**COVID-19 HAEMATOLOGY GUIDANCE version 1.0**

* In light of new data and guidelines this SOP will be regularly reviewed and updated. Please ensure that you are using the most up to date version

**COVID-19 Disseminated Intravascular Coagulation**

Early data from Wuhan, China suggests that DIC is more commonly seen in patients with severe disease and may be a predictor of increased mortality (Tang et al, 2020).

A FBC, clotting screen, fibrinogen and D dimers should be undertaken on all patients with suspected or proven COVID-19. These can be requested as a test group (DIC score) on tQuest or individually.

The results can be used to calculate a DIC score (BSH H+T Task Force 2020), supporting the presence or absence of DIC, and therefore may be used to aid decision-making when used in conjunction with other clinical parameters.



Consideration should be given to repeating this every 24-48 hours, especially if on ICU/HDU or if clinical deterioration.

**Treatment**

**No active bleeding / No planned significant procedure:**

Maintain platelet count >20. Request a single unit of platelets only.

Do not treat abnormal INR/APTT/fibrinogen parameters.

Do not give tranexamic acid / Prothrombin complex concentrate / recombinant factor VIIa

**Active Bleeding**

Maintain platelet count >50 in the first instance.

Transfuse FFP 15-25mls/kg to maintain INR/APTT ≤ 1.5

Transfuse 1-2 adult doses cryoprecipitate to maintain fibrinogen ≥ 1.5 (or fibrinogen concentrate off-license)

Do not give tranexamic acid / Prothrombin complex concentrate / recombinant factor VIIa

For major bleeding please follow the major haemorrhage protocol. Suggest discuss all bleeding COVID-19 patients with evidence of DIC, with the SpR or Consultant Haematologist on-call.

Tang, N., Li, D., Wang, X. & Sun, Z. (2020) Abnormal Coagulation parameters are associated with poor prognosis in patients with novel coronavirus pneumonia. *Journal of thrombosis and haemostasis : JTH***,** 1–4.

British Society for Haematology, Haemostasis and Thrombosis Task Force, 2020, b-s-h.org.uk/media/18151/dic-score-in-covid-19-pneumonia\_19-03-2020.pdf

Hunt B, Retter A, McClintock C. 2020. Practical guidance for the prevention of thrombosis and management of coagulopathy and disseminated intravascular coagulation of patients infected with COVID-19 **https://thrombosisuk.org/covid-19-thrombosis.php**

**COVID-19 Venous Thromboembolism Guidance**

**Key points:**

* Patients with COVID-19 are at **high** VTE risk. **All patients should have VTE risk assessment and pharmacological prophylaxis administered within 14h of admission unless contraindicated**. There is evidence to support survival advantage in patients who received thromboprophylaxis.

**Recommendations:**

**On admission**

* On admission perform VTE risk assessment as per Trust guidelines
* Prescribe/administer pharmacological thromboprophylaxis as per Trust guidelines

**Once confirmed COVID-19 positive**

* Prescribe pharmacological thromboprophylaxis if **platelets >50x109/L** unless other contraindications (e.g. active bleeding, invasive procedure planned within 12h, prescribed and taking therapeutic anticoagulation, fibrinogen <1.0g/l).

 **Note** abnormal INR/APTT alone is **NOT** a contraindication to prophylactic anticoagulation

* All completely immobilised patients would benefit from intermittent pneumatic compression in addition to pharmacological thromboprophylaxis
* In patients at high VTE risk (e.g. previous VTE within last 3 months, or previous VTE on anticoagulation, or underlying malignancy) in the absence of bleeding a **lower platelet threshold >30 x109/L** is considered safe to give prophylactic dose anticoagulation. If in doubt please discuss with haematology consultant.
* Mechanical thromboprophylaxis should be used alone if platelets <30 x109/L or bleeding
* **Consider** switching patients already on oral anticoagulants (warfarin or DOACs) for e.g. stroke prevention or previous VTE to LMWH because of lower risk of drug interactions
* **Consider** possibility of pulmonary embolism (PE) in patients with sudden onset of oxygen desaturation, respiratory distress or hypotension

**Thereafter**

* Reassess VTE risk regularly and especially if there is a change in clinical condition.

Hunt B, Retter A, McClintock C. 2020. Practical guidance for the prevention of thrombosis and management of coagulopathy and disseminated intravascular coagulation of patients infected with COVID-19 **https://thrombosisuk.org/covid-19-thrombosis.php**

**COVID-19 Related Haemophagocytic Lymphohistiocytosis**

Reactive or secondary haemophagocytic lymphohistiocytosis (HLH) is an underecognised hyperinflammatory syndrome caused by excessive immune activation. This results in hypercytokinaemia and haemophagocytosis, and is typically characterised by:

* Persistent fever
* Cytopenias
* Hyperferritinaemia
* Hyperfibrinogenaemia
* Hypertriglyceridaemia
* Hepatosplenomegaly
* (Multi-) organ failure
* Evidence of haemophagocytosis on tissue biopsy

In a subset of COVID-19 patients, a cytokine storm consistent with or mimicking HLH has been identified, possibly as a predictor of worse outcomes.

Suggested baseline investigations in addition to usual COVID investigations:

* FBC
* Clotting screen
* Fibrinogen
* LFT
* Ferritin
* LDH
* Triglycerides
* Soluble CD25 on discussion with haematology

These results can be used to calculate the HScore, to support the clinical decision making but should not be used to exclude HLH:

<https://www.mdcalc.com/hscore-reactive-hemophagocytic-syndrome>

Investigations to exclude alternative secondary causes should be undertaken as directed by the haematology team.

Usual treatment for secondary HLH is immunosuppression but concern has been raised about this in COVID-19 patients. Other options include IVIG or selective cytokine blockade although no standardised treatment has been agreed. Please discuss any case of possible HLH with the haematology consultant on-call.

Mehta P, McAuley D, Brown M, Sanchez E, Tattersall R, Manson J. 2020. COVID-19: consider cytokine storm syndromes and immunosuppression.  *The Lancet Volume 395, 1033-1034*