## **ROLE OF IRON CHELATION IN COVID**

SARS-CoV-2 infection is characterized by a protean clinical picture that can range from asymptomatic patients to life-threatening conditions.

Severe COVID-19 patients often display a severe pulmonary involvement and develop neutrophilia, lymphopenia, and strikingly elevated levels of IL-6.

There is an over-exuberant cytokine release **with hyperferritinemia** *leading to the idea that COVID-19 is part of the hyperferritinemic syndrome spectrum.* 

Indeed, very high levels of ferritin can occur in other diseases including hemophagocytic lymphohistiocytosis, macrophage activation syndrome, adult-onset Still's disease, catastrophic antiphospholipid syndrome and septic shock.

Numerous studies have demonstrated the immunomodulatory effects of ferritin and its association with mortality and sustained inflammatory process.

High levels of free iron are harmful in tissues, especially through the redox damage that can

lead to fibrosis. (eg, in patients of Thalassemia receiving Blood requires thus regular Iron chelation)

Iron chelation represents a pillar in the treatment of iron overload.

## It was proven to have an anti-viral and anti-fibrotic activity.

## In this small write up the Criteria is:

Inclusion Criteria: Clinical diagnosis of COVID-19 Disease

Exclusion criteria

- Previous history of allergy to Deferoxamin,
- Pregnancy,
- kidney dysfunction



Evidence from the literature and a compelling hypothesis on the potential immunomodulatory, iron chelating and anti-oxidant effects of iron chelators (**Desferioxamine**) in the treatment of COVID-19 and its complications.

Interestingly, iron chelation has been shown in vitro to suppress endothelial inflammation in viral infection, which is the main pathophysiologic mechanism behind systemic organ involvement induced by SARS-CoV-2, by inhibiting IL-6 synthesis through decreasing NF-kB. Iron chelators exhibit iron chelating, antiviral and immunomodulatory effects in vitro and in vivo, *particularly against RNA viruses*.

These agents could attenuate ARDS and help control SARS-CoV-2 via multiple mechanisms including:

- inhibition of viral replication
- decrease of iron availability
- upregulation of B cells
- improvement of the neutralizing anti-viral antibody titre.
- inhibition of endothelial inflammation and

• prevention of pulmonary fibrosis and lung decline via reduction of pulmonary iron accumulation.

Both retrospective analyses of data in electronic health records, as well as proof of concept studies in humans and large RCTs are needed to fully elucidate the efficacy and safety of iron chelating agents in the therapeutic armamentarium of COVID-19, probably as an adjunctive treatment.