

Overview Of Pneumothorax And Bullectomy Surgery – The Way Forward.

(Study Of Lungs Cirrhosis – Thoracic Cancer Due To Bullas)

By

Dr RitendraRathore

MBBS , MD , PhD In Medical Oncology

Department Of Oncology

All India Institute Of Medical Science , New Delhi , India.

Under Guidance

Of

Dr Pranav Modi Sir

(M. Ch , Cardio Vascular Thoracic Surgeon – CVTS)

Marengo CIMS Hospital Ahmedabad Gujarat India.

Abstract :-

In this research paper, we would study pneumothorax and bullectomy surgery and also lungs—thoracic cancer due to bullas. This is the study in which we would develop the understanding about the chances of the development of lung cirrhosis and thoracic cancer due to bullae, which is mainly developed because of COPD (Chronic Obstructive Pulmonary Disorder). In this research, we have collected the medical reports of the patient who has gone through bullectomy surgery and removed the bullae from the left side of the upper lobe (lungs). We have done a biopsy test of the removed cells.

This research will reveal certain facts about whether bullous development and the resultant bullae develop the carcinogenic cells or tissues or not. Here in this paper we would develop the basis of understanding about bullas and their relation with lung cirrhosis.

Key Words :-

Lung Cirrhosis, Bullectomy, Bullas , COPD , Biopsy etc.

Note: special thanks to Dr. Pranav Modi, Sir, for his support and cooperation in this research. Without Dr. Pranav Sir's coordination, I would not be able to conduct this research. He provided me all the information and reports (biopsy) for making this research successful and effective. This research is dedicated to Dr. Pranav Modi Sir.

Introduction:

The chronic obstructive pulmonary disorder (COPD) is a heterogeneous disease entity that is linked with multiple comorbidities and systematic manifestation. The pulmonary emphysema is a phenotype of COPD characterized by hyperinflation leading to breathlessness and reduced quality of life.

COPD is one of the leading causes of deaths worldwide (4th leading cause of death).

The pharmacological therapy is the cornerstone and very important for the treatment of the lungs with emphysema. The bullectomy and lung volume reduction surgery (LVRS), which has aimed to reduce hyperinflation and thus lead to optimized the lung function, exercise capacity, and also survival.

The bullectomy is lead to transit alleviation of dyspnea and certain indices of respiratory function that dissipate over time.

The National Emphysema Treatment Trail (NETT), published in 2003, is the largest randomized controlled trial related to LVRS. Overall, the lung volume reduction surgery increased the chance of improved exercise capacity but did not confer a survival advantage over the medical therapy.

The most important thing is that, during the era of bronchoscopic LVRS, some centers have nearby abandoned the use of surgical LVRS.

Atelectasis Vs Pneumothorax.

Both are the conditions in which a collapsed lung can be caused, but both are different somehow in terms of varying the degrees of lung collapse. Atelectasis is total collapse of the lungs due to blockage in the air passages or the pressure in the lungs. This could be due to conditions of anaesthesia, prolonged bed rest, shallow breathing, or underlying lung disease.

Pneumothorax.

When the air leak in the pleural space, the area between the lung and chest wall, caused the lung to collapse. Thus caused by the chest trauma , excess pressure on the lung's , or the lung disease.

The symptoms of the Pneumothorax are....

- 1) Bluish skin due to lack of oxygen.
- 2) Chest tightness.
- 3) Lightheadness.
- 4) Extreme Fatigue.
- 5) Irregular breathing pattern.
- 6) Rapid Heart Beat.
- 7) Shock & Collapse.

=> The chest X-ray will show whether someone has pneumothorax or atelectasis, and according to the symptoms, the treatment could be started.

The other diagnostics methods or tests are....

- 1) Computed Tomography (CT) Scan.
- 2) Bronchoscopy.
- 3) Arterial blood gas
- 4) Pulse Oximetry.

Patient Information.

The information obtained from the patients, which included smoking habits and pre-existing comorbidities. All the patients of the cohort were active smokers or ex-smokers and ceased smoking at least 2 weeks prior to the surgical interventions. The patients with the cardiomyopathy, arterial hypertension and type II non-insulin-dependent diabetes mellitus were adequately controlled with medical treatment prior to the surgical interventions.

Surgical Procedure.

The Retrospective analysis of the patient would be divided into the three groups.

- A) Bullectomy (Group 1 , N = 19).
- B) Unilateral LVRS (Group 2 , N = 31)
- C) Sequential Bilateral LVRS (Group 3 , N = 8)

Definition Of Bullectomy.

=> Bullectomy is defined as atypical lung resection margin between bullae and healthy and potentially affected lung parenchyma.

Lung Volume Reduction Surgery.

=> A unilateral no anatomical lung resection of the most damaged lung segments with a planned volume reduction of 20-30% of the lung's volume.

I. In the case of extended emphysema—both lobes of the lungs (upper and lower)—LVRS included both upper and lower lobes and resections in order to achieve a 20–30% reduction of the lung's volume.

Whether COPD Is responsible for the lung cancer ?

Yes , COPD is significant factor for the lung cancer or lung Cirrhosis.

=> The people with COPD are 2-3 times more likely to develop the lung cancer than Non- COPD.

Note :- About 1 % Of People With COPD develop the lung cancer each year.

Causes :-

COPD and lung cancer share many risk factors including.....

- A) Smoking :- Both linked with Smoking and COPD.
- B) Inflammation:- Due to toxic gases.
- C) Genetic Predisposition :- Both diseases may be linked to the genetic susceptibility.

=> Both COPD and lung cancer are major health concern worldwide.

The relation between lungs and lungs cirrhosis.

=> COPD is a risk factor for lung cancer beyond their shared aetiology.

=> Both are driven by oxidative stress.

=> Both are linked to the cellular aging , senescence and telomere shortening.

=> Both have been linked to genetics predisposition.

=> Both show altered epigenetic regulation of gene expression.

=> The lung cancer and COPD May be different aspects of the same diseases, with the same underlying predisposition, telomere shortening, mitochondrial dysfunction, or premature aging.

=> In the majority of smokers, the burden of smoking may be dealt with by the body's defense mechanisms: anti-oxidants such as superoxide dismutase, anti-proteases, and DNA repair mechanisms.

Note: approximately 50% of the smokers will have their lives curtailed by cigarettes smoked, reducing the life expectancy by 11 minutes, such that males and females each lose an average of 13.2 and 14.5 years of life, respectively.

=> Lung cancer is the leading cause of cancer-related deaths worldwide, with only 16% 5-year survival rates.

=> The number of lung cancer deaths is expected to rise to 10 million deaths per year by 2030.

Lung Cancer :-

The lung cancer is caused by the mutations in oncogenes leading to the proliferation of the mutated cells and the formation of a tumor.

The addition mutations can further transform the benign tumor to the invasive cancer , a process marked.

- A) Metastasis(Spread)
- B) Invasiveness.
- C) Anaplasia (loss of the cell types specific features)

The lungs cancer cells are classified in two segments....

The lung cancer cell is originated from the basal epithelial cell...

- a) Non Small Cell Lung Cancer (NSCLC) – 80 % lung cancer cell with adenocarcinoma accounting for 40 % of total and large cell carcinoma for 5 – 10 %.
- b) Small Cell Lung Cancer (SCLC)–remaining 30 % derive from the squamous cells.

The COPD.

COPD is the progressive and ultimately fatal deterioration of lung function over time.

COPD is the third most common cause of death worldwide in 2010 and ranked 5th worldwide in terms of the burden of the disease.

When the person has COPD and mainly lungs could be damaged due to oxidative stress (both exogenous from smoking and endogenous), inflammatory cytokines release, protease activity (due to protease: anti-protease imbalance), and autoantibody expressions.

The relations between lungs cancer and COPD.

=> Both are related with each other and both are increasing evidence linking the two diseases beyond a common etiology.

=> Both are caused by cigarette smoking.

=> COPD is an independent risk factor for lung carcinoma particularly for the squamous cell carcinoma.

=> The lung cancer is occurred five time more likely occur in smokers with airflow obstruction than those with the normal lung function.

=> **The other factors are the over diagnosis of the COPD which increase the risk of the lungs cancer.**

=> **The Higher Prevalence of the Lung cancer in the COPD that would suggest the common mechanisms such as.....**

- 1) **Premature aging in the lungs.**
- 2) **Genetic Predisposition.**
- 3) **Common pathogenic factors –growth factors , intracellular pathway or epigenetic .**

The disease of the aging lungs – COPD & Lung Cancer.

The probability of the lung cancer can be increased with the age and approximate 66 years is the lung cancer age.

The COPD is affect smokers age of 40 years which is 2.5 times higher in over 60 years old.

The normal declination of the lung function because of premature loss of the lung function. The aging is the principally driven by the failure of the organs to repair DNA damage by Oxidative Stress (the non programmed aging) and from telomeres shortening as a result of repeated cell division.

The Oxidative Stress is caused of the both COPD and Lung Cancer.

The free radicals like Reactive Nitrogen and oxygen species (RNOS) which are free radicals and around 10x15 times more reactivities. The main source of the RNOS's generation is mitochondrial respiration and mitochondrial dysfunction.

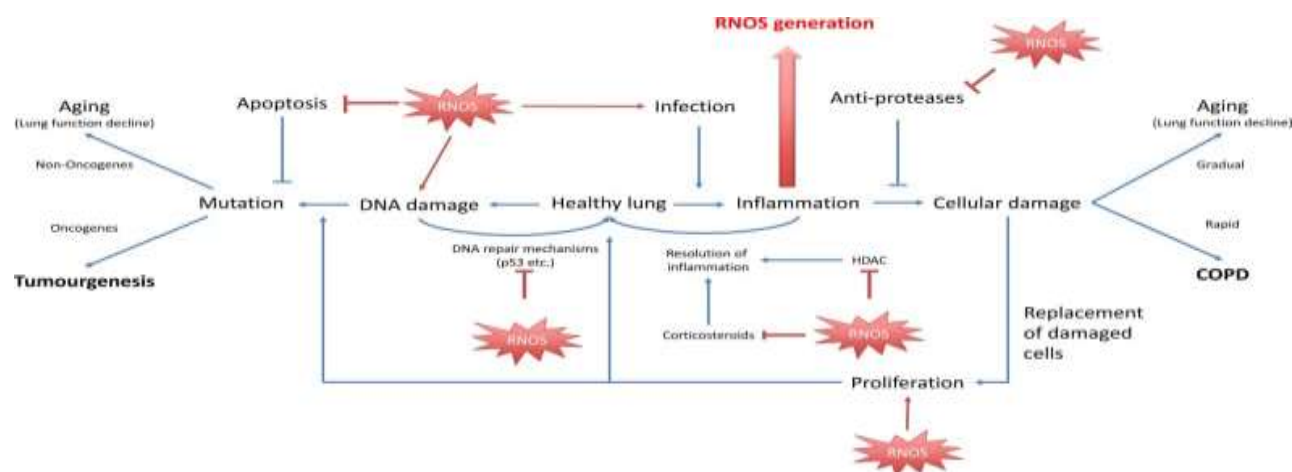
RNOS which damaged the DNA especially mitochondrial DNA , lipid peroxidation , the oxidation of amino acids and oxidation of the inorganic enzyme co- factors.

Oxidative Stress Of The Damages DNA.

The Oxidative Stress drives cancer initiation through DNA Damage :-

- 1) **Point Mutations.**
- 2) **Single Strand Breaks (SSB)**
- 3) **Double Strands Breaks (DSB).**
- 4) **DNA Cross Linking.**

Due to continuous RNOS exposure , which lead number of somatic mutations.



Reactive oxygen and nitrogen species (RNOS) derived from both exogenous and endogenous sources drive many of the pathways in both COPD and lung cancer. RNOS can react with DNA, leading to DNA damage, which if not correctly repaired leads to mutations. Mechanisms that prevent mutation, including DNA repair and apoptosis can be inhibited by RNOS activity. Additionally RNOS can contribute to susceptibility to infection and drive inflammation in the lungs. Inflammation can lead to further cellular and DNA damage, both through the generation of further RNOS and also through the action of cytokines and proteases. RNOS are capable of inhibiting the protective mechanisms, such as anti-proteases. Damage to the lungs is repaired by processes including cellular proliferation, which can in turn promote tumourgenesis.

The Telomere Shortening Of The DNA Replication During the mutation.

Definition Of The Telomere Shorter.

Repetitive nucleotide sequences located at the ends of the chromosomes which against the progressive shortening which would be occurred with the DNA Replication.

Telomere shortening is associated with the cell aging and senescence , with the cells unable to divide once their Telomere shortened beyond the crucial points that is called **HAYFLICK LIMIT**.

The several studies and the research paper shown the link between the short Telomere length and the development of the lung cancer at the same time the evidence that shorter the Telomeres are associated with COPD and the short Telomere which leads the inflammation of the COPD.

Epigenetic in lungs cancer and COPD.

The genetic changes, the epigenetic changes including DNA methylation, covalent histone modification, microRNAs (miRNAs) expressions, and nucleosome remodelling, which is playing a vital role in the development of cancer and COPD. DNA methylation, which is a reversed modification of DNA structure, adds the methylene group to the 5th position of the cytosine residue. The hypermethylation of the tumor suppressor and other gene promoters is observed in the majority of the cancer.

Medical Case Study.

Recently, I came to know one of the medical cases, which is of my father, Mr. N M Rathore. He has been suffering Of COPD and acute respiratory syndrome. Why I am taking my father's case in the consideration ? Because I have been observing this case from the very beginning and I knew each and every aspects of this case.

Since my childhood, I have been seeing my whooping, shortness of breath, asthma, COPD, and extended emphysema. One year before I came to know, there was a development of the bullas in his left lung. After getting an X-ray and CT scan report, there was a bulla in his lung, and the overall size of the bulla was approx. 20 mm. It was huge in size, and I approached Dr. Amit Patel regarding this matter at CIMS Hospital, Ahmedabad, Gujarat, India.



According to the PFT Test , the lung vital capacity was suppressed or being reduced day by day due to that bulla which was found at the left apical bulla (Left lobe of the lung) , hence the frequently reduction of the lung's vital capacity , we would be prescribed to opt for Bullectomy in order to reduce that bulla through the surgical procedure.

Result Of this PFT Test.

FVC (L) – Total 46%

FEV1(L) -26%

FEV1/FVC – 57%

FEF25- 75% (L/S) – 21 %

- ⇒ The value of the outside normal range or significant post change.
- ⇒ There was clearly found the severe obstruction in the respiration due to bulla.

He prescribed us to perform a PFT test, and in the PFT test, we got the result that his left lung has 46% vital capacity and has been reducing day by day; hence he asked us to go for surgery, but at the same time he suggested us to consult with Dr. Pranav Modi, sir, for the final consultation on whether the surgery has been done or not. At that time, Dr. Pranav Modi didn't prescribe going for bullectomy, and 8 months later, the bullas leaked air, and air blocked the entire thoracic passage; hence, immediately he had to go for ICD, and he underwent ICD surgery. When it is done, he feels relaxation; somehow he is feeling well, but we need to remove that bulla from the left lobe of the lungs; hence we approach Dr. Pranav Modi, sir, for further diagnosis. We underwent bullectomy and removed the bullae.

The Other pathological tests and reports.

Marango CIMS Hospital

DEPARTMENT OF LABORATORY SERVICES

Patient: Mr. KATHIRUHAN M BATHOD
Lab No/Barcode: 9625759
Collection Date: 10/12/2024 8:15PM
Age/Gender: 33 Years/Male
Resolving Date: 10/12/2024 7:00PM
Bed No/Ward: HDL 4TH FLOOR EAST
Report Date: 10/12/2024 7:00PM
Referring Doctor: Dr. Praveen Arvindhath Mudi
Report Status: Final
Sample Quality: Final

Test Name	Result	Unit	Ref. Range	Method	Sample
Haematology					
PRE-OPERATIVE PROFILE (MAJOR)					
2019-10-10					
Haemoglobin	13.7	g/dL	13.5 - 16.0	Si-B Method	Result
Hematocrit (HCT)	44.0	%	43.0 - 52.0	W. Hanging Method	
RBC COUNT	5.08	mill/Cmm	4.70 - 6.20	W. Hanging Method	
MCV	87.0	fL	85.0 - 101.0	Calculated	
MCH	27.0	pg	27.0 - 31.0	Calculated	
MCHC	30.9	g/dL	32.0 - 36.0	Calculated	
RDW-CV	11.6	%	11.5 - 14.0	Calculated	
Platelet count	284,000	/cumm	150,000 - 450,000	W. Hanging Method	
Mean Platelet Volume (MPV)	8.9	fL	8 - 12	Calculated	
Total Leucocyte Count (TLC)	11730/CC	/cumm	4300.00 - 10800.00	Flow Cytometry	
Differential Leucocyte Count				Manual Count	
Neutrophils	75	%	40 - 70		
Lymphocytes	18	%	20 - 40		
Eosinophils	02	%	1 - 6		
Basophils	00	%	1 - 4		
Monocytes	05	%	3 - 11		
Immature Granulocytes	00	%	0 - 2		
Peripheral Smear Study					
RBCs are Normochromic & Normocytic. WBCs (Stained) Normal. No Leucocytosis. Platelets are adequate in number. Malaria parasites not seen. No Premature cells are seen.					
Absolute Leucocyte Count					
Neutrophil Count	8798	/cumm	1000 - 7000		
Lymphocyte Count	2100.6	/cumm	1000 - 4000		
Monocyte Count (AEC)	584.4	/cumm	200 - 400		
Basophil Count	0	/cumm	0 - 100		

Dr. Praveen Arvindhath Mudi
Reg No. 10170

Prepared By: RAJESH K
Printed at: 10/12/2024 7:00PM

Marango CIMS Hospital

DEPARTMENT OF LABORATORY SERVICES

Patient: Mr. KATHIRUHAN M BATHOD
Lab No/Barcode: 9625759
Collection Date: 10/12/2024 8:15PM
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Referring Doctor: Dr. Praveen Arvindhath Mudi
Report Status: Final
Sample Quality: Final

Test Name	Result	Unit	Ref. Range	Method	Sample
Biochemistry					
PRE-OPERATIVE PROFILE (MAJOR)					
Glucose	84.20	mg/dL	80 - 110	POCT (Glucose)	Result
Creatinine	1.01	mg/dL	0.70 - 1.20	POCT (Creatinine)	Result
Random Glucose	84.2	mg/dL	< 140.0	POCT (Glucose)	Result
As per HbA1c, Diabetes is diagnosed at Random blood glucose of greater than or equal to 200 mg/dL.					
SERUM ELECTROLYTES					
Sodium (Serum)	138.0	mmol/L	136.0 - 145.0	ISE Method	
Potassium (Serum)	4.69	mmol/L	3.50 - 5.10	ISE Method	
Chloride (Serum)	100.1	mmol/L	98.0 - 107.0	ISE Method	
End of Report					

Dr. Praveen Arvindhath Mudi
Reg No. 10170
Consultant Pathologist

Prepared By: RAJESH K
Printed at: 10/12/2024 7:00PM

Above Blood Test (CBC) and Creatinine Test clearly indicated the all parameter are normal and nothing found serious.

- Haemoglobin – 13.7g/dL.
- RBC Count – 5.08 mill/ Cmm
- Platelet count – 2,84,000 / cumm.
- Mean Platelet Volume (MPV) – 8.9.
- Neutrophils – 75 %
- Lymphocytes – 18 %
- Eosinophils – 02 %
- Monocytes – 05%
- Basophils – 00%
- Immature Granulocytes 00%

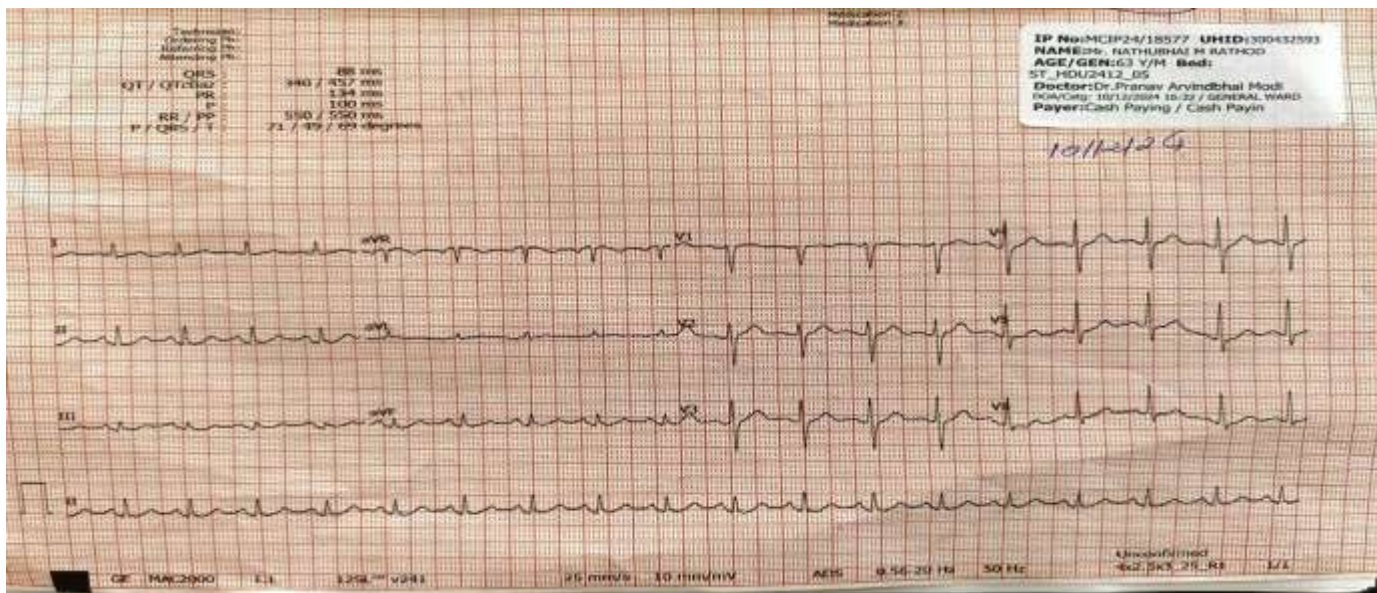
Peripheral Smear Study shows that RBCs are normochromic & Normocytic. WBCs Shows Neutrophilic Leucocytosis. Platelets are adequate in number. malaria parasites are not seen. No premature cells are seen.

As per creatinine test , No Diabetes is diagnosed (Random Blood Glucose of greater than or equal to 200 mg/dL- that shows 84.2 mg/dL.

Serum Electolytes levels were also normal.

- Sodium – 138.0 mmol/L (Normal Range – 136.0-145.0
- Potassium (Serum) – 4.69 mmol/L (Normal Range – 3.50-5.10)
- Chloride (Serum) 100.1 mmol/L (Normal Range – 98.0- 107.0)

The Echo and electrocardiogram Test Report.



Non Invasive Cardiology			
UHID / IP NO	300432593 (MCHP-24/18577)	UHID	300432593
Patient Name	Mr. NATHURAM RATHOD	Age/Gender	63 Y/M
Admitting Doctor	Dr. Pranav Arvindbhai Modi	Treating Doctor	Dr. Pranav Arvindbhai Modi
Cath No.		Date of Procedure	11/12/2024
Procedure Code	Echo Screening For LV Function		

2D ECHO-CARDIOGRAPHY WITH COLOUR DOPPLER.

Comment: Poor echo window

1. Normal sized LA, LV, RA, RV.
2. Normal LV systolic function, LVEF: 55%
3. No significant RWMA.
4. Grade-I diastolic dysfunction.
5. All cardiac valves are structurally normal
6. Mild MR, Trivial TR, Trivial PR, No AR.
7. No PAH.
8. Normal RV systolic function.
9. No clot/vegetation/pericardial effusion.
10. Normal sized IVC.

Dr. Anish Chandarana

DR. ANISH CHANDARANA
(CARDIOLOGIST)

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- ⇒ Both reports found normal , dystolic and systolic functions – LVEF – 55%
- ⇒ All cardiac valves are structurally normal.
- ⇒ Mild MR , Trival TR , Trival PR , No AR.

The specimen or the removed bullas have been sent for Biopsy Test , we have analysed the biopsy.



DEPARTMENT OF LABORATORY SERVICES			
Patient Name	MR. MAHESH K. K. K.	Lab Number	SG257894567890
MR. MAHESH K. K. K.	MR. MAHESH K. K. K.	Collection Date	12/12/2024 9:42PM
Age/Gender	63 Year/Male	Receiving Date	12/12/2024 9:42PM
Bed No/Ward	MR. 4TH FLOOR EACB	Report Date	12/12/2024 12:00PM
Referring Doctor	Dr. Praveen Aravindan Mool	Report Status	Final
Referring Doctor	Dr. Praveen Aravindan Mool		

HISTOPATHOLOGY

Report:

Specimen
 Excised, left apical lung tissue.

Clinical Diagnosis
 Left apical tumor - unspecified.

Gross Description
 Single brownish irregular soft tissue bits - 6x3x2.5 cm. Outer surface shows focally collapsed flap-like bullous wall. Cut surface - Brownish hemorrhage. No solid area. Representative sections taken in 4 blocks (A1-A4).

Microscopic Description
 Shows congested lung tissue with focally dilated bullae lined by flat to cuboidal epithelium with moderate chronic active inflammation and marked recent and old haemorrhage. Adjacent lung tissue shows dilated and ruptured alveoli with marked haemorrhage. There is many congested thin walled blood vessels. No granulomas/honeycomb change/fungi/malignancy.

Diagnosis
 Mild chronic active inflammation with congestion and bulla formation. Excised, left apical lung tissue.
 No granulomas/honeycomb change/fungi/malignancy.


 Dr. Ganesha Manish Shrivastava
 MBBS, MD, FRCP
 Consultant Histopathologist

Prepared By: RAB Processed By: RAB Page: 1 of 1

1. Lab
 2. Histopathology
 3. Clinical Pathology

According to the Biopsy Test Report , The removed bulla at the left lobe of the lung , which found No Granulomas/ honeycomb change / fungi / and malignancy. This report which is clearly indicated that bulla was not contained with the carcinogenic cells / Tissue.

The Microscopic Description Of the bulla which clearly indicated that congested lung tissue with focally dilated bullae lined by flat to cuboidal epithelium with moderate chronic active inflammation and marked recent and old haemorrhage. adjacent lung tissue show dilated and ruptured alveoli with marked haemorrhage. this is many congested thin wall blood vessels. No granulomas / Honeycomb change / Fungi / Malignancy.

Result.

The result of the above test reports clearly show , there was no relation of the lung cancer with bulla. The Biopsy report clearly mentioned " No granulomas / Honeycomb Change / fungi / Malignancy ".

Different Pathology for the bulla's creation and also the creation of the cancerous cell in lungs. lung cirrhosis can increase the risk of developing lung cancer and have a significant impact on the outcomes of the lung cancer patients undergoing surgery.

Lung Bullae and lung cirrhosis both have a different causes for the development. The prime causes of the bullae development are...

- 1) emphysema.
- 2) smoking.
- 3) alpha – 1 anti aspirin deficiency.
- 4) illicit drug use.
- 5) Human immunodeficiency virus (HIV)
- 6) Aging.
- 7) Ritalin Lung - lung damaged by injecting talc – containing methylphenidate.

It has clearly shown that different factors are responsible for lung cancer and lung bullae development.

Conclusion :-

when we conclude this research , we understand the different pathological development for the lung cancer and lung cirrhosis but the COPD is responsible for the development of the Lung Cancer and Cirrhosis but there was not found any links between Lung's Bullae and Lung Cirrhosis.

Author Opinion :-

The COPD (Chronic Obstructive Pulmonary Disorder) which has sub type Emphysema , which is a group of lung disease that make it hard to breathe however the lung's bullae are not accountable for the lung cirrhosis but the prolonged or chronic emphysema is the cause of the lung cirrhosis or cancer. The emphysema which damages the air sacs in the lungs. symptoms include shortness of breath, wheezing and a cough that produces a lot of mucus.

we have to develop the understanding bullae are because of emphysema and COPD but Bullae wont be considered as the cell's of cancer. chronic bronchitis and emphysema could be the cause of lung cirrhosis.

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