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Case Report

COVID-19 as a potential trigger for mirror syndrome: exploring an infectious etiology in a rare obstetric triad

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ABSTRACT

Mirror syndrome (Ballantyne's syndrome)—the triad of maternal edema, fetal hydrops, and placental hydrops—is a sporadic, life-threatening condition. Its etiology is diverse, including infections, isoimmunization, and fetal anomalies. This case report aims to explore and highlight a possible correlation between COVID-19 infection and the precipitation of mirror syndrome in a primigravida. In this case, a 25-year-old primigravida (PCOS history) presented to OPD at 28 weeks 4 days gestation with dyspnea and grade IV edema/anasarca. Ultrasound and MRI confirmed fetal hydrops (ascites, pleural effusion), polyhydramnios (Liquor 25-26 cm), and placentomegaly, fulfilling the diagnostic criteria for mirror syndrome. Further evaluation revealed maternal bilateral pleural effusion and consolidation, confirmed as a new-onset COVID-19 infection by CT and RT-PCR. Due to PPROM, the pregnancy was terminated, resulting in a stillbirth. Mirror syndrome's pathogenesis is thought to involve trophoblastic damage and endothelial dysfunction, similar to preeclampsia. Given that COVID-19 infection is known to induce systemic inflammation, coagulopathy, and endothelial injury, the timing of the maternal infection in this case suggests a potential infectious etiology. While a direct causal link requires further study, this case supports the hypothesis that COVID-19 may act as a trigger, contributing to the angiogenic-antiangiogenic imbalance implicated in the syndrome. This case highlights an unusual presentation of mirror syndrome coinciding with acute maternal COVID-19 infection. Due to the high morbidity/mortality of mirror syndrome, we advise wider screening for this condition in COVID-19 positive pregnant patients, and vice versa, to ensure early detection and prompt, etiology-based management.

Keywords: Polycystic ovarian syndrome, Coronavirus disease 2019, Premature prelabour rupture of membranes, Reverse transcriptase polymerase chain reaction, Computerised tomography

INTRODUCTION

Mirror syndrome (MS), also referred to as Ballantyne's syndrome or Maternal hydrops syndrome, is a rare and severe complication of pregnancy, characterized by the clinical triad of fetal hydrops, placental edema (placentomegaly), and maternal edema.[1,2] First described in 1892 by John William Ballantyne, this condition is named for the way maternal symptoms—particularly edema—*mirror* the hydropic state of the fetus.[1] Fetal hydrops is a relatively rare complication of pregnancy, but when it does occur, 5%-30% of cases are further complicated by the occurrence of MS.[3] MS has been described complicating approximately 0.02% of all pregnancies during the second and third trimester.[4] However, given the clinical overlap with preeclampsia, its prevalence is likely underreported.[4] The underlying pathophysiology of MS remains poorly understood. Current theories suggest a genesis similar to that of preeclampsia, where a dysregulation of placenta-mediated angiogenic and antiangiogenic factors leads to uteroplacental ischemia.[3] Many of these same factors,

including soluble FMS-like tyrosine kinase 1 (sFlt-1), which is believed to be produced by the hydropic placenta, appear to play a role in the development of MS.[4] Clinically, MS is often underdiagnosed or misdiagnosed due to its preeclampsia-like manifestations. The mother may develop proteinuria, hypertension, and even severe preeclampsia; approximately 60% of patients with MS have hypertension.[5] Due to irreversible fetal hydrops and a consistently poor fetal prognosis, with mortality reported as high as 67.26%.[6] Most literature focuses on recognizing MS as a unique maternal condition. The primary treatment strategy remains pregnancy termination or delivery for maternal indications.[6]

Interestingly, the reversal of these maternal symptoms can occur after intrauterine transfusion in parvovirus-induced hydrops without the need for delivery.[5] This observation, along with others, implies that while MS and preeclampsia share similar molecular pathways, the inciting event in MS is the underlying fetal pathology.[7] Here, we present an unusual and challenging case of MS in a primigravida diagnosed with acute COVID-19 infection. Given that infectious etiologies are a recognized cause of fetal hydrops, and COVID-19 is known to induce systemic endothelial damage, this report aims to explore a possible correlation between the viral infection and the precipitation of MS.

CASE REPORT

A 25-year-old primigravida from Guntur presented to the outpatient department (OPD) at 28 weeks and 4 days of gestation with a two-week history of worsening shortness of breath (dyspnea, Grade 2) and significant generalized swelling (Grade IV edema/anasarca). Her history was notable for polycystic ovary syndrome (PCOS) with irregular cycles, but she conceived spontaneously after four years of marital life. She had no prior history of chronic conditions, including hypertension, diabetes, or cardiovascular, respiratory, or thyroid disorders. Importantly, she reported no history of fever or cough on initial presentation.

Table 1: Patient vitals.

Vitals	At admission	After 2 days
BP	110/70 mm hg	110/80 mm hg
PR	82/min	114/min
SpO2	99% at room air	99% at room air
Temperature	98.4 F	100.7 F
RR	14/min	18/min

Upon admission, her vitals were charted: blood pressure (BP) was 110/70 mmHg, pulse rate (PR) 82/min, respiratory rate (RR) 14/min, and SpO2 99% on room air, with a temperature of 98.4°F. Over the next two days, her condition changed, with her temperature rising to 100.7°F and PR to 114/min. Initial laboratory investigations were notable for leukocytosis (TLC -19700/mm), mild anaemia (Hb-10.2 g/dl), and elevated ESR-29/mm. HbA1c was 6.2%, suggesting pre-diabetic tendencies, but OGTT was normal. Renal and liver profiles, including Creatinine-0.69 mg/dl, and indirect Coomb's test - negative.

Obstetric ultrasound demonstrated a single live foetus corresponding to 32-week period of gestation with severe polyhydramnios (25-26cm). Crucially, the foetus exhibited features of hydrops fetalis (Figure 1), including significant pleural effusion and ascites. Fetal MRI further confirmed the findings of fetal hydrops and placentomegaly (Figure 2). Maternal chest X-ray showed bilateral pleural effusion and consolidation of the left lower lobe of the lung. Alongside the clinical development of fever, these radiological findings were confirmed by CT scan and RT-PCR, leading to the diagnosis of COVID-19 positive infection.

Figure 1: Hydrops foetalis.

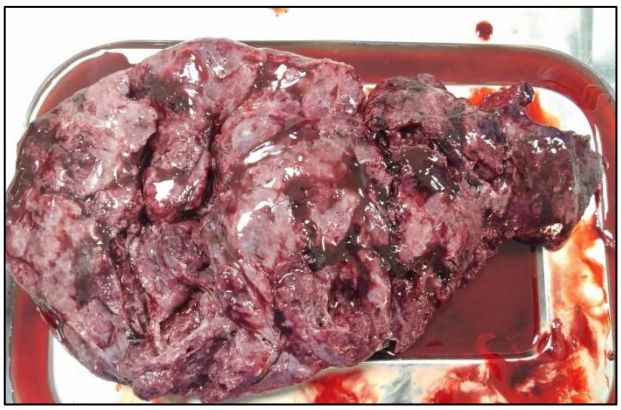

Figure 2: Placentomegaly.

Given the diagnosis of MS in the setting of COVID-19 and the development of pre-labour rupture of membranes (PPROM), consent was obtained for termination of the

pregnancy. She was induced using two dinoprostone gels intra cervically and progressed well to a vaginal delivery. The delivery resulted in a stillbirth of a hydropic baby with noted placentomegaly and no other external anomalies. Tissue samples were sent for autopsy, with reports currently pending. The mother recovered well from both the acute COVID-19 infection and the severe edema/dyspnea, and was subsequently discharged.

Table 2: Patient investigations.

Hematology		**Infectious**	
HB	10.2 g/dl	HIV	NR-non reactive
TLC	19700/mm^3	HBSAG	NR
Platelet	4.04 lakh/mm^3	Indirect coombs	NEG
ESR	29 mm	**Cardiac**	
Renal and electrolyte		ECG	Normal sinus rhythm
Urea	24 mg/dl		
CREAT	0.69 mg/dl		
NA	140 meq/		
K	4.2 meq/l		
CL	103 meq/l		
Urine ALB/SUG	Trace/nil		
Glucose metab			
HBAIC	6.2%		
FBS	94 mg/dl		
PPBS	154 mg/dl		
OGTT	92 mg/dl		
Mean blood glucose	141 mg/dl		

DISCUSSION

MS remains a diagnostic challenge due to its rarity and significant clinical overlap of symptoms with severe preeclampsia. In our patient, the classic clinical triad was present: fetal hydrops, placentomegaly, and severe maternal edema (anasarca), which pointed us towards MS.[1,2] MS is frequently misdiagnosed because its symptoms—such as proteinuria and hypertension—mirrorpreeclampsia.[4-6] While approximately 60% of patients with MS develop hypertension.[1] Our patient was notable for the initial absence of hypertension and significant proteinuria. This prompted a more comprehensive investigation that eventually uncovered the severe fetal pathology.[2,3] Comparing our study with other studies, the period of gestation when MS classically presents is the late second trimester and third trimester. (It was 28 weeks in our study).

Current theories suggest a genesis for MS similar to preeclampsia, where a dysregulation of placenta-mediated angiogenic and antiangiogenic factors and endothelial dysfunction leads to systemic issues.[5] Factors such as sFlt-1, believed to be produced by the hydropic placenta, appear to play a key role in the development of the syndrome by inducing widespread endothelial dysfunction.[4,5] The etiology in this case appears to be infectious, coinciding with the acute diagnosis of COVID-19 infection. Viral infection is a known cause of fetal hydrops that can trigger MS.[3-7] As SARS-CoV-2 is characterized by widespread inflammation and profound endothelial injury, it likely serves as a precipitating factor that exacerbates the fetal hydropic process, leading to the rapid decompensation observed as MS.[5] COVID-19 is a systemic disease characterized by widespread inflammation, microvascular thrombosis, and profound endothelial injury, often presenting with pulmonary and systemic edema. The key differentiators in this case—early onset (28 weeks), profound edema, and the rapid confirmation of severe fetal hydrops/placentomegaly on imaging—were essential for accurate diagnosis. Fetal prognosis in most cases was guarded with most cases being either a second-trimester abortion, stillbirth, or intrauterine death. Our case had a stillbirth. While fetal therapy can occasionally reverse symptoms in specific etiologies which lead to live birth of about 55% in study by sichitiu et al the primary treatment remains delivery.[1,2]

Maternal outcome improved after delivery in almost all cases, including our case. Prompt delivery was necessitated by the worsening maternal respiratory status (due to pleural effusion/consolidation from COVID-19) and PPROM, which is the definitive treatment path for improving maternal outcome, given the consistently guarded fetal prognosis.

The successful resolution of our patient's edema and dyspnea post-delivery substantiates the diagnosis, as maternal symptoms characteristically resolve within two weeks following delivery.2-6 Maternal complications like ICU stay, blood transfusion, hypertension, and postpartum haemorrhage were present in many cases and were mostly individualised and managed like covid 19 infection in our case. The striking temporal association between the acute

onset of COVID-19 and the presentation of MS suggests that the infectious agent is a strong contributing or precipitating factor. We have compared our study with 4 other studies in Table 3.

Table 3: Discussion.

Study	Age of onset	Associated etiology	Symptoms	Outcome -fetal	Outcome -maternal
Mogharbel et al[1]	median age at DIA-23w 3 day Median age at birth -25 weeks	Fetal hydrops (10/276)	Hypertension, Weight gain, Anaemia and hyperuricaemia	66.7% still birth 25% neonatal death Structural anomalies -50%	Improved after delivery
Chen et al[2]	2nd and 3rd trimester	Fetal hydrops (16/85)	Hypertension, Hellp, Cardiac failure Lab-anaemia, Proteinuria	Neonatal deaths and still births mostly	Improved after delivery
Shichitiu et al[3]	27±3.8 weeks	Fetal hydrops-21 cases	TTTS, anaemia	Livebirths: 55.5% (15/27). Neonatal deaths: 25.9% (7/27). Intrauterine deaths: 18.5% (5/27).	Improved after termination or fetal intervention
Han et al[6]	22.6-34 weeks	Fetal hydrops - 10/37	Anaemia, weight gain, proteinuria and hypertension	Two very preterm Twin infants survived and one preterm neonate died. Rest all were 2nd trimester abortions	Postpartum haemorrhage (7/14), placenta accreta (5/14), acute left heart failure (3/14), renal dysfunction (3/14), pulmonary edema (2/14), Hellp syndrome (2/14), liver dysfunction (1/14), placental abruption (1/14), and disseminated intravascular coagulation (DIC) (1/14). outcome improved After delivery
Our study	28 weeks	COVID-19 - infectious agent causing fetal hydrops	Proteinuria, fever, tachypnoea, and tachycardia	Fetal -still birth	Improved after delivery, Recovered from COVID-19

CONCLUSION

MS (Ballantyne's syndrome) is a rare and severe obstetric complication requiring urgent recognition to prevent high maternal and fetal morbidity and mortality. Its accurate detection and differentiation from similar conditions like preeclampsia is paramount. This case report highlights the critical finding of MS coinciding with a confirmed acute maternal COVID-19 infection. Given that known etiologies of this syndrome include viral infections, and considering the extensive endothelial damage associated with SARS-CoV-2, we propose that COVID-19 infection in pregnancy may serve as a potential trigger or contributing factor in the precipitation of MS. Therefore, we strongly recommend a wider clinical index of suspicion for MS in pregnant patients presenting with edema and acute COVID-19 infection, and vice versa. Early multidisciplinary detection and prompt, etiology-based management remain the primary goals for optimizing outcomes.

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