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An Early Clinical Case of COVID-19 in New York

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Abstract

SARS-CoV-2, a novel coronavirus that causes the human disease COVID-19, was determined to be the cause of a cluster of pneumonia cases in Wuhan, China which began in December 2019 [1]. The first case in the United States reportedly occurred on 20 January 2020 in Washington state in a patient with a history of travel to Wuhan [2]. We report an earlier case of COVID-19 in Queens, New York in November 2019. While it is not perfectly clear from the initial November 2019 emergency department case presentation, if the patient had influenza alone, COVID-19 alone, or combined influenza and COVID-19 infection, presenting lung lesions and constitutional symptoms, later follow-up antibody and immune cell analyses, and the possibility of false-positive RIDT, do strongly suggest initial COVID-19 infection (with or without initial influenza infection). This clinical paper becomes important, because it may describe the earliest now-reported COVID-19 case in the United States, and because emergency department and post-emergency department treatments contributed to a successful patient outcome.

Keywords

SARS-COV-2, COVID-19, New York, November 2019

1. Case Report

On 6 November 2019, a 60-year-old Caucasian male with a five-day history of wet cough, headache, subjective fever, and dyspnea presented to the emergency department of the Mount Sinai Hospital in New York City after visiting an urgent care clinic where his oxygen saturation levels were found to be low (90% - 93%). The patient, a long-time resident of Queens, New York, reported becoming acutely ill on the evening of 2 November 2019 while attending a conference of approximately one hundred people in upstate New York (Otsego County). Assuming influenza, he self-isolated at the home of a friend in Ulster County,

New York, from 3 - 5 November. His host did not subsequently become ill, and it is not known if any of the conference attendees reported similar symptoms. His partner, a 51-year-old Haitian American female and licensed practical nurse, became ill with similar signs and symptoms shortly after picking him up from Ulster County. She did not seek treatment, and her illness fully resolved within a month. Both the patient and his partner subsequently tested positive for serum IgG antibodies to SARS-CoV-2.

2. Emergency Department Findings and Diagnostic Evaluation

The patient's medical history includes coronary artery disease (CAD) and a sustained myocardial infarction in 2007. He was treated with angioplasty and staged stent placement, the most recent of 11 total stents was placed in 2017. Management of his CAD and associated hypertension and hyperlipidemia included: aspirin 81 mg, losartan 25 mg, isosorbide 60 mg, atorvastatin 80 mg, metoprolol 50 mg and niacin 500 mg. Since 1989 symptoms of depression and anxiety have been controlled by fluoxetine 20 mg. A reducible umbilical hernia was noted. Past surgical history included lithotripsy and cystoscopy for renal calculi.

3. Physical Examination, Laboratory Findings and Admission to Hospital

The patient is a 60-year-old white male with a Body Mass Index (BMI) > 30, afebrile, and not in acute distress. Symptoms include a five-day history of subjective fever, headache, wet cough, chills, myalgia, sore throat, and dyspnea. Patient denied nausea, vomiting, abdominal pain, or urinary symptoms. On arrival to the emergency department blood pressure was 136/71, pulse 74, respiration 20, and temperature was 97.1°F.Oxygen saturation level was 96% but subsequently fell to 92% - 93%. Auscultation of chest was positive for scattered rhonchi. Placement on 2L NC effected improvement to 97%. Patient was started on IV ceftriaxone and azithromycin to cover possible bacterial etiology. At this time, the patient was admitted to hospital. Serology over three days was mostly unremarkable but several abnormal values indicating infection and low oxygen levels were noted (Table 1). Computed Tomography Angiography (CTA) of the lungs was ordered to rule out pulmonary embolism and pneumonia but showed multiple, scattered, indeterminate ground-glass nodular opacities and diffuse bronchial wall thickening (Figure 1). A nasal swab returned a positive PCR result for Influenza A on Rapid Influenza Diagnostic Test (RIDT).

4. Diagnosis and Treatment

Based on clinical signs and symptoms, a positive Influenza A PCR nasal swab test, and non-specific inflammatory findings on lung images, a diagnosis of acute hypoxic respiratory failure secondary to flu was made. IV antibiotics were discontinued, and the patient was prescribed the antiviral Tamiflu (Oseltamivir)

Table 1. Patient's serology results over three days indicating normal and flagged values.

Serology 11/06/2019	Value	Serology 11/07/2019	Value	Serology 11/08/2019	Value
ANION GAP (mEq/L)	9.7	TROPONIN-1 (NG/ML)	0.02	ANION GAP (mEq/L)	9.8
BASE EX.VEN (POCT) (%)	4.0 (H)	PROCALCITON IN (ng/mL)	0.06	BASOPHIL # (x10E3/uL)	0
BASOPHIL # (x10E3/uL)	0			BASOPHIL (%)	0.5
BASOPHIL (%)	0.6			CALCIUM (mg/dL)	8.2 (L)
CALCIUM (mg/dL)	8.6			CHLORIDE-BLD (MEQ/L)	109 (H)
CHLORIDE-BLD (MEQ/L)	108			CO2 TOTAL (MEQ/L)	23.2
CO2 TOTAL (MEQ/L)	22.3			CREATININE (mg/dL)	1.03
CREATININE (mg/dL)	1.19			EOSINOPHIL # (x10E3/uL)	0
EOSINOPHIL # (x10E3/uL)	0			EOSINOPHIL (%)	0.6
EOSINOPHIL (%)	0.7			GLUCOSE (mg/dL)	105 (H)
GLUCOSE (mg/dL)	110 (H)			HEMATOCRIT (%)	45.9
HCO3.VEN (POCT) (MEQ/L)	29 (H)			HEMOGLOBIN (G/DL)	15.4
HEMATOCRIT (%)	47.3			LYMPHOCYTE # (x10E3/uL)	1.1
HEMATOCRIT-VEN (POCT) (%)	47			LYMPHOCYTE (%)	27.3
HEMOGLOBIN (%)	15.9			MEAN CORP.HGB (PG)	31.2
LYMPHOCYTE # (x10E3/uL)	1.3			MEAN CORP.HGB CONC. (G/DL)	33.6
LYMPHOCYTE (%)	26.3			MEAN CORP.VOLUME (FL)	93
MEAN CORP.HGB (PG)	30.9			MEAN PLT VOLUME (FL)	8.5
MEAN CORP.HGB CONC. (G/DL)	33.6			MONOCYTE # (x10E3/uL)	0.8
MEAN CORP.VOLUME (FL)	92.2			MONOCYTE (%)	19.7 (H)
MEAN PLT VOLUME (FL)	8.4			NEUTROPHIL # (x10E3/uL)	2.1
MONOCYTE # (x10E3/uL)	0.8			NEUTROPHIL (%)	51.9
MONOCYTE (%)	15.5 (H)			PLATELET (x10E3/uL)	102 (L)
NEUTROPHIL # (x10E3/uL)	2.8			POTASSIUM (MEQ/L)	3.7
NEUTROPHIL (%)	56.9			RBC BLOOD CELL (x10E3/uL)	4.94
O2 SAT.VEN (POCT) (%)	67			RBC MORPHOLOGY-1	Normal
PCO2.VEN (POCT) (MM HG)	45			RED DISTRIB.WIDTH (%)	13.9
PH.VEN (POCT)	7.42			SODIUM-BLD (MEQ/L)	142
PLATELET (x10E3/uL)	109 (L)			UREA NITROGEN (mg/dL)	13
PO2.VEN (POCT) (MM HG)	34			WHITE BLOOD CELL (x10E3/uL)	4.1 (L)
POTASSIUM (MEQ/L)	4.2				
RBC BLOOD CELL (x10E6/uL)	5.13				
RBC MORPHOLOGY-1	Slight Ovalocytes				

Continued

RBC MORPHOLOGY-1	Slight Burr Cells
RED DISTRIB WIDTH (%)	13.9
SODIUM-BLD (MEQ/L)	140
TOT CO2. VEN (POCT) (MEQ/L)	31
UREA NITROGEN (mg/dL)	20
WB CA++. VEN (POCT) (MMOL/L)	1.05 (L)
WB GLUCOSE-VEN (POCT) (mg/dL)	119
WB K VEN (POCT) (MEQ/L)	52 (H)
WB LACTATE-VEN (POCT) (MMOL/L)	1.6
WB NA VEN (POCT) (MEQ/L)	136
WHITE BLOOD CELL (x10E3/uL)	4.9
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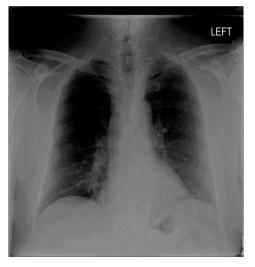


Figure 1. Computed Tomography Angiography (CTA) of the lungs (with contrast) of our patient showing multiple, scattered, indeterminate ground-glass nodular opacities and diffuse bronchial wall thickening.

75 mg b.i.d. to be continued for 4 days after discharge from hospital. An inhaled steroid and supplemental oxygen improved the patient's symptoms while hospitalized and Flovent was prescribed to manage lingering respiratory symptoms after discharge on 8 November 2019. A follow-up appointment with his primary care physician was scheduled for 14 November 2019.

5. Follow-Up

The patient was seen by his primary care physician 6 days later. No issues of concern were noted. He was afebrile, without cough and lung sounds were normal. On 8 May 2020, well into the COVID-19 pandemic, the patient, recognizing

his illness in November 2019 might have been COVID-19, returned to the urgent care clinic and requested a SARS CoV-2 serum antibody test. The test was positive (7.1) for IgG antibodies to SARS CoV-2 (+ = >1.4). On 19 August 2020, prior to a previously scheduled biopsy for a laryngeal granuloma, protocol required him to undergo the nasal swab PCR test for SARS CoV-2 antigens. The result was negative. Finding the clinical picture and positive IgG antibody to novel coronavirus interesting, the patient's primary medical provider ordered another serum antibody assay on 14 October 2020. This returned a moderate positive titer of 320. A positive result on the SARS-CoV-2 Antibody Spike, Semi-quantitative test (78.20 U/mL) taken on 1 August 2021 indicated long-lasting immunity. On 17 February 2022, the patient was evaluated for memory T-cell adaptive immunity to SARS CoV-2 which returned a positive result suggesting long-lasting protection from reinfection [3]. Considering these findings, in part, the patient applied for, and was granted, a medical exemption from COVID-19 vaccination required by his employers. His accommodation requires weekly testing (by nasal swab) for SARS CoV-2 antigen. As of this writing he has been tested>25 times all with negative results and has been asymptomatic since his hospital admission in early November 2019.

6. Discussion

The presentation, clinical findings and course of disease suggest an early case of COVID-19 in New York that predates the first reported case from Washington state by over two months. In addition to constitutional symptoms now known to be common in the disease, the patient had lung manifestations that, while not pathognomonic for COVID-19 sensu stricto, are currently well known in the literature on COVID-19 [4] [5] [6] [7] [8]. That the patient subsequently tested positive for serum antibodies and an adaptive T-cell immune response to SARS Co-V-2 confirm infection with SARS Co-V-2. Demographic and temporal factors should be considered in this case as well. The patient lives in the New York borough of Queens, parts of which have high populations of Chinese immigrants, some of whom may have attended the Olympic games in Wuhan in October of 2019 [9] became infected and returned home with the first cases on the east coast earlier than November 2019.

The most confounding factor in this case is the positive RIDT taken in the emergency department which elucidated a diagnosis and change in treatment course. Explanations include a false positive RIDT [10] and the phenomenon of co-infection with coronavirus disease and influenza [11] [12]. Another possibility is that the original disease was influenza, and the patient later sustained an asymptomatic case of COVID-19 which produced the immune response. The robust IgG response suggest otherwise as do the patients subjective sense that past bouts with influenza felt different than this disease which lacked high fever, intestinal or gastric distress and other common features of influenza virus infection. This clinical report establishes an influenza etiology (assuming RIDT valid-

ity) and suggests COVID-19 as also present in the patient at the time of initial symptomatology and hospital admission. There is compelling evidence of COVID-19 infection through antibody and immune cellular analyses later (May and October 2020, August 2021, and February 2022), and initially from patient symptomatology and lung X-ray analysis. That definitive identification of COVID-19 was not made in November 2019 must be considered in this report. However, COVID-19 was not being widely considered in the differential diagnosis of respiratory presentations at this time in the United States and there were no COVID-19 testing protocols in place. Genetic data on the "novel SARS-CoV-2" was not available until January 2020 [13] and vaccines would not be available in the U.S. until December 2020 [14].

7. Overview

582,752,785 cases of COVID-19 have been reported as of 1 August 2022 [15]. In a pandemic era, this exemplar's presentation, constellation of symptoms and laboratory findings, would urge a viral etiologic agent. Epidemiologic factors would command correlation to be causation. Despite the paucity of constitutional symptoms at the outset, laboratory and positive imaging findings would confirm the presence of a viral illness *i.e.*, coronavirus.

The longitudinal study of this case does not provide for the earlier history of immunological integrity to determine a *de novo* viral entity, compromised immune state, or normal integrity of the patient during a pandemic. This case has evolved over the past year with weekly follow up testing, per mandate, for the presence of coronavirus or COVID-19 which never tested positive.

It is suggested that immune test baseline parameters be established before administration of preventive measures, antiviral treatment, or vaccination be undertaken.

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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