

# Hematological finding in Covid-19

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# Introduction

- \* It is well documented that COVID-19 is primarily manifested as a **respiratory tract infection**
- \* Emerging data indicate that it should be regarded as a **systemic disease** involving multiple systems including cardiovascular, respiratory, gastrointestinal, neurological, hematopoietic and immune system.
- \* The possible mechanisms could be systemic inflammatory response, stasis, and SARSCoV- 2 – Angiotensin converting enzyme (ACE2) binding and **direct endothelial cell damage**

# Introduction

- \* Endothelial cell injury induced by viral infection activates a multitude of **pro-inflammatory cytokines** such as interleukin IL-1, IL-6, and TNF-alpha
- \* Hyper-cytokemia that often lead to **multi-organ dysfunctional syndrome** COVID-19 has a **high rate of hospitalization and mortality**.
- \* Multiple hematologic parameters in COVID-19 patients can **predict prognosis as well as severity of illness** thus may help with proper triage of patients within the hospital.
- \* Herein, we summarize some hematologic findings and complications of COVID-19 and we provide guidance for early prevention and management of the latter.

# Thrombosis and hemostasis and COVID-19

- \* **Coagulation disorders** are relatively frequently encountered among COVID-19 patients, especially among those with **severe diseases**.
- \* The up-regulation of tissue factor (TF) - activated VIIa complex is associated with **thrombin generation and fibrin deposition** in various organs including the **bronco-alveolar system**.
- \* **Thrombotic complications** have thus emerged in COVID-19 patients as an **important aspect** of their clinical presentation
- \* some form of **anticoagulation** is being used in many COVID-19 **treatment** protocols.

# D-Dimer

## (Thrombosis and hemostasis and COVID-19)

- \* Studies so far have suggested that **coagulopathy associated** with COVID-19 (CAC) is predominantly a **pro-thrombotic DIC with elevation in D-dimer levels**, and fibrinogen levels, and a **decrease in anti-thrombin levels**.
- \* The clinical implications of these coagulopathy perturbations are pulmonary congestion with **microvascular thrombosis and micro occlusion with an increased rate of thromboembolic events**, central line thrombosis, and strokes
- \* Guan et al. in their study on 1099 hospitalized COVID-19 patients found **elevated D-dimers ( $\geq 0.5$  mg/L) as an indicator of severe illness**.
- \* Tang et al. reported D-dimers as a mortality predictor with the **median value of  $2.12 \mu\text{g/ml}$  (range  $0.77\text{--}5.27 \mu\text{g/ml}$ ) in the non-survivors patients, compared to  $0.61 \mu\text{g/ml}$  (range  $0.35\text{--}1.29 \mu\text{g/ml}$ ) in the survivors**
- \* Huang et al. was that a **higher at admission median d-dimers was associated with an increased chance of requiring critical care**
- \* Based on these findings, D-dimer has become a reliable marker of prognosis, hospital mortality, and need for ICU level care

# Anti-phospholipid antibodies

## (Thrombosis and hemostasis and COVID-19)

- \* These antibodies are well known to rise **transiently in various infections** (tuberculosis, syphilis, Human immunodeficiency virus, hepatitis C) or other auto-immune disorders
- \* in a study in France reported that out of 56 patients with COVID-19, 25 **cases (45%) had lupus anticoagulant (LAC)** positive
- \* Zhang et al. reported three patients with COVID-19 who were evaluated for cerebral infarctions and peripheral limb **ischemia with digit discoloration**.
- \* Mao et al. reported **4 cases of ischemic stroke** in their 214 patient's data
- \* Li et al. reported an incidence of **stroke as 5%** among hospitalized patients
- \* **Large vessel stroke** in 5 COVID-19 patients younger than 50 yrs was recently reported by Oxley et al.
- \* Hence, so far significance of **antiphospholipid** antibodies in precipitating **ischemic/thrombotic events** in COVID-19 patients is unknown and needs further evaluation.

# Thrombocytopenia

## (Thrombosis and hemostasis and COVID-19)

- \* Thrombocytopenia is a marker of poor clinical outcome and was associated with a three-fold enhanced risk of worsening disease.
- \* The platelet count was lower in patients with very severe COVID-19. Twenty percent of COVID-19 patients who died had a platelet count  $<100 \times 10^9/l$ , compared with 1% of survivors.
- \* Very low platelet counts of  $<20 \times 10^9/l$ , or a sudden fall in the platelet count  $>50\%$  over 24-48 hours can occur in the pre-terminal stages of COVID-19
- \* Underlying liver issues, drug side effects, heparin-related thrombocytopenia (HIT), primary hematological disease (Immune thrombocytopenia, Thrombotic thrombocytopenic purpura etc.), impending DIC, viral infection and overt inflammatory response are few of the many such causes

# Thrombocytopenia

(Thrombosis and hemostasis and COVID-19)

Few of the potential mechanisms proposed are:

- \* **Consumptive** thrombocytopenia.
- \* **Reduced release** in the peripheral circulation.
- \* Robust **auto-immune response** against platelets and platelet destruction.
- \* Virus directly infecting the hematopoietic stem cells, megakaryocytes, and platelets (via CD13 or CD66a) and **apoptosis**.



# Leukocyte in Covid-19

- \* Several studies demonstrated that **neutrophilia** that might be related to the **cytokine storm** (absolute neutrophil count above the normal range;  $3-7.5 \times 10^9/L$ )
- \* **lymphocytopenia** (lymphocyte count  $< 1.5 \times 10^9/L$ ) were present in severe cases of COVID-19 pneumonia and were associated with **poor prognosis**.
- \* Neutrophil to lymphocyte ratio (**NLR**) has also been found to **predict disease severity in the early stages** of SARS CoV-2 infection.
- \* In contrast, small studies reported a significant reduction in granulocytes in severe as compared to non-severe patients.

- \* Lymphocytopenia is a reliable indicator of early SARS CoV-2 infection and helps in tracing of contacts besides assessment of disease progression along the course of COVID-19 pneumonia.
- \* In the clinical practice of treating patients with COVID-19, we have observed that the NLR of severe patients is higher than that in mild patients
- \* When NLR was  $\geq 4.21$ , and the patient's age was  $< 50$ , the sensitivity and specificity were 70.3% and 93.7%, respectively
- \* strong correlation between NLR and CRP might suggest the use of NLR to differentiate between non-severe and severe cases, especially in a remote healthcare facility.



**Thanks for  
attention**