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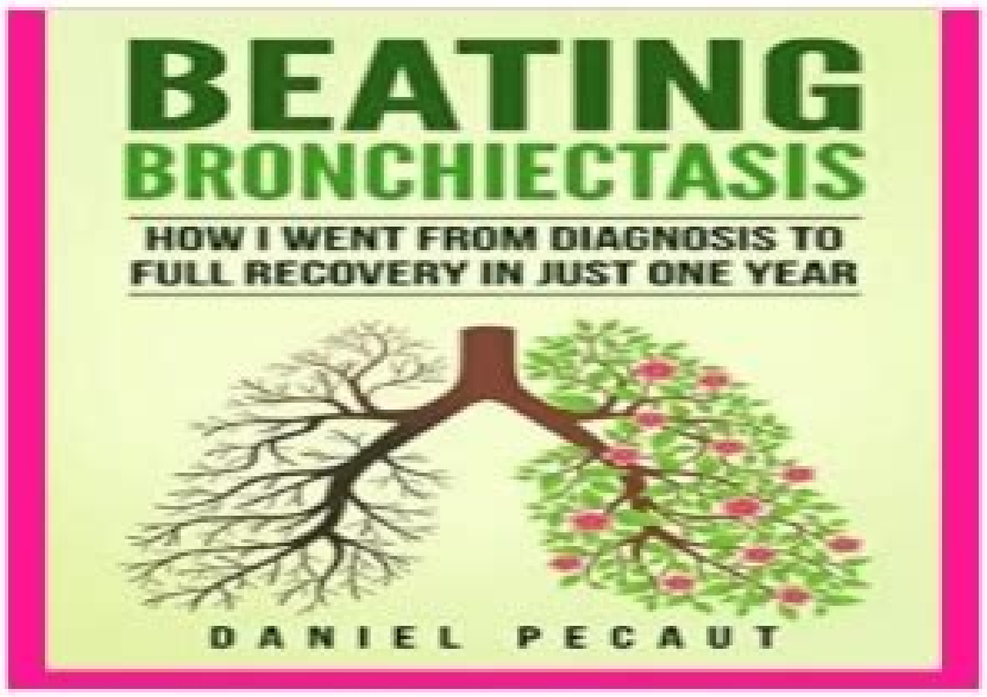

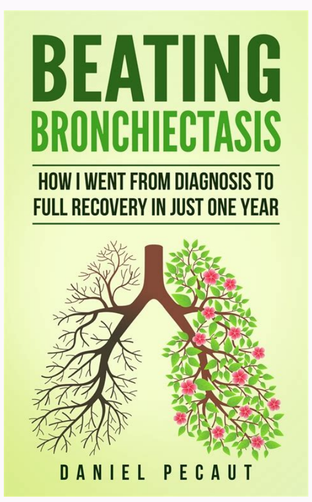

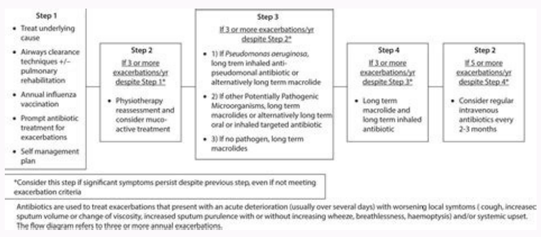

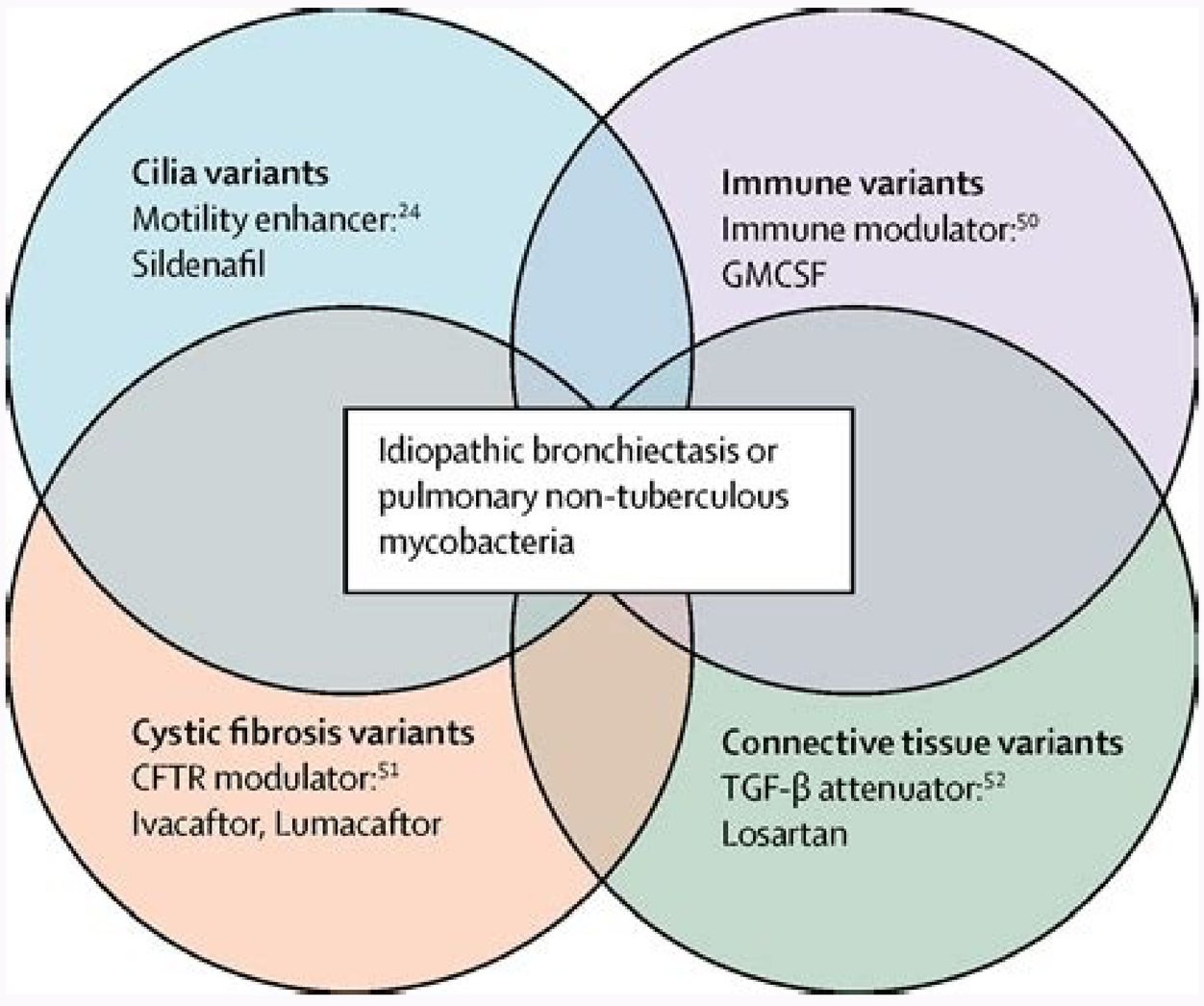

Beating bronchiectasis book. Beating bronchiectasis daniel pecaut. What helps bronchiectasis. Can you recover from bronchiectasis. What makes bronchiectasis worse. How do you prevent bronchiectasis from getting worse.

Medically reviewed by Adithya Cattamanchi, M.D., Pulmonology — Written by Carmella Wint and Matthew Solan — Updated on April 27, 2022SymptomsCausesRisk factorsDiagnosisPreventionTreatmentOutlookBronchiectasis is a condition where the bronchial tubes of your lungs are permanently damaged, widened, and thickened. These damaged air passages allow bacteria and mucus to build up and pool in your lungs. This results in frequent infections and blockages of the airways.There’s no cure for bronchiectasis, but it’s manageable. With treatment, you can usually live a typical life. However, flare-ups must be treated quickly to maintain oxygen flow to the rest of your body and prevent further lung damage. Read on for more information about bronchiectasis and its symptoms, causes, treatment, and more.Share on PinterestThe left image is a bronchus without bronchiectasis compared with an image of bronchiectasis. Illustration by Maya ChastainThe risk of developing bronchiectasis increases with age, though younger people can have it. Women are more likely to have it than men.Other health conditions that can put you at risk of having bronchiectasis include:A chest computed tomography (CT) scan is the most common test for diagnosing bronchiectasis because a chest X-ray does not provide enough detail.This painless test creates precise pictures of your airways and other structures in your chest. A chest CT scan can show the extent and location of lung damage. After bronchiectasis is confirmed with the chest CT scan, your doctor will try to establish the cause of the bronchiectasis based on your history and physical exam findings. It’s important to find out the exact cause so the clinician can treat the underlying disorder to prevent the bronchiectasis from getting worse. There are numerous causes that can induce or contribute to bronchiectasis. The evaluation for the underlying cause mainly consists of laboratory and microbiologic testing and pulmonary function testing.Your initial evaluation will likely include:If your doctor suspects CF, they’ll order a sweat chloride test or genetic test.The exact cause of bronchiectasis is unknown in about 50 percent of the cases of non-CF bronchiectasis.For others, it’s related to atypical genetic characteristics and other medical conditions that affect the lungs. Ways to prevent bronchiectasis include:not smoking tobacco products or quitting smokingavoiding polluted airgetting your vaccination against the flu, whooping cough, pneumonia, measles, and COVID-19taking antibiotics to prevent and treat infections if you have conditions that put you at risk (studies are currently being done on new formulations of inhaled antibiotics)When the cause is unknown, prevention may be challenging. Early recognition of bronchiectasis is important so that you can get treatment before significant lung damage occurs.There’s no cure for bronchiectasis in general, but treatment is important to help you manage the condition. The main goal of treatment is to keep infections and bronchial secretions manageable. The most common treatment options for bronchiectasis include:Chest physiotherapy. One form of chest physiotherapy is a high frequency chest wall oscillation vest to help clear your lungs of mucus. The vest gently compresses and releases your chest, creating the same effect as a cough. This dislodges mucus from the walls of the bronchial tubes.Surgery. If there’s bleeding in your lung, or if the bronchiectasis is only in one part of your lung, you may need surgery to remove the affected area.Draining secretions. Another part of daily treatment involves draining the bronchial secretions, aided by gravity. A respiratory therapist can teach you techniques to aid in coughing up the excess mucus.Treating underlying conditions. If conditions like immune disorders or COPD are causing your bronchiectasis, your doctor will also treat those conditions.Lifestyle changes. Things like exercise, eating a healthy diet, and drinking plenty of water may help improve the symptoms of bronchiectasis.The outlook for people with bronchiectasis depends on the severity of the condition and what is causing it.Bronchiectasis affects 350,000 to 500,000 people in the U.S. While severe bronchiectasis can be fatal, individuals with the non-CF type generally have a good outlook with treatment.Early diagnosis is important so that treatment can begin and additional lung damage can be prevented.Read this article in Spanish. 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He used to suffer from frequent cough and cold since childhood. Clinical examination revealed bilateral coarse basal crepitations and rhonchi. His apex beat was on right 5th intercostal space in mid-clavicular line. Investigation revealed situs inversus, bi-lateral bronchiectasis, and chronic sinusitis. His semen analysis revealed the complete absence of sperm. The Saccharin test revealed impaired nasal ciliary movement. Considering all the finding, he was diagnosed as a case of Kartagener's syndrome. We are reporting this case because of its rarity and rare presence of aspermia in Kartagener's syndrome.Keywords: Aspermia, bronchiectasis, dextrocardia, Kartagener's syndrome, sinusitisKartagener's syndrome (KS) is an important as well as rare subgroup of primary ciliary dyskinesia (PCD). In KS, defective ciliary movement results in sinusitis, bronchiectasis, and dextrocardia. During the embryonic stage, organ position is determined by uniform ciliary beating but in KS, due to ciliary dysmotility heart along with the other organs fail to move on to the left side, resulting in dextrocardia and situs inversus. These patients usually present with repeated lower respiratory infection in childhood leading to bronchiectasis and infertility in adulthood. Utmost care is needed during surgery in KS. Here, we are reporting a case of KS, who presented to us with cough and breathlessness.A 16-year-old boy presented with 3 months dry cough with intermittent breathlessness. He had no fever or chest pain or hemoptysis. Cough and breathlessness were not associated with any postural or diurnal variation. He used to suffer from frequent cough and cold since childhood. No family history of asthma or atopy was present. Clinical examination revealed normal physical development with mild pallor and digital clubbing. His apex beat was palpable on the right 5th intercostal space in mid-clavicular line on palpation otherwise inspection and percussion findings were normal. Chest auscultation revealed bi-lateral polyphonic rhonchi and basal coarse crepitations with clearly audible heart sounds on the right side. Cardio-vascular examination revealed right-sided 1st heart sound without any murmur. Other system examinations were normal.Chest X-ray revealed dextrocardia with normal lung field [Figure 1]. Sputum smear examination for acid fast bacillus was negative and aerobic culture showed growth of Staphylococcus aureus. A high-resolution computerized tomography (HRCT) scan of thorax revealed bronchiectasis in lingul [Figure 2]. X-ray of paranasal sinuses revealed bi-lateral maxillary sinusitis with the absence of both frontal sinuses [Figure 3]. Ultrasonography of the abdomen revealed complete situs inversus. Observing the presence of bronchiectasis, sinusitis, and dextrocardia, his semen analysis was done and it revealed complete aspermia with normal seminal fluid content. Fine needle aspiration cytology (FNAC) of the both testes revealed the presence of normal mature sperm, which indicated the inability of the sperm to be transported to the seminal fluid. The Saccharin test revealed delayed movement (35 minutes) of the particle toward oropharynx, which was indicative of impaired nasal ciliary movement. Echocardiography showed dextrocardia with normal atrioventricular concordance without any structural abnormality. Routine blood count was within normal range. The pulmonary function test was of mixed pattern with significant bronchodilator reversibility.He improved significantly after treatment with intravenous 1.2 g co-amoxyclav three times daily, oral 600 mg linezolid twice daily along with nebulized salbutamol and budesonide for 2 weeks.PCD also known as immotile cilia syndrome is a very rare disorder, found 1 in 26,000-40,000 live births. But probably this is an underestimation because of misdiagnosis.[1] KS and Young's syndrome are the two important variants of PCD.[2] The prevalence of KS is around 1 in 60,000 populations,[3] more common among people with consanguineous marriages. Diagnostic triad of KS is bronchiectasis, chronic sinusitis, and situs inversus. Siewert first described the above combination in 1904 and later Manes Kartagener identified this clinical triad as a distinct congenital syndrome in 1933.[2] It is also known as Afzelius’ syndrome, Kartagener's triad, Siewert's syndrome, dextrocardia–bronchiectasis–sinusitis syndrome. It is inherited via an autosomal recessive pattern with incomplete penetrance.[4] All the defects develop due to defective ciliary movement. Among the many ciliary ultrastructural defects, the absence of one or both rows of dynein arms and the absence of spoke heads or central sheath are commonly

seen in KS.[4] These dynein arms are ATPase radial projections, two of which normally arise from each of nine pairs of microtubules that run longitudinally and which are arranged circumferentially around the cilia. Ciliary bending occurs when outer microtubules slide over the inner microtubules in a coordinated fashion. Here, the energy is supplied by the dynein arms. This microtubular shortening initiates bending motion because the outer pairs of microtubules are tethered to each other and to the central sheath. Because of the same ultrastructural plane of symmetry, all cilia work in a coordinated fashion to propel the mucus blanket in a cephalic direction. Such coordinated sliding and bending cannot occur either in the absence of dynein arms or radial spokes,[5] or in the presence of spatial transposition of microtubules[6] or in the presence of random ciliary orientation in which the cilia are anchored in disorganized fashion at the cell surface.[7] Because of the impaired muco-ciliary clearance, a patient frequently suffers from repeated cough and cold since childhood leading to chronic sinusitis. Recurrent lower respiratory tract infection leads to bronchiectasis. An adult person commonly seeks medical help because of infertility. A patient may have dextrocardia because of disorganized ciliary beating, which results in random rotation of the primitive organ precursors to either left or right. For this reason, dextrocardia is not found in all cases of KS. Situs inversus, nasal polyps, rhinitis, corneal abnormalities, and conductive deafness are the other abnormalities found in KS.[8] However, in our case, this adolescent boy came to us because of his recurrent chest infection and breathlessness, which was due to bronchiectasis.Dextrocardia, hyperinflation of lungs, bronchial wall thickening, peribronchial infiltrates are found in chest X-ray though bronchiectasis and paranasal sinuses are better visualized by a CT scan.[9] Semen analysis of postpubertal males may reveal either abnormal sperm motility or aspermia. The Saccharin test is also used for diagnosis. A 0.5 mm saccharin particle is placed near inferior turbinate and its sweet test is felt in mouth within 30-60 minutes. Measuring exhaled nasal nitric oxide is a good screening test for immotile-cilia syndrome with a good negative predictive value.[10] Pulmonary function tests usually reveal an obstructive pattern in spirometry with a variable bronchodilator response. Static lung volumes may show hyperinflation. Gold-standard investigation is electron microscopic examination of cilia. Brush cytology from tracheal or bronchial mucus membrane provides the best specimen but cilia of the nasal epithelium are easy to collect. Sperm tail is the alternative good source of specimen. Video or electron microscopy of cilia for ciliary movement, beat frequency, co-ordination, and amplitude is commonly examined. In future, genetic testing will be the principal means of establishing the diagnosis.[11]Haemophilus influenza, Staphylococcus aureus, and Pseudomonas species commonly colonize the bronchial tree of PCD patients. Antibiotics, intravenous or oral, intermittent or continuous, are used to treat upper and lower respiratory tract infections. In children, long-term low dose prophylactic antibiotics are sometimes necessary. Patients are also treated with inhaled bronchodilators inhaled corticosteroids, mucolytics, and chest physiotherapy. Influenza and pneumococcal vaccination should be routinely given.Kartagener's syndrome will always be kept in mind in young patients with bronchiectasis and when Kartagener's syndrome is diagnosed, always look for the rare presence of aspermia.Source of Support: NilConflict of Interest: None declared.1. Kuehni CE, Frischer T, Strippoli MP, Maurer E, Bush A, Nielsen KG, et al. Factors influencing age at diagnosis of primary ciliary dyskinesia in European children. Eur Respir J. 2010;36:1248–58. [PubMed] [Google Scholar]2. Kartagener M. Zur pathogenese der bronchiectasien. I Mitteilung: Bronchiectasien bei situs viscerum inversus. Betr Klin Tuberk. 1933;83:498–501. [Google Scholar]3. Boat TF, Carson JL. Ciliary dysmorphology and dysfunction: Primary or acquired? N Engl J Med. 1990;323:1700–2. [PubMed] [Google Scholar]4. Afzelius BA. Immotile Cilia syndrome: Past, present and prospects for the future. Thorax. 1998;53:894–7. [PMC free article] [PubMed] [Google Scholar]5. 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