

Brain CT Study of Vitamin B₁ Deficiency in Infants

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Abstract: Infantile vitamin B₁ deficiency, also known as beriberi, is caused by insufficient supply of vitamin B₁ in infants. Infants who lack essential vitamins are stunted and have poor resistance. Severe cases suffer from related diseases due to malnutrition. This paper mainly studies the brain CT study of infant vitamin B₁ deficiency, discusses the CT manifestations and diagnostic value of infant vitamin B₁ deficiency encephalopathy, and improves the level of clinical diagnosis. 35 children with vitamin B₁ deficiency encephalopathy were scanned by conventional CT, and their CT manifestations and clinical data were analyzed. The main clinical symptoms were convulsion, mental disorder, ocular depression and vomiting. Brain CT showed: bilateral lenticular nucleus (20 / 35), caudate nucleus head (15 / 35, thalamus (3 / 35), anterior limb internal capsule (4 / 35), lateral ventricle of external capsule (1 / 35) and white matter (2 / 35) symmetrical low-density area, 18 cases of cerebral sulcus enlargement, 16 cases of ventricular enlargement, 1 case of cerebral cortex density was generally low. After treatment with vitamin B₁, the clinical symptoms were significantly improved and disappeared in 2-5 days. The CT low density changes of bilateral basal ganglia in children with vitamin B₁ deficiency encephalopathy have certain characteristics. The study of brain CT can quickly diagnose the clinical symptoms of vitamin B₁ deficiency in infants, and it has important value in the diagnosis of infant diseases.

1. Introduction

In this paper, we will study the CT features and diagnostic value of vitamin B₁ deficiency, to understand the pathogenesis of vitamin B₁ deficiency syndrome, the related role of vitamin B₁ group, the help of CT in the treatment of craniocerebral diseases, and its role [1]. In order to better study the subject, we collected the clinical data of 35 cases of infants with vitamin B₁ deficiency mainly with nervous system symptoms and related literature, and made a retrospective analysis of

the correlation between clinical and brain lesions, and explained from the perspective of etiology and pathology.

Studies have found that vitamin B₁ deficiency can lead to attention loss, confusion, memory instability, nausea, irritability and depression. Alcohol has neurotoxic effects. Long term drinking can lead to vitamin B₁ deficiency, decrease of brain glial cells, increase of homocysteine level and decrease of casein kinase activity, which may lead to cognitive dysfunction caused by alcohol. Yes, the results related to vitamins showed that: (1) the acetylcholinesterase activity in cortex and hippocampus decreased significantly due to vitamin B₁ deficiency; (2) acetylcholinesterase activity in hippocampus was not affected and decreased significantly; (3) acetylcholinesterase activity in cortex and hippocampus decreased significantly after vitamin B₁ deficiency for one week. Severe malnutrition in patients with chronic alcoholism often leads to vitamin B₁ deficiency or rapid deterioration of mental state. When the body's vitamin deficiency often leads to deep cortical diencephalon (periventricular gray matter and central aqueduct gray matter) damage, the performance is anterograde amnesia [2].

The characteristics of vitamin B₁ deficiency in infants are as follows: (1) vitamin B₁ deficiency in the diet of lactating mothers and children with beriberi symptoms; (2) prominent neurological symptoms of infants; (3) decreased binding capacity of blood carbon dioxide; (4) vitamin B₁ treatment can rapidly improve within 1-2 days. In clinical practice, due to the lack of understanding of the disease, the blind use of hormones, glucose, dehydrating agents and respiratory stimulants can make the disease worse and even lead to death. Because hormone can prevent pyruvate oxidation into carbon dioxide and acetyl coenzyme A, increase blood sugar, accumulate lactic acid and pyruvate, and make the disease worse and more complicated.

The clinical manifestations of this disease are nonspecific and lack of objective diagnostic indexes. To prevent misdiagnosis and abuse, the following conditions should be considered. (1) Infants from rural areas have obvious central nervous system symptoms, no pathological reflexes, normal cerebrospinal fluid and liver function, and a history of vitamin B₁ deficiency with the mother. (2) Clinical suspected central nervous system infection, failure or deterioration after the corresponding treatment. (3) The suspected cases improved rapidly after vitamin B₁ treatment. (4) Infants with sudden MSOF [3], especially severe central respiratory failure, should consider the possibility of disease if there is no other major disease investigation, and adequate vitamin B₁ treatment should be given. (5) When clinical identification is difficult, vitamin B₁ treatment can be tried first, and blind application of glucose and hormone should be avoided in order to avoid aggravating the disease, leading to serious consequences.

Medical image processing [4] and analysis has always been a hot research direction in the field of image processing, and the main direction of these research will inevitably go to computer-aided diagnosis system and even computer intelligent diagnosis system, which is also a main trend in this field. For example, CT in infant vitamin B₁ deficiency encephalopathy research also has great help.

This text is to let readers understand the cause and characteristics of the disease comprehensively. We will start with vitamin B₁ in detail, combining pictures and texts, and strive to be detailed. Analysis of the characteristics of patients with disease and related performance, combined with theoretical knowledge and clinical experience, this paper puts forward the relevant treatment methods and some aspects that need attention.

2. Vitamin B₁ Deficiency in Infants

2.1. Overview of Vitamin B

Vitamin B is an essential nutrient for human body Common vitamin B₁, vitamin B₂, vitamin B₃,

vitamin B₆. In addition to being used as a health care product, it can also be made into drugs to help treat some diseases.

Vitamin B₂ can treat glossitis. Vitamin B₂, also known as riboflavin, mainly participates in the redox reaction in the form of coenzyme and is closely related to the metabolism of vitamin B₆ and vitamin B₃. Vitamin B₂ participates in the metabolism of energy and fat to maintain the health of mucous membrane. Therefore, for tongue ulcers and other glossitis symptoms, can be under the guidance of doctors to check whether lack of vitamin B₂, if necessary, use drug treatment.

Vitamin B₃: pellagra, also known as niacin [5], plays an important role in maintaining skin health. Lack of hydrochloric acid easy to appear rough skin: Patients with erythema, burning, itching, exudation, accompanied by herpes, bullae, and then scab, pigmentation, rough skin surface, dandruff. Take hydrochloric acid tablets and other drugs when necessary. Vitamin B₃ can also help control blood lipids, but do not overdose it, or you will have dizziness, rapid heartbeat, nausea and vomiting and other adverse reactions.

Vitamin B₆ can treat seborrheic dermatitis [6]. When the scalp appears dark yellow red spots, greasy scales or scab on the surface, it may be seborrheic dermatitis. It has been found that vitamin B₆ deficiency may cause seborrheic dermatitis and insomnia. However, there are many reasons for the above diseases, which should be symptomatic treatment under the guidance of doctors.

Vitamin B₉: stroke. Vitamin B₉ is also known as folic acid. Folic acid is butterfly glutamate. With the gradual recognition of the importance of folic acid in diet, especially with the deepening research on folic acid and birth defects, cardiovascular diseases and tumors, folic acid is considered to be a very important micronutrient. Folic acid exists in intestinal wall, liver and bone marrow. Tetrahydrofolate is reduced to tetrahydrofolate with biological activity by folate reductase. As one carbon unit of coenzyme, its role is carbon unit. It participates in the synthesis of purine and thymine, the conversion of amino acids, the synthesis of hemoglobin and heavy methyl compounds, such as adrenal cord, choline, creatine, etc., and promotes the hematopoietic function of bone marrow. In some patients, metabolic abnormalities lead to increased homocysteine and cysteine, Especially H-type hypertension patients. These are the risk factors of acute myocardial infarction, stroke and cardiovascular and cerebrovascular diseases [7]. At the same time, folic acid can also participate in the metabolism of amino acids, as a carrier between homocysteine and methionine, thus reducing the value of homocysteine in vivo.

Vitamin B₁₂ can treat anemia. Vitamin B₁₂ helps to maintain the stability of red blood cells in the process of energy production. In some cases, megaloblastic anemia may be caused by vitamin B₁₂ deficiency. In addition, some fatigue, loss of appetite, emaciation symptoms are also related to the lack of vitamin B₁₂, it can also assist the treatment of the above symptoms. But always consult your doctor before taking it. And what we're going to focus on today is vitamin B₁.

2.2. Vitamin B₁

Vitamin B₁ is a water-soluble vitamin [8] containing amino and sulfur elements, so it is also known as thiamine. What we can buy on the market is usually thiamine hydrochloride or nitrate [9]. VB₁ has an important nutritional and health care function in our body, which participates in the important link of carbohydrate metabolism and heat metabolism in our body, so VB₁ demand is calculated according to the proportion of individual body heat consumption. VB₁ deficiency is also known as beriberi. The earliest symptoms of VB₁ deficiency are anorexia, dyspepsia, mental fatigue, and dyskinesia. If it is more serious, it will endanger the heart and eventually die of heart failure. The earliest record of VB₁ deficiency in history is in China. In 2600 BC, the related symptoms were recorded in Neijing. In the Jin Dynasty, Tao Hongjing described beriberi in detail. It was not until the 18th century that the disease was gradually recognized and studied by medicine.

Generally, can be divided into dry beriberi, wet beriberi and infantile beriberi. Brain type beriberi is a part of dry beriberi. It was first reported by Carl Wernicke in 1881, also known as Wernicke encephalopathy. In recent years, due to the improvement of living standards and the lack of nutrition knowledge guidance, the increase of white rice and flour consumption, and alcoholism, vitamin B₁ deficiency is on the rise. Long term fever, chronic consumptive diseases, metabolic diseases and heavy physical labor will also appear. This paper reviews the diagnosis and treatment of beriberi and the clinical application of vitamin B₁ in related diseases. The next focus of this paper is infantile beriberi.

2.3. Food Sources of Vitamin B₁

Vitamin widely exists in natural food, the most abundant source is sunflower seed kernel, soybean, peanut. The second is wheat, millet, rice, corn and other cereals, vegetables and fruits are relatively less. If the daily intake is insufficient, it needs to be supplemented with drugs to meet the needs of the body.

2.4. Development and Application of Vitamin B₁

(1) Compared with the control group, the treatment course of diarrhea, infantile diarrhea and intramuscular injection of vitamin B₁ in children was significantly shorter than that in the control group. Acupoint injection of vitamin B₁ has a significant effect on severe refractory not bacterial diarrhea caused by diabetes and hyperthyroidism.

(2) Lead poisoning: a survey of lead workers in a battery factory showed that the blood lead level and dietary vitamin B₁, B₂ were negatively correlated with phosphorus intake. At the same time, vitamin B₁ can prevent the accumulation of lead in the blood, liver and brain of experimental animals, and accelerate the elimination of lead in tissues.

(3) Close sweat, hot bath can expand skin capillary, can make sweat gland mouth open, oryzanol, vitamin B₁ can adjust autonomic nerve, thus can cure sweat.

2.5. Etiology of Vitamin B₁ Deficiency

Before liberation, fine white rice was mainly used by urban residents in southern China, and beriberi was very popular. However, there were few cases of beriberi among rural residents, and it was later found that the reason was that rural residents mainly used brown rice. After liberation, China stipulated the processing standard of commercial grain, and urban residents changed to eat 92 meters and 81 noodles, which greatly reduced the number of patients with beriberi. Later, due to the upgrading of food processing technology of rural residents, water mills and cattle mills were replaced by machines. The rate of bran production was too high and the VB₁ content of processed grain was too low, which once again led to the prevalence of beriberi.

2.6. Evaluation Criteria for Vitamin B₁ Deficiency in Infants

Infantile beriberi (i.e. vitamin B₁ deficiency encephalopathy) is a nutritional deficiency caused by vitamin B₁ deficiency. Due to the acute onset, it is difficult to diagnose early clinical manifestations and easy to be misdiagnosed. Vitamin B₁ deficiency can lead to vitamin B₁ deficiency (infantile beriberi). Excessive intake of thiamine is usually harmless because it can be excreted quickly. Vitamin B₁ deficiency is mainly due to insufficient supply rather than absorption disorders. It is suggested that the AI value of vitamin B₁ in DRIs is 0.1 mg / D for infants aged 0-6 months and 0.3 mg / D for infants aged 7-12 months. Vitamin B₁ was 0.6 mg / D for children aged

12-36 months. The survey shows that in China, the milk production of lactating women is close to that of developed countries, and the average vitamin B₁ content of breast milk is $17 \pm 7 \mu\text{g} / 100 \text{g}$, which is also close to that of the United States. During lactation, the secretion of vitamin B₁ from milk is about 0.2 mg per day. Some studies have carried out a comprehensive survey and analysis of vitamin B₁ intake in urban and rural infants, vitamin B₁ content in infant urine, differences between urban and rural areas, gender differences in infant urine, vitamin B₁ content and vitamin B₁ content in age-related infant urine.

Table 1. Comparison of vitamin B₁ intake between urban and rural infants

	Six months		Twelve months		Twenty-four months	
	City	Countryside	City	Countryside	City	Countryside
Intake mg/d	0.30±0.17	0.22±0.13	0.65±0.25	0.59±0.18	0.68±0.22	0.62±0.21
RNIs(%)	96.70	70.10	106.68	96.69	113.34	103.32

Shown as Table 1, the intake of vitamin B₁ is lower than the recommended intake mainly occurs in rural areas of infants aged 6-12 months. After six months of age, infants and young children begin to add complementary food, if they do not take enough vitamin B₁, they will have insufficient nutrition supply.

Table 2. Statistics of vitamin B₁/ creatinine in urban and rural infants

	Not Enough	Flat	Suitable	Adequate	Amount To
City	0(0)	2(1.6)	3(2.9)	100(95.5)	105(100)
Countryside	7(2.6)	6(1.9)	24(8.2)	254(87.3)	291(100)

Shown as Table 2, the intake of vitamin B₁ in urban infants is higher than that of rural infants in the same period of the time.

Table 3. Comparison of vitamin B₁ limits in different countries / regions

Nutrient	CAC		USA		EU		ANZ		Taiwan	
	Min	Max/GUL	Mi	Ma	Mi	Ma	Mi	Ma	Mi	Ma
			n	x	n	x	n	x	n	x
VB ₁ (Baby)	15	73	-	-	9.7	73	11	49	15	73
	61	301	41	-	41	302	-	-	61	302
VB ₁ (Older baby)	11(15)	N.S(72)	-	-	9.6	72	10	49	15	73
	41(65)	N.S(302)	41	N.S.	41	300	-	-	61	302
VB ₁ (infant)	11	N.S.							15	73
	41	N.S.							61	302

Shown as Table 3, we can see that the local governments' intake requirements of vitamin B₁ according to local conditions. According to the requirements, we can regulate the intake requirements of complementary food for infants and young children.

3. Analysis of CT Data of Infants with Vitamin B₁ Deficiency

3.1. Main Symptoms of Patients

35 cases of infantile cerebral beriberi were collected, including 15 males and 20 females, aged from 1 month to 1 year old. Among them, there were 3 two month infants, 4 three month infants, 3 four month infants, 6 five month infants, 5 six month infants, 2 seven month infants, 3 eight month infants, 1 nine month infant, 3 10 month infants, 2 11 month infants and 3 one year old infants. All the 35 cases were from rural areas, most of them were rice producing areas. The main food is

refined white rice. The children are breastfed without supplementary food. The mother has a partial feeding habit. Clinically, 7 cases had vomiting, 6 cases had fever, 19 cases were in low spirits and 20 cases had convulsions. The main symptoms and signs in this case were mental retardation, apathy, blindness, slow response, hoarseness, silent crying or loss of voice, soft limbs, weak sucking, vomiting, etc.

3.2. Mechanism of Vitamin B₁ Deficiency

Vitamin B₁ is a water-soluble vitamin and a coenzyme involved in glucose metabolism. Glucose metabolism provides almost all the energy required by nerve tissue. The body is unable to synthesize, reserves are not rich, mainly rely on food intake. Vitamin B₁ participates in glucose metabolism in the form of thiamine pyrophosphate [10]. It is pyruvate dehydrogenase [11], enzyme ketoglutarate dehydrogenase and transketolase coenzyme. Vitamin B₁ deficiency can lead to thiamine pyrophosphate deficiency, hinder oxidative dehydrogenation of pyruvate, lead to disorder of glucose metabolism and accumulation of lactic acid and pyruvate in local tissues. The energy supply of nerve tissue is mainly provided by glucose oxidation, so the deficiency of vitamin B₁ easily affects the energy supply of nerve tissue, leading to degeneration and necrosis of nerve tissue.

According to the literature reports, the autopsy pathological changes of vitamin B₁ deficiency encephalopathy are mainly the proliferation of capillary endothelial cells in caudate nucleus and small nucleus of bilateral basal ganglia region, narrowing or occlusion of lumen, resulting in local tissue edema, degeneration and necrosis. In infancy, brain tissue develops rapid and oxygen consumption is high (accounting for 50% of the total oxygen consumption of human body). Therefore, vitamin B₁ deficiency in infants and young children is easy to cause damage to the central nervous system. However, white matter, inner capsule and thalamus were not sensitive to abnormal metabolic changes, and the lesions appeared late and less in children. In addition, brain atrophy [12] is a common feature of brain atrophy in infants. Therefore, brain atrophy can be used as one of the characteristics of the disease.

Vitamin B₁ mainly exists in the seed coat and germ, and excessive processing of rice or discarding rice soup when cooking will cause a large loss of vitamin B₁ in diet. However, the main source of our case is from the rice producing areas in rural areas. In rural areas, the pure white rice which has been rolled for 2-3 times is generally used, and the cooking method of cooking rice with discarded rice soup is adopted. This is the main cause of vitamin B₁ deficiency.

3.3. Pathological Changes of Nervous System in Vitamin B₁ Deficiency

The proliferation of main capillary endothelial cells, such as the bilateral basal ganglia of caudate nucleus [13], lenticular nucleus [14], and bilateral basal ganglia of Globus pallidus, as well as luminal stenosis and occlusion, lead to ischemia, hypoxia, edema and / or degeneration of neurons in the lentiform nucleus and caudate nucleus in varying degrees. Microscopically, the nucleus was condensed or expanded, the structure was not clear, the number of nerve cells decreased, and the proliferation of glial cells was formed. In some cases, atrophy and vacuolation may occur. Autopsy also found that papillary body, putamen, Globus pallidus, inner capsule capillary endothelial cells significantly proliferated, resulting in lumen stenosis or occlusion. There were many small soft lesions in the Globus pallidus and inner capsule. The cerebral cortex of temporal lobe and anterior and posterior central gyrus showed chronic degeneration. The injury of basal ganglia is reversible. The reversibility of basal ganglia is confirmed by the deprivation and supplement of thiamine in animal experiments. These lesions can be shown by CT, which is also an important means to evaluate the prognosis of the disease.

3.4. Development of CT Technology and Its Application in the Study of Infant Vitamin B₁ Deficiency Encephalopathy

Since the introduction of CT in 1973, CT has revolutionized the field of Radiology and become the basic equipment of modern radiology. People have been making great efforts to study better system structure and more advanced image reconstruction algorithm for important biomedical applications. As an important and widely used examination method, CT is becoming more and more common in children's emergency. Children are more sensitive to ionizing radiation than adults. Therefore, the application of CT in children should be paid more and more attention. By understanding the use of CT examination in children in emergency, it will remind and promote emergency physicians in pediatric emergency CT, further standardize the use of CT, and help the management to formulate corresponding strategies and rules, so as to reduce the use of emergency CT in children and reduce the damage caused by ionizing radiation.

CT plays an important role in tissue volume quantification, diagnosis, pathological localization, anatomical structure research, treatment plan, partial solvent correction of functional imaging data, and computer integrated surgery. This paper mainly introduces the CT study of infantile vitamin B₁ deficiency encephalopathy. Firstly, DICOM file is converted into bmp image; secondly, the image is preprocessed; secondly, in order to reduce the interference of skull and other tissues on the extraction of lesion area; finally, the intracranial structure is clustered and the region growing algorithm is used to extract the bleeding lesions in the classified image. GE 8800 and Somatom plus 5 scanners were used for CT scanning, and the slice thickness and interval were both 10 mm. 38 cases were plain scan, 3 cases were plain scan and enhanced scan. 60% meglumine diatrizoate was intravenously injected 1.5ml/kg during enhanced scanning.

In the CT study of infantile vitamin B₁ deficiency encephalopathy, sometimes it is difficult to differentiate according to CT findings. Therefore, clinical data and laboratory examination should be closely combined in the diagnosis, such as the determination of pyruvic acid and thiamine in blood and cerebrospinal fluid, or experimental treatment of vitamin B₁. because the disease has a good response to this method, it is the best verification method.

4. Results and Discussion

4.1. Misdiagnosis of Vitamin B₁ Deficiency Encephalopathy

In clinical treatment, vitamin B₁ deficiency will be misdiagnosed due to various reasons. Next, we will give specific directions and causes of misdiagnosis for future clinical treatment.

Shown as Figure 1 that there are three main misdiagnosis directions of vitamin B₁ deficiency encephalopathy. (1) Hepatolenticular degeneration (HLD) is an autosomal recessive disorder of copper metabolism. However, the general population is 3-60 years old, which is rare in infants. (2) CT findings of carbon monoxide poisoning were similar to those of vitamin B₁ deficiency encephalopathy in infants. It is very possible for infants to contact with other diseases. (3) Encephalitis, such misdiagnosis is is mainly due to our lack of thorough understanding of this disease.

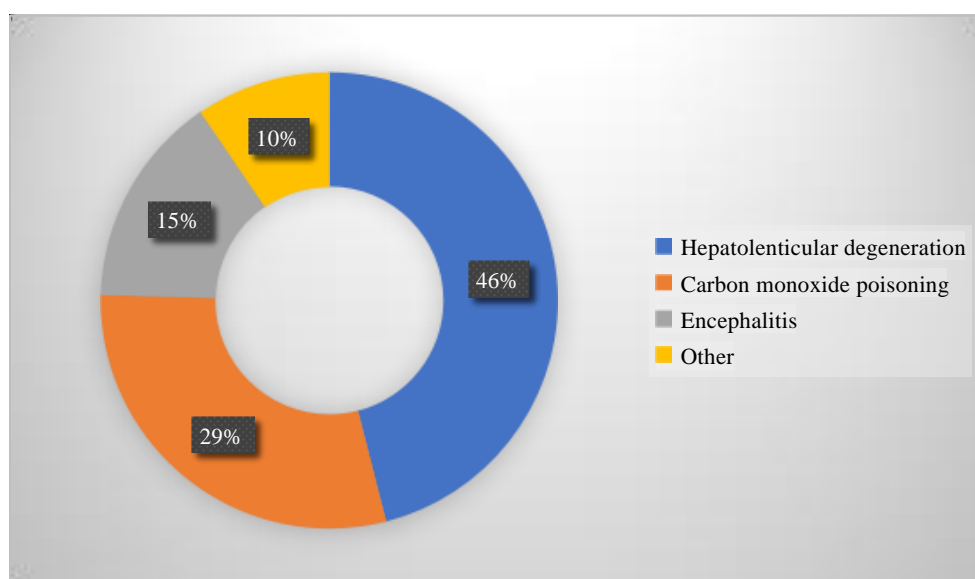


Figure 1. Analysis of misdiagnosis of vitamin B₁ deficiency encephalopathy

4.2. Misdiagnosis Rate of Vitamin B₁ Deficiency Encephalopathy before and After CT Diagnosis

The causes of misdiagnosis are as follows: (1) clinicians lack of understanding of vitamin B₁ deficiency and only consider common diseases and frequently occurring diseases, leading to misdiagnosis in many hospitals. The main reasons for misdiagnosis are that the out-patient doctors do not ask the medical history in detail and the physical examination is not serious, which will only make the diagnosis based on some manifestations or unilaterally emphasize some signs, lack of the overall situation and the whole process of the disease, and ignore the history of vitamin B₁ investigation, clinical manifestations and the history of infant feeding and lactating women. (2) When vitamin B₁ is deficient, energy is insufficient, acetylcholine synthesis is decrease and multiple organs are damaged. The symptoms of central nervous system in infants are particularly prominent, which is easy to be confused with intracranial diseases and misdiagnosed. Doctors only focus on the common manifestations of brain diseases, such as drowsiness, coma and convulsion. Neglecting the collection of medical history, paying little attention to the decrease of muscle tension, weakness of neck and limbs, disappearance of deep reflex, negative pathological signs, etc., which lead to misdiagnosis. (3) This disease can be caused by other diseases, increase the consumption of vitamin B₁, the clinical manifestations are more complex. 25 cases were accompanied with fever, and central nervous system symptoms appeared after upper limb sensation, which increased the difficulty of diagnosis. (4) Some diseases and complications are covered up. The symptoms of rickets and febrile convulsion were found in 20 cases. Only hypocalcemia convulsion was diagnosed. The diagnosis was corrected after the symptoms could not be controlled by calcium supplement. 5 cases of sudden convulsion, somnolence and moderate anemia were misdiagnosed as intracranial hemorrhage and were excluded by lumbar puncture and head CT. (5) The knowledge of subclinical and fulminant subtypes was insufficient. In recent years, the number of subclinical cases has increase and it is not easy to find. Although fulminant disease is rare, it is sudden, dangerous and rapid. Most of the patients were in coma at the time of admission. Central respiratory failure is the most prominent manifestation. Most of them are accompanied by heart failure or brain failure. If they're not identified and there's no targeted treatment, they're going to die soon.

4.3. Help of Medical Imaging Technology for Disease Examination

In recent years, with the continuous development of medical imaging technology and the rapid promotion of equipment including multi-slice spiral CT and high field intensity MRI, the extremely high image resolution and super large capacity image information provide the possibility for more accurate diagnosis. It reduces the misdiagnosis caused by the symptoms are not obvious.

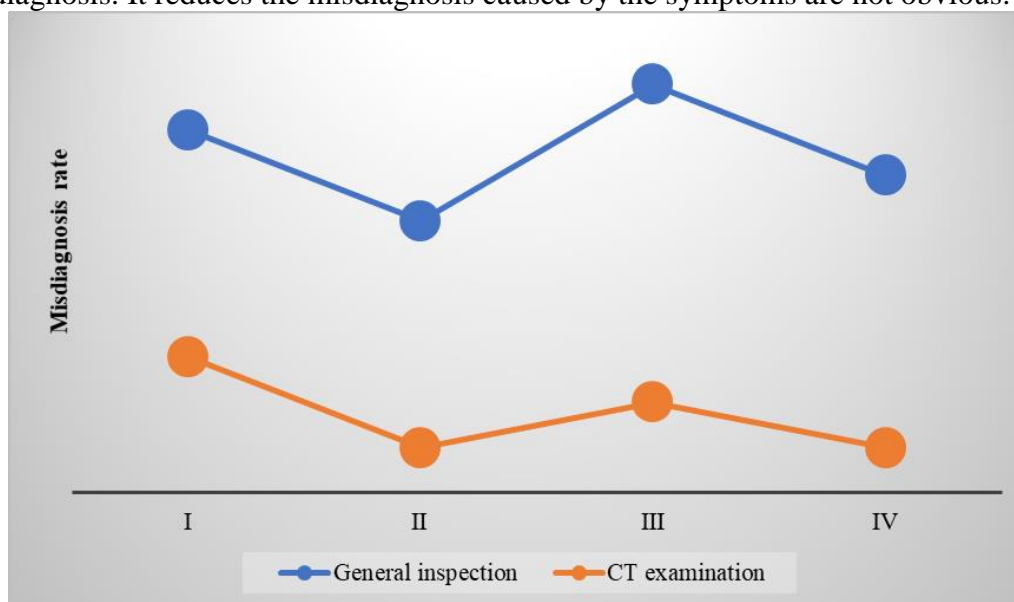


Figure 2. The change of misdiagnosis rate of vitamin B₁ deficiency encephalopathy before and after using CT technology

Shown as Figure 2, I stand for one to three months, II for four to six months, III for seven to nine months, and IV for ten to two months. After the use of medical imaging technology, the misdiagnosis rate of vitamin B₁ deficiency encephalopathy has been greatly reduced, at the same time, the doctor's work pressure has been reduced, the focus is obvious, which provides great help and convenience for the follow-up treatment of vitamin B₁ deficiency encephalopathy.

4.4. Characteristics of Vitamin B₁ Deficiency Encephalopathy

Vitamin B₁ deficiency encephalopathy mainly causes symmetrical low-density changes in bilateral basal ganglia and thalamus on CT, especially lenticular nucleus. In addition, it should be emphasized that vitamin B₁ deficiency encephalopathy can also involve the inner capsule, the outer capsule and the periventricular white matter area, making it a symmetrical low-density change.

As can be seen from Figure 3, sample. 1 stands for anti-limb, sample. 2 for deep white matter of bifrontal lobe, sample. 3 for external capsule, sample. 4 for trigonal white matter of lateral inventory, and sample. 5 for other. The ordinate is probability of involved parts. Among the 35 cases, 8 cases involved the anterior limb of the internal capsule, 9 cases involved the deep white matter of the double frontal lobe and 7 cases of the capsule, and 5 cases involved the white matter of the trigonometry of the lateral ventricle. The results of autopsy showed that the pathological changes of low-density white matter were similar to those of gray matter in basal ganglia, which were related to the obvious proliferation of capillary endothelial cells, stenosis or occlusion of capillary lumen, ischemic necrosis and softening. In addition, demyelination of white matter was observed. Therefore, we believe that the disease not only causes symmetrical low density of gray

matter nuclei such as bilateral basal ganglia and thalamus, but also causes low density changes in the inner capsule, outer membrane and lateral ventricle white matter.

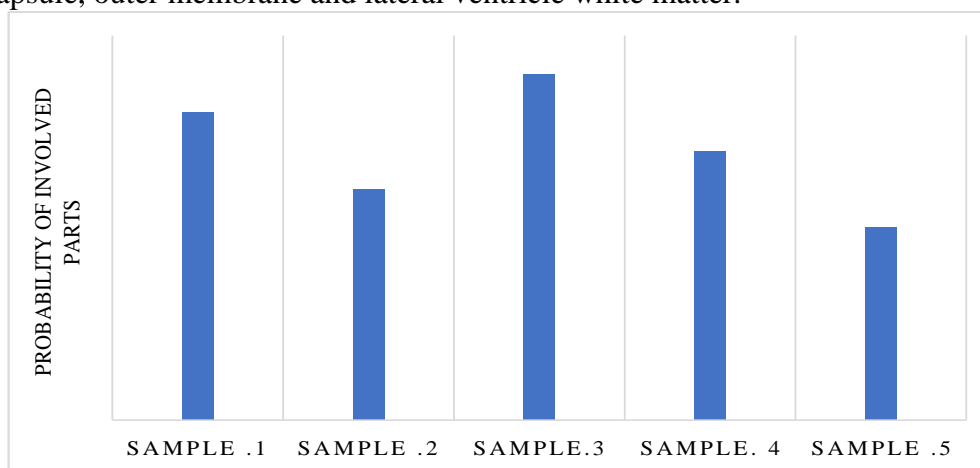


Figure 3. Involved parts of vitamin B₁ deficiency

4.5. Proportion of Children with Vitamin B₁ Deficiency in Urban and Rural Areas in Recent Ten Years

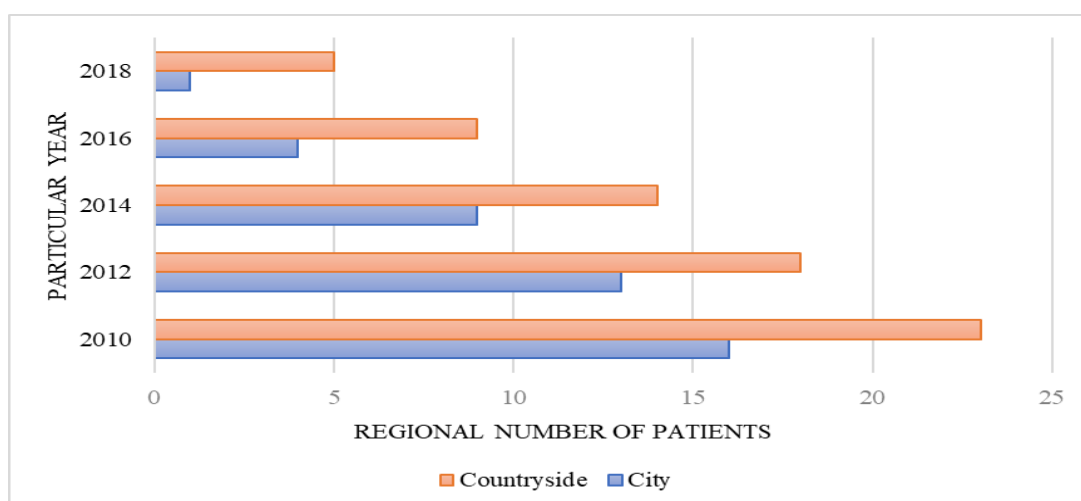


Figure 4. Changes of vitamin B₁ deficiency encephalopathy in urban and rural areas in recent ten years

Shown as Figure 4 that the proportion and number of infants with vitamin B₁ deficiency encephalopathy have changed in recent ten years in the area we investigated. This is due to the increasingly developed medical technology and the development of related impression technology.

In addition, symmetrical low-density changes of basal ganglia, thalamus, inner capsule, outer membrane and paraventricular white matter caused by the disease are important CT signs, which can provide important diagnostic reference for clinical. However, the symptoms of the disease are not specific to the disease, such as Lee's disease, Wilson's disease, Parkinson's disease, bilateral basal ganglia infarction, methanol, cyanide, hydrogen sulfide and mildew cane poisoning, as well as atrophy of substantia black striatum. Sometimes it is difficult to distinguish according to CT findings, so the diagnosis should be closely combined with clinical data and laboratory tests, such as pyruvate and thiamine in blood and cerebrospinal fluid, or vitamin B₁ experimental treatment,

because the disease has a good response to this method, it is the best verification method. In addition, we believe that the age of onset is also important in differential diagnosis.

5. Conclusion

Vitamin B₁ deficiency encephalopathy (VDE) is a common nutritional deficiency disease. Bilateral hemispheric injury is symmetrical. In serious cases, it can lead to cerebral palsy and even death. The clinical manifestations were nonspecific, and CT signs had certain diagnostic value. The physiological function of vitamin B₁ is to promote the complete oxidation of carbohydrate and other substances, and maintain the normal functions of nerve, heart and digestive system. Its deficiency not only affects glucose metabolism, but also involves fatty acid and energy metabolism, resulting in the accumulation of pyruvate and lactic acid in tissues, resulting in dysfunction of digestive system, nervous system and cardiovascular system. The clinical manifestations were polydipsia, inflammation, muscle atrophy, tissue edema, cardiac enlargement, circulatory system disorder and gastrointestinal symptoms. Vitamin B₁ deficiency_ It is difficult for pyruvic acid in the blood to enter the tricarboxylic acid circulation and oxidize. Pyruvic acid and lactic acid are mainly present in tissues and blood, which affect the energy source and function of nerve tissue and muscle (skeletal muscle and myocardium). Pyruvic acid and lactic acid are accumulated in neuromuscular tissues, resulting in neurological and muscular symptoms.

Medical imaging technology is a very valuable tool in today's medicine. Computed tomography (CT) provides an effective method for noninvasive acquisition of anatomical structures. These techniques greatly improve the understanding of normal tissue structure and lesions in medical research and are key components of diagnosis and treatment planning. With the increasing of the size and quantity of medical images, it is necessary to process and analyze images by computer. The misdiagnosis rate of infant vitamin B₁ deficiency encephalopathy is high. If CT technology is used, the misdiagnosis rate can be effectively reduced. For the diagnosis and treatment of the disease has great help.

Vitamin B₁ deficiency encephalopathy is common in infants with unreasonable diet structure, long-term partial diet, picky food, reduced digestive function, resulting in reduced absorption of vitamin B₁, or long-term fever, chronic consumptive diseases, metabolic diseases, and increased body requirements under specific occupational environment and living conditions. In the future, more attention should be paid to the prevention of the disease and try to stop the disease from the source. In the scientific research work of medical workers, it is necessary to seek a better evaluation of thiamine nutritional status and specific diagnostic methods. The expanded application and innovative technology of CT can be further explored. We should be alert to suspected cases and supplement vitamin B₁ for diagnostic treatment immediately.

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Data Availability

Data sharing is not applicable to this article as no new data were created or analysed in this study.

Conflict of Interest

The author states that this article has no conflict of interest.

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