BRITISH MEDICAL JOURNAL

10.5281/zenodo.7308619
British Medical Journal
Volume 2, No 4., 2022
Internet address: http://ejournals.id/index.php/bmj
E-mail: info@ejournals.id
Published by British Medical Journal
Issued Bimonthly
3 knoll drive. London. N14 5LU United Kingdom
+44 7542 987055
Chief Editor
Dr. Fiona Egea

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British Medical Journal Volume-2, No 4
10.5281/zenodo.7308619

SCIENTIFIC ASSESSMENT OF THE MORPHOFUNCTIONAL CHARACTERISTICS OF THE LUNGS IN DIABETES

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Abstract: Diabetes mellitus is associated with metabolic and morphofunctional changes in the brain and the development of diabetic encephalopathy, which is characterized by cognitive, sensorimotor and psychoemotional disorders. There are given main information about scientific assessment of the morphofunctional characteristics of the lungs in diabetes in this article.

Keywords: Diabetes, morphofunctional, International Health Organization, health, sugar in the blood.

INTRODUCTION

Diabetes occurs when the body is unable to maintain a normal level of sugar in the blood. According to the International Health Organization, more than 420 million people in the world have diabetes. This is 25% more than 40 years ago. Diabetes is a chronic disease and there is no way to get rid of it completely. However, in modern times, ways to fully control the amount of sugar in the blood have been developed. This ensures that patients with diabetes live a long and happy life without being separated from other healthy people. A person can stop it by changing his lifestyle and some habits.

Diabetes mellitus is divided into several types. The most common type of diabetes is type 2 diabetes. In this type of diabetes, the production of the hormone insulin, which is responsible for the acceptance of sugar in the blood by the cells of the body as food, is either reduced, or (usually) some reasons prevent its full functioning. These changes usually occur in middle-aged and elderly people.

Who is more likely to develop diabetes? If a person leads a sedentary lifestyle, is obese, eats wrong (many and high-calorie), and has high blood pressure, the probability of early onset of this disease increases. Heredity also plays a big role in the origin of the disease.

MAIN BODY

How is blood sugar controlled? The blood of all healthy people contains a certain amount of sugar (glucose) as the main source of energy. Sugar enters the blood in two ways:

- With foods that contain carbohydrates.
- From glucose (glycogen) reserves in the liver.

As a source of energy, sugar enters muscle cells (for work), fat and liver tissue (for reserves). Sugar (glucose) enters the cells with the help of the insulin hormone produced by the pancreas. After a person eats, the amount of sugar in the blood rises. But the pancreas immediately produces insulin and distributes it into the blood, and this insulin opens the gates of all cells for glucose. After glucose enters the cells, the amount of sugar in the deposit returns to normal. Between meals and during sleep,

10.5281/zenodo.7308619
the blood sugar level is replenished from the reserve in the liver. This process is also controlled by insulin.

In type 2 diabetes, the pancreas produces insulin. But:

Insulin is not produced at the right time and in the right amount, and insulin production decreases over time.

- Normally, say, 1 molecule of insulin is needed to open the "gate" of 1 cell to glucose, but in type 2 diabetes several insulin molecules are needed. In other words, the body's resistance to insulin increases.
- To make a diagnosis of diabetes, it is necessary to measure the amount of sugar in the blood in a laboratory.

What are the symptoms of diabetes? Diabetes is manifested by constant weakness, frequent thirst, weight loss for no reason, feeling of hunger, frequent and large urination, wounds that do not heal for a long time, decreased sensation and pain in the legs, decreased vision. Why is diabetes dangerous? At the onset of diabetes, the patient is not known for a long time. In most cases, the diagnosis is made when the symptoms of the disease appear. Late diagnosis and late start of treatment cause the disease to be severe and complicated. Complications of diabetes can be divided into early and late types. Early complications are associated with a sharp drop or rise in blood sugar. These include hypo- and hyperglycemia, diabetic ketoacidosis, and others.

Late complications of diabetes are caused by effects on many organs and systems of the body. In this, the brain, eyes, heart and vascular system, kidneys, nerve fibers, legs and other internal organs are mainly damaged. Eye disease (diabetic retinopathy) occurs as a result of long-term high blood sugar levels in patients with diabetes. This can lead to a decrease in vision and even its loss. The effects of diabetes on the eyes begin long before vision loss. Therefore, patients with diabetes should be under the constant supervision of an ophthalmologist. Vascular complications are the main cause of disability and death in diabetes. These include angina pectoris (pains and pains in the chest area), stroke, vascular diseases of the legs (purulent ulcers and blackheads caused by poor blood flow to the legs), chronic heart failure with water accumulation in the lungs and other tissues, and others. These complications are aggravated by high blood pressure, high cholesterol and high blood sugar.

Diabetes is the leading cause of chronic kidney diseases. It is associated with damage to the most delicate blood vessels in the kidney, which causes the kidney to decrease and even stop. Damage to nerve fibers in diabetes (diabetic neuropathy) is also associated with long-term high blood sugar levels. In most cases, peripheral neuropathy occurs in the legs. This condition is accompanied by a decrease in sensitivity, cramps and pain in the legs. Due to the decrease in sensitivity, the patient may not feel the wounds on his leg. Secondary infection of these wounds can lead to deep ulcers and even amputation. These foot complications are called "diabetic heel", "diabetic ankle" and "diabetic foot". In addition, patients with diabetes suffer from blockage of blood vessels in the legs and blood circulation disorders. Impaired blood

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circulation is another major factor in the severity of wounds and the increase in amputations. Such a risk can be avoided if the patient checks his feet on time and keeps his blood sugar at a normal level. Women with diabetes require constant medical supervision during pregnancy. In men, it can cause erectile dysfunction, indigestion (diabetic enteropathy), and problems with urination.

At present, after reconsidering the ventilation and hemodynamics of the lungs, evidence has been gathered that indicates not only the ability of the surfactant system to be extremely adaptable, but also the true sensitivity of the components to many unfavorable factors of the tuberculosis process; The cause of the tuberculosis process is the long-term persistence of this process, which is manifested by the wave-like course of the process, the microcirculatory flow disorder. The changes taking place in it include not only the place of formation of the foci of infection, but also the active working areas of the lung parenchyma. In this regard, assessment of the morphofunctional smoothness of various components of the surfactant system is very important in the diagnosis and timely correction of surfactant-related disorders of respiratory function. Early signs of lung surfactant destruction can be observed using specific lung fixation in experimental models. At the initial stage of the development of tuberculosis inflammation, they are local and are mainly detected in areas where there is swelling inside the alveoli. It is possible to observe the transition of the outer thin layer of the surfactant membrane and the different stages of its erosion by the swelling liquid in the electron microscope. These changes are most pronounced in the inflammatory foci of tuberculosis, where the absorbed surfactant material is found everywhere in the contents of the alveoli. When tuberculosis inflammation develops, a different picture is observed in the respiratory organs, because the inflammatory surfactant has a bad effect on the processes of intracellular synthesis. Direct inoculation of Mycobacterium tuberculosis into the lungs of dogs (through the chest with a needle) showed that the cytoplasmic network and mitochondrial profiles were altered within the first 15-30 minutes; and after a few hours, alveolocytes are completely destroyed at the place of infection. The rapid development of deficiency of surface-active substances leads to collapse of alveoli and spread of inflammation to the surrounding parenchyma. In the alveoli close to the foci of tuberculosis, small secretory granules are dominated by smaller young A2 cells, or the intracellular structures are large cells with signs of vacuolization, sometimes with completely eroded cytoplasm. Giant osmiophilic plate-like bodies are detected in alveolates, which are widespread elements of the cytoplasmic network and plate-like complex, which indicates that the intracellular surfactant cannot be brought to the surface of the alveoli.

Of much greater interest are diet-induced modeling of the pathological process. In the natural environment, most often, the development of type 2 diabetes is caused by a chronic excess of carbohydrate nutrition. Endocrinologists assign a significant role in the development of type 2 diabetes to simple carbohydrates, which supply significant amounts of glucose during their breakdown during digestion in the gastrointestinal tract. One of the most important and accessible donors of glucose is food sugar (sucrose), which in the evolutionary process of the development of life began to be widely used by man in the recent past.

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Excess sugar causes a violation of carbohydrate metabolism and the development of hyperglycemia, which can initiate biochemical and structural changes characteristic of type 2 diabetes. Hyperglycemia stimulates the level of secretory activity of beta cells, leading to the depletion of insulin production, induces the processes of glycosylation of proteins and lipids. As a result of violations of carbohydrate metabolism in the cells of the pancreas, changes in lipid and protein metabolism develop. An increase in blood glucose levels increases insulin resistance and leads to a decrease in the sensitivity of P-cells, causing a violation of insulin secretion. A vicious circle develops: an increase in glucose levels increases insulin resistance, which contributes to the development of even more pronounced hyperglycemia. Hyperglycemia is accompanied by activation of peroxidation processes, which lead to excessive formation of free radicals that have a cytotoxic effect on cells and tissues.

The model of intraperitoneal administration of glucose is characterized by the absence of direct damage to the islets of Langerhans of the pancreas, however, at high doses of intraperitoneal administration of glucose, it causes a disorder of the water and electrolyte balance, the development of dehydration and the appearance of crystalluria and kidney stone formation.

Absorption of glucose from the gastrointestinal tract leads to greater stimulation of B cells and release of insulin than when glucose enters the bloodstream, by passing the gastrointestinal tract, which contributes to the creation of more prolonged hyperglycemia. We induced hyperglycemia in rats by natural daily oral administration of glucose with an unbalanced carbohydrate diet predominantly sugar for a month. Purpose of the study: To study the nature of biochemical and structural changes in the pancreas of rats with an unbalanced carbohydrate diet with food sugar.

Diabetes mellitus is associated with metabolic and morphofunctional changes in the brain and the development of diabetic encephalopathy, which is characterized by cognitive, sensorimotor and psychoemotional disorders. Dement disorders are perceived most painfully by patients and their relatives. Cognitive impairments are classified according to the severity of disorders into mild, moderate and severe [2]. With mild cognitive impairment, the speed of information processing, decreased concentration of attention, the ability to quickly switch from one type of activity to another, and working memory are more affected. Moderate cognitive impairments are characterized by memory impairment, disorientation in an unfamiliar area, impaired performance of counting operations, and deterioration in labor competencies. With moderate cognitive dysfunction, patients themselves turn to the doctor with complaints of memory impairment, learning difficulties. Dementia is considered a manifestation of severe cognitive impairment, characterized by complete social and everyday maladjustment. In patients with diabetic encephalopathy, the risk of complications of diabetes mellitus, including life-threatening ones, increases due to a decrease in the ability to adequately take recommended drugs and control glycemia.

Morphofunctional Changes in the Brain in Diabetes Mellitus In diabetes mellitus, with increasing age and duration of diabetes, the risk of developing neurodegenerative diseases, psychoemotional, sensorimotor, and cognitive

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impairments increases. In type 1 diabetes, hypoglycemic reactions are more common, and less common in type 2 diabetes, in response to insulin and, very rarely, to other antidiabetic agents. Hypoglycemic reactions cause deeper functional (cognitive) and morphological changes in the brain, especially in the hippocampus, that is, in the structures most sensitive to glucose and oxygen deficiency. This assumption is confirmed by the influence of glucose fluctuations on the state of hippocampal neurons. In the formation of cognitive dysfunction, DM is an independent risk factor. However, additional risk factors, angiopathy, hyperglycemia, lipid metabolism disorders, oxidative and nitrosative stress led to endothelial dysfunction, dyslipidemia and atherosclerosis, impaired microcirculation, arterial hypertension, impaired blood supply to the myocardium and brain, which increases the likelihood of cognitive dysfunction in diabetes mellitus. At the same time, anxiety-depressive manifestations are diagnosed with high frequency with diabetes mellitus. This is based on metabolic disorders in the limbic system, which may be associated with depressive manifestations. Structural changes in the brain in diabetes mellitus are largely the result of micro and macroangiopathies caused by endothelial dysfunction associated with glyco- and lipotoxicity, activation of inflammatory factors: the transcription factor Nf-kb, which stimulates the production of pro-inflammatory cytokines. Changes in cerebral vessels are observed in almost all patients with type 2 diabetes.

CONCLUSION

Changes that occur in the brain in DM manifest themselves as a violation of structural elements, cognitive and psycho-emotional functions. Cerebral angiopathy was detected in all patients with DM. Marked sclerosis of cerebral vessels, capillary congestion and stasis of blood in the cortical substance of the cerebral hemispheres, fibrosis and hyalinosis of capillaries. The most frequently registered changes are sclerosis of the temporal lobes of the brain, a decrease in the volume of white matter in the cerebral cortex, mainly in the temporal and frontal lobes, as well as a decrease in the volume of gray matter in the hippocampus, thalamus, and insular cortex. In most cases, DM leads to mild to moderate cognitive impairment in the form of a decrease in attention, memory, information processing, as well as a decrease in psychomotor efficiency. Severe cognitive impairment in DM is a manifestation of dementia associated with Alzheimer's disease or cerebrovascular pathology.

The duration of the disease, age, disease severity, glycemic control, episodes of hypoglycemia, and the presence of comorbidity have a negative impact on neurodegeneration processes and cognitive functions in patients with DM. Animals with experimental DM showed pronounced morphofunctional changes in the synaptic apparatus, damage to neurons and a decrease in their total volume with an increase in the permeability of brain capillaries. Early reversible structural changes in neurons are swelling of neurons (hydropic dystrophy of nerve cells), local hyperchromatosis. Irreversible structural damage to neurons is manifested by cell death, incl. by apoptosis, the formation of shadow cells, the formation of vacuoles in the cytoplasm, the lysis of axons with neurophagy. A combination of acute neuronal damage with chronic ischemic damage is also characteristic. The above changes are combined with atrophy of the cortex, hippocampus and vascular changes. Thus, functional and

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structural changes in the brain in DM are accompanied by a wide range of morphofunctional disorders.

At present, it remains relevant to study the processes of damage and regeneration in various parts of the brain, in particular, in the hippocampus, cerebellum, cortex, hypothalamus, taking into account the processes of neuroinflammation, reactions from glial cells and vessels of the microvasculature. Particular attention should be paid to the study of the mechanisms of neuroprotection, including GABA-dependent and autophagy, the influence of these mechanisms on the survival of neurons and the state of neurogenesis.

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