

La IC aguda incidente se reconoció como una complicación en el 2% y la lesión miocárdica en el 10% de todos los pacientes hospitalizados con COVID-19.¹ Los niveles elevados de **NT-proBNP** al ingreso se asociaron con una mayor mortalidad,² y los microARN específicos de miocitos cardíacos aumentaron en pacientes críticos con COVID-19, lo que indica compromiso cardíaco.³ La disminución de las tasas generales de admisión por IC⁴ y las tasas más altas de mortalidad fuera del hospital⁵ durante el confinamiento se reconocieron como problemas alarmantes, lo que refleja la falta de acceso a la atención entre los pacientes con IC establecida. Los ensayos aleatorios demostraron la seguridad de la continuación de los IECA o ARB entre pacientes hospitalizados con COVID-19.⁶⁻⁷ El tratamiento con dapagliflozina no redujo significativamente la disfunción orgánica o la muerte, pero fue bien tolerado en pacientes hospitalizados con COVID-19 (ensayo DARE-19).⁸ La miocarditis surgió como una complicación rara de las vacunas de ARNm de COVID-19, especialmente en hombres jóvenes.⁹ La evaluación de riesgos y beneficios de la vacunación contra la COVID-19 fue favorable para todos los grupos de edad y sexo; y casi todos los pacientes con miocarditis tuvieron resolución de síntomas y signos.¹⁰

Las complicaciones a largo plazo de la infección por SARS-CoV-2 incluyen **taquicardia sinusal persistente, síndrome de taquicardia ortostática postural, arritmia auricular y miocardiopatía.** ¹¹

Entre los atletas que se recuperan de COVID-19, varios estudios de CMR informaron tasas y grados variables de anomalías cardíacas que sugerían miocarditis. ^{12, 13} La detección mediante troponina, ECG, ecocardiografía y CMR adicional y/o ecocardiografía de estrés si es anormal, resultó en que solo el 0.6 % de los atletas se restringieron para regresar a los deportes y ninguno tuvo eventos cardíacos. ¹⁴

Aunque la lesión miocárdica es común en COVID-19 y el ARN del SARS-CoV-2 se puede detectar en el corazón, la miocarditis es un diagnóstico patológico poco común que ocurre en el 4,5 % de los casos muy seleccionados que se someten a autopsia o biopsia endomiocárdica. ¹⁵ Durante la convalecencia después de una infección grave por COVID-19 con elevación de troponina, la CMR puede detectar una lesión similar a la miocarditis, sin embargo, con una extensión limitada y mínima. Figura 1)¹⁶

Figura 1

Hospitalizado con COVID-19

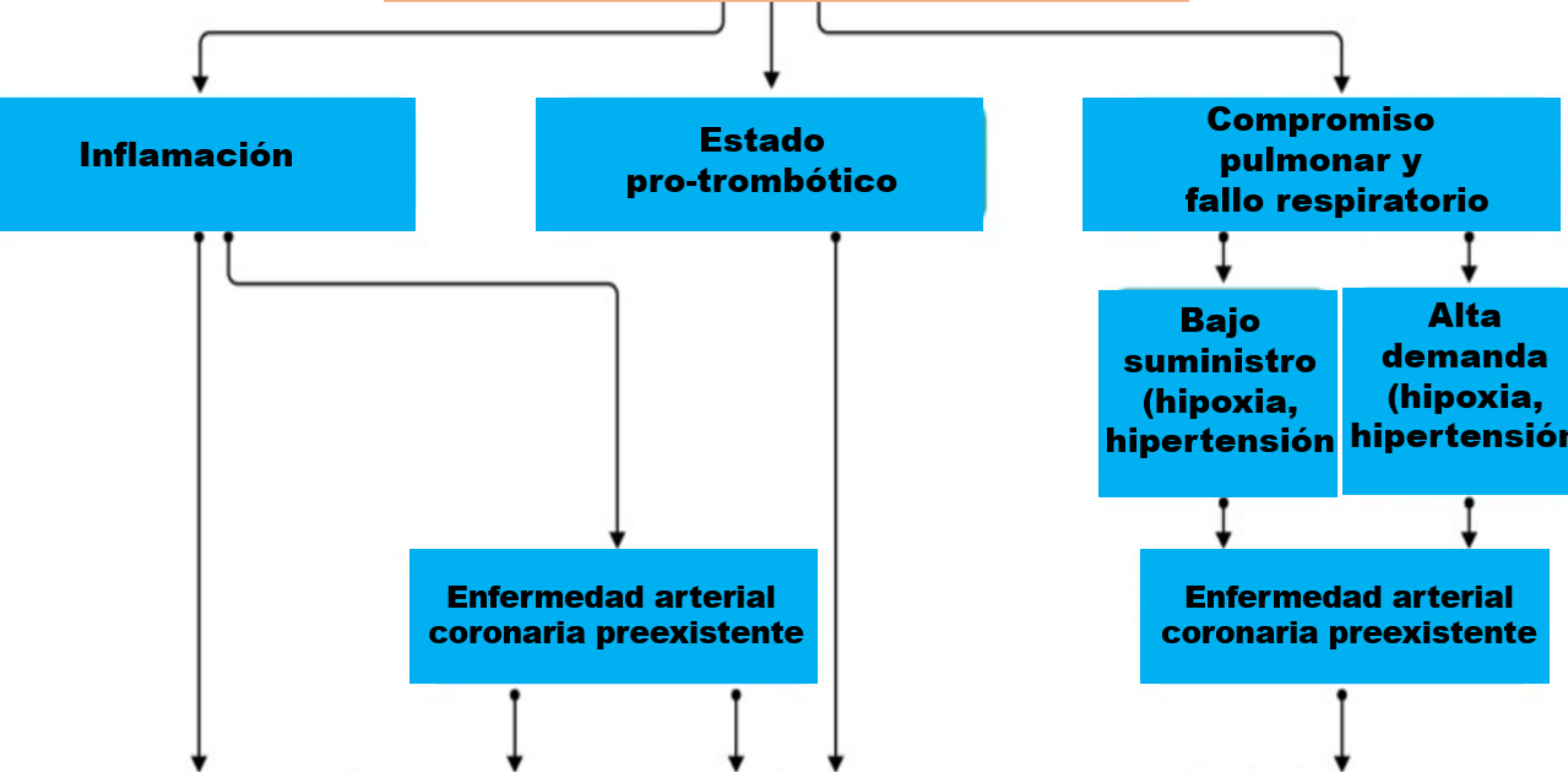
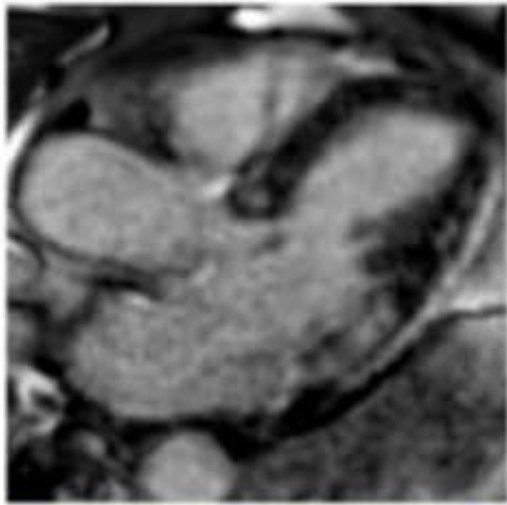
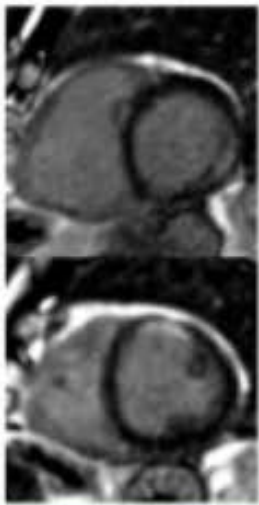


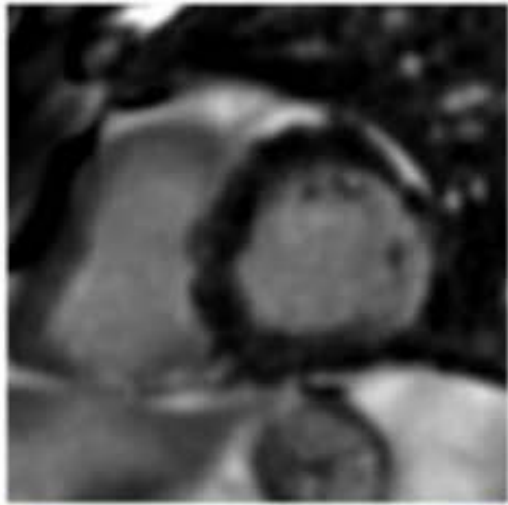
Figura 1



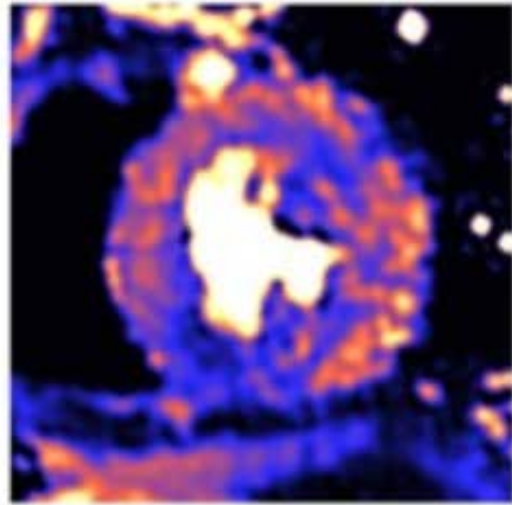
Miocarditis



Miocarditis +IM



IM



Isquemia

Lesión miocárdica con liberación de troponina

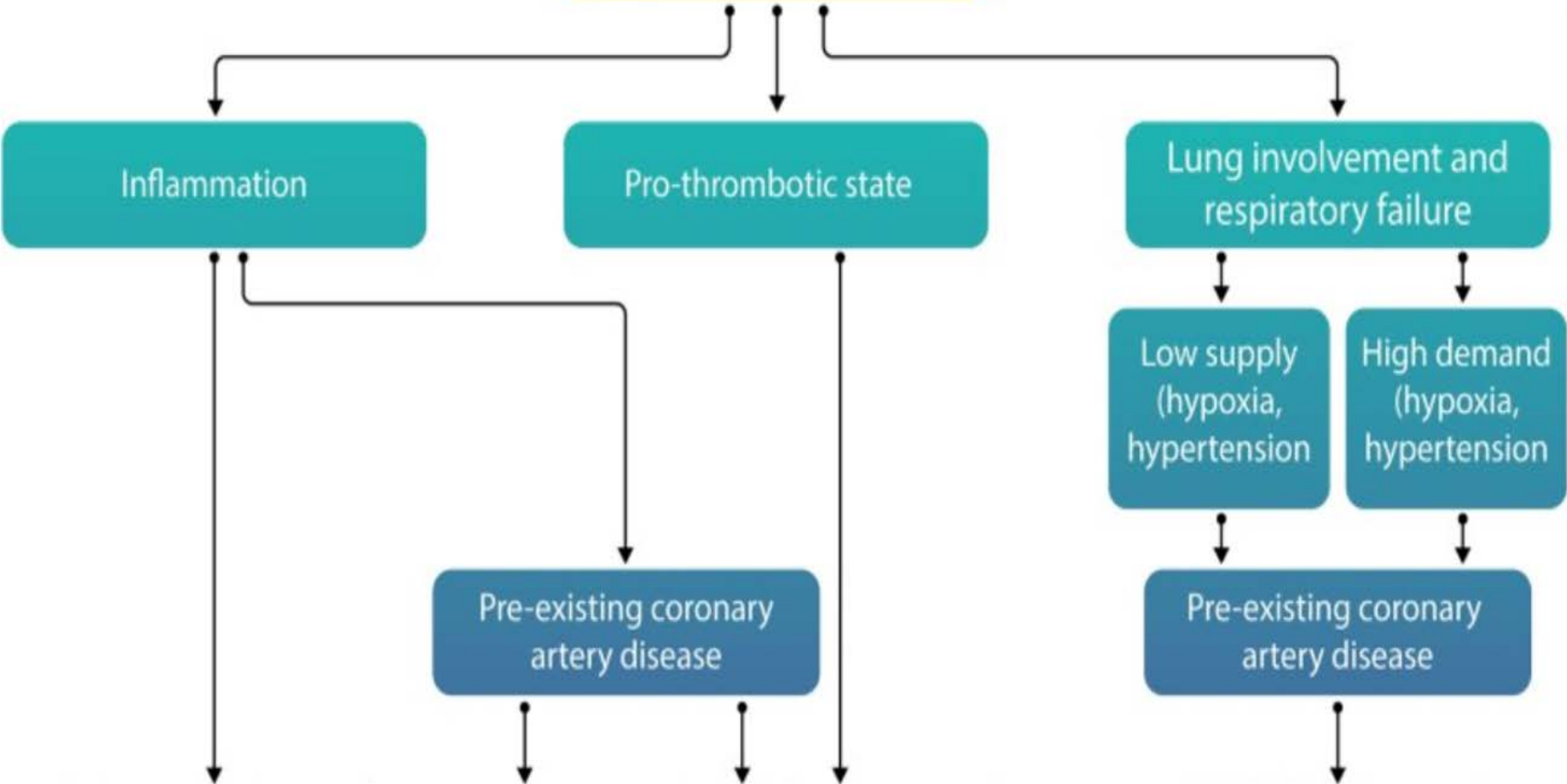
Heart failure during the COVID-19 pandemic

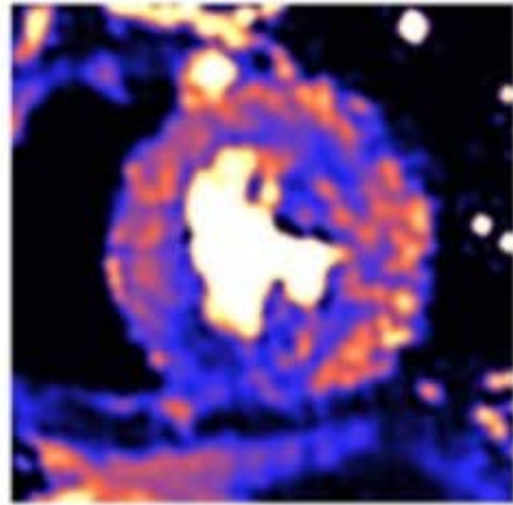
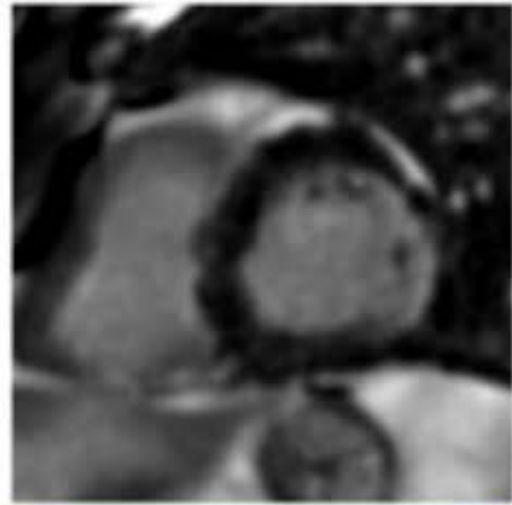
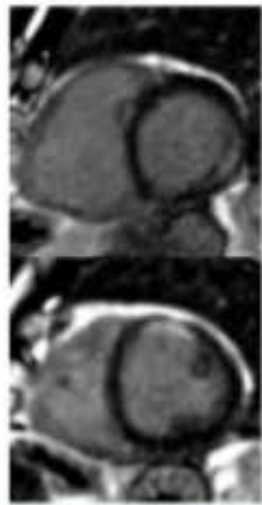
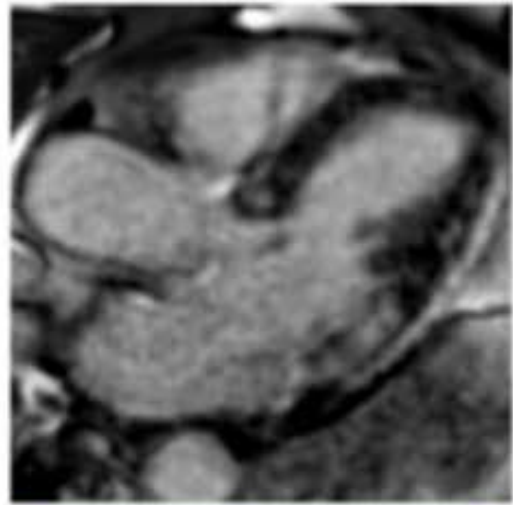
Incident acute HF was recognized as a complication in 2%, and myocardial injury in 10% of all patients hospitalized with COVID-19.¹ Elevated admission NT-proBNP levels were associated with higher mortality,² and cardiac myocyte-specific microRNAs were upregulated in critically ill COVID-19 patients indicating cardiac involvement.³ Declining overall admission rates for HF⁴ and higher out-of-hospital mortality rates⁵ during lockdown were recognized as alarming issues, reflecting lack of access to care among patients with established HF. Randomized trials demonstrated the safety of continuation of ACE inhibitors or ARB among patients hospitalized with COVID-19.^{6;7} Dapagliflozin treatment did not significantly reduce organ dysfunction or death, but was well tolerated in patients hospitalized with COVID-19 (DARE-19 trial).⁸ Myocarditis emerged as a rare complication of COVID-19 mRNA vaccinations, especially in young men.⁹ Benefit–risk assessment for COVID-19 vaccination was favorable for all age and sex groups; and almost all patients with myocarditis had resolution of symptoms and signs.¹⁰

Long-term complications of SARS-CoV-2 infection include persistent sinus tachycardia, postural orthostatic tachycardia syndrome, atrial arrhythmia, and cardiomyopathy.¹¹

Among athletes recovering from COVID-19, several CMR studies reported varying rates and degrees of cardiac abnormalities suggestive of myocarditis.^{12,13} Screening by troponin, ECG, echocardiography, and additional CMR and/or stress echocardiography if abnormal, resulted in only 0.6% of the athletes being restricted to return to sports, and none had cardiac events.¹⁴ Though myocardial injury is common in COVID-19, and SARS-CoV-2 RNA can be detected in the heart, myocarditis is an uncommon pathologic diagnosis occurring in 4.5% of highly selected cases undergoing autopsy or endomyocardial biopsy.¹⁵ During convalescence after severe COVID-19 infection with troponin elevation, myocarditis-like injury can be detected by CMR, however, with limited extent and minimal functional consequence (*Figure 1*).¹⁶

Hospitalised COVID-19





Myocarditis

Myocarditis + MI

MI

Ischaemia

Myocardial injury with troponin release

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