COVID-19: A Review on Epidemiology, Clinical Features and Possible Potential Drugs Based on Available Case Studies

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Abstract: The emerging novel coronavirus disease 2019 has caused a global outbreak and significant public health concern. The World Health Organization (WHO) has announced a coronavirus disease outbreak a pandemic with a global public health emergency of international concern. As of now, 12\textsuperscript{th} April 2020 almost 18,37,404 cases have been confirmed globally (in 209 countries) with almost 1,13,274 fatalities. This increasing number has created anxiety throughout the world, which has severely affected the whole world's culture, societies, behavioral patterns, peace and economics. At present, research on novel coronavirus is in the preliminary stage. There is no vaccine or specific antiviral to treat coronavirus disease. Also, very few case studies are available; hence it has become difficult to treat and to control this pandemic situation. In view of this, the present systematic review is done to highlight clinical epidemiology features, radiographic characteristics, and potential drugs based on available clinical case reports. Biomarkers for early diagnosis and impact of age, sex, pre-existing comorbidity on COVID-19 is also discussed. Further, this paper also outlines various possible antiviral chemical drug agents that can be potential and promising to treat this coronavirus disease in 2019. This review may be helpful for the medical practitioner, public health workers and government authorities to manage and deal with novel coronavirus disease 2019.

Keywords: COVID-19 epidemiology, coronavirus, clinical characteristics, potential drugs, antiviral agents, SARS-CoV-2, pediatric, pregnant women.

1. INTRODUCTION

Since the first week of December 2019, some cases of severe pneumonia from an unknowing aetiology/pathogen have been reported in Wuhan (Hubei, China) [1, 2]. Most patients showing characteristics and symptoms of pneumonia were regular workers, customers or visitors from the local Huanan sea-food wholesale market where live as well as dead animals were always on trade [1, 2]. Coronaviruses (CoV) cause respiratory tract, gastric and neurological diseases in birds, reptiles and mammals [3-6], till date six human coronaviruses (HCoV) have been reported in which four are endemic (i) HCoV-OC43, (ii) HCoV-229E, (iii) HCoV-NL63, and (iv) HCoV-HKU1 and two are epidemic (i) SARS-CoV and (ii) MERS-CoV) [3, 4]. Coronavirus belongs to the family Coronaviridae, which is an enveloped positive-sense single-stranded RNA virus [1-3]. These coronaviruses are divided into four genera named as, (i) alpha-coronavirus, (ii) beta-coronavirus, (iii) gamma-coronavirus and (iv) delta-coronavirus. The isolation of this mysterious virus from the infected patient and corresponding phylogenetic analyses showed close resemblance with bat coronavirus, which is designated by WHO as “2019 novel-coronavirus” (2019-nCoV) disease (COVID-19) and the reference name as “severe acute respiratory syndrome coronavirus-2” (SARS-CoV-2) [5-7]. At present, no effective drug, vaccine, or treatment is available for this virus, which creates a pandemic situation globally. Moreover, high mortality rates, contagious nature and its potential to cause pandemic grabs the serious attention of the whole world [5].

As of now (12\textsuperscript{th} April 2020), almost 18,37,404 cases have been confirmed with almost 1,13,274 fatalities globally; this number is going on increasing day by day and has created anxiety throughout the world which has severely affected the whole world culture, societies, peace and economics [8] (Fig. 1A, 1B). The World Health Organization (WHO) declared the outbreak of 2019-novel coronavirus as a pandemic
with a global public health emergency of international concern [7-10].

Currently, no vaccine or appropriate antiviral treatment is available due to complicated virus-cell receptor interaction and surface structure of spike glycoprotein [7, 9]. Moreover, knowledge about epidemiology and clinical features of the COVID-19 is much limited [9, 10]. All medical professionals are busy in the service to manage COVID-19 pandemic and hence it is a prime duty of researchers to present a review article that can emphasize the clinical characteristics of COVID-19 on the basis of scientific evidences and case studies/literature. Considering this primary objective, we make an attempt to explore the evidences/case studies of early findings of the clinical characteristics and therapeutics used to control COVID-19. For this, we conducted a related literature search (scientific evidences/case studies) of the last three months, which are analyzed critically and summarized into (i) clinical characteristics (in adults, pediatric patients and pregnant women), (ii) antimicrobial treatments to control COVID-19 and (iii) impact of age, sex and pre-existence of comorbidities in the severity of COVID-19. Thus, this review can provide significant information about clinical features (symptoms, diagnosis, biochemistry, anti-viral chemical drug agents, prevention plans, prophylaxis strategies, the possibility of re-infection and second-wave). Furthermore, this review may be helpful to medical practitioners, health workers, clinical researchers, and decision making government authorities to manage this global public health emergency.

2. METHODOLOGY

We searched related literature from various search engines such as PubMed, Sci-finder, Scopus, ScienceDirect and Google Scholar using the terms: anti-viral chemical drug agents, therapeutics, clinical cases studies/trials, for- novel coronavirus disease, COVID-19, nCoV-19 and SARS-CoV-2 etc in adults, pediatric patients and pregnant women. All available publications with above queries are strictly reviewed, analysed and segregated appropriately into population data, mean age, symptoms, laboratory finding, chest study, biochemistry, treatment and clinical outcomes as reported in case study/evidence (Table 1, entries 1-18) [11-28]. A similar search methodology is used to find out the literature about hydroxychloroquine for COVID-19, the possibility of COVID-19 re-infection, and second-wave.

3. COVID-19 INFECTION AND SYMPTOMS

COVID-19 virus follows the same mechanism to enter into host cells like the SARS-CoV virus [10, 11]. It has a higher incubation period and is found to be more selective and contagious than SARS-CoV [10]. Furthermore, ACE-2, which was known receptor for SARS-CoV, is likely to act as a receptor for SARS-CoV-2 due to amino-acid homology. It has spike protein which involves the two subunits [10]. The first subunit shows binding ability to host protein receptor ACE-2 in lung and adipose tissues, while the second subunit causes the membrane fusion and introduction of RNA into the cell. The basic clue for the disease COVID-19 infection is the travel history, visit exposure and direct contact. The initial and most typical characteristic symptoms for COVID-19 patients involve fever (37.6-38.6°C), dry-cough, myalgia, and sore-throat, which are very general in respiratory viral infection/influenza/pneumonia (Table 1, entries 1-18) [11-28]. Kim et al. [11] reported the presence of pneumonia in the patient at the beginning of illness with symptoms such as fever (38.4°C), chill, myalgia, sore-throat, cough, nasal congestion and pleuritic chest discomfort [11]. Wang et al. [22] reported that pneumonia with dry-cough, fever and fatigue are common onset symptoms whilst, upper respiratory tract infection like symptoms (pharyngalgia and rhinorrhea) are not common [22]. However, the possibility of COVID-19 in the patients cannot be ruled out who showing the symptoms of nasal tampon, rhinorrhea, and pharyngalgia [22, 24]. Wang et al. [25] observed fever (~98%), fatigue (~70%), dry-cough (~60%), myalgia (~35%) and dyspnea (~31%) as the most common symptoms appear at the start of sickness, while headache, dizziness, abdominal pain, diarrhea, nausea, and vomiting are found to be less common symptoms [25]. The onset of symptoms and its appearance assist physicians in predicting the severity of illness [25].

In conclusion, one might have COVID-19 without any substantial initial symptoms that can be observed. A person with COVID-19 may not have a cough, cold and fever depending on strong immunity. However, such a person may cause the transmission of the virus/infection to a normal one. Thus the person with no symptoms can continue to transmit the virus/infection for 2 to 14 days. On average COVID-19 patients took a week from exposure to show their initial symptoms. Primarily it shows the symptoms similar to that of pneumonia or common flue, which becomes more severe
Table 1. COVID-19 in adult: Symptoms, biochemistry, CT radiography and treatment.

<table>
<thead>
<tr>
<th>Entry</th>
<th>Population</th>
<th>Avg. Age</th>
<th>Symptoms (% or No. in Population)</th>
<th>CT Chest/ Chest Radiography (% or No. in Population)</th>
<th>Lab Findings (% or No. in Population)</th>
<th>Confirmatory Test</th>
<th>Treatment (% or No. in Population)</th>
<th>Outcome</th>
<th>Refs.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1</td>
<td>35</td>
<td>Fever temperature of 38.4°C, chill, myalgia, sore-throat, cough, nasal congestion, pleuritic chest discomfort, diarrhea</td>
<td>No infiltrations, ground-glass opacities in both subpleural spaces (on fourth day of illness)</td>
<td>Leukopenia, thrombocytopenia. Increase of liver enzyme</td>
<td>PCR assay-swab throat sample</td>
<td>Antiviral therapy lopinavir 400 mg/ritonavir 100 mg</td>
<td>NM</td>
<td>[11]</td>
</tr>
<tr>
<td>2</td>
<td>13</td>
<td>34</td>
<td>Fever, cough (46.3%), upper airway congestion (61.5%), myalgia (23.1%), and headache (23.1%)</td>
<td>Scattered opacities in left lower lung (8%), ground glass opacity in the right or both lungs (52 %), no consolidation/scarring (40 %).</td>
<td></td>
<td>q-PCR assay-throat swab samples</td>
<td>Antiviral therapy lopinavir 200 mg/ritonavir 50 mg</td>
<td>All patients recovered.</td>
<td>[12]</td>
</tr>
<tr>
<td>3</td>
<td>1</td>
<td>54</td>
<td>Fever and dry-cough on fifth and seventh day of sickness</td>
<td>Ground-glass opacities in both lower lobes while small consolidation in right upper lobe</td>
<td></td>
<td>RT-PCR assay-swab throat sample</td>
<td>Antiviral therapy lopinavir 200 mg/ritonavir 50 mg</td>
<td>NM</td>
<td>[13]</td>
</tr>
<tr>
<td>4</td>
<td>17</td>
<td>45</td>
<td>Fever (6.6 days), cough (83.3%), fatigue (41.7%), myalgia (16.7%) and diarrhoea (16.7%)</td>
<td>Patchy shadows or ground glass opacity (58.3%). Bilateral lobes or multi-lobed infectious lesions of both lungs (41.7%) and inflammatory lesions (60%).</td>
<td>Lower lymphocytes levels, lower monocytes levels. Abnormal increased C-reactive protein levels (100%).</td>
<td>q-PCR assay-throat swab samples</td>
<td></td>
<td>12 patients quarantined in hospital and 5 patients discharged</td>
<td>[14]</td>
</tr>
<tr>
<td>5</td>
<td>99</td>
<td>55.5</td>
<td>Fever (83%), cough (82%), shortness of breath (31%), muscle ache (11%), confusion (9%), headache (8%), rhinorrhoea (4%), sore-throat (5%), diarrhoea (2%), chest pain (2%), nausea/vomiting (1%).</td>
<td>Multiple mottling and ground glass opacity (14%), pneumothorax (1%)</td>
<td>Lymphocyte level in most patients was reduced (35%). Haemoglobin decreased (51%). Neutrophils level increased (35%)</td>
<td>RT-PCR-throat swab sample</td>
<td>Antiviral therapy oseltamivir, ganciclovir, lopinavir and ritonavir (76%), antibiotics (25%), combination therapy (45%), methyl-prednisolone sodium succinate, and dexamethasone (19%)</td>
<td>Hospitalized, discharged and died 57, 31, 11 % respectively</td>
<td>[15]</td>
</tr>
<tr>
<td>6</td>
<td>NM</td>
<td>NM</td>
<td>NM</td>
<td>NM</td>
<td>NM</td>
<td>RT-PCR</td>
<td>Antiviral therapy lopinavir and remdesivir</td>
<td>NM</td>
<td>[16]</td>
</tr>
<tr>
<td>7</td>
<td>NM</td>
<td>NM</td>
<td>NM</td>
<td>NM</td>
<td>NM</td>
<td>Antiviral therapy lopinavir/ritonavir and remdesivir</td>
<td>NM</td>
<td>[17]</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>125</td>
<td>NM</td>
<td>NM</td>
<td>NM</td>
<td>NM</td>
<td>Antiviral (33.3%), TCM (33%), anti-inflammation or immunomodulator (14.7%), cell-based therapy (9.3 %), antioxidants (2.3%) and other approaches (7.0%), Probiotics/ corticosteroids, Regulating Intestinal Flora.</td>
<td>NM</td>
<td>[18]</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>1</td>
<td>33</td>
<td>Sore-throat, chills, and myalgias, fever of 39.1°C, developed, along with a productive cough.</td>
<td></td>
<td></td>
<td>RT-PCR, Two nasopharyngeal swabs and one sputum</td>
<td>Antimicrobial therapy including oseltamivir, arbidol, lopinavir/ritonavir and moxifloxacin</td>
<td>Discharged home quarantine for at least 14 days</td>
<td>[19]</td>
</tr>
<tr>
<td>10</td>
<td>1</td>
<td>46</td>
<td>Fever of 37.3-38.3°C with no other obvious symptoms, sore-throat, cough and chest distress</td>
<td>Multiple patchy ground glass opacities in bilateral sub-pleural areas.</td>
<td></td>
<td>RT-PCR oropharyngeal or nasopharyngeal</td>
<td>Antimicrobial therapy including oseltamivir, arbidol, lopinavir/ritonavir and moxifloxacin</td>
<td></td>
<td>[20]</td>
</tr>
</tbody>
</table>

(Table 1) contd....
<table>
<thead>
<tr>
<th>Entry</th>
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<th>Lab Findings (% or No. in Population)</th>
<th>Confirmatory Test</th>
<th>Treatment (% or No. in Population)</th>
<th>Outcome</th>
<th>Refs.</th>
</tr>
</thead>
<tbody>
<tr>
<td>11</td>
<td>19</td>
<td>48</td>
<td>Fever (78.9%), cough (47.37%), sore-throat (21.05 %), headache (10.53%), fatigue (10.53%), Diarrhea (5.26%) and chest tightness (5.26%)</td>
<td>Multiple patchy ground glass opacities in bilateral subpleural areas.</td>
<td>Normal WBC, lymphocytes were decreased in (63.18%). Increased ratio of neutrophils (61.11%). High levels of alanine aminotransferase, aspartate aminotransferase, lactate dehydrogenase, γ-glutamyl transpeptidase and α-hydroxybutyric dehydrogenase</td>
<td>RT-PCR-throat sample/putum</td>
<td>Antiviral therapy lopinavir and ritonavir</td>
<td>NM [21]</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>69</td>
<td>42</td>
<td>Fever (87%), temperature reached 38.1-39.0°C, cough (55%), fatigue (42%), and myalgia (33%), less common symptoms were sputum production, oppression in the chest, dyspnea, diarrhea, and headache</td>
<td>Manifestations of ground glass density which get solidify after some days.</td>
<td>Decreased neutrophils (39%), lymphocytes (42%), &amp; eosinophils (72%), Zero number of eosinophils (42%), Increase in alanine amino-transferease (33%), increase in aspartate amino-transferase (28%), increase in lactate dehydrogenase (41%), increase in c-reactive protein (67%).</td>
<td>RT-PCR-throat sample</td>
<td>Most patients received antiviral therapy (98.5%) and antibiotic therapy (98.5%). Anti-fungal (11.9%), Corticosteroids (14.9%)</td>
<td>As of Feb 4, 2020, 26.9% patients had been discharged, and five patients had died (mortality rate 7.5%).</td>
<td>[22]</td>
</tr>
<tr>
<td>13</td>
<td>36</td>
<td>45</td>
<td>NM</td>
<td>NM</td>
<td>NM</td>
<td>RT-PCR nasopharyngeal swab</td>
<td>Hydroxychloroquine + Azithromycin</td>
<td>1 death reported</td>
<td>[23]</td>
</tr>
<tr>
<td>14</td>
<td>5</td>
<td>50</td>
<td>Fever (100%), cough (100%), shortness of breath (100%), nasal tampon (60%), pharyngalgia (60%), myalgia (40%), fatigue (40%), headache (40%), and expectoration (40%)</td>
<td>Ground-glass opacities (GGO), multifocal patchy consolidation.</td>
<td>Reduced lymphocytes (80%), Increased aspartate amino-transferease (40%), alanine amino-transferease (40%) C-reactive protein (80%) and procalcitonin(40%)</td>
<td>NM</td>
<td>Antiviral (with oseltamivir), oxygen inhalation, and antibiotics. Three patients were treated with glucocorticoids</td>
<td>Fortunately, all patients did not need ICU care and were discharged from hospital without death</td>
<td>[24]</td>
</tr>
<tr>
<td>15</td>
<td>138</td>
<td>56</td>
<td>Fever (98.6%), fatigue (69.6%), dry cough (59.4%), dyspnea (31.2%), anorexia (39.9%), myalgia (34.8%), expectoration (26.8 %), pharyngalgia (17.4 %)</td>
<td>Bilateral patchy shadows or ground glass opacity in the lungs of all patients.</td>
<td>Marked lymphopenia while non-survivors have more severe lymphopenia WBC, neutrophil and level of D-dimer were higher in non-survivors. Levels of blood urea and creatinine were higher,</td>
<td>RT-PCR-respiratory tract</td>
<td>Most patients received antiviral therapy (oseltamivir, 89.9%) and many received antibacterial therapy such as moxifloxacin (64.4 %), ceftriaxone, 24.6 %, azithromycin, (18.1 %), glucocorticoid therapy (49.3 %) and oxygen inhalation (76 %)</td>
<td>34.1% were discharged, 6 died (4.3%), and 61.6% remain hospitalized</td>
<td>[25]</td>
</tr>
<tr>
<td>16</td>
<td>137</td>
<td>57</td>
<td>Fever (81.8%), cough (48.2%), muscular pain/ fatigue (32.1%), with less typical symptoms like low frequency, including heart palpitations, headache and diarrhea.</td>
<td>Lesions in multiple lung lobes, ground glass opacity co existed with consolidations and cord-like shadows</td>
<td>Normal or decreased WBC count (80%). Lymphopenopnia (72.3%).</td>
<td>NM</td>
<td>Symptomatic and respiratory support treatment. Immuno-globulin G is prescribed for critical patients. Early respiratory supports assist disease recovery and improved prognosis.</td>
<td>NM [26]</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>41</td>
<td>49</td>
<td>Fever (98%), cough (76%), myalgia/fatigue (48%), dyspnea (55% mean time 8 days), less common symptoms were sputum production (28%), headache (8%), haemoptysis (5%) and diarrhoea (3%).</td>
<td>ICU patient: bilateral multiple lobular and consolidation at sub-segmental areas. Non-ICU patient: bilateral ground glass opacity and subsegmental areas of consolidation</td>
<td>ICU patient: Increased prothrombin time and D-dimer level. Increased Levels of aspartate and amino transferase (37%), leucopenia (25%), lymphopenia (63%)</td>
<td>RT-PCR-lower respiratory tract sample</td>
<td>Antiviral, (93 %), antibiotics (100 %) and corticosteroid (22%)</td>
<td>Hospitalization (17 %), discharge (68 %) and death (15 %)</td>
<td>[27]</td>
</tr>
</tbody>
</table>

(Table 1) contd....
after a week from the onset of illness, which involves severe dyspnea and chest pain. Thus the infectivity during the incubation period is the biggest challenge in controlling COVID-19 [29, 30]. Home quarantine, isolation hand hygiene, respiratory etiquette and social distancing are the ways to control the transmission of the virus and subsequent pandemic situations.

4. COVID-19: BIOCHEMISTRY AND CHEST RADIOGRAPHY FEATURES

Biochemistry and chest radiography features are very important for the early diagnostic [11-28] (Table 1, entries 1-18). Kim et al., [11] reported slight changes in laboratory tests with thrombocytopenia, leukopenia, and increment of liver enzyme. Moreover, chest radiography displayed no infiltrations whereas; high-resolution CT on the fourth day of sickness showed multiple ground-glass opacities in both subpleural places [11]. Lim et al., [13] also reported small consolidation (right upper lobe) and ground-glass opacities (both lower lobes). Li et al., [14] mentioned that novel corona virus pneumonia patients have lesser lymphocytes, monocytes level and elevated C-reactive protein levels and anomalous CT images of the chest [14]. Wang et al., [22] reported a decrease in neutrophils (39%), a decrease in lymphocytes (42%) and decrease in eosinophils (72%). Surprisingly, eosinophil’s count found to be zero in 46 % patients [22]. Furthermore, lab finding displayed elevation in alanine aminotransferase (33%) and aspartate aminotransferase (28%). Besides this, inflammation indicators displayed rise of lactate dehydrogenase (41%), C-reactive protein (67%) and erythrocyte sedimentation rate (52%). Liu et al., [28] reported that, level of the Angiotensin II in plasma was notably raised and it is linearly allied with the viral load and lung injury.

In conclusion, abnormal chest CT is observed in COVID-19 patients, mostly ground glass opacities are detected (depending on the severity of infection) which are get solidified (consolidate) after some days and cause trouble in breathing. Lymphopenia is most commonly observed in COVID-19 patient that leads to affect or inhibit the body’s defence mechanism. Improvement in the lymphocytes, normalization of aspartate and amino transferase level in COVID-19 patient demonstrated faster recovery and better therapeutics outcome. Thus, affected immunity hinders the recovery of the patient. Liver injury/damage and neutrophilia are found to be common in the severe COVID-19 case. Consequently, the aspartate aminotransferase, lactate dehydrogenase, alanine aminotransferase, α-hydroxybutyric dehydrogenase and γ-glutamyl transpeptidase are considered as markers for the evaluation of liver in COVID-19 cases and severity. Excessive neutrophil count accounted for acute lung damage. In severe cases, abnormal neutrophil count (higher), blood urea (higher), D-dimer (higher), and creatinine levels (higher) were observed and can be linked to fatalities. The viral load of COVID-19 patients predicts the severity of patients related to lung disease. Thus, in short, viral load, the extent of lung injury, CBC count, liver and renal indicators may act as predictors for the COVID-19 disease severity.

5. COVID-19 ANTIVIRAL CHEMICAL DRUG TREATMENT AND CLINICAL OUTCOME

No appropriate therapy is available to treat COVID-19. Various drugs are used by in vitro analysis study (Table 1, entry 1-18) [11-28]. Kim et al., [11] and Lim et al., [13] mentioned the use of lopinavir/ritonavir which showed a decrease in virus load. Chen et al., [15] gave combination therapy, which includes the use of antibiotics, antifungals, antivirals, glucocorticoids, and intravenous immunoglobulins. Antiviral drugs include oseltamivir, ganciclovir, lopinavir/ritonavir (median 3 days) while antibiotic drugs include cephalosporins, quinolones, carbapenems, tigecycline and linezolid for the duration of 3-17 days (median 5 days). Their reported clinical outcome involves hospitalization, discharge and death of 57%, 31% and 11 % patient, respectively, from the population of 99 admitted cases [15]. Wang et al., [22] reported the use of arbidol in treatment which could improve the discharging rate and decrease the mortality rate, with a notable observation that all deaths occurred in the arbidol-untreated group [22]. Gautret et al., [23] observed that viral load gets reduced by the use of hydroxychloroquine treatment and its impact is reinforced by the use of azithromycin [23]. Their clinical outcome involved the death of one patient in a population of 36 patients. Liu et al., [28] reported the use of anti-viral (ribavirin and interferon) treatments. Furthermore, they suggested diagnosis biomarkers and use of angiotensin receptor blocking agents for repurposing treatment of COVID-19.

In conclusion, various kinds of drug trials are reported in the literature to treat COVID-19, which involves the use of antimicrobial chemical drug agents (antiviral treatment, antibiotics including anti-fungal), interferon, corticosteroids, oxygen therapy. It was seen that a combination of two or more therapies/drugs are more effective and essential for faster recovery. However, the use of any therapy and dosage
is varied from patient to patient and their health condition. Before the administration of the drugs, it is advisable to consider the history of the patient. In most of the cases oxygen therapy is found to be compulsory since COVID-19 causes damage to the respiratory system. Thus there is an urgent need for appropriate therapeutics guideline and effective drug for the treatment of COVID-19. The lethal nature of COVID-19 produces anxiety among the people as well as the health sector. The increasing numbers of COVID-19 patients create a worldwide emergency, instability and critical situation. However, the dedicated efforts of the medical doctors/practitioners/health workers/medical researchers are appreciable to manage and to control the pandemic COVID-19.

6. COVID-19: AGE, GENDER, AND COMORBIDITY FACTOR

Age, gender and comorbidities are found to be important factors in COVID-19 (Fig. 2A and 2B). Li et al., [14] suggested that higher risk and poor recovery is associated with older age citizens [14]. Chen et al., [15] mentioned that, smoking habit, older age with obesity and comorbidity may increase the rate of mortality. Wang et al., [22] reported that the median age for SpO$_2$ < 90% is 70 years and all five patients who deceased in the reported case study had SpO$_2$ < 90% [22]. The reason behind this may be the weak immunity with older age and comorbidities associated with them such as hypertension, diabetics, cardiovascular disease, tumor, obesity, cancer, chronic liver or renal injury [14, 15, 22]. Liu et al., [26] also found that patients treated in ICU with comorbidities were of older age; most of them had dyspnea and anorexia. Fig. (2A) shows global COVID-19 death rate (%) of patients with pre-existing comorbidities. Thus age could be the factor for severity in COVID-19. More recent survey showed that (Fig. 2B), the death rate is 14.8%, 8%, 3.6% and 1.3% for 80+ years, 70-79 years, 60-69 years and 50-59 years old age respectively. The recent survey showed that, the fatality rate in males and females is 4.7 % and 2.8 % respectively [8]. Chen et al., [15] proposed that, female candidates are less susceptible to the COVID-19 infection since females have more innate and adaptable immunity from the X-chromosome and sex-hormones. A similar kind of observation is also noted by Li et al. [14].

7. COVID-19 AND PEDIATRIC PATIENT

Very few research articles reported the pediatric clinical cases (Table 2, entries 1-6) [31-36]. Sun et al., [36] reported polyneu in all children in their case study. Most common features of the CT study involve ground-glass opacities, consolidation with surrounding halo-sign, lung auscultation, multiple patchy shadows whereas, less common observations are fine mesh shadow, tiny nodules, pleural effusion and white lung like change. Qui et al., [31] reported increased creatine kinase MB (31%), reduced lymphocytes (31%), leucopenia (19%), elevated procalcitonin (17%), elevated D-dimer, and creatine level. Similar kinds of biochemistry results are also reported by Xia et al., [34] and Sun et al., [36]. Besides this, Sun et al., [34] mentioned drop of CD16 + CD56 (50%), increased CD3 (26%), CD4 (50%) and CD8 (14%), IL-6 (14%), IL-10 (62%) and IFN-γ (26%).

Qiu et al., [31] reported recovery of all pediatric patients by treatment of the use of interferon alfa by aerosolization, lopinavir-ritonavir and oxygen inhalation. Xia et al., [34] reported co-infection (mycoplasma, influenza A/B, respiratory syncytial virus, cytomegalovirus) along with COVID-19. Sun et al., [36] reported recovery of all pediatrics patients by the use of antiviral treatments (virazole, oseltamivir) (100%), high-flow oxygen therapy (75%), invasive ventilation (25%), other therapies such as antibiotic therapy, traditional chinese medicine, intravenous glucocorticoid, immunoglobulin therapies are also used as per patient’s condition and response.

![Fig. (2). (A): Global COVID-19 death rate for pre-existing comorbidity date 1$^{st}$ April 2020 (11.00 PM) Indian Standard Time. (B): Global COVID-19 death rate for given age range by date 1$^{st}$ April 2020 (11.00 PM) Indian Standard Time. (A higher resolution/colour version of this figure is available in the electronic copy of the article).](image-url)
higher level of antioxidants, which boost their immunity [33]. However in pediatric patients the occurrence of leukopenia, lymphopenia and increment of myocardial enzymes is observed in COVID-19 [31-34]. Remarkably, an increase of procalcitonin elevation in pediatric patients may act as a marker to identify infection which is not common in adults [36]. The lung injury is more manifest in critical patients which are linked with a cytokine storm. Besides antiviral treatment, routine anti-bacterial treatment is also recommended to avoid the co-infection in children [37-40]. Finally, children have distinct immune responses than adults and hence they may show clinical features and therapeutic responses that need to be studied with more detailed clinical cases [32].

8. COVID-19: PREGNANCY AND NEONATES

Very few research articles reported the pregnancy and neonatal cases (Table 3, entries 1-8) [41-48]. Wang et al., [41] and Chen et al., [42] mentioned no possible intrauterine fetal infections, since testing of amniotic fluid, cord, and neonatal throat swab were seen to be negative for SARS-CoV-2. Yu et al., [43] found that, COVID-19 infected pregnant women had similar kinds of symptoms and clinical features to non-pregnant COVID-19 infected adult patients. Furthermore, they reported that breast milk, cord blood and placenta samples are free from COVID-19 [43]. Their clinical outcome has no fetal or neonatal death or neonatal asphyxia [41,43]. However, Yu et al., [43] represented a case of COVID-19 infection in one neonate (out of 7) after 36 hour. Hence, they proposed further investigation to confirm mother-to-child vertical transmission; conversely, they concluded the possible infection by physical contact [43]. Dong et al., [45] reported the birth of neonate (from COVID-19 infected mother) with increased antibody (IgG and IgM) levels and anomalous cytokine results after two hours of delivery [45]. The increased antibody level did not rule out the chances of COVID-19 in the neonate. However, no COVID-19 infection was found in swab samples of neonate, which indicated that the presence of passive antibodies in the neonate is a result of a defensive mechanism. Wang et al., [48] mentioned a case of COVID-19 infected pregnant woman who delivered (preterm) a baby without COVID-19 infection and was kept in isolation and given formula instead of mother’s milk. At the time of delivery, amniotic fluid, umbilical cord, placenta, throat swabs of neonate were tested, which are found negative for SARS-CoV-2 [48].
Table 3. COVID-19 in pregnant women: Mother and neonate characteristic.

<table>
<thead>
<tr>
<th></th>
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<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1</td>
<td>34</td>
<td>Suspect of COVID-19</td>
<td>RT-PCT of the pharyngeal swab for SARS-CoV-2 was positive after 36 hours of birth. Chest CT on the fourth day showed high-density nodular shadow under the pleura of the posterior segment of the upper lobe.</td>
<td>Negative test for SARS-CoV-2 of cord blood, breast milk sample and placenta specimens. The possibility of mother-to-child transmission of SARS-CoV-2 cannot be ruled out.</td>
<td>Baby recovered and discharged</td>
<td>[41]</td>
</tr>
<tr>
<td>2</td>
<td>9</td>
<td>30</td>
<td>Confirm COVID-19</td>
<td>Six neonates were tested for the SARS-CoV-2 test, which was found to be negative</td>
<td>Sample of amniotic fluid, cord blood, neonate throat swab, and breast milk were negative for SARS-CoV-2. Pneumonia in pregnant women with COVID-19 was similar to normal non-pregnant adult patients with COVID-19.</td>
<td>No fetal death, neonatal death, or neonatal asphyxia.</td>
<td>[42]</td>
</tr>
<tr>
<td>3</td>
<td>7</td>
<td>32</td>
<td>Confirm COVID-19</td>
<td>1 neonatal confirmed with COVID-19 after 36 h of birth</td>
<td>The pregnant patient’s symptoms with COVID-19 was similar to a non-pregnant adult patient with COVID-19.</td>
<td>Outcomes appear very good as patients are discharged</td>
<td>[43]</td>
</tr>
<tr>
<td>4</td>
<td>11</td>
<td>32</td>
<td>Confirm COVID-19, antiviral therapy given only after delivery</td>
<td>No COVID-19 infection was found in neonates.</td>
<td>Inflammation and liver injury of neonate support the possibility of vertical transmission. Further, newborn have elevated IgM antibodies possibly from mother, however, surprisingly, COVID-19 was found to be negative.</td>
<td>Neonatal and mother discharged</td>
<td>[44]</td>
</tr>
<tr>
<td>5</td>
<td>1</td>
<td>29</td>
<td>Confirm COVID-19</td>
<td>The infant exposed to mother with COVID-19 (from diagnosis to delivery). No COVID-19 infection was found in the neonate. (2 hours to 16 days).</td>
<td>Pregnancy a well as delivery, did not exacerbate the severity of COVID-19. The impact of antiviral therapy is necessary to assess for pregnant women with COVID-19 due to possible potential hazard/danger linked with the fetus.</td>
<td>Neonatal mother discharged</td>
<td>[45]</td>
</tr>
<tr>
<td>6</td>
<td>9</td>
<td>30</td>
<td>Confirm COVID-19, antiviral treatment not given before delivery</td>
<td>No evidence of vertical transmission COVID-19 and no antiviral treatment was given to neonates. NAT was negative for neonates.</td>
<td>Pregnant women are at a high risk of COVID-19 infection due to the immune suppressive stage</td>
<td>5 neonates discharged, 1 death reported and 4 neonates were hospitalized in stable condition. (twin case)</td>
<td>[46]</td>
</tr>
<tr>
<td>7</td>
<td>2</td>
<td>31.5</td>
<td>Confirm COVID-19</td>
<td>Negative PCR test in both neonates</td>
<td>Mature serum, cord blood, placenta tissue, amniotic fluid, vaginal swab, breast milk, and newborn’s nasopharyngeal swab were collected at or after delivery. All are negative for SARS-CoV-2</td>
<td>Both mothers and newborns had excellent outcomes</td>
<td>[47]</td>
</tr>
<tr>
<td>8</td>
<td>1</td>
<td>28</td>
<td>Confirm COVID-19</td>
<td>Negative PCR test for throat swab and stool sample.</td>
<td>Neonate was kept in isolation ICU of neonatal nursery without any contact with the mother after birth. The newborn was given formula instead of mother’s milk.</td>
<td>NM</td>
<td>[48]</td>
</tr>
</tbody>
</table>

NAT: Nucleic acid amplification test.

In conclusion, according to the literature survey, COVID-19 infected pregnant women displayed similar symptoms like non-pregnant adults with COVID-19. Very limited clinical studies are available for infection cases of COVID-19 in the neonate. Vertical transmission of COVID-19 through placenta is not observed in the reported literature; however, it requires strong evidence and further studies. Hence at present, the possibility of vertical transmission cannot be ruled out. Most studies reported the absence of the SARS-CoV-2 virus in various delivery products [41-49]. Further, continuous follow-up observation of pregnant women is essential to avoid complications during delivery since pregnant women are highly vulnerable to the respiratory pathogens due to their immunosuppressive state and physiological adaptive variations in the body such as the elevated diaphragm, high oxygen requirement, and higher Hb requirement. Isolation of neonate from COVID-19 infected mother is essential. Further, all medical practitioners and health care workers associated with pregnancy management may become asymptomatic carriers and need prophylaxis.

9. POSSIBLE POTENTIAL THERAPEUTIC APPROACHES FOR COVID-19

The list of various drugs presented in Table 4 shows possible potential use in the treatment of COVID-19 (Table 4, entries 1-12) [50-69]. Some of the drugs have been tested in various case studies, while some are under clinical trial study. Chloroquine or hydroxychloroquine are anti-malarial drugs that display potential in the treatment of COVID-19 and utilized by Gautret et al. [23]. These drugs increase an endosomal pH, also interfere with the cellular receptors glycosylation (ACE-2) process [51, 52]. Remdesivir is a nucleotide analogue that may obstruct nucleotide synthesis, which inhibits the replication of the virus and reduces the viral load [53-57]. Lopinavir and ritonavir were used in combination...
Table 4. Possible potential antiviral drugs to treat COVID-19.

<table>
<thead>
<tr>
<th>Entry</th>
<th>Drug</th>
<th>Remarks for the Mode of Action</th>
<th>Clinical Case Study Analysis Ref.</th>
<th>Refs. for Mode of Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Chloroquine</td>
<td>Increases endosomal pH needed for virus/cell fusion, also interfere with cellular receptors glycosylation.</td>
<td>51</td>
<td>[50-52]</td>
</tr>
<tr>
<td>2</td>
<td>Hydroxy-chloroquine</td>
<td>Interfere with ACE2 glycosylation and inhibits entry of virus with endolysosomes.</td>
<td>23</td>
<td>[52]</td>
</tr>
<tr>
<td>3</td>
<td>Remdesivir</td>
<td>A nucleotide analogue that may obstruct nucleotide synthesis, which inhibits replication of the virus and reduces the viral load. Proposed based on in vitro analysis</td>
<td>[50, 53-57]</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Lopinavir</td>
<td>Acts as protease inhibitors which hamper viral proteases (3CLpro or PLpro), also in vitro studies it displayed anti-SARS property.</td>
<td>13,15,20,21,31</td>
<td>[54-60]</td>
</tr>
<tr>
<td>5</td>
<td>Ritonavir</td>
<td>Acts as a protease inhibitor and interferes with replication of the virus.</td>
<td>13,15,20,21,31</td>
<td>[54-56, 60]</td>
</tr>
<tr>
<td>6</td>
<td>Ganciclovir</td>
<td>A nucleoside analogue which inhibits replication of the virus</td>
<td>15</td>
<td>[59]</td>
</tr>
<tr>
<td>7</td>
<td>Ribavirin</td>
<td>Interfere RNA metabolism needed for replication of the virus</td>
<td>28</td>
<td>[61]</td>
</tr>
<tr>
<td>8</td>
<td>Arbidol (Umifenovir)</td>
<td>Prevent viral entry at the host cell by distracting the binding of the viral coated protein to the host cell.</td>
<td>20</td>
<td>[62, 63]</td>
</tr>
<tr>
<td>9</td>
<td>Darunavir</td>
<td>Prevent viral replication and acted as a protease inhibitor</td>
<td>Proposed</td>
<td>[62]</td>
</tr>
<tr>
<td>10</td>
<td>Oseltamivir</td>
<td>Inhibits the neuraminidase enzyme and further movement of the virus from the infected host cell of the respiratory tract.</td>
<td>15,20,25,36</td>
<td>[64]</td>
</tr>
<tr>
<td>11</td>
<td>Favipiravir</td>
<td>Attacks viral genetic material and inhibits RNA polymerase enzyme</td>
<td>Proposed</td>
<td>[65, 66]</td>
</tr>
<tr>
<td>12</td>
<td>IFN-alpha</td>
<td>Restrict the viral replication in vitro.</td>
<td>28,31</td>
<td>[54, 60, 61, 67]</td>
</tr>
<tr>
<td>13</td>
<td>Ikarugamycin, molsidomine</td>
<td>Proposed based on the connectivity map and ACE-2 function.</td>
<td>Awaited</td>
<td>[68]</td>
</tr>
<tr>
<td>14</td>
<td>Losartan</td>
<td>Exerts an inhibitory effect on ACE-2 and protects lung damage.</td>
<td>Awaited</td>
<td>[69]</td>
</tr>
</tbody>
</table>

for the treatment of COVID-19 by Lim et al., [13], Chen et al., [15] and Qiu et al., [31]. These drugs acted as protease inhibitors that hamper viral proteases (3CLpro or PLpro), moreover, in vitro studies, it displayed anti-SARS property. Nucleoside ganciclovir is used by Chen et al., [15] along with lopinavir and ritonavir. Ribavirin is a ribonucleic analogue that interferes with RNA metabolism needed for the replication of virus [61], it is used in the treatment of COVID-19 by Liu et al., [28]. Arbidol is an indole derivative with several active sites has been successfully used by Chen et al., [20] in the treatment of COVID-19, which prevents viral entry at host cell by distracting the binding of the viral coated protein to host cell [62, 63]. Darunavir prevents viral replication and acts as a protease inhibitor [62]. Oseltamivir is a cyclohexene-carboxylate ester with active amine and amide side arms which show inhibition of neuraminidase enzyme and further movement of virus from infected host cells of the respiratory tract [64]. Favipiravir is a pyrazine carboxamide derivative which attacks viral genetic material and inhibits RNA polymerase enzyme [65, 66]. IFN-alpha is immune interferon which is supposed to restrict the viral replication [54, 60, 61, 67]. Ikarugamycin, molsidomine and losartan are the drugs that show the inhibitory effect with ACE-2 receptor in vitro analysis [68, 69] however, their results for clinical case studies are awaited which may find scope in the treatment of COVID-19. These all above-mentioned drugs may be used in combination such as (remdesivir+ chloroquine) [50], (lopinavir + ritonavir) [13, 15, 20, 21, 31], (arbidol + lopinavir + ritonavir) [20] (oseltamivir + lopinavir + ritonavir) [15, 20] in order to have pronounced effect. Further, most of the above-mentioned drugs are reported to be used along with antibiotics (to avoid co-infection), glucosteroids, and herbal medicines for effective treatment of COVID-19.

In conclusion, no specific antiviral chemical drug(s) has been available/ identified/ confirmed/ approved for the treatment of COVID-19. At present existing available antiviral drugs are used based on clinical case studies or in vitro evidences or computational modelling studies. In most of literature cases combination therapy (antiviral + antibiotics) [21, 24, 25, 36], (two antivirals) [13, 15, 20, 25], (antiviral + glucosteroids) [8, 36], (antiviral + antibiotics + herbal medicines) [8, 36], (antiviral + symptomatic treatment) [8, 15, 20, 36] is applied (depends on patients situation) in order to have fast, speedy recovery and victory over COVID-19.
10. USE OF HYDROXYCHLOROQUINE AS PROPHYLAXIS

At present, some reports recommended prophylactic use of hydroxychloroquine to prevent likely infection of COVID-19 on the basis of in-vitro analysis [70, 71]. There are two reasons which promote the use of hydroxychloroquine in the treatment of COVID-19 (i) cytokine storm in the COVID-19 infection can be modulated by immunomodulatory effect of hydroxychloroquine [72-74], further, (ii) hydroxychloroquine alter the pH of cell-organelles (endosome, Golgi vesicles, lysosomes) which hamper the virus fusion process (replication, glycosylation of viral proteins, endocytosis, virion transport and virion release) by affecting enzymatic hydrolysis process [72]. Gautret and the research group [23] have confirmed the effective use of the hydroxychloroquine to treat COVID-19 [23]. However, at the same time Molina et al., [75] reported no clinical benefit by the use of hydroxychloroquine for COVID-19 [75]. Thus it may be possible that, hydroxychloroquine may be effective in the early phase of the disease and hence recommended to use as prophylaxis [70-72]. Various clinical trials are ongoing and hence confirmatory results are still awaiting the use of hydroxychloroquine as prophylaxis and in treatment of COVID-19 [76]. It is noteworthy to mention that random use of the hydroxychloroquine may result in detrimental side effects [72], hence prophylactic use of the hydroxychloroquine should be done only after medical advice.

11. PERSONAL PROTECTIVE EQUIPMENT (PPE) KITS FOR HEALTHCARE WORKERS

SARS-CoV-2 is highly contagious in nature, which can be very easily transmitted by the direct-indirect contact transmission (surface, utensils, patient’s contaminated room items) and via airborne droplets (talking, laughing, coughing, or sneezing) [77-85]. Healthcare-professionals are a highly susceptible population for the infection due to the close proximity of COVID-19 patients [77-80]. Hence, use of the personal protective equipment kit is of high priority and of great importance to avoid the risk of infection to physicians, nurses, and ward-workers during the treatment of COVID-19 patients [81-85]. Personal protective equipment involves the face mask (N95, surgical), face shield, goggles, isolation gown/ medical suit/ apron, gloves, hair protection/ cap and shoe/ sole cover etc. [77-80]. Lau et al., [83] reported the transmission of coronavirus disease in most of the healthcare professionals who were practicing without the use of personal protective equipment kit in high risk contaminated ward [83]. More recently, Liu et al., [84] studied impact of the use of personal protective equipment among 420 healthcare professionals. Their study reported that, during the deployment period, no specific COVID-19 symptoms are observed (among all 420 healthcare professionals) who used personal protective equipment in high-risk areas [84]. Further, all those 420 healthcare professionals did not show the development of any protective immunity against COVID-19 or viral-flu disease [84]. Thus, it is recommended that healthcare professionals should give self-safety priority by using personal protective equipment.

In conclusion, the safety of the healthcare professionals should be on high priority and use of personal protective equipment is MUST for healthcare professionals who are giving divine service in this critical period of pandemic. Correct use of the PPE offers protection or reduces the possible chances of infection by SARS-CoV-2. The Centre for Disease Control and Prevention provides a guideline and the use of personal protective equipment [85]. Further, training of the correct use of PPE (appropriate sequence of putting on and taking off) is essential to avoid the possible transmission of the SARS-CoV-2 virus.

12. POSSIBILITY OF RE-INFECTION OF COVID-19

A chance and possibility of re-infection is the major question about the COVID-19 [86-90]. The pathogenicity of SARS-CoV-2 is very close to that of SARS-CoV. Further, genome sequencing showed almost 80% similarity and hence scientific reports about the re-infection of SARS-CoV are very important [72]. Cao et al., [87] found the presence of IgG and neutralizing antibodies in 74% and 84% recovered patients respectively, after 3 years [87]. Similarly Wu et al., [88] also mentioned the presence of SARS-CoV antibodies in almost 90 % after 2 years [88]. Further they concluded that SARS-CoV antibodies were reduced after 3 years and chances of re-infection may be possible after 3 years [88]. Recently, Kirkcardly et al., [89] reported a view point wherein they confirmed no case of re-infection of COVID-19 (till publication date) may be due to post-infection immunity [89]. More recently, Bao et al., [90] reported that re-infection and replication of SARS-CoV-2 virus do not appear in rhesus macaques, which are re-infected (at 28th day) after complete recovery from the COVID-19 [90].

Thus, in our opinion, limited literature is available about the study of re-infection and antibody responses to SARS-CoV-2 viral infection. The available past SARS-CoV illness evidences suggest that chances of re-infection are very less, since recovery from COVID-19 infection may offer post-infection immunity at least for 2 years. However, the possibility of re-infection (in lesser time duration) cannot be ruled out in patients having comorbidity and lower immunological response, which can be well judged by cellular immune response assay.

13. POSSIBILITY OF SECOND-WAVE OF COVID-19

The possibility of the appearance of a second-wave outbreak is the major concern about COVID-19 [91-94]. Limited research data is available for the prediction and revisiting of COVID-19 second wave [91-94]. The SARS history stated that, (i) SARS in the years 2003-04 was revisited in the form of the second wave in Toronto and Canada after relaxation in civil restriction/regulations [95, 96] (ii) while the form of the second wave in Toronto and Canada was split-up by Influenza A epidemic [97]. Thus the previous historical evidence about coronavirus outbreak suggested that, quick relaxation of infection controlling management parameters may call up possible second wave outbreak. Ali [91] proposed that SARS-CoV-2 infectivity will remain active at least for two years and progress throughout the world again in successive wave form [91]. Further, various experts from the health sector and CDC officials warn about the revisiting of another wave of COVID-19 [91-94, 98]. The possible second wave of the outbreak
may put a noteworthy strain on healthcare professionals [99]. Hence, Government officials must ensure the availability of human resources in the health care sector and medical facilities to minimize the risk of a second-wave outbreak.

In our opinion, lock-down, social distancing, personal hygiene, the use of PPE and quarantine phases are able to control somehow the first wave (at some extent), whereas very quick relaxation in above these controlling parameters may call for the potential second wave which may be possibly even worse than the first wave. Further government needs to be ready with the must policy, planning and management to control/avoid the possible predicted loss in the successive reappearance of the COVID-19 wave. More specifically, the health care sector needs to be ready with appropriate planning, human resource, primary medical facilities, medicine stockpile, ventilators, and other essential principle medical needs for the future possible revisiting outbreak.

14. COVID-19: PREVENTION AND CHALLENGES

The biggest danger of COVID-19 is its potential for rapid exponential rise due to community transfer, which can be controlled by means of social distancing. To achieve social distancing in densely populated places city, lock-down helps. Isolation and quarantine of patients or suspects at least for 2-3 weeks, along with early detection, is key to break the chain. The mean incubation period of SARS-CoV-2 is 2-14 days. The rate of transmission is very high within a short time span to understand and to control. Further, asymptomatic carriers also possess enough viral load to spread the disease without showing any noteworthy symptoms. SARS-CoV-2 virus can be spread via droplets and aerosols; hence personal hygiene, hand hygiene, respiratory etiquette, the use of masks can be used to control the infection of COVID-19.

The major limitations and challenges associated with the COVID-19 therapeutics are insufficient scientific data/reports regarding drugs, vitro analysis and case studies. Many case study reports are in the Chinese language and hence difficult to translate. Furthermore due to busy schedules, medical practitioners are unable to communicate case studies. COVID-19 rapid testing centre, the number of testing centres and availability of testing kits are also limited considering the rate of rapid transmission. Moreover, potential vaccine development in a short period of time is a challenging task. Besides this, there is a threat of the possibility of second-wave, which could be worse than the first one. Limited literature is available about the post-infection immunity data of SARS disease and hence the probability of re-infection is a major unanswered question. Thus the controlling of the pandemic situation is itself a big challenge at present, which creates worldwide anxiety and fear about COVID-19.

CONCLUSION

COVID-19 shows symptoms similar to that of pneumonia or common flu (fever, fatigue, dry cough, myalgia, sore throat, dyspnea and chest pain) which becomes more severe after a week from the onset of sickness. The abnormal chest CT is observed in COVID-19 patients with ground glass opacities which are consolidated after some days and cause trouble in breathing. The extent of lung injury, CBC count, liver and renal indicators may act as predictors for the COVID-19 disease severity. Patients with pre-existing comorbidities are more susceptible to COVID-19. Pediatric patients found to be less susceptible than adults for pneumonia in COVID-19, however, the severity of COVID-19 cannot be excluded in pediatric patients. Further, an increase of procalcitonin elevation in pediatric patients may act as a marker to identify infection which is not common in adults. COVID-19 infected pregnant women displayed similar symptoms like non-pregnant adults infected with COVID-19. Vertical transmission of COVID-19 is not observed; however, the possibility of vertical transmission cannot be ruled out which requires further studies. Various kinds of therapies can be used, which involves the use of antimicrobial agents (antiviral treatment, antibiotics including anti-fungal), interferon, corticosteroids, oxygen therapy. The most commonly used antiviral drugs include oseltamivir, lopinavir, ritonavir, remdesivir, arbidol, chloroquine and hydroxychloroquine. Combination of two or more therapies/drugs is found to be more effective and essential for faster recovery. Thus, the present review provides significant information about epidemiology, clinical features, radiographic characteristics and possible potential chemical drugs based on available case studies and reports. Further, this review also highlights various available antiviral medications to control/manage COVID-19. Besides this, the present review also discussed prophylaxis strategy, possibility of re-infection and second wave. This review may be helpful to medical practitioners, health workers, clinical researchers and decision making government authorities to manage this global public health emergency.

NOTE FOR READERS

Readers must refer to the cited research article(s) for more detailed information. Clinical treatment should NOT be given solely based on this review article.

CONSENT FOR PUBLICATION

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CONFLICT OF INTEREST

The authors declare no conflict of interest, financial or otherwise.

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