



## **GESTATIONAL HYPERTENSION: ABOUT 59 CASES**

**M. Sebti\*, M. C Fourati, F. Zidane, PR M. Yousfi, PR S. Bargach**

Department of Gynaecology-Obstetrics, Cancerology and High Risk Pregnancies Unit, Maternity Souissi, Hospital Ibn Sina, Rabat.

**\*Corresponding Author: M. Sebti**

Department of Gynaecology-Obstetrics, Cancerology and High Risk Pregnancies Unit, Maternity Souissi, Hospital Ibn Sina, Rabat.

**Article Received on 03/03/2020**

**Article Revised on 24/03/2020**

**Article Accepted on 14/04/2020**

### **I-INTRODUCTION**

Gestational hypertension (GH) is a heterogeneous group of pathological conditions whose common denominator is an increase in blood pressure with a systolic pressure greater than or equal to 140mm-Hg and/or a diastolic pressure greater than or equal to 90mm-Hg, obtained on two occasions separated by at least 4 hours. It is a major health problem of global proportions, firstly because of its frequency, which can reach up to 15% of pregnancies, and secondly because of the high maternal and foetal morbidity and mortality for which it is responsible. The pathophysiology of this condition remains poorly defined despite the numerous studies on it.

Most of the current recommendations for the treatment of these disorders are based on expert opinion and observational studies, with a lack of evidence from randomized controlled trials. The overall strategy in the treatment of hypertension in pregnancy is to prevent maternal cerebrovascular and cardiac complications, while preserving the uteroplacental and fetal circulation and limiting medication toxicity to the fetus.

Close management and rigorous multidisciplinary follow-up involving the cardiologist and the obstetrician are therefore essential to limit complications, hence the importance of early management and early detection of gestational high blood pressure.

### **II-MATERIAL AND METHODS**

This is a descriptive retrospective study conducted at the obstetrical gynaecology department of high-risk pregnancy within the hospital of maternity SOUSSI in RABAT, MOROCCO, involving 59 women with pregnant arterial hypertension over 586 women consulted.

The study lasted 2 years, from January 2018 to January 2020.

We included pregnant women with systolic blood pressure greater than or equal to 140mm-Hg and/or diastolic blood pressure greater than or equal to 90mm-Hg, with or without proteinuria, and those with a

complication related to gestational hypertension at the time of the consultation.

We have adopted the American College of Obstetricians and Gynecologists (ACOG 2002) criteria for defining preeclampsia (BP Obstétricien 140/90 mm-Hg + proteinuria greater than or equal to 300 mg/24h or greater than or equal to 2 crosses at the labstix) and its severity criteria.

Are excluded from this study the lost of sight and patients whose follow-up and delivery were not performed in our department.

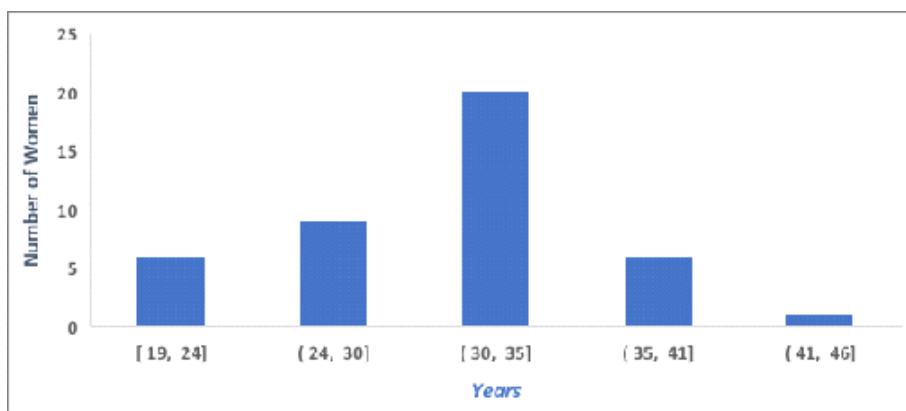
Each of these patients received rigorous and methodical follow-up with multidisciplinary anesthesiologic-cardiologic-obstetric follow-up.

### **III-RESULTS**

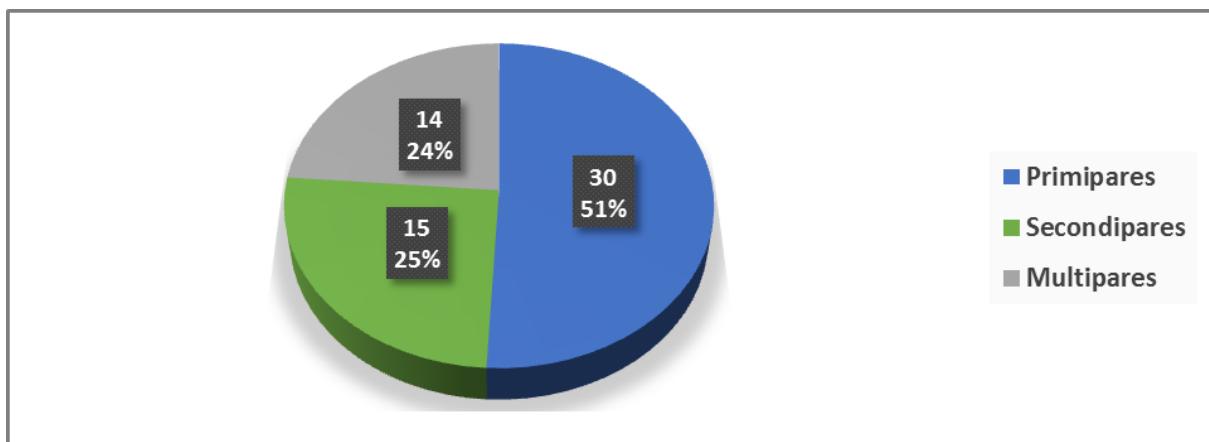
#### **A/ Epidemiology**

A total of 59 patients have been studied out of 586 consultations, i.e. a prevalence in the order of 10%. About patients characteristics, our study has shown an average age of 29 years with a rate of 51% of women with an age inferior to 30 years.

Patients aged between 20 and 30 years seem to be the most exposed to develop hypertension during pregnancy with a frequency rate of 47% in our study. On the other hand, patients over 40 years of age seem to be the least exposed since they represent 9% of all parturients.

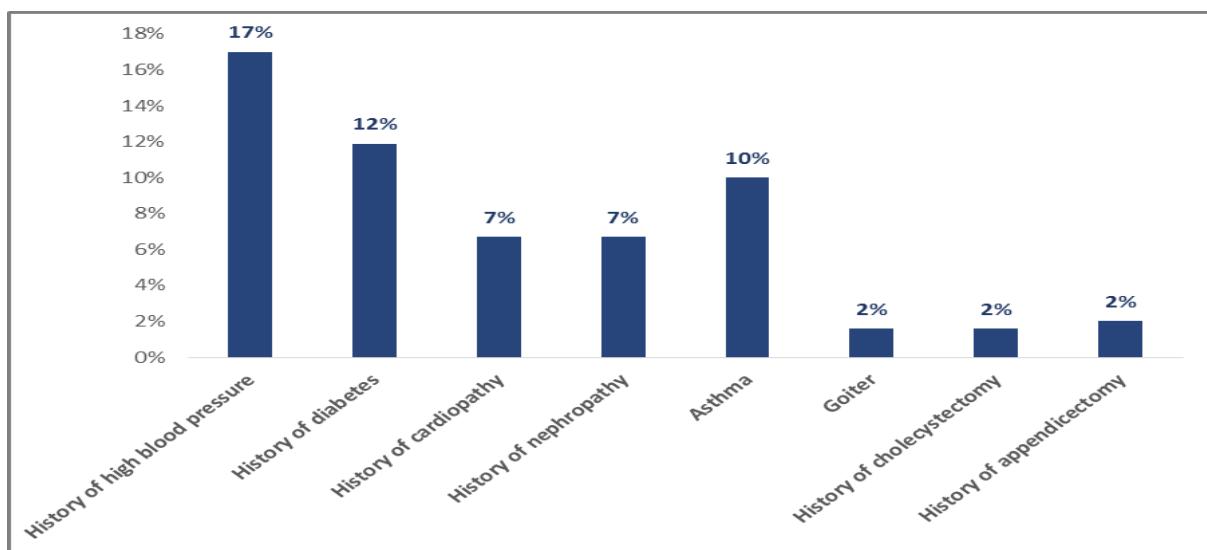
**Diagram 1: Age distribution of our study population.**

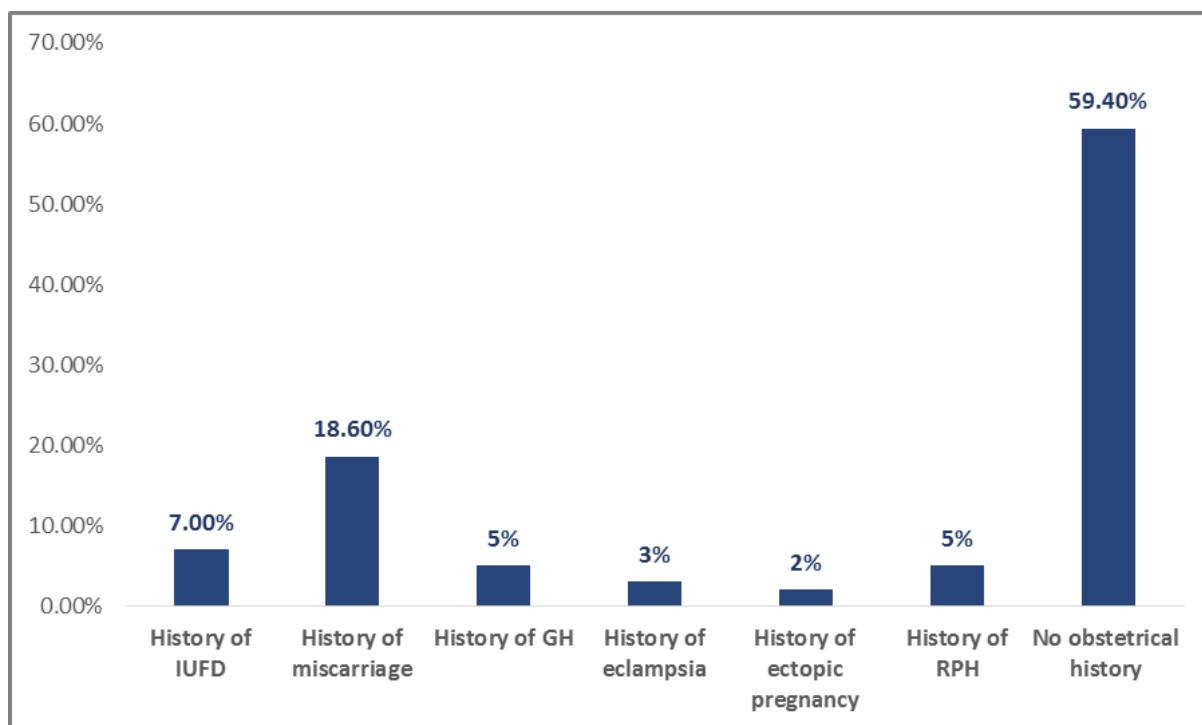
Concerning the parity, we've found that 30 were primiparous (51%), 15 pauciparous (25%) and 14 multiparous (24%).

**Diagram 2: Distribution of GH in women parity.**

Regarding to the weight, 27 women had a body mass index (BMI) superior to 30 Kg /m<sup>2</sup> (45,7%) and the average BMI was 28,3 Kg/m<sup>2</sup>.

We've noticed in 38,9% multiple risk factors of GH as shown on diagrams 3 and 4.

**Diagram 3: Distribution of GH's medical and surgical risks factors in our study population.**



**Diagram 4: Distribution of GH's obstetrical risks factors in our study population.**

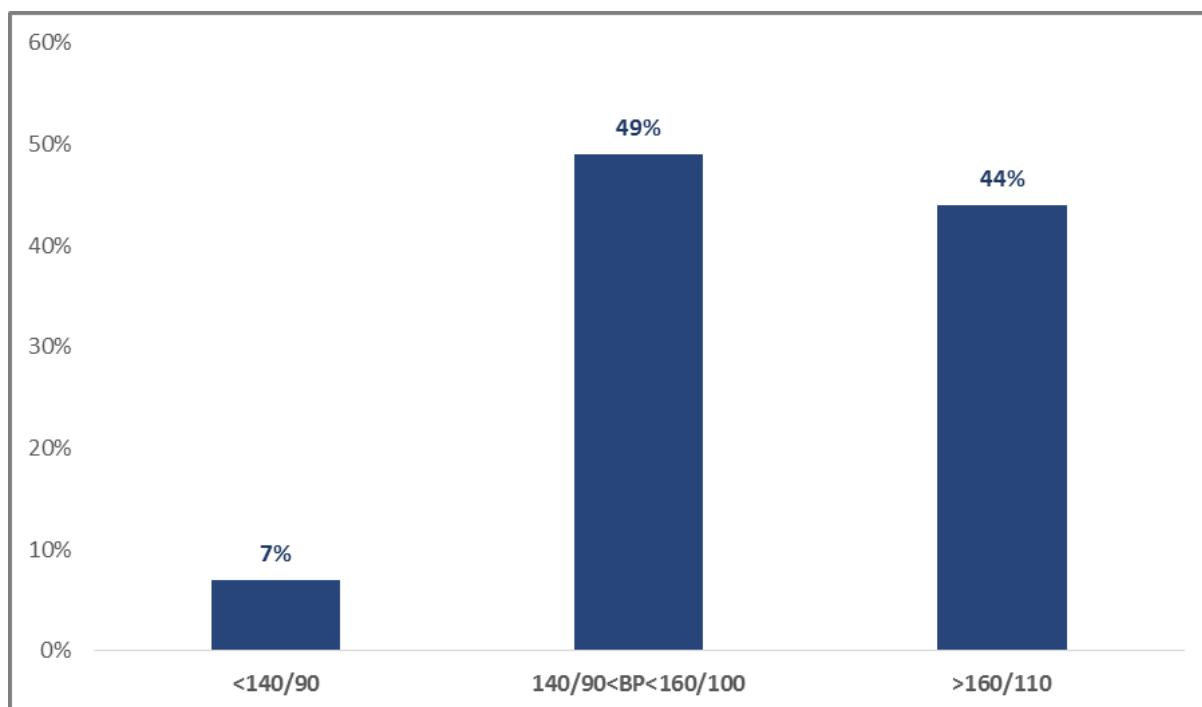
#### B/ Clinical and obstetrical profil

In all patients, blood pressure figures were recorded, and the existence of oedema and the search for proteinuria (urine strips) were mentioned in all observations.

Patients' blood pressure ranged from 120 to 200 mm-Hg for systolic blood pressure (SBP) and from 60 to 160

mm-Hg for diastolic blood pressure. This pressure was measured at patient's first consultation and monitored after 15 to 30 minutes of rest.

The diagram 5 shows the distribution of patients by blood pressure controlled at the first consultation.



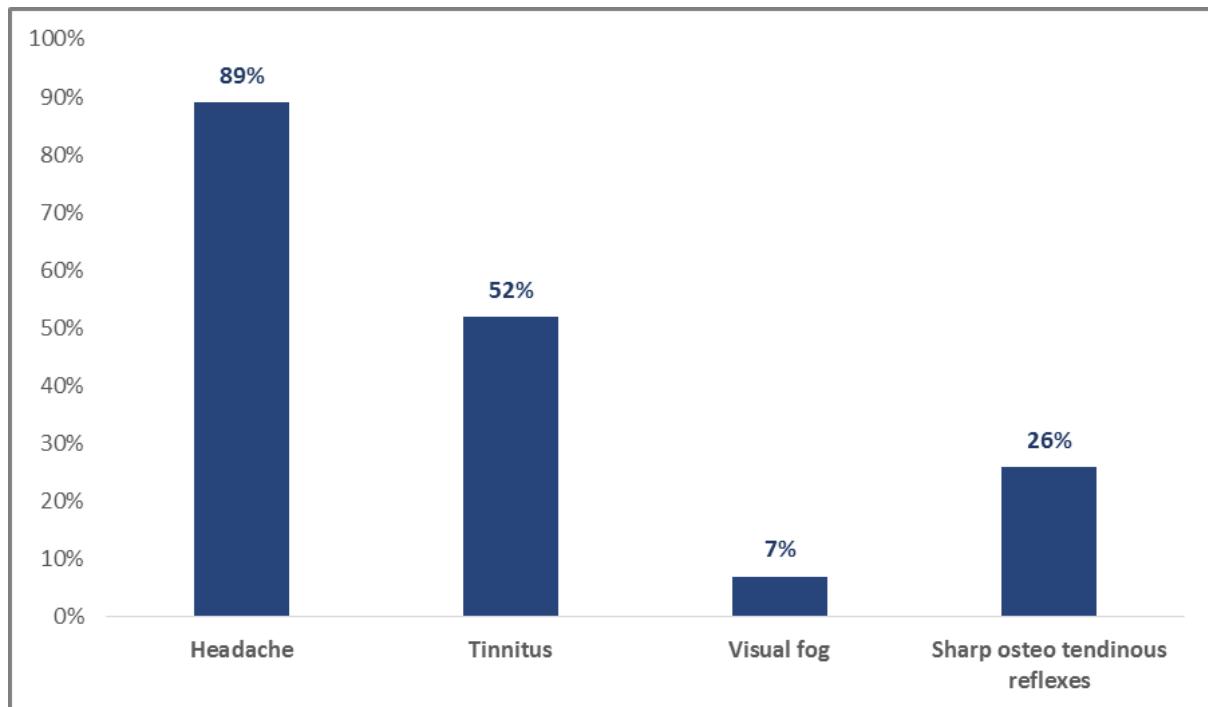
**Diagram 5: Distribution of patients by blood pressure during the first consultation.**

Regarding the presence of oedemas, we have found 30 parturients, i.e. 58.3%, with a predominance in the lower limbs.

The search for proteinuria should be done in every pregnant woman using of urinary strips (Labstix), and all our patients benefited from this examination at the 22th week of gestational

age. Our study found 54,2% of the women positive and 45,8% were negative.

27 patients showed neurosensory signs during follow-up. Headache was the most common major sign (89%), followed by tinnitus (52%) and visual fog (7%). It should be noted that the signs were either isolated or associated with each other.



**Diagram 6: Patient Distribution by Neurosensory Signs osteo tendinous reflexes.**

Vomiting and the epigastric bar were considered digestive signs. These signs were present in 9 patients or 15%.

As for metrorrhagia, they were observed mainly during the third trimester and were present in 4 cases, i.e. 7%, most of them presenting with a retro-placental hematoma.

The follow-up and impact of this hypertension was rigorously monitored through a paraclinical biological and radiological assessment.

This assessment was carried out in all patients during their prenatal follow-up.

Biologically, the patients were given a CBC, a renal and hepatic check-up, uricemia and 24-hour proteinuria, in addition to a haemostasis check-up.

The results of these tests are summarized in the following table:

**Table 1: Distribution of patients according to the results of the bioassessment.**

BIOLOGICAL TEST	NUMBER OF PATIENTS		FQ %
<i>CBC :</i>	-----		-----
<i>Hb</i> <i>(g/dL)</i>	<7	7	12%
	7-11	22	37.2%
	>11	30	50.8%
<i>Platelets</i> <i>(e/mm3)</i>	<50.000	1	2%
	50-100.000	5	9%
	100-150.000	14	24%
	>150.000	39	65%
RENAL BALANCE	NORMAL	52	88%
	ABNORMAL	7	12%
HEPATIC BALANCE (ASAT/ALAT)	NORMAL	48	81%
	ABNORMAL	11	19%
HEMOSTASIS BALANCE	NORMAL	54	92%
	ABNORMAL	5	8%
24 HOUR PROTEINURIA	+	21	36%
	-	38	64%

The main biological abnormalities found during our study are therefore 24-hour positive proteinuria, hepatic disorders and in particular transaminases, a slight to moderate decrease in blood platelets as well as anaemia in 50% of cases.

During follow-up, each patient received one dating ultrasound, two morphological ultrasounds at the 22th week of gestational age and the 32th , and a Doppler was relaid in 13 patients with abnormalities during their evolution.

Obstetrical ultrasound revealed intrauterine growth retardation in 8 cases and morphological abnormalities in 1 case.

Doppler was pathological in 4 parturients, i.e. 7%.

In terms of therapeutic management, hygieno-dietary measures and antihypertensive monotherapy have been

the most widely used. Alpha methyl dopa and nifedipine were the most commonly used during our consultations, with methyl dopa predominating in 75% of patients.

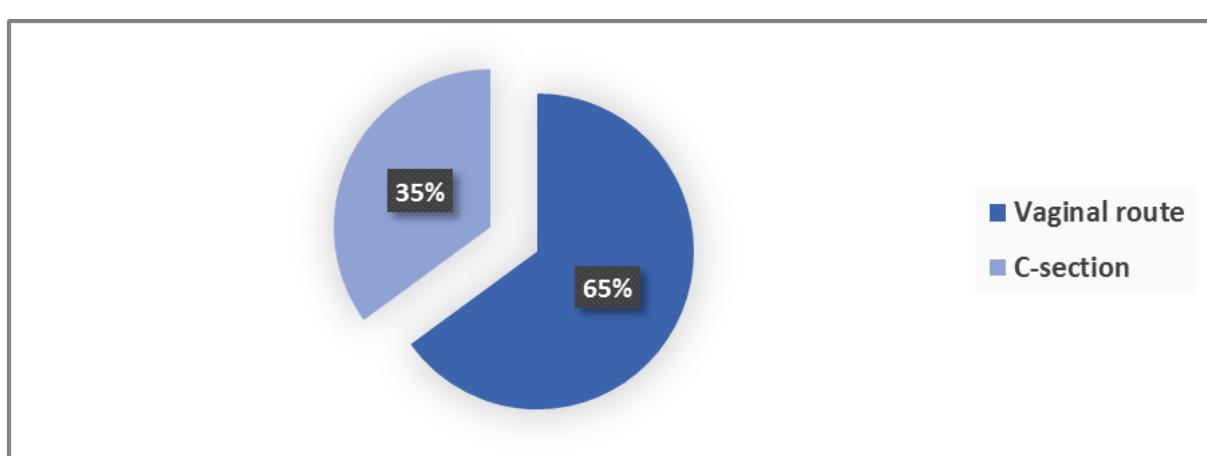
Dual therapy was used in 12 patients (20.3%) at the beginning of the third trimester after difficulties in controlling blood pressure levels with monotherapy.

In our study, the blood pressure control was obtained in 48 women (81%).

39 patients gave birth by vaginal route, i.e. 65%, while Caesarean section was performed in 35% of cases (20 patients).

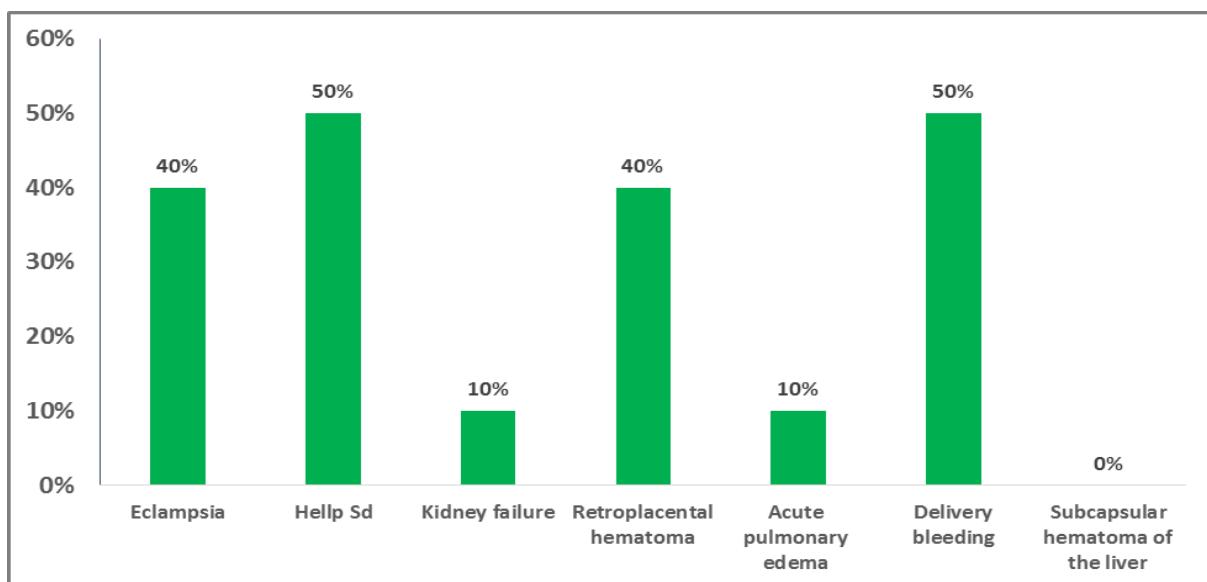
The upper route was indicated in 12 patients.

Secondary Caesarean section was performed in 8 patients for labour abnormalities, fetal distress or failure of induction.

**Diagram 7: Distribution of patients according to the mode of delivery.**

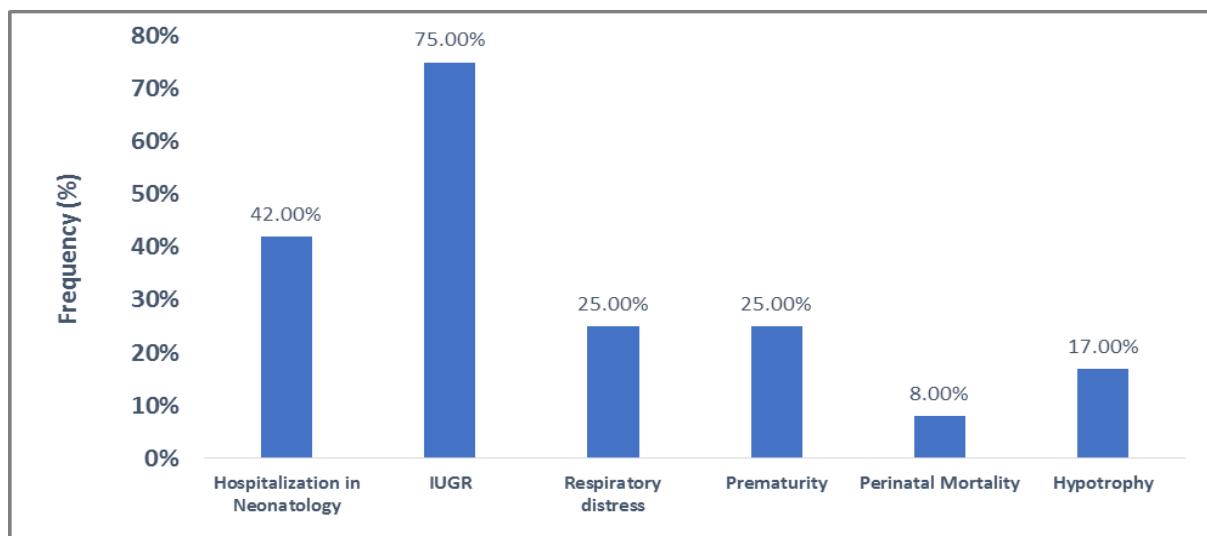
49 patients progressed well and presented no complications, i.e. 83.2% of all parturients.

10 parturients had at least one complication, i.e. 16.8% of all patients. They are distributed as follows:



**Diagram 8 : Distribution of maternal complications recorded in our study series**

Among our 59 births, 47 newborns had a favourable trend, i.e. 79.6%. 12 cases had presented at least one complication (20.4%) distributed as follows:



**Diagram 9: Distribution of neonatal complications in our series**

The average term of childbirth is 37.1 weeks of amenorrhea. The most common complication noted is the IUGR (9 cases). 5 newborns were transferred in the neonatology unit for intensive care.

Prematurity was noted in 3 cases(25%).

Respiratory distress (25%) and hypotrophy (17%) were noted too.

#### IV-DISCUSSION

##### 1-The prevalence

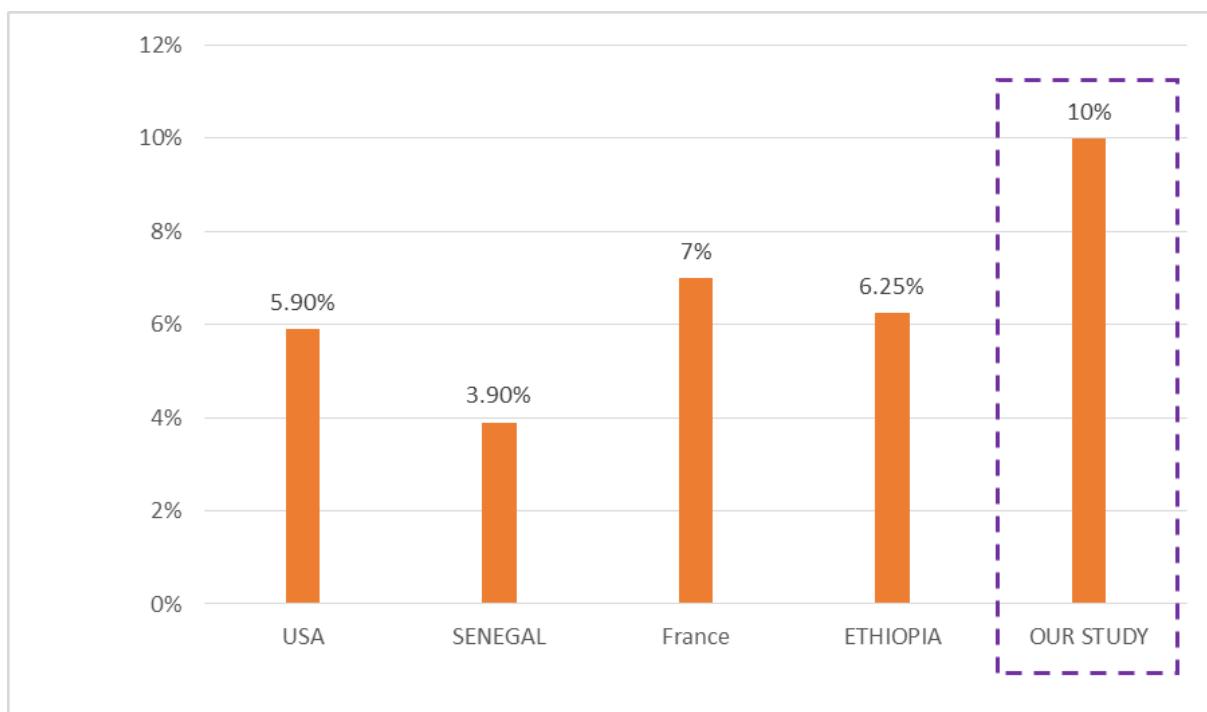
The prevalence of GH varies depending on the source from 5 to 15% of pregnancies.

It varies from one country to another and is believed to be closely linked to ethnic and socio-economic disparities in the different populations.

Thus, Zhang J. et al.<sup>[1]</sup> found a prevalence of 5.9% in the USA, M.Thiam.<sup>[2]</sup> 3.9% in Senegal, M.Beaufils.<sup>[3]</sup> 7% in France.

Our study finds a prevalence of 10%, a higher percentage than the majority of studies conducted.

A study conducted by F.Diallo has shown a high prevalence compared to other studies at 16%.<sup>[4]</sup>



**Diagram 10: Frequency of GH in different countries**

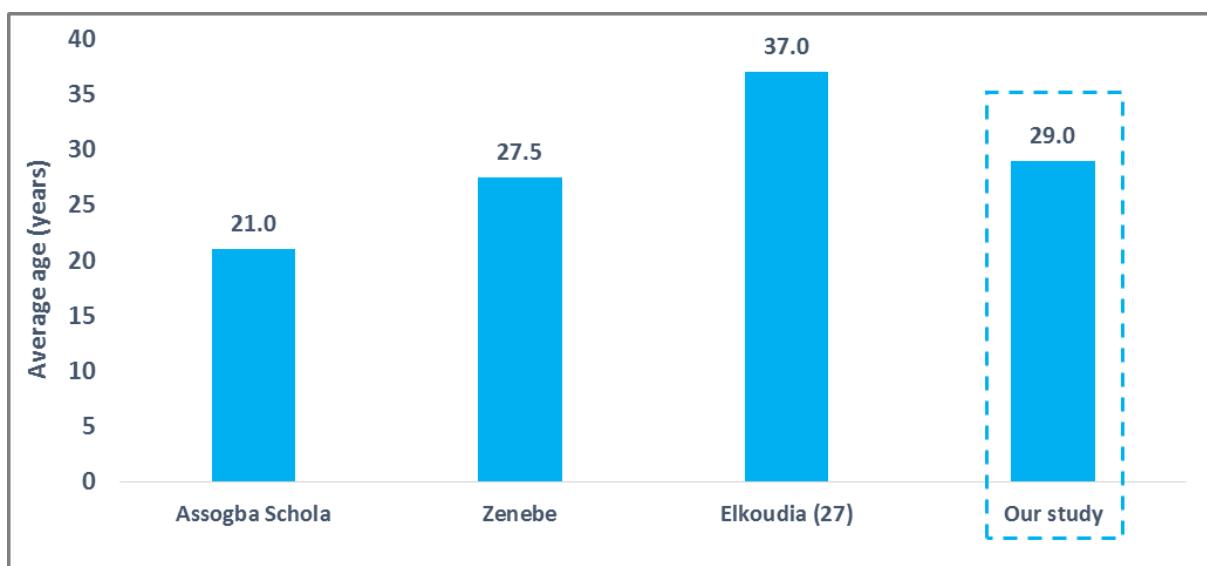
## 2-Patients characteristics

The age of our patients ranges from 17 to 42 years, with a predominance of women under the age of 30 (51%).

The average age is 29, this is in line with the study put forward by Assogba Schola C.<sup>[5]</sup> Indeed, the age group most affected by the disease is 13-29 years in more than

70% of cases, of which 32.9% of parturients were aged 20-24 years.

Similarly, Zenebe W. noted a high frequency in young women with a rate of 52.5% of patients aged 25-30 years.<sup>[6]</sup>

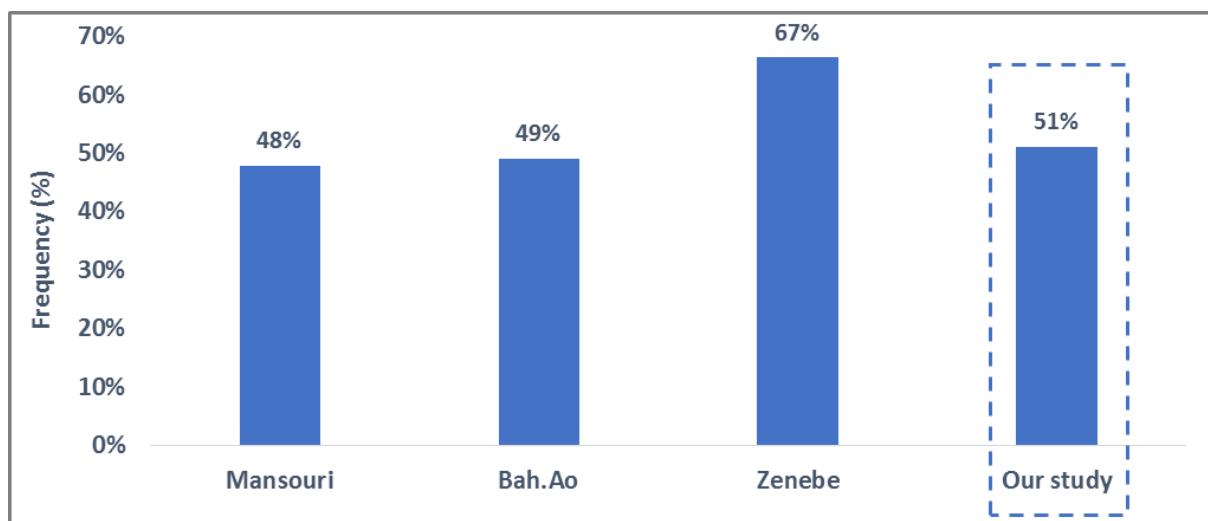


**Diagram 11: Distribution of average age by series.**

Some authors consider multiparity greater than or equal to 3 as a risk factor for the GH.<sup>[7]</sup> In our study, 51% of the patients are primipares, which is consistent with the results at the national level, M.Elkoudia,<sup>[8]</sup> Moukaddime,<sup>[9]</sup> Elfalaki,<sup>[10]</sup> and Mansouri,<sup>[11]</sup> report primiparity rates of 48.06%, 52.44%, 41.01% and 47.89% respectively.

Internationally, W.Zenebe et al,<sup>[12]</sup> and Bah.Ao,<sup>[13]</sup> reported rates of 66.5% and 49.11% respectively of primiparous patients.

According to numerous studies, primiparity is a considerable risk factor in the development of the disease. Duckitt and Harington,<sup>[14]</sup> attribute a relative risk of 2.91 to it.



**Diagram 12: Frequency of primiparous patients by series**

### 3-Obesity and risk factors of PAH

Duckitt and Harrington,<sup>[14]</sup> found a relative risk of developing the disease of 2.47 in cases of obesity. According to a study carried out on more than 15,000 pregnancies in the United States (Boston), the risk of developing preeclampsia was multiplied by 2.1 in the event of a BMI > 30 kg/m<sup>2</sup>,<sup>[15]</sup>

This same observation has been reported by other authors, notably Rosenberg,<sup>[16]</sup> Frederick,<sup>[17]</sup> and Stone.JI,<sup>[18]</sup>

In our series, we noted that 45.7% had a BMI > 30 kg/m<sup>2</sup>.

This confirms the major role played by this factor in the development of GH and especially preeclampsia.

A field of GH-eclampsia was present in 8% of cases.

The literature incriminates a history of PAH, preeclampsia, IUFN or other complications of PAH in the risk of developing a second episode of the disease and especially its severe forms.

A history of preeclampsia has been shown to be the most significant risk factor in many studies, with a relative risk of 7.19,<sup>[3]</sup> and a recurrence rate of 14% in a study of 19960 women in Norway and 15% in Sweden,<sup>[19]</sup>

Zhong et al,<sup>[20]</sup> showed in their study that the risk of hypertension for this population during second pregnancy is 19%, 32% and 46% respectively. The study also showed that IUGR without hypertension in the first pregnancy doubles the risk of developing GH in subsequent pregnancies.

In our series, 38.9% of our patients had one or more obstetrical histories dominated by miscarriages and IUGD.

In the same vein, they noted that women who were even normotensive in their first pregnancies but who had

IUGR in their first births had a two-fold risk of developing hypertension in subsequent pregnancies.

The literature lists various personal medical histories likely to induce GH or even preeclampsia, the most important of which are represented by chronic hypertension,<sup>[7,20,21]</sup> diabetes,<sup>[7,14]</sup> anti-LP CA syndrome (RR=9.72).<sup>[14]</sup>

Kidney diseases are also incriminated with notably nephropathy and urinary tract infections.<sup>[22,23,24]</sup>

In our series, we found that 12% of patients had a vascular risk field dominated by chronic hypertension followed by diabetes.

The history of heart disease and nephropathy was found in a small percentage.

### 4-Clinical gravity signs

It is generally accepted that a systolic blood pressure greater than or equal to 160 mmHg or a diastolic blood pressure greater than or equal to 110 mmHg are elements of severity.<sup>[25,26,27,28]</sup>

In our series, systolic blood pressure greater than 160 mmHg and diastolic blood pressure at 110 mmHg in 44% of patients.

The neurosensory signs during pregnancy are very important in that they may be a precursor to the diagnosis of severe preeclampsia.<sup>[25,29]</sup>

For Ducarme,<sup>[30]</sup> 59-75% of convulsive attacks were preceded by prodromal symptoms and the most frequent symptom found in his study was headache (50-75%).

In our study, 45.7% of patients presented neurosensory signs on admission marked by headache in 89%. The osteo tendinous reflexes were sharp in 26% and digestive signs were present in 15%.

According to the American College of Obstetrician Gynecologist,<sup>[28]</sup> massive proteinuria, i.e. urine strips greater than or equal to 3 crosses twice, is considered a sign of severe preeclampsia.

In our series, massive proteinuria was recorded in 38.9% ( $\geq 3$  cross).

#### **5-Paraclinical gravity signs**

The 24H proteinuria is evaluated by the 24H urine assay, it shows glomerular lesions.<sup>[31]</sup> It is significant if greater than 0.3 g/24H, it is considered as a criterion of disease severity when it exceeds 3.5 g/24H.<sup>[32,33]</sup> It should be systematically investigated.

In our series, 24H proteinuria was performed in 36% of patients.

In our series, 7 parturients had impaired renal function, i.e. 12% of all patients receiving this test, and all had a severe form of the disease. This finding is supported by Moulin B, in his study where kidney acute failure occurs in 5-10% of cases in severe preeclampsia.<sup>[31]</sup>

The study of liver function tests is based mainly on transaminase assays, and is performed in the search for a HELLP syndrome, a serious complication of GH. In our series, this assessment was carried out in all the parturients and was found to be disturbed in 11 patients, 7 of whom had levels greater than or equal to 3 times normal. These results are higher than those found by Boukhchach F (6,25%) in 2008.<sup>[34]</sup>

During preeclampsia, the state of compensated excessive hypercoagulability is characterized by endothelial and platelet activation and excess thrombin formation.

For our patients, hemostasis exploration consists of a PT and/or APTT assay only. It was performed in 100% of our patients and was normal in 92% of cases.

During a normal pregnancy, hemoglobin, hematocrit and hemoglobin levels decrease due to physiological hemodilution. In our series, all the parturients benefited from this assessment. This assessment revealed 29 cases of anaemia, i.e. 49.7%, including 12% of cases of severe anaemia requiring transfusion and 20 cases of thrombocytopenia.

Ultrasound is an essential examination in the monitoring of any pregnancy, and even more so in the case of pregnant arterial hypertension. It allows the study of fetal vitality, morphology, and well-being thanks to Manning's biophysical score, and another essential element to be determined is fetal biometry. In our series, ultrasound was performed in 100% of our patients.

It allowed us to detect: 2 cases of MFIU, 4 cases of HRP, 3 cases of oligoamnios and 8 cases of hydramnios.

In our series, Doppler was performed in 21 parturients, i.e. 35.5%. It was normal in 17 patients, pathological in 4 cases.

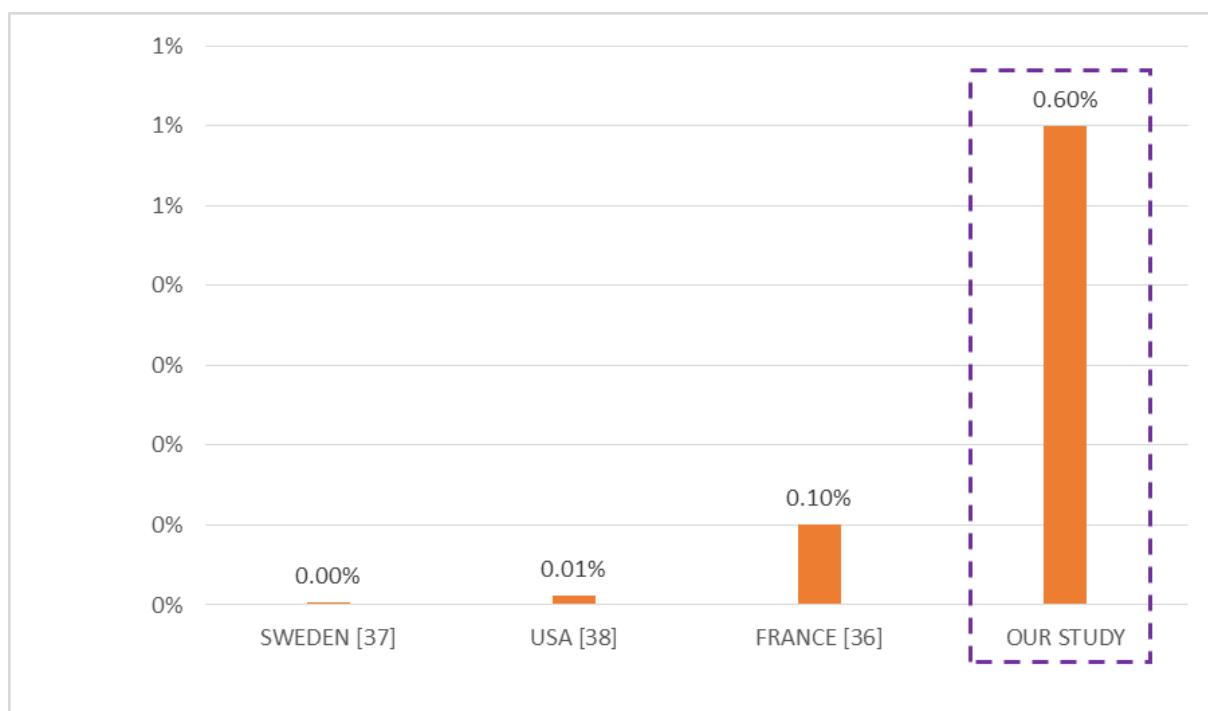
#### **6-Maternal complications**

##### **A/Eclampsia**

No deaths were noted in our study.

Eclampsia is estimated to be responsible for 50,000 maternal deaths annually worldwide, particularly in developing countries.<sup>[35]</sup>

The following table shows the frequency of eclampsia in selected international studies:



**Diagram 13: Frequency of eclampsia in selected international studies.**

The frequency of eclampsia still remains high in our context compared to developed countries and is low compared to an African country such as Senegal (0,8%).<sup>[39]</sup>

As for the developed countries, maternal mortality is on the other hand lower, it varies between 0 and 1.8%.<sup>[40]</sup>

In our series, eclampsia has an incidence of 0,6% and was responsible for 20% of the indications for the upper route.

In France, a study carried out on a small series of 16 cases showed that eclampsia occurred in 56% prepartum, 6% per-partum and 38% post partum.<sup>[30]</sup>

B.Sabiri finds that postpartum eclampsia accounts for 19% of his series of 305 eclamptics. The mean time to onset of the attack in relation to delivery was 36 hours with extremes ranging from 15 minutes to 2 months.<sup>[41]</sup>

In our study, we noted 75% prepartum eclampsia and 25% postpartum eclampsia.

#### **B/ Retroplacental hematoma:**

According to a study by Tbieba.B,<sup>[42]</sup> the occurrence of RPH in the context of GH is 31.1%.

In our series, a frequency of 7% was recorded.

#### **C/Hellp Sd:**

The HELLP syndrome complicates preeclampsia and eclampsia in 4-12% and 30-50% of cases respectively.<sup>[43]</sup>

In our series, there were 5 cases of HELLP Syndrome or

50%. 3 cases complicated eclampsia and 2 cases complicated GH.

#### **D/Other complications:**

The acute kidney failure was found in 10% in our study which is near El koudia's study (5,82%).<sup>[8]</sup> The pulmonary acute oedema (PAO) was diagnosed in 10% in our complicated parturients. According to Sibai et al, PAO complicates 2-5% of severe preeclampsia.<sup>[44]</sup> According to Matthys, PAO is the second most common complication in postpartum preeclamptic women with an incidence of 5.9%.<sup>[45]</sup> In a French series, PAO complicated 1.6% of patients.<sup>[46]</sup>

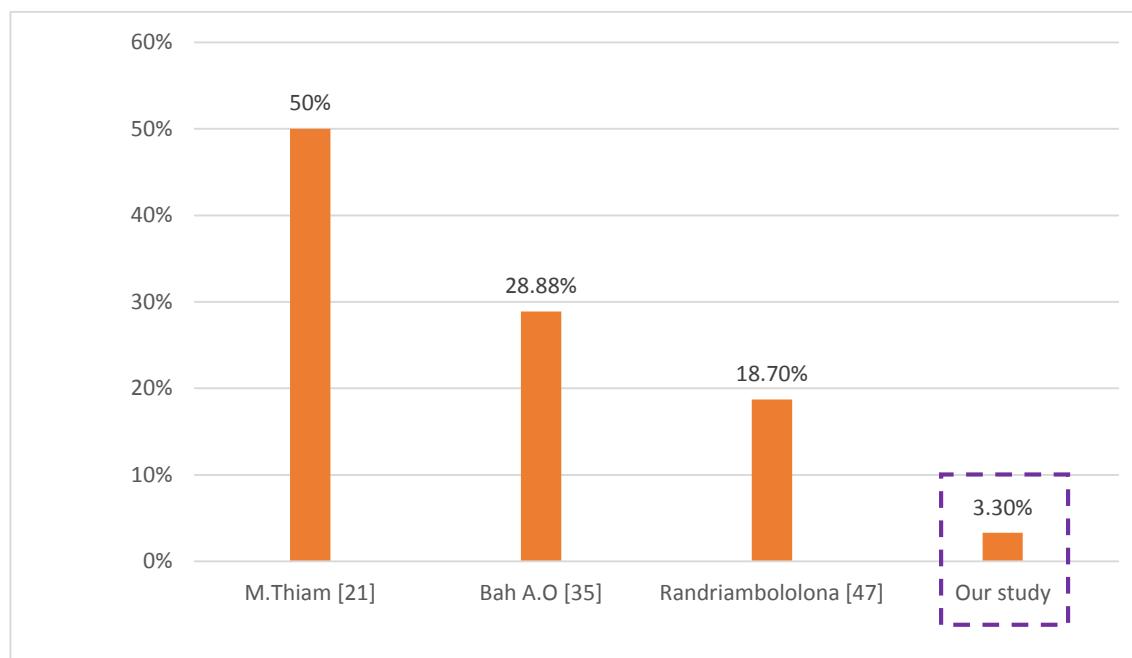
The deliverance bleeding represents 50% of the complications and no case of subcapsular hematoma of the liver was noted.

#### **7- Foetal complications**

##### *A/ Perinatal mortality*

In our series, in utero fetal death (IUFD) accounts for 1,7% (1 case), while neonatal mortality is around 3.3% (2 cases).

Our results are better than the other studies, thanks to the early management of pregnant women with GH, as well as of newborns.



**Diagram 14: Incidence of perinatal mortality due to pregnant high blood pressure in selected national and international studies.**

**B/Prematurity**

In the United States, an estimated 15% of preterm births in the United States are due to preeclampsia (82,000 births per year).<sup>[48]</sup> In France, hypertensive pathologies in pregnancy are the third cause of great prematurity (22%).<sup>[49]</sup>

In our series, prematurity comes first with an incidence of 5%.

**C/Hypotrophy**

In a recent study, 1.4% of the total incidence of IUGR was thought to be secondary to hypertensive disorders, and the incidence of this complication in hypertensive women is approximately 35%; however, it appears that the course of IUGR is independent of the presence or absence of gestational hypertension.<sup>[23]</sup>

In our series, hypotrophy was the first perinatal complication with an incidence of 15% (birth weight<2500g including the IUGR).

Our figures are higher to those reported by Moukaddime.A,<sup>[17]</sup> who noted a frequency of 7%, while Thiam.M,<sup>[2]</sup> observed a higher rate of around 40%.

**D/Neonatal Suffering**

In the Casablanca series, neonatal suffering was of the order of 8.97% with 11% progression to perinatal death.<sup>[9]</sup> In our series, the frequency of neonatal suffering was around 6%.

**8-Medical and obstetrical care****A/Medical care**

Alpha methyl dopa is the main antihypertensive drug used for the medical management of gestational hypertension.<sup>[50]</sup>

This drug provides gentle blood pressure control without fetal consequences. It is prescribed at a dose of 250 mg twice daily for 48 hours and then at a maintenance dose with an increase of 250 mg in 48-hour increments to a usual dose of 0.75 to 1.5 g per day.

The most commonly observed adverse effect is sedation, usually transient, occurring at the beginning of treatment or when doses are increased.

However, caution should be exercised with regard to the possible occurrence of psychological disorders (nightmares, depression) or cardiovascular disorders with the appearance of oedema.<sup>[51]</sup>

In our study, alpha-methyl dopa was the most commonly used drug, 75% in monotherapy and 20% in dual therapy.

Nifedipine is used in variable doses.<sup>[51,52]</sup> In our study it was mainly used in dual therapy with alpha methyl dopa in the context of uncontrolled gestational hypertension.

Magnesium sulphate is mainly used in severe preeclampsia to prevent eclampsia and especially to prevent recurrence of the eclampsia attack.<sup>[53,54,55,56]</sup>

The Magpie study has shown that the use of magnesium sulphate reduces the risk of eclampsia by 50% compared to placebo.<sup>[55]</sup>

The initial treatment regimen involves a bolus of 4g of magnesium sulphate in 20-30 min, followed by a continuous intravenous infusion of 1g/h.<sup>[57,58]</sup>

Monitoring of treatment should be based on repeated assessment of consciousness (Glasgow=15), OTR, respiratory rate (>12c/min) and diuresis (>30 ml/h). In case of manifestations of overdosage, the infusion should be stopped, calcium gluconate injection considered and magnesemia measured.<sup>[58]</sup>

In our study, magnesium sulphate was used in 6 patients, i.e. 10% of the women, 4 of whom had eclamptic seizures and 2 for severe pre-eclampsia to prevent the eclamptic seizure.

Aspirin was used preventively from the first weeks of amenorrhea in all patients at risk and those diagnosed in time, i.e. 92%.

In our study, corticosteroid therapy was also used in the context of fetal pulmonary maturation for 10 women, i.e. 16%.

**B/Obstetrical care**

There is no specific treatment. The only curative treatment for severe GH remains to this day the termination of pregnancy and delivery of the placenta.

In cases of non-severe gestational arterial hypertension above 36 weeks of amenorrhea.

Severe gestational hypertension prior to 24 weeks of amenorrhea requires a medical termination of the pregnancy which must be clearly discussed with the parents.<sup>[58]</sup>

Indications for termination of pregnancy in severe GH between 24 and 34 weeks of amenorrhea may be maternal or fetal,<sup>[84]</sup> for immediate maternal reasons (uncontrolled hypertension, eclampsia, PAO, PRH, thrombocytopenia less than 50,000, hepatic subcapsular haematoma) or after corticosteroid therapy for fetal maturation in cases of rapidly worsening renal failure and/or persistent oliguria (< 100 ml/4 hours) despite adequate vascular filling, persistent signs of impending eclampsia (headache or visual disturbances), persistent epigastric pain, HELLP syndrome evolving.

Indications are extended to fetal reasons such as repeated decelerations of baby's heart rate, severe IUGR above 32

weeks of amenorrhea, inverted arterial umbilical diastole above 32 weeks of amenorrhea.

The choice between Caesarean section and vaginal delivery depends on the term, local conditions, fetal and maternal status. A vaginal delivery is reserved for GH without fetal impact where fear of a worsening of any late pregnancy may (if obstetrical conditions are favourable) lead to a decision to induce labour instead of spontaneous delivery. If the maternal or fetal condition is severe, extraction is most often performed by Caesarean section.<sup>[59]</sup>

In our study, 39 patients gave birth by vaginal route, i.e. 65%, while Caesarean section was performed in 35% of cases (20 patients).

The upper route was indicated in 12 patients. Secondary Caesarean section was performed in 8 patients for labour abnormalities, fetal distress or failure of induction.

## V-CONCLUSION

Hypertensive disorders of pregnancy include a wide range of relatively diverse conditions under the same title, with the threatening and enigmatic shadow of preeclampsia looming over them.

If antihypertensive medication is introduced, it is important to weigh the potential maternal benefits against the potential risks to the fetus due to placental hypoperfusion.

Many questions, especially about preeclampsia, remain unanswered and are the subject of much research. Although, at present, each new discovery seems to add to the immense complexity of this disease and reveal more grey areas than it sheds light on, the progress of the work allows us to remain confident that new therapeutic strategies will be implemented. Until then, vigilance remains the best treatment.

## VI-BIBLIOGRAPHIC REFERENCES

- Zhang J, Meikle S, Trumble A. Severe maternal morbidity associated with hypertensive disorders in pregnancy in the United States. *Hypertens Pregnancy*, 2003; 22: 203-12.
- Thiam M, Goumbala M, Gning SB, Fall PD, Cellier C, Perret JL. Pronostic maternel et foetal de l'association hypertension et grossesse en Afrique Subsaharienne (Sénégal). *J Gynecol Obstet Biol Reprod*, 2003; 32: 35-8
- Beaufils M. Hypertensions de la grossesse. *Néphrologie et thérapeutique*, 2010; 6: 200-14.
- Daouda Diallo F. Hypertension arterielle et grossesse dans le service de gynécologie obstétrique du CHU Gabriel Touré de 2003 à 2006. *Thèse Doctorat Médecine*, Bamako, 2008; 10: 147.
- Assogba schola C. La prééclampsie à l'hôpital de la mère et de l'enfant Lagune de Cotonou. *Thèse Doctorat Médecine*, Bamako, 2005; 24,139.
- Zenebe W Hailemariam S, Mirkuzie W. Hypertensive disorders of pregnancy in Jimma university specialized hospital. *Ethiop J Health Sci.*, 2011; 21(3): 147-54.
- Goffinet F. *Epidémiologie. Annales Françaises d'Anesthésie et de Réanimation*, 2010; 29: 7-12
- Elkoudia ML. Prééclampsie au CHU Hassan II de Fès. *Thèse Doctorat Médecine*, Fès, 2011; 94: 337.
- Moukkadime A. Pronostic foetal et maternel au cours de la toxémie gravidique à la maternité Lalla Meryem du CHU Ibn Rochd de Casablanca à propos de 330 cas. *Thèse Doctorat Médecine*, Casablanca, 2001; 306: 143.
- El falaki S. Toxémie gravidique à l'hôpital Hassan II de la wilaya d'Agadir (à propos de 307 cas). *Thèse Doctorat Médecine*, Rabat, 2003; 234: 146.
- MANSOURI I. Hypertension artérielle gravidique: Expérience du service de maternité Souissi II (355 cas). *Thèse Doctorat Médecine*, Rabat, 2005; 167: 141.
- Zenebe W Hailemariam S, Mirkuzie W. Hypertensive disorders of pregnancy in Jimma university specialized hospital. *Ethiop J Health Sci.*, 2011; 21(3): 147-54.
- Bah AO, Diallo MH, Diallo AA, Keita N, Diallo MS. Hypertension artérielle et grossesse: aspects épidémiologiques et facteurs de risques. *Médecine d'Afrique noire*, 2000; 47(10): 422-5.
- Duckitt K, Harrington D. Risk factors for pre-eclampsia at antenatal booking: systematic review of controlled studies. *BMJ*, 2005; 330: 565.
- Thadhani R, Stampfer MJ, Hunter DJ, Manson JE, Solomon CG, Curhan GC. High body mass index and hypercholesterolemia: risk of hypertensive disorders of pregnancy. *Obstet Gynecol*, 1999; 94: 543-50.
- Rosenberg TJ, Garbers S, Lipkind H, Chiasson MA. Maternal obesity and diabetes as risk factors for adverse pregnancy outcomes: differences among 4 racial/ethnic groups. *Am J Public Health*, 2005; 95: 1545-51.
- Moukkadime A. Pronostic foetal et maternel au cours de la toxémie gravidique à la maternité Lalla Meryem du CHU Ibn Rochd de Casablanca à propos de 330 cas. *Thèse Doctorat Médecine*, Casablanca, 2001; 306: 143.
- Stone JL, Lockwood CJ, Berkowitz GS, Alvarez M, Lapinski R, Berkowitz RL. Risk factors for severe preeclampsia. *Obstet Gynecol*, 1994; 83: 357-61.
- Bramham K, Briley AL, Seed P, et al. Adverse maternal and perinatal outcomes in women with previous preeclampsia: a prospective study. *Am J Obstet Gynecol*, 2011; 204: 512.e1-9.
- Poon LC, Kametas NA, Maiz N, Akolekar R, Nicolaides KH. First trimester prediction of hypertensive disorders in pregnancy. *Hypertension*, 2009; 53: 812-8.

21. MC Cowen LM, Buist RG, North RA, Gamble G. Perinatal morbidity in chronic hypertension BJOG, 1996; 103: 123-9.
22. Bodnar LM, Ness RB, Harger GF, Roberts JM. Inflammation and triglycerides partially mediate the effect of prepregnancy body mass index on the risk of preeclampsia. Am J Epidemiol 2005; 162: 1198–206.
23. Villar J, Carroli G, Wojdyla D, Abalos E, Giordano D, Baaqeel H, et al. Preeclampsia, gestational hypertension and intrauterine growth restriction, related or independent conditions? Am J Obstet Gynecol, 2006; 194: 921–31.
24. Schieve LA, Handler A, Hershow R, Persky V, Davis F. Urinary tract infection during pregnancy: its association with maternal morbidity and Perinatal outcome. Am J Public Health, 1994; 84: 405–10.
25. Berkane N. Définitions et conséquences des hypertensions artérielles de la grossesse. Annales Françaises d'Anesthésie et de Réanimation 2010; 29: 1–6
26. Société française d'anesthésie et de réanimation (SFAR), Collège national des gynécologues et obstétriciens français (CNGOF), Société française de médecine périnatale (SFMP), Société française de néonatalogie (SFNN). Prise en charge multidisciplinaire de la pré-éclampsie: Recommandations formalisées d'experts communes 2008. Annales Françaises d' Anesthésie et de Réanimation, 2009; 28: 275–81.
27. Édouard D. Prééclampsie. Éclampsie EMC [36-980-A-10],
28. ACOG practice bulletin Diagnosis and management of preeclampsia and eclampsia. Clinical management guideline for obstetrician-gynecologists, 2002; 33: 159-67
29. Raphael V, Levasseur J. Éclampsie. EMC [25-070-B-20].
30. Ducarme G, Herrnberger S, Pharsien I, Carillon L, Uzan M. Eclampsie : étude rétrospective de 16 cas. Gynécologie Obstétrique & Fertilité, 2009; 37: 11-7.
31. Moulin B, Hertig A, Rondeau E. Rein et prééclampsie. Annales Françaises d'Anesthésie et de Réanimation 2010; 29: 83–90.
32. Pottecher, et al. Réanimation des formes graves de prééclampsie : conférence d'experts, Société Française d'anesthésie et de réanimation, Société française de médecine périnatale, Société française de pédiatrie, Collège national des gynécologues obstétriciens français. J Gynecol Obstet Biol Reprod, 2001; 30: 121-32.
33. Trabya C, Rudigoz RC, Dubernard G, Huissoud C. Les troubles biologiques au cours des états préclamptiques : aspects physiopathologiques et cliniques. Revue francophone des laboratoires, 2010; 421: 43-50.
34. Boukhchach FE. Épidémiologie de l'hypertension artérielle gravidique à propos de 544 cas. Thèse Doctorat Médecine, Marrakech, 2009; 10: 151.
35. Beye MD, Diouf E, Kane O, Ndoye MD, Seydi A, Ndiaye PI, et al. Prise en charge de l'éclampsie grave en réanimation en milieu tropical africain à propos de 28 cas. Annales Françaises d' Anesthésie et de Réanimation, 2003; 22: 25–9.
36. Pourrat O. Prééclampsie et éclampsie: progrès thérapeutiques. Actualités néphrologiques. Paris: Flammarion Médecine Sciences, 2004: 177-89.
37. Rugarn O, Carling Moen S, Berg G. Eclampsia at a tertiary hospital 1973–99. Acta Obstet Gynecol Scand, 2005; 84: 496.
38. Miguil M, El Mabady E, Khatib G, El Youssoufi S, Salmi S. Facteurs pronostiques de l'éclampsie : à propos de 408 cas. Réanimation, 2008; 17: 65.
39. Cisse CT, Faye Dieme ME, Ngabo D, Mbaye M, Diagne PM, Moreau JC. Indications thérapeutiques et pronostic de l'éclampsie au CHU de Dakar J Gynecol Obstet Biol Reprod, 2003; 32: 239-45.
40. Ghulmiyyah L, Sibai B. Maternal Mortality From Preeclampsia/Eclampsia. Seminars in perinatology. Elsevier, 2012; 36: 56-9
41. Sabiri B, Moussalit A, Salmi S, El youssoufi S, Miguil M. L'éclampsie du post-partum : épidémiologie et pronostic J Gynecol Obstet Biol Reprod, 2007; 36: 276–80.
42. Thieba B, Lankoande J, Akotionga M, Kyelem C, Ouedraogo A, et al. Hématome rétroplacentaire : aspects épidémiologiques et pronostiques à propos d'une série de 177 cas. Gynécologie Obstétrique & Fertilité, 2003; 31(5): 429–33
43. Martin JN, Rinehart BK, May WL, Magann EF, Terrone DA, Blake PG. The spectrum of severe preeclampsia: comparative analysis by HELLP (hemolysis, elevated liver enzymes levels, and low platelet count) syndrome classification. Am J Obstet Gynecol, 1999; 180: 1373–84.
44. Sibai BM, Dekker G, Kupferminc M. Pre-eclampsia. Lancet, 2005; 365: 785–99
45. Matthys LA, Coppage KH, Lambers DS, Barton JR, Sibai BM. Delayed postpartum preeclampsia: An experience of 151 cases. Am J Obstet Gynecol, 2004; 190: 1464-6
46. Merviel P, Touzart L, Deslandes V, Delmas M, Coicaud M, Gondry J. Facteurs de risque de la prééclampsie en cas de grossesse unique J Gynecol Obstet Biol Reprod, 2008; 37: 477-82
47. Randriambololona DMA, Botolahy ZA, Randrianantoanina FE, Randriamahavony R, Hery Rakotovao A. Hypertension artérielle et grossesse: pronostic materno-fœtal. Revue Tropicale de Chirurgie, 2009; 3: 32-4
48. Hupuczi P, Sziller I, Hruba E, Rigo B, Szabo G, Papp Z. The rate of maternal complications in 107 pregnancies complicated with HELLP syndrome. Orv Hetil, 2006; 147: 1377-85.
49. Zeitlin J, Ancel PY, Larroque B, Kaminski M, EPIPAGE Study. Fetal sex and indicated very preterm birth : results of the EPIPAGE study. Am J Obstet Gynecol, 2004; 190: 1322–5.

50. Pourrat O.Etendu et noté : Atelier FMC sur l'hypertension artérielle au cours de la grossesse, 63e Congrès de la Société nationale française de médecine interne, Poitiers, 2011.Revue de médecine interne, 2011; 32: 649-51
51. Boutroy Mj, Bayoumen F .Utilisation des antihypertenseurs en obstétrique. EMC, 5-036-A-20.
52. Duley L, Henderson-Smart DJ, Knight M, King JF.Antiplatelet agents for preventing pre-eclampsia and its complications. Cochrane Database Syst Rev, 2004. CD004659.
53. Jabeen M, Yawar M, Imdad A, Bhutta z.Impact of interventions to prevent and manage preeclampsia and eclampsia on stillbirths.BMC Public Health, 2011; 11(3): 1-11
54. Rozenberg P.Intérêt du sulfate de magnésium dans la prise en charge de la prééclampsie. Gynécologie Obstétrique & Fertilité, 2006; 34: 54–59.
55. Altman D, Carroli G, Duley L, Farrell B, Moodley J, Neilson J. et al.Do women with preeclampsia, and their babies, benefit from magnesium sulphate ? The magpie trial: a randomised placebo-controlled trial. Lancet, 2002; 359: 1877-90.
56. Sibai BM. Diagnosis, prevention, and management of eclampsia. Obstet Gynecol 2005; 105: 402-10.
57. Winer N, Tsasaris V. État des connaissances : prise en charge thérapeutique de la prééclampsie. La Revue Sage-Femme, 2008; 7: 27—37
58. Pottecher T. Prise en charge multidisciplinaire des formes graves de pré-éclampsie. La Revue Sage-Femme, 2009; 8: 247-52
59. Uzan S, Beaufils M, Uzan M HTA et grossesse in papernik E cabrol" JC obstétrique medicine science flammarion paris, 1995.