Review Article

A Review of Covid -19 Detailed study of History, Life cycle, Diagnosis, Prevention and Treatment of Corona virus.

ABSTRACT:

Coronavirus Disease-2019 is a new life threatening quickly spreadable pandemic disease. It is a huge family of viruses known to cause sickness from the breathing trouble, fever, fatigue, cough, sore throat, breathlessness, and common cold to the continuation of acute respiratory tract infection and the severity of the infection sometimes visible as pneumonia, acute respiratory syndrome and even death. The disease is commonly known as covid-19 Since December 2019, in this time Covid-19 emerged in Hunan seafood market at Wuhan in this area first discovered in coronavirus, country of South China and rapidly spread throughout the world, the virus epidemic has been proclaimed a public health emergency of International concern by World Health Organization (WHO). The virus spread extensively in Wuhan region of China and has obtained entry to over 210 countries and territories. An exceedingly potential for spreading resulted in the universal coronavirus disease 2019 (COVID-19) pandemic in 2020. In spite of the aggravate trends of COVID-19, no drugs are validated to have significant effectiveness in clinical treatment of COVID-19 patients in large-scale studies. Though specialist mistrust that the virus is transmitted from animals to humans, there are mixed results on the origin of coronavirus. Without treatment options in the virus as such, limited to the use of antiviral drugs, anti-malarial drugs, plasma therapy, and oxygen therapy and by the immune system. Antiviral drugs and immune modulating treatments are present being tested. In this review of the detail study about history of covid-19, virus life cycle, diagnosis, prevention and treatment.

Keywords: COVID-19, SARS, Symptoms, Respiratory syndrome, Treatment.

1. INTRODUCTION:

Up to SARS outbreak (2002), in which coronaviruses displayed their possible for epidemic spread and significant pathogenicity in humans, in which mainly known as causes of mild respiratory syndrome and gastrointestinal disease [1]. Finished in first two decades third novel Beta coronaviruses, it is Severe Acute Respiratory Syndrome (SARS)-CoV, Middle East Respiratory Syndrome (MERS)-CoV and SARS-CoV2, in which crossed the species barrier and caused significant epidemic characterized by increase case-fatality rates in humans [2–4]. The little reemergence of SARS in late 2003 after the restarting of the wildlife market in southern China and the current discovery of a very alike virus in horseshoe bats, bat SARS-Corona virus, suggested that SARS can return if circumstances are fit for the introduction, amplification, mutation, and transmission of this threatening virus [5-8]. Seven coronaviruses can bring about

infection and people surrounding in the world but generally people get infected with these four human coronaviruses such as 229E, NL63, OC43, and HKU1[9].The virus is typically quickly spread from one person to another person via respiratory droplets produced throughout coughing and sneezing. It's considered most infectious when people are symptomatic, although transmission may be achievable before symptoms show in patients. Time from revelation and symptom onset is commonly between 2 and 14 days, with an average of 5 days. General symptoms such as fever, cough, sneezing and shortness of breath. Complications involved such as pneumonia, throat pain and acute respiratory syndrome. Recently, there is no specific treatment or vaccine; efforts consist of symptom terminate supportive therapy. Recommended preventive measures such as washing your hands with soap, covering the mouth when coughing by using face mask or hand kerchief, maintaining one-meter gap from other people and monitoring and self-isolation for 14 days for people who suspect they are infected [10].The disease diagnosis by using a standard tool of Reverse Transcription Polymerase Chain Reaction (RT-PCR) from a throat swab or nasopharyngeal swab [11]. Diagnostic approaches to COVID-19 can be divided into two broad categories included in Clinical diagnostics and in vitro diagnostics [12–14]. Clinical diagnostics for COVID-19 include to begin with assessment of possible COVID-19 related symptoms and exposure the past. These should be considered in the conditions of the SARS-CoV-2 incubation period, which is evaluated to be up to 14 days from exposure, with a median of 4 to 5 days [15–17]. Invitro diagnostics for SARS-CoV-2 infection is confirmed by observation of SARS-CoV-2 RNA using NAAT [18].It is a two-step assay usually takes 3.5–4 h and need three reagent kits such as RNA extraction, cDNA synthesis, and amplification and detection of the target nucleic acid, as well as specialized lab equipment [19]. Clinical management of SARS has relied increasing upon supportive care. Broad-spectrum antimicrobial coverage for community obtained pneumonia should be given while virological confirmation is pending. Such antibiotics should be over once the diagnosis of SARS is confirmed, but nosocomial infections as an outcome of extent intubation and used in corticosteroids should be appropriately managed. The correlation between viral loads and clinical result suggests that suppression of viral replication by effective antiviral agents should be the key to fend off morbidity and mortality [20-22].

2. HISTORY AND LIFE CYCLE OF VIRUS:

2.1 History

The first sufferer of coronaviruses in human present in 1965 by Tyrrell and Bynoe. They noticeable that they could passage a virus named B814. It's noticeable in human embryonic tracheal organ cultures obtained from the respiratory tract of an adult in common cold symptom. The first sufferer were seen in Wuhan City of Hubei region China in December 2019, and have been associated to the Huanan Seafood Market at South China and the infection has spread to several countries around the world [23].

SARS is a first known major pandemic disease caused by a coronavirus. During the outbreak in 2003 in which 8,096 cases with 774 deaths had occurred in over 30 countries in the middle of 5 continents [24- 39]. The disease emerged in late 2002, when a pandemic of acute community acquired atypical pneumonia syndrome was first observed in the Guangdong region.

Retrospective surveillance divulge severe cases of the disease in five cities around Guangzhou above a period of 2 months [40]. The index case was announced in Foshan, a city 24 km away from Guangzhou. The second sufferer involved a chef from Heyuan who worked at restaurant in Shenzhen. The patient had continuous contact with wild game food animals. His wife, 2 sisters, and 7 hospital staff who had contact with him were also affected. Since 16 November 2002 to 9 February 2003, totally 305 cases were reported in mainland China, with 105 of those sufferer involving health care workers. The devastating outbreak started in Hong Kong Special Administrative Region (HKSAR), when a professor of nephrology since a teaching hospital in Guangzhou who had received the disease from his patients came to HKSAR on 21 February 2003. In this day, he transmitted the viral infection to 16 other people in the hotel where he resided. His brother-in-law, one of the secondary sufferer, underwent an open lung biopsy since which the etiological agent was find out and first isolated [32]. It is a novel coronavirus, named in SARS-CoV.

2.2 Life cycle

Trimers of the S protein form the peplomers. Protein S is a class I fusion protein such as amino-terminal S1 and carboxyl-terminal S2 subunits connected by a fusion peptide. In the two subunits are indispensable for receptor binding and membrane fusion in respectively. A receptor binding domain of S1 has been mapped to residues 318 to 510 [41- 42]. Protein S1 is binding to the cellular receptor will trigger conformational changes, in which collocate the fusion peptide upstream of the 2 heptad repeats of S2 is a transmembrane domain and finally resulting fusion of the viral and cellular lipid envelopes. Moreover, this process could be facilitated by the infected cell membrane that associated with protease, like factor Xa, which can cleavage of S into S1 and S2. In this proteolytic cleavage is specifically inhibited by a protease inhibitor such as Ben-HCl [43]. The host cell of the key receptor attached by S is angiotensin-converting enzyme II (ACE II), it is a metalloprotease expressed in the cells of the lung, intestine, liver, vascular endothelium, heart, testis, and kidney [44]. From ACE II was appear to protect against acute lung injury in a mouse model and from the binding of S protein to host cells finally in the down regulation of ACE II. Aiming this mechanism may contribute to the severity of lung damage in SARS [45]. Cells expressing some lectins, including DC-SIGN, L-SIGN, and LSECtin, have been appear to augment the cellular entry of pseudo type virus expressing S but only in the concomitant presence of Angiotensin Converting Enzyme II [46-49]. Nonsusceptible cells are expressing the lectins and in these absence of ACE II, like dendritic cells, in this able to promote the cell-mediated transfer of SARS-CoV to susceptible cells [46]. Although lysosomotropic agents are used to block the viral entry, it's indicates that endosomal acidification is required for entry and the activation of S protein by protease can bypass this prevent and result in cell-to-cell fusion. In spite of the role in pH-sensitive endosomal protease cathepsin L in the entry pathway [50-51], viral culture does not needed pretreatment with trypsin. But, pH-sensitive cathepsin L some time target for agents including chloroquine, thus elevates endosomal pH [52-53]. In this process of viral take-apart in the cytoplasm for the release of viral RNA for translation and replication remains elusive. Translation begin with two large polyproteins since Orf1a and Orf1ab that are posttranslationally cleaved by the two viral proteases including nsp1 to nsp16. In this cleavage products form the replication-transcription complex, in which replicates the viral

genome and transcribes a 3 conterminal nested set of 8 subgenomic RNAs.Therefore conceivable that infected cells contain an increasing number of transcripts containing genes towards the 3 terminus of the viral genome. In this basis, Reverse Transcriptase Polymer Chain Reaction (RT-PCR) using the N gene may have a better sensitivity than using the other genes. And other coronaviruses, SARS-CoV may attach by the hydrophobic domains of their replication equipment to the restrict membrane of autophagosomes and form double-membrane vesicles. Once adequate viral genomic RNA and structural proteins are accumulated, viral assembly by budding of the helical nucleocapsid at the endoplasmic reticulum to the Golgi intermediate compartment take place. At this time, the triplemembrane-spanning M protein interact with N protein and viral RNA to create the basic structure. It is interacts with E and S proteins to induce viral budding and release. Unlike other coronaviruses and the M protein of SARS-CoV also indicate another triple-membrane-spanning protein of Orf3a into the virion [54]. The N protein is the most copious expressed viral protein is infected cells in which the mRNA levels were amplified 3 to 10 times increasing at 12 h postinfection than other structural genes [55] and therefore an main target for immunohistochemistry and antigen detection in the clinical specimens. Such as Various diagnostic tests, antiviral drugs, and vaccines are designed on the basis of our comprehension of the structure and function of the several viral proteins involved in the life cycle of this virus.

3.DIAGNOSIS:

RT-PCR assays are conventional or automated type of assay. Alternative terminologies such as rRT-PCR or RT-qPCR.

NAAT (Nucleic Acid Amplification Test) used to detect the presence of viral RNA [56]. Purified RNA from clinical specimens is reverse transcribed into complementary DNA (or) cDNA, next added to a master mix containing target primers and fluorophore-quencher probe. The RT-PCR process is carried through in a thermal cycler. The fluorophore-quencher probe is cleaved and creating a fluorescent signal that corresponds to the amplified outcome [57-58]. While conventional NAAT start from manual RNA preparation, followed by rRT-PCR; automated systems unsegregated RNA extraction, purification, amplification, detection and outcome in rapid, high-throughput results and decreased contamination [59–62].Pre-heating specimens to omit RNA extraction [63–66]. Accuracy with alternative, reduced invasive specimens (e.g., Saliva) in comparison with standard NP specimens [67–70]. Less respiratory specimens may provide pros later in the disease course [71], while non-respiratory specimens may correlate with local symptoms (e.g., stool) or clinical seriousness (e.g., blood) [72-74]. Swab pooling to be increase testing capacity [75]. Various PCR target regions may act on sensitivity [76–79]. Monitoring effect of SARS-CoV-2 genome mutations on RT-PCR showing [80- 81].First-step (consolidated RT and PCR) versus. Second-step (separate RT and PCR) assays, and uniplex versus. Multiplex RT-PCR [82- 83, 58]. Subgenomic RNA or Ct value in the surrogate for infectious or live virus [84].

3.1 FUTURE MANAGEMENT:

Availability of the diagnostic technologies has enabled researchers to quickly adapt them to COVID-19 [58]. Lessons since the 2002 SARS epidemic have guided development of COVID-19 detection strategies. Only three weeks elapsed since visualization of the virus by using transmission electron microscopy to the elucidation of SARS-CoV-2 genetic sequence, while SARSCoV took five months to be grant [58, 85]. Control of outbreak requires extensive, going on surveillance, and quick sharing of epidemiological data [86]. Smartphones are used in which increased exponentially and including in sub-Saharan Africa, can be leveraged for this motive as they possess link, computational power, and hardware to ease electronic reporting, epidemiological data basing and sharp end of care testing [58, 87]. Combining diagnostics tools with smartphone combination could support better management, curb transmission of infection and decrease mortality [58]. Safety of laboratory employee who conduct COVID-19 testing is also paramount. Concern for laboratory-associated infection is of specific concern in the setting of individual or personal Protective Equipment (PPE) shortages, inappropriate microbiological techniques, insufficiency of training, and inadequate detoxification protocols or biosafety measure [88]. Totally which are more expected to occur when systems are overwhelmed. Optimization of mechanisms to save laboratory employee should occur in parallel with optimization of COVID-19 diagnostics.

4. PREVENTION:

To diminished COVID-19 transmission since potentially asymptomatic or presymptomatic people, the ECDC recommends to the use of face masks [89].While included social distancing, travel restrictions on visitors arriving from increasingly risk province, quarantine for nationals returning from increasingly risk locations, and closure of schools, colleges and certain types of workplaces. The full or partial closure of educational institutions and certain workplaces, Restrict the number of visitors and restrict the contact betwixt the residents of confined settings, including prolonged-term care facilities and prisons, Cancellation, barring and reduction of mass gatherings and smaller meetings, obligatory quarantine of buildings or residential areas, Internal or external boundary closures, and Stay-at-home restrictions for whole regions or countries. All ministries published common instructions on COVID-19 prevention and control measures in their organizations [90]. In April 13, approximately 40,000 tests have been reached per day with a total of 73 authorized laboratories, and the number of daily tests is step by step increasing.

5. TREATMENT:

5.1 Antimalarial Drugs against SARS-CoV-2

Hydroxy Chloroquine:

Chloroquine is a phosphate and sulphate derivative drug it can be medically used the drug administered as antimalarials, and hydroxychloroquine is an immunomodulatory agent in systemic lupus erythematosus. Chloroquine present antiviral activity against Influenza, seasonal CoVs, Chikungunya virus and SARS [91-94]. Chloroquine derivatives against SARS-CoV2 was identified in vitro early on [95].The drug was quickly introduced into clinical use, and preliminary reports suggested enhanced viral clearance and clinical result in COVID-19 patients receiving a 10 days course of Hydroxychloroquine [96]. And randomizing 36 patients with

COVID-19 suggested accelerated viral clearance in patients treated with a combination of hydroxychloroquine and azithromycin [97]. However, others have challenged outcome and found no pros in either disease results or viral clearance [98]. Disappointingly, the largest retrospective study to date assessing Hydroxychloroquine on its own or in combination with azithromycin found no pros, but indeed an enhance mortality risk among patients receiving hydroxychloroquine [99]. A study exploring chloroquine diphosphate in two dosing regimens was forced to terminate early for concerns over in fact mortality in the high dose arm. The authors conclude that treatment with high dose chloroquine for 10 days is not sufficiently safe and should no longer used in severe SARS-CoV2 patients [100].

Azithromycin produced synergistic effects while azithromycin and hydroxychloroquine against SARS-CoV2 have been observed in vitro, which seem to translate into clinical practice [97,101-102].

Interestingly, azithromycin is a weak base, and it's accumulates in endosomes, with an alkalinizing effect at least parallel to Hydroxychloroquine. Inclusion to its antimicrobial properties, azithromycin is sometimes used for its immunomodulatory properties, mainly in patients with chronic pulmonary disorders. Azithromycin polarizes macrophages towards an anti-inflammatory M2 phenotype, and prevent pro-inflammatory STAT1 and NFκB signaling pathways [103,104]. In the context of anti-inflammatory effects, in particular interest that azithromycin is used for patients requiring intensive care for nonCOVID-19 related ARDS and is associated with a significant reduction in mortality and low time to extubation [105–107].

5.2 Antiviral Drugs against SARS-CoV-2

The antiviral drugs are especially used in case of HIV/AIDS, such as Lopinavir and Ritonavir. Other drugs and nucleoside analogs such as Favipiravir, Ribavirin, Remdesivir, and Galidesivir have been tested for present activity in the prevention of viral RNA synthesis [108]. Among these drugs, Lopinavir, Ritonavir, and Remdesivir are listed in the Solidarity trial by the World Health Organization (WHO).

5.3 Plasma from recuperating patients:

Recuperating plasma, the plasma since individuals following COVID19 resolution and affluent in immunoglobulins directed against SARSCoV2, is being entertained as believable treatment option [109-110]. Unscientific use in SARS, MERS, Ebola and Influenza patients supports its use of neutralizing or immunomodulatory agent [111-112].But, a larger randomized controlled estimate of hyperimmune intravenous immunoglobulin use for severe influenza [113-114] and Ebola [115] manifest this intervention without superior to placebo. Similarly, rigorously estimated data used in coronaviral infections is lacking but not only for its use in SARS-CoV2 [116], and a possible study exploring use in MERS found that in numerous survivors, antibody titres were not high enough, so further restrict the donor pool [117]. Different dosing, issues surrounding donor recruitment in times of quickly increasing patient numbers, and drawbacks concerning safety of widespread use of human blood products all limit availability and its benefit widely available treatment option. Eventually, the viruses that are subject to ADE (such as SEARS-CoV2) by non-neutralizing antibodies, the choice of plasma therapy also holds

significant risks. This complication has lately exemplified by anti-Zika virus antibodies increasing Dengue virus infection [118].so, the administration of hyperimmune/convalescent plasma may carry the threat of significant sickness upon future exposure to related or yet-to-emerge coronaviruses.

6. CONCLUSION

In this review, we conclude that the disease description of COVID-19 is dynamic and continues to quickly evolve. As more and more suspected cases of COVID 19 infection arises, crisis prospect of RT-PCR kits may also be enhanced. This has led to chest CT being utilized to aid diagnosis is not presence of RT-PCR, as demonstrated in a recent case reported since China and all over world. The development of the lung changes of COVID-19 on CT imaging is also similar to SARS, with the ground-glass and consolidation far worse or better over several days. This would be expected, as the 2 infectious agents in the part of the coronavirus family. We are only beginning to understand host factors, such as various expression of cell surface proteins that may determine infection risk, disease presentation and results. Unveiling tissue and stage specific factors contributing to pathology will outcome in new, effective and disease stage particular therapeutic approaches that control virus replication while restrict inflammatory damage until vaccinations become available.

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