A survey on the relationship between pneumonia and covid-19 pneumonia in Dhaka at present circumstance

M. Pharm (Master's) Thesis Report

A thesis report submitted to the Department of Pharmacy, Daffodil International University for the partial fulfillment of Masters of Pharmacy Degree.

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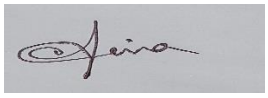

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Dedicated to

My Parents and Supervisor

Abstract

The signs and symptoms of COVID-19 pneumonia are comparable to different sorts of viral pneumonia; this makes it tough to inform the distinction between the two until we get examined for COVID-19 and different respiratory infections. Pneumonia due to Covid-19 impacts each lung whilst pneumonia due to different respiratory prerequisites tends to have an effect on solely one. In this thesis form there are 105 responses, though there are 32 respondent's responses positively they are suffering from pneumonia /covid-19 pneumonia, among them 50% are pneumonia and 50% are covid-19 pneumonia. They face lots of symptoms, found relationship between pneumonia and covid-19 pneumonia, taken treatment, following some side effect of medicine, maintain their lifestyle for controlling pneumonia /covid-19 pneumonia.

Keywords: WHO, COVID-19, Pneumonia, Lifestyle

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List of Abbreviations

DIU-Daffodil International University

WHO-World Health Organizations

SARS-Severe acute respiratory syndrome

MERS-Middle East respiratory syndrome

PCR-Polymerase chain reaction

LRTI-lower-respiratory-tract contamination

ARDS-Acute respiratory distress syndrome

ECMO-Extra corporeal membrane oxygenation

HFNC-High Flow Nasal Cannula

NMB-Neuromuscular blockade

PEEP-Positive give up expiratory stress

CHF-Coronary heart failure

RCTs-Randomized Controlled Trials

CHAPTER-1

INTRODUCTION

1.1. INTRODUCTION

Pneumonia is a frequent sickness that continues to be the primary killer of younger adolescents in growing international locations and aged human beings in developed countries. Many microorganisms are related with pneumonia, and now interest is turning to the significance of viruses as pathogens. Widespread introduction of Haemophilus influenzae kind b and pneumococcal conjugate vaccines into immunisation programmes has led to hypothesis about the developing predominance of viruses as motives of childhood pneumonia. The emergence of extreme acute respiratory syndrome (SARS), avian influenza A (H5N1) virus, and the 2009 pandemic influenza A (H1N1) virus has re-emphasised the essential position of respiratory viruses as motives of extreme pneumonia. New respiratory viruses—such as human metapneumovirus, coronaviruses NL63 and HKU1, and human bocavirus—have been found all through the previous decade. Importantly, the availability of molecular diagnostic assays (such as PCR) has radically expanded our capacity to realize and characterise the epidemiology of respiratory virus infections. Findings of preceding studies, in which traditional virological diagnostic methods have been used, have most probably underestimated the function of viruses as pneumonia pathogens.[1–5] In this Seminar, we evaluate viral neighborhood received pneumonia in immunocompetent youngsters and adults, focusing on research that have used cutting-edge molecular diagnostic techniques.

The ongoing outbreak of the coronavirus sickness 2019 (covid-19) has posed giant challenges for the lookup and clinical communities. This overview focuses on the epidemiologic and scientific aspects of covid-19, the pathophysiologic mechanisms, inpatient respiratory support, and the proof to date on drug treatments. It additionally covers the recuperation and lengthy time period administration of sufferers with covid-19 pneumonia. The evaluate is aimed at clinicians and intensivists caring for sufferers with extreme covid-19 pneumonia as described via the National Institutes of Health,[6] referring to men and women with SARS-CoV-2 contamination demonstrated via polymerase chain response (PCR) checking out who have SpO2 30 breaths/min, or lung infiltrates >50%.

At the cease of 2019, a novel coronavirus (COVID-19) emerged in Wuhan, Hubei Province, China [7]. Reports confirmed that the COVID-19 contamination triggered clusters of onset comparable to extreme acute respiratory syndrome (SARS) coronavirus [7, 8]. A preceding learn about has proven that coronaviruses can purpose respiratory and intestinal infections

in animals and human beings [9]. Generally, coronaviruses had been now not viewed to be notably pathogenic to human beings till the outbreak of SARS in 2002 and 2003 in Guangdong, China[10,11]. Another noticeably pathogenic corona virus, Middle East respiratory syndrome (MERS) coronavirus, emerged in Middle Eastern international locations in 2012[12]. COVID-19 is one greater pretty pathogenic coronavirus to human beings in history.

The virus has raised world issue due to the fact of its excessive transmission functionality as properly as excessive mobility and mortality[8,13–15]. As of 14 February 2020, greater than 60 zero instances with over 8000 sufferers with extreme contamination with the virus have been reported, and greater than 1500 sufferers died. In addition to China, there have been sufferers detected in 25 nations globally. Early reviews confirmed that nearly all of proven sufferers have proof of pneumonia[13,15]. However, pneumonias are very frequent at some point of a time of yr when respiratory ailments prompted with the aid of different pathogen infections are particularly customary[16,17]. Therefore, it is a hard time for public fitness as nicely as physicians in this outbreak.

CHAPTER-2

LITERATURE REVIEW

2.1. EPIDEMIOLOGY OF PNEUMONIA AND COVID 19 PNEUMONIA

According to WHO estimates, 450 million instances of pneumonia are recorded each year; about four million human beings die from this illness, accounting for 7% of complete mortality of fifty-seven million people. [18,19] The very best incidences occur in youth youthful than 5 years and in adults older than seventy-five years (figure 1).[20] In creating countries, incidence ought to be 5 instances greater than in developed regions. In children, 156 million episodes of pneumonia are recorded annually, of which 151 million are existing in creating countries. [18,19] In 2008, 1·6 million youth youthful than 5 years died from pneumonia.[21] 5 million instances of childhood community-acquired pneumonia are said every year in developed countries; however, mortality has declined strikingly and is now very rare. In a Canadian study,[25] 319 admissions for childhood pneumonia took area in the course of the 9-year find out about period; eleven deaths have been recorded and solely one demise did now not have a comorbid condition.[22] Mortality of 1·2 per million formerly healthful younger adults has been recorded in the UK.[23] In the USA alone, the monetary burden of community-acquired pneumonia has been estimated to be extra than US$17 billion annually.[24]

At the time of writing, covid-19 is accountable for 116 million instances globally and 2.5 million deaths.[25] The most hanging attribute of the sickness is its heterogeneity, ranging from no signs to integral illness.[26] Older age, male sex, race (particularly Black, Hispanic, and South Asian), and comorbidities (including hypertension, diabetes, cardiovascular disease, continual pulmonary disease, continual kidney disease, cancer, and persistent liver disease) have been related with worse outcomes.[27-32] Genetic elements can also play a section as well, with blood kind A related with a greater danger for extreme disease.[33] A frequent attribute of SARS-CoV-2 is asymptomatic transmission,[34] which is in all likelihood the purpose of rampant unfold and transmission.[35] Given SARS-CoV-2 entry is mainly by means of the respiratory tract, higher and decrease respiratory tract involvement is the most frequent manifestation.[36] About one 1/3 of sufferers hospitalized with SARS-CoV-2 contamination meet criteria for acute respiratory misery syndrome.[37] In-hospital mortality, whilst firstly very excessive in sure sequence (60% for these intubated in a massive find out about from New York City in April 202014) has been declining at some stage in the route of the pandemic, with in-hospital survival enhancing from 74.4% (March 2020) to 92.4% (August) in a find out about from New York City,[38] and intensive care

unit (ICU) survival enhancing from 58% (March) to 80% (June) in a giant country wide surveillance database from England.[39]

2.2. PATHOPHYSIOLOGIC MECHANISMS

Structure of sars-cov-2

SARS-CoV-2 is a high-quality sense, single stranded RNA enveloped virus in the Beta coronavirus genus.[40] Bats and pangolins may additionally be the animal hosts of SARS-CoV-2 as there is a >90% gene homology to the SARS-CoV-2 discovered to infect humans.[41] Currently it stays uncertain if SARS-CoV-2 used to be at once transferred from bat/pangolins to human beings or an intermediate host was once required for transmission. In mild of the modern pandemic, researchers first in contrast SARS-CoV-2 with the preceding endemic SARS-CoV (2002-03) and MERS-CoV (2012).[42] SARS-CoV-2 has overlapping genetic sequences with SARS-CoV and MERS-CoV, with 79% and 50% homology, respectively. [43,44]

SARS-CoV-2 is characterized by way of 4 most important structural proteins that are essential for infectivity and replication. These proteins consist of the spike (S), membrane (M), envelope (E), and nucleocapsid (N) proteins. [45,46] The S protein, which consists of two protein subunits (S1 and S2), offers the virus its properly recognized look as the S protein protrudes from the membrane. The tip of the protruding S protein has a crown (Latin corona)-like shape.[47] The S protein is additionally necessary for binding to the angiotensin changing enzyme two (ACE2) receptor, which is the factor of entry of the virus to the human and animal host.[48] Furthermore, the S protein is notion to be a fundamental contributor to the immunogenic response; consequently the S protein is the goal of most vaccines.[49] The M protein is a transmembrane protein essential in viral pathogenesis.[50] Little is understood about the E protein; however, it is recognized to play a function in viral replication and infectivity.[51,52] Finally, the N protein approves for law of viral RNA replication, transcription, and synthesis.[53]

SARS-CoV-2 mutations

Emerging facts exhibit one-of-a-kind mutations in the SARS-CoV-2 genome remoted from patients.[54] SARSCoV-2 mutated variations consist of B.1.1.7 (UK variant), P.1 (Brazilian variant) [55], and B.1.351 (South African variant).[56] The fundamental location of mutation for these variations is in the spike protein. The B.1.1.7 variant has a increased price of

infectivity and spread,55 which might also be associated to binding affinity to the ACE2 receptor.[57]

SARS-CoV-2 invasion and replication in cells (fig 1)

Early understanding of the entry method of SARS-CoV-2 into host cells, by using the binding of the S protein to the ACE2 receptor, used to be extrapolated from what was once regarded from SARS-CoV.[58,59] Human ACE2 (hACE) receptor is the identical receptor used by way of SARS-CoV for viral entry. hACE receptor is comparable throughout animal species however with a assorted binding efficiency.[60] Older age and male intercourse of the host are additionally determinants of S protein-ACE2 binding efficiency.[61] ACE2 receptors are enormously expressed in the top respiratory tract of humans. Proteolytic cleavage of the S protein via serine proteases along with transmembrane protease serine two (TMPRSS2), cathepsin L, and furin, are required for binding to the ACE2 receptor. Similar to the ACE2 receptor, protease expression varies by means of tissue kind and location, with a excessive expression in the nasal and bronchial epithelium.[62] In addition, human epithelial cells that line mucosal surfaces and cowl organs such as conjunctiva, gastrointestinal tract, liver, and kidney additionally categorical ACE2 and TMPRSS2.[63,64] Once the virus attaches to the host telephone receptors, it undergoes endocytosis, viral maturation, replication, and launch of extra virus inside the cytoplasm of the host cell. SARS-CoV-2 contamination starts off evolved with viral replication and partly avoids host focus throughout the preliminary contamination and earlier than the host innate response is enabled.[65]

Host response

Limited mechanistic statistics are handy on the innate immune response to SARS-CoV-242 though enlargement of in vitro studies, animal models, and covid-19 affected person serum profiles has been significant.[66] It is now evident that over the first few days after SARS-CoV infection, activation of toll-like receptors (TLR 3, 7, and 8) by way of pathogen focus receptors (PRRs) induces transcriptional upregulation of interferons (type I and III interferons) and recruitment of leukocytes.[66]

The magnitude of the innate antiviral response has been related with the diploma of infection, which would possibly account for the heterogeneous viral response amongst these contaminated with covid-The adaptive immune response begins with IgA, IgG, and

IgM particular antibody launch comparable to the response to SARS-CoV. The timing of antibody launches and the persistence of detectable tiers has various amongst patients.[67] Case and observational research in sufferers with SARS-CoV-2 confirmed early detection of precise IgA and IgM antibodies (within 5 days) and late detection of particular IgG antibodies (after 14 days). In addition, sickness severity has these days been proven to power and more suitable antibody response, [68,69] which correlates with scientific outcomes.[70]

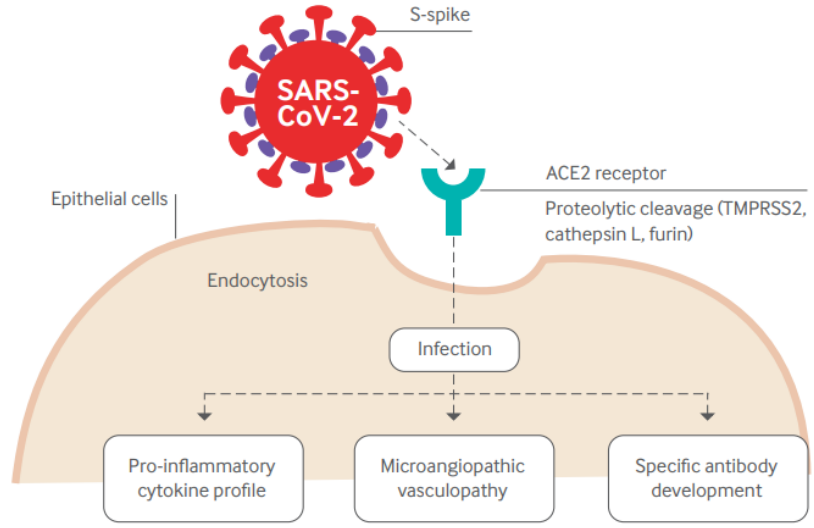

Fig 1 | SARS -CoV-2 S spike protein binds to the ACE2 receptor, which leads to proteolytic cleavage by TMPRSS2, cathepsin L, and furin in the epithelial cell of the respiratory tract. The virus undergoes endocytosis, viral maturation, replication, and release of more virus within the cytoplasm infecting the host cell. Consequences of infected cells include pro-inflammatory cytokine secretion, microangiopathic vasculopathy, and B cell secretion of specific SARS - CoV-2 antibodies.

Clinical remark of lymphopenia has been obvious when you consider that the begin of the covid-19 pandemic and may additionally be related with worsening disease.[71] An enough T telephone response (both CD4+ and CD8+ T cells) directed towards SARS-CoV-2 has been proven to be related with milder disease.[72] Aging is properly mounted to be related with failure of regeneration of naive T cells and T mobile phone activation.[73] In covid-19, dysregulation of T mobile homeostasis has been postulated as a mechanism for extreme ailment considered in older adults. Direct anti-SARS-CoV-2 antibodies have been manufactured for therapy by using Regeneron (REGN10933 and REGN 10987) and Eli Lilly (LY-CoV016) to bind to the viral receptor binding domain. Concern is ongoing that the mutations would provide the virus the capability to break out direct binding to the

unique antibodies. More lookup is wished to entirely perceive the have an impact on the virus mutations have on the remedy modalities available.

Early descriptions of covid-19 covered improvement of a cytokine storm as a harbinger for scientific deterioration.[74] Clinical and serologic proof factors to excessive tiers of serum IL-6, IL-1β, and TNF-α which are related with scientific instability and different biomarkers of inflammation.[75-77] More current research evaluating serum cytokine measurements with different recognized cytokine mediated illnesses such as sepsis and cytokine launch syndrome have cited that covid-19 patients' serum cytokine ranges have been considerably lower.[78] As a result, the direct function of cytokines in ailment pathogenesis has been challenged.[78] Many unanswered questions associated to the pathogenesis of infection and the mechanism of motion of corticosteroids in covid-19.

Autopsy research of sufferers who have died from extreme SARS CoV-2 contamination disclose presence of alveolar wall damage and diffuse alveolar injury steady with ARDS.[79,80] However, in contrast with basic ARDS, post-mortem research additionally point out greater thrombus burden in pulmonary capillaries, which suggests a increased pathogenic function of thrombotic and microangiopathic vasculopathy in covid-19 associated ARDS. Studies together exhibit that thromboembolism takes place greater often and is related with a greater mortality in sufferers with covid-19.[84,82] Additional research are wanted to delineate the direct medical penalties of accelerated thrombosis and its affiliation with mortality in covid-19, which have predominant implications for the administration of respiratory failure. Current research are ongoing to look into remedy with anticoagulants, which may also shed mild on the significance of thrombosis in covid-19 ARDS.

2.3. CAUSES OF VIRAL PNEUMONIA

Pediatric research

We recognized 9 research of community-acquired pneumonia (n=4279 episodes) in which the viral motive had been searched for with the aid of PCR. In most of these investigations, virus lifestyle and antigen detection had been also used. [21,30,40–46] Seven research have been undertaken in developed nations and two in creating countries. Evidence of viral contamination used to be recorded in 49% (range 43–67) of cases. Prevalence of community-acquired pneumonia related with respiratory syncytial virus (11%), influenza

viruses (10%), parainfluenza viruses (8%), and adenovirus (3%) used to be comparable to that said in research in which solely traditional diagnostic methods have been used. [1,2] Exact numbers of specific viruses are challenging to examine from one learn about to every other due to the fact numerous methods have been applied. When serological assays on my own had been used, proof of a viral reason used to be received in 20–43% of youth with community-acquired pneumonia, and respiratory syncytial virus was once dominant. [47–50] PCR has expanded detection of rhinoviruses (18%) and enteroviruses (7%). Of newly described viruses, human bocavirus was once recorded in 5% of instances and human metapneumovirus in 8%. Coronaviruses had been considered in 22 (7%) of 338 youngsters in one study.[41] In a 3-year potential learn about in Finland, the basic probably motive of pneumonia was once recorded in 85% of children, with bacterial contamination in 53% and viral contamination in 62%.[30] The most complete find out about from a virological viewpoint searched for 14 viruses in 338 kids with pneumonia over a 2-year period.[41] Prevalence of viral contamination used to be 67%, with respiratory syncytial virus, rhinoviruses, human bocavirus, human meta pneumo virus, and parainfluenza viruses being the most frequent agents.

	Rhinovirus (n=580)	Respiratory syncytial virus (n=1655)	Adenovirus (n=902)	Parainfluenza virus 1 (n=94)	Parainfluenza virus 2 (n=49)	Parainfluenza virus 3 (n=315)	Influenza A virus (n=544)	Influenza B virus (n=139)
Pneumonia	18%	16%	8%	9%	6%	14%	9%	8%
Wheezy bronchitis	22%	12%	2%	2%	4%	8%	6%	6%
Otitis media	23%	59%	24%	27%	20%	30%	26%	19%
Non-specified acute respiratory infection	14%	32%	37%	27%	22%	50%	44%	53%
Bronchiolitis	3%	34%	1%	2%	10%	5%	1%	1%
Laryngitis	2%	2%	1%	37%	53%	10%	5%	4%
Tonsillitis	2%	0	30%	1%	0	2%	5%	4%
Fever without a focus	2%	1%	5%	10%	0	2%	1%	2%
Febrile convulsion	1%	2%	7%	4%	0	5%	12%	9%
Fever ≥38°C	44%	63%	81%	77%	76%	63%	94%	89%

Rhinovirus infections are from 1987 to 2006; other respiratory virus infections are from 1980 to 1999. Modified from reference 51, with permission of John Wiley and Sons.

Table 1: Occurrence of pneumonia and other findings in 4277 children with laboratory-confirmed viral respiratory infection at Turku University Hospital, Finland

Many researchers have centered on the position of single respiratory viruses as a purpose of childhood neighborhood received pneumonia or have studied sole virus infections and regarded for pneumonia in their scientific profiles (table 1). Globally, respiratory syncytial virus continues to be the primary causative viral agent of pneumonia in young people and may want to be the predominant viral purpose of extreme pneumonia in this population.[52,53] With the introduction of PCR techniques, rhinoviruses have been detected

an increasing number of in childhood pneumonia.[54] The scientific profile of 643 rhinovirus infections in adolescents admitted to health center has been stated in seven studies,[55–61] and 11–53% had pneumonia. However, the function of rhinoviruses in pneumonia is nevertheless wondered due to the fact of the commonplace detection of rhinoviruses in asymptomatic people (mean occurrence 15%), strikingly greater than for different respiratory viruses (prevalence 1–5%).[62] Jartti and colleagues cautioned that PCR is probably to become aware of a authentic however asymptomatic infection.[62] A problem with rhinoviruses is the paucity of serological checks to affirm acute infection. In immunocompetent individuals, rhinoviral clearance after symptomatic contamination is fast (average 1–3 weeks).[18]

Pneumonia was once identified in 10% of adolescents admitted with acute human metapneumovirus respiratory infection,[63–66] with the best occurrence (44%) in babies youthful than 12 months.[66] It has additionally been recorded in 11–75% of youth with human bocavirus infection.[67] In a find out about from Thailand of toddlers youthful than 5 years admitted with pneumonia, human bocavirus used to be the 0.33 most frequent agent detected, after rhinovirus and respiratory syncytial virus, accounting for 12% of all cases.[68] Although the position of human bocavirus in pneumonia is nonetheless being clarified, serological proof suggests it is a purpose of human infection. With a novel IgM and IgG enzyme immunoassay, 96% of teenagers with a excessive load of human bocavirus in nasopharyngeal aspirates and 92% of wheezy youngsters with viraemia had diagnostic seroresponses.[69] Human bocavirus used to be recognized serologically in 12 (12%) of one hundred and one teenagers with community-acquired pneumonia in Italy.[70]

Although incidence of adenovirus-associated pneumonia is pretty low (range 2–12%), this kind of contamination is essential to realize due to the fact it may result in extreme and deadly necrotizing pneumonia (especially serotypes 3, 7, and 14).[30,40–46,71] In China, adenovirus DNA used to be detected in 9% of autopsy pulmonary tissue specimens from one hundred seventy five teens with deadly pneumonia.[72] Of note, PCR is drastically greater touchy for identification of adenovirus than is antigen detection.[73]

Human coronaviruses 229E and OC43, and newly observed kinds NL63 and HKU1, have been linked to community-acquired pneumonia in children. [74,75] Infection with human coronavirus used to be detected in 3% of teenagers and youth in a giant pneumonia learn about in Thailand.[76]

Research in adults

We identified ten research of adults with community? acquired pneumonia (n=2910 episodes) in which PCR was used to take a look at for respiratory viruses. Evidence of viral infection used to be detected in 22% of cases. [19,20,77–85] In most of these studies, a complete array of traditional virological strategies had been additionally applied to higher defi ne the function of viruses in adults with community? acquired pneumonia. Similar to findings of pediatric studies, occurrence of contamination with influenza viruses (8%), respiratory syncytial virus (3%), parainfluenza viruses (2%), and adenovirus (2%) is similar with values recorded with traditional diagnostic techniques alone.[5,36] Serological methods solely had been used in 4 studies; proof of viral community-acquired pneumonia was mentioned in 10–23% of patients.[86–89] Use of PCR has augmented detection of viruses that are difficult to identify with traditional methods, which include rhinoviruses (6%), human coronaviruses (5%), and human metapneumovirus (1%). As a result, ordinary prevalence of respiratory viral contamination in PCR research (15–56%) is usually greater than for research in which PCR used to be now not implemented. With a full set of tests, findings of three reviews advocate that a 0.33 of person cases of community-acquired pneumonia are related with viral infection. [19,20,85]

Other researchers have centered on the function of specific respiratory viruses in adults with community-acquired pneumonia. Respiratory syncytial virus is known increasingly as a purpose of sickness in adults,[90] and roughly 2–9% of aged sufferers admitted with pneumonia in the USA have contamination related with this virus.[91] Infections with respiratory syncytial virus are linked to good sized mortality.[92] Several outbreaks of extreme respiratory ailment (including deadly pneumonia) in aged residents of nursing homes have been related with rhinoviruses.[93,94] Adenoviruses have been implicated in 90% of pneumonia? related admissions in simple navy trainees.[95] An outbreak of pneumonia related with adenovirus serotype 14 has been reported.[96] When searched for systematically, coronaviruses have been detected in samples from a small proportion (2–6%) of adults with pneumonia.[76,97] These patients had medical ailments indistinguishable from these in men and women with community-acquired pneumonia associated with different micro-organisms. 2% of asymptomatic controls additionally had human coronavirus infection.[76]

Infections with human metapneumovirus occur throughout adulthood. Outbreaks of this

viral contamination associated with deadly result have been suggested from long-term care facilities. [98,99] Of sufferers admitted with human metapneumovirus infection, 27% had chest radiographic infiltrates, 12% required ventilatory support, and 7% died.100 Human bocavirus is a distinctive reason of pneumonia in adults. As phase of a surveillance assignment in Thailand, this virus used to be detected in five (1%) of 667 adults (age 20 years or older) admitted with pneumonia and in one of 126 (1%) controls except febrile or respiratory illness.[68]

Pneumonia related with SARS, avian influenza, and 2009 pandemic influenza

During 2002 and 2003, the SARS coronavirus precipitated severe respiratory contamination in greater than 8000 humans and led to 774 deaths. Up to a 0.33 of sufferers with SARS became seriously ill. Pneumonia with lung damage arose in about 16% of all people contaminated with the virus and in 80% of significantly unwell patients. By distinction with different viral pneumonias, kids had been pretty nicely blanketed from extreme illness.[101] Since November, 2003, avian influenza A (H5N1) virus has triggered extra than 450 human infections, with a case-fatality percentage of about 60%. Multiorgan failure usually develops inside 1 week from onset of illness, with lymphopenia, thrombocytopenia, and raised concentrations of aminotransferase and creatinine. Almost all sufferers with avian influenza increase pneumonia. Cause of loss of life is most normally modern respiratory failure.[102]

Since March, 2009, pandemic influenza A (H1N1) virus has unfolded in greater than 200 nations over the world, inflicting about 18 zero deaths. In the USA alone, more than fifty-nine million human beings have been infected.[103] In Australia, the price of admission used to be 23 per 100 zero population. Critical sickness arose most often in adults with a median age of forty years and has been rare in these older than sixty five years.[103–105] Half of sufferers with imperative sickness had viral pneumonitis or acute respiratory misery syndrome.[103,106] In Germany, pneumonia used to be recognized in 275 (0·7%) of 40729 sufferers with pandemic H1N1 virus infection; half of these have been admitted.[107] In the UK, 102 (29%) of 349 sufferers with chest radiographs had findings consistent with pneumonia. Median age of sufferers with pneumonia used to be 26 years.[108] Poor consequences from H1N1 virus contamination have been recorded in pregnant women, indigenous populations, and men and women with full-size obesity or serious comorbidities.

2.4. PHYSIOLOGICAL CHANGES IN THE RESPIRATORY SYSTEM ASSOCIATED WITH AGEING

Maximum characteristic of the respiratory device is reached at about the age of 20–25 years.[83] Thereafter, getting older is related with an innovative reduce in lung performance; however, except affected through disease, the respiratory device stays successful of retaining sufficient fuel trade throughout the whole lifestyles span. Physiological adjustments related with aging have essential penalties on the purposeful reserve of older people, and their potential to cope with the minimize in lung compliance and enlarge in airway resistance related with lower-respiratory-tract contamination (LRTI).

The most vital physiological adjustments related with growing old are: a limit in the elastic turn away of the lung, a reduce in compliance of the chest wall, and a minimize in the power of respiratory muscles. Alterations in lung parenchyma (enlargement of alveoli, or "senile emphysema", decline in small airway diameter) and the related decline in elastic pull away of the lung motive a make bigger in practical residual potential (FRC): older sufferers consequently breathe at greater lung volumes, growing the workload imposed on respiratory muscles. Calcification and different structural modifications inside the rib cage and its articulations lead to stiffening of the chest wall (ie, diminished compliance), similarly growing the work of breathing. Changes in the structure of the thorax additionally take place as an end result of osteoporosis and vertebral fractures, ensuing in dorsal kyphosis and extended anteroposterior diameter ("barrel chest"), which decreases the curvature of the diaphragm and has a bad impact on its force-generating capabilities. Respiratory muscle overall performance is hence impaired by using the age-related make bigger in FRC, the minimize in chest-wall compliance and the geometric adjustments in the rib cage.83 Respiratory muscle power is additionally affected by way of dietary status, frequently poor in the elderly, and by using age-associated sarcopenia.[84,85] Dysfunction of respiratory muscle groups in conditions the place an extra load is positioned on the respiratory muscles, such as pneumonia, may additionally lead to hypoventilation and hypercapnic respiratory failure. Noteworthy is the reality that everyday values for most inspiratory strain in humans over eighty are under the threshold described in a person populace for clinically applicable respiratory dysfunction. Respiratory muscle feature additionally relies upon on power availability (ie, blood flow, oxygen content); indeed, reduced respiratory muscle energy has been described in sufferers with persistent coronary heart failure (CHF), a established incidence in older patients. [86,87] Other well-known

scientific conditions reducing respiratory muscle characteristic in the aged consist of Parkinson's disorder and sequelae of cerebral vascular disease. [88,89]

Forced expiratory volumes and top expiratory float exhibit an age-related linear decrease, probable indicating structural adjustments and persistent low-grade infection in peripheral airways.[90] In the very old, lowered pressured expiratory float costs and lung elastic pull away might also compromise the efficacy of clearance of airway secretions by way of coughing. It has additionally been advised that, even in the wholesome aged population, mucociliary clearance costs are slowed by using contrast with the young. Indeed, each smoking and non-smoking aged humans have decreased tracheal mucus speed in contrast with youthful individuals. [91,92]

Lower sensitivity of respiratory centers to hypoxia or hypercapnia in older sufferer's effects in a diminished ventilatory response in instances of acute sickness such as coronary heart failure, infection, or aggravated airway obstruction, and for this reason delays essential scientific signs and symptoms such as dyspnea and tachypnoea, which are essential for analysis of pneumonia and understanding of the severity of the related respiratory impairment.[93]

2.5. BURDEN OF CARDIAC COMPLICATIONS IN PATIENTS WITH PNEUMONIA

For countless decades, investigators have mentioned that acute respiratory infections, such as pneumonia, regularly precede the improvement of acute cardiac events, and a causal relation has been proposed.[10,11] For acute coronary syndromes specifically, this affiliation satisfies most of Bradford Hill's standards for causality and is mentioned elsewhere.[11,12] The excessive occurrence of cardiac arrhythmias after an episode of pneumonia and the temporality of this affiliation additionally advise a causal function for pneumonia in instances of pneumonia-associated arrhythmias.[9] Although a comparable argument can be made for the affiliation between pneumonia and coronary heart failure, this relation is likely extra complex. Results of scientific research propose that sufferers with coronary heart failure have decreased immunological responses, and experimental proof shows that pulmonary congestion can promote the increase of frequent micro-organism such as Streptococcus pneumoniae (pneumococcus) and Staphylococcus aureus in the lungs.[13–15] Epidemiological records additionally advise that pre-existing coronary heart failure is a chance aspect for the improvement of pneumonia.[16] Therefore, the cause–effect relation between pneumonia and coronary heart failure would possibly be bidirectional. A causal

relation between infections in different organs, such as the urinary or gastrointestinal tracts, and acute cardiac occasions has additionally been suggested, however has now not but been characterized. [17,18]

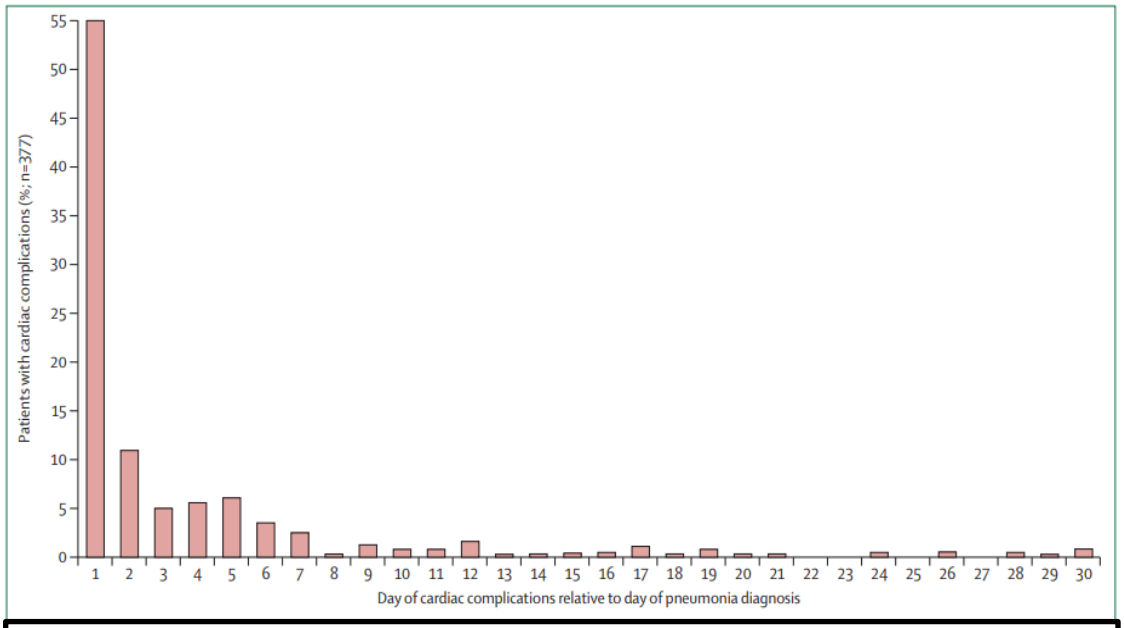

Figure 2: Timing of cardiac complications in patients with community-acquired pneumonia Cardiac complications included any of the following cardiac events: new or worsening heart failure, new or worsening arrhythmias, or myocardial infarction. Data from the Pneumonia Patient Outcomes Research Team cohort study. Adapted from Corrales-Medina et al,9 with permission of Wolters Kluwer Health

Although acute cardiac occasions have been regarded as essential problems in sufferers with pneumonia for the reason that the early twentieth century, the magnitude of this hassle has solely these days begun to be liked fully.[19] A meta-analysis of 25 research reporting the incidence of cardiac activities inside 30 days of pneumonia prognosis mentioned cumulative prices of new or worsening coronary heart failure (14%, vary 7–33%), new or worsening arrhythmias (5%, vary 1–11%), and the acute coronary syndromes myocardial infarction or unstable angina (5%, vary 1–11%) in sufferers admitted to health center with pneumonia.[20] Most studies, however, did now not use clear definitions for the effects investigated, relied on retrospective chart evaluate for their ascertainment, or have been confined to high-risk populations (eg, veterans, sufferers with diabetes, and aged patients). In their 2012 evaluation of a prospective multicenter cohort of 2344 unselected sufferers with community-acquired pneumonia (1343 inpatients and 944 outpatients), Corrales Medina and colleagues mentioned the 30-day incidence of well-defined cardiac complications.[9] In this cohort, new or worsening coronary heart failure, new or worsening arrhythmias, and myocardial infarction happened in, respectively, 21%, 10%, and 3% of

inpatients, and 1·4%, 1·0%, and 0·1% of outpatients. Overall, cardiac issues (defined as any of the aforementioned events) happened in 27% of inpatients and 2% of outpatients. Cardiac arrest happens in up to 3% of sufferers admitted to health facility with community-acquired pneumonia.[21]

The occurrence of two or extra kinds of cardiac match in a affected person with pneumonia is no longer amazing and has been pronounced in 20–40% of sufferers who increase cardiac complications.[9,19] In this setting, the attention of myocardial infarction is normally preceded by means of the prognosis of different cardiac occasions (69% of patients).9 Conversely, new or worsening heart failure and arrhythmias are the first recognized, or only, pneumonia-associated cardiac tournament in most instances (85% and 69%, respectively).

Risk for cardiac issues is greater in the first few days after a pneumonia diagnosis, with about 90% of these activities known inside 7 days of diagnosis, and extra than 1/2 recognized inside the first 24 h (figure 2).[9,22] Risk elements for cardiac problems consist of older age (about 86% of cardiac problems manifest in human beings aged ≥60 years), nursing domestic residence, pre-existing cardiovascular disease, and larger severity of pneumonia at presentation.[9,22,23] Nonetheless, about a 1/3 of pneumonia-associated cardiac problems take place in sufferers with no records of medical cardiac disease, a quarter of instances are in sufferers viewed to be at low danger on the foundation of their pneumonia severity index score, and about three-quarters of instances occur in sufferers concept now not to want intensive care after their first assessment.[9,19,21,24]

Cardiac issues have a necessary impact on the scientific route of sufferers with pneumonia. In sufferers admitted to clinic with pneumonia who trip scientific failure to treatment, about a 1/3 do so due to the fact of cardiac complications.[25] Diagnostic standards for myocardial infarction are current in as many as 50% of sufferers with pneumonia who want intensive care unit remedy inside 24 h of admission to hospital.[23] Cardiac issues are additionally the direct or underlying purpose of demise in 27% of pneumonia-associated deaths.[26] Death inside 30 days of pneumonia analysis is 5 times greater frequent in sufferers who boost cardiac problems than in these who do not. Even after adjustment for baseline risk, cardiac issues are related with a 60% amplify in pneumonia-associated temporary mortality, and lead to one in 4 readmissions after hospitalization for pneumonia.[27]

2.6. RESPIRATORY CARE FOR SEVERE COVID-19 PNEUMONIA

Severe covid-19 pneumonia as described through NIH1 overlaps drastically with the scientific definition of "classic" ARDS.[60] However, a number of unique pathophysiological approaches are postulated to be at play for CARDS, such as intravascular thrombosis induced by way of loss of endothelial barrier, outstanding loss of hypoxic pulmonary vasoconstriction ensuing from endothelial dysfunction, and immoderate blood go with the flow to collapsed lung tissue.[61] Further, now not all case sequence furnish a clear semantic difference between extreme covid-19 pneumonia and CARDS, which confounds interpretation. In this section, we summarize the modern literature on the use of respiratory remedy tools in sufferers with extreme covid-19 pneumonia. To date, no managed potential trials inform the respiratory administration of extreme covid-19 pneumonia. Notwithstanding, amongst sufferers with extreme covid-19 pneumonia, affected person respiratory gadget mechanics and medical results performed with trendy ARDS administration are comparable to basic ARDS. Consequently, cutting-edge respiratory care revolves round supportive measures and is primarily based on the administration of basic ARDS. We start via offering a regular assessment of these concepts.

Titration of oxygen remedy to keep away from hyperoxemia[62,63] and hypoxemia[64] is strongly encouraged for acute hypoxemic respiratory failure. A vary of 90-96% oxygen saturation, tested via cooximetry, is a lifelike target.[63] For sufferers who require invasive mechanical air flow (IMV), the first aim is avoidance of excessive tidal volumes, which are related with ventilator precipitated lung injury.[65,66] Evidence suggests that comparable harm should take place due to the fact of sustained excessive tidal volumes for the duration of spontaneous breathing, additionally recognized as affected person self-induced lung damage (P-SILI).[67-69] Although now not validated in managed scientific trials, an evaluation of pressure regarded as tidal stress or riding pressure[70,71] (defined as the ratio of tidal extent to tidal respiratory device compliance) permits matching of quantity transport with respiratory gadget mechanics and permits finest mechanical ventilatory settings. In an observational learn about of non-covid ARDS trials, mediation evaluation published that 75% of the really helpful impact of remedy group task was once attributable to discount in tidal pressure.[70]

The 2nd purpose of mechanical air flow in ARDS is to forestall the consistent opening and closing of alveoli which may additionally be injurious to the lung (atelectrauma). Positive

give up expiratory stress (PEEP) is titrated to preserve alveolar devices open in the course of the respiratory cycle. Several RCTs that aimed to optimize recruitment in the intervention arm confirmed comparable scientific results to controls[72,73] and a sign for practicable damage which used to be attributed to recruitment maneuvers.[74] To that end, the advantages of greater PEEP are evident solely when decreasing tidal pressure—ie, much less pressure for a given tidal volume.[70] Recruit ability (the capacity to open and hold alveoli open) can be assessed at the bedside by means of calculating the recruitment/inflation (R/I) ratio.[75,76] For sufferers who are tested recruitable, using the excessive PEEP and Fi O2 table 2 may additionally be preferable whilst monitoring cardiac output and respiratory mechanics to keep away from concurrent hyperinflation.[77,78]

Prone air flow and neuromuscular blockade (NMB) are ordinary adjuncts in the cure of ARDS. Prone air flow promotes lung recruitment and improves ventilation/perfusion matching by means of developing an extra even distribution of transpulmonary stress for the duration of the chest. A multicenter, potential RCT confirmed that amongst sufferers with extreme hypoxemic respiratory failure (Pa O2 /Fi O2 sixteen hours a day was once related with decreased 28-day mortality.[79] NMB in early ARDS probably reduces lung stress by using casting off spontaneous respiratory activity. Despite before encouraging findings, a latest meta-analysis of 5 RCTs confirmed no mortality benefit, with a modest discount in barotrauma hazard and elevated oxygenation if utilized after forty-eight hours in sufferers with extreme ARDS.[80]

The faith that respiratory care standards to deal with traditional ARDS ought to practice in CARDS used to be challenged when until now collection of covid-19 sufferers appeared to point out two extraordinary respiratory failure phenotypes.[81] A case collection (n=16) mentioned that sufferers had low elastance, low air flow perfusion matching, low recruit ability and lung weight which they named the “L type.” Conceivably, such discrepancy of air flow perfusion matching with extraordinarily everyday mechanics used to be attributed to loss of lung perfusion rules and hypoxic vasoconstriction. The rest of the instances have been greater constant with basic ARDS (high elastance, excessive ventilation/perfusion ratio, excessive recruit ability and lung weight) referred to as the “H type.” The authors recommended that sufferers who had the L kind may also now not require low tidal extent air flow and tries at recruitment ought to carry harm. Further, they reasoned that sufferers who current with a paucity of infiltrates, low elastance, and hypoxemia need to be positioned on mechanical air flow previously to stop spontaneous excessive tidal volumes

generated by means of the patients. This proposed want for a distinct administration has been contested on the grounds of inconclusive proof for P-SILI and CARDS case collection that published respiratory device mechanics comparable to basic ARDS. [82,83]

Current observational reviews replicate our trip and fortify our view that a widespread percentage of sufferers with covid-19 pneumonia can be handled non-invasively (ie, excessive glide nasal cannula (HFNC) or non-invasive air flow (NIV)) in lieu of invasive mechanical air flow (IMV). This method may additionally optimize utilization of mechanical ventilators, a scarce useful resource for the duration of the pandemic. We suggest the use of the complete spectrum of non-invasive and invasive gadgets for respiratory help (fig 2). Figure 2 is based totally on our exercise in treating extreme covid-19 pneumonia, and attracts generally from the trip in traditional ARDS. Close monitoring and interest to symptoms of non-invasive machine failure are indispensable for superior outcomes. Extra corporeal membrane oxygenation (ECMO) is handy for sufferers who have refractory hypoxemia after these measures [84] however is sometimes needed.[85]

2.7. DIAGNOSIS OF VIRAL PNEUMONIA

Laboratory prognosis of viral pneumonia has relied on detection of virus or viral antigen in upper-respiratory specimens (eg, nasopharyngeal aspirates) and decrease respiratory samples (eg, brought on sputum) by way of tradition or immune fluorescence microscopy, and on dimension of antibodies in paired serum samples. Introduction of PCR has accelerated the capability to realize respiratory viruses, which include these that are challenging to culture. At least 26 viruses have now been related with neighborhood obtained pneumonia (panel).

Despite technological advances, setting up the reason of pneumonia stays challenging.[13] Specimens from the lower-respiratory tract can be challenging to obtain, and distinguishing feasible extended shedding or colonization from contamination can be difficult. For analysis of viral pneumonia, reliance on checking out of nasopharyngeal specimens offers its very own challenges; detection of a virus in the nasopharynx should characterize coincidental upper-respiratory contamination or a pneumonia pathogen. Measurement of history occurrence of asymptomatic nasopharyngeal viral contamination in a manipulate crew may assist to make clear the dimension of this diagnostic trouble at a populace level, however this method has been used solely not often in etiological studies. Furthermore,

most lookup has centered on sufferers admitted to health facility and, therefore, findings may now not be consultant of mild to moderate disease.

Several distinctive kinds of specimen from the top and decrease airway have been used in etiological research of community-acquired pneumonia, including: nasopharyngeal aspirates or washes; swabs from the nasopharynx, nose, or throat; mixed nasopharyngeal and throat swabs; expectorated and brought on sputum; tracheal aspirates; bronchoalveolar lavage; and lung puncture.[14,15] Recovery of virus fluctuates in accordance to specimen type, which in all likelihood money owed for some of the variability of findings between studies.

Most research of the motive of viral pneumonia have used upper-respiratory specimens to take a look at for viruses. In children, nasopharyngeal aspirates are normally deemed the specimen of desire due to the fact each nasal and nasopharyngeal mucus sample are gathered. Respiratory viruses have been cited in 95% of mucus samples bought by means of nasopharyngeal aspiration from kids with respiratory infection.[16] Obtaining an aspirate is, however, disagreeable and requires a suction device. Nasal swabs taken with a sterile cotton swab from a depth of 2–3 cm have similar sensitivity to nasopharyngeal aspirates for subculture of all principal respiratory viruses, without respiratory syncytial virus.[17] Flocked swabs with nylon fibres in a perpendicular trend are now favored by using many clinicians due to the fact they are handy to use and have a comparable sensitivity to nasopharyngeal aspirates for detection by way of PCR of respiratory viruses.[15,17,18] In adults, nasopharyngeal swabs have a greater sensitivity than throat swabs, however they can be much less touchy than nasopharyngeal washes. Transnasal nasopharyngeal flocked swabs also have excessive virus detection costs in adults. [19,20]

2.8. MANAGEMENT OF PNEUMONIA

Do all sufferers with community-acquired pneumonia, inclusive of these with proof of viral infection, want to be dealt with antibiotics? To date, no clear consensus exists on this issue. Some professionals advocate that all sufferers with pneumonia must get hold of antibiotic treatment, due to the fact exclusion of the presence of bacterial contamination is impossible. Recommendations of the British Thoracic Society are that antibiotic therapy can be withheld in younger youth with moderate sickness in whom viral contamination is likely. As some distance as we know, solely one randomized placebo-controlled learn about has been accomplished to check out the want for antibiotic therapy in childhood

community-acquired pneumonia.[94] In 136 children, no clinically extensive efficacy of antibiotics was once recorded. Most find out about youth had pretty moderate ailment and the investigation was once undertaken all through an epidemic of respiratory syncytial virus, so most members likely had pneumonia precipitated by way of this virus. Further randomized placebo-controlled trials of antibiotic remedy for pneumonia are not going to take place due to the fact of moral concerns.

Opportunities are presently restricted in scientific exercise for use of antivirals in the cure of pneumonia (table 2).[95] Neuraminidase inhibitors, such as oseltamivir and zanamivir, had been developed at some point of the Nineteen Nineties and now have installed roles in early cure of influenza A and B infections. In teens and adults, neuraminidase inhibitors decrease median time to decision of signs and symptoms by using 0·5–2·5 days when administered inside forty eight h of onset of symptoms.[96] Importantly, early use of neuraminidase inhibitors can decrease improvement of issues such as pneumonia.[97] The Infectious Diseases Society of America extends remedy with neuraminidase inhibitors to admitted influenza sufferers whose onset of signs and symptoms is extra than forty eight earlier than presentation.[98] Selection of the most fantastic antiviral to deal with influenza must be made on the groundwork of applicable susceptibility data. Before emergence of the 2009 pandemic H1N1 virus, the seasonal H1N1 virus developed resistance to oseltamivir, and cure with both zanamivir or amantadine or rimantadine used to be recommended, whereas the seasonal H3N2 virus was once resistant to amantadine and rimantadine. If subtype statistics is unavailable, zanamivir or an aggregate of oseltamivir and rimantadine is recommended.[98] The 2009 pandemic H1N1 virus stays inclined to neuraminidase inhibitors, and oseltamivir has been used broadly for cure of pneumonia precipitated by way of this virus. Although resistance to oseltamivir has been mentioned in human beings with 2009 pandemic H1N1 virus infection, it has been generally confined to immunocompromised individuals. All isolates are nonetheless inclined to zanamivir.

Intravenous use of peramivir or zanamivir should be lifesaving in seriously unwell sufferers with influenza. [99,100]

Experience with antivirals for community-acquired pneumonia prompted with the aid of viruses different than influenza is scarce, with present expertise often from case reviews and some therapy research in immunosuppressed patients. Ribavirin has a vast antiviral range, together with respiratory syncytial virus, human metapneumovirus, and

parainfluenza and influenza viruses.[101] Efficacy of ribavirin aerosol remedy for bronchiolitis and pneumonia brought about with the aid of respiratory syncytial virus contamination is modest at best. Intravenous ribavirin should be regarded for remedy of extreme pneumonia precipitated through contamination with respiratory syncytial virus, human metapneumovirus, or parainfluenza virus, on the foundation of ride in immunosuppressed patients.[102]

New antiviral marketers are in improvement for respiratory syncytial virus infection, which includes small interfering RNAs.[103] In quite a few case research of immunocompromised patients, scientific efficacy of cidofovir has been proven for extreme adenovirus pneumonia.[104] Cidofovir must be viewed for therapy of new adenovirus subtype 14 pneumonia. Researchers stated profitable administration of human metapneumovirus pneumonia with a mixture of intravenous ribavirin and immune-globulin.[105] Varicella pneumonia ought to be handled with aciclovir.[106]

	Treatment	Prevention
Influenza A and B viruses	Oseltamivir (oral); zanamivir (inhalation, intravenous); peramivir (intravenous)	Vaccines (inactivated, live); oseltamivir; zanamivir
Influenza A virus	Amantadine (oral); rimantadine (oral)	..
Respiratory syncytial virus	Ribavirin (inhalation, intravenous)	Palivizumab (intramuscular)
Adenovirus	Cidofovir (intravenous)	Vaccine for types 4 and 7*
Rhinovirus	Pleconaril†	Alfa interferon (intranasal)
Enteroviruses	Pleconaril†	..
Human metapneumovirus	Ribavirin (intravenous)	..
Hantavirus	Ribavirin (intravenous)	..
Varicella-zoster virus	Aciclovir (intravenous)	Vaccine

*Long successful use in US military conscripts, no production now. †Has been used for compassionate cases.

Table 2: Possibilities for antiviral treatment and prevention of severe viral pneumonia

Use of corticosteroids for therapy of viral neighborhood obtained pneumonia is controversial and can fluctuate in accordance to the causative virus. The ineffectiveness of these sellers for therapy of respiratory syncytial virus infections is properly established.[107] For administration of SARS, inconclusive consequences have been suggested in 26 cure studies, and feasible damage used to be indicated in 4 trials.[108] High-dose corticosteroids had been administered to a 0.33 of sufferers with 2009 pandemic H1N1 virus infection,[109] however use of these marketers is now not advocated due to the fact of extended viral shedding in seasonal influenza and elevated mortality in avian H5N1 and, possibly, 2009

pandemic H1N1 virus infections. On the different hand, some statistics advocate that corticosteroids can increase consequence of pneumonia brought about with the aid of contamination with varicella-zoster virus (in mixture with aciclovir) and hantavirus.[110]

2.9. PREVENTION OF PNEUMONIA

Possibilities to forestall viral community-acquired pneumonia are limited. Influenza vaccines have been used considering the fact that the mid Forties and they now have a hooked-up function in prevention of influenza A and B virus infections. Importantly, inactivated influenza vaccine is tremendous in younger children, such as these youthful than two years.[111] During the 2009 H1N1 pandemic, a monovalent vaccine in opposition to the virus used to be developed. Its energetic use ought to have performed a phase in the route of the preliminary pandemic wave in some countries—eg, in Finland, solely fourty four deadly instances have been recorded. In addition to vaccines, influenza A and B virus infections can be avoided through prophylactic use of neuraminidase inhibitors. Severe respiratory syncytial virus infections in high-risk neonates have been averted efficaciously with palivizumab, a humanized monoclonal antibody, which is administered throughout a respiratory syncytial virus epidemic.[112] This agent has been proven to forestall admissions associated to respiratory syncytial virus by using 50% in untimely infants. Since the 1960s, various kinds of vaccines for respiratory syncytial virus have been developed besides success. Live-attenuated vaccines produced via reverse genetics are now in medical studies. Pneumonia prompted with the aid of adenovirus kinds four and 7 has been avoided in navy trainees by using an oral vaccine, with 95% efficacy. Unfortunately, struggle over the manufacturing manner stopped manufacturing in 1996.[95] Pneumococcal conjugate vaccine used to be proven to stop a 1/3 of viral pneumonia instances in a learn about in South Africa, most probable via prevention of superimposed bacterial co-infections.[113]

2.10. OUTCOME AND TREATMENTS OF COVID-19 SUFFERERS

By 14 February 2020, no affected person wished to be admitted to the intensive care unit (ICU) and administered mechanical air flow in these investigated COVID-19 and non-COVID-19 patients. Except for two COVID-19 sufferers who had a transient lowering pulse oxygen saturation (SpO2) (92–93%) on admission, SpO2 of the different sufferers remained at 95–99%. All of the COVID-19 sufferers had been dealt with the antiviral drug lopinavir and ritonavir drugs and symptomatic supports, whilst non-COVID-19

sufferers have been dealt with antibiotics (moxifloxacin) and different symptomatic helps which include ample sleep, vitamin and oxygen remedy if necessary. Besides drug treatments, a good deal of the therapy comprised psychological counseling for these COVID-19 sufferers due to the fact of the panic and nervousness about the illness.

CHAPTER-3

OBJECTIVES OF THE STUDY

3.1. OBJECTIVES

- To understand the relationship between pneumonia and covid-19 pneumonia
- To know the impact of pneumonia and covid-19 pneumonia in our social and physical life
- To understand the awareness among the people
- To observe maintain the hygiene among the people during covid-19 pneumonia and pneumonia
- To understand the knowledge about pneumonia and covid-19 pneumonia medicine and their side effect among the people

3.2. RESEARCH QUESTION

- Gender
- Age
- Occupation
- Are you or any members of your family suffering from Pneumonia/ Covid-19 Pneumonia?
- If yes, what kind of pneumonia/ Covid-19 Pneumonia did you/ they suffer?
- How did you/ your family know that you/ they have Pneumonia/ Covid-19 Pneumonia?
- What were the symptoms of Pneumonia/ Covid-19 Pneumonia?
- Have any relationship between Pneumonia and Covid-19 Pneumonia, what do you/ they think?
- If yes, what is the relationship between Pneumonia and Covid-19 Pneumonia?
- Did you/ they take any medicine for Pneumonia/ Covid-19 Pneumonia?
- If yes, mention the name of medicine that you/ they are taken?
- Did you/ your family members feel any side effects after taking medicine?
- If yes, what kind of side effect did you/ they feel?
- Do you/ they believe non-pharmacological treatment (life style modification) can fully control pneumonia/ Covid-19 Pneumonia?
- What kind of lifestyle you/ your family members are maintained for controlling Pneumonia/ Covid-19 Pneumonia do you think?

CHAPTER-4

METHODOLOGY

4. METHODOLOGY

4.1. Introduction

This chapter discusses the methodology used in this study. It describes the research setting, the study design, the study population, the study sample, the research instrument, the procedure, and the data analysis. The chapter ends with the ethical consideration.

4.2. Thesis Design:

This thesis has been designed after a through literature review and then a structured questionnaire was prepared and circulated among the respondents to collect data

4.3. Study Population:

After collection of the response 112 data was finalized for the analysis

4.4. Method of Data Analysis

After collection of data, all interviewed questionnaires were checked for accuracy and inward consistency to prohibit absent or conflicting information and those were disposed of. Data analysis was done through Microsoft excel updated version.

4.5. Ethical Considerations

Verbal informed consent was taken from the study participants before starting data collection. The anonymity of the respondents was kept confidential and study subjects were informed that they can be able to leave the program at any stage of data collection. The study was approved by Department of Pharmacy, Faculty of Allied Health Sciences, Daffodil International University, Dhaka, Bangladesh.

CHAPTER- 5

RESULT & DISCUSSION

5. RESULT & DISCUSSION

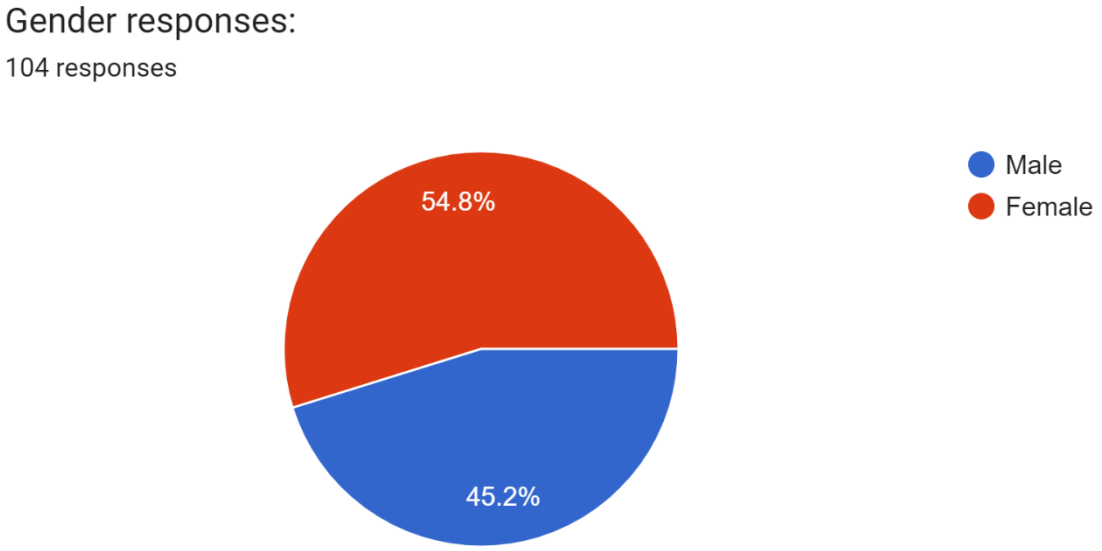

Fig 3: Gender responses

In this survey: Among the total number of respondents 54.8% of people are female, while 45.2% of people are male.

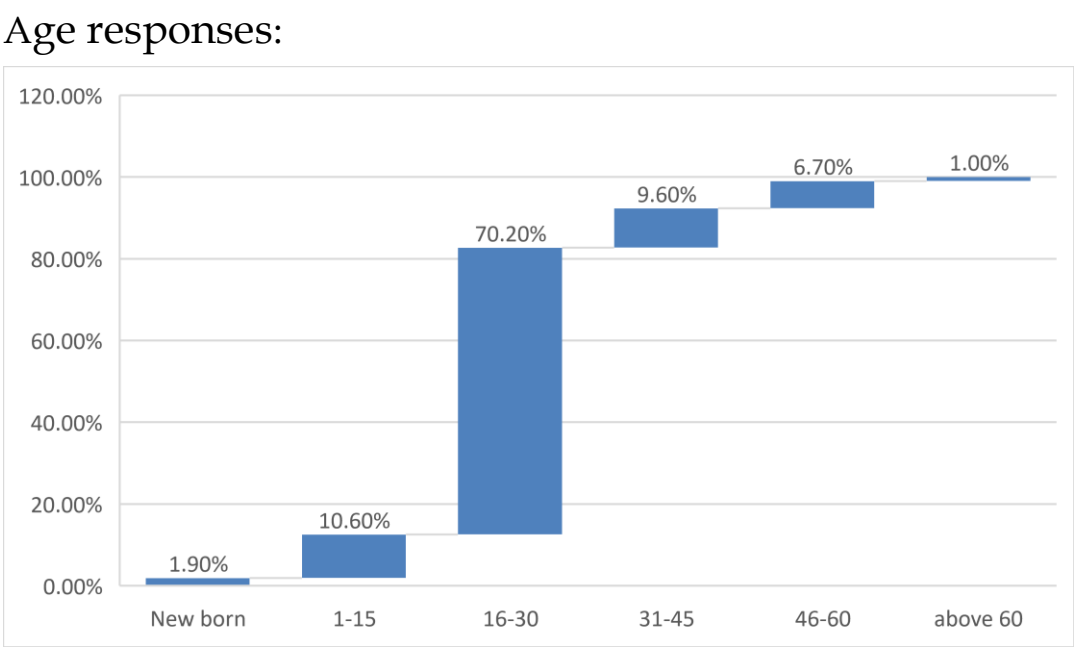

Fig 4: Age responses

Most of respondents of the survey are from 16 to 30 years old and that proportion is 72.2% of the total number. There is 9.6% of responses from 31-45 age group, 6.7% of response are from 46-60 age group. Though 1.9% and 1% are new born and above 60 years.

Occupation responses:

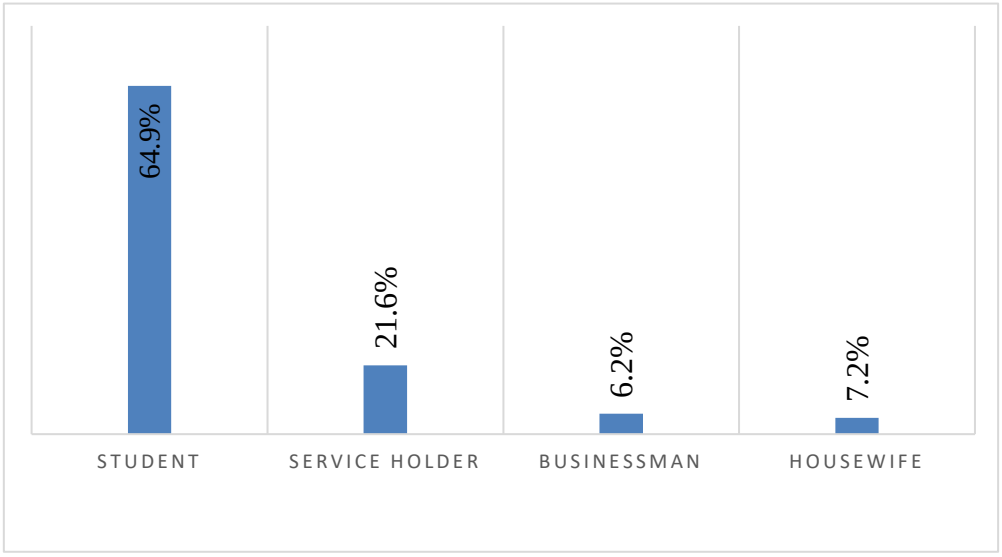

Fig 5: Occupation respondents

In this occupation criteria people responses on four groups. A significant percentage of people are students which is 64.9% of the total. The second highest participants are service holder 21.6%. Businessmen and housewife, response to these criteria were 6.2% and 7.2% respectively.

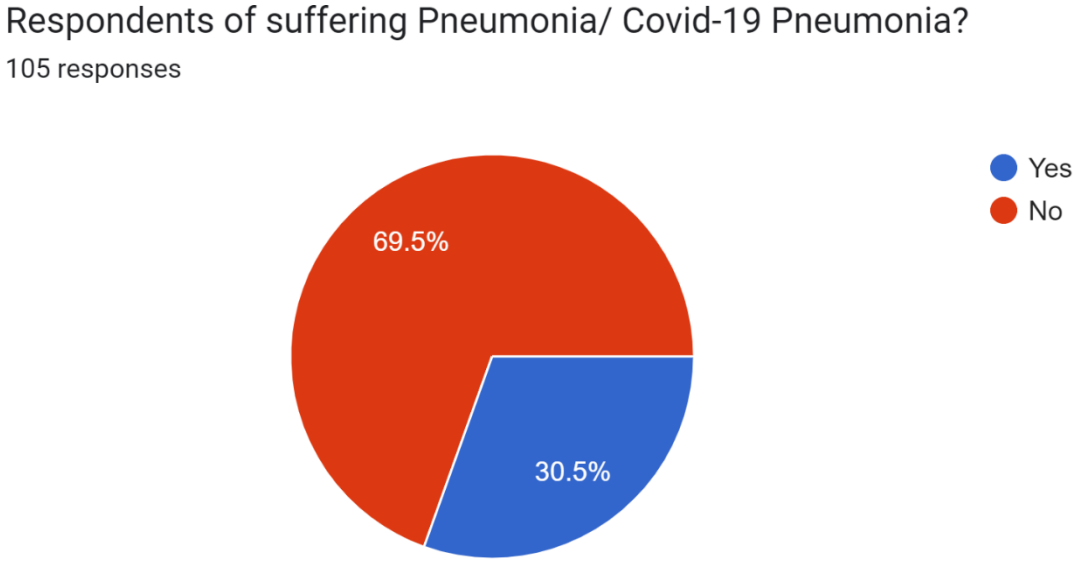

Fig 6: Respondents of suffering in Pneumonia/ Covid-19 Pneumonia

From 105 responses about whether they have suffered in Pneumonia/ Covid-19 Pneumonia problem or not. 30.5% of people responses positively. But 69.5% them never faced any problem relatives to Pneumonia/ Covid-19 Pneumonia.

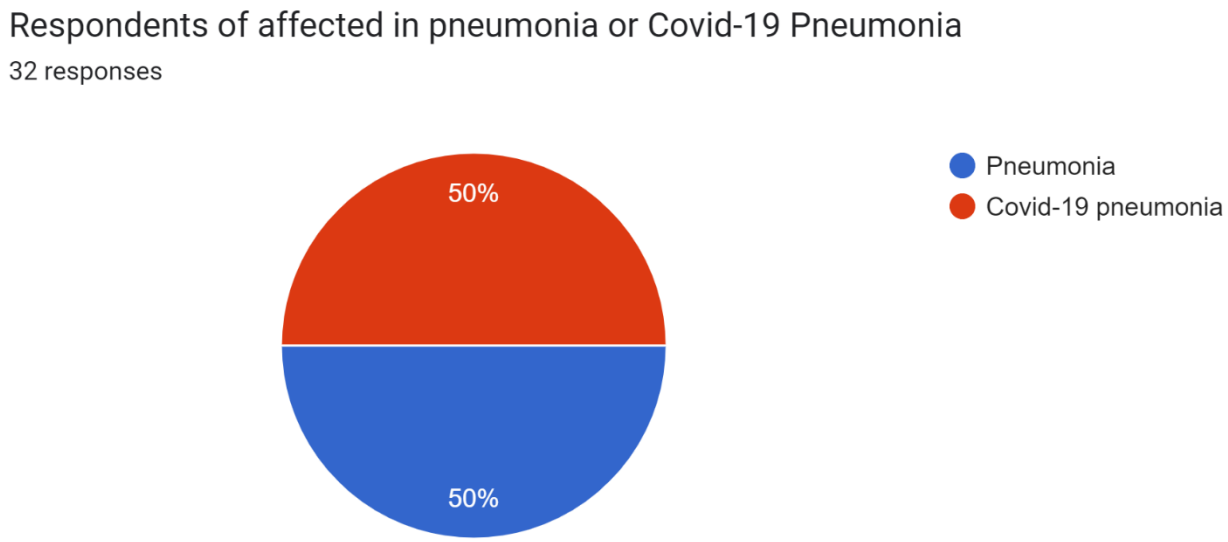

Fig 7: Respondents of affected in Pneumonia or Covid-19 Pneumonia

From total 105 responses, 32 responses are taken among affected about whether 50% respondent are affected Pneumonia and 50% respondent are affected Covid-19

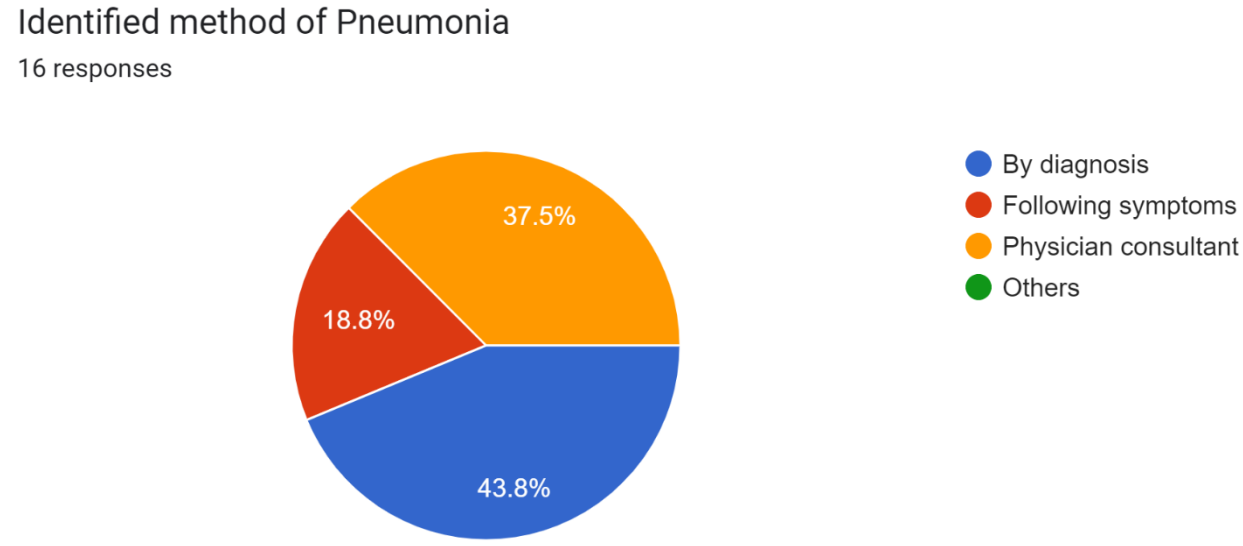

Fig 8: Identified method of Pneumonia

In this part of thesis people were asked how did they/their family know that they/ their family have pneumonia. There are 16 responses. Majority of the people 43.8% are knowing by diagnosis another is 37.5%are knowing physician consultant and 18.8%are knowing following symptoms.

Respondents of the symptoms in Pneumonia

Table 3
Symptoms of pneumonia
Chest pain
Fever
Fatigue and muscle pain
Productive cough
Sore throat
gray or bluish skin color

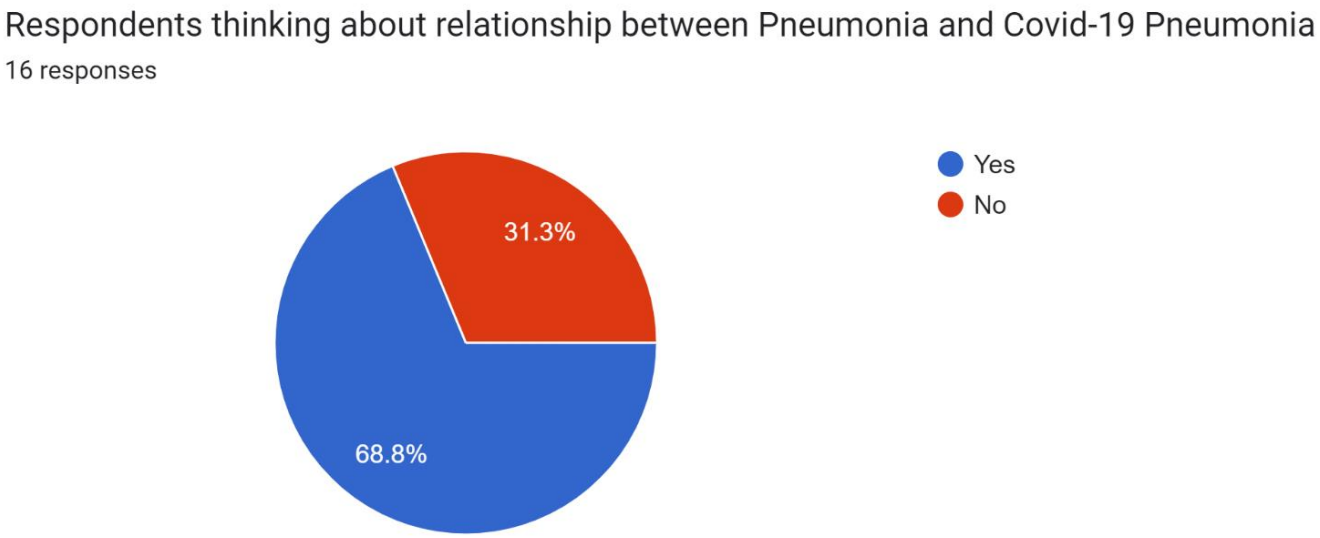

Fig 9: Respondents thinking about relationship between pneumonia & covid-19 pneumonia responses from pneumonia patient

There are about 16 responses whom are affected in pneumonia that whether the maximum people are thinking about relationship that is 68.8% people and 31.3% people are not thinking.

The relationship between Pneumonia and Covid-19 Pneumonia responses from Pneumonia patient

Table 4

Relationship between Pneumonia and Covid-19 Pneumonia
Lung's infection
Respiratory illness
Fever
Cold
Cough

In table 4, some symptoms are similarly shown in Pneumonia and Covid-19 Pneumonia Patients.

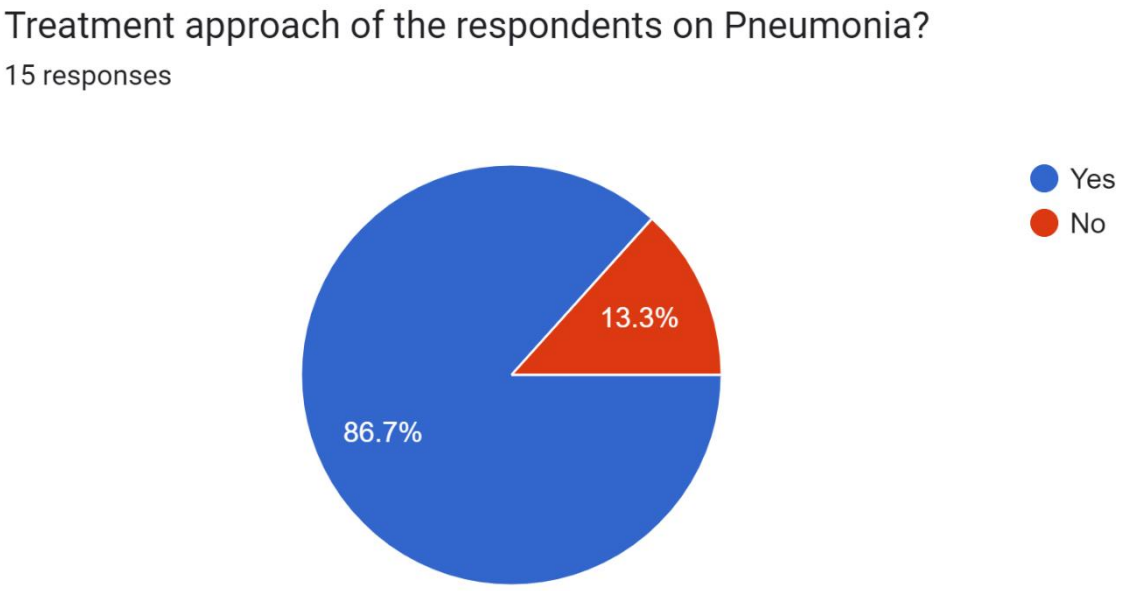

Fig 10: Treatment approach of the respondents on pneumonia

From 15 responses of affected people, who have ever experienced pneumonia in their life, 86.8% of them have taken any kind of medicine that is maximum but 13.3% of them have not.

Drug name for use of pneumonia disease

Table 5

Drug use in pneumonia
Normal saline
Paracetamol
Montelukast
Inj. Ceftriaxone
Inj. ampicillin
Inj. Gentamicin
Flucloxacillin
Nosomist nasal drop

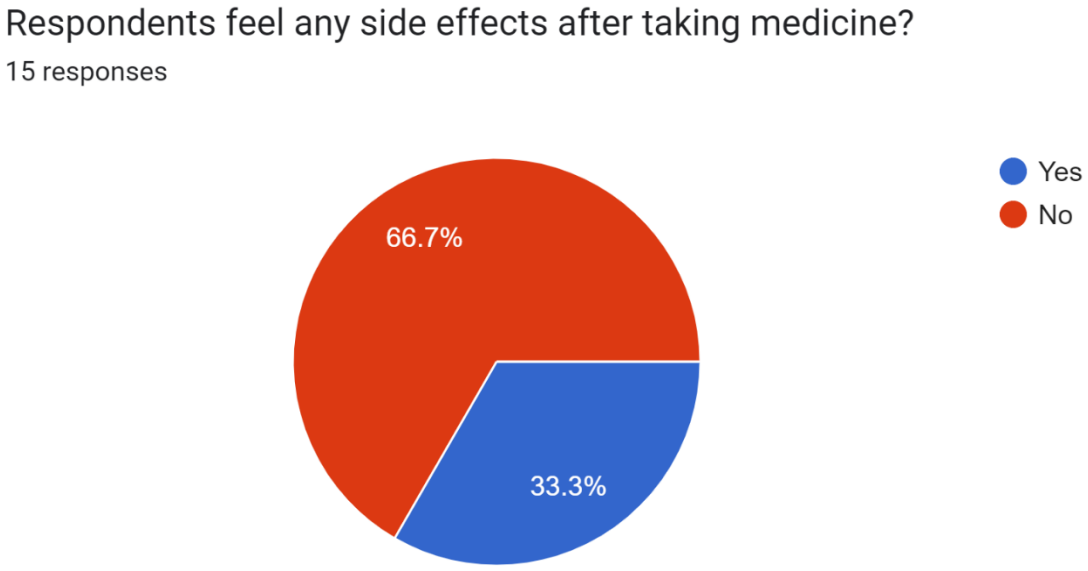

Fig 11: Respondents feel any side effects after taking medicine

From 15 responses of affected people, who have ever experienced side effect after taking medicine in their life, 33.3% of them have positively feel side effect but 66.7% of them have not feel.

Side effect of pneumonia patient after taking medicine

Table 6
Side effect
Rash
Irritation
Dizziness
Nausea
Vomiting
Diarrhea
Sickness

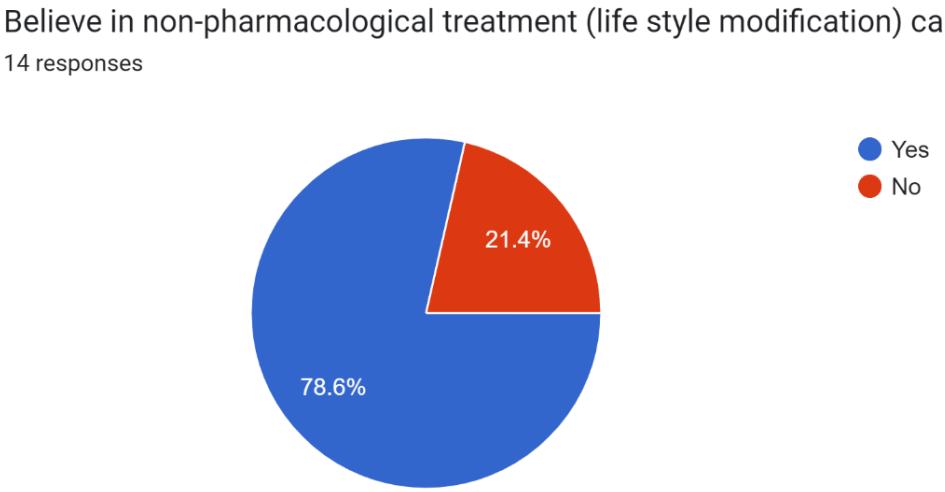

Fig 12: Respondents believe in non-pharmacological treatment

From 14 responses pf affected people, who have ever believed in non-pharmacological treatment is 79.6% but 21.4% of them have not believe in non-pharmacological treatment.

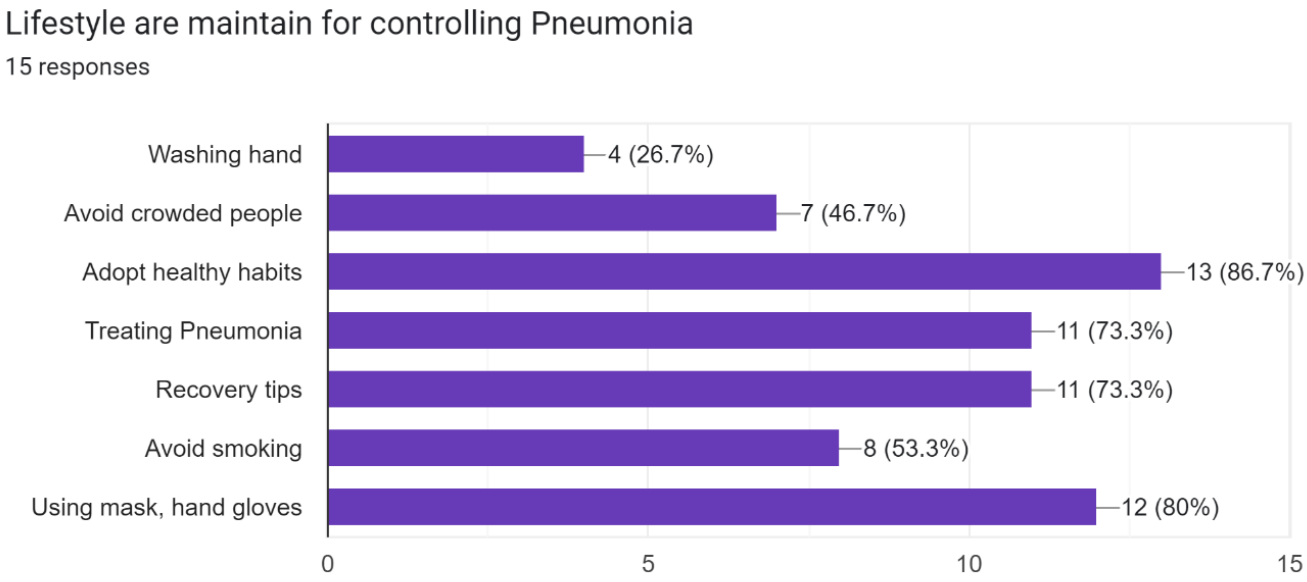

Fig 13: Lifestyle are maintained for controlling covid-19 pneumonia

Exactly 15 people responses in this part of thesis whether, we have noticed that maximum number of respondents people (86.7%) are maintaining adopt healthy habit, (80%) number of respondents are using hand gloves, mask, one the other hand (73.3%) of respondents are maintaining recovery tips and threating pneumonia others (53.3%), (46.7%), (26.7%) respondents are maintaining avoid smoking, avoid crowded people and washing hand.

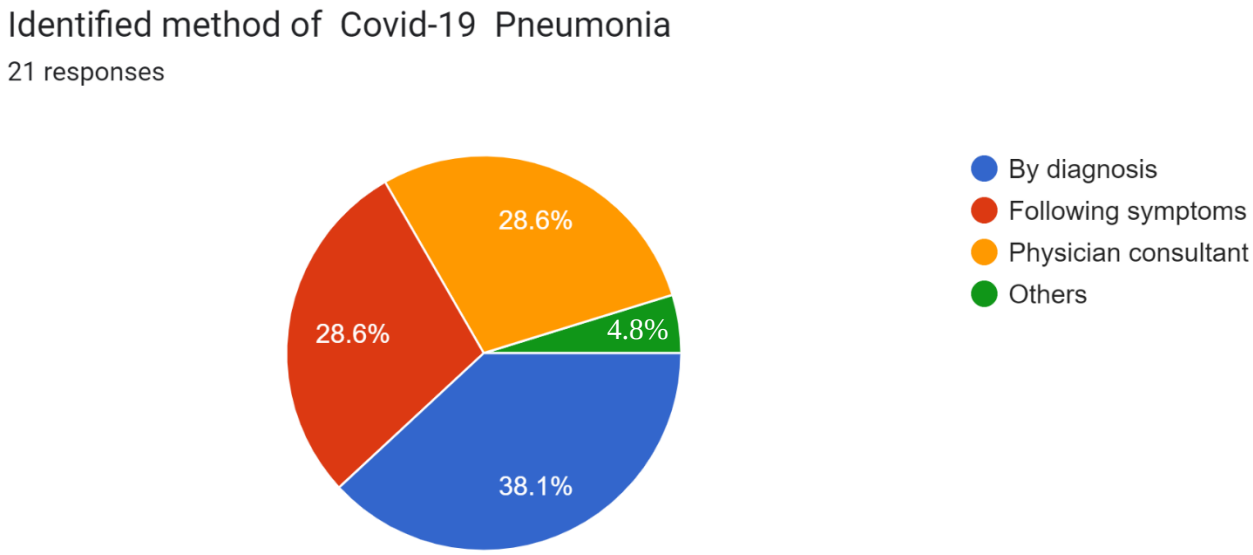

Fig 14: Identified method of covid-19 Pneumonia

In this part of thesis people were asked how did they/their family know that they/ their family have pneumonia. There are 21 responses. Majority of the people 38.1% are knowing by diagnosis another is 28.6% are knowing physician consultant and 28.6% are knowing following symptoms. And last 4.8% are knowing by others.

Respondents of the symptoms in covid-19 Pneumonia

Table 7

Symptoms of covid-19 pneumonia
Shortness of breath
Fever
Cough
Bluish lips, skin or nails (cyanosis)
Chest pain or tightness
gray or bluish skin color

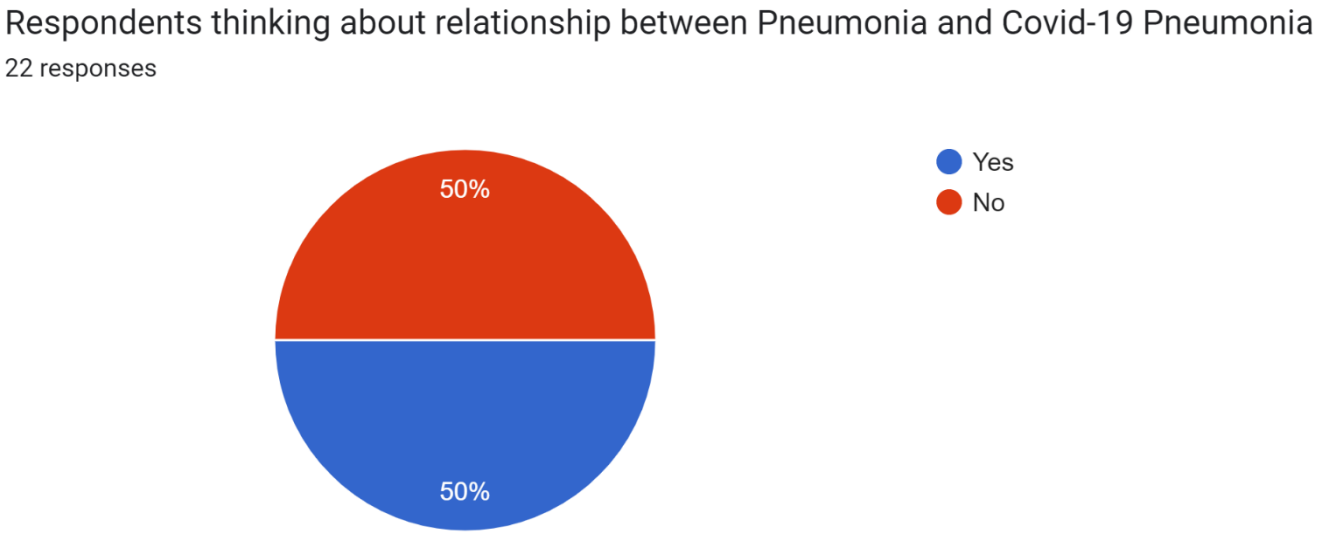

Fig 15: Respondents thinking about relationship between pneumonia & covid-19 pneumonia responses from covid-19 pneumonia patient

There are about 22 responses whom are affected in pneumonia that whether the maximum people are thinking about relationship that is 50% people and 50% people are not thinking.

The relationship between Pneumonia and Covid-19 Pneumonia responses from Covid-19 Pneumonia patient

Table 8

Relationship between Pneumonia and Covid-19 Pneumonia
Lung's infection
Respiratory illness
Fever
Cold
Cough

In table 4, some symptoms are similarly shown in Pneumonia and Covid-19 Pneumonia Patients.

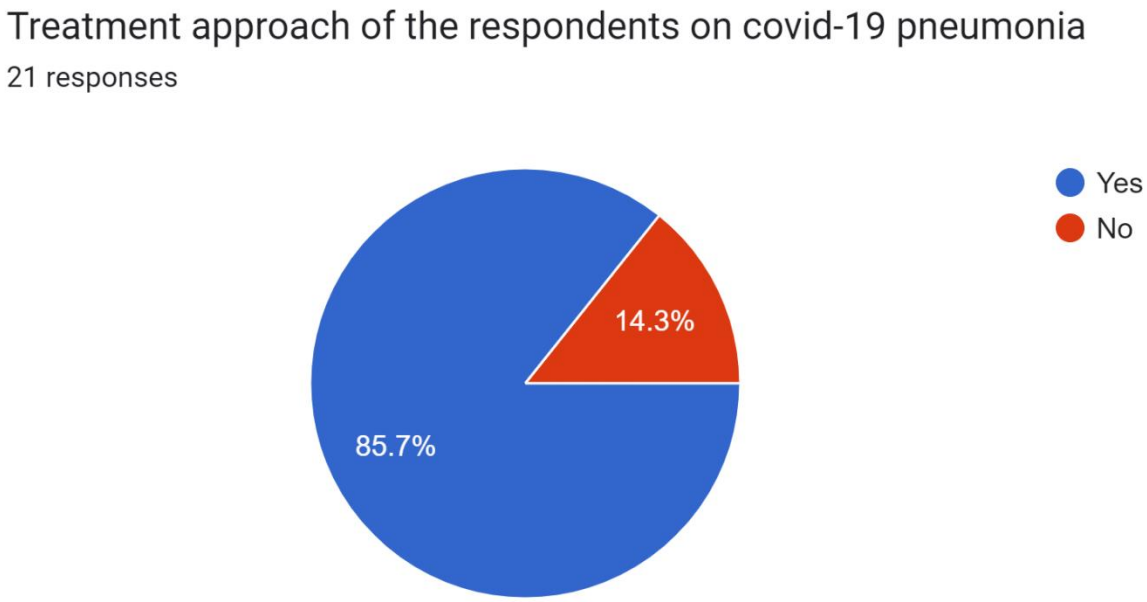

Fig 16: Treatment approach of the respondents on covid-19 pneumonia

From 21 responses of affected people, who have ever experienced pneumonia in their life, 85.7% of them have taken any kind of medicine that is maximum but 14.3% of them have not.

Drug name for use of covid-19 pneumonia disease

Table 9

Drug use in covid-19 pneumonia
Azithromycin
Paracetamol
Montelukast
Ceevit
Vitamin B &D
Fexofenadin
Guaifenesin + Levomenthol + Diphenhydramine

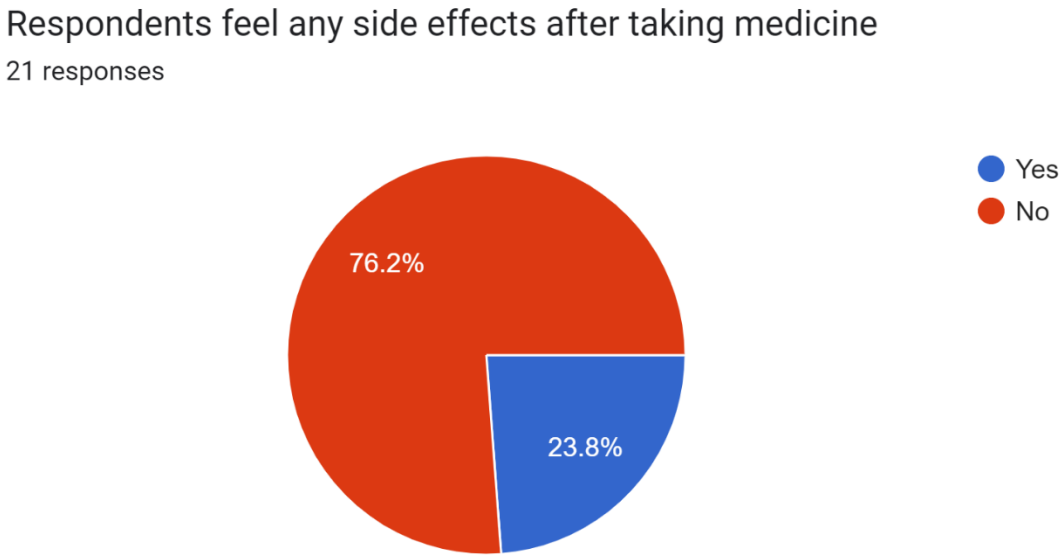

Fig 17: Respondents feel any side effects after taking medicine

From 21 responses of affected people, who have ever experienced side effect after taking medicine in their life, 23.8% of them have positively feel side effect but 76.2% of them have not feel.

Side effect of covid-19 pneumonia after taking medicine

Table 10

Side effect
Rash
Irritation
Dizziness
Nausea
Vomiting
Diarrhea
Sickness

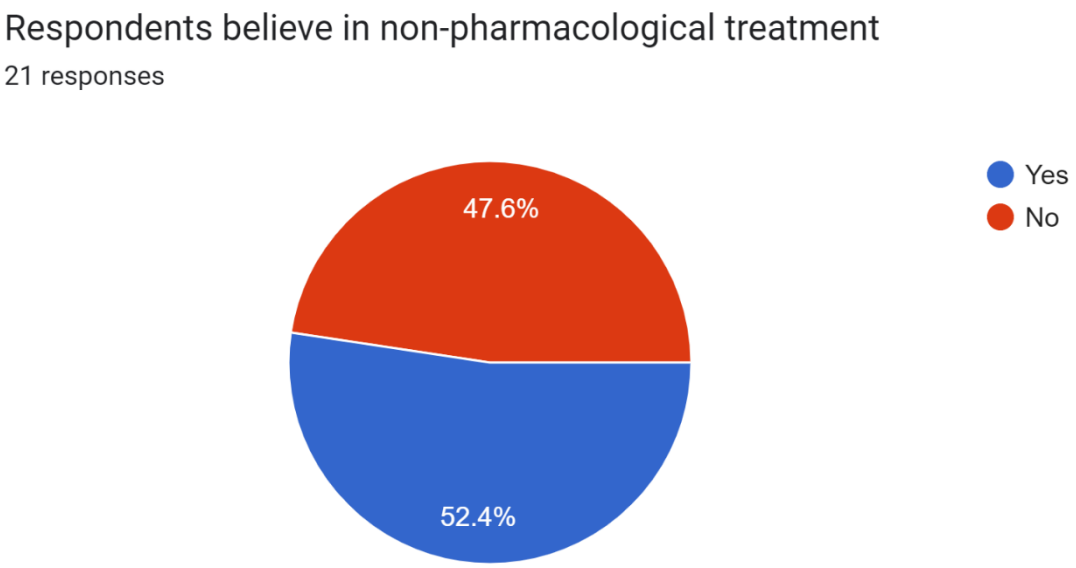

Fig 18: Respondents believe in non-pharmacological treatment

From 21 responses pf affected people, who have ever believed in non-pharmacological treatment is 52.4% but 47.6% of them have not believe in non-pharmacological treatment.

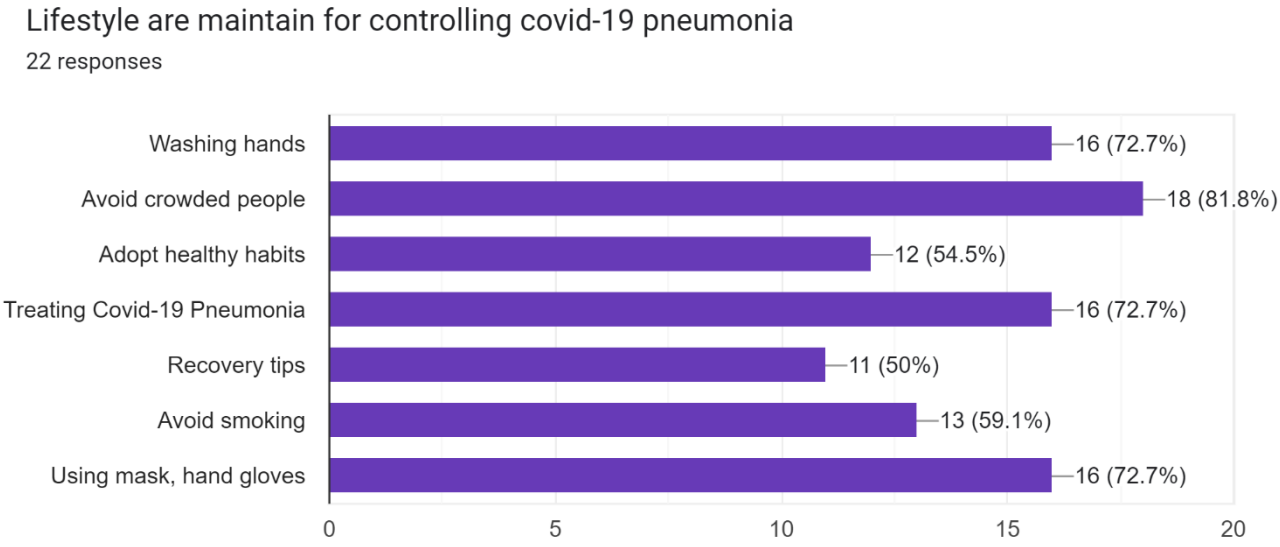

Fig 19: Lifestyle are maintained for controlling covid-19 pneumonia

Exactly 22 people responses in this part of thesis whether, we have noticed that respondent's people (54.5%) are maintaining adopt healthy habit, (72.7%) number of respondents are using hand gloves, mask, one the other hand (50%) of respondents are maintaining recovery tips and threating covid-19 pneumonia is (72.7%). And (59.1%), (81.8%) respondents are maintaining avoid smoking, avoid crowded people and washing hand respectively.

CHAPTER-6

CONCLUSION

6. CONCLUSION

The signs and symptoms of COVID-19 pneumonia are comparable to different sorts of viral pneumonia; this makes it tough to inform the distinction between the two until we get examined for COVID-19 and different respiratory infections. All pneumonias purpose irritation and fluid in our lungs. But lookup suggests that the SARS-CoV-2 virus that reasons COVID pneumonia strikes in another way through our lungs than different viruses and microorganism that reason pneumonia. COVID pneumonia spreads throughout your lungs slowly, the use of our very own immune machine to spread, which capacity it tends to final longer and reason injury in extra places. Other pneumonia's purpose acute sickness signs and symptoms come on all at as soon as however don't closing as long. Pneumonia due to Covid-19 impacts each lung whilst pneumonia due to different respiratory prerequisites tends to have an effect on solely one. Lungs have an attribute "ground glass" look by way of CT scan in pneumonia linked to Covid-19. Abnormalities are considered in a liver characteristic for pneumonia related with Covid-19. Vaccination towards Pneumonia has already been developed and permitted whereas there are no vaccines but developed towards Covid-19.

CHAPTER 7

REFERENCES

1. British Thoracic Society of Standards of Care Committee. BTS guidelines for the management of community acquired pneumonia in childhood. Thorax 2002; 57–24.
2. McIntosh K. Community-acquired pneumonia in children. N Engl J Med 2002; 346: 429–37.
3. File TM. Community-acquired pneumonia. Lancet 2003; 362: 1991–2001.
4. Durrington HJ, Summers C. Recent changes in the management of community-acquired pneumonia in adults. BMJ 2008; 336: 1429–33.
5. Lim WS, Baudoin SV, George RC, et al. British Thoracic Society guidelines for the management of community acquired pneumonia in adults: update 2009. Thorax 2009; 64 (suppl III): iii1–55.
6. National Institutes of Health. NIH covid-19 treatment guidelines. Clinical spectrum of SARS-CoV-2 Infection. 2020.
7. Zhu N, Zhang D, Wang W, et al. A novel coronavirus from patients with pneumonia in China, 2019. N Engl J Med 2020. doi:10.1056/NEJMoa2001017
8. Huang C, Wang Y, Li X, et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. Lancet 2020. doi:10.1016/S0140-6736(20)30183-5
9. Cui J, Li F, Shi ZL. Origin and evolution of pathogenic coronaviruses. Nat Rev Microbiol 2019; 17:181–92.
10. Drosten C, Günther S, Preiser W, et al. Identification of a novel coronavirus in patients with severe acute respiratory syndrome. N Engl J Med 2003; 348:1967–76.
11. Ksiazek TG, Erdman D, Goldsmith CS, et al; SARS Working Group. A novel coronavirus associated with severe acute respiratory syndrome. N Engl J Med 2003; 348:1953–66.
12. Zaki AM, van Boheemen S, Bestebroer TM, Osterhaus AD, Fouchier RA. Isolation of a novel coronavirus from a man with pneumonia in Saudi Arabia. N Engl J Med 2012; 367:1814–20.
13. Chen N, Zhou M, Dong X, et al. Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a descriptive study. Lancet 2020. doi:10.1016/S0140-6736(20)30211-7
14. Phelan AL, Katz R, Gostin LO. The novel coronavirus originating in Wuhan, China: challenges for global health governance. JAMA 2020. doi:10.1001/ jama.2020.1097
15. Li Q, Guan X, Wu P, et al. Early transmission dynamics in Wuhan, China, of novel Coronavirus-infected pneumonia. N Engl J Med 2020. doi:10.1056/ NEJMoa2001316

16. González DA, Victora CG, Gonçalves H. The effects of season at time of birth on asthma and pneumonia in childhood and adulthood in a birth cohort in southern Brazil. Cad Saude Publica 2008; 24:1089–102.
17. Viasus D, Marinescu C, Villoslada A, et al; Influenza A (H1N1) Study Group of the Spanish Network for Research in Infectious Diseases (REIPI). Community acquired pneumonia during the first post-pandemic influenza season: a prospective, multicentre cohort study. J Infect 2013; 67:185–93.
18. in adults: update 2009. Thorax 2009; 64 (suppl III): iii1–55. 6 Rudan I, Boschi-Pinto C, Biloglav Z, Mulholland K, Campbell H. Epidemiology and etiology of childhood pneumonia. Bull World Health Organ 2008; 86: 408–16.
19. WHO. Revised global burden of disease 2002 estimates. 2004. http://www.who.int/healthinfo/global_burden_disease/estimates_ regional_2002_revised/en/ (accessed Nov 5, 2010).
20. Jokinen C, Heiskanen L, Juvonen H, et al. Incidence of community acquired pneumonia in the population of four municipalities in eastern Finland. Am J Epidemiol 1993; 137: 977–88.
21. Black RE, Cousens S, Johnson HL, et al, for the Child Health Epidemiology Reference Group of WHO and UNICEF. Global, regional, and national causes of child mortality in 2008: a systematic analysis. Lancet 2010; 375: 1969–87.
22. de Wals P, Robin E, Fortin E, Thibeault R, Ouakki M, Douville-Fradet M. Pneumonia after implementation of the pneumococcal conjugate vaccine program in the province of Quebec, Canada. Pediatr Infect Dis J 2008; 27: 963–68.
23. Simpson JCG, Macfarlane JT, Watson J, Woodhead MA. A national confidential enquiry into community acquired pneumonia deaths in young adults in England and Wales. Thorax 2000; 55: 1040–45
24. File TM, Marrie TJ. Burden of community acquired pneumonia in North American adults. Postgrad Med 2010; 122: 130–41.
25. Dong E, Du H, Gardner L. An interactive web-based dashboard to track COVID-19 in real time. Lancet Infect Dis 2020;20:533-4.
26. Grasselli G, Zangrillo A, Zanella A, et al, COVID-19 Lombardy ICU Network. Baseline characteristics and outcomes of 1591 patients infected with SARS-CoV-2 admitted to ICUs of the Lombardy region, Italy. JAMA 2020;323:1574-81.

27. Richardson S, Hirsch JS, Narasimhan M, et al, the Northwell COVID-19 Research Consortium. Presenting characteristics, comorbidities, and outcomes among 5700 patients hospitalized with covid-19 in the New York City Area. JAMA 2020;323:2052-9.
28. Docherty AB, Harrison EM, Green CA, et al, ISARIC4C investigators. Features of 20133 UK patients in hospital with covid-19 using the ISARIC WHO Clinical Characterisation Protocol: prospective observational cohort study. BMJ 2020;369:m1985.
29. Garg S, Kim L, Whitaker M, et al. Hospitalization rates and characteristics of patients hospitalized with laboratory-confirmed coronavirus disease 2019—covid-NET, 14 States, March 1-30, 2020. MMWR Morb Mortal Wkly Rep 2020;69:458-64.
30. Torres Acosta MA, Singer BD. Pathogenesis of COVID-19- induced ARDS: implications for an ageing population. Eur Respir J 2020;56:2002049.
31. Yang Y, Zhao Y, Zhang F, Zhang L, Li L. covid-19 in elderly adults: clinical features, molecular mechanisms, and proposed strategies. Aging Dis 2020;11:1481-95.
32. Ellinghaus D, Degenhardt F, Bujanda L, et al, Severe Covid-19 GWAS Group. Genomewide association study of severe covid-19 with Respiratory Failure. N Engl J Med 2020;383:1522-34.
33. Yang X, Yu Y, Xu J, et al. Clinical course and outcomes of critically ill patients with SARS-CoV-2 pneumonia in Wuhan, China: a single-centered, retrospective, observational study. Lancet Respir Med 2020;8:475-81.
34. Lauer SA, Grantz KH, Bi Q, et al. The incubation period of coronavirus disease 2019 (covid-19) from publicly reported confirmed cases: estimation and application. Ann Intern Med 2020;172:577-82.
35. Hu B, Guo H, Zhou P, Shi Z-L. Characteristics of SARS-CoV-2 and COVID-19. Nat Rev Microbiol 2020;6:1-14.
36. Tzotzos SJ, Fischer B, Fischer H, Zeitlinger M. Incidence of ARDS and outcomes in hospitalized patients with COVID-19: a global literature survey. Crit Care 2020;24:516.
37. Petrilli CM, Jones SA, Yang J, et al. Factors associated with hospital admission and critical illness among 5279 people with coronavirus disease 2019 in New York City: prospective cohort study. BMJ 2020;369:m1966.
38. Horwitz LI, Jones SA, Cerfolio RJ, et al. Trends in covid-19 risk adjusted mortality rates. J Hosp Med 2021;16:90-2.
39. Dennis JM, McGovern AP, Vollmer SJ, Mateen BA. Improving survival of critical care patients with coronavirus disease 2019 in England: a national cohort study, March to June 2020. Crit Care Med 2021;49:209-14.

40. Lan J, Ge J, Yu J, et al. Structure of the SARS-CoV-2 spike receptor binding domain bound to the ACE2 receptor. Nature 2020;581:215- 20.
41. Meyerowitz EA, Richterman A, Gandhi RT, Sax PE. Transmission of SARS-CoV-2: a review of viral, host, and environmental factors. Ann Intern Med 2020.
42. da Costa VG, Moreli ML, Saivish MV. The emergence of SARS, MERS and novel SARS-2 coronaviruses in the 21st century. Arch Virol 2020;165:1517-26.
43. Shang J, Ye G, Shi K, et al. Structural basis of receptor recognition by SARS-CoV-2. Nature 2020;581:221-4.
44. Zhang L, Lin D, Sun X, et al. Crystal structure of SARS-CoV-2 main protease provides a basis for design of improved α-ketoamide inhibitors. Science 2020;368:409-12.
45. Walls AC, Park Y-J, Tortorici MA, Wall A, McGuire AT, Veesler D. Structure, function, and antigenicity of the SARS-CoV-2 spikeglycoprotein. Cell 2020;181:281-292.e6.
46. Siu YL, Teoh KT, Lo J, et al. The M, E, and N structural proteins of the severe acute respiratory syndrome coronavirus are required for efficient assembly, trafficking, and release of virus-like particles. J Virol 2008;82:11318-30.
47. Satarker S, Nampoothiri M. Structural proteins in severe acute respiratory syndrome coronavirus-2. Arch Med Res 2020;51:482-91.
48. Huang Y, Yang C, Xu XF, Xu W, Liu SW. Structural and functional properties of SARS-CoV-2 spike protein: potential antivirus drug development for COVID-19. Acta Pharmacol Sin 2020;41:1141-9.
49. Duan L, Zheng Q, Zhang H, Niu Y, Lou Y, Wang H. The SARSCoV-2 spike glycoprotein biosynthesis, structure, function, and antigenicity: implications for the design of spike-based vaccine immunogens. Front Immunol 2020;11:576622.
50. Hu Y, Wen J, Tang L, et al. The M protein of SARS-CoV: basic structural and immunological properties. Genomics Proteomics Bioinformatics 2003;1:118-30.
51. Singh Tomar PP, Arkin IT. SARS-CoV-2 E protein is a potential ion channel that can be inhibited by Gliclazide and Memantine. Biochem Biophys Res Commun 2020;530:10-4.
52. Sarkar M, Saha S. Structural insight into the role of novel SARS-CoV-2 E protein: A potential target for vaccine development and other therapeutic strategies. PLoS One 2020;15:e0237300.
53. Dutta NK, Mazumdar K, Gordy JT. The nucleocapsid protein of SARS CoV-2: a target for vaccine development. J Virol 2020;94:e00647- 20.

54. Lopez-Rincon A, Perez-Romero CA, Tonda A, et al. Design of specific primer sets for the detection of B.1.1.7, B.1.351 and P.1 SARS-CoV-2 variants using deep learning. bioRxiv [Preprint] 2021.
55. Vrancken B, Dellicour S, Smith DM, Chaillon A. Phylogenetic analyses of SARS-CoV-2 B.1.1.7 lineage suggest a single origin followed by multiple exportation events versus convergent evolution. bioRxiv [Preprint] 2021.
56. Centers for Disease Control and Prevention. Emerging SARS-CoV-2 variants. 2021. https://www.cdc.gov/coronavirus/2019-ncov/ more/science-and-research/scientific-brief-emerging-variants. html
57. Starr TN, Greaney AJ, Addetia A, et al. Prospective mapping of viral mutations that escape antibodies used to treat COVID-19. Science 2021:eabf9302.
58. Wang Q, Zhang Y, Wu L, et al. Structural and functional basis of SARS CoV-2 entry by using human ACE2. Cell 2020;181:894-904.e9.
59. Samavati L, Uhal BD. ACE2, much more than just a receptor for SARS-COV-2. Front Cell Infect Microbiol 2020;10:317.
60. Yuki K, Fujiogi M, Koutsogiannaki S. COVID-19 pathophysiology: A review. Clin Immunol 2020;215:108427.
61. Swärd P, Edsfeldt A, Reepalu A, Jehpsson L, Rosengren BE, Karlsson MK. Age and sex differences in soluble ACE2 may give insights for COVID-19. Crit Care 2020;24:221.
62. Hoffmann M, Kleine-Weber H, Schroeder S, et al. SARS-CoV-2 cell entry depends on ACE2 and TMPRSS2 and is blocked by a clinically proven protease inhibitor. Cell 2020;181:271-280.e8.
63. Ma D, Chen C-B, Jhanji V, et al. Expression of SARS-CoV-2 receptor ACE2 and TMPRSS2 in human primary conjunctival and pterygium cell lines and in mouse cornea. Eye (Lond) 2020;34:1212-9.
64. Bao R, Hernandez K, Huang L, Luke JJ. ACE2 and TMPRSS2 expression by clinical, HLA, immune, and microbial correlates across 34 human cancers and matched normal tissues: implications for SARS-CoV-2 COVID-19. J Immunother Cancer 2020;8:e001020.
65. Bergmann CC, Silverman RH. COVID-19: Coronavirus replication, pathogenesis, and therapeutic strategies. Cleve Clin J Med 2020;87:321-7.
66. Blanco-Melo D, Nilsson-Payant BE, Liu W-C, et al. Imbalanced host response to SARS-CoV-2 drives development of covid-19. Cell 2020;181:1036-1045.e9.
67. Guo L, Ren L, Yang S, et al. Profiling early humoral response to diagnose novel coronavirus disease (covid-19). Clin Infect Dis 2020;71:778-85.

68. Woodruff MC, Ramonell RP, Nguyen DC, et al. Extrafollicular B cell responses correlate with neutralizing antibodies and morbidity in COVID-19. Nat Immunol 2020;21:1506-16.
69. Long Q-X, Tang X-J, Shi Q-L, et al. Clinical and immunological assessment of asymptomatic SARS-CoV-2 infections. Nat Med 2020;26:1200-4.
70. Ren L, Fan G, Wu W, et al. Antibody responses and clinical outcomes in adults hospitalized with severe coronavirus disease 2019 (covid-19): a post hoc analysis of LOTUS China trial. Clin Infect Dis 2020.
71. Tan L, Wang Q, Zhang D, et al. Lymphopenia predicts disease severity of COVID-19: a descriptive and predictive study. Signal Transduct Target Ther 2020;5:33.
72. Rydyznski Moderbacher C, Ramirez SI, Dan JM, et al. Antigen-specific adaptive immunity to SARS-CoV-2 in acute covid-19 and associations with age and disease severity. Cell 2020;183:996-1012.e19.
73. Goronzy JJ, Weyand CM. Mechanisms underlying T cell ageing. Nat Rev Immunol 2019;19:573-83.
74. Wilk AJ, Rustagi A, Zhao NQ, et al. A single-cell atlas of the peripheral immune response in patients with severe COVID-19. Nat Med 2020;26:1070-6.
75. Del Valle DM, Kim-Schulze S, Huang H-H, et al. An inflammatory cytokine signature predicts COVID-19 severity and survival. Nat Med 2020;26:1636-43.
76. Leisman DE, Ronner L, Pinotti R, et al. Cytokine elevation in severe and critical COVID-19: a rapid systematic review, meta-analysis, and comparison with other inflammatory syndromes. Lancet Respir Med 2020;8:1233-44.
77. RECOVERY Collaborative Group. Dexamethasone in hospitalized patients with Covid-19—preliminary report. N Engl J Med 2020;
78. JG, Simpson LJ, Ferreira A-M, et al. Cytokine profile in plasma of severe COVID-19 does not differ from ARDS and sepsis. JCI Insight 2020;5:140289.
79. Maiese A, Manetti AC, La Russa R, et al. Autopsy findings in COVID 19-related deaths: a literature review. Forensic Sci Med Pathol 2020.
80. Arrossi AV, Farver C. The pulmonary pathology of COVID-19. Cleve Clin J Med 2020.
81. Malas MB, Naazie IN, Elsayed N, Mathlouthi A, Marmor R, Clary B. Thromboembolism risk of COVID-19 is high and associated with a higher risk of mortality: A systematic review and metaanalysis. EClinical Medicine 2020;29:100639.
82. Chi G, Lee JJ, Jamil A, et al. Venous thromboembolism among hospitalized patients with covid-19 undergoing thromboprophylaxis: a systematic review and meta-analysis. J Clin Med 2020;9:2489.

83. Janssens JP, Pache JC, Nicod LP. Physiological changes in respiratory function associated with ageing. Eur Respir J 1999; 13: 197–205.
84. Tolep K, Kelsen S. Effect of aging on respiratory skeletal muscles. Clin Chest Med 1993; 14: 363–78.
85. Enright PL, Kronmal RA, Manolio TA, Schenker MB, Hyatt RE. Respiratory muscle strength in the elderly: correlates and reference values. Am J Respir Crit Care Med 1994; 149: 430–38.
86. Evans S, Watson L, Hawkins M, Cowley A, Johnston I, Kinnear W. Respiratory muscle strength in chronic heart failure. Thorax 1995; 50: 625–28.
87. Nishimura Y, Maeda H, Tanaka K, Nakamura H, Hashimoto Y, Yokoyama M. Respiratory muscle strength and hemodynamics in chronic heart failure. Chest 1994; 105: 355–59.
88. Tzelepis GE, McCool FD, Friedman JH, Hoppin FG Jr. Respiratory muscle dysfunction in Parkinson's disease. Am Rev Respir Dis 1988; 138: 266–71.
89. Brown LK. Respiratory dysfunction in Parkinson's disease. Clin Chest Med 1994; 15: 715–27.
90. Meyer K, Ershler W, Rosenthal N, Xing-Gu L, Peterson K. Immune dysregulation in the aging human lung. Am J Respir Crit Care Med 1996; 153: 1072–79.
91. Ho JC, Chan KN, Hu WH, et al. The effect of aging on nasal mucociliary clearance, beat frequency, and ultrastructure of respiratory cilia. Am J Respir Crit Care Med 2001; 163: 983–88.
92. Fein AM, Feinsilver SH, Niederman MS. Atypical manifestations of pneumonia in the elderly. Clin Chest Med 1991; 12: 319–36.
93. Kronenberg R, Drage G. Attenuation of the ventilatory and heart rate responses to hypoxia and hypercapnia with aging in normal man. J Clin Invest 1973; 52: 1812–19.
94. Friis B, Andersen P, Brenoe E, et al. Antibiotic treatment of pneumonia and bronchiolitis. Arch Dis Child 1984; 59: 1038–45.
95. Wong SSY, Yuen K-Y. Antiviral therapy for respiratory tract infections. Respirology 2008; 13: 950–71.
96. Shun-Shin M, Thompson M, Heneghan C, Perera R, Harnden A, Mant D. Neuraminidase inhibitors for treatment and prophylaxis of infl uenza in children: systematic review and meta-analysis of randomised controlled trials. BMJ 2009; 339: 3172–80.
97. Yu H, Liao Q, Yuan Y, et al. Eff ectiveness of oseltamivir on disease progression and viral RNA shedding in patients with mild pandemic 2009 infl uenza A H1N1: opportunistic retrospective study of medical charts in China. BMJ 2010; 341: c4779.

98. Harper SA, Bradley JS, Englund JA, et al. Seasonal infl uenza in adults and children: diagnosis, treatment, chemoprophylaxis, and institutional outbreak management—clinical practice guidelines of the Infectious Diseases Society of America. Clin Infect Dis 2009; 48: 1003–32.

99. Birnkrant D, Cox E. The emergency use authorization of peramivir for treatment of 2009 H1N1 infl uenza. N Engl J Med 2009; 361: 2204–07.

100. Härter G, Zimmermann O, Maier L, et al. Intravenous zanamivir for patients with pneumonitis due to pandemic (H1N1) 2009 infl uenza virus. Clin Infect Dis 2010; 50: 1249–51.

101. Yin MT, Brust JCM, van Tieu H, Hammer SM. Antiherpes, anti-hepatitis virus, and anti-respiratory virus agents. In: Richman DD, Whitley RJ, Hayden FG, eds. Clinical virology, 3rd edn. Washington: ASM Press, 2009: 217–64.

102. Hopkins P, McNeil K, Kermeen F, et al. Human metapneumovirus in lung transplant recipients and comparison to respiratory syncytial virus. Am J Respir Crit Care Med 2008; 178: 876–81.

103. 103. Empey KM, Pebbles S, Koll JK. Pharmacologic advances in the treatment and prevention of respiratory syncytial virus. Clin Infect Dis 2010; 50: 1258–67.

104. Doan ML, Mallory GB, Kaplan SL, et al. Treatment of adenovirus pneumonia with cidofovir in pediatric lung transplant recipients. J Heart Lung Transplant 2007; 26: 883-89

105. Bonney D, Razali H, Turner A, Will A. Successful treatment of human metapneumovirus pneumonia using combination therapy with intravenous ribavirin and immune globulin. Br J Haematol 2009; 145: 667–69.

106. Frangites CY, Pneumatikos I. Varicella-zoster virus pneumonia in adults: report of 14 cases and review of the literature. Eur J Intern Med 2004; 15: 364–70.

107. Jartti T, Vanto T, Heikkinen T, Ruuskanen O. Systemic glucocorticoids in childhood expiratory wheezing: relation between age and viral etiology with effi cacy. Pediatr Infect Dis J 2002; 21: 873–78.

108. Stockman LJ, Bellamy R, Garner P. SARS: systematic review of treatment effects. PLoS Med 2006; 3: e343.

109. Falagas ME, Vouloumanou EK, Baskouta E, Rafailidis PI, Polyzos K, Rello J. Treatment options for 2009 H1N1 infl uenza: evaluation of the published evidence. Int J Antimicrob Agents 2010; 35: 421–30.

110. Cheng VCC, Tang BSF, Wu AKL, Chu CM, Yuen KY. Medical treatment of viral pneumonia including SARS in immunocompetent adult. J Infect 2004; 49: 262–73.

111. Heinonen S, Silvennoinen H, Lehtinen P, Vainionpää R, Ziegler T, Heikkinen T. Eff ectiveness of inactivated infl uenza vaccine in children aged 9 months to 3 years: an observational cohort study. Lancet Infect Dis (published online Nov 23, 2010).

112. American Academy of Pediatrics. Policy statement: modifi ed recommendations for use of palivizumab for prevention of respiratory syncytial virus infections. Pediatrics 2009; 124: 1694–701.

113. Madhi SA, Klugman KP, The Vaccine Trialist Group. A role for Streptococcus pneumoniae in virus associated pneumonia. Nat Med 2004; 10: 811–13

APPENDIX

QUESTIONNAIRES

Gender

- Male
- Female

Age

- New born
- 01-15
- 16-30
- 31-45
- 46-60
- Above 60

Occupation

- Student
- Job holder
- House Holder
- Businessman

Are you or any members of your family suffering from Pneumonia/ Covid-19 Pneumonia?

- Yes
- No

If yes, what kind of pneumonia did you/ they suffer?

- Pneumonia
- Covid-19 pneumonia

How did you/ your family know that you/ they have Pneumonia?

- By diagnosis
- Following symptoms
- Physician consultant
- Others

What were the symptoms of Pneumonia?

- Chest pain
- Fever
- Fatigue and muscle pain
- gray or bluish skin color
- Sore throat
- Productive cough

Have any relationship between Pneumonia and Covid-19 Pneumonia, what do you/ they think?

- Yes
- No

If yes, what is the relationship between Pneumonia and Covid-19 Pneumonia?

_____.

Did you/ they take any medicine for Pneumonia?

- Yes
- No

If yes, mention the name of medicine that you/ they are taken?

_____.

Did you/ your family members feel any side effects after taking medicine?

- Yes
- No

If yes, what kind of side effect did you/ they feel?

_____.

Do you/ they believe non-pharmacological treatment (life style modification) can fully control pneumonia?

- Yes
- No

What kind of lifestyle you/ your family members are maintained for controlling Pneumonia do you think?

- Washing hands
- Recovery tips
- Avoid smoking
- Avoid crowded people
- Adopt healthy habits
- Treating Covid-19 Pneumonia
- Using mask, hand gloves

How did you/ your family know that you/ they have Covid-19 Pneumonia?

- By diagnosis
- Following symptoms
- Physician consultant
- Others

What were the symptoms of Covid-19 Pneumonia?

- Shortness of breath
- Cough
- Chest pain or tightness
- Fever
- Extreme fatigue/ tiredness
- Bluish lips, skin or nails (cyanosis)

Have any relationship between Pneumonia and Covid-19 Pneumonia., what do you/ they think?

- Yes
- No

If yes, what is the relationship between Pneumonia and Covid-19 Pneumonia?

_____.

Did you/ they take any medicine for Covid-19 Pneumonia?

- Yes
- No

If yes, mention the name of medicine that you are taken?

_____.

Did you/ your family members feel any side effects after taking medicine?

- Yes
- No

If yes, what kind of side effect did you/ they feel?

_____.

Do you/ they believe non-pharmacological treatment (life style modification) can fully control Covid-19 pneumonia?

- Yes
- No

What kind of lifestyle you/ your family members are maintained for controlling Covid-19 Pneumonia do you think?

- Washing hands
- Recovery tips
- Avoid smoking
- Avoid crowded people
- Adopt healthy habits
- Treating Covid-19 Pneumonia
- Using mask, hand gloves