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Review Article

Post COVID Cardiovascular Care - A Review

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Abstract:

Over the last one-year COVID 19 has affected millions of people around the world. Though initially it was thought to be an infection of respiratory system, but it involves any organ of the body. Its morbidity and mortality are more when it involves patients with preexisting cardiovascular diseases and also when it causes a cardiovascular complication. COVID 19 can produce a number of cardiovascular complications including myocardial infarction, heart failure, myocarditis, arrhythmia, thromboembolism etc. Following recovery from COVID 19 infection evaluation, follow up and management of these cardiovascular conditions, which may be sometimes in subclinical form, is essential. Though there is lack of adequate studies or guidelines, we tried to discuss the different aspects of Post- COVID cardiovascular care depending on the existing evidence and previous guidelines.

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Introduction:

COVID 19 has got a tremendous impact in the cardiovascular health. It has changed the cardiovascular disease profile and practice of cardiovascular medicine a lot. Though primarily it involves the respiratory system; but it can involve any other organ of the body and cardiovascular system is the most important among them, which has got serious morbidity and mortality. These cardiovascular sequelae along with other multiorgan disorder and also cognitive and mental disorders may persist even after recovery from infection. Usually, patients who survive the acute hospitalization and/or ICU stay, are at significant risk of these cognitive, mental and physical sequelae, known as Post- Intensive care syndrome (PICS) and Post- Hospital syndrome (PHS).[1,2] Large number studies report some sort of persistent symptoms even among those who were not hospitalized. In combination all these clinical conditions can be defined as Post COVID syndrome. Many of the features of this post COVID syndrome mimics with cardiac symptoms. Sometimes cardiovascular complications are not well recognized or evaluated during the phase of acute infection due to lack of use or availability of investigations. Many of the patients present for evaluation of their preexisting or newly developed cardiovascular conditions. Sometimes they also present to find whether COVID has done any damage to their cardiovascular system. Though there are not many studies about how to deal with these COVID related cardiovascular conditions after recovery from acute infection, cardiologists have to manage these patients. These patients include both groups, patients with cardiac illness before & now suffered from COVID infection and patients who have got cardiac disease as a consequence of COVID infection. We have to use our experience and guidelines to learn how to manage these post COVID cardiovascular disorders.

Definition of Post COVID syndrome

There is no well accepted definition of Post COVID syndrome. The National Institute for Health and Care Excellence (NICE), the Scottish Inter-collegiate Guidelines Network (SIGN) and the

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Royal College of General Practitioners (RCGP) have defined post-COVID syndrome as:[3]

Signs and symptoms that develop during or following an infection consistent with COVID- 19 which continue for more than 12 weeks and are not explained by an alternative diagnosis. The condition usually presents with clusters of symptoms, often overlapping, which may change over time and can affect any system within the body. Many people with post-COVID syndrome can also experience generalized pain, fatigue, persisting high temperature and psychiatric problems.

Post-COVID-19 syndrome may be considered before 12 weeks while the possibility of an alternative underlying disease is also being assessed.

Epidemiology of Post COVID syndrome

An UK based study reveals around 10% of patients who have tested positive for SARS-CoV-2 virus remain unwell beyond three weeks, and a smaller proportion for months.[4] A recent US study found that only 65% of people had returned to their previous level of health 14-21 days after a positive test.[5]

Reason behind the prolong symptoms is damage to multiple organs including lungs, heart, vascular system, gastrointestinal tract, liver, kidney and brain. Damage may be due to direct infiltration, inflammatory process, thrombotic micro-angiopathy, venous thrombosis and hypoxia. Many of the infected patients may suffer from silent hypoxia in the asymptomatic or presymptomatic stage. Organ damage has been documented in some patients with mild symptoms also. Some of the patients also suffer from Post intensive care symptoms (PICS), like- muscle weakness, cognitive impairment, balancing problem and psychological problems.[6]

Cardiovascular complications of COVID

cardiovascular complications accounted for 7% of deaths at 30 days in patients with COVID-19 compared with sepsis in nearly 90%. Perhaps 20% of patients admitted with covid-19 have clinically significant cardiac involvement;[49,50-7] occult involvement may be even commoner.[7,8] These include myocarditis, pericarditis, myocardial infarction, dysrhythmias, and pulmonary embolism; they may present even several weeks after acute covid-19. They are commoner in patients with pre-existing cardiovascular disease,[9] but they have also been described in young, previously active patients.[10-12] Myocardial injury is common in severe COVID-19 as a function of baseline comorbidities, advanced age and multisystem organ dysfunction. The adverse prognosis of myocardial injury in COVID-19 is a function of multisystem organ involvement, similar to generic ARDS.[13]

Various pathophysiological mechanisms have been proposed, including viral infiltration, inflammation and microthrombi, and down-regulation of ACE-2 receptors. Depending on the type of myocardial injury, there may be important sequelae if residual inflammation or fibrosis exists. As SARS-CoV-2 is a new pathogen, there are no long-term data on cardiovascular abnormalities or dysrhythmias that may occur in the convalescent phase.

Delayed or absent presentation of ACS

The European Society of Cardiology (ESC) administered an online questionnaire to cardiovascular specialists (cardiologists and cardiovascular nurses) across 6 continents. Nearly 80% responded that the number of STEMI patients was reduced since the COVID-19 outbreak and 65.2% indicated that the reduction in STEMI presentations was > 40%. Additionally, about 60% of all respondents reported a delay in ED presentation among STEMI patients and that > 40% presented beyond the optimal window for PCI or thrombolysis.[14]

Many of the centers in Europe and USA reported reduced and delayed presentation of acute cardiovascular conditions.[15,16] In Bangladesh also, there was marked reduction in hospital admissions.

This was mainly in the early part of pandemic and partly due to the lack of measures to detect COVID patients, to make separate arrangements in the for COVID and non- COVID patients in the same institute, treatment policy of the institutes and apprehension of getting infected in medical facilities by the patients.[17] During this pandemic many of the organizations recommended for primary PCI as standard of care for STEMI patients at PCI capable hospitals when it can be provided in a timely fashion, with an expert team outfitted

with PPE in a dedicated CCL room and a fibrinolysis-based strategy may be entertained at non-PCI capable referral hospitals or in specific situations where primary PCI cannot be executed or is not deemed the best option.[13] The impact of this strategy is also not well studied. In some centers in India, the outcome of acute MI was worse because of delayed presentation, like more reduced ejection fraction and longer hospital stay.[18]

The impact of this delayed treatment increased the rate of out of hospital cardiac arrest in Lombardy, Italy.[19]

The number of elective procedures were also deferred worldwide. This is also true for the COVID positive patients along with the general population. This delay or lack of treatment in COVID patients can lead their cardiovascular health to a worse state. Though the impact of this delay and lack of treatment is not well studied so far. It's expected that more patients will present with cardiovascular complications including heart failure, chronic coronary syndrome and arrhythmia.

Type I Myocardial infarction

As the incidence of COVID is more among the patients having cardiovascular risk factors, the incidence of MI is also high in this group of patients. Besides this, COVID produces a hypercoagulable state and also produces endothelial injury leading to plaque rupture. Thus, COVID can precipitate an episode of MI with a different pathophysiology. There is increased thrombus burden, multiple culprit arteries, increased need for heparin and GP IIb IIIa inhibitors and increased morbidity and mortality in the patients with MI with COVID19.[20] Because of system delay and patient's apprehension and reluctance there is a delay in reperfusion also. The COVID-19 pandemic had significant impact on the treatment of patients with STEMI, with a 19% reduction in PPCI procedures, especially among patients suffering from hypertension, and a longer delay to treatment, which may have contributed to the increased mortality during the pandemic.[21]

It has already been established that anticoagulation can improve the outcome of hospitalized COVID patients. But the recommendation for anticoagulation in post hospital phase differs among the experts.[22,23] What should be the anticoagulation and antiplatelet protocol of these group of patients with acute MI with or without PCI and COVID is not well established. At the moment, anticoagulation (NOAC) for 4-6 weeks followed by standard therapy is recommended. Dual antiplatelet therapy should be added to anticoagulants in the first one month after acute MI with or without primary PCI. Then only dual antiplatelet therapy should be given. The use of ticagrelor and prasugrel is not recommended along with NOAC. For the patients who will be treated for DVT or pulmonary embolism, anticoagulation should be continued for three to six months. On this case, aspirin should be stopped after one month and clopidogrel should be continued for the period of anticoagulation. After the period of anticoagulation, antiplatelet drugs should be tailored as per patient's requirement.

Regarding other standard post MI care, standard follow-up and care are recommended.

Type II Myocardial Infarction

Type II myocardial infarction is one of the cardiovascular manifestations of COVID. In non-COVID patients with type 2 MI and/or cardiac injury has got significantly higher rates of major cardiovascular events (after adjustment for clinical covariates) and higher rates of noncardiovascular death.[24] This may also be true for the COVID patients. So, these patients should be treated and followed up with standard care of treatment, including beta blocker, ACEi/ARB and antiplatelet drugs and management of comorbid conditions.

Myocarditis

This is one of the most important cause of morbidity and mortality in COVID patients. The long- term sequelae of COVID myocarditis are not well established. If we consider about the other form of viral myocarditis, the survival is reduced and 20% patients die in one year and 40% patients die in four years. There is increased chance of sudden cardiac death, ventricular and atrial arrhythmia.[25] Ventricular arrhythmias occur even in patients with a left ventricular ejection fraction of >50%.[26] These observations may also be applicable for COVID myocarditis.

COVID myocarditis may develop even after hospital discharge. Most of these patients are being

treated with steroids and other anti-inflammatory drugs. There should be some well controlled studies regarding their efficacy and safety. Complications of myocarditis like heart failure and arrhythmia should be managed like the other forms of myocarditis.

Patients who develop COVID myocarditis, should be regularly followed up to manage the complications and improve their outcome.

Tako Tsubo syndrome

During the COVID-19 pandemic, the incidence of Tako Tsubo syndrome (TTS) has risen 4.5-fold even in individuals without severe acute respiratory syndrome coronavirus 2 infection.[27] This is presumably due to increased emotional stress. Though TTS has got a relatively benign long- term course, in acute condition it results in serious in-hospital adverse events in 20% of patients, with estimates of in-hospital mortality of about 2.0% to 5.6%[28] and rates of major adverse cardio- vascular and cerebrovascular events (MAC- CEs) ranging from 5% to 10%.[29,30] By definition, when associated with COVID 19, this is a secondary TTS and this secondary TTS has got worse prognosis than primary TTS.[31] These patients may develop cardiogenic shock, arrhythmia and thrombus formation along with chest pain and respiratory distress. Recovery from TTS can be slow, with persistent abnormalities on echocardiography and cardiac MRI, prolonged NT-proBNP elevation, impaired quality of life, and even long-term myocardial fibrosis in up to 10% of patients with TTS. These patients demonstrated long-term symptomatic and functional impairment despite normalized ejection fraction.[32] Although atrial and ventricular arrhythmias are common during the acute phase, there does not appear to be a significant risk of ventricular arrhythmias in the convalescent phase after recovery from takotsubo cardiomyopathy.[33] Patients with TTS are at risk for a repeat episode of stress-induced cardiomyopathy; the largest study reported a recurrence rate of 1.8% per patient-year.[34, 35] So, these patients should be followed up. There is not much long-term trial in TTS. The use of angiotensin-converting enzyme inhibitors or angiotensin-receptor blockers was associated with improved survival at 1-year follow-up even after propensity matching. In contrast, there was no evidence of any survival benefit for the use of beta-blockers.

Psychiatric disorders (e.g., depression, anxiety) are common in TTS patients, and those might benefit from a combined psychocardiologic rehabilitation.[36]

Spontaneous coronary artery dissection (SCAD)

SCAD is one of the important cardiovascular effect's manifestations of COVID. The potential mechanisms for SCAD are that SARS-CoV-2 viral infection can lead to T-cell activation and infiltration in adventitia and periadventitial fat, which in turn produce more cytokines and proteases, thereby increasing the risk of plaque rupture or erosion and subsequent dissection (inside-out mechanism of SCAD).[37] Another mechanism is that SARS-CoV-2 may stimulate angiogenesis and lead to proliferation of the vasa vasorum. The newly formed vasa vasorum is relatively leaky, fragile, and prone to disruption, which results in intramural hematoma (outside-in mechanism of SCAD).[38] Vasa vasorum can also serve as the conduit for the entry of inflammatory cells into the tunica media and adventitia, facilitating inflammation and disruption of vasa vasorum.[39] Sometimes there is a complex interplay between SCAD and TTC. COVID-19 is associated with greater mortality and the potential risk of both SCAD and TTC, it is advisable to screen for SCAD in the presence of TTC in order to improve patient outcomes.[40] Whether these COVID induced SCAD needs any special treatment or it can be managed as like the traditional SCAD is not known. But at the moment, they can be managed conservatively by dual antiplatelet therapy as well as beta-blockers and anti-arrhythmic medications to prevent the dysrhythmia.[41]

Arrhythmia

All sorts of cardiac arrhythmia can develop during COVID-19 infection. This may be due to preexisting cardiac illness, or may be due to newly developed MI, myocarditis, pericarditis, hypoxia, drug interaction or electrolyte imbalance.[42] The way the arrhythmia to be dealt after COVID 19 is not well established, and needs further research. But at the moment they should be treated like conventional arrhythmia. Common causes of palpitation are sinus tachycardia, atrial and ventricular ectopic beats and atrial fibrillation.

Reduction of sympathomimetic bronchodilators and cough suppressants and explanation and reassurance of the benign nature of these arrhythmia to the patient, can help in most of the cases. In AF/atrial flutter the therapeutic choices of rate and rhythm control should be re-assessed, and long-term anticoagulation should be continued based on the CHA2DS2-VASc score. The need for permanent pacing in bradycardia and for catheter ablation, secondary prophylactic implantable cardiac defibrillator (ICD) or wearable defibrillator in ventricular tachyarrhythmia needs to be re-evaluated.[43]

Pulmonary pathology

More than a third of recovered patients develop fibrotic abnormalities in radiology. Also, 47 % of patients have an abnormal diffusion capacity of the lungs for carbon monoxide (DLCO), and 25 % have reduced total lung capacity (TLC). This seemed even worse in patients with severe disease.[44] This is added to the cardiac abnormalities, if there is any. The consequence of this fibrosis is not known and at the moment there is no treatment to reverse it. In long term if this fibrosis persists or progresses, it can produce pulmonary hypertension and right heart failure.

Hypertension

Many reports mentioned a high prevalence of hypertension among patients with COVID-19,[45-47] Increased incidence of CVD and CKD among the hypertensive patients may be one of the causes of high incidence and mortality in this group of patients. Because ACE inhibitors and ARBs may increase the amount of ACE2, whether these drugs should be discontinued during the COVID-19 pandemic has been a topic of discussion.[48,49] Number of studies conducted in China, Italy, Spain and New York have showed that there is no relation between prevalence and severity of COVID with ACE inhibitor or ARB.[50-55] Moreover, a recent meta-analysis showed the potential benefit of ACE inhibitors or ARBs in patients with hypertension.[56] None of the anti- hypertensive agents except loop diuretics were associated with an increased risk of COVID-19 in number of studies.[57,58] As the patients lose their follow up schedule and there is increased number of uncontrolled hypertension. There is also increased incidence of mental disorders in this pandemic period owing to, for example, anxiety, economic issues, and decreased physical activity—all of which can potentially compromise BP control. Similar things happened in the Great East Japan Earthquake in 2011, medical care for patients with hypertension was compromised ("disaster hypertension").[59] It is currently unknown whether the COVID-19 pandemic will affect BP control and the development of CVDs in the long term; nonetheless, it is necessary to carefully monitor each patient's blood pressure and control it with guideline directed medication.

Renin angiotensin aldosterone antagonist (RAS antagonist)

Severe acute respiratory-syndrome coronavirus-2 (SARS-CoV-2) host cell infection is mediated by binding to angiotensin- converting enzyme 2 (ACE2). Because angiotensin II receptor blockers (ARB), ACE inhibitors (ACEi), and mineralocorticoid receptor antagonists (MRA) increase ACE2 receptor expression, it was believed that the use of these agents may facilitate viral disease; thus, they should not be used in high-risk patients with cardiovascular disease. But considering the beneficial effect on cardiovascular conditions, anti-inflammatory, anti- fibrotic, and antithrombotic support as well as reduction in progression of vascular and/or cardiac remodeling and heart failure, most of the societies recommended to continue the treatment with RAS antagonists unless there is a specific contraindication.[60,61] In a recent study discontinuing ACEI or ARB therapy for 30 days did not affect the number of days alive and out of the hospital in patients hospitalized with mild to moderate COVID-19. There were no significant between-group differences in death, cardiovascular outcomes, or COVID-19 progression. Over all, this and other available data do not support routinely discontinuing ACEIs or ARBs among patients hospitalized with mild to moderate COVID-19 if there is an indication for their use.[62] In the post COVID patients with newly developed or preexisting cardiovascular condition RAS antagonist should be prescribed.

Heart Failure

Patients with HF may be at increased risk for severe disease and complications from COVID-19.[63] COVID-19 may cause or worsen HF through a variety of mechanisms including myocardial

ischemia or infarction, increased oxygen demand, elevations in pulmonary pressures, pulmonary embolism, myocarditis, stress cardiomyopathy, and diffuse cytokine release.[64] Burdens of HF incidence, prevalence, and undertreatment will likely grow as a result of new COVID-19-related heart disease, delays in the recognition and treatment of ischemic heart disease, rising unemployment, and loss of income and health benefits for large segments of the population.[63] Patients with HF hospitalized with COVID-19 are at high risk for complications, with nearly 1 in 4 dying during hospitalization.[65]

Patients should be treated with diuretics and vasodilators as per standard protocols. But the exact role of beta blocker, ACE inhibitor, ARB, ARNI, SGLT-2 inhibitors are not yet established in patients, regarding how long they should be prescribed and at what dose.

In the time of COVID infection there is postponement and cancellation of complete diagnostic and therapeutic procedures.[66] Once the patients with advanced HF are stabilized, these procedures include echocardiograms, stress testing, cardiopulmonary exercise testing, right heart catheterizations, coronary angiography, and implantation or interrogation of cardiac electronic devices should be done. Individualized risk assessment is needed when these procedures are done.[67]

Postural orthostatic tachycardia syndrome (POTS)

Recovered patients with post COVID syndrome may develop dysautonomia, in which changes in functioning of one of the components of the autonomic nervous system (ANS) adversely affect health. POTS is characterized by a sustained heart rate increment of >30 beats/min within 10 minutes of standing or head-up tilt. Cardiologic symptoms include chest pain, palpitations, exercise intolerance, and orthostatic intolerance. POTS can develop by a number of mechanisms- i) Hypovolaemia and deconditioning during the acute illness, ii) SARS-CoV-2 virus might infect and destroy extracardiac postganglionic SNS neurons, secondarily increasing cardiac SNS outflow in a manner analogous to neuropathic POTS. This might include splanchnic venous pooling or a failure of reflexive mesenteric vasoconstriction during orthostasis, iii) SARS-CoV-2 virus could invade the brainstem and alter functions of medullary centers, resulting in increased central sympathetic outflows in a manner analogous to takotsubo cardiopathy. There could be alterations in brain perfusion manifesting with "brain fog", iv) Autoimmunity against autonomic nervous system.[68]

The condition can be easily diagnosed by measuring pulse and blood pressure with change of posture, Valsalva maneuver and tilt table test. They should be managed by adequate hydration, betablocker and minimizing the dose of drugs which can exaggerate the condition. Physical training and special maneuvers which is used for treatment of other patients POTS can also be applied in this post-COVID POTS.

Pericardial disease

Pericardial involvement in COVID 19 infection is also common. In a study among young athletes with RT PCR positive for COVID 19, 27.1% patients have got pericardial involvement; 14.6% of them were symptomatic and 12.5% of them were asymptomatic.[69] Long term consequences of this pericardial disease is not known. The effect of conventional drugs like steroid, colchicine and NSAID is not known in these patients. But at the moment NSAID and colchicine may be the drugs of choice, and in resistant cases steroid can be considered.

Thromboprophylaxis

COVID 19 is a hypercoagulable state. Arterial or venous thromboembolism and major adverse cardiovascular events are common over 30 days in ICU patients with COVID-19.[70] Most of the hospitalized patients are treated with prophylactic thromboembolism. The role of post hospital thromboprophylaxis is debatable. The patients who develop thromboembolism (Deep vein thrombosis, pulmonary embolism or ventricular thrombus) should be treated with standard protocol with thromboprophylaxis for three months.[71] But it is not known whether they should get the thromboprophylaxis beyond three months in case of COVID.[72]

Cardiometabolic care

There is an association between cardiometabolic based chronic disorders and COVID 19. There is a

reduction in physical activity during this pandemic period as well as lack of follow up and healthy dietary style. All these conditions along with the acute illness and use of different drugs like steroid can lose the control of cardiometabolic status of COVID patients. This may lead to a condition like COVID related cardiometabolic syndrome (CIRCS).[73] Along with cardiovascular injury induced by COVID, this CIRCS can increase the incidence of cardiovascular diseases. To minimize these following measures should be taken-

- Restructure routines at home.
- Greater emphasis on healthy weight.
- Continue same healthy eating recommendations.
- Increase exercise time at home.
- Adapt sleep routines to "normal" schedule.
- No tobacco products.
- Abstinence from alcohol.
- Continue positive attitudes.
- Avoid overprescribing.
- Arrange telemedicine contacts.
- Improving infrastructure for comprehensive prevention plans to reduce CMBCD burden.

Investigations in Follow up

Though there is no definitely prescribed follow up schedule for Post COVID patients. This mainly depends on patient's severity and complications. During follow up some investigations should be done routinely, like- Complete blood count, ESR, C reactive protein, ferritin as inflammatory marker. Renal and liver functions are also to be evaluated. Troponin and d-Dimer can be done. Though there may be false positive results, a negative test will be reassuring.

ECG should be done to exclude arrhythmia including QT interval, features of myocardial infarction & myocarditis. Echocardiography should be done to detect any cardiac abnormality and evaluate the LV function, pericardial effusion and specially RV function. Stress tests may be required for professional athletes with history of COVID before joining their usual training programs.[74]

In Wuhan, fifty eight percent patients who recovered from COVID infection shows cardiac involvement in CMR. Most of them has got no LV dysfunction.[75] Their long-term consequences are not known. Cardiac MRI may be done if patient's clinical condition demands, specially to exclude myocarditis, myocardial infarction and ventricular dysfunction. In a study of a cohort of German patients recently recovered from COVID-19 infection, CMR revealed cardiac involvement in 78 patients (78%) and ongoing myocardial inflammation in 60 patients (60%), independent of preexisting conditions, severity and overall course of the acute illness, and time from the original diagnosis. As CMR is expensive and not readily available in every center, whether it should be routinely done in all cases is not known and the long-term effects of these changes in myocardium is also not known.

Many patients may have developed MI during COVID infection, which may be type I or type II. For evaluation of exact mechanism and further treatment modality coronary angiogram, standard or CT, may be done.

Self-care of the patients

Lifestyle changes- Dietary habit should be standard like primary and secondary prevention of cardiovascular diseases. Smoking should be avoided, because it increases the probability of cardiac and pulmonary complications. Limitation of drinking alcohol and caffein should be advised.

Exercise after COVID [76]

We don't know the impact of cardiopulmonary exercise in post COVID patients. But following rules may be followed-

- After recovery from mild illness- one week of low level stretching and strengthening before strenuous exercise.
- Mild post COVID symptoms- limit exercise to slow walking. Increase rest period if symptoms worsen. Avoid strenuous exercise.
- Persistent symptoms (such as fatigue, cough, breathlessness, fever): limit activity to 60% maximum heart rate until 2-3 weeks after symptoms resolve.
- Patients who had lymphopenia or required oxygen need respiratory assessment before resuming exercise.

- Intense cardiovascular exercise must be avoided for three months in all patients after myocarditis or pericarditis; athletes are advised to take three to six months of complete rest from cardiovascular training followed by specialist follow-up, with return to sport guided by functional status, biomarkers, absence of dysrhythmias, and evidence of normal left ventricular systolic function.[60-7]

Post COVID clinic

To support this huge patient population with COVID 'Post-COVID assessment clinics' should be established with the following criteria:

- Be available at least in every medical colleges and district level hospital, following clinician referral, to all affected patients, whether hospitalized or not.
- Have access to a multidisciplinary team of professionals to account for the multi- system nature of post-COVID syndrome.
- Have access to diagnostic tests.
- Ensure coverage of the population in that geography.
- Have a local communications plan for raising awareness within the clinical community
- Have an external communication plan for informing and raising awareness with patients

Multi-disciplinary approach

As COVID is a multi -system disease, many of the patients will have multiorgan disorder along with cardiovascular illness, like renal impairment, hepatic impairment, neurological problems, nutritional problems. So multi-disciplinary approach will be needed to treat these patients. They require a separate rehabilitation unit where physicians from required speciality, general physician, nutritionist, occupational therapist, physiotherapist and psychiatrists will work together.

Vaccination

Vaccines should be given to all the patients who are infected with COVID, once the active infection is over. Whether this should be repeated every year is not known yet.

Future research

Substantial cardiovascular abnormality develops in COVID patients who are hospitalized and ICU survivor, even in patients with mild or without symptom. Research activities are demanded to evaluate the acute and long- term consequences of these abnormalities and also how to manage it.

Conclusion:

Cardiovascular involvement of COVID is one of the important causes of short term and probably of long-term morbidity and mortality of COVID. And these patients most often have got multiorgan involvement. Post COVID cardiac syndrome is a new clinical condition. There is lack of adequate information regarding their management. All the COVID positive patients should go through a cardiac evaluation even after clinical improvement and those who has developed cardiovascular abnormality should go through long term follow-up schedule. We need more researches regarding their management and assessment of long-term consequences to minimize those.

Conflict of Interest - None.

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