

Bronchiectasis etiological diagnosis recommendations

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ABSTRACT

In the past years, more diagnoses of bronchiectasis have been made for a variety of reasons. A meaningful percentage of patients' approaches and treatments, as well as their prognosis, were shown to shift in response to an etiological diagnosis, according to recent research. It is currently advised to conduct comprehensive research on the etiology, especially for those disorders that can be treated specifically. Given the difficulty of the etiological diagnosis, the Bronchiectasis Study Committee

of the Pulmonology Portuguese Society put together a working group that created a document to direct and standardize the etiological research using the material that was accessible and its own competence. Facilitating research, allocating resources wisely, and enhancing care delivery, patient quality of life, and prognosis are the objectives.

Key Words: *Etiological; Undernutrition; Bronchiectasis, Interventional pulmonology*

INTRODUCTION

The disease Bronchiectasis (BE) is characterized by increasing bronchial damage, recurrent infection, and irreversible airway dilatation and lesion. Due to the fact that this pathology's frequency had not diminished in response to better treatment of respiratory infections as would be anticipated, there have been many indications of a resurgence in interest in it during the past few years. This finding is mostly attributable to improved diagnostic capabilities, awareness of the link between BE and specific systemic illnesses, and rising population survival rates, particularly for chronic patients. As a result, BE epidemiological characteristics have changed, with fewer occurrences of post-infectious BE, more adult patients, and more cases linked to other common disorders including Chronic Obstructive Pulmonary Disease (COPD). BE can be brought on by a number of regional, systemic, genetic, or acquired disorders. Even after a thorough investigation, the etiology cannot be determined in a sizable majority of instances. As a result, the percentage of diagnoses acquired in published series varies greatly. It's common to believe that getting an etiologic diagnosis is pointless and expensive. However, there are a number of reasons to look into the etiology, a better prognosis is related to the precise treatments given for individual disorders that cause BE. The prognosis of the underlying disease is

made worse by BE, which also greatly lowers quality of life and speeds up the loss of pulmonary function. It's crucial to diagnose inherited conditions in order to determine the risk of transmission and provide genetic counselling. BE is a complicated, diverse, and perhaps multifaceted phenomenon. After BE has been identified, a thorough etiologic research should be conducted. Based on the material that was accessible and its own experience, the working group established by the Pulmonology Portuguese Society Bronchiectasis Study Group created a statement to direct and standardize the etiologic research. The objective is to better care delivery, quality of life, and prognosis for patients with BE while rationalizing resource use. A systematic analysis of the underlying etiology carried out in accordance with a predetermined protocol improves resource management and increases the likelihood of making a diagnosis. The initial assessment must be driven by a thorough clinical history and physical examination in order to identify the most plausible hypotheses and conduct the most efficient testing. A thorough clinical history should cover the patient's age at onset, symptoms at presentation, clinical evolution, previously identified illnesses, risk factors, history of infertility, non-respiratory symptoms, and family history, including consanguinity information. These inquiries should be made methodically since patients might not value some clinical

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information if it does not materially affect their quality of life or if they are unaware of its significance to the diagnosis. When symptoms appear following a major infection, it is typical to presume that the diagnosis is post-infectious BE. However, there are instances where symptoms don't show up until years later, after the presence of another predisposing condition has been confirmed (for instance, some degree of immunodeficiency). However, one should inquire about any respiratory symptoms the patient may have experienced before to the viral episode. Therefore, even if infectious diseases are still a common cause of BE, caution should be used when making the diagnosis of post-infectious BE, particularly when upper airway symptoms, non-respiratory symptoms, or other systemic diseases have already been identified. It is crucial to stay alert to new clinical information throughout the follow-up period in case etiology is still unclear despite thorough inquiry. This could need a reevaluation. BE may occur before the diagnosis of various systemic illnesses, including inflammatory bowel disease and rheumatoid arthritis. There might be a gradual progression in other circumstances. The examination must be thorough in some circumstances where etiologic diagnoses are thought to be precluded. Only those causes of BE that now benefit from specialized monitoring and treatment strategies with prognostic implications have been mentioned because it would be impossible to discuss the investigation of all BE causes in this paper. As a powerful inhibitor of neutrophil elastase, Alpha-1-Antitrypsin (AAT), a glycoprotein mostly generated in the liver, works to protect the lung against proteolytic injury. AAT Deficiency (AATD) is characterized by a decreased serum level of AAT and/or the discovery of a faulty genotype. The only known genetic risk factor for COPD, AATD also increases the likelihood of developing severe emphysema. Additionally, it can result in panniculitis, vasculitis, and, less frequently, liver damage.

When making a clinical diagnosis, the significant overlap between AATD and COPD is a confounding factor. In AATD patients, dyspnea, coughing, sputum expectoration, wheezing, and recurrent exacerbations are the most typical respiratory symptoms. The following are some distinctive and suggestive characteristics of the emphysema connected to AATD: disproportionate involvement of the lung bases in emphysema despite these indicative similarities, there are no clinical traits that can reliably separate COPD from AATD. Based on laboratory results, AATD is diagnosed. AAT genotyping, AAT protein phenotyping of serum or plasma, and measurement of serum or plasma protein level are the three methods that are frequently used to diagnose AATD. It is advised to conduct additional testing using protein phenotyping or genotyping if the AAT protein level is below the normal range. Serum concentrations of AAT, an acute-phase protein, can only be relied upon for diagnostic testing if there is no inflammation present at the time the blood sample is taken. The Alpha Kit Quick Screen, a brand-new diagnostic instrument, can quickly and accurately identify the Z protein in a blood sample. It is a screening test that may be done during a clinic visit and has results available (a positive result suggests the need for phenotyping or genotyping. An uncommon, heterogeneous autosomal recessive genetic condition with an estimated frequency of births is Primary Ciliary Dyskinesia (PCD). It has a wide clinical spectrum, is defined by aberrant ciliary ultrastructure or function, and frequently involves recurrent respiratory infections because of insufficient mucociliary clearance.

The pattern of inheritance is typically autosomal recessive. The age range at presentation is from infancy through maturity. Age-appropriate symptoms include: during the infant stage; lacking any evident risk factors, pneumonia in newborns or respiratory distress, rhinorrhea that has persisted since the first day of life, Laterality disorders, complex congenital heart disease, heterodoxy, hydrocephalus, cleft palate, bilateral cervical ribs, biliary atresia, esophageal abnormalities, anal atresia, and other conditions are associated with each other. The younger youngsters and older children, Recurrent upper and lower respiratory infections, chronic rhino sinusitis, and a persistent productive cough. In actual practice, disorders with similar traits (such as CF and immunodeficiency) should be avoided by patients whose clinical presentation suggests PCD. Confirmatory diagnostic testing is necessary for any positive screening test in order to show that the cilia have an aberrant structure or function. Before undertaking measurements, the screening test and the diagnostic test should be carried out on clinically stable patients who have not experienced an acute upper or lower respiratory infection in at least a few weeks. When a patient has a consanguineous history or is a newborn or infant, a lower screening threshold should be taken into account. In adults, if BE is not present, the PCD diagnosis should be questioned. According to a defined procedure, patients with BE and clinical signs of PCD should be tested using a nasal NO assay. To confirm the diagnosis, a ciliary structure study using electron microscopy or ciliary function analysis using video recording should be used. The initial diagnostic tests shouldn't include genetic testing. A non-invasive condition known as Allergic Bronchopulmonary Aspergillus (ABPA) is brought on by an intolerance to the spores of the common fungus *Aspergillus fumigatus*. Almost invariably, patients with CF or asthma will also have ABPA. According to estimates, ABPA can develop in CF patients with asthma, although it is still underdiagnosed, with a mean diagnostic delay of years following the onset of symptoms. Occasionally, ABPA may exacerbate certain chronic lung conditions, such as idiopathic BE or due to other causes. When it comes to Caucasians, CF is the most prevalent autosomal recessive illness. It is brought on by mutations in the Cystic Fibrosis Transmembrane Conductance Regulator (CFTR) gene, which impairs the CFTR protein's ability to regulate chloride and sodium transport in secretory epithelial cells. This results in abnormal ion concentrations across the apical membranes of these cells. Multisystem illness is CF. It is linked to a significant number of mutations that lead to various forms of CFTR protein malfunction and, as a result, to a great deal of clinical diversity. A clinical entity that is also linked to CFTR malfunction but does not meet all the requirements for the diagnosis of CF is referred to as a "CFTR-related illness." These phenotypes are best represented by three key clinical entities. Other than the *Mycobacterium tuberculosis* complex and *Mycobacterium leprae*, a vast array of mycobacterial species with a wide range of pathogenicity are responsible for Nontuberculous Mycobacterial (NTM) infections. NTM are common bacilli that live primarily in soil and water. In this situation, there is continuous exposure to these microbes. According to the period of time of growth in culture, the typical classification of NTM is established. *Mycobacterium avium* complex is the most frequent cause of lung NTM infection due to the slower-growing, more pathogenic NTM. Fast-growing NTM can cause a disease that is more difficult to treat and has a worse prognosis than *Mycobacterium*

abscesses complex due to its less aggressive range of organisms. The most frequent clinical manifestation of NTM infections is pulmonary involvement. The chronic progressive type and, in rare cases, hypersensitivity pneumonitis, are the two most common presentations. Patients with pre-existing structural abnormalities characterized by BE and/or cavitation's originating from prior lung diseases, particularly in elderly males, are two significant patterns that can be distinguished in the chronic clinical presentation of Nontuberculous Mycobacterial Pulmonary Disease (NTM-PD). Patients in non-smoking women with slow-evolving multifocal nodular BE and no underlying lung disease Respiratory symptoms like a persistent cough, frequently purulent sputum, occasionally hemoptysis, dyspnea, and systemic symptoms like asthenia, weight loss, and intermittent fever are typical presenting signs of NTM-PD. A cause-and-effect cycle that contributes to escalating structural damage to the airways and advancing clinical deterioration is maintained by BE's association with NTM-PD as both a contributing factor to the disease and a symptom of it. The host must typically have some degree of local or systemic immune weakness given the NTM's poor pathogenicity in order to cause sickness. Although not yet clinically validated, the ATS/IDSA recommended diagnostic criteria for NTM-PD, which include the following, exclude other diseases with a similar clinical presentation and imaging results. Patients who have a strong suspicion of having NTM-PD but do not meet the prerequisites should be monitored until this diagnosis is firmly established or eliminated. NTM infections as a whole have dramatically grown in recent years. However, there is a lot of variation in terms of isolated species and prevalence between different regions. Infection with NTM and the prevalence of tuberculosis are inversely correlated. In this regard, there has been a substantial rise in NTM sickness in regions with low tuberculosis incidence. GERD was recognized as a causative component in patients when many BE etiological aspects were taken into account. In the Western world, GERD prevalence has been observed to vary. In a recent observational prospective study, it was discovered that the prevalence of proximal and distal gastric reflux in COPD and BE patients was significantly higher than that of a control group. Clinically silent reflux was the most common kind of GERD in BE.