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ORIGINAL ARTICLE

The Alteration in Parathyroid Hormone and Calcitonin of Pregnant Women in Babylon Governorate

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Abstract

Background The peptide hormones parathyroid hormone (PTH) and calcitonin (CT) both work on osteoblasts (bone-forming cells) and osteoclasts (bone-resorbing cells) to regulate calcium homeostasis.
Aims of the study This study aimed to evaluate the changes in serum levels of parathyroid hormone and calcitonin in pregnant and non-pregnant women in Babylon Governorate, Iraq.
Materials and methods This study designs as a case-control study performed on pregnant women who attended Women's and Children's Hospital in Hilla and Shomali General Hospital in Babylon Governorate. Forty pregnant women with a range age (18-40 years) and 50 healthy subjects with non-pregnant women with range age (18-40 years) visited the hospital for routine check-ups without any history of chronic diseases, acute illness and infection. The biochemical parameters were determined by the sandwich ELISA technique.
Results The results show a significant increase in levels of parathyroid hormone (PTH) and calcitonin (CAL) in pregnant as compared with the non-pregnant group (277.7 ± 47.87 ng/mL),(72.58 ± 12.76 ng/mL) and (21.89 ± 4.412 ng/mL), (9.65 ± 1.921 ng/mL) respectively. The significant difference (p-value < 0.0001).
Conclusion In conclusion, the present study finds that pregnant women experience significant hormonal changes in PTH and CT, which indicates the necessity of diagnosing these changes and treating them for the success of this pregnancy.

Keywords: Parathyroid hormone; Calcitonin; Pregnancy.

1 Introduction

The hormone that has the most influence on regulating the rate of the body's metabolism is the thyroid hormone [1]. Thyroid hormone is essential for a baby's brain development. Thyroxine (T4) and triiodothyronine (T3), the thyroid gland's two primary hormones, are combined to form thyroid hormones [1]. Because T4 is mainly inactive, meaning it has little effect on cells, and T3 is active, they are frequently referred to as "thyroid hormone" collectively. The human body's organs convert T4 that has been released by the thyroid into T3 so that it can affect cells and metabolism [2]. To control the amount of calcium in the blood, the thyroid also secretes a hormone called Calcitonin. Calcitonin does not affect your body's metabolism in the same way as T3 and T4 do, and it is not classified as a thyroid hormone [3].

Calcitonin, a 32-amino-acid peptide hormone, is released by the thyroid's parafollicular cells (C cells in humans and other chordates). It lowers blood calcium (Ca^{2+}) unlike parathyroid hormone. Calcitonin, discovered over 50 years ago, rapidly reduces blood calcium [4]. Many species have determined the amino

acid sequences of CTs, which have an intramolecular disulfide bridge between the cysteine residues at positions 1 and 7 and an amidated carboxy-terminal with identical amino acid residues [5]. The calcitonin family contains CGRPs (αCGRP and βCGRP), amylin, adrenomedullin, and intermedin. The peptides signal via comparable receptors and share certain biological functions [6].

Parafollicular cells (C-cells) in the thyroid gland generate Calcitonin. The CALCA gene controls its production from procalcitonin, a bigger peptide. Calcitonin regulates calcium levels. This hormone lowers excessive calcium levels. Calcitonin, a potent bone resorption inhibitor, inhibits calcium efflux from the bone. Calcitonin has been used clinically for Paget's disease and osteoporosis, but its usage has waned due to the introduction of more effective bone resorption inhibitors [7, 8]. Four spherical, rice-sized particles make up the parathyroid gland. Two reside beneath the thyroid gland on either side of the neck. The superior and inferior parathyroid glands originate from the epithelial lining of the third and fourth pharyngeal pouches. Since embryological structures migrate, the inferior and superior glands, termed for their ultimate locations, shift position [9,10].

Parathyroid hormone (PTH) is a polypeptide produced by parathyroid chief cells and cleaved into an active form in the gland [11]. Parathyroid hormone increases blood calcium (Ca^{2+}), whereas Calcitonin, generated by the thyroid glands' parafollicular cells (C cells), decreases it (20,21). PTH increases blood calcium by acting on the parathyroid hormone 1 and 2 receptors [12]. The parathyroid hormone controls blood calcium and releases calcium from the vast bone reserve. It increases distal tubule and thick ascending limb calcium and magnesium active reabsorption [13]. Activated vitamin D synthesis increases intestinal calcium absorption. The parathyroid hormone is the primary body hormone. Synthesis, cleavage, and storage take under an hour [14]. Low blood calcium may activate PTH production in seconds. Exocytosis releases hormones from membrane vesicles that merge with the cell membrane. Activated PTH has a few-minute serum half-life and is swiftly eliminated by the kidney and liver [15].

2 Materials and Methods

2.1 Ethical Considerations

The Women's and Children's Hospital in Hilla, Shomali General Hospital, and the College of Medicine Committee at the University of Babylon authorized the research, which involved collecting blood samples and experimental methods. Before collecting any samples, all research participants gave their informed permission. Additionally, all procedures and methodologies adhered to the rules and regulations set out by the Babylon Province Ethical Committee of the Central Directorate of Health.

2.2 Study Design

This study designs as a case-control study performed on pregnant women who attended Women's and Children's Hospitals in Hilla and Shomali General Hospital. They were admitted as pregnant women whom expert physicians diagnosed.

2.3 Subjects

There were 40 pregnant women in the study, ranging in age from 18 to 40. Fifty healthy non-pregnant subjects were enrolled in the study between November 2022 and March 2023 at Women's and Children's Hospital in Hilla and Shomali General Hospital. They ranged in age from 18 to 40 years old and had no history of chronic illnesses, acute illnesses, or infections. The Women's and Children's Hospital in Hilla, Shomali General Hospital, and the Chemistry and Biochemistry Department of the College of Medicine at the University of Babylon conducted all laboratory testing and analyses. Each Subject completed an informed consent form in writing. Body weight (kg) divided by the square of height (meters) yields the body mass index (BMI). According to the manufacturer manual, the Calcitonin and parathyroid hormone (PTH) were determined using Mybiosource® (China) sandwich ELISA technique.

3 Results and Discussion

3.1 Parathyroid hormone

The results show an increased concentration of parathyroid hormone (PTH) (pg/mL) in pregnant as compared with the non-pregnant group (277.7 $\pm$ 47.87), (72.58 $\pm$ 12.76) (pg/mL) respectively; the significant difference (p-value <0.0001). as shown in Table 1, Figure 1.

Table 1: Comparison of mean values of Parathyroid hormone in women pregnant and non-pregnant.

Characteristic	Non-pregnant	Pregnant	P value
	n= 50	n= 40	
Parathyroid hormone. pg/ml			
Range	40.87 - 96	160 - 356.8	<0.0001
Mean ± SD	72.58 ± 12.76	277.7 ± 47.87	

SD:Standard Definition

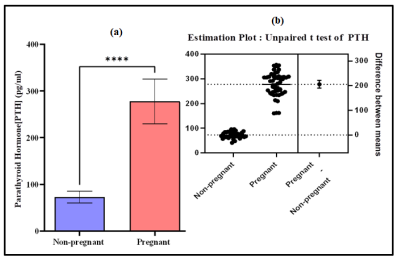

Figure 1: Estimation of serum parathyroid hormone (PTH) (pg/mL). (a) a comparison between the non-pregnant and pregnant group, (b) an estimation plot that illustrates the presence of a significant increase in the level of PTH in the pregnant group as compared to the non-pregnant, the significant difference (p-value <0.0001). Data are expressed as means ± SD. indicates *significant differences compared to the non-pregnant, P≤0.05.

The results show an increased concentration of parathyroid hormone (PTH) (pg/mL) in the third Trimester (290.9± 47.48) (pg/mL) compared with the second Trimester and first Trimester (285.8± 29.60), (253.5 ± 54.44) (pg/mL) respectively; the significant difference (p-value = 0.0431) The measurement of PTH (pg/mL) showed an insignificant difference was present in mean values between the first Trimester and with second Trimester (p-value = 0.1242); the significant difference in mean values between the first Trimester and third Trimester (p-value= 0.0497); the non-significant difference in mean values between second Trimester and third Trimester (p-value = 0.9451), As shown in Figure 2.

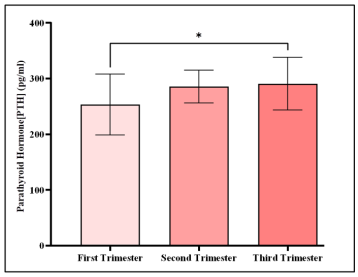

Figure 2: Estimation of serum parathyroid hormone (PTH) (pg/mL)in Stages of Pregnancy. First Trimester (0-13 Weeks), Second Trimester (14-26 Weeks), and Trimester (27-40 Weeks).

The hormone parathyroid hormone (PTH) is crucial for maintaining calcium homeostasis. It is affected by minute variations in blood calcium levels and has a brief half-life of just 5 minutes. The need for calcium rises during pregnancy. Concerning the fetal upper arm, calf, and birth weight, maternal PTH levels are positively correlated [16, 17]. The parathyroid hormone regulates the fetoplacental mineral homeostasis, skeletal development, and stimulation of placental calcium transfer [18]. The maintenance of ionized calcium in the blood, raising the level of calcium phosphate released from bone tissue, conserving calcium, reducing tubular phosphate reabsorption, and increasing intestinal calcium absorption through vitamin D are some of the processes that parathyroid hormone is involved in [18]. Compared to non-pregnant women, our estimate revealed a highly significant elevation of parathyroid hormone in pregnant women. Additionally, our estimate is comparable to estimates from populations in other pregnancy-related studies [19–21]. According to the results of our research and those of other studies, the rise in PTH may be explained as follows: Plasma proteins, mostly albumin, carry the other half of the serum calcium in bound form [22]. Total blood calcium slightly decreases during pregnancy due to hypoalbuminemia, increased renal clearance, and placental transfer to the baby. To maintain calcium homeostasis, the parathyroid glands release a parathyroid hormone. PTH concentrations are repressed into the low normal range and may even decrease below the normal range, according to other investigations that reported results inconsistent with our findings [23, 24]. A remarkable set of physiological changes occur throughout pregnancy to maintain the mother's calcium homeostasis and to meet the baby' needs for development and skeletal mineralization. The fundamental component maintaining the mother's calcium homeostasis may be increased in blood 1,25 (OH) 2D levels during pregnancy. Uncertainty surrounds the cause of increased renal and/or placental 1-alpha-hydroxylase activity. PTH levels have been reported to increase during pregnancy, and some researchers have hypothesized that this increase may encourage the production of 1,25 (OH) 2D [25–28]. The use of antibodies with different

specificities to PTH and the heterogeneity of inactive fragments of the hormone resulting from peripheral metabolism and increased glomerular filtration that occurs in pregnancy may cause conflicting information regarding PTH concentrations in pregnancy (24) (24). A 16-subject longitudinal study was reported by Naylor et al. During the first Trimester of pregnancy, PTH levels were reported to decline by 47%, and they thereafter gradually rose but remained below baseline [29]. According to research by Ardawiet et al., intact-PTH concentrations rose from 1.31 pmol/l in the first Trimester to 2.26 pmol/l in the second Trimester before falling back to first trimester levels and then dramatically increasing postpartum [28]. It was proposed that the main mediator of alterations in maternal calcium metabolism during pregnancy was an increase in calcitriol concentration [30]. Similarly to this, Rasmussen et al.'s study of 20 healthy pregnant women concluded that pregnancy does not result in a physiological hyperparathyroidism state [31].

It is known that parathyroid hormone (PTH) levels are increased in women during pregnancy and lactation [32]. Pitkin et al. [33] suggested that physiologic hyperparathyroidism exists during pregnancy [34]. In this regard, Heaney and Skillman [35] found that intestinal absorption of calcium doubled in pregnancy, a fact they attributed, in part, to increased PTH. Perhaps the osteolytic activity of PTH is countered by the hypersecretion of Calcitonin. This action may permit the calcium retaining actions of PTH to be exerted on the gut and kidney while the calcium needs of the fetus are met, thus, sparing the maternal skeleton [30].

3.2 Calcitonin

The results show increased levels of Concentration of Calcitonin (CAL) (pg/mL) in pregnant as compared with the non-pregnant group (21.89 ± 4.412),(9.65 ± 1.921) (pg/mL) respectively; the significant difference (p-value <0.0001) as shown in Table 2, Figure 3.

Table 2: Comparison of mean values of Parathyroid hormone in women pregnant and non-pregnant.

Characteristic	Non-pregnant n= 50	Pregnant n= 40	P value
Calcitonin. pg/ml			
Range	6.335 - 14.26	14.37 - 29.36	<0.0001
Mean ± SD	9.65 ± 1.921	21.89 ± 4.412	

SD:Standard Definition

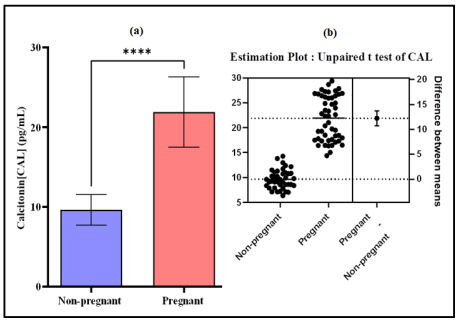

Figure 3: Estimation of serum calcitonin (CAL) (pg/mL). (a) a comparison between the non-pregnant and pregnant group, (b) an estimation plot that illustrates the presence of a significant increase in the level of CAL in the pregnant group as compared to the non-pregnant, the significant difference (p-value <0.0001). Data are expressed as means ± SD. indicates *significant differences compared to the non-pregnant, P≤0.05.

The results show an increased concentration of Calcitonin (CAL) (pg/mL) in the third Trimester (25.55± 3.291) (pg/mL) compared with the second Trimester and first Trimester (21.21 ± 3.991),(19.65 ± 3.915) (pg/mL) respectively; the significant difference (p-value = 0.0003). The measurement of CAL (pg/mL) showed an insignificant difference was present in mean values between First Trimester and with Second Trimester (p-value = 0.4368); the significant difference in mean values between first Trimester and third Trimester (p-value= 0.0002); the significant difference in mean values between second Trimester and third Trimester (p-value = 0.0059), As shown in Figure 4.

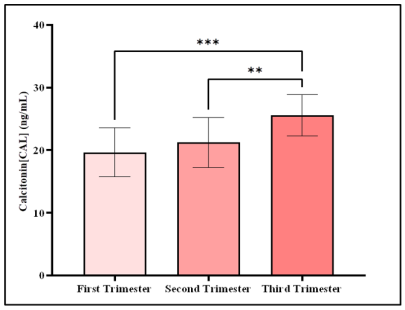

Figure 4: Estimation of serum calcitonin (CAL) (pg/mL) in Stages of Pregnancy. First Trimester (0-13 Weeks), Second Trimester (14-26 Weeks), and Trimester (27-40 Weeks).

The C-cells of the thyroid gland manufacture and release the hormone calcitonin. Its biological purpose in humans is to play a minor part in maintaining a healthy calcium balance. During pregnancy, significant amounts of Calcitonin are in the blood [36]. The thyroid's hypertrophied C cells are the most probable suppliers, while breast and placental cells may also be involved. Human studies have not conclusively shown any major impact of Calcitonin on calcium metabolism during pregnancy, even though it is hypothesized to influence the maternal bone [37]. Thyroid volume (TV) and function fluctuate due to pregnancy's substantial effects on thyroid homeostasis.

Additionally, it has been shown that Calcitonin (CT) and its gene-related peptide are crucial for the implantation process [38]. Our research shows pregnant women had higher blood calcitonin levels than non-pregnant women. The results were corroborated by those of another research, which showed that levels of this hormone were assessed in 56 pregnant women throughout all trimesters and found to be 72% higher than normal. A few weeks after delivery, the elevated blood calcitonin levels found during pregnancy progressively return to normal. Uncertain is its function in human pregnancy's physiology of the bones [39, 40].

Konopka et al [41] measured the serum calcitonin of no pregnant and pregnant women by bioassay and found that 57.4% of pregnant women had increased values [41]. The second and third trimesters of pregnancy saw a statistically significant rise. Second and third-trimester levels were comparable; however, postpartum values were somewhat higher. According to these scientists, hypercalcitonemia may protect the bones against demineralization when a woman is pregnant. The rise in CT levels in women during birth was initially noted by Samaan et al. [17]. It would appear that the hypercalcitonemia of pregnancy is not explicable exclusively based on maternal to fetal calcium transfer or hyperestrogenism. However, there was no significant increase in Calcitonin from the second to the third Trimester. Similarly, estrogen production by the placenta is not likely to be the sole stimulus to calcitonin secretion since placental estrogen increases progressively until delivery [42, 43].

Modifications in calcium homeostasis are a characteristic of pregnancy and the perinatal period. PTH, 1,25-dihydroxy vitamin D (1,25 (0H)*D), and Calcitonin (CT) are all involved in the control of calcium homeostasis; the elevated levels of CT during pregnancy suggest that this hormone contributes to the protection of the mother's bones. However, precisely what each one does throughout pregnancy and on the first day of life is unclear. In the third Trimester of pregnancy, some writers discovered high quantities of the hormone's amino-terminal form and enhanced biological activity, while others observed typical levels of the hormone's carboxyl-, amino-, and complete forms [44–46].

4 Conclusions

The present study finds that pregnant women experience significant hormonal changes in parathyroid hormone and Calcitonin, which indicates the necessity of diagnosing these changes and treating them for the success of this pregnancy.

Conflict of Interest: No conflicts of interest exist between the authors and the publication of this work.
Ethical consideration: The ethical committee approved the study at University of Babylon, Hillah, Iraq.

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