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COVID-19, A Complex Multisystem Clinical Syndrome

I think this is now a good time to review and understand the COVID-19 infection and to look at presentation and complications.

A helpful article in The BMJ of 1 May 2020 by Dr Michael Roberts and others helps us to assess this complex clinical syndrome.

The COVID-19 infection can start as a local upper respiratory tract infection but can then spread to affect multiple organ systems with subsequent consequences.

Following spread in the body the multi-system critical illness can be associated with a high risk of death. This is caused by a combination of specific host defence responses with associated inflammatory activity and micro and vascular involvement with distinct coagulopathy (clotting) and a strong propensity to develop thromboembolic complications.

This hyperinflammatory tissue response goes hand in hand with a compromised circulatory system which leads to multi-organ dysfunction which affects the lungs, heart, kidneys, nerves, muscles, gastrointestinal tract and brain.

Those patients who are most severely affected have a cytokine storm which is characterised by very high levels of pro-inflammatory cytokines and tumour necrosis factor A, interleukin, granulocyte-colony stimulating factor and several chemokines. These are features of the inflammatory response and occur to suppress the infection; however, in COVID-19 they go into overdrive.

Those patients at highest risk of this multi-system failure and significant mortality are males and those with pre-existing hypertension or coronary disease. This is because there is expression which is located on the X chromosome.

This widespread nature reflects the ability of the SARS-CoV-2 virus to infect the endothelial cells with the likely release of cytokines which makes them more adhesive, sticky and hence increased coagulation. Patients with COVID-19 manifest with hypercoagulability, increased clotting with high levels of D-dimer, fibrinogen and Factor VIII. This leads to venous thromboembolism and a mixture of large proximal pulmonary artery thrombi and fibrin micro-thrombi in the presence of vascular inflammation. Thus, in the lungs, the post-mortem samples from critically ill patients have shown that there was local haemorrhage. Within the cardiovascular system, cardiac enzyme release is observed even in the early stages – this suggests myocardial inflammation and damage. Cardiologists report that patients present with chest pain as the primary symptom in the absence of temperature and other disease manifestations.

Renal failure is also a common complication of severe COVID-19 and there may be direct infection of the proximal renal tubular cells with SARS-CoV-2 which causes the renal insult. There, however, may also be indirect injury through endothelial inflammation or as a consequence of medical management. Perversely, reduced fluid administration to minimise pulmonary vascular congestion may increase pressure in the renal vein impairing renal blood flow.

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Gastrointestinal and liver dysfunction is shown by deranged tests of liver function and slow tolerance of the enteral feeding and occur frequently in severe illness.

In 50% of the most severely ill patients, neurological abnormalities are found. These include impaired consciousness, acute cerebrovascular events, and muscle diseases. It is thought that SARS-CoV-2, may directly involve and invade the central nervous system and neuromuscular tissue, both via a vascular route and by retrograde neuronal transmission.

COVID-19 is therefore a complex clinical syndrome and not a straightforward viral pneumonia.

Sadly, there is an ever present risk of damage by the medical treatment that the patient is being given. Early management of all these multisystem issues is essential. An early proactive management to support renal function can improve later survival in intensive care and aggressive thromboprophylaxis can reduce pulmonary emboli, while understanding the extent of organ involvement guides the management decision taking.

What about long-term consequences?

It is too early to say but they are likely to include a post-intensive care syndrome. More than a quarter of patients suffer cognitive impairment with variable duration, psychological distress and physical issues.

The sequalae of COVID-19 infection itself is yet unknown but if one looks at SARS survivors, there is a sustained reduction in lung and exercise capacity with high levels of psychological distress.

Follow-up of all these patients is essential.

The London General Practice has kept itself abreast of all the current research involving testing, diagnosis and management of COVID-19 infections. So long as a patient is not suffering with current acute COVID-19 infection, we are happy to undertake face to face consultations and home visits as appropriate wearing full PPE protection.

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