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***denotes equal contributions of the first and second author, thus should be considered of equivalent importance**

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NON-STANDARD ABBREVIATIONS (Glossary of Terms)

SARS, Severe Acute Respiratory Syndrome

SARS-CoV-1, viral etiology of the 2003 SARS epidemic

SARS CoV-2, novel coronavirus 2019-nCoV

COVID-19, disease associated with SARS CoV-2 infection

MERS-CoV, Middle East respiratory syndrome coronavirus, viral etiology of the 2013 MERS epidemic

ACE2, Angiotensin converting enzyme-2

NAT, Nucleic Acid Testing

HD, Hospital Day (where day of admission is HD1)

PPD, Post Partum Day (where PPD0 is day of delivery, and PPD1 is the day after delivery)

ABSTRACT

Background. Despite 2.5 million infections and 169,000 deaths worldwide (current as of April 20, 2020), no maternal deaths and only a few pregnant women afflicted with severe respiratory morbidity had been reported to be related to COVID-19 disease. Given the disproportionate burden of severe and mortal respiratory disease previously documented among pregnant women following other related coronavirus outbreaks (SARS-CoV in 2003 and MERS-CoV) and influenza pandemics over the last century, the absence of reported maternal morbidity and mortality with COVID-19 disease is unexpected.

Objectives. To describe maternal and perinatal outcomes and death in a case series of pregnant women with COVID-19 disease.

Study design. We describe here a multi-institution adjudicated case series from Iran which includes 9 pregnant women diagnosed with severe COVID-19 disease during their latter 2nd or 3rd trimester. All 9 pregnant women were diagnosed with SARS-CoV-2 infection by rRT-PCR nucleic acid testing (NAT). Outcomes of these women were compared to their familial/household members with exposure to the affected patient on or after their symptom onset. All data were reported at death or after a minimum of 14 days from date of admission with COVID-19 disease.

Results. Among 9 pregnant women with severe COVID-19 disease, at the time of reporting 7 of 9 died, 1 of 9 remains critically ill and ventilator-dependent, and 1 of 9 recovered after prolonged hospitalization. We obtained self-verified familial/household cohort data in all 9 cases, and in each and every instance the maternal outcomes were more severe when compared to other high and low-risk familial/household members (n=33 members for comparison).

Conclusion. We report herein maternal deaths due to COVID-19 disease. Until rigorously collected surveillance data emerges, it is prudent to be aware of the potential for maternal death among pregnant women diagnosed with COVID-19 disease in their latter trimester(s).

Word count for abstract: 298

Key words: Maternal death, SARS CoV-2 virus, COVID-19 disease, pregnancy, maternal mortality, maternal respiratory morbidity, coronavirus disease in pregnancy, respiratory failure with COVID-19, lower respiratory infections in pregnancy .

INTRODUCTION

Over the last several decades, it has been shown that emerging novel strains of influenza and coronaviruses which cause severe respiratory disease[1-4] typically disproportionately affect pregnant women, in part due to the adaptive immunology and cardiopulmonary physiology of pregnancy.[5-7] During three of the major influenza pandemics of the last 100 years (1918, 1957-58, and 2009), pregnant women in their 2nd or 3rd trimester of pregnancy were considerably more likely to be hospitalized or die when compared with the general population.[3,8] For example, in the 1918 H1N1 pandemic, the case fatality proportion among pregnant women was 27%.[3] In the most recent influenza pandemic (2009 H1N1), although pregnant women in the U.S. generally represent only 1% of the population, they accounted for 6.4% of all hospitalizations and 4.3-5.7% of all deaths.[6,8] In the SARS-CoV-1 outbreak, the general population mortality rate was 10.5%, while that of pregnant women approximated 25% with 33% requiring mechanical ventilation.[1] The risks of morbidity with severe lower respiratory infections are not limited to maternal outcomes, as there is a known increased occurrence of preterm birth, fetal demise, and delivery of low birth-weight infants with nearly all maternal severe lower respiratory viral infections.[1-4,8]

However, based on initial reports largely from China, SARS-CoV-2 does not appear to follow these historical patterns of worsened disease risk in pregnancy. We identified 16 reports of SARS-CoV-2 infection or COVID-19 disease in pregnancy or in neonates (current as of April 8, 2020).[9-24] Unexpectedly, although these case reports or retrospective case series include some information on a total of 154 pregnant women and 118 live born neonates (1 set of twins, 1 2nd trimester termination, 1 fetal death, 34 undelivered), we identified only a few gravidae reported as having suffered morbidity requiring respiratory support and critical care.[9-24] For example, in a detailed case series of nine gravida from Wuhan, China, all delivering at 36 to 40 weeks gestation with symptomatic COVID-19 viral pneumonia, Chen and colleagues[10] reported that none required mechanical ventilation nor respiratory support. Of note, however, the upper confidence interval limit for zero mortality in a series of nine women would be 33%. The sole reported severe cardiopulmonary maternal morbidity reported from China was suffered by a gravida admitted at 34 weeks gestation with a severe COVID-19 pneumonia.[12] Subsequent to this patient's Cesarean delivery for a stillborn fetus while in septic shock, her cardiopulmonary status acutely deteriorated with multiple organ system dysfunction requiring extracorporeal membrane oxygenation (ECMO). At the time her care was reported, she remained on ECMO heart-lung bypass.[12] In a recently published two-week surveillance cohort arising from paired clinical affiliate institutions in New York City, Breslin and colleagues[18] reported that among 43 confirmed cases of SARS-CoV-2 infection during pregnancy, the estimated rate of severe maternal disease approximated that of the non-pregnant population at 9.3%. Interestingly, the two gravida which progressed to critical disease (4.3%) were among the 14 of 43 (32.6%) who were initially asymptomatic, and both required admission for ICU care in the post-partum period.[18]

SARS-CoV-1 (the viral pathogen of the 2003 SARS epidemic) and SARS-CoV-2 are both enveloped virions containing one positive-strand RNA genome belonging to the *Betacoronavirus* genus of the *Coronaviridae* family. SARS-CoV-2 uses the same angiotensin converting enzyme 2 (ACE2) as its putative cell entry host receptor as SARS-CoV-1 (the viral pathogen of the 2003 SARS epidemic), and bears 80-85% nucleotide homology to SARS-CoV-1.[25,26] While both SARS-CoV-1 and CoV-2 bind ACE2 via the viral surface spike glycoprotein (S protein, 76% protein identity), there are some suggested distinctions regarding the role of specific serine and cysteine proteases in cleavage of the S protein in priming for enhanced cell entry.[25-27] Specifically, while the S protein of both CoV-1 and CoV-2 S is cleaved by the same transmembrane protease serine 2 (TMPRSS2) to facilitate efficiency of entry and viral replication, there is emerging evidence that SARS-CoV-2 co-opts and recruits additional host proteases for transmissibility.[27,28] Nonetheless, given overall phylogenetic and functional similarities between the viruses, the suggestion of zero pregnant fatalities is unexpected and further inconsistent with data documenting severe disease and death among similarly aged adults who are not pregnant and of low-risk.[29]

While accurate case fatality rates and attributable and relative risk of maternal mortality following SARS-CoV-2 infection will be reported in the future, one of the critical immediate questions faced by providers caring for pregnant women in the midst of the current pandemic is straightforward: are pregnant women at risk of death with COVID-19 disease? We detail herein 7 maternal deaths in a case series of 9 women with severe COVID-19 disease, and compare these deaths to self-verified outcomes among their familial/household members.

METHODS

Study design. The intent of this retrospective case series was to document maternal death and describe maternal, fetal, neonatal, and familial self-reported characteristics among 9 patients known to have experienced severe maternal cardiopulmonary morbidity or mortality following admission to any one of seven level III maternity hospitals in Iran over a 30 day period of time (mid-February to mid-March, 2020; precise dates of admission gated to protect patient identity).

This case series and its detailed reporting was approved by the Ethics Committee of Tehran University of Medical Sciences (IRB IR.TUMS.VCR.1398.1082; IRB PI S.H.) and Baylor College of Medicine (IRB H-47407); a data use agreement (DUA) between Baylor College of Medicine and Tehran University of Medical Sciences was executed for the purpose of this reporting. Subject consent was waived by both review boards, and all familial data was voluntarily self-reported and no familial medical records were reviewed. Additional protections for participants beyond not disclosing exact dates of admission or death included gating maternal age in 5 year increments, and using controlled-access encrypted electronic records for data transfer of primary source data, including digital images of patients' medical records. Index case subjects were assigned Case 1 through 9 designations for the purposes of publication and

communication of non-identifying information, and do not reflect the order of their care nor presentation of first symptoms. The hospitals in which each patient received her care is similarly not reported in an effort to protect subject identity.

Cases were not selected by any form of systematic surveillance, but rather arose through a voluntary reporting of maternal cases with known morbidity or mortality attributable to COVID-19. Our definition of severe disease was comparable to that of others (see further definitions below).[29] Severe cardiopulmonary disease was defined as need for ventilator support and/or cardiopulmonary collapse. With IRB approval from the Ethics Committee of Tehran University of Medical Sciences, starting in February 2020 the Iranian Perinatology Society generated a secure reporting structure to share cases, outcomes and management of pregnant women with severe morbidity or death. For the purposes of this case series, all 9 known cases with severe COVID-19 disease over a 30 day span of time at any of the 7 hospitals or centers are reported herein. Severe disease was classified by provision of having met inclusion criteria of (1) severe morbidity (dyspnea, blood oxygen saturation [SaO2] ≤93% on room air, or partial pressure of arterial oxygen to fraction of inspired oxygen <300), (2) an available death certificate, and (3) there was at least one positive SARS-CoV-2 NAT. Centers were not selected for participation *a priori*, and all reporting of cases was voluntary. Ultimately, all nine cases arose from seven centers which provide high level maternity care in Iran and are staffed by Perinatology (Maternal-Fetal Medicine) consultant specialists and critical care specialists. These 7 centers included: Shariati Hospital and Imam Khomeini Hospital at Tehran University of Medical Sciences, Tehran, Iran; Shohada Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran; Baqiyatallah Hospital, Baqiyatallah University of Medical Sciences, Tehran, Iran; Mousavi Hospital, Zanjan University of Medical Sciences, Zanjan, Iran; Kamkar-Arabnia Hospital, Qom University of Medical Science, Qom, Iran; Alzahra Hospital, Guilan University of Medical Sciences, Rasht, Iran. Of these 9 known cases of severe COVID-19 disease from this 30 day time period, 7 resulted in maternal death (current as of April 20, 2020).

General case management. All pregnant women in this series tested positive for SARS-CoV-2 by use of reverse transcription-polymerase chain reaction (rRT-PCR) nucleic acid testing (NAT) on nasopharyngeal, with or without oropharyngeal and sputum specimens; some subjects were tested multiple times. All collected specimens were tested for SARS-CoV-2 using the TIB-MOLBIOL (Germany) and/or Sansure Biotech 2019-Ncov (China) reagents and protocols, complying with WHO guidelines for use. According to the COVID-19 national guidelines issued by Iran's National Ministry of Health and Medical Education on March 8th, 2020, a three-drug regimen for all COVID-19 pneumonia was recommended, and allowed extension for up to 14 days. This included oseltamivir 75 mg PO every 12 hours for 5 days; hydroxychloroquine sulfate 400 mg PO daily or chloroquine sulfate 1000 mg tablet PO as a single dose; and lopinavir/ritonavir 400/100 mg PO every 12 hours for 5 days. Changing to a four-drug regiment was recommended with the presence of any of the following signs indicative of severe disease: loss of consciousness, tachypnea

(respiratory rate >24, hypotension [blood pressure <90/60], multi-lobar lung infiltration with consolidation by chest imaging [CT or radiograph], or hypoxemia [SaO2 <90%]. The four-drug regiment added ribavirin 1200 mg PO twice daily for 5 days. Antibiotic administration (choice, dose and duration) was deferred to the attending physician's best clinical judgement, and corticosteroids were not recommended. All but one patient in this series was first admitted prior to March 8th. In addition, all subjects received either enoxaparin (40 mg subcutaneous, daily) or heparin (5000 units subcutaneous, twice daily) for thromboprophylaxis.

Case ascertainment and adjudication of outcomes. Following IRB and DUA approval, digital and electronic images of all available patient information from the time of admission through discharge, death, or reporting was securely sent to a single investigator (AAS) and encrypted. All records were converted to paper form, subsequently stripped of any identifiers, and assigned a unique study code. A systematic process of independent interpretation/translation to distill essential elements of clinical care and outcomes from Farsi to English was undertaken by two bilingual investigators boarded in Obstetrics and Gynecology (AAS, SEA); this included conversion of dates from the Solar Hijri calendar (Iranian chronology) to the Gregorian calendar (Western chronology). The de-identified and translated data was then independently adjudicated by two boarded and practicing maternal-fetal medicine clinicians (KMA, AAS). A series of clarifying questions generated by any of the three case interpreters/adjudicators (KMA, AAS, SEA) were communicated to the co-authors in Farsi through three means: video (Skype/WhatsApp/FaceTime), e-mail, and/or direct verbal communication by phone. Once all co-authors and the primary adjudicators reached consensus of all known and reportable outcomes, the case was considered fully adjudicated.

RESULTS

A total of 9 pregnant women are reported in this case series: 7 died and 2 survived and were alive at the time of reporting (April 20, 2020). A schematic summary of each of the 7 fatal adjudicated cases (cases 1 through 7) and comparative outcomes relative to their familial/household members (self-reported) is provided in Figures 1 and 2, respectively; more granular details are found in Table 1 and Table 2. The 2 adjudicated cases of severe morbidity without death (cases 8 and 9) are documented in Supplemental Figure S1, and Supplemental Tables S1 and S2. Key clinical aspects relative to the interpretation and findings of each case include:

Case 1: A 25 to 29-year-old previously healthy gravida at 30 3/7 weeks gestation was seen in the outpatient antenatal clinic the day prior to admission with a complaint of sore throat and rhinorrhea with a dry cough, but was afebrile and did not have dyspnea. She was admitted the next day after become febrile (39°C) and short of breath, and was normotensive (125 systolic, 80 diastolic). On the day of admission, chest CT had features of a viral pneumonia, laboratory values were significant for lymphopenia and pancytopenia, and antepartum testing for fetal well-being was reassuring. The SARS-CoV-2 testing was sent, but ultimately did not result positive until the day of death. Nonetheless, she was initiated on oseltamivir, azithromycin, and ceftazidime at the time of admission. 24 hours later she suffered

acute respiratory distress syndrome (ARDS), and was transferred to the ICU and intubated; linezolid and amantadine were added. Later that evening, onset of spontaneous labor accompanied persistent oxygen desaturations (SaO2 88% on ventilator support), with spontaneous vaginal delivery of an intrauterine fetal death (IUFD, stillbirth) the following morning. With persistent postpartum ARDS (SaO2 85-90% on maximal ventilator support), meropenem, vancomycin and lopinavir/ritonavir were added. 8 hours later she suffered acute hypotension and bradycardia, and died despite cardiopulmonary resuscitative efforts. There are three other household members in her familial cohort: her son was diagnosed with mild COVID-19 disease by clinical symptoms, and her father and husband had prolonged exposure. He recovered and all are alive and well.

Case 2: A 25 to 29-year-old previously healthy primigravid at 38 3/7 weeks gestation was admitted with a 24 hour history of fever, dyspnea and dry cough and was initiated on oseltamivir, azithromycin, and ceftriaxone; fetal wellbeing was reassuring. She was normotensive on admission (110 systolic, 70 diastolic). On HD2, a chest radiograph demonstrated bilateral patchy features typical of a viral pneumonia, her SARS-CoV-2 testing resulted positive, and antepartum fetal monitoring was reassuring. Within 48 hours of admission, she developed acute hypoxemia, spontaneous uterine contractions were noted, and the fetal heart rate tracing regressed to non-reassuring. Following a Cesarean delivery of a viable neonate, she acutely decompensated and was transferred to the ICU and intubated for ARDS. Despite maximal ventilator support, she suffered cardiopulmonary collapse and died within 24 hours after failed resuscitative efforts. Her neonate's pharyngeal swab was SARS-CoV-2 negative. There are four other adults in her familial/household cohort, and her mother and niece were positive for mild symptoms consistent with COVID-19 disease. All recovered, and are alive and well.

Case 3: A 40 to 44-year-old gravida with a history of subclinical hypothyroidism (normal free T4 ng/dL and negative anti-TPO antibody studies) at 30 5/7 weeks gestation was admitted with a one week history of intermittent fever, dyspnea and persistent dry cough; she was normotensive and fetal wellbeing on admission was reassuring (biophysical profile 8 of 8). Her chest CT on admission had bilateral patchy ground glass features, and her SARS-CoV-2 resulted positive. Within 36 hours, she reported decreased fetal movement and acutely decompensated (SaO2 50%). An emergency Cesarean was performed, and she was intubated post-delivery with ARDS. After 24 hours of no improvement, oseltamavir, vancomycin, and meropenum were added. Despite a normal echocardiogram on HD4/PPD2, within 24 hours she suffered persistent hypoxia despite maximal ventilator support with end organ failure; she died one day later following unsuccessful cardiopulmonary resuscitative efforts. Her husband is the only other member of her cohort and was asymptomatic and not tested for SARS-CoV-2, and did not have contact with the neonate at birth nor during the first week of life. Her preterm neonate tested negative by nasopharyngeal swab on day-of-life one, but presumptively acquired SARS-CoV-2 postnatally and subsequently tested positive on day-of-life seven with an accompanying lymphopenia (nadir white blood cell 8.9, with 26% lymphocytes). The neonate was intubated for prematurity, developed a pneumonia on day-of-life two, and remains intubated but stable in the newborn unit.

Case 4: A 30 to 34-year-old gravida at 24 0/7 weeks gestation was admitted with a suspected COVID-19 pneumonia with dyspnea (respiratory rate 30 breaths/minute), tachycardia (heart rate 130 beats/minute), normotensive and fever (39.1°C). She had experienced mild dyspnea with a dry cough several days prior, and her chest CT on admission had bilateral patchy ground glass features; nasopharyngeal swab for SARS-CoV-2 resulted positive. She was initiated on hydroxychloroquine, oseltamivir, azithromycin, lopinavir/ritonavir and ceftriaxone. Over the next several hours, she suffered dyspnea with acute hypoxemia (SaO2 88%) requiring exogenous O2 supplementation; a fetal death occurred within 72 hours of admission. 24 hours later, she acutely decompensated with ARDS, was intubated and arrested, but was successfully resuscitated albeit complicated by a pneumothorax requiring chest tube placement. She required hemodialysis for acute renal failure (serum creatinine 6 mg/dL), and within 24 hours suffered cardiopulmonary collapse and died after failed resuscitation. There are six other household members in her familial cohort, her father was admitted to the hospital with COVID-19 pneumonia and her sister had clinical disease managed outpatient. They recovered, and all are alive and well.

Case 5: A 30 to 34-year-old previously healthy gravida at 36 0/7 weeks gestation was admitted to the ICU with a diagnosis of COVID-19 pneumonia, and initiated on ceftriaxone, oseltamivir, and lopinavir/ritonavir. She had experienced mild cold symptoms for two weeks prior to admission, and on the day prior to admission reported being febrile with new onset dyspnea and dry cough. Her medical history was significant for type A2 gestational diabetes, managed on low dose metformin (500 mg, twice daily). With concern for impending cardiopulmonary collapse, she underwent Cesarean delivery of a viable neonate. 24 hours later, with persistent hypoxia (SaO2 70%), tachypnea, and impending respiratory collapse she was intubated and her antibiotic regiment was changed to vancomycin and meropenum. Over the next 5 days, her cardiopulmonary status worsened despite, a chest tube was placed, and despite maximal ventilator support she died one day later and 3 hours after an initially successful resuscitation. Her neonate's pharyngeal swab was SARS-CoV-2 negative. There are four other household members in her familial cohort: her six year-old son was diagnosed with clinical COVID-19 disease, and three others had prolonged exposure. He recovered, and all are alive and well.

Case 6: A 35 to 39-year-old previously healthy gravida with a current dichorionic/diamniotic twin gestation at 24 0/7 weeks gestation was admitted for a two-day history of fever, dyspnea and persistent dry cough. She had experienced mild cold symptoms for two weeks prior to admission, and her medical history was significant only for infertility requiring *in vitro* fertilization (IVF) for the current pregnancy. Her chest CT on admission was significant only for bilateral patchy ground glass features. With a SARS-CoV-2 nasopharyngeal swab resulting positive; she was immediately initiated on hydroxychloroquine, oseltamivir, and lopinavir/ritonavir, and fetal wellbeing was reassessed reassuring. 24 hours later, she acutely decompensated from ARDS with profound hypoxemia (SaO2 65%). She was intubated and ceftriaxone, azithromycin, vancomycin, and meropenum were added sequentially over the next 4 days given concern for secondary bacterial pneumonia. On HD4, with an ongoing viable twin gestation but clinical worsening, she was provided empiric intravenous immunoglobulin (IVIG) for 3 days, and initially improved enough to

be extubated with documentation of an ongoing and viable twin gestation while remaining inpatient for monitoring. However, two weeks later (HD20), her ARDS acutely recurred (SaO2 77%), and she suffered septic shock, disseminated intravascular coagulopathy, and required re-intubation with an IUFD of both twins within 24 hours. She progressed to left heart failure with an ejection fraction of 25%, suffered cardiopulmonary arrest 18 hours later, and died after failed resuscitative efforts. There are four household members in her cohort, but only her husband was diagnosed with clinical COVID-19 disease. He recovered, and all remain alive and well.

Case 7: A 45 to 49-year-old previously healthy gravida at 28 0/7 weeks gestation with a DCDA twin gestation was admitted with a 14 day history of fever and persistent dry cough. Her history was significant only for age-related infertility requiring IVF with donor oocytes for the current pregnancy. As anticipated for maternal age over 45, prior to IVF therapy she was seen by cardiology with a normal echocardiogram. On the day of admission, she complained of worsening dyspnea over two days and a CT study demonstrated a bilateral patchy, ground glass pneumonia; she was normotensive (systolic 120, diastolic 80). Her SARS-CoV-2 testing on admission resulted positive, and she was initiated on hydroxychloroquine, oseltamivir, and lopinavir/ritonavir; 24 hours later empiric intravenous immunoglobulin (IVIG) for 3 days was added. On HD2 she developed intermittent hypoxemia, with concerns for fetal well-being. She underwent a Cesarean delivery of two viable but premature neonates. She continued on exogenous oxygen support until HD5/PPD3, when she acutely decompensated and was transferred to the ICU, was intubated, and vancomycin and meropenum were added. Both neonate's pharyngeal specimens were SARS-CoV-2 negative and neither twin had evidence of COVID-19 disease post-delivery (absence of lymphopenia, thrombocytopenia, and normal chest radiographs), but they experienced complications of premature birth and both died on day-of-life 3. She died after 18 days of maximal ventilator support following a failed cardiopulmonary resuscitation. Her husband and their surviving child are the only other members of her familial/household cohort, and he was positive for COVID-19 disease with mild symptoms. He recovered, and both are alive and well.

Case 8: A 35 to 39-year-old previously healthy primigravid at 33 5/7 weeks gestation was admitted for inpatient care due to suspected COVID-19 pneumonia (CT with bilateral patchy ground glass features) with accompanying lymphopenia, tachypnea and hypoxemia (SaO2 88%) with hemoptysis. Four days prior to admission she reported a dry cough and fever, with intermittent dyspnea. Despite being normotensive on admission (systolic 130, diastolic 80), within 24 hours she acutely worsened and underwent Cesarean delivery for non-reassuring fetal status (fetal tachycardia) and breech presentation. She was initiated on hydroxychloroquine, oseltamivir, and lopinavir/ritonavir and intubated post-delivery for ARDS. With absence of improvement despite maximal ventilator support, over the following 72 hours meropenum, vancomycin, azithromycin, and levofloxacin were added. She necessitated a tracheotomy on HD6/PPD5 and remains conscious but on ventilator support as of HD35, despite having received immunotherapy (convalescent plasma transfusion) under a separate IRB approval from a recovered COVID-19 donor with known seropositivity. Her neonate's pharyngeal swab was SARS-CoV-2 negative. There are five other

members in her household/familial cohort: her father and sister were diagnosed with clinical COVID-19 disease, and three others had prolonged exposure. All recovered and remain alive and well.

Case 9: A 35 to 39-year-old gravida with gestational diabetes (diet-controlled, type A1) at 36 0/7 weeks gestation was admitted with a fever and persistent dry cough. Her presentation to the hospital was prompted by preterm premature rupture of membranes (PPROM) and decreased fetal movement. On arrival to labor and delivery, an IUFD in breech presentation was diagnosed and a Cesarean delivery was performed. She was normotensive on admission, and a chest CT showed bilateral patchy ground glass features. She decompensated within 18 hours of Cesearen delivery for her IUFD, and was transferred to the ICU with a diagnosis of coagulopathy and received fresh frozen plasma and cryoprecipitate, and a second-generation cephalosporin was added. On HD3/PPD2, with persistent hypoxia, tachypnea, and impending respiratory collapse she was intubated and her antibiotic regiment was changed to vancomycin and meropenum. 48 hours later (HD5/PPD4), following negative influenza A and B testing with an initial SARS-CoV-2 NAT negative test but with persistent ventilator dependence, she was initiated on oseltamavir. Her condition gradually improved by HD14/PPD13, when she was briefly extubated but re-intubated within 8 hours for acute worsening of ARDS; on that same day two repeat SARS-CoV-2 NAT tests returned positive. She later experienced a gastrointestinal bleed requiring endoscopy, and colistimethate sodium was started and meropenum was held. With persistent pulmonary disease, atazanavir and piperacillin/tazobactam were added. She experienced a slow recovery and was extubated after 20 days. She remains in the hospital, and in our series of 9, is the only patient recovering and extubated at the time of reporting. There are four other adults in her familial household cohort, and despite prolonged exposure to the patient prior to admission, only her sister was diagnosed with COVID-19 disease by symptoms. She recovered, and all are alive and well.

DISCUSSION

Primary findings. Potentially consistent with reported outcomes from other severe viral lower respiratory infections,[1-6,8] we show that pregnant women with SARS-CoV-2 infection and COVID-19 disease in their 2nd or 3rd trimester of pregnancy may experience cardiopulmonary complications and die. Our reported outcomes are significantly different from 104 pregnant women from China,[9-17,19-24] but are consistent with one (gravida suffering stillbirth and remaining on ECMO at the time of the reporting[12]), and potentially consistent with some level of respiratory support but recovery in two others.[9,14] Because our study is a case series and not a surveillance cohort, while our report demonstrating any occurrence of morbidity or death generally aligns with the reported trend of severe maternal outcomes (9.3% of the surveillance cohort) and critical morbidity (4.7%) of Breslin and colleagues, we cannot report on the rate of occurrence in any of our hospitals or centers.[18] When considering our maternal deaths relative to these and others reports,[9-24] there are two key distinguishing features of our series. First, ours is a case series reporting death of pregnant women in their latter 2nd or 3rd trimester presenting with severe COVID-19 disease over a 30 day interval in Iran. In contrast, Breslin and colleagues[18] reported all outcomes (n=43) SARS-CoV-2 positive gravida over two weeks from their paired

affiliate hospitals, 1/3 of which were asymptomatic and diagnosed by universal surveillance testing on routine obstetrical admission. Second, all of our subjects were delivered (or died with their periviable fetuses *in utero*); other case series totaled 22% undelivered gravida at the time of reporting and retained only sparse maternal outcomes data.[9-24] Longer term follow-up may be important in revealing instances of severe maternal outcomes, since most of our patients died days to weeks after initial symptoms and often in the post-partum interval.

There are additional characteristics of the patients in our case series which are distinct, but unlikely to be confounding our reported SARS-CoV-2 associated mortality. Five of our gravidae were 35 years of age or older, with two of these five being of elder advanced maternal age (>40 years). However, of the 2 of 9 still alive, both were 35 years or older (Table 1). We observed a statistically significant distinction in the mean maternal age in our case series when compared to all others (average maternal age 36.7 ± 7.3 years versus 30.3 ± 3.6 years, $p<0.001$); the clinical significance of this difference is unknown.[9-24] No patient in our series had pre-existing comorbidities above baseline population risk (*ie.,* GDM and subclinical hypothyroidism), and none had hypertension, cardiovascular disease, asthma, nor renal disease. Finally, all women on admission were normotensive, excluding a comorbid diagnosis of preeclampsia. Similarly, we think it is unlikely that the quality of delivered obstetrical care is the source of these outcome discrepancies, as the WHO-reported maternal mortality ratio in Iran is lower than that of China (16 versus 19.6 per 100,000, 2017 normalized data; see https:// linked to who.int/gho/maternal_health/countries/irn.pdf and who.int/china/health-topics/maternal-health). Maternity care delivered in Iran during the pandemic has remained a high national and regional priority, and ICU capacity at all of the centers was non-limiting and in no instance was admission, ICU transfer, delivery, intubation, nor medication delayed for lack of availability of resources.

Rather, we think it more probable that delays in reporting or underreporting, alongside non-random selection bias, may be contributing to these first-pass differences. Assessment of epidemiologic characteristics including case-fatality ratios during the course of a pandemic may be affected by right (Type I) censoring and ascertainment bias.[30] As recently demonstrated by Mizumoto et al,[30] breakdowns in an overwhelmed healthcare delivery system with the SARS-CoV-2 pandemic may result in an underestimated death risk in epidemic epicenters even within a given country (right censoring). We emphasize that large upper limit confidence intervals will always accompany small case series with zero mortality, and this could additionally lead to a false reassurance regarding risk of death in early reporting.[9-24]

Strengths and limitations. We acknowledge that our series is limited by lack of surveillance data and is prone to adverse outcome ascertainment bias. Accordingly, we are not attempting to quantify risk or estimate rates in our small series and explicitly discourage others from doing so. Surveillance data will ultimately define the impact of pregnancy among women who died or experienced severe morbidity attributed to COVID-19 disease. Our case series lends critical information to the current narrative based on previously published reports, which presently suggests zero mortality

among pregnant women.[9-24] Determining the proportionate case fatality rate and risk of severe morbidity in pregnancy will require rigorous population-wide surveillance data from many countries, inclusive of data identifying potential modifiers and co-morbidities adjusting risk. However, the cases we have reported herein demonstrate that COVID-19 maternal mortality is not zero, and suggest caution against complacency and early assumptions of protection with pregnancy.[9-17,19-24]

Despite these limitations, there are a number of strengths to our report. First, we took a rigorous approach to collecting primary source data and adjudicating each case and its outcomes. Second, we compared our outcomes to familial/household members as a proxy for comparative risk (**Figure 2 and Figure S1, panel B**). Given the R0 estimate of SARS-CoV-2 ranging from 2.1-3.11,[25-30] it would be anticipated that the same viral strain infected the case and her familial/household cohort and access to quality care and baseline demographic variances would be anticipated to be small. We recognize that ideally we would compare our outcomes to all severely ill non-pregnant subjects over the same time period. However, the level and rigor of surveillance data that such a study would require is not feasible at this time. Thus, our conclusions are best summated in a simple statement with clinically practical implications: in this case series documenting 7 deaths among 9 pregnant women with severe COVID-19 disease, when compared to their spouses, children, or family members living in the same household, the pregnant patients were the only reported deaths. We emphasize the risk for ascertainment bias in our case series, and we make no statements regarding either relative or attributable risk for severe morbidity nor death among pregnant women when compared to non-pregnant women from the population at-large.

Interpretation of our findings in the context of other studies. Our reporting of a number of adverse perinatal and neonatal outcomes, including preterm birth, fetal death, and neonatal death are consistent with several other adverse outcome reports from China and New York[9-24] and prior adverse perinatal outcomes of fetal death, fetal growth restriction, and placental abruption seen with SARS-CoV-1 in 2003 and MERS-CoV in 2012.[1-4] Of note, the intent of our series was not to determine whether or not neonates can vertically acquire SARS-CoV-2 via intrauterine maternal transmission. However, in one instance (case 3), the premature neonate initially tested SARS-CoV-2 negative by nasopharyngeal swab and later tested positive on day-of-life seven while intubated in the NICU. Evidence regarding perinatal outcomes, including vertical mother-to-child transmission of SARS-CoV-2, is presently unclear, with 13 of 16 publications suggesting no evidence of transmission.[9-24] In contrast, Wang et al[15] provided a single case report from the generally same region (Wuhan) of a pregnant woman living approximately 1.2 km from the Huanan Seafood Wholesale Market. In that instance, the patient wore an N95 mask during the delivery, and there was no maternal-neonatal contact nor breastmilk feeding.[15] Nevertheless, at 36 hours of life the neonate nasopharyngeal swab was SAS-CoV-2 positive.[15] Three other groups have recently reported on rare occasions of possible vertical transmission,[22-24]

albeit inclusive of serologic testing with IgM antibodies with yet unproven SARS-CoV-2 specificity which unexpectedly declined in their levels within the first two weeks of postnatal life.[24]

We acknowledge that when considering the potential for vertical transmission, both the placenta and stool/meconium maybe of importance, as a less lethal common *Coronaviridae* (229E) is known to be transplacentally transmitted but its use of the same or different cell entry receptors as SARS-CoV-2 (*i.e.,* ACE2) is unknown.[31,32] Moreover, while *ACE2* mRNA is more highly expressed in early gestation human placental syncytiotrophoblasts, and ACE protein localizes to fetal endothelium, the placental expression of host proteases (such as TMPRSS2 and others) necessary for cleavage of the S protein and receptor priming is unknown but has generally only been described in lung and airway cells, or their progenitors.[26-28] As a result, whether the necessary and sufficient host molecular machinery to enable efficient transplacental vertical transmission is present or absent in the second or third trimester human placenta is presently unknown, and our current case series offers no further insight. With regards to fecal-oral transmission, we were struck by a recent report suggesting that as great as 23.2% of non-pharyngeal detected SARS-CoV-2 may be detected by rRT-PCR NAT in the stool.[33] In this case series of 73 SARS-CoV-2 infected patients ranging from aged 10 months to 78 years, 53.4% tested persistently positive in the stool for as great as 12 days and immunofluorescence visualization of biopsy specimens was consistent with viral uptake in the glandular cells of the gastric, duodenal, and rectal epithelia.[33] This raises concern for the possibility of fecal-oral transmission, which would have potential implications in obstetrical practice and risk of vertical transmission if women were known to harbor infectious virions in their stool or vagina at the time of vaginal delivery. However, infectious SARS-CoV-2 virion load in stool nor vagina is not known, and demonstration of fecal-oral transmission remains purely speculative. In our series, case 3 was delivered by Cesarean.

We make no conclusions regarding the relative likelihood (or not) that pregnant women may be at higher risk for infection with SARS-CoV-2. While it is frequently stated that pregnant women are 'immunosuppressed', such assumptions are incorrect.[7] Rather, human pregnancy represents highly adaptive immunity, allowing the mother to become tolerant to her fetus yet remain immunocompetent to ward off pathogenic infection. This includes competency of B cell mediated humoral immune responses, as well as innate and T cell mediated responses to intracellular pathogens.[7] Ours and others general finding of lymphopenia in both pregnant[9-24] and non-pregnant[25-28] SARS-CoV-2 infected patients is a result of viral infection, and not pregnancy *per se*. As evident by comparison with our referencing to familial/household members, our patients were never alone in their occurrence of infection nor symptomatic disease. Rather, once they were infected and with the development of COVID-19 pneumonia, they suffered severe respiratory and/or cardiopulmonary morbidity and death. It is likewise important to refrain from conclusions regarding safety, harm or efficacy of any medications or treatment decisions made in the care of these patients. Hydroxychloroquine and chloroquine are cellular autophagy modulators that interfere with endosome-mediated viral entry and latter stages of replication of enveloped viruses, including retroviruses, coronaviruses and

404 flaviviruses.[34-37] However, HIV-infected patients receiving hydroxychloroquine in the absence of other antiretroviral
405 therapy showed increased viral replication and poorer outcomes in a randomized controlled trial.[37] Its use among the
406 patients in our case series was consistent with regional practice and did include concomitant antiviral agents.
407
408 *Clinical implications.* These 7 maternal deaths due to severe COVID-19 disease should prompt reexamination of any
409 current guidelines and recommendations by professional societies that might be potentially construed as providing yet
410 unproven reassurance of the absolute absence of death among pregnant women with COVID-19 disease. Our case
411 series is in contrast to prior reports suggesting no known mortality among pregnant women infected by SARS-CoV-2.[9-
412 24] Whether the maternal case fatality rate or maternal morbidity estimates will ultimately be the same, less, or greater
413 than that of other populations is as yet unknown. However, the fatal cases reported herein demonstrate it is not zero,
414 and should inspire caution against complacency and guide restraint in rushing estimates of relative or attributable risk
415 with pregnancy.
416

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AUTHOR CONTRIBUTIONS

All authors except for KMA, MDS, AAS and SEA cared for these women and communicated their clinical care. SH and AAS coordinated all communications of data. KMA designed the study to include familial/household members as a proxy for proportionality comparisons. KMA and AAS adjudicated the outcomes. AAS and SEA interpreted the content of the medical records to English summary data, and verified the source data with the co-authors, alongside available death certificates and receipt of source medical records. KMA, MDS, and AAS analyzed the data. KMA wrote the manuscript, curated all literature to date, and summated the systematic review. KMA and MDS projected the data, designing and preparing the figures and tables. All authors reviewed and edited the manuscript, and agreed and verified its content.

Figures with Legends

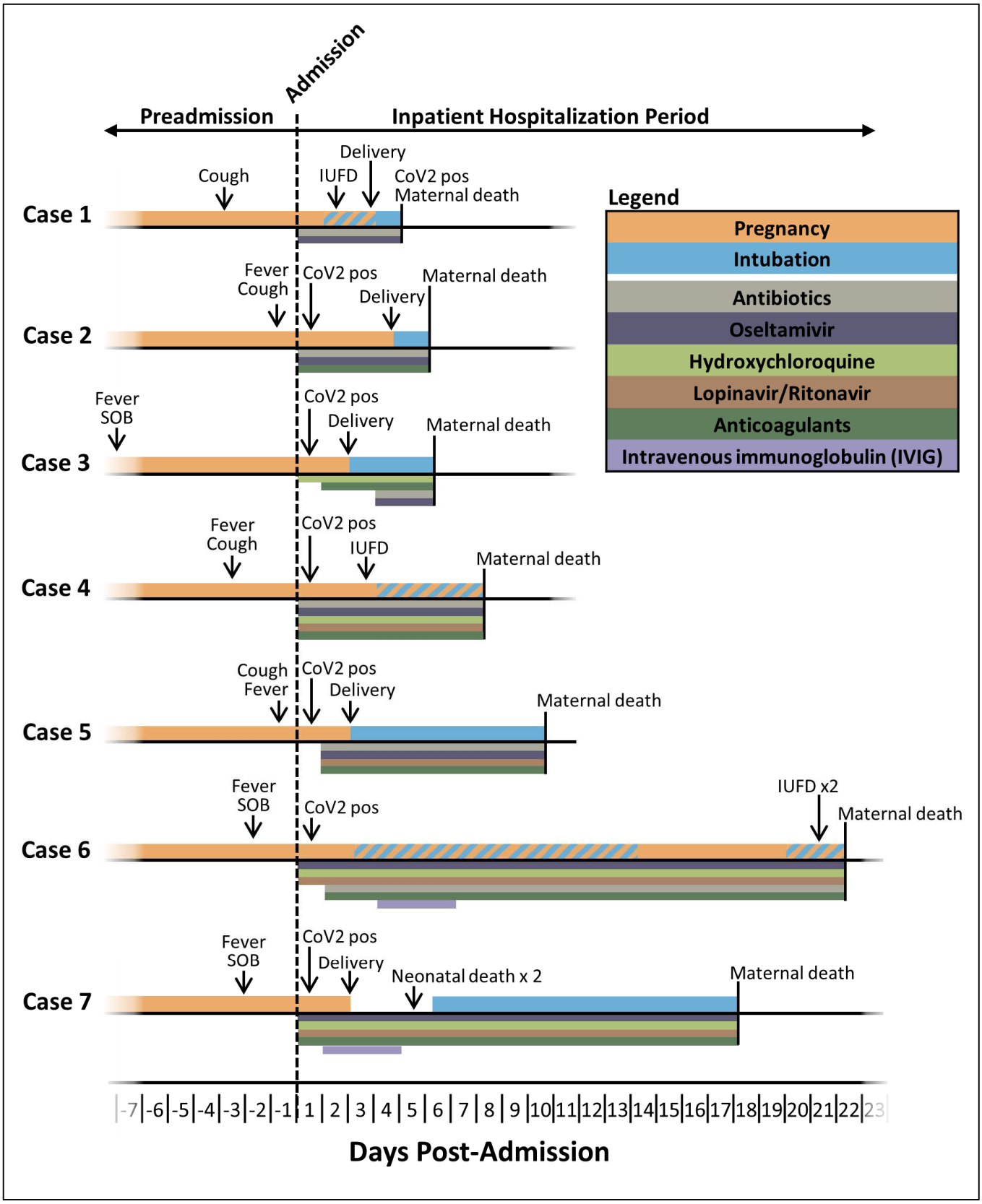


Figure 1. **Summary timeline of patients' events, procedures and medications prior to death.** Narrative summaries are provided in the text, and further details are in Table 1 and Table 2. The order of cases does not represent chronology nor site of care. DCDA: dichorionic, diamniotic twin gestation; rRT-PCR NAT: nucleic acid testing for SARS-CoV-2 (CoV) virus. DFM, decreased fetal movement; resp. distress, respiratory distress; IUFD, intrauterine fetal death. No patient was positioned in the prone position, either while pregnant nor in the post-partum interval. Dosages of medications are provided in methods, timing is indicated by the bars, and constituent drug therapies are detailed in each case report narrative. For all cases, anticoagulation therapy was comprised of enoxaparin (cases 1 to7 & 9, at 40 mg subcutaneous daily) or heparin (case 8, heparin 5000 units subcutaneous twice daily).

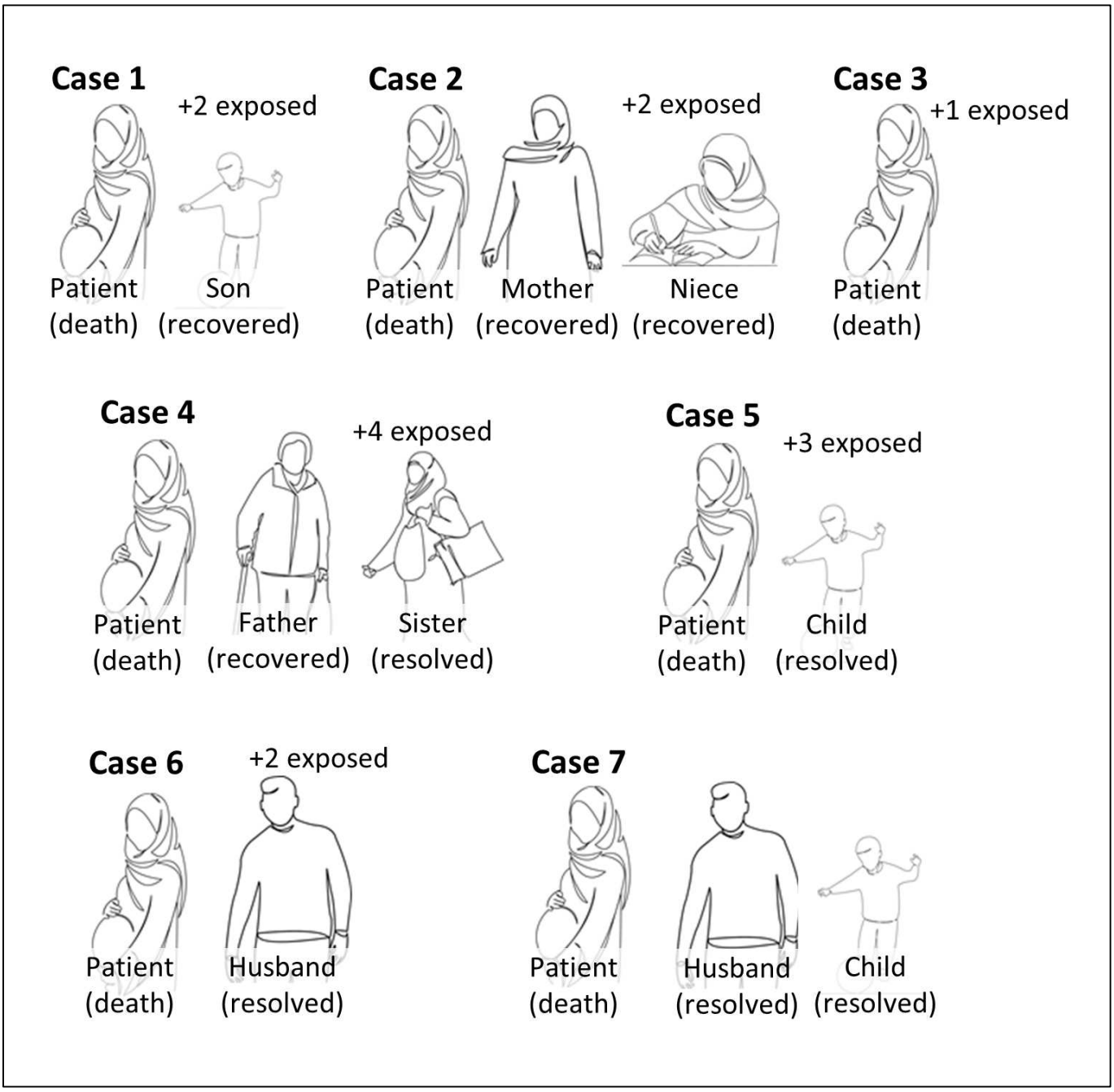
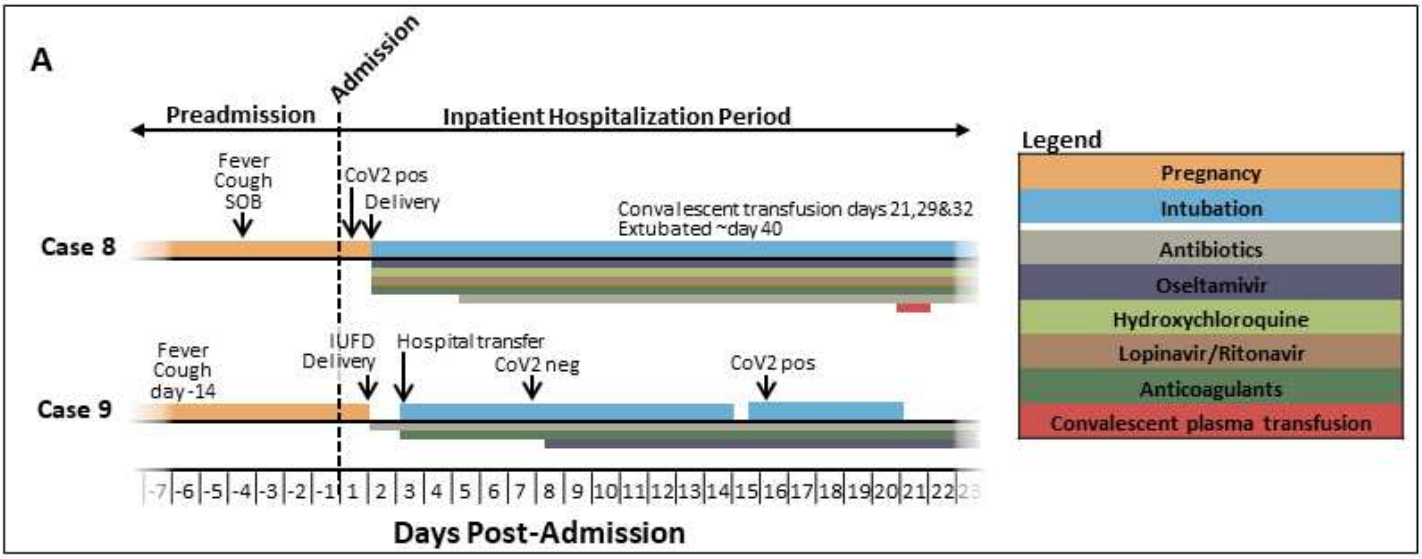
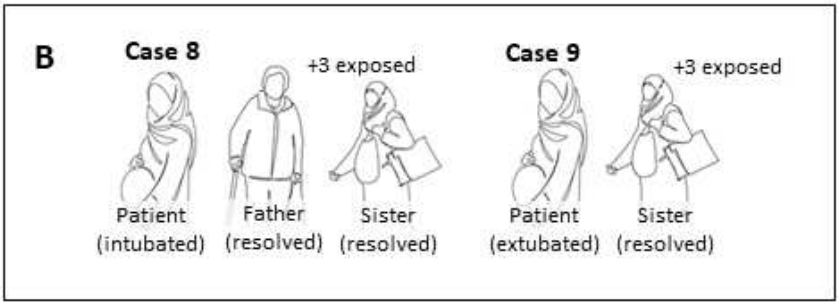


Figure 2. **Outcomes among familial and household members of the 7 pregnant patients who suffered death following SARS-CoV-2 infection.** All of our pregnant patients had available self-reported data, and the only member suffering death was the pregnant patient. All occurrences of prolonged exposure occurred as a result of duration of symptoms prior to patient admission.





Supplemental Figure S1. **Panel A. Summary timeline of patients' events, procedures and medications in cases of severe morbidity but without death (current as of April 20, 2020).** Narrative summaries are provided in the text, and further details are in Supplemental Table S1 and Table S2. The order of cases does not represent chronology nor site of care. DCDA: dichorionic, diamniotic twin gestation; rRT-PCR NAT: nucleic acid testing for SARS-CoV-2 (CoV) virus. DFM, decreased fetal movement; resp. distress, respiratory distress; IUFD, intrauterine fetal death. No patient was positioned in the prone position, either while pregnant nor in the post-partum interval. Dosages of medications are provided in methods, timing is indicated by the bars, and constituent drug therapies are detailed in each case report narrative. For all cases, anticoagulation therapy was comprised of enoxaparin (case 9, at 40 mg subcutaneous daily) or heparin (case 8, heparin 5000 units subcutaneous twice daily). **Panel B. Outcomes among familial and household members of the 2 pregnant patients who suffered severe morbidity but did not die (current as of April 20, 2020).** All of our pregnant patients had available self-reported data, and the only member suffering severe cardiopulmonary morbidity was the pregnant patient. All occurrences of prolonged exposure occurred as a result of duration of symptoms prior to patient admission.

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	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7
Maternal characteristics							
+Maternal age (yr)	25-29	25-29	40-44	30-34	30-34	35-39	45-49
Gravida, para	G2 P1001	G1 P0	G2 P1001	G3 P0020	G2 P1001	G2 P0010	G2 P1001
Co-morbidities	none	obesity	Subclinical hypothyroid AMA	none	GDMA2 (metformin)	AMA	AMA Underweight
Blood type (Rh)	A (+)	B (+)	A (+)	B (+)	A (+)	O (+)	A (-)
Influenza vaccinated	No^	Yes	No	No	Yes	Unknown	No
Admit BMI (kg/m^2)	23	36	26	unk	23	24	18
Presenting symptoms							
Fever	yes	yes	yes	yes	yes	yes	yes
Cough	yes	yes	yes	yes	yes	yes	yes
Dyspnea	yes	no	no	yes	no	yes	yes
Myalgia	yes	yes	no	yes	no	yes	no
Medications							
Antivirals	yes	yes	yes	yes	yes	yes	yes
Antibiotics	yes	yes	yes	yes	yes	yes	yes
Anticoagulants	no	yes	yes	yes	yes	yes	yes
Other	none	none	HCQ	HCQ	none	HCQ, IVIG	HCQ
Laboratory or relevant clinical values*							
SARS-CoV-2 NAT	positive	positive	positive	positive	positive	positive	positive
Hemoglobin, g/dL	9.6 (9.2,9.6)	9.0 (8.5,10)	11.6(10.8,11.8)	10.8(10.2,14.3)	9.9 (8.2,10)	8.1 (8,10.2)	12.3(9.9,12.5)
Platelets, x 10^3/µL	51 (48,43.4)	68 (62,280)	224(220,265)	206(206,333)	305(265,328)	177(122,188)	380(172,380)
WBC, x 10^9/L	3.8 (3.2,7.8)	8 (7.2,8.2)	7 (4.2,13.3)	13.3(13,35.6)	20.3(13.7,26)	7 (7,8.6)	16.4(8.8,18)
#Lymphocyte, % 10^9/L	6.8% (5.5,7.8)	unknown	5% (5,6.8)	7.7% (unk)	8.5% (7.5,8.8)	9% (8.8, 9)	7% (6.2, 8.4)
&CRP, mg/L	41 (38,87)	18 (18,22)	25 (20,25)	56 (unk)	64 (60,68)	117.5 (37,12)	81.9
AST (U/L)	52 (47,58)	60 (52,76)	160(152,220)	28 (unk)	40 (32,48)	29 (22,29)	66 (52,68)
ALT (U/L)	68 (62,78)	40 (32,65)	143(123,148)	26 (unk)	17 (15,40)	18 (14,22)	38 (34,62)
Cr (mg/dL)	0.8 (0.8-1.6)	0.5 (0.5-1.1)	0.6 (0.6-1.4)	0.7 (0.6-6.0)	0.8 (0.8-1.3)	0.9 (0.9-1.4)	0.7 (0.6-1.5)
^^O_2 Sat, % (SaO2)	85	70	50-60	83	70-75	65	60-65
Maternal status (current as of April 20, 2020)							

Death, intubated, or inpatient recovery	death	death	death	death	death	death	death

Table 1. Maternal characteristics and outcomes among pregnant patients with SARS-CoV-2 infection and suffering death.

GDM, gestational diabetes mellitus; HCQ, hydroxychloriquine; IFN, interferon-alpha nebulizers; plasma, under a separate IRB, received immunotherapy through convalescent plasma transfusion from a recovered COVID-19 donor with known seropositivity; unk, unknown values; AMA, advanced maternal age; NAT, nucleic acid testing by rRT-PCR; WBC, white blood cell count; Cr, serum creatinine.

For antiviral and antibiotic regiments, please see methods and case narratives.

+For protection of patient identification, maternal age was gated in inclusive 5 year blocks.

^Case 1 did not receive seasonal influenza vaccination, but did have negative influenza testing during hospitalization for SARS-CoV-2.

*For all laboratory values, the initial value at the time of admission is provided, with the trough and peak from the hospitalization interval (trough, peak).

[#] Lymphopenia was defined as 10%; [&]Elevated C-reactive protein (CRP) was defined as >10 mg/L.

^^SaO2 values are as reported at the time of diagnosis of ARDS and intubation.

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7
Fetal death	yes	no	no	yes*	no	yes*	no
Gestational age, weeks	30 3/7	38 3/7	30 5/7	24 0/7 (undelivered)	36 0/7	24 0/7 (undelivered)	28 0/7
Neonatal demise	n/a	no	no	n/a	no	n/a	yes (twins)
Mode of delivery	NSVD	Cesarean	Cesarean	n/a	Cesarean	n/a	Cesarean
Birthweight, grams	1700	2800	2100	n/a	3200	n/a	1180; 1340
APGARS, 1,5 minute	0,0	8,9	9,10	n/a	7,9	n/a	8,9; 7,9
DCDA twin gestation	no	no	no	yes	no	yes	yes
SARS-CoV-2 NAT	n/a	negative	negative[&]	n/a	negative	n/a	negative
Neonatal pneumonia	n/a	no	yes	n/a	no	n/a	no; no
Neonatal lymphopenia	n/a	no	yes	n/a	no	n/a	no; no

Table 2. Perinatal outcomes among pregnant patients with SARS-CoV-2 infection and suffering death.

DCDA, dichorionic, diamniotic twin gestation; NSVD, normal spontaneous vaginal delivery; NAT, nucleic acid testing by rRT-PCR.

*24 week singleton (case 4) or DCDA twin gestation (case 6) in utero at the time of maternal death, undelivered

n/a, not applicable.

[&]As detailed in the case description, case 3 was negative on day of life 1, but converted to positive on day of life 7.

In no instance was magnesium sulfate given intrapartum nor antenatal for the purpose of neuroprotection, and not patient in the series had preeclampsia.

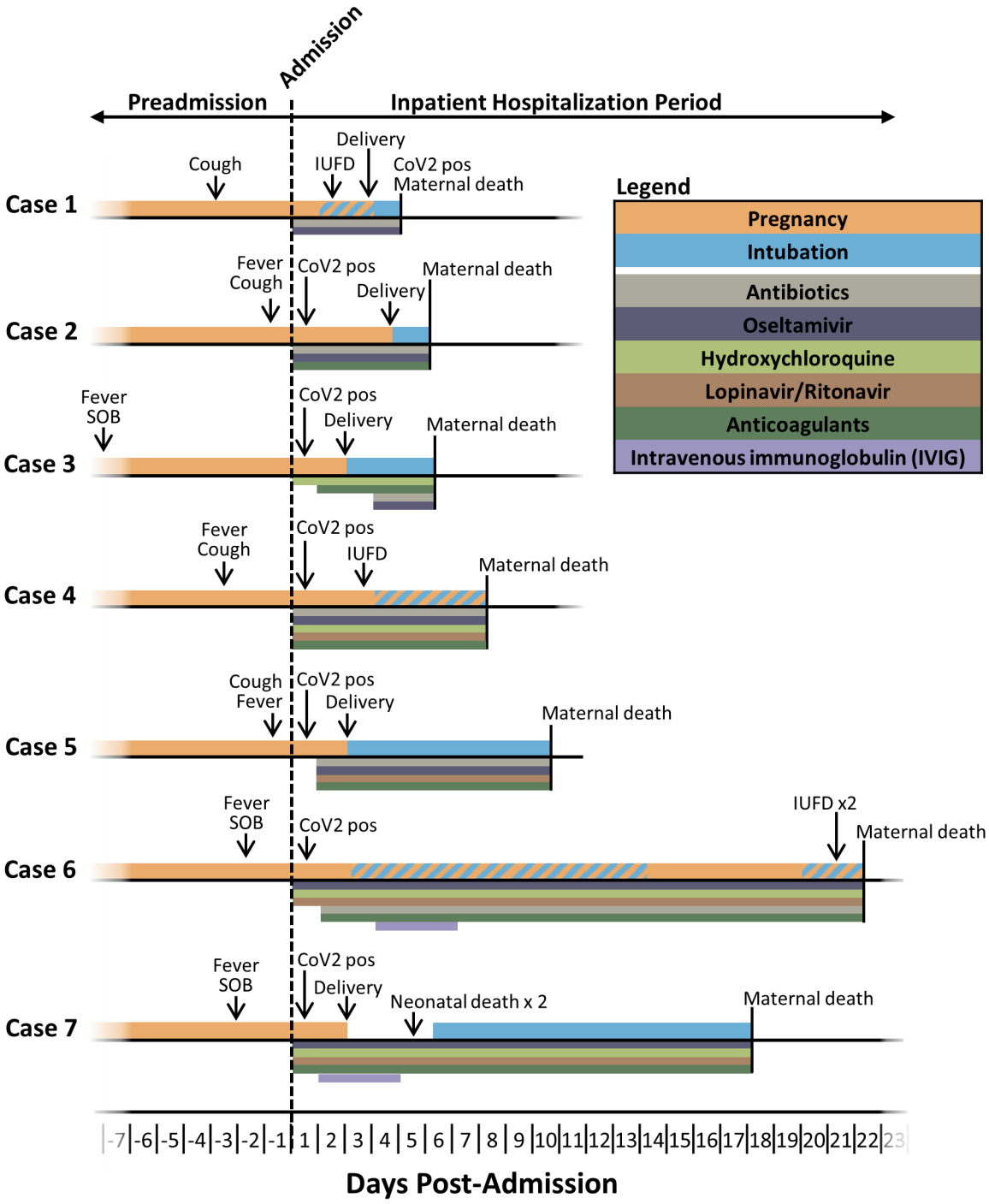
	Case 8	Case 9
Maternal characteristics		
+Maternal age (yr)	35-39	35-39
Gravida, para	G1 P0	G2 P0010
Co-morbidities	AMA Obesity	GDMA1 (diet controlled) AMA Obesity
Blood type (Rh)	O (+)	O (+)
Influenza vaccinated	No	Yes
Admit BMI (kg/m^2)	32	31
Presenting symptoms		
Fever	yes	yes
Cough	yes	yes
Dyspnea	yes	yes
Myalgia	no	no
Medications		
Antivirals	yes	yes
Antibiotics	yes	yes
Anticoagulants	yes	yes
Other	HCQ, plasma	HCQ
Laboratory values*		
SARS-CoV-2 NAT	positive	positive
Hemoglobin, g/dL	8 (8,10)	7.6 (7.2,7.8)
Platelets, x 10^3/μL	275(262,284)	145(122,270)
WBC, x 10^9/L	9.4 (8,9.8)	26(16,32)
#Lymphocyte, % 10^9/L	9% (8.5, 9.4)	8.5% (8.2,8.8)
&CRP, mg/L	45 (38,47)	210(120,235)
AST (U/L)	80 (66,94)	172(88,178)
ALT (U/L)	6 2(26,68)	126(48,132)
Cr (mg/dL)	0.6(0.6-1.2)	0.6(0.5-1.7)
^^O_2 Sat, % (SaO2)	60	85
Maternal status (current as of April 20, 2020)		
Intubated or inpatient recovery	extubated, inpatient	discharged

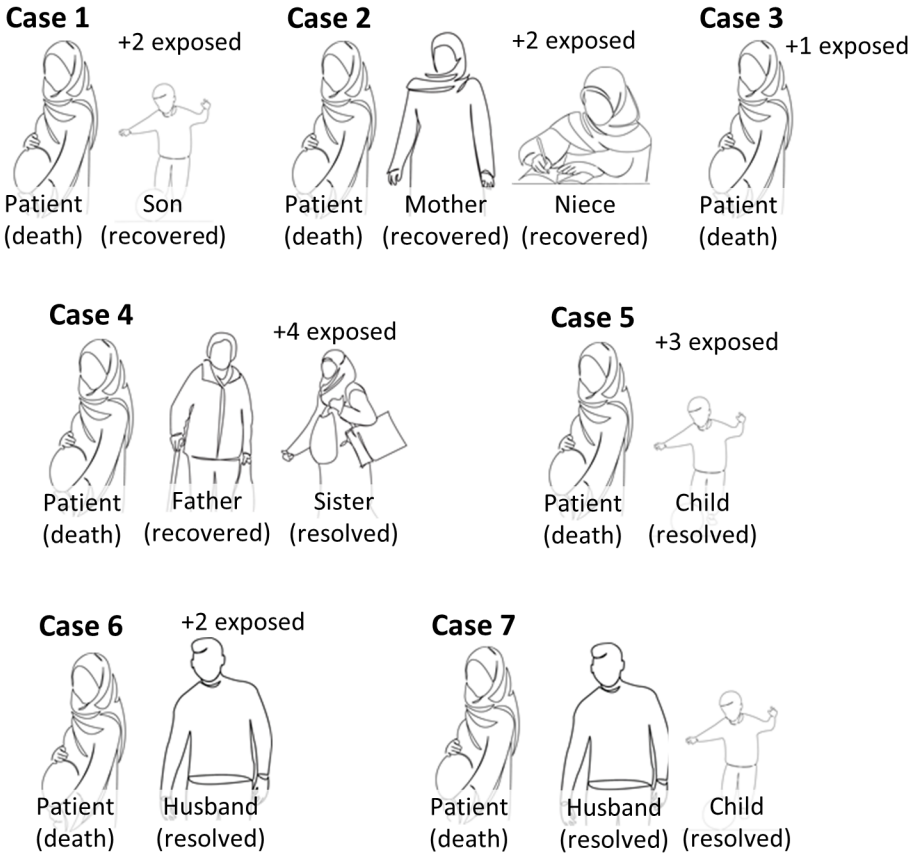
Table S1. Maternal characteristics and outcomes among pregnant patients with SARS-CoV-2 suffering severe morbidity, but not death (current as of April 20, 2020).
GDM, gestational diabetes mellitus; HCQ, hydroxychloriquine; IFN, interferon-alpha nebulizers; plasma, under a separate IRB, received immunotherapy through convalescent plasma transfusion from a recovered COVID-19 donor with known seropositivity; unk, unknown values; AMA, advanced maternal age; NAT, nucleic acid testing by rRT-PCR; WBC, white blood cell count; Cr, serum creatinine.
For antiviral and antibiotic regiments, please see methods and case narratives.
+For protection of patient identification, maternal age was gated in inclusive 5 year blocks.
^Case 1 did not receive seasonal influenza vaccination, but did have negative influenza testing during hospitalization for SARS-CoV-2.
*For all laboratory values, the initial value at the time of admission is provided, with the trough and peak from the hospitalization interval (trough, peak).
Lymphopenia was defined as 10%;
&Elevated C-reactive protein (CRP) was defined as >10 mg/L.
^^SaO2 values are as reported at the time of diagnosis of ARDS and intubation.

	Case 8	Case 9
Fetal or neonatal outcome (current as of April 20, 2020)		
Fetal death	no	yes
Gestational age, weeks	33 6/7	36 0/7
Neonatal demise	no	n/a
Mode of delivery	Cesarean	Cesarean
Birthweight, grams	1800	3000
APGARS, 1,5 minute	6,7	0,0
DCDA twin gestation	no	no
SARS-CoV-2 NAT	negative	n/a
Neonatal pneumonia	no	n/a
Neonatal lymphopenia	no	n/a

Table S2. Perinatal outcomes among pregnant patients with SARS-CoV-2 suffering severe morbidity, but not death (current as of April 20, 2020).

DCDA, dichorionic, diamniotic twin gestation; NSVD, normal spontaneous vaginal delivery; n/a, not applicable.


Admission
Preadmission
Inpatient Hospitalization Period
Case 1
Cough
Delivery
IUFD
CoV2 pos
Maternal death
Case 2
Fever
Cough
CoV2 pos
Delivery
Maternal death
Case 3
Fever
SOB
CoV2 pos
Delivery
Maternal death
Case 4
Fever
Cough
CoV2 pos
IUFD
Maternal death
Case 5
Cough
Fever
CoV2 pos
Delivery
Maternal death
Case 6
Fever
SOB
CoV2 pos
IUFD x2
Maternal death
Case 7
Fever
SOB
CoV2 pos
Delivery
Neonatal death x 2
Maternal death
Legend
Pregnancy
Intubation
Antibiotics
Oseltamivir
Hydroxychloroquine
Lopinavir/Ritonavir
Anticoagulants
Intravenous immunoglobulin (IVIG)
-7 -6 -5 -4 -3 -2 -1 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23
Days Post-Admission


Case 1
+2 exposed
Patient (death)
Son (recovered)
Case 2
+2 exposed
Patient (death)
Mother (recovered)
Niece (recovered)
Case 3
+1 exposed
Patient (death)
Case 4
+4 exposed
Patient (death)
Father (recovered)
Sister (resolved)
Case 5
+3 exposed
Patient (death)
Child (resolved)
Case 6
+2 exposed
Patient (death)
Husband (resolved)
Case 7
Patient (death)
Husband (resolved)
Child (resolved)