COVID-19 and cardiac considerations in the community

Our understanding of severe COVID-19 has moved on from its early characterisation as a pneumonitis, with frequent heterogenous cardiac and thromboembolic complications. We now understand its multisystem nature, from head-to-'COVID-toe', and protracted symptoms in around 10% of all people infected with SARS-CoV-2.[1] To date there has been little guidance for GPs on identification and management of cardiac effects, whether acute, sub-acute, or chronic, which is therefore the focus of this editorial.

ACUTE MYOCARDIAL INJURY

Studies of patients hospitalised with COVID-19 have reported the prevalence of 'acute myocardial injury', defined as an acute rise of cardiac troponin T/I, to be 12%–36%.[2] The umbrella term 'acute myocardial injury' encompasses ischaemic causes, including Type 1 myocardial infarction (MI) due to atherosclerotic plaque disruption; and Type 2 MI due to microthrombosis, or to demand-perfusion mismatch. Non-ischaemic causes include myocarditis, stress cardiomyopathy, or right ventricular strain from pulmonary embolism (Figure 1).[2] Acute myocardial injury is associated with a worse prognosis in COVID-19, particularly in the older population and those with cardiovascular comorbidities.[2] The association with worse outcomes during hospitalisation led to the National Institute for Health and Care Excellence publishing a rapid guidance for hospital practitioners.[3] The array of cardiac effects supports the hypothesis that *'COVID-19 is, in the end, an endothelial disease'*.[4]

POST-ACUTE AND CHRONIC CARDIAC INJURY

The prevalence and implications of post-acute cardiac injury are less clear. At first sight, a recent community study of 100 German patients recovering from COVID-19, of whom only one-third had been hospitalised, suggests a large proportion (78%) have cardiac abnormalities when assessed at a single center with cardiovascular magnetic resonance (CMR) imaging.[5] Subsequent debates[6] highlight possible methodological and statistical issues, but similar high rates of CMR-detectable myocardial abnormalities were reported elsewhere in very small studies; namely 58% (15/26) of Chinese,[7] 56% (9/16) of Hong Kong,[8] and 66% (19/29) of UK[9] study participants who were also recovering in the community.

While acute myocardial injury, defined as acute rise and fall of cardiac troponin, in hospitalised patients with COVID-19 is associated with excess risk, the short-term clinical significance and long-term implication of CMR abnormalities in recovering patients are unknown.[6] Nor is it apparent to what extent cardiorespiratory pathophysiology explains the persistent fatigue, dyspnoea, or chest pain in up to 74% of patients 2–3 months post-admission.[1,10] Large population based multi-organ studies are underway to address these questions. Perhaps we should be reassured that most of the patients in the CMR studies had normal ventricular function, N-terminal pro B-type natriuretic peptide (NT-proBNP) levels, and cardiac troponin levels at the time of the scan. This suggests that, at around 1–2 months after presentation with COVID-19, heart failure was not a complication of the abnormalities seen on CMR. Despite speculation,[5,6] progression to future heart failure is as yet unproven.

Theoretically, myocardial injury can present at any stage after a viral infection.[3,6,11] Myocardial injury during a viral respiratory illness is not surprising as it was reported during the avian influenza A(H7N9), SARS, and MERS pandemics.[11,12] Influenza infection is associated with a doubling of short-term acute myocardial infarction rates.[13] Myocarditis can be driven by immediate and delayed immune responses, with some patients progressing to develop dilated cardiomyopathy.[11,14]

DIAGNOSTIC CHALLENGES FOR GPs

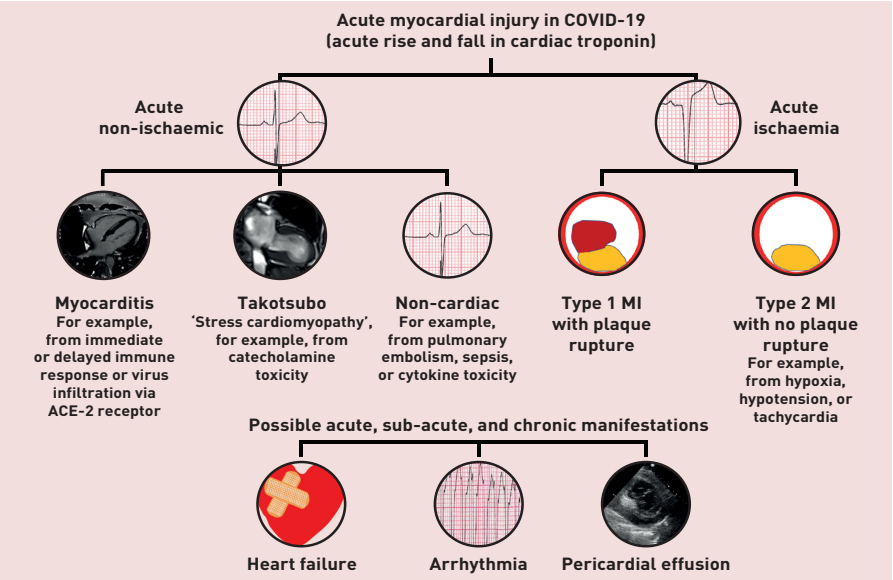
Pending longitudinal studies, GPs need to reassure the majority of patients who have post-COVID-19, but be alert to the development of acute myocardial injury in the early weeks, or delayed complications (for example, pericardial effusion, arrhythmias, and heart failure) (Figure 1).[3,4,8] While most suspected cases would merit referral to secondary care,[3,12] GPs may occasionally find themselves managing patients declining

Box 1. GP's toolkit for post-COVID-19 patients with cardiac symptoms

1. Clinical history and examination.
2. Electrocardiogram.
3. BNP or NT-proBNP.
4. Cardiac troponin.[a]
5. Referral for direct access outpatient echocardiogram.
6. Referral for cardiology review.

[a]Includes Troponin T and Troponin I. NT-proBNP = N-terminal pro B-type natriuretic peptide.

Figure 1. Possible mechanisms and clinical manifestations of acute myocardial injury in COVID-19. MI = myocardial infarction. ACE-2 = angiotensin converting enzyme 2.



hospital attendance or whose ceiling of care does not include admission. In this situation, for diagnostic assistance, cardiac troponin (Troponin I or Troponin T) levels might be measured but interpretation may be difficult. Evaluation of a potential myocardial infarction should be familiar territory but in COVID-19 alternative causes of cardiac troponin rise need consideration (Figure 1). A rise and fall in cardiac troponin helps distinguish acute myocardial injury from causes of chronic elevation such as heart failure or renal failure. In myocarditis, the magnitude of the cardiac troponin rise is variable and electrocardiogram (ECG) findings such as arrhythmias, ST changes, or QRS prolongation are non-specific. In post-viral pericarditis, no troponin rise is expected. Typically, the chest pain is relieved by sitting up and forward but resolves if an effusion develops. Classic ECG findings like concave ST elevation with PR segment depression are often absent but a targeted echocardiogram can be diagnostic. Where heart failure is suspected, natriuretic peptides may be requested, being mindful of false positives and negatives. In all patients provoking concern, the combination of a detailed history, examination (incorporating remote measurements where helpful), blood profiling, and ECG are needed, and often, advice from secondary care (Box 1).

COMMUNITY MANAGEMENT CONSIDERATIONS

If a GP diagnoses pericarditis or myocarditis outside a secondary care setting, and if specialist involvement remains inappropriate, symptom control is required. In acute post-viral pericarditis analgesia is generally achieved with nonsteroidal anti-inflammatory drugs (for example, ibuprofen) when there is no evidence of heart or renal failure. Low dose colchicine is a helpful adjunct in post-viral pericarditis to reduce the likelihood of tamponade or chronic restrictive pericarditis.[15] Pending clinical trials,[11] current management of myocarditis hinges around pain control with simple analgesia, suppression of arrhythmias, and heart failure symptoms. Excepting rare cases of sudden cardiac death, myopericarditis and myocarditis (of all aetiologies) generally carry a good long-term prognosis. Resumption of competitive exercise should be delayed for 3–6 months,[15] or until symptoms, ECG, biomarkers, and cardiac dysfunction if seen on imaging have all normalised.[16] The short- and longer-term impact of 'mild' COVID-19 on the heart in patients with no formal diagnosis of myocardial injury is unknown at the time of writing[14,16] but longitudinal studies across the disease spectrum are underway. Pending further evidence we suggest that following 'mild' COVID-19, asymptomatic patients do not need to restrict exercise, but patients developing cardiac symptoms merit cardiology referral and an interim precautionary approach with avoidance of intense exercise.

Understanding the various, sometimes co-existing, elements of acute myocardial injury in this multisystem disorder could help GPs make sense of discharge summaries of patients admitted with severe COVID-19 and evaluate newly presenting patients. GPs in the UK and their international counterparts need to stay vigilant for occult, residual, or delayed cardiorespiratory disease while trying not to add anxiety to patients afflicted by a traumatic hospital admission or an unexpectedly delayed recovery from seemingly 'mild' COVID-19, either of which can have far-reaching biopsychosocial effects.

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