Thromboprophylaxis and anticoagulation in COVID-19 infection

A striking feature of COVID-19 infection is the acute phase response (APR). Several pro-coagulant factors are positive acute phase reactants: Factor VIII, VWF, Fibrinogen and the APR is associated with an increased risk of thrombosis. Published data and local experience confirm that fibrinogen is often markedly elevated in the COVID patients.

Pneumonia and sepsis are often complicated by DIC, but although COVID patients do have abnormalities of coagulation, they are not typical of DIC. The most marked abnormality is an elevation of d-dimer but without a parallel fall in platelets or prolongation of clotting times. This suggests that local rather than disseminated thrombin generation and fibrinolysis is taking place.

The site of thrombin and fibrin formation appears to be the lungs, based on limited post-mortem data and clinical observations from CT scans and ventilation parameters (V/Q mismatch). Some patients have overt pulmonary emboli, but in others it is presumed to be microvascular thrombi.

The above is consistent with limited evidence from China indicating that patients receiving prophylactic dose UFH/LMWH had significantly better survival than those who did not.

In response to these findings the COVID-19 Treatment Guidelines Working Group, with expert input from haematology, makes the following recommendations for medical patients admitted with suspected or confirmed COVID-19 in Level 1 and 2 beds, unless such agents are contraindicated:

D-Dimer	Weight	LMWH
<1000	<100kg	Enoxaparin 40mg OD
	100-150kg	Enoxaparin 40mg BD
	>150kg	Enoxaparin 60mg BD
1000-3000	<100kg	Enoxaparin 40mg BD
	100-150kg	Enoxaparin 80mg BD
	>150kg	Enoxaparin 120mg BD
>3000	<100kg	Tinzaparin 175 units/kg OD

As per usual practice, all doses may need adjusting based on renal function. In the absence of bleeding, coagulopathy is not a contraindication to heparin unless platelets fall below 30 for prophylaxis or below 50 for therapeutic heparin.