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Research Article

GRANULOMATOSIS WITH POLYANGIITIS: LITERATURE REVIEW

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ABSTRACT

Wegener's granulomatosis (granulomatosis with polyangiitis) is a rare autoimmune disease characterized by vasculitis of small and medium-sized vessels and the formation of granulomas. Most often, Wegener's granulomatosis (WG) affects the upper respiratory tract, lungs, kidneys, and eyes. Without treatment, the disease progresses rapidly and leads to death. GV was first described at the end of the 19th century. The characteristic clinical picture of hepatitis B was described by the German pathologist Friedrich Wegener. After the reports of F. Wegener in 1936 and 1939. In cases of necrotizing vasculitis with granulomatous inflammation as a special nosological form of vasculitis, this disease became known as Wegener's granulomatosis. In 2006, F. Wegener's connection with Nazism was discovered, and it was proposed to abandon the term "Wegener's granulomatosis" in favor of the term "granulomatosis with polyangiitis," which, in general, more reflects the modern view of the essence of this disease. The term "Wegener's granulomatosis", however, is today used along with the term "granulomatosis with polyangiitis".

KEYWORDS

Wegener's granulomatosis, vasculitis, polyangiitis.

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INTRODUCTION

Granulomatosis with polyangiitis (GPA), formerly known as Wegener's granulomatosis (WG), is a rare disease from the group of systemic ANCA-associated vasculitides (systemic vasculitis associated with the presence of antineutrophil cytoplasmic antibodies), characterized by granulomatous inflammation and necrotizing vasculitis of small and medium-sized vessels (capillaries, venules, arterioles, arteries), affecting mainly the upper and lower respiratory tract, as well as the kidneys [1-4]. The prevalence of hepatitis B in the population is 25-60 cases per 1 million population, the incidence is 3-12 cases per 1 million. However, the incidence of hepatitis B in European countries, according to some data, has increased 4 times over the past 30 years. An increase in new cases of the disease has been noted in the autumn-winter period [3].

The German pathologist F. Wegener described this disease as a separate nosological unit, noting the characteristic clinical and morphological features, and for the first time identified this disease as a special form of vasculitis [5-8]. In 1954, G. Gudman and J. Churg formulated clinical and morphological criteria for hepatitis B, including a triad of pathological signs: systemic necrotizing vasculitis, necrotizing granulomatous inflammation of the respiratory tract and necrotizing glomerulonephritis. The first description of GW in Russian literature belongs to A.I. Strukov and V.V. Badmaeva [9].

The reasons for the development of this malignant necrotizing vasculitis are still unknown. The morphological substrate of the disease is granulomas, accompanied by necrosis [10-12]. Therefore, the gold standard for confirming the diagnosis today remains histological examination of the affected tissue (nasal mucosa, lung tissue, skin or kidney, granulation tissue of the orbit of the eye), obtained during surgery or by biopsy. Two types of changes are detected in the tissues studied: necrotizing granuloma and necrotizing vasculitis. The disease is characterized by a widespread necrotizing process in blood vessels of all levels with predominant damage to muscular arteries and the microvasculature. Panarteritis with the spread of inflammatory phenomena to all three membranes of the vessel is typical for hepatitis B. As a result of destructive-productive vasculitis, giant cell granulomas are formed, followed by their destruction or necrosis [1, 3-15].

GV is divided into local, limited and generalized forms. The local form is an isolated lesion of the upper respiratory tract, organ of hearing and organ of vision. The limited form implies damage to the lungs with the presence or absence of local lesions of the upper respiratory tract, hearing and vision. The generalized

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form is characterized by the development of glomerulonephritis, regardless of the presence or absence of damage to other organs. During the GW there are three periods. The onset of the disease is characterized by local changes in the upper respiratory tract, middle ear or eyes [16]. During the period of generalization, internal organs, primarily the lungs and kidneys, are involved in the pathological process. In the terminal period, the development of renal and/or pulmonary heart failure progresses. The clinical picture of hepatitis B is very diverse. Common symptoms at the onset of the disease are fever, general weakness, weight loss, myalgia/arthralgia [17]. The onset of the disease can be subacute (with the development of clinical symptoms over several weeks) or primary chronic. One of the leading manifestations is damage to the ENT organs, which occurs in the advanced stage of the disease in 90-94% of patients.

Early diagnosis of hepatitis B is a difficult clinical task and requires a thorough examination of the patient using modern research methods to identify pathognomonic symptoms. A targeted search for damage to the respiratory tract is necessary, including rhinoscopy, laryngoscopy, computed tomography of the paranasal sinuses and lungs, since for a long time the disease can be asymptomatic or accompanied by scanty clinical symptoms. Only in 50% of patients the diagnosis is verified in the first 3-6 months from the onset of the disease, and in 7% of patients, HB remains undifferentiated for 5-16 years from the onset of the first symptoms [18].

In 1990, the American College of Rheumatology developed classification criteria for GPA [3]:

1. Inflammation of the nose and mouth, ulcers in the mouth, purulent or bloody discharge from the nose.

2. Changes in the lungs during X-ray examination (nodules, infiltrates or cavities).

3. Microhematuria (>5 red blood cells per field of view) or accumulations of red blood cells in the urine sediment.

4. Biopsy: granulomatous inflammation in the arterial wall or in the perivascular and extravascular space.

The presence of two or more criteria allows a diagnosis to be made with a sensitivity of 88% and a specificity of 92%. In the absence of biopsy results, it is proposed to include an additional symptom – hemoptysis – into the diagnostic criteria.

CONCLUSION

Early diagnosis of hepatitis B is a difficult clinical task and requires a thorough examination of the patient using modern research methods to identify pathognomonic symptoms. A targeted search for damage to the respiratory tract is necessary, including rhinoscopy, laryngoscopy, computed tomography of the paranasal sinuses and lungs, since for a long time

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