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# Acute Cytomegalovirus Infection Mimicking Atypical Preeclampsia and Complicated by Pulmonary Embolism in Late Pregnancy

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## Abstract

Primary cytomegalovirus (CMV) infection during pregnancy is the most common congenital viral infection worldwide, yet maternal infection is often asymptomatic or presents with non-specific symptoms. In rare cases, acute CMV infection causes significant maternal hepatitis and systemic illness, closely resembling pregnancy-specific conditions such as preeclampsia or acute fatty liver of pregnancy (AFLP). Awareness of these atypical presentations is important to avoid unnecessary intervention while ensuring timely management of maternal and fetal complications.

We report a 30-year-old gravida 2 para 1 woman who presented at 33 + 5 weeks' gestation with acute chest and shoulder pain. Initial investigations, including electrocardiography and computed tomography pulmonary angiography (CTPA), were unremarkable, apart from an isolated elevation in alanine aminotransferase (ALT), and she was discharged. Two days later, she re-presented with worsening chest pain, dyspnea, headache, pruritus, swollen hands, dark urine, pale stools, reduced appetite, abdominal discomfort, and proteinuria. Despite normal blood pressure, progressive respiratory compromise and persistently deranged liver function prompted further investigation. Repeat contrast-enhanced CT demonstrated small bilateral pulmonary emboli, and therapeutic low molecular weight heparin (LMWH) was commenced. Ongoing elevation of liver enzymes, oliguria, splenomegaly, proteinuria, headache, and evidence of mild fetal growth restriction raised concern for atypical preeclampsia despite persistent normotension. She received magnesium sulfate, antenatal corticosteroids, and close maternal and fetal surveillance, and delivery was initially planned within 24 hours. However, subsequent maternal improvement and positive CMV IgM serology prompted reconsideration of the diagnosis. Gastroenterology review attributed the liver dysfunction to acute CMV infection, and conservative multidisciplinary management was pursued. Following further monitoring and a subsequent brief admission for recurrent respiratory symptoms, labor was induced at 37 weeks of gestation, resulting in an uncomplicated vaginal delivery. The neonate tested positive for CMV with a very low viral load. The mother made a good recovery following transition from LMWH to apixaban postpartum.

This case highlights the diagnostic complexity of acute CMV infection in late pregnancy, particularly when presenting with abnormal liver function tests, respiratory symptoms, and features suggestive of atypical preeclampsia. It also illustrates the rare coexistence of acute CMV infection and pulmonary embolism, emphasizing the need to consider multiple concurrent pathologies rather than attributing all clinical findings to a single diagnosis. Multidisciplinary collaboration, repeated clinical reassessment, and careful interpretation of evolving investigations were essential in avoiding unnecessary preterm delivery while ensuring maternal and fetal safety.

**Categories:** Obstetrics/Gynecology
**Keywords:** cytomegalo virus (cmv), high-risk obstetrics, maternal and fetal medicine, plum embolism, pre-eclampsia

## Introduction

Cytomegalovirus (CMV), an enveloped double-stranded DNA herpes virus, is the most common congenital viral infection. It is also known as human herpesvirus 5 (HHV-5), and like other herpes viruses, it becomes latent after a primary infection but can reactivate with renewed viral shedding or from a new strain. A recently published systematic review and meta-analysis estimated a global CMV seroprevalence of 83% in the general population, 86% in women of childbearing age, and 86% in donors of blood or organs. For each of these three groups, the highest seroprevalence was seen in the World Health Organization (WHO) Eastern Mediterranean region (90%) and the lowest in the WHO European region 66% [1].

CMV is the most common congenital infection, affecting approximately 0.48% of live-born infants in high-income countries and 1.42% in low- and middle-income countries. It can cause significant morbidity, particularly in infants who are symptomatic at birth. CMV is also the leading non-genetic cause of sensorineural hearing loss (SNHL) and is an important cause of long-term neurological impairment [2].

Transmission of CMV to susceptible individuals occurs through direct contact with body fluids such as saliva, urine, or semen of someone who is actively shedding the virus. The diagnosis of primary CMV in pregnant women based on clinical signs and symptoms is difficult and unreliable, as up to 90% of infected mothers are asymptomatic and signs, when present, are non-specific (rhinitis, pharyngitis, myalgia, arthralgia, headache, and fatigue). Pregnancy does not appear to affect clinical severity. Routine antenatal screening for CMV is not currently recommended outside of research settings. This is mainly because CMV screening does not meet all the criteria required for an effective screening program, particularly as there is currently no established treatment available to prevent or reduce the effects of congenital CMV infection [1].

CMV serological testing should be considered in pregnant women who develop influenza-like or glandular fever-like symptoms, particularly when Epstein-Barr virus (EBV) testing is negative, as well as in those presenting with symptoms suggestive of hepatitis. Testing should also be considered when routine antenatal ultrasound identifies fetal abnormalities that may be associated with congenital CMV infection. These include ventriculomegaly, microcephaly, intracranial calcifications, intraventricular synechiae, intracranial hemorrhage, periventricular cysts, cerebellar hypoplasia, cortical abnormalities, echogenic bowel, fetal growth restriction (estimated fetal weight below the third centile), pericardial effusion, ascites, and fetal hydrops [2].

The diagnosis of primary CMV infection in pregnancy can be made by one of the following findings: (1) the appearance of CMV-specific IgG in a woman who was previously seronegative or (2) the detection of CMV IgM antibody with low IgG avidity, indicating recent primary infection [2]. Amniocentesis can be offered to assess for fetal infection, but this should generally be performed at least seven to eight weeks after the suspected maternal infection and after 21 weeks of gestation.

Antiviral treatment for CMV during pregnancy is limited due to concerns about drug toxicity and fetal safety. Valaciclovir has been investigated in pregnancies affected by fetal CMV infection and is preferred over aciclovir because of its better oral absorption. However, both drugs have limited activity against CMV, and their use in pregnancy requires careful consideration of the potential benefits and risks [2]. The routine use of CMV hyperimmune globulin is not recommended for the prevention of congenital CMV infection. All pregnant women should also be provided with advice on appropriate hygiene measures to reduce the risk of CMV transmission, regardless of their CMV serostatus [3].

Neonatal testing for CMV should be performed using PCR within the first three weeks of life. Symptomatic infants, particularly those with central nervous system or hearing abnormalities, should be considered for treatment with valganciclovir, ideally starting within the first month after birth. Infants with congenital CMV infection should receive ongoing follow-up, with monitoring for developmental and hearing problems [4]. All infants with CMV infection at birth should be followed up at a minimum of up to two years of age or later, depending upon the disease status, to check hearing and brain development [3].

Preeclampsia is a pregnancy-related disorder characterized by new-onset hypertension after 20 weeks of gestation, accompanied by proteinuria and/or maternal organ dysfunction. However, atypical presentations may occur without hypertension or proteinuria, making diagnosis challenging [5]. Symptoms may include persistent headache, visual disturbances, upper abdominal or epigastric pain, abnormal liver function tests, thrombocytopenia, and, in severe cases, seizures. These features may overlap with other conditions, including CMV-associated hepatitis, acute fatty liver of pregnancy (AFLP), and intrahepatic cholestasis of pregnancy. Therefore, distinguishing atypical preeclampsia from infectious and other pregnancy-related liver disorders is essential to guide appropriate maternal and fetal management. This highlights the importance of considering CMV infection in pregnant women presenting with unexplained liver dysfunction, particularly when the clinical presentation is atypical.

We present a challenging case of CMV infection during pregnancy, characterized by abnormal liver function tests that raised suspicion of atypical preeclampsia and complicated by pulmonary embolism.

## Case Presentation

A 30-year-old gravida 2 para 1 woman presented to the emergency department at 33 + 5 weeks of gestation with acute-onset chest and shoulder pain, which had started earlier that morning. She described the pain as an “icy” sensation in her chest. There was no associated shortness of breath, cough, or fever. Her vital signs were within normal limits, and physical examination, including cardiovascular and respiratory examination, was unremarkable. Her body mass index (BMI) was 35.96 kg/m$^2$.

Her past medical history was significant for COVID-19 infection during her first pregnancy, complicated by transiently elevated liver enzymes, which resolved postpartum. She had no other medical or obstetric complications. She smoked fewer than five cigarettes per day at the start of pregnancy. Initial investigations included an ECG and CT pulmonary angiography (CTPA), both of which were unremarkable. Blood tests were unremarkable (Table *1*) apart from an isolated elevation in alanine aminotransferase (ALT) (Figure *1*). She was reassured and discharged home from the emergency department.

| Parameters | Results | Normal Values |
|---|---|---|
| WCC | 7.1 | 4.0-10.0 x $10^9$/L |
| HB | 133 | 120-150 g/L |
| Platelets | 193 | 150-410 x $10^9$/L |
| CRP | 36 | 0-5 mg/L |
| Potassium | 3.9 | 3.5-5.1 mmol/L |
| Urea | 2.6 | 2.8-8.1 mmol/L |
| Sodium | 134 | 136-145 mmol/L |
| eGFR | >90 | >90 mL/min/1.73 $m^2$ |
| Creatinine | 53 | 45-84 µmol/L |
| PT | 13.5 | 11.6-14.6 s |
| INR | 1 | 0.9-1.2 |
| APTT | 32.9 | 25.7-39.5 s |
| Albumin | 25 | 35-52 g/L |
| ALP | 296 | 35-104 U/L |
| Total bilirubin | 15 | 0-21 µmol/L |
| Total protein | 58 | 64-83 g/L |

**TABLE 1: Initial blood tests**

WCC: White cell count; HB: Hemoglobin; CRP: C-reactive protein; eGFR: Estimated glomerular filtration rate; PT: Prothrombin time; INR: International normalized ratio; APPT: Activated partial thromboplastin time; ALP: Alkaline phosphatase.

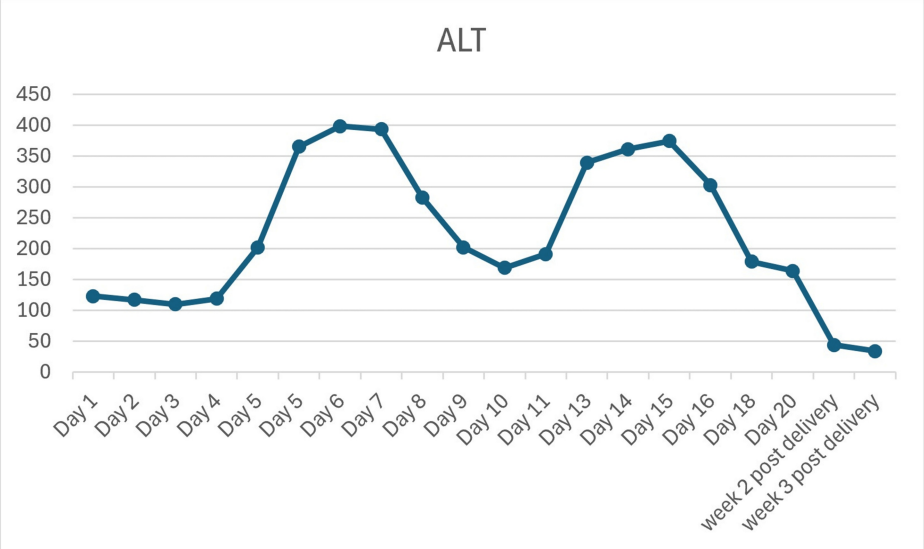



**FIGURE 1: ALT levels during the clinical course**

ALT: Alanine aminotransferase.

## Antenatal course before admission

The pregnancy had been uncomplicated until this point. Booking blood tests were normal, including CMV

IgG and IgM. She reduced smoking to two to three cigarettes per day and stopped completely by 17 weeks of gestation. She had a normal glucose tolerance test, and serial fetal growth scans were normal.

## Re-presentation and clinical deterioration

Two days later, she re-presented to the maternity unit with worsening chest pain and shortness of breath. She was unable to speak in full sentences and additionally reported headache, pruritus of the lower limbs, swollen hands, reduced appetite, mild abdominal pain, dark urine, and pale stools.

On examination, her abdomen was soft and non-tender. Chest auscultation was clear, with no peripheral edema or calf tenderness. Her vital signs were normal apart from oxygen saturation of 94% on room air. Urinalysis demonstrated proteinuria (+). She was reviewed by the medical team, who advised exclusion of obstetric causes, including intrahepatic cholestasis of pregnancy and preeclampsia, given the recent normal CTPA and persistently elevated ALT (Figure *1*).

Over the following hours, her condition deteriorated with an increasing respiratory rate up to 35 breaths per minute. Her oxygen saturation was 95%, blood pressure was 109/73 mmHg, pulse was 88 bpm, and temperature was 36.4°C. She was commenced on the sepsis pathway, and a surgical review was requested.

The surgical team advised abdominal imaging to exclude AFLP and biliary obstruction. However, the patient's oxygen saturations dropped to 89%, requiring supplemental oxygen. A repeat contrast-enhanced CT scan of the head, thorax, abdomen, and pelvis was therefore performed, which demonstrated multifocal bilateral pulmonary emboli.

She was transferred to the high-dependency unit (HDU) and commenced on therapeutic-dose low molecular weight heparin (LMWH). Despite anticoagulation, her ALT continued to increase (Figure *1*), and she developed oliguria with dark urine and brisk reflexes. A comprehensive plan was made including liver ultrasound, liver disease screen (Table *2*), daily full preeclampsia blood panel (Table *3*), fetal growth scan, and continuous fetal monitoring. Her urine protein-creatinine ratio was elevated (34.1 mg/mmol), and liver ultrasound revealed splenomegaly. Blood pressure remained within the normal range throughout. She continued to report persistent headaches and swollen hands, without visual disturbance or epigastric pain. She had a fetal growth scan at 34+1 weeks, which demonstrated mild fetal growth restriction with estimated fetal weight of 2.275 g, with normal umbilical artery Doppler and normal liquor. Cardiotocography (CTG) remained reassuring, and a repeat growth scan at 35+5 weeks showed normal growth with estimated fetal weight of 2.707 g and normal umbilical artery Doppler and normal liquor.

| Parameters | Results | Normal values |
|---|---|---|
| EBV IgM | Negative | N/A |
| EBV IgG | Negative | N/A |
| EBV nuclear antigen IgG antibody | Negative | N/A |
| Hepatitis B core total antibody | Negative | N/A |
| Hepatitis C antibody | Negative | N/A |
| Hepatitis B surface antigen | Negative | N/A |
| Hepatitis A IgM antibody | Negative | N/A |
| IgA (0.70-4.00) g/L | 2.02 | 0.70-4.00 g/L |
| IgM (0.40-2.30) g/L | 0.93 | 0.40-2.30 g/L |
| IgG (7.0-16.0) g/L | 8.73 | 7.0-16.0 g/L |
| LDH (0-250) U/L | 295 | 0-250 U/L |
| Uric acid (143-339) µmol/L | 325 | 143-339 µmol/L |
| PT (11.6-14.6) s | 13.8 | 11.6-14.6 s |
| INR (0.9-1.2) | 1 | 0.9-1.2 |
| APTT (25.7-39.5) s | 49.2 | 25.7-39.5 s |
| Albumin (35-52) g/L | 24 | 35-52 g/L |
| ALP (35-104) U/L | 258 | 35-104 U/L |
| Total bilirubin (0-21) µmol/L | 9 | 0-21 µmol/L |
| ALT (0-35) U/L | 164 | 0-35 U/L |
| Total protein (64-83) g/L | 63 | 64-83 g/L |
| AST (5-40) U/L | 32 | 5-40 U/L |
| GGT (6-50) U/L | 41 | 6-50 U/L |
| Ammonia (11-51) µmol/L | 11 | 11-51 µmol/L |
| Bile acid (0-10) µmol/L | 10.3 | 0-10 µmol/L |

**TABLE 2: Liver screen**

EBV: Epstein-Barr virus; IgA: Immunoglobulin A; IgM: Immunoglobulin M; IgG: Immunoglobulin G; LDH: Lactate dehydrogenase; PT: Prothrombin time; APTT: Partial thromboplastin time; INR: International normalized ratio; ALP: Alkaline phosphatase test; ALT: Alanine transaminase; AST: Aspartate aminotransferase test; GGT: Gamma-glutamyl transferase.

| Parameters | Day 1 | Day 2 | Day 3 | Day 4 | Day 5 | Day 6 | Day 7 | Day 8 | Normal values |
|---|---|---|---|---|---|---|---|---|---|
| WCC | 6.5 | 6.2 | 6 | 4.8 | 5.2 | 4.9 | 6.1 | 5.9 | 4.0-10.0 x $10^9$/L |
| HB | 129 | 120 | 117 | 105 | 115 | 123 | 123 | 115 | 120-150 g/L |
| Platelets | 164 | 183 | 183 | 203 | 179 | 158 | 156 | 151 | 150-410 x $10^9$/L |
| CRP | 48 | 45 | 50 | 19 | 21 | 30 | 33 | 29 | 0-5 mg/L |
| Potassium | 4 | 3.8 | 4.2 | 4.1 | 3.9 | 4.1 | 3.9 | 4 | 3.5-5.1 mmol/L |
| Urea | 2.1 | 2 | 3 | 2.1 | 2.7 | 2.5 | 2.6 | 2.7 | 2.8-8.1 mmol/L |
| Sodium | 134 | 135 | 134 | 138 | 136 | 137 | 135 | 137 | 136-145 mmol/L |
| eGFR | >90 | >90 | >90 | >90 | >90 | >90 | >90 | >90 | >90 mL/min/1.73 $m^2$ |
| Creatinine | 52 | 57 | 51 | 50 | 49 | 58 | 62 | 61 | 45-84 µmol/L |
| Albumin | 22 | 21 | 20 | 20 | 20 | 21 | 19 | 18 | 35-52 g/L |
| ALP | 309 | 289 | 304 | 260 | 250 | 280 | 292 | 279 | 35-104 U/L |
| Total bilirubin | 30 | 52 | 34 | 21 | 20 | 29 | 20 | 16 | 0-21 µmol/L |
| Total protein | 55 | 53 | 53 | 50 | 51 | 54 | 50 | 50 | 64-83 g/L |

**TABLE 3: Daily blood tests**

WCC: White cell count; HB: Hemoglobin; CRP: C-reactive protein; eGFR: Estimated glomerular filtration rate; ALP: Alkaline phosphatase.

Given the constellation of findings, she was managed as atypical preeclampsia despite being normotensive. Management included fluid restriction, magnesium sulfate for seizure prophylaxis, and antenatal corticosteroids. A plan was made for delivery within 24 hours, and she was reviewed by the neonatal team to discuss risks and benefits of preterm delivery.

## Revised diagnosis and outcome

The following day, her clinical condition improved, and her blood pressure remained within the normal range, making preeclampsia less likely. Investigations performed to date (Tables *2*, *3*) also helped exclude other differential diagnoses, including obstetric cholestasis, AFLP, and viral hepatitis. Further investigations subsequently revealed positive CMV serology (Table *4*). She was then transferred to the maternity unit for close clinical observation and serial blood tests.

| Parameters | Results | Normal values |
|---|---|---|
| CMV IgM | Positive | N/A |
| CMV IgG | Positive | N/A |
| CMV IgG avidity | Moderate | N/A |
| CMV DNA level | 32361 IU/mL | <1000 IU/mL |

**TABLE 4: CMV serology**

IgM: Immunoglobulin M; IgG: Immunoglobulin G; CMV: Cytomegalovirus.

The gastroenterology team reviewed her and considered acute CMV infection a possible explanation for the deranged liver function test results, given the normal liver imaging, which excluded hepatic steatosis, and the resolution of symptoms initially suggestive of atypical preeclampsia in the absence of hypertension. They recommended CMV PCR testing. Regional maternal medicine and fetal medicine consultations were also obtained. As she remained clinically stable, she was discharged after one week with a plan for twice-weekly CTG, weekly liver function tests, and induction of labor at 37 weeks of gestation. The fetal medicine

review recommended postnatal neonatal CMV testing and treatment with ganciclovir if indicated.

### Further admission and delivery

She was re-admitted three days after with shortness of breath, fatigue, and chest pain. Vital signs and examination were normal. Investigations including blood tests (Table 5), ECG, chest X-ray, echocardiography, D-dimer (Table 4), and viral swabs (influenza, COVID-19, and respiratory syncytial virus (RSV)) were unremarkable.

| Parameters | Day 1 | Day 2 | Day 3 | Day 4 | Normal values |
|---|---|---|---|---|---|
| WCC | 7.5 | 7.1 | 6.3 | 6.5 | 4.0-10.0 x $10^9$/L |
| HB | 124 | 122 | 121 | 118 | 120-150 g/L |
| Platelets | 265 | 220 | 254 | 236 | 150-410 x $10^9$/L |
| CRP | 21 | 23 | 20 | 19 | 0-5 mg/L |
| Potassium | 3.9 | 4.4 | 4.1 | 3.6 | 3.5-5.1 mmol/L |
| Urea | 3.2 | 2.6 | 2.3 | 2.8 | 2.8-8.1 mmol/L |
| Sodium | 136 | 133 | 135 | 135 | 136-145 mmol/L |
| eGFR | >90 | >90 | >90 | >90 | >90 mL/min/1.73 $m^2$ |
| Creatinine | 64 | 60 | 59 | 59 | 45-84 µmol/L |
| PT | 13.7 | 13.5 | 13.8 | 13.6 | 11.6-14.6 s |
| INR | 1 | 1 | 1 | 1 | 0.9-1.2 |
| APTT | 44.9 | 45.1 | 49.2 | 48.2 | 25.7-39.5 s |
| Albumin | 24 | 22 | 23 | 21 | 35-52 g/L |
| ALP | 301 | 302 | 307 | 284 | 35-104 U/L |
| Total bilirubin | 19 | 18 | 16 | 12 | 0-21 µmol/L |
| Total protein | 59 | 59 | 59 | 59 | 64-83 g/L |

**TABLE 5: Re-admission blood tests**

WCC: White cell count; HB: Hemoglobin; CRP: C-reactive protein; eGFR: Estimated glomerular filtration rate; PT: Prothrombin time; INR: International normalized ratio; APPT: Activated partial thromboplastin time; ALP: Alkaline phosphatase.

She was admitted for observation and daily blood tests; she was advised to continue her therapeutic LMWH and commenced on ursodeoxycholic acid for liver dysfunction. Her symptoms improved during her admission, and her blood tests were unremarkable (Table 5). She was discharged with a planned induction of labor. Her delivery was an uneventful vaginal delivery. Postnatally, the patient was transitioned from LMWH to apixaban and made a good recovery. The neonate had a urine CMV test in the first two weeks, which was positive. However, his CMV PCR level was very low.

## Discussion

This case illustrates the diagnostic complexity of liver dysfunction and cardiorespiratory symptoms in late pregnancy, particularly in the absence of sustained hypertension. Preeclampsia remains a leading cause of maternal and perinatal morbidity; however, atypical presentations without hypertension are increasingly recognized and may include neurological symptoms, proteinuria, and hepatic dysfunction [6].

In this patient, the initial clinical picture was concerning for preeclampsia, intrahepatic cholestasis of pregnancy, and AFLP. The presence of headaches, brisk reflexes, proteinuria, rising transaminases, and fetal growth restriction supported a provisional diagnosis of atypical preeclampsia, despite consistently normal blood pressure readings. The decision to initiate magnesium sulfate and corticosteroids was therefore clinically appropriate while further investigations were ongoing.

The subsequent identification of acute CMV infection provided an alternative explanation for the hepatic dysfunction, systemic symptoms, and splenomegaly. CMV infection in pregnancy is often asymptomatic in the mother but can present with hepatitis, malaise, and deranged liver function tests. Importantly, CMV infection may mimic obstetric conditions and delay diagnosis if not considered early [7,8].

The diagnosis was further complicated by the development of bilateral pulmonary emboli, highlighting the hypercoagulable state of pregnancy and the need for continued clinical vigilance even after initially negative imaging [9]. The coexistence of CMV infection and venous thromboembolism has been described, with CMV thought to contribute to a prothrombotic state through endothelial activation and inflammatory mechanisms [10,11].

Multidisciplinary management involving obstetrics, internal medicine, gastroenterology, maternal medicine, fetal medicine, neonatology, and radiology was essential in guiding both maternal and fetal care. Conservative management with close surveillance allowed safe prolongation of pregnancy to 37 weeks, balancing maternal stability with fetal maturity.

This case emphasizes that not all cases of liver dysfunction and neurological symptoms in pregnancy are due to hypertensive disorders and that viral infections such as CMV should be included in the differential diagnosis. Early recognition may prevent unnecessary preterm delivery while ensuring appropriate neonatal follow-up.

## Conclusions

This case highlights the difficulty of diagnosing liver dysfunction and cardiorespiratory symptoms in late pregnancy, particularly when the symptoms overlap with other conditions such as preeclampsia and pulmonary embolism. The presence of headache, proteinuria, brisk reflexes, elevated liver enzyme levels, and fetal growth restriction initially raised concern for atypical preeclampsia, despite the patient remaining normotensive. However, the later diagnosis of acute CMV infection provided an alternative explanation for the abnormal liver function and other systemic symptoms. The case emphasizes the importance of considering a broad range of diagnoses when a pregnant woman presents with abnormal liver function tests and symptoms that do not clearly fit one condition. Infectious causes such as CMV should be considered alongside obstetric and hepatic conditions. The development of pulmonary emboli also highlights the importance of continued clinical assessment when symptoms worsen, even when previous investigations have been reassuring.

This case also demonstrates the importance of multidisciplinary management. Input from obstetric, medical, gastroenterology, maternal medicine, fetal medicine, radiology, and neonatal teams helped guide both maternal and fetal care. Close monitoring allowed the pregnancy to continue safely to 37 weeks, while appropriate treatment was provided for the pulmonary emboli and liver dysfunction. Overall, this case demonstrates that atypical presentations in pregnancy require careful and repeated assessment, particularly when the clinical findings do not fit a single diagnosis. It highlights the importance of avoiding a narrow focus on preeclampsia in the absence of hypertension and considering other causes, including CMV infection and thromboembolic disease. Early recognition, appropriate investigation, and multidisciplinary management can help achieve good maternal and neonatal outcomes while avoiding unnecessary preterm delivery.

## Additional Information

### Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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