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An Overview of Hypertensive States During Pregnancy for Primary Care Physicians

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ABSTRACT

With the aim of informing the first contact physician about the hypertensive states of pregnancy, we carried out this bibliographic review. Sources in English and Spanish were consulted for the writing of this article. Hypertensive status of pregnancy refers to a group of disorders that constitute, as a group, one of the most common complications of pregnancy [1]. The incidence of this group of disorders is close to 1 in 10 pregnant women [2], due to its high frequency, it is important for all doctors to know about these states, for their early identification, particular diagnostic method to each of them and the treatment of choice. Thus through prevention at the first level of care and its treatment at the second level of care, the incidence of both maternal and perinatal complications is reduced, generating a benefit to the Mexican public health.

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INTRODUCTION

Hypertensive disorders of pregnancy affect approximately 10% of pregnant women worldwide. This group of disorders includes preeclampsia, eclampsia, gestational hypertension, and chronic hypertension. As a group of diseases they are an important cause of death in women, fetuses and newborns, preeclampsia and eclampsia stand out as the main causes among these disorders, recognized worldwide, of maternal-fetal morbidity and mortality [3][4].

Hypertensive states of pregnancy are considered the second cause of death in pregnant women, only preceded by hemorrhagic causes [5]. In Latin America, one in four maternal deaths are related to complications secondary to hypertensive states of pregnancy, most of which could be avoided by providing timely and effective care to pregnant women [4].

To think about hypertension in pregnancy systolic blood pressure exceeds 140mmHg or diastolic pressure is greater than or equal to 90mmHg, other data that suggest one of the hypertensive states of pregnancy is the presence of proteinuria, thrombocytopenia and elevation of liver enzymes [3]. Among the risk factors associated with this condition are nulliparity, multiple pregnancy, age younger than 20 years or older than 35, and comorbidities such as previously diagnosed hypertension and diabetes mellitus. [6][7]

Despite the importance of preeclampsia as a public health issue, the pathogenesis of this condition is partially understood and is related to 2 related situations: placental abnormalities and the mother's inflammatory response. In a normal pregnancy, there is an increase in the blood flow that irrigates the uterus, to ensure correct nutritional supplementation to the intervillous space and therefore adequate fetal growth. To achieve this increase in blood flow, the spiral arterioles have to be remodeled through 4 steps promoted by trophoblastic invasion in their walls [5].

At first, the decidua is invaded, followed by intra-arterial trophoblastic migration with subsequent intramural invasion of the vessels with loss of the middle muscular layer of the vessels, replaced by fibrous material and connective tissue. The final step in this remodeling is reendothelialization. All this process causes the diameter of these vessels to be greater than that of non-pregnant women, thus reducing the resistance to blood flow, increasing the blood supply. On the other hand, the arcuate and radial arteries will present greater pressure on their walls, resulting from the greater flow, which by generating endothelial stress stimulates the production of nitric oxide, a vasodilator [5].

The previously mentioned process occurs physiologically in approximately 90% of the uterine vessels. In the pathophysiology of preeclampsia, it has been found that these

changes are qualitatively reduced or do not occur, with an association with atherosclerotic processes in the vessels, thus reducing blood flow and increasing the resistance of these vessels. The pathophysiology of preeclampsia also includes platelet activation and consumption, vasospasm, and prostacyclin deficiency, which has a vasodilator effect and inhibits platelet aggregation. In addition to the increased production of thromboxane A2, which is involved in vasospasm and increased platelet aggregation [5]. Its diagnostic process, screening tests, severity classification and treatment continue to be controversial worldwide. [4]

DEFINITION

Hypertensive disorders of pregnancy are one of the most common complications during pregnancy and in the early postpartum period. The principal hypertensive disorders in pregnancy can be divided into two, 1) non-urgent hypertensive disorders of pregnancy and 2) urgent hypertensive disorders of pregnancy;

1) Non-Urgent Hypertensive Disorders of Pregnancy

- Chronic hypertension is diagnosed before pregnancy or in the first 20 weeks of pregnancy. [1]
- Gestational hypertension, defined as pregnancy-induced hypertension (systolic BP >140mmHg and diastolic BP >90mmHg on two occasions at least 4 hours apart), diagnosed after 20 weeks gestation in patients without proteinuria or target organ damage and a history of hypertension, can only be diagnosed if the patient was normotensive before 20 weeks of gestation. [1, 8]
- Postpartum hypertension is persistent after childbirth and resolves 12 weeks after delivery; if it lasts more than this time, it should be considered a secondary cause. [9. 10]

2) Urgent Hypertensive Disorders of Pregnancy

- Preeclampsia, defined as new-onset stationary hypertension with proteinuria or target organ dysfunction after 20 weeks of gestation, can occur in patients with chronic hypertension; in such a situation, it would be called superimposed preeclampsia. It is subdivided into mild and severe preeclampsia. [8]
- Eclampsia is a convulsive manifestation in patients with preeclampsia, characterized by tonic-clonic seizures, focal or multifocal, new onset in the absence of other causes. [1]
- HELLP syndrome is a life-threatening form of preeclampsia characterized by hemolysis, elevated liver enzymes, and thrombocytopenia and may occur with or without hypertension or proteinuria. [8]

EPIDEMIOLOGY

The incidence of hypertensive disorders in pregnancy generally affects approximately 6 to 8% of all pregnancies, thus affecting almost 1 in 10 pregnancies. The most common hypertensive disorder during pregnancy is preeclampsia, the cause of discharges in the blood pressure in approximately 5 to 7% of all pregnancies, followed by HELLP syndrome, which affects 0.5 to 0.9% of all pregnancies, and eclampsia <0.1% of all deliveries. [2, 11, 12, 13]

ETIOLOGY

Although the etiology is not fully understood, a higher prevalence can be observed in patients with a more significant number of risk factors, which are divided into two groups, general risk factors, and pregnancy-related risk factors. [6]

General risk factors include, but are not limited to, thrombophilia, <20 or >35 years of age, African descent, diabetes mellitus or gestational diabetes, chronic hypertension, chronic kidney disease, and obesity. [6, 7]

Pregnancy-related risk factors include nulliparity, multiple gestations, hydatidiform mole, prior preeclampsia, chromosomal abnormalities, or congenital structural abnormalities, as well as concordant family history. [6]

PATHOPHYSIOLOGY

Placental hypoperfusion produces maternal hypertension and other consequences due to multiple maternal, fetal, and placental factors in circulation. The most important factors are the following:

- Abnormal implantation of the placenta or trophoblast in the uterus.
- The uterine spiral arteries usually become high-capacity blood vessels; in patients with preeclampsia, this process is dysfunctional, leading to placental flow at high pressures as a compensatory method.
- Systemic vasoconstriction causes placental hypoperfusion, which increases the release of vasoactive substances, increasing arterial pressure.
- Systemic endothelial dysfunction causes placental hypoperfusion resulting in increased placental factors causing endothelial injury leading to microthrombosis.[14, 15]

As a consequence of vasoconstriction and microthrombosis, tissue ischemia and damage to target organs are caused, with the kidneys being the main affectation in preeclampsia, cerebral in eclampsia, and multi-organ hemorrhage and liver disease in HELLP syndrome. In addition, placental hypoperfusion causes insufficiency of the uteroplacental unit and fetal growth restriction. Below is a table with the systemic effects of different hypertensive disorders of pregnancy and their organic involvement. [15, 16]

Table 1.

Organic alterations in hypertensive states during pregnancy			
Organ	Pathophysiological mechanism	Clinic/Laboratory	hypertensive state
Kidney	Glomerular endothelial dysfunction and hypertension-induced vasoconstriction	Proteinuria Renal insufficiency Edema	Preeclampsia Eclampsia HELLP syndrome
Lung	Increased systemic vascular resistance and volume overload	Pulmonary edema respiratory distress	Severe preeclampsia HELLP syndrome
Liver	Vasoconstriction and microthrombotic obstruction of hepatic sinusoids	Liver failure and non-alcoholic hepatitis	Preeclampsia Eclampsia HELLP syndrome
CNS	Hypertension-induced vasoconstriction and endothelial damage, CNS vasospasm	Seizures	Eclampsia
Blood	Systemic microthrombi and overactivation of coagulation as well as microangiopathic hemolysis	Disseminated intravascular coagulopathy thrombocytopenia Anemia	Severe preeclampsia HELLP syndrome

CLINICAL MANIFESTATIONS

The clinical characteristics can often go unnoticed as non-specific symptoms. However, blood pressure can always be measured objectively, as in gestational hypertension, where the patient is generally asymptomatic or with symptoms such as headache, fatigue, and nausea. In preeclampsia, the symptomatology changes depending on the severity of the disease and generally occurs after 34 weeks (up to 90% of cases) of pregnancy. However, mild preeclampsia is generally asymptomatic, and non-specific symptoms may include headaches, visual disturbances, right upper quadrant pain, or epigastric pain, as well as the development of edema and proteinuria. In severe preeclampsia, we can find blood pressure figures systolic BP >160mmHg or diastolic BP >110mmHg, proteinuria, oliguria, headache, visual disturbances, pain in the epigastrium and/or right upper flank, pulmonary edema, and neurological symptoms such as altered consciousness or clonus. [12, 17]

Eclampsia is characterized by eclamptic seizures, generalized tonic-clonic seizures that are generally self-limited, most often associated with severe preeclampsia, and mainly intrapartum and postpartum. Some warning signs of deterioration in a patient who may develop eclampsia are headache, right upper quadrant pain, hyperreflexia, and visual changes. [11]

HELLP syndrome commonly occurs at >27 weeks of gestation; however, 30% occur after the delivery, and preeclampsia is present in up to 85% of cases and may be accompanied by nausea, vomiting, diarrhea, and abdominal pain. Hepatic capsule and rapid clinical deterioration due to complications specified below. In HELLP syndrome, hypertension and proteinuria may be mild or absent and present a series of non-specific symptoms. [18]

DIAGNOSIS

The initial diagnosis should be done to all pregnant patients during attendance at prenatal appointments. The initial study for all suspected hypertensive disorders of pregnancy is the measurement of blood pressure; we can name it hypertension when >140/90mmHg in at least two separate measurements at least 4 hours apart, and if the systolic blood pressure is >160mmHg and the diastolic blood pressure is >110mmHg, it is considered severe hypertension. Diagnostic criteria for hypertensive disorders of pregnancy are listed in Table 2. [1, 19].

For the evaluation of proteinuria, the 24-hour urine collection can be used, which is considered the gold standard (proteinuria >300mg/24hrs), the urine protein: creatinine ratio (>0.3), and the use of reactive strips are considered less accurate but can be used in the absence of other studies (>2+). [1, 19, 20]

For the assessment of target organ dysfunction, laboratory studies should be performed, including blood count, liver chemistry, renal function tests, and lactate dehydrogenase, in addition to the need in specific cases for serum electrolytes, electrocardiogram, uric acid, peripheral smear, and coagulation studies, as well as head CT in exceptional cases. The alterations that can be observed in the studies are:

- Hematic Biometry; decreased hemoglobin and platelets may be seen in severe preeclampsia or HELLP syndrome.
- Liver Chemistry; An elevation of transaminases suggests severe preeclampsia or HELLP syndrome.
- Kidney function tests; A decrease in GFR is indicative of severe preeclampsia.
- Lactate Dehydrogenated; may be elevated in HELLP syndrome.
- Serum electrolytes, electrocardiograms, and uric acid levels are studies that should be performed in patients with chronic hypertension. [1, 19, 20]

For patients in whom HELLP syndrome is suspected, thrombocytopenia and impaired liver function may be seen, schistocytes on the peripheral blood smear indicate hemolysis, as well as elevated D-monetary, TP, and TTP, decreased fibrinogen, and antithrombin III suggest disseminated intravascular coagulation. [21]

Patients with chronic hypertension should undergo 24-hour urine protein, liver function, and kidney function tests from the first appointment for prenatal care to maintain control and observe an increase in baseline values, which can indicate superimposed preeclampsia. [19, 21]

In patients with headaches refractory to treatment or with neurological symptoms, a head CT should be performed to rule out intracranial hemorrhage or any alternative pathology. [21]

DIAGNOSTIC CRITERIA

Table 2.

Disorder		Diagnostic Criteria
Chronic Hypertension		Hypertension diagnosed before pregnancy or in the first 20 weeks of pregnancy with or without end-organ dysfunction.
Gestational Hypertension		Hypertension diagnosed at ≥ 20 weeks of gestation. Before 20 weeks gestation, patients were normotensive at all previous antenatal care visits. No history of pre-existing hypertension Patients are asymptomatic with normal laboratory studies (no proteinuria, no target organ dysfunction).
Preeclampsia	…without severe features	Hypertension (≥ 140/90 mm Hg) Moreover, proteinuria is evidenced by any of the following: 24-hour urine collection: ≥ 300 mg/24 hours Urine protein:creatinine ratio: ≥ 0.3 Urine dipstick : > 2+ protein
	…with severe features	Gestational hypertension, + ≥ 1 of the following: Severe hypertension Proteinuria Thrombocytopenia (e.g., platelets < 100,000 cells/mm 3) Renal insufficiency Serum creatinine > 1.1 mg/dL Doubling of serum creatinine Impaired liver function Persistent right upper quadrant pain Pulmonary edema The new appearance of any of: Headache that does not respond to medication visual disturbances
	HELLP syndrome	Preeclampsia plus all of the following: H = Hemolysis EL = Elevated liver enzymes LP = Low platelets (< 100,000 cells/mm3)
	Chronic hypertension with superimposed preeclampsia	History of chronic hypertension with any of the following: New occurrence of ≥ 1 of the following: Proteinuria Thrombocytopenia Impaired kidney or liver function Symptoms of preeclampsia Or sudden worsening of proteinuria or existing hypertension
Eclampsia		New-onset seizures in a patient with preeclampsia (may be the presenting symptom in some cases)

Fetal evaluation is part of the diagnosis of these pathologies and must be carried out in parallel with the studies of the mother; cardiotocography must be performed to monitor the fetal heart rate and uterine contractions, and ultrasound will serve to evaluate fetal movement and tone to assess signs of fetal distress. Doppler ultrasound for evaluation of fetal and placental blood flow, data range from increased resistance and abnormal flow pattern in atypical uterine arteries, as well as bilateral grimacing (early diastolic bleeding). The most common complications are: [22, 23, 24]

- Fetal growth restriction
- placental abruption

- Oligodhramnios

Differential diagnoses

Although the diagnosis of hypertension in pregnancy can be done with the previously mentioned criteria, the differential diagnoses for a hepatic chemical alteration, or eclampsia, as well as for HELLP syndrome, are expressed in Table 3.

DIFFERENTIAL DIAGNOSES

Table 3 [25, 26, 27, 28, 29]

Altered liver chemistry	Eclampsia	HELLP syndrome
Hyperemesis gravidarum Intrahepatic cholestasis of pregnancy Liver acute degree of pregnancy HELLP syndrome	Epilepsy Encephalitis Metabolic disorders Hemorrhagic seizure Stroke Abstinence syndrome	Thrombotic microangiopathy (thrombocytopenia) Acute fatty liver of pregnancy Intrahepatic cholestasis of pregnancy Acute liver failure

TREATMENT

The main treatment is antihypertensives, which should be administered 30 to 60 minutes after diagnosis in urgent hypertensive disorders. Antihypertensives used in hypertensive emergencies during pregnancy include but are not limited to: [1, 30]

- Parenteral Labetalol (avoid in patients with contraindication to B-Blockers)
- Nifedipine (immediate release)
- Parenteral hydralazine.

For non-urgent hypertensive states, Labetalol, Nifedipine (extended-release), and Methyldopa can be used orally; ACE inhibitors and BRAs should be avoided during pregnancy with teratogenic effects. [1, 30]

Patients with severe preeclampsia, HELLP, or eclampsia require immediate blood pressure control and treatment for possible complications (in an ideal world, in a 3rd level care institution); these actions reduce maternal and fetal morbidity and mortality. [1, 30, 31]

In patients with the above diagnoses, the use of antihypertensives should be started urgently, as well as the administration of magnesium sulfate to prevent seizures; in addition, the indications for immediate delivery should be evaluated after the hemodynamic stabilization of the mother. [31, 32]

Indications for accelerated labor: [32]

- Eclampsia (with neurological symptoms)
- Pulmonary edema
- Disseminated intravascular coagulation
- Placental abruption
- Severe hypertension refractory to antihypertensive drugs
- Signs of fetal distress
- Fetal death or fetus is unlikely to survive.

Delivery should be accelerated after administration of corticosteroids for fetal maturity (before 34 weeks of gestational age) if any of the following are present: [31, 32]

- Labor or premature rupture of membranes
- Severe oligodrahmnios
- Inverted end-diastolic flow in the umbilical artery Doppler
- New-onset or worsening kidney failure
- Moderate or severe thrombocytopenia
- Abnormal liver chemistry

The management of preeclampsia, eclampsia and HELLP syndrome are described in Table 4, which mentions medical and obstetric management in general. [1, 31, 32]

Table 4

Medical and obstetric management			
	Severe Preeclampsia	Eclampsia	HELLP syndrome
Medical Management	Initiation of antihypertensive treatment, prophylactic administration of magnesium sulfate, and control of blood pressure, oxygen saturation, and uresis are essential for a better prognosis and management of complications.	Treat eclamptic seizures by placing the patient in the left lateral decubitus position to prevent placental hypoperfusion due to compression of the inferior vena cava and reduce the risk of aspiration. Anticonvulsant therapy should start with magnesium sulfate and blood pressure management.	Administer blood products as needed to control bleeding and coagulopathy. Start of antihypertensives. Administration of magnesium sulfate.

Obstetric Management	Suppose the indications for immediate delivery are not completed. In that case, delivery acceleration should be performed if the pregnancy is >34 weeks gestation, the mother should be stabilized, administration of pulmonary maturation, tests of explanatory management, and close maternal surveillance. Fetal. Vaginal delivery is preferred, but a cesarean section is usually required in poor maternal-fetal conditions.	Eclampsia is an indication for immediate delivery regardless of gestational age; it should only be performed after stabilizing maternal symptoms.	Accelerated delivery is indicated for all patients regardless of gestational age. >34 weeks: Immediate delivery 24-34 weeks: Administer lung maturers; delivery should be delayed 24-48 hours after administration of corticosteroids.

The management of non-urgent hypertensive pathologies is based on a series of maternal-fetal evaluations to determine severity. Gestational age should be initially evaluated. Labor induction is recommended if gestational age is >37 weeks of gestation. If the pregnancy is below 37 weeks of gestation, expectant management should be maintained until the 37th week of gestation; unless there are other indications to accelerate delivery, close control of these patients should be maintained, evaluating from 1 to 2 times per week, assessing blood pressure, laboratory studies and fetal evaluation. The start of antihypertensives is necessary once the pathology has been identified and the start of preeclampsia prophylaxis with aspirin. Early delivery is considered after the administration of antenatal corticosteroids for accelerating fetal lung maturation, and if it is between 24-34 weeks of gestation, and as in any pathology, it is necessary to educate and provide the necessary information on signs and symptoms of fetal distress in order to receive immediate medical care. [1, 30, 31]

Management for chronic hypertension in pregnant patients is based on promoting lifestyle modifiers; the threshold for initiating antihypertensives in patients without previous treatment is >140/90 mmHg. [1]

For mild hypertension (>140/90mmHg), treatment with the antihypertensives mentioned above is started, and treatment is continued in patients who are already receiving medical treatment. In case of severe hypertension (>160/110mmHg) lasting more than 15 minutes, antihypertensive therapy should be started within the first hour after diagnosis. [30, 31]

For gestational hypertension or preeclampsia without severe features, if the patient has >37 SDG, she should be hospitalized until delivery; in patients with <37 SDG, a complete obstetric ultrasound should be performed with fetal estimation and fluid volume and start with the antihypertensive treatment, as well as weekly maternal-fetal follow-up. [1, 30]

Outpatient management is based on serial blood pressure monitoring, an evaluation of the development or worsening of preeclampsia, a platelet laboratory, serious creatinine, liver chemistry, and proteinuria. Fetal monitoring is based on an amniotic fluid index, nonstress fetal testing, and echocardiographic evaluation (every 3-4 weeks). Unless otherwise specified, it should be performed once to twice a week until the 37 SDGs are met. [1, 30, 31]

COMPLICATIONS

Complications can be divided into two large groups, maternal complications and fetal complications; the complications are listed in Table 5: [33, 34]

Maternal complications	Fetal complications
Placental abruption	Fetal growth restriction
Disseminated intravascular coagulation (20% of patients with HELLP)	Premature labor
Cerebral hemorrhage	Seizure-induced fetal hypoxia
Acute respiratory distress syndrome	Fetal death
Acute renal failure	
Hepatic subcapsular hematoma	
Aspiration pneumonia	
Retinal detachment	
Diabetes Mellitus and Chronic Kidney Disease	
Maternal death	

PROGNOSIS 35, 36, 37

The prognosis of hypertensive disorders of pregnancy depends almost entirely on the severity of the involvement and the complications that have occurred; most cases have a resolution that ranges from hours to days after delivery. Table 6 shows the recurrence rate, maternal mortality, and fetal mortality. [35, 36, 37]

Table 6

Recurrence rate in subsequent pregnancies	Preeclampsia: 10-20% Eclampsia: 1-2% HELLP syndrome: 3-5%
Maternal mortality	Eclampsia 5-10% HELLP syndrome: 1-3.5%
Fetal mortality	Eclampsia: 5-11% HELLP syndrome: 24%

PREVENTION

The primary prevention is weight loss and exercise. It has been proven that for exercise to start doing a positive effect in the pregnant and nor-pregnant woman with risk of preeclampsia has to be al least 3 days a week, 25 minutes per workout. [38] An early diagnosis is probably the most important action for preventing hypertensive states complications, and the later it starts has a positive association with poor fetal outcome [39, 40]. Low dose aspirin is the most successfully proven preventive pharmacological intervention; low calcium supplementation has also been proven beneficial. [38] Other studies have tried with alternative treatments such as nicotine analogues or plant extractive from Eriosema kraussianum, also demonstrated helpful in prevention or treatment of preclampsia in early pregnancies. [39] The only curative treatment is the ending of pregnancy [40], if the medical criteria decides to postpone this event the patient has to be in close clinical and laboratorial surveliance, due to risk of developing an alteration in the coagulation cascade, correlated with previous appearance of kidney and liver failure, seen more often in patients with HELLP syndrome. [41]

DISCUSSION

Hypertensive states of pregnancy are in fact a major concern for all physicians, or should be, due to their high incidence of approximately 1 in 10 pregnant women [2,3], constituting one of the most important causes of morbidity and mortality in the pregnancy [3,4], considered the second cause of death in pregnant women [5], in Latin America one of every 4 deaths can be related to a hypertensive state of pregnancy [4].

The reviewed sources inform us that to exist a hypertensive state of pregnancy we have to find a blood pressure that is greater than 140/90mmHg in its systolic or diastolic value in at least 2 shots with more than 4 hours of difference, after this they must of requesting laboratory studies due to the different results that can be found in each hypertensive state [1,19]

Hypertensive states are divided depending on the week in which the alteration in blood pressure is found, prior to week 20 it is considered chronic hypertension because the pathophysiology that was recognized in the different sources cannot be a cause prior to this gestational week [5, 14, 15] and then we assume that it was a finding of hypertension prior to pregnancy. After 20 weeks of gestation, we can refer as such to the hypertensive states of pregnancy, now differentiated by the different objective conditions that they may produce.

Gestational hypertension is considered with the same high blood pressure figures (>140/90), with the same diagnostic criteria (at least 2 doses with 4 hours separation between doses), in which proteinuria is not demonstrated (or this is less than 300mg/ 24hrs), target organ damage or history of hypertension at 20 weeks of gestation, this is considered a non-urgent hypertensive state of pregnancy. [1,8] Another of the non-urgent conditions of the hypertensive state of pregnancy is postpartum hypertension, which should resolve prior to 12 weeks after birth. [9,10]

CONCLUSION

The hypertensive state of pregnancy is a potentially fatal complication of gravidity that unfortunately is presented by many pregnant women in our country, so the early identification of these states is relevant to prevent the previously mentioned complications that can endanger the life of the binomial. Reducing the incidence of hypertensive states in pregnancy, especially those with a higher mortality rate, eclampsia for the mother and HELLP syndrome for the product, is a great challenge and a health goal committed by the country, therefore, prevention strategies are a priority.

Each hypertensive ailing in pregnancy has unique characteristics, which must be discovered by the treating physician. This can be done by doing corresponding laboratory studies, which were recommended in this review article (CBC, liver function tests, lactic dehydrogenase, collection of urine of 24 hours in search of proteinuria), in addition to serial blood pressure measurements to make the proper diagnosis and determine the severity of hypertension. Each case must be individualized and the correct clinical analysis must be carried out with additional studies if considered necessary (e.g. coagulation times and D-dimer if disseminated vascular coagulation is suspected).

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