Optimizing the outcome of pregnancy in obese women: From pregestational to long-term management

F. Galtier, I. Raingeard, E. Renard, P. Boulot, J. Bringer

Abstract

The obesity epidemic is of some concern in women of reproductive age. Maternal obesity is associated with many pregnancy complications, especially gestational diabetes and hypertensive disorders of pregnancy. Delivery in obese women is characterized by a high caesarean-section rate and an increased risk of anaesthetic and postoperative complications. Weight retention after birth may increase the risk of type 2 diabetes in the long term. Foetal risks include macrosomia, malformations and increased perinatal mortality, with the long-term infant health marked by a higher risk of obesity and metabolic disorders. Optimal management includes preconception counselling, pregravid weight-loss programmes, monitoring of gestational weight gain, repeated screening for pregnancy complications and long-term follow-up to minimize the social and economic consequences of pregnancy in overweight women.

Keywords: Obesity; Pregnancy; Management; Gestational diabetes; Macrosomia; Malformations

In France, the number of obese individuals increases by more than 15% every three years, and the prevalence of childhood obesity has tripled over the past 15 years, now reaching 5%, with a further 11% of children being overweight. Maternal obesity before and during pregnancy has a considerable impact on the course of pregnancy as well as on the development of the foetus, the condition of the infant and its future health (Table 1). This evidence highlights the importance of appropriate management of high-risk pregnancies due to excess weight in the mother.

1. Maternal complications

1.1. Impaired glucose tolerance

Excess weight increases the risk of glucose intolerance. Even in moderately overweight subjects [body mass index (BMI)
Women with higher baseline BMIs and greater increases were demonstrated during the third trimester of pregnancy and snoring are common in pregnancy and narrower upper airways resistance. Sleep-disordered breathing documented. Excess weight reduces thoracic-wall compliance and increases airways resistance. Sleep-disordered breathing and snoring are common in pregnancy and narrower upper airways were demonstrated during the third trimester of pregnancy [9]. Women with higher baseline BMIs and greater increases in neck circumference during pregnancy report higher apnoea symptom scores [10], and preeclamptic toxaemia is associated with a high-respiratory disturbance index [11]. Sleep apnoea should be looked for in obese women if either foetal growth retardation or hypertension is detected [7,12].

1.4. Complications and morbidity during delivery

Obese women have a higher frequency of induced labour and more caesarean sections than normal-weight women. Commonly reported indications for caesarean delivery include cephalopelvic disproportion, failed cervical dilatation, foetal distress and risk of shoulder dystocia [7,13]. Caesarean-section practices vary widely, but around one in two severely obese women (BMI > 40 kg/m²) is delivered by C-section and each one-unit increase in pregravid BMI increases the risk of a caesarean delivery by 7% [14]. This higher caesarean delivery rate is driven by prepartum complications of obesity: in the severely obese, caesarean sections represent around 40% of deliveries in the presence of no other risk factors, 44% in case of concomitant hypertension, 50% in case of preexisting or gestational diabetes, and 59% in class 3 obesity (BMI ≥ 40 kg/m²) with concomitant hypertension and diabetes [15].

Anaesthesia risks are high in obese patients who pose technical challenges. Difficulties in inserting epidural catheters include positioning the patient correctly, identifying the midline, identifying the epidural space and dislodging the catheter. Regional anaesthesia is preferred for caesarean sections, as obesity is characterized by an increased incidence of difficult intubation, rapid desaturation and an increased risk of aspiration during anaesthesia [16].

Although most obese patients receive Pfannenstiel’s incision, whether the incision is transverse or vertical remains a matter of controversy, particularly in the severely obese. The advantages of a transverse incision in terms of postoperative pain, and a lower risk of hypoxia and atelectasis, are offset, in some studies, by a higher postoperative infection rate. Incision-site infections are two to three times more frequent in overweight women. Postoperative morbidity is further increased by a higher thromboembolic risk and longer operating time, with more blood loss, which prolongs hospitalization [13].

2. Infant complications

2.1. Macrosomia and neonatal morbidity

Prepregnancy obesity considerably increases the risks of macrosomia and associated shoulder dystocia, observed in 33% of infants weighing more than 4.5 kg compared with only 2% among those under this weight at birth [13,17,18]. Even being moderately overweight (BMI 25–30 kg/m²) increases the risk of macrosomia [1] and the incidence is multiplied by 1.4–18 in obese women, depending on the degree and type of obesity. A prospective study showed that a 0.1 increase in prepregnancy waist-to-hip ratio predicted an increase of 120 g in body weight, 0.51 cm in height and 0.31 cm in head circumference in the newborn [3]. The risk of macrosomia persists even in the absence
of GDM, although the latter is a predisposing factor if blood glucose is not satisfactorily controlled. In a multivariate analysis of 1000 neonates, including 209 with macrosomia, maternal excess weight was a stronger predictor of birth weight than the oral glucose-tolerance test \[19\]. GDM does not increase the risk of macrosomia if blood glucose is well controlled, particularly with insulin treatment \[18\]. The risk of late foetal death is three times higher in morbidly obese women, whereas neonatal mortality is similar to that of control infants \[20,21\]. However, infants of obese mothers more often require neonatal intensive care due to obstetrical trauma and low Apgar scores \[1\].

### 2.2. Malformations

Even in the absence of GDM, most studies report an increased frequency of neural-tube defects (Table 2; Fig. 1), cardiac malformations (\(OR\) for atrial or ventricular septal defect = 2.2, 95% CI = 1.0–4.9 or multiple abnormalities (\(OR\) = 2.0, 95% CI = 1.0–3.8) in the offspring of obese women \[22–26\]. Inadequate gestational weight gain \[27,28\] and GDM \[29\] further increase these risks while, conversely, pregravid BMI predicts the risk of malformation in the infants of mothers with GDM (Fig. 2 \[24\]). This increased risk of malformation probably arises from metabolic disturbances present before pregnancy or manifesting in early pregnancy, such as unrecognized diabetes and insulin resistance. Insulinaemia and hyperglycaemia may play a role, as suggested by the correlation between postpartum insulinaemia and neural-tube defects. Furthermore, the benefit of folic-acid supplementation is reduced in obese women compared with the nonobese \[30\]. In addition, maternal obesity increases the rate of suboptimal ultrasound visualization of foetal cardiac structures by 49.8% and of craniospinal structures by 31% \[31\].

<table>
<thead>
<tr>
<th>Abnormality</th>
<th>N</th>
<th>Adjusted (OR^a)</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>All neural-tube defects</td>
<td>254</td>
<td>2.6</td>
<td>1.7–4.0</td>
</tr>
<tr>
<td>Anencephalus</td>
<td>97</td>
<td>2.3</td>
<td>1.2–4.3</td>
</tr>
<tr>
<td>Spina bifida</td>
<td>157</td>
<td>2.8</td>
<td>1.7–4.5</td>
</tr>
<tr>
<td>Hydrocephaly</td>
<td>103</td>
<td>2.7</td>
<td>1.5–5.0</td>
</tr>
</tbody>
</table>

\(a\) Odd ratios adjusted for ethnicity (white, Hispanic, other); age (18, 18–24, 25–29, 30 years); maternal education (high-school graduate, some higher education); smoking (three months before pregnancy); alcohol use (three months before pregnancy) and periconceptional folic acid use. Excluded are mothers with pregestational (type 1 or 2) diabetes and those lacking the variables used for adjustment.

Fig. 1. Maternal obesity, gestational diabetes and central nervous system malformations (from Anderson et al.) \[29\].

Fig. 2. Overall odd ratios for congenital defects (cardiovascular defects, renal/urinary anomalies, spine/rib alterations, holoprosencephaly) in infants of mothers with GDM stratified by pregestational BMI (from Martinez-Frias et al.) \[24\].
obesity in young adulthood is predicted independently by maternal expression (epigenetic factors) [35,38,39]. 

Exposure to a diabetic environment in utero is associated with an increased risk of impaired glucose tolerance and defective insulin secretion in adulthood. These prediabetic abnormalities are independent of genetic predisposition [35]. Obesity in young adulthood is predicted independently by maternal obesity (OR = 3.6, 95% CI = 2.1–5.9), but also by paternal obesity (OR = 2.9, 95% CI = 1.7–4.9) [36]. Rapid postnatal growth (before two years of age) also partially determines future weight and fat mass [37]. Thus, both the nutritional environment in utero and dietary conditions during the first few years of life contribute to influence future weight, possibly via modulation of gene expression (epigenetic factors) [35,38,39].

<table>
<thead>
<tr>
<th>Management of pregnancy in obese women</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pregestational management</strong></td>
</tr>
<tr>
<td>Information</td>
</tr>
<tr>
<td>Screening for complications of obesity</td>
</tr>
<tr>
<td>Diet and physical activity</td>
</tr>
<tr>
<td>Treatment of nutritional deficiencies</td>
</tr>
<tr>
<td><strong>Management during pregnancy</strong></td>
</tr>
<tr>
<td>Diet (folic acid supplementation)</td>
</tr>
<tr>
<td>Support for physical activity</td>
</tr>
<tr>
<td>Weight and blood pressure monitoring</td>
</tr>
<tr>
<td>Screening for GDM</td>
</tr>
<tr>
<td>Screening for foetal malformations</td>
</tr>
<tr>
<td>Monitoring of foetal biometry</td>
</tr>
<tr>
<td>Anaesthetic assessment</td>
</tr>
<tr>
<td><strong>Postpartum follow-up</strong></td>
</tr>
<tr>
<td>Screening for type 2 diabetes</td>
</tr>
<tr>
<td>Prevention of weight retention</td>
</tr>
</tbody>
</table>

2.3. Long-term postnatal outcomes

Childhood obesity is frequent in the offspring of obese mothers, particularly in cases of gestational diabetes or macrosomia. Follow-up of children of diabetic mothers, particularly infants with macrosomia, suggests an impact of nutrition in utero on later growth [32–34]. The symmetry index (ratio of relative weight to relative height) is increased starting at age of three years in the children of diabetic mothers. This excess weight is correlated with amniotic fluid insulin concentration and with the C-peptide-to-glucose ratio in umbilical cord blood [34]. Exposure to a diabetic environment in utero is associated with an increased risk of impaired glucose tolerance and defective insulin secretion in adulthood. These prediabetic abnormalities are independent of genetic predisposition [35]. Obesity in young adulthood is predicted independently by maternal obesity (OR = 3.6, 95% CI = 2.1–5.9), but also by paternal obesity (OR = 2.9, 95% CI = 1.7–4.9) [36]. Rapid postnatal growth (before two years of age) also partially determines future weight and fat mass [37]. Thus, both the nutritional environment in utero and dietary conditions during the first few years of life contribute to influence future weight, possibly via modulation of gene expression (epigenetic factors) [35,38,39].

3. Therapeutic recommendations (Table 3)

3.1. Pregestational management of obese women

Even today, overweight women of reproductive age are rarely informed of the risks of pregnancy or the need for preconceptional care and optimal obstetrical management. However, the substantial morbidity incurred by both mother and child makes it necessary for healthcare professionals to implement a strict programme of preconceptional information and care. (Table 3).

Such a programme should comprise an evaluation of the determinants and complications of obesity, screening for impaired glucose tolerance and monitoring blood pressure. Lifestyle recommendations should include the prescribing of an adequate diet, folic-acid supplementation, correction of nutritional deficiencies and a physical-activity programme with realistic targets. Women with anovulation-related infertility should be prescribed a weight-loss programme before any attempt to stimulate ovulation, as weight loss improves the quality of both spontaneous and induced ovulation [40]. Eating disorders should be identified and treated, with psychological counselling provided if necessary.

3.2. Weight gain during pregnancy

Gestational weight gain is an independent risk factor for the outcomes of macrosomia, hypertensive complications, induction of labour and caesarean section [41]. Losing weight during pregnancy is always inadvisable, regardless of the degree of obesity. The American Institute of Medicine (IOM) established a set of guidelines in 1990 based on analysis of foetal mortality, premature birth and birth weight [42]. The target weight gain during pregnancy is 7 kg in obesity, 7–11 kg in overweight women, 11–16 kg in normal-weight women and 12–18 kg for thin women. During the second and third trimesters of pregnancy, obese women should gain about 1.3 kg/month.

Considering the combined weight of the foetoplacental unit and the mammary hypertrophy of late pregnancy, a weight gain of less than 7 kg would reflect weight loss in the mother, which may be detrimental to foetal development. Although the risk of growth retardation in utero is low in the infants of obese mothers, it remains high with weight gains below the recommended limits [43]. Also, the risk of neural-tube defects increases for weight gains of less than 5 kg [27]. On the other hand, the risk of macrosomia in obese women increases for weight gains of 13 kg or more whereas, in normal-weight women, the risk is only increased after a weight gain of more than 17 kg [43]. A recent study suggested that a more limited weight gain would be beneficial in obese glucose-tolerant women in terms of reducing macrosomia, hypertension, induction of labour and caesarean section. In this study, the rate of small-for-gestational-age infants was not significantly related to low-weight increases. However, the sample size was too small to allow firm conclusions to be drawn [41]. In practice, such guidelines remain difficult to implement, particularly when the woman’s own target differs from the recommended weight gain [44].

3.3. Dietary recommendations

Overweight or obese women should keep to a minimum daily caloric intake of 1500 kcal. An additional 100 kcal/day over the dietary intake that allowed a stable prepregnancy weight is generally sufficient although, in late pregnancy, an additional 200 kcal/day is sometimes recommended. It is preferable to restrict simple carbohydrates but to maintain fruit, with three main meals and one or two snacks. Complex carbohydrates in the form of starches, legumes, seeds and bread should be limited to reasonable quantities. Protein can be provided by meat, fish, cheese and dairy products (source of calcium), supplemented with small amounts of butter and vegetable fats.

Vitamin supplements, including folic acid, can be added to these dietary measures, particularly for patients with eating disorders, cognitive restraint or selective diets. Iron supplements are generally not necessary as the incidence of
iron-deficiency anaemia is lower during pregnancy in obese women.

The impact of bariatric surgery on pregnancy outcome has not been clearly established. However, the increasing frequency of adjustable gastric banding and vertical gastroplasty with or without bypass, associated with improved fertility and sexual activity after weight loss, raises concerns over the management of the specific problems associated with such situations during pregnancy. To date, series reported in the literature are relatively small and indicate reduced risks of GDM, hypertension, macrosomia and caesarean delivery in comparison with obese controls [45–48]. However, gastric bypass can lead to several metabolic complications, including vitamin B12 deficiency, iron-deficiency anaemia, hypokalaemia, hypoglycaemia, decreased serum albumin and severe vitamin-deficiency states, such as Wernicke’s encephalopathy. Identifying and correcting these deficiencies before pregnancy are necessary to ensure adequate nutritional supplies for the foetus. Clinical, biological and ultrasound monitoring – and, if necessary, band adjustments during pregnancy – provide more favourable maternal outcomes [45].

3.4. Physical activity

Physical activity can be promoted provided that it is reasonable and compatible with pregnancy. The patient’s motivation is necessary, but reeducation and support are often also required in obese women, particularly during the later stages of pregnancy. Participation in physical activity during pregnancy can reduce the risk of GDM and preeclampsia and help to prevent excess maternal weight gain. The risk of uterine contractions may be increased with lower-extremity exercise, whereas upper-extremity exercise produces no uterine contractions [49]. For a comparable training intensity, biking or walking are four to five times more likely to cause contractions than rowing or arm exercises. Activities such as swimming or water aerobics can be recommended throughout pregnancy.

3.5. Management during pregnancy

Weight and blood pressure should be measured, and symptoms of leg venous stasis looked for at least monthly. Women with diagnosed gestational hypertension–preeclampsia should be repeatedly evaluated for symptoms of organ dysfunction (severe headaches, visual changes, epigastric pain, decreased urine output) and abnormal laboratory tests (24-h urine protein, serum creatinine, platelet count, liver enzymes). Foetal monitoring should include ultrasound estimation of foetal weight and Doppler flow velocimetry. Severe disease should be managed while in hospital.

Screening for GDM should be performed early and repeated monthly throughout pregnancy. As oral glucose challenges cannot be reasonably carried out at this frequency, fasting and postprandial glycaemia should be monitored closely (at least once a month) and an oral glucose challenge performed at week 24 of pregnancy if necessary. Fasting glycaemia greater than 5.3 mmol/l (0.95 g/l) and/or postprandial glycaemia greater than 6.7 mmol/l (1.20 g/l) indicate the need to reinforce dietary measures and to institute insulin therapy if appropriate. Insulin therapy is required more often in obese women than in normal-weight women and leads to a significant reduction of maternal and foetal morbidity [17,18]. Indeed, it appears that obese women who meet blood-glucose targets by diet alone still have a two- to threefold higher risk of adverse maternal and foetal outcomes than normal-weight women with similarly controlled GDM (Figs. 3 and 4) [17]. When appropriately adjusted, insulin does not cause extra maternal weight gain.

Careful ultrasound assessment of foetal biometry is needed, especially as maternal obesity impairs the accuracy of ultrasound examination for detecting foetal abnormalities [31]. Maternal

![Fig. 3. Influence of maternal weight and treatment of gestational diabetes on the rate of adverse maternal outcomes (from Langer et al.) [17].](image-url)
obesity is also a risk factor for anaesthesia-related maternal morbidity and mortality. Antenatal anaesthetic assessment and the growing use of regional techniques have contributed to reducing anaesthesia-related maternal complications.

3.6. Postnatal and long-term follow-up

Prevention of infection and thrombosis should be emphasized in the immediate postpartum period. Particularly following caesarean section, early mobilization, aggressive chest physiotherapy and adequate pain control are essential components of effective care. In the long-term, preventing weight retention is a major issue. Weight retention varies widely among individuals and depends on prepregnancy weight, gestational weight gain and postpartum lifestyle factors, including smoking cessation, breastfeeding, dietary habits, eating disorders and the ability to maintain regular physical activity [50,51]. Weight retention in obese or even nonobese women leads to increased complications during a subsequent pregnancy [52] and a consequently higher risk of type 2 diabetes that is further increased in case of GDM. In such patients, an oral-glucose challenge should be performed two months after delivery or at the end of breastfeeding. Thereafter and depending on the results, annual or biannual glycaemia monitoring is recommended, as the yearly incidence of type 2 diabetes after gestational diabetes is 5%.

4. Conclusion

In the general population, both prepregnancy maternal BMI and gestational hyperglycaemia are independent risk factors for adverse pregnancy outcomes. However, due to its higher prevalence, maternal obesity has a much greater population impact than gestational abnormal glucose tolerance on pregnancy outcomes [53]. This is especially true for pregnancy-induced hypertension and large-for-gestational-age babies. Even today, far too little attention is focused on the appropriate care of obese women before and during pregnancy, despite precise data on the adverse consequences of obesity on maternal morbidity and infant development. However, the high motivation of women who wish to become pregnant offers a good opportunity to effectively implement a healthier lifestyle, provided that they are informed of the favourable impact of weight loss before pregnancy, and have access to suitable medical, dietary and psychological support systems.

References


