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# Fibrotic-Cavernous Pulmonary Tuberculosis: Modern Diagnosis and Treatment

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**Abstract:** Pathologic substrate of these forms of secondary tuberculosis is considered in detail. The possibilities and advantages of an innovative approach of valvular bronchial blocking used in complex treatment of destructive forms of pulmonary tuberculosis are presented. The technique enables to close completely the cavities in 70% of patients. The advantages of the method are described. The characteristics of clinical presentation and radiodiagnosis of the disease are given, and treatment techniques are presented.

**Keywords:** tuberculosis, caverna, bronchial blocking,radiodiagnosis.

**Relevance.** Fibrous-cavernous tuberculosis and its complications are the main cause of death in patients with pulmonary tuberculosis (75-80%). The disintegration of the focus of specific inflammation in the lungs and the formation of a cavity can be observed with the progression of any form of tuberculosis, if predisposing conditions arise for this in the form of changes in the reactivity of the body, increased sensitization, massive superinfection, the addition of other diseases and the effects of various harmful factors that reduce overall resistance[3,9,11]. Under the influence of these factors, the permeability of vascular walls increases in the zone of tuberculous changes, where the reproduction of mycobacteria increases. The granulation tissue and caseous masses contained here are infiltrated by lymphoid elements and polynuclears, which secrete proteolytic enzymes, and a zone of perifocal inflammation appears around[5,1,8]. In the future, as a result of necrobiosis and purulent melting of curd masses, destruction is formed. For some time it remains closed and only after its contents are emptied through the draining bronchus and atmospheric air penetrates into the vacated space, a destructive cavity is formed. The wall of the newly formed decay cavity first consists of two layers: the inner — pyogenic-necrotic and the outer — granulation shaft. Then, collagen fibers are gradually formed in the outer part of the granulation layer, which form a thin, sometimes interrupted fibrous layer[6,11,15]. Over time, a three—layer wall is formed around the decay cavity, characteristic of the cavern. The size of the cavern varies widely - from a few millimeters to 10-20 cm or more. Medium (from 2 to 4 cm) are more common, less often — large (4-6 cm) and giant (more than 6 cm) cavities. The size of the cavities depends not only on the volume of the destroyed lung tissue and the elasticity of the surrounding parenchyma, but also on the condition of the draining bronchi, which are often involved in the pathological process. With the progression of the process, the walls of the bronchi are infiltrated by lymphoid and epithelioid cells, the mucous membrane is replaced by specific granulations, scars appear, which lead to the formation of various degrees of stenosis[9,14,18]. As a result, the normal patency of the bronchi is disrupted. When a valve mechanism is formed, the cavity stretches or swells, in such cases its dimensions often significantly exceed the actual volume of the destroyed lung tissue, and an atelectasis or distelectasis zone forms around the cavity. Cavernous tuberculosis Destructive cavity as a kind of manifestation of tuberculous inflammation is characterized by dynamism. With effective

treatment, and much less often, spontaneous resorption of the zone of perifocal inflammation and fresh bronchogenic foci occurs[12,20,17]. The cavity is clearly delimited from the surrounding lung tissue, but its walls do not yet have a pronounced fibrous-sclerotic character (elastic cavity). The clinical picture of cavernous tuberculosis is determined by the initial form of tuberculosis and the time of formation of the cavern. With a recent decay, the symptoms characteristic of the original form of the disease dominate.

**Material and methods.** The course of cavernous tuberculosis has the following features: with prolonged cavernous tuberculosis, the process is characterized by a wave-like flow with periodic outbreaks. Signs of an exacerbation of the process are increased ESR, a shift of the leukocyte formula to the left, lymphopenia. With the formation of the cavity, the bronchogenic spread of infection and persistent bacterial excretion begins to dominate. Intracananicular spread of infection with damage to other organs (larynx, intestines) is possible. There is a tendency to hemoptysis and bleeding, which sometimes lead to asphyxia or aspiration pneumonia, followed by bronchogenic insemination. The cavern can cause spontaneous pneumothorax and empyema. If the cavity is small and does not communicate with the bronchus, it is difficult to determine it during physical examination. With healthy and blocked cavities, Mycobacterium tuberculosis is usually not found in the sputum. They can sometimes be detected after puncture and washing of the cavity with saline solution. In such cases, the hemogram, ESR, protein content and other biochemical parameters are within the norm. During bronchoscopy, deformation and varying degrees of bronchial stenosis are detected. Outcomes of cavernous tuberculosis. 1. An important condition for this is the absence of pleural adhesions, which fix the affected lung to the chest and prevent the cavity from collapsing. 2. In other cases, after the rejection of the pyogenic membrane, the cavity is filled with a growing granulation tissue, which subsequently undergoes connective tissue transformation. 3. It is possible to close the cavity by performing it with caseous masses, lymph and tissue fluid with the formation of a homogeneous focus resembling tuberculosis (Fig. 2, c). This healing option is far from perfect. Often, under the influence of unfavorable factors, an aggravation of the process occurs and then a cavern is found again at this place. 4. It is possible to improve the cavity in an open way. In such cases, while maintaining the drainage function of the bronchi, most of the inner caseous-necrotic layer is rejected, and many macrophages, polyblasts, epithelioid and giant cells with basal granularity and a large number of nuclei are determined in the granulation layer. Small blood vessels and lymphoid follicles develop intensively here. Gradually, the inner surface of the cavity becomes smooth and is lined with a flat epithelium for a greater or lesser extent. A capsule of concentrically arranged argyrophilic collagen fibers is formed around it. However, only in some patients there is a complete connective tissue transformation and epithelization of the walls of the cavity. In most others, even despite complete clinical well-being and the prolonged absence of bacillus excretion, encapsulated tuberculous foci, elements of specific granulation tissue remain in the walls of such residual cavities. Active tuberculous changes sometimes remain in the areas of lung tissue adjacent to the cavern, and in the bronchi — tuberculous tubercles and epithelioid cells. Thus, cavernous tuberculosis is a process that is not homogeneous in its origin, pathomorphological substrate and final outcome. With ineffective treatment or its absence, the process progresses, repeated episodes of bronchogenic insemination, infiltrative outbreaks occur, followed by the development of fibrosis in and around the cavity wall, resulting in fibrocavernous pulmonary tuberculosis. Fibrotic-cavernous pulmonary tuberculosis Fibrotic-cavernous tuberculosis is the final stage in the progressive course of the destructive tuberculosis process. For all such patients, despite the variety of clinical and pathomorphological manifestations of the disease, common signs are the presence of a fibrous cavity or caverns, the development of fibrous changes in the surrounding lung tissue and polymorphic foci of bronchogenic dissemination, often in both lungs . The walls of the cavity in such cases have a three-layer structure with a predominance of a coarse fibrous layer, turning into fibrosis of the interlobular, interalveolar septa and pleura. Progressive fibrous-cavernous tuberculosis is characterized by a specific lesion of peribronchial tissue, smooth muscles and cartilaginous

plates of large bronchi, as well as the presence of tubercular and infiltrative-ulcerative changes in the submucosal layer and bronchial mucosa. Especially often small bronchi and bronchioles suffer, the walls of which are exposed to caseous necrosis. As a result, narrowing, amputation, obliteration of the bronchi are formed, cylindrical and small bagged bronchiectasis occur. With the progression of fibrous-cavernous tuberculosis, a giant cavern can form, often occupying an entire fraction of the lung or even almost the entire lung. Such a cavity can be multi-chamber with the presence of blood vessels in the beams crossing its lumen. In addition, large, often aneurysmally dilated blood vessels are detected near such a cavity, with a violation of the integrity of which massive pulmonary bleeding occurs. Complete scarring of the fibrous cavity occurs relatively rarely, since massive fibrosis in its walls and in the surrounding lung tissue prevents the collapse of such a cavity and its scarring. In fibrotic-cavernous pulmonary tuberculosis, the visceral pleura is often involved in the process first, and then the parietal pleura. Areas of perifocal inflammation, tuberculous foci, limited or extensive planar accretions appear in it. For this reason, not only the mobility of the lung is limited, but also the stretching increases and the healing of cavities becomes difficult, especially if they are located at the apex or in the cortical layers. An open cavern is a permanent source (reservoir) of the spread of Mycobacterium tuberculosis. It is estimated that this reservoir contains 1010-1012 mycobacteria — this is a huge bacterial population that is in an unstable state, multiplies and all the time supports the inflammatory process with the presence of necrosis in the cavity wall. During the outbreak, a rather pronounced perifocal inflammatory reaction develops around the cavity, foci of bronchogenic dissemination occur. With each new exacerbation, new foci of dissemination appear, the process becomes even more widespread. Individual foci of dissemination can merge into larger conglomerates, these conglomerates can also undergo destruction, destruction. This is how new caverns, or "child caverns", appear. Thus, with fibrotic cavernous tuberculosis, there is a threat not only of perifocal inflammation, not only bronchogenic dissemination, but also the appearance of new cavities in both the same and the opposite lung. In the terminal phase of the process, areas of caseous pneumonia are formed. This is the polymorphic pathomorphological picture of destructive tuberculosis, which also determines its peculiar clinical picture. The clinical picture of fibrous-cavernous tuberculosis is very diverse, due to numerous morphological and functional changes.

**Results and diccusion.** There are three clinical forms. 1. Limited and relatively stable fibrous-cavernous process (this form is rare). This is fibrous-cavernous tuberculosis with a limited lesion and a stable course of the disease, rare outbreaks; in such patients there is often no release of mycobacteria or it occasionally occurs during rare exacerbations. The interval between exacerbations stretches for several months, and sometimes even for several years. Such persons, in general, do not suffer much and do not really feel their disease. There is even a feeling of recovery. Patients say that the cavities they have do not interfere with them much. However, such a condition, such stability of the tuberculosis process is noted mainly in patients who strictly observe the regime and adapt their vital activity to new conditions — to the presence of the tuberculosis process in them. If they do not comply with these rules, especially if they lead a disorderly lifestyle, abuse alcohol, are exposed to hyperinsolation and other adverse external influences, exacerbation and progression of the tuberculosis process occur. This variant of the course of the disease is possible only in patients who regularly and for a long time take chemotherapy drugs. With the "chaotic" use of therapeutic agents, it is not possible to achieve stabilization of the fibrous-cavernous process. Due to the fact that patients are treated with chemotherapy for a long time, adequate chemotherapy is often hampered by two factors — drug resistance of mycobacteria and poor tolerability of chemotherapeutic agents. These two points do not always allow for adequate therapy, which also leads to the progression of the fibrous-cavernous process. 2. Progressive fibrous-cavernous tuberculosis. The progressive course of fibrous-cavernous tuberculosis (fast or slow) can develop from the very beginning of the disease without a previous period of stability. It (especially rapidly progressing) is characterized by undulation, i.e. frequent change of flashes and intervals. During the outbreak, intoxication is

expressed, which may persist during the interval. Patients are concerned about cough, sputum, hemoptysis, chest pain, and eventually shortness of breath. These clinical manifestations in the period of exacerbation correspond to the development of perifocal inflammation around the cavity, bronchogenic dissemination, concomitant endobronchitis. Sometimes there may be a lesion of the pleura, pleurisy develops. If the cavity breaks into the pleural cavity, spontaneous pneumothorax, purulent pleurisy occurs. Some patients develop meningitis, but currently this is observed infrequently. An objective examination determines pallor, adynamia, body weight deficiency, tachycardia. The chest on the side of the lesion is flattened, lags behind the healthy one in the act of breathing. With percussion, a shortening of the percussion sound is detected, and with large and rigid cavities, a box sound is detected. During auscultation, weakened or bronchial breathing, local wet and dry wheezing "squeak of a cavern", "creak of a cart" are heard. Bronchial or amphoric respiration is determined over large and giant caverns. In such patients, low blood pressure, tachycardia, and the accent of the II tone over the pulmonary artery are noted. Progressive forms of fibrous-cavernous tuberculosis are characterized by constant and massive bacterial excretion, the presence of drug-resistant Mycobacterium tuberculosis. Tuberculin sensitivity decreases. In peripheral blood, leukocytosis with a pronounced shift to the left, a significant increase in ESR are detected, there may be signs of anemia. There is a pronounced imbalance of protein fractions of blood serum, an increase in the content of fibrinogen, C-reactive protein, etc. With bronchoscopy, it is relatively often possible to detect specific changes in large, and especially in small bronchi. As the disease progresses, the function of respiration and blood circulation deteriorates, pulmonary hypertension progresses, the intensity of oxidative processes decreases, all types of metabolism are disrupted, blood oxygenation decreases, hypoxemia increases, secretion and acidity of gastric juice decrease, dystrophic changes and dysfunction of various parts of the nervous and endocrine systems occur. The general condition of the patient is adversely affected by specific (tuberculosis of the larynx or intestines) or non-specific (amyloidosis of parenchymal organs, pulmonary heart failure, etc.) complications. Frequent complications are pneumothorax, pleural empyema, pleural tuberculosis. Among other complications, arthropathies, arthralgias (and even polyarthritis, such as universal hyperplastic periostitis), endocrinopathies such as Cushing's syndrome or pituitary cachexia, adissonism, thyroid dysfunction can be observed in a patient with fibrotic cavernous tuberculosis. Radiological diagnostics of cavernous and fibrous-cavernous pulmonary tuberculosis, A destructive cavity in the lung is radiologically detected only if, after rejection of the molten contents, air enters it through the draining bronchus. Therefore, one of its radiological signs is the display of enlightenment against the background of darkening. The latter, in turn, depends on many factors — the initial form of the process, the structure of the cavity wall, the condition of the lung tissue. The main radiological symptom of a destructive cavity is the presence of an annular or wider border shadow around the illumination with a continuous closed contour that persists in at least two mutually perpendicular projections. Along with the main radiological sign of a destructive tuberculous cavity in the lung, additional radiological symptoms may be detected: the presence of a horizontal or meniscus-like fluid level within the lung tissue; signs of a draining bronchus, which becomes visible as a result of infiltration or sclerosis of its walls and, like a pointer, targets the location of the cavity in the lung; in some cases, in the absence of obvious X-ray bronchogenic dissemination, which indirectly indicate its presence. They are usually large, irregular in shape, without clear contours, sometimes of a draining nature, numerous and have a typical localization in the lungs. Such foci are located below the source of their formation and in greater numbers in the anterior (3rd, 4th, 5th) and lower (7th, 8th, 9th, 10th) segments, which are better ventilated when breathing. In the radiological picture of pulmonary tuberculosis in the decay phase, the initial form of the process dominates. Thus, in disseminated tuberculosis, the X-ray picture of the decay phase is characterized by the presence of foci of dissemination and one or more thin-walled, round, as if stamped. Tomogram of the lungs in direct projection with cavernous tuberculosis: a cavern is determined in the upper lobe of the left lung (indicated by an arrow), the surrounding lung tissue is not changed without perifocal inflammation. With focal tuberculosis in the decay phase, a

small, relatively round and thin ring—shaped shadow with separate foci in its composition or adjacent to its outer contour is usually determined against the background of limited polymorphic foci (the symptom of the "necklace" is an alterative cavity). As long as the cavity in the tuberculoma retains this appearance and occupies only a part of it, the process should be defined as a tuberculoma in the decay phase. Only after complete emptying and uniform thinning of the wall can it be considered as a cavity formed from tuberculoma. The X-ray picture of the cavernous form of pulmonary tuberculosis is characterized by the following signs: limited localization of the process, usually within one or two segments; absence of typical signs of the initial form of the disease; a formed cavern with well-emphasized external and internal contours of its walls, which can have different sizes, more often round or oval, with relatively thin or medium in thickness, but with uneven walls and the presence of moderate fibrosis and few compacted foci around it. In rare cases, several cavities are found in the cavernous form, but they correspond to the above signs. The cavernous form of tuberculosis is not always stable and may have different phases of the course. With its progression, the size of the cavity increases, perifocal infiltration appears around it or bronchogenic insemination occurs. The formation of fresh foci of bronchogenic dissemination is an indicator of the phase of insemination, which, with vigorous treatment, can be eliminated with complete resorption of foci, without the formation of fibrosis, but with the preservation of the cavity itself. However, more often the progression of the cavernous form of tuberculosis leads to the development of fibrous-cavernous pulmonary tuberculosis. The X-ray picture of the fibrous-cavernous form of the process is characterized not only and not so much by the formed cavernous, but also by the presence of pronounced and to varying degrees widespread fibrous and specific changes in the lungs. Bronchographic examination determines deforming bronchitis or bronchiectasis of varying severity and prevalence. With a volumetric decrease in individual segments and lobes, the mediastinal organs shift towards the lesion. The main method of treating patients with destructive tuberculosis is combined chemotherapy using 4-5 tuberculostatic drugs, taking into account the sensitivity of Mycobacterium tuberculosis. Antibacterial therapy at the first stage is usually carried out for 4-6 months, and after achieving favorable results — by an intermittent method until full clinical effect. In addition, various types of pathogenetic treatment are carried out. Currently, it is mandatory to use alternative methods of drug administration — intrapulmonary intracavitary and pericavitary administration of drugs. The valve is made of a rubber compound 52-336/4, indifferent to the human body, and is a hollow cylinder. The inner opening of the valve on the one hand has a smooth round shape, on the other it is made in the form of a falling lobe valve, which is locked by excessive external pressure due to its own elastic properties of the material. The valve is installed with both a rigid bronchoscope and a bronchofibroscope. The size of the valve depends on the localization of the tuberculous process, which is the source of bleeding, and the diameter of the draining bronchus, where the valve is installed (main, lobar, segmental), and should exceed the diameter of the bronchial lumen by 2-2.5 times. The criteria for including the valve in the treatment complex are drug resistance of Mycobacterium tuberculosis, exhaustion of the possibilities of chemotherapy and traditional collapse therapy for closing cavities and cavities. The advantages of this method are: the ability, in the absence of conditions for radical resection, to assess the functional reserves of the patient for the use of surgical methods of treatment; the ability to reduce cavities and achieve abacillation in unresectable patients. However, the extent of the lesion, the high frequency of multidrug resistance of Mycobacterium tuberculosis, severe, sometimes fatal pleuro-pulmonary and systemic complications, especially in patients with fibrotic-cavernous tuberculosis, present significant difficulties for both conservative and surgical treatment of destructive forms of pulmonary tuberculosis.

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