Research Article

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Study on the Modulation of MDA and Lipid Profile Status of Some Women during Menstrual Cycle

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ABSTRACT

Study was carried out to evaluate levels of Malondialdehyde and lipid profile parameters of some women in different phases of menstrual cycle. All subjects were premenopausal within childbearing age between 18 years to 50 years who had normal 28 days cycle. Blood samples were collected at follicular, ovulatory and luteal phase and their lipid profile and Malondialdehyde levels were determined spectrophotometrically. Applying analysis of variance with P value at (P<0.05) cholesterol and low-density lipoprotein showed significant difference at follicular and luteal phases. Marked variation was observed in all three phases for malondialdehyde with a peak at ovulatory phase. Pattern of result could be used to explain the interplay of lipids and hormonal behavior in menstruation.

Keywords
Malondialdehyde, Oxidative stress, Lipid Profile, Menstrual cycle.

Introduction
Variation in levels of lipid has a significant role in menstrual cycle since it could elicit clinical implications. There have been several epidemiological studies carried out in attempt to situate the functions of lipids and the outcome during the menstrual cycle of women have been revealing [1]. While some have observed marked variations, others have not, probably due to the design of the study, variation in sample collection time, genetic factors and other environmental factors [2].

The female reproductive system is structured in a way that several organs are interconnected and act in synergy to enhance production of steroid hormones, synthesis of oocytes and the integration of sperm to egg cells. The aggregate of this result in the development of foetus [3].

The menstrual cycle is the regular natural changes that occur in the female reproductive system that makes pregnancy possible [4]. Three phases have been clearly identified in the menstrual cycle and they are the follicular phase, ovulatory phase and the luteal phase. The proper functioning of each of these phases and production of adequate quality and quantity of hormones are precursors to pregnancy.

The proxidation of lipid involves the formation and propagation of lipid radicals, the uptake of oxygen, rearrangement of the double bonds in unsaturated lipids, which result in intercalation, and destruction of membrance lipids terminating in the release of variety of products such as ketones, alkanes, ethers and aldehydes. A major lipid peroxidation product is Malondialdehyde [5]. There are a plethora of evidence pointing to variations in lipoprotein cholesterol level during the menstrual cycle [6,7].

Reactive oxygen species (ROS) especially when they are reduced can act as precursors of cell injury and result in lipid Peroxidation. One product of lipid peroxidation that can be used to determine indirectly the product of free oxygen radical is Malondialdehyde. Oxygen free radicals are present in both the endometrium of normal cycling women and infertile women and could create unpleasant surrounding within the microenvironment of embryo implantation [8].

The interplay of environmental agents and free oxygen radicals have not been fully studied and understood. However, it is known

that free radicals have the capacity to induce DNA damage and cause alteration of membrane.

Materials and Methods

The study was carried out at the Federal Medical Centre Yenagoa. 80 women that met the inclusion criteria were recruited for the research. Their consent was obtained. Fasting venous blood was collected from them and transferred into plain bottles. They were allowed to clot and were separated to obtain serum that was used for the analysis.

Cholesterol was determined spectrotophometrically by the enzymatic methods. Triglyleride was evaluated by the enzymatic colorimetric method. Low-density lipoprotein (LDL) and very low-density lipoprotein (VLDL) were precipitated quantitatively by the addition of phosphotungstic acid in the presence of magnesium ions to obtain fractions. After centrifugation, the cholesterol concentration in the High-density lipoprotein (HDL) fraction, which remained in the supernatant, was determined.

All reagents were product of Randox (Randox Laboratories Limited, UK) The low-density lipoprotein was determined mathematically using the Friedewald's formula (LDL = Total Cholesterol – $^{T}/22$)-HDL). The method of Varshney and Kale (1990) was used for the determination of malondialdehyde (MDA). MDA reacts with thiobarbituric acid (TBA) to form red or Pink-coloured complex, which absorbs maximally in solution at 532 nm.
MDA + 2TBA – MDA: TBA adduct +H_20

Results

Result obtained from the analysis of lipid profile and Malondialdehyde for the three phases of menstrual cycle are shown in Table 1 and 2 respectively.

Table 1: Showing 3 phases of menstrual cycle with respective lipid profile parameter value.

Parameters	**Phases of Menstrual cycle**			
Values	Follicular	Ovulatory	Luteal	P-values
TC (mmol/L)	4.62 ± 0.77	5.83 ± 0.86	4.38 ± 0.77	.014*
LDLC (mmol/L)	2.14 ± 1.10	3.81 ± 1.10	2.15 ± 1.21	0.001*
HDLC (mmol/L)	1.70 ± 0.79	1.38 ± 0.47	1.34 ± 0.55	.054
TG (mmol/L)	0.87 ± 0.40	0.71 ± 0.18	0.77 ± 0.24	.375

Values are mean ± SD of triplicate determination * P<0.05 (significant)

Te-Total cholesterol, LDLc-Low density lipoprotein cholesterol HDLC: High-density lipoprotein cholesterol; TG: Triglyceride.

The result shows a trend in which total cholesterol and low-density lipoprotein has higher values at the ovulatory phase with TG and HDLc higher at the follicular phase.

Table 2: Obtained values of Malondialdehyde in respective phase of the cycle.

Parameters	**Phases of Menstrual cycle**			
Values	Follicular	Ovulatory	Luteal	P-values
MDA (mmol/L)	5.32 ± 3.78	7.31 ± 4.38	4.42 ± 3.68	0.001

Values were mean SD of triplicate determinations. Comparison of the value of MDA in the three phase reveals that it was highest at the ovulatory phase.

Discussion

Free radicals are known to be molecular species that have the potential of independent existence possessing unpaired electrons in an orbital atom. Its production presents as a continuum in the normal metabolic cellular processes. There is now understanding that excess free redicals wether produced endogenously or introduced exogenously into the environment are capable of causing several diseases.

Research work aimed at providing support for oxidative stress theory has been examined to explain the pathological basis of many medical conditions. The purpose of this research is to evaluate the oxidative stress pattern using lipids and Malondialdehyde as markers in the three phases of the menstrual cycle of women in productive age. We observed higher values of low-density lipoprotein, triglyeride and total cholesterol in pre-menopausal women. In term of the phases, our results show that triglyceride and high-density lipoprotein were higher at the follicular phase. This observation correlates with the work of [9]. A further revelation from the work shows that the low-density lipoprotein level was highest at the ovulatory phase. Support for this finding can be seen in [10]. Menopause is a physiological condition, which is characterized by inhibition of the menstrual period and ovarian function because of the altered hormone levels. Arising from this, there is usually changes in the physical appearance of most women occasioned by increased adiposity and greater body mass on transition to menopause.

We observed from our data raised value of Malondialdehyde at the ovulatory phase when compared to value at the follicular and luteal phase. A plausible biochemical explanation that can be offered for this which may be due to production of high estrogen levels from developing follicles at the proliterative phase of the menstrual cycle, an assertion with the findings of [11,12].

To examine if the formation of MDA is reduced or increase during menstrual cycle, it has earlier been suggested that it is a marker of oxidant stress and has been known to be changed in diverse stages of the development of hyperlipidaemia. Menopausal status is known to be associated with some dyslipidaemia but, the administration of some hormone's replacement is in use [13,14].

Evidence has been shown in this work that oxidative stress occur in cells as outcome of normal physiological process due to interplay of environmental interaction and antioxidant defence system is in place to play protecting role.

References

1. Suliga E, Koziel D, Gesla E, et al. Factors Associated with Adiposity, Lipid profile Disorders and Metabolic Syndrome Occurrence in Premonopausal and Post menopausal women. PLOS one. 2016; 11: e015411.

2. Serviddio G, Loverro G, Vicino M, et al. Modulation of endometrial Balance during the menstrual cycle: Relation with sex hormones. J. Clin Endocrinol Metab. 2002; 87: 2843-2848.
3. Jones RE, Lopez KH. Human Reproductive Biology. Academic Press Cambridge. MA. USA. 2013.
4. Andrew C Silverthorn, Dee Unglaub Silverthorn, Bruce R Johnson, et al. Human physiology: An integrated Approach, 6th Edition. Glenview, Pearson Education. 2013; 850-890.
5. Cakir T, Goktas B, Mutlu MF, et al. Advanced oxidative protein products and malondialdehyde the new biological marker of oxidative stress are elevated in postmenopausal women. Ginekol Pol. 2016; 87: 321-325.
6. Rui-li Yang, Yong-Hui Shi, Gang Hao, et al. Increasing oxidative stress with progressive Hyperlipidaemua in human: Relation between malondialdehyde and Atherogenic index. J. Clin Biochem Nutr. 2008; 43: 154-158.
7. Sagar S, Kalio IJ, Ganguly NK, et al. Oxygen free radicals in essential hypertension. mol cell Biochem. 1992; 111: 103-108.
8. Betteridge DJ. What is oxidative stress?. Metabolism. 2000; 49: 3-8.
9. Agarwal A, Apponte-Mellado BJ, Premkuma BJ, et al. The effects of oxidative stress on female reproduction. A review. Repro Biol Endocrinol. 2012; 10: 49.
10. Strehlow K, Rotter S, Wassmanns S. modulation of antioxidant enzyme expression and function by eostrogen. circ Res. 2003; 93: 170-177.
11. Massafra C, Gioia D, De Felic Picciolini E, et al. Effects of Estrogen and Androgens on erythrocyte antioxidant Superoixe dismutase, catalase and glutathione peroxidase activities during the menstrual cycle. J, Endocrinol. 2000; 167: 447-452.
12. Kenneth Chiang, Sampath Parthasarathy, Nalini Santanam. Estrogen, Neutrophil oxidation. Life Sci. 2004; 75: 2425-2438.
13. Browne RW, Bloom MS, Sehisterman EF, et al. Analytical and Biological variation of Biomarkers of oxidative stress during the menstrual cycle. Biomarkers. 2008; 13: 160-183.
14. Doshi SB, Agarwal A. The role of oxidative stress in Menopause. J. Midlife Health. 2013; 4: 140-146.