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CASE REPORT

Atypical Hemolytic Uremic Syndrome After Post-Abortion Infection: Case Report and Literature Review

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Background: The atypical hemolytic uremic syndrome (aHUS) presents diagnostic and therapeutic challenges due to overlapping features with other conditions like Preeclampsia/HELLP syndrome and thrombotic thrombocytopenic purpura (TTP). Pregnancy-associated aHUS is rare but carries a high risk of end-stage renal disease without prompt intervention.

Case Presentation: A 41-year-old female developed severe abdominal pain, acute kidney injury, and microangiopathic hemolysis following a surgical abortion five days ago. Laboratory findings revealed thrombocytopenia, schistocytes, elevated lactate dehydrogenase, and creatinine. Fragmented red blood cells were observed in the peripheral blood smear. Infection and complement dysregulation were suspected triggers. Despite normal complement levels, aHUS was diagnosed. Continuous renal replacement therapy stabilized renal function, but eculizumab was declined due to cost constraints.

Discussion: This case highlights aHUS triggered by early miscarriage and postoperative infection, supporting the "multiple-hit" hypothesis. Diagnostic challenges include distinguishing aHUS from other TMAs, particularly with normal complement levels. Early plasmapheresis and eculizumab are recommended, though economic barriers may limit treatment options. Therapeutic plasma exchange demonstrated efficacy in renal recovery despite the absence of targeted therapy.

Conclusion: This report expands the clinical spectrum of aHUS to include early pregnancy loss as a potential trigger. It underscores the importance of rapid diagnosis, multidisciplinary management, and the need for accessible therapies in resource-limited settings. Further research is needed to optimize diagnostic criteria and treatment protocols for abortion-associated aHUS.

Keywords: thrombotic microangiopathy, hemolytic uremic syndrome, pregnancy, therapeutic plasma exchange, eculizumab

Introduction

Thrombotic microangiopathy (TMA) is characterized by organ failure, microangiopathic hemolytic anemia, and thrombocytopenia. It is particularly relevant to the brain (unconsciousness, seizures), kidneys (acute renal injury), and heart (elevated blood troponin levels, ischemia, sudden death).[1] Concurrent consumptive thrombocytopenia and microangiopathic hemolytic anemia are defining characteristics of the biological symptoms of TMA.[2]

TMA may arise during pregnancy, including pre-eclampsia, thrombotic thrombocytopenic purpura (TTP), complement-mediated hemolytic uremic syndrome (CM-HUS), and antiphospholipid syndrome (APS). Hemolytic uremic syndrome (HUS) has a poor prognosis, with 50% of patients progressing to end-stage renal failure within three to five years.[3] Among women aged 18 to 45, 16% of all cases of atypical HUS (aHUS) are connected with pregnancy.[4] The pregnant aHUS is generally recognized as a subtype of CM-HUS, with an estimated frequency of 1 in 25,000.[5] Past practice has included that both complement-mediated aHUS and pregnancy-associated HUS present with similar severe clinical symptoms: between 41% and 71% of patients need dialysis; without targeted treatment, renal outcomes are catastrophic (53% of individuals reach end-stage renal disease). Early identification is essential for focused therapies to

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enhance outcomes for both mother and fetus. Women of childbearing age represent over 20% of all instances of CM-HUS, with pregnancy frequently serving as the initial or secondary identification of the illnesses.[6] aHUS after abortion has been reported in the past, but it is very rare.[7]

This report presents a clinical case of aHUS secondary to post-abortion infection in early gestation. This study seeks to offer clinical insights for the early detection and management of aHUS in future cases through literature review and case analysis.

Case Presentation

A 41-year-old female presented with abdominal pain and nausea. Five years ago, she was diagnosed with uterine fibroids and adenomyosis, and twenty month ago caesarean section was taken. Five days before admission, she underwent an uncomplicated surgical abortion with normal blood laboratory test results except a preoperative hemoglobin of 84 g/L and platelet count of 336 × 10^9/L. The patient underwent negative pressure suction induced abortion at 6 weeks of gestation because she did not continue to pregnant, and the operation was successful. Two days post-procedure, she developed abdominal pain accompanied by nausea and vomiting, which persisted despite self-administered ibuprofen.

Upon admission, the patient's vital signs were stable, with a blood pressure recording of 119/69 mmHg. Physical examination disclosed tenderness and rebound pain in the lower abdominal region. The uterus was in an anteverted position, enlarged to the approximate size of a 4-month pregnancy, and exhibited tenderness upon palpation. No cervical motion tenderness or adnexal abnormalities were identified. Vaginal examination revealed blood-tinged discharge, with the cervix appearing smooth.

Laboratory findings were significant for leukocytosis (37.53 × 10^9/L) with a neutrophil count of 34.59 × 10^9/L (92.20%), anemia (hemoglobin 76 g/L), thrombocyte decrease (platelets 153 × 10^9/L), hypoalbuminemia (28.7 g/L) and acute kidney injury (creatinine 816 μmol/L). Ultrasound findings were consistent with adenomyosis, uterine fibroids, intrauterine fluid collection, and heterogeneous endometrial echo.

Initial management included intravenous antibiotics (ornidazole 0.5 g every 12 hours and cefuroxime 1.5 g every 8 hours). However, the patient's clinical condition showed no significant improvement, with persistent abdominal pain and intermittent fever, peaking at 37.5°C. Additionally, a progressive decline in urine output was observed, indicating worsening renal function. One day after admission, antibiotic therapy was escalated to piperacillin-tazobactam 4.5 g every 12 hours and doxycycline 100 mg orally every 12 hours. Repeated laboratory tests showed elevated white blood cell count (28.74 × 10^9/L), anemia (hemoglobin 65 g/L), and elevated inflammatory markers (procalcitonin 31.708 ng/mL, C-reactive protein 269.35 mg/L). Renal function continued to deteriorate (creatinine 816 μmol/L, urea 25.3 mmol/L).

Following a multidisciplinary consultation, the patient was transferred to a tertiary care center for further management two days after admission later. Upon transfer, she was febrile (38.0°C) with a blood pressure of 153/67 mmHg. Repeated laboratory tests showed further elevation of inflammatory markers (procalcitonin 47.83 ng/mL, IL-6460 pg/mL) and worsening renal function (creatinine 1083.7 μmol/L, urea 39.08 mmol/L). Coagulation studies revealed prolonged prothrombin time (18.2 seconds), hypofibrinogenemia (1.48 g/L), and elevated D-dimer (46 μg/mL) and fibrinogen degradation products (150 μg/mL). Fragmented red blood cells were observed in the peripheral blood smear. The results of rheumatic immunological examination (including ANA spectrum, antiphospholipid antibody spectrum, lupus anticoagulant, complement, etc.) were negative.

A diagnosis of thrombotic microangiopathy was considered. Given the absence of hypertension, significant thrombocytopenia, or marked elevation of liver enzymes, Preeclampsia/HELLP syndrome was less likely. The patient was conscious with stable vital signs, no significant bleeding tendency, and no jaundice, making thrombotic thrombocytopenic purpura (TTP) less likely, though ADAMTS13 activity testing was still required to exclude TTP definitively. The absence of diarrhea, bloody stools, and negative stool cultures for Shiga toxin-producing bacteria made Shiga toxin-associated HUS unlikely. Complement levels were within normal limits (C3 1.03 g/L, C4 0.35 g/L), but aHUS could not be excluded.

The patient was managed with meropenem and linezolid for infection, along with packed red blood cell and plasma transfusions. The therapeutic plasma exchange was initiated. The result of Thrombospondin type 1 motif, member 13

(ADAMTS13) activity testing was 101%. Eculizumab was recommended for potential aHUS but was declined by the family due to cost concerns. Over the following 15 days, the patient's infection markers normalized, and urine output gradually improved to approximately 1500 mL per day. She remained on dialysis twice weekly, with serum creatinine levels stabilizing between 400 and 500 μmol/L. Now her renal function has basically recovered, and she does not require dialysis.

Discussion

The exact processes behind all TMA symptoms remain enigmatic; nevertheless, experts suggest that a complex interplay of hormonal, immunological, coagulation, and hemodynamic factors contributes to this heightened susceptibility.[8] The presence of other diseases or risk factors increases the likelihood of dysregulated endothelial activation, resulting in the development of TMA.[9]

Patients with aHUS possess either hereditary or acquired autoantibodies that regulate the alternative complement pathway.[10] This underscores the importance of aberrant complement activation, shown by the uncontrolled activation of C3 convertase followed by the subsequent stimulation of C5 convertase. The release of C5a anaphylatoxin and, crucially, the C5b-9 terminal complement complex culminates in endothelial injury and the initiation/progression of thrombosis.[11] Infections, autoimmune exacerbations, and pregnancy are identified as potential environmental triggers for the clinical manifestations of CM-HUS in accordance with the hypothesized "multiple-hit" hypothesis.[12] In this case, we believe that the miscarriage and subsequent infection were the causes that led to the occurrence of aHUS. There is less knowledge regarding the role of complement in infection-associated HUS. Approximately 25% of all cases of HUS may result from infections.[13] The range of pathogens encompasses bacteria such as Enterobacteriaceae, *Staphylococcus aureus*, and *Streptococcus pneumoniae*, as well as viruses including influenza.

Criteria for diagnosing pregnancy-associated TMA include a platelet count below 100 × 10^9/L, hemoglobin level under 10 g/dL, serum lactate dehydrogenase over 1.5 times the upper limit of normal, undetectable serum haptoglobin, a negative direct antiglobulin test, and the appearance of schistocytes on a blood smear.[1] The characteristic trio of acute renal failure, thrombocytopenia, and thrombotic microangiopathic hemolytic anemia is apparent in patients with aHUS. aHUS can be challenging to identify due to its parallels with various illnesses, including Preeclampsia/HELLP syndrome, acute fatty liver of pregnancy, and TTP. Establishing a differential diagnosis between these disorders may be exceedingly difficult, due to their indistinct boundaries and potential coexistence.

Preeclampsia is the predominant cause among pregnancy-related TMAs. This disorder is defined by the onset of new hypertension after 20 weeks of gestation, accompanied with proteinuria and/or acute organ failure.[14] While acute kidney injury (AKI) may occur in both TTP and preeclampsia/HELLP syndrome, it is generally less severe than in CM-HUS, where substantial renal impairment is more frequently noted. Serum creatinine and lactate dehydrogenase (LDH) concentrations can differentiate Preeclampsia/HELLP syndrome from aHUS. Deranged liver tests which are more likely in Preeclampsia/HELLP syndrome than aHUS, which often presents with strikingly high creatinine levels. The ideal diagnostic thresholds for pregnancy-associated aHUS are serum creatinine levels of ≥1.9 mg/dL, LDH levels of ≥1832 U/L, or a combination of serum creatinine ≥1.9 mg/dL and LDH ≥600 U/L.[15] Upon transfer, the patient's LDH level was 1875 U/L, and creatinine level was 1083.7 μmol/L, indicating significant renal impairment. Furthermore, the patient's onset transpired during early gestation, and her blood pressure was consistently within the normal limits previous to and at the onset of the disease, rendering preeclampsia/HELLP syndrome improbable.

There exists considerable clinical overlap among TTP, CM-HUS, and preeclampsia/HELLP syndrome, resulting in diagnostic difficulties. Thrombocytopenia is quite prevalent in pregnancy, impacting 5% to 10% of gestations.[16] The majority of cases of thrombocytopenia were mild, with 15% showing normal platelet counts, in a large multinational European study included 87 cases of pregnancy-associated HUS.[4] Deficiency of a disintegrin and metalloproteinase with a ADAMTS-13 arises from autoantibodies targeting ADAMTS-13 in immune-mediated TTP or from pathogenic mutations in the ADAMTS-13 gene in congenital TTP. ADAMTS-13 is present in trophoblasts and fetal vascular endothelial cells within the chorionic core during pregnancy, exhibiting peak levels in early gestation that diminish as pregnancy advances.[5] Two recently developed clinical risk assessment models for TTP, the PLASMIC score and the French score, can determine the pre-test likelihood of TTP as well as by combining clinical and laboratory data, may aid

in the prediction of ADAMTS-13 insufficiency.[17] In this case, the patient's platelet count was 336 before the miscarriage. Despite a decline following the commencement of the disease, it persisted within the range of 150 to 160, which exceeds the lower threshold of the normal range and does not satisfy the diagnostic criteria for thrombocytopenia. Furthermore, there was no notable reduction in ADAMTS-13 activity, therefore excluding the likelihood of TTP.

The diagnosis of aHUS can be confirmed by exclusion once the previously mentioned diagnoses are eliminated, as there is currently no definitive positive diagnostic test available. Just like the genetic tests and other serological tests, the renal biopsy would have provided objective evidence and much stronger scientific weight to the diagnosis. However, we were unable to conduct the examination as we could not obtain the patient's consent due to her financial limitation. In addition, the diagnosis and treatment of aHUS do not need complement testing findings, as normal results do not rule out the diagnosis of complement-mediated aHUS. The detection of complement variants can retrospectively confirm the diagnosis of complement-mediated hemolytic uremic syndrome; however, normal test findings, as seen in this case, do not exclude the possibility of CM-HUS. In a comprehensive investigation, approximately 50% of CM-HUS patients demonstrated blood concentrations of C3, C5a, and sC5b-9 within the normative range during the acute phase.[18] The quantification of complement regulatory protein levels lacks specificity and sensitivity in distinguishing CM-HUS from other forms of pregnancy-associated TMA.[19] In the acute phase of HUS, sC5b-9 levels are often elevated, irrespective of any familial predisposition.[20] Patients suspected of having pregnancy-associated aHUS/CM-TMA may benefit from a screening test that indicates enhanced complement activity, such as urinary or plasma sC5b-9. Elevated sC5b-9 in the urine or plasma does not prove aHUS/CM-TMA, but it does lend credence to the idea that complement activation is the root cause of the TMA condition.[21] Nonetheless, elevated soluble C5b-9 levels were observed in only 21% of CM-HUS patients, as indicated by a recent study.[22]

Renal injury is defined by the presence of vascular microthrombi, subendothelial edema leading to vascular lumen obstruction, fibrinoid necrosis, and glomerular basement membrane thickening exhibiting a double-contour appearance.[23] Renal failure may manifest in all pregnancy-related TMA forms; however, fast progressive acute kidney injury is more suggestive of hemolytic uremic syndrome or severe obstetric consequences. Oliguria may precede increased creatinine levels and can act as an early sign of the severity of renal damage. Anuria may indicate renal cortical necrosis, a possible consequence of hemolytic uremic syndrome and severe prenatal circumstances.

In this case, given the insufficient diagnostic evidence for aHUS and the patient's poor financial capacity, we did not choose to use complement inhibitors. Therapeutic plasma exchange seemed to be the optimal treatment option under the circumstances at that situation. As a result, the therapeutic plasma exchange (TPE) was commenced promptly, and its efficacy was substantial. Traditionally, pregnancy-associated hemolytic uremic syndrome has been managed with therapeutic plasma exchange and/or plasma infusion, albeit plasma therapy is effective in only fifty percent of patients.[4] TPE was previously advocated as a primary treatment for HUS linked to severe renal impairment or neurological damage, with or without eculizumab. Early therapeutic plasma exchange has been correlated with markedly improved renal recovery outcomes across various causes of aHUS.[24] Given the uncertainty around the diagnosis and terminology, the severity of thrombocytopenia, microangiopathic hemolytic anemia, and neurological complications should determine the decision to initiate therapeutic plasma exchange. The extensive application of therapeutic plasma exchange therapy contributes to enhanced survival rates after this condition. Rather than awaiting the definitive diagnosis of HUS through complement gene mutation analysis and ADAMTS13 activity assessment, which may be time-consuming and costly, it is advisable to initiate plasmapheresis promptly for optimal outcomes. Patients suspected of aHUS must initiate TPE treatment within 24 hours of diagnosis, following the acquisition of all essential laboratory findings, including ADAMTS13, but without awaiting these results. Additionally, a kidney biopsy contributes to confirming histological evidence of TMA.

It is advised to begin eculizumab early in the course of postpartum CM-HUS in order to improve renal outcomes, in addition to providing supportive care.[25] Eculizumab is a recombinant humanized monoclonal antibody that obstructs the cleavage of C5, hence inhibiting the development of C5 convertase and the subsequent cleavage into C5a and C5b, which prevents the commencement of the membrane assault complex.[26] Eculizumab markedly enhanced platelet count and renal function in the patient cohort and correlated with considerable renal recovery and improved clinical outcomes in individuals with atypical hemolytic uremic syndrome.[25] In a retrospective study of 22 women with aHUS, 10 achieved

hematological and renal remission following treatment with a C5 inhibitor. Furthermore, none of the patients developed end-stage renal disease by the end of the 2-year follow-up period.[27] Through the administration of eculizumab, a separate trial revealed a complete remission rate of 87.5%, with a median renal recovery period of 31 days, and dialysis was terminated in all instances after 21 days.[28] In this case, we initially intended to administer eculizumab early, but due to delays in obtaining the medication and the patient's limited financial capacity, we ultimately decided against attempting its use.

Innovation

This article first reports early miscarriage (as opposed to postpartum or late-term pregnancy) as a triggering factor for aHUS, thereby expanding the clinical spectrum of pregnancy-associated TMA. It also highlights the potential role of infection (eg, postoperative infection) in the pathogenesis of aHUS, providing new evidence supporting the "multiple-hit" hypothesis. Furthermore, the study realistically reflects the impact of economic factors on treatment options (eg, the inaccessibility of eculizumab), offering insights for clinical decision-making in resource-limited settings. Additionally, it validates the efficacy of therapeutic plasma exchange in managing acute kidney injury, demonstrating rapidly partial renal function recovery even in the absence of targeted therapy.

Conclusion

This case underscores the diagnostic difficulties and management intricacies of thrombotic microangiopathy following abortion, especially in differentiating among various etiologies and addressing therapy alternatives in resource-constrained environments. This study enhances the understanding of abortion-associated aHUS through a rare case report and comprehensive literature review. Its novelty lies in the expansion of clinical scenarios, refined considerations for diagnostic workflows, and practical insights for resource-limited settings, thereby providing new directions for research on aHUS. Successful treatment outcomes rely on various aspects, including prompt diagnosis to commence TPE and the administration of eculizumab at the earliest opportunity.

Data Sharing Statement

The datasets analysed during the current study are not publicly available due to the patience's wish but are available from the corresponding author on reasonable request.

Ethics Statements

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. This research had obtained the approval of the Qingdao Municipal Hospital and Ethics Committee of Qingdao Municipal Hospital to publish the case details and it was performed in accordance with the regulations on Scientific Research Management of Qingdao Municipal Hospital and the declaration of Helsinki.

Consent to Participate

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

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Disclosure

The authors report no conflicts of interest in this work.

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