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CLINICAL REVIEW

Treatment of bronchiectasis in adults

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Patients with bronchiectasis usually need lifelong medical support from their doctor, especially given the frequent episodes of infection. This article focuses on the treatment of bronchiectasis in adults and does not include a discussion of bronchiectasis caused by cystic fibrosis. The prevalence of bronchiectasis not linked to cystic fibrosis is unclear, but every general practitioner in the United Kingdom probably has a few patients.

How do we diagnose bronchiectasis?

Bronchiectasis refers to the permanent abnormal dilatation of the central and medium sized bronchi as a result of a vicious cycle of transmural infection and inflammation with mediator release.[1] Symptoms include chronic productive cough, wheeze, and dyspnoea. Infective exacerbations are associated with worsening of symptoms and signs of pneumonia. Haemoptysis can occur, but amounts of blood are usually small, and serious haemoptysis requiring selective arteriography and embolisation or surgery is rare.

The most frequently used classification system distinguishes between cylindrical, varicose, and saccular or cystic bronchiectasis.[w1] Although insightful, this classification has no clinical or therapeutic uses. A modern clinical definition includes the daily production of mucopurulent phlegm and chest imaging that demonstrates dilated and thickened airways.[w2] The clinical suspicion of bronchiectasis can be confirmed by high resolution computed tomography. Characteristic findings include internal bronchial diameters greater than that of the adjacent pulmonary artery, lack of bronchial tapering, presence of bronchi within 1 cm of the costal pleura, presence of bronchi abutting the mediastinal pleura, and bronchial wall thickening.[w3]

A diagnosis of bronchiectasis should prompt an investigation of possible causes and associated conditions (table 1), some of which can be treated.[2] Extrinsic factors, particularly childhood respiratory infections, were an important cause of permanent bronchial damage in the past. These days, especially in Western countries with early immunisation and widespread use of antibiotics, post-infectious damage is a less prominent cause of the disease, and intrinsic defects are more common causes. A study of 150 adults with bronchiectasis in the UK found that 53% of cases were idiopathic; 29% were post-infectious; 8% were caused by an immune defect, 7% by allergic bronchopulmonary aspergillosis, 4% by aspiration, 3% by Young's syndrome, 3% by cystic fibrosis, 3% by rheumatoid arthritis, 1.5% by ciliary dysfunction, and <1% by miscellaneous causes.[3]

How is it treated?

Bronchiectasis can be treated by pharmacological and non-pharmacological means (tables 2 and 3).[w4] The box shows an arbitrary step-up scheme that takes into account the level of evidence and safety risks.

Antibiotics

Antibiotics are used to treat acute exacerbations, to prevent exacerbations, or to reduce the bacterial burden. In general, the outcome of treatment with antibiotics depends on the severity of the disease. In mild to moderate bronchiectasis the infection can be completely eradicated, whereas in severe disease the bronchial tree remains chronically colonised. Antibiotics with a high penetrance (macrolides, azalides, and quinolones) are recommended in severe cases because high concentrations of bacteria are located intraluminally in association with mucus, and because thickening and scarring of the bronchial wall may reduce local bioavailability. *Pseudomonas aeruginosa* is inherently resistant to most antibiotics at concentrations that are achieved in vivo. The organism is susceptible to (oral) quinolones,[2] but after one or two courses resistance may develop. Patients who are clinically unwell or do not respond to oral antibiotics should be admitted to hospital and given intravenous antibiotics.

Patients who relapse quickly might need prophylactic antibiotics. Three strategies have been described—a high oral dose for a prolonged period (at least four weeks), aerosolised antibiotics (for example, during alternate months), or regular pulsed courses of intravenous antibiotics (for example, two to three week courses with one or two months in between).

Suggested step-up scheme for treating bronchiectasis

Step 1: Inhaled fluticasone (500 µg twice daily)
Step 2: Physical therapy including exercise training
Step 3: Prolonged use of oral antibiotics. The choice of antibiotic should depend on sputum culture results, with a preference for clarithromycin

Web references w1-w22 are on bmj.com

Prolonged use of oral antibiotics for purulent bronchiectasis was investigated in a recent meta-analysis. It identified six trials that included 302 patients.[5] The results of five studies were positive in terms of parameters pertaining to sputum, such as volume or purulence. However, no positive effects were seen on rates of exacerbation, lung function, or death. This contradicts the findings of a recent controlled study that investigated 500 mg of azithromycin given twice a week for six months in 30 patients.[6] Pulmonary function tests did not change, but azithromycin significantly decreased the incidence of exacerbations compared with usual care (5 *v* 16). Inhalation of nebulised antibiotics might be a better route of administration because of its favourable benefit-risk ratio. Gentamicin 40 mg inhaled twice daily for three days improved sputum production, sputum neutrophil activity, airway obstruction, exercise capacity, and nocturnal desaturation compared with placebo.[7] Tobramycin 300 mg inhaled twice daily for four weeks eradicated *P auruginosa* in 35% of patients and improved the medical condition of 62%.[8] Ceftazidime 1 g and tobramycin 100 mg inhaled twice daily over 12 months decreased the number of hospital admissions and inpatient days.[9]

Flu vaccinations reduced the number of flu related exacerbations in chronic obstructive pulmonary disease,[w5] so vaccination might have a beneficial effect in bronchiectasis. However, randomised controlled trials of vaccination in patients with bronchiectasis are lacking.

Mucolytics

Mucolytics target hypersecretion or the physiochemical characteristics of sputum seen in bronchiectasis. They aim to improve tracheobronchial clearance. In one study, oral bromhexine (30 mg three times daily) was added to an antibiotic during acute infective exacerbations. It improved expectoration, the quantity and quality of sputum, and auscultatory findings.[10] In another study, nebulised recombinant human DNase I (2.5 mg once or twice a day for two weeks) was given to patients with stable bronchiectasis. The study found no improvements in spirometry, quality of life, dyspnoea, safety, or ciliary transportability of the sputum.[11] In a large placebo controlled study, 349 patients with idiopathic bronchiectasis in a stable condition inhaled 2.5 mg recombinant human DNase I for 24 weeks. Pulmonary exacerbations were more frequent and the decline in FEV_1 (forced expiratory volume in one second) was greater in patients who received DNase than in controls. These results contrast greatly with the positive effects of this drug in cystic fibrosis.[12]

Inhaled mannitol may improve impaired mucociliary clearance by inducing an influx of fluid into the airways, and thereby changing the mucous rheology. One study found that mucociliary clearance was doubled in the central and intermediate lung regions of 11 patients with bronchiectasis directly after inhaling 300 mg mannitol.[w6] A follow-up study showed that these effects lasted for at least 24 hours.[w7] Inhalation of 400 mg mannitol once daily for 12 days improved the tenacity and hydration of sputum, as well as health status.[w8] Mannitol is therefore a promising new drug, especially as it is easier and more hygienic to inhale a dry powder than to use a nebuliser. Large randomised controlled studies are needed, however, to establish its efficacy and tolerability during long term administration.[13]

Anti-inflammatory agents

Inhaled corticosteroids may reduce inflammation and improve airway obstruction, as they do in asthma. To date, three randomised controlled studies have investigated this hypothesis.[w9-w11] Inhaled fluticasone decreased the density of leucocytes and concentrations of the inflammatory mediators interleukin 1β, interleukin 8, and leukotriene B4 in sputum.[w11] However, a systematic review found no significant improvements in lung function.[14] Recently, a large study was published in which 86 patients inhaled fluticasone 500 μg twice daily or placebo for 12 months.[15] Twenty four hour sputum volume improved significantly in patients treated with fluticasone, but no change was seen in the frequency of exacerbations, the purulence of sputum, or lung function. Systemic corticosteroids may be better at penetrating the bronchial wall and therefore be more effective, but data from randomised controlled trials are available only for bronchiectasis associated with cystic fibrosis.[16]

Leukotriene receptor antagonists are a new class of drugs that is effective in the treatment of asthma. These drugs may be of benefit in bronchiectasis because they inhibit neutrophil mediated inflammation. To date, no randomised controlled trials of these agents have been published, however.[17]

Non-steroidal anti-inflammatory drugs may also be of use because they inhibit neutrophil function and the release of neutrophil elastase. In a non-controlled open study, eight patients with stable bronchiectasis

Table 1 | Causes of bronchiectasis and associated conditions[2]

Cause	Details or associated conditions
Post-infectious damage	Tuberculosis, whooping cough, non-tuberculous mycobacteria
Mechanical obstruction	Intrinsic (tumour or foreign body), extrinsic (lymph node)
Congenital	Defective bronchial wall, pulmonary sequestration
Inflammatory pneumonitis	Aspiration of gastric contents, inhalation of toxic gases
Excessive immune response	Allergic bronchopulmonary aspergillosis, lung transplant rejection, chronic graft versus host disease
Abnormal mucous clearance	Primary ciliary dyskinesia, cystic fibrosis, Young's syndrome
Fibrosis	Cryptogenic fibrosing alveolitis, sarcoidosis
Diffuse panbronchiolitis	Predominantly seen in Japanese patients
Deficient immune response	Hypogammaglobulinaemia, human immunodeficiency
Infertility	Cystic fibrosis, Young's syndrome, primary ciliary dyskinesia
Inflammatory bowel disease	Ulcerative colitis, Crohn's disease, coeliac disease
Connective tissue disease	Rheumatoid arthritis, systemic lupus erythematosus
Malignancy	Acute or chronic lymphatic leukaemia
Yellow nail syndrome	Discoloured nails, lymphoedema, pleural effusions
α1 antiproteinase deficiency	More commonly causes emphysema
Mercury poisoning	May cause Young's syndrome

received 25 mg oral indometacin three times daily for four weeks.[w12] Indometacin had a pronounced effect on two of the three neutrophil functions studied—neutrophil chemotaxis and fibronectin degradation were inhibited by more than 50%. However, lung inflammation, sputum volume, and sputum quality did not change. Interestingly, high dose ibuprofen taken consistently for four years significantly slowed the progression of lung disease in patients with cystic fibrosis.[w13] Ibuprofen has not yet been investigated in non-cystic fibrosis bronchiectasis.

Macrolides suppress inflammation—independent of their antimicrobial actions—in airway diseases like asthma, chronic obstructive pulmonary disease, cystic fibrosis, and diffuse panbronchiolitis. Indeed, macrolides improved clinical status and lung function in a few small studies of bronchiectasis.[w14-w18] In addition, a randomised placebo controlled trial in children showed that three months of treatment with clarithromycin decreased the total number of leucocytes, proportion of neutrophils, and the concentration of interleukin 8 in bronchoalveolar lavage fluid.[w18] Because bacterial growth was not eliminated the authors suggested that direct anti-inflammatory effects of clarithromycin were responsible. This contradicts a randomised placebo controlled study in adults, however, which showed that eight weeks of low dose erythromycin did not reduce the number of leucocytes or the concentrations of interleukin 1α, interleukin 8, tumour necrosis factor α, or leukotriene B4 in sputum.[w15]

Table 2 | Pharmacological and non-pharmacological treatments for stable bronchiectasis

Treatment	Level of evidence	Grade of recommendation
Drugs		
Antibiotics:		
Prolonged use of oral antibiotics	1++	A
Aerosolised antibiotics	1+	A
Regular pulsed courses of intravenous antibiotics	4	D
Flu vaccination	1−	C
Mucolytics:		
Bromhexine*	1+	B
N-acetylcysteine	?	?
Recombinant human DNase aerosol	1+	A†
Mannitol inhalation powder	2++	B
Anti-inflammatory or immunomodulating drugs:		
Oral corticosteroids	2+	D
Inhaled corticosteroids	1+	B
Oral leukotriene receptor antagonists	4	D
Indometacin or ibuprofen	2−	D
Macrolides (clarithromycin)	1+	B
Flu vaccinination	2+	C
Bronchodilators:		
Short acting β2 adrenergic agonists	2+	D
Long acting β2 adrenergic agonists	4	D
Short acting anticholinergics	2+	D
Long acting anticholinergics	4	D
Methylxanthines	4	D
Non-pharmacological treatments		
Bronchopulmonary hygiene physical therapy[w14 w15]:		
Forced expiratory technique‡	3	D
Autogenic drainage‡	3	D
Positive end expiratory pressure therapy‡	3	D
Flutter device or RC-Cornet device‡	3	D
Postural drainage§	3	D
Mechanical vibration§	3	D
Percussion§	3	D
Intrapulmonary percussive ventilation§	3	D
High frequency chest compression§	3	D
Training		
Exercise training with or without inspiratory muscle training‡	1+	B
Surgery		
Segmental, lobar, or lung resection	2+	B

*Studied in acute exacerbations; †not recommended; ‡active participation of the patient needed; §passive techniques.
See table 3 for definitions of level of evidence and grade of recommendation.

SOURCES AND SELECTION CRITERIA

We searched PubMed using the keywords "bronchiectasis", "diagnosis", "pathogenesis", "treatment", and "management". We also searched the Cochrane Library databases and *Clinical Evidence* and used references from our personal archives. Papers and reports on cystic fibrosis and childhood bronchiectasis were excluded.

Bronchodilators

The mechanisms of expiratory airflow obstruction in non-cystic fibrosis bronchiectasis are not clear, but may include excessive production of mucus, distortion of the bronchial wall, and constriction of smooth muscle. Increased bronchial hyper-reactivity and some reversibility of the airflow obstruction with an inhaled bronchodilator are common. As bronchiectasis may coexist with asthma or chronic obstructive pulmonary disease, some studies have a high degree of uncertainty as to whether airway obstruction is due to asthma, chronic obstructive pulmonary disease, or bronchiectasis (or a combination). Nevertheless, as many patients show signs of airway obstruction and hyper-responsiveness, they often receive bronchodilators.

One small Malaysian study gives some information about the reversibility of airway obstruction in bronchiectasis. It included 24 patients with confirmed bronchiectasis but no signs of an acute exacerbation. The study compared 400 µg fenoterol, followed by 5 mg fenoterol 30 minutes later, with 40 µg ipratropium, followed by 500 µg ipratropium.[w19] FEV_1 increased more than 15% in response to one or both bronchodilators in 11 patients—five responded to both, three to fenoterol alone, and three to ipratropium alone. This small study suggests a significant response to bronchodilators in a subset of patients.

Cochrane reviews found no randomised controlled trials on short acting β2 adrenergic agonists,[18] long acting β2 adrenergic agonists,[19] anticholinergic

Table 3 | Revised grading system for levels of evidence and recommendations in evidence based guidelines[4]

Grade	Evidence
Level of evidence	
1++	High quality meta-analyses, systematic reviews of RCTs, or RCTs with a very low risk of bias
1+	Well conducted meta-analyses, systematic reviews of RCTs, or RCTs with a low risk of bias
1−	Meta-analyses, systematic reviews or RCTs, or RCTs with a high risk of bias
2++	High quality systematic reviews of case-control or cohort studies; or high quality case-control or cohort studies with a very low risk of confounding, bias, or chance and a high probability that the association is causal
2+	Well conducted case-control or cohort studies with a low risk of confounding, bias, or chance and a moderate probability that the association is causal
2−	Case-control or cohort studies with a high risk of confounding, bias, or chance and a significant risk that the association is not causal
3	Non-analytic studies, such as case reports, case series
4	Expert opinion
Grade of recommendations	
A	At least one meta-analysis, systematic review, or RCT rated as 1++ and directly applicable to the target population; or a systematic review of RCTs or a body of evidence consisting principally of studies rated as 1+ directly applicable to the target population and showing overall consistency of results
B	A body of evidence including studies rated as 2++ directly applicable to the target population and demonstrating overall consistency of results or extrapolated evidence from studies rated as 1++ or 1+
C	A body of evidence including studies rated as 2+ directly applicable to the target population and demonstrating overall consistency of results or extrapolated evidence from studies rated as 2++
D	Evidence level 3 or 4 or extrapolated evidence from studies rated as 2+

RCT=randomised controlled trial.

therapy,[20] or oral methylxanthines[21] in patients with non-cystic fibrosis bronchiectasis. Good quality studies are therefore urgently needed in this field.

Bronchopulmonary hygiene physical therapy

Bronchopulmonary hygiene physical therapy is a form of chest physical therapy that aims to remove lung secretions in patients with acute and chronic airway diseases. Many active and passive techniques are available (table 2), and the technique chosen varies between institutes and physical therapists.[w20 w21] The evidence in support of these techniques is variable and the literature is conflicting.[w20] Two systematic reviews found insufficient evidence to support or refute this form of therapy.[22 23]

ADDITIONAL EDUCATIONAL RESOURCES

Resources for healthcare professionals

UpToDate online (www.uptodate.com)—Website designed to answer the clinical questions that arise in daily practice

Clinical Evidence (www.clinicalevidence.com)—Medical resource for informing treatment decisions and improving patient care

Cochrane Collaboration (www.cochrane.org)—Library of systematic reviews of the effects of healthcare interventions to aid decision making

Resources for patients

National Heart Lung and Blood Institute (www.nhlbi.nih.gov/health/)—Information for professionals on diseases of the heart, lung, and blood

British Lung Foundation (www.britishlungfoundation.org/)—Website dedicated to diseases of the lung

Patient UK (www.patient.co.uk/)—Information and support for patients

Bronchiectasis Chat Group (bronchiectasis-subscribe@yahoogroups.com)—Email support group for people with bronchiectasis and their relatives

Exercise training

The role of pulmonary rehabilitation and inspiratory muscle training has been investigated in only one randomised controlled trial. It compared an eight week training programme of pulmonary rehabilitation alone, pulmonary rehabilitation plus inspiratory muscle training, and a control group.[w22] The authors concluded that pulmonary rehabilitation does improve exercise tolerance. Simultaneous inspiratory muscle training offered no additional benefit.

Surgery

If the area of bronchiectasis is localised and the patient's symptoms are debilitating or life threatening, surgical resection has long been thought to be of benefit. This assumption was based on several non-randomised controlled studies of uncertain value. A Cochrane review updated in 2002 found no randomised clinical trials—just case series or case-control studies.[24] The largest case-control study included pneumonectomy in 190 cases, lobectomy in 202 cases, bilobectomy in 23 cases, and lobectomy combined with segmental resection in 72 cases.[25] Overall mortality was 3.5%, and 71% of patients had no symptoms during follow-up (four months to 10 years). The authors therefore concluded that surgery was preferable to conventional medical treatment.

Conclusion

The general consensus is that acute exacerbations should be treated promptly with short courses of systemic antibiotics. In stable bronchiectasis, a high level of evidence exists for the use of prolonged and aerosolised

SUMMARY POINTS

- Bronchiectasis refers to abnormal bronchial dilatation caused by a vicious cycle of transmural infection and inflammation
- Symptoms include chronic productive cough, wheeze, and dyspnoea; repeated respiratory infections may dominate the clinical picture
- Diagnosis is based on daily production of mucopurulent phlegm and dilated and thickened airways on computed tomography
- Diagnosis should lead to investigation and treatment of possible causes and associated conditions
- Acute exacerbations should be treated promptly with short courses of antibiotics
- Frequent exacerbations may be treated with prolonged and aerosolised antibiotics
- The role of mucolytics, anti-inflammatory agents, and bronchodilators is not clear
- Surgery is a possibility if the area of bronchiectasis is localised and symptoms are debilitating or life threatening

antibiotics, but this form of treatment is mostly reserved for patients with frequent exacerbations. Because of the low level of evidence we cannot recommend the use of mucolytics, anti-inflammatory agents, or broncho-pulmonary hygiene therapy on a routine basis. Surgery may be considered if the area of bronchiectasis is localised and the patient's symptoms are debilitating or life threatening. Well powered studies to investigate the effects of well defined treatment regimens on important short and long term outcomes of bronchiectasis are urgently needed.

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An all-round thumbs up

A 43 year old street cleaner was brought to her local emergency department in the early hours of the morning by a concerned set of firefighters. The patient had inadvertently trapped her non-dominant thumb in the end of a standpipe. The activated self-locking mechanism had clamped on her thumb within the heavyweight tubing, and the fire crew had been unable to prise her free.

After the administration of a digital nerve block and some fairly hefty head scratching, a far fetched plan came to mind. The standpipe had two holes (for a cross bar) about 10 cm from the locked thumb. By inserting a fine-bore optic naso-endoscope into one of the holes, I was able to see the pin compressing the patient's thumb and simultaneously introduce a right-angled Lahey clamp. This enabled me to retract the pin, and the patient slide her thumb free to a resounding round of applause from the attendant fire crew.

The benefits of an old fashioned basic surgical training shone through as my accident and emergency, ENT, and general surgery skills combined for an all-round thumbs up.

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