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COMPLICATIONS OF PRE-ECLAMPSIA: POTENTIALLY SERIOUS OBSTETRIC EMERGENCY

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Abstract: Introduction: Pre-Eclampsia (PE) is characterized by variable degrees of poor placental perfusion, with release of soluble factors into the circulation. **Methodology:** This is a literature review carried out through the PUBMED, LILACS and SciELO databases, using the descriptors in Portuguese: "Pre-eclampsia", Pregnancy" and "Obstetrics". 10 articles were selected, corresponding to the theme. **Theoretical Background**: PE is a multisystem disorder of pregnancy characterized by new onset hypertension (i.e., systolic blood pressure (SBP) ≥ 140 mmHg and/or diastolic blood pressure (DBP) ≥ 90 mmHg) and proteinuria (> 300 mg/ 24h). **Conclusion:** In all contexts, clinical history must not be underestimated as it provides important data and remains the most effective way of identifying pregnant women at increased risk of developing pre-eclampsia.
Keywords: Pregnancy Toxemia. Emergency

INTRODUCTION

Pre-eclampsia is a life-threatening determinant during pregnancy, which only occurs in humans, and is among the main causes of maternal and neonatal morbidity and mortality. (PE) is characterized by variable degrees of poor placental perfusion, with release of soluble factors into the circulation. These factors cause maternal vascular endothelial injury, which leads to hypertension and multiple organ damage. It is a hypertensive disorder in pregnancy related to 2% to 8% of pregnancy-related complications worldwide. It results in 9% to 26% of maternal deaths in low-income countries and 16% in high-income countries. Therefore, a study of the possible complications inherent to pre-eclampsia is crucial for adequate management of the clinical conditions presented by patients.

METHODOLOGY

This is a literature review carried out through the PUBMED, LILACS and SciELO databases, using the descriptors in Portuguese: "Pre-eclampsia", Pregnancy" and "Obstetrics". 30 articles were selected for reading, of which 10 articles were included for writing this literature review.

THEORETICAL FOUNDATION

Pregnancy is a physiological state associated with increased metabolism and oxygen demand. And it can be marked by complications, such as Hypertensive Pregnancy Syndromes (HGS), whose incidence ranges from 7.3 to 15.3%.

Preeclampsia (PE) is a multisystem disorder of pregnancy characterized by new onset hypertension (i.e., systolic blood pressure (SBP) ≥ 140 mmHg and/or diastolic blood pressure (DBP) ≥ 90 mmHg) and proteinuria (> 300 mg/24 h). (PE) is more common in industrialized countries and more common in African-American women. African American women are more likely to have comorbidities associated with preeclampsia and more likely to experience an adverse outcome during peripartum care.

(PE) is defined as the presence of new-onset hypertension and proteinuria or other end-organ damage that occurs after 20 weeks of gestation, while eclampsia is defined as the development of grand mal seizures in a woman with pre -eclampsia. Pathological studies show abnormal development of an ischemic placenta with high-resistance vasculature, which cannot provide an adequate blood supply to the fetoplacental unit. Endothelial dysfunction plays a central role in the pathogenesis of maternal syndrome. Furthermore, the general risk factors for pre-eclampsia are family history, as women of mothers who had pre-eclampsia during pregnancy have a higher risk of developing it during pregnancy,

prolonged sexual cohabitation, smoking, age, pregnancies arising from in vitro fertilization, pre-existing hypertension, diabetes, chronic kidney disease, obesity, multiple pregnancies, trisomy 13.

Limited data suggest that excess circulating soluble tyrosine kinase 1 (sFlt-1), which binds placental growth factor (PlGF) and vascular endothelial growth factor (VEGF), may have a pathogenic role. Placental growth factor is a member of the vascular endothelial growth factor (VEGF) family and is predominantly expressed in the placenta, although it is also expressed at low levels in many other tissues, including heart, lung, thyroid, liver, skeletal muscle and bone. The human PlGF gene is located on chromosome 14q14 and encodes 4 PlGF isoforms. Serum and urinary PlGF have been found to be decreased in women both at the time of diagnosis with preeclampsia and well before the onset of the syndrome. PlGF deficiency is likely due to a combination of decreased PlGF expression and reduced free PlGF due to binding to sFLT-1, which is elevated in affected women.

Women who survive preeclampsia have decreased life expectancy, with increased risks of stroke, cardiovascular disease, and diabetes, while babies from a preeclamptic pregnancy have increased risks of premature birth, perinatal death, and neurodevelopmental disability and cardiovascular and metabolic diseases throughout life. It is unknown whether the long-term cardiovascular disease risks associated with preeclampsia result from persistent vascular damage induced during the affected pregnancy or simply reflect pre-existing common risk factors that are shared by preeclampsia and cardiovascular disease. Finally, a study showed through a series of autopsies of 317 mothers who died of eclampsia, it identified brain lesions with perivascular edema in 68.4% of these women, hemorrhage in 36.8%, hemosiderin in 31.6%, thrombosis of small vessels in 10.5%, parenchymal necrosis in 15.8%, liver lesions with periportal and portal necrosis and sinusoidal fibrin in 72.2% and medial hepatic arterial necrosis in 44.4% - these are complications arising from Eclampsia in the deaths studied. Therefore, it is crucial to find intra-hospital and extra-hospital measures to minimize these deaths.

CONCLUSION

Women who survive pre-eclampsia have a reduced life expectancy, with increased risks of stroke, cardiovascular disease and diabetes, while babies from a pre-eclamptic pregnancy have increased risks of premature birth, perinatal death and developmental disability. neurological and cardiovascular and metabolic diseases later in life. Prophylactic low-dose aspirin may reduce the risk of premature preeclampsia, but once preeclampsia is diagnosed, there are no curative treatments except for delivery, and no medication has been shown to influence disease progression. Finally, in all contexts, clinical history must not be underestimated as it provides important data and remains the most effective way to identify pregnant women at increased risk of developing pre-eclampsia. Regardless of risk quantification, the identification of these conditions must guide the expansion of prenatal surveillance, avoiding causing generalized anxiety in patients.

REFERENCES

Chappell LC, Cluver CA, Kingdom J, Tong S**. Pre-eclampsia.** Lancet. 2021 Jul 24;398(10297):341-354. doi: 10.1016/S0140-6736(20)32335-7. Epub 2021 May 27. PMID: 34051884.

Dimitriadis E, Rolnik DL, Zhou W, Estrada-Gutierrez G, Koga K, Francisco RPV, Whitehead C, Hyett J, da Silva Costa F, Nicolaides K, Menkhorst E. **Pre-eclampsia.** Nat Rev Dis Primers. 2023 Feb 16;9(1):8. doi: 10.1038/s41572-023-00417-6. Erratum in: Nat Rev Dis Primers. 2023 Jul 3;9(1):35. PMID: 36797292.

Karrar SA, Hong PL. **Preeclampsia.** 2023 Feb 13. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan–. PMID: 34033373.

Lambert G, Brichant JF, Hartstein G, Bonhomme V, Dewandre PY. **Preeclampsia: an update**. Acta Anaesthesiol Belg. 2014;65(4):137-49. PMID: 25622379

Garovic VD, White WM, Vaughan L, et al. I**ncidence and Long-Term Outcomes of Hypertensive Disorders of Pregnancy.** J. Am. Coll. Cardiol. 2020; 75(18):2323-2334.

Hecht JL, **A patologia da eclâmpsia: uma série de autópsias.** Hipertens. Gravidez 36, 259–268 (2017).

Pruthi D, Khankin EV, Blanton RM, Aronovitz M, Burke SD, McCurley A, Karumanchi SA, Jaffe IZ. **A exposição à pré-eclâmpsia experimental em camundongos aumenta a resposta vascular a futuras lesões.** Hipertensão. 2015 Abr; 65(4):863-70. DOI: 10.1161/HYPERTENSIONAHA.114.04971. EPub 2015 Fev 23. PMID: 25712723; PMCID: PMC4359068.

Chau K, Hennessy A, Makris A**. Fator de crescimento placentário e pré-eclâmpsia.** J Hum Hipertensos. 2017 Dez; 31(12):782-786. DOI: 10.1038/jhh.2017.61. EPub 2017 24 de agosto. PMID: 29115294; PMCID: PMC5680413.

Shahul S, Tung A, Minhaj M, Nizamuddin J, Wenger J, Mahmood E, Mueller A, Shaefi S, Scavone B, Kociol RD, Talmor D, Rana S. **Disparidades raciais em comorbidades, complicações e desfechos maternos e fetais em mulheres com pré-eclâmpsia/eclâmpsia. Gravidez de hipertens.** Novembro de 2015; 34(4):506-515. DOI: 10.3109/10641955.2015.1090581. EPub 2015 Dez 4. PMID: 26636247; PMCID: PMC4782921.

Phipps EA, Thadhani R, Benzing T, Karumanchi SA. **Pré-eclâmpsia: patogênese, novos diagnósticos e terapias.** Nat Rev Nefrol. Maio de 2019; 15(5):275-289. DOI: 10.1038/s41581-019-0119-6. Errata em: Nat Rev Nephrol. Junho de 2019; 15(6):386. PMID: 30792480; PMCID: PMC6472952.