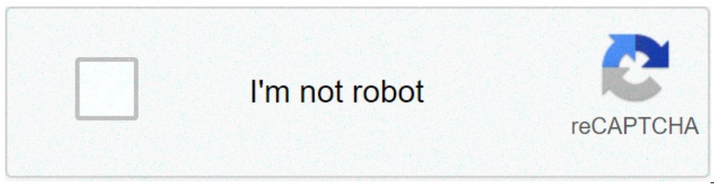

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Pathogenesis and etiology of recurrent varicose veins

Causes of bronchiectasis include: primary ciliary dyskinesia allergic bronchopulmonary aspergillosis Congenital anatomical defect connective tissue disorder A1-antitripsin (AAT) deficiency I idiopathic inflammatory disease Autosomal dominant polycystic renal traction from other processes of bronchiectasis can be the aftereffects of various necrotic infections that are poorly treated or not treated at all. Primary infection (i.e., without inherent defects or non-infectious exogenous insults) was a particularly common cause of bronchial disodia in developed countries before widespread use of antibiotics[14], and is still important in developing countries where antibiotics are used consistently. [15, 16] For typical unpleasant organisms known to cause bronchidiasis, the following [17,14]: Certain types of adenovirus infections with respiratory reticulum viruses in childhood, mycobacterium tuberculosis nontuberculous mycobacterium tuberculosis, can also cause bronchodylosis. Mycobacterium abium complex (MAC) infections deserve special mention. They tend to occur in human immunodeficiency virus (HIV) infection settings and in immune-capable hosts. [18] MAC infections have been observed, especially in women who are nonsmokers. I'm over 60 years old. Does not have known predisposition lung disorder; The application of phlegm in these cases is positive for acidic bacteria, and CT scans show findings of small regular nodles and bronchidrosis. [20, 21, 19] When patients develop bronchidia, many of these same organisms colonize damaged bronchi, which can cause continuous damage and episodic worsening. The most typically found organisms include hemophilus species (47-55% of patients) and pseudomonas species (18-26% of patients). [22, 23] Although not the main cause of bronchial dilation, P.aeruginosa often causes chronic bronchial infections in non-CF bronchidia patients through mechanisms with biofilm formation and release of pathogenic factors. This suggests that pseudomonas species may promote disease progression, and that infection with these species may be linked to worsening lung function and increased morbidity and mortality. [24] Post-occlusive bronchial diropathy can occur in many clinical settings (e.g., bronchial tumors, bronchial ishosis, bronchial stenosis due to infection, invasion of hiller lymph nodes, foreign body aspiration). Right middle lobe syndrome is a certain type of bronchial obstruction that can cause bronchial disodia. This is due to abnormal angulation of the rover bronchi at its origin, predisposition to blockages, subsequent infections, and the development of bronchidia. In adults, the desire of foreign d'ies is often carried out in a changed mental state setting, accompanied by un bitten food. The patient may also aspirat the chewed material.Contains food, digestive acids, microorganisms. After suction, pneumonia after blockage occurs, followed by focal bronchidiasis. Bronchiodyrosis can also develop in the setting of chronic aspiration. In addition, we recognize that the history of gastroesoesophobic refly is a risk factor for aspiration, and that helicobacter pylori in patients in this group may play a role in the development of bronchodilation. [25, 26, 27] CF is a multi-phylogenetic disorder that affects the chloride transport system in exocrine tissues and is primarily secondary to defects in cf permembrosis regulator (CFTR) proteins. CF and its variants are the most common causes of bronchidia in the United States and other developed countries. CF is an autosomal recessive disease that affects about 1 in 2,500 whites and 1 in 1 black person in the United States. In 2005, it was estimated that 10,000 U.S. adults had CF, or 40 percent of the total CF population. The risk to patients with multiple genetic variants of CF and genetic variant mutations remains to be elucidated. However, a reasonable assumption is that CF patients can be divided into two groups: (1) patients with classic diseases that are easily diagnosed based on clinical and laboratory data, and (2) patients with less severe diseases that appear later in life and have ambiguous genetic test results. [30, 31, 32] The main lung discovery of CF is bronchial disodia, which is an almost universal feature of the disease. It may be the only feature of CF in adults or those with genetic variations of the disease. Bronchiodyrosis associated with CF is thought to occur secondary to the mucus of the near respiratory tract and chronic lung infections, especially in mucosal P-greening. Young syndrome is clinically similar to CF and may represent a genetic variant of CF. [34] Young syndrome patients have bronchidia (often predominant in the lower lobe), sning and obstructive apermia. However, while the etiology of bronchial diration in these patients is thought to be similar to that of bronchial dilegation of CF, the diagnostic criterion for Young syndrome is electron microscopic analysis of the structure of cilia. Primary hermetic dyskinesi is a group of genetic disorders that can affect one in a population of 15,000 to 30,000 people. It appears by immutable or carcinogenic cilia and / or sperm. This can lead to poor mucus clearance, recurrent lung infections and eventually bronchidia. [35, 36] Variants of the condition were first described by Cartagener and encompassed a clinical triad of subsocia, nasal polyps or sinusitis and bronchidrosis in the setting of immutable cilia of the airways. [37] Allergic Bronchopulmonary Aspergillosisis an hypersensitivity reaction to inhaled aspergillus antigen characterized by immunological evidence of responses to bronchospasm, bronchidilosis, and aspergillus species. ABPA should be suspected in patients with productive coughs with a long history of asthma-type symptoms that do not respond to conventional treatment. Bronchial dilopathy is thought to be secondary to the difference of the outer trachea, including the aspergillus species of hifae. The resulting bronchidrosis is thin-fleshed and affects the central and medium airways. A CT scan of the chest shows the central airway of bronchidermia, distinguishing the condition from other causes of bronchidia. Other features of ABPA include eosinophilicity, increased immunoglobulin E (IgE) levels, and a dramatic response to therapeutic corticosteroids. Immunodeficiency conditions can be congenital or acquisition. The most common congenital conditions (albeit infrequently) are accompanied by B lymphocyte function. Hypoganglobulinemia in these cases can take one of the following forms [39,40,41, 42] : Immunoglobulin G (IgG) Subclass Deficiency X-Bound Aganglobulin Immunoglobulin A (IgA) Deficiency Immunoglobulin M (IgM) Deficiency Immunoglobulin E (IgE) Deficiency Gammaglobulin, which is usually present in childhood with hypoganglobulinemia It is important to establish a diagnosis, as it can reduce the number of infections and the resulting lung damage. HIV disease, along with the resulting gonth natural immunodeficiency syndrome (AIDS), is involved in the development of bronchidia, indicating accelerated bronchial injury that can occur from repeated infections in immunosuppressed patients. Bronchiodyrosis in HIV infection occurs before or without an obvious lung infection and can occur secondary from HIV disease itself to impaired immune function. [18, 43, 44] Bronchial disodia can result from various congenital anatomical defects. Isolation of the bronchopulmonary is a congenital abnormality that is classified in the eye or in the outer lobe, resulting in chronic lower respiratory tract infections that cause bronchidiasis. Williams-Campbell syndrome (congenital cartilage defect) is a lack of cartilage from the rover to the first- to second-generation sectional airways that results in widespread peripheral bronchidilation. [45] Meunier-Kuhn syndrome (tracheobronchoma) is a rare disease characterized by dileation of the trachea and compartmental bronchi (central bronchidiasis). [46] Swier-James syndrome (unilateral hyperjunctival lung) is likely a developmental disorder that causes unilateral bronchitis, hyperinflies, and in some cases bronchidilosis. Yellow nail syndrome is rare. It results in exudative trial exudate. [47] Bronchial disolosis has been noted to occur in this rare condition, bothIn patients with true AAT deficiency and in patients of the iskyrodia type. [48, 49, 50, 51] The etiology of bronchial dilatation in this setting is unknown, but it is believed that patients are susceptible to respiratory tract infections and subsequent bronchial injuries due to AAT abnormalities. Rheumatoid arthritis was associated with bronchial disodia in 3.2 to 35 percent of patients reported, and in one series, it was associated with an unfavorable prognosis. [55] Pathology of bronchidermia may increase susceptibility to infections in these patients. Lung disease may occur before the onset of the rheumatic process. Bronchiodyrosis is noted in patients with Sjogren's syndrome and can be secondary to an increase in the viscosity of mucus with poor airway clearance. [56] Ankylosing spondylitis is associated with bronchidylosis, but is a small number. [57] Phylogenetic erythematosis can present a variety of pulmonary pathologies, including bronchial dilopathy. [58] In recurrent polychondritis, bronchial dirosis appears to be secondary to primary bronchial injury with recurrent infection. In inflammatory bowel disease, bronchidia was seen in both ulcerative colitis and Crohn's disease. The etiology remains unknown. Lung symptoms may occur before the onset of intestinal diseases. Sarcoidosis can cause bronchial dilation by various mechanisms, including scarring of the bronchi, bronchial granulomatosis, or exogenous compression of the bronchi. Marfan syndrome is connective tissue disorder [61] The general consensus is that weakness of connective tissue in the bronchial wall predisposes to bronchial dilopathy. [62] Autosomal dominant polycystic nephropathy (ADPKD) patients have also been shown to have an increased incidence of bronchidia in radiation screening. ADPKD is another disease of so-called syriopathy, or a disease in which defects in ciliary function are the main pathological findings. [63] Traction bronchial diration is secondary airway distortion from bronchial fibrosis of the surrounding lungs to mechanical traction of the bronchi. In this situation the airways can expand, but other symptoms of bronchiodyrosis are lacking. Traction bronchodilopathy tends to have an upper lobe distribution in the case of radiation fibrosis and sarcoidosis, and the lower lobe is mainly involved in cases of intersodic pulmonary disease / idiopathic pulmonary fibrosis (ILD / IPF). Exposure to toxic gases can often cause irreversible damage to the bronchial airways and cystic bronchidrosis. Commonly involved drugs include chlorine gas and ammonia. Ammonia.

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