Potential long-term health consequences of covid-19

Abstract

The Covid 2019 (COVID19) problem is the causal factor for very harsh symptoms of the breathing apparatus of the human body disorders Covid 2 (SARCoV2). Despite the fact that lung problems are the most severe, additional respiratory signs of COVID19 are plentiful. Confirmed cases of COVID19 have now exceeded 64 lakhs worldwide as of the month of nov of last year . With the expected rate of decline (the number of COVID19 transmissions broken down into an extended category of COVID19) varies between 1 and 7%, c It is possible that a large number of individuals have recovered from COVID19 which can accumulate a large number of long-term benefits. Despite the fact that multi-organism COVID19 outbreaks have been widely archived, the drawing ramifications of such waves continue to be revealed. MERSCoV]) all had distinct ramifications for SARSCoV2 infection status to predict limited COVID19 survival outcomes, combined respiratory, cardiovascular, hematologic, renal, essential neurologic, and emerging gastrointestinal and psychosocial utility, although well-known post-mortem care conditions. Show people with post-release COVID19 that they are aware of the extent and seriousness of the results. This can be done by reusing or starting to centralize a large number of companions who have so far focused not only on the effects learned from SARSCoV2, but also on the attributes of invulnerability due to ethnicity and imbalances. wages. COVID19 and during hospitalizations. The ultimate fate of COVID19 survivors remains unsettled or undeterrmined, and if this infection persists in us for a long time, the results could increase dramatically as well. Covid 2019 has affected vital organs adversely specially in long term and mental health.

Keywords: renal, cardiovascular, hematological, gastrointestinal manifestation

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Introduction

Severe respiratory distress Covid 2 (SARSCoV2) is the expert cause of Covid disease 2019 (COVID19). The World Health Association (WHO) first more than 57 times. 22, 2020 . Addition of SARSCoV2 allows entry into the cell via restriction of its peak glycoprotein (S) typically to the human movement cascade chemoreceptor 2 (ACE2) and the opportunistic receptor. , 290 L cleavage group (CD290L) .[1] Similar to increased cross section for receptors such as SARSCoV1 . Rich in the ACE2 receptor, the epithelial cells of the nasal and alveolar spaces are the major reception sites for SARSCoV2.[2] In view of the unique pluripotent RNA sequencing dataset, Zou et al. has given dangerous indications to various organs predisposed to SARSCoV2 that depend on the ACE2 claim, combining the respiratory tract and lungs, such as the coronary heart, kidneys, digestive system and other organs . Then, while pneumothorax problems were the biggest sign evoked, the article describes the extrapulmonary result of COVID19 as being excellent , its pathophysiology has been exhaustively described elsewhere . The intracellular infusion of SARSCOV2 in a conjugate layer promotes the leads to their decreased reactivity to deangiotensin II . angiotensin II .[3] These more frequent respiratory occurrences may be due to the widespread spread and replication of SARSCoV2 due to direct disseminated toxicity or to immunopathological findings of toxicity, including endothelial damage, mobility, abscess-related irritation, and the deregulation of the response. impregnable framework (called "cytokine storm") .[4] According to Johns Hopkins University, the cost of death (category of births separated by type of time shown) for COVID19 patients typically ranges from 1% to 7%, well-defined costs based on many factors, including dates of death. 'deadline. in the main case indicated, the choice of viability, the rules of local response to the pandemic, and the age of the population . For the model, in Italy, the costs of death vary from 3% in people over 60 years to more than 30% in people 80 years and over . Therefore, it is possible that a large number of individuals with COVID19 will be recovered. Despite the fact that sick people have been infected with SARSCoV2, some of the consequences of the consequences can also significantly affect their health later in life. Multi-organ markers of COVID19 are now extensively archived , however, the design ramifications of these occurrences have yet to be found. The United States alone has more than four. Three times have been indicated , with the potential to exceed the 0 million infections found from the seroconversion study . In a year of shutdown, it is almost certain that the most severe category will exceed

100,000,000.[5] Therefore, the number of individuals able to cope with the persistent problem of COVID19 contamination is very large. Since COVID19 has not been with us long enough to assess the majority of the adverse effects of poisoning, we need to be open to similar Covids in the past by trying to anticipate recommendations already. a large part of the new Covids that have spread to humans are SARSCoV1 and East Central Respiratory Disease (MERSCoV). SARSCoV1 generally causes a severe pandemic of respiratory disorders (SARS) 2002-2003, causing multiples, resulting in 77 deaths in mid-July 2003 . 866 cases out of 2,519 cases in January 2020 .

While COVID19 appears to have added infectivity and reduced mortality when assessing SARSCoV1 and MERSCoV, older Covids have shown similarity to SARSCoV2 , which makes it conceivable. -term We desperately need measurements from observations of COVID19 patients over time It takes a long time to teach healthcare professionals the most effective approach to demonstrate survivors' fitness, provide intervention early which can be beneficial and reduce the risk of overpowering clinical equipment.[6] Prepare the insides of the things that are about to happen because of an impending migraine.of several volumes of thousands of patients. In the interim, this evaluation looks at numerous extra aspiratory and pneumonic signs of COVID19 with regards to what is known from past Covids forthcoming long haul wellbeing ramifications of COVID19. supplant this evaluation every year with the huge and quickly developing writing..Potential long haul results of SARSCoV2 disease. Expected extra aspiratory and pneumonic appearances of COVID19 are given fundamentally dependent on an assessment of recorded long haul results of SARSCoV1 and MERSCoV, notwithstanding the COVID19 indications reported to date. The most splendid long haul outcomes incorporate the basic disturbing gadget, psychosocial, cardiovascular, aspiratory, hematologic, renal and gastrointestinal appearances, following the distribution of expanded consideration condition COVID19, Covid sickness 2019; MERSCoV, Center East respiratory disorder Covid; SARSCoV1, extreme intense respiratory disorder Covid 1; SARSCoV2, intense respiratory pain disorder Covid 2; CHD, coronary vein infection; GI, gastrointestinal; MI, myocardial dead tissue.[7]

Pulmonary manifestations

Few probes have demonstrated deactivated physical capacity of human and diffusivity for carbon monoxide(CO) (DLCO) in SARSCoV1 survivors from 7 months to fifteen years of follow-up , a proposed impedance of the common pathway in the alveoli. For eg., a survey of 111 survivors of SARS-Co-V1 contamination revealed minor abnormalities on chest x-rays, further reducing the ability to practice and the safety of the workup for a single normal subject 8 months after the onset of side effects .[8] A 2365-day follow-up examination showed that fifty-two percent of SARS-Co-V1 manifests exhibited pervasive weakness and decreased exercise utility . In a 15-year ad review of 71 patients who had recovered from SARSCoV1, the amount of abnormal pulmonary interstitial superwound and the adjusted utility reduction in the first 2 years after infection, with .6 % of victims with an interstitial lung malformation after 15 years . Abnormalities of comparable duration are recommended for MERSCoV, with 36% of patients recovering from MERSCoV presenting with bizarre chest x-rays, including pulmonary fibrosis, and glass floor opacity and pleural thickening, on average 0 three days later release .[9] Finally, 1 year after overcoming desperation with high intensity breathing (ARDS), diffuse utility appears to persist normally in up to eighty percent of victims, with 20% exhibiting wind flow obstruction and 20% thoracic limit .Immunopathological sports due to infection are linked to the tracheal indications of SARSCoV1 and MERSCoV .[10] In particular, these may also include rapid infectious replication resulting in more severe and cytotoxic contamination of alveolar epithelial cells (eg, lung type I and II cells) and slowly induce support from fiery cytokines and chemokines, which in turn select fibroblasts and achieve their cleavage into fibroblasts . In addition, the alienation capacity of SARSCoV1 and MERSCoV and ultimately suppression of interferon responses disrupts the fiery response .[11]

Cardiovascular manifestations

Cardiovascular headaches are associated with MERSCoV, SARSCoV1 , and pneumonia . For example, in a study of 121 patients admitted with SARSCoV1 infection (25% of existing SARSCoV1-infected patients), hypotension (50% of patients), tachycardia (71%), bradycardia (1.9%). The presence of cardiovascular headaches such as was detected.)[12] And cardiac hypertrophy (10.7%) . However, the largest complications were normalized by the useful support of the application 3 weeks after discharge in addition to tachycardia and were detected in 35% of patients 3 weeks later . Similarly, individuals infected with SARSCoV1 suffered systolic-

independent asymptomatic diastolic insufficiency indefinitely in the future of acute infection, but the decline was reversible 30 days after clinical recovery . In a longer follow-up suggesting an accelerated risk of cardiovascular disease (CVD) after coronavirus infection, 68%, including 12-month test compliance, respectively. Is reported.[13] Persistent work-related hyperlipidemia, abnormalities in cardiac equipment, and problems with glucose metabolism . Hospitalization for pneumonia may also be associated with long-term cardiovascular complications. In a review of the large cohort (cardiovascular health study cohort [n = 5,888, n = 591 lung inflammation cases] and atherosclerosis-community risk study cohort [n = 15,792, n = 680 cases of pneumonia]), hospitalization was Due to the failure of pneumonia associated with an accelerated risk of CVD (ie, myocardial infarction, stroke and fatal coronary artery disease) 30 days, 90 days, 1 year and 10 years (best in the latter cohort) after admission .[14] In addition, excessive blood stress and coronary artery disease are some of the greatest risk factors for COVID19, especially because perivascular pericytosis and cardiomyocyte ACE2 in patients with these diseases are upregulated . In summary, a significant amount of literature makes it possible to consider lung infections and cardiovascular complications. Although many cardiovascular symptoms appear reversible immediately after infection, epidemic studies have accelerated post-infection cardiovascular threats. In addition, COVID19 is associated with cardiovascular diseases such as acute myocardial damage , cardiomyopathy , myocarditis , and arrhythmias .[15] A study of 44 primary patients with COVID 19 recorded in Wuhan, China, found myocardial damage in 5 of patients.

Hematologic manifestations

Although information is generally scarce, MERSCoV has been associated with hematologic markers and thrombotic entrapment, as well as thrombocytopenia and disseminated intravascular coagulation (DIC) sixty-seven.[16] The basis for comparison quickly became clear with the SARSCoV2 contamination. Several hematologic indications have shown terrible prognostic rates in patients with coronavirus, which are associated with lymphopenia , leukocytosis and thrombocytopenia and coagulopathy (delayed dimer and fibrinogen) . Coagulopathy has become an excellent possibility of infection with SARSCoV2,generally considered to be a country favorable to thrombosis, in terms of vein and vascularization . Thrombotic migraine due to SARSCoV2 contamination was first presented in China And the

Netherlands in patients up to 30% of large consideration units (ICU). Regardless of thromboprophylaxis, an unlikely occurrence of thrombotic difficulties (usually thromboembolic exercises) has been demonstrated . In addition, cases of DIC andmicrothrombi have been expressed in patients with coronavirus. In particular, posthumous assessment showed alveolar slim microthrombi to be multiple times additional now no longer unusualplace in patients who kicked the bucket of Coronavirus in appraisal to the people who passed on of flu .[17]

Renal manifestations

Severe renal failure has occurred in patients with SARSCoV1 and now even more so in patients with SARSCoV2. A had an analysis of 536 SARSCoV1 patients with 6.7 severe kidney damage (AKI) who developed 5 to 0 eight days after the onset of viral infection .[18] Of these patients, 91.7% kicked the bucket, a rate significantly higher than the death rate for SARSCoV1 victims without renal failure (8.8%). It has been suggested that ARI occurs more frequently in coronavirus victims than in those infected with SARSCoV1, including 0.5-29% of coronavirus victims in China and 37% in the United States. United, with 1 % requiring dialysis . In addition, a meta-evaluation including more than thirteen,000 victims with coronavirus showed an ARI prevalence of 17% . Another renal failure presenting in coronavirus victims is hematuria, which is reported in almost half of all coronavirus patients , and proteinuria, which presents as debris in 87% of all patients with diseases caused by the coronavirus . However, research is now out of reach to clarify long-term kidney failure in victims infected with Covid. [19]. Possibly due to unreasonable death rates in beneficiaries of kidney failure .

Central nervous system manifestations

SARSCoV2 can enter the central nervous system, presumably via platform development via destruction of thin endothelium to gain legal recognition of contemplations or via cribriform disc and smell (reviewed in). Studies have shown a residual strength of RNA from different Covid covering the CNS after a particular disease passage, causing neuronal misfortune (investigation in). One or a combination of direct disseminated encephalitis, underlying disease, marginal visceral problems, as well as cerebrovascular changes render coronavirus survivors helpless in the face of persistent neurological findings .[20] Apparently, on release, 33% of coronavirus patients presented with intellectual impedance and motor deficits . Basal contamination is well

known in SARSCoV2 disease , and the fact that basic pollution increases intellectual disability and neurodegenerative infections , it is recommended that people living with Coronavirus survivors likely benefit from the neurodegeneration. long-term. Moreover, the relentless wind is very clearly identified with assiduous intellectual decadence, government problems and a reduced presence (studied in . Therefore, longitudinal concordance tests are needed to determine the long-term neurological effects of SARSCoV2 infection and to recognize its neural invaders.[21]

Gastrointestinal manifestations

Patients treated with SARSCoV1 have been prescribed for entry into the gastrointestinal tract , liver and pancreas . Additionally, suggested gastrointestinal side effects and signs of coronavirus include eating disorders , nausea , loss of power , discomfort in the abdomen,stools which are very loose , and G.I.T. diarrhea .[22] Likewise, segregation of the SARSCoV2 virus from waste collection and endoscopic mucosal lesions also allow the utility of SARSCoV2 to persist, reflect and contaminate the environment of the digestive batch. Hepatic inclusions, including hepatotoxicity, have been reported in coronaviruses, the aminotransferases alanine and persistent aspartate . Severe hepatitis with abnormal hepatic manifestations with previously usual manifestations and side effects as well as signs and indications of coronavirus have also been demonstrated . Examination of the biopsy revealed the possibility of liver neurological damage, all due to lobular contamination and mild entry versus mild microvascular stenosis . In addition, the pancreatic markers of Coronavirus have been well demonstrated, as well as an increase in lipase and amylase leading to severe acute pancreatitis [23]. Even so, long-term observational studies are needed to determine whether gastrointestinal, hepatic and pancreatic indications can be maintained after severe toxicity.

Psychosocial Manifestations

Another urgent recreational activity given the long-range impact of the Coronavirus is the impact on the academic health of survivors, including stress, distress and suffering. concessions, likewise for the significant signs and indications, associated with exacerbation at rest and non-arrest asthenia / myosomyelitis [24]. The long-term effects of SARSCoV1 include constant pain, exhaustion, hopelessness and long-term discomfort, resulting in the inability to return

mechanically unproven works of art one year after being heavily polluted [25]. A review of SARSCoV1 survivors in Hong Kong revealed reasonable judgments on PTSD (55%), depression (39%), wasting disease (36%), warning disease (33%) and on maximum urgency (16%) at 31-50 months of release from contamination, a sharp increase over the frequency of pre-infection psychiatric assessment of 3% [26][27]. Another person examined 195 victims of SARSCoV1, predicting that 10-18% of victims had identified signs and side effects of PTSD, stress and sadness, with more severe manifestations in those at risk. . Additionally, a survey of 117 survivors from Toronto, Ontario found that at the time of consent for up to one year, 33% of victims expressed a much lower level of well-being and had a total of 668 visits by 51 victims to psychiatrists or brain researchers. [28]. These victims reported stressors such as friendly scams, loss of media secrets, sharing of the individual's family and associates, lack of ability to take a stand. -be, and the inconvenience of sending the infection to those who are appreciated [29]. Loss of effort can be a major contributor to the academic health of people with disabilities after SARS.

Review of SARSCoV1 Survivors

Seventy-eight percent details are again acceptable to complete the time creation job after 2 years [30]. While current facts about the increased health status and physical side effects of the coronavirus are scarce, an analysis of 71 hospitalized patients, but solid coronavirus patients, is recommended to distribute symptoms, but at this point not stress, when assessed for isolated individuals [31]. Psychosocial signs of SARSCoV2. In addition, the coronavirus can also have a significant impact on high levels of survivors, as well as anxiety, hopelessness, PTSD, OCD, alarm problems, and industrious anxiety. Additionally, physical manifestations in light of SARSCoV2 disease may involve disruptive effects of rest and relentless weakness. Potential stressors that can cause these signs include death of family and co-workers, inability to attend memorial services due to shared ties, inability to work in the arts, inconvenience while communication with friends and family, the enthusiastic pressure of isolation and isolation, defamation, lack of media secrecy and radically seen viability. Coronavirus, Covid 2019 infection; OCD, habitual fanaticism; PTSD, Distributed Stress Disease; SARSCoV2, Paroxysmal Severe Respiratory Arrhythmias Covid 2. Embody the whole approach from various angles which has its focus on the healing of the ill person in the long run of life that is in the long term

period , especially for pathologies related with the mental state of a person & burnout, although recovery Feedback and formulation valuable are needed. People are beginning to make various applications for the mobile phones so that the ill people can get help in their pathway to full and a long term that is lifelong recovery .[32-38]

REFRENCES

1. Xu X, Chen P, Wang J, Feng J, Zhou H, Li X, Zhong W, Hao P. Evolution of the novel coronavirus from the ongoing Wuhan outbreak and modeling of its spike protein for risk of human transmission. Science China Life Sciences. 2020 Mar;63(3):457-60.
2. Amraie R. CD209L/L-SIGN and CD209/DC-SIGN act as receptors for SARS-CoV-2 and are differentially expressed in lung and kidney epithelial and endothelial cells. bioRxiv 182, 436-418, doi: papers3. publication/doi/10.1101/2020.06. 2020;22.
3. Ou X, Liu Y, Lei X, Li P, Mi D, Ren L, Guo L, Chen T, Hu J, Xiang Z, Mu Z. Characterization of spike glycoprotein of 2019-nCoV on virus entry and its immune cross-reactivity with spike glycoprotein of SARS-CoV.
4. Jeffers SA, Tusell SM, Gillim-Ross L, Hemmila EM, Achenbach JE, Babcock GJ, Thomas WD, Thackray LB, Young MD, Mason RJ, Ambrosino DM. CD209L (L-SIGN) is a receptor for severe acute respiratory syndrome coronavirus. Proceedings of the National Academy of Sciences. 2004 Nov 2;101(44):15748-53.
5. Zou X, Chen K, Zou J. Single-cell RNA-seq data analysis on the receptor ACE2 expression revealsthepotentialriskofdifferenthumanorgans vulnerable to 2019-nCoV infection. Front Med.
6. Wadman M, Couzin-Frankel J, Kaiser J, Matacic C. A rampage through the body.
7. Gupta A, Madhavan MV, Sehgal K, Nair N, Mahajan S, Sehrawat TS, Bikdeli B, Ahluwalia N, Ausiello JC, Wan EY, Freedberg DE. Extrapulmonary manifestations of COVID-19. Nature medicine. 2020 Jul;26(7):1017-32.
8. Verdecchia P, Cavallini C, Spanevello A, Angeli F. The pivotal link between ACE2 deficiency and SARS-CoV-2 infection. European journal of internal medicine. 2020 Jun 1;76:14-20.
9. Johns Hopkins University of Medicine. Coronavirus resource center: mortality analyses.

10. AbouGhayda R, Lee KH, Han YJ, Ryu S, Hong SH, Yoon S, Jeong GH, Lee J, Lee JY, Yang JW, Effenberger M. Estimation of global case fatality rate of coronavirus disease 2019 (COVID-19) using meta-analyses: comparison between calendar date and days since the outbreak of the first confirmed case. International Journal of Infectious Diseases. 2020 Nov 1;100:302-8.
11. Signorelli C, Odone A. Age-specific COVID-19 case-fatality rate: no evidence of changes over time. International journal of public health. 2020 Nov;65(8):1435-6.
12. Fang B, Meng QH. The laboratory's role in combating COVID-19. Critical reviews in clinical laboratory sciences. 2020 Aug 17;57(6):400-14.
13. World Health Organization. Coronavirus disease 2019 (COVID-19): situation report, 73.
14. Havers FP, Reed C, Lim T, Montgomery JM, Klena JD, Hall AJ, Fry AM, Cannon DL, Chiang CF, Gibbons A, Krapiunaya I. Seroprevalence of antibodies to SARS-CoV-2 in 10 sites in the United States, March 23-May 12, 2020. JAMA internal medicine. 2020 Dec 1;180(12):1576-86.
15. Wu A, Peng Y, Huang B, Ding X, Wang X, Niu P, Meng J, Zhu Z, Zhang Z, Wang J, Sheng J. Genome composition and divergence of the novel coronavirus (2019-nCoV) originating in China. Cell host & microbe. 2020 Mar 11;27(3):325-8.
16. Ngai JC, Ko FW, Ng SS, TO KW, Tong M, Hui DS. The long-term impact of severe acute respiratory syndrome on pulmonary function, exercise capacity and health status. Respirology. 2010 Apr;15(3):543-50.
17. Su MC, Hsieh YT, Wang YH, Lin AS, Chung YH, Lin MC. Exercise capacity and pulmonary function in hospital workers recovered from severe acute respiratory syndrome. Respiration. 2007;74(5):511-6.
18. Liu YX, Ye YP, Zhang P, Chen J, Ye H, He YH, Li N. Changes in pulmonary function in SARS patients during the three-year convalescent period. Zhongguoweizhongbingjijiuyixue= Chinese critical care medicine= Zhongguoweizhongbingjijiuyixue. 2007 Sep 1;19(9):536-8.
19. Hui DS, Joynt GM, Wong KT, Gomersall CD, Li TS, Antonio G, Ko FW, Chan MC, Chan DP, Tong MW, Rainer TH. Impact of severe acute respiratory syndrome (SARS) on pulmonary function, functional capacity and quality of life in a cohort of survivors. Thorax. 2005 May 1;60(5):401-9.

20. Zhang P, Li J, Liu H, Han N, Ju J, Kou Y, Chen L, Jiang M, Pan F, Zheng Y, Gao Z. Long-term bone and lung consequences associated with hospital-acquired severe acute respiratory syndrome: a 15-year follow-up from a prospective cohort study. Bone research. 2020 Feb 14;8(1):1-8.
21. Das KM, Lee EY, Singh R, Enani MA, Al Dossari K, Van Gorkom K, Larsson SG, Langer RD. Follow-up chest radiographic findings in patients with MERS-CoV after recovery. The Indian journal of radiology & imaging. 2017 Jul;27(3):342.
22. Orme Jr J, Romney JS, Hopkins RO, Pope D, Chan KJ, Thomsen G, Crapo RO, Weaver LK. Pulmonary function and health-related quality of life in survivors of acute respiratory distress syndrome. American Journal of Respiratory and Critical Care Medicine. 2003 Mar 1;167(5):690-4.
23. Channappanavar R, Perlman S. Pathogenic human coronavirus infections: causes and consequences of cytokine storm and immunopathology. InSeminars in immunopathology 2017 Jul (Vol. 39, No. 5, pp. 529-539). Springer Berlin Heidelberg.
24. Kinaret PA, Del Giudice G, Greco D. Covid-19 acute responses and possible long term consequences: what nanotoxicology can teach us. Nano Today. 2020 Dec 1;35:100945.
25. Cao Y, Liu X, Xiong L, Cai K. Imaging and clinical features of patients with 2019 novel coronavirus SARS-CoV-2: a systematic review and meta-analysis. Journal of medical virology. 2020 Sep;92(9):1449-59.
26. Wang D, Hu B, Hu C, Zhu F, Liu X, Zhang J, Wang B, Xiang H, Cheng Z, Xiong Y, Zhao Y. Clinical characteristics of 138 hospitalized patients with 2019 novel coronavirus–infected pneumonia in Wuhan, China. Jama. 2020 Mar 17;323(11):1061-9.
27. Shi H, Han X, Jiang N, Cao Y, Alwalid O, Gu J, Fan Y, Zheng C. Radiological findings from 81 patients with COVID-19 pneumonia in Wuhan, China: a descriptive study. The Lancet infectious diseases. 2020 Apr 1;20(4):425-34.
28. Spagnolo P, Balestro E, Aliberti S, Cocconcelli E, Biondini D, Della Casa G, Sverzellati N, Maher TM. Pulmonary fibrosis secondary to COVID-19: a call to arms?. The Lancet Respiratory Medicine. 2020 Aug 1;8(8):750-2.
29. Guzik TJ, Mohiddin SA, Dimarco A, Patel V, Savvatis K, Marelli-Berg FM, Madhur MS, Tomaszewski M, Maffia P, D'acquisto F, Nicklin SA. COVID-19 and the cardiovascular

system: implications for risk assessment, diagnosis, and treatment options. Cardiovascular research. 2020 Aug 1;116(10):1666-87.

30. Inui S, Fujikawa A, Jitsu M, Kunishima N, Watanabe S, Suzuki Y, Umeda S, Uwabe Y. Chest CT findings in cases from the cruise ship diamond princess with coronavirus disease (COVID-19). Radiology: Cardiothoracic Imaging. 2020 Mar 17;2(2):e200110.
31. Bandirali M, Sconfienza LM, Serra R, Brembilla R, Albano D, Pregliasco FE, Messina C. Chest radiograph findings in asymptomatic and minimally symptomatic quarantined patients in Codogno, Italy during COVID-19 pandemic. Radiology. 2020 Jun;295(3):E7-.
32. Alhogbani T. Acute myocarditis associated with novel Middle East respiratory syndrome coronavirus. Annals of Saudi medicine. 2016 Jan;36(1):78-80.
33. Burhani, Tasneem Sajjad, and Waqar M. Naqvi. “Telehealth - A Boon in the Time of COVID 19 Outbreak.” JOURNAL OF EVOLUTION OF MEDICAL AND DENTAL SCIENCES-JEMDS 9, no. 29 (July 20, 2020): 2081–84. https://doi.org/10.14260/jemds/2020/454.
34. Gawai, Jaya Pranoykumar, Seema Singh, Vaishali Deoraoji Taksande, Tessy Sebastian, Pooja Kasturkar, and Ruchira Shrikant Ankar. “Critical Review on Impact of COVID 19 and Mental Health.” JOURNAL OF EVOLUTION OF MEDICAL AND DENTAL SCIENCES-JEMDS 9, no. 30 (July 27, 2020): 2158–63. https://doi.org/10.14260/jemds/2020/470.
35. Naqvi, Waqar M., and Arti Sahu. “Paradigmatic Shift in the Education System in a Time of COVID 19.” JOURNAL OF EVOLUTION OF MEDICAL AND DENTAL SCIENCES-JEMDS 9, no. 27 (July 6, 2020): 1974–76. https://doi.org/10.14260/jemds/2020/430.
36. Prasad, Narayan, Mansi Bhatt, Sanjay K. Agarwal, H. S. Kohli, N. Gopalakrishnan, Edwin Fernando, Manisha Sahay, et al. “The Adverse Effect of COVID Pandemic on the Care of Patients With Kidney Diseases in India.” KIDNEY INTERNATIONAL REPORTS 5, no. 9 (September 2020): 1545–50. https://doi.org/10.1016/j.ekir.2020.06.034.
37. Wanjari, A. K., Ayush Dubey, Sourav Chaturvedi, and Sunil Kumar. “Young COVID 19 Presenting as Fatal Subarachnoid Hemorrhage: Association or Chance?” MEDICAL SCIENCE 24, no. 104 (August 2020): 2712–15.

38. Kolhe, Seema, Minal Dambhare, Priya Dhankasar, Pallavi Dhole, Ashwathy Nair, and Priya Rewatkar. “Home Remedies During Covid Pandemic Lockdown.” JOURNAL OF RESEARCH IN MEDICAL AND DENTAL SCIENCE 8, no. 6 (2020): 103–7.