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Bronchiectasis Revisited: Imaging-Based Pattern Approach to Diagnosis

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A B S T R A C T

Background: Bronchiectasis is one of the causes of non-resolving, persistent or recurrent pulmonary infection which, if uncorrected may have deleterious consequences on the lung parenchyma and pulmonary circulation. High-resolution computed tomography (HRCT) is needed for the confirmation, localization and directing management accordingly.

Contents: Bronchiectasis is one of the major cause of morbidity worldwide. Chest radiograph is done at the initial suspicion which is supplemented by HRCT to confirm the diagnosis. Imaging diagnosis supplemented by the recognition of the pattern of involvement is essential to outline the differential diagnosis, map the complications and, hence, guiding the further management. Identification of the causative aetiology may not only prevent its further progression but obviate recurrent insults to the lung parenchyma as well. This article focuses on an algorithmic approach to bronchiectasis based on the distribution on imaging

Introduction

Bronchiectasis refers to the permanent irreversible dilatation of cartilage containing airways. Although its exact etiopathogenesis is not known, it is believed to be the end result of multiple factors including recurrent infection or inflammation, impaired mucociliary clearance and attrition of the bronchial wall occurring in concurrence leading to the subsequent bronchial dilatation.[1,2] It remains a disease of international concern with high prevalence worldwide, although the etiology differs. Clinically, there may be hypoxia, dyspnea, or hemoptysis depending upon the severity of affliction and superimposed infection. Milder disease presents with occasional cough and dyspnea. With chronicity and increasing severity, there is worsening of symptoms. Abnormalities in pulmonary function tests ensue with an obstructive pattern of abnormality.

Diagnosis of bronchiectasis is essentially based on imaging which morphologically defines airway enlargement. Imaging may also help in determining the etiology in certain cases, guiding management (surgical vs medical) and to rule out secondary complications. In children, it is essential to diagnose bronchiectasis early to prevent irrevocable damage to the developing airways and lung parenchyma, besides prognostication and genetic counseling in certain causes.[2]

This article discusses the criteria for diagnosing bronchiectasis, evaluation of complications and then proposes an imaging-based algorithmic approach toward an etiologic diagnosis.

Criteria for Diagnosing Bronchiectasis on HRCT

The foremost step in managing bronchiectasis is ascertaining its presence on HRCT, which is the most sensitive imaging modality to establish its diagnosis. With the available multidetector row CT scanners, it is possible to visualize airways as small as 2 mm in diameter with a wall thickness of up to 0.2 mm. In general, there should not be any visible airways within 1 cm of the costal pleura, although this criterion does not hold true for mediastinal pleura; which may show airways within 1 cm. Further, the internal diameter of the bronchus is equal to the accompanying pulmonary artery at the same branching level. This is referred as *broncho-arterial ratio*. Special CT reconstructions like minimum intensity projection are very useful in providing an overview of involvement and may identify subtle abnormalities in certain cases.[1,3-5]

Bronchiectasis is diagnosed on HRCT on the basis of certain criteria (Table 1 and Fig 1). Most reproducible among these criteria is the measurement of broncho-arterial ratio.[6-14]

Ancillary findings in bronchiectasis include mucoid impaction in distal smaller airways, leading to bronchiolitis. It results in characteristic “tree in bud” appearance with branching opacities in “V” and “Y” pattern. Secondary changes in the neighboring lung parenchyma include volume loss, mosaic attenuation due to air trapping, fibrosis, and scarring.[6,9,10,15,16]

Complications associated with bronchiectasis may be superimposed infection, which is suggested by an increase in the peribronchial thickening, presence of air-fluid levels, centrilobular nodules, or consolidation in the adjacent lung parenchyma. Other complications include pulmonary artery hypertension, mosaic attenuation and volume loss of the affected lung parenchyma (Fig 2).

Role of HRCT in the Management of Bronchiectasis

Imaging may provide a clue toward the etiology of bronchiectasis by recognizing its pattern of distribution, lobar predisposition as well as other ancillary features. The causes of bronchiectasis may be local or systemic. Most cases of bronchiectasis encountered in clinical

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TABLE 1
Criteria for diagnosing bronchiectasis on HRCT

Broncho-arterial ratio (BAR)	Other criteria
BAR >1.0 is abnormal in adults*	Lack of bronchial tapering or tram track appearance—no change in the caliber of bronchi at least 2 cm distal to the branching point. It is best seen in axial images in middle lung zones due to horizontal orientation of airway. Highly sensitive and earlier change to occur than bronchial dilatation.
Suggested BAR in children >0.8 due to smaller caliber of airways.	
Eccentrically placed arteriole around dilated bronchus results in "signet-ring" appearance.	Visualization of peripheral airways within 1 cm of the costal pleura (except mediastinal pleura).
	Peribronchial thickening—the bronchial wall thickness more than twice of the normal bronchus.

*Cut-off for abnormality in adult.

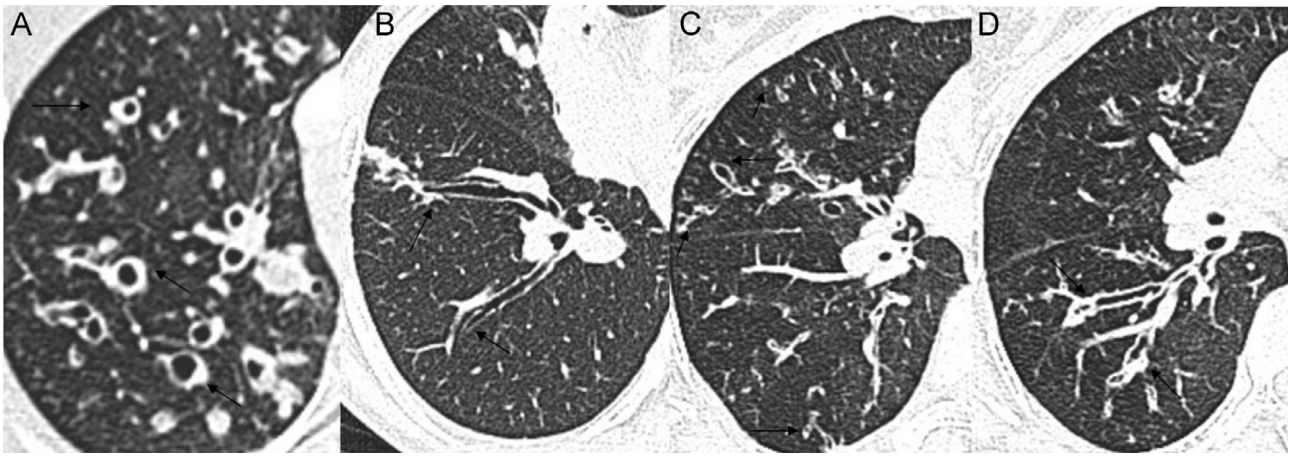

FIG 1. Criteria for bronchiectasis. (A) Increased broncho-arterial ratio (BAR > 1), "signet- ring" sign (arrows). (B) Lack of bronchial tapering (arrows). (C) Visualization of peripheral bronchi (arrows). (D) Peribronchial thickening (arrows).

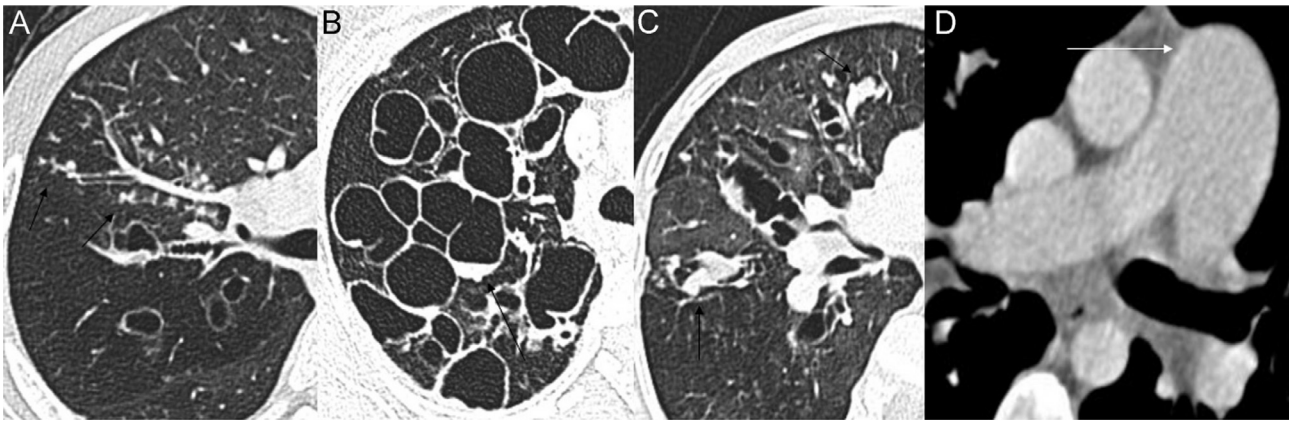

FIG 2. Complications of bronchiectasis. (A and B) Superimposed infection suggested by centrilobular nodules (arrows) and air-fluid level (arrow in B). (C) Mucus impaction (arrows). (D) Pulmonary artery hypertension (arrow).

practice are postinfectious (bacterial, tuberculosis [TB], pertussis, and viral) in origin. Other commoner causes are abnormal mucociliary clearance, allergic bronchopulmoary pulmonary aspergillosis (ABPA) or idiopathic (Table 2). Apart from establishing the diagnosis, HRCT is crucial in the management of bronchiectasis and the complications consequent to it.

Etiological Diagnosis of Bronchiectasis Based on Distribution Pattern

For simplicity, bronchiectasis is broadly divided into "focal" or "diffuse" based on the extent of involvement.[1,17] Based on the spatial distribution of bronchiectasis on HRCT, clue to the possible etiology may be hinted for which an algorithmic approach is proposed (Fig 3). This approach may help in narrowing the differential diagnosis; nonetheless, arriving at a conclusive diagnosis is a holistic approach additionally requiring correlation of symptomatology and laboratory investigations.

TABLE 2
Etiology of bronchiectasis

Postinfectious	Systemic immunodeficiency
Tuberculosis	Aspiration syndromes
Nontuberculous mycobacteria	Chronic obstructive airway disease
Bacterial	Interstitial lung disease
Viral	Toxins and drug injury
ABPA	Idiopathic
Congenital	
Cystic fibrosis	
Ciliary dyskinesia	
Mounier-Kuhn syndrome	
Willliam-Campbell syndrome	
Localized obstruction	
Foreign body	
Stricture	
Neoplasms	

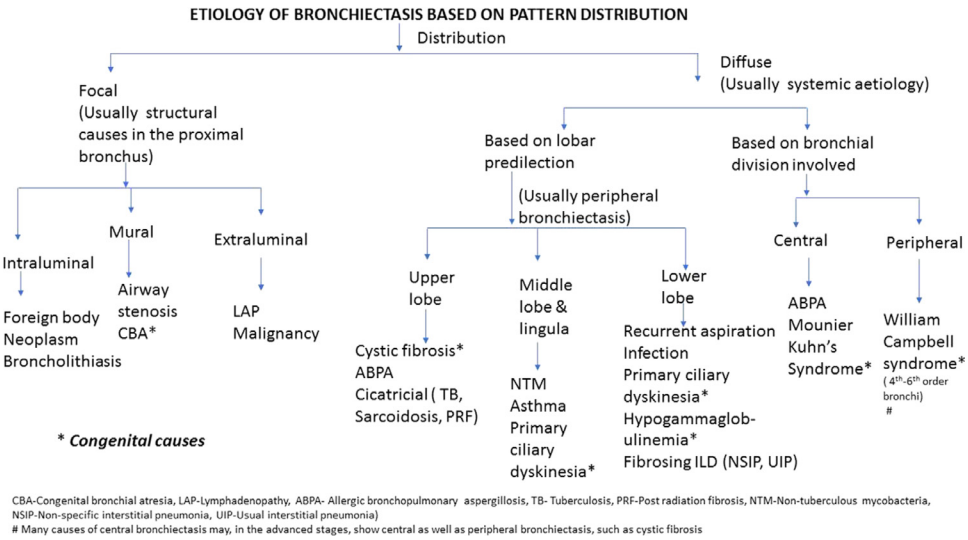

FIG 3. Etiology of bronchiectasis based on pattern distribution. (Color version of the figure available online.)

Focal Bronchiectasis

The term focal is used here to refer to the bronchiectasis, which is confined to a part of the lung supplied by a single lobar or segmental bronchus or even the main bronchus. Herein, the bronchiectasis is confined to the distribution of lung distal to the affected proximal airway. The importance of this is that when bronchiectasis is confined to a segment, lobe or even single lung, the proximal airway should be meticulously evaluated. For airway obstruction to result in bronchiectasis, it may be intraluminal, mural, extraluminal or extrinsic. In general, focal bronchiectasis is due to the structural abnormality and due

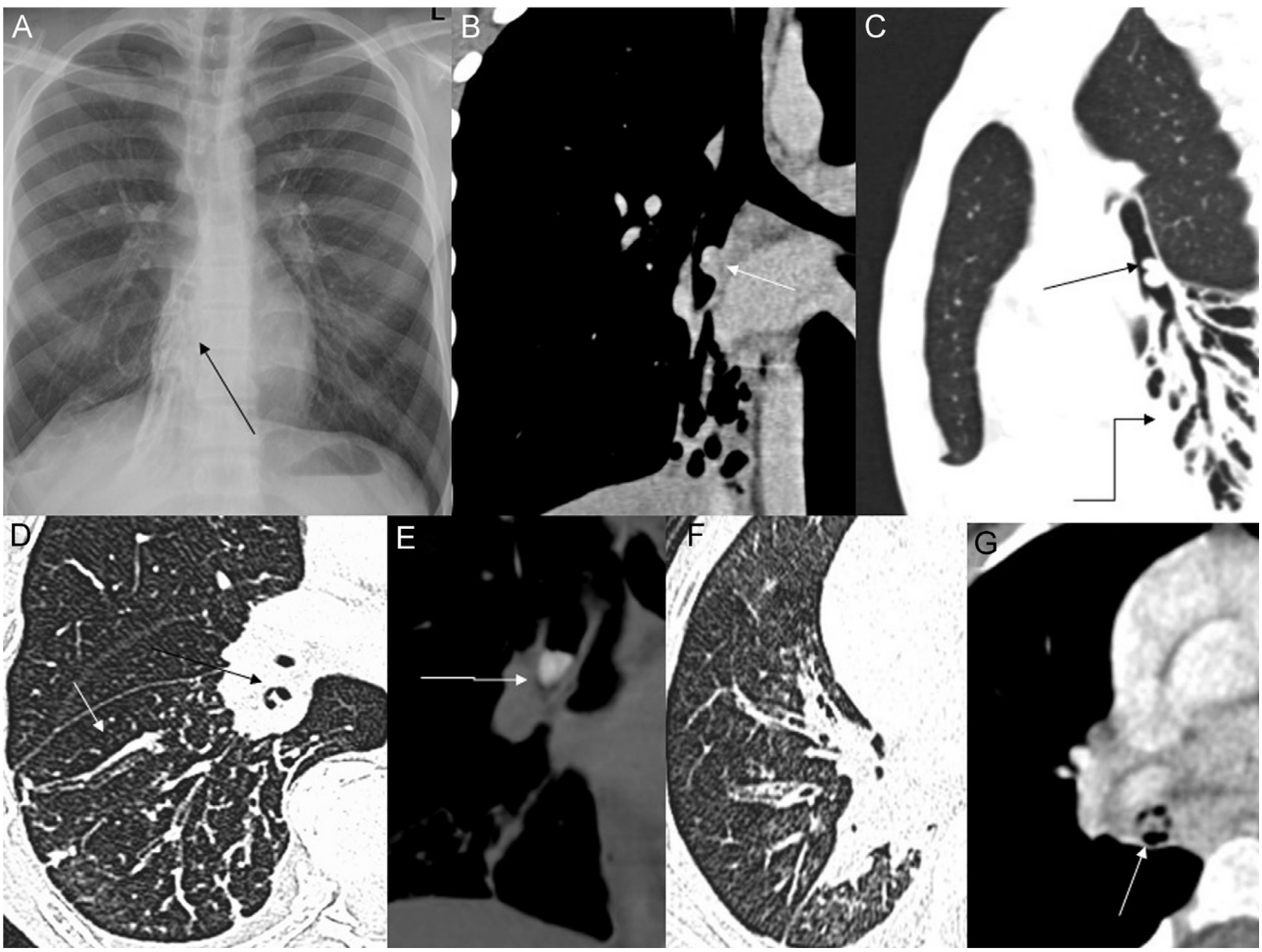

FIG 4. Focal bronchiectasis—intraluminal occlusion. (A-C) Carcinoid in right lower lobe bronchus. (A) Chest radiograph showing localized right lower lobe bronchiectasis (arrow). (B) Coronal CECT in mediastinal window demonstrating intraluminal enhancing polypoidal mass (arrow) in the lobar bronchus with (C) distal localized bronchiectasis (curved arrow). (D and E) Right lower lobe (RLL) bronchiectasis due to broncholith (arrow), better appreciated on coronal bone window. (F and G) RLL bronchiectasis in a child due to chronically retained inhaled foreign body (whistle, arrow in G).

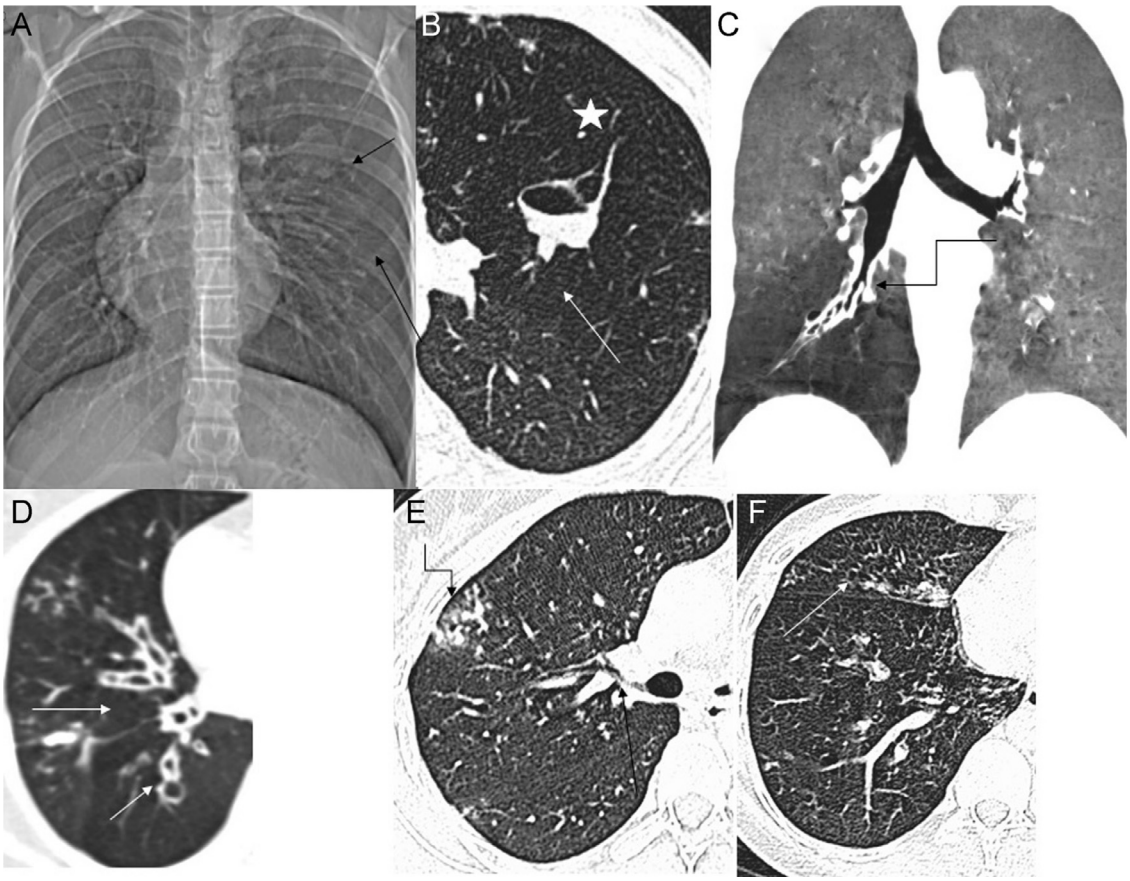

FIG 5. Focal bronchiectasis—mural narrowing (A-D) Congenital bronchial atresia: Topograph in a child showing round opacity in left upper lobe (straight arrow) with surrounding hyperlucent lung s/o air trapping (curved arrow). (B) Axial HRCT showing blind ending fluid-filled tubular opacity (straight arrow) with distal bronchiectasis (curved arrow) and surrounding mosaic attenuation (asterisk). (C and D) Posttubercular narrowing of bronchus intermedius (arrow in E) with localized bronchiectasis in right middle and lower lobes. (E and F) Another case of active tuberculosis with irregular narrowing of right middle lobe bronchus, centrilobular nodules (curved arrow) as well as bronchiectasis (arrow) in lung parenchyma.

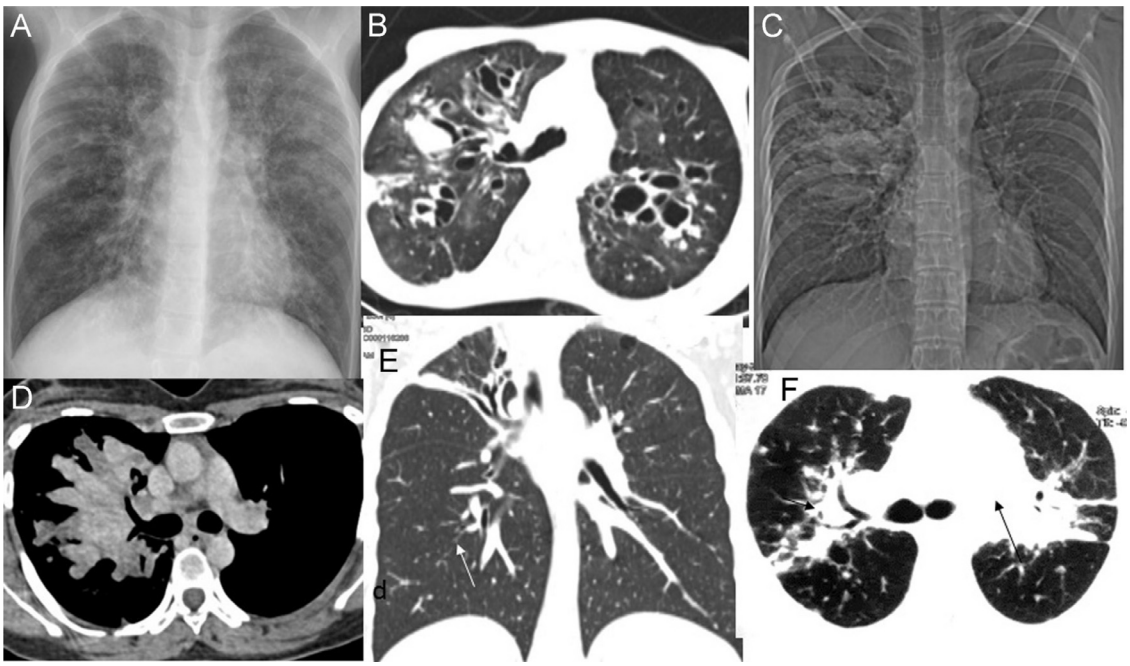

FIG 6. Diffuse bronchiectasis—upper lobe predominant (A and B). Cystic fibrosis—bilateral bronchiectasis with upper lobe predominance (C and D). ABPA with upper and central lobe bronchiectasis, "finger-in-glove" appearance and high attenuation mucus (arrow in D). (E) Posttuberculosis right upper lobe bronchiectasis (arrow). (F) Sarcoidosis, on treatment-mild bilateral upper lobe bronchiectasis (arrow), peribronchovascular thickening and perilymphatic nodules.

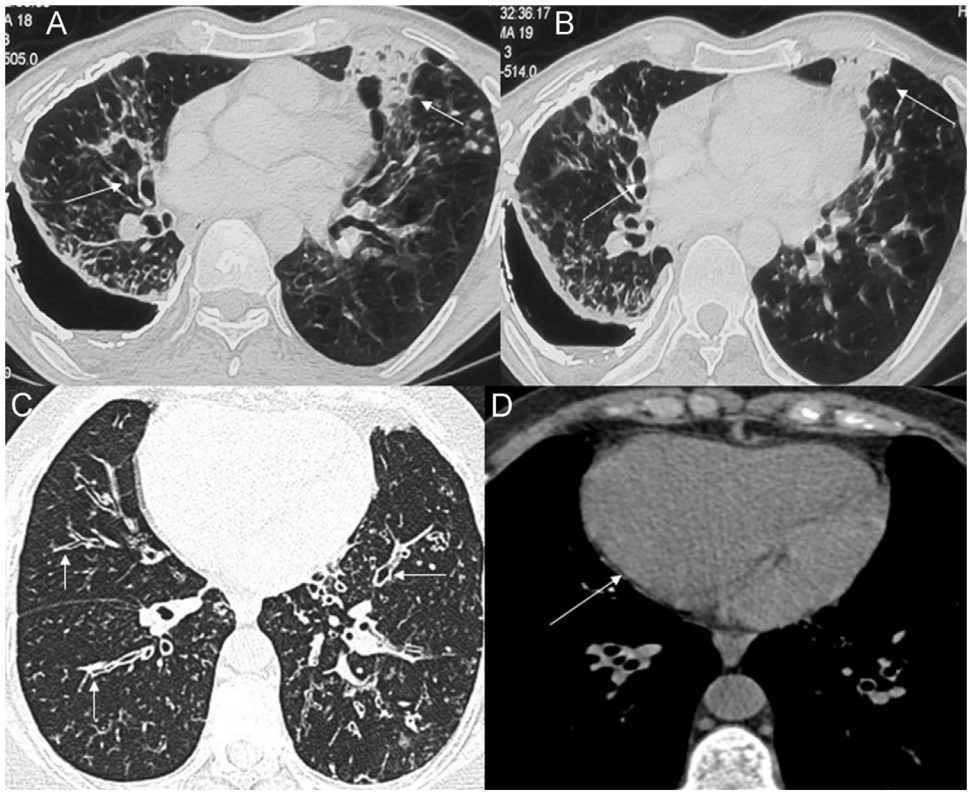

FIG 7. Diffuse bronchiectasis—right middle lobe (RML) and lingula (A and B). Nontuberculous mycobacteria with bronchiectasis in RML and lingual (arrow), pleural calcification and iatrogenic pneumothorax. (C and D) Kartagener's syndrome with bronchiectasis in RML, lingula and bilateral lower lobes (arrows) and dextrocardia (arrow in D).

to confined involvement management is either surgical or bronchoscopic, depending on the etiology.

Intraluminal. Foreign body is the most common intraluminal cause of bronchiectasis in children. Aspirated foreign body, if remains undiagnosed may cause recurrent infection leading to bronchiectasis. Intraluminal occlusion in adults is usually due to neoplasms, of which carcinoid is the commonest cause of long standing occlusion. Other rare causes include mesenchymal tumors (Fig 4). More aggressive neoplasms such as bronchial carcinoma present early and hence, bronchiectasis is not usually seen. A detailed discussion on various malignancies affecting the tracheobronchial tree is beyond the scope of this review. Broncholithiasis is a nonneoplastic cause of bronchiectasis, which occurs due to the calcified granulomatous mediastinal or hilar lymph nodes eroding into the adjacent bronchus.[1]

Among these intraluminal causes of bronchiectasis, distinction may be made on the basis of density and enhancement of the obstructing soft tissue. Foreign body may be of soft tissue or high density lesions and lack any enhancement. Organic foreign body may show internal air foci due to entrapped air. Neoplasms, on the contrary, are of soft tissue density and are usually enhancing. Typical carcinoids are characteristically intensely enhancing central masses affecting the central, main or segmental bronchi with low malignant potential. Presence of extraluminal invasion and lymphadenopathy are other corroborative findings to suggest a neoplasm.

Age of the patient and location of the lesion may be helpful in certain cases. For instance, foreign body aspiration is usually seen in children, and most commonly involves the right mainstem bronchus followed by left mainstem bronchus due to their alignment in line with the trachea. Broncholithiasis, on the other hand, most commonly involves bronchi of anterior segment of upper lobes and right middle lobe in their proximal part, explained by their anatomical proximity to lymphadenopathy. In broncholithiasis, apart from the calcified intraluminal density there may be other findings to suggest infective sequelae.

Mural Abnormality. Focal mural narrowing or airway stenosis may be congenital or acquired. Congenital bronchial atresia (CBA) or congenital mucocele is the most common cause of localized congenital bronchiectasis. It may involve main, lobar or segmental bronchus and most commonly involves left upper lobe. Acquired stenosis may result from infections (TB, bacterial, pertussis, and respiratory syncytial virus), inflammation (sarcoidosis, amyloidosis, and relapsing polychondritis) or prolonged intubation (Fig 5).

Imaging in a classic case of CBA may demonstrate triad of tubular or ovoid opacity suggesting mucocele, focal hyperlucent affected segment due to air trapping along with its hypoperfusion due to scarcity of vessels. CBA may be differentiated from acquired stenosis by earlier age of presentation in infancy or childhood with recurrent infection, although few cases may remain asymptomatic. Acquired stenosis on the other hand usually presents in adults with a history specific to the underlying etiology, usually predating the bronchiectasis. CBA is always associated with a mucocele as opposed to the acquired causes of bronchostenosis, which may have mucocele depending on the degree of stenosis. Mucocele in the CBA lacks communication with the proximal airway due to focal interruption as opposed to the acquired mucocele. Moreover, hyperinflation of the adjoining lung parenchyma is a feature which is present in CBA and is lacking in acquired mucocele. Flexible fibreoptic bronchoscopy is invaluable in demonstrating blind ending atretic bronchus in CBA besides excluding secondary causes of stenosis.[1,3,17]

Extraluminal Causes. Bronchial narrowing may result from adjoining mediastinal lymphadenopathy (infectious and neoplastic) or mediastinal masses. Although any lobe may be involved, they most commonly involve anterior segment of right upper lobe or middle lobe due to the proximity of the origin of their lobar bronchi to the hilum.

Necrotic mediastinal lymphadenopathy as in TB can involve the airway in multiple ways wherein the mechanism can be extraluminal (compression), mural (by inflammation of the wall), or intraluminal

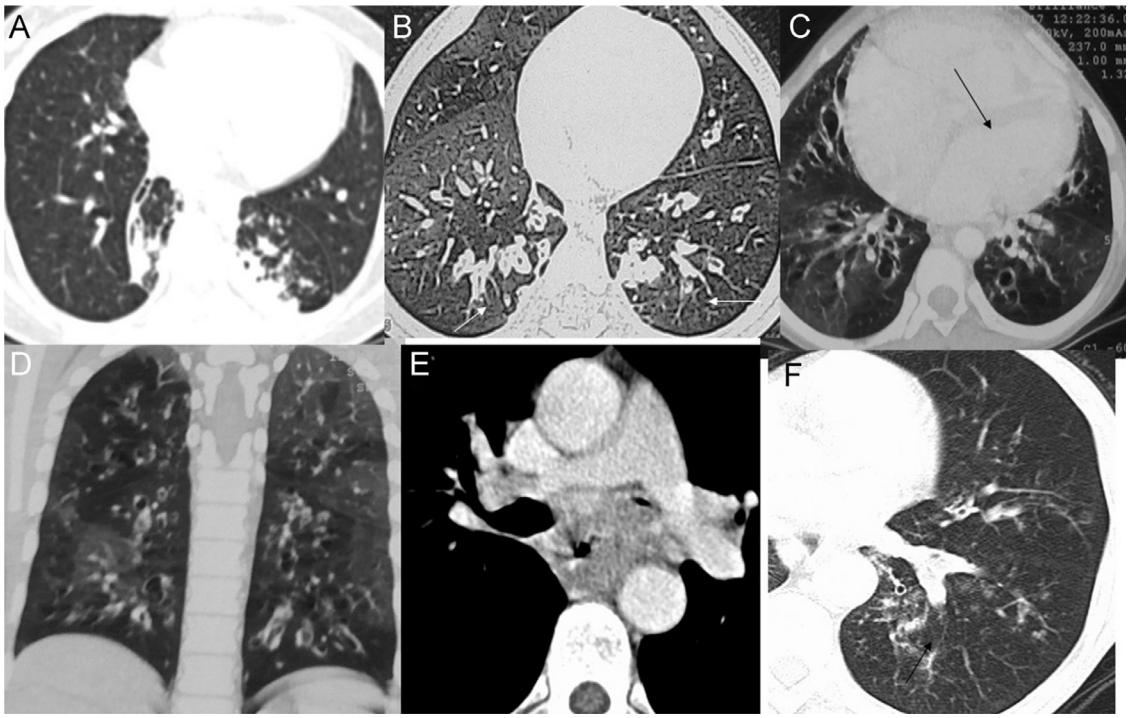

FIG 8. Diffuse bronchiectasis—lower lobe predominant. (A) Postinfective, tuberculosis sequelae. (B) Combined variable immunodeficiency with bronchiectasis and peribronchial thickening (arrow) in bilateral lower lobes. (C and D) Hyper Ig E syndrome with lower lobe predominant bronchiectasis. (E and F) Carcinoma mid thoracic esophagus infiltrating the left mainstem bronchus (arrow in E) with centrilobular nodules and mild bronchiectasis (arrow in F) in left lower lobe due to aspiration.

(due to erosion of a necrotic node into the bronchus). On subsequent healing, mural inflammation may result in bronchial stenosis whereas the intraluminal contents may result in formation of bronchiolitis. The common sites of involvement are bronchus intermedius leading to the bronchiectasis in right middle and lower lobes. Extraluminal compression of the bronchus is common; however, it more frequently leads to collapse than bronchiectasis.[1,17,18]

Diffuse Bronchiectasis

Diffuse bronchiectasis refers to the bilateral or multifocal involvement with no endobronchial cause to it. It is usually due to a systemic pathology, which may be inflammatory, infectious or congenital. Based on the lobar distribution within the lung, the involvement may be further categorized into upper, middle or lower lobe dominant disease. Central bronchiectasis refers to involvement of central bronchial tree, which includes mainstem bronchus up to the lobar division. Due to involvement of more than one lobe, diffuse bronchiectasis is managed conservatively with treatment directed toward underlying etiology and preventing associated complications.

Based on Lobar Predilection

Upper Lobe Predominance. Bronchiectasis with upper lobe predominance is seen in cystic fibrosis (CF), allergic bronchopulmonary aspergillosis (ABPA), or may be cicatricial, for instance in TB, sarcoidosis or postradiation fibrosis (Fig 6).

CF usually manifests in childhood with bilateral symmetrical involvement. Apart from recurrent pulmonary infections, sinusitis, infertility, and pancreatic insufficiency are the other systemic

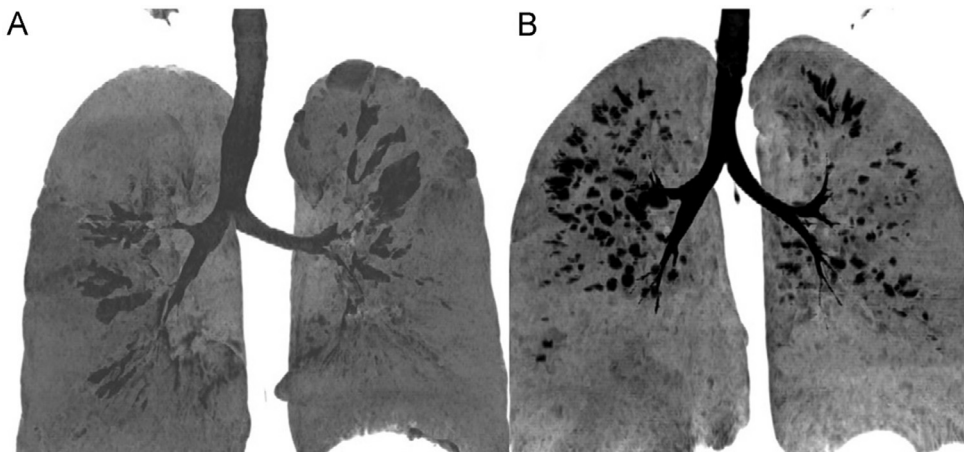

FIG 9. Central vs peripheral bronchiectasis. (A) Central bronchiectasis involving medial two-third of the lung. (B) Peripheral bronchiectasis involving lateral one-third of the lung.

manifestations. Bronchiectasis is initially localized to right upper lobe, however, with further progression, there is diffuse lung involvement.[1,3,19]

ABPA may present in childhood (in CF) or adults (asthmatics) and although the involvement is bilateral but it is usually asymmetrical. Involvement of central airways is a constant finding in ABPA in the early stages. Ancillary features to further differentiate these entities include presence of peribronchial thickening and more severe bronchiectasis in CF compared to ABPA. On the other hand, fleeting pulmonary opacities (migratory pneumonitis) in the initial stages and high attenuation mucus (HAM, >70-100 HU) favours ABPA.[20,21]

Posttubercular bronchiectasis has predilection for upper lobes which parallels the site of occurrence of parenchymal TB in the lung apices. It may involve single or multiple lobes. Bilateral symmetrical upper lobe bronchiectasis involvement if present, favours sarcoidosis. Furthermore, degree of bronchiectasis in TB is more severe as compared to sarcoidosis. Both may have calcified lymph nodes. In postradiation fibrosis, there will be history of prior radiotherapy. Fibrobronchiectasis occurs in a wedge shaped, triangular distribution corresponding to the radiation portal. It, characteristically, has a sharp margination from the surrounding lung parenchyma with nonanatomical distribution. Bilateral paramediastinal involvement is present if there is mediastinum irradiation.[1,2,5,6,18]

Right Middle Lobe and Lingula Predominance. Bronchiectasis in this distribution is commonly seen nontuberculous mycobacterial infections (NTM) (Fig 7). NTM infection usually affects elderly immunocompetent females who voluntarily suppress their cough reflex. Impaired cough response, which is a predisposing factor explains its predilection for right middle lobe and lingual.[19-22] It can also be seen in patients with structural lung lesions like COPD, pneumoconiosis or prior tuberculosis.[23] It is usually resilient to treatment as opposed to mycobacterium tuberculosis. On imaging, bronchiectasis (usually cylindrical) and bronchiolectasis may be seen involving right middle lobe and lingual.[22-25]

Lower Lobe Predominance. Lower lobe bronchiectasis may be due to aspiration, recurrent childhood infections, in primay ciliary dyskinesia (PCD) or in immunodeficiency states such as hypogammaglobulinemia. In middle aged or elderly patients, lower lobe involvement is commonly due to traction bronchiectasis seen in fibrosing interstitial lung diseases such as usual interstitial pneumonitis and nonspecific interstitial pneumonia (Fig 8).

Aspiration related bronchiectasis usually occurs secondary to the prolonged persistent aspiration, which may be present in various aspiration syndromes notably, gastro-esophageal reflux disease, hiatus hernia, and trachea-esophageal fistula. On imaging, there may be bronchiectasis and peribronchial thickening involving bilateral lower lobes.[1,2,4] HRCT may additionally show ill-defined centrilobular nodules if there is superimposed active involvement. Recurrent or chronic childhood bacterial or viral infections may lead to lower lobe bronchiectasis. Childhood TB may also heal by middle and lower lobe bronchiectasis.[1,3,17]

Primary ciliary dyskinesia or immotile cilia syndrome is an autosomal recessive condition resulting from defective ciliary motility thus affecting the clearance of secretions. Altered mucociliary clearance further leads to recurrent sinusitis, chest infections and infertility. Kartagener's syndrome is a subtype of PCD manifesting with the triad of bronchiectasis, sinusitis and situs inversus. Bronchiectasis in PCD has predilection for right middle lobe, lingula and bilateral lower lobes and is predominantly central in distribution.[26] Severity of affliction may differ in various regions giving rise to heterogeneous

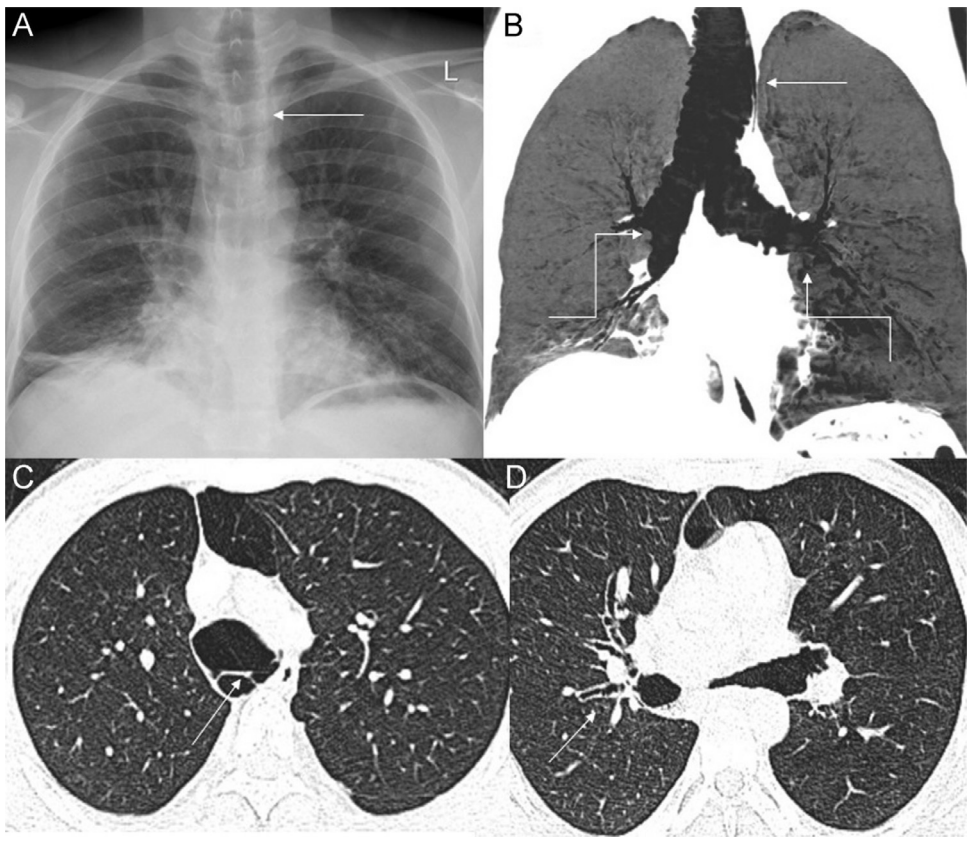

FIG 10. Central bronchiectasis. Mounier-Kuhn's syndrome (A) chest radiograph and (B) coronal MinIP axial image showing tracheomegaly (straight arrow) and grossly dilated mainstem and lobar bronchi (curved arrow). (C and D) Additionally, posteriorly projecting tracheal diverticula (arrow in c) and central bronchiectasis are present (arrow in D). MIP, minimum intensity projection.

morphological patterns. In Kartagener's syndrome, situs inversus and sinusitis are additional findings.[27]

Based on Bronchial Division Involved

On the basis of involvement of bronchial division, bronchiectasis is categorized into central and peripheral. In most causes leading to bronchiectasis, there may be eventual involvement of both central as well as peripheral airways due to recurrent inflammation in the obstructed segment. However, some entities show characteristic involvement of the central or peripheral airways.[28,29]

Central Bronchiectasis

Central bronchiectasis refers to the involvement of proximal large airways affecting medial two-third of lung (Fig 9). Central involvement is usually seen in ABPA and rarely may be inherited as in Mounier-Kuhn's disease.[28,29]

Tracheobronchomegaly (Mounier-Kuhn's disease) occurs due to congenital weakness of airway wall resulting in progressive thinning of its various components including cartilage, muscle, and elastin, ultimately leading to tracheobronchomegaly. Subsequently, mucosal herniations (diverticulosis) occur through thin walled airways. Despite being a congenital condition, it initially manifests with recurrent pulmonary infections in third to fifth decade having male propensity, although it may present as early as 18 years of age. Lungs are hyperinflated with dilatation of trachea and bronchi (up to fourth order) leading to central bronchiectasis. HRCT confirms the diagnosis of tracheobroncheomegaly which is objectified by the tracheal diameter exceeding 30 mm in sagittal and coronal scans (Fig 10). Characteristic feature is the absence of mural thickening. On dynamic HRCT, involved airways show further dilatation in inspiration and collapse during expiration. Additionally, inspissated mucus can be seen in more distal airways with infection and fibrosis in surrounding lung parenchyma.[30]

Peripheral Bronchiectasis

Peripheral bronchiectasis involves outer one-third of lungs. As stated earlier that most of the stated causes of central bronchiectasis may have eventually peripheral bronchiectasis as well,[27,28] except Mounier-Kuhn syndrome. Exclusive involvement of peripheral lung in the absence of central bronchiectasis or other cicatricial lung disease has limited differential diagnosis.

William Campbell syndrome: it is characterized by cartilage abnormality involving smaller peripheral bronchi (fourth to sixth generation), which may be congenital or acquired as a sequelae to childhood infections. Lung involvement may be localized or diffuse. Bronchiectasis is usually peripheral with cystic morphology, which on dynamic imaging may show expansion in inspiration and collapse on expiration.[31]

Conclusion

Bronchiectasis may root from various causes. Establishing its diagnosis necessitates corroborating clinical history and symptomatology with the radiographic findings. Its early diagnosis and treatment is imperative to halt the vicious cycle of recurrent infection and continuing damage to the lung parenchyma. A simplified diagnostic approach based on the distribution pattern of involvement on imaging narrows the differential diagnosis and guides the further work up accordingly.

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