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Atypical Hemolytic Uremic Syndrome in Patients with Pregnancy Related Renal Cortical Necrosis: An Observational Study From Western Part of India

Dear Editor,

Renal cortical necrosis (RCN) is the most catastrophic form of AKI, associated with increasing maternal morbidity and mortality. Prolonged low renal perfusion causes ischemic cortical injury via spasm, clotting, and capillary damage. Obstetric RCN is an important sequela of pregnancy-related AKI, particularly in developing countries. and is most linked to massive blood loss from conditions such as placenta previa, abruptio placentae, post-partum hemorrhage, amniotic fluid embolism, and septic abortion.[1]

Of late, complement-mediated thrombotic microangiopathy (CM-TMA) or atypical hemolytic uremic syndrome (aHUS) are distinct causes of AKI occurring in late pregnancy or postpartum.[2] In this study, we describe the clinico-epidemiological profile and outcomes of pregnancy-related RCN, emphasizing the potential contribution of aHUS, diagnosed clinically or through renal histology, and its impact on renal recovery in postpartum RCN patients from the western part of India. Materials and methods of the study have been provided in the Supplementary File.

A total of 20 patients with pregnancy-related AKI and biopsy-proven RCN were followed. The mean age at presentation was 28.1 (range: 19-42) years. Preeclampsia was seen in four patients (20%). Post-partum hemorrhage occurred in six patients (30%). A majority of patients (17/20) had an institutional delivery. The time from first medical contact to referral to our centre, as well as the delay in referral, were associated with an adverse outcome [Supplementary Table S1]. All patients presented in an anuric state after delivery and were initiated on hemodialysis. About 65% had sepsis; 17 (85%) exhibited classical microangiopathic hemolytic anemia (MAHA). Of these, 13 had histological changes of TMA on kidney biopsy. Five patients had low C3 levels, while five had normal C3 levels [Supplementary Table S2]. Nine patients were treated with plasma exchange. Patients with diffuse RCN with or without TMA on biopsy remained dialysis-dependent at 3 months. No deaths were observed during follow-up; one patient was lost to follow-up. Neonatal outcomes included intrauterine death (9), molar pregnancy (1), and live birth (10).

We assessed the relationship of various clinical and laboratory parameters with dialysis dependency at 3 months [Table 1]. In multivariate regression analysis, diffuse RCN was a statistically significant independent predictor (p= 0.006) of dialysis dependency at 3 months. Among the factors associated with the diffuse RCN, only age > 30 years had a significant association (p=0.01) [Supplementary Table S3]. Both clinical HUS and histological TMA were not associated with diffuse cortical necrosis (p=1.00 and p=0.16, respectively).

The median age at presentation was 28 years, similar to the findings from the systematic review by Gupta *et al*.[3]

Table 1: Factors affecting dialysis dependency

Factors	Dialysis dependency at 3 months	*p*-value
Age (years)		0.01
≤30	37.5%	
>30	62.5%	
Blood pressure		0.11
Hypertension	31.25%	
Hypotension	75%	
Gestation age of presentation		0.13
≤ 2nd or trimester	0%	
3rd trimester	47.10%	
Gravida		0.04
Primigravida	12.5%	
Multigravida	87.5%	
Pre-eclampsia		0.49
Yes	25%	
No	43.75%	
Post-partum hemorrhage		0.55
Yes	50%	
No	35.71%	
Mode of delivery		0.30
Vaginal	9.1%	
LSCS	25%	
Neonatal outcome		0.58
Live birth	36.36%	
Other (e.g. stillbirth/molar)	44.44%	
Sepsis		0.85
Yes	38.46%	
No	42.86%	
Plasma exchange		0.14
Yes	22.22%	
No	54.54%	
Histology (cortical necrosis)		0.44
with TMA	46.15%	
without TMA	28.57%	
RCN		<0.001
Diffuse	87.50%	
Patchy	9.10%	

LSCS: Lower segment ceaserean section, D&C: Dilatation and curretage, TMA: Thrombotic microangiopathy, RCN: Renal cortical necrosis

Despite 65% (13/20) of patients presenting with sepsis, none had derangement of coagulation profile to suggest Disseminated Intravascular Coagulation. A few patients (20%) were diagnosed with preeclampsia, but none had transaminitis or evidence of endothelial damage. Six patients had postpartum hemorrhage and septic abortion, with 5/6 exhibiting the aHUS triad.[4,5] We could not identify a possible triggering factor for atypical HUS; however, an underlying genetic defect of complement dysregulation remains a possible etiology. Patients not being followed in their antenatal period in our institution leave the possibility of any antenatal events predisposing to the condition unanswered. The onset occurred at a gestational age of 34 weeks or beyond, primarily in the postpartum period, except for two cases that occurred within 28 weeks similar to the findings of the Spanish aHUS registry by Huerta *et al*.[6] which reported an 88% postpartum onset and the French cohort of aHUS patients, which reported a postpartum incidence of 79%.[7] About 43% had low C3 levels, highlighting a probable pathophysiological role involving complement dysregulation at the cell surface.

In resource-limited setting, plasma exchange (PLEX) serves as a mainstay treatment for aHUS, where dysfunctional complement-regulating proteins are replaced by fresh frozen plasma.[8] Anti-Complement Factor H antibodies, which cause 5-10% of aHUS cases, tend to respond well to PLEX when combined with immunosuppression.[9] Only nine (45%) of our patients received PLEX [Supplementary Table 4], though effectiveness has been reported in only a few case studies of pregnancy-related TMA. In our study, PLEX did not appear to affect renal outcomes at 3 months, in contrast to a study where 50% patients had improvement. Age, multiparity, baseline hemoglobin level, and diffuse RCN were significantly associated with dialysis dependency at 3 months, with diffuse RCN being independently associated.

Diffuse RCN is a key predictor of dialysis dependency at 3 months. Larger studies with diverse populations could clarify the roles of gestational age, parity, and PLEX in preventing cortical necrosis. The limited sample size and follow-up, along with the unavailability of a comprehensive genetic workup, have restricted deeper pathogenic insights.

Conflicts of interest: There are no conflicts of interest.

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Hepatitis C Infection Among Maintenance Hemodialysis Patients in Low Resource Settings: Time to Reorient Public Payer Programs

Dear Editor,

Between 1990-2022, the prevalence of hepatitis C virus (HCV) infection among patients on maintenance hemodialysis (MHD) globally and in India has been estimated to be 24.3% and 19.3%, respectively. In low- and middle-income countries (LMICs), the prevalence is 26.8%, compared to 24.4% in high-income countries (HICs).[1] A survey in 2019 among patients on HD showed HCV seropositivity from 1.4% to 28.3% and 4.7% to 41.9% in developed and developing countries, respectively.[2] The latter have multiple risk factors, such as poor infrastructure, overcrowding, suboptimal infection control practices, hygiene and waste management issues, and inadequately trained staff. All these are drivers of HCV transmission within dialysis centres.[3] The standard of diagnosis and treatment remains inaccessible and unaffordable for health systems in LMICS. Hence, the objectives of this study are to describe the implementation of screening and treatment intervention for hepatitis C and to evaluate the cost of screening and treating HCV in patients on dialysis in India.

We conducted this study in six dialysis centers, operating under a public-private partnership, with 795 patients in Andhra Pradesh. The HCV prevalence was from 8 to 10% between May 2022 and April 2023. The selection of patients has been described in Figure 1. HCV IgG ELISA detected hepatitis C positivity in 69 patients who were then screened with the TaqMan HCV quantitative test (quantitative HCV RNA). Methodology: A real-time PCR kit was used for HCV quantification (COBAS AmpliPrep COBAS TaqMan kit). The analytical detection limit of the assay is ≥15 IU/mL, with a hit rate ≥95%).

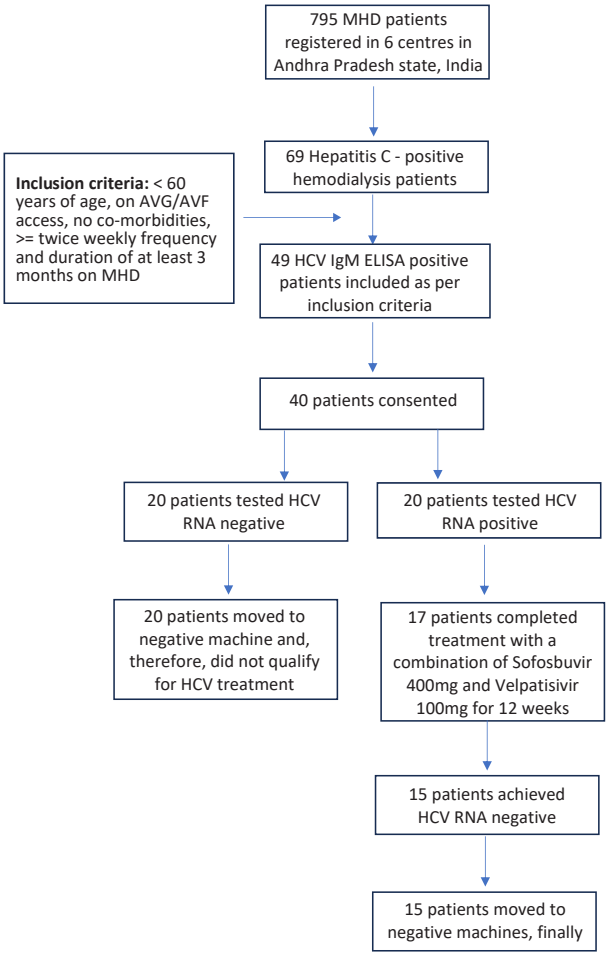

Figure 1: Selection of patients for the study.

We developed a 4-year state-transition model to estimate the costs and benefits associated with implementing a screening (ELISA followed by HCV RNA) and treatment intervention for HCV among dialysis patients compared with the no-intervention case. We modelled a cohort of 2,000 dialysis patients in the first year. Inputs for the model were primarily drawn from the observational study, as shown in Table 1. The model assumes that once cured, HCV does not recur in that patient for the period and that the intervention results in a 3% per cent