Evaluation of interstitial lung diseases patients admitted at Abbasia Chest Hospital

Thesis submitted for

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List of Abbreviations

%	Percent
µl	Microliters
6MWT	Six-minute walk test
ABG	Arterial Blood Gases
ACE	Angiotensin converting enzyme
AEP	Acute eosinophilic pneumonia
AIP	Acute interstitial pneumonia
ANA	Anti-nuclear antibodies
ANCA	Anti-neutrophil cytoplasmic antibodies
ARDS	Acute respiratory distress syndrome
ATS	American Thoracic Society
BAL	Bronchoalveolar lavage
BM	Basement membranes
BOOP	Bronchiolitis obliterans organising pneumonia
CPK	Creatine phosphokinase
CFA	Cryptogenic fibrosing alveolitis
CI	Confidence interval
CMV	Cytomegalovirus
COP	Cryptogenic organising pneumonia

COPD	Chronic obstructive pulmonary disease
CT	Computed tomography
CTD	Connective tissue disease
DAD	Diffuse alveolar damage
DIP	Desquamative interstitial pneumonia
DL CO	Diffusing capacity of the lung for CO
DNA	Deoxyribonucleic acid
DPLD	Diffuse parenchymal lung disease
EBV	Epstein Barr virus.
ELISA	Enzyme-Linked-Immune-Sorbent-Assay
ERS	European Respiratory Society
ESR	Erythrocyte Sedimentation Rate
FEV1	Forced expiratory volume in one second
Fig.	Figure
FiO_2	Fraction of inspired Oxygen
FRC	Functional residual capacity
FVC	Forced vital capacity
GGO	Ground glass opacity
GIP	Giant cell interstitial pneumonia
Hb	Hemoglobin

HCT	Hematocrit
HDL	High-density lipoprotein
HIV	Human immunodeficiency virus
HLA	Human leukocyte antigen
HP	Hypersensitive pneumonitis
HRCT	High resolution computed tomography
HSV	Herpes simplex virus
IBW	Ideal body weight
ICU	Intensive care unit
IHS	Idiopathic hypereosinophilic syndrome
IIP	Idiopathic interstitial pneumonia
ILD	Interstitial Lung Disease
IPF	Idiopathic pulmonary fibrosis
Kg	Kilogram
LAM	Lymphangioleiomyomatosis
LBW	Lean body weight
LDH	Lactate dehydrogenase
LDHC	Low-dose Hydrocortisone
LDL	Low-density lipoprotein
LIP	Lymphoid interstitial pneumonia
MCP-1	Monocyte chemoattractant protein-1

MDR	Multidrug Resistant
Mg	Milligram
$MgSO_4$	Magnesium Sulphate
mm	Millimeter
mm Hg	Millimeter mercury
MRI	Magnetic resonant image
MRSA	Methicillin Resistant Staphylococcus aureus
n.	Number
NSIP	Non-specific interstitial pneumonia
OR	odds ratio
PaO2	arterial O2 pressure
PAP	pulmonary alveolar proteinosis
PCP	Pneumocystis pneumonia
PCR	Polymerase chain reaction
Plt	Platelets
PO 2	Alveolar–arterial pressure difference for O2
RBILD	Respiratory bronchiolitis-associated interstitial lung disease
REM	Rapid eye movement
RNA	Ribonucleic acid
RSV	Respiratory syncytial virus

RV	Residual volume
S.aureus	Staphylococcus aureus
SaO2	arterial O2 saturation
SD	Standard Deviation
SGOT	Serum glutamic oxaloacetic transaminase
SGPT	Serum glutamic pyruvic transaminase
SPE	Simple pulmonary eosinophila
TBB	Transbronchial biopsy
TLC	Total lung capacity
TLC	Total leucocytic count
UIP	Usual interstitial pneumonia
VATS	video-assisted thoracoscopic
WBC	White blood cells
WHO	World health organization

INTRODUCTION

Interstitial Lung Disease is also known as diffuse parenchymal lung disease (DPLD) and refers to a large group of acute and chronic pulmonary diseases characterized by damage to the lung parenchyma, with varying patterns of inflammation and/or fibrosis **(Leslie, 2006).**

Presence of inflammation was proven by research on alveolitis conducted during the mid-1970s and early 1980s following the introduction of a new medical procedure called bronchoalveolar lavage (BAL); Later with the introduction of high resolution computed tomography (HRCT), the presence of ground glass opacities was found to be consistent with edema and/or inflammation, features viewed as 'less fibrotic' occurring in very stages of the disease. This was the rationale behind treating these patients with high-dose of anti-inflammatory drugs such as dexamethasone. However, as clinicians became confronted with the fact that many forms of ILD are recalcitrant to corticosteroid treatment, a new hypothesis of disease pathogenesis emerged, namely that fibrosis itself could arise and progress in the absence of inflammation **(Chapman, 2004).**

The new hypothesis relating to the absence of inflammation in the progression of lung fibrosis and this new pathologic classification have been well-received by specialists because the histopathology of different types of lung fibrosis is clearly defined and provides a basis for research on antifibrotic therapeutics, instead of anti-inflammatory agents (**Du Bois & King, 2007).**

With the general acceptance of these distinct histological patterns together with the changing nature of the pathophysiologic model during the final three decades of the last millennium, the American Thoracic Society (ATS) and the European Respiratory Society (ERS) put forth in the years 2000-2002 joint consensus statements that defined classification, diagnosis, and management of ILD **(ATS/ERS, 2002)**.These guidelines classify the ILDs based on clinical, radiologic, and pathologic findings into four main categories: (1) ILD of known cause (e.g. environmental exposure, drug exposure, association with a connective tissue disease (CTD), etc.) (2) Granulomatous ILD (i.e. sarcoidosis, Wegener's Granulomatosis) (3) extremely rare ILD with well-defined clinico-pathologic features (e.g. pulmonary alveolar proteinosis (PAP), eosinophilic pneumonia, lymphangioleiomyomatosis (LAM), etc.) (4) Idiopathic interstitial pneumonia (IIP) (Figure 4). The latter is further subdivided into seven distinct clinico-pathologic disease entities, the causes of which are unknown by definition. In the order of relative frequency, they are: idiopathic pulmonary fibrosis (IPF), non-specific interstitial pneumonitis (NSIP), cryptogenic organizing pneumonia (COP), acute interstitial pneumonitis (AIP), respiratory bronchiolitis-interstitial disease (RBILD), desquamative interstitial pneumonia (DIP), and lymphoid interstitial pneumonia (LIP)