Heartbeat: an increase in preventable cardiovascular deaths during the COVID-19 pandemic due to avoidance of medical care

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Patients with cardiovascular disease (CVD) have an increased mortality risk with COVID-19 infection yet several studies have shown fewer hospital-based CVD diagnoses and procedures during the COVID-19 pandemic. In this issue of *Heart*, Wu and colleagues[1] show that despite a decrease in the number of patients presenting with an acute CVD event there was an 8% excess of CVD deaths in England between March and June 2020 (during the COVID-19 pandemic), compared with the previous 6 years (figure 1). About ½ of these deaths occurred outside the hospital with the most frequent causes of CVD death being stroke (35.6%), acute coronary syndrome (24.5%), heart failure (23.4%) pulmonary embolism (9.3%) and cardiac arrest (4.6%). Most of these deaths were not related to a known COVID-19 infection, suggesting they were most likely due to delays in seeking medical care or undiagnosed COVID-19 infection.

As Singh and Newby[2] emphasise in an editorial: 'the evidence presented by Wu and colleagues[1] provides us with an important message to our patients and society: it is important to seek emergency medical attention for symptoms indicative of serious life-threatening cardiovascular disease even during the height of the pandemic. Here, the risk of fatal stroke and myocardial infarction outweighs the COVID-19 risk to the patient, and the healthcare system had capacity within acute specialities outside of the intensive care and dedicated COVID-19 units to provide life-saving treatments. This ultimately begs the question: is the fear of disease worse than the disease itself?'

Another important study in this issue of heart describes a 12-year cohort study of 419 patients with infective endocarditis in South Korea.[3] Overall, hospital mortality was 14.6% with risk factors for mortality including aortic valve infection, *Staphylococcus aureus*, neurological complications multi-organ failure, and an increased number of comorbidities. Surgical intervention was associated

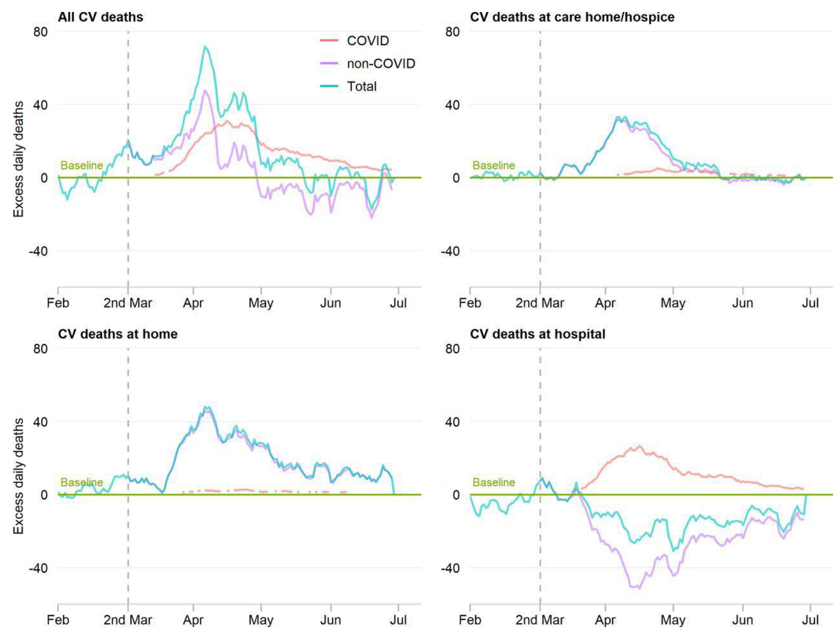

Figure 1 Time series of acute cardiovascular (CV) deaths, by place of death. The number of daily CV deaths is presented using a 7-day simple moving average (indicating the mean number of daily CV deaths for that day and the preceding 6 days) from 1 February 2020 up to and including 30 June 2020, adjusted for seasonality. The number of non-COVID-19 excess CV deaths each day from 1 February 2020 were subtracted from the expected daily death estimated using Farrington surveillance algorithm in the same time period. The green line is a zero historical baseline. The red line represents daily COVID-19 CV death from 2 March to 30 June 2020; the purple line represents excess daily non-COVID-19 CV death from 2 March to 30 June 2020 and the blue line represents the total excess daily CV death from 1 February to 30 June 2020.

with a markedly lower risk of in-hospital mortality (OR 0.25, p<0.001) and improved long-term outcomes (figure 2).

'We could (and should) do better' in preventing and treating infective endocarditis plead Scully *et al*.[4] They conclude that: 'As the present data from South Korea demonstrate, IE remains associated with poor outcomes and its incidence is increasing in many countries around the world. Greater public health awareness is warranted alongside renewed emphasis on education of patients at risk (with particular regard to prompt symptom reporting and maintenance of good oral and cutaneous hygiene), early diagnosis, timely referral and specialist care. Once suspected or diagnosed, early involvement of a dedicated Endocarditis Team is essential in managing these patients combined with early, appropriate antibiotic therapy and decisions regarding the need for surgery and its timing.'

Another interesting paper in this issue of *Heart* by Onishi and colleagues[5] describes the diagnosis and outcomes of triglyceride deposit cardiomyovasculopathy (TGCV) which is seen in about 20% of haemodialysis patients with suspected coronary artery disease. At median follow-up of 4.7 years, the composite primary endpoint of CVD death, non-fatal myocardial infarction and non-fatal stroke occurred in 52.3% of the definite TGCV patients compared with 27.3% in those with probable TGCV and 9.1% of the non-TGCV patients. In the accompanying editorial, Nakajima[6] explains the causes of TGCV and discusses the diagnostic approach. In brief, 'The principal disorder in TGCV is defective intracellular lipolysis, which causes excessive triglyceride accumulation in

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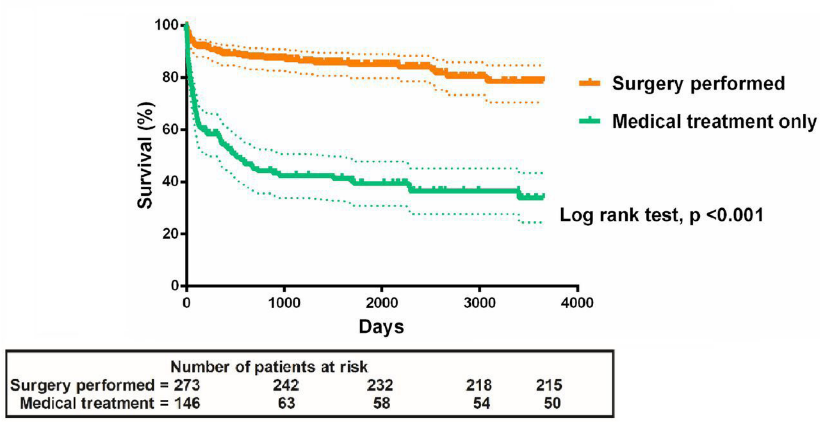

Figure 2 Kaplan-Meier curves of the long-term survival rates of patients with infective endocarditis who underwent surgery versus those who underwent medical treatment only.

the myocardium and coronary artery vascular smooth muscle cells, leading to heart failure and coronary artery disease with a poor prognosis.' Diagnosis is based on the presence of impaired long-chain fatty acid metabolism or triglyceride deposition in the myocardium in combination with clinical major and minor criteria and supportive items.

The *Education in Heart* article in this issue[7] reviews the prevalence and predictors of neurocognitive and psychosocial impairment among adults with congenital heart disease followed by a discussion of how these issues can be mitigated over the patient's lifespan.

Readers will also want look at the review article[8] on the emerging mechanistic models that link atrial fibrosis, atrial fibrillation and stroke given the implications of these models for new approaches to prevention of adverse clinical events (figure 3). Boyle *et al* outline 'a vision of a future paradigm integrating simulations in formulating personalised treatment plans for each patient.'

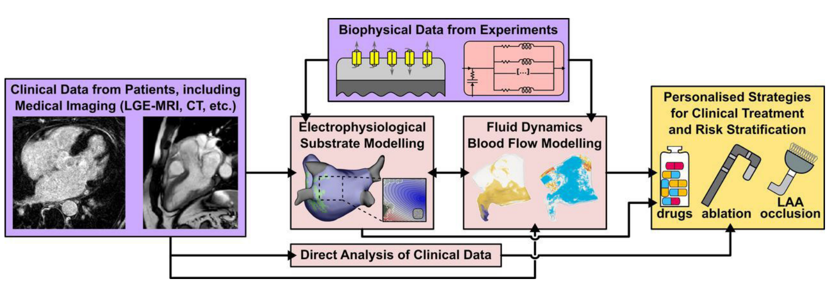

Figure 3 Schematic for envisioned use of modelling and simulation to augment imaging, resulting in better, personalised treatment strategies for patients who had stroke, atrial fibrillation or both. Electrophysiological simulations facilitate detailed assessment of patient-specific consequences of fibrotic remodelling. Computational fluid dynamics simulations enable prediction of thrombus formation and can be further integrated with modelling tools to reflect the coagulation cascade and clot transport towards the brain. Both modelling methodologies integrate medical imaging with measurements from biophysical experiments to produce patient-specific predictions that can be integrated with direct analysis of clinical data to produce better treatment options (eg, custom-tailored drug dosing, recommendations for ablation procedures or appendage closure). LAA, left atrium appendage; LGE-MRI, late-gadolinium enhancement-MRI.

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