ORIGINAL RESEARCH PAPER
Volume - 10 | Issue - 04 | April - 2021 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

A RARE AND INTERESTING PRESENTATION OF EMPHYSEMATOUS BULLAE

General Surgery

Dr.Sri Naga Maithry Gudala* 2ND YR GENERAL SURGERY PG *Corresponding Author

Dr.Raju S Iyer MS,MCh,F.C.C.P,F.I.A.C.S – (CTVS).

Dr.L.Nageswara Rao M.D (ANESTHESIA)

ABSTRACT

The term bullous disease is reserved for multiple bullae in lungs that are otherwise normal.This entity is different in etiology and pathogenesis from that in which bullae occur in conjunction with underlying chronic obstructive pulmonary disease (COPD).The present report is regarding a case of emphysematous bullae in a 31 yr old female in right lower lobe.The patient underwent right lower lung segmental resection where fungal ball was found intraoperatively and was removed.The patient had an uneventful recovery postoperatively.

KEYWORDS

INTRODUCTION :

Emphysema is defined as dilation and destruction of the terminal air spaces. These air cavities are blebs (subpleural air space separated from the lung by a thin pleural covering with only minor alveolar communications) or bullae (larger than a bleb with some destruction of the underlying lung parenchyma) (1).A bulla is an air-containing space within the lung parenchyma that arises from destruction, dilatation, and confluence of airspaces distal to terminal bronchioles and is larger than 1 cm in diameter.Its walls are composed of attenuated and compressed parenchyma (2). The term bullous disease is reserved for multiple bullae in lungs that are otherwise normal(3). Giant bullae are those that encompass more than one-third of the lung volume. They are uncommon but when present can lead to compression of adjacent normal lung tissue. The presence of emphysema associated with large bullae is referred to as bullous emphysema (4).

We present a case of 31 yr old female with right sided bullous emyphysema where we found intraop fungal ball in the cavity

CASE PRESENTATION :

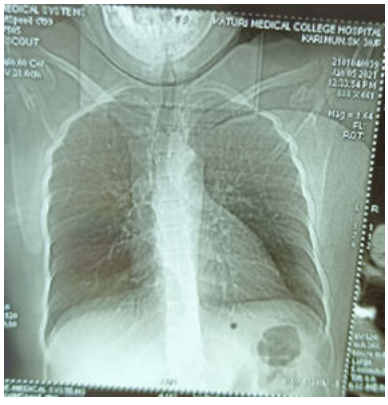
A 31 year old female non diabetic non smoker presented in january 2021 with chief complaint of chestpain since 20 days associated with shortness of breath during mild exercise and persistent fatigue 5 months prior she developed similar complaints such as chestpain which was vague,intermittent onset and gradually progressive, associated with shortness of breath while exertion and relieved with rest.She did not have not cough and hemoptysis or fever

No previous history of surgeries,non diabetic,non hypertensive,age of menarche-13yrs,P3L3,regular menstrual cycles,bowel and bladder regular,normal sleep and apettite,no significant family history

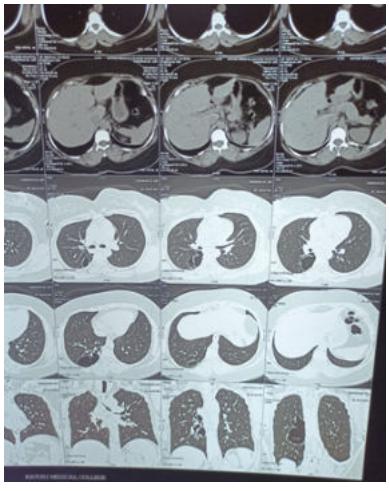
Physical examination revealed young female alert and oriented without any respiratory distress speaking full sentences.Vitals stable.There was no cyanosis,clubbing or edema.On chest examination,chest movements symmetrical with accesory muscles of respiration working. Trachea was shifted to left.Tactile fremitus was absent on right side with ipsilateral diminution of breath sounds,resonant to percussion with no added sounds.Vitals stable. Rest of the examination was unremarkable. Preoperative pulmonary function tests (PFT) were consistent with an obstructive pattern (Forced expiratory volume in 1 sec (FEV 1) 1.71L; 45% predicted).

INVESTIGATIONS :

Routine blood investigations – NORMAL
CHEST XRAY PA VIEW – FIG.1
HRCT CHEST FIG.2 – approx.10X5.6 cavitatory lesion with internal soft tissue density component and internal septations noted in posterobasal segment of right lower lobe with perilesional centrilobular nodules and tree in bud opacities-likely bronchiectatic cavity with mycetoma and surrounding active infection component.Perihilar bronchiectasis noted in right lower lobe.Few subcentric pretracheal,prevascular and aortopulmonary window lymohnodes noted



CHEST XRAY PA VIEW - FIG.1



HRCT CHEST FIG.2

PROCEDURE :

Under general anesthesia and left endobronchial intubation with double lumen ET tube,right chest opened by right posterolateral thoracotomy through the 5th intercostal space.Under one lung anesthesia the emphysematous bullae of the right superior segment and lower lobe opened after releasing the lung adhesions.Fungal ball identified and retrieved.The bronchiolar leaks closed with 4-0 prolene and the segmental bronchial opening also closed with 4-0 prolene by 2 layers.Betadine lavage given.The bullous cavity closed after excising

the superior segments.No air leaks.Hemostasis achieved.2 chest drains placed.Intercostal block done.Chest closed in layers with NO.1 ethilone and muscles with 1-0 vicryl and routine closure done.Patient extubated on table.Patient without airleaks on ICD and shifted to ICU.Specimen sent for HPE.

Operative findings :

1) Large emphysematous bullae occupying entire superior segment and right lower lobe with multiple bronchial leaks (FIG.3)
2) A 2X1 sized fungal ball (FIG.4)

Rest of lung segments and lobes normal
Patient was stable postop and shifted to ICU

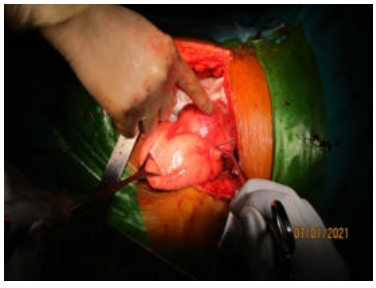


FIG.3

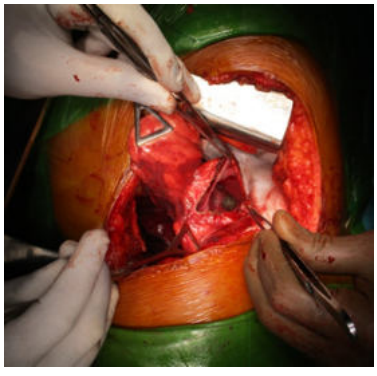


FIG.4

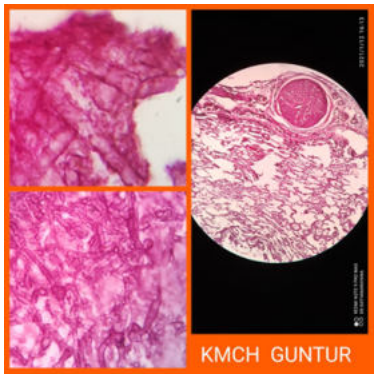


FIG.5

HISTOPATHOLOGY :

Bronchiectasis with focal emphysematous change and aspergillus fungal structures (FIG 5)

DISCUSSION :

Bullous emphysema was originally described by Burke in 1937 as an idiopathic, distinct clinical syndrome of severe progressive dyspnea caused by extensive, predominantly asymmetric upper lobe emphysema that may eventually lead to respiratory failure (4)

Bullae may originate in a variety of clinical and pathogenetic settings:
(1) with emphysema of distal acini
(2) in the setting of cigarette smoking
(3) in conjunction with scar tissue formation, which "traps" areas of normal lung, enlarges airspaces by traction on surrounding intact alveoli, or produces retraction or shrinkage of intact walls of adherent alveoli
(4) in the setting of intravenous drug abuse
(5) as a result of chronic inflammation and destructive changes in terminal and first-order respiratory bronchioles,resulting in airspace distention from delayed emptying; and
(6) with α1-antitrypsin deficiency

Several hypotheses have been proposed over the years for how bullae develop, although none have been proved.These include

1) weakness of the alveolar walls predisposes to the formation of bullae,particularly at the apices of the lungs, where pleural pressures are most negative. This theory underscores the proclivity of bullae for the upper lobes and stresses the influence of mechanical forces acting upon flawed tissue. 2) Inflammatory disease of a bronchiole leads to progressive air trapping and "tension airspaces." 3) Disordered collateral ventilation produces the findings. 4) The same mechanisms responsible for generalized emphysema are operative in the formation of bullae. 5) Underlying paraseptal emphysema produces bullous disease (5).

Bullae within the intact chest are molded and compressed to fit adjacent anatomic configurations.However, if the lung is released from these constraints (e.g., when removed from the chest cavity), bullae project as shiny bubbles at the lung surface.Within the thoracic cavity, large bullae cause crowding of adjacent lung parenchyma, and structures such as bronchi are displaced, stretched, and narrowed over the bullae surfaces.

EVALUATION AND DIAGNOSIS :

In asymptomatic individuals, bullae may be detected in the course of routine chest radiography. Small bullae rarely become visible on the chest radiograph but are usually easily visible by computed tomography (CT). As a rule, small bullae usually produce no symptoms, signs, or discernible alterations in pulmonary function (6). In some patients bullae give rise to progressive dyspnea or chest pain (7),Radiographically identified by air fluid levels.If giant bullae,may lead to localised decrease in air entry,with absent breast sounds and increase in resonance on percussion. Measuring an α1-antitrypsin level should be obtained to diagnose α1-antitrypsin deficiency (8).Finally evaluation of arterial blood gases, performed while the patient is breathing room air if possible, is generally indicated in patients with severe respiratory insufficiency and those being evaluated for possible bullectomy.Chest radiography suggests areas of increased radiolucency that are sharply delineated by fine radiopaque lines representing the walls of the bullae. These lines, or "hairline shadows," are composed of compressed and fused interlobular septae or pleura. CT scans are the most accurate means of detecting emphysema, determining its type and extent and distinguishing giant bullae from pneumothorax.On Chest CT,Bullae are identified as areas of radiolucency that usually do not contain blood vessels and that are confined by visible walls.Also,giant bullae are predominantly located in the upper lobes and are generally subpleural. The major complications of bullous lung disease are fluid accumulation (including infection) in the bulla,spontaneous pneumothorax, bronchogenic cancer, chest pain, and hemoptysis.

TREATMENT :

Many patients with bullous lung disease can be managed medically (9).Chronic bronchitis, asthma, or emphysema associated with bullae require treatment in their own right. For patients with α1-antitrypsin deficiency augmentation therapy with antiproteases may be appropriate. Indications for surgery with giant bullae are (1) increasing bulla size (2) pneumothorax (3) pulmonary insufficiency, and (4) infection within the bulla (10).The most common indication for bullectomy is severe dyspnea due to a bulla occupying 30% or more of the hemithorax or spontaneous secondary pneumothorax (11). Pre-operative evaluation can be done by nuclear medicine techniques.A key factor suggesting that bullectomy may be beneficial is a bulla that occupies greater than 50% of the hemithorax with radiographic evidence that the bulla is compressing adjacent normal pulmonary parenchyma. Candidates not suitable for surgery include older age, ongoing cigarette smoking, significant comorbid disease, lower pulmonary function (FEV1 and DLCO), hypercapnia, poorly defined bullae on chest imaging, and pulmonary hypertension.

CONCLUSION :

This case has been presented in view of the rarity of the presentation of the combination of Bullous Emphysema with Bronchiectasis and

Fungal Ball in the ectatic bullous segment of the lung in a non diabetic patient.

Bullous emphysema is either congenital without general lung disease or a complication of COPD with more or less generalized lung disease. The challenge is to separate the disability related to the bullae from that caused by the chronic emphysema or chronic bronchitis.Careful clinical, radiographic, and physiologic assessment allows identification of patients whose dyspnea may be substantially improved through a variety of medical,surgical, and bronchoscopic interventions.

REFERENCES :

1) Sabiston textbook of surgery – 20 edition
2) Fishman's pulmonary diseases and disorders - 5 edition
3) Laurenzi GA, Turino GM, Fishman AP. Bullous disease of the lung. Am J Med. 1962;32:361–378.
4) Burke R. Vanishing lungs: a case report of bullous emphysema. Radiology 1937;28: 367-71.
5) Fishman's pulmonary diseases and disorders - 5 edition
6) Boushy SF, Kohen R, Billig DM, Heiman MJ. Bullous emphysema: clinical, roentgenologic and physiologic study of 49 patients. Dis. Chest. 1968;54(4):17–24.
7) Martinez F. Evaluation and medical management of giant bullae in COPD. In: Rose B, ed. UpToDate.Waltham, MA: UpToDate; 2013.
8) Stoller JK, Aboussouan LS. A review of α1-antitrypsin deficiency. Am J Resp Crit Care Med.2012;185(3):246–259.
9) Byrd RP Jr, Roy TM. Spontaneous resolution of a giant pulmonary bulla: what is the role of bronchodilator and anti-inflammatory therapy? Tenn Med. 2013;106(1):39–42.
10) Laros CD, Gelissen H1, Bergstein PGM, Van den Bosch JM, Vanderschueren RG, Westermann CJ et al. Bullectomy for giant bullae in emphysema. J Thorac Cardiovasc Surg 1986; 91: 63-70. Back to cited text no. 17 AND Mehran R1, Deslauriers J. Indications for surgery and patient work-up for bullectomy. Chest Surg Clin N Am 1995; 5: 717-34.
11) Meyers B, Patterson G. Chronic obstructive pulmonary disease. 10: bullectomy, lung volume reduction surgery, and transplantation for patients with chronic obstructive pulmonary disease.Thorax. 2003;58:634–638