Plasma colloid osmotic pressure and intravenous albumin replacement in preeclampsia. A Review

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Background
Preeclampsia is the hypertensive disorder that most frequently complicates human pregnancy. It is a disease of placental origin with adverse effects on maternal organs, the fetus and the placenta itself. The endothelial lesion is the first manifestation of the maternal stage, the disturbances in the capillary permeability condition alterations of the plasmatic colloid osmotic pressure (PCOP) that can precede the clinical manifestations. Capillary leakage of albumin into the tissue space and proteinuria are the pathophysiological mechanisms that have been identified to explain hypoalbuminemia in patients with preeclampsia. It has also been established that hypoalbuminemia and reduced PCOP are adverse factors that increase maternal morbidity. However, intravenous albumin replacement has been a controversial issue.
This narrative review analyzes the most relevant data on PCOP in general, PCOP in normal pregnancy and in preeclampsia, intravenous albumin as a drug, results of intravenous albumin replacement in preeclampsia, expert opinion on the North American region and the most relevant research that has been carried out in Mexico. The intention of this review is to provide readers with basic knowledge and the most current concepts on the subject so that they have sufficient elements to establish their clinical judgment on management with intravenous albumin in preeclampsia and its maternal and fetal implications. This is an interesting topic for future clinical research.

Keywords: Plasma colloid osmotic pressure; Intravenous albumin; Capillary leak syndrome; Preeclampsia; High risk pregnancy; Obstetric intensive care.

Preeclampsia is the main hypertensive disease complicating human pregnancy. [1] Over time it continues as the disease of theories. More than 30 hypotheses have been reported in the medical literature to explain its etiology and pathophysiological mechanisms. [2] It is known that its origin is placental with devastating effects on maternal organs, the fetus and the placenta itself. [3,4] Redman [5] has proposed that preeclampsia has two stages, stage 1 corresponds to the placental phase and stage 2 includes maternal disease. Evidence has documented that the endothelium is the first maternal organ affected by preeclampsia. Endothelial swelling and apoptosis, perivascular edema, capillary leak, thrombosis, and hemorrhage of the smallest vessels are the characteristic structural findings in practically all maternal tissues and organs.[3,4] The lesions correspond to a small-vessels disease. **Figures 1 to 6** Wall abnormalities and arteriolar vasoconstriction manifesting clinically as systemic hypertension represent the transition from preeclampsia to disease of the great arterial vessels.

Ruiz Alvarez et al. [6] have documented differences in the pattern of the synthesis of vasoactive substances from the early stages of preeclampsia that clearly explain the imbalance in favor of vasoconstriction. Anym-Nyame et al.[7] studied microvascular permeability in preeclampsia, their data showed that microvascular filtration capacity is increased in preeclampsia with a significant correlation with maternal circulating levels of Tumor Necrosis Factor α (TNF α) but not with Leptin or vascular endothelial growth factor (VEGF) concentrations. Undoubtedly, other pathophysiological factors are involved, but the progressive evolution and severity of preeclampsia seem to be mainly due to high blood pressure, hypertensive complications, and generalized tissue hypoperfusion.

In preeclampsia, increased capillary permeability, endothelial damage and rupture of its basement membrane cause a state of capillary leakage of fluids, electrolytes, and macromolecules that alters the natural forces described by Starling. Thus, the reduction of the colloid osmotic pressure of plasma

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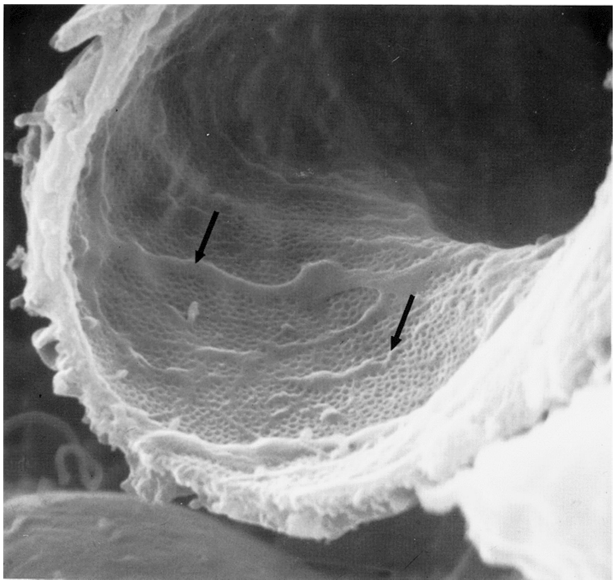

Figure 1. Appearance of a normal capillary endothelial cell seen with electron microscopy. The arrows show roughness that increase their luminal surface. Courtesy of the Department of Pathological Anatomy. High Specialty Medical Unit Gynecology and Obstetrics Hospital No. 3. National Medical Center "La Raza". Mexican Institute of Social Security. Mexico City, Mexico.

proteins (PCOP) occurs from the leakage of proteins into the interstitial space and urine. [3,4]

Plasma colloid osmotic pressure

Plasma colloid pressure (PCP) is one of the hemodynamic forces described in 1896 by Starling [8] that makes it possible to maintain fluid balance in the capillary circulation. The direction of flow between the intravascular space and the interstitial space depends on the balance of hydrostatic pressure forces, protein concentration on either side of the capillary membrane, and tissue lymphatic flow.

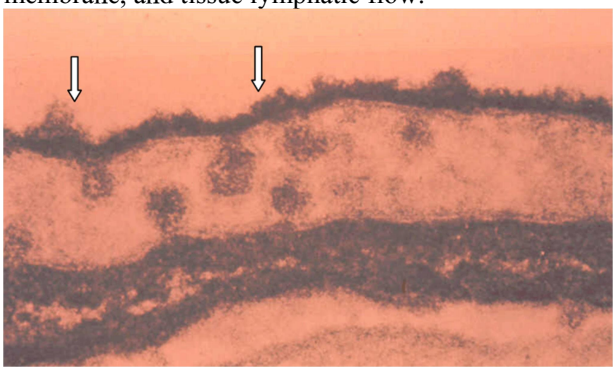

Figure 2. nterior of an endothelial cell seen with electron microscopy. Areas of injury to its luminal membrane are observed as a result of mechanical trauma due to hypertension (arrows) and the formation of vesicles that internalize them into the cytoplasm (endocytosis) as a repair mechanism. Courtesy of the Department of Pathological Anatomy. High Specialty Medical Unit Gynecology and Obstetrics Hospital No. 3. National Medical Center "La Raza". Mexican Institute of Social Security. Mexico City, Mexico.

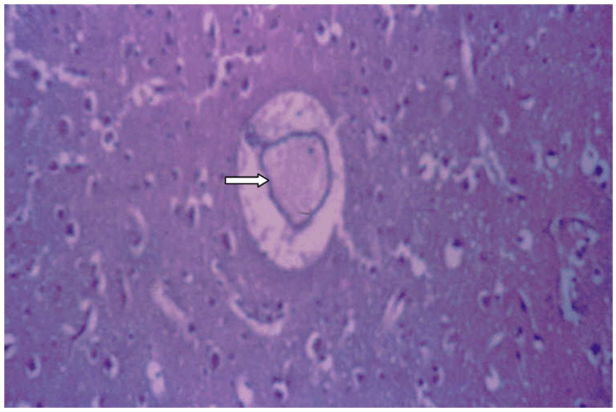

Figure 3. Cross section of a cerebral blood vessel. The arrow points to your wall. Inside there is a clot and a clear halo around the vessel which corresponds to perivascular edema. This picture is described as "eclamptic vasculopathy". Courtesy of the Department of Pathological Anatomy. High Specialty Medical Unit of Gynecology and Obstetrics Hospital No. 3. National Medical Center "La Raza". Mexican Institute of Social Security. Mexico City, Mexico.

Under physiological conditions, PCP is determined by the normal amounts of the main circulating proteins (albumin, globulins, and fibrinogen) and presupposes the integrity of the capillary wall and glycocalyx, which functions as a semi-permeable barrier. [9] When the osmotic effect of electrolytes (sodium) that are naturally bound to the protein structure (albumin) is added, the sum is identified as PCOP.

PCOP received the attention it deserved until Morissette [10] reported its importance in the prognosis of seriously ill patients because the author found that patients with PCOP values greater than 18 mmHg survived in 100% and all patients with values less than 9 mmHg died. Capillary leak syndrome is the name given to the massive passage of plasmatic fluid into the interstitial compartment due to the imbalance of Starling pressures at the level of the microcirculation. [9] In preeclampsia, this phenomenon is produced mainly by three mechanisms, the first is the increase in

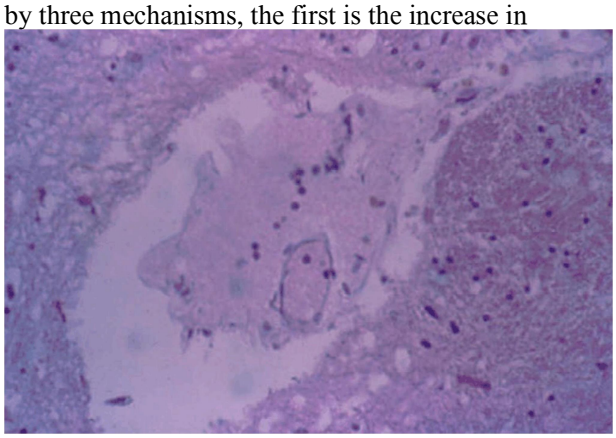

Figure 4. Microscopic image of the leakage of an erythrocyte (diapedesis) through a site of rupture of the vascular wall. Courtesy of the Department of Pathological Anatomy. High Specialty Medical Unit Gynecology and Obstetrics Hospital No. 3. National Medical Center "La Raza". Mexican Institute of Social Security. Mexico City, Mexico.

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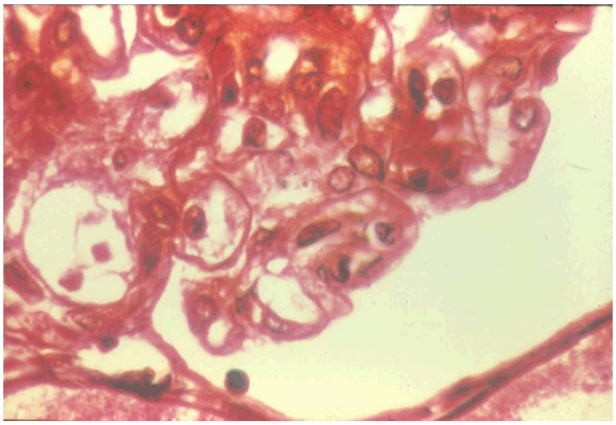

Figure 5. Microscopic image of glomerular endotheliosis. Courtesy of the Department of Pathological Anatomy. High Specialty Medical Unit Gynecology and Obstetrics Hospital No. 3. National Medical Center "La Raza". Mexican Institute of Social Security. Mexico City, Mexico.

hydrostatic pressure that is inherent to disorders of vascular mechanic that is characterized by increased blood pressure, the second is the increase in the permeability index. secondary to endothelial dysfunction, and the third is the drop in PCOP.

The colloid osmotic pressure of a liquid is proportional to the number of solute particles (proteins) it contains. In the case of human plasma, the main osmotically active proteins are albumin, globulins, and fibrinogen. Albumin is responsible for 75% to 85% of normal PCOP while the remainder is dependent on globulins and fibrinogen. [9] Traditionally, the PCOP has been calculated with the equation originally described by Landis and Pappenheimer: [11]

PCOP can also be measured directly in the patient with a device called a colloidosmometer that uses a transducer with a semipermeable membrane. This device was used by Wu et al. [12] in 1981 to measure PCOP in newborns (Wescor Colloid Omometer Model 4100, Amicon PM30 semipermeable membrane).

Plasma colloid osmotic pressure and pregnancy

In normal pregnancy, PCOP gradually decreases during the first and second trimester, reaches its lowest value between 30 and 34 weeks and increases thereafter. The variations occur due to the volume gain in the intravascular fluid that dilutes the albumin and produces a reduction in its plasmatic concentration. [13] The movement of fluid and protein during the third trimester of normal pregnancy and with preeclampsia does not follow the pattern of a circadian cycle. [14] PCOP in women with normal term pregnancy is 22 mmHg and decreases to around 16 mmHg postpartum as a result of blood loss and administration of crystalloid solutions during labor. [15]

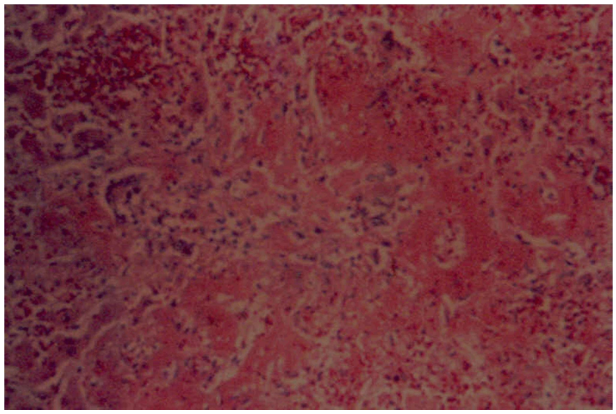

Figure 6. Microscopic image of liver tissue showing multiple sites of microbleeding. Courtesy of the Department of Pathological Anatomy. High Specialty Medical Unit Gynecology and Obstetrics Hospital No. 3. National Medical Center "La Raza". Mexican Institute of Social Security. Mexico City, Mexico.

Plasma colloid osmotic pressure in preeclampsia

In normal pregnancy, PCOP is physiologically reduced without major systemic repercussions. [1] In contrast, in women with preeclampsia, the plasmatic concentration of albumin decreases due to the increase in its passage into the interstitial space (capillary leak syndrome) and the urinary loss that characterizes the disease. Bathia et al. [16] reported in 1987 an investigation about the mechanisms for the reduction of PCOP in preeclampsia. The authors studied 32 patients with preeclampsia and 32 control patients and found a more marked reduction in PCOP and greater proteinuria in the group with preeclampsia, the correlation was very high with elevated levels of Fibronectin suggesting that endothelial injury is the main mechanism for reduction of PCOP in preeclampsia, rather than proteinuria.

In patients with pregnancy and preeclampsia, PCOP has been reported to be 15 to 17 mmHg before delivery and 13 to 14 mmHg in the immediate postpartum period. [17] As in the case of women with normal pregnancy, the decrease in PCOP in preeclampsia also depends on the type and volume of parenteral fluids used for circulating volume resuscitation and the amount of intrapartum hemorrhage. [15] Na et al. [18] have proposed that serial PCOP measurements in the peripartum stage may be useful to guide optimal fluid management in preeclampsia, but other notable factors are added; the severity of preeclampsia, the nutritional characteristics of the population, the anthropometric variations of the patients and the PCOP values related to the geographic region. [15,17,19]

The PCOP has been used to measure capillary leak in patients with preeclampsia by calculating the Briones index (BI) which is obtained by dividing the PCOP by the mean arterial pressure (MAP). [17]

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Authors year	Research site	No. cases	Clinical condition	PCOP mmHg
Briones 2000 [17]	Toluca, State of México	125	Severe preeclampsia (n=87)	15.30±4.50
2006 [38]			Severe preeclampsia (n=38)	16±3.54
Mean				15.65±4.02
Vázquez 2010 [23]	Mexico City	517	Severe preeclampsia (n=225)	20.14±2.52
2011 [39]			Severe preeclampsia (n=92)	19.45±2.37
2017 [19]			Severe preeclampsia (n=200)	19.58±8.94
Mean				19.72±4.50
Rodríguez [40] 2014	Mexico City	106	Severe preeclampsia (n=106)	19.34±2.79
Garzón [41] 2016	Mexico City	30	Severe preeclampsia (n=30) and biopsy of the parietal peritoneum	18.79±2.64
Vázquez [19] 2017	Obregón City, Sonora	172	Severe preeclampsia (n=172)	18.10±2.18
Total	----	950	----	----
Average of the means	----	----	----	18.32±3.24
PCOP; plasma colloid osmotic pressure				

Table 1. Research in Mexico on plasma colloid osmotic pressure in pregnant patients with preeclampsia. [19]

Briones index = PCOP mmHg / MAP mmHg

The PCOP is also used to calculate the hydrostatic gradient. [20]

Hydrostatic gradient = PCOP mmHg – Pulmonary artery wedge pressure mmHg

Martell-Claros et al. [21] have found that PCOP and BI are useful tools for measuring capillary leak syndrome in preeclampsia and not circulating albumin concentration.

Capillary leak syndrome is clinically identified by the exaggerated increase in maternal weight with edema of the pelvic limbs, generalized edema, and fluid collection in the cavities formed by serous membranes (pericardium, pleurae, peritoneum). In extreme cases, anasarca can occur with acute pulmonary edema and cerebral edema. These findings are considered complications that can increase the morbidity of the patients. [22.23]

Albumin as a drug

Albumin is the most abundant protein in plasma. Its molecule is made up of 585 amino acids with 17 disulfide bridges and has a molecular weight of 67,000 Daltons. [9] The total albumin content in the body exceeds 300 grams of which 40% (120 grams) are found in plasma. For every 500 ml of bleeding, only 12 grams of albumin (4% of total body albumin) are lost. Albumin deficiency is replaced by hepatic synthesis within 3 days. Under normal conditions, the concentration of total plasma proteins varies between 6.2 and 7.9 g/dL, while the albumin concentration is between 3.6 and 5.2 g/dL. When the plasma albumin concentration is <2 g/dL, edema usually occurs.[24] Albumin exerts between 75% and 85% of the oncotic pressure of blood, is synthesized by liver cells, enters the circulation via sinusoidal vessels, and remains in the bloodstream for approximately 21 days. Hepatic sinusoidal spaces are permeable to albumin and other high molecular weight proteins such as fibrinogen. [9.24]

After intravenous administration, human albumin has a half-life of 16 hours and 48 hours are required to reach equilibrium between the intravascular and interstitial compartments. It has about 75 to 85% intravascular osmotic capacity as 1 gram of albumin binds to about 18 mL of water. Exogenous albumin solution at a concentration of 5% is iso-osmotic with plasma and has been used as a circulatory volume expander. [9] Solutions with 20% and 25% albumin have concentrations 4 and 5 times higher than plasma. Therefore, when administered intravenously, for each volume of solution the blood volume increases 3.5 times in a period of 15 minutes.

The effect is due to the passage of fluid from the interstitial to the intravascular space. This movement of fluid in patients with heart failure can cause acute circulatory overload. There is also a risk of a potential aggravation of interstitial dehydration in patients with tissue hypoperfusion due to pathologies such as hypovolemic or septic shock. For this reason, it is recommended that in cases of dehydration,

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solutions with 0.9% sodium chloride or lactated Ringer-type solutions be infused at the same time. Due to the osmotic capacity of albumin, it is recommended that for every 100 ml of 20% albumin infused, about 360 ml of saline should be administered. [9]

Albumin replacement has been used specifically in patients with edema and severe hypoalbuminemia (≤2 g/dL), but without evidence of heart failure. The dose is 37 grams in 24 hours, which is equivalent to a 50 ml vial containing 25% albumin administered every 8 hours. The expansion power of albumin is the equivalent of 450 ml in a period of 60 min. Furthermore, 1 gram of albumin exerts an oncotic pressure of 5.54 mmHg and 1 gram of globulins produces an oncotic pressure of 1.43 mmHg. These physiological data are used in the clinical setting of the patients to calculate the PCOP in an approximate way, the average values in healthy adult subjects are 21.1±1.2 mmHg. [24,25]

The use of human albumin solutions has varied over time due to the imprecise and occasionally contradictory conclusions of clinical studies, to the lack of clear direction and the existence of misconceptions. Joannidis et al. [26] published a narrative review in 2022 in which they addressed ten myths and summarized the current evidence. The authors address the belief that albumin leaks from the intravascular space into the interstitial compartment and contributes to edema. The authors respond that this concept is incorrect and argue that up to 5% of intravascular albumin leaks per hour into the extravascular space with a mean distribution time of 15 hours. The transcapillary leak rate depends on the endothelial barrier function and the glycocalyx, after the extravascular leak the albumin is incorporated into the bloodstream through the lymphatic system at a rate similar to that of the transcapillary leak rate and does not remain in the interstitium. In the pulmonary territory, the lymphatic system is capable of increasing up to seven times its capacity to recover interstitial fluid and proteins. Myth number two refers to the intravascular expansion capacity of albumin, the authors argue that the intravascular expansion with albumin is greater than that of crystalloid solutions. This property can be exploited to temporarily increase the effect of loop diuretic agents administered after infusing albumin solutions and improve uresis in selected cases. Other myths are discussed and the answers are argued with current data. The authors conclude that correction of hypoalbuminemia and PCOP do not prevent the occurrence of acute kidney injury in all cases and that it is still uncertain whether albumin replacement can improve survival in cases with sepsis, it does not directly increase injury cerebral trauma, does not increase the mortality of critically ill patients and yes, replacement with intravenous albumin can increase the sodium chloride load, but in an irrelevant way. Some of these statements may apply to patients with preeclampsia, but the discussion of the ten myths does not include it as a specific topic. [26]

Intravenous albumin has the following advantages: (a) It is a commercially available drug in many countries. (b) Does not necessarily require an infusion pump or special equipment. (c) Sun protection of the infusion line is not a priority. (d) Does not transmit germs (bacteria, viruses, fungi). (e) It has not been associated with allergic processes. (f) It does not alter coagulation mechanisms or interfere with other laboratory measurements. (g) The sodium chloride overload is irrelevant. (h) The rate of the infusion is easily regulated. (i) Accidents due to rapid drip are rare because the colloidal solution that contains it cannot be infused at a high rate. (j) Its administration can be repeated periodically, that is, there is no maximum dose. The main disadvantages are: (a) It is a relatively expensive drug. (b) It needs to be stored in a cool place without direct exposure to the sun. (c) It is recommended to have access to the patient's hemodynamic monitoring during her infusion, but nonetheless in an Intensive Care Unit (ICU). (d) A chest X-ray and arterial blood gases may be necessary in selected cases. (e) Periodic determinations of PCOP and plasma albumin concentration are recommended to regulate criteria for continuing or ending its administration. (f) Fluid overload is more likely in patients with heart failure and oliguria-anuria of any cause. (g) Albumin infusion does not prevent or recover from acute kidney injury.

Intravenous albumin replacement in preeclampsia

Intravenous replacement with exogenous human albumin has shown encouraging results in critically ill patients, with tissue destruction from extensive burns, septic shock, acute respiratory distress syndrome, chronic kidney disease with nephrotic syndrome ("heavy" proteinuria), pancreatic insufficiency, liver disease with ascites, and in patients undergoing extensive surgery. [24,25,27] In the field of preeclampsia, the effect of albumin replacement on intrauterine growth restriction, [28] intervillous blood flow, [29] and maternal blood pressure has been studied. [30] Despite the fact that it has been documented for decades that PCOP is reduced in patients with preeclampsia and that capillary leak syndrome causes greater maternal deterioration [6,7,16-18,21-23] there has been no sufficient evidence that intravenous human albumin replacement is entirely useful in this type of patient.
Initially, plasma albumin concentration has not shown its strength as a criterion of severity of preeclampsia, [31] and studies of replacement with crystalloid solutions, plasma-expanding agents, and intravenous albumin related to maternal benefit have been inconclusive. Crystalloid solutions do not expand maternal

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circulating volume and significantly reduce PCOP. [9,15,24,25] Previous research has found that the administration of albumin solutions only reduces the postpartum drop in PCOP, but does not correct it. Jones et al. [32] reported in 1986 the results of a clinical study carried out to compare the effect of the infusion of crystalloid solutions (1,000 ml), Plasma-Lyte A solution (2,000 ml) and a 5% albumin solution (1,000 ml) on the peripartum PCOP. Before elective caesarean section, 45 patients received one of the three infusions. The lowest PCOP (16.6±1.1 mmHg, $p<0.05$) occurred in the infusion group with 2,000 ml crystalloid solutions 8 to 16 hours after delivery. Although PCOP fell in all groups, the reduction was significantly less ($p <0.05$) in the 5% albumin infusion group. The authors concluded that the only advantage of infusing 5% albumin solutions would be to minimize hydrostatic pressure gradient reduction to avoid acute pulmonary edema in selected patients.

Maternal circulatory volume expansion has also shown no maternal and fetal benefits. Based on the premise that plasma volume expansion may benefit both mother and newborn in early-onset and severe hypertensive disorders of pregnancy, Ganzevoort et al. [33] conducted a randomized clinical trial at two university hospitals in Amsterdam, the Netherlands. They included 216 patients with a pregnancy between 24 and 34 weeks with severe preeclampsia, HELLP syndrome, or severe fetal growth restriction who were admitted between April 1, 2000 and May 31, 2003. The treatment group (expansion of plasmatic volume with 200 ml of 6% Starch (Hydroxy Ethil Starch) infused for 4 hours every 12 hours was formed with 111 patients and the control group (intravenous fluid restriction) included 115 cases. The authors found that plasma volume expansion did not improve maternal or fetal outcome.

As a consequence, over the years the plasmatic albumin concentration has not been considered as a criterion of severity of preeclampsia and PCOP has not been included as a goal of peripartum pharmacological management of preeclampsia by experts from official organizations of the countries that make up the geographic area of North America (Canada, United States of America, Mexico). [34-37]

Investigations in Mexico of intravenous albumin replacement in preeclampsia.

In Mexico, PCOP in pregnant patients with preeclampsia has been a research topic for many years. **Table 1** shows the most representative studies. [19]

Three tertiary care centers that belong to the national health sector system (Gynecology and Obstetrics Hospital of the Maternal-Infant Institute of the State of Mexico, Gynecology and Obstetrics Hospital of the Institute for the Integral Development of the Family, Toluca, State of Mexico , General Hospital of México in Mexico City) have implemented a protocol that includes colloid solutions (25% albumin in a bottle containing 50 ml, or fresh frozen plasma 15 ml/K weight intravenously every 8 hours) to which attribute better results compared to other previous management schemes that did not include them. [42,43]

The High Specialty Medical Unit, Gynecology and Obstetrics Hospital No. 3 of the National Medical Center "La Raza" which belongs to the Mexican Institute of Social Security in Mexico City has the resource of an ICU in which nine parameters have been implemented of prenatal management of preeclampsia. The protocol includes the PCOP calculated with the formula [(plasma albumin g/dL x 5.54) + (plasma globulins g/dL x 1.43)]. The therapeutic goal is set at 24±2 mmHg and the resource for its correction consists of 25% albumin in a bottle containing 50 ml, administered as an intravenous infusion for 30 minutes every 8 hours. The therapeutic goal was achieved in only 11% of the pregnant patients with severe preeclampsia reported by Vázquez et al. [44] in 2020.

In a review of the Mexican literature from 1997 to 2018 on PCOP in preeclampsia, intravenous albumin administration is recommended in patients with prepartum PCOP <20 mmHg and postpartum PCOP <18 mmHg with continuous monitoring of the peripartum period in the ICU to detect fluid overload early and prevent acute pulmonary edema and cerebral edema. [45]

Conclusion

In preeclampsia, clinical data on capillary leak syndrome (anasarca, pleural effusion, pericardial effusion, ascites), PCOP calculation, and Briones index may be the most useful tools for selecting potential patients for albumin replacement. intravenously. The medical team should consider that albumin replacement has not shown direct maternal-fetal benefits and that it is not a substitute for more radical measures in the management of preeclampsia, such as the use of antihypertensive drugs, crystalloid solutions, termination of pregnancy, continuous follow-up in a ICU for early detection of complications and multidisciplinary management.

The window of opportunity remains open to carry out research on endothelial damage and its recovery mediated by selective drugs, the role of the glycocalyx in preeclampsia, alterations of the vascular

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wall in the maternal territory, and the role of albumin replacement to correct disturbances of Starling forces that occur in maternal microcirculation. [45] These themes have not been exhausted.

Conflicts of interests

The authors have no conflicts of interest to declare.

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