**NOTE: This is a condensed, summarized version of an interview with a frontline medical practitioner in Seattle. To be updated with the actual interview soon.**

*This is an 'easier-to-read' version regarding what the ICU doctor shared with us from Seattle on his/her experience with treating/managing COVID19:*

Regarding COVID19: some of these patients are healthy with no other medical problems in their 20s. Currently the entire intensive care unit is for critical patients. The regular hospital floor beds are being used for stable patients and those at the end of life. The progressive care unit (like an ICU but a little less serious) and half of the ER are for patients with respiratory symptoms.

Personal protection equipment (PPE) for healthcare providers is inadequate.

Clinical information- agree with data from China of the patients that are confirmed sick with this virus. Of those confirmed 80% are mildly ill, 14% are sick enough to be hospitalized, and 6-8% are critically ill. However the data are skewed by under-testing and the number of elderly getting comfort-only care.

Perfectly healthy young people are also getting sick, needing ICU and ventilator support, and dying. The time-frame to death is about a week.

COVID19 has some unique features: 1/3 have mild lower respiratory symptoms (cough, shortness of breath) and need oxygen by nasal cannula. Another 1/3 are sicker and require face masks (or oxygen non-rebreather masks). And, finally, 1/3 are intubated (tube into their airway on a ventilator) with acute respiratory distress syndrome (ARDS).

These sick patients seem to all have normal white blood cell (WBC) count with low lymphocytes. Sometimes there are elevated neutrophils, but combined with the low lymphocytes the total WBC is normal. And it is stable even at 10 days. The BAL (bronchoalveolar lavage- which is when sterile saline is put into the airway, and then sucked out, and sent to the lab for analysis) shows elevated lymphocytes (despite the fact that they were low in the blood). But try not to do a BAL on these patients.

They have high, intermittent, persistent fevers for over 10 days. It is not the medication, it's the virus. The procalcitonin levels are low and checking it initially and then watching its trend may be useful to help watch for ventilator-associated pneumonia (because in VAP the levels rise).

Liver function tests (AST/ALT and sometimes alk phos) increase; but without full hepatitis. It seems like higher levels at admission heralded deterioration and progression to severe lung disease and respiratory failure.

There appears to be evidence emerging that several patients have died from a form of fulminant viral myocarditis, causing severely reduced LV ejection fraction. Arrhythmias tend to be rare, and this does not look like Takatsubo cardiomyopathy. CPK levels tend to be elevated and may be useful as a screening test on hospital admission. Some providers are even checking twice daily to monitor for this complication.

CXR is always a bilateral, patchy, or reticular infiltrates that are sometimes peri-hilar. May look like a cardiac issue (like early heart failure with a backup of fluid into the lungs), but the heart function (ejection fraction, EF) is normal and the patient is dehydrated at presentation. This CXR finding has always been present! CT scans have not been more helpful than CXRs.

When respiratory failure occurs it is FAST. It may be happening about 7-10 days from symptom onset, but very rapid from the time of hospital admission. Common scenario is: admit the patient on 1 liter of oxygen by nasal cannula (a very little bit). Twelve hours later the patient needs more help with a device that pushes oxygenated air into their nose/mouth and down their airway into their lungs (NPPV, think like a CPAP machine). Twelve to 24 hours later they are intubated, on a ventilator, turned onto their bellies (pronated) in the ICU, and on Flolan. With this treatment, their oxygen levels aren't as bad as with H1N1.

Patients are dying from this from cardiac arrest and not respiratory failure.

Because of the rapid deterioration from onset and presentation to respiratory failure, it's important to intubate early. Sometimes people try a facemask, but if they need high-flow oxygen by nasal cannula, or positive pressure to push air into the airway, they are going to need a tube- so go ahead and do that early.

We have not seen multi-organ system failure. Liver seems to react some, and maybe the kidneys too, but no florid failure. Except heart failure- we are seeing heart failure which is how the patients die.

Regarding the heart: we have patients who have normal functioning hearts and normal heart ultrasounds. Normal heart labs too. These patients then go into the described respiratory failure (but don't have shock, blood pressure is okay, no sepsis). Then they start looking good. They get off the Flolan, are turned over onto their backs, and weaned off the ventilator.

Then, 12 hours later they are suddenly in shock. Cold, clammy, and blood pressure drops and they need pressors (medication to keep the BP up). Their heart fails with the EF (ejection fraction) going from normal (55-70%) to critical at less than at 10%. Then they either develop a deadly arrhythmia, like ventricular tachycardia, ventricular fibrillation, to pulseless electrical activity, to asystole in less than one day! Actually, the most common rhythm we see is asystole.

Therefore there appears to be a viral cardiomyopathic component (i.e. the virus seems to attack the heart directly later in the course).

Treatments: Remdesivir (originally developed for Ebola) seems to help the ARDS, but we don't have enough to give everyone the recommended 10 days, so we do it until they seem better-ish. Steroids are questionable. It is likely that steroids increase ventilator associated pneumonia and/or hospital acquired pneumonia. The general consensus here is that steroids do more harm than good, to the extent that inhaled steroids should be stopped.