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Current state of the art in pathophysiology, diagnosis, treatment and post partum care

Hypertension in pregnancy

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Summary

Cardiovascular diseases in pregnancy are increasing, and account for the majority of pregnancy-induced maternal deaths. Among them, hypertensive disorders are the most frequent, affecting 6–8% of all pregnancies.

More severe forms, like preeclampsia and HELLP syndrome, represent serious complications and are associated with far-reaching consequences for mother and child, such as stillbirth, peripartum cardiomyopathy, diastolic heart failure, eclampsia and a long-term increased risk for cardiovascular disease. While risk factors such as obesity, smoking, diabetes mellitus, twin pregnancy, multiparity, advanced age of the mother, in-vitro fertilisation (IVF) are well known, the exact pathophysiology of preeclampsia has still not been precisely clarified.

Therefore, specific treatment options are limited, also in part due to the challenge of performing clinical trials in pregnant women.

The present review provides an overview of the current state of knowledge and summarises treatment strategies and therapy options.

Keywords: Hypertension, pregnancy, preeclampsia, PPCM, risk-factors, pathophysiology, diagnosis, treatment

Introduction

In recent decades, there has been an increase in maternal age, cardiovascular diseases (CVD), and preexisting risk factors among pregnant women (table 1). This is reflected by the number of high-risk pregnancies and requires interdisciplinary care, ideally six months before conception [1-3].

A distinction must be made between women with preexisting hypertension and women who acquire hypertension during pregnancy.

Nevertheless, hypertension is a crucial risk factor for the development of severe complications such as preeclampsia (PE), eclampsia and HELLP syndrome. The present review provides an overview of the hypertensive disorders in pregnancy, their pathophysiology, diagnosis and management.

Preexisting hypertension

Preexisting hypertension and hypertension diagnosed in the first 20 weeks of gestation need close monitoring and intensive care [4]. Patients with chronic hypertension represent a special risk group and their medication and blood pressure values should optimally be adjusted before pregnancy. The switch from potentially embryotoxic drugs to pregnancy-compatible medication is important in the first weeks of gestation (table 3) [5], [6]. Women with congenital heart defects (CHD) represent a special subgroup and should receive prenatal and postnatal care in specialised interdisciplinary centres [7-9]. Early and comprehensive care can preventively influence maternal and foetal outcome and prevent or at least minimise secondary complications [10]. In preparation for pregnancy, the genesis (renal, endocrine, essential) of the CVD must be elicited to ensure optimal individualised care [2, 11]. The recommendations regarding the threshold values in chronic hypertension vary in international guidelines, however it should be aimed for systolic values between 120-160 mmHg and diastolic values between 80-110 mmHg [12]. Higher levels are associated with an increase in pregnancy-associated complications and should be avoided [13]. Overall, women with chronic hypertension experience more maternal (preeclampsia, pulmonary oedema) and foetal complications (intrauterine growth restriction, stillbirth, transfer to neonatal intensive care unit [NICU]). These risks increase significantly if other risk factors, i.e., preexisting renal disease and metabolic disorders, exist [14, 15].

The strongest preventive measure is adequate management of blood pressure, also in absence of risk factors, so that emerging hypertension can be detected and treated early to avoid severe complications for the mother and subsequently for the foetus [16].

Gestational hypertension

Pregnancy-induced hypertension develops during pregnancy with values >140/90 mmHg, severe progression is associated with blood pressure values >160/110 mmHg [17]. Gestational hypertension affects approximately 6-8% of pregnancies [18]. Preexisting risk factors, i.e., obesity, smoking, chronic medication, diabetes mellitus, dyslipidaemia and renal diseases should be registered [19, 20]. The risk for early pregnancy-induced hypertension is increased in women conceived by IVF, especially when frozen embryo transfer was performed [21]. It is accepted, that in IVF, the ovarian stimulation hormone treatment causes endometrial dysplasia, which leads to deficient trophoblastic invasion and defective placenta-

tion. Additionally, the formation of chorion is initiated in vitro, which may lead to a difference in the nature of the placenta, placental vascular lesions and insufficient uteroplacental circulation. Oocyte donation is acknowledged to be an independent risk factor for preeclampsia due to immunological intolerance [22].

Diagnosis of hypertension in pregnancy

Maternal blood pressure should be checked on both arms using an upper arm cuff after an adequate resting phase (five minutes). To prevent inaccurate measurements, an adequate cuff fitting the circumference of the upper arm is recommended [23]. A blood pressure measurement of >140/90 mmHg is considered pathological. In case of elevated values at first the measurement, it should be repeated after an adequate time interval. If results are pathological, blood pressure is measured by the patient at home (at least two measurements per day). If this leads to the suspicion of hypertension, 24-hour blood pressure measurement may be indicated [17]. Women who have white coat hypertension (high blood pressure in the presence of a health care provider) already early during pregnancy have an increased risk of developing manifest hypertension (up to 40%) or preeclampsia (up to 8%) later in gestation [24].

As part of the screening, maternal urine analyses should be carried out regularly using dipsticks for proteinuria. In the case of conspicuous results, quantification using 24-hour collected urine is proposed. Proteinuria >300 mg/d is considered pathological [17]. Further laboratory chemistry parameters are not primarily indicated in hypertensive pregnancy disorders. However, if there is a suspicion of an adverse pregnancy outcome (APO), further laboratory tests are needed to make an adequate diagnosis and initiate targeted treatment strategies (table 2). The determination of the ratio of angiogenic factors (sFlt-1 and PIGF) can be used in addition to the clinical examination in cases of suspected preeclampsia. The PROGNOSIS study showed that a quotient of <38 excludes the occurrence of preeclampsia in the next week. This assessment is essential, especially with regard to the further care of the patient (outpatient/inpatient) and for the assessment of further medical interventions (antenatal corticosteroid therapy, transfer to a perinatal centre) depending on the gestational age. It must be emphasised that the quotient is a supplement to the clinical examination and assessment but does not establish the diagnosis alone [25, 26].

Pathophysiology of gestational hypertension

Pregnancy requires an adaptation of the female body to optimally supply the foetus with nutrients and oxygen and to prepare the body for delivery. To understand the pathophysiology of pregnancy induced hypertension, it is important to explain the physiological changes starting in early pregnancy [27, 28]. Pregnancy changes the maternal homeostasis profoundly, including an increase in blood volume, heart rate and heart size, a switch in energy substrate use from glucose to fatty acids as well as changes in the immune system. These rapid changes are largely controlled by hormones, i.e., progesterone, oestrogen, oxytocin and prolactin, which are mainly but not exclusively produced by the placenta. Progesterone leads to a decrease in total peripheral resistance by lowering the tone of the vascular musculature. This leads to an increase in heart rate, cardiac output and a decrease in blood pressure [29]. After delivery all this needs to be reversed in a controlled manner. It can take up to twelve weeks for peripheral resistance to return to pre-pregnancy values [30]. Thereby, the placenta is a temporary organ of mostly foetal origin which orchestrates the reprogramming of the maternal physiology to suit the needs of the foetus. After delivery, rapid hormonal, metabolic and immunological changes take place in the mother as well as a massive loss of fluid volume sometimes enhanced by bleeding complications. Furthermore, the body prepares to nurse the infant.

The cause of early hypertensive pregnancy disorders is attributed to impairment of the implantation of the placenta. The placenta develops from the trophoblast which infiltrates the maternal spiral arteries via so-called trophoblastic invasion, whereby the endothelial layer of the spiral arteries is replaced by trophoblastic cells (remodelling). It is followed by the loss of vasoregulation and the transformation into large non-muscular channels resulting in an increase in maternal blood flow to the placenta [31]. Trophoblastic invasion occurs in two stages: in a first step the spiral arteries involve in the decidua at eight weeks of gestation, the second invasion of the spiral arteries of the inner third of the myometrium occurs between 14-18 weeks of gestation [32].

In the case of defective trophoblastic invasion, this process does not extend to the myometrial segments, but remains confined to the vessels of the decidua [33]. There is a

Table 1: Risk factors for the development of hypertensive pregnancy disease and severe risk factors [92]

General risk factors	Pregnancy-associated risk factors
Obesity, BMI >30	Multiple pregnancy
Diabetes mellitus	**IVF**
Family disposition	Gestational diabetes mellitus
Preexisting kidney disease	Increased resistance of uterine arteries >24 weeks of gestation
Maternal age >40 years	Chromosomal aberration of the foetus
Autoimmune disease e.g., SLE	**Preeclampsia in previous pregnancy**
Antiphospholipid syndrome	Nulliparous
Chronic hypertension	
Ethnicity, African American	

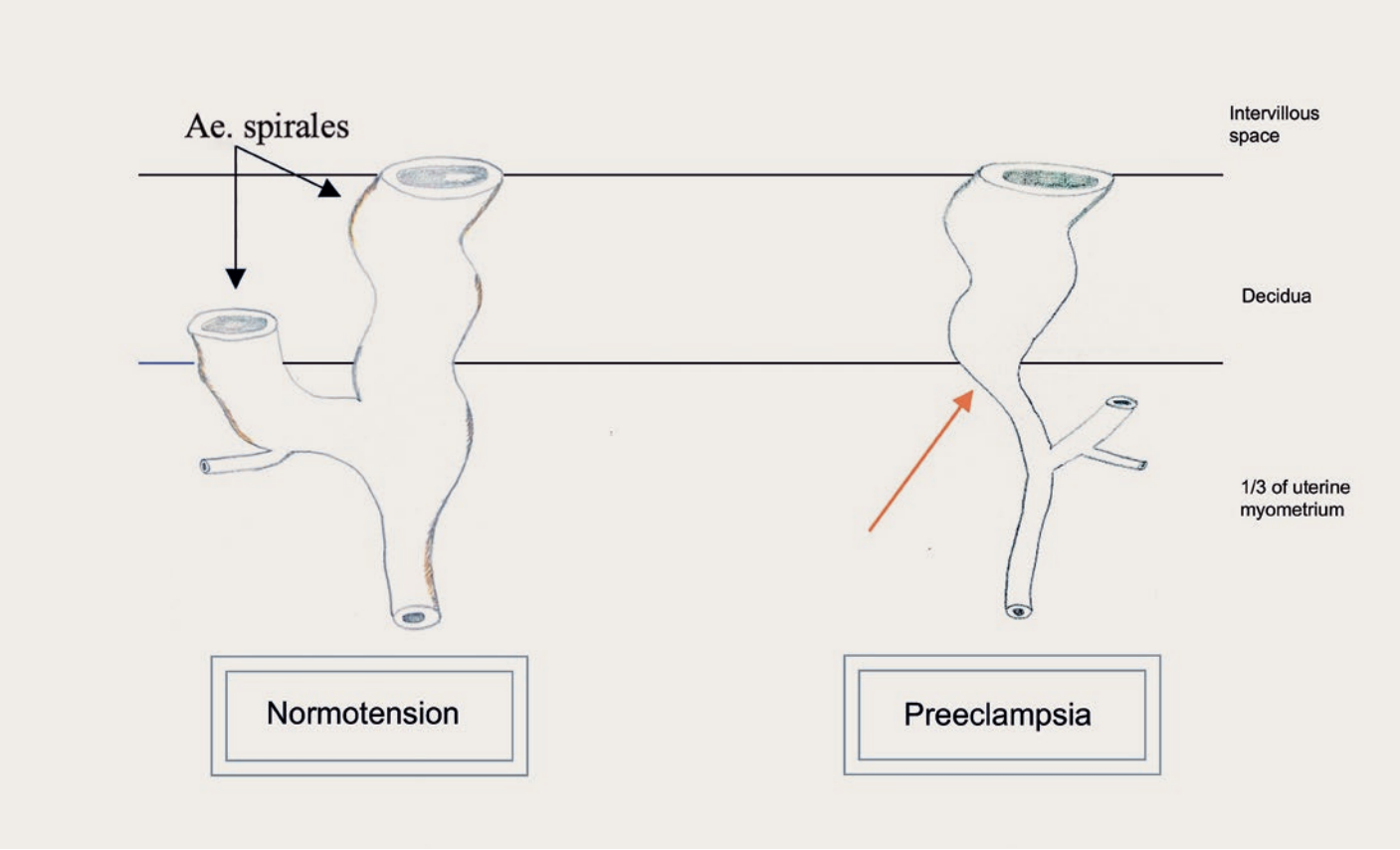

Figure 1: Illustration of persistent narrowing of spiral arteries in insufficient trophoblast invasion compared with normal trophoblast invasion. The orange arrow marks the persistent constriction of the spiral arteries and a reduced placental blood supply in preeclampsia.

persistent constriction of the spiral arteries and a reduced placental blood supply (fig. 1). The exact reason for the insufficient placentation is still unknown. Maternal hypertension is the compensatory mechanism to ensure sufficient placental blood flow in the presence of persistently increased uteroplacental resistance. The reduced placental perfusion results in oxidative stress and an increased release of anti-angiogenic factors such as soluble fms-like tyrosine kinase 1 (sFlt-1) that counteracts pregnancy induced proangiogenic factors such as placental-like growth factor (PlGF) (fig. 2). sFlt-1 itself causes generalised endothelial dysfunction in the maternal circulation [34], resulting in various organ manifestations including the heart and thereby increases the risk for peripartum cardiomyopathy [35].

Adverse pregnancy outcomes of hypertensive disorder (APO)

APOs (table 2) include eclampsia, cerebral vascular accidents, disseminated intravascular coagulopathy (DIC), peripartum cardiomyopathy (PPCM) and HELLP syndrome. Other complications may include pulmonary and cerebral oedema, renal and hepatic failure. Overall, the complications are associated with a high mortality [36].

Preeclampsia (PE)

PE is one of the main causes of maternal and perinatal morbidity and mortality [37]. In preeclampsia, there is a simultaneous occurrence of maternal hypertension with a further organ manifestation. Renal, hepatic and central nervous disorders as well as respiratory, haematological or placental dysfunction may occur. In Europe, about 2% of all pregnancies are affected, but the incidence worldwide is up to 8%, especially in low-income countries [38, 39].

Eclampsia

The vasoconstriction and endothelial cell damage leads to cerebral microcirculatory disturbance, which may result in a generalised seizure (eclampsia) due to ischaemia. Eclampsia is a generalised tonic-clonic seizure that is not associated with other causes of seizure and usually occurs in the context of hypertensive derailment in pregnancy. 0.5-3% of pregnancies affected by preeclampsia develop eclampsia, this affects 0.1% of all pregnancies [17].

HELLP syndrome

HELLP syndrome is characterised by the typical laboratory constellation of haemolysis (lowered haptoglobin), elevated liver enzymes and low platelet count <100 G/l. It can be accompanied by right-sided upper abdominal pain due to tension of the hepatic capsule. Typically, it progresses in episodes and can exacerbate within a very short time leading to severe maternal bleeding, disseminated intravascular coagulation (DIC), organ failure and foetal impairment and death. It occurs in 0,1-0,2% of all pregnancies and increases up to 10-20% of all women with preeclampsia [17].

Thrombotic microangiopathies (TMA)

Differential diagnosis should include diseases from the group of TMAs. Differentiation is sometimes difficult because the individual clinical pictures overlap and cannot always be distinguished from each other [17]. TMAs are characterised by the formation of microthrombosis in arterial and venous vessels due to endothelial damage. This leads to ischaemia and results in organ dysfunction up to fulminant organ failure. TMAs relevant in pregnancy include thrombotic thrombocytopenic purpura (TTP) and atypical haemolytic uremic syndrome (aHUS) and represents a heterogeneous spectrum that can be triggered by various causes [40]. TMA outside of the HELLP syndrome are extremely rare conditions. The incidence is estimated to be 1-3/10,000 pregnancies [41].

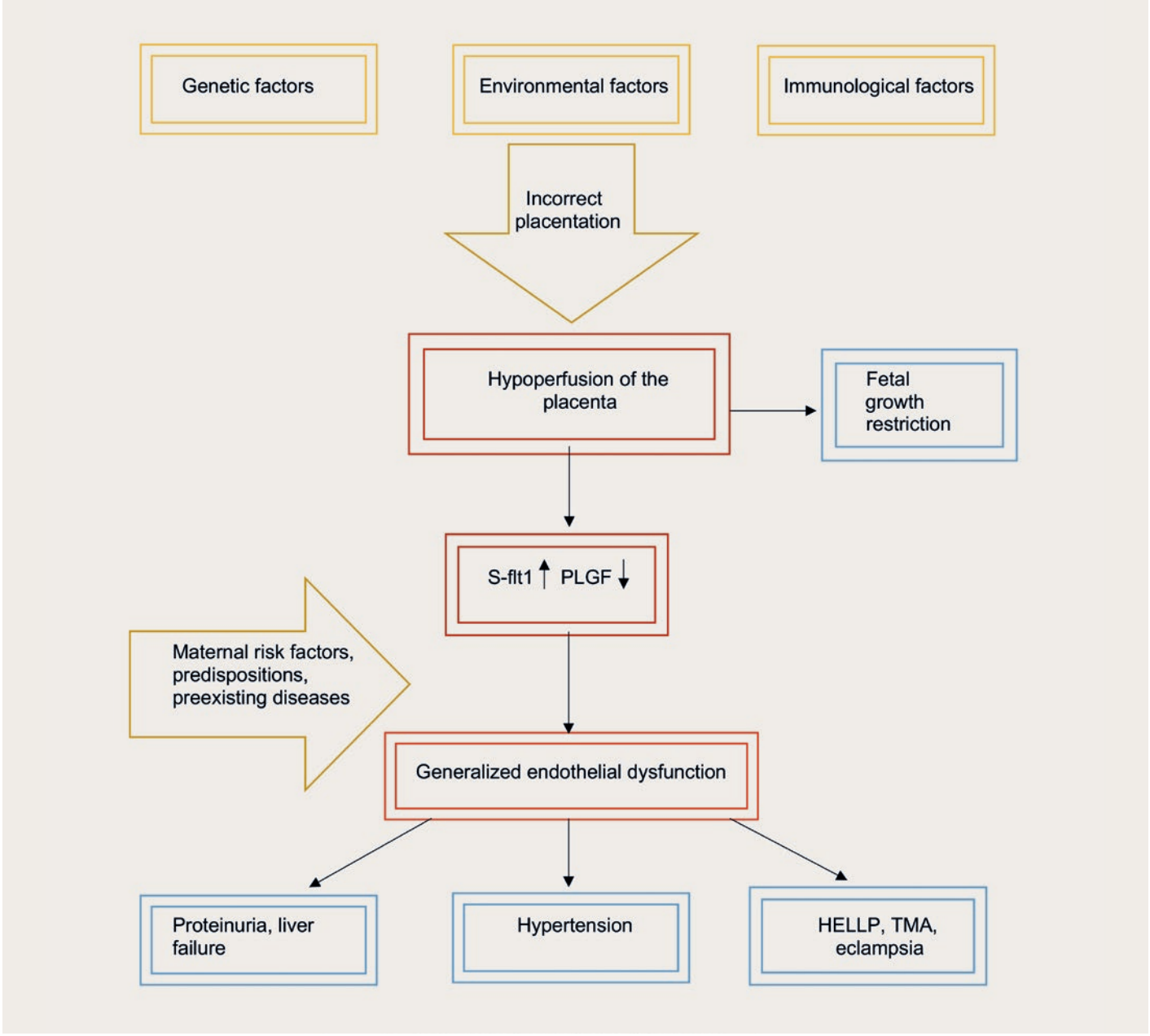

Figure 2: Overview of influencing factors and pathomechanisms for the development of preeclampsia.

Table 2: Clinical differential diagnosis of TMA modified from [42]

	HELLP	aHUS	TTP
Haemolysis	++	+++	+++
Elevation of liver enzymes	++	-/+	-/+
Thrombocytopenia	++	+++	+++
Hypertension	++	+++ (secondary)	-/+
Proteinuria	+++	++	+
Renal insufficiency	+++	+++	+
Neurological symptoms	+++	+	+++
Activity of ADAMTS-13	normal	normal	degraded

aHUS, atypical haemolytic uremic syndrome; TTP, thrombotic thrombocytopenic purpura.

Thrombotic thrombocytopenic purpura (TTP)

TTP is caused by a deficiency of the metalloprotease ADAMTS-13, which cleaves the large von Willebrand (VWF) multimers. The increased concentration of VWF multimers leads to an aggregation of platelets with microthrombus formation, because of a genetic defect or acquired auto-antibody formation. In principle, the acquired form often manifests itself in the last trimester, while the genetic form can occur throughout the whole pregnancy. Clinically, thrombocytopenia, coagulopathy up to DIC, neurological and gastrointestinal symptoms, as well as acute renal failure occur [42].

Atypical haemolytic uremic syndrome (aHUS).

Adult-onset aHUS is associated with pregnancy in 20%. In approximately 40-60% of cases, there is a genetic defect in one of the complement-regulating genes. Currently, eleven different genetic defects are known. It is suspected that pregnancy leads to an activation of the complement system. Due to the sudden cessation of the production of complement-regulating factors by the placenta, an imbalance with excessive complement activation occurs after birth. Gene carriers are unable to regulate this, which is why aHUS typically manifests postpartum. Clinically, haemolysis and acute renal failure is seen [42].

Peripartum cardiomyopathy (PPCM)

PPCM is a potentially life-threatening condition typically presenting as heart failure with reduced ejection fraction (HFrEF) in the last month of pregnancy or the postpartum period [43, 44]. Clinically, a reduced left ventricular ejection fraction (LVEF) <45 % is seen. Symptoms may include fatigue, oedema, heart failure and cardiogenic shock [28]. Hypertensive pregnancy disorder is a significant risk factors for the development of PPCM [45-48]. The aetiology is still unclear but it is suspected that multiple factors induce the disease. It is hypothesised that these factors merge in a common pathway involving increased oxidative stress and increased cleavage of prolactin into an anti-angiogenic 16 kDa PRL fragment. Consequently, impairment of endothelial function, frequently enhanced by additional anti-angiogenic factors (s-flt-1) and impaired neuregulin (NRG) signalling results in heart failure [44, 49, 50].

Foetal complications

Placental dysfunction leads to impaired foetal growth [51], which increases the risk of intrauterine foetal death (IUFD). In a considerable proportion of women, premature delivery is necessary for maternal and/or foetal reasons and newborns are exposed to the risks of premature birth. The prognosis is determined by the gestational age and the neonatal weight [52]. Children with intrauterine exposure to preeclampsia and premature delivery have twice the risk of cerebral palsy due to prematurity and growth restriction. In childhood and adolescence, they show higher blood pressure values, a higher body mass index and have an increased risk of CVD and diabetes mellitus [53, 54].

Therapy

Conservative options

Non-drug therapies play a minor role in the treatment of hypertensive pregnancy disorders. Limited data is available and only show minimal effects through dietary and life-style interventions. Moderate exercise of 150 min/week and the avoidance of excessive weight gain is recommended, especially in the presence of obesity [55, 56]. However, these recommendations are based on heterogeneous study data resulting from differences in duration, type and intensity of interventions [57].

Drug therapy

Drug therapy for hypertensive pregnancy disorders is not subject to evidence-based data, since the only study that also mapped the foetal outcome after 7.5 years was conducted more than 40 years ago [58, 59]. Pharmacotherapy should achieve optimal maternal blood pressure control while ensuring the safety of the foetus. However, due to ethical reasons and high risks, randomised prospective studies are rarely performed and most data derive from retrospective analyses and observational studies. Table 3 summarises the current recommendations for the treatment of hypertension in pregnancy and gives an overview of the medications.

In mild and moderate hypertension (systolic 140-159 mmHg, diastolic 90-109 mmHg) drug therapy includes the use of a-methyldopa, beta blockers (best tested: labetalol) or calcium channel blockers [60, 61]. Since labetalol is not licensed in all countries, alternatively a-methyldopa or slow-release nifedipine can be used [2, 17].

In severe hypertension (systolic >160 mmHg, diastolic >110 mmHg) the current guidelines recommend inpatient treatment of the pregnant woman. Drug therapy can be intravenous (IV) or intramuscular (IM) and close monitoring of the foetal condition must be ensured. The IV administration of labetalol, alternatively urapidil or nifedipine can be used [17, 62]. Dihydralazine is approved in pregnancy but has a significantly higher side effect profile (reflex tachycardia, headache) compared to other drugs, so that differentiation from preeclampsia can be difficult. Nevertheless it is often used in routine clinical practice when other therapeutic regimes fail [63, 64].

Delivery as the last option for treatment

The only option to avoid associated APO of hypertensive pregnancy disorders is delivery, especially when the limit of preterm birth is exceeded from 37+0 weeks of GA. The German guidelines recommend induction of labour from 37+0 weeks of GA for women with gestational hypertension and 38+0 weeks of GA for women with chronic hypertension [65]. Compared to waiting, this can improve the maternal outcome without increasing the rate of caesarean sections [66].

If severe hypertension, preeclampsia or other associated complications develop during pregnancy, delivery is the only causal therapy. Depending on the GA there is often a difficult balancing act between maternal outcome and foetal prognosis [67-69]. If possible antenatal corticosteroids should be given up to 34+0 weeks of gestation to induce lung maturity of the child. In case of neurological symptoms, magnesium should be administered IV to prevent eclampsia [70] and up to 32+0 weeks of gestation for neuroprotection for the child when delivery is expected within in the next twelve hours. In case of mild symptoms, symptomatic therapy by means of blood pressure control under close clinical monitoring should be carried out. In general, regular checking of the reflex status should be part of the daily rounds and assessment of foetal status using cardiotocography and ultrasound should be carried out regularly. In case of APO, delivery is indicated at any time in pregnancy [17, 71].

TMA

When TTP is diagnosed, the main treatment is therapeutic plasma exchange, which delivers the autoantibody to ADAMTS13 and supplies functional ADAMTS13 in the replacement plasma via the infusion [42].

In addition to plasmapheresis, in case of aHUS, antibodies (eculizumab) should be administered. Eculizumab inhibits the complement system by binding to complement factor C5 and preventing its cleavage into C5a and C5b to prevent permanent kidney damage [41, 72].

Postpartum care

Postnatal care needs close monitoring, as hypertension and other CVD can persist [73]. In

Table 3: Overview of antihypertensive therapy, therapy options and contraindicated agents

Classification	Drug	Placenta permeable category	Transfer to breast milk (foetal dose)	Preclinical/clinical safety data
Beta blocker				No foetal malformations, foetal hypoglycaemia and bradycardia
	Metoprolol	Yes	Yes	
	Bisoprolol	Yes	Yes	
Calcium channel blocker				Well tolerated
	Verapamil (oral)			
	Verapamil (intravenous)	Yes	Yes	IV use is associated with a greater risk of hypotension and subsequent foetal hypoperfusion
	Nifedipine	Yes	Yes	Tocolytic, sublingual application and potential synergism with magnesium sulphate may induce hypotension (mother) and foetal hypoxia, no teratogenic effects, however, increased perinatal asphyxia, caesarean delivery, prematurity and intrauterine growth retardation
Central a-agonist				No teratogenic effects
	α-Methyldopa	Yes	Yes	First line if labetalol is not available
α- & β-blocker				
	Labetalol	Yes (animal model)	Yes (animal model)	No foetal malformations
	Urapidil (intravenous)	Unknown	Unknown	Inadequate human data, use in severe hypertension
	Carvedilol	Yes (animal model)	Yes (animal model)	No adequate human data, bradycardia and hypoglycaemia in foetus, use only if potential benefit outweigh potential risks
Vasodilator				Use in postpartum management of hypertension
	Hydralazine	Yes	Yes (1%)	Maternal side effects: lupus-like symptoms. Foetal tachyarrhythmia
ACE-inhibitor	Because ACE inhibitors should be avoided during pregnancy and breastfeeding	Medication switch at least one month before conception [11]	During pregnancy medication change within two days [11]	Contraindicated renal or tubular dysplasia, oligohydramnios, growth retardation, ossification disorders of skull, lung hypoplasia, contractures, large joints, anaemia, intrauterine foetal death
	Captopril	Yes	Yes (1.6%)	Use in postpartum management of hypertension
	Enalapril			Use in postpartum management of hypertension
Angiotensin II receptor blocker	ARB should be avoided during pregnancy and breastfeeding			Contraindicated, can cause foetal harm
	Valsartan, Candesartan	Unknown	Yes	

Overview about placental transfer and concentration in breast milk. Modified from [36] with additional information from [2].

1. First line
2. Second line
3. Contraindicated

cases of mild to moderate hypertension without APO, monitoring blood pressure for 1-2 weeks is recommended. The target range is <150/100 mm Hg and if necessary, oral medication must be adjusted [74]. Women with severe hypertension or hypertensive derailment often require intensive medical care immediately postpartum. Intravenous therapy options include labetalol, urapidil or hydralazine. Oral maintenance therapy can be given with either substance. Alternatively, ACE inhibitors, AT1-blockers, beta blockers or calcium channel blockers can be used (table 3). Dose reduction is indicated for continuous values below 140/90 mm Hg [17, 75].

Postpartum medication must be seen in the context of breastfeeding tolerance (table 3). The majority of antihypertensives pass only in small proportions into breast milk and only nifedipine and propranolol have almost plasma-identical values in breast milk [2]. Recommendations by designated embryonal toxicity institutes regarding the use of medication during pregnancy and breastfeeding can be obtained via web-based services [76]. Taking any medication while breastfeeding confuses many women, which can lead to discontinuation of therapy. In these cases, outpatient interdisciplinary care can improve treatment adherence [77]. A check-up by the attending specialist after the first three postpartum weeks is recommended, but about 40% of women do not take advantage of this [75]. Moreover, it should be noted that hypertensive disorders in pregnancy increase the risk for heart failure and PPCM. Therefore, measuring NT-proBNP as a marker for cardiac dysfunction and an echocardiogram is recommended within the first postpartum weeks, especially in severely affected patients.

Low socioeconomic status, lack of health insurance coverage as well as belonging to certain ethnic groups or living in rural areas leads to a lower utilisation of medical care [78, 79]. In this context, the use of telemedicine can improve communication and the care of women by medical providers [80].

Outpatient care by a midwife is important in achieving comprehensive care for mother and child and identifying complications at an early stage [81, 82].

Long-term outcome and subsequent pregnancies

In the postpartum check-up, the risk of recurrence in subsequent pregnancies should be pointed out and, if necessary, treatment by other disciplines should be initiated [77]. In general, annual monitoring of blood pressure and metabolic factors (lipid balance, fasting glucose, HbA1c) is recommended [2, 83].

Jowell et al. showed in a review that linkage to specialised centres improved patient education, lifestyle changes and referral to appropriate specialists over a period of 0-6 months [84].

Overall, women with gestational hypertension have an increased risk of developing CVD later in life, this risk being further increased by preeclampsia. They are significantly more likely to develop chronic hypertension, stroke, venous thrombosis, coronary artery disease (CAD), both in the short-term and long-term interval. Vascular central nervous disorders also occur more frequently in this context [85-88]. Furthermore, they are more often affected by postpartum depression [89].

Interdisciplinary care must play an important role in pointing out long-term consequences for women, especially in further pregnancies and to inform the patient about preventive strategies and treatments. This can initiate and support the patient's own initiative and self-care. In subsequent pregnancies, screening examinations in the first trimester are an option for early intervention. In the first trimester screening (11+6 - 14+1 weeks), a risk calculation can be made by collecting anamnestic factors, determining the mean arterial pressure and sonographic measurements of foetal and maternal factors [90]. In the case of increased risk, taking aspirin 150 mg/d starting before 16 weeks GA is recommended, which can result in a significant risk reduction for preeclampsia before 37 weeks of gestation (63%) and especially for severe preeclampsia before 32 weeks GA [91]. Since it is a screening method, this examination should be offered to all pregnant women in the first trimester.

Summary and conclusion

Hypertension in pregnancy bears a high risk for mother and baby. Preexisting hypertension requires counselling prior to pregnancy regarding changes in the medication as blood pressure lowering drugs could be fetotoxic. Furthermore, close monitoring is recommended during pregnancy in these patients. Pregnancy induced hypertensive disorders including preeclampsia, eclampsia and HELLP syndrome, are frequent and involve a complex pathophysiology. Screening examinations in the first trimester can help to identify women at increased risk and provide them with adequate care [91], especially if any of the risk factors (increased age of the mother, in vitro fertilisation, twin pregnancy, smoking, obesity) are present. Comprehensive interdisciplinary care and treatment is needed to identify and reduce maternal and child complication rates. Among suspected causes are a dysfunctional placenta resulting from insufficient trophoblastic invasion in early pregnancy [31]. Subsequently, maternal hypertension is a compensatory mechanism to ensure sufficient blood supply to the placenta for the growing embryo/foetus and is an expression of generalised endothelial dysfunction, which can lead to further complications [10, 34]. Pregnant women with known risk factors need to be identified and monitored more intensively [19]. Preventive measurement should be started as well as close monitoring and, if needed, pharmacological treatment during pregnancy. Delivery is the ultimate therapy, especially when the limit of preterm birth is exceeded, but needs to be carefully evaluated prior to this time point. Postpartum surveillance is equally essential, as hypertensive disorders can persist beyond pregnancy and have far-reaching consequences for maternal health in subsequent life and pregnancies.

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