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Pregnancy complicated with acute interstitial nephritis and myocarditis in postpartum period; a case report

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ABSTRACT

The co-occurrence of postpartum acute interstitial nephritis (AIN) and myocarditis is a rare but serious complication of pregnancy following HELLP (hemolysis, elevated liver enzymes, and low-platelet) syndrome. We present the case of a 42-year-old woman who developed HELLP syndrome at the 31st week of gestational age. Termination of pregnancy and eight sessions of plasmapheresis were conducted. The patient exhibited acute kidney injury (AKI), dyspnea, bilateral pleural effusion, and 30% left ventricular (LV) ejection fraction. The diagnosis of AIN was established by kidney biopsy, while the post-partum cardiomyopathy (PPCM) was confirmed by cardiac magnetic resonance imaging (CMRI). The AKI and PPCM, in addition to hematologic abnormalities, were successfully resolved with intravenous methylprednisolone, followed by oral prednisolone.

Case Report

Implication for health policy/practice/research/medical education:
This case report presents a rare and complex clinical presentation of a postpartum patient who developed both acute interstitial nephritis and myocarditis.

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Introduction

Acute kidney injury (AKI) during pregnancy or in the postpartum period, postpartum acute kidney injury (PR-AKI), is a serious condition with an increasing incidence and is concomitant with significant maternal and fetal morbidity and mortality rates (30% to 60%) (1).

Acute kidney injury during pregnancy can be attributed to acute tubular necrosis, renal cortical necrosis, thrombotic microangiopathy, preeclampsia spectrum disorders, glomerulonephritis, acute interstitial nephritis (AIN) and acute fatty liver of pregnancy (1,2).

Postpartum acute kidney injury may lead to long-term renal, cardiovascular, and neurocognitive consequences that persist beyond the postpartum period. Hypertensive conditions during pregnancy, particularly preeclampsia and hemolysis-elevated liver enzymes-low platelet syndrome (HELLP syndrome) are associated with an increased risk of developing cardiovascular disease in later life (1).

Post-partum cardiomyopathy (PPCM) is a rare form of heart failure that may present with mild or severe symptoms. PPCM typically occurs during the final month of pregnancy or up to five months after. Although the most common etiology for this condition is idiopathic, recent evidence implies that it is a type of myocarditis that can be caused by an infectious, autoimmune, or idiopathic process. A number of case reports have documented the coexistence of peripartum cardiomyopathy/myocarditis with pre-eclampsia complicated by PR-AKI (3-6).

Acute myocarditis is a rare occurrence during pregnancy,

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and it typically manifests in the third trimester (5). It is associated with peripartum cardiomyopathy. The clinical presentation varies from asymptomatic, mild, nonspecific symptoms to cardiogenic shock and/or life-threatening arrhythmias (7).

This case report presents a patient with AKI and cardiac involvement following delivery.

Case Presentation

A 42-year-old woman, gravida 8, parity 1, abort 7, at 31st weeks' gestation, by in vitro fertilization of a twin fetus, was admitted to the nephrology unit on the 15th day of her postpartum as a second hospitalization, following the premature termination of her pregnancy at 31st weeks' gestation due to HELLP syndrome. At the time of her first admission, her hemoglobin level was 6.6 g/dL and the platelet count was 102 000/µL. Her serum creatinine level was 4.3 mg/dL. Additionally, mildly elevated liver enzymes, lactate dehydrogenase (LDH) of 859 U/L (normal range 125-220 U/L) and schistocytes of 3% in peripheral blood smear were observed. She underwent plasmapheresis and hemodialysis every other day for eight and seven sessions, respectively, following the pregnancy termination. Subsequently, she was referred to our nephrology ward for further evaluation.

Upon admission to our hospital, the patient had symptoms of dyspnea and mild chest pain. She denied the use of over-the-counter drugs, tobacco, or alcohol as well as exposures to communicable disease, she also had a history of infertility for 20 years.

The serum creatinine level was 3.6 mg/dL, accompanied by microscopic hematuria on urine analysis and 24-hour proteinuria of 1468 mg. Mild hypokalemia (serum potassium: 3 mEq/L), leukocytosis (white blood cells: 30×10^3/µL), anemia (hemoglobin; 8.5 g/dL), and thrombocytopenia (platelets: 105×10^3/µL) were observed. Liver enzyme levels returned to normal range, however, LDH levels were elevated at 596 U/L. Immunologic tests and serum protein electrophoresis were unremarkable, while urine protein electrophoresis demonstrated a proteinuria pattern consistent with glomerular origin (Table 1).

The electrocardiogram (ECG) revealed low voltage complex waves, yet trans-thoracic echocardiography (TTE) demonstrated mild cardiomegaly and evidence of pericardial effusion. The left ventricular (LV) ejection fraction was 30%, indicating moderate LV systolic dysfunction, and a significant bilateral pleural effusion was observed in the chest computed tomography scan. Subsequent cardiac magnetic resonance imaging (CMRI) revealed focal myocardial inflammation in the basal and mid-lateral walls, as well as global pericardial inflammation, in favor of acute myopericarditis and PPCM (Figures 1 and 2).

Table 1. Immunologic test

Name	Result	Normal range
ANA-ELISA	0.1	Neg < 1
C-ANCA-ELISA	Negative	
P-ANCA-ELISA	Negative	
Anti-dsDNA IgG-ELISA	0.2	Neg < 0.9
Anti-cardiolipin IgG	1.2	Neg <10
B2 glycoprotein Ab (IgG)	1.9	Normal range <12
B2 glycoprotein Ab (IgM)	3	Neg <20
C3	1.17	0.89-1/87
C4	0.18	0.165-0.380
CH50	105	50-150
SPEP	Normal	
UPEP	Normal	

ANA: Antinuclear antibody; ELISA, Enzyme-linked immunosorbent assay; C-ANCA: Anti-neutrophil cytoplasmic antibody; Anti-dsDNA: Anti-double stranded DNA; SPEP: Serum protein electrophoresis; UPEP: Urine protein electrophoresis.

Moreover, a kidney biopsy was performed, which results were compatible with acute tubulointerstitial nephritis, the presence of inflammatory cells in the interstitium,

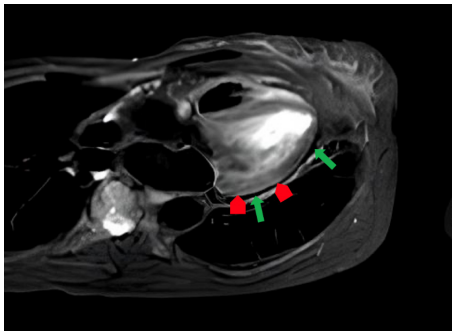

Figure 1. Cardiac magnetic resonance imaging (C MRI), STIR, Four-chamber view; myocardial inflammation in the basal to mid lateral wall is evident (Red arrow) global pericardial effusion (Green arrow).

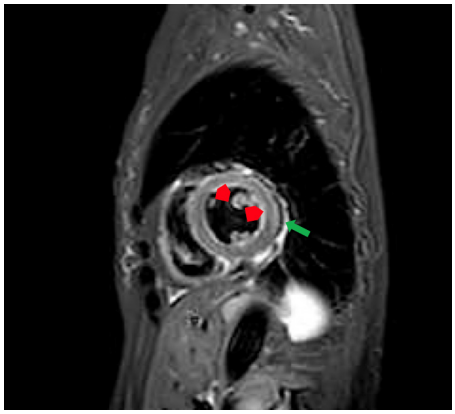

Figure 2. Cardiac magnetic resonance imaging (CMRI), STIR, short axis view, myocardial inflammation in the lateral wall (Red arrow), pericardial effusion (Green arrow).

predominantly lymphocytes, and evidence of tubulitis with atrophic changes of 5%-10% (Figures 3 and 4), The immunofluorescence study reveals 1+ mesangial deposition for both IgA and IgM, with C3 exhibiting trace mesangial deposition.

In favor of postpartum acute tubulointerstitial nephritis and myocarditis diagnosis, methylprednisolone was initiated at a dosage of 500 mg over three consecutive days, followed by a prednisone dose of 1 mg/kg orally, with gradual tapering and discontinuation over a two-month period. At the 4-month follow-up, the clinical course had improved. The patient no longer experienced chest discomfort when exerting herself, her serum creatinine level had declined to 0.82 mg/dL, and the proteinuria and hematuria had resolved. Her hematologic and serum electrolyte abnormalities had returned to normal, with a white blood cell count of 9.7 $\times 10^3$/µL, a platelet count of 247 $\times 10^3$/µL, a hemoglobin level of 11.9 g/dL, across with a potassium level of 4 mEq/L.

The myopericardial involvement was resolved, as evidenced by echocardiography, which demonstrated a left ventricular ejection fraction of 50% without pericardial effusion and normal diastolic function. The subsequent pharmacological regimen comprised sacubitril/valsartan 49 mg/51 mg twice daily, empagliflozin 10 mg daily, carvedilol 6.25 twice daily and eplerenone 25 mg daily.

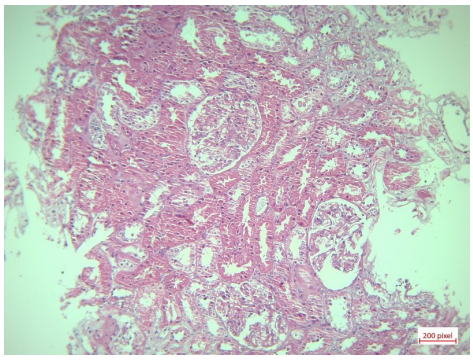

Figure 3. Kidney biopsy; light microscopy, hematoxylin and eosin staining (H&E), All glomeruli are of a typical size with a normal mesangium. No spikes, holes, or corrugations are observed. There is no evidence of endocapillary proliferation or fibrinoid necrosis.

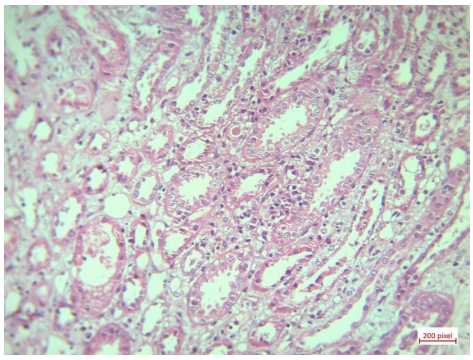

Figure 4. Kidney biopsy; light microscopy with H&E staining. The tubules display focal proteinaceous casts within their lumen, with approximately 5-10% exhibiting atrophic changes. An inflammatory cell infiltration is evident in the interstitium, predominantly comprising lymphocytes with focal tubulitis.

Discussion

The co-occurrence of postpartum AIN and myocarditis is a rare but serious pregnancy complication following HELLP syndrome. A limited number of case reports have been published that provide precise identification and management of this catastrophic syndrome (PPCM and multi-organs failure, including kidneys) (8). The following criteria are used to diagnose PPCM; 1) diagnosis of cardiac failure in the latest 30 days of pregnancy or occurring 5 months postpartum, 2) no substitute etiology, 3) lack of past detectable heart disease, 4) LV systolic dysfunction with an ejection fraction of <45% on echocardiogram (8, 9). Our patient had the ejection fraction of 30% with moderate LV systolic dysfunction on transthoracic echocardiography and MRI displayed evidence of focal myocardial inflammation. In addition, other differential diagnoses of kidney and heart diseases should be considered during the peripartum period, including complement pathways abnormalities (10). The term "thrombotic microangiopathies" encompasses two distinct conditions; thrombotic thrombocytopenic purpura (TTP) and atypical hemolytic uremic syndrome (a-HUS) (11). Despite the presence of clinical and laboratory markers consistent with HELLP syndrome during the initial hospitalization and persistent AKI, kidney biopsy did not reveal the presence of thrombotic microangiopathy (TMA). Indeed, the incidence of such occurrences during the peripartum period is relatively uncommon (12). A more common etiology is systemic lupus erythematosus (SLE), which frequently affects women of reproductive age. The risk of developing lupus nephritis and cardiomyopathy is a significant concern (13). Our patient was evaluated for SLE, but did not meet the criteria for lupus or antiphospholipid syndrome. Furthermore, the renal biopsy did not reveal lupus nephritis.

Conclusion

Myocarditis during pregnancy is an uncommon occurrence, particularly when accompanied by AIN. The clinical manifestations of the disease may range from the absence of symptoms to the presence of mild, non-specific signs, or even fatal arrhythmias. It is important for physicians to be aware of this potential complication, in addition to other organ dysfunctions such as interstitial nephritis.

Authors' contribution

Conceptualization: Fereshteh Saddadi, Bahareh Marghoob.
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Investigation: Mohammad Hassan Fallahkohan.
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Project administration: Bahareh Marghoob.
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Supervision: Fereshteh Saddadi.
Validation: Hounaz Akbari, Neda Najafi.
Visualization: Mohammad Hassan Fallahkohan.
Writing–original draft: Bahareh Marghoob, Hounaz Akbari.
Writing–review & editing: Fereshteh Saddadi, Mohammad Hassan Fallahkohan.

Conflicts of interest

All authors declare that they have no conflicts of interest.

Ethical issues

This case report is in concordance with the principles outlined in the Declaration of Helsinki. Ethical issues including plagiarism, data fabrication, double publication have been completely observed by the authors. The patient has given us a written informed consent for publication as a case report.

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References

1. Szczepanski J, Griffin A, Novotny S, Wallace K. Acute Kidney Injury in Pregnancies Complicated With Preeclampsia or HELLP Syndrome. Front Med (Lausanne). 2020;7:22. doi: 10.3389/fmed.2020.00022.
2. Liu Y, Ma X, Zheng J, Liu X, Yan T. Pregnancy outcomes in patients with acute kidney injury during pregnancy: a systematic review and meta-analysis. BMC Pregnancy Childbirth. 2017;17:235. doi: 10.1186/s12884-017-1402-9
3. Akbar A, Ata Ur-Rehman HM, Tameez Ud Din A, Malik A. Peripartum Cardiomyopathy during puerperium co-existing with preeclampsia and complicated by acute renal failure: a case report. Cureus. 2019;11(12):e6319. doi: 10.7759/cureus.6319.
4. Brown CS, Bertolet BD. Peripartum cardiomyopathy: a comprehensive review. Am J Obstet Gynecol. 1998, 178:409-414. 10.1016/s0002-9378(98)80034-3
5. Flores-Sanga HS, Salas-Cuadros D, Saucedo-Chinchay JL, Arriola-Montenegro J, Torres-Valencia J, Muñoz-Moreno JM. Clinically Suspected Acute Myocarditis in the First Trimester of Twin Gestation: A Case Report. Cureus. 2023;15(12):e50851. doi: 10.7759/cureus.50851
6. GOLDBERG WM. Idiopathic myocarditis of pregnancy. Can Med Assoc J. 1963;88:1247-50.
7. Elkayam U. Cardiac Problems in Pregnancy. Myocarditis and Pregnancy. 4th ed. Wiley-Blackwell; 2019. doi: 10.1002/9781119409861.
8. Quevedo S, Bekele C, Thompson PD, Philkhana M, Virani S, Consuegra A, et al. Peripartum cardiomyopathy and HELLP syndrome in a previously healthy multiparous woman: A case report. SAGE Open Med Case Rep. 2020;8:2050313X20979288. doi: 10.1177/2050313X20979288.
9. Kota LN, Garikapati K, Kodey PD, KB G. Study on HELLP syndrome-maternal and perinatal outcome. Int J Reprod Contracept Obstet Gynecol 2017;6:715. doi:10.18203/2320-1770.ijrcog20170411.
10. Lynch AM, Salmon JE. Dysregulated complement activation as a common pathway of injury in preeclampsia and other pregnancy complications. Placenta. 2010;31:561-7. doi: 10.1016/j.placenta.2010.03.010.
11. Cosmai EM, Puzis L, Tsai HM, Lian EC. Thrombocytopenic purpura and cardiomyopathy in pregnancy reversed by combined plasma exchange and infusion. Eur J Haematol. 2002;68:239-242. doi: 10.1034/j.1600-0609.2002.01605.x.
12. Fujimura Y, Matsumoto M, Kokame K, Isonishi A, Soejima K, Akiyama N, et al. Pregnancy induced thrombocytopenia and TTP, and the risk of fetal death, in Upshaw-Schulman syndrome: a series of 15 pregnancies in 9 genotyped patients. Br J Haematol. 2009;144:742–754. doi: 10.1111/j.1365-2141.2008.07515.x.
13. Clowse ME, Magder LS, Witter F, Petri M. The impact of increased lupus activity on obstetric outcomes. Arthritis Rheum. 2005;52:514-521. doi: 10.1002/art.20864.