Chapter

Adolescence and Preeclampsia

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Abstract

Adolescent pregnancy is defined as that which occurs in a woman between 10 and 19 years of age. Approximately 10% of all women aged 15–19 become pregnant. It is estimated that 11% of births worldwide occur in this population. In teenage population, preeclampsia has a prevalence twice as high as that in adult population. Adolescent population is exposed to different maternal-fetal adverse outcomes such as preterm birth, low birth weight, and gestational diabetes mellitus, associated with the outcomes of preeclampsia like seizures, pulmonary edema, defects in coagulation, liver or kidney failure, and death. The risk of adverse outcome remained increased in adolescent compared to young adult mothers (20–24 years). That is why it's important to know the approach of preeclampsia in adolescent pregnancy. We will describe the principal chance in the adolescent pregnancy, related risk factors, major complications for mother and fetus, and management and late complication for both.

Keywords: adolescent pregnancy, preeclampsia

1. Introduction

Pregnancy at any age constitutes a very important biopsychosocial event, but in adolescence it encompasses a series of situations that may place the mother's and the fetus' health at risk, thus becoming a public health issue not only in the present but also in the future due to potential complications and as a reflection of the social conditions of a country.

Maternal, late fetal, neonatal, and infant mortalities have the highest rates in women under 20 years of age than in any other reproductive period.

Complications in adolescent mothers are a constant dilemma since many factors are involved, such as inadequate prenatal control, race, family factors, and economic and sociodemographic conditions.

Hypertensive problems associated with pregnancy are another public health issue that in developing countries still represent the first cause of maternal death; worldwide, their prevalence varies but hovers around 10% of all pregnancies.

Whatever causes triggering complications, maternal and neonatal deaths are significant indicators in all countries, and in the case of pregnant adolescents, developing preeclampsia and their high and varied worldwide prevalence make this combination, specifically this group of patients, a point of reference when establishing health policies.

2. Definition of adolescence

The WHO defines adolescence as the period of human growth and development after childhood and before adulthood, between the ages of 10 and 19. It is one of the most important transitional phases in human life, characterized by an accelerated rhythm of growth and changes, only surpassed by those in infants. This growth and development phase is conditioned by several biological processes. The beginning of puberty is the hallmark of passage from childhood to adolescence. From this stage on, the reproductive system's capacity to procreate is potentially latent [1].

3. Classification of adolescence

It is difficult to establish the chronological boundaries of this period, but in accordance with conventionally accepted concepts accepted by the World Health Organization (WHO), adolescence may be divided into two phases:

1. Early (10–14 years).

2. Late (15–19 years).

Likewise, other authors refer that there are three stages:

1. Early adolescence, between the ages of 10 and 13.

2. Mid-adolescence, between the ages of 14 and 17.

3. Late adolescence, between the ages of 17 and 21 [2].

4. Impact of pregnancy on an adolescent

The beginning of sexual activity in adolescents currently occurs at a younger age and carries immediate unwanted consequences such as an increased frequency of sexually transmitted diseases (STD) and unwanted pregnancy that may lead to miscarriage or other complications during pregnancy [3].

From a biological viewpoint, some of the consequences of adolescent pregnancy include hypertensive disease of pregnancy, anemia, gestational diabetes, and complications during childbirth that lead to an increase in maternal and fetal mortality [4]; complications in the newborn include higher rates of low birth weight, premature delivery, respiratory diseases, dystocia, and an increased frequency of neonatal complications and greater infant mortality [5].

Risk factors in adolescent pregnancy include low educational level, beginning sexual activity before the age of 15, absence of the partner, maternal history of pregnancy in adolescence, and the lack of knowledge and access to birth control methods. There is also a high percentage of school dropouts, a lack of plans for the future, low self-esteem, alcohol and drug abuse, ignorance on sexuality, and an inadequate use of birth control [6].

A correlation has been shown between minimal or absent family communication on birth control and sexuality and a higher risk of adolescent pregnancy and infection with sexually transmitted diseases [7].

Medical risks associated with pregnancy in adolescent mothers such as hypertensive pathology, anemia, low birth weight, prematurity, insufficient nutrition,

etc. lead to an increase in maternal morbidity and mortality and an estimated two- to threefold increase in infant mortality among patients in the age range between 20 and 29.

In adolescents, the higher observed compared risk does not appear to be due to special physiologic conditions but to sociocultural variables and the medical care provided to these patients.

These pregnancies are frequently unwanted or unplanned events within a weak couple relationship, which leads to an attitude of rejection and concealment because of fear of the family group that, in turn, conditions late or insufficient prenatal care.

According to various publications, we must emphasize that 73–93% of cases of pregnant adolescents are women bearing their first child. The first pregnancy carries specific risks resulting from physiological immaturity in the pregnant

		<16 years	**>16 years**
Kawakita [37]	CS	OR = 0.49; CI 95% = 0.42–0.59	OR = 0.75; CI 95% = 0.71–0.79
	CA	OR = 0.63; CI 95% = 0.47–0.84	OR = 0.83; CI 95% = 0.75–0.91
	MA	OR = 1.25; CI 95% = 1.07–1.45	OR = 1.15; CI 95% = 1.09–1.22
	PD < 37 weeks	OR = 1.36; CI 95% = 1.14–1.62	aOR = 1.16; CI 95% = 1.08–1.25
	PPH* and BT**	*OR = 1.46; CI 95% = 1.10–1.95	**OR = 1.21; CI 95% = 1.02–1.43
	Preeclampsia and HELLP Sd	OR = 1.44; CI 95% = 1.17–1.77	
	PL		OR = 0.82; CI 95% = 0.71–0.95

Bostanci et al. [21]		Early adolescent (%)	Late adolescent (%)	Adult (%)	P value
	Preeclampsia	4.8	2.7	5.9	<0.001
	PD	37.2	12.8	2.2	<0.001
	PPROM	37.2	10.2	8.5	<0.001
	IUGR	9	3.3	5.4	<0.001
	Postterm	0.7	5.9	8.7	<0.001
	Episiotomy	79.3	69.8	70	>0.05
	NICU	18	11.7	10	0.009
	Neonatal outcome	2.1	1.1	2.1	>0.05
	LBW	17.9	13.2	13.1	>0.05
	VLBW	4.1	3.4	2.7	>0.05
	CS	17.2	25.7	29.6	0.001

CS, cesarean section; CA, chorioamnionitis; MA, maternal anemia; PD, preterm delivery; PPH, postpartum hemorrhage; BT, blood transfusion; PL, perineal laceration; PPROM, preterm premature rupture of membranes; IUGR, intrauterine growth restriction; NICU, neonatal intensive care unit admission; LBW, low birth weight; VLBW, very low birth weight.

Table 1.
Comparison of some pregnancy complications and outcome among early and late adolescent.

adolescent. For example, preeclampsia or gestation-induced hypertension is more frequent in young pregnant women, from a low socioeconomic level, and specifically, in the first pregnancy, conditions all frequently met by a pregnant adolescent. When developing this clinical entity, a possible failure in the adaptive immune response has been posited, although it normally permits the development of a close interrelation between the maternal organism and its host. Since 50% of the fetal antigenic structure is of paternal origin, it acts like a graft and has been associated to factors such as immaturity of the maternal immune system or a functional abnormality that may be associated to maternal malnutrition, a very common condition in pregnant adolescents. Morbidity may be classified according to the gestational periods, whereby miscarriage, anemia, urinary tract infections, and asymptomatic bacteriuria are more common in the first half. In the second half, there are hypertensive manifestations, hemorrhage associated to placental pathology, scarce weight gain and associated maternal malnutrition, symptoms of premature delivery (abnormal contractility), and premature membrane rupture [8, 9].

Several authors have suggested that there is a relation between hypertensive disorders and pregnancy, which will be further discussed in this chapter. Other complications are summarized in **Table 1**.

A strategy to confront these problems and adolescent pregnancy is to increase the availability of high-quality sexual and reproductive health services for adolescents [10].

5. Frequency of preeclampsia in adolescents

Preeclampsia refers to a relatively common hypertensive disorder during pregnancy that develops progressively. Its cause remains unknown, and it frequently leads to severe maternal and perinatal complications.

Its incidence is broad since it depends on various aspects such as geographic location, race, nutritional or immunological factors, and associated comorbidities, and even a humid and cold climate has been related to a higher incidence of affected women.

It is estimated that about 7% of pregnancies will develop preeclampsia. The incidence of preeclampsia is about 25% in primiparous women. In industrialized nations, the rate of maternal morbidity due to preeclampsia varies between 3.8 and 12 per 1000 births. In Latin America, there are few reports on the subject, so the real magnitude of the problem remains unknown; there are only some studies from Brazil and Cuba [11].

Throughout the world, 25% of all maternal deaths occur in adolescent women. In Latin America, adolescent pregnancy is an independently associated factor conferring greater risk for adversities during pregnancy [12, 13]. In the United States, the maternal mortality rate due to preeclampsia-eclampsia is approximately 1 for every 100,000 live births. In Mexico, maternal mortality has decreased in the past six decades, whereby 1281 maternal deaths were registered in the year 2009, in women between the ages of 15 and 34 [14].

The main causes of death in pregnant women were:

1. Hypertensive disease induced by pregnancy (20.4%).

2. Obstetric hemorrhage (19%).

3. Sepsis (4.1%).

These percentages are five to ten times greater than those reported in industrialized nations or in those with more developed national health systems [15, 16].

6. Definitions of hypertensive states in pregnancy

According to the American College of Obstetricians and Gynecologists (ACOG) [17–19], the classification of hypertensive disorders in pregnancy is as follows:

Hypertensive disease induced by pregnancy:

- Preeclampsia
  - Mild
  - Severe
- Eclampsia

Chronic hypertension before pregnancy (any etiology).

Chronic hypertension and pregnancy-induced hypertension:

- Preeclampsia
- Eclampsia

6.1 Pregnancy-induced hypertensive disorders

Pregnancy-induced hypertension (PIH) is defined as the disorder that develops during gestation, delivery, or postpartum, characterized by elevated blood pressure values ≥140/90 mm Hg, accompanied by signs and symptoms allowing it to be classified according to its severity.

6.2 Preeclampsia

Preeclampsia refers to the presence of hypertension with values above those previously mentioned and proteinuria, in a pregnant female after the 20th week, after excluding hydatidiform mole or hydrops fetalis.

Arterial hypertension (AH) is diagnosed if the obtained values exceed, on two separate occasions and with at least 6 hours between each measurement, 140/90 mm Hg or if there is an increase in systolic arterial pressure (SAP) of at least 30 mm Hg or an increase in diastolic arterial pressure (DAP) of at least 15 mm Hg.

Proteinuria can be diagnosed with a urine test strip but must be confirmed with a quantitative method (urine sample or 24-hour urine).

Proteinuria must be above >300 mg in the urine collected in 24 hours or the protein: creatinine ratio must be ≥0.3.

6.3 Severe preeclampsia

Severe preeclampsia is defined as a systolic BP ≥160 mm Hg and/or a diastolic BP ≥110 mm Hg, if detected on two separate occasions, with a 6-hour difference, with proteinuria greater than 5 g/24 hours and no previous history of arterial hypertension, diabetic nephropathy, or renal disease.

HELLP syndrome is the acronym of hemolysis, elevated liver enzyme levels, and low platelet levels; this syndrome occurs in 10–20% of women with preeclampsia or eclampsia and is considered a severe form of the disease since it leads to increased mortality.

6.4 Eclampsia

It is defined as the presence of seizures or coma in a patient with preeclampsia, and that cannot be explained by another underlying cause.

6.5 Chronic hypertension

BP > 140/90 mm Hg before pregnancy or the same obtained values on two separate occasions prior to week 20 of gestation or persistent hypertension after the sixth week postpartum.

6.6 Chronic hypertension and pregnancy-induced hypertension

It is defined as an increase in SAP greater than 30 mm Hg or an increase greater than 15 mm Hg in DAP, on two separate occasions, prior to week 20 of gestation, initial proteinuria, and generalized edema.

6.7 Transient or late arterial hypertension

This is hypertension developing in the postpartum without previous preeclampsia; values return to baseline after the tenth day postpartum.

7. Physiopathology of preeclampsia

Preeclampsia is a syndrome that compromises all maternal organs and systems.

The etiology of hypertensive disorders in pregnancy remains to be identified since searching for their origin has led to an infinite number of hypotheses that encompass practically all maternal and fetal organs.

Its physiopathology has not been totally elucidated, and it is no different in the *adolescent* patient than in the rest of the affected population.

Several factors have been implicated in its physiopathology such as oxidative stress, the inflammatory response, abnormal circulatory adaptation, metabolic abnormalities, and even abnormalities in placental development, releasing circulating factors that interfere with vascular endothelial growth factor (VEGF) and placental growth factor (PGF).

Aside from the physiopathogenic factors to be discussed ahead, many factors predisposing to preeclampsia have been reported, such as extreme ages (very young or older), nulliparity, obesity, smoking, a history of preeclampsia in a previous pregnancy, etc. Other less studied factors include some infections, asthma, and the time period between pregnancies [20].

7.1 Systemic endothelial dysfunction

Endothelial abnormality leads to dysfunction in the control of muscle tone in blood vessels which, in turn, may cause hypertension, edema due to increased permeability, and also proteinuria.

Likewise, the abnormal expression of procoagulant factors by the endothelium favors the development of coagulopathy. All of these abnormalities injure target organs such as the kidney, the liver, the central nervous system, and the placenta. Women with previous vascular disease are at greater risk of developing preeclampsia, quite possibly a result of preestablished vascular injury.

7.1.1 First phase: abnormal placentation and placental ischemia

The placenta plays a pivotal role in the development of preeclampsia since it only develops in its presence and symptoms rapidly remit after delivery.

During the development of normal placentation, the cytotrophoblast invades the spiral arteries which leads to their remodeling; they will have low resistance and high elasticity or capacitance. This cytotrophoblastic vascular invasion not only affects the most superficial layers but reaches the muscle tunica. Trophoblast penetration has also been reported as incomplete and is not invasive in patients with preeclampsia; after complete remodeling of the spiral arteries, placental perfusion decreases. Although remodeling of the spiral arteries begins in the first trimester, it is not considered complete until weeks 18–20 of gestation.

Recently, great importance has been attributed to angiogenesis because of molecules such as VEGF, angiopectin, and other proteins in the ephrin family. The invasive trophoblast expresses VEGF, P1GF, VEGF, and their respective receptors. Likewise, in in vitro studies in which these signals were blocked, integrin alpha-1, a marker of pseudovasculogenesis, decreased alarmingly.

7.1.2 Second phase: systemic endothelial dysfunction

As previously mentioned, systemic endothelial dysfunction in these patients may explain all or almost all of the clinical signs, such as hypertension, proteinuria, or abnormalities in target organs such as the liver, the central nervous system, or the kidneys.

Among the various findings upholding this theory, we can mention the following:

- The plasma elevation in some biomarkers, such as fibronectin, factor VIII, and thrombomodulin, reflects endothelial cell injury in patients with preeclampsia.

- Vasodilation mediated by flow has also been reported in the vessels of women with preeclampsia, suggesting altered endothelial function.

- Decreased production of vasodilators such as prostacyclins or an increase in the production of angiotensin II also suggests endothelial injury.

- In these patients, renal biopsies show diffuse glomerular injury caused by glomerular endotheliosis.

- Likewise, the serum of women with preeclampsia has been shown to activate the endothelium in in vitro studies using endothelial cells from the umbilical veins.

An important factor for future consideration is that the increased concentrations of sFlt-1 generally precede, by 5 weeks, the development of clinical manifestations and appear to be most elevated in the initial phase of severe preeclampsia. However, neither PlGF nor VEGF, measured during gestation, appears to decrease prior to the onset of preeclampsia symptoms.

Most recently, decreases in urinary PIGF have been described before the development of preeclampsia. Some authors have speculated that sFlt-1 plays a beneficial role in fetal circulation and that preeclampsia is a reflection of a maladaptive effect of its release into the maternal circulation. Thus, in the setting of some spiral arterioles with increased resistance, vasoconstriction of the nonplacental maternal circulation would theoretically increase the cardiac output percentage reaching the

placental sub-circulation. Although most cases of preeclampsia are sporadic, some authors suggest that genetics play a role in the development of this entity based on a series of findings:

- Primiparous women with a positive family history of preeclampsia have a two- to fivefold greater risk of developing preeclampsia than primiparous women without this history.

- In sisters with preeclampsia, the genetic imprint plays a major role in the development of the disease.

- Studies in women pregnant with males that were the result of a gestation with preeclampsia have greater probabilities of developing the disease in their pregnancies.

- Women who became pregnant with men whose previous partner had preeclampsia have greater probabilities of developing the disease if gestation with the previous partner was normotensive [20].

7.2 Preeclampsia-eclampsia in adolescents

Traditional references accept that the risk of preeclampsia-eclampsia in adolescents is twice that in the adult population. However, literature reviews are contradictory; while one group of authors refer to an increased risk of preeclampsia in adolescents [21], others detect no differences between these and adult pregnant women, as shown in **Table 2**.

Several studies and meta-analysis [22] detect a significant difference between pregnant adolescents and adult women, with up to 20% more preeclampsia events in pregnant adolescents than in adult women [23]; they actually report a greater risk of preeclampsia-eclapmsia in the group of adolescents between the ages of 13 and 16 when compared with women between the ages of 20 and 34, OR 2.97 (95%CI 1.62–5.42) [24], as well as lower frequencies of preeclampsia in adolescents (5%) vs. 1.5% in the adult group, and the difference was statistically significant, OR 3.66 (95%CI 1.67–7.72) [25].

Therefore, the frequency of preeclampsia in adolescents is currently different according to the studied population as a result of many factors such as prenatal care and body weight changes in pregnancy [26–29].

There is significant bias in observational studies that depend on the type of design and case identification; in these cases, no significant association is found suggesting an increased frequency of preeclampsia in pregnant adolescents, RR 0.88 (95%CI 95% 0.73–1.23). On the contrary, if we take into account follow-up studies, the pregnant adolescent is 23% less likely to develop preeclampsia when compared with pregnant women in other age ranges, RR 0.77 (95%CI 0.64–0.92).

Therefore, the available evidence suggests that adolescence is not a determining factor in the development of preeclampsia and eclampsia, but geographic area does appear to be an additional factor.

In a systematic review [30] describing pregnant adolescents that participated in integral prenatal care programs, they had a lower risk of developing pregnancy-induced hypertension (RR 0.59) than those following traditional prenatal care programs. This means that integral prenatal care programs focused on adolescents decrease the frequency of pregnancy-induced pregnancy by 41% [10].

Due to the heterogeneous presentations of preeclampsia in adolescents from different regions, age is probably not a determining factor but is rather associated to

Parra [38]	Under 19 year 13.5%		The prevalence is greater than 17–19 years before the age of 17 p < 0.001		
Zeck et al. [25]	5% in adolescents vs 1.5% in adults		OR 3.666 (1.627, 7.723)		
Tebeu et al. [24]	Risk of 13–16 vs 20–34 years		OR 2.974 (1.627, 5.427)		
Gronvik [22]	Meta-analysis		OR 3.52 (2.26, 5.48)		
Bostanci et al. [21]		Age of resolution P < 0.001	Number of appointments P < 0.001	Prevalence of preeclampsia P < 0.001	aOR p < 0.001
	Early adolescent 11–13 years	36 weeks	3.0	4.8%	1.14 (0.031, 0.63)
	Middle adolescent 14–16 years	37 weeks	4.78		
	Late adolescent 17–19 years	38 weeks	5.54	2.7%	0.44 (0.31, 0.63)
	Adult			5.9%	2.1 (1.51, 3.01)
Garmer [39]	Hypertensive disorders related with pregnancy P = 0.99				
	<20 years		20–34 years		
	8.7%		9.2%		
Azevedo et al. [6]	Hypertensive disorders in pregnancy		10%		

Table 2.
Prevalences of preeclampsia in adolescents.

other variables, such as excess weight, excessive weight gain during pregnancy, and inadequate prenatal care, among others.

Two factors warrant analysis in terms of the development of preeclampsia in pregnant adolescents; the first is its association with obesity. Obesity is currently considered an epidemic in which one of every four women in reproductive age is obese and over half of women between the ages of 20 and 39 have excess weight or obesity. Obesity has been reported to increase the risk of adverse maternofetal outcomes due to its association to and development of comorbidities such as gestational diabetes, fetal macrosomia, an increase in cesarean sections, and preeclampsia. Its genesis is the increase in oxidative stress; an increase in circulating pro-inflammatory biomarkers such as C-reactive protein, tumor necrosis factor-alpha, interleukin-6, and interleukin-8; the presence of dyslipidemia, insulin resistance, and abnormalities of endothelial function, all playing a role in the pathogenesis of preeclampsia [31].

Another factor leading to a poor maternal-perinatal outcome and that also increases the risk of preeclampsia is an inadequate nutrient intake, including calcium, zinc, vitamin C, vitamin E, and essential fatty acids. This factor has a doubly negative effect and results from the fact that adolescents are still growing, and during pregnancy, nutrients compete with the fetus for development, therefore leading to nutritional deficiencies [31].

As to the studies recommended in pregnant adolescents to screen for preeclampsia, there are currently no specific tests that can help establish a preclinical diagnosis, so the methods used in the general population are the only option:

1. First trimester combined test: markers (weight, blood pressure), ultrasound markers (uterine Doppler), and biochemical markers (PAPP-A, sFlt-1/PIGF).

2. Prophylactic aspirin before the 16th week of gestation in patients at greatest risk, based on an abnormal uterine Doppler in the first trimester.

3. In the second trimester, a uterine Doppler combined with other variables has a high negative predictive value.

Since there is no defined pattern in adolescent preeclampsia, its diagnosis and treatment must be the same as in the general population.

Therefore, the following recommendations have been established [32]:

In pregnant adolescents, screening, diagnostic, and therapeutic interventions should be similar to those applied in the rest of the population. **Level of evidence, moderate. Recommendation, strong.**

Care of pregnant adolescents must be provided in *ex profeso* clinics with complete, integral, and multidisciplinary programs to decrease maternal and perinatal risks, including pregnancy-induced hypertension. **Level of evidence, moderate. Recommendation, strong.**

Although there is no evidence on the beneficial effect of interventions used to curb weight gain during pregnancy, offering a medical evaluation and nutritional counseling is a good practice to recommend. **Level of evidence, low. Recommendation, strong.**

Every adolescent clinic must establish the incidence of preeclampsia and eclampsia in its patient population and determine which factors are associated to their development. **Level of evidence, low. Recommendation, strong.**

According to the diagnostic situation of preeclampsia and eclampsia in each adolescent clinic, screening, preventive, early detection, and therapeutic programs must be designed. **Level of evidence, moderate. Recommendation, strong.**

In adolescents with preeclampsia, the disease is generally manifested in the latter part of gestation, close to full-term delivery, so good prenatal care fosters a timely diagnosis of hypertensive disease in earlier stages and improves maternal and fetal outcomes. In the case of the induction of delivery, outcomes will also be improved by decreasing the need for cesarean delivery even in the cases of severe preeclampsia: the neonatal outcome also improves since age is not an influencing factor but disease severity is.

8. Long-term fetal complications in hypertensive mothers

Follow-up of the offspring of mothers with any pregnancy-associated hypertensive state [33] has shown that by age 7, their systolic blood pressure is increased although within normal parameters—SBP of 104 mm Hg (95%CI 101–106 vs. SBP 99 mm Hg, 95%CI 99–100, p = 0.001)—and this cardiometabolic injury is evident from the age of 2 years.

This abnormality is only observed in full-term births and not in premature offspring; a posited explanation is that the stress caused by preterm delivery protects the fetus from sequelae.

Another consequence observed in the offspring of hypertensive mothers is the development of hypertension and cerebral vascular disease when these children reach adulthood. Also, their risk of developing hypertension increases if their body mass index is elevated, and this has been observed since the ages of 4–10, even if the mother had hypertension with no proteinuria; the presence of elevated liver enzymes or thrombocytopenia has also been associated with hypertension in young offspring [34, 35].

Fetal changes due to maternal hypertension have been associated with genetic and environmental mechanisms that condition modifications in fetal programming [36]. Among the effects caused by preeclampsia, one hypothesis suggests that superficial invasion of spiral arteries leads to fetal malnutrition. Changes in fetal programming may also involve abnormalities in the inflammatory response and endothelial dysfunction associated with preeclampsia.

An important point in this description of long-term sequelae in the offspring of pregnant adolescents is that they are not modified by age.

9. Points to remember

- Adolescent pregnancy is a very important biopsychosocial event, a public health issue, and a continuous social reflection.

- Hypertensive problem associated with pregnancy including the period of adolescence is another public health issue; its prevalence in the worldwide is around 10% of all pregnancies.

- Preeclampsia in adolescents refers to a relatively common hypertensive disorder during pregnancy that develops progressively. Its cause remains unknown, and it frequently leads to severe maternal and perinatal complications.

- The incidence is broad since it depends on various aspects such as geographic location, race, nutritional or immunological factors, and associated comorbidities.

- Traditional references accept that the risk of preeclampsia-eclampsia in adolescents is twice that in the adult population, but in recent studies including meta-analysis, the presence of preeclampsia in pregnant adolescents varies from a 20% more frequent and specifically in the group of 13–16 years (OR 2.97, 95% CI 1.62–5.42) to 23% less. Therefore, the available evidence suggests that adolescence is not a determining factor in the development of preeclampsia and eclampsia, but geographic area does appear to be an additional factor.

- Two other determinants of risk factor for developing preeclampsia in pregnant adolescent are obesity and inadequate nutrient intake (calcium, zinc, vitamin C, vitamin E, and essential fatty acids).

- In adolescents with preeclampsia, the disease is generally manifested in the latter part of gestation, close to full-term delivery.

- A good prenatal care fosters timely diagnosis of hypertensive disease in earlier stages and improves maternal and fetal outcomes.

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