

Case presentation: Cavitary lung lesion in COVID19

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Patient characteristics

Age: 61yo Gender: M BMI: 37

Hospital course: Vent day 23 (COVID pneumonia)

PMHx: HFrEF, HTN, HLD, obesity, childhood rickets

PsurgHx: None

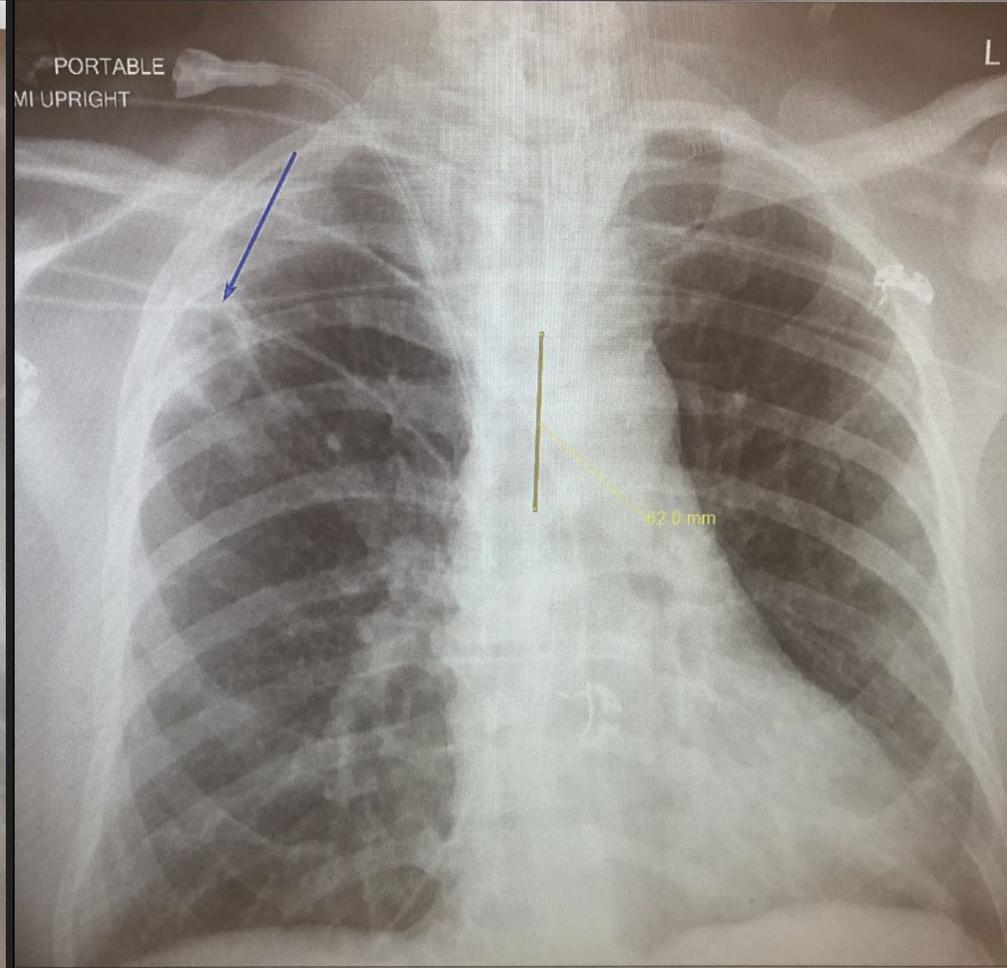
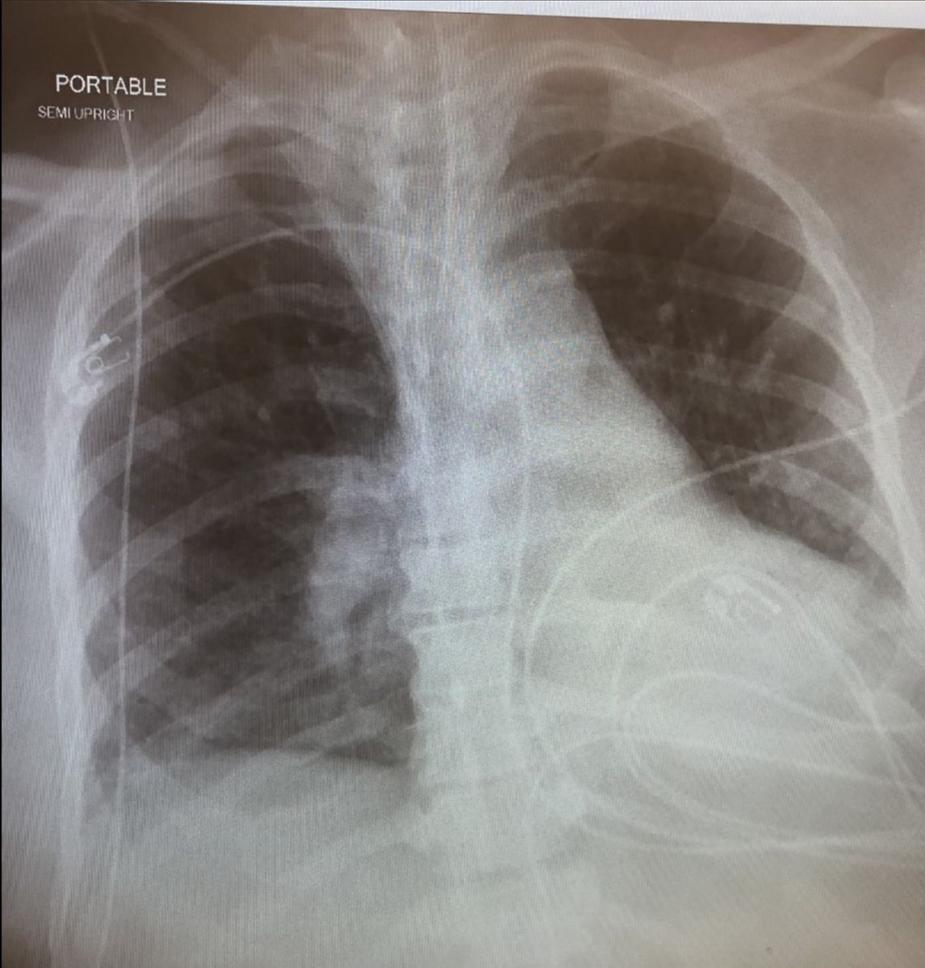
SHx: Smoker, former EtOH, no IVDU

FHx: n/c

Rx (home): Lisinopril, aldactone, aspirin, statin, coreg, zofran



Day 24: slightly increasing O₂ requirement



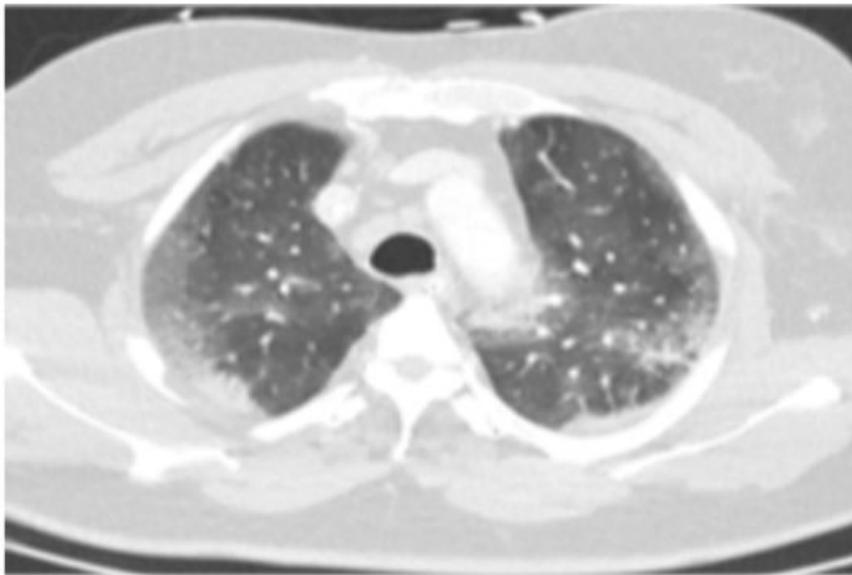
Day 28: continued low grade fever despite antibiotics

Bojanowski and Deere, 2020

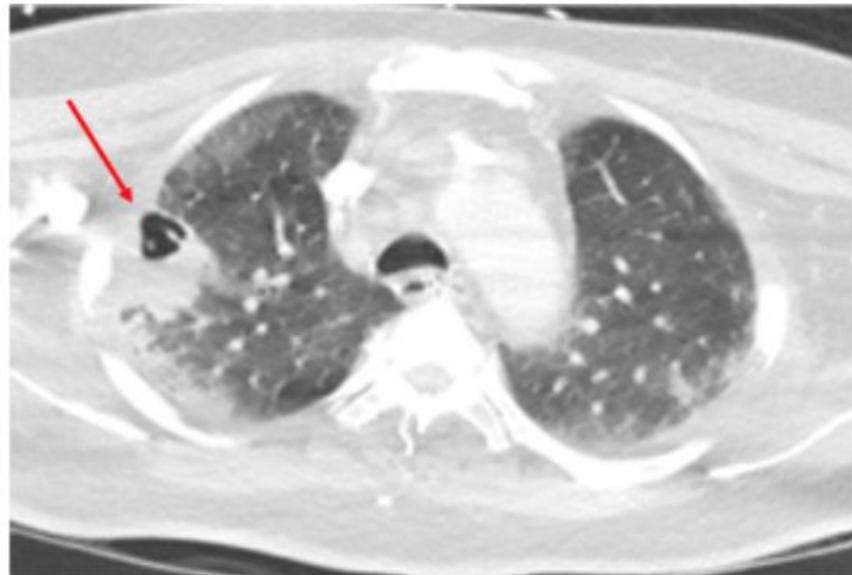
Figure A: Computerized tomography (CT) thorax on admission with bilateral peripherally based ground glass opacities consistent with the diagnosis of COVID-19. Axial view.

Figure B: Repeat CT thorax of the same patient revealing a new cavitary lung lesion (red arrow) in the right upper lobe one month after initial diagnosis of COVID-19. Axial view.

A.



B.



Labs

WBC: Elevated, but unchanged

Procalcitonin: Elevated, but unchanged

Sars CoV 2: Negative as of hospital day 28

Respiratory culture: Normal flora, plus some mold

Serum galactomannan: Negative



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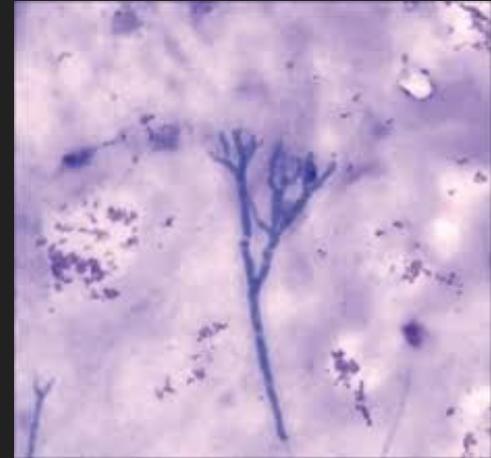
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*Mold speciates to *Aspergillus fumigatus*;

**Antibiotic therapy tailored to voriconazole



Outcome

Voriconazole initiated on hospital day 31 (plan for 10-12 week course)

Patient defervesced by hospital day 33

Underwent trach and PEG on hospital day 43

Awake and conversing with family and staff as of hospital day 49

Discharged from inpatient care on hospital day 53; currently recovering in LTAC

Invasive Pulmonary Aspergillosis in COVID19

Frequent complication of severe influenza pneumonia (possibly 7-23% of cases)

Often only diagnosed post-mortem (mortality of 40-60%)

European case series suggest IPA may complicate ~33% of COVID19 ARDS

- *Alanio et al, Lancet Respi Med, 5/20/2020*
- *Rutsaert et al, Annals of Int Care, 6/1/2020*
- *Koehler et al, Mycoses, 4/27/2020*
- *Van Arkel et al, AJRCCM, 5/12/2020*

Radiopaedia.org



Diagnosis and Management

Bronchoscopic biopsy and histopathology is gold standard of diagnosis

Clinical diagnosis often requires high suspicion and mycologic testing

Voriconazole is the preferred therapy

Amphotericin and echinocandins are alternative therapies

Take away points

Increasing concern for co-infection with COVID19 and IPA

Poor sensitivity of serum galactomannan (21-65%)

Consider BAL galactomannan (non-bronchoscopic lavage)

Maintain high index of suspicion

Consider initiation of anti-fungal therapy

Key questions:

- Role of screening
- Role of prophylaxis
- Standard case definitions

Panel: Crucial next research questions for COVID-19 associated invasive pulmonary aspergillosis

Diagnosis of CAPA

- What is the positive predictive value of culture isolation of *Aspergillus* species in samples from the upper respiratory tract (infections vs colonisation)?
- In light of low sensitivity of serum galactomannan, are there alternative blood tests for CAPA? What is the performance of *Aspergillus* PCR, β -D-glucan, and the *Aspergillus*-specific lateral flow device and lateral flow assay?
- Do radiological signs differ in people with CAPA from what's seen in COVID-19 without CAPA

Prophylaxis and treatment of CAPA

- Is the incidence of CAPA in intensive care unit patients high enough to justify antifungal prophylaxis trials?
- What is the clinical relevance of CAPA? Is there a survival benefit with antifungal treatment?
- What is the CAPA-associated mortality? Autopsy study vs post-mortem lung biopsy?
- What is the optimal treatment of CAPA? Considerations of efficacy, dosing, adverse events, and drug-drug interactions

Immunology or host factors

- Underlying host factors (neutropenia, lymphopenia, monocytopenia, polymorphisms; eg, PTX3, dectin-1, and NADPH-oxidase) or a role for concomitant medication, such as (hydroxy)chloroquine being either harmful (causing defective autophagy) or having direct antifungal or protective effect like in chronic granulomatous disease?
- Role of the kallikrein-kinin system in antifungal host defense
- Is there an underlying antifungal defect caused by COVID-19 or associated with CAPA, such as defective reactive oxygen species production, defective T-helper cell 1 responses, defective LC3-associated phagocytosis, or defective neutrophil extracellular traps activation or release, such as in chronic granulomatous disease?