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Contemporary Topics in Gestational Diabetes Mellitus



Editor
Veeraswamy Seshiah

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Hormono-metabolic Adaptations in Pregnancy

2

Subhash K Wangnoo

INTRODUCTION

Maternal metabolism changes substantially during pregnancy. Amongst the functioning systems regulating the metabolism in pregnancy, endocrine system is the earliest to develop and start functioning. The endocrinology of pregnancy involves endocrine and metabolic changes as a consequence of physiological alterations at the fetoplacental boundary between mother and fetus. During the early pregnancy, most of the physiologic changes are hormone-mediated; while in the second and third trimesters, the uteroplacental vascular system and mechanical factors associated with the enlarging gravid uterus combine with the hormonal environment to have an effect on each and every system. The vast physiological variations in maternal hormones during the course of pregnancy complicate the assessment of the normal level of most hormones.

GENERAL ADAPTATIONS

The average weight gain during normal pregnancy is approximately 12.5 kg, with the fetus accounting for 3.4 kg, placenta 0.65 kg, amniotic fluid 0.8 kg, uterus 1 kg, breasts 0.4 kg, blood 1.5 kg, extravascular fluid 1.5 kg, and maternal fat stores approximately 3.3 kg. The volume of uterine cavity increases to about 5 L at term from about 10 mL and blood flow through the uteroplacental circulation reaches 450–650 mL/min.

The blood volume increases by 40–45% at term compared to nonpregnant state to maintain appropriate perfusion of maternal-fetal unit. The plasma volume also increases by 40–50% due to the action of aldosterone leading to sodium and water retention. The red cell mass increases approximately 20% as a response to enhanced erythropoietin secretion. As a result, hematocrit declines to about 15% at term.[1]

The renal blood flow and the glomerular filtration rate increase rapidly reaching a peak during second trimester. There is a 50% reduction in creatinine clearance resulting in reduction in serum creatinine.[2] Heart rate increases by 10–15 beats/minute, cardiac output by 30–50%, reduction in diastolic pressure with no effect on systolic pressure, and a reduction in peripheral vascular resistance by about 20%. The pulmonary vascular resistance is reduced by about one-third. There are no changes in respiratory rate, maximum breathing capacity, or forced or timed vital capacity. Gastric emptying time is reduced by 50% at term; tone of lower

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esophageal sphincter is reduced leading to a marked symptom of reflux. Intestinal motility is reduced leading to constipation and gall bladder emptying is delayed leading to a more lithogenic bile and increased incidence of cholelithiasis.[1]

THE ROLE OF PLACENTA IN DEFINING ENDOCRINE METABOLISM

Pregnancy-related proteins can be found in the maternal circulation shortly after conception, and human chorionic gonadotropin (hCG) is detectable in maternal serum after initiation of implantation, as early as from the 8th day after ovulation. hCG functions to prolong the hormonal activity of the corpus luteum to continue production of progesterone and maintain the early pregnancy endometrium. The decidua is the site of maternal steroid and protein biosynthesis in order to maintain and protect the pregnancy from immunologic rejection which acts in conjunction with hCG and progesterone secreted by the conceptus to suppress the maternal immune response conferring the immunologic privilege required by the implanting conceptus.[3] The decidua furthermore produces prolactin, insulin-like growth factor binding protein-1 and other protein hormones (Table 1).

The placenta plays an important role in balancing fetal growth and development with maternal homeostasis. It functions partly as a hypothalamic-pituitary-end organ-like entity with stimulatory and inhibitory feedback mechanisms, to

Table 1: Placental hormones and their functions

Placental hormones	*Functions*
Human chorionic gonadotropin	• Maintains corpus luteum of pregnancy • Stimulates the secretion of testosterone by the developing testes of XY embryo
Estrogen (also secreted by the corpus luteum of pregnancy)	• Stimulates growth of the myometrium, increasing uterine strength for parturition • Helps prepare mammary glands for lactation
Progesterone (also secreted by the corpus luteum of pregnancy)	• Suppresses uterine contractions to provide a quite environment for the fetus • Promotes formation of a cervical mucus plug to prevent uterine contamination • Helps prepare mammary glands for lactation
Human chorionic somatomammotropin (also called placental lactogen; has a structure similar to both prolactin and growth hormone)	• Helps prepare mammary glands for lactation • Believed to reduce maternal utilization of glucose and to promote the breakdown of stored fat so that greater quantities of glucose and free fatty acids may be shunted to the fetus
Relaxin (also secreted by the corpus luteum of pregnancy)	• Softens the cervix in preparation for cervical dilatation at parturition • Loosens the connective tissue between the pelvic bones in preparation for partuition
Placental parathyroid hormone-related protein	• Increases maternal plasma calcium levels for use in calcifying fetal bones; if necessary, promotes localized dissolution of maternal bone, mobilizing her calcium stores for use by the developing fetus

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regulate dynamic factors affecting fetal growth and development under a variety of conditions. However, in the placenta there are no direct neural inputs as in the fully developed hypothalamo-pituitary-end organ and the exact mechanism(s) responsible for regulation of the secretion of hypothalamic-like placental peptides is unknown.

The placenta as a site of active steroidogenesis depends on interactions with both mother and fetus, and is characterized by significant aromatase, sulfatase, and 11β-hydroxysteroid dehydrogenase type 2 activities in conjunction with lack of 17α-hydroxylase and 17/20 lyase activities. The principal precursor for decidual and placental steroid production is maternal low-density lipoproteins (LDL)-cholesterol.[4]

HYPOTHALAMUS AND PITUITARY

The maternal pituitary enlarges two- to threefolds during pregnancy and may become hyperintense on scan[5,6] primarily caused by estrogen-stimulated hypertrophy and hyperplasia of the lactotrophs.[7] Thus, maternal plasma prolactin levels parallel the increase in pituitary size throughout gestation. Gonadotrophs decline in number, and corticotrophs and thyrotrophs remain constant.[8] Somatotrophs are generally suppressed due to negative feedback suppression[9,10] by the placental production of growth hormone (GH), and may function as lactotrophs.[11]

The placental GH differs from pituitary GH by 13 amino acids and is synthesized by the syncytiotrophoblast. The regulation of placental GH secretion remains unknown, and it has similar carbohydrate, lipid, and somatogenic properties as pituitary GH, with less lactogenic activity.

Corticotropin-releasing hormone (CRH) levels rise several 100-folds by term.[12] It stimulates both syncytotrophoblastic placental and pituitary adrenocorticotropic hormone (ACTH) production.[13,14] ACTH levels consequently increase throughout gestation, with a further increase in labor mirroring an increase in corresponding cortisol levels. Cortisol binding globulin levels rise secondary to estrogen-stimulated production, leading to an increase in total cortisol of two- to threefolds by term.[15]

The posterior pituitary gland diminishes in size during pregnancy. The levels of maternal plasma arginine vasopressin levels are not believed to play a pivotal role in human pregnancy. Maternal oxytocin levels are also low throughout pregnancy, until they increase during labor.[16]

THYROID GLAND

Maintaining maternal euthyroidism during pregnancy is of absolute importance for growth and development, in particular neurodevelopment of the fetus. Maternal thyroid gland enlarges by approximately 18% during gestation. This enlargement is due to an increase in the size of follicles with increased colloid and enhanced blood volume.[17]

The maternal thyroid status is subject to substantial pregnancy-related physiological changes. hCG has a thyroid-stimulating hormone (TSH)-like activity and the steep increase in hCG levels during the first trimester may result

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in an increased production of thyroid hormones and thus decreased TSH levels. Pregnancy-related hyperestrogenism induces an increased production of thyroxine-binding globulin (TBG) and thus an approximately 50% physiological increase of total thyroxine (T_4). In the second and third trimesters, the hCG-induced stimulation of the thyroid gland decreases, while the maternal level of total T_4 continues to be in the high end or above the nonpregnant reference values, and levels of TSH in the low end of the range.[18,19] The renal iodine clearance and the maternal blood volume are increased, and in addition, maternal thyroid hormone is metabolized by or crosses the placenta to reach the fetus.[20,21] As a result of the increased need for thyroid hormone production, vascularity of the thyroid gland increase and glandular hyperplasia occurs, leading to slightly increased size of the thyroid gland during pregnancy.

During gestation, the mother remains in a euthyroid state, if sufficient iodine is available. Total triiodothyronine (T_3) and T_4 levels increase, but do not result in hyperthyroidism because of the parallel increase in TBG. Transthyretin transports thyroid hormones across the blood-placenta barriers. However, the placental exchange of maternal thyroid hormones is strictly regulated by placental deiodinases, securing a minimal, but highly significant supply of thyroid hormone to the fetus.[22] Since iodine is an essential component in the synthesis of thyroid hormones, maternal iodine status becomes vital to fetal development, also in the light of increased renal clearance of iodine.

In the first trimester, the fetus relies solely on thyroid hormones and iodine from the mother. The placenta is relatively impermeable to TSH and T_4, so the fetal hypothalamo-pituitary-thyroid axis develops and functions independently of that of the mother. The levels of TSH and T_4 are low in fetal blood until mid-gestation. At 24–28 weeks of gestation, serum T_4 as well as the biologically inactive reverse T_3 begins to rise progressively, while the TSH concentration peaks.

PARATHYROID GLANDS AND CALCIUM HOMEOSTASIS

The amount of calcium to the fetus is regulated by transfer of calcium across the placenta from the mother. Approximately 30 g of calcium is transferred from the maternal compartment to the fetal compartment, with the major chunk of transport occurring in the last trimester. Maternal compartment changes permit calcium accumulation by increase in maternal dietary intake and doubling of intestinal calcium absorption by end of first trimester and increases in maternal 1,25-dihydroxyvitamin D_3 levels due to increase in estrogen-induced rise in vitamin D binding globulin.[23] Parathyroid hormone (PTH) levels generally remain constant, but the circulating levels of PTH-related protein (PTHrp) keep on rising during pregnancy which is probably responsible for placental calcium transport. The higher calcitonin levels during pregnancy may be partly responsible for the elevated 24-hour calcium excretion, and an increased risk of kidney stones during pregnancy may be clinically relevant. By approximately 10–12 weeks' gestation, the fetal parathyroid glands secrete PTH. Maternal PTH is not transferred across the placenta, and therefore, the changes in fetal calcium levels are probably related to the fetal changes in PTH and calcitonin, and consistent with an adaptation to conserve and stimulate fetal bone growth.[24]

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ADRENAL GLANDS

The maternal adrenal gland does not change morphologically during pregnancy. Plasma adrenal steroid levels increase with advancing gestation. However, the increase in total plasma cortisol concentration is mostly due to a concomitant increase in cortisol-binding globulin due to hyperestrogenemia of pregnancy. Plasma and urinary free cortisol increase 2–3 times, but pregnant women normally do not exhibit any overt clinical features of hypercortisolism probably due to antiglucocorticoid effects of elevated progesterone. The diurnal rhythm of cortisol secretion is maintained; the enhanced cortisol production is due to an increase in maternal plasma ACTH concentrations and hyperesponsiveness of adrenal cortex during pregnancy.[25]

Levels of renin and angiotensin rise during pregnancy, leading to elevated angiotensin II levels and markedly elevated levels of aldosterone, up to eight- to tenfolds. The aldosterone levels peak in midgestation and are maintained until delivery. Despite baseline elevations, the various components of rennin-angiotensin-aldosterone system demonstrate a normal response to positional changes, sodium restriction and sodium loading. Another mineralocorticoid, 11-deoxycorticosterone shows a six- to tenfold elevation in concentration at term. Levels of androstenedione and testosterone are elevated because of estrogen-induced increase in hepatic synthesis of sex hormone-binding globulin. Free androgen levels remain normal or low. The production rates of dehydroepiandrosterone and dehydroepiandrosterone sulfate are increased twofold.[26]

Adrenal medullary function remains normal during pregnancy. The measurements of 24-hour catecholamines and plasma epinephrine and norepinephrine are same as that of nonpregnant state.[27]

INSULIN AND METABOLIC HOMEOSTASIS

Hyperplasia and hypertrophy of the beta cells are probably due to results of stimulation by estrogen and progesterone.[28] In early pregnancy, glucose tolerance is generally normal and peripheral sensitivity to insulin and glucose similar to pregravid levels.[29] Although basal insulin levels are normal, there is hypersecretion of insulin in response to meals. Over the course of gestation, the insulin response to nutrients further increases, while glucose tolerance changes only slightly. The balance is maintained by a progressive increase in basal and postprandial insulin concentrations to counteract with the increase in insulin resistance.[30] Towards the end of pregnancy, the basal and 24-hour mean insulin concentration may be twofold greater than nonpregnancy, with first and second phases of insulin secretion threefold greater[31] due to elevated levels of human placental lactogen (hPL) and glucocorticoids. With increases in peripheral insulin resistance slightly higher levels of postparandial concentrations of metabolic fuels—including glucose, lipids, and amino acids—are available for the fetus.

Lipid metabolism is also altered with normal pregnancy. Overall, there is an increase in triglycerides, fatty acids, cholesterol, and lipoproteins throughout pregnancy.[32] Whereas, accumulation of fat depots occurs during the first two-thirds of gestation, increased adipose tissue lipolysis and hyperlipidemia develops in the last trimester. Insulin resistance and increased estrogens in late pregnancy

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contribute to these changes. Lipoprotein receptors and fatty acid binding proteins in the placenta allow the transfer of long-chain polyunsaturated fatty acids to the fetus. Enhanced oxidative stress in pregnancy may be related to maternal hyperlipidemia. Maternal plasma nonesterified fatty acids and cholesterol correlate with those in the fetus, and maternal adipocytokines have been associated with fetal growth. At the molecular level, cytokines are important links between lipid metabolism and insulin resistance.

POINTS TO REMEMBER

- Pregnancy is a dynamic state and evaluation for metabolic dysfunction needs astute observation and planned investigations rather than a blanket of investigations
- Estimation and interpretation of hormonal status during pregnancy is to be correlated to the understanding of normal physiology
- A suppressed TSH level during pregnancy in a previously healthy female does not always point to thyroid dysfunction
- Estimation for hypercortisolism and hypercortisol states is fraught with confounders. Urinary and free cortisol are better markers but maybe spuriously elevated.

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Veeraswamy Seshiah MD FRCP DSc was instrumental in establishing a separate department of diabetology in Madras Medical College and "Feto-Placental-Maternal Unit" in the Institute of Obstetrics and Gynecology, Chennai, Tamil Nadu, India. Both of them are the first of their kind in the country. He is a Founder Chairman Diabetes in Pregnancy Study Group India (DIPSI), Vice-Chairman International Association of Diabetes and Pregnancy Study Group (IADPSG), expert committee member of the World Health Organization (WHO), and Indian Council of Medical Research (ICMR). He is also Chairman of the Board of Studies, of the Tamil Nadu Dr MGR Medical University, Chennai. He has innumerable publications in both national and international journals. He is also reviewer of articles for many journals. Dr Seshiah is a recipient of prestigious Dr BC Roy National Award for the development of the specialty, diabetology in the country.

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ISBN 978-93-5152-372-7
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