IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 17, Issue 7 Ver. 7 (July. 2018), PP 04-10
www.iosrjournals.org

Ultrasound morphology of the thyroid gland among pregnant women in the moderate iodine deficient region of Lubumbashi, DR Congo

TwiteKabange Eugène[1], Laurence Habimana[2], Pierre Bernard[3], BienvenueIlunga Banza[4], Etienne Kasongo Mpoyo[1], Damien Gruson[5], Philippe Donnen[6], Philippe DeNayer[5], NyemboMukenaChristophe[1], KalengaMuenzeKayambaProsper[1], Annie Robert[2].

1. Biomedical sciences department, Faculty of medicine, University of Lubumbashi (UNILU).2. Biostaticsunity, Faculty of Public health, Catholic University of Leuven (UCL). 3. Obstetric service, Saint Luc Hospital, Catholic University of Leuven(UCL),4.Chemistry department, Faculty of Sciences, University of Lubumbashi(UNILU), 5.Endocrinology laboratory, Saint Luc hospital, Catholic University of Leuven (UCL), 6. Epidemiology,NutritionandDévelopment unity, ,Public health School, Free University of Brussels (ULB)
Corresponding authors: TwiteKabangeEugène

Abstract:
Background: *Iodine deficiency occurs in varying degrees worldwide. When it occurs during pregnancy, it leads to morphological and functional changes in the maternal thyroid gland with fatal consequences on the pregnancy's way out. The aim was to evaluate morphological condition of the thyroid gland and to compare this to thyroid stimulating hormone (TSH) levels as well as to urinary iodine levels among pregnant women in Lubumbashi*
Method:*a cross-sectional study among 225 pregnant women from three areas: Lubumbashi University Clinic (urban zone), Bongonga Health Center (semi-urban zone) and KatubaHospital (urban-rural zone) between March 2009 and February 2012. Thyroid ultrasounds were performed using 7.5 MHz probe frequency. TSH, T4L and T3L was performed by enzyme immunoassay using ETI Diasorin equipment. Urinary iodine was measured after oxidation by ammonium persulfate. In order to analyze the data, the software Epi-Info™ Version 3.3.2 was used.*
Results:*12% of pregnant women had thyroid abnormality. Pregnant women with a subclinical goiter or a nodule were on average older than those which no morphological abnormalities (p=0.01) and the family history of goiter was more frequent among them (p=0.01). Women with subclinical goiter had a lower median urinary iodine concentration. Among the 28 women with subclinical hypothyroidism, only 4 (14%) showed morphological abnormalities on the ultrasound.*
Conclusion: *morphological and functional thyroid changes are relatively frequent among pregnant women in Lubumbashi. Given the impact of these disorders on pregnancy and the fetus, quality control on commercialized salt in these areas must be strengthened and Thyroid function monitored.*
Keys words: *Thyroid, pregnancy, goiter, nodule, iodine, Lubumbashi*

Date of Submission: 30-06-2018 Date Of Acceptance: 16-07-2018

I. Introduction

Iodine deficiency is one of the most common worldwide nutritional deficiencies. It affects about two billion people and is a serious public health issue(1). It is the leading preventable cause of mental retardation among children, occurring in varying degrees throughout developing countries, and is still not completely under control in developed countries. According to the ICCIDD, 64% of the 600 million Western and Central Europeans had an insufficient iodine intake in 2003 (2). All over the world, the global estimate of iodine nutrition including 130 countries showed that iodine intake was insufficient in 15 countries, sufficient in 102 countries and excessive in 10 countries (3). When iodine deficiency occurs during pregnancy, it accentuates the negative maternal iodine balance by the transplacental transfer of iodine from the mother towards the fetus, and by an increased glomerular filtration rate. Iodine requirements are therefore higher during pregnancy; thyroid gland activity is amplified both as a result of the stimulating effect of the human chorionic gonadotrophin (hCG) and as a result of the increased synthesis of thyroxin binding globulin (TBG), secondary to the increased levels of estrogen, which induce a diminished free thyroxine (T4) concentration (4,5). With sufficient iodine

intake, thyroid function is preserved. Insufficient intake, on the other hand, causes a disruption in the fragile equilibrium and thyroid function deteriorates, leading to hypothyroidism. Hypothyroidism has many negative effects on pregnancy including miscarriage, intrauterine growth retardation, and threatened pre-term labor (6).

In cases of severe iodine deficiency during pregnancy, neonatal mortality is increased and the neurological consequences for the child are deplorable, especially when the deficiency occurs during the first half of the pregnancy (7, 8). In addition to fatal consequences for the fetus, iodine deficiency leads to morphological changes in the maternal thyroid gland, going from nodules to goiter, whose size is proportional to the length and severity of the deficiency. In most cases, the goiter only partially regresses after pregnancy, and the mother subsequently risks getting a multinodular goiter which can later turn to toxicity (9).

In the Democratic Republic of Congo, an evaluation carried out in 2001 showed that iodized salt was available in 97% of households and that median urinary iodine was 495 µg/l. In addition, 10% of subjects had a urinary iodine level below 100 µg/l, and the prevalence of goiter was 5.7% (10). These results demonstrated great progress towards eliminating iodine deficiency disorders (IDD) as a public health problem. A recent study in Lubumbashi by Habimana et al. (11)looking at pregnant women found a median urinary iodine of 138 µg/L and the extent of the iodine deficiency increased with gestational age. In the previous year, Kitwa et al (12). showed that among samples of table salt coming from varying markets in Lubumbashi, 36.3% had less than 15ppm of iodine. These studies suggest that iodine deficiency remains a problem in the DR Congo. Our current study aims to evaluate the morphological condition of the thyroid gland and to compare this to thyroid stimulating hormone (TSH) levels, as well as to urinary iodine levels among pregnant women living in Lubumbashi.

II. Materials And Methods

We conducted a cross-sectional study in Lubumbashi at the South-east of the Democratic Republic of Congo exactly in the High Katanga province. 225 pregnant womenwere recruited during prenatal consultations at the Lubumbashi University Clinic (urban zone), at the Bongonga Health Center (semi-urban zone) and at the Katuba General Referral Hospital (urban-rural zone) between March 2009 and February 2012. Within each participating structure, there were 25 women for each of the trimesters of pregnancy. This ensured a representative sample that included all three trimesters and all three socio-economic backgrounds. The sample size was calculated in order to be able to detect an iodine deficiency prevalence of 0.25, with a 10% precision in each zone in Lubumbashi.

For each of the selected women, after voluntary consent, gestational age was determined based on the date of the woman's last period, if she remembered, counting the first day of menstruation. In order to minimize recall bias, women were interviewed by two different personsat a two hour interval. For women who did not know the date of their last period, we measured fundal height using a metric tape measure, going from the upper edge of the pubis to the fundus. Leroy reference tables (13) were subsequently used to calculate the clinical gestational age. We compared clinical age to the age calculated by ultrasound using various fetal biometric parameters (crown-rump length, gestational sac, biparietal diameter, abdominal circumference, and femur length). We also considered socio-demographic variables, maternal age, parity, occupation and family history of goiter.

Thyroid ultrasounds were performed coupled with a color Doppler, using a probe frequency of 7.5MHz. The thyroid volume was obtained, in most cases, by taking the sum of the two lobes' volumes. The isthmus volume was also taken if increased. Lobe volume was determined using the following formula: height x length x width x 0.52 (14). We subsequently searched for the presence of nodules and explored their specific characteristics (size, ultrasound structure, echogenicity, location and vascularization). Subclinical goiter (a thyroid volume > 18ml) and/or the presence of a nodule were main morphological abnormalities observed in the thyroid gland.

For all pregnant women, blood and urine samples were taken in order to respectively measureserum TSH and urinary iodine levels. The TSH dosage was performed by enzyme immunoassay using ETI Diasorin equipment. The kits used came from the Diametra Company (15). Normal TSH values went from 0.03 to 2.5 mUI/l for the first trimester, 0.1 to 3 mUI/l for the second trimester, and 0.2 to 3 mUI/l for the third trimester. Hypothyroidism was defined as a TSH level above 2.5 mUI/l (first trimester), or 3 mUI/l (second and third trimester). Hyperthyroidism was defined as a TSH level below 0.03 mUI/l (first trimester), 0.1 mUI/l (second trimester) or 0.2 mUI/l (third trimester) (16, 17, 18, 19, 20, 21)

Urinary iodine was measured after oxidation by ammonium persulfate; dose assessment was based on the Sandell Kolthoff reaction using a microplate that measured the speed of the color change of cerium ions (Ce IV) catalyzed by iodide ions (I^-) in the presence of arsenic acid (As III) (22). This technique has a within subjects coefficient of variation (repeatability or precision, n=10) of 1.5% and a between subjects coefficient of variation (reproducibility, n=10) of 3.5%. Standard iodine concentrations set by the World Health Organization for pregnant women are as follows: median less than 150µg/l: iodine deficiency; 150 - 249 µg/l: adequate iodine

intake; 250 - 499 µg/l: elevated iodine; and over 500 µg/l: excess iodine. Iodine deficiency itself is subdivided into the following three categories: mild deficiency: 100 - 149 µg/l; moderate deficiency 50 - 99 µg/l; severe deficiency: 20 - 49 µg/l (23).

In order to analyze the data, the software Epi-Info™ Version 3.3.2 was used. Given the non-Gaussian distribution of the thyroid volume values, a logarithmic transformation was performed [24]. Data was presented as the geometric mean (geometric standard deviation). Comparison of thyroid volumes according to trimester of pregnancy was done using an ANOVA Fisher F-test. A non-parametric Mann-Whitney test was used to compare urinary iodine medians between the different groups of pregnant women. The threshold for statistical significance for all tests was set at $p<0.05$, and all tests were two-sided.

III. Results

Table 1 presents socio-demographic characteristics of the 225 pregnant women who were examined. Women were older in the urban zones than in the semi-urban or urban-rural zones. In each zone, women reported a family history of goiter (5.3 to 6.6%).

As shown in Table 2, 45 to 63% of the pregnant women examined had urinary iodine concentration below 150 µg/l; median urinary iodine in semi-urban zones and in urban-rural zones was below the threshold of deficiency. On the other hand, the median thyroid stimulating hormone (TSH) serum concentration was within normal limits in all three zones, although slightly higher in the semi-urban and urban-rural zones, where subclinical hypothyroidism was more frequent. In total, 18 (8%) of the 225 pregnant women examined had hypothyroidism and 28 (12%) of women had subclinical hypothyroidism. Hypothyroidism was most frequent in the urban-rural zone, but the difference between zones was not statistically significant.

Thyroid ultrasounds showed that 11 (4.9%) women had a subclinical goiter and nodules were detected in 15 (6.7%) of the screened women. As shown in Table 3, pregnant women with a subclinical goiter or a nodule were on average older than those in which no morphological abnormalities were detected. A family history of goiter was more frequent in women with a subclinical goiter. Although the results were not statistically significant, women with a subclinical goiter had a lower median urinary iodine concentration. Subclinical hypothyroidism was also more common among these women ($p<0.01$). One should note, however, that among the 28 women with subclinical hypothyroidism, only 4 (14%) showed morphological abnormalities on the ultrasound.

IV. Discussion

The assessment of iodine levels in our study showed that median urinary iodine concentrations among pregnant women in semi-urban and rural zones was below the WHO's standard values, which are between 150 and 249 µg/l for pregnant women (4). Overall, 53% of the tested women had an insufficient iodine intake. This is consistent with the observations of Kitwa et al. (12), who reported that 36.3% of commercialized salt in Lubumbashi was inadequately iodized and that 14% of salt was entirely non-iodized. In the study lead by Al-Yatama et al. in Kuwait (25), 56.8% of pregnant women had aurinary iodine concentration below 140 µg/l; this was particularly the case for women in their second and third trimester. Pregnancy contributes to the negative maternal iodine balance. This is due both to the transplacental transfer of iodine from the mother to the fetus, as well as to the increased renal clearance of iodine during pregnancy (26). The thyroid gland has an immense capacity to adapt to increased thyroid hormone requirements during pregnancy. Iodine deficiency during pregnancy, however, renders this adaptation insufficient, resulting in insufficient thyroid hormone production both for the mother and the fetus, with deplorable consequences for the fetus' future neurological development (27). In addition to insufficient iodine intake, poor appetite for salt and salt-restriction during pregnancy in order to prevent dysgravidia, possible poor salt conservation by both vendors and consumers, and cooking style are all factors that may reduce the bioavailability of iodine content in table salt, thus affecting the function and morphology of the thyroid gland during pregnancy (5).

In the unstable environments of Lubumbashi, like the semi-urban zones, the salt that is sold in the various open markets is often exposed to air and sunlight, leading to a reduction in the iodine content. This reduction in bioavailability is also linked to other factors including poor conservation methods and inappropriate cooking techniques. These factors could explain the large number of subclinical hypothyroidism cases observed in semi-urban (25%) and urban-rural areas (9%), especially when compared to the urban zone (3%). In contrast, in urban zones, pregnant women generally get their supply of salt in stores, where the salt is packaged and therefore maintains its iodine content. Hieronimus et al. (28), in their study on 330 pregnant French women in their third trimester, found hypothyroidism in 30% of cases. Iodine stock repletion during pregnancy is harmless as long as iodine intake remains sufficient, either by increasing food sources, or by taking iodine supplements. However, in areas where iodine intake is below the daily recommended values (200 µg per day), the negative maternal iodine balance, caused by an increased renal clearance and by transplacental transfer of iodine, may be responsible for hypothyroidism (29, 26). Hypothyroidism impacts the maternal-fetal relationship in many ways.

It may cause, in part, abortions, premature labor, and intra-uterine growth retardation. Hypothyroidism also disrupts proper development of the fetal brain, especially if it occurs in the beginning of pregnancy (30; 31). In developed countries, the most common cause of hypothyroidism during pregnancy is Hashimoto's thyroiditis, an auto-immune disease. However, in developing countries, iodine deficiency is the most common cause of hypothyroidism (32). Because the symptoms of this disorder are frequently absent and non-specific in pregnant women (33), one does not often consider the thyroid as being the cause of the aforementioned obstetrical complications. This is especially the case in underequipped regions considering the fact that a diagnosis during pregnancy is primarily based on blood assay. Technical support in these underequipped regions is therefore clearly necessary in order to screen for thyroid dysfunction during pregnancy. Authors need to come to an agreement and establish a screening procedure motivated by a family history of thyroid dysfunction, or by the consequences within the general population in regions with a sufficient iodine intake. We believe that a systematic iodine supplementation during pregnancy is essential in iodine deficient countries in order to ensure proper functioning of the thyroid gland, even for cases in which the thyroid is morphologically abnormal (12% in our study). In our study, one out of five women had hypothyroidism or subclinical hypothyroidism; a bioassay of thyroid hormones, even though it requires sizable equipment, must be performed on pregnant women in theDR Congo in order to detect subclinical or clinical hypothyroidism, which is treatable by thyroxin.

In our study group, in cases of morphological thyroid abnormalities, namely subclinical goiter, the thyroid function was highly impaired. Subclinical hypothyroidism was the most frequent of impairments. In the study by Vila et al. (34), pregnant women who were iodine deficient in the beginning of their pregnancy, and who did not benefit from iodine supplements during pregnancy, showed an increased thyroid volume during the third trimester when compared to women who had received iodine supplements. Pregnancy is often associated with morphological changes in the thyroid. About 80% of women undergo changes in their thyroid gland during their pregnancy, going from a simple increase in volume to the occurrence of a goiter and/or a nodule (35). However, this volume increase attributed to pregnancy may mask cases caused by iodine deficiency, which is a major cause of goiter in developing regions. The duration and severity of the iodine deficiency, genetic predispositions as evidenced by a high family history of goiter during pregnancy (27%), goitrogenic foods, and smoking are all factors that, when combined with pregnancy, prevent the gland from adapting, leading to goiter and/or nodules (36). According to the 2000 yearly report from the Scientific Committee on Food Health(37), physiological iodine supplementation (100 to 200 µg of iodine) does not have any adverse effects on pregnant women, even if they already have sufficient intake, provided that the thyroid is healthy. However, excessive intake of iodine on a pathological thyroid, including hot nodules, can lead to hyperthyroidism (38) due to the decreased release of iodine (Wolf-Chaikoff effect).

In our study in the DR Congo, 26 (12%) of the 225 pregnant women who were recruited showed a morphological abnormality upon thyroid ultrasound. This high number supports the argument arguing the importance of an ultrasound exploration of the thyroid gland before, during and after iodine supplementation. Any subclinical or clinical modification in the gland during pregnancy in a region where iodine intake is unstable, like the DR Congo, must attract physicians' attention; the gland must undergo a functional evaluation in order to detect a possible clinical or subclinical hypothyroidism, which is most often asymptomatic. Let us note, however, that among the 28 women with subclinical hypothyroidism, 24 (86 %) had a normal thyroid morphology. These findings highlight the need for both bioassays and ultrasounds to detect subclinical hypothyroidism cases that could benefit, or not, from early treatment to maximize the chances of good fetal neural development in the DR Congo.

V. Conclusion

Our results show that morphological changes in the thyroid gland (goiter or nodules) are relatively frequent among pregnant women in Lubumbashi. In pregnant women with goiter, the urinary iodine was the lowest; thyroid function was the most perturbed for those with thyroid nodules. The majority of pregnant women who had an altered thyroid function came from semi-urban and rural zones of Lubumbashi. Given the impact of these disorders on pregnancy and what follows, as well as on the neurological future of the fetus, quality control on commercialized salt in these areas must be strengthened. Thyroid morphology and function in pregnant women must also be monitored in order to reduce the risk for mothers, and maybe also the risk of developing neurological damage in fetuses in DR Congo.

Acknowledgments

This study was supported by a grant from the Commission Universitaire pour le Développement (CUD - PIC 2008 RD Congo, www.cud.be) of the Belgium Government.

Conflicts of interest:theauthors declaredthat thereis no conflicts of interest

Autors' Contributions:

- ✓ **Twite KE:**implemented research, analyzed bloodand urinary samples, realized thyroid ultrasound, wrote the paper
- ✓ **Habimana L :** analyzed data, revised the paper
- ✓ **Bernard P :** revised the paper
- ✓ **Banza IB:** analyzed bloodand urinary samples
- ✓ **Mpoyo KE** : implementedresearch
- ✓ **Gruson D :** revised the paper
- ✓ **Donnen Ph :** revised the paper
- ✓ **DeNayer Ph :** revised the paper
- ✓ **Nyembo MC :** revised the paper
- ✓ **Kalenga MK :** took the responsability of the research
- ✓ **Robert A. :** took the responsability of theresearch

References

[1]. OMS Assessment of Iodine Deficiency Disoders and monitoring their elimination.2007

[2]. Pinchera A.Foreward. In: First symposium of ICCIDD West-Central Europe: Iodine deficiency in Europe. J Endocrinol Invest.2003; 26: 6 -7.

[3]. Gizak M, Gorstein J, Andersson M. Epidemiology of Iodine deficiency. In: Iodine deficiency and their elimination; Springer: Berlin, Germany, 2017; pp. 29-43.

[4]. Caron P, Glinoer D, Lecompte P, Orgiazzi J, WemeauJL.Apport iodée en France : prévention de la carence iodée au cours de la grossesse et de l'allaitement. Ann Endocrinologie.2006; 67(4): 281-6.

[5]. Ducarme G, Berthera TJ, Vuillard E, Polak M, Guibourdenche J, Luton D. Pathologie thyroidienne et grossesse. Rév. Méd. Interne. 2007; 28(5): 314-321.

[6]. GlinoerD.Thyroid disease during pregnancy In: Werner and Ingbar's The Tyroid: A Fondamental and Clinical Text. Ed. LE Braverman and RD Utiger. JB Lippincott-Raven, Philadelphia.2005: 1086-108.

[7]. Delange FM, Dunn JT. Iodine deficiency. In: Braverman LE, Utiger RD (eds) Werner and Ingbar's The Thyroid: A Fundamental and Clinical Text, 9th edition. Lippincott, Williams and Wilkins, Philadelphia.2005: 264-288.

[8]. Abalovich M, Amino N, barbour LA, Cobin RH, De Groot LJ, Glinoer D. Management of thyroid dysfunction during pregnancy and postpartum: An Endocrine Society clinical practice guideline J ClinEndocrinolMetab. 2007; 92 (8): S1-S47.

[9]. Dunn J, Delange F. Damaged reproduction: the most important consequence of iodine deficiency. J Clin EndocrinolMetab.2001; 86 : 2360-2.

[10]. Ntambwe T, Banea JB, Lebuki E, Tusuku T, Mubiala J, Mfumungani M, Kamanda A, Nahimana D. Enquête nationale sur la lutte contre les troubles dus à la carence en iode en R.D.Congo. Rapport Ministère de la Santé. Programme National de Nutrition Kinshasa. 2001, 21 pages.

[11]. Habimana L, Twite KE, Wallemacq P, De Nayer P, Daumerie C, Donnen P, Kalenga

[12]. MK, Robert A .Iodine and iron status of pregnant women in Lubumbashi, Democratic Republic of Congo. Public Health Nutr. 2013; 16(8):1362-70.

[13]. Kitwa KE, Habimana L, Simbi JB, DonnenPh, Twite KE, Kasongo ME, DeNayerPh, Kalenga MK, Robert A . Evaluation of Iodine content in table salt consumed in Democratic Republic of Congo. Food and Nutrition Bulletin.2012; 33 (3): 217-23.

[14]. Leroy B, Leffort F.A propos du poids et de la taille des nouveau-nés à la naissance. Rév. Fr. GynecoObst. 1971 ; 66 (6-7) : 391-6.

[15]. Tramalloni J, Monpeyssen H. Goitres. In: Tramalloni Jet Monpeysen H (Eds) Echographie thyroidienne. Paris, Masson. 2006: 31 - 72.

[16]. Diametra. Direct immunoenzymatic determination of TSH, T4L and T3L. 2009www.Diametra.com

[17]. Panesar NS, Li CY, Rogers MS. References intervals for thyroid hormones in pregnant Chinese women. Ann ClinBiochem. 2001; 38: 329-32.

[18]. Haddow JE, Knight GJ, Palomaki GE. The reference range and within-person variability of thyroid stimulating hormone during the first and second trimesters of pregnancy. J Med Screen.2004; 11(4):170-4.

[19]. Spencer CA, Lee R, Kazarosyan M. Thyroid references ranges in pregnancy: studies of an iodine sufficient cohort. Thyroid. 2005; 15(1): S-16.

[20]. Walker JA, Illions EH, Huddleston JF, Smallridge RC. Racial comparisons of thyroid function and autoimmunity during pregnancy and the postpartum period. Obstet Gynecol.2005; 106: 1365-71.

[21]. Stricker R, Echenard M, Eberhart R, Chevailler MC, Perez V, Quinn FA, Stricker R Evaluation of maternal thyroid function during pregnancy: the importance of using gestational age specific reference intervals. Eur J Endocrinol. 2007; 157(4): 509-514.

[22]. Mitchell AL, Pearce SH.How should we treat patients with low serum thyrotropin concentrations?Clin.Endocrinol. 2010; 72(3):292-6.

[23]. Pino S, Fang SL, BravermanLE.AmmoniumPersulfate: a safe alternative oxidizing reagent for measuring urinary iodine. Clin chem.1996; 42: 239-43.

[24]. WHO, UNICEF & ICCIDD.Assessment of iodine deficiency disorders and monitoring their elimination. A guide for programme managers.2007 ; 3rd edn.

[25]. Marc MT, Mario FT. Biostatistique pour les sciences de la vie, Pearson Education. 2009 ; pp.448.

[26]. Al-Yatama FI, Al-Bader MD, Al-Mazidi ZM, Ali A, Al-Omair A, Al-AjmiNH.Iodine status among pregnant women in Kuwait. J Endocrinol Invest.2007; 30 (11): 914-9.

[27]. GlinoerD.Thyroïde et grossesse. Med Clin Endocrinol Diab.2004 ; 65-70.

[28]. Caron P, Glinoer D, Lecomte P,OrgiazziJ, Wemeau JL. Apport iodé en France : prévention de la carence iodée au cours de la grossesse et de l'allaitement. Annales d'endocrinologie. 2006 ; 67 (4) : 281-286.

[29]. Hiéronimus S, Bec-Roche M, Ferrari P,Chevalier N, Fénichel P, Brucker-Davis F. Iodine status and thyroid function of 330 pregnant women from nice area assessed during the second part of pregnancy. Ann Endocrinol. 2009; 70(4):218-24.

[30]. Smallridge RC, Ladenson PW. Hypothyroidism in pregnancy: consequences to neonatal health, J ClinEndocrinol Metab.2001; 86(6): 2349–2353.
[31]. Haddow JE, Palomaki GE, Allan WC, Williams JR, Knight GJ, Gagnon J et al. Maternal Thyroid deficiency during pregnancy and subsequent neuropsychological development of the child. N Engl J Med.1999; 341(8): 549-555.
[32]. Alexander EK, Marqusee E, Lawrence J, Jarolim P, Fischer GA, Larsen PR .Timing and magnitude of increases in levothyroxine requirements during pregnancy in women with hypothyroidism. N Engl J Med. 2004;351(3): 241–249.
[33]. Brucker-Davis F. Carence iodée et grossesse. Médecine Thérapeutique Endocrinologie & Reproduction. 2004 ; 6 (5) : 293-9.
[34]. Roberts CG, Ladenson PW Hypothyroidism. Lancet. 2004 ; 363(9411): 793–803.
[35]. Vila L, Legaz G, Barrionuevo C, Espinel ML, Casamitjana R, Muñoz J et al. Iodine status and thyroid volume changes during pregnancy: results of a survey in Aran Valley (Catalan Pyrenees) J Endocrinol Invest. 2008; 31(10):851-5.
[36]. Glinoer D. Pregnancy and iodine. Thyroid. 2001; 11: 471-81.
[37]. Schlienger JL, Sapin R (2005) Basic of the Thyroid function. Roche Diagnostics.2005; 1:41-45.
[38]. Scientific committee of Food Health in Elsevier. 2000. 249p.
[39]. Roti E, Uberti ED Iodine excess and hyperthyroidism. Thyroid. 2001; 11: 493-500.

Table 1. Demographic characteristics of pregnant women examined in the three maternity wards in Lubumbashi, DR Congo

	Lubumbashi Zone			
	Urban	Semi-urban	Urban-rural	
	n= 75	n= 75	n= 75	P-value
Age (years)				
Mean ± SD	29 ± 7	26 ± 6	26 ± 7	<0.001
Min- max	17 - 42	16 - 41	15 - 43	
Parity				
Mean ± SD	2	2	2	0.64
Min- max	0 - 9	0 -7	0 - 7	
Married- n (%)	74 (99)	74 (99)	73 (97)	0.77
Family history of goiter - n (%)	5 (6.6)	4 (5.3)	5 (6.6)	0.92

Table 2. Biological characteristics of the thyroid gland in pregnant women examined in the three maternity wards in Lubumbashi, DR Congo

	Lubumbashi Zone			
	Urban	Semi-urban	Urban-rural	
	n= 75	n= 75	n= 75	P-value
Urinary Iodine (µg/l)				
Median	160	135	95	0.06
Interquartile	70 -326	57 - 372	35 - 280	
Geometric Mean (SD)	256.8 (3.8)	214.3 (1.28)	103 (8.7)	0.004
Abnormal- n (%)				
< 150	34 (45)	39 (52)	47 (63)	0.09
> 250	26 (35)	24 (32)	20 (27)	0.39
TSH (mUI/l)				
Mean ±SD	1,6 ± 0.9	2,2 ± 1,56	2,2 ± 1,4	0.009
Geometric Mean (SD)	3.3(1.5)	4.0 (1.9)	4.0 (1.8)	
Abnormal Function -n (%)				
- Hypothyroxinemia	29 (39)	19 (25)	28 (37)	0.16
- Subclinical Hypothyroidism	2 (3)	19 (25)	7 (9)	0.001
- Hypothyroidism	6 (8)	4 (5)	8 (11)	0.47
- Subclinical Hyperthyroidism	0	6 (8)	2 (3)	

Hypothyroidsm: TSH > 2.5 mUI/l in 1st trimester, > 3 in 2nd and 3rd trimester;
Euthyroidism: TSH between 0.3 and 3 m UI/l;
Hyperthyroidism: TSH < 0.1 mUI/l in 1st trimester, < 0.2 in 2nd trimester, and < 0.3 mUI/l in 3rd trimester.
Subclinical Hypothyroidism: Elevated TSH and normal free-T4;
Hypothyroidism: Elevated TSH and low free-T4;
Subclinical Hyperthyrodism: Low TSH and normal free-T4.

Table 3. Factors associated with ultrasound abnormalities in the thyroid gland among pregnant women examined in Lubumbashi, DR Congo

	Morphological Abnormality Detected			
	Goiter	Nodule	None	
	n = 11	n = 15	n = 199	P - value
Age (Years)				
Mean ± SD	29 ± 6	31 ± 6	26 ± 6	0.01
Parity				
Mean ± SD	3	3	2	0.39
Interquartile	2 - 7	1 - 6	2 - 9	
Family History of Goiter - n (%)	3 (27)	1 (7)	10 (5)	0.01
Urinary Iodine (µg/l)				
Median	117	215	137	0.79
Interquartile	87 - 303	33 - 469	52 - 320	
Geometric Mean (SD)				
Abnormal -n (%)				
< 150	7 (64)	7 (47)	106 (53)	0.69
> 250	3 (27)	4 (27)	63 (32)	0.61
Thyroid Stimulating Hormone TSH (mUI/l)				
Mean ±SD	1.9 ± 1.8	1.7 ± 0.9	2.0 ± 1.3	0.60
Geometric Mean (SD)	3.2 ± 2.2	3.4 ± 1.6	3.7 ± 1.7	
Abnormal Function -n (%)				
- Hypothyroxinemia	3 (27)	0	66 (33)	0.02
- Subclinical Hypothyroidism	4 (36)	0	24 (12)	0.01
- Hypothyroidism	0	0	17 (9)	0.30
- Subclinical Hyperthyrodism	0	1 (7)	7 (4)	0.68
Gestational Age - n (%)				0.90
- First Trimester	4 (36)	6 (43)	66 (33)	
- Second Trimester	4 (36)	3 (21)	64 (32)	
- Third Trimester	3 (27)	5 (21)	70 (35)	

TwiteKabangeEugène " Ultrasound morphology of the thyroid gland among pregnant women in the moderate iodine deficient region of Lubumbashi, DR Congo."IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 17, no. 7, 2018, pp 04-10.