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Dietary intake to help prevent Covid-19 implications: The devil is in the detail

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Abstract

One of the most frequent antecedents of mortality in world is coronavirus that proceeds multitude metabolic disorders and is consequence of damaged lungs that is ultimately caused by contagious infection and virus called coronavirus. It effects people of every age mostly dominating in child, aged, people who have already poor immune system and lungs related disorders. It has tremendous effects on body affecting physical, emotional, cognitive and intellectual abilities as well. It leads to various clinical moderate to severe complications including acute respiratory distress syndrome (ARDS), pneumonia, coughing up blood, persistent chest pain, fever, SOB (shortness of breath), coughing up sputum, chills, sore throat, respiratory sputum production (phlegm), loss of sense and smell, headache, muscle aches, rashes and diarrhea etc. One of the most imprudent factors examined in these patients is acute respiratory distress syndrome (ARDS) as well which refers to respiratory failure, a mechanism in which body is unable to respond normally sequentially results by high level of widespread inflammation in lungs. Penurious immune system which contributes to higher rate of mortality worldwide present strong association with COVID-19. Metabolic disturbances contribute to the development and progression of respiratory distress, as well as they have serious consequences in next stages of life that can have harmful effects on health. In this review we will discuss the pathophysiology as well as the signs and symptoms of COVID-19, the morbidities related to this disease and the treatment required controlling this disorder.

Keywords: Covid-19, Diet, Health Benefits

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1. INTRODUCTION

COVID-19 is a disorder that executes due to SARS-CoV-2 that can trigger a respiratory tract infection. "Upper respiratory tract (sinuses, nose, and throat) or lower respiratory tract (windpipe and lungs) is affected and poor immune system which is bottom line of this disease". One of the most prevalent metabolic disarray as well as disaster was declared as public health emergency of international concern (PHEIC) by WHO on 30 January 2020. This outbreak was caused by virus known as SARS-CoV-2, one of seven types of corona virus and was

mostly related to bat coronavirus, pangolin COV etc. The scientific concurrence provides evidence that COVID-19 has a natural origin[1].

Table 1. COVID-19 pandemics by country and territory

Location	Cases	Deaths	Recovery
US	2,041,179	114,592	606,819
Brazil	775,581	39,803	380,300
Russia	502,436	6,532	261,150
United Kingdom	290,143	41,128	No data
India	286,579	8,102	141,029
Spain	242,280	27,136	150,376
Germany	186,530	8,845	171,046
Iran	180,156	8,584	142,663
Turkey	173,036	4,746	146,839
France	155,136	29,319	71,832
Canada	148,496	2,475	No data
Mexico	124,301	14,649	91,412
Pakistan	119,536	2,356	38,391
Saudi Arabia	116,021	857	80,019

Cases refer to number of people who have been exposed to testing for COVID-19 and resulted positive for it. Analysis in China was performed and data according to age depicts that there were fewer positive cases of coronavirus in people less than 20 years of age. Number of deaths attributed to COVID-19 was 417,000 till 11 June 2020 in Pakistan and China, 80% cases prevailed in those people aged over 60 and those who already had pre-existing health conditions like CVD[2]. In Wuhan on January 9, 2020, first death case was confirmed regard COVID-19[3,4]. "Corona virus has a spike glycoprotein which mediates viral entry into the host cell. Spike protein binds angiotensin converting enzyme 2 receptor on host cell and mediates fusion of viral membrane and host membrane". Spike protein has two subunits, S1 and S1 and has two structural conformations, pre fusion and post fusion. S1 subunit mediates binding and s2 subunit mediates fusion and viral entry into the host cell which requires proteolytic activation by host cell proteases which includes endosomal cathepsins, cell surface trans membrane protease/serine (TMPRSS) proteases, furin and trypsin. After entering the host cell, virus uncoils in the cytoplasm where replication takes place. RNA genome of virus looks like host RNA as it has methylated cap on 3' end and polyadenylated-A tail on 5' end which allows its attachment to ribosome for translation. Coronavirus encoded protein, replicase allows translation of RNA viral genome to RNA using the host cell machinery and a long polyprotein is formed. A non-structural protein of corona virus, Protease allows separation of proteins in chains[5]. Transcription of corona virus occurs in discontinuous fashion. Infection by coronavirus controls the cell macromolecular synthesis by locating proteins in the nucleus of host cell[6].

2. COMMON SIGNS AND SYMPTOMS OF COVID-19

Majority of people with infection does not develop symptoms. However, the people with underlying problems of heart, lung, kidney or liver disease, diabetes mellitus and immunocompromising condition may develop severe disease[7]. SARS-CoV-2 infection is often asymptomatic but may cause: Mild flu, fever, body aches, Pneumonia, Severe acute respiratory distress syndrome, Gastroenteritis, Encephalitis, Acute haemorrhagic necrotizing encephalopathy, Thrombotic complications like pulmonary embolism and ischemic stroke, Upper respiratory tract infection may present as fever, sore throat and loss of smell and taste sensation because of inflammatory response. While lower respiratory tract infection presents as pneumonia which may leads to shortness of breath and difficulty in breathing[7].

3. PATHOPHYSIOLOGY OF COVID-19

The exact pathophysiology of COVID-19 is very complex. COVID-19 is a type of virus that can be transmitted from one person to another Pneumonitis is a pauci-inflammatory septal capillary injury characterised by

mural and luminal fibrin accumulation in the septal capillaries, as well as neutrophil infiltration of the alveolar septa. C5b-9 (membrane attack complex), Cd4, and mannose binding lectin (MBL)-associated serine protease (MASP)-2 are all terminal complement components that are deposited in the pulmonary microvasculature leads to inflammatory changes in the lung, which is a sign of activation of complement pathways (both alternative pathway and lecithin pathways). Co-localization of COVID-19 spike glycoprotein with Cd4 and Cd5 in inter alveolar septa is also seen. Respiratory distress syndrome seen in COVID-19 seems to be different from typical Acute respiratory distress syndrome[8]. Cytokine storm syndrome can have infectious or non-infectious causes. Cytokines associated with cytokine storm syndrome are interferon (Type I, II and III), interleukins (IL-1alpha, IL-1beta and IL-18), chemokines (CXC, CC, C, CX3C), CSFs (GM-CSF and M-CSF) and tumour necrosis factor (TNF). Release of these cytokines leads to local and systemic inflammatory signs[9,10].

4. CLINICAL SIGNS OF COVID-19

Inflammatory response to SARS-Cov-19 may leads to systemic inflammatory response syndrome (SIRS)[11], which is defined as presence of 2 of the following findings:

Clinical sign	Indication
Hyperthermia	Fever more than 100.4 F
Tachycardia	More than 90 beats per minute
Tachypnea	Respiratory rate of more than 20 breaths per minute or arterial carbon dioxide of less than 32mmHg.
Lymphocytosis	(WBCs >12,000/uL)

Local signs include redness, swelling, edema, fever, pain, and loss of function. Later, systemic spread of cytokines leads to systemic manifestations of inflammatory response. Cytokine storm in lungs can lead to acute lung injury (ALI) which is characterized by acute neutrophilic recruitment followed by fibro proliferative changes in the lungs[12].

5. PATHOGENESIS OF COVID-19 PNEUMONIA

Community Acquired pneumonia is defined as pneumonia which occurs outside the hospital setting. It can be bacterial or viral. Typical Bacterial agents which cause pneumonia include Hemophilus Influenzas, pneumococcus, Moraxella catarrhalis, Group A streptococcus and anaerobic gram-negative bacteria. Atypical bacteria are legionella, mycoplasma, and chlamydia. Common viruses which cause pneumonia are influenza virus, respiratory syncytial virus (RSV), parainfluenza virus and adenoviruses[13]. Coronavirus usually causes cold and the flu, but it can cause pneumonia which can become severe. Usually, the viral pneumonias are mild. Immunocompromised individuals such as HIV patients and organ transplant recipients can have fungal infection. Histoplasma, Blastomyces and coccidioides are the common fungi which cause fungal pneumonia. Human body has defence mechanisms which provide protection against pneumonia causing microbial agents. Pneumonia occurs when these physiological defensive processes are disrupted due to any reason like impaired cellular or humoral immunity, impaired complement mediated immunity, kartergener syndrome, cystic fibrosis, bronchial obstruction, and cigarette smoking. Other risk factors include Children with age of 2 or younger and people over age of 65[14].

6. DIAGNOSIS

Radiological tests are chest X-ray and CT-scan of chest which shows consolidation in the lung and cavity lesion is severe disease. COVID-19 Pneumonia has bilateral chest infiltrates, which are seen on x-ray and CT-scan of chest. Laboratory tests include “blood culture, sputum culture, complete blood count and lymphocyte count”. Urinary antigen testing and bronchial aspirate are used for legionella. To differentiate the bacterial infection from viral, CRP levels are done. Nasopharyngeal swab test is done to rule out viral causes. Real time PCR of Nasopharyngeal swab sample is positive in COVID-19 pneumonia.

7. DISEASES IN ASSOCIATION WITH COVID-19

7.1. COVID-19 and acute respiratory distress syndrome (ARDS)

Multiple studies have shown link between ARDS and COVID-19, COVID-19 in patients with ARDS take place earlier as compared to women who do not have ARDS. It is confirmed from studies that in Italy having ARDS the incidence of COVID-19 is 2.6 times greater when compared to other people without this disease[12]. ARDS play its role in the increased risk of COVID-19. Inflammatory response in coronavirus infection leads to structural and functional alteration in lungs. Protein rich fluid leaking through endothelial cell gaps leads to pulmonary edema. The accumulating fluid disrupts the exchange of oxygen between blood vessels and alveoli leading to hypoxia and respiratory failure[15]. High protein content of alveolar fluid in ARDS differentiates it from pulmonary edema of cardiogenic cause which has low protein content. Alveoli are lined by type 1 squamous epithelial cells which form the tight barrier to fluid and protein to make the alveoli relatively dry for better exchange of gases[16]. Disrupted function of type 2 epithelial cells leads to impaired reabsorption of fluid from alveoli and subsequent development of pulmonary edema. Oxygen-carbon dioxide exchange occurs through thin alveolar-capillary barrier. Pulmonary edema leads to disruption of this barrier and impaired gas exchange. All these processes are disrupted in ARDS. Increased Capillary permeability leads to physical modifications in pulmonary capillary endothelium. ROS, Rho Guanosine triphosphate enzymes (GTPases) and tyrosine phosphorylation of junctional proteins are the regulator of junctional permeability[17].

7.2. Coronavirus associated gastritis

The main symptom of coronavirus gastritis is Diarrhea, seen in up to 30% of patients. It may also present as anorexia, nausea, and vomiting. Coronavirus infects by binding to Angiotensin converting Enzyme 2 ACE2 receptor which are also found in gastrointestinal tract (e.g., esophagus, gallbladder, pancreas, liver and colon. Spike protein has two subunits, S1 and S1 and has two structural conformations, pre fusion and post fusion. S1 subunit mediates binding and s2 subunit mediates fusion and viral entry into the host cell. The virus enters and replicates in the cells of gastrointestinal tract and leads to shedding of cells lining the GI tract[18].

7.3. COVID-19 Associated Encephalitis

Coronavirus spreads from respiratory tract to central nervous system due to its neuro-invasive capacity. SARS-CoV-2 causes the neurologic disease by damaging the nervous system. Clinical evidence has showed CNS involvement in COVID-19[19]. Encephalitis is an infection of brain substance. Infection in brain causes an inflammatory response which leads to inflammation of cerebral cortex, white matter, basal ganglia and brain stem. Area of brain involvement depends upon the causative agent such as in case of herpes virus, temporal lobes are involved[20]. While in Cytomegalovirus, area adjacent to ventricles is involved. Inflammatory response also causes the neuronal degeneration, diffuse glial proliferation and cerebral edema. Pathophysiological process of SARS-CoV-2 associated encephalitis is not well understood but it is believed that inflammatory response to infection causes neurologic injury and subsequent cerebral edema leading to altered conscious levels. Clinically encephalitis may present as acute onset of headache, fever, seizures and focal neurologic deficits[21,22]. Diagnosis of encephalitis is made by CT-scan brain which may shows low density lesions. But more sensitive imaging modality is MRI, which can reveal abnormalities at early stages[23].

7.4. COVID-19 associated thrombotic complications

COVID-19 has also been associated with thrombotic complications such as pulmonary embolism (20%-30% of cases), deep vein thrombosis (DVT) and catheter related thrombosis. In addition large arterial thrombotic complications such as ischemic stroke and microvascular thrombosis are also been seen[24]. Pathophysiological process associated with Thrombosis involves the binding of virus to Angiotensin converting enzyme 2 (ACE2), which are widely present on alveolar epithelium of lungs, cardiac myocytes and Vascular Endothelium. A massive inflammatory response is evoked in response to "SARS-CoV-2 infection which causes the release of inflammatory cytokines (IL-1beta, IL-6 and TNF-alpha)". This cytokine storm set ups systemic inflammatory response syndrome. Local and Systemic inflammatory response leads to thrombosis by two mechanisms, Hypercoagulability or endotheliopathy[25].

8. NUTRITIONAL THERAPY FOR COVID-19 PATIENTS

Tube feeding, oral nutrition supplementation (ONS), Supplementary parenteral nutrition, Total parenteral nutrition considered as best nutritional therapy which play fundamental role in recovery of infected patients.

When poor nutritional status exists is COVID-19 patients, it is mandatory to administer enteral nutrition (EN) or parenteral nutrition (PN). Oral administration with ONS is first line route to COVID-19 patients. EN or PN is administer to patients when ONS is insufficient to meet the nutritional needs of patients accordingly. Considering the clinical characteristics of COVID-19 patients the method, plan, route, and formula of nutrition therapy should be calibrated dynamically and conveniently (Saul, 2020a). There should be smooth transition between EN, PN and ONS. Based on international guidelines following decalogue will help in managing nutritional therapy for COVID-19 patients (Livingston and Bucher, 2020). With respiratory insufficiency, in ICU admitted patient, MNT should be initiated with following priorities:

- If there are no symptoms present so early EN must be started within 48 hours.
- PN can be started slowly on day 3 and the infusion rate gradually increased up to the 7th day and on day 7, in patients at risk of malnutrition, with stable clinical conditions. PN can be started by changing the fluid therapy.
- Overfeeding must be prevented; EN and PN should be authorize at increasing speed to prevent overfeeding and the target speed must be stick out within 3-4 days (an internal or endogenous production of 2092-5857 kJ/day is present in the first 72 hour in delicate phase).
- It is feasible to initiate an integrated medical nutritional therapy; if the mandatory prerequisites are not met with EN and PN is favored to administer poly amino acids.

8.1. Proteins rich diet for Covid-19 Patient

It is very censorious to maintain the energy balance of patients suffering with COVID-19. Patients of COVID-19 required more energy than normal person. Exacts estimate of caloric needs is functional to circumvent overfeeding or underfeeding[26]. It is recommended to supply 84-126 kJ/kg/day (1 kcal = 4.184 kJ). Body's energy utilization rises by 10%, for every 1°C increase in body temperature. As a top priority it is stipulated to enhance protein supply, to decrease the catabolism due to the inflammatory negotiator[27]. It is suggested 1.3 g/kg/day increasing the supply of amino acids to 50%, to prohibit muscle loss, raises the strength of respiratory muscles.

8.2. Carbohydrates and Fats rich diet for Covid-19 Patient

Due to respiratory failure in COVID-19 patient which are in the critical phase; carbohydrates management must be limited. The carbohydrate requirement is 2 g/kg/day and should not increase 150 g per day. In respiratory failure, to decrease the respiratory quotient CO2 production should be prevented. In critically ill patient the lipid requirements are 1.5 g/kg/day. Commonly, the 0.5 g/kg/day of lipids derive from the management of sedatives in lipid-solution. Increase in the proportion of ω-3 fatty acids and ω-9 fatty acids occur due to the preference in the use of medium and long chain fatty acids. Fatty acids which are necessary; play a major role in immune responses by altering the manufacturing of cell membranes and modulating cell signalling.

8.3. Fluid volume and Micronutrient's requirement

It is necessary to maintain balance in neutral fluid of patients which are suffering critically with COVID-19 with particular consideration to renal and prerenal failure[28]. For stable patients in ICU: 30 mL/kg/day of fluid for adult and 28 mL/kg/day for elderly. It is specially suggested to control the number of intravenous fluids for elderly patients and large areas of pulmonary consolidation. Supplement is taken 3-5 mL/kg for every 1℃ increase in body temperature. In patients suffering from COVID-19, division must be done in the demand of micronutrients and the managements according to the nutritional therapy

8.4. Immuno-nutrients

These nutrients have been shown to have a significant impact on immune function as well as increase metabolic and nutritional indices including nitrogen balance and serum proteins. Immune-nutrients, such as arginine, nucleotides, glutamine, and omega-3 fatty acids, come in a variety of forms, each with its own set of functions and mechanisms. Immuno-nutrients can help patients heal by inhibiting inflammation and

synchronizing immune function. Also, different plants can be used to boost up immune response to combat COVID-19[29].

9. GENERAL AND DIETARY GUIDELINES REGARDING COVID-19

A well-known proverb states that prevention is preferable to treatment. To combat the harmful and life-threatening effects of COVID-19, the following lifestyle changes and common recommendations should be followed:

- The most well-known ways to avoid infection are good sterility practise, social distancing, and isolating those who are infected.
- Before and after cooking or consuming food or social exposure, wash your hands for 20 seconds with soap or sanitise your hands. Viruses on your hands are killed by alcohol-based hand rub.
- Keeping a one-meter (3-foot) distance between yourself and others is needed because when someone coughs, sneezes, or talks, tiny liquid droplets from their nose or mouth are transferred, which may contain virus. If you are near someone who has COVID-19, you can breathe in the droplets, which could contain the virus.
- Avoid going to rabble places because when people congregate in groups, you are more likely to come into direct contact with someone who has COIVD-19 and maintaining a physical distance of one metre is more difficult (3 feet)[3].
- If you have a slight symptom like a cough, headache, or moderate fever, stay at home and isolate yourself until you feel better. They will be protected from COVID-19 and other viruses by avoiding contact with others.
- Maintain respiratory hygiene by wearing the mask.
- If you have symptoms such as a fever, cough, or difficulty breathing, seek medical help using modern technologies such as calling or following the guidelines given by your local health authority. According to the situation and sites where you are located, national and local authorities provide more knowledge about the disease, signs, cures, and preventive measures. Calling in advance will allow your health care provider to quickly direct you to the right health facility[30].
- This will also protect you and aid in the prevention of virus and infection spread. Before and after use, sanitise surfaces and objects.

10. CONCLUSIONS

Based on clinical and general observation regarding COVID-19, it is incontrovertible that despite COVID-19 can affect people of all ages, but it has worse prognosis, severe susceptibility and high mortality rate in people who are malnourished, already have chronic diseases and low immunity. Immuno-compromised people are at major threat as COVID-19 progresses abruptly in week immune system and malnourished persons. Good nutrition is not only a key to immunity against diseases, including COVID-19 but also a barrier promotion of diseases as well. Metabolic disturbances contribute to the development and progression of respiratory distress, as well as they have serious consequences in next stages of life that can have harmful effects on health. There are number of metabolic and environmental factors that can contribute to the development of COVID-19 including pneumonia, fever, diarrhea and lungs dysfunctions. More studies are needed to be executed in accordance to know the link between COVID-19 and mortality.

CONFLICT OF INTEREST

No conflict of interest

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