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Is it possible to use radiotherapy to kill intrapulmonary COVID 19 to stop its destroying effect on lungs?

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In this critical era of the Covid 19 pandemic. I hope my idea may help worldwide patients who become contaminated by this aggressive virus.

I Asked Myself at the beginning of this pandemic: Is there a direct way to kill coronavirus to save the patient as there is no up till now any vaccine or treatment particularly for the deteriorated cases when the virus arrived at the lung started destroying it. At this stage, is it possible to destroy the viruses inside the lungs by using radiotherapy? Even the dead coronavirus patients can be sterilized by radiotherapy before burring them.

It is mentioned in the literature Genomes (DNA and RNA) are the major targets for the biological effects of ionizing radiation in killing microbes. The main cause of virus inactivation is protein damage. The dose of radiation required to inactivate an infectious virus or its nucleic acid is much greater under direct than under indirect conditions. **(**1, 2**)**.

The world uses the classic ways in the treatment of covid 19 by antimalarial drugs without complete cure of patients. So, treating Covid 19 patients by radiotherapy could be promising if tried on severe cases to save them.

I think applying this new idea "Radiotherapy" can come true if studied by a radiotherapist.

For my question, many of the researchers and Doctors shared their responses and their work as below.

Radiotherapy targeted to areas of the lung involved by COVID-19 may inactivate the virus, with coronaviruses requiring 1Mrad (10 KGy) for this to occur. However, the acute radiation-associated toxicities may be severe in such patients. This is especially true for COVID-19 patients with several pre-existing co-morbidities. These may include radiation pneumonitis, pericarditis and oesophagitis.

In addition, although most late toxicities will develop in the period following the COVID-19 infection, these toxicities, such as pulmonary fibrosis and pleural effusions, will further reduce lung function following recovery.

Reports of up to a 30% loss in lung volume following COVID-19 recovery have already arisen, thereby highlighting the importance of preserving lung function.

Although modern radiotherapy techniques allow for more precise irradiation, such as Stereotactic body radiotherapy, the dose required for viral inactivation is still significant. To my knowledge, viral inactivation using radiation is only performed successfully in vitro, where no dose constraints are required. This is done for highly virulent viruses to be made safer to handle and test.

Radiotherapy for COVID-19 also presents a logistical issue, which is often overlooked. Planning radiotherapy treatments is time-consuming and requires significant resources. Moreover, due to the current situation, radiotherapy departments are working with reduced staffing and alterations in protocols. This, therefore, limits the amount of COVID-19 patients who can be treated with radiotherapy. Bringing COVID-19 positive patients into a radiotherapy department would also put oncology patients at risk, especially since such patients may be immunocompromised.

Therefore, radiotherapy would not be a suitable alternative treatment for COVID-19 for all patients. However, it may be utilized in select cases taking the appropriate precautions to safeguard other patients, given further research to evaluate whether low doses of radiotherapy are effective. As I previously stated, however, I do not recommend radiotherapy for such a purpose due to the development of early and late radiation-associated toxicities.

Some of the studies show pulmonary disorder due to radiotherapy dose collimation as appeared in the below result of PET scan images that can be acquired online (Affidea-irmet-covid19-paziente-asintomatico-jpg).

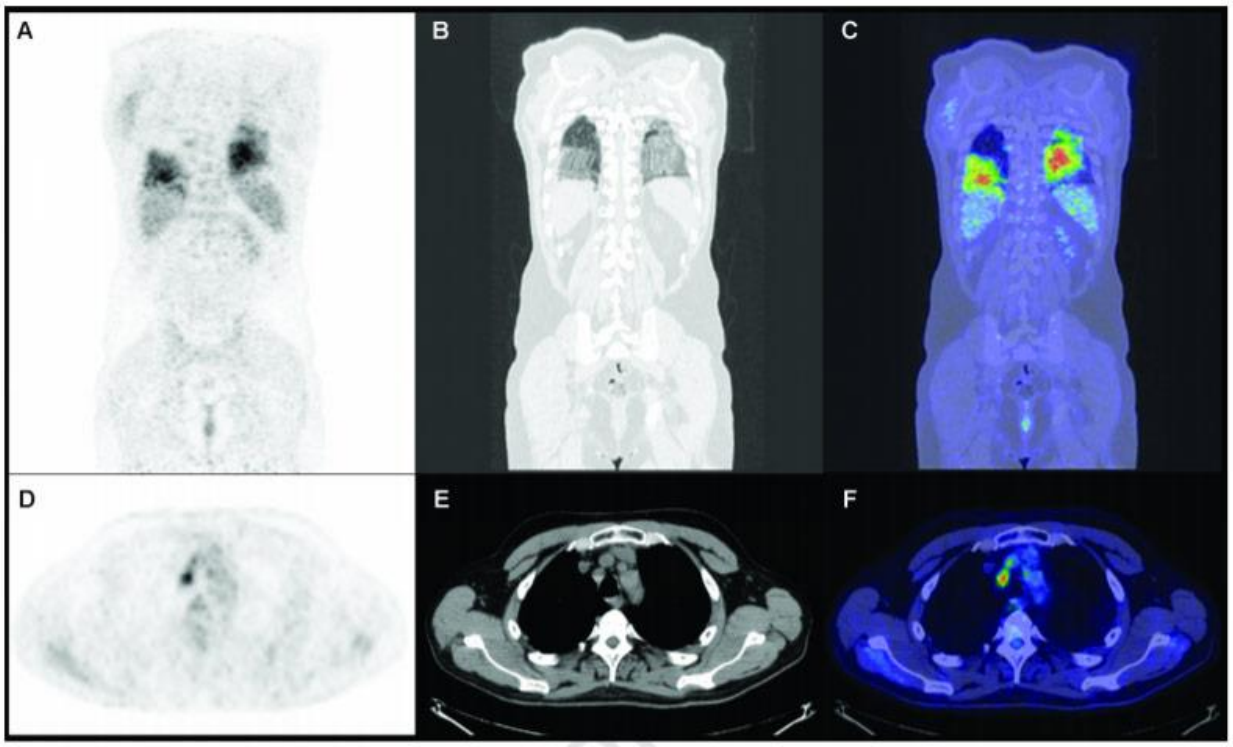

Figure 1

Some researchers presented the updated discovery of COVID 19 disease pathogenesis which will alter the trend of treatment. They advised reading this from doctors in China, it all makes sense and this is how COVID-19 we should be treated.

[They presented trials to understand novel Corona Virus in form of question & answer]

In COVID- 19

-Why there is a high ferritin level?

-Why there are very high DDimer levels disproportionate to the severity of infection?

-Why ARDS in those are nearly

Not responding to high PEEP and Fio2 levels?

-Why all CT chest patterns are exclusively Ground glassing and associated with rapid and marked hypoxemia disproportionates with the geographical CT findings size?

- Why did Early Chinese protocol for Covid includes high dosing of systemic steroids which is questioned and rejected by WHO?

- Why body immune response against Covid is not like other respiratory viruses by lymphocytosis (Cytotoxic T cells and Natural killer cells), instead body prefers to react against covid by phagocytosis (monocytosis)?

- Why does Covid Attack mature red blood cells while it is one of the body cells that does not contain nucleus and DNA?

-Why Critically ill Patients are responding well to anticoagulation, Hydroxychloroquine and novel antiviral Favipiravir?

The following molecular pathogenesis is the only one for the time being that can answer all these questions.

COVID -19 may not cause pneumonia either typical or atypical or classical ARDs. It seems like we are dealing with a presumed wrong disease.

The Key pathogenic molecular step of SARS-Cov2 is to attack the 1-Beta Chain of Hemoglobin and hunt the porphyrins dissociating the iron from it and releasing iron into the circulation.

Thus, Hb loses its capacity to bind with oxygen, so oxygen is not supplied to major organs. That is why we see resistant hypoxia coupled with very rapid multi-organ failures.

Moreover, the free iron released into the circulation is so toxic that it causes powerful oxidative damage to the lungs.

Free iron toxicity causes inflammation of alveolar macrophages- leads to CT scan characteristic changes.

The body tries to compensate for this by elevating the rate of Hb synthesis which explains why Hb is high in those patients.

Another compensatory mechanism to deal with such iron load is increasing ferritin production (iron store) which explains the very high ferritin levels observed in those patients.

Therefore, the cause of monocytosis is the body needs excess macrophages to engulf the excess iron load.

And the cause of Lymphopenia is the WBCs differentiation favored towards monocytes line rather than lymphocytes line.

This makes ferritin a bad prognostic marker (too much iron means too much Hb lost its O2 carrying capacity).

Also, this iron load and increased Hb production lead to increased blood viscosity with recurrent and diffuse micro and macro circulatory thrombosis that is why there are high levels of DDimer in those patients and this explains the cause of sudden deterioration and death in some sporadic cases

This disseminated thrombosis is proved by postmortem examinations of ARDS victims (it is not a real ARDS).

This theory could explain why we are losing patients so rapidly and why mechanical ventilation is not so effective in the treatment and using ARDS mechanical ventilation protocol is not causing any benefit. actually, it could be futile and cause more lung damage.

On the other hand, this is crucially explaining the very rapid and good response of those patients to full therapeutic anti-coagulation.

Chloroquine as the antimalarial drug is working by protecting Hb against invasion by malaria parasites. It is doing the same here but just protecting the Hb against invasion by the virus.

The chemical components in chloroquine phosphate compete with the porphyrin and bind to the viral protein, thereby inhibiting the viral protein's attack on heme or binding to the porphyrin.

Favipiravir is the latest anti-novel coronavirus drug with specific therapeutic effects.

In Favipiravir, the most critical ligand is 1RP, which is 6 - fluoro - 3 - oxo - 4 - (5 - O - phosphono - beta - D - ribofuranosyl) - 3, 4 - dihydropyrazine - 2 - carboxamide.

Favipiravir cannot be bound to E2 glycoprotein and Nucleocapsid, and its binding energy to viral envelope protein, ORF7a, orf1ab is higher than that to porphyrin.

It is useful to note that the binding energy of Envelope protein and Favipiravir is more than 2700 times the binding energy of porphyrin.

The primary function of Envelope protein is to help the virus enter host cells, which shows that Favipiravir can effectively prevent the virus from infecting human cells. **(**3, 4, 5**)**.

Recommendations: -

According to these clinical observations, correlations and understandings:

1-Hydroxychloroquine, Favipiravir and early full coagulation therapy should be involved as early as possible in the management protocol of Covid-19.

According to French, Covid-19 disease is considered a catastrophic antiphospholipid vasculitis syndrome & it is not ARDS.

It has been proposed that a very low dose of radiotherapy (50 to 100 cGy) to the entire lung could be used in the treatment of covid-19. Pay careful attention to the fact that the intended use of radiotherapy in this setting would not be to kill the SARS-Cov2 virus, but to prevent the inflammatory cytokynes storm that can be lethal for many patients. **(**6**)**

Some studies in labs shows that the low dose of ionizing radiation can be used to activate the virus on study for increasing its numbers by multiplication in the plate media. But to prepare a vaccine against that virus, a high dose of ionizing radiation is used to kill firstly the virus. I think there is a level of radiation that may be previously studied that it will not affect the nature of the virus during applying such low radiation to treat classic infectious pneumonia. What is the dose of low radiotherapy which avoids activating the virus during treating the lung? I think it is important to test that on diseased animals with COVID 19 to study the effect of low radiotherapy on the virus side aside the lungs to know the safe dose which will be useful to the lung without activating intrapulmonary COVID 19 virus.

Is there any risk of COVID 19 activation during low-dose radiotherapy?

Ionizing radiation-induced viral reactivation has long been known, e.g., in hepatitis virus (HBV, HCV), HIV, and Epstein-Barr virus, but I have recognized no data relevant to SARS-CoV-2 and other coronaviruses. As such, I do not think that there is an underpinning scientific basis sufficient to discuss the possibility for radiogenic reactivation of coronaviruses, although I am not in a position to rule out that possibility. **(**6, 7**)**.

It is mentioned in the literature that there are currently no approved treatments for COVID-19, some have suggested that 0.5 to 1 Gy of whole thorax radiation therapy would present a very low risk to COVID-19 patients in a clinical trial. Some researchers introduced the concept of LDRT and recommended doses were as low as 100 mGy. Moreover, to reduce any potential risk, in their model patients receive a conditioning dose of 2mGy. This dose not only maximizes the anti-inflammatory effects of the main dose (100/180/250 mGy), but reduces the risk of cancer and any potential circulatory disease.

Conclusions:

- The evidence of radiotherapy's role in the treatment of Covid-19 patients is still low.

- Experimental study on animals is recommended to confirm the effectiveness of low versus high dose radiotherapy on the Covid-19 virus in vivo.

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