2490/prm.20180021
17. Bielecki JE, Gupta V. Cyclothymic disorder. [Updated 2021 Nov 20]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK557877/
18. Perugi G, Hantouche E, Vannucchi G. Diagnosis and treatment of cyclothymia: the "primacy" of temperament. Curr Neuropharmacol 2017; 15(3): 372–9. https://doi.org/10.2174/1570159X14666160616120157
19. Byrne R, Kirschner K. Unusual behaviors. In: Kliegman RM, Lye PS, Bordini BJ, Toth H, Basel D, eds. Nelson pediatric symptom-based diagnosis. Elsevier, 2018; 421–38.e1. https://doi.org/10.1016/B978-0-323-39956-2.00027-3
20. Moini J, Koenitzer J, LoGalbo A. Bipolar and related disorders. In: Global emergency of mental disorders. London: Academic Press, 2021; 119–36. https://doi.org/10.1016/B978-0-323-85837-3.00024-8

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NEUROLOGINIAI IR PSICHIATRINIAI SIMPTOMAI, SUKELTI ĮGIMTOS VENINĖS ANOMALIJOS

Santrauka

Vienos dažniausiai sutinkamų galvos smegenų kraujagyslių malformacijų yra įgimtos venų anomalijos (ĮVA). Manoma, kad įprastai šios anomalijos simptomų nesukelia, tačiau literatūroje pasitaiko atvejų, kai ĮVA pasireiškia neurologine klinika dėl neurovaskulinio suspaudimo, obstrukcinės hidrocefalijos, venų infarkto ar intracerebrinio kraujavimo. Aprašytų ĮVA atvejų, kurie pasireiškia psichiatriniais simptomais, literatūroje nėra daug. Šiame straipsnyje pristatomas klinikinis ĮVA atvejis, kai pacientei nuotaikos sutrikimai pasireiškė kartu su neurologiniais simptomais, būdingais parietalinės galvos smegenų skilties pažeidimui. Kartu su klinikiniu atveju pristatoma literatūros apžvalga: galvos smegenų kraujagyslių malformacijų klasifikacija, ĮVA paplitimas, klinikinis pasireiškimas ir charakteristika vaizdiniuose tyrimuose. Taip pat straipsnyje apžvelgiamos parietalinės skilties funkcijos, aptariami nuotaikų sutrikimai, galimai susiję su parietalinės skilties pažeidimu, ir trumpai pristatomas ciklotiminis sutrikimas.

Raktažodžiai: afektiniai sutrikimai, ciklotimija, įgimta venų anomalija, parietalinė skiltis.

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