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ORIGINAL RESEARCH

Effect of Infection on the Incidence of Hyperglycemic Hemichorea: A Case-Series

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Objective: To explore the effect of infection on hyperglycemic hemichorea.

Methods: The clinical data of 11 patients with hyperglycemic hemichorea admitted to the Affiliated People' s Hospital of Xinxiang Medical College and the Second People's Hospital of Xinxiang were retrospectively analyzed, including gender, age, clinical symptoms, imaging features, blood glucose, glycated hemoglobin, infection indicators, and treatment conditions.

Results: Eleven patients had acute or sub-acute onset, including 9 females and 2 males, with an average age of 74.55 years. Nine patients presented with unilateral limb involuntary movement and 2 patients presented with bilateral limb involuntary movement. Imaging findings of 9 patients showed abnormalities in the basal ganglia region. Random blood glucose levels were all elevated at admission, with an average blood glucose value of 26.20 mmol/l. Urine ketone bodies were positive in 2 patients, inflammatory indexes were elevated in 7 patients, symptoms of hypoglycemic treatment and anti-infection treatment in 10 patients disappeared, and symptoms of 1 patient disappeared after improved microcirculation treatment.

Conclusion: The disease tends to occur in middle-aged and elderly women, and the blood glucose may fluctuate significantly during the onset. Infection may lead to the occurrence and development of the disease. Active control of blood sugar, inflammation and improvement of brain metabolism can effectively control symptoms and prevent recurrence.

Keywords: hyperglycemia, infection, hemichorea, clinical analysis

Hyperglycemic hemichorea is a kind of continuous, non-repetitive, aimless and regular movement of the limbs, which may be accompanied by involuntary facial movements, usually acute or subacute onset, more unilateral involvement, bilateral rare. There is no clear conclusion on the pathogenesis of hyperglycemic hemichorea. Foreign case reports[1] suggest that infection causes the disease. Some studies have pointed out that neutrophils, C-reactive protein and other indicators reflecting non-specific inflammation can also predict the prognosis[2] of the disease, considering that infected patients are prone to cause the disease when they have blood sugar fluctuations. Based on this, this study analyzed the influence of infection on hyperglycemic hemichorea through case collection.

Materials and Methods

General Data

A total of 28 chorea patients admitted to the Affiliated People's Hospital of Xinxiang Medical College and the Second People' s Hospital of Xinxiang City from September 2008 to May 2023 were collected, and 11 patients were diagnosed as hyperglycemic hemichorea. Through the analysis of clinical symptoms, imaging results and test results, the chorea caused by Huntington's disease, acute cerebrovascular disease, drugs, poisoning, intracranial infection, hyperthyroidism, hepatolenticular degeneration and other diseases were excluded. Among the 11 patients, 9 were females. Nine patients had a clear history of diabetes and met the WHO diagnostic criteria for diabetes. Two patients were found to have elevated blood glucose after admission, and 7 patients had elevated inflammatory indicators.

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Clinical Manifestations

All of the 11 patients had acute or subacute onset, the shortest course of disease was 3 days, the longest was more than 3 months, and the average course of disease was 29. 67 days. All of them were conscious at the time of onset, and no obvious positive signs were found in nervous system physical examination. Two patients showed bilateral dance-like movements and 9 patients showed unilateral dance-like movements.

Laboratory Examination

The blood routine examination, urine routine examination, liver function, renal function, and blood sugar. Two patients had positive urinary ketone bodies; Infection indexes were elevated in 7 patients, including 1 patient with ankle III degree scald before onset, and white blood cells increased 25.3×10 ^ 9/l and 1049 urinary white blood cells on admission. One patient was admitted in November 2014 due to chorea, leukocyte 19.3×10 ^ 9/l, urine leukocyte 56ul, chorea reappeared 5 months later after upper respiratory tract infection, leukocyte 26.9×10 ^ 9/l, blood glucose 27mmol/L, ketone body production, 3 cases of pneumonia, 1 case of elevated blood glucose combined with urinary tract infection. One case of acute renal failure combined with pulmonary infection, and the inflammatory indicators were elevated, as shown in Table 1.

Imaging Examination

All the 11 patients underwent imaging examination during hospitalization, 8 patients underwent skull magnetic resonance examination, and the symptoms of T1 in the basal ganglia were patchy and high signal. Among them, 1 patient' s symptoms worsened 5 months later, and there was no abnormality in the basal ganglia in the skull magnetic resonance; Head CT examination was performed in 5 patients, 2 patients showed high-density changes in the basal ganglia area, and 3 patient s showed no obvious abnormalities on brain CT.

Treatment

Ten patients received hypoglycemic treatment after admission, and 1 patient received hypoglycemic treatment alone; 3 cases were treated with hypoglycemic combined with sedative drugs; 1 case of diabetes was not diagnosed before hospital, and the blood glucose was elevated immediately. Insulin combined with estazolam tablet 1mg or ally before going to bed was given to the patient. Due to the combination of left foot scald III and infection, the patient was given levofloxacin anti -infection treatment. Two cases of diabetes complicated with urinary tract infection, of which 1 case received anti-infection treatment with insulin combined with haloperidol 2 mg, Bid, ceftriaxone combined with moxifloxacin, the inflammation index was normal after 5 days, and the symptoms were significantly improved; 5 months later, the upper respiratory tract infection was admitted to hospital, the symptoms were worse than the first time, the inflammation index was significantly increased, and the symptoms were improved after insulin pump combined with cefotaxime sodium treatment. One case was treated with insulin and moxifloxacin. Three patients with pneumonia, including 2 patients with diabetes, were given antibiotics combined with hypoglycemic treatment. The clinical and imaging findings of 1 patient were supportive of the disease. The blood glucose was elevated, and the symptoms disappeared after treatment with butylphthalide and ceftriaxone. The chorea of 1 patient lasted for 10 years and was aggravated after long-term administration of benzhexol with acute renal failure complicated with lung infection. The symptoms were improved after the third generation of cephalosporin combined with hypoglycemic drugs was given, benzhexol was discontinued and haloperidol 1mg, Qd was given. See Table 2 for details.

This study was approved by the Ethics Committee of Henan Veterans Hospital. All the data in this study came from the hospital information management system. As this study was a retrospective analysis, the collection and processing of relevant data had been completed when patients received medical services, so the informed consent of patients could not be performed. We have taken strict measures to desensitize and keep private patient information confidential to ensure that personally identifiable patient information is not disclosed. In addition, all procedures and operations of this research are strictly in accordance with the ethical guidelines of the Declaration of Helsinki to ensure the ethics and legality of the research. All data used in the study was properly processed and protected to ensure that patients' privacy rights were not violated.

Table 1 Test Results and Clinical Manifestations of 11 Patients

Patient ID	Sex	Age (Years)	Glc (mmol/l)	HbAlc (%)	WBC (10 ^ 9/l)	ESR (0–20 mm/h)	CRP (0–5 mg/l)	Urine WBC (0–9/ul)	Clinical Symptoms	Duration (d)
1*	F	79	7.96	19	25.3	–	95	1049	Involuntary shaking of both upper limbs	2
2	F	84	13.6	14.5	19.3	–	–	56	Involuntary movement of the right limb	60
3	F	70	27.62	13.4	–	–	–	–	Involuntary movement of the right limb	3
4	F	60	29.55	–	–	–	–	–	Left upper limb involuntary movement	30
5a	F	72	18.5	10.2	18	–	–	–	Involuntary movement of the right limb	60
6	M	78	23.55	9.6	–	–	–	–	Involuntary movement of the right limb	60
7	F	84	24. 14	9.9	27	–	–	109	Bilateral limbs involuntary dance sample	>90
8a	F	72	23. 12	8.57	2.24	86	170. 5	–	Left limb involuntary dance	30
9*a	M	82	17.37	13.9 5	–	60	–	–	Right limb involuntary dance	15
10 b	F	74	7.9	8.7	–	68	83.5	48. 18	Right limb continuous involuntary dance	–
11	F	65	23.76	15.8	–	–	–	–	Right upper limb involuntary dance pattern	7

Notes: The test result is normal; Blank: Data missing; *: No history of diabetes; a: Pneumonia; b: Acute renal failure.
Abbreviations: F, female; M, male.

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Table 2 Imaging, Treatment and Prognosis Data of 11 Patients

Patient ID	CT	MRI Site	T1 WI	T2WI	Treatment Options	Prognosis
1*	None	Basal ganglia region	High Signal	Low signal	Moxifloxacin,insulin, Esa zolam	Disappearance of symptoms
2	None	Basal ganglia region	High Signal	Normal	Ceftriaxone, hypo glycemic agents, insulin, haloperido l (1mg)Bid	Recurrent symptoms
3	Normal	Left striatum lentiform nucleus	High Sign al	Equisignal	Insulin, haloperidol (2 mg) Bid	Symptom improvement
4	None	Right lentiform nucleus	High Signal	Normal	Insulin, Estazolam (1 mg) Qd	Symptom improvement
5a	Normal	–	–	–	Hypoglycemic drugs, broad-spectrum penicillin,	Disappearance of symptoms
6	High density in left basal ganglia	–	–	–	Hypoglycemic agents	Symptom improvement
7	None	Left putamen, right putam en, caudate nucleus	High Signal	Low signal	Insulin, moxifloxacin, broad-spectrum penicillin	Disappearance of symptoms
8a	Normal	–	–	–	Hypoglycemic drugs, broad-spectrum penicillin, phenobarbital	Symptom improvement
9*a	None	Bilateral lenticular and caudate nuclei	High Signal	Low signal	Butylphthalein, broad-spectrum penicillin	Disappearance of symptoms
10b	None	Right caudate nucleus	Slightly higher signal	Low signal	Hypoglycemic agents moxifloxacin, Benzoxol, Haloperidol (1mg) Bid	Symptom improvement

Notes: Blank: Data missing; *: No history of diabetes; a: Pneumonia; b: Acute renal failure.

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Results

Of the 11 patients in this study, 9 were female with an average age of 74.55 years and 9 had history of diabetes before admission, immediate blood glucose examination results of the 11 patients were clear, the blood glucose range was 7.90 to 29.55mmmol/l, and the average blood glucose value was 19.73mmol/l. Ten patients provided HbA1c% value, with an average of 12.36%. Upon admission, all patients had involuntary dance-like symptoms, the patients' blood sugar, inflammation control, and the frequency and amplitude of involuntary movement gradually decreased, 5 patients' symptoms improved significantly before discharge, 4 patients' symptoms disappeared completely, 1 patient was discharged after improvement, and relapsed 5 months later. The symptoms of 1 patient lasted for 10 years, and the long-term oral administration of benzhexol before hospital did not improve, and the symptoms were significantly improved when haloperidol was changed to haloperidol.

Discussion

Hyperglycemic hemichorea can be classified according to its clinical and imaging characteristics:[3] 1. According to the typical, the most common symptoms are elevated blood glucose without ketone body production, hemichorea symptoms, brain CT showing high-density image of the contralateral basal ganglia and high signal in the basal ganglia area of MRI-T1WI. 2. No diabetes was found in the past, and the immediate blood sugar was slightly high, with symptoms of hemichorea, and the imaging findings were typical. 3. Elevated blood sugar, no typical imaging changes, typical clinical symptoms, symptoms improved after hypoglycemic treatment, which is helpful to distinguish from other diseases. 4. Elevated blood glucose with or without ketone body production, typical on imaging, but no dance-like symptoms. When there are typical neuroimaging manifestations, even if there are no clinical symptoms, it should be considered as a diagnosis. These patients are the most neglected and miss examination and treatment. 5, poor blood sugar control, both limbs show dance-like symptoms, imaging can be shown as unilateral or bilateral lesions. Dubey recently proposed that symptomatic down syndrome in the classification of diabetic steatosis is a fingerprint-like neuroimaging lesion associated with clinically significant movement disorders and hyperglycemia.[4]

In this study, 3 patients had symptoms, but no corresponding lesions were found on head CT. Among them, 1 patient with negative head CT showed high T1 patchy signal in bilateral basal ganglia by head magnetic resonance examination. Literature studies have shown that MRI is more sensitive[5] than CT, and the appearance time of lesions on CT or MRI is inconsistent, so false negative manifestations[6] on CT or MRI cannot be ruled out. The severity and even the lateral differentiation of the symptoms were not consistent with the magnetic resonance imaging. Considering that the imaging of the disease was in a dynamic process, it also indicated that the occurrence of the disease was not only caused by changes in blood glucose. This study collected patients from two tertiary hospitals in Xinxiang City since 2008, and found that infection may be the cause of the disease, or the cause of aggravating symptoms and prolonging cure. It was reported in foreign literature that all 3 case shad a history of infection before the onset of the disease, and serum immunoglobulin IgG, IgA and IgM were significantly elevated by detection A foreign case[7] of type 1 diabetes was reported that when chorea appeared[1] for the first-time blood glucose was significantly elevated, but the symptoms still existed after hypoglycemic treatment, and the symptoms were improved and did not disappear after being given haloperidol and sodium valproate successively. Three years later, the patient was infected with dengue fever, and the blood sugar was not well controlled, and the symptoms of chorea worsened and spread. After improvement, the symptoms worsened again due to paronychia.[8] In this study, 7 patients had a history of infection before admission, and the inflammatory indexes were significant elevated. The infection index and blood glucose were dynamically monitored, and the symptoms improved after stabilization. This disease is more common in elderly women with poor blood sugar control. It may be because estrogen can reduce the function of dopamine transmitter in the nigrostriatal system, up-regulate the function of dopamine receptor, increase to[9] the level of dopamine,[10] activate the direct pathway, inhibit the indirect pathway, promote the excitability of the cerebral cortex, and lead to hemichorea. In addition, changes in hormone levels lead to changes in women's internal environment, vaginal microbiome, and special urinary system structure, increasing the chance of infection. At present, with the increase of viral infection, the incidence of this disease is increasing. For patients with dance symptoms, detailed examination of inflammation indicators improve the diagnosis rate, and help to control symptoms. At present, there are several viewpoints:

(1) Microvascular theory: A beet[11] al conducted a biopsy of the striatum of the diseased side of a patient, and the results showed that the intima, media and outer membranes of the small arteries were severely thickened, blood vessels were occlusive in severe areas, and neovascularization was formed, accompanied by flaky ischemic necrosis, red blood cell leakage, lymphocyte migration and macrophage infiltration. These changes indicated that the structure constituting the blood–brain barrier was destroyed, and after the barrier was destroyed, some metabolic substances and regulatory peptides could freely enter and exit, resulting in functional changes. These changes are similar to those in diabetic retinopathy (DR), where microvascular lesions (such as peri-cell loss, microaneurysms, and capillary shedding) in the early stages of DR Emerge after years of clinically hidden damage. The early biochemical effects of hyperglycemia are marked by increased production of reactive oxygen species (eg, superoxides, peroxynitrates, hydrogen peroxide, etc.). Over time, as capillary permeability increases, the function of the retinal neurovascular unit (NVU) changes, altering blood flow regulation. The above evidence indicates that the destruction of micrangium and neurovascular units may occur in patients with long-term diabetes, and patients with impaired fasting blood glucose and abnormal glucose tolerance may also have symptoms. For these patients, it is necessary to inquire in detail whether there is a history of infection before the onset of the disease. Infection causes metabolic disorders, aggravates brain metabolism abnormalities, and leads to symptoms. Studies have shown[4,12] that the high-signal site of MRI T1 in some patients is not correlated with the disease side. For patients with diabetes for many years, the relationship between imaging findings and the severity and duration of symptoms is not clear. Compared with retinal NVU, our understanding of brain NVU function and dysfunction in patients with normal blood glucose and diabetes is not deep enough. The difference[13] between normal and disease cannot be judged.

(2) Theory of metabolic turbulence: Studies[14,15] have found that in patients with hyperglycemic hemichorea, cerebral blood flow increases and glucose metabolism increases in the early stage, while cerebra l blood flow decreases in the late stage. Some patients with pathological sites are in a state of reduced blood flow at the beginning, which may cause long-term high blood sugar to cause microvascular arteriosclerosis in the basal ganglia and lead to insufficient blood supply. It was also reported that a patient with old thalamic infarction developed chorea due to poor blood sugar control. Elevated blood sugar stimulate increase blood flow to the thalamus, resulting in a significant asymmetry in cerebral blood flow between the basal ganglia and the thalamus, and a decrease in blood flow ratio in the basal ganglia, which weakened the inhibitory signal from the globus pallidus to the thalamus, thus leading to cortical excitation.[16] Infection leads to significant changes in the basal metabolic rate of the body, which in turn affects brain metabolism and causes symptoms. Some scholars have found that intracellular hyperglycemia induces excess superoxide production in mitochondria, which then activates the polyol pathway and amino-hexose pathway, resulting in increased formation of advance glycosylation end products (AGE), and activation of protein kinase C and nuclear factor-γ B (NF-γ B). In the hyperglycemic environment, mitochondrial protein glycosylation induces the production of excess supernegative oxygen ions. Even after the hyperglycemia is corrected, the glycated mitochondria continue to produce excess hypernegative oxygen ions, activating related pathways and producing metabolites that lead to the progression of diabetic complications. In this study, 1 patient had obvious abnormalities in imaging examination, but the clinical symptoms appeared 4 months later, and the symptoms were delayed with different duration, which may be related to the above conclusions. Secondly, the basal ganglia very sensitive to changes in blood flow, and factors such as blood pressure and infection will affect the normal function of the basal ganglia, causing dance-like symptoms.[17] At present, the number of patients with hemichorea in patients with diabetes combined with ketoacidosis has gradually increased,[18] and the control of blood sugar, blood pressure and heart rate in some patients is obviously unstable[19] at the onset of the disease, or in the case of combined infection, the basal metabolic rate (BMR) changes significantly, the body energy imbalance, even if the ketone body increases, it cannot compensate for the loss of energy. The production of metabolic waste leads to the increase of the permeability of the blood–brain barrier, and the anti-glutamate decarboxylase 65 antibodies are easier to pass through the blood–brain barrier, resulting in an immune response. The neurons in the basal ganglia area are attacked by this immune attack, and chorea symptoms appear. Propsonet al found that during aging, astrocytes release an increase in C3 complement, which binds to C3 receptors on endothelial cells, triggering endothelial inflammation, causing damage[20] to the blood–brain barrier, and inflammation accelerates the destruction of the blood–brain barrier, leading to disease progression. In this study, 7 patients had elevated infection indicators (including ESR, CRP, urine routine, and WBC),

and the nursing blood pressure recording curve of some patients indicated that their blood pressure was more unstable than before at the onset of the disease, resulting in a large daily variation of BMR. As the development of the disease is a dynamic process, the current application of SPECT and PET can measure cerebral blood flow reaction brain metabolism, but the cost is high, cannot be dynamically monitored, by measuring the change of daily basal metabolic r ate, perhaps reflect the changes of brain metabolism to a certain extent, because the main energy supply material of brain tissue is glucose, glucose concentration changes have a huge impact on brain metabolism. The data of this study show that when the changes in basic metabolism increase, symptoms appear, may indicate that the changes in basic metabolism al so produce corresponding changes in brain metabolism, due to the small sample size, previous studies did not involve the basic metabolic rate, so it is necessary to expand the sample size, supplement evidence, to provide a basis for treatment.

(3) Neurodegeneration theory: Glucose metabolism disorder can induce glucose collateral metabolism, resulting in the production of sorbitol and fructose, and then damage nerve fiber demyelination and nerve cells. Hyperglycemia in turn causes oxidative[21] stress, which further causes activation of polyol pathway, non-enzymatic glycosylation, protein kinase C activation, and hexosamine pathway activation, leading to tissue damage. There introduce a new magnetic resonance spectroscopy (MRS) though a 79 year old man affected by sudden onset hemichoreic movements whose cause was a non-ketotic hyperglycaemia diagnosed despite the normal blood glucose levels, the normal brain CT and magnetic resonance imaging.[22] The decrease of cerebral blood flow in GABA pathway first leads to ischemic pathological changes of astrocytes and GABAergic neurons in the brain. The injury of GABAergic neurons induces disequilibrium between excitation and inhibition in the brain, leading to excitotoxic nerve injury and ischemic nerve cell death. Chronic hyperglycemia and loss of insulin signaling pathways lead to a cascade of inflammatory pathway activation, oxidative stress, and endothelial dysfunction.

In summary, this study proposes the occurrence of infection-induced disease s, aiming to propose new diagnostic ideas and specify a more perfect treatment plan. Currently, there is a gradual controversy over the use of sedative drugs for treatment. The sample size should be continuously expanded, the evidence of infection and metabolic disorders should be improved, and the cause treatment plan should be formulated to avoid the occurrence of irreversible symptoms.

Disclosure

The authors report no conflicts of interest in this work.

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