e-ISSN: 2319-9865
p-ISSN: 2322-0104

Research and Reviews: Journal of Medical and Health Sciences

Trends in the Epidemiological Study of the Most Common Pulmonary and Respiratory Disorders: A Systematic Overview

Vijay Kumar Mittapally[1]* and Divya Lanka[2]

[1]Department of Biotechnology, Periyar University, Salem, Tamil nadu
[2]Department of Pharmaceutics, Gland Institute of Pharmaceutical Sciences, Kothapet, Medak dist.

Review Article

Received: 04/01/2015
Revised: 25/01/2015
Accepted: 03/02/2015

*For Correspondence

Vijay Kumar Mittapally, Master of Sciences, Department of Biotechnology, Periyar University, Salem, Tamilnadu, India, Tel: 91-9949985675, E-mail: vijay.mittapally20@gmail.com.

Keywords: Chronic Obstructive Pulmonary Disease (COPD), Pulmonary embolism, Pulmonary hypertension, Dyspnea, Pulmonary Tuberculosis.

ABSTRACT

Different types of pulmonary and respiratory disorders like pulmonary hypertension, dyspnea, Chronic Obstructive Pulmonary Disease (COPD), pulmonary embolism etc., are emerging as important public health problems. This development has occurred in large measure due to the increasing number of population at risk. In addition, advances in therapeutic technologies and in particular the development of novel immunosuppressive therapies for the disorders have prolonged the period of risk for many individuals. This review deals with the prevalence, causes and effects of various pulmonary and respiratory disorders in defined populations. Although many novel therapies are obtained these days, an understanding of risk factors is required if prevention strategies, such as chemoprophylaxis and environmental control measures are widely applicable and cost-effective.

INTRODUCTION

Chronic obstructive pulmonary diseases (COPD), chronic dyspnea, pulmonary hypertension continue to be heavy health problems and economic burdens around the world. Recent studies showed the evidence of various risk factors including smoking, occupational exposures, air pollution, airway hyper responsiveness, and certain genetic variations [1].

Chronic obstructive pulmonary disease (COPD):

COPD is the fourth leading cause of chronic morbidity and mortality in the United States [2], and is ranked fifth in 2020 in burden of disease worldwide, according to a study published by the World Bank/World Health Organization [3-9].

It can be called as a collection of lung diseases including chronic bronchitis, dyspnea, emphysema and chronic obstructive airways disease. Cigarette smoking is one of the most important risk factors for the development of COPD [10-12].

The likelihood of the disease occurrence increases along with an increased rate of smoking in a person. It usually affects a person at the age of 35, even though most people are not diagnosed until they are in their 50s. It is estimated that there are more than 3 million people living with the disease in the UK, of which only about 900,000 were diagnosed [13].

Although its prevalence is more common in male smokers, females have also showed an increased rate of diseased condition. Evidences of a study based on the effect of gender differences on smoking show that Females reported more symptoms compared with males with more severe airway obstruction [14].

e-ISSN: 2319-9865
p-ISSN: 2322-0104

Global Initiative for Chronic Obstructive Lung Disease (GOLD) [15] defined COPD as a common treatable and preventable disease[9] which is characterized by limitation of the airflow that usually progresses and is associated with an enhanced chronic inflammatory response in the airways and the lungs to noxious particles and gases (last 2011 update).

According to the World Health Organization (WHO), COPD was responsible for over 3 million deaths annually at the beginning of this millennium. It is also known as the 7th leading cause of death in Hispanics [16].

Approximately 2.7 million deaths from COPD occurred in 2000 of which half are from western pacific region, majority occurring in China. About 400,000 deaths caused by COPD are mainly from industrialised and developed countries [17].

Pulmonary Arterial Hypertension:

Pulmonary Arterial Hypertension (PAH) [18-23] is defined as a rare and devastating condition which is characterized by a sustained increase in Pulmonary Vascular Resistance (PVR) leading to right ventricular failure [24]. This disease is affected in people of any age, although some types mostly are common in youngsters. In the UK, around 6,000-7,000 people are expected to have pulmonary hypertension [25]. It's also thought that many remain undiagnosed. Its Symptoms include shortness of breath, fatigue, dizziness, angina, palpitations etc. [26] Dyspnea, arrhythmia and premature death are the common features of PAH [27].

The clinical classification of pulmonary hypertension was introduced at the 4th World symposium on PH held at Dana Point [28,29], CA in 2008 (Table 1). PH is now divided into five classs based on etiology [30].

Table 1: Clinical classification of pulmonary hypertension

Class 1: Pulmonary arterial hypertension • Idiopathic • Heritable: associated with specific gene mutations like BMPR2, ALK1, endoglin, etc • Drug- & toxin-induced • Associated with: connective tissue diseases, HIV infection, portal hypertension, congenital heart diseases, schistosomiasis & chronic hemolytic anemia • Persistent pulmonary hypertension of the newborn **Class 1': Pulmonary veno-occlusive disease and/or pulmonary capillary hemangiomatosis**
Class 2: PH secondary to left heart disease • Systolic dysfunction • Diastolic dysfunction • Valvular disease
Class 3: PH secondary to lung diseases and/or hypoxia • Chronic obstructive pulmonary disease • Interstitial lung disease • Other diseases with a mixed restrictive & obstructive pattern • Sleep disordered breathing • Alveolar hypoventilation disorders • Chronic exposure to high altitude • Developmental abnormalities
Class 4: Chronic thromboembolic pulmonary hypertension
Class 5: PH due to multi-factorial/unclear mechanisms.

e-ISSN: 2319-9865
p-ISSN: 2322-0104

- Hematologic: myeloproliferative disorders, splenectomy
- Systemic: sarcoidosis, pulmonary Langerhans cell histiocytosis
- Metabolic disorders: glycogen storage disorders, Gaucher disease, thyroid disorders
- Others: tumoral obstruction, fibrosing mediastinitis, chronic renal failure on dialysis

This system was based on earlier world symposia on PH in 1998 and 2003 [31].

Recent studies suggest that the epidemiology of the disease has changed dramatically over the past three decades [32]. The patients in the landmark National Institutes of Health (NIH) registry conducted in the 1980s were predominantly young (mean age of 36 at presentation) and female [33] (1.7:1) and had idiopathic, familial, or anorexigen-associated PAH. Their 1-, 3-, and 5-year survival were 67, 45, and 37%, respectively hypertension connections (PHC—single-center U.S.-based registry), and the French national registry [34].

Dyspnea

Dyspnea is defined as an uncomfortable sensation of breathing with varying intensity [35]. Ventilation is normally controlled by the autonomic nervous system, with only limited voluntary override. The sensation of dyspnea may be developed by the sense of respiratory effort, chemoreceptor stimulation, mechanical stimuli arising in lung and chest wall receptors and neuroventilatory dissociation [36,37].

In the 1950s and 1960s much of the work on dyspnea was mainly focused on the impact of mechanical loads on respiratory symptoms [38-44]. While there was an intention that there may be several different qualities of dyspnea, the general appreciation was that the sense of effort was the primary element of breathing discomfort.

Patients with chronic pulmonary disease often limit their activities due to respiratory discomfort [45]. Reductions in physical functioning status, quality of life, and disability are frequent consequences of this symptom. Diseases producing chronic dyspnea may make the patient suffer with significant breathlessness [46-49].

Dyspnea is the term generally applied to the individuals who complain of unpleasant or uncomfortable respiratory sensations [50].

In obstructive lung diseases such as asthma and COPD, dyspnea is a common respiratory symptom [51-55] with different characteristics and different pathogenic mechanisms: in COPD initially it can occur during exertion but then it increases progressively along with the airflow obstruction, whereas in asthma it occurs in concurrent episodes and is caused by transient bronchoconstriction [56-61].

Pulmonary embolism (PE)

It is known as the blockade of the pulmonary arteries of the lungs. It is a life-threatening disease which is caused by the formation of blood clots in the arteries that travel to the lungs from the other parts of the body (deep vein thrombosis) [62-65].

As this disorder occurs in conjunction with the deep vein thrombosis, it is usually termed as “Venous thromboembolism.”

Its usual symptoms are shortness of breath, chest pain, cough, cyanosis, dizziness, fever etc. Sometimes other than blood clots, some other substances like fat within the marrow of a broken bone, air bubbles, any part of the tumour may also cause the blockage of the arteries [66-72].

There has been a belief that sometimes the obesity may also increase the severity of pulmonary embolism, but it didn’t show satisfactory results when examined in defined population [73].

The incidence of venous thrombo-embolism in India is 17. 46 per 10,000 hospital admissions and pulmonary embolism are diagnosed in 14. 9% of these patients with a 13. 5% mortality rate [74].At present there has been no appropriate information on the optimal management of the patient with

pulmonary embolism [75]. Treatment given depends upon the patient response towards the treatment, local health guidelines and available local expertise [76].

Pulmonary embolism can be detected by various forms like Spectral Doppler waveform analysis [77] (example: for the detection of right ventricular dysfunction) and also can be successfully managed by Unilateral Utrasound-assisted catheter-directed thrombolysis (example: in cardiac surgery) [78].

Also the specificity of the pulmonary embolism and deep vein thrombosis can be analysed by the quantification of a specific protein named D-dimer [79], which is present in the blood as a product of fibrin clot lysis due to the action of plasmin, which begins at the same time the clot begins to form [80].

However most of the cases of pulmonary embolism result from clot fragmentation of lower limb Deep Venous Thrombosis. Free-Floating Thrombus (FFT) [81] is present in 10-26% of thrombi detected with ultrasound and is often considered being a risk factor for PE in patients with Deep Venous Thrombosis [82].

Pulmonary Tuberculosis

Pulmonary tuberculosis (TB) is caused by the bacterium Mycobacterium tuberculosis (M. tuberculosis). Its symptoms include breathing difficulty, chest pain, cough (usually with mucus), coughing up blood, excessive sweating, fatigue, fever etc [83]. It is a known as the disease of poverty, overcrowding, and inadequate nutrition associated with sub-standard public health conditions, which infects 7% of people exposed [84].

It is the leading cause of death worldwide and the number of new cases is rising at a rate of 2% annually. It is generally associated with poverty, 95% of cases and 98% of deaths occur in developing countries [85], of which most of these deaths occur in young people. It is been estimated that in 2008 there were 9.4 million new cases of TB, out of which 1.8 million people died. The severity of this problem is so great that in 1993 the World Health Organization (WHO) declared it as a global emergency [86]. The World Health Organization also estimated that 8.8 million new cases of tuberculosis (TB) and 1.45 million deaths due to TB occurred worldwide in 2009, and almost 160 people die of TB each hour [87].

Before the invention of antitubercular drugs, therapies against TB were ineffective. Surgical treatment for TB included various forms of collapse therapy like wax or Lucite ball plombage, pneumoperitoneum, induced pneumothorax, thoracoplasty and phrenic nerve crush or interruption. Thoracoscopy for pulmonary tuberculosis was introduced by Jacobeus [88-91].

Asthma

It is known as the chronic lung disease which causes inflammation and narrowing of the airways [92,93]. It is affected in people of all age groups, but it most often starts during childhood. In the United States, more than 25 million people are known to have asthma out of which 7 million are children [94]. Its common symptoms include chest tightness, wheezing, coughing, shortness of breath etc. [95-99]. It is the most common chronic inflammatory disease, characterized by hyper reactivity of the airways and is triggered by different stimuli. Environmental factors play a key role in the allergy development and disease prevalence in genetically susceptible people. Management of asthma symptoms before hospitalizing the patient and during transportation to the emergency department can reduce the overall risk of death [100-106].

CONCLUSION

Respiratory and pulmonary disorders have been emerging from the past decades and the various epidemiological studies show that the disease prevalence is being increasing over the time with an increase in population. Several factors which are the causal agents for the diseases must be preventively organized. The overall risk of the diseases can be minimized by selectively choosing the therapies and by proper management of the diseased symptoms which can further bring down the mortality rate.

REFERENCES

1. Joyce CC et al. Worldwide epidemiology of chronic obstructive pulmonary disease. Current Opinion in Pulmonary Medicine. 1999;5:93.
2. Rabe et al. Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease. American Journal of Respiratory and Critical Care Medicine. 2007; 176: 532-555.
3. Powell Tand Williams EM. Assessing Perceptual Sensitivity of Respiratory Load Using Constant Airway Resistance. J Pulm Respir Med. 2015; 5:236.
4. Kumar S, et al. To Assess the Quality of Life (QOL) of Caregivers and Patients Suffering from Chronic Obstructive Pulmonary Disease (COPD). J Aller Ther. 2012; S2:003.
5. Burel JG, et al. Evaluation of Immune Responses to Influenza Vaccination in Chronic Obstructive Pulmonary Disease. J Vaccines Vaccin. 2012; S4:001.
6. Kirakli C. Chronic Obstructive Pulmonary Disease. J Pulmon Resp Med. 2012; S9:e001.
7. Toraldo DM, et al. Systemic Inflammation in Chronic Obstructive Pulmonary Disease: May Diet Play a Therapeutic Role? J Aller Ther. 2013; S2:005.
8. Afshar K.Treatments Based on Phenotypic Variants in Chronic Obstructive Pulmonary Disease. J Pulm Respir Med. 2013; 3:e128.
9. Gharabaghi MA, et al. High Dose Vitamin D3 Improves Exercise Tolerance in Elderly Patients with Chronic Obstructive Pulmonary Disease. J Gerontol Geriat Res. 2013; 2:127.
10. Kvangarsnes M, et al. Nursesâ€™ Perspectives on Compassionate Care for Patients with Exacerbated Chronic Obstructive Pulmonary Disease. J Allergy Ther. 2013; 4:158.
11. Deerpaul D and Hui SY. The Study of Association between Helicobacter pylori (H. pylori) and Chronic Obstructive Pulmonary Disease (COPD). J Pulm Respir Med. 2014; 4:171.
12. Zaghloul MZ. The Contribution of Anaerobic Bacteria that Causes Periodontitis in the Pathogenesis of Chronic Obstructive Pulmonary Disease (COPD). 2014.
13. Sherman CB. The health consequences of cigarette smoking. Pulmonary diseases.Med Clin North Am. 1992; 76: 355-375.
14. Patel AK, et al. Sputum Bacteriology and Antibiotic Sensitivity Pattern of Patients Having Acute Exacerbation of COPD in India â€“ A Preliminary Study. J Pulm Respir Med. 2015; 5:238.
15. Smythe MA, et al. Aligning Experiential Opportunities with Institutional Needs: Focus on Chronic Obstructive Pulmonary Disease. J Pharma Care Health Sys. 2014; S1-009.
16. Bulcun E, et al. Gender Differences on Airway Obstruction in Smokers. J Pulm Respir Med. 2015; 5:229.
17. Simonetti JA, et al. Pulmonary Function in HIV-Infected Recreational Drug Users in the Era of Anti-Retroviral Therapy. J AIDS Clin Res. 2014; 5: 365.
18. Wei M, et al. A Novel Mutation in BMPR2 in Patients with Congenital Heart Disease and Pulmonary Arterial Hypertension. J Clinic Experiment Cardiol. 2012; 3:181.
19. Durongpisitkul K, et al. Outcome of Atrial Septal Defects versus Ventricular Septal Defects in Response to Bosentan Treatment: Proof of Concept Controlled Study in Pulmonary Arterial Hypertension Related to Eisenmenger Syndrome. J Pulmon Resp Med. 2012; 2:120.
20. Yang Q. Targeting Epigenetic Changes for the Reprogramming of Vascular Walls in Pulmonary Arterial Hypertension, the Role of Histone Deacetylases and their Inhibitors. Cardiol Pharmacol. 2012; 2:e106.
21. Yu W, et al. Myocardial Infarction and Pulmonary Arterial Hypertension in a Young Patient with Systemic Lupus Erythematosus. J Clin Case Rep. 2012; 2:214.
22. Yang Q and Sun M. Role of Bone Morphogenetic Protein Type II Receptor Signaling in Pulmonary Arterial Hypertension. Cardiol Pharmacol. 2013; 2:e120.
23. Bradley EA and Bradley D. Pulmonary Arterial Hypertension and Insulin Resistance. J Mol Genet Med. 2014; S1:015.

24. Kirakli C. Chronic Obstructive Pulmonary Disease. J Pulmon Resp Med. 2012; S9:e001.
25. Kinney GL, et al. The Protective Effect of Hispanic Ethnicity on Chronic Obstructive Pulmonary Disease Mortality is Mitigated by Smoking Behavior. J Pulm Respir Med. 2014; 4:220.
26. Lopez et al. Chronic obstructive pulmonary disease: current burden and future projections. 2006; 27: 397-412.
27. GaliÃ¨ N, et al. Guidelines on diagnosis and treatment of pulmonary arterial hypertension. The Task Force on Diagnosis and Treatment of Pulmonary Arterial Hypertension of the European Society of Cardiology. Eur Heart J. 2004; 25: 2243-2278.
28. Cappelleri JC, et al. Cumulative Distribution Functions of Sildenafil Citrate on Exercise Capacity and Hemodynamic Function in Children with Pulmonary Arterial Hypertension. J Pulmon Resp Med. 2013; S4:002.
29. Banjar HH. Pulmonary Hypertension (PHT) in Patients with Down Syndrome: The Experience in a Tertiary Care Center in Saudi Arabia. J Pulmonar Respirat Med. 2012; 2:115.
30. Yang Q and Sun M. Role of MicroRNAs in Hypoxia-Induced Pulmonary Hypertension. Cardiol Pharmacol. 2014; 3:e124.
31. Ramachandraiah V, et al. Treatment of Pulmonary Hypertension. J Pulmon Resp Med. 2012; S4:001.
32. Boixeda R, et al. Hospital Admissions in Patients with COPD during the 2009 Flu Pandemic. J Med Microb Diagn. 2014 ; 3:161.
33. María-Teresa GS, et al. Description of Hospital Admissions for Acute Exacerbation of COPD. J Pulm Respir Med. 2014; 4:200.
34. Lisa G, et al. Clinical Impact and Prognosis of Pulmonary Hypertension after Transcatheter Aortic Valve Implantation. J Cardiovasc Dis Diagn. 2014; 2:143.
35. María-Teresa GS, et al. Description of Hospital Admissions for Acute Exacerbation of COPD. J Pulm Respir Med. 2014; 4:200.
36. Colvin KL and Yeager ME. Animal Models of Pulmonary Hypertension: Matching Disease Mechanisms to Etiology of the Human Disease. J Pulm Respir Med. 2014; 4:198.
37. Alencar AKN, et al. A Novel Adenosine A2a Receptor Agonist Attenuates the Progression of Monocrotaline-induced Pulmonary Hypertension in Rats. J Pulmon Resp Med. 2013; S4:005.
38. Rich S, et al. Primary pulmonary hypertension: a national prospective study. Ann Intern Med 1987; 107:216–223.
39. Puri A, et al. Asthma in HIV-Infected Population: A Review of Respiratory Symptoms, Pulmonary Function Abnormalities and Pathophysiology. Epidemiol. 2014; 4:164.
40. Siyoum K, et al. Respiratory Symptoms and Associated Factors among Cement Factory Workers and Civil Servants in North Shoa, Oromia Regional State, North West Ethiopia: Comparative Cross Sectional Study. Occup Med Health Aff. 2014; 2:182.
41. Gumus A, et al. An Evaluation of Chronic Dyspnea in a Chest Disease Clinic. J Pulm Respir Med. 2014; 4:173.
42. Rosengarten D and Kramer MR. Pulmonary Hypertension and Pregnancy: Management and Outcome. J Pulmon Resp Med. 2013; S4:004.
43. Aziz A, et al. Proteomic Profiling of Early Chronic Pulmonary Hypertension: Evidence for Both Adaptive and Maladaptive Pathology. J Pulm Respir Med. 2015; 5:241.
44. Svensson A, et al. Exposure to Human Herpes Virus Type 6 Protects Against Allergic Asthma in Mice. J Aller Ther. 2010; 1:101.
45. Lewkowich IP. IL-17A in Asthma - A Question of Severity. J Clin Cell Immunol. 2011; 2:107.
46. Wu AC, et al. Acceptance of Asthma Pharmacogenetic Study by Children and Adults. J Pharmacogenomics Pharmacoproteomics. 2011; 2:103.

47. Master Z, et al. Balancing Efficiency and the Protection of Research Participants: Canadian Allergy/Asthma Researchersâ€™ Perspectives on the Ethics Review of Multi-Site Health Research. J Clinic Res Bioeth. 2011; 2:104e.
48. Pritpal K, et al. Prevalence of Asthma in Elderly versus Young in Rural and Urban Area of India. J Pulmonar Respirat Med. 2011; 1:102.
49. Islami H, et al. Adrenergic Agonist and Antagonist Action in Airways of Patients with Bronchial Asthma. J Pulmonar Respirat Med. 2011; 1:104.
50. Elmorsy SM and Khafagy YW. Does Asthma and Aspirin Hypersensitivity Affect the Outcome of Endoscopic Sinus Surgery for Chronic Rhinosinusitis with Nasal Polyps. J Aller Ther. 2011; S5:002.
51. Pinto S, et al. Erythrocyte and Plasma Antioxidants in Bronchial Asthma Before and After Homeopathic Treatment. J Homeopat Ayurv Med. 2012; 1:103.
52. Lowder TW and Kunz HE. Regulatory T Cells in Asthma and Airway Hyperresponsiveness. J Aller Ther. 2011; S1:002.
53. Theofilou P and Saborit AR. Predictors of Asthma Treatment Adherence. J Psychol Psychother. 2012; S3:e001.
54. Morin C, Fortin S, Rousseau Ã. New Omega-3 Derivatives Reduce Airway Inflammation and Prevent Rho-Kinase Activation in an Allergic Model of Asthma. J Aller Ther. 2012; S1:003.
55. Vieira RP. Has Aerobic Exercise Anti-inflammatory Effects for Asthma? J Nov Physiother. 2012; 2:e108.
56. Aydemir G, et al. Finding Thrombocytosis at the Time of the Diagnosis in the Patients With Pneumonia, Bronchiolitis and Asthma, and Its Importance in Terms of the Diagnosis. Pediatr Therapeut. 2012; 2:118.
57. Antonogeorgos G and Panagiotakos DB. Obesity and Asthma: Is Diet a Therapeutic Mean? J Aller Ther. 2012; 3:e105.
58. Hostrup M, et al. Urine Concentrations of Inhaled Salmeterol and its Metabolite a-Hydroxysalmeterol in Asthmatic and Non-Asthmatic Subjects. J Sports Med Doping Stud. 2012; 2:110.
59. Torabi M. Spatial Disease Cluster Detection: An Application to Childhood Asthma in Manitoba, Canada. J Biomet Biostat. 2012; S7:010.
60. Kumar D, et al. Descriptive Study of Head Injury and its Associated Factors at Tertiary Hospital, Northern India. J Community Med Health Educ. 2012; 2:141.
61. Loh LC. Risks of Long-Acting Beta-Agonist in Asthma-Perceived or Real? J Pulmonar Respirat Med. 2012; 2:e115.
62. Lee and Hsu AAL. Pulmonary Embolism-A Mechanical Compression Effect on Lower Limb Deep Venous Thrombosis. J Blood Disorders Transf. 2013; 5:183.
63. Sobhy E, et al. Successful Treatment of Right- Sided Heart Thrombus with Pulmonary Embolism with Thrombolytic Therapy. J Cardiovasc Dis Diagn. 2013; 1:121.
64. Poowanawittayakom N and Clarke C. Overweight and Obesity does not Increase Severity of Pulmonary Embolism. J Obes Weight Loss Ther. 2014; 4:206.
65. Atilla N, et al. Bone Cement Pulmonary Embolism after Percutaneous Vertebroplasty. J Clin Case Rep. 2014; 4:416.
66. Lobo JL, et al. Right Atrial Size and 30-Day Mortality in Normotensive Patients with Pulmonary Embolism. J Pulm Respir Med. 2014; 4:218.
67. Agrawal H, et al. Deep Vein Thrombosis /Pulmonary Embolism in a Patient with Retroperitoneal Fibrosis: A Case Report. J Clin Case Rep. 2012; 2:154.
68. Demir N, et al. The Value of Cardiac Troponins in Diagnosis and Differential Diagnosis of Pulmonary Embolism. J Pulmon Resp Med. 2012; 2:134.
69. West M, et al. Does Deep Vein Thrombosis Usually Precede a Pulmonary Embolism. J Trauma Treat. 2013; 2:168.

70. Alwassia AA, et al. Pericarditis Post Pacemaker Implantation Progressing To Cardiac Tamponade after Treatment of an Incidental Pulmonary Embolism. J Cardiovasc Dis Diagn. 2013; 1: 119.
71. Barsoum EA, et al. Uncommon Presentation of Fatal Pulmonary Embolism. Angiol. 2013; 1:106.
72. Wu D. New Insights into the Pathological Features of Asthma/COPD and Pulmonary Arterial Hypertension. Air Water Borne Dis. 2012; 1:e113.
73. Rappaport H and Bonthapally V. The Direct Expenditures and Indirect Costs Associated with Treating Asthma in the United States. J Aller Ther. 2012; 3:118.
74. Askin D, et al. Spontaneous subcutaneous and muscular bleeding due to Acquired Hemophilia a in Association with Poorly- Controlled Asthma: A Case Report. J Blood Lymph. 2012; 2:107.
75. Reyes LR, et al. Clinical Characteristics and Survival of Patients with Pulmonary Hypertension: A 40-Month Mean Follow-Up. J Pulm Respir Med. 2015; 5:235.
76. Seimetz M, et al. Pulmonary Hypertension Precedes Emphysema: Paradigm Shift or Artifact of Rodent Studies?. Angiol Open Access. 2014; 2:e108.
77. Houck PD. An Unusual Case of Dyspnea. Primary Health Care. 2014; 4:169.
78. Gumus A, et al. An Evaluation of Chronic Dyspnea in a Chest Disease Clinic. J Pulm Respir Med. 2014; 4:173.
79. Oâ€™Neill ML, et al. 22 Year Old Female with Worsening Dyspnea. OMICS J Radiology. 2013; 2:131.
80. Rohani A. Dyspnea after CABG, What Do You Think: A Case Report. J Clin Case Rep. 2012; 2:162.
81. Ghosh S, et al. Lung Bullae, Cavitation and Right Ventricular Thrombus Formation with Saprophytic Aspergillus Colonisation: A Rare Presentation of Idiopathic Pulmonary Embolism. J Clin Case Rep. 2014; 4:442.
82. Agrawal H, et al. Deep Vein Thrombosis /Pulmonary Embolism in a Patient with Retroperitoneal Fibrosis: A Case Report. J Clin Case Rep. 2012; 2:154.
83. Poowanawittayakom N and Clarke C. Overweight and Obesity does not Increase Severity of Pulmonary Embolism. J Obes Weight Loss Ther. 2014; 4:206.
84. Nath MP, et al. Anaesthetic Management of Massive Pulmonary Embolism: Case report and Review. J Clin Case Rep. 2014; 4:466.
85. Sachithanandan A. The Role for Surgery in the Contemporary Management of Patients at High or Intermediate Risk of A Pulmonary Embolism-Related Death- is a Paradigm Shift Required? J Pulm Respir Med. 2014; 4:204.
86. Atilla N, et al. Bone Cement Pulmonary Embolism after Percutaneous Vertebroplasty. J Clin Case Rep. 2014; 4:416.
87. Taute BM, et al. Spectral Doppler Waveform Analysis of Common Femoral Veins for the Detection of Right Ventricular Dysfunction in Acute Pulmonary Embolism. J Cardiovasc Dis Diagn. 2015; 3:187.
88. Koray Ak, et al. Successful Management of Massive Bilateral Pulmonary Embolism by Unilateral Ultrasound-Accelerated Thrombolysis after Cardiac Surgery. Angiol Open Access. 2014; 2:138.
89. Vicente Gomez V, et al. D-Dimer Specificity for the Diagnosis of Acute Symptomatic Pulmonary Embolism. J Hematol Thrombo Dis. 2015; 3:177.
90. Lobo JL, et al. Right Atrial Size and 30-Day Mortality in Normotensive Patients with Pulmonary Embolism. J Pulm Respir Med. 2014; 4:218.
91. Lee and Hsu AAL. Pulmonary Embolism-A Mechanical Compression Effect on Lower Limb Deep Venous Thrombosis. J Blood Disorders Transf. 2013; 5:183.
92. Sobhy E, et al. Successful Treatment of Right- Sided Heart Thrombus with Pulmonary Embolism with Thrombolytic Therapy. J Cardiovasc Dis Diagn. 2013; 1:121.
93. Ekpe EE and Obot V. Indications and Outcome of Surgery in Pleuropulmonary Tuberculosis. Trop Med Surg. 2014; 2:174.
94. Agbaji O, et al. Factors Associated With Pulmonary Tuberculosis-HIV Co-Infection in Treatment- Naive Adults in Jos, North Central Nigeria. J AIDS Clin Res. 2013; 4:222.

95. Rosado-Quiab U, et al. Influence of Family System Characteristics on Adherence to Directly Observed Treatment, Short-Course (Dots) in Pulmonary Tuberculosis-A Cohort Study. J Mycobac Dis. 2014; 4:166.
96. Mboowa G. Tuberculosis and Genetics of Sub-Saharan Africa Human Population. J Mycobac Dis. 2014; 4:164.
97. Onal CO and Kibaroglu E. Pulmonary Tuberculosis Caused By Immunosuppressive Treatment. J Clin Case Rep. 2014; 4:383.
98. Pingle P. Application of Bleach Concentration Method in Tissue Samples Received for Diagnosis of Extra Pulmonary Tuberculosis Diagnosis. J Med Microb Diagn. 2014; 3:168.
99. Das S. Changing Trend of Surgery in Pulmonary Tuberculosis. J Pulm Respir Med. 2015; 5:225.
100. Trabulo D, et al. Sweet Syndrome and Pulmonary Tuberculosis in a Crohn's Disease Patient Treated with Anti-TNFα. J Gastrointest Dig Syst. 2015; 5:262.
101. Vishvender S, et al. Preventive and Curative Aspect of Yoga in Management of Asthma in Children. J Homeop Ayurv Med. 2014; 3:152
102. Hudd TR, et al. Survey of Certified Asthma Educator (AE-C) Pharmacists â€"Who are they and how is this Credential Being Used?. J Pulm Respir Med. 2014; 4:223.
103. Mincarini M, et al. Allergen Specific Immunotherapy in Asthma. J Allergy Ther. 2014; 5:190.
104. Sabbah I, et al. Influence of Air Quality Conditions on Asthmatic Patient Visits in Kuwait. J Allergy Ther. 2014; 5:197.
105. Brooks SM. Irritant-Induced Asthma and Reactive Airways Dysfunction Syndrome (RADS). J Allergy Ther. 2014; 5:174.
106. Hasanloei MAV and Athari SS. Proper Care of Allergic Asthma before Hospitalization. J Allergy Ther. 2014; 5:161.