Deaths	Number of patients	Percentage
Maternal deaths	67	21.54
Fetal deaths	148	47.58

Conclusions:
1. Most common cause of PRAKI in our study was PIH (43.4%)
2. Most common biopsy finding was cortical necrosis.
3. Most common PRAKI presentation was in third trimester and puerperium.
4. IUD was associated in 47.58% cases.
5. Sepsis and multisystem involvement was in 74.27 and 21.86% patients respectively.
6. Maternal deaths was in 21.54% patients and fetal deaths was in 47.58%.
7. Total patients receiving RRT was 223 (71.70%) while 88 (28.29%) patients was managed conservatively.

No conflict of interest

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PREECLAMPSIA SYNDROME: PLATELETS AND COMPLEMENT MEMBRANE ATTACK COMPLEX.

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Introduction: The hemostatic alterations that occur in preeclampsia suggest that there is increased thrombin generation and complement system activation, increased antiangiogenic factors, and alteration in the total number of platelets and platelet index. This study seeks to evaluate the platelet system and the complement membrane attack complex (MAC) in patients with preeclampsia syndrome (PES) in comparison with normotensive pregnant women (NT) or with gestational hypertension without pathological proteinuria (GH).

Methods: Observational analytical study with pregnant women over 18 years of age receiving care at a university hospital in Brazil. Patients were randomly selected and included in the study after signing an informed consent form. Participants were classified according to their clinical status as: a) NT, b) PES, and c) GH. Three 4 mL blood samples were collected in 3 tubes containing EDTA, sodium citrate, or saline. Outcomes: total platelet count, mean platelet volume (MPV), immature platelet fraction (IPF), and soluble membrane attack complex (sC5b9). ELISA was used to measure sC5b9. Platelet numbers and platelet indices were obtained using automated XE-5000 and XN-3000 counters. The university ethics committee approved the study.

Groups (N)	PES (47)	GH (30)	NT (34)	P
Age (Years, mean ± SD)	28.5 ± 6.3	29.4± 6.8	29.9± 6.3	0.309
Gestational age (Weeks, mean±SD)	34.9± 4.2	36.3±4.5	37.9±3.4	0.007
MPV (fL; mean±SD)	12.18±1.6	11.5±1.2	10.8±0.99	<0.001
IPF (%; median min-max)	7.4 (1.9-21.8)	6.8 (2.4-17)	4.9 (1.3-9.7)	0.004
Platelets Count (n/uL, mean±SD)	199127±52864	225827±80728	240323±54321	0.012
sC5b9 (ng/dL; median min-max)	1040 (706-1433)	1221 (849-1771)	1471 (1085-1986)	0.023

Results: A total of 111 women participated in the study (PES: 47, GH: 30, and NT: 34). Results are shown in Table 1. MPV and IPF were increased in patients with PES compared to controls, but total platelet counts were reduced. The sC5b9 was increased in NT controls in comparison to SPE. No significant difference in sC5b9 levels between NT controls and GH patients was disclosed. There was no association between platelet indices and sC5b9.

Conclusions: MPV and IPF may have a potential role as biomarkers of preeclampsia, are easily accessible, can be obtained quickly, and have low cost. Its incorporation into the clinical care of pregnant women may be justified to assist in the diagnosis of PES. More studies are needed on the activation of the complement system and the interaction between platelets and the complement pathway.

No conflict of interest

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FIRST REPORTED CASE OF GELLER SYNDROME OUTSIDE NORTH AMERICA. ABSTRACT PAID FOR SUBMISSION 2022-A-WCN23-1137(200026936629)

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Introduction: Geller syndrome is a rare autosomal dominant inherited missense mutation S810L of mineralocorticoid receptor, characterized by worsening hypertension and hypokalaemia during pregnancy. This is thought to be due to upregulation of mutated mineralocorticoid receptor by abundant progesterone during pregnancy.

Methods: We report the first suspected case of Geller syndrome in Australasia to our knowledge. Our patient was a 36-year-old New Zealand European woman G4P3 who presented at 29 weeks' gestation with hypertension and asymptomatic severe hypokalaemia. Investigations confirmed hypokalaemia with potassium 2.8 mmol/L associated with 24 hour urinary potassium 29 mmol/day, hypomagnesemia, suppressed serum aldosterone level, borderline low serum renin, and normal serum pH.

She had a history of episodes of hypokalaemia and hypomagnesaemia in previous pregnancies, which were treated with potassium and magnesium supplements. Her first pregnancy was complicated by pre-eclampsia due to hypertension. Hypertension and hypokalaemia had fully resolved between previous pregnancies.

Results: In this fourth pregnancy, she was initially treated with potassium and magnesium replacement with persistent hypokalaemia. Geller syndrome was suspected and therefore Amiloride was commenced with subsequent normalization of potassium, magnesium, and blood pressure. Amiloride was stopped at one month prior to delivery. Rapid resolution of hypokalaemia occurred with delivery allowing potassium supplementation to be discontinued within days of delivery. At 3 weeks post-delivery, her serum potassium and magnesium remained normal.

Conclusions: This case fits well with the diagnosis of Geller syndrome. Other causes of hypertensive hypokalaemia are ruled out based on the clinical course of this disease. Geller syndrome has been reported in North America patients (United States of America and Canada), since it was first reported by Geller et al in year 2000. No case prior to this has been reported outside North America to our knowledge, after reviewing English based literatures. Therefore, this syndrome should be considered by clinicians throughout the world when they encounter Hypertensive Hyperkalaemic pregnant patients.

No conflict of interest