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An Introduction of Corona Virus along with its Transmission & Managements

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Abstract: ***Corona virus is most notorious virus in the world. Now we are facing a huge problem of corona virus disease. No one knows the proper character of corona virus which is necessary to develop the vaccine and drugs against the virus. So that I am focusing here the nature, types of the virus as well as mode of transmission and pharmacological and emergency management of the severe pandemic viral disease. My main moto is to focus on a details about corona virus and it's management within few papers. I hope it is helpful to all scientists, doctors to progress the development of drug and vaccine against corona virus.***

Keywords: Pharmacological Management, Mode of Transmission, Types of Virus, Lesions from SARS MARS

1. Introduction

Coronaviruses are pleomorphic, single-stranded RNA viruses that measure 100–160 nm in diameter. The name derives from the crownlike appearance produced by the club-shaped projections that stud the viral envelope. Recently, new human coronaviruses were identified as causes of Middle East Respiratory Syndrome (MERS) and severe acute respiratory syndrome (SARS). The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is the most recently identified human coronavirus.

Virion subtypes:
Coronaviruses were previously subtyped based on their protein components. These subtypes are recently defined by RNA genotyping, resulting in the four main subgroups of coronaviruses: alpha, beta, gamma, and delta. Alpha and beta coronaviruses are found in both humans and animals. Gamma and delta coronaviruses have only been identified in animals.

Human corona virus:
In 1960s, four types of human corona virus have been identified as common causes of human respiratory system illnesses such as the common cold.

1) 229E (alpha coronavirus)
2) NL63 (alpha coronavirus)
3) OC43 (beta coronavirus)
4) HKU1 (beta coronavirus

Increased genetic diversity and provides opportunities for viruses in animal reservoirs to emerge as novel human pathogens through genetic recombination in between the members of the same and different corona virus group. Such genetic recombination led to emergence of three newer human coronaviruses:

5) MERS-CoV (beta coronavirus) - middle east respiratory syndrome (MERS)
6) SARS-CoV (beta coronavirus) - severe acute respiratory syndrome (SARS)
7) SARS-CoV-2 (novel beta coronavirus) - coronavirus disease (COVID-19)

Virus entry into host cell: The coronavirus spike (S) protein contains two major sections that are cleaved apart during host cell protease priming:

a) S1 (attachment) includes a receptor binding domain
b) S2 (fusion) includes a transmembrane domain

Coronaviruses enter into the host cell through viral spike(S) protein binding to host angiotensin-converting enzyme 2 (ACE2), which is a membrane-bound aminopeptidase majorly present in the lungs and heart. Binding of the S1 unit of the viral S protein to the host ACE2 cellular receptor facilitates viral attachment to the surface of target host cells. Viral S protein priming then requires S protein cleavage of S1 from S2 (and at another S2' site) by the host cell serine protease TMPRSS2. The viral S2 subunit then drives fusion of the viral and host cell membranes.

Lesions from SARS and MARS

Spread of SARS and MERS between people has generally occurred between close contacts. Person-to-person spread is thought to have happened mainly via respiratory droplets (coughs, sneezes), although some spread may have occurred by touching contaminated objects then touching the mouth, nose, or eyes

SARS
Severe acute respiratory syndrome (SARS) is a respiratory illness caused by the SARS-CoV beta coronavirus, usually resulting in pneumonia. Symptoms include a high fever, headache, body aches, and possibly diarrhoea. There have been no known cases of SARS reported anywhere in the world since 2004.[1]

SARS was first reported in 2003, when it emerged in Asia. The illness spread by close person-to-person contact to countries in North America, South America, Europe, and greater Asia before the global outbreak was contained later in 2003. The virus is currently contained in research laboratory samples.

The World Health Organization recorded SARS as the cause of illness for 8,098 people worldwide during the outbreak. Of these, 774 (9.6%) died [2]. In the United States, only 8

people had laboratory evidence of SARS-CoV infection during the 2003 outbreak.

MERS

Middle East respiratory syndrome (MERS) is an illness caused by the MERS-CoV beta coronavirus. Symptoms include fever, cough, and shortness of breath. Pneumonia is common, but not always present. The spectrum of illness due to MERS-CoV infection is not fully defined. Although most reported cases have had severe acute lower respiratory illness, mild infections, and infections with no apparent symptoms, have been reported. Additionally, in some cases, diarrhoea preceded respiratory symptoms. Other early symptoms have included headache, chills, myalgia, nausea/vomiting, and diarrhoea.

Centers for Disease Control[3]

MERS was first reported in Jordan and Saudi Arabia in 2012. All cases of MERS have since been linked through travel to, or residence in, the Arabian Peninsula region. Spread is through close person-to-person contact. The World Health Organization has identified 2,484 laboratory-confirmed cases of MERS since 2012. With 858 attributed deaths, about 35% of people with MERS-CoV illness have died[3.]

Only two people in the U.S. have ever tested positive for MERS-CoV infection in 2014. Currently, MERS represents a very low risk to the general public in the U.S. However, MERS-CoV remains a threat in the Arabian Peninsula with the potential to spread further, causing more cases both globally and within the U.S.

Symptomatic contacts should be evaluated and, depending on their clinical history and presentation, considered for MERS-CoV testing. Testing includes rRT-PCR testing of respiratory and serum specimens. MERS-CoV serology should be considered if symptom onset was more than 14 days prior to evaluation.

Salient features

1) Spreading of SARS and MERS between people has generally occurred between the close contacts, mainly via respiratory droplets (coughs, sneezes).
2) Testing is performed based on a patient's clinical history and presentation, with rRT-PCR testing of respiratory and serum specimens.
3) An outbreak such as SARS can be fully contained through coordinated infection control efforts

Transmission of SARS-CoV 2 Infection:

Characterization of SARS-CoV-2 transmission is ongoing but initial data reveal both asymptomatic and symptomatic spread:

SARS-CoV-2 can spread from person-to-person through respiratory droplets in air or deposited on surfaces, and possibly by the fecal-oral route[17] Based on reports of early-onset infection and serology in newborns in Wuhan, China, vertical transmission cannot be ruled out. Detection of SARS–CoV-2 in sputum and feces has been reported in infected patients after their pharyngeal swabs became negative, but it is not known whether a positive RT-qPCR result in those samples indicates that a patient continues to pose a risk for infection to others[51]

SARS-CoV-2 can remain viable and infectious in aerosols for hours, and on surfaces up to days; the half-life of SARS-CoV-2 was approximately 1.1 hours in aerosols, 5.6 hours on stainless steel and 6.8 hours on plastic,4 hours on copper no viable virus was measured on cardboard after 24 hours, but virus was still detectable (depending on the inoculum shed) on plastic and stainless steel after 72 hours[23].

Asymptomatic transmission: It is likely that SARS-CoV-2 is transmitted by asymptomatic persons during the incubation period. High titers of virus have been found in the oropharynx early in the course of disease during the period of minimal symptoms[14]. Case histories and population monitoring of incubation period compared with serial interval demonstrate asymptomatic spread[16].

Incubation period: Analysis of confirmed COVID-19 cases have estimated the median incubation period to be 5.1 -5.2 days[18,19], and estimated that 97.5% of those who develop symptoms will do so within 11.5 days (CI, 8.2 to 15.6 days) of infection [18]. Clinical descriptions of asymptomatic phases after possible exposure range from 2 to 14 days. A 14 days period for monitoring after potential exposure is generally recommended, and modelling predicts that 101 out of every 10,000 cases (99th percentile, 482) will develop symptoms after 14 days of active monitoring or quarantine[18].

Serial interval: The serial interval (the time between successive cases in a chain of transmission) has been calculated as 4 to 4.6 days in some studies[15]. A small study of 10 confirmed cases estimated a serial interval distribution mean (±SD) of 7.5±3.4 days (95% CI, 5.3 to 19)[36]. The short serial interval compared with the described incubation period means asymptomatic spread is likely. The serial interval for SARS-CoV-2 is also likely shorter than the 2003 SARS mean calculated serial interval of 8.3 days[16].

Reproductive Number (R_0): The reproductive number is the number of cases, on average, an infected person will cause during their infectious period. Based on an early estimate of mean serial interval of 7.5 days as described for initial cases in Wuhan, China, R_0 was estimated to be 2.2 (95% CI, 1.4 to 3.9)[36]. The actual R_0 for SARS-CoV-2 remains uncertain but estimates generally range between 1.5 and 3.5[37].

Close Personal Contact Defined

The Centers for Disease Control (CDC) defines close contact as:

"a) being within approximately 6 feet (2 meters), or within the room or care area, of a novel coronavirus case for a prolonged period of time while not wearing recommended personal protective equipment or PPE (e.g., gowns, gloves, NIOSH-certified disposable N95 respirator, eye protection); close contact can include caring for, living with, visiting, or sharing a health care waiting area or room with a novel coronavirus case. (or)

b) having direct contact with infectious secretions of a novel coronavirus case (e.g., being coughed on) while not wearing recommended personal protective equipment."[7]

SARS-CoV-2 Infection
ACE2 has cardiovascular and immune system functional roles in healthy persons, and has a role in development of hypertension and diabetes mellitus[21]. SARS- CoV-2 mainly invades alveolar epithelial cells, resulting in the respiratory symptoms seen in Covid-19.

Because hypertension, heart disease, and chronic lung disease are risk factors for more severe disease in patients with Covid-19, questions have arisen about a link between disease severity and increased ACE2, including concerns about increases related to the use of renin–angiotensin–aldosterone system inhibitors[21]. For patients with Covid-19 and hypertension, the benefits and risks of continuing or changing ACEI/ARB/renin-acting agents is uncertain and controversial[21] (see Treatment section).

Transmission electron microscopic image of an isolate from the first U.S. case of COVID-19. The spherical viral particles, colorized blue, contain cross-sections through the viral genome, seen as black dots.

Clinical Features:

Symptoms
Infection with (SARS-CoV-2) may be asymptomatic, or may cause an acute respiratory disease. The acute respiratory disease may be mild to severe viral pneumonia. Full characterization of the spectrum of the novel coronavirus disease (COVID-19) is ongoing, but the primary presentation is an influenza-like illness with lower respiratory tract symptoms:[4]

1) **Fever** develops in 87-98% of Covid-19 patients[5]. However, fever may be absent on presentation; a study of 1,099 hospitalized COVID-19 patients in Wuhan, China reported that fever was present in only 44% at hospital admission, then increased to 89% during hospitalization[13]. Fever may be prolonged and intermittent[5].
2) **Cough** may be severe and is often non-productive; hemoptysis may occur.
3) **Shortness of breath** may be severe and may progress with development of pneumonia.

Additional symptoms include: **myalgias**, **fatigue**, **sore throat**, **nausea/vomiting**, **diarrhoea**, and **headache**[5, 13]. Some patients have experienced gastrointestinal symptoms such as diarrhea and nausea prior to developing fever and lower respiratory tract signs and symptoms[5].

Anosmia with resultant **dysgeusia** have been additionally reported[53], stated by American Academy of Otolaryngology–Head and Neck Surgery (AAO-HNS)

Physical Examination
Reports describe diverse findings, but common presentations include:

- **Vital signs**: fever, increased respiratory rate, and reduced oxygen saturation
- **Lungs**: relatively benign/quiet auscultation relative to dyspnea and hypoxia

Laboratory Findings
Routine laboratory testing is nonspecific, but the following are commonly reported:

- Lymphocytopenia[5, 13, 50]
- Thrombocytopenia[5,13]
- Liver transaminase elevations[5,13]
- C-reactive protein and ESR elevation[5,13, 50]
- Albumin decreased[50]
- LDH elevated [50]

TnT and myocardial injury: mortality has been found to be markedly higher in patients with elevated troponin T levels (TnT) levels than in patients with normal TnT levels (59.6% vs 8.9%).[55]

Chest Radiological Picture:
Imaging is nondiagnostic, but in persons with Covid-19 who have undergone imaging the characteristic reported findings are:

- Chest radiograph: bilateral ground glass opacities[13, 50]
- Chest CT: ground-glass opacity and bilateral patchy shadowing[13]

2. Clinical Course

The acute respiratory disease of Covid-19 may progress to bilateral **pneumonia**, acute respiratory distress syndrome (**ARDS**), or death. Diffuse alveolar damage has been identified on postmortem histopathology from the lungs of a patient with COVID-19 who had respiratory failure and radiographic bilateral ground-glass opacities[19].

Intensive care requirements: Reports describe the potential for clinical deterioration during the second week of illness, with roughly 25-30% of hospitalized patients requiring intensive support[10, 11]. ARDS developed in 17–29% of hospitalized patients in Wuhan, China[10,11] and median time between initial symptoms to onset of ARDS was reported in one study as 8 days[5,10]. In a series of 24 patients requiring intensive care in Seattle-area hospitals, all were admitted for hypoxemic respiratory failure, 18 needed mechanical ventilation, and 17 also had hypotension and needed vasopressors; 12 died between ICU day 1 and day 18, including 4 patients who had a DNR order on admission, and 7 remained hospitalized (3 still on mechanical ventilation) at the time of data cutoff[56].

Cardiac injury: Elevation of TnT is common among patients hospitalized with COVID-19, and is associated with higher risk of in-hospital mortality[54]. In one study, patients with underlying cardiovascular disease but without presenting myocardial injury had a relatively favorable prognosis[55].

Viral Diagnostic Testing: To test for SARS-CoV-2, specimen testing (nasopharyngeal or oropharyngeal aspirates or washes, swabs, broncheoalveolar lavage, tracheal aspirates, sputum, and serum) is performed using a real time reverse transcription PCR (rRT-PCR) assay for SARS-CoV-2

3. Collection of Respiratory Specimen

Lower Respiratory Specimen
Collect sputum, if productive (bronchoalveolar lavage or tracheal aspirate when appropriate).

To collect a sputum sample: have the patient rinse the mouth with water and then expectorate deep cough sputum directly into a sterile, leak-proof, screw-cap sputum collection cup or sterile dry container. Induction of sputum is not recommended if cough is not productive.

Upper Respiratory Specimen
Collect a nasopharyngeal swab if possible (or nasopharyngeal wash/aspirate when clinically appropriate). If unable to collect a nasopharyngeal aspirate, collect an oropharygeal swab. If both swabs are used, NP and OP specimens should be combined at collection into a single vial.

To collect nasopharyngeal or oropharyngeal swabs: Use only synthetic fiber swabs with plastic shafts. [Calcium alginate swabs and swabs with wooden shafts may contain substances that inactivate viruses and inhibit PCR testing.] Place the swab immediately into a sterile tube containing 2-3 ml of viral transport media. Label the tube with the site of origin. Nasopharyngeal swab: Insert a swab into the nostril parallel to the palate. Leave the swab in place for a few seconds to absorb secretions. [Swabbing the anterior nares is insufficient for sample collection.]

Oropharyngeal swab [collect only if NP swab is not obtainable]: Swab the posterior pharynx, avoiding the tongue.

Process, Store and Submit Specimens
Refrigerate specimens at 2-8°C and ship ASAP to testing on ice pack. Label each specimen container with the patient's ID number (e.g., medical record number), unique specimen ID (e.g., laboratory requisition number), specimen type (e.g., nasopharynx, sputum) and collection date.

Testing through local laboratories is approved bylocal health departments and state health departments; follow your local laboratory processing and shipping requirements.

Turnaround time for testing varies by test availability, shipping time, and lab throughput; anecdotal reports of waiting times vary from hours to 5 days. Availability of FDA approved rapid commercial testing is to be determined.

Treatment: Supporative + Intensive Care

Supportive Care Basics
1) Promptly implement infection control measures.
2) Provide standard supportive management for respiratory disease and complications, including advanced organ support if indicated.
3) Unless otherwise required to treat septic shock or other disease processes (acute exacerbation of COPD, etc), do not use corticosteroids because of the potential for prolonging viral replication, based on lessons learned from observed MERS-CoV patients[5]. See the section on Treatment-Pharmacotherapy for other considerations.

Critical Care Control Measures
1) **Treatment of SARI/ARDS:** Best practices for severe acute respiratory infection (SARI) including infection prevention and control (IPC), triage, and optimized supportive care are included
2) **Surviving Sepsis Campaign:** An international expert consensus panel has issued 54 statements relating to infection control, laboratory diagnosis and specimens, hemodynamic support, ventilatory support, and COVID-19 therapy. The final version of the article will be published as soon as approved on ccmjournal.org. Key recommendations for management of critically ill adults include[47]

Shock Hemodynamic Support
1) Measure dynamic parameters to assess fluid responsiveness; use a conservative fluid administration strategy; using crystalloids over colloids (balanced crystalloids preferred over unbalanced)
2) Use norepinephrine as first-line vasoactive; use vasopressin or epinephrine as first line if norepinephrine is not available; dopamine is NOT recommended if norepinephrine is not available; add vasopressin as a second-line agent if target MAP (60-65 mm Hg) cannot be achieved by norepinephrine alone

Ventilatory Support
1) Start supplemental oxygen if Spo2 <90%; maintain Spo2 no higher than 96%
2) For acute hypoxemic respiratory failure despite oxygen therapy, use high-flow nasal cannula (HFNC) over conventional oxygen and NIPPV; if HFNC is not available, a trial of NIPPV is suggested; closely monitor for worsening respiratory status and intubate early if worsening
3) For mechanical ventilation of ARDS, use low tidal volume (4-8 mL/kg of predicted body weight); target plateau pressures of <30 cm H2O; use a higher PEEP strategy
4) For moderate to severe ARDS, prone ventilate for 12 - 16 hours; use as-needed (instead of continuous) neuromuscular blocking agents to facilitate protective lung ventilation
5) For severe ARDS and hypoxemia despite optimizing ventilation, a trial of inhaled pulmonary vasodilator is suggested but if no rapid improvement in oxygenation is observed, taper the treatment; use lung recruitment manoeuvers such as 40 cm H2O inspiratory hold for 40 seconds, but staircase (incremental PEEP) recruitment is not recommended; use veino-veinous circulation for ECMO

Pharmacotherapy

1) For ARDS, use corticosteroids; do not routinely use corticosteroids in the absence of ARDS
2) In COVID-19 patients receiving mechanical ventilation who have respiratory failure, use empiric antimicrobial/antibacterial agents; assess for de-escalation.
3) For fever, use pharmacologic agents for temperature control

Treatment: Pharmacotherapy

1) Remdesivir: Clinical Trials and Compassionate Use

Remdesivir is a prodrug of an adenosine analog with potent activity against an array of RNA virus families including Filoviridae, Paramyxoviridae, Pneumoviridae, and Orthocoronavirinae, through the targeting of the viral RNA dependent RNA polymerase. Open label dosage trials of remdesevir are ongoing.

Note that the safety and efficacy of remdesevir and other agents is currently unknown, and only data from clinical trials (not individual compassionate use cases) will contribute to determinations of safety and efficacy of remdesivir in treating COVID-19.

Other Antiviral Agents

Lopinavir–ritonavir: An open label trial for the treatment of 199 hospitalized patients with severe Covid-19 found no observed benefit of lopinavir–ritonavir beyond standard care[24].

Convalescent Plasma and Hyperimmune Globulin

An uncontrolled case series of 5 critically ill patients in in Shenzhen, China with COVID-19 and ARDS observed improvement in patient clinical status after administration of convalescent plasma containing neutralizing antibody[45]. The treatment group was not compared with controls, along with other important limitations[46]. C***linical trials are needed regarding safety and effectiveness of administering convalescent plasma to patients with COVID-19***.

The US FDA announced on March 24, 2020 that convalescent plasma may be collected from recovered COVID-19 patients and considered for emergency administration under a single patient emergency Investigational New Drug (IND) application for individual patients with life threateningly severe disease[35] Due to the limitations of clinical trial entry and the volume of expected patient need while awaiting such trial results, the FDA is facilitating this access to COVID-19 convalescent plasma for use in individual patients with serious or immediately life-threatening COVID-19 infections[35]. .

4. Evidence Based Therapeutic Management

Chloroquine & Hydroxychloroquine

Chloroquine is an immunomodulant drug. Prior studies on SARS-CoV demonstrated that it can block the SARS virus infection by reducing virus/cell fusion[26]. It is primarily used to treat malaria that has also been shown effective in reducing viral replication of SARS-CoV and MERS-CoV[25]. For SARS-CoV-2, early data suggest effectiveness in reducing viral infection in vitro[26]. The safety and efficacy for use in Covid-19 caused by SARS-CoV-2 is not known. Nonrandomized observational reports suggest benefit[31], but a prospective controlled clinical trial of hydroxychloroquine with 30 enrolled patients demonstrated no clinical benefit over usual supportive care[41].

Hydroxychloroquine scarcity considerations: Because of its importance in the treatment of systemic lupus erythematosus (SLE) and rheumatoid arthritis, and because even brief hydroxychloroquine (HCQ) withdrawal can result in SLE flare, the American College of Rheumatology (ACR) has posted guidance on scarce resource allocation of HCQ.

Chloroquine + Azithromycin

A small, open-label, non-randomized, practical study enrolled 26 patients [6 asymptomatic, 22 with URI symptoms, and 8 with LRTI symptoms] and 16 controls to observe outcomes on viral load at day 6 of treatment[31]. Of the 26 patients in the treatment group, 6 also received azithromycin with daily electrocardiographic monitoring) and these 6 demonstrated more rapid resolution of viral load in nasopharyngeal swabs[31]. Aside from the small sample size, other limitations of the study included the method of identifying controls, confounders of baseline viral load, imputed data, and loss of patient follow up data[51]. Studies with more severe presentations, carefully identified controls, clinical data points, safety data, and data from high-quality clinical trials remain urgently needed. Furthermore, practical trial data on pre-exposure and post-exposure prophylaxis remain unavailable.

Corticosteroids

Unless otherwise required to treat septic shock or other disease processes (acute exacerbation of COPD, etc), **do not use corticosteroids** because of the potential for prolonging viral replication, based on lessons learned from observed MERS-CoV patients[5].

Other immunomodulators

Other investigational treatment approaches include interferon-1β and a variety of traditional Chinese medicines[14].

ACEI/ARBs: Questions and Evidence

As noted in the chapter on Transmission & Infection, questions have arisen related to the binding of SARS-CoV-2 to the ACE2 functional receptor[20]. This has led to questions about whether ACEI/ARB antihypertensive agents may increase risk for morbidity/mortality due to Covid-19 by increasing ACE2 levels[21]. However, there is no evidence linking anti-hypertensive agents to Covid-19 disease severity, and discontinuing or changing antihypertensive therapy without medical indication and supervision could lead to adverse effects and may be harmful[41,57]. In a study of 187 confirmed Covid-19 positive patients with underlying cardiovascular disease, the mortality of those treated with or without use of ACEI/ARB did not show a significant difference in outcome[55].

The followingjoint statement from the ACC, American Heart Association and Heart Failure Society of America was posted online on March 17 and addresses

using renin angiotensin aldosterone system (RAAS) antagonists in COVID-19: *"... there are no experimental or clinical data demonstrating beneficial or adverse outcomes among COVID-19 patients using ACE-I or ARB medications ... We urge urgent, additional research that can guide us to optimal care for the millions of people worldwide with cardiovascular disease and who may contract COVID-19."*

The following statement is from the Council on Hypertension of the European Society of Cardiology: "*...speculation about the safety of ACEI or ARB treatment in relation to COVID-19 does not have a sound scientific basis or evidence to support it. Indeed, there is evidence from studies in animals suggesting that these medications might be rather protective against serious lung complications in patients with COVID-19 infection, but to date there is no data in humans. The Council on Hypertension of the European Society of Cardiology ... strongly recommend that physicians and patients should continue treatment with their usual anti-hypertensive therapy because there is no clinical or scientific evidence to suggest that treatment with ACEI or ARBs should be discontinued because of the Covid-19 infection.*"

NSAIDs: Controversy and Questions

While there are some reports of associations between non-steroidal anti-inflammatory (NSAID) medication use and severity of disease/outcomes for persons with Covid-19, there is no clear causative relationship or definitive correlation[44]. Individuals who use NSAIDs regularly for medical reasons should contact their physician for guidance.

5. How to Reduce Risk of Viral Respiratory Illnesses

Influenza remains the major cause of severe respiratory illness for most otherwise healthy adults. Therefore, influenza vaccination and standard respiratory hygiene remain essential for health.

There is currently no vaccine protecting against SARS-CoV, so infection control requires reduction of person-to-person transmission by doing the following[6]:

1) Use mask always in outside
2) Wash hands often with soap and water for at least 20 seconds. If soap and water are not available, use alcohol-based hand sanitizer containing at least 60% alcohol.
3) Avoid touching your eyes, nose, or mouth with unwashed hands.
4) Avoid close contact with people who are sick.
5) Stay home when you are sick.
6) Cover your cough or sneeze with a tissue, then throw the tissue in the trash.
7) Clean and disinfect frequently touched objects and surfaces like phones, door knobs, faucets, and light switches.
8) Maintain social distancing.

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