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Renal histopathology of prolonged acute kidney injury in HELLP syndrome: a case series and literature review

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Abstract

Purpose Acute kidney injury (AKI) is a severe complication of hemolysis, elevated liver enzymes, and low platelet count (HELLP) syndrome. However, renal pathological investigation of AKI in this syndrome has rarely been reported. We aimed to evaluate the renal pathological changes of persistent AKI and its relationship with renal outcomes in HELLP syndrome.

Methods Women with HELLP syndrome who had a renal biopsy because of persistent AKI were investigated. The cases describing renal pathology of AKI in HELLP syndrome reported in PubMed were also reviewed.

Results Among the 41 patients diagnosed with AKI complicated by HELLP syndrome, 6 patients had renal biopsy. Four of these patients had anuria and required renal replacement therapy. Renal histopathology showed thrombotic microangiopathy (TMA) that coexisted with acute tubular necrosis (ATN) (3), acute renal cortical necrosis (ARCN) (1), and glomerular disease (2). Two patients who had ARCN and ATN with TMA lesions developed chronic renal dysfunction. Ten cases reported in the literature showed ATN (4), TMA (1), TMA with ATN (1), ARCN (2) and mesangial proliferative glomerulonephritis (1). All of them required temporary renal replacement therapy. Two patients developed chronic renal dysfunction including one patient with ARCN.

Conclusions ATN was the most common finding for persistent AKI in HELLP syndrome. Patients with ARCN or TMA with ATN may have the potential to develop chronic renal dysfunction. Renal biopsy should be performed in patients with prolonged AKI to determine the renal prognosis and guide the appropriate treatment.

Keywords HELLP syndrome · Acute kidney injury · Renal pathology · Outcome · Pregnancy

Introduction

Hemolysis, elevated liver enzymes, and low platelet count (HELLP) syndrome is a serious complication of pregnancy, which often causes a high incidence of maternal and perinatal morbidity and mortality [1–3]. Acute kidney injury (AKI) is a severe complication of this syndrome, with an incidence of 7.7–60% [1–6]. Although AKI associated with HELLP syndrome usually has a favorable renal outcome, there are some patients who develop chronic kidney disease [2, 4, 7]. However, the pathogenesis involved in AKI in HELLP syndrome remains unclear. Because of the increased risk of bleeding related to thrombocytopenia and rapid recovery of renal function in most patients, there is a lack of information on renal histology. To date, only about ten cases on the renal histology of AKI in this syndrome have been reported [8–12].

Herein, we studied the renal pathological features for prolonged AKI complicated by HELLP syndrome and evaluated its association with the outcome of renal failure. We believe that our patients' renal biopsy findings can explain the pathogenesis and the clinicopathological relationship of persistent AKI in HELLP syndrome.

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Materials and methods

The diagnosis of HELLP syndrome was made based on the presence of the following criteria [2, 13]: (1) microangiopathic hemolytic anemia with an abnormal blood smear and elevated lactate dehydrogenase levels > 600 IU/L or bilirubin levels > 20.52 μmol/L; (2) liver dysfunction indicated by aspartate transaminase levels > 70 IU/L; and (3) a platelet count < 100×10^9/L. The following Kidney Disease Outcomes Quality Initiative (KDOQI) criteria were used to diagnose AKI [14]: (1) an increase in serum creatinine of 0.3 mg/dL (26.5 μmol/L) within 48 h; (2) an increase in serum creatinine to 1.5-times baseline within the previous 7 days; or (3) urine volume 0.5 mL/kg/h for 6 h. Among all patients with HELLP syndrome diagnosed at the Peking Union Medical College (PUMC) Hospital between January 2008 and March 2017, seven patients underwent a renal biopsy for the indication of renal lesions in HELLP syndrome. Six patients who had AKI that persisted for more than 2 weeks, which was arbitrarily called prolonged AKI, were included in this study. One patient who had a renal biopsy because of proteinuria that developed in the first term of gestation and persisted postpartum, which was not considered to be prolonged AKI, was excluded. The following maternal and fetal variables for these patients were recorded: demographics, onset time of HELLP syndrome, blood pressure, maternal complications and management, fetal outcome, and relevant laboratory tests. Specific investigations to exclude other causes of AKI, including antinuclear antibody, anti-dsDNA antibody, antiphospholipid antibody, and complement (C)3 and C4 were also recorded. The histopathological diagnoses were made by two pathologists based on light microscopic and immunofluorescence studies. The renal ultrastructure was examined in five of the six patients using electron microscopy. The pathologic findings of thrombotic microangiopathy (TMA) include endothelial swelling and mesangiolysis in active lesions, and double contours of the basement membrane in chronic lesions observed using electron microscopy, with or without the renal arteriole occlusion with endotheliosis, and lumen and vessel wall fibrin observed using light microscopy [15, 16]. All patients were followed up for at least 1 year.

The cases of patients who had renal pathology in HELLP syndrome were searched in PubMed and papers published in English were included. There were six papers about renal histopathology in patients with HELLP syndrome, and five of these studies, which used AKI as an indication of renal pathology, were reviewed.

Results

Characteristics of patients undergoing renal biopsy

There were 85 women diagnosed with HELLP syndrome over 10 years at the PUMC hospital. AKI developed in 41 (48.2%) patients. Renal function in most patients with AKI recovered within 1 week after delivery. Six patients with a mean age of 29.2 ± 6.1 years had a renal biopsy after 2–6 weeks postpartum because of persistent AKI. One patient had a history of hypertension for 5 years and her blood pressure was well controlled before pregnancy. No patient had preexisting chronic kidney disease or diabetes mellitus. All of the patients had preeclampsia before HELLP syndrome developed. The characteristics of these patients are shown in Table 1.

Renal histopathological manifestation of acute kidney injury in HELLP syndrome

In our study, renal histopathology showed acute tubular necrosis (ATN) in three patients, all of whom had coexisting TMA (Fig. 1a, b). Acute renal cortical necrosis (ARCN) was present in one patient (Fig. 1c, d), and glomerular disease was present in two patients (Table 2). In patients with ATN and TMA, two patients (patients 1 and 2) had typical renal arteriole lesions including arterial endotheliosis, occlusion, and vessel wall fibrin that was observed under light microscopy, and patient 6 was diagnosed by glomerular endotheliosis using electron microscopy.

Light microscopy in patient 4 showed severe mesangial proliferation with cellular crescent formation in one-third of glomeruli and patchy interstitial hemorrhage. Patient 5 gradually developed nephrotic syndrome after 1 week of anuria. Membranous proliferative glomerulonephritis (MPGN) presenting with severe endocapillary proliferation with a lobular pattern and duplication of the glomerular basement membrane (GBM) was observed (Fig. 2a, b). Immunofluorescence showed staining of immunoglobulin (Ig) G (+−++), IgA (+), and complement (C)3 (++), which was mainly located along the GBM, with some in the mesangium (Fig. 2c). Electron microscopy showed large, amorphous, electron-dense deposits in the subendothelial and mesangial areas (Fig. 2d). Although lupus nephritis was highly suspected, no serum immunological markers were detected during regular monitoring. Except for two patients with glomerulonephritis, immunofluorescence studies for all immunoglobulins and complement in four other patients were negative. Electron microscopy in 4 of 5 patients revealed endothelial swelling and widening of

Table 1 Characteristics of patients with acute kidney injury who had a renal biopsy

Patient no.	1	2	3	4	5	6
Age	40	26	23	32	26	28
History of hypertension	Yes	No	No	No	No	No
Onset time of HELLP (weeks)	26^{+5}	35	35	34	36	32^{+4}
Bleeding volume (mL)	700	1000	1000	200	2400	2000
Blood pressure (mmHg)	200/100	148/100	160/100	150/90	150/110	160/105
Lowest hemoglobin (g/L)	74	79	72	70	46	61
Lowest platelet (10^9/L)	32	37	41	10	21	39
Proteinuria at biopsy	+	3+	0.83 g/24 h	NA	11.55 g/24 h	2.14 g/24 h
Serum albumin at biopsy (g/L)	42	43	45	33	28	43
Highest SCr (μmol/L)	463	584	731	760	614	341
Anuria duration (days)	No	21	30	22	7	No
Dialysis duration (weeks)	No	4	6	4	2	No
Duration from delivery to biopsy	2 weeks	26 days	6 weeks	2 weeks	6 weeks	2 weeks
SCr during follow-up	Normal after 3 months	195 μmol/L at 1 year	127 μmol/L at 2 years	Normal after 2 years	Normal after 6 months	Normal after 6 months
Fetal outcome	Stillbirth	Viable (twin)	Stillbirth	Viable (twin)	Viable	Viable

HELLP hemolysis, elevated liver enzymes, and low platelet count, *SCr* serum creatinine, *NA* not available because of anuria

the subendothelial space with fluffy material that was similar to the ultrastructure lesions of preeclampsia. Another patient showed glomerular ischemia, which was consistent with ARCN that was diagnosed using light microscopy.

Ten cases were reviewed in the literature (Table 3). All of the patients in these cases had oliguria or anuria. ATN was diagnosed in five patients, one of whom had coexisting TMA. ARCN was found in two patients, and other forms involving AKI were TMA alone and mesangial proliferative glomerulonephritis.

Outcome of acute kidney injury with different renal pathologies in HELLP syndrome

In our study, four of six patients had anuria with a duration of 7–30 days and they received hemodialysis for 2–6 weeks. Two patients (patients 2 and 5) had plasma exchange and another two patients (patients 4 and 6) were administered plasma transfusion in the early stage of the disease.

During follow-up, two patients in this series developed chronic renal dysfunction. Their renal pathological diagnoses were TMA with ATN and ARCN. The serum creatinine level in patient 2, who had TMA and ATN, was 195 μmol/L after 1 year of follow-up. ARCN was found in patient 3. Her serum creatinine level slowly decreased and remained abnormal after 2 years. Two patients who had proliferative glomerulonephritis were administered immunosuppressive therapy. Patient 4, who had severe mesangial proliferation and cellular crescents, was administered prednisone 1 mg/kg/day, which was gradually tapered with the total course of 1 year. Proteinuria and renal function completely recovered after 1 year. Patient 5 gradually developed nephrotic syndrome after 1 week of anuria. Renal pathology showed MPGN. She was administered prednisone with an initial dose of 1 mg/kg/day and cyclophosphamide with a cumulative dose of 10 g. The patient's renal function was completely recovered after 6 months of treatment. After 1 year of follow-up, her urine protein levels returned to the normal range.

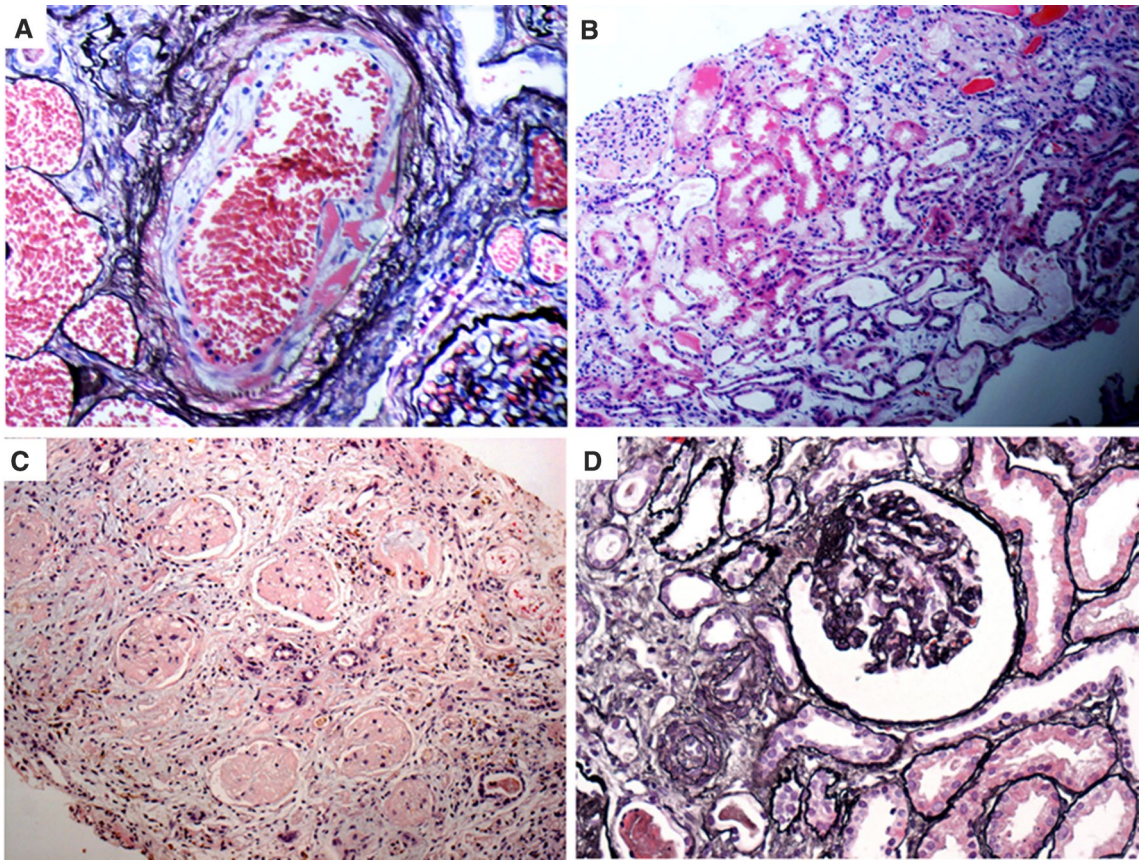

Fig. 1 **a**, **b** Thrombotic microangiopathy with acute tubular necrosis in patient 2. **a** The interlobular arteriole showed marked intimal mucoid swelling and wall thickness (periodic acid-silver methenamine (PASM), × 200). **b** Proximal tubules showed extensive epithelial attenuation, brush border loss, and dilated lumens with prominent interstitial edema (hematoxylin and eosin (HE), × 100). **c**, **d** Acute renal cortical necrosis in patient 3. **c** Most (82.9%; 34/41) glomeruli show necrotic dissolution with a loss of glomerular integrity (HE, × 100). **d** Ischemic collapse with wrinkling of the GBM and an expanded Bowman's capsule cavity in the remaining glomeruli. An adjacent small arteriole shows narrowing of the lumen because of wall thickening (PASM, × 200)

Table 2 Renal histopathological findings of persistent acute kidney injury in HELLP syndrome

Patient no.	Immunofluorescence	Light microscopy	Electron microscopy
1	(−)	TMA and ATN	Changes of TMA
2	(−)	TMA and ATN	NA
3	(−)	Acute renal cortical necrosis	Glomerular ischemia
4	IgM(+), granular staining along GBM and mesangium	Mesangial and endothelial hypercellularity with cellular crescent formation (7/24); interstitial hemorrhage	Endothelial swelling and widened subendothelial space; no electron-dense deposit
5	IgG(+~++), IgA(+) and C_3(++), focal and granular staining along GBM and mesangium	MPGN	Endothelial swelling, endocapillary hypercellularity with large electron-dense deposit in subendothelial and mesangial area
6	(−)	ATN	ATN with endothelial swelling and widened subendothelial space

HELLP hemolysis, elevated liver enzymes, and low platelet count, *TMA* thrombotic microangiopathy, *ATN* acute tubular necrosis, *IgG* immunoglobulin G, *IgM* immunoglobulin M, *IgA* immunoglobulin A, *GBM* glomerular basement membrane, *MPGN* membranous proliferative glomerulonephritis, *NA* not available

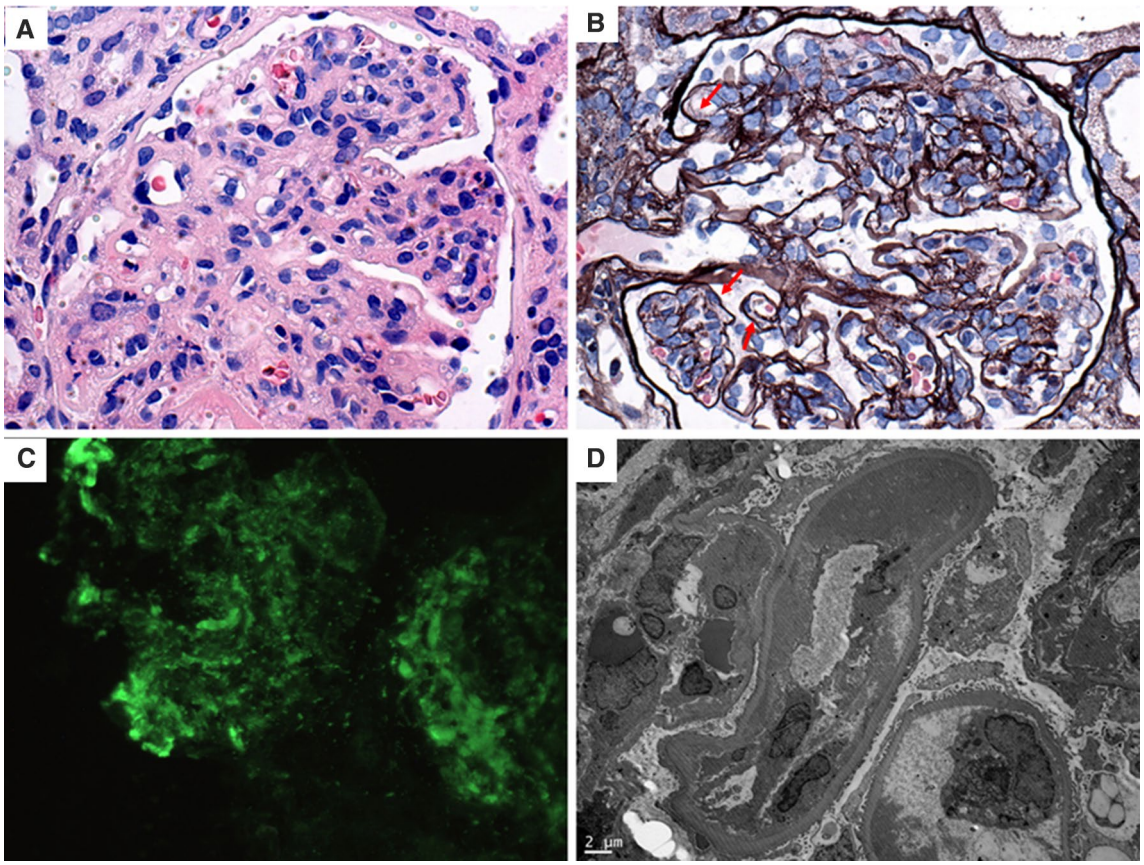

Fig. 2 Membranous proliferative glomerulonephritis in patient 5. Light microscopy showed severe endocapillary hypercellularity with a lobular pattern and duplication of the GBM (red arrows; **a** HE, ×400; **b** PASM, ×400). Immunofluorescence showed IgG (+−++) staining in the mesangial area and along the glomerular basement membrane (**c** ×400). Electron microscopy showed prominent narrowing of the lumen with endocapillary hypercellularity, subendothelial interposition, and large, amorphous, electron-dense deposits in the subendothelium (**d** ×4000)

Table 3 Summary of published cases with renal histopathological descriptions of AKI complicated by HELLP syndrome

First author (year)	Patient no.	Age	Gestation (weeks)	BP (mmHg)	Oligo-anuria	Hemodialysis	Pathological diagnosis	Renal outcome	Fetus outcome
Kahra (1998) [27]	1	32	37	150/100	Oliguria	Yes	TMA	Normal after 1 year	Viable
Sheikh (1998) [12]	6/7	27.6 ± 6.1 (22–37)	34.1 ± 1.8 (32–36)	140–170/90–100	7/oligo-anuria	Yes	4 ATN; 1 ARCN; 1 no diagnosis	CRF in 1 patient	2/7 viable
Fang (2000) [9]	1	42	33	166/110	Oliguria	Yes	MesPGN	Normal	Stillbirth
Abraham (2003) [8]	1	31	26	190/105	Oliguria	Yes	TMA and ATN	107 µmol/L at 4th month	Stillbirth
Gupta (2012) [10]	1	32	29	180/108	Anuria	Yes	ARCN	Hemodialysis dependent	Stillbirth

BP blood pressure, *TMA* thrombotic microangiopathy, *ATN* acute tubular necrosis, *ARCN* acute renal cortical necrosis, *MesPGN* mesangial proliferative glomerulonephritis, *CRF* chronic renal failure

Discussion

Although the incidence of pregnancy-related AKI has significantly declined over the last 3 decades in developing countries, it is still an important cause of maternal and fetal morbidity and mortality [17]. HELLP syndrome develops in 0.1–0.9% of all deliveries and in 10–20% of women with preeclampsia [3, 5, 10, 18, 19]. It is the common cause of AKI in pregnancy, accounting for 36–50% of all cases of pregnancy-related AKI [7, 20]. AKI associated with HELLP syndrome usually has a favorable renal outcome, but some patients develop chronic renal dysfunction [2, 4, 7]. To date, there has been little research on renal pathology of AKI complicated by HELLP syndrome.

ATN is the most common histopathological finding in both the present study and published reports. The only series about AKI renal histology in HELLP syndrome was performed by Sheikh et al. [12]. Among 52 patients with HELLP syndrome, 7 patients were diagnosed with AKI and renal histopathological information was acquired from 5 of these patients [12]. Four (80%) had ATN and one (20%) had ARCN. In the present study, ATN occurred in three (50%) of six patients. The reason for the discrepancy between these two studies may be explained by differences in patient enrollment. Sheikh et al. included all patients with AKI and HELLP syndrome, except for one patient who did not agree to a renal biopsy [12]. However, in our study, patients with prolonged AKI were selected. We excluded those with mild or moderate AKI; these patients rapidly recovered postpartum. Patients with ATN alone exhibited complete recovery of renal function in our series and in the cases we reviewed [2, 21]. The finding of ATN as the dominant lesion in HELLP syndrome can explain the reversibility of acute renal failure in most patients, even in those who experienced anuria and required dialysis.

TMA was the second most common renal pathological finding in AKI patients with HELLP syndrome. Overall, among 5 (31.3%) of 16 patients (including patients in our study and in published cases) who were diagnosed with TMA, 4 patients had TMA in combination with ATN, and only 1 patient had TMA alone. The renal pathology showed intimal mucoid swelling and thickening of the vascular wall in interlobular arteries. The changes in response to TMA, which included endovascular stenosis up to lumen occlusion, may cause renal failure by directly reducing glomerular perfusion and filtration, or lead to downstream tubular ischemia that indirectly causes ATN if the changes persist [8]. This observation suggested that ATN induced by TMA is an important part of pathogenesis in AKI development in patients with HELLP syndrome and it may explain the close association between TMA, ATN, and renal failure. Renal function in four patients with ATN only recovered completely, while two of four patients with ATN and TMA developed chronic renal dysfunction. This result showed that TMA is the common cause of prolonged renal failure, and it indicates an adverse prognosis of AKI in HELLP syndrome, especially when it coexisted with ATN.

ARCN, glomerulonephritis, and nephrosclerosis have also been noted in patients with HELLP syndrome [10, 22]. This was observed in one of our patients and two of ten patients in published studies [10, 12]. ARCN often results in irreversible renal failure, but the patchy variety can present with initial oliguria and even anuria, which can be followed by a variable recovery of renal function [10]. The patient with ARCN in our study developed mild renal insufficiency. After having anuria that lasted for 30 days, the patient achieved a partial recovery, with a serum creatinine level of 127 mol/L after 2 years of follow-up. In Gupta's study, the patients with extensive ARCN remained hemodialysis dependent [10]. The finding showed that ARCN was a devastating pathology for an adverse renal outcome in patients with AKI complicated by HELLP syndrome. ARCN should be suspected when renal failure persists for several weeks after delivery or the patient does not achieve a complete recovery after long-term follow-up. Glomerulonephritis was observed in 3 of 16 patients (18.8%), including mesangial proliferative glomerulonephritis in Fang's study [9] and MPGN and severe mesangial proliferative glomerulonephritis with cellular crescents in our patients. This type of renal pathology requires a renal biopsy to make a pathological diagnosis.

Management of AKI in HELLP syndrome included the following: control of blood pressure, termination of pregnancy, plasma exchange, and renal replacement therapy if necessary. Termination of pregnancy is the main and effective treatment in the presence of maternal complications and/or fetal distress. Most patients with HELLP syndrome start to improve within the first 24–48 h after delivery [23]. Patients who fail to recover after delivery are at major risk of more severe morbidity or mortality. Postpartum plasma exchange is effective for patients with severe HELLP syndrome [24, 25]. Recently, Simetka et al. recommended that plasma exchange should be considered in all patients who show no significant improvement in aspartate transaminase levels, and especially platelet levels, within 24–48 h after delivery [25]. In our study, two women received plasma exchange. Dialysis was performed in four patients in this study. For patients with glomerulonephritis, prednisone with/without immunosuppressants was administered. Renal function recovered and proteinuria completely resolved after 1–2 years. Complete resolution of renal injury in these patients indicates the important role of renal biopsy for diagnosis and predicting the prognosis in these patients.

The pathogenesis of AKI in HELLP syndrome remains unclear. Our study revealed that the most common renal

histology is ATN for those with persistent AKI. Several pathogenic factors may be involved in this process. First, acute anemia is thought to be the underlying mechanism of AKI that results from hemolysis or hemorrhage, which is the main manifestation of HELLP syndrome. A rapid decrease in hemoglobin levels occurred in all patients, with the lowest hemoglobin level of 46 g/L. Second, microvascular dysfunction may increase the kidney's sensitivity to ischemia in patients with HELLP syndrome. Beller et al. reported that 11 of 12 patients with HELLP syndrome and renal pathology showed endotheliosis as the main finding [22]. Although the authors did not provide any information on the renal clinical status in their study, it is speculated that some of the patients had normal renal function, because all patients with HELLP syndrome were enrolled. Electron microscopy-demonstrated glomerular endothelial damage that was similar to preeclampsia lesions was observed in most patients in our study. Therefore, endotheliosis may be the basic lesions in HELLP syndrome with or without AKI. Recently, Ospina-Tascon et al. observed substantial changes in microcirculatory blood flow and capillary density in patients with HELLP syndrome [26]. The resulting hemodynamic and endothelial changes superimposed on an already existing state of intravascular volume depletion make the kidneys more vulnerable to developing AKI. Third, TMA caused tubular ischemia in some patients. ATN was present in combination with TMA in four of eight patients with ATN in our study and the published cases. It is well known that the primary event in TMA is endothelial damage, which was identified histologically in our patients and has been reported previously in patients with HELLP syndrome [9, 22]. We speculate that part of the pathogenic process of AKI in HELLP syndrome is endothelial damage of intrarenal arterioles and glomeruli caused by TMA, leading to glomerular ischemia, tubular necrosis, or ARCN. Additionally, our study showed that severe glomerulonephritis may be a cause of AKI in HELLP syndrome. Therefore, AKI in HELLP syndrome may involve all renal compartments, including tubules, arterioles, and glomeruli. Ischemia caused by acute anemia, endotheliosis, and TMA was the main factor that caused AKI in patients with HELLP syndrome.

There were some limitations to the present study. First, patients with prolonged AKI were included in this study. We did not perform renal biopsy in patients whose renal function recovered in a few days after delivery and in those with severe complications who were not suitable for this procedure. Thus, the pathological findings in this study only represented the cases of some patients with severe AKI in HELLP syndrome. Second, the indication for renal biopsy was based on clinical experience of the physicians instead of on uniform criteria because of the nature of a retrospective study. Third, HELLP syndrome overlaps with hemolytic uremic syndrome (HUS), especially atypical HUS and thrombotic thrombocytopenic purpura (TTP) during the perinatal period and a differential diagnosis is challenging in some cases. The levels of ADAMTS13 activity and HUS-related complement system activation and regulation were not tested to exclude the possibility of TTP/HUS, because these tests were not available at our hospital. However, the obvious liver dysfunction and the rapid recovery of AST, ALT, and hemolysis after delivery supported a diagnosis of HELLP syndrome rather than HUS/TTP.

In summary, AKI is a serious complication of HELLP syndrome during pregnancy and the postpartum period. ATN is the most common pathological finding, and ACRN or ATN that coexists with TMA may potentially contribute to the adverse renal prognosis. Renal lesions may involve all compartments of the kidney, including vessels, tubules, and glomeruli. A renal biopsy is required to make a definitive diagnosis and facilitate appropriate treatment in patients with severe AKI.

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Compliance with ethical standards

Conflict of interest All authors declare that they have no conflict of interest.

Ethics approval The research protocol was approved by the Ethics Committee at PUMCH. All procedures performed in this study were in accordance with the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards. For this type of study, formal consent was not required.

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