From: [redacted]
Sent: Sunday, March 15, 2020 9:03 PM
To: [redacted]
Subject: RE: read this!

An email found it's way to me from a colleague… pretty crazy stuff, and likely a look into our future…

This is from a front-line ICU physician in a Seattle hospital.

This is his personal account:

* we have 21 pts and 11 deaths since 2/28.
* we are seeing pts who are young (20s), fit, no comorbidities, critically ill. It does happen.
* US has been past containment since January
* Currently, all of ICU is for critically ill COVIDs, all of floor med/surg for stable COVIDs and EOL care, half of PCU, half of ER. New resp-sx pts Pulmonary Clinic offshoot is open
* CDC is no longer imposing home quarantine on providers who were wearing only droplet iso PPE when intubating, suctioning, bronching, and in one case doing bloody neurosurgery. Expect when it comes to your place you may initially have staff home-quarantined. Plan for this NOW. Consider wearing airborne iso PPE for aerosol-generating procedures in ANY pt in whom you suspect COVID, just to prevent the mass quarantines.

* we ran out of N95s (thanks, Costco hoarders) and are bleaching and re-using PAPRs, which is not the manufacturer's recommendation. Not surprised on N95s as we use mostly CAPRs anyway, but still.

*terminal cleans (inc UV light) for ER COVID rooms are taking forever, Enviro Services is overwhelmed. Bad as pts are stuck coughing in the waiting room. Rec planning now for Enviro upstaffing, or having a plan for sick pts to wait in their cars (that is not legal here, sadly).

* **CLINICAL INFO** based on our cases and info from CDC conf call today with other COVID providers in US:
* the Chinese data on 80% mildly ill, 14% hospital-ill, 6-8% critically ill are generally on the mark. Data very skewed by late and very limited testing, and the number of our elderly pts going to comfort care.
* being young & healthy (zero medical problems) does not rule out becoming vented or dead - probably the time course to developing significant lower resp sx is about a week or longer (which also fits with timing of sick cases we started seeing here, after we all assumed it was endemic as of late Jan/early Feb).
* based on our hospitalized cases (including the not formally diagnosed ones who are obviously COVID - it is quite clinically unique) about 1/3 have mild lower resp sx, need 1-5L NC. 1/3 are sicker, FM or NRB. 1/3 tubed with ARDS.
* Thus far, everyone is seeing:
    ** nl WBC. Almost always lymphopenic, occasionally poly-predominant but with nl total WBC. Doesn't change, even 10days in.

** BAL lymphocytic despite blood lymphopenic (try not to bronch these pts; this data is from pre-testing time when we had several idiopathic ARDS cases)
** fevers, often high, may be intermittent; persistently febrile, often for >10d. It isn't the dexmed, it's the SARS2.
** low ProCalc; may be useful to check initially for later trending if later concern for VAP etc.
** up AST/ALT, sometimes alk phos. Usually in 70-100 range. No fulminant hepatitis. Notably, in our small sample, higher transaminitis at admit (150-200) correlates with clinical deterioration and progression to ARDS. LFTs typically begin to bump in 2nd week of clinical course.
** mild AKI (Cr <2). Uncertain if direct viral effect, but notably SARS2 RNA fragments have been identified in liver, kidneys, heart, and blood.
** characteristic CXR always bilateral patchy or reticular infiltrates, sometimes perihilar despite nl EF and volume down at presentation. At time of presentation may be subtle, but always present, even in our pts on chronic high dose steroids. NO effusions. CT is as expected, rarely mild mediastinal LAD, occ small effusions late in course which might be related to volume status/cap leak.

    * Note - China is CT'ing everyone, even outpts, as a primarily diagnostic modality. However, in US/Europe, CT is rare, since findings are nonspecific, would not change management, and the ENTIRE scanner and room have to be terminal-cleaned, which is just impossible in a busy hospital. Also, transport in PAPRs. Etc. 2 of our pts had CTs for idiopathic ARDS in the pre-test era; they looked like the CTs in the journal articles. Not more helpful than CXR.

** when resp failure occurs, it is RAPID (likely 7-10d out from sx onset, but rapid progression from hospital admit). Common scenario for our pts is, admit 1L NC. Next 12hrs -> NPPV. Next 12-24hrs -> vent/proned/Flolan.
** interestingly, despite some needing Flolan, the hypoxia is not as refractory as with H1N1. Quite different, and quite unique. Odd enough that you'd notice and say hmmm.
** thus far many are dying of cardiac arrest rather than inability to ventilate/oxygenate.
** given the inevitable rapid progression to ETT once resp decompensation begins, we and other hosps, including Wuhan, are doing early intubation. Facemask is fine, but if needing HFNC or NPPV just tube them. They definitely will need a tube anyway, & no point risking the aerosols.
** no MOSF. There's the mild AST/ALT elevation, maybe a small Cr bump, but no florid failure. except cardiomyopathy.
** multiple pts here have had nl EF on formal Echo or POCUS at time of admit (or in a couple of cases EF 40ish, chronically). Also nl Tpn from ED. Then they get the horrible resp failure, sans sepsis or shock. Then they turn the corner, off Flolan, supined, vent weaning, looking good, never any pressor requirement. Then over 12hrs, newly cold, clamped, multiple-pressor shock that looks cardiogenic, EF 10% or less, then either VT->VF-> dead or PEA-> asystole in less than a day. Needless to say this is awful for families who had started to have hope.
** We have actually had more asystole than VT, other facilities report more VT/VF, but same time course, a few days or a week after admit, around the time they're turning the corner. This occurs on med-surg pts too; one today who is elderly and chronically ill but baseline EF

preserved, newly hypoTN overnight, EF<10. Already no escalation, has since passed, So presumably there is a viral CM aspect, which presents later in the course of dz.
** of note, no WMAs on Echo, RV preserved, Tpns don't bump. Could be unrelated, but I've never seen anything like it before, esp in a pt who had been HD stable without sepsis.

Treatment -

* Remdesivir might work, some hosps have seen improvement with it quite rapidly, marked improvement in 1-3 days. ARDS trajectory is impressive with it, pts improve much more rapidly than expected in usual ARDS.
    * Recommended course is 10d, but due to scarcity all hosps have stopped it when pt clinically out of the woods - none have continued >5d. It might cause LFT bump, but interestingly seem to bump (200s-ish) for a day or 2 after starting then rapidly back to normal - suggests not a primary toxic hepatitis.
* unfortunately, the Gilead compassionate use and trial programs require AST/ALT <5x normal, which is pretty much almost no actual COVID pts. Also CrCl>30, which is fine. CDC is working with Gilead to get LFT reqs changed now that we know this is a mild viral hepatitis.
* currently the Gilead trial is wrapping up, NIH trial still enrolling, some new trial soon to begin can't remember where.
* steroids are up in the air. In China usual clinical practice for all ARDS is high dose methylpred. Thus, ALL of their pts have had high dose methylpred. Some question whether this practice increases mortality.
* it is likely that it increases secondary VAP/HAP. China has had a high rate of drug resistant GNR HAP/VAP and fungal pna in these pts, with resulting increases mortality. We have seen none, even in the earlier pts who were vented for >10d before being bronched (prior to test availability, again it is not a great idea to bronch these pts now).
* unclear whether VAP-prevention strategies are also different, but wouldn't think so?
* Hong Kong is currently running an uncontrolled trial of HC 100IV Q8.
* general consensus here (in US among docs who have cared for COVID pts) is that steroids will do more harm than good, unless needed for other indications.
* many of our pts have COPD on ICS. Current consensus at Evergreen, after some observation & some clinical judgment, is to stop ICS if able, based on known data with other viral pneumonias and increased susceptibility to HAP. Thus far pts are tolerating that, no major issues with ventilating them that can't be managed with vent changes. We also have quite a few on AE-COPD/asthma doses of methylpred, so will be interesting to see how they do.