PHYSIOLOGICAL CHANGES IN 50 WOMEN DURING PREGNANCY

Afsheen Mehmood[1], Naila Noor[2], Zubia Shah[1], Mohammad Irfan[3]

ABSTRACT

Background: Pregnancy involves physiological adoptive changes in almost all organs & system to prepare a pregnant woman for the growth & development of growing embryo. The objective to assess physiological changes in our own set up. We monitored physiological changes in 50 women at the department of Physiology & gynecology & Obstetric at Hayat Abad Medical Complex.

Results: On initial booking their median weight was 74 Kg, mean Hemoglobin 10.24 gm/dl SD 1.098. Median platelets count 267, median urea 28 mg/dl, median sugar 76 mg/dl. Median blood pressure 110/70 mm Hg. Out of 50, three had twin pregnancy & 47 had single pregnancy. In the second trimester the median weight 80 Kg, Hb 10 gm/dl, platelets count was 346, Urea 29 mg/dl, sugar 76 mg/dl & blood pressure was 100/70 mm Hg. In the third trimester their median weight was 88 Kg, Hb 9 gm/dl, platelets count 359, Urea 30 mg/dl, sugar 87 & blood pressure 100/75 mm Hg.

Conclusion: Pregnancy is associated with statistically highly significant increase in weight, reduction in systolic & diastolic blood pressure, drop in hemoglobin. There was statistically insignificant effect on urea & sugar level.

Key words: Physiological changes, Pregnancy

INTRODUCTION

Female physiological changes in pregnancy are the adaptations during pregnancy that a female's body undergoes to accommodate the growing embryo. These physiologic changes are entirely normal, and involve cardiovascular hematologic, metabolic, renal, posture, and respiratory systems. Increases in blood sugar, breathing, and cardiac output are all expected changes that allow a pregnant person's body to facilitate the proper growth and development of the embryo 8). The pregnant woman and the placenta also produce many other hormones that have a broad range of effects during pregnancy. Pregnant women experience numerous adjustments in their endocrine system that helps to support the developing fetus. The fetal-placental unit secretes steroid hormones and proteins that alter the function of various maternal endocrine glands[2,3,5]. Sometimes, the changes in certain hormone levels and their effects on their target organs can lead to gestational diabetes and gestational hypertension 2.

Place of the study

This study was conducted at the department of Physiology & Gynecology/obstetric/Antenatal clinic Hayat abad medical Complex, Peshawar Nov 2017 till February 2018.

[1] Department of Physiology KGMC Peshawar
[2] Department of Gynae KGMC HMC Peshawar
[3] 2nd Year Student

Address for correspondence:
Afsheen Mehmood
Department of physiology KGMC Peshawar
Cell No.03469370321
Email: am_100@hotmail.com

Study Design

A random sample of convince involving 50 consecutive pregnant women were selected from antenatal clinic in their first trimester. The data was recorded on a proforma at first trimester, second & third trimester. Later it was entered into SPSS & analyzed.

Inclusion & exclusion criteria. All pregnant women with normal pregnancy in their first trimester who were willing to participate were eligible to be included. All pregnant women who had underlying chronic illness involving respiratory, cardiovascular, respiratory, hematological or neurological illness, not willing to participate, from far off area who were not going to come subsequently or pregnancy beyond first trimester were excluded.

RESULTS

On initial booking their median weight was 74 Kg, mean Hemoglobin 10.24 gm/dl SD 1.098. Median platelets count 267, median urea 28 mg/dl, median sugar 76 mg/dl. Median blood pressure 110/70 mm Hg. Out of 50, three had twin pregnancy & 47 had single pregnancy. In the second trimester the median weight 80 Kg, Hb 10 gm/dl, platelets count was 346, Urea 29 mg/dl, sugar 76 mg/dl & blood pressure was 100/70 mm Hg. In the third trimester their median weight was 88 Kg, Hb 9 gm/dl, platelets count 359, Urea 30 mg/dl, sugar 87 & blood pressure 100/75 mm Hg. The decrease in both systolic & diastolic BP from first & third trimester was statistically highly significant (p .0001). There was statically highly significant increase in weight from first to third trimester (p .0001). Platelets count, urea & sugar level did not change to a statistically significant value (P 0.29) (P 0.132) (P0.158) respectively. There was

Statistics

		platelets1	urea1	sugar1	weight1	systolic1	dia-stolicbp1	hb1
N	Valid	50	50	50	50	50	50	50
	Miss-ing	3	3	3	3	3	3	3
Mean		5035.9000	82.6600	244.0200	72.7600	112.5000	67.8000	10.2400
Median		267.5000	28.0000	76.0000	74.5000	110.0000	70.0000	10.0000
Mode		345.00	24.00	77.00a	74.00	120.00	70.00	11.00
Std. Deviation		33515.34926	381.76805	1203.57368	9.59923	9.38138	6.78835	1.09842

Statistics

		hb2	platelets2	urea2	sugar2	systolicbp2	dia-stolicbp2	weight2
N	Valid	50	50	50	50	50	50	50
	Miss-ing	3	3	3	3	3	3	3
Mean		9.9800	336.1200	31.0800	76.8000	103.8000	74.4000	77.3800
Median		10.0000	346.5000	29.0000	76.0000	100.0000	70.0000	80.0000
Mode		9.00a	450.00	29.00	76.00	100.00	70.00	80.00
Std. Deviation		.99980	65.50701	6.10065	9.15401	4.90314	5.01427	10.41955

a. Multiple modes exist. The smallest value is shown

Statistics

		hb3	platelets3	urea3	sugar3	systolicbp3	dia-stolicbp3	weight3
N	Valid	50	50	50	50	50	50	50
	Miss-ing	3	3	3	3	3	3	3
Mean		9.5600	381.6600	31.1600	85.6200	105.7000	70.5000	85.4000
Median		9.0000	359.0000	30.0000	87.0000	100.0000	70.0000	88.0000
Mode		9.00	359.00	26.00	87.00	100.00	75.00	88.00
Std. Deviation		.90711	67.69427	4.31589	3.96356	6.22782	7.01674	9.56396

One-Sample Test

	Test Value = 0					
	T	Df	Sig. (2-tailed)	Mean Difference	95% Confidence Interval of the Difference	
					Lower	Upper
systolic1	84.795	49	.000	112.50000	109.8338	115.1662
systolicbp3	120.012	49	.000	105.70000	103.9301	107.4699

One-Sample Test

	Test Value = 0					
	T	Df	Sig. (2-tailed)	Mean Difference	95% Confidence Interval of the Difference	
					Lower	Upper
diastolicbp1	70.624	49	.000	67.80000	65.8708	69.7292
diastolicbp3	72.961	49	.000	70.70000	68.7527	72.6473

One-Sample Test

	Test Value = 0					
	T	Df	Sig. (2-tailed)	Mean Difference	95% Confidence Interval of the Difference	
					Lower	Upper
weight1	53.597	49	.000	72.76000	70.0319	75.4881
weight3	62.325	49	.000	85.82000	83.0529	88.5871

One-Sample Test

	Test Value = 0					
	T	df	Sig. (2-tailed)	Mean Difference	95% Confidence Interval of the Difference	
					Lower	Upper
platelets1	1.062	49	.293	5035.90000	-4489.0569	14560.8569
platelets3	39.646	49	.000	383.60000	364.1559	403.0441

One-Sample Test

	Test Value = 0					
	T	Df	Sig. (2-tailed)	Mean Difference	95% Confidence Interval of the Difference	
					Lower	Upper
hb1	65.920	49	.000	10.24000	9.9278	10.5522
hb3	74.807	49	.000	9.58000	9.3226	9.8374

One-Sample Test

	Test Value = 0					
	T	Df	Sig. (2-tailed)	Mean Difference	95% Confidence Interval of the Difference	
					Lower	Upper
urea1	1.531	49	.132	82.66000	-25.8373	191.1573
urea3	52.013	49	.000	31.28000	30.0715	32.4885

One-Sample Test

	Test Value = 0					
	T	df	Sig. (2-tailed)	Mean Difference	95% Confidence Interval of the Difference	
					Lower	Upper
sugar1	1.434	49	.158	244.02000	-98.0319	586.0719
sugar3	148.878	49	.000	85.38000	84.2275	86.5325

statically highly significant drop in hemoglobin (p 0.001)

DISCUSSION

A retrospective study of 1145 pregnant women showed that there is trend in mean maternal weight gain from the time of booking until delivery but it is not linear [3]. Statistically significant lower rates of maternal weight gain were seen before 16 weeks, after 36 weeks and between 28 and 32 weeks gestation (P less than 0.05). The mean maternal weight gain was 10.71 kg (SD 4.3) and the mean weekly weight gain was 0.38 kg (SD 0.16). A wide variation of maternal weight gain was seen in women with a normal outcome. The mean weight gain in heavy (greater than 68 kg) and light (less than 55.4 kg) women was less than that in women whose weight was in the third quartile (60-68 kg, P less than 0.05). The mean maternal weight gain was less in young (less than 20 years) women than in older women (greater than 25 years; P less than 0.05), less in parous than in primigravid women from week 37 onwards (P less than 0.05), less in smokers than in non-smokers from 20 weeks onwards (P less than 0.05), and greater in hypertensive women (BP less than 140/90) than in normotensive women (P less than 0.05) from week 24 onwards. The mean weight gain in women who had small for gestational age (SGA) infants was not significantly different from that in women who had infants that were of appropriate size for gestational age. After taking into account infant and placental weight using multiple regression analysis, the factors that were associated with statistically significant differences in average weekly weight gain were parity, body mass index, and smoking habit and raised blood pressure[4,5,6,7,8]. In our study there was highly significant increase in weight from first to third trimester (P 0.0001).

An increase in blood volume has been documented in a number of studies; however, there is variability among studies with regard to the magnitude and timing of this increase[5,6,7]. The increased blood volume delivered to the ventricle during pregnancy increases preload and can be estimated by examining ventricular diastolic volume and pressure. Blood volume begins to increase in week 6 of gestation and by the end of pregnancy it will have reached approximately 50% more than in the pre pregnant state one study demonstrated individual increases from 20% to 100% above pre-pregnant blood volume. All studies have shown that blood volume progressively increases, at least until mid-pregnancy; some studies have found that it plateaus in the third trimester. Whereas others suggest that it increases continuously until term. The increase in blood volume is more pronounced in twin pregnancies. Red cell mass increases as much as 40% above pre-pregnancy levels. The plasma volume increase is proportionally greater than the increase in red blood cell mass, and the resulting hemodilution explains the so-called 'physiological anemia of pregnancy'. Reduced plasma volume expansion has been associated with low birth weight and intrauterine growth retardation. A number of mechanisms are postulated for the hypervolemia of pregnancy. Estrogen increases renin levels and causes sodium retention and an increase in total body water[6,9,10,11,12,13]. Other hormones, such as prolactin, placental lactogen, prostaglandins and growth hormone, are increased during pregnancy and may contribute to fluid retention. In our study there was there highly significant reduction in hemoglobin (P 0.0001) but statically insignificant effect in platelet count (P 0.29)

Despite increasing blood volume and atrial and ventricular distension, cardiac filling pressures have not been shown to be higher in women at term compared with women 11–13 weeks' postpartum The ability of a normal heart to adapt to chronic volume overload probably prevents pressures from increasing in women without heart disease During pregnancy, there is a fall in systemic (peripheral) vascular resistance beginning in week 5 of gestation with a nadir between weeks 20 and 32. After week 32 of gestation, the systemic vascular resistance slowly increases until term. There is a corresponding initial decrease in the systemic arterial pressure, which begins in the first trimester and reaches its nadir at mid-pregnancy. Thereafter, systemic pressure begins to increase again and ultimately reaches or exceeds the pre-pregnancy level. The overall fall in systemic vascular resistance is a result of changes in resistance and flow in multiple vascular beds. During pregnancy, blood flow increases in the low impedance utero placental circulation, reaching up to 500mL/min at term, measured in the supine position, and even higher in the left lateral decubitus position. Placental flow increases until week 25 of gestation and then remains unchanged. In addition there is a fall in resistance caused by increased levels of peripheral vasodilators, in particular prostacyclin (PGI2)[14,15,16,17]. In our study there was significant reduction of both systolic & diastolic blood pressure (P 0.0001).

Renal blood flow increases during pregnancy, peaking in the third trimester at about 60–80% above pre-pregnancy levels. This coincides with a 50% increase in the glomerular filtration rate Changes in renal blood flow are primarily caused by renal vasodilatation and are also altered by positional changes. Supine recumbency and sitting result in lower glomerular filtration rates. Changes in renal function roughly parallel those in cardiac function. GFR increases 30 to 50%, peaks between 16 and 24 wk gestation, and remains at that level until nearly term, when it may decrease slightly because uterine pressure on the vena cava often causes venous stasis in the lower extremities. Renal plasma flow increases in proportion to GFR[18,19,20,21,22,23]. As a result, BUN decreases, usually to < 10 mg/dL (< 3.6 mmol urea/L), and creatinine levels decrease proportionally to 0.5 to 0.7 mg/dL (44 to 62 µmol/L). Marked dilation of the ureters (hydroureter) is caused by hormonal influences (predominantly progesterone) and by backup due to pressure from the enlarged uterus on the ureters, which

can also cause hydronephrosis. Postpartum, the urinary collecting system may take as long as 12 wk to return to normal. Postural changes affect renal function more during pregnancy than at other times; ie, the supine position increases renal function more, and upright positions decrease renal function more. Renal function also markedly increases in the lateral position, particularly when lying on the left side; this position relieves the pressure that the enlarged uterus puts on the great vessels when pregnant women are supine[24,25,26,27,28,29,30]. This positional increase in renal function is one reason pregnant women need to urinate frequently when trying to sleep. In our study there was statistically insignificant effect on urea from first to third trimester (P 0.132)

Pregnancy alters the function of most endocrine glands, partly because the placenta produces hormones and partly because most hormones circulate in protein-bound forms and protein binding increases during pregnancy. The placenta produces the beta subunit of human chorionic gonadotropin (beta-hCG), a trophic hormone that, like follicle-stimulating and luteinizing hormones, maintains the corpus luteum and thereby prevents ovulation. Levels of estrogen and progesterone increase early during pregnancy because beta-hCG stimulates the ovaries to continuously produce them. After 9 to 10 wk of pregnancy, the placenta itself produces large amounts of estrogen and progesterone to help maintain the pregnancy. The placenta produces a hormone (similar to thyroid-stimulating hormone) that stimulates the thyroid, causing hyperplasia, increased vascularity, and moderate enlargement. Estrogen stimulates hepatocytes, causing increased thyroid-binding globulin levels; thus, although total thyroxine levels may increase, levels of free thyroid hormones remain normal. Effects of thyroid hormone tend to increase and may resemble hyperthyroidism, with tachycardia, palpitations, excessive perspiration, and emotional instability. However, true hyperthyroidism occurs in only 0.08% of pregnancies. The placenta produces corticotropin-releasing hormone (CRH), which stimulates maternal ACTH production. Increased ACTH levels increase levels of adrenal hormones, especially aldosterone and cortisol, and thus contribute to edema. Increased production of corticosteroids and increased placental production of progesterone lead to insulin resistance and an increased need for insulin, as does the stress of pregnancy and possibly the increased level of human placental lactogen[31,32,33,35,34,36,37]. Insulinase, produced by the placenta, may also increase insulin requirements, so that many women with gestational diabetes develop more overt forms of diabetes 38. In our study there was statistically insignificant effect on blood sugar from first to third trimester (P 0.158)

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