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Advances in the pathogenesis of vestibular migraineassociated anxiety and depression

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Abstract: The common symptoms of vestibular migraine (VM) are spontaneous or positional vertigo attacks accompanied by migraine, and anxiety and depression are prone to be a common clinical concomitant. VM-associated anxiety and depression disorders are interconnected with the clinical symptoms of VM, which mainly involves functional changes such as paroxysmal vertigo and vestibular abnormalities, and is affected by gender and attack frequency. Impact. Vertigo-anxiety loops of action, periaqueductal grey matter, and adrenocorticotropic hormone-releasing factor serve as anatomical and physiological bases of VM-related anxiety and depression, and abnormal release of neurotransmitters such as norepinephrine (NA), 5-hydroxytryptophan (5-HT), and dopamine (DA) may ultimately lead to the development of VM-related cognitive deficits. Acupuncture therapy, cognitive-behavioural therapy and drugs may have a therapeutic effect on VM-related anxiety and depression.

1. Introduction

Vestibular migraine (VM), a vestibular disorder in which vertigo and migraine coexist, is one of the common clinical causes of recurrent dizziness and one of the most common causes of episodic vestibular symptoms^[1]. Vestibular migraine is a common cause of spontaneous vertigo in adults and children, with a lifetime prevalence of VM in adults of 1%^[2]. Modern research classifies VM as a subtype of migraine with two main clinical symptoms: migraine and dizziness, which are characterised by recurrent episodes of dizziness followed by migraine, with episodes lasting 24h-72h being the most common. Studies have shown that these two symptoms are one of the most common complaints of ENT and neurologists^[3]. The common concomitant symptoms of VM vary, and usually include dizziness, nausea, vomiting, unsteady gait, and visual aura, including blurred vision, hemianopsia, etc.; in a few episodes, there is a loss of hearing. Repeated episodes of the above symptoms have a serious impact on the patient's physical and mental health, and are easily associated with anxiety and depression and sleep disorders. In addition, studies have shown that the neuropsychological deficits of patients with migraine aura are more severe than those of other patients^[4]. In recent years, VM-associated anxiety and depression has received increasing attention from many medical researchers, and this article reviews the clinical characteristics, influencing factors and potential mechanisms of VM-associated anxiety and depression in patients with VM.

2. Correlation of VM clinical symptoms with anxiety and depression

2.1 Paroxysmal vertigo

Typical vestibular symptoms of VM are spontaneous vertigo, positional vertigo, visual and head movement induced vertigo, and head movement induced dizziness with nausea^[2]. This kind of paroxysmal vertigo leads to the restriction of the patient's activities, which greatly interferes with the patient's daily life and work, and is very likely to cause the patient to experience negative emotions such as anxiety and depression. Liu Bing^[5] et al. analysed the psychosomatic state of children with benign paroxysmal vertigo and VM with recurrent vertigo attacks, and found that children with recurrent vertigo attacks were more prone to negative emotions such as anxiety and depression than normal children. Liu Bo^[6] et al. found that the frequency of vertigo episodes affects the psychological feelings of adults with vertigo. As the number of episodes of vertigo gradually increases, the risk of anxiety and depression increases, suggesting that more frequent episodes of vertigo, especially frequent episodes and persistent episodes, can cause a great deal of mental stress, leading to the emergence of anxiety and depression in patients. Many patients may even experience the psychological problem of "fear of vertigo". At the same time, the emotional distress caused by vertigo may prolong the course of the disease. Therefore, there is a correlation between recurrent episodes of paroxysmal vertigo caused by VM and anxiety and depression.

2.2 Abnormal vestibular function

As vertigo is a clinical symptom caused by different etiologies, and vestibular system dysfunction is one of the common causes, anxiety is often accompanied by decreased vestibular/balance function^[7]. Some patients with VM show abnormal vestibular function on examination^[8]. Vestibular dysfunction may be secondary to recurrent vestibular symptoms and the severity of vestibular impairment is associated with treatment prognosis^[9]. The most common symptoms are vertigo and nystagmus. Patients are often in a complex visual state due to vestibular dysfunction. Loss of spatial orientation and visual disturbances are often emotionally draining. Studies have shown that patients with vestibular symptoms have higher Hamilton Anxiety Scale (HAMA) scores and are more fearful of the disease than VM patients without vertigo, and that patients with vestibular syndromes have a significantly higher probability of comorbid anxiety and depression than normal subjects, and that chronic vestibular syndromes have a higher incidence than acute vestibular syndromes. Kim et al. Analysed^[10] the results of a multifactorial analysis by means of a logistic regression model. Logistic regression model multifactorial analysis of 47 patients with VM, the results of this analysis showed that peripheral vestibular dysfunction and severe intensity of vestibular symptoms were responsible for anxiety and depression in patients with VM^[11] . Therefore, there is a correlation between abnormal vestibular function and the development of anxiety and depression.

3. Characteristics of VM-related anxiety and depressio

Best C found^[12] that vertigo and psychiatric co-morbidity were more prevalent in some organic vertigo/dizziness subgroups.Lahmann C^[13] found that 48.8% of patients with vertigo, especially those with vestibular paroxysms or vestibular migraines, had current psychiatric co-morbidities in 547 patients with organic or non-organic vertigo. Patients with psychiatric co-morbidities reported more vertigo-related disorders, more depression, anxiety and somatisation symptoms, and lower psychological quality of life compared to patients without psychiatric co-morbidities.Patients with VM had the highest prevalence of psychiatric disorders compared to other patients with organic or

non-organic vertigo. Furthermore, the results of this study point out that patients with VM are a particularly vulnerable subgroup and that anxiety and fear disorders are their most common psychiatric disorders, followed by somatoform and affective disorders^[14]. Therefore, patients with VM are at greater risk of developing psychiatric disorders and are more likely to develop psychiatric disorders such as anxiety and fear disorders, especially anxiety disorders, compared to patients with other causes of vertigo.

4. Possible Factors Influencing VM-Related Anxiety and Depression

4.1 Frequency of vertigo attacks

Episode frequency refers to the number of episodes of vertigo per unit of time. Liu Bo[6] et al. assessed the mental and psychological status of 253 patients with vertigo and performed logistic regression on the possible factors affecting vertigo. The study grouped the patients according to the duration of frequent attacks of vertigo and found that the values of anxiety assessment scores as well as the rate of abnormality gradually increased with the duration of frequent attacks of vertigo, suggesting that the anxiety state of the patients may increase with the duration of vertigo. Pollak L et al. [15] found that patients with acute vertigo tend to experience extreme anxiety and a more severe condition. As a result, the frequency of vertigo attacks in VM patients, as well as the fear of vertigo reoccurrence, severely affects the patient's psychological state and is highly susceptible to the development of psychosomatic problems. At the same time, the existence of anxiety and depression greatly affects the patient's mental health and aggravates the psychological burden, which may lead to the aggravation of the patient's symptoms and the frequency of attacks, and the two may be mutually causative, thus creating a vicious circle. However, due to the lack of other large-sample clinical studies on the relationship between vestibular migraine and anxiety and depression, the relationship between the frequency of vertigo attacks and anxiety and depression in VM patients is still worth exploring further in the next study.

4.2 Gender Factors

As women have a higher prevalence of dizziness or vertigo^[16], headache or migraine sufferers are also more likely to be female^[17], with anxiety and depression following. Studies have shown^[6] that women who suffer from vertigo are more likely to experience anxiety and depression if they suffer from a higher frequency of attacks. Monzani D^[18] has shown that women are more likely to experience anxiety and depression, which may be related to women's social roles and mental capacity. Studies have confirmed that anxiety and depression are related to sex hormone levels, such as oestrogen and progesterone, which play a role in the regulation of neurotransmitters such as 5-H T, NA, and CGRP, and these neurotransmitters are involved in the neural regulation of mood-related nuclei, which forms the neurophysiological basis of the development of anxiety and depression in female patients. From a psychosocial point of view, women are generally more delicate and sensitive in their personality traits, and are more susceptible to stress, illness and pain, which makes them more prone to mood disorders.

5. Possible mechanisms of VM-related anxiety and depression

As a variant of migraine with vestibular manifestations, the pathogenesis of VM-associated anxiety and depression is not fully understood, and most of its studies have been based on migraine pathogenesis. However, there is extensive overlap between the vestibular sensory pathway, the migraine pathway and the emotion perception regulation pathway. Therefore hypotheses of possible

mechanisms of VM-related anxiety and depression are generally associated with migraine.

5.1 Anatomical and physiological basis

5.1.1 Vertigo-Anxiety Loop of Action

The co-morbidity mechanism of VM interacting with anxiety and depression may be related to the presence of extensive cross-over effects between the vestibular pathway, the migraine pathway, and the emotion-related regulatory pathway^[19]. From a neurological point of view, vestibular migraine combined with anxiety and depression can be regarded as a co-product of sensorimotor, endosensory, and cognitive adaptations, and the extensive overlap between vestibular sensory pathways, migraine pathways, and emotion-related regulatory pathways may be the neuroanatomical basis of their co-morbidities^[20]. Balaban C D^[21] and other researchers have pointed out that the co-morbidities of migraine and depression are a product of sensorimotor, endosensory, and cognitive adaptations. The extensive fibre connections between the vestibular-parabrachial nucleus network, the dorsal nucleus of the middle suture-vestibular network, the blue-spot-vestibular network, the dorsal nucleus of the middle suture-blue-spot ring, and the cortical networks (including insulae, orbitofrontal cortex, prefrontal cortex, and anterior cingulate cortex) form the basic vertigoanxiety loop, which is in turn connected to the brain via 5-hydroxytryptamine (5-HT), norepinephrine (NA), dopamine (DA), and calcitonin gene-related peptide (CGRP). Calcitonin gene-related peptide (CGRP) and other neuropeptides interact with the trigeminal vascular system and thus with the migraine pathway.

5.1.2 Grey matter around conduits

It has also been suggested that periaqueductal grey matter (PAG) is associated with dizziness, pain, and anxiety^[22]. PAG is an or simultaneously involved in dizziness, pain, and anxiety, and fibres from the supra vestibular nucleus project to the ventral and ventral aspects of the PAG, and these areas of the PAG receive projections from both the frontal-orbital cortex as well as the insular cortex, which is also interconnected with the central amygdala. The ventral and ventral PAGs appear to be joints that connect vestibular, migraine and anxiety-related pathways, forming a link between balance disorders, migraine, and emotional and behavioural responses.

5.1.3 Adrenocorticotropin-releasing factor (ARF)

WANG et al.^[23] found that corticotropin releasing factor (CRF), a neuropeptide mainly distributed in the central nervous system and associated with stress and anxiety, regulates muscle tone, maintains postural balance, and prevents falls by acting on the lateral vestibular nucleus in the vestibular nucleus complex of the brainstem. The above evidence shows that the vestibular nuclei play a key role in maintaining postural balance, stress and anxiety, and it can be inferred that the internal anatomy of the brain plays an important role in the vestibular system and emotions, and it also strengthens the theoretical foundation for the study of the correlation between the vestibular system and negative emotions of anxiety and depression.

5.2 Neurotransmitters

Since the vestibular nerve is connected to many emotion-related nuclei such as the parabrachial nuleus, locuscoeruleus, dorsal raphe nucleus, and central nucleus of the amygdala/inferior limbic cortex that control emotions, and the vestibular nucleus is connected to the hippocampus, frontal lobe, and dentate gyrus that control emotions. Structures controlling emotions are linked^[24].

The results of basic research indicate^[25] that mood disorders such as anxiety and depression mainly act through noradrenergic (NA), 5-hydroxytryptaminergic (5-HT) and dopaminergic neural pathways, and that the mechanism of VM vertigo is related to the asymmetric release of neurotransmitters, such as NA, 5-HT, and dopaminergic neurotransmitters, caused by the abnormality of trigeminal vascular pathways triggered by the dysfunction of the central nervous system. Therefore, abnormal vestibular stimulation not only promotes the release of neurotransmitters such as 5-HT, dopamine, and norepinephrine, which play an important role in the development of anxiety/depression, through the connection between the vestibular nerve and the parabrachial nucleus, but the vestibular nerve and the nucleus of the vestibular nerve also promotes the release of neurotransmitters, such as 5-HT, through the connection with the dorsal nucleus of the median suture, which can stimulate anxiety or depression^[26].

6. Prevention and treatment of VM-related anxiety and depression

6.1 Drug therapy

Due to the relatively late emergence of the concept and diagnostic criteria of VM, its pathogenesis has not yet been clarified in studies, there is a lack of high-level clinical controlled studies, and there is no specific treatment at present, and clinically, the main reference is to the comprehensive treatment program of migraine, including the treatment of the attack period and the treatment of the intermittent period. Tretinoin analgesics and vestibular inhibitors are mostly used to relieve symptoms such as vertigo and vomiting during the exacerbation period. For prophylactic medication in the intermittent period, most of the Tretinoin non and steroidal anti-inflammatory drugs are used, such as flunarizine, propranolol and topiramate [27]. Gui Yongkun et al. showed^[25] that flunarizine hydrochloride is a calcium channel blocker, which can easily cross the bloodcerebrospinal fluid barrier and play a role in the peripheral and central vestibular system. Flunarizine hydrochloride capsule in the peripheral vestibular system mainly through the increase of cochlear blood flow, improve the blood circulation of the vestibule, inhibit vertigo attacks; in the central vestibular system, the drug can be specific and selective effect on the intracranial vascular smooth muscle, inhibit the calcium ion inward flow, relieve intracranial smooth muscle spasm, dilate the cerebral blood vessels, improve the ischemia and hypoxia of cerebral tissues, protect the vascular endothelial cells and their brain cells, and reduce cytotoxicity. At the same time, the drug can also inhibit the subsequent occurrence of intracranial and extracranial vasodilatation-induced migraine, inhibit the release of neurotransmitters such as norepinephrine, block the abnormal electrical activity produced by the cerebral cortex after stimulation, and then avoid the activation of the brainstem vestibular nerve nucleus, thus reducing the onset of vertigo symptoms. Although flunarizine hydrochloride capsules can effectively reduce the occurrence of vertigo symptoms, the effect is rapid, but can not cure VM, during the drug will produce weakness, drowsiness and nausea and other adverse reactions, long-term use of the drug may produce adverse reactions of the central nervous system, common extrapyramidal symptoms. However, hydrobromide camptothecin tablets can be used for the treatment of migraine, and the treatment of VM is generally referred to the treatment of migraine, so the researchers through the comparison of the clinical efficacy of flunarizine and hydrobromide camptothecin found that hydrobromide camptothecin can be used to dilate blood vessels by inhibiting receptor-operated calcium channels and the voltage-dependent calcium channel-mediated calcium inward flow to effectively alleviate the vasospasm; and also to regulate neurotransmitters release, such as NA, 5-HT and so on. HT, etc. Therefore, camptothecin hydrobromide can improve the symptoms of vestibular migraine and prevent recurrence, especially reduce anxiety and depression with better effect and high safety. Therefore, camptothecin hydrobromide has a good prospect for research and development for the treatment of VM-related anxiety disorders.

6.2 Non-pharmacological treatment

Cognitive behavioural therapy is the use of cognitive techniques and behavioural change techniques to change the patient's adverse cognition of the disease, to achieve the purpose of reducing adverse emotions. Sun Fengli^[28] et al. divided 65 patients with VM with anxiety and depression into two groups, the control group was given conventional treatment and general psychological care, and the experimental group implemented a 3-week cognitive behavioural therapy on this basis, and the comparison concluded that cognitive behavioural intervention therapy can effectively alleviate anxiety and depression in patients with vestibular migraine. In addition, the clinical Chinese acupuncture treatment of VM with anxiety state has also achieved significant efficacy. Acupuncture and moxibustion are rich in therapeutic means, including ordinary acupuncture, head-needle therapy, electroacupuncture, auricular acupuncture points, moxibustion, and other methods, each with its own characteristics, and are widely used clinically. According to a study conducted by Guo Sweet^[29] and others, acupuncture can effectively reduce many of the adverse psychological emotions of patients, reduce the incidence of many adverse symptoms, and shorten the duration of symptoms in patients. Studies at^[30] have shown that acupuncture can improve VM symptoms at multiple targets and has unique advantages in regulating neurotransmitter levels and improving blood supply to the brain. However, there is a lack of sufficient animal experiments and basic research on the mechanism of acupuncture for VM-related anxiety and depression, and future research on the mechanism is a priority.

7. Summary and outlook

Vestibular migraine is an extremely common disease in the clinical nervous system, and there is variability in the onset of symptoms in different patients, with a wide variety of clinical symptoms and a lack of specific laboratory evidence to support the diagnosis of VM, so the diagnosis rate is not high. The prevalence of this disease in the clinic is not high enough, and clinicians do not know it well enough, which leads to a high incidence of underdiagnosis, misdiagnosis, and mistreatment, and inaccurate diagnosis and inappropriate treatment can lead to recurrent symptoms. Patients with VM often suffer from a combination of mood disorders, with anxiety and depression being the most common, and the incidence of anxiety is higher than the incidence of depression, which makes the clinical treatment somewhat difficult. Researchers have not explored the pathogenesis of anxiety and depression in depth, and there is still no definitive pathway to explain the occurrence of anxiety and depression associated with VM. Therefore, future research should focus on the clinical features, early screening and pathogenesis of VM-related anxiety and depression, in order to improve the diagnosis of VM and early intervention of VM vertigo symptoms, which can help to reduce the incidence of anxiety and depression. Clinicians can reduce the frequency of VM episodes and improve the prognosis of VM by improving the mood disorders of the patients, and the daily life of the patients is seriously affected by the dual problems of vertigo, anxiety, and depression. Therefore, we recommend that patients with VM should be screened by the Anxiety and Depression Scale, and that clinical psychologists be involved in the early detection of and intervention in psychological problems when necessary. It is an important guidance for the diagnosis and treatment of VM and the improvement of prevention.

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