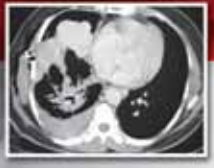
101 CASES IN RESPIRATORY MEDICINE



Supriya Sarkar

JAYPEE

101 Cases in Respiratory Medicine

Supriya Sarkar
MD (Pulmonary Medicine)
Professor and Head
Department of Chest Medicine
College of Medicine and Sagore Dutta Hospital
Kolkata, West Bengal, India

The Health Sciences Publisher
New Delhi | London | Panama

Jaypee Brothers Medical Publishers (P) Ltd

Headquarters

Jaypee Brothers Medical Publishers (P) Ltd
4838/24, Ansari Road, Daryaganj
New Delhi 110 002, India
Phone: +91-11-43574357
Fax: +91-11-43574314
Email: jaypee@jaypeebrothers.com

Overseas Offices

J.P. Medical Ltd
83 Victoria Street, London
SW1H 0HW (UK)
Phone: +44 20 3170 8910
Fax: +44 (0)20 3008 6180
Email: info@jpmedpub.com

Jaypee-Highlights Medical Publishers Inc
City of Knowledge, Bld. 235, 2nd Floor, Clayton
Panama City, Panama
Phone: +1 507-301-0496
Fax: +1 507-301-0499
Email: cservice@jphmedical.com

Jaypee Brothers Medical Publishers (P) Ltd
17/1-B Babar Road, Block-B, Shaymali
Mohammadpur, Dhaka-1207
Bangladesh
Mobile: +08801912003485
Email: jaypeedhaka@gmail.com

Jaypee Brothers Medical Publishers (P) Ltd
Bhotahity, Kathmandu
Nepal
Phone: +977-9741283608
Email: kathmandu@jaypeebrothers.com

Website: www.jaypeebrothers.com
Website: www.jaypeedigital.com

101 Cases in Respiratory Medicine

First Edition: **2018**

ISBN: 978-93-5270-311-1

Printed at

Dedicated to

My friend, philosopher and guide,
my teacher *Late* Professor Subal Kargupta
and
My beloved
future generations of pulmonologists

Preface

As a teacher, I feel, it is my utmost duty to transmit my knowledge and experience to the future generations. I received lots of love, admiration and respect from my students. I have tried to teach them with my best capabilities. Often, I feel, in this teaching–learning process, I have learnt more than I have taught.

In my long journey, I have received blessings and affections from teachers. My teachers have facilitated and guided me in this long teaching–learning process. During this long journey, sometimes I like to believe, 'I have mastered the art of clinical medicine'. But next day, some of my patients or students have unequivocally showed me the hard reality that I know nothing. Thereby I get enriched by my patients as well as by my students. With their love and respect, my patients by sharing their pains and sufferings have made my journey on clinical medicine interesting and challenging.

In this rapidly changing global scenario, medical science has changed a lot. I personally feel that clinical medicine cannot be confined to history and clinical examination, but it should include interpretation of investigations on the backgroud of clinical setting.

The book is intended primarily to the postgraduate students and residents. In this book, I have presented a longitudinal view of sufferings of 101 of my patients. I have narrated their story as they were unfolded before me, and I have included my successes as well as my mistakes also. To make readers understand my narratives, I have written chapters on 'Clinical Approach Respiratory System' and 'Clinical Data Analysis'.

This book is a small and humble endeavor from my side to transmit my knowledge and experience to future generations of pulmonologists. I will be happy, if my attempts reach them and help them in their day-to-day clinical practice. Education means a combination of knowledge, attitude and practice. Junior doctors have treasures of knowledge, but they do not know how to apply them. I think this book will help them in filling those gaps.

Supriya Sarkar

Acknowledgments

I acknowledge all my teachers *Late* Professor Subal Kargupta, *Late* Professor D Sen, *Late* Professor RS Bhakta, *Late* Professor Samir Kumar Saha, Professor AG Ghoshal, Professor M Maitra, Professor RS Mukherjee, Professor Sumita Mukherjee, Professor Somenath Kundu, Professor Atin Dey, Dr PS Bhattacharyya and Professor Arup Kumar Kundu, who have taught and encouraged me to enter into the field of clinical medicine.

I am especially thankful to Professor Atin Dey, who has helped me in writing the chapter on clinical approach. I am also thankful to Professor Arup Kumar Kundu, who have encouraged me and taught me the art of writing.

I acknowledge my colleagues of different medical colleges, where I have worked, i.e. Calcutta National Medical College, North Bengal Medical College and NRS Medical College, for their help and contribution in achieving diagnosis. I am thankful to my friend Professor Amitava Sengupta for two cases admitted in our college under his care. I earnestly thank Departments of Thoracic Surgery, Oncology, Radiology, Pathology, Microbiology and Biochemistry of medical colleges and private institutes for their contribution in those cases. I should be especially thankful to all my colleagues of college of Medicine and Sagore Dutta Hospital, for their cooperation in writing the book.

I am thankful to my friend Dr S Guha, Thoracic Surgeon, for his help. I especially acknowledge my colleagues and staff of different private hospitals of Kolkata, for helping me to learn clinical medicine and for helping my patients with their knowledge, expertise and skills.

I am thankful to my junior colleagues, junior doctors and postgraduate students, who have helped me to achieve diagnosis and thereby have enriched my knowledge.

I am greatly thankful to my patients, who are the main contributors in the book. I express my gratitude for their love, respect and blessings. I specially acknowledge their help in my long teaching–learning process in clinical medicine.

Contents

Abbreviations

°C	-	Degree Centigrade
°F	-	Degree Fahrenheit
µg	-	Microgram
ABG	-	Analysis Blood Gas
ABPA	-	Allergic Bronchopulmonary Aspergillosis
ACTH	-	Adrenocorticotropin Hormone
ADA	-	Adenosine Deaminase
AECOPD	-	Acute Exacerbation of COPD
AFB	-	Acid Fast Bacilli
AFP	-	Alpha Fetoprotein
AIDS	-	Acquired Immunodeficiency Syndrome
AMI	-	Acute Myocardial Infarction
ANA	-	Antinuclear Antibodies
anti-CCP	-	Anti-cyclic Citrullinated Peptides
anti-Scl 70	-	Anti-topoisomerase Antibody
ARDS	-	Acute Respiratory Distress Syndrome
ART	-	Antiretroviral Treatment
ATD	-	Antitubercular Drugs
AV malformation	-	Arteriovenous Malformation
B	-	Basophils
BAL	-	Bronchoalveolar Lavage
BiPAP	-	Bi-level Positive Airway Pressure
BMI	-	Body Mass Index
BOOP	-	Bronchiolitis Obliterans Organizing Pneumonia
BP	-	Blood Pressure
CAP	-	Community-acquired Pneumonia
CAT score	-	COPD Assessment Test Score
CAT	-	Category
CBG	-	Capillary Blood Glucose
CBNAAT	-	Cartridge-based Nucleic Acid Amplification Test
CCAM	-	Congenital Cystic Adenomatoid Malformation
CECT	-	Contrast-enhanced Computed Tomography
CMI	-	Cell-mediated Immunity
COPD	-	Chronic Obstructive Pulmonary Disease
CPAP	-	Continuous Positive Airway Pressure
CT	-	Computed Tomography

DAH	-	Diffuse Alveolar Hemorrhage
DIP	-	Desquamative Interstitial Pneumonia
dL	-	Deciliter
DOTs	-	Directly Observed Treatment Short-course
DPLD	-	Diffuse Parenchymal Lung Disease
dsDNA	-	Double-Stranded DNA
DTH	-	Delayed-type Hypersensitivity
E	-	Eosinophils
EBUS	-	Endobronchial Ultrasonography
ECG	-	Electrocardiography
ELISA	-	Enzyme-linked Immunosorbent Assay
EPAP	-	Expiratory Positive Airway Pressure
ESR	-	Erythrocyte Sedimentation Rate
ET tube	-	Endotracheal Tube
ETB/E	-	Ethambutol
FDG-PET	-	^{18}F-Fluoro-2-Deoxyglucose Positron Emission Tomography
$FEF_{25-75\%}$	-	Forced Expiratory Flow Rate at 25–75% of the pulmonary volume
FEV_1	-	Forced Expiratory Volume in 1st Second
FEV_1/FVC	-	Ratio between FEV_1 and FVC
FiO_2	-	Fraction of Inspired Oxygen
FNAC	-	Fine Needle Aspiration Cytology
FOB	-	Fiberoptic Bronchoscopy
FVC	-	Forced Vital Capacity
GOLD	-	Global Initiative for Obstructive Lung Disease
HAART	-	Highly Active Anti-retroviral Therapy
HAP	-	Hospital-acquired Pneumonia
Hb	-	Hemoglobin
HCAP	-	Health Care Associated Pneumonia
hCG	-	Human Chorionic Gonadotropin
HCO_3^-	-	Bicarbonate
HDL	-	High Density Lipoprotein
HIV	-	Human Immunodeficiency Virus
HP	-	Hypersensitivity Pneumonia
HRCT	-	High-resolution Computed Tomography
I:E ratio	-	Inspiratory-expiratory Ratio
IBW	-	Ideal Body Weight
ICS	-	Inhaled Corticosteroids
ICU	-	Intensive Care Unit
Ig	-	Immunoglobulin
ILD	-	Interstitial Lung Disease
IMV	-	Invasive Mechanical Ventilation
INH/H	-	Isoniazid

IPAP	-	Inspiratory Positive Airway Pressure
IPF	-	Idiopathic Pulmonary Fibrosis
IRIS	-	Immune Reconstruction Syndrome
IV	-	Intravenous
JVP	-	Jugular Venous Pressure
K^+	-	Potassium
L	-	Liter
L	-	Lymphocytes
LABA	-	Long-acting Beta-2 Agonist
LAM	-	Lymphangioleiomyomatosis
LAMA	-	Long-acting Muscarinic Receptor Antagonist
LDH	-	Lactate Dehydrogenase
LDL	-	Low Density Lipoprotein
LFT	-	Liver Function Test
LIP	-	Lymphoid Interstitial Pneumonia
LVF	-	Left Ventricular Failure
LVH	-	Left Ventricular Hypertrophy
M	-	Monocytes
M.	-	*Mycobacterium*
MCTD	-	Mixed Connective Tissue Disease
MDCT	-	Multi-detector CT
MDR	-	Multi-drug Resistant
mEq	-	Milli-equivalent
Mg^{++}	-	Magnesium
MGIT	-	Mycobacterial Growth Indicator Tube
mL	-	Milliliter
mmol	-	Millimol
mMRC	-	Modified Medical Research Council
MODS	-	Multi-organ Dysfunction Syndrome
MRI	-	Magnetic Resonance Imaging
MRSA	-	Methicillin Resistant *Staphylococcus aureus*
N	-	Neutrophils
Na^+	-	Sodium
NIV	-	Non-invasive Ventilation
NNRTI	-	Non-Nucleoside Reverse Transcriptase Inhibitors
NRTI	-	Nucleoside/Nucleotide Reverse Transcriptase Inhibitors
NSAIDs	-	Nonsteroidal Anti-inflammatory Drugs
NSCLC	-	Non-small Cell Lung Cancer
NSIP	-	Nonspecific Interstitial Pneumonia
NT pro BNP	-	N-Terminal B-Type Natriuretic Peptide
O_2	-	Oxygen
OAD	-	Obstructive Airway Disease
OSA	-	Obstructive Sleep Apnea

PA view	-	Posteroanterior View
$PaCO_2$	-	Partial Pressure of Arterial Carbon Dioxide
PAH	-	Pulmonary Arterial Hypertension
PaO_2	-	Partial Pressure of Arterial Oxygen
PAS	-	Para-aminosalicylic Acid
PCR	-	Polymerase Chain Reaction
PEEP	-	Positive End-expiratory Pressure
PEFR	-	Peak Expiratory Flow Rate
PET- CT	-	Positron Emitting Tomography-Computed Tomography
pg	-	Picogram
PIE	-	Pulmonary Infiltrates with Eosinophilia
PLCH	-	Pulmonary Langerhans Cell Histiocytosis
PMDT	-	Programmatic Management of Drug Resistant Tuberculosis
PMF	-	Progressive Massive Fibrosis
PO	-	Per Oral
PPI	-	Proton Pump Inhibitor
Pplat	-	Plateau Pressure
PS	-	Pressure Support
PTE	-	Pulmonary Thromboembolism
PZN/Z	-	Pyrazinamide
RA Factor	-	Rheumatoid Arthritis Factor
RA	-	Rheumatoid Arthritis
RB	-	Respiratory Bronchiolitis
RBC	-	Red Blood Corpuscle
RB-ILD	-	Respiratory Bronchiolitis–Interstitial Lung Disease
RICU		Respiratory Intensive Care Unit
RIF/R	-	Rifampicin
RNTCP	-	Revised National Tuberculosis Control Programme
RR	-	Respiratory Rate
SABA	-	Short-acting Beta-2 Agonist
SABD	-	Short-acting Bronchodilators
SACE	-	Serum Angiotensin-converting Enzyme
SAMA	-	Short-acting Muscarinic Receptor Antagonist
SaO_2	-	Oxygen Saturation in Arterial Blood
SBT	-	Spontaneous Breathing Trial
SCLC	-	Small-cell Lung Cancer
SGOT/AST	-	Aspartate Aminotransferase
SGPT/ALT	-	Alanine Aminotransferase
SIRS	-	Systemic Inflammatory Response Syndrome
SLE	-	Systemic Lupus Erythematosus
SM/S	-	Streptomycin
SPN	-	Solitary Pulmonary Nodule

SpO_2	-	Oxygen Saturation in Peripheral Blood
SVC	-	Superior Vena Cava
TB	-	Tuberculosis
TBLB	-	Transbronchial Lung Biopsy
TBNA	-	Transbronchial Needle Aspiration
TNM	-	Tumor-Lymph Node-Metastasis
TR grading	-	Tricuspid Regurgitation Grading
TU	-	Tuberculin Unit
Tv	-	Tidal Volume
U/L	-	Unit/Liter
UIP	-	Usual Interstitial Pneumonia
USG	-	Ultrasonography
VAP	-	Ventilator-associated Pneumonia
VILI	-	Ventilator-associated Lung Injury
WBC	-	White Blood Corpuscle
XDR-TB	-	Extensively Drug Resistant Tuberculosis
ZN stain	-	Ziehl-Neelsen stain

Introduction

"Pustakastha tu ja vidya para hastha gatam dhanam
Karjya kale samutpanne na sa bidya na tat dhanam"

The shloka means bookish knowledge, like occupied wealth, is of no use at the time of needs. It is not possible to learn medicine from book/internet. For learning medicine, you have to go to the hospital wards and connect with patients. Only patients can teach you medicine and in return they demand a little care and empathy. Here teachers' job is to facilitate the teaching–learning process. You have to listen to her story and you have to understand his clinical setting and then only you can learn medicine.

Young doctors are often confused whether they should adapt the shortcut, easy and glamorous way of modern technologies, or they should take the long, difficult and cumbersome way of clinical medicine. My feeling is that those who can converge clinical medicine with modern technologies will be a master in the field of medicine.

It is often wrongly perceived that clinical medicine will automatically die in the era of modern technology and instrumentalization. I beg to defer and I think clinical medicine is now becoming more relevant. Your every decision-making step will be challenged by modern technologies. There will always be a plethora of causes for a particular manifestation. You cannot ask for a battery of investigations due to socioeconomic constrain and other reasons. Any delay or unnecessary investigation will be challenged by patients.

In the era of information technology, information is available everywhere. Anybody can just download them and learn them. So, at a particular time, on a particular disease, patient/patients' relative may have gather more knowledge than the treating physician. Only your hold on clinical medicine and your interpretation skill will establish your supremacy.

I truly feel, clinical medicine needs to be reinvented. Now, clinical medicine cannot be confined to history-taking and physical examination with inspection, palpation, percussion and auscultation. Clinical medicine should include interpretation of history, clinical findings and investigation results (chest X-ray, CT scan, ABG, spirometry, sputum report, pleural fluid reports and other relevant tests). Deep-seated lesions surrounded by normal lung parenchyma may not have any sign and only chest X-ray can unveil them. HRCT-thorax has revolutionized the outlook on DPLD. Obstructive airway diseases cannot be diagnosed without spirometry. The diagnosis of tuberculosis needs sputum examination. Analysis of pleural fluid reports is an essential step in pleural diseases.

My book has started with a small chapter on clinical approach to respiratory medicine followed by a chapter on clinical data analysis and finally by hundred-one examples of cases, taken from life. These cases, I am presenting, are not mere case reports, but the pains and sufferings of some of my patients. I have gone step-by-step as these cases are explored along with my impression and background knowledge. I have also depicted my mistakes what, did and what I have learned from these mistakes. I request readers to be empathic to my patients and have an illuminating journey with them. I will be a proud facilitator in your learning process.

Reading is of two types. Before examination, we read with aims to commit to memory and to vomit in the examination hall. The process is cumbersome, strenuous and boring. I believe that after getting degrees, the true examination begins where every patient will be our examiner. Secondly, reading is for learning and enjoying the process of getting a rim of knowledge. The process is a nice, charming and smooth journey. I want my readers to be in the second category. Please enjoy the world of clinical medicine.

In my book, I am sharing my learning process with you, helps you in your day-to-day clinical practice, my endeavor would be successful.

CHAPTER 3

Cases

RESPIRATORY DISEASES DISCOVERED ACCIDENTALLY BY CHEST RADIOLOGY

CASE 1: ASYMPTOMATIC MAN WITH HILAR CALCIFICATION

CASE REPORT

A 45-year-man had upper abdominal pain and ultrasonography (USG) detected gallstones. Surgeon planned cholecystectomy. For preoperative checkup a chest X-ray postero-anterior (PA) view was done and that showed some abnormalities. He was referred to chest department for opinion.

He was a non-smoker and did not have any respiratory complain or past history of respiratory disease. His general survey and respiratory system examination did not reveal any abnormality. His chest X-ray PA view (Fig. 1)

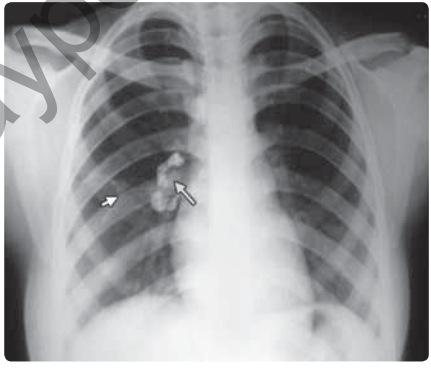


Fig. 1: Chest X-ray PA view showing primary complex with large hilar (long arrow) and small parenchymal calcification (small arrow)

showed an irregular calcification at right hilum suggestive of calcification of right hilar lymph nodes. A careful search into the X-ray detected an irregular small calcified opacity in the right mid-zone. Lung parenchymal calcification was smaller than right hilar gland calcification.

Discussion

Tuberculosis (TB) is caused by *Mycobacterium tuberculosis* and seven other closely related species of mycobacterium (*M. bovis, M. africanum, M. microti, M. caprae, M. pinnipedii, M. canetti* and *M. mungi*). TB bacilli spread as droplet nuclei (1–5 microns) generated during coughing, sneezing and talking of TB patients (pulmonary and laryngeal). Each aerosol droplet contains 1-400 bacilli. When a person inhales the droplet nuclei, bacilli reach alveoli of the lungs. In first time infection, human immune system is not ready to deal with them. After initial nonspecific immune response, alveolar macrophages come into action; they engulf bacilli and phagosomes are formed. Bacilli can survive within phagosomes either by preventing their fusion with lysosomes or by surviving within phagolysosomes. The capacity of bacilli to survive within adverse situation is due to the peculiarity and integrity of their cell wall. Cell wall of TB bacilli consists of a peptidoglycan layer covered by arabino-galactan layer, and they are covalently linked to an outward layer of mycolic acids and long chain fatty acids. This cell wall skeleton forms a waxy, non-fluid barrier that restricts penetration of both hydrophobic and hydrophilic molecules.

TB bacilli multiply within macrophages and ultimately macrophages burst. Bacilli then spread to regional lymph node and reach systemic circulation via thoracic duct. Thereby bacilli spread to different organs of human body. In the meantime, antigen presenting cells (dendritic cells and alveolar macrophages) come into action; engulf and carry bacilli to hilar lymph nodes; and present them to TH_1 lymphocytes. Ultimately cell mediated immunity (CMI) and delayed-type hypersensitivity (DTH) develops by 2 to 6 weeks time. There is a basic difference between CMI and DTH. CMI is due to cytotoxic T lymphocytes and natural killer cells capable of lysing macrophages infected with TB bacilli and killing individual bacteria. DTH kills bacilli en masse along with destruction of surrounding tissues and thereby forming caseous necrosis.

Tubercular granuloma contains central caseous necrosis surrounded by epithelioid cells, Langerhan giant cells and T lymphocytes with an outer layer of fibroblasts. TB bacilli are contained within granuloma by acidic pH, poor oxygen level and toxic fatty acids. Primary complex consists of a larger glandular component and a smaller parenchymal component. Previous textbooks described an additional 3rd lymphatic component of primary complex.

Fate of primary complex depends on the balance between virulence of bacilli on one side and CMI on the other side. More than 90% of primary complex heals spontaneously and they may be calcified. Within the focus bacilli remain dormant for long time. In favorable condition they may start multiplying causing post-primary TB. Primary complex may progress causing

progressive primary TB; it may rupture into surrounding structures causing pleural effusion, pericardial effusion; hilar lymph nodes may compress bronchus in children causing consolidation and collapse of distal lung (epituberculosis); in low immunity state it may progress to military TB; and focus in different organs may be activated causing TB of that organ or surrounding structures like meningitis.

In our country, about 38% people are infected with TB and infection usually occurs in childhood. Primary TB may be totally asymptomatic or may cause symptoms like viral fever. In most of the cases, they heal spontaneously and remain asymptomatic. Primary TB can be identified by positive tuberculine test. Healed primary complex can be accidentally detected by chest X-ray as calcified hilar glandular and lung parenchymal lesions. Characteristically glandular components are much more prominent than lung parenchymal component. Approximately 10% of healed primary complexes are activated in individual's lifetime. In persons living with HIV, the rate of activation increased to about 10% per year. In general no treatment is required for healed primary complex.

My Learning

Radiological findings of primary complex, a common condition in our country, is typical but we are less aware of.

Management

We diagnosed the case as healed primary complex, reassured the patient and sent him back to surgeon for gallbladder surgery.

CASE 2: CALCIFICATION IN SOLITARY PULMONARY NODULE

CASE REPORT

A 50-year-male smoker had right sided inguinal hernia. For preoperative checkup he had an X-ray chest (Fig. 2), and that showed a solitary pulmonary nodule (SPN) in the right mid-zone. He was referred to department of pulmonary medicine for preoperative checkup.

On enquiry he told that he had cough with little mucoid expectoration for more than 2 years. He was a smoker and he smoked about 10 cigarettes per day for more than 20 years. He ignored cough as smoker cough. He denied any other respiratory symptom including dyspnea, wheeze, chest pain or hemoptysis. His general survey and examination of respiratory system did not reveal any abnormality. Examination of sputum was negative for acid fast bacillus (AFB). Spirometry was done as preoperative checkup as he was a smoker and spirometry findings were within normal limits.

Discussion

Solitary pulmonary nodule is defined as a single nodule that is not more than 3 cm in diameter usually surrounded by lung parenchyma. Opacity more than

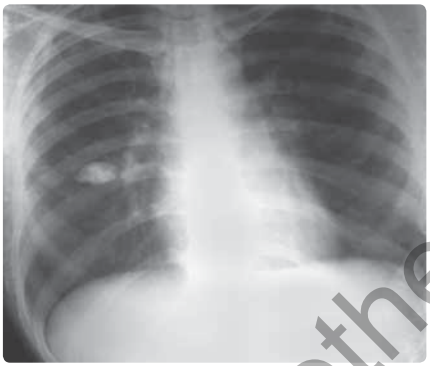


Fig. 2: Chest X-ray PA view showing solitary pulmonary nodule with popcorn calcification

3 cm in diameter is generally considered as mass lesion. Causes of SPN are malignant neoplasm, benign neoplasm, inflammatory granulomas and some less common causes like arteriovenous malformation, pulmonary infarct, lung cysts, etc. SPN is an important clinical problem and main issue here is to rule out malignancy. SPN needs a detailed evaluation as early detection of malignancy and surgical management will improve its prognosis. SPN should be carefully examined regarding its size, shape, border, density, homogeneity, calcification and rate of growth.

Previous X-ray if available should be examined and compared with present X-ray to assess the rate of growth (tumor doubling time). Tumor doubling time is the time taken by the tumor to double its volume (measured as 1.25 time increase in diameter of tumor). Less than a month doubling time usually indicate inflammatory lesion whereas a doubling time more than 18 months indicates benign tumor. Doubling time in between should be evaluated for malignancy. No change in tumor size when compared with old X-ray done before 1 year or more, usually suggests benign tumor.

Regarding size, incidentally detected SPNs of less than 6 mm diameter are usually benign and more than 3 cm goes in favor of malignant tumor. Lobulated structure indicates benign lesion. Distinctly smooth border goes in favor of benign lesion. Satellite lesions are also important as they may give some clue, and they are usually found in tubercular lesion.

Calcification within SPN usually indicates benign lesion but does not rule out malignancy, particularly in eccentric calcification. Specific types of calcification as central, diffuse, stippled, laminar, concentric or popcorn calcification indicate benign lesion. High-resolution computed tomography (HRCT) thorax or sometimes contrast-enhanced computed tomography (CECT) thorax are useful in detailed evaluation of SPNs. Biopsies and

resections by thoracoscopy or thoracotomy should be considered for nodules more than 3 cm or lesions less than 3 cm without clear-cut evidence suggestive of benign lesion.

Ultimately the probability of cancer to be estimated considering nodule size, location, growth rate (doubling time < 20 days or > 400 days), margin characteristics, age of the patient, smoking history, prevalence of malignancy in the community, and occult calcification on computed tomography (CT) densitometry. ^{18}F-fluoro-2-deoxyglucose positron emission tomography (FDG-PET) has sensitivity of 97% and specificity of 78% for diagnosis of malignancy in nodules ≥ 10 mm in diameter. FDG-PET can have false negative result in carcinoid, bronchoalveolar cell carcinoma and small nodules less than 10 mm in diameter. On the other hand, false positive results may be found in inflammatory granulomas like tuberculosis, histoplasmosis and rheumatoid nodules.

Four imaging observations reasonably exclude malignancy: (i) benign pattern of calcification, (ii) rate of growth too short or too long, (iii) specific shape indicating benign lesion and (iv) equivalent evidence suggesting of benign process like infarction or granuloma.

Concentric calcification (laminated) is specific for tuberculosis and fungal granuloma. Punctate calcifications are caused by granuloma, hamartoma, amyloidoma, carcinoid and metastasis (osteosarcoma). Eccentric calcification may occur in bronchial carcinoma. Uniform calcification is virtually diagnostic of granuloma. Fat density within SPN is almost diagnostic of hamartoma and very rarely it can occur in lipoid pneumonia and metastatic liposarcoma. Contrast enhancement favors malignant neoplasm than benign tumor.

Popcorn calcifications are randomly distributed and overlapping small rings of calcification suggest the presence of cartilage as in hamartoma and cartilage tumor. Hamartoma is a benign neoplasm probably derived from bronchial wall mesenchymal cell characterized by the presence of cartilage, loose fibroblastic tissue and adipose tissue. Some pathologists consider it as developmental anomaly. They are sharply defined smoothly marginated nodule with focal areas of fat density and multiple coarse foci of calcification or popcorn calcification. Though popcorn calcification is characteristic it is not a common finding. CT thorax can make a confident diagnosis in 50–60% cases by demonstrating focal areas of fat density in smooth marginated nodule. Definite diagnosis can be made by transthoracic core needle biopsy for peripheral hamartomas and bronchoscopic biopsy for endobronchial hamartomas. Management of hamartoma is not surgical but by giving assurance that it will not cause any harm. In intrabronchial hamartoma causing obstruction and collapse of lung, surgery may be required.

Management

His chest X-ray PA view (Fig. 2) showed a SPN in the right mid-zone less than 3 cm in diameter with clear lobulated margin without satellite lesion and hilar enlargement. There was popcorn calcification within the nodule. We diagnosed

the case as hamartoma. We assured the patient that it was a benign tumor and it will not cause harm. We advised him to stop smoking and gave him symptomatic treatment for cough. We gave him fitness clearance for hernia operation after two weeks of smoking cessation.

My Learning

It is the specific type of calcification not merely the presence of calcification in SPN that exclude malignancy.

CASE 3: ASYMPTOMATIC SPN IN SMOKER

CASE REPORT

Step I

A 55-year-old man was diagnosed as chronic cholecystisis. He was admitted in the surgery in-patient department for cholecystectomy. As preoperative check-up he was advised a chest X-ray. The chest X-ray came one day before surgery, and X-ray was reported as round opacity in right upper zone. The case was referred to us for opinion.

He was a smoker about 5–10 cigarettes per day for about 25 years, and he stopped smoking for about 2 weeks as advised by anesthetist. He was a school teacher. He denied any respiratory symptoms except occasional cough. He had no other significant history. General survey and systemic examination failed to reveal any abnormality. His routine blood and biochemistry were within normal limits. His chest X-ray PA view (Fig. 3A) showed one faintly outlined, low density, small round opacity at the anterior end of right second rib. His spirometry was normal. Patient denied any past illness; he never gone to doctor before and he never had a chest X-ray previously.

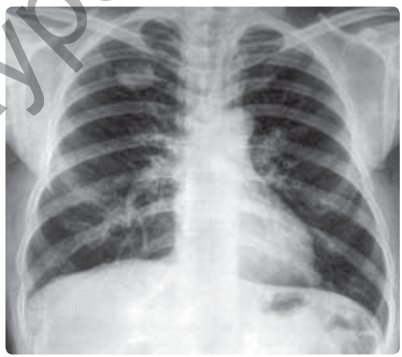


Fig. 3A: Chest X-ray PA view showing a solitary pulmonary nodule in right upper zone

Discussion

In a 55-year-old smoker without having any respiratory symptoms and signs, a SPN was accidentally detected during routine preoperative checkup. In persons aged more than 35 years with or without smoking history, SPN should not be ignored and must be investigated properly. If old X-ray was available we could have assessed the tumor doubling time. Roughly speaking if no change in tumor size was found over a period of 1 year or more, we could ignore that opacity as benign lesion.

Lungs have no pain receptor and pain in lung cancer occurs when it invades mediastinum or chest wall. Lungs have a large physiological reserve, so manifestations of lung cancer occur late. Early detection and management is the key in success of lung cancer management. Lung cancer detected as SPN, stage –I cancer, has a good prognosis with 10 years survival more than 88%.

Step II

I started with CECT-thorax, a non-invasive test. CECT-thorax (Fig. 3B) showed a round, heterogeneous, nodular lesion with smooth regular margin in right upper lobe that took little contrast. There was no satellite lesion or mediastinal lymphadenopathy. CT-guided fine-needle aspiration cytology (FNAC) suggested the diagnosis of adenocarcinoma of lung.

There was no sign of metastasis, no pleural involvement or mediastinal involvement. Fiber-optic bronchoscope did not show any intraluminal lesion up to sub-segmental bronchi. TNM (tumor-node-metastasis) classification was T_{1b}, N_0, M_0. We staged the tumor as stage I lung cancer. There was no history of myocardial infarction or cardiac disease. His forced expiratory volume in 1st second (FEV_1) was 1.8 liter (L) and his arterial blood gas (ABG) analysis findings were within normal limits (without carbon-dioxide retention).

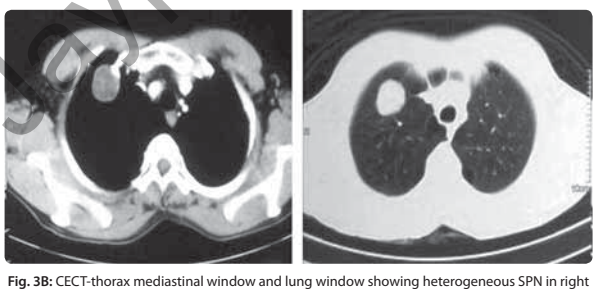


Fig. 3B: CECT-thorax mediastinal window and lung window showing heterogeneous SPN in right upper lobe

Discussion

A SPN in elderly smoker should be investigated for evidence of malignancy. At that stage surgery is possible and prognosis will be good. SPN should be considered benign with smaller size, regular margin and denser lesion. Calcification if present usually indicate benign lesion but did not rule out malignancy. Some patterns of calcifications like egg cell, popcorn, central, laminated, etc. suggest benign tumor. Homogeneous nodule may be benign or malignant but the chance of malignancy increased with heterogeneous lesion. Non-calcified nodules are classified into pure ground-glass, partly solid and solid lesions. Possibility of malignancy in pure ground-glass nodules varies from 18 to 60%. Alveolar cell carcinoma or atypical adenomatous hyperplasia can present as ground-glass opacity. Chance of malignancy in partly solid nodule is very high. Solid nodules are usually benign but when occur in persons aged more than 35 years and in smokers should be evaluated.

The risk of lung cancer in SPN depends on diameter (more the diameter more the risk); age of the patient (our patient aged 55 years had intermediate risk); smoking status (our patient smoking less than 20 cigarettes per day had intermediate risk); smoking cessation status (duration of smoking cessation above or below 7 years); and character of nodule margin (low risk for smooth, intermediate risk for scalloped and high risk for corona radiata or spiculated margin). In our case, all findings were nonspecific but they indicated an intermediate to high risk for malignancy. To confirm the diagnosis invasive procedures should be adopted. CT-guided FNAC/biopsy should be the first choice; and open thoracotomy or video-assisted thoracoscopy should be considered if CT-guided procedures failed.

Management

We confirmed the diagnosis of adenocarcinoma by CT- guided FNAC. The next step should be anatomical staging of tumor to find out resectibility of tumor. We staged the tumor as T1b as the tumor was surrounded by lung parenchyma without invasion into proximal part of lobar bronchus, and the size of SPN was more than 2 cm but less than 3 cm in diameter. We classified the tumor as N_0 and M_0 as there was no evidence of lymph node involvement or metastasis.

We searched for any contraindications for surgery like recent myocardial infarction. He was ambulatory with normal lung function. His ABG was within normal limits. As his anatomical staging, physiological staging and performance status were good we planned surgical resection.

We referred the case to thoracic surgery department and they did right upper lobectomy. Staging at the time of surgery was not different and they took hilar lymph nodes for histopathological examination. Histopathological examination of tumor confirmed adenocarcinoma and that of hilar lymph nodes did not show any evidence of malignancy. We referred the case to oncology department, and they suggested to keep patient under surveillance. Patient was followed up with quarterly chest X-ray, and no clinical or radiological evidence of relapse was found during 1 year of surveillance.

Discussion

I should have a PET scan before advising surgery but that facility was not available at that time. Moreover, at that time the opinion was against searching for metastasis if patients did not have manifestations suggestive of metastasis in non-small cell lung cancer. It is now well accepted that all non-small cell lung carcinoma should have whole body PET scan before surgery. As PET is not effective in detecting brain metastasis, additional CECT/magnetic resonance imaging (MRI) brain is suggested.

The next step should be to assess the operability of patient including the contraindications for surgery and assessment of tolerability of lung resection. For resectable and operable tumor, surgery (lobectomy or pneumonectomy) should be done at the earliest. At the time of operation staging should be performed (surgical staging) for planning adjuvant chemo/radiotherapy. If patient's tumor is found to be resectable but patient's physiological staging does not permit operation, then curative chemo/radiotherapy should be considered.

My Learning

Early detection and appropriate investigation of SPN is important for increasing survival time of lung cancer.

CASE 4: ACCIDENTAL DISCOVERY OF IRREGULAR CALCIFICATION

CASE REPORT

A 60-year man consulted his family physician for cough with expectoration and fever. He was treated with antibiotics (co-amoxiclav 625 mg 8 hourly for 7 days) and was advised to have a chest X-ray. He responded with treatment. His chest X-ray showed calcified opacities. He was referred to us for evaluation of X-ray abnormality.

When we saw him, he had no symptom and he denied any past history of illness. He was nonsmoker, nondiabetic and nonhypertensive. General survey, examination of respiratory system and other systems were normal. His chest X-ray PA view (Fig. 4) showed opacity with calcification in right mid zone near paracardiac region. Calcifications were linear, beaded and vertically placed like candle wax or branches of banyan tree. The typical findings were suggestive of pleural calcification.

Discussion

Pleural fibrosis may be focal or diffuse and may be associated with blunting of costophrenic angle. Diffuse pleural thickening is often associated with volume loss and it may have focal and extensive calcification. Diffuse pleural fibrosis is almost always preceded by exudative pleural effusion. It may be asymptomatic or may cause dyspnea as a result of entrapment of underlying lung. Pleural thickening may be caused by previous empyema, hemothorax,

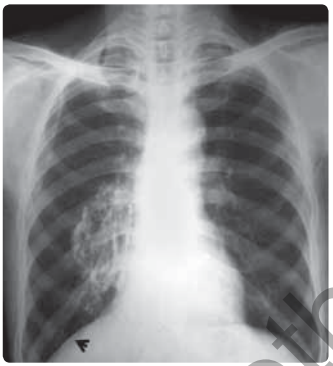


Fig. 4: Chest X-ray PA view showing right sided pleural calcification and calcification of right diaphragmatic pleura (arrowhead)

thoracic surgery, thoracic irradiation, asbestos related pleural diseases, malignant mesothelioma, pleural metastasis, collagen vascular diseases and drugs (methysergide, methotrexate, bromocriptine, mitomycin etc.). In our country, possibly tubercular pleural effusion is the commonest cause of pleural fibrosis.

Pleural plaque is a dense, acellular collagen deposit in the parietal pleura and often associated with dystrophic calcification. Calcifications are usually punctated, linear or coalescent. They present as focal, pleural based opacities with irregular margin, typically distributed in the posterolateral chest wall, lateral chest wall, dome of diaphragm and paravertebral pleura. They are usually bilateral and asymptomatic. It may take 20–30 years (latency time) after exposure of asbestos fibers to develop plaque. Asbestos fibers are carried to pleura by lymphatic channels or by direct penetration into pleura causing inflammation, fibrosis and ultimately calcification. Chest X-ray may be negative up to 50% cases and CT scan is superior in detecting pleural plaque. Isolated pleural plaque may be seen in tuberculosis, trauma and hemothorax.

Pleural calcification can be easily identified by its peculiar appearance (candle wax pattern). It usually found posterolaterally. It is dense, linear and parallel to the inner chest wall. Sometimes they may produce 'veil-like opacity' or 'holly leaf opacity'. Causes of pleural calcification are empyema, hemothorax, tuberculous effusion and asbestos plaque. Empyema usually started as parapneumonic effusion. Empyema as a cause of pleural calcification can be diagnosed by long history of high fever, chest pain and signs of pleural effusion that needs treatment with prolong antibiotics and intercostal tube drainage. Pleural calcification in infection and hemorrhage are difficult to differentiate. They are unilateral and vary from minimal to massive. In massive calcification they are sheet-like, hazy veil-like opacity, closer look detect dense and linear opacity parallel to the inner chest wall.

Asbestos related pleural plaques usually involve parietal pleura whereas calcification in empyema can affect both layers of pleura. Asbestos plaques involve posterolateral and diaphragmatic pleura sparing apex, costophrenic sulci or mediastinal pleura. Now benign asbestos, chrysolite, is mostly used and carcinogenic asbestos like amphiboles, crocidolite and amosite are usually not used in industry. The use of chrysolite is thought to be the cause of pleural plaque without parenchymal fibrosis and intrathoracic malignancy. History of exposure may be absent in small and unilateral plaque as asbestos are widely used and individuals may get exposure unknowingly.

Management

I diagnosed the case as asbestos plaque with calcification though the history of exposure to asbestos was not available. The points in favor were involvement of posterolateral pleura and diaphragmatic pleura without involvement of apex, costophrenic sulcus and mediastinal pleura. I assured the patient about the benign nature of opacity and referred him back to his physician.

My Learning

Pleural calcification can be easily differentiated from lung parenchymal calcification by its characteristic radiological appearance. The presence of diaphragmatic pleural calcification is strongly suggestive of asbestos induced pleural plaque.

CASE 5: AN ADOLESCENT BOY WITH ILL-DEFINED NODULAR OPACITIES

CASE REPORT

Step I

A 16-year-old boy came to me with shortness of breath during exertion for 1 month. As per patient's statement he had insidious onset dyspnea and that was increasing. Dyspnea occurred during strenuous exercise that restricted his activities and outdoor play. He had occasional cough without expectoration. There was no fever, wheeze, hemoptysis, paroxysmal nocturnal dyspnea or chest pain. General survey and systemic examination revealed no abnormalities. His routine blood and biochemistry were normal. His spirometry was within normal limits. His chest X-ray PA view (Fig. 5A) showed some non-specific paracardiac reticulonodular opacities. I gave a course of antibiotics (azithromycin 500 mg orally daily for 5 days) and salbutamol inhaler on as needed basis, but there was no response.

Discussion

The boy had dyspnea of short duration as primary symptoms with no other clinical and laboratory findings. I thought there might be infection but that did not responded to antibiotics. Asthma should be considered as a possibility as normal spirometry does not rule out asthma. Miliary

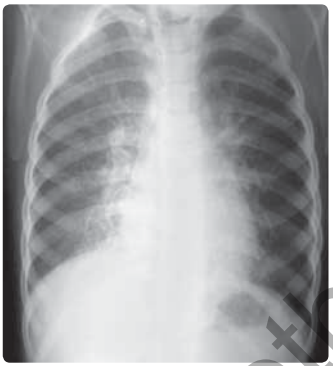


Fig. 5A: Chest X-ray of younger brother showing ill-defined parahilar opacities

tuberculosis was considered but absence of fever, toxicity and normal general condition went against that diagnosis. I also considered hypersensitivity pneumonia as a possibility and took detail history regarding exposure to organic dust, particularly exposure to pigeon and other birds, but no such history was available. Cardiac disease like mitral stenosis was another possibility. Sometimes classical mid-diastolic rumbling murmur might not be audible due to rotation of heart. There was no clinical or radiological evidence of pulmonary hypertension. Atrial septal defect usually presented with respiratory tract infection and wide splitting of second heart sound, and that was clinically ruled out.

Step II

His echocardiography was normal and IgE was within normal limits (525 mg/dL). I took detailed history. There was no history suggestive of atopy: the boy did not have urticaria, rhinitis, eczema or family history of asthma or allergic diseases. But his elder brother was suffering from dyspnea. The elder brother was under treatment with a homoeopathic doctor. The younger brother himself came to me for medical help, probably alarmed by the fate of his elder brother. I asked him to bring his elder brother next day.

Elder brother had progressive and persistent dyspnea and at that time he was dysphonic even at rest. Examination revealed RR—36/min, accessory neck muscles were working during respiration but examination of respiratory system revealed no abnormalities except reduced lung expansion suggesting bilateral decreased respiratory movement. Elder brother's chest X-ray PA view (Fig. 5B) showed bilateral reticulonodular dense (more than rib density) opacities involving mainly lower and mid zones. The most astonishing finding

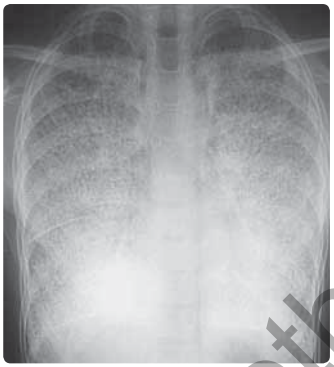


Fig. 5B: Chest X-ray of elder brother showing bilateral dense lung opacities with relatively lucent heart

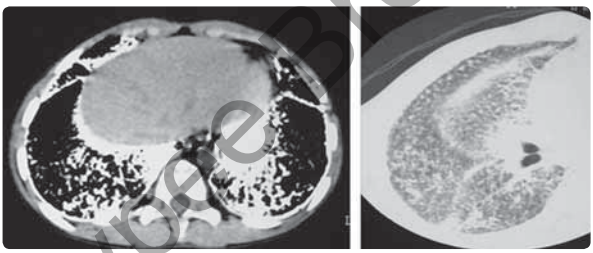


Fig. 5C: CT-thorax of elder brother mediastinal window and lung window showing the dense nodules of alveolar microlithiasis involving fissure lines

was that lung opacities were denser than cardiac silhouette, and it appeared as if heart had been vanished. The radiological findings were consistent with the diagnosis of alveolar microlithiasis. The radiological picture of elder brother was confirmatory of alveolar microlithiasis. CT-thorax of elder brother (Fig. 5C) showed the typical dense nodules of alveolar microlithiasis those conglomerated in some areas. Nodules also invoved the fissure and septal lines.

Discussion

Alveolar microlithiasis is a disease characterized by accumulation of numerous calcified bodies mainly in the alveoli. Microliths contains calcium phosphate with mean diameter 200 micrometer that may be surrounded by dystrophic calcification. Alveolar wall is not involved in early disease

but later the disease may involve interstitium. Sixty percent cases occur in families and the rest occur sporadically. The disease is usually detected in third and fourth decades. Dyspnea is the main symptom. Cough developed at a later stage. Hemoptysis, clubbing, pneumothorax and cor pulmonale may occur rarely. Chest X-ray characteristically showed enumerable, tiny, widely distributed (mainly basal) and pinpointed calcified nodules. Nodules may coalesce to form ground glass opacity or coarse nodules. In late stage heart may be relatively radiolucent than lungs (vanishing heart). Blebs and bullae are better seen in CT-thorax. The differential diagnosis includes metastatic calcification, renal failure, pneumoconiosis, hypersensitivity pneumonia and pulmonary infection like varicella and histoplasma. The disease may remain stable or progresses.

Management

I diagnosed the younger brother as early alveolar microlithiasis as the disease has propensity to run in family. I decided to keep the younger brother under observation, assuring him that the disease might not progress as rapidly as his elder brother.

My Learning

Family history is important and sometimes that may give vital clue leading to diagnosis.

CASE 6: AN ELDERLY MAN CARRYING SIGNS OF LONG-STANDING PAIN

CASE REPORT

A 74-year-old man came to our outpatient department with hemoptysis. Hemoptysis was described by him as streaks of blood with mucoid expectoration, particularly occurring in the morning. There was long history of cough with mucoid and mucopurulent expectoration, and progressive dyspnea. Patient was an exsmoker, he used to smoke *biri* 1 packet per day (1 packet contains 20 *biries*) for more than 30 years. He left smoking for about 10 years. He was diagnosed as chronic obstructive pulmonary disease (COPD) and was treated irregularly with oral bronchodilators and sometimes with antibiotics. He had pulmonary tuberculosis 15 years back and he was treated with daily antitubercular chemotherapy from private clinic. Old records were not available but as per his statement, he took medicines regularly for 1 year.

General survey revealed no abnormality; his blood pressure (BP) was 110/70 mm of Hg; pulse was 96/min and respiratory rate (RR) was 22/min. His upper respiratory tract was normal. Examination of lower respiratory tract revealed vesicular breath sound with prolonged expiration. Other systems were essentially normal. His routine peripheral blood and blood biochemistry were within normal limits. His X-ray chest PA view showed (Fig. 6) heterogeneous opacities in right upper zone along with some small, round, discrete, dense and well-circumscribed opacities.

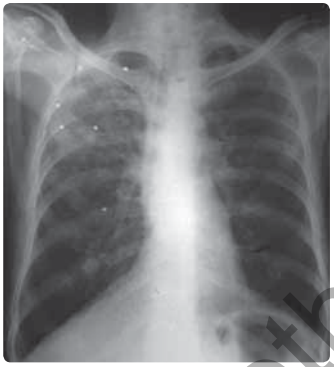


Fig. 6: Chest X-ray of elderly man showing pellets inside and outside lungs apart from some parenchymal opacities

Discussion

As the patient came with hemoptysis, my first job was to treat hemoptysis and second job was to identify the cause of bleeding. In TB hemoptysis is usually less and can be easily controlled with conservative treatment. Massive hemoptysis in active TB can occur from cavity due to rupture of Rasmussen's aneurysm, and as a result of post TB bronchiectasis or from aspergilloma in a cavity in patients with past history of TB. The old man had right upper zone opacities and past history of TB. I looked for the activity of TB. The patient had no symptoms suggestive of active TB like fever, and cough could be explained by associated COPD. Though his repeated negative sputum smears for AFB, I sent sputum for mycobacterial culture in solid media (BACTEC was not available at that time) and report came as negative after 6 weeks.

On those days (CT scan was not available), we used to classify radiological opacities as hard and soft opacities where hard lesions indicated old tubercular lesion and soft lesions were considered as active disease. The differentiation of hard and soft lesions required long experience and was often subjective. Presence of cavity is usually indicative of activity. Any change of lesion (deterioration or even improvement) in the follow up X-rays was considered as signs of activity.

The next question was about those tiny discrete round opacities. The opacities did not look like calcified parenchymal lesions found in pulmonary TB. The opacities were homogeneously dense, surprisingly round and remarkably discrete. The possibility of artifact was seriously considered and that was confirmed by detection of a similar opacity over right shoulder joint. The rule is to look outside lung border whenever there is a suspicion of artifact. If similar opacities are found outside lung border or the opacity can

be stretched outside thoracic cage, we can diagnose the opacity as artifact with confidence. Sometimes buttons of garments can produce artifact and that can be confirmed by taking another X-ray after putting off the garment. Sometimes nipple may produce artifact and that can be confirmed by taking X-ray putting a marker over the nipple. In that X-ray there were nipple shadows in both lower zones.

Final Diagnosis

The opacities were pellets. I asked the old man, and he told me his story that he was eager to tell but nobody was listening. He was carrying those pellets for more than 50 years. There was a notorious riot in Calcutta, the great Calcutta killing, just before partition to take control of our beloved city. The riot was selectively deleted from the contemporary Indian history. During that riot the old man had a pellet-gunshot injury, and he was carrying those pains for more than 50 years.

Management

I treated hemoptysis with rest, bronchodilators and etamsylate. I avoided sedations and cough suppressants as he had COPD. Hemoptysis stopped with conservative management. I gave him long acting theophyllin tablets for COPD. His sputum samples were negative for acid fast bacilli (AFB). I asked for sputum culture of mycobacterium. Subsequently the report came as negative. I did not start anti-TB treatment, and gave him antibiotics (co-amoxiclav 625 mg 8 hourly for 7 days). I assured him that those dense round lesions were pellets and no treatment was required for them.

My Learning

Doctors are part of society, and a clear understanding of social and political events, without getting involved into it, is essential.

CASE 7: ACCIDENTAL DETECTION OF BILATERAL HILAR LYMPHADENOPATHY

CASE REPORT

Step I

A 46-year-old housewife went to gynecology outpatient department with excessive vaginal bleeding and was diagnosed as fibroid uterus. As a routine preoperative check-up one X-ray chest was done, and that showed some abnormalities. She was referred to chest department.

We took history and examined her. She told me that she had occasional cough. Dyspnea occurred on exertion and modified Medical Research Council (mMRC) grading was grade II. She had no fever, weight loss, night sweat but had weakness. Examination revealed no abnormality except pallor (probably related to blood loss). Routine blood and biochemistry were normal except hemoglobin (Hb) was 7 g/dL and erythrocyte sedimentation rate (ESR) was

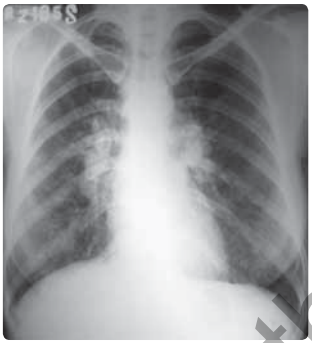


Fig. 7: Chest X-ray showing bilateral symmetrical hilar lymphadenopathy with ill-defined parenchymal opacities

60 mm in 1st hour. Her chest X-ray PA view (Fig. 7) showed bilateral almost symmetrical hilar lymphadenopathy with parenchymal ill-defined reticulonodular opacity.

Discussion

In a middle-aged lady with minimal respiratory symptoms, radiological abnormalities were detected by routine preoperative check-up. She had dyspnea, and that could be explained by anemia. The main radiological abnormality was bilateral hilar lymphadenopathy.

In chest X-ray hilar opacity can be distinguished from mediastinal opacity by the presence of a radiolucent gap between opacity and mediastinal contour. Opacity that merges with mediastinum is usually considered as mediastinal opacity. Hilar lymphadenopathy can be differentiated from vascular hilar shadow by 'hilar convergent sign' (when a peripheral branch of pulmonary artery is traced centripetally it will merge with hilar vesicles in case of vascular enlargement not with hilar lymphadenopathy). The main causes of hilar lymphadenopathy are tuberculosis, sarcoidosis, lymphoma, bronchogenic carcinoma, etc. Tuberculosis and bronchogenic carcinoma usually have unequal hilar lymphadenopathy, while sarcoidosis and lymphoma may have equally enlarged hilar lymph nodes.

Step II

Contrast enhanced computed tomography (CECT)-thorax showed bilateral hilar lymphadenopathy along with right paratracheal lymphadenopathy and few nodular opacity scattered over both lung field. Nodules could not be classified in CECT plates, and that would need high resolution CT (HRCT)-

thorax. Her Mantoux test was negative 5 x 6 mm with 10 TU. Her serum calcium was 9.6 mg/dl (normal value 8.7–10.2 mg/dl) and urine examination did not revealed calciuria. Her serum angiotensin-converting enzyme (SACE) level was 52 U/L (normal level 9–67 U/L). Fiber-optic bronchoscopy showed no intraluminal pathology. Bronchoalveolar lavage (BAL) fluid did not give any clue and BAL fluid for AFB was negative. We sent BAL fluid for BACTEC culture for tuberculosis. Transbronchial lung biopsy reports (TBLB) came as ill-formed granuloma without evidence of caseation. We planned endobronchial ultrasonography (EBUS) but patient could not afford that investigation.

Discussion

Lung parenchymal nodules are classified into (i) centrilobular (a gap can be visible along subpleural areas and along fissures) found in hypersensitivity pneumonia; (ii) perilymphatic (nodules are visible over fissures) found in sarcoidosis and (iii) random nodules found in miliary tuberculosis and metastases. The classification of nodules requires good quality HRCT-thorax.

Transbronchial lung biopsy has the problem of getting tiny tissue and the tissue available is usually crushed. Transbronchial lung biopsy is useful for detecting infective etiology particularly tuberculosis, disseminated malignancy and sarcoidosis (as the lesions are mainly peribronchovascular). The finding of ill-formed granuloma may be found in tuberculosis and sarcoidosis, but may not exclude lymphoma or carcinoma.

In our case bronchogenic carcinoma was excluded by combination of factors like non-smoker, female sex, no chest symptoms, absence of lung mass in CECT thorax, symmetrical hilar lymphadenopathy and absence of intraluminal lesion in bronchoscopy. Absence of peripheral lymphadenopathy, hepatosplenomegaly went against lymphoma though pure intrathoracic lymphoma that confined within thorax is now considered as a definite entity. Considering clinico-radiological picture of that lady we restricted the differential diagnosis within tuberculosis and sarcoidosis. Absence of constitutional symptoms, negative Mantoux test, symmetrical lymphadenopathy, right paratracheal lymphadenopathy and absence of AFB in BAL fluid pointed towards sarcoidosis. Bilateral symmetrical hilar lymphadenopathy in an asymptomatic individual with negative tuberculin test favored the possibility of sarcoidosis.

Management

We decided not to start treatment of sarcoidosis, because of lack of symptoms and lack of urgency of starting treatment. We also considerded that corticosteroid might increase perioperative complications. We sent back the patient to gynecologist for hysterectomy after correction of anemia with blood transfusion. She had undergone hysterectomy. In between BAL fluid BACTEC culture for mycobacterium report came as negative. After 1 month of surgery, we reviewed the case and decided to keep her under observation as at that time she did not have any respiratory symptoms.

Discussion

Sarcoidosis is a multisystemic granulomatous disease of unknown etiology. Lungs are involved in more than 90% cases. The common radiological findings are bilateral hilar lymphadenopathy, paratracheal lymphadenopathy and reticulonodular opacities in lungs. Lung involvement can be staged as: (i) Stage 0—normal chest X-ray (found in 8–16% cases); (ii) Stage I—bilateral hilar lymphadenopathy with or without right paratracheal lymphadenopathy (25–65% cases); (iii) Stage II—bilateral hilar lymphadenopathy with lung parenchymal infiltration (14–49% cases); (iv) Stage III—parenchymal infiltration without hilar lymphadenopathy (10% cases); and Stage IV—parenchymal infiltration with overt pulmonary fibrosis (5% cases). In HRCT-thorax, nodules in sarcoidosis are classically perilymphatic in distribution.

Manifestations of other system involvement include erythema nodosum, peripheral lymphadenopathy, hepatosplenomegaly, keratoconjunctivitis, peripheral neuritis, pituitary gland involvement, polyarthralgia, lupus pernio, hypercalcemia and/or hypercalciuria and renal stone. SACE level may be helpful in diagnosis of sarcoidosis but it has low sensitivity and specificity. High SACE level is found in about 60% in acute and 20% in chronic sarcoidosis. SACE level may be increased in other conditions like disseminated tuberculosis, leprosy, hyperthyroidism and Gaucher's disease, etc.

The differentiation between sarcoidosis and tuberculosis is sometimes difficult. The presence of caseating granuloma, detection of tubercular bacilli by smear, culture or molecular methods and a positive tuberculin test should be considered as evidence for tuberculosis. The absence of evidence for tuberculosis and presence of multiorgan involvement with non-caseating granuloma can be taken as evidence for sarcoidosis.

Treatment of sarcoidosis depends on the symptoms, presentations and organ involved. Treatment is mainly with oral corticosteroids; prednisolone 40–60 mg daily followed by maintenance dose of 5–10 mg daily. Lung parenchymal involvement usually requires treatment with corticosteroids. Our patient was asymptomatic and lung involvement was trivial and doubtful, and we kept her under observation.

My Learning

Differentiation of sarcoidosis from tuberculosis is difficult and sometimes impossible. A pragmatic approach is the key for decision making regarding treatment.

CASE 8: A YOUNG LADY WITH CHRONIC NONSPECIFIC SYMPTOMS

CASE REPORT

Step I

A 30-year-old lady presented with central chest pain for more than 3 years. She was changing doctors as none of them gave her some relief. General physicians

gave her proton pump inhibitors (PPI) and she had continued with PPI for more than two years but there was no relief to pain. She was referred to a cardiologist and routine cardiological checkup was done. Cardiac investigations including electrocardiography (ECG) and echocardiography were with normal limits. She was referred to a psychiatrist. She did not go to psychiatrist probably due to social stigma and consulted a physician. The physician at last advised her a chest X-ray. He found abnormality in chest X-ray and referred her to me.

The patient gave me history that she had insidious onset of chest pain in the front of central chest in the lower part. The pain was vague, dull aching, not increased with exertion or respiration. The pain was persisting but not progressive and that did not affect her normal day to day activity or sleep. She denied any history suggestive of acidity, reflux, heart-burn and chest trauma. Her general survey, examination of respiratory system and other systems were essentially normal.

Her peripheral blood and routine biochemistry were within normal limits. Her chest X-ray PA view (Fig. 8A) showed homogeneous opacity at right lower zone obliterating right costophrenic angle. The opacity had sharp lateral and upper margins, but medial and lower borders were obscured. There was no mediastinal shifting. Her chest X-ray right lateral view (Fig. 8B) showed that the opacity was placed in the anterior mediastinum obliterating anterior costophrenic angle and anterior part of right dome of diaphragm.

Discussion

Central chest pain is a common symptom in middle-aged female. The causes include acidity, heart burn, cardiac pain, costochondritis – Tietze syndrome (usually upper part of chest with tenderness of costochondral junction). But in most cases the cause of chest pain cannot be identified. Sometimes, it is considered as psychological in origin. The pain, when no definite cause is found, is treated with PPI, analgesics and sometimes antidepressants.

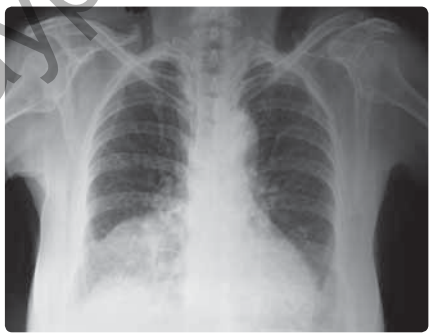


Fig. 8A: Chest X-ray PA view showing right lower zone opacity at the right cardiophrenic angle

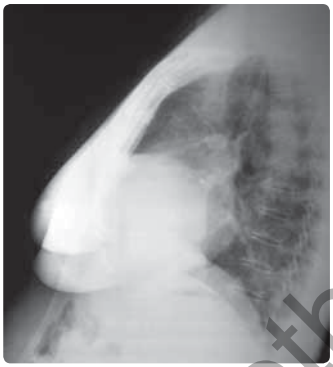


Fig. 8B: Chest X-ray right lateral view showing opacity in anterior costophrenic angle

Any opacity that could not be separated from mediastinum should be considered as mediastinal mass. The differential diagnosis of opacity in anterior mediastinum can be described as '6 T' (teratoma, thymoma, thyroid and parathyroid mass, terrible lymphoma, tortuous vessels and trauma). In our case the mediastinal mass was situated in the right cardiophrenic angle. Two conditions (i) Morgagni hernia and (ii) pleuropericardial cyst are specifically found in that particular area.

Step II

I advised CT scan of thorax, and CT–thorax mediastinal window showed (Fig. 8C) a low density mass lesion with clear and sharp margin at the right cardiophrenic angle. The density of the lesion was similar to fat density. There was lobulation/septation within the mass lesion. We diagnosed the case as Morgagni hernia with herniation of omentum.

Discussion

Morgagni hernia is a congenital hernia that occurs through the gap between sternal and costal origins of the diaphragm. It is usually filled with loose connective tissue and in some cases omentum may be herniated through the opening. Less commonly viscera may herniate and they include colon, liver, gallbladder, etc. Morgangi hernia is usually in the right side. Though it is congenital, presentation in childhood is very rare. In adult they usually present with vague symptoms like feelings of tightness, fullness or pain in the right anterior chest. Occasionally, the pain may be referred to shoulder tip. Strangulation of bowel within hernia may present with acute symptoms.

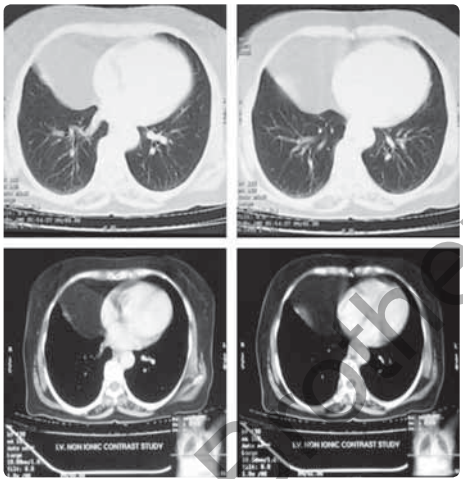


Fig. 8C: CT-thorax showing mass lesion in right cardiophrenic angle with fat density and loculation

Chest X-ray usually shows a rounded density in the cardiophrenic angle. Barium studies may be useful to detect the presence of intestine, and the hitched up transverse colon in omental herniation. Management is the surgical repair of the gap in the diaphragm.

The lady was diagnosed as Morgagni hernia by vague symptoms, the characteristic site of lesion, low density in CT-thorax indicating fat/omentum, and was confirmed during surgery. Fat density in mediastinum may be found in teratoma where fat density within a heterogeneous mass is typically found. As CT-thorax did not give any suggestion of gut herniation I avoided barium meal X-ray. Despite of milder symptoms repair of gap was done to avoid the risk of strangulation of hernia.

Management

We sent the patient to thoracic surgeons and they repaired the gap after pulling down omentum into abdomen. She was discharged from hospital after 10 days of operation.

My Learning

Morgagni hernia should be considered in differential diagnosis of opacity in right cardiophrenic angle.