CME LIVE OPEN ACCESS

Journal of
Gynecology and Reproductive Health

Research Article

ISSN: 2574-2728

Association Between Quantity Ovarian Hyper Stimulation and Endometrial Thickness on Women 20 To 46 Years

Emeonye Odochi. P[1*], Obiyo Franklin.C[2] and Okoroafor C[3]

[1]*Maternal and child health care nursing, department of nursing science, faculty of health sciences, college of medicine Abia State University uturu.*

[2]*Collage of medicine Department of Anatomy, Abia State University Uturu, Abia State, Nigeria.*

[3]*Department Of Nursing Sciences, Abia State University Uturu, Abia State, Nigeria.*

Corresponding Author: Emeonye odochi peace, Maternal and Child Health Care Nursing, Department of Nursing Science, Faculty of Health Sciences, College of Medicine Abia State UniversityUturu.

Received: 2024 Oct 08 **Accepted:** 2024 Oct 30 **Published:** 2024 Nov 30

Abstract

Ovarian stimulation causes the release of more eggs in ovulation every increasing the chances of sperm cells contacting eggs leading to fertilization. The method of ovarian hyperstimulation has been used extensively by (Anna Carby (2019), Cohen J (2003), (Balen A.H and Rutherford 2007), (Brown SE et al, 2020). Endometrial thickness and growth during ovarian stimulation is a possible predator of implantation in invitro fertilization (F. Casper et al., 2016).

Objective: *This study evaluated the association between the ovarian stimulation and its response to the uterine thickness. This research helped the medical practitioners (fertility experts) in the management of women with anovulatory infertility.*

Methodology: *with the use of use225IU of menopur and becinolyn injected in the ovary for 13days, it showed that 103 eggs where released in an endometrial thickness of 79MM, these shows a linear relationship.*

Summary: *The more the ovary is been stimulated the chance of conception and the readiness of endometrial wall.*

Keywords: Association, Ovarian, Hyper Stimulation, Endometrial Thickness

1. Introduction

Ovulation is [t]he release of an egg from the ovary, it must occur for pregnancy to be achieved naturally. Anovulation means a lack of ovulation or absent ovulation. When ovulation is irregular but not completely absent. It is called oligo-ovulation. Both anovulation and oligo-ovulation are types of ovulatory dysfunction. Ovulatory dysfunction is a common cause of female infertility. Up to 40% of infertile people with ovaries experience dysfunctional ovulation. Reelin and aromatase cooperate in ovarian follicle development. Controlled ovarian hyperstimulation (COH) is based, in the development of a large number of follicles in the same cycle to obtain the optional number and quality of oocytes from ovary within assisted reproductive technology (ART) which is a common practice in infertility treatment. Many COH protocols have been developed for this purpose [1]. The COH technique applied in the framework of ART is largely hope light for couples who cannot have children in normal conditions, but the long-term effects have not yet been fully clarified. Agents used to stimulate ovulation have been 2reported to have negative effects on reproductive organs. Such as ovaries uterus and cervix [2]. The uterus is a dynamic tissue under the control of ovarian steroids, estrogen and progesterone. The endometrium layer of the uterus undergoes proliferation, secretion and menstrual cycle to reach the receptive state in the implantation of the embryo. When the endometrial carries out its natural functions by stimulation of the hormones, the effects of this stimulation arises as transient secretion of endometrial proteins, changes in cell behavior and thickness. When a couple is not experiencing infertility, the chances of conception are about 25% each month however, even when ovulation happens normally, a couple is not guaranteed to conceive. When a person is anovulatory, they cannot get pregnant because there is no egg to be fertilized. If a person has irregular ovulation, they will have fewer chances to conceive because they ovulate less frequently [3]. Often cycles fall within the normal range 21 to 26 days but the length of an individual cycles varies widely from month to month, that could also be a sign of ovulatory dysfunction.

For instance, that experiences a monthly cycle of 22days but present fluctuates to 35 days cycle signifies an ovulation problem. The treatment for anovulation will depend

on what is causing it. Some cases can be treated with changes to lifestyle or diet. For instance, if low body weight or extreme exercise habit is the cause of anovulation, gaining weight or easing up on workout o routine might be enough to restart ovulation. On the other hand, certain anovulation caused by obesity, losing weight (about 10% of body weight) may restart ovulation. Anovulation is also treated by fertility drugs, the most common brand being clomid (clomifencitrate). Clomid has been observed to trigger ovulation in 80% of anovulatory women [3]. Insulin-sensitizing drugs like moetornin has been found to trigger ovulation [4]. The period is 6 months other available remedies within gives similar therapentic properties with metformin [5,6].

A combined therapy of metfornial has been found useful in anovulatory women who have only administered clomid drug. Women with PCOS, the cancer treating drug letrozole brand name femora are used to trigger ovulation in such women, with anovulation. Anovulaton and ovulatory dysfunction can have several causes. The most common cause of ovulatory dysfunction is polycyct.c ovarian syndrome [pcos]. Other potential causes of irregular or absent ovulation includes extreme exercise, extreme high stress levels, low body weight, excess porolactin, obesity and thypriod dysfunction (hyper thyrodisim) [6-15].

2. Methodology

The researcher sort for the women who were still ovulating within the ages of women bearing age from the fertility units in different clinic in Umuahia Abia State, Nigeria. The researcher applied convince sampling in selection [15-19]. The researcher excluded all the women who were below 18 years and above 65 years. The questionnaire was made of sections: A: socio demographic data, B: methods use in stimulation and C: procedure for stimulation with the use of ultrasound Machine, Ovulation Microscopy, Ovulation strip and Laparoscopy. Ethical clearance. Because the study involved human being, ethical consent was sorted from the individuals and the hospitals [19-22].

Testing the predictors				
Predictor	Coef	SEcoef	T	P
Constant	-2.517	9.052	-0.28	0.792
AGE (X1)	-0.2336	0.1953	-1.20	0.285
THICKNESS(X2)	2.1904	0.5615	3.90	0.011
S = 4.10148; R-Sq = 81.08; R-Sq (adj) = 73.4%				

Table 1: Quantity of Eggs Produced in the Study Population

3. Results

After hyperstimulation by using injections 225IU of the drug becinolyn and menopur for 13days, the age group 20-25 produced the greatest quantity of eggs (53.8%) while the age group 36-40 produced the least quantity of eggs (0.77%). This is further highlighted in fig3 which shows a bar chart of the age group of the study population (x-ck3) against the quantity of eggs produced (y- ck3) this displayed a Gaussian distribution with 70 eggs as the modal range (53.8%) while the mean quantity of eggs produced in the study population being 16 eggs. N=8 Q (egg) =130 Mean 130/8= 16.3 e quantity of follicles (eggs) produced were recorded separately for the different age groups [22].

Age Range in Years	No of Follicles (Eggs) Produced.	Percentage (%)
20-25	70	53.8
26-30	20	15.4
31-35		
36-40	1	0.77
41-46	12	9.2
Total	103	

Table 2: showed Association between Quantities of Eggs Released by the Ovary after Hyper Stimulation and the thickness of the Endometrim

The association between quantities of eggs released by the endometrium as presented in table 5 shows that the highest endometrial thickness was realized when 24 eggs were released by the ovary after hyper stimulation based on the total number of eggs produced which is 103 eggs. The least endometrial thickness was realized when 1 egg was released by the ovary after hyper stimulation based on the total number of eggs produced which is 103 eggs [23,24]. This is further highlighted thus:

- Quantity of eggs produced after ovarian hyper stimulation is103eggs
- Thickness of endometrium after ovarian hyper stimulationis79NM
- Association between quantity of eggs produced and endometrial thickness showed linear relationship.

Regression Analysis: EGGS(Y) versus AGE (X1), Thickness(X2)

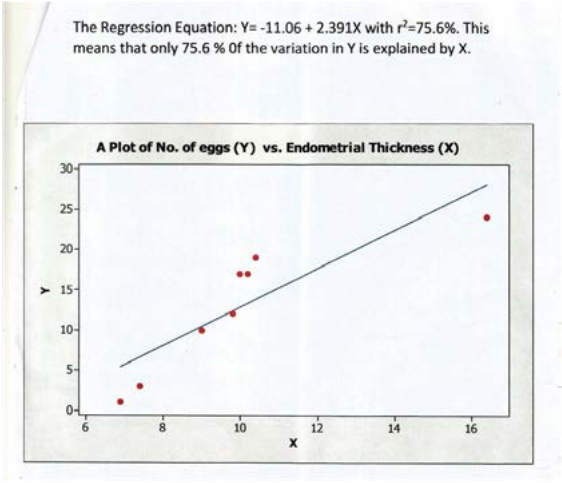
The regression equation is $Y = -2.5 - 0.234 X_1 + 2.19 X_2$

Where: Y= No. of EGGS;

X_1= AGE OF THE PATIENT

X_2= ENDOMETRIAL THICKNESS

Citation: Emeonye-Odochi, P., Obiyo-Franklin, C., Okoroafor, C. (2024). Association Between Quantity Ovarian Hyper Stimulation and Endometrial Thickness on Women 20 To 46 Years. J. Gynecol. Reprod. Health, 2(3). 1-4.



The present study has highlighted the association between quantity of eggs produced after ovarian hyper stimulation and the thickness of the endometrium based on age groupings. This presented in table 1, and showed that the frequency of eggs produced was not significantly different ($p>0.05$) in ages (36-40 yrs) but showed a significant difference ($p<0.05$) in the ages (20-25yrs) [25].

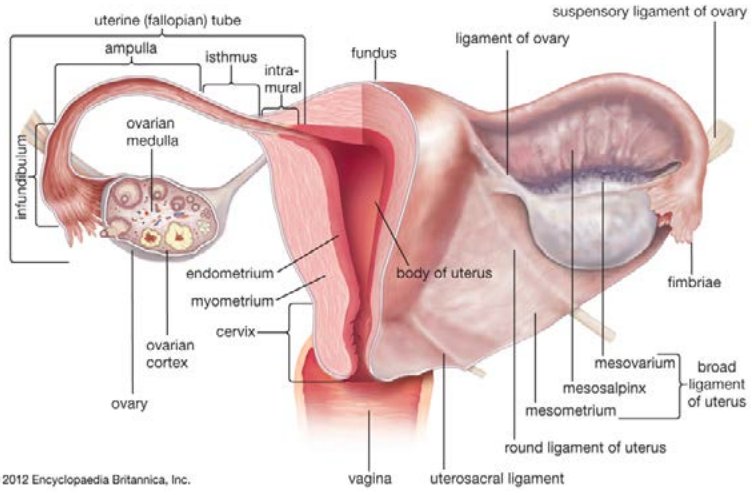
From Table 3, only the thickness is significant ($p<0.05$). On the whole and from the Analysis of variance in Table 2, the regression is significant ($p<0.05$). With the adjusted coefficient of determination (r2) = 73.48, we can also infer that 73.4%, we can also infer that 73.4% of the variation in the production of eggs is explained by the age and the endometrial thickness) account for the unexplained 26.6%.

Age Range in Years	Quantity of Eggs Realesed	Endometrial Thickness
20-25	70	45MM
26-30	20	17MM
31-35		
36-40	1	7MM
41-46	12	10MM

Table 3: showed Association between Quantities of Eggs Released by the Ovary after Hyper Stimulation and the thickness of the Endometrium

4. Conclusion

The ovaries secrete both estrogen and progesterone into the bloodstream, and thus they are important endocrine glands. They produce and release the egg(ovum) that is fertilize to form a fetus. Hyper-stimulation of ovaries with hormonal drugs result to multiple egg release, higher endometrial thickness and in-turn multiple pregnancy. Also, production of constant egg depends on the age of the individual even when the ovaries are stimulated.



ANATOMY OF THE UTERUS

The uterus is divided into the cervix, isthmus and Corpus.

References

1. Brown, S. E., Mandelin, E., Oehninger, S., Toner, J. P., Seppala, M., et al (2000). Endometrial glycodelin-A expression in the luteal phase of stimulated ovarian cycles. Fertility and sterility, 74(1), 130-133.
2. Siristatidis, C. S., Gibreel, A., Basios, G., Maheshwari, A., Bhattacharya, S. et al (2015). Gonadotrophin-releasing hormone agonist protocols for pituitary suppression in assisted reproduction. Cochrane Database of Systematic Reviews, (11).
3. Leyla Bilali, RN (Medical review August 10, 2020). Marieb, Elaine (2013). Anatomy & physiology. Benjamine-Cummings.P. 903.
4. Kettel, L. M., Roseff, S. J., Berga, S. L., Mortola, J. F., Yen, S. S. et al (1993). Hypothalamic-pituitary-ovarian response to clomiphene citrate in women with polycystic ovary syndrome. Fertility and sterility, 59(3), 532-538.
5. Budinetz, T. H., Benadiva, C. A., Griffin, D. W., Engmann, L. L., Nulsen, J. C., et al (2015). Ovulation rate and cycle characteristics in a subsequent clomiphene citrate cycle after stair-step protocol. Fertility and Sterility, 103(3), 675-679.
6. WALDSTREICHER, J., SANTORO, N. F., HALL, J. E., FILICORI, M., CROWLEY JR, W. F. et al (1988). Hyperfunction of the hypothalamic-pituitary axis in women with polycystic ovarian disease: indirect evidence for partial gonadotroph desensitization. The Journal of Clinical Endocrinology & Metabolism, 66(1), 165-172.
7. Broekmans, F. J., Knauff, E. A., te Velde, E. R., Macklon, N. S., Fauser, B. C. et al (2007). Female reproductive ageing: current knowledge and future trends. Trends in Endocrinology & Metabolism, 18(2), 58-65.
8. Brown, H. M., Russell, D. L. (2014). Blood and lymphatic vasculature in the ovary: development, function and disease. Human reproduction update, 20(1), 29-39.
9. Hussain, A., Anatomy, B. B., Thorax, W. (2020). StatPearls Publishing. Treasure Island.
10. Chandler, T., Machan, L. S., Cooperberg, P. L., Harris, A. C., Chang, S. D. et al (2009). Müllerian duct anomalies: from diagnosis to intervention. The British journal of radiology, 82(984), 1034-1042.
11. Colvin, C. W., Abdullatif, H. (2013). Anatomy of female puberty: The clinical relevance of developmental changes in the reproductive system. Clinical anatomy, 26(1), 115-129.
12. Daftary, Shirish; Chakravarti, Sudip (2011). Obsterics, 3rd Edition. Elsevier. Pp 1-16.
13. de Ziegler, D., Pirtea, P., Galliano, D., Cicinelli, E., Meldrum, D. et al (2016). Optimal uterine anatomy and physiology necessary for normal implantation and placentation. Fertility and sterility, 105(4), 844-854.
14. Larsen, R., Kronenberg, H. (2011). et ad. Williams textbook of endocrinology 12th edition.
15. Gonen, Y., Casper, R. F., Jacobson, W., Blankier, J. (1989). Endometrial thickness and growth during ovarian stimulation: a possible predictor of implantation in in vitro fertilization. Fertility and sterility, 52(3), 446-450.
16. Guioli, S., Sekido, R., Lovell-Badge, R. (2007). The origin of the Mullerian duct in chick and mouse. Developmental biology, 302(2), 389-398.
17. Kobayashi, A., Behringer, R. R. (2003). Developmental genetics of the female reproductive tract in mammals. Nature Reviews Genetics, 4(12), 969-980.
18. Robbins, J. B., Broadwell, C., Chow, L. C., Parry, J. P., Sadowski, E. A. et al (2015). Müllerian duct anomalies: embryological development, classification, and MRI assessment. Journal of Magnetic Resonance Imaging, 41(1), 1-12.
19. Roly, Z. Y., Backhouse, B., Cutting, A., Tan, T. Y., Sinclair, A. H., et al (2018). The cell biology and molecular genetics of Müllerian duct development. Wiley Interdisciplinary Reviews: Developmental Biology, 7(3), e310.
20. Ross, M. H., Pawlina, W. (2006). Histology. Lippincott Williams & Wilkins.
21. Rzepka-Górska, I., Tarnowski, B., Chudecka-Głaz, A., Górski, B., Zielińska, D., et al (2006). Premature menopause in patients with BRCA1 gene mutation. Breast cancer research and treatment, 100, 59-63.
22. Thomas, D. F. (2020). The embryology of persistent cloaca and urogenital sinus malformations. Asian journal of andrology, 22(2), 124-128.
23. Titus, S., Li, F., Stobezki, R., Akula, K., Unsal, E., et al (2013). Impairment of BRCA1-related DNA double-strand break repair leads to ovarian aging in mice and humans. Science translational medicine, 5(172), 172ra21-172ra21.
24. Troiano, R. N., McCarthy, S. M. (2004). Mullerian duct anomalies: imaging and clinical issues. Radiology, 233(1), 19-34.
25. Warne, G. L., Kanumakala, S. (2002). Molecular endocrinology of sex differentiation. In Seminars in reproductive medicine (Vol. 20, No. 03, pp. 169-180). Copyright© 2002 by Thieme Medical Publishers, Inc., 333 Seventh Avenue, New York, NY 10001, USA. Tel.: + 1 (212) 584-4662.