

# High Blood Pressure During Pregnancy

## About 544 Cases

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### Abstract:-

#### ➤ Background :

Arterial hypertension and pregnancy is common and remains a leading cause of maternal and fetal mortality and morbidity. The aim of this work is to study the peculiarities of this high-risk pregnancy. This is a retrospective study of 544 cases of hypertension and pregnancy collected at the LallaMeryem maternity hospital Ibn Rochd hospital in Casablanca for a period of 2 years. The incidence is 9.2%. The average age of onset was 30 years with an age range of 15 to 45 years. The primiparous were the most exposed 261 cases (48%). 310 cases (57%) have an unguarded pregnancy. 290 cases (53.3%) had a systolic blood pressure greater than or equal to 160 mm Hg, and 160 cases (29.4%) had a diastolic blood pressure  $\geq$  110 mmHg. The most used medical conduct was the combination of rest and antihypertensives. Obstetrical behavior was marked by the frequency of vaginal deliveries (63.4%). Maternal complications represent (14.7%) dominated by retro-placental hematoma (5.1%) and eclampsia (3.7%). Perinatal mortality represents 57 cases (9.9%). The factors of bad foeto-maternal prognosis are for the fetus: the low gestational age, the low parity, the non monitoring of the pregnancy, the massive proteinuria and the hyperuricemia. For the mother, young maternal age, primiparity, non-pregnancy status, diastolic blood pressure  $\geq$  110 mm Hg, systolic blood pressure  $\geq$  160 mm Hg, and massive proteinuria. Careful monitoring of pregnancies, early diagnosis of high blood pressure and better management of the mother and child, as well as a better knowledge of the fetal and maternal prognostic factors, contribute to the reduction of complications of arterial hypertension during pregnancy.

#### ➤ Conclusion:

This study concluded that high blood pressure and pregnancy is a common condition and remains a major cause of maternofetal mortality and morbidity. These complications can be reduced by a better physiopathological understanding and a better knowledge of the fetal and maternal prognostic factors. This condition mobilizes obstetricians and researchers looking for effective treatment. The only truly effective treatment is the termination of pregnancy, all other

therapies are intended only to prolong the pregnancy to a term acceptable to the fetus.

### I. INTRODUCTION:

The definition of hypertension in pregnancy is based only on office (or inhospital) BP values [systolic BP (SBP)  $\geq$ 140 mmHg and/or DBP  $\geq$ 90 mmHg] and distinguishes mildly (140–159/90–109 mmHg) or severely ( $\geq$  160/110 mmHg) elevated BP, in contrast to the grades used by the joint ESC/ESH Hypertension Guidelines. Hypertension in pregnancy is not a single entity but comprises:

**Pre-existing hypertension:** precedes pregnancy or develops before 20 weeks of gestation. It usually persists for more than 42 days post-partum and may be associated with proteinuria.

**Gestational hypertension:** develops after 20 weeks of gestation and usually resolves within 42 days post-partum.

**Pre-eclampsia:** gestational hypertension with significant proteinuria ( $>0.3$  g/24 h or ACR  $\geq$ 30 mg/mmol). It occurs more frequently during the first pregnancy, in multiple pregnancy, in hydatidiform mole, in antiphospholipid syndrome, or with pre-existing hypertension, renal disease, or diabetes. It is often associated with foetal growth restriction due to placental insufficiency and is a common cause of prematurity. The only cure is delivery.<sup>363</sup> As proteinuria may be a late manifestation of pre-eclampsia, it should be suspected when de novo hypertension is accompanied by headache, visual disturbances, abdominal pain, or abnormal laboratory tests, specifically low platelets and/or abnormal liver function.

**Pre-existing hypertension plus superimposed gestational hypertension with proteinuria**

**Antenatally unclassifiable hypertension:** This term is used when BP is first recorded after 20 weeks of gestation and hypertension is diagnosed; reassessment is necessary after 42 days post-partum.

Hypertensive disorders in pregnancy are the most common medical complications, affecting 5–10% of pregnancies worldwide. They remain a major cause of

maternal, foetal, and neonatal morbidity and mortality. Maternal risks include placental abruption, stroke, multiple organ failure, and disseminated intravascular coagulation. The foetus is at high-risk of intrauterine growth retardation (25% of cases of pre-eclampsia), pre-maturity (27% of cases of preeclampsia), and intrauterine death (4% of cases of pre-eclampsia). Its etiology remains uncertain to date despite the many epidemiological and pathophysiological studies carried out for this purpose, the latter made it possible to define a high-risk population and to evaluate the prognostic factors, this will allow a better therapeutic, medical and obstetric and therefore a decrease in maternofetal mortality, number of complications and severe forms.

#### Patients and Study Methods: I-Study Population:

The objective of this work is to specify the epidemiological, clinical, paraclinical, therapeutic and prognostic aspects of high blood pressure during pregnancy, as well as the risk factors for maternal and fetal complications in our study population.

This is a retrospective analysis of 544 cases of hypertension during pregnancy out of a total of 5946 deliveries collected at the Gynecology and Obstetrics Department "A" at the LallaMeryem maternity hospital of Ibn Rochd Hospital, Casablanca. Over a period of two years. Included in this study were all pregnant women with the diagnosis of hypertension defined by blood pressure values greater than or equal to 140 / 90mmHg with or without proteinuria with or without edema, and all maternal complications: eclampsia, Hematoma and placental, HELLP syndrome, Intravascular Coagulation Disseminated.

We used for making observations:

- hospital record
- the registers of births
- operating room reports
- Resuscitator reports about the evolution of patients with severe preeclampsia. We try to clarify from our study this particularity of this entity.

## II. RESULTS

### Global Study

#### Epidemiological Characters

##### ▪ Frequency

Out of a total of 5946 births registered for two years, we recorded 544 cases of hypertension and pregnancy giving an incidence rate of 9.2%.

Year Delivery number	Number	%
2012 2858	262	9,2
2013 3088	282	9,1

Table 1: Incidence of high blood pressure and pregnancy.

- **Origin Of The Patients:**339 cases (62, 3%) our parturients were of urban origin.

Types	N
Gestational hypertension	228
Transient hypertension	163
Preeclampsia	136
Preeclampsia superadded	17

Table 2: Types of Hypertension met.

- **Frequency According to the Months**

Month	%
January	30
February	38
March	63
April	60
May	44
June	53

<b>July</b>	43
<b>August</b>	30
<b>September</b>	28
<b>October</b>	24
<b>November</b>	50
<b>December</b>	46

**Table 3:** Arterial Hypertension Frequency in Month Period.

- **Mother's age**

The age of the patients varies between 15 and 46 years with an average of 30 years.

<b>AGE</b>	<b>N</b>
<b>15-20</b>	49
<b>21-25</b>	100
<b>26-30</b>	150
<b>31-35</b>	97
<b>36-40</b>	105
<b>Superior à 40</b>	43

**Table 4:** Frequency by age.

- **Antecedents**

- **Medical**

363 cases (66.7%) of our parturients had no medical history.

<b>Antecedents:</b>	<b>N</b>	<b>%</b>
<b>High blood pressure</b>	24	4,4
<b>Diabetes</b>	15	2,7
<b>Heart disease</b>	13	2,4
<b>Nephropathy</b>	6	1,1
<b>Other</b>	42	7,7
<b>Total</b>	100	18,4

**Table 5:** Distribution according to medical antecedent.

- **Obstetrics**

210 cases (38.6%) with an obstetrical history are distributed as follows:

	<b>N</b>	<b>%</b>
<b>Abortion</b>	68	12,5
<b>Fetal death</b>	65	12
<b>Caesarean</b>	26	4,8
<b>Isolated blood pressure</b>	13	2,4
<b>Complicated hypertension</b>	7	1,3
<b>Eclampsia</b>	13	2,4
<b>Retro-placental hematoma</b>	10	1,8
<b>Prenatal Mortality</b>	8	1,5

**Table 6:** Distribution of Patients by Obstetrical History.

### ▪ Parity

We divided the patients by parity into three groups:

- Primiparous: a childbirth after 28 weeks
- Multipares: 2 or 3 deliveries
- Large Multiparous: 4 or more deliveries.

N	%
<b>Primiparae 262</b>	48
<b>Multiparous 167</b>	31
<b>-Plus 4 childbirth 115</b>	21

**Table 7:** Frequencies according to parity.

### ▪ Examen Paraclinic

	N	%
<b>Groupage + rhesus</b>	280	51,5
<b>Blood count + platelets</b>	180	33,1
<b>Fasting blood glucose</b>	50	9,2
<b>Kidney function</b>	30	5,5
<b>Uric acid</b>	8	1,5
<b>liver function</b>	12	2,2
<b>Hemostasis assessment</b>	25	4,6
<b>Proteinuria 24h</b>	20	3,7
<b>Echography</b>	187	34,4

**Table 8:** Before Admission

### ▪ Gestational Age

AGE GESTATIONEL	N
<b>Lower than 28</b>	40
<b>28-32</b>	53
<b>33-36</b>	100
<b>37-42</b>	331
<b>Better than 42</b>	20

**Table 9:** Distribution by gestationa

### Clinical Data

#### General Examination

- Blood pressure
- Systolic blood pressure

SBP	<140	140/150	160/170	180/190	>20	Total
N	74	180	170	90	30	544
%	13,6	33,1	31,3	16,5	5,5	100

**Table 10:** Systolic blood pressure figures in mmHg

440 cases (94.5%) of our parturients had a SBP between 140-190mmHg against 180 cases (33.1%) with blood pressure figures between 140 and 150 mm Hg.

#### ▪ Diastolic blood pressure

Diastolic blood pressure	<90	90-100	110-120	130-140	>150	Total
N	64	320	140	15	5	544
%	11,8	58,9	25,8	2,8	0,9	100

**Table 11:** Diastolic blood pressure figures in mmHg.

460 cases (84.6%) of patients had a DBP between 90-120 mmHg in our series.

#### ▪ Maternal weight

Weight (Kg)	<70	(70-80)	(80-90)	(90-100)	>100	Not pris	Total
N	53	85	74	53	40	239	544
%	9,7	15,6	13,6	9,7	7,4	44	100

**Table 12:** Distribution according to maternal weight (in Kg).

Weight was not taken only at 305 parturients. 93 cases (17%) weighed more than 90 kg.

#### ▪ Proteinuria (test strips)

Proteinuria	N	%
O or traces	69	12,7
One or two crosses	36	6,6
> Two ++	70	12,9
Not pris	369	68
Total	544	100

**Table 13:** Proteinuria cross

Proteinuria was estimated in only 175 cases (32.2%) by means of test strips and revealed a significant proteinuria > two ++ in 70 cases (12.9%).

#### ▪ Others (edema)

Edema	N	%
Located at the lower 288 limb		53
Generalized	16	3
Absent	240	44
Total	544	100

**Table 14:** frequency of edemas.

#### ▪ Obstetrical observation

##### ▪ Uterine contractions

208 cases (38.2%) presented to the maternity ward outside of work.

##### ▪ Uterine height

Uterine height was measured in almost all patients in our series.

232 cases (42.6%) had an HU  $\geq$  31 cm, of which 70 cases (13%) had an excessive uterine height greater than 35 cm.

	N	%
< 24	34	6,3
24- 30	208	38,2
31-35	232	42,6
> 35	70	13
<b>Total</b>	<b>544</b>	<b>100</b>

**Table 15:** uterine height (cm).

#### ▪ Sounds of the Fetal Heart

The auscultation of fetal heart sound is determined using the obstetrical stethoscope of PINARD, unspecified in only 27 cases (5%) because of edema of the abdominal wall.

Fetal heart sounds were negative in 62 cases (11.4%).

Fatal heart Sound	N	%
Negative	62	11,4
Positive	455	83,7
Not pris	27	5
<b>Total</b>	<b>504</b>	<b>100</b>

**Table 16:** fetal noise.

#### ▪ Para-clinical data

##### ▪ Fetal Heart Rate Recording

Of the fetal heart rate recordings in our series, 87 (16%) were pathological.

##### ▪ Radiological examinations: a- Obstetrical ultrasound

This examination performed at 516 (94.9%) parturientes showed:

	N	%
GrossesseGemellaire	30	5,8
Fetal death	62	12
Intra-uterine growth delay	17	3,3
Oligoamnios	25	4,8
Anaminios	7	1,4
Hydramnios	13	2,5
Retro-placental hematoma	38	7,4
Placenta praevia	9	1,7
Normale	315	61

**Table 17:** Results of Obstetrical Ultrasound.

##### ▪ Echopdpler

The Doppler echo is more and more practiced, in our series 164 cases (30.14%) benefited from this examination. This examination is based essentially on the exploration of the uterine arteries and the umbilical artery. The exploration of the cerebrovascular territories was not carried out among the parturientes of our series.

The different anomalies observed are distributed in the following tables:

	N	%
Diastole decreased	30	5,5
Diastole nothing	17	3,1

**Table 18:** Umbilical Doppler Results.

Umbilical doppler was pathological in 33 cases (5.5%).

	N	%
Normale	139	25,5
Notch	20	3,7

**Table 19:** Result of Uterine Doppler

The presence of Notch in the uterine artery was noted in 20 cases (3.7%).

### Biological examinations

#### ▪ Uricemia

This examination was of considerable importance in 253 cases (46.5%) and revealed hyperuricemia > 60 mg / l in 78 cases (14.3%).

	<40	40-60	>60	NOT PRIS	Total
N	45	130	78	291	544
%	8,3	23,9	14,3	53,5	100

**Table 20:** Uricemia in (mg / l)

#### ▪ Proteinuria 24h

The examination was performed in 303 cases (55.7%).

	0 ou traces	<0,3	0,3-0,5	0,5-1	>1	NOT PRIS	Total
N	133	54	37	28	51	288	544
%	24,4	9,9	6,8	5,2	9,4	52,9	100

**Table 22:** Distribution according to the proteinuria of 24h

Proteinuria was negative in 187 cases (34.3%) while 51 cases (9.37%) had a proteinuria > 1g / 24h.

#### ▪ Kidney Function

Urea and serum creatinemia were determined in 437 cases (80.3%), with a serum creatinine level  $\geq$  15 mg / l in 13 cases (2.4%) and uremia  $\geq$  0.45 g / l in 17 patients. case (3.1%).

#### ▪ Liver Fonction

The liver test is based essentially on the determination of transaminases. This assay was performed in 375 cases (67%) of our series, it showed:

- Hepatic function checkup normal : in 319 cases (58.6%).
- Disrupted status in 17 cases (3.1%), including 7 cases (1.3%) with a transaminase level > 100 IU / ml.

#### ▪ Hemostasis Assessment

This assessment was carried out only in 519 cases (95.4%) of which 7 cases (1.3%) had a disrupted balance with elevation of the rate of fibrin degradation products especially in complicated arterial hypertension. retro-placental hematoma or eclampsia seizures.

#### ▪ Blood Count

Almost all of our patients benefited from this examination, he showed:

- 90 cases (16.5%) of hemoglobin anemia  $\leq$  8g / 100ml (39).
- 18 cases (3.3%) haemoconcentration Hematocrit  $\geq$  40%.
- Platelet count: 23 cases (4.2%) had thrombocytopenia (platelets <100,000 elements / cc).

### ▪ Fasting Blood Glucose

382 cases (5.4%) had a blood glucose level > 1.26 g / l.

### ▪ Therapeutic Data

#### ▪ Medical Treatments

Hospitalization, rest in the left lateral decubitus and normal sodium diet were the rule in the patients of our series. These hygiene and dietary measures were sufficient in 64 cases (11.8%) while 480 cases (88.2%) received antihypertensive treatment.

#### ▪ Antihypertensive Treatment

The main molecules used in our series were:

Alpha-methyl-dopa (aldomet)

Dihydralazine (nepressol)

Nicardipine (loxen)

And atenolol (tenorine)

Either as monotherapy or dual therapy, and in cases of severe pre-eclampsia, triple therapy is used.

#### ▪ The different therapeutic protocols are distributed in the following table

		N	%
* Monotherapy	Alpha-methyl-dopa	221	40,6
	Dihydralazine	19	3,5
	Nicardipine	42	7,7
* Bithérapie	alpha-methyl-dihydralazine	103	18,9
	alpha-methyl-nicardepine	35	6,4
* Trithérapie	Alphamethyl-dihydralazine-nicardepine	47	8,6
	Alphamethyl-dihydralazine-atenolol	7	1,3
	alphamethyl-nicardepine-furosemide	6	1,1
	Not medically treated	64	11,76
	<b>Total</b>	544	100

**Table 23:** Distribution according to adopted pharmacological protocols.

282 cases (52%) treated as monotherapy. In 138 cases (25.4%), dual therapy was prescribed based on the combination of alpha-methyl-dopa and dihydralazine in 103 cases (18.9%), and in 35 cases (6.4%) case based on alphamethyl-dopa and nicardipine.

#### ▪ Sedative Treatment

The sedative treatment was prescribed either for treatment or prevention, it is associated with anti-hypertension treatment in 34 cases (6.3%) including 27 cases of eclampsia and 7 cases of severe pre-eclampsia where the treatment was used as a preventive measure, the main product used was valium.

#### ▪ Other Medications

We had to transfuse 7 cases (13%) with red blood cells, and in those who were in a state of eclamptic ecstasy the vascular filling was based on physiological saline, Hemacel or fresh frozen plasma.

#### ▪ Obstetric driving

Out of 544 cases there were 20 twin pregnancies, bringing the number of deliveries to 564 deliveries.

We postponed the delivery of 32 cases (5.9%) of the cases that were not at term, and out of work, after stabilization of their blood pressure.

#### Delivery

Artificial induction of labor was necessary in 180 cases (33.1%) generally with misoprostol while 289 cases (87%) spontaneously entered work.

Delivery was deferred in 43 cases (8%).

Trigger indications were distributed as follows

	N	%
<b>Term pregnancy</b>	70	51,5
<b>Exceeding term</b>	10	7,4
<b>In utero fetal death</b>	47	34,5
<b>Signs of gravity</b>	9	6,6
<b>Total</b>	136	100

**Table 24:** Trigger indications.

▪ **Way of delivery**

▪ **Low way**

The low pathway was indicated in 345 cases (63.4%).

Spontaneous delivery was observed in 450 cases (82.7%), compared with 94 cases (17.3%) which required instrumental extraction.

▪ **High way**

Caesarean section was reported in 193 cases (35.5%).

	N	%
<b>* Acute fetal distress</b>	87	45,1
<b>* Chronic fetal distress</b>	13	6,7
<b>* Retro-placental hematoma</b>	15	7,8
<b>* Maternal rescue - eclampsia</b>	10	5,2
<b>- Hellyp syndrome</b>	2	1
<b>* Failure to agulate complete dilatation with</b>	16	8,3
<b>* Dystocia</b>	11	5,7
<b>* Macrosomia</b>	21	10,1
<b>* Irregular presentation</b>	5	2,6
<b>* Other</b>	13	6,7
<b>Total</b>	193	100

**Table 25:** Indications for cesarean section

N	%
415	85.2
487	100

**Table 26:** Apgar score at the 1st minute

193 cases (35.5%) were caesarean section of which 87 cases (45.1%) related to acute fetal distress.

▪ **State of Newborns at Birth**

Of the 544 deliveries, we revealed 487 live newborns (89.5%).

▪ **Apgar Score**

\* ≤ 3 15 3,1

\* 4-6 57 11,7

\* ≥ 7

▪ **Total**

In our series, the Apgar score was mentioned in all newborns. Of these, 487 (85.2%) had an Apgar  $\geq 7$  score while 15 (3.08%) were in apparent death with an Apgar score of  $\leq 3$ .

### ▪ Sex

51.5% of the born were male in our series.

Fetal weight at birth in (g)

In our series, fetal weight was taken as well in term, premature infants, and fetal deaths in utero.

160 cases (28%) had a weight of  $\leq 2500$  g.

Poids	N	%
Lower than 2500	160	28
2500-3000	115	20
3001-3500	149	26
3501-4000	57	16
Better than 4000		10
Total	574	100

**Table 27:** Frequencies according to fetal weight.

### ▪ Fetal Evolution

Of the 574 births, 413 born (72%) had a favorable evolution.

102 cases (18%) had had at least one complication.

Acute Fetal Suffering 89 cases (15.5%), perinatal mortality 57 cases (15.3%) and prematurity 75 cases (13.1%) were the most frequently observed complications.

		N	%
Mortality	In utero fetal death	42	7,7
	Mortality neonatal	15	2,6
Morbidity	Prematurity	75	13,1
	hypotrophy	43	7,5
	Acute fetal distress	89	15,5
	Neonatal suffering	65	11,9

**Table 28:** Frequency of fetal complications.

### ▪ Maternal Evolution

461 cases (84.7%) had a favorable evolution without any complications. While 80 cases (14.7%) presented at least one complication.

28 cases (5.1%) complicated the retro-placental hematoma.

20 cases (3.7%) of eclampsia.

We recorded the death of three cases (0.6%) of all our parturients.

	N	%
Retro-placental hematoma	28	5.1
Eclampsia	20	3,7
Haemorrhagic shock	9	1,7
Acute renal failure	4	0,7
Hellp syndrome	2	0.4
	5	0.9
encephalopathy	1	0.1
Death	3	0.6
Other	8	1.4
Total	80	14,7

**Table 29:** Frequency of maternal complications.

### ▪ State of Newborns at Birth

Of the 544 deliveries, we revealed 487 live newborns (89.5%).

### ▪ Fetal Weight at Birth in (g)

In our series, fetal weight was taken as well in term, premature infants, and fetal deaths in utero. 160 cases (28%) weighed less than 2500 g.

## III. ANALYSIS OF MATRNL COMPLICATIONS

### Based on Epidemiological Parameters

#### • Maternal age

	Retro-placental hematoma	Eclampsia	hemorrhage shock	acute kidney failure	Disseminated intravascular coagulation	Other	Total	%
<25	3	3	2	1	2	2	13	17,6
25-35	20	13	4	2	2	5	46	62,1
>35	5	4	3	1	1	1	15	20,3
Total	28	20	9	4	5	8	74	100

**Table 30:** Maternal complications according to maternal age.

46 cases (62.1%) of maternal complications occurred in patients aged 25-35 years.

#### • Antecedents Vasculo-renal:

	Retro-placental hematoma	Eclampsia	hemorrhage shock	acute kidney failure	Disseminated intravascular coagulation	Other	Total	%
With	6	3	1	3	0	2	15	20,3
Without	22	17	8	1	5	6	59	79,7
Total	28	20	9	4	5	8	74	100

**Table 31:** Maternal complications according to Vasculo-renal antecedents.

#### • Follow-up of the pregnancy

	Retro-placental hematoma	Eclampsia	hemorrhage shock	acute kidney failure	Disseminated intravascular coagulation	Other	Total	%
Followed	3	2	1	0	0	4	10	13,5
not Followed	25	18	8	4	5	4	64	86,5
Total	28	20	9	4	5	8	74	100

**Table 32:** Maternal complications according to pregnancy monitoring

64 cases (86.5%) of maternal complications in patients not followed obstetrically.

#### • The Parity

	Retroplacental hematoma	Eclampsia	hemorrhage shock	acute kidney failure	Disseminated intravascular coagulation	Other	Total	%
primiparous	10	13	6	3	3	2	37	50
multiparous two -three	14	4	2	1	1	5	27	36,5
multiparous > three	4	3	1	0	1	1	10	13,5
Total	28	20	9	4	5	8	74	100

**Table 33:** Maternal complications according to parity.

37 cases (50%) of complications occurred in primiparous women.

- **Gestational Age**

Retroplacental hematoma	Eclampsia	hemorrhage shock	acute kidney failure	Disseminated intravascular coagulation	Other	Total	%
<34		8	4	2	5	32	43,2
≥34	17	12	5	2	3	42	56,8
Total	28	20	9	4	8	74	100

**Table 34:** Maternal complications according to gestational age.

42 cases (56.8%) of complications were observed at gestational age <34 weeks.

#### BASED ON CLINICAL DATA

- **Systolic Blood Pressure in mm Hg**

	Retro-placental hematoma	Eclampsia	hemorrhage shock	acute kidney failure	Disseminated intravascular coagulation	Other	Total	%
< 160	5	1	3	1	2	3	15	20,3
≥160	23	19	6	3	3	5	59	79,7
Total	28	20	9	4	5	8	74	100

**Table 35:** Maternal complications according to systolic blood pressure.

59 cases (79, 7) of maternal complications occurred in patients with systolic blood pressure ≥ 160 mm Hg.

- **Diastolic Blood Pressure in mm Hg:**

	Retro-placental hematoma	Eclampsia	hemorrhage shock	acute kidney failure	Disseminated intravascular coagulation	Other	Total	%
< 110	10	3	2	1	1	3	20	27,02
≥ 110	18	17	7	3	4	5	54	72,98
Total	28	20	9	4	5	8	74	100

**Table 36:** Maternal complications as a function of diastolic blood pressure.

54 cases (73%) of maternal complications were in patients with diastolic blood pressure ≥110 mm Hg.

- **According to Other Clinical Data:**

The study of other clinical parameters (edema, maternal weight and test strip proteinuria) was not allowed due to insufficient data.

#### 1. Based on Biological Parameters

- **Uricemia in mg / l**

47 cases (63.5%) were seen in patients with serum uric acid ≤60 mg / l versus 27 cases (36.5%) in patients with serum uric acid > 60 mg / l.

	Retro-placental hematoma	Eclampsia	hemorrhage shock	acute kidney failure	Disseminated intravascular coagulation	Other	Total	%
≤60	18	15	4	3	2	5	47	63.5
> 60	10	5	5	1	3	3	27	36.5
Total	28	20	9	4	5	8	74	100

**Table 37:** Maternal complications according to serum uricemia.

- **Proteinuria of 24 hours (g / 24h)**

	<b>Retro-placental hematoma</b>	<b>Eclampsia</b>	<b>hemorrhage shock</b>	<b>acute kidney failure</b>	<b>Disseminated intravascular coagulation</b>	<b>Other</b>	<b>Total</b>	<b>%</b>
< 0.3	6	1	0	0	0	1	8	15,1
0.3-1	3	6	2	0	0	3	14	26,4
>1	14	8	4	2	1	2	31	58,5
Total	23	15	6	2	1	6	53	100

**Table 38:** maternal complications according to the 24h proteinuria.

31 cases (58.5%) of maternal complications occurred in patients with proteinuria > 1g / 24h.

#### IV. DISCUSSION

A frequent and serious condition, the hypertensive pathology of pregnancy is one of the leading causes of maternal and perinatal mortality and morbidity, especially because of its severe forms. The prognosis of the severe forms of this disease can be improved by the detection of hypertension and the medical care of the patient (particularly the resuscitation measures) and obstetric adaptations, this study is not free from methodological limitations. It is retrospective and limited to 544 patients. It does not lead to statistically significant results. However, the results of this study may be of interest to the subject of hypertension, and it may be used for subsequent management. This paper describes the diagnosis, therapeutic, prognosis, and proposes solutions for better prognosis.(1)

In order to identify the epidemiological profile, the study of the diagnosis, the therapeutic and prognostic aspects of high blood pressure during pregnancy and the subsequent review of definitions, classifications and pathophysiology, we will analyze our results in relation to the literature.

##### Hypertensive Disorders

Hypertensive disorders in pregnancy are the most common medical complications, affecting 5–10% of pregnancies worldwide. They remain a major cause of maternal, foetal, and neonatal morbidity and mortality. Maternal risks include placental abruption, stroke, multiple organ failure, and disseminated intravascular coagulation. The foetus is at high-risk of intrauterine growth retardation (25% of cases of pre-eclampsia), pre-maturity (27% of cases of preeclampsia), and intrauterine death (4% of cases of pre-eclampsia).(2)

- **Diagnosis and Risk Assessment**

Repeated BP readings should be performed, preferably on two occasions,  $\geq 15$  min apart in severe hypertension (i.e.  $\geq 160/110$  mmHg in the obstetric literature).

- **Blood Pressure Measurement**

BP in pregnancy should be measured in the sitting position (or the left lateral recumbent during labour) with an appropriately-sized arm cuff at heart level and using Korotkoff V for diastolic BP (DBP). Mercury

sphygmomanometers are still the gold standard for BP measurement in pregnancy. Automatic devices tend to under-record the true BP and are unreliable in severe pre-eclampsia. Therefore, only devices validated according to recognized protocols should be used in pregnancy.

The diagnosis of hypertension in pregnancy by ambulatory BP monitoring (ABPM) is superior to routine BP measurement for the prediction of pregnancy outcome. The devices used for ABPM are technically more accurate than those used for office or home BP measurement. ABPM avoids unnecessary treatment of white-coat hypertension, and is useful in the management of high-risk pregnant women with hypertension and those with diabetic or hypertensive nephropathy.(3)

- **Laboratory Tests**

Basic laboratory investigations recommended for monitoring pregnant hypertensive patients include urinalysis, blood count, haematocrit, liver enzymes, serum creatinine, and serum uric acid (increased in clinically evident pre-eclampsia, hyperuricaemia in hypertensive pregnancies identifies women at increased risk of adverse maternal and foetal outcomes).

All pregnant women should be assessed for proteinuria in early pregnancy to detect pre-existing renal disease and, in the second half of pregnancy, to screen for pre-eclampsia. A dipstick test of  $\geq 1+$  should prompt further investigations, including an albumin:creatinine ratio (ACR), which can be quickly determined in a single spot urine sample. A value  $< 30$  mg/mmol can reliably rule out proteinuria in pregnancy, but a positive test should possibly be followed by a 24 h urine collection. In cases of proteinuria  $> 2$  g/day, close monitoring is warranted. However, the result of a 24 h urine collection is often inaccurate and delays the diagnosis of pre-eclampsia. Consequently, an ACR cut-off of 30 mg/mmol can be used to identify significant proteinuria.(4)

In addition to basic laboratory tests, the following investigations may be considered:

- Ultrasound investigation of the adrenals, and plasma and urinary fractionated metanephrine assays in hypertensive pregnant women with a suggestive clinical presentation of pheochromocytoma in particular.
- Doppler ultrasound of uterine arteries (performed after 20 weeks of

- Gestation) is useful to detect those at higher risk of gestational hypertension, preeclampsia, and intrauterine growth retardation.
- A sFlt1 to placental growth factor (sFlt1:PIGF) ratio  $\leq 38$  can be used to exclude the development of pre-eclampsia in the next week when suspected clinically.

#### • Definition and classification of hypertension in pregnancy

The definition of hypertension in pregnancy is based only on office (or inhospital) BP values [systolic BP (SBP)  $\geq 140$  mmHg and/or DBP  $\geq 90$  mmHg] and distinguishes mildly (140–159/90–109 mmHg) or severely ( $\geq 160/110$  mmHg) elevated BP, in contrast to the grades used by the joint ESC/ESH Hypertension Guidelines.

Hypertension in pregnancy is not a single entity but comprises:

- **Pre-existing hypertension:** precedes pregnancy or develops before 20 weeks of gestation. It usually persists for more than 42 days post-partum and may be associated with proteinuria.
- **Gestational hypertension:** develops after 20 weeks of gestation and usually resolves within 42 days postpartum.
- **Pre-eclampsia:** gestational hypertension with significant proteinuria ( $>0.3$  g/24 h or ACR  $\geq 30$  mg/mmol). It occurs more frequently during the first pregnancy, in multiple pregnancy, in hydatidiform mole, in antiphospholipid syndrome, or with preexisting hypertension, renal disease, or diabetes. It is often associated with foetal growth restriction due to placental insufficiency and is a common cause of prematurity. The only cure is delivery. As proteinuria may be a late manifestation of pre-eclampsia, it should be suspected when *de novo* hypertension is accompanied by headache, visual disturbances, abdominal pain, or abnormal laboratory tests, specifically low platelets and/or abnormal liver function. (5)
- **Pre-existing hypertension plus superimposed gestational hypertension with proteinuria.**
  - **Antenatally unclassifiable hypertension:** this term is used when BP is first recorded after 20 weeks of gestation and hypertension is diagnosed; re-assessment is necessary after 42 days post-partum.

#### • Prevention of Hypertension and Pre-Eclampsia

Women at high or moderate risk of pre-eclampsia should be advised to take 100–150 mg of aspirin daily from week 12 to weeks 36–37.

High risk of pre-eclampsia includes any of the following:

- hypertensive disease during a previous pregnancy
- chronic kidney disease

- autoimmune disease such as systemic lupus erythematosus or antiphospholipid syndrome
- type 1 or type 2 diabetes
- Chronic hypertension.
- Moderate risk of pre-eclampsia includes more than one of the following risk factors:
  - first pregnancy
  - age 40 years or older
  - pregnancy interval of more than 10 years
  - BMI of  $\geq 35$  kg/m<sup>2</sup> at first visit
  - family history of pre-eclampsia
  - Multiple pregnancy.

Calcium supplementation (1.5–2 g/day, orally) is recommended for the prevention of pre-eclampsia in women with low dietary intake of calcium ( $<600$  mg/day), to be commenced at the first antenatal clinic.

Vitamins C and E do not decrease pre-eclampsia risk; on the contrary, they are more frequently associated with a birth weight  $<2.5$  kg and adverse perinatal outcomes. (6)

#### Management of Hypertension in Pregnancy

##### • Background

Management of hypertension in pregnancy depends on the BP, gestational age, and the presence of associated maternal and foetal risk factors. Most women with pre-existing hypertension and normal renal function have nonsevere hypertension (140–159/90–109 mmHg) and are at low-risk for cardiovascular complications. Some are able to withdraw their medication in the first half of pregnancy because of the physiological fall in BP. Evidence-based data regarding treatment of hypertension in pregnancy are lacking. The only trial of treatment of hypertension in pregnancy with adequate infant follow-up (7.5 years) was performed 40 years ago with  $\alpha$ -methyldopa. (7)

In terms of treatment benefit, tight vs. less-tight control of hypertension in pregnancy in the Control of Hypertension in Pregnancy Study was associated with less severe maternal hypertension, but no difference in the risk of adverse perinatal outcomes and overall serious maternal complications. However, a secondary analysis of the data showed that women developing severe hypertension had higher rates of adverse maternal (pre-eclampsia, platelets  $<100 \times 10^9/L$ , elevated liver enzymes with symptoms, and maternal length of hospital stay  $\geq 10$  days) and perinatal outcomes (perinatal death, high-level neonatal care for  $>48$  h, birth weight  $<10$ th percentile, pre-eclampsia, and pre-term delivery). Thus, there is no evidence currently supporting target BP values in pregnancy. (8)

##### • Non-pharmacological management

Non-pharmacological management of hypertension during pregnancy has a limited role to play, with randomized studies of dietary and lifestyle interventions showing minimal effects on pregnancy outcome. Regular exercise might be continued with caution and obese women

( $\geq 30 \text{ kg/m}^2$ ) are advised to avoid a weight gain of more than 6.8 kg.(9)

- **Pharmacological management**

While the goal of treating hypertension is to reduce maternal risk, the agents selected must be effective and safe for the foetus.

- **Treatment of severe hypertension**

There is no agreed definition of severe hypertension, with values ranging between 160–180 mmHg to  $>110$  mmHg. This Task Force recommends considering an SBP  $\geq 170$  mmHg or DBP  $\geq 110$  mmHg in a pregnant woman an emergency, and hospitalization is indicated. The selection of the antihypertensive drug and its route of administration depend on the expected time of delivery. ACE inhibitors, ARBs, and direct renin inhibitors are strictly contraindicated (see section 12). Pharmacological treatment with i.v. labetalol, oral methyldopa, or nifedipine should be initiated; i.v. hydralazine is no longer the drug of choice as its use is associated with more perinatal adverse effects than other drugs. However, hydralazine is still commonly used when other treatment regimens have failed to achieve adequate BP control as most obstetricians find its side effect profile acceptable. Use of i.v. urapidil can also be considered. Sodium nitroprusside should only be used as the drug of last choice since prolonged treatment is associated with an increased risk of foetal cyanide poisoning. The drug of choice when pre-eclampsia is associated with pulmonary oedema is nitroglycerin (glyceryl trinitrate), given as an i.v. infusion of  $5 \mu\text{g/min}$ , and gradually increased every 3–5 min to a maximum dose of  $100 \mu\text{g/min}$ .(10)

- **Treatment of mild–moderate hypertension**

Despite a lack of evidence, the European Guidelines recommend the initiation of drug treatment in all women with persistent elevation of BP  $\geq 150/95$  mmHg and at values  $>140/90$  mmHg in women with:

- gestational hypertension (with or without proteinuria)
- pre-existing hypertension with the superimposition of gestational hypertension
- Hypertension with subclinical organ damage or symptoms at any time during pregnancy.

Methyldopa, beta-blockers (most data available for labetalol), and calcium antagonists (most data available for nifedipine) are the drugs of choice. Betablockers appear to be less effective than calcium antagonists and may induce foetal bradycardia, growth retardation, and hypoglycaemia; consequently, their type and dose should be carefully selected, with atenolol best avoided. Women with pre-existing hypertension may continue their current antihypertensive medication unless on ACE inhibitors, ARBs, and direct renin inhibitors, which are contraindicated due to adverse foetal and neonatal outcomes. The plasma volume is reduced in pre-eclampsia, therefore diuretic therapy is best avoided unless in the context of oliguria, when low-dose furosemide may be considered. Delivery of i.v. magnesium sulfate is recommended for the prevention of eclampsia and treatment of seizures, but should not be given

concomitantly with CCBs (there is a risk of hypotension due to potential synergism).(11)

- **Delivery**

Delivery is indicated in pre-eclampsia with visual disturbances or haemostatic disorders, and at 37 weeks in asymptomatic women.(12)

- **Prognosis after pregnancy**

- **Blood pressure post-partum**

Post-partum hypertension is common in the first week. Methyldopa should be avoided because of the risk of post-partum depression.

- **Hypertension and lactation**

Breastfeeding does not increase BP in the nursing mother. Cabergoline, rather than bromocriptine, is recommended for lactation suppression. However, there is some evidence that bromocriptine might be beneficial in PPCM, although it may induce hypertension.(13)

All antihypertensive agents taken by the nursing mother are excreted into breast milk. Most of the antihypertensive drugs are present at very low concentrations, except for propranolol and nifedipine, which have breast milk concentrations similar to those in maternal plasma.(14)

- **Risk of recurrence of hypertensive disorders in a subsequent pregnancy**

Women experiencing hypertension in their first pregnancy are at increased risk in a subsequent pregnancy. The earlier the onset of hypertension in the first pregnancy, the higher the risk of recurrence in a subsequent pregnancy.(15)

- **Long-term cardiovascular consequences of gestational hypertension**

Women who develop gestational hypertension or pre-eclampsia are at increased risk of hypertension, stroke, and ischaemic heart disease in later adult life. Lifestyle modifications are primarily indicated to avoid complications in subsequent pregnancies and to reduce maternal cardiovascular risk in the future. Therefore, annual visits to a primary care physician to check BP and metabolic factors are recommended.(16)

- **Fertility Treatment**

There is no clear evidence that fertility treatment increases the risk of hypertension or pre-eclampsia.

**Recommendations for the management of hypertension**

Recommendations	Class*	Level <sup>†</sup>
Low-dose aspirin (100–150 mg daily) is recommended in women at high or moderate risk of pre-eclampsia from week 12 to weeks 36–37. <sup>141,144</sup>	I	A
In women with gestational hypertension or pre-existing hypertension superimposed by gestational hypertension, or with hypertension and subclinical organ damage or symptoms, initiation of drug treatment is recommended at SBP >140 mmHg or DBP >90 mmHg. <sup>152</sup> In all other cases, initiation of drug treatment is recommended if SBP ≥150 mmHg or DBP ≥95 mmHg. <sup>148,175</sup>	I	C
SBP ≥170 mmHg or DBP ≥110 mmHg in a pregnant woman is an emergency, and hospitalization is recommended.	I	C
Methyldopa (B), labetalol (C), and calcium antagonists (C) are recommended for the treatment of hypertension in pregnancy. <sup>11,179,389</sup>	I	B (methyldopa) C (labetalol and calcium antagonists)
In women with gestational hypertension or mild pre-eclampsia, delivery is recommended at 37 weeks. <sup>163</sup>	I	B
It is recommended to expedite delivery in pre-eclampsia and with adverse conditions such as visual disturbances or haemostatic disorders.	I	C
In pre-eclampsia associated with pulmonary oedema, nitroglycerin given as an intravenous infusion is recommended. <sup>161</sup>	I	C
In severe hypertension, drug treatment with intravenous labetalol, or oral methyldopa or nifedipine, is recommended. <sup>11</sup>	I	C
Limitation of weight gain to <6.8 kg should be considered in obese women. <sup>177</sup>	IIa	C
ACE inhibitors, ARBs, or direct renin inhibitors are not recommended. <sup>11,183,361</sup>	III	C

ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; BP = blood pressure; DBP = diastolic blood pressure; SBP = systolic blood pressure.  
\*Class of recommendation.  
†Level of evidence.

**Frequency**

**• Overall Frequency:**

The frequency of high blood pressure and pregnancy in our hospital practice is currently on the rise in recent years due to the increasingly medical surveillance of pregnant women. According to reports from the World Health Organization (WHO), this frequency ranges between 0.1% and 17.5% of pregnancies.(17)

These wide intervals are explained not only by the heterogeneity of the populations studied but also by the difference in the criteria used to define pregnancy-induced hypertension. According to the results of the literature, the incidence of hypertension and pregnancy is:

It is estimated between 5 and 10% in the United States, 10 to 15% in France, 9% in China. In our study we have a frequency of 9.2.(18)

**• Frequency of Maternal Complications**

Complications induced by high blood pressure during pregnancy are a source of mortality and morbidity for the mother and her fetus. The retro-placental hematoma, the neurological disorders dominated by eclampsia, coagulation disorders, Hellp syndrome, liver, pulmonary and renal accidents are the essential complications of this pathology. The authors describe them separately but they are the more often associated. Faced with these complications, a vigorous assessment of the severity of maternal-fetal status is required. The management of these patients requires the collaboration of the anesthesiologist - resuscitator, obstetrician, pediatrician and cardiologist.

The study of the evolution of the patients of our series allowed us to note 80 cases (14, 2%) which presented / displayed at least one complication.(19)

**• Maternal Mortality**

In recent years, maternal mortality has been reduced, particularly in countries with high socio-economic status,

thanks to a better understanding of the early severity factors of severe arterial hypertension and also to the progress of resuscitation. Cases of maternal death reported in the literature occur either in a context of poorly followed pregnancy or during an accident difficult to predict. The quality of care and management different according to the degree of development of the health structure of the countries can explain the variability of the values found thus the maternal mortality secondary to the arterial hypertension and these complications varies between 0 and 31% in Literature. In our series, we found three maternal deaths secondary to hemorrhagic disorders occurring in a context of eclampsia.(20)

**• Eclampsia**

A frequent and serious complication of vascular-renal syndromes, it represents a real public health problem all over the world. Eclampsia is defined by the occurrence in a context of gravid arterial hypertension of a generalized seizure, single or repeated epilepticus or a state of epilepticus, it is usually preceded in (80% of cases) a phase severe pre-eclampsia, marked by rapid worsening of arterial hypertension, proteinuria and minor neurological disorders such as: headache, visual disturbances, tinnitus, osteotendinous hyper-reflexivity.(21)

The incidence of eclampsia in relation to the total number of deliveries varies greatly from one country to another depending on the level of antenatal care of pregnancies. In our institution, this complication remains frequent compared to the series of developed countries or those of some African countries with a frequency of 4.4% of all hypertension and pregnancy and 3.7 in our study.(22)

**• Placental Retro Hematoma**

The retro-placental hematoma is the premature detachment of a placenta normally inserted, it is an acute haemorrhagic stroke of the third quarter of sudden onset, unpredictable, not preceded by prodromes unlike eclampsia and therefore difficult to prevent. The frequency of the retro-placental hematoma variously appreciated by the authors according to the mode of diagnosis, is between 2 to 7%. Although prevention has improved the incidence of eclampsia, the authors did not find any influence on the occurrence of retro-placental hematoma. In our series, the retro-placental hematoma accounted for 5.1% among all cases, our results seem similar to those found in the literature.(23)

**• Hellp Syndrome**

Hellp syndrome is a rare but serious complication of high blood pressure. It occurs in 2 to 12% of pre-eclamptic and surviving pregnancies, usually in the third trimester of pregnancy with an average gestational age of 34 weeks of amenorrhoea (21 to 40 weeks of analyzes). It consists of the combination of 3 biological phenomena: Hemolysis, hepatic cytolysis and thrombocytopenia. In fact, there is a disparity in the definitions of Hellp Syndrome. In our series, we found 2 cases of Hellp syndrome or 0.4%. A rate that remains too low compared to the data of the literature. All authors recognize that Hellp syndrome has a severe maternal and

fetal prognosis. This syndrome is accompanied by a high maternal mortality between 0 and 24% according to the authors and a very important fetal mortality which is between 6.7 and 50% depending on the number of cases examined. Most authors currently believe that the Hellp syndrome is a barrier to the continuation of pregnancy. However, neonatal complications are largely secondary to prematurity which is why some authors defend the conservative attitude.(24)

#### • Acute Renal Insufficiency

Extremely rare complication, acute renal failure is most often found in advanced forms of gestational hypertension, 15% in eclampsia, 10% in retro-placental hematoma and 36% in Hellp syndrome. Physiopathologically, this acute renal failure is related to tubular or cortical necrosis secondary to glomerular lesions endotheliosistype. It is characterized by swelling of endothelial cells, mesangial fibrin deposits and immunoglobulins. Moderate functional renal failure is common in severe forms of arterial hypertension during pregnancy its essential feature is its total reversibility after childbirth. Under the term acute renal failure, we grouped functional and organic renal insufficiency. In our series, acute renal failure complicated 4 cases (0.7%) of all parturients with high blood pressure during pregnancy. On the other hand, acute renal failure has not been responsible for maternal or perinatal mortality in our series. It is important to associate a hemodynamic study during Oligo-Anuria, which avoids volemic expansions. Cause of heart failure and death. Finally, we could not specify the cause of acute renal failure in our parturients.(25)

#### • Disseminated Intravascular Coagulation (DIC)

During arterial hypertension and pregnancy, appear first biological and clinical stigmata of disseminated intravascular coagulation, most often its expression is minor, but it can be severe as in the retro-placental hematoma, the eclampsia, the Hellp syndrome or in case of fetal death in-utero. A haemorrhagic syndrome and biologically frank thrombocytopenia, a drop in fibrinogen and plasma levels of haemostasis factors, as well as the presence of fibrin degradation products, are observed clinically. Disseminated intravascular coagulation has been the initiator mechanism of Hellp syndrome according to some authors. For others, disseminated intravascular coagulation would be secondary to the Hellp syndrome may be through intercurrentretroplacental hematoma.(26)

All of these disorders involve maternal and often fetal vital prognosis and involve the immediate termination of pregnancy, cesarean section is the usual method of extraction. The incidence of disseminated intravascular coagulation in our series is 5 cases (1%), our low percentage compared to the rates calculated by most authors is probably explained by our recruitment criteria.(27)

#### • Acute Pulmonary Edema

Pulmonary edema is observed during severe forms of arterial hypertension during pregnancy in older multiparous women with chronic arterial hypertension, and during the

postpartum period when compartment redistribution occurs. Fluidity of the body is a contributing factor. The main mechanisms that can lead to the development of acute pulmonary edema are described by the authors:

- Decrease in plasma oncotic pressure due to significant blood depression during delivery.
  - The increase of the capillary pressure by an overload in infusion.
  - A lesion of the endothelium of the capillaries because of the increase in permeability, that of the interstitial oncotic pressure.
  - Cardiogenic causes related to an abnormality of myocardial contractility.
- Iatrogenic origins is also to be feared when corticosteroid treatment is combined with infusion overload. In our series, we did not note any cases of acute pulmonary edema. (28)

#### • Frequency of Fetal Complications

High blood pressure during gestational pregnancy is still a common cause of prenatal mortality (10-25%) and fetal morbidity despite advances in maternal and neonatal resuscitation. In our series, of the 574 children born to mothers with high blood pressure who gave birth at the LallaMeryem maternity hospital at Ibn Rochd University Hospital, we found 413 newborns with a favorable evolution (75.9%) and 102 newborns (18.7%) who had at least one complication, this figure remains intermediate compared to those found in the literature.(29)

#### ▪ Perinatal Mortality

Perinatal mortality includes both stillbirth (deaths or deaths beyond 28 weeks of amenorrhea and before birth) and early neonatal mortality (born alive and died before 7 days of age). Fetal mortality is the usual consequence of acute placental ischemia, and neonatal mortality is related to either acute pre-, post- or postpartum anoxia, or prematurity. In our series, perinatal mortality represents 57 cases (9.9%) among the 574 born with 42 cases (7.3%) of in-utero fetal death and 15 cases (2.6%) of neonatal mortality. remain higher compared to some authors and intermediate to others. This is because patients often consult at obstetric disaster stages. In addition, there is the absence of prenatal consultations and adequate care.(30)

#### ▪ Prematurity

It is the birth before 37 weeks of gestation. Prematurity is one of the main causes of neonatal morbidity and mortality, secondary to respiratory, cerebral, digestive and immune immaturity. This is, in general, an induced prematurity. Indeed, fetal extraction can be decided in case of maternal rescue (Hellp syndrome, eclampsia, severe arterial hypertension), or fetal rescue (severe hypotrophy, abnormal fetal heart rate) the prognosis of these premature babies depends mainly on gestational age and is dominated by still significant perinatal mortality despite progress in management. (31)

In our study, prematurity comes second with an incidence of 13.8% of all newborns after acute fetal distress, this rate is consistent with many other studies conducted both in our country and elsewhere. In our series, it was defined as the 1st cause of perinatal mortality 164 cases (28.6%). (32)

#### ▪ **Intrauterine Growth Retardation**

It complicates 7 to 20% of pregnancies with high blood pressure. Its appearance is most often late in the third trimester of pregnancy. It is often accompanied by prematurity. Fetal hypotrophy is defined by a fetal term weight of less than 2500 g or a weight less than the 10th percentile curve for premature infants. The risk of occurrence of hypotrophy has been evaluated, according to some authors according to 3 maternal parameters:

The level of diastolic pressure (the risk is multiplied by 5 for a diastolic blood pressure > 110 mm Hg). The date of onset of pregnancy-induced hypertension (the risk increases between 28 and 35 weeks of gestation). Two types of intrauterine growth retardation can be seen. Most often, it is segmental or disharmonious but more rarely it can be harmonious in early attacks. (33)

The detection of intrauterine growth retardation is done clinically by the measurement of the uterine height, echoed graphically by the measurement of fetal biometrics (biparietal diameter, femur length and antero-transverse diameter), Doppler allows a hemodynamic study. The fetus and the uteroplacental circulation, its applications are diagnostic, etiological and therapeutic. In our series, hypotrophy accounted for 47 cases (7.5%) of all children born to mothers with high blood pressure during pregnancy. Our low percentage compared to literature data is probably explained by our recruitment criteria, the lack of early detection of intrauterine growth retardation and the insufficiency in fetal monitoring during hospitalization. (34)

#### • **Therapeutic Attitude**

Because of the physiopathological circumstances of the onset of arterial hypertension during pregnancy, it is understood that antihypertensive treatment is only a symptomatic treatment of this pathology and the best treatment remains fetal extraction. Such a decision balances most often the risk of prematurity and the severity of the maternal state.

#### • **Goal**

In arterial hypertension and pregnancy, the goal of treatment is twofold, maternal and fetal; to protect the mother from life-threatening complications and to put the fetus in the best conditions of survival until sufficient maturity is reached.

#### • **Therapeutic Means**

There is still no specific treatment for high blood pressure during pregnancy, it is currently symptomatic.

#### ▪ **Hygiene and Dietary Measures**

Hospitalization is usually necessary. Although brief, it allows the maternofetal situation to be classified according to severity. An outpatient treatment is conceivable only in mild arterial hypertension without biological anomalies and without fetal damage under strict surveillance. In our series, hospitalization was routine in any patient with high blood pressure during pregnancy. Strict bed rest and quietness is advocated by most authors and its usefulness is beyond doubt. If possible in left lateral decubitus which improves placental and renal perfusions as well as maternal hemodynamics. In fact, psychic rest is just as important as physical rest.

The diet must be normal in salt and calories, the diet is not indicated because it aggravates the relative hypovolemia inherent in this pathology, and caloric restriction can promote fetal hypotrophy. In our series, dietary and lifestyle measures were the rule in all our parturients, alone or in combination with antihypertensive treatment, they were sufficient in 64 cases (11.8%). (35)

#### ▪ **Medication treatments**

In case of arterial hypertension during pregnancy, especially during preeclampsia, the prescription of an antihypertensive is relatively limited. The molecule, the dosage and the route of administration depend on the severity of the arterial hypertension, the therapeutic urgency and the existence of associated maternal complications. The use of antihypertensive drugs is indicated in case of rest failure and dietary measures. The lowering of the blood pressure should be gradual and moderate so as not to alter the utero-placental blood flow. (36)

The ideal antihypertensive drug for use during high blood pressure during pregnancy should have consistent, rapid, titratable efficacy, a high therapeutic index, and no toxic effects for the mother and fetus. It should not expose to a rebound effect when treatment is stopped. Finally, it should be available in intravenous form for attack and oral treatment for relaying. Few antihypertensives meet so many requirements. (37)

#### ▪ **Indications**

For maternal and / or maternofetal rescue depending on the term. Similarly, in case of suffering When the arterial hypertension during the pregnancy is moderate (systolic blood pressure <160mmHg, diastolic blood pressure <100mmHg and proteinuria <at 1g / 24heures) a hospitalization is desirable for the realization of a balance sheet and initiation of antihypertensive treatment initiated by low doses. Blood pressure should be monitored twice a week, uric acid dosing should be done weekly, and fetal ultrasound every 15 days. In cases of severe hypertension, hospitalization and antihypertensive treatment are required, all antihypertensive agents may be used (unless contraindicated) at gradually increasing doses. Always start as far as possible with mono therapy. (38)

Fetal maternal surveillance should be rigorous (including a fetal heart rate three times a day). Two situations can then arise. When arterial hypertension is controlled, surveillance is maintained until delivery, which can be vaginal in the event of a term  $\geq 36$  weeks of amenorrhea, fetal weight  $> 2000g$ , in the absence of fetal distress and if obstetric conditions allow. When the situation is not perfectly controlled, childbirth is desired as soon as possible. Thus, a caesarean section is performed for the terms  $> 28$  weeks of amenorrhea. Conservative treatment is justified only when the risk of prematurity is deemed too high, but it should not put the mother at excessive risk. (39)

▪ **Indications for termination of pregnancy may be**

▪ **Maternal**

- Eclampsia
- Pre-eclampsia severe uncontrolled and / or complicated disorders of consciousness, hematological abnormalities, HELLP syndrome or oliguria resistant to filling.
- Fetal order
- Acute fetal distress
- Hypotrophy with no growth on ultrasound.

▪ **Choose the route of delivery in case of hypertension during pregnancy**

- If the fetus is dead, the work is triggered after administration of prostaglandins in the cervix, if the work proves long and difficult, if the cardiovascular state of the mother is poorly controlled and if there is a risk major retro-placental hematoma, a caesarean section is performed. From 36 weeks of amenorrhea: trigger when the obstetric situation is very favorable. In the other cases: either the extraction is urgent (or the fetal and / or maternal state do not allow to consider the low way) and it is a caesarean section that will be made, either the interruption of the pregnancy can be delayed for 24 to 48 hours and artificial ripening of the uterine cervix may be used, the purpose of which is to secondarily trigger under favorable conditions. Before 36 weeks of amenorrhea, it is most often cesarean section that is the only acceptable method, especially since these are often fragile fetuses. In some cases, beyond 34 weeks of amenorrhea, if the cervical conditions are very favorable, the fetus in cephalic presentation in a multiparous, we could consider a trigger of work. (40)

## V. MONITORING AND PREVENTION

### Monitoring

• **Monitoring Pregnancy**

In order to implement the first therapeutic measures as soon as necessary: rest or medication, any hypertension during pregnancy, even isolated and moderate should be given special attention.

This surveillance is based on clinical elements:

- Regular intake of blood pressure.

- Regular weight gain.
- Search for edema of the lower limb and / or the face.
- Measurement of uterine height.
- Appreciation of active fetal movements.
- Auscultation of the fetal cardiac sound with Pinard stethoscope.

And on para-clinical examinations none of which has any absolute diagnostic value, but which all have a very great prognostic value: proteinuria, serum uric acid, haemostasis assessment, a blood count. Ultrasound, Doppler examination and fetal heart rate recording are of great importance especially in the assessment of fetal prognosis. In our series, the notion of prenatal consultation was known in only 180 cases (33.1%). In the absence of follow-up of the pregnancy, there was a high rate of fetal and maternal complications, the non-follow-up of pregnancy was a factor of bad prognosis as well maternal as fetal. (41)

• **Post Partum Surveillance**

The risk of complications of arterial hypertension persists, even increases in the consequences of childbirth. The occurrence of acute pulmonary edema, acute renal failure, disseminated intravascular coagulation or eclampsia crisis is always possible especially during the first three days, sometimes up to a week after delivery. It is therefore advisable to maintain a close surveillance during this period (blood pressure every 4 hours, diuresis, weight, proteinuria ...). Further treatment is to be continued. A study of renal function, hemogram and hemostasis assessment is performed before discharge. Antihypertensives can be decreased in a time varying from one woman to another. After discharge, a dosage of proteinuria and regular blood pressure checks are recommended until their normalization. If arterial hypertension or proteinuria persist, nephrology management is necessary. In all cases, medium and long-term blood pressure monitoring is mandatory because 10% to 20% of patients will remain or become permanent hypertensives and must be treated. Moreover, regardless of blood pressure values, women who had eclampsia or pre-eclampsia during the first pregnancy more often develop obstetric complications in subsequent pregnancies (higher number of retro-placental hematoma, premature infants, hypotrophy and neonatal mortality) and have a significantly higher rate of chronic hypertension than normotensive pregnant women during their first pregnancy. (43)(42).

## VI. CONCLUSION

This study concluded that high blood pressure and pregnancy is a common condition and remains a major cause of maternofetal mortality and morbidity. These complications can be reduced by a better physiopathological understanding and a better knowledge of the fetal and maternal prognostic factors. This condition mobilizes obstetricians and researchers looking for effective treatment. The only truly effective treatment is the termination of pregnancy, all other therapies are intended only to prolong the pregnancy to a term acceptable to the fetus. The

prevention of placental insufficiency by the early management of risky parturients (rigorous clinical, biological, ultrasound and velocimetric monitoring, treatment with platelet antiaggregants at low doses) and the rapid and adapted treatment of hypertension and pregnancy reported, notably thanks to the progress of neonatal resuscitation, contribute to the improvement of the results already recorded. Finally, any doctor should be alert to the discovery of a blood pressure higher than normal or any abnormality (even minimal) occurring in a pregnant woman.

### SUMMARY

Arterial hypertension and pregnancy is common and remains a leading cause of maternal and fetal mortality and morbidity. The aim of this work is to study the peculiarities of this high-risk pregnancy. This is a retrospective study of 544 cases of hypertension and pregnancy collected at the LallaMeryem maternity hospital Ibn Rochd hospital in Casablanca for a period of 2 years. The incidence is 9.2%. The average age of onset was 30 years with an age range of 15 to 45 years. The primiparous were the most exposed 261 cases (48%). 310 cases (57%) have an unguarded pregnancy. 290 cases (53.3%) had a systolic blood pressure greater than or equal to 160 mm Hg, and 160 cases (29.4%) had a diastolic blood pressure  $\geq$  110 mmHg. The most used medical conduct was the combination of rest and antihypertensives. Obstetrical behavior was marked by the frequency of vaginal deliveries (63.4%). Maternal complications represent (14.7%) dominated by retro-placental hematoma (5.1%) and eclampsia (3.7%). Perinatal mortality represents 57 cases (9.9%). The factors of bad foeto-maternal prognosis are for the fetus: the low gestational age, the low parity, the non-monitoring of the pregnancy, the massive proteinuria and the hyperuricemia. For the mother, young maternal age, prim parity, non-pregnancy status, diastolic blood pressure  $\geq$  110 mm Hg, systolic blood pressure  $\geq$  160 mm Hg, and massive proteinuria. Careful monitoring of pregnancies, early diagnosis of high blood pressure and better management of the mother and child, as well as a better knowledge of the fetal and maternal prognostic factors, contribute to the reduction of complications of arterial hypertension during pregnancy.

### REFERENCES

- [1]. Villar J, Carroli G, Wojdyla D, Abalos E, Giordano D, Ba'aqee, et al. (2006) Preeclampsia, gestational hypertension and intrauterine growth restriction, related or independent conditions? *Am J Obstet Gynecol* 194:921-194931.
- [2]. National high blood pressure education program working group report on high blood pressure in pregnancy. (1990) *Am J Obstet Gynecol* 163:1691-1712.
- [3]. Levine RJ, Ewell MG, Hauth JC, Curet LB, Catalano PM, et al. (2000) Should the definition of preeclampsia include a rise in diastolic blood pressure of  $\geq$ 15 mm Hg to a level  $<$ 90 mm Hg in association with proteinuria? *Am J Obstet Gynecol* 183: 787-792.

- [4]. Mancina G, Fagard R, Narkiewicz K, Redon J, Zanchetti A, et al. (2013) 2013 ESH/ESC Guidelines for the management of arterial hypertension: the Task Force for the management of arterial hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology. *J Hypertens* 2013; 31:1281–1357.
- [5]. dabl@Educational Trust. [www.dablededucation.org](http://www.dablededucation.org).
- [6]. Blood Pressure UK. <http://www.bloodpressureuk.org>.
- [7]. Penny JA, Halligan AW, Shennan AH, Lambert PC, Jones DR, et al. (1998) Automated, ambulatory, or conventional blood pressure measurement in pregnancy: Which is the better predictor of severe hypertension? *Am J Obstet Gynecol* 178: 521-526.
- [8]. Magee LA, Ramsay G, von Dadelszen P (2008) What is the role of out-of-office BP measurement in hypertensive pregnancy? *Hypertens Pregnancy* 27: 95-101.
- [9]. Schmella MJ, Clifton RG, Althouse AD, Roberts JM (2015) Uric acid determination in gestational hypertension: Is it as effective a delineator of risk as proteinuria in high-risk women? *Reprod Sci* 22: 1212-1219.
- [10]. Cade TJ, de Crespigny PC, Nguyen T, Cade JR, Umstad MP (2015) Should the spot albumin-to-creatinine ratio replace the spot protein-to-creatinine ratio as the primary screening tool for proteinuria in pregnancy? *Pregnancy Hypertens* 5: 298-302.
- [11]. Chappell LC, Shennan AH (2008) Assessment of proteinuria in pregnancy. *BMJ* 336: 968-969.
- [12]. Cote AM, Firoz T, Mattman A, Lam EM, von Dadelszen P, et al. (2008) The 24-hour urine collection: Gold standard or historical practice? *Am J Obstet Gynecol* 199: 625 e621-626.
- [13]. Cnossen JS, Morris RK, ter Riet G, Mol BW, van der Post JA, et al. (2008) Use of uterine artery Doppler ultrasonography to predict pre-eclampsia and intrauterine growth restriction: A systematic review and bivariable metaanalysis. *CMAJ* 178: 701-711.
- [14]. Zeisler H, Llorba E, Chantraine F, Vatish M, Staff AC, et al. (2016) Predictive value of the sFlt-1:PIGF ratio in women with suspected preeclampsia. *N Engl J Med* 374: 13-22.
- [15]. Leanos-Miranda A, Campos-Galicia I, Isordia-Salas I, Rivera-Leanos R, Romero-Arauz JF, et al. (2012) Changes in circulating concentrations of soluble fms-like tyrosine kinase-1 and placental growth factor measured by automated electrochemiluminescence immunoassays methods are predictors of preeclampsia. *J Hypertens* 30: 2173-2181.
- [16]. American College of Obstetricians and Gynecologists; Task Force on Hypertension in Pregnancy. Hypertension in pregnancy (2013). Report of the American College of Obstetricians and Gynecologists' Task Force on Hypertension in Pregnancy. *Obstet Gynecol* 122: 1122-1131.
- [17]. Magee LA, Pels A, Helewa M, Rey E, von Dadelszen P (2014) Diagnosis, evaluation, and management of the hypertensive disorders of pregnancy. *Pregnancy Hypertens* 4: 105-145.

- [18]. Lowe SA, Bowyer L, Lust K, McMahon LP, Morton MR, et al. (2015) The somanz guidelines for the management of hypertensive disorders of pregnancy 2014. *Aust N Z J Obstet Gynaecol* 55: e1-29.
- [19]. Bartsch E, Medcalf KE, Park AL, Ray JG (2016) High Risk of Pre-eclampsia Identification Group. Clinical risk factors for pre-eclampsia determined in early pregnancy: Systematic review and meta-analysis of large cohort studies. *BMJ* 353: i1753.
- [20]. National Collaborating Centre for Women's and Children's Health (UK). Hypertension in pregnancy: The management of hypertensive disorders during pregnancy. London: RCOG Press; 2010. Rolnik DL,
- [21]. Rolnik DL, Wright D, Poon LC, O'Gorman N, Syngelaki A, et al. (2017) Aspirin versus placebo in pregnancies at high-risk for preterm preeclampsia. *N Engl J Med* 377: 613-622.
- [22]. Hofmeyr GJ, Lawrie TA, Atallah AN, Duley L, Torloni MR (2014) Calcium supplementation during pregnancy for preventing hypertensive disorders and related problems. *Cochrane Database Syst Rev* 6: CD001059.
- [23]. Xu H, Perez-Cuevas R, Xiong X, Reyes H, Roy C, et al. (2010) An international trial of antioxidants in the prevention of preeclampsia (INTAPP). *Am J Obstet Gynecol* 202: 239.e1-239.e10.
- [24]. Villar J, Purwar M, Merialdi M, Zavaleta N, Thi Nhu Ngoc N, et al. (2009) World Health Organisation multicentre randomised trial of supplementation with vitamins C and E among pregnant women at high-risk for pre-eclampsia in populations of low nutritional status from developing countries. *BJOG* 116: 780-788.
- [25]. Spinnato JAII, Freire S, Pinto ESJL, Cunha Rudge MV, Martins-Costa S, et al. (2007) Antioxidant therapy to prevent preeclampsia: A randomized controlled trial. *Obstet Gynecol* 110: 1311-1318.
- [26]. Poston L, Briley AL, Seed PT, Kelly FJ, Shennan AH (2006) Vitamin C and vitamin E in pregnant women at risk for pre-eclampsia (VIP trial): Randomised placebo-controlled trial. *Lancet* 367: 1145-1154.
- [27]. Redman CW (1976) Fetal outcome in trial of antihypertensive treatment in pregnancy. *Lancet* 2: 753-756.
- [28]. Cockburn J, Moar VA, Ounsted M, Redman CW (1982) Final report of study on hypertension during pregnancy: The effects of specific treatment on the growth and development of the children. *Lancet* 1: 647-649.
- [29]. Magee LA, von Dadelszen P, Rey E, Ross S, Asztalos E, et al. (2015) Less-tight versus tight control of hypertension in pregnancy. *N Engl J Med* 372: 407-417.
- [30]. Magee LA, von Dadelszen P, Singer J, Lee T, Rey E, et al. (2016) The CHIPS randomized controlled trial (Control of Hypertension in Pregnancy Study): Is severe hypertension just an elevated blood pressure? *Hypertension* 68: 1153-1159.
- [31]. Abalos E, Duley L, Steyn DW (2014) Antihypertensive drug therapy for mild to moderate hypertension during pregnancy. *Cochrane Database Syst Rev* 2: CD002252.
- [32]. Dodd JM, Turnbull D, McPhee AJ, Deussen AR, Grivell RM, et al. (2014) Antenatal lifestyle advice for women who are overweight or obese: LIMIT randomised trial. *BMJ* 348: g1285.
- [33]. Leddy MA, Power ML, Schulkin J (2008) The impact of maternal obesity on maternal and fetal health. *Rev Obstet Gynecol* 1:170-178.
- [34]. Magee LA, Cham C, Waterman EJ, Ohlsson A, von Dadelszen P (2003) Hydralazine for treatment of severe hypertension in pregnancy: Meta-analysis. *BMJ* 327: 955-960.
- [35]. Vigil-De Gracia P, Lasso M, Ruiz E, Vega-Malek JC, de Mena FT, et al. (2006) Severe hypertension in pregnancy: Hydralazine or labetalol. A randomized clinical trial. *Eur J Obstet Gynecol Reprod Biol* 2006;128:157-162.
- [36]. Shekhar S, Gupta N, Kirubakaran R, Pareek P (2016) Oral nifedipine versus intravenous labetalol for severe hypertension during pregnancy: A systematic review and meta-analysis. *BJOG* 123: 40-47.
- [37]. Clark SM, Dunn HE, Hankins GD (2015) A review of oral labetalol and nifedipine in mild to moderate hypertension in pregnancy. *Semin Perinatol* 39: 548-555.
- [38]. Altman D, Carroli G, Duley L, Farrell B, Moodley J, et al. (2002) Do women with pre-eclampsia, and their babies, benefit from magnesium sulphate? The Magpie Trial: A randomised placebo-controlled trial. *Lancet* 359: 1877-1890.
- [39]. Koopmans CM, Bijlenga D, Groen H, Vijgen SM, Aarnoudse JG, et al. (2009) Induction of labour versus expectant monitoring for gestational hypertension or mild pre-eclampsia after 36 weeks' gestation (HYPITAT): A multicentre, open-label randomised controlled trial. *Lancet* 374: 979-988.
- [40]. Podymow T, August P (2010) Postpartum course of gestational hypertension and preeclampsia. *Hypertens Pregnancy* 29: 294-300.
- [41]. Beardmore KS, Morris JM, Gallery ED (2002) Excretion of antihypertensive medication into human breast milk: A systematic review. *Hypertens Pregnancy* 21: 85-95.
- [42]. Ray JG, Vermeulen MJ, Schull MJ, Redelmeier DA (2005) Cardiovascular health after maternal placental syndromes (CHAMPS): Population-based retrospective cohort study. *Lancet* 366: 1797-1803.
- [43]. Black MH, Zhou H, Sacks DA, Dublin S, Lawrence JM, et al. (2016) Hypertensive disorders first identified in pregnancy increase risk for incident prehypertension and hypertension in the year after delivery. *J Hypertens* 34: 728-735.
- [44]. Wang YA, Chughtai AA, Farquhar CM, Pollock W, Lui K, et al. (2016) Increased incidence of gestational hypertension and preeclampsia after assisted reproductive technology treatment. *Fertil Steril* 105: 920-926 e922.