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Nulliparity, Delayed Child Birth and Obesity: Potential Risk Factors for Development of Uterine Fibroid in South-South Nigeria

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Abstract

Background: Causes of fibroid are still unknown. Despite series of cytogenetic investigations, the uncertainty remains as to the primary or secondary genetic changes and their impact on the initiation or promotion of fibroid development.

Purpose: To determine the effect of obesity, nulliparity and delayed child birth on the occurrence of uterine fibroid.

Materials and Methods: A prospective sonographic survey was carried out in a population of women in South-South region of Nigeria. Ultrasound scan was performed in 240 randomly selected women aged 20 – 45 years, presenting for scans at the time of study. Subjects' ages and reproductive history was obtained to ascertain their parity status and ages at which parous women had their first children.

Results: Results showed a weak positive association ($r = 0.1$; $p<0.05$) between uterine fibroid and delayed child birth and nulliparity at 31-35 years. A strong positive association ($r = 0.9$; $p<0.05$) was observed at 36 – 45 years. There was no statistically significant association between low, normal and high body mass index with uterine fibroid however, a significant association ($r = 0.85$; $p<0.05$) was observed with obesity.

Conclusion: Delayed child birth and nulliparity after 36 years of age appeared to be the most potent risk factor for uterine fibroid in the population studied.

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Introduction

Uterine fibroids or leiomyomas are benign tumors of the uterus usually of smooth muscle origin. It is a localized proliferation of smooth muscle cells into a concentric whorl. The exact initiating factors are unknown. It is believed that it may be physical or mechanical in nature

and operate within the rapidly contracting uterine smooth muscle resulting it its most often multiplicity. Uterine leiomyoma is one of the benign tumors prevalent in the South-South region of Nigeria [1]. It is believed that these tumors develop in the majority of African women and become symptomatic in one third of these women [2].

The causes of fibroid are unknown, however, a few theories of initiation have been offered in the literature. Despite the series of cytogenetic investigations, uncertainty remains as to the primary or secondary genetic changes and their impact on the initiation or promotion of fibroid development.

The role of growth promoters of fibroid seems to be attributed to ovarian hormones estrogen and progesterone. The clinical and laboratory evidence has been cited [3]. Although the causes of fibroids are unknown, several predisposing factors have been identified including genetic predisposition, early menarche, nulliparity, delayed child birth and obesity [4]. It is not very clear why leiomyomas develop in some women and not others.

Despite their prevalence, little attention has been directed toward the causes and pathogenesis of fibroids until recently because of the rarity of malignant transformation. Regardless of their general neoplastic character, uterine fibroid have been responsible for significant infertility in a large segment of the female population by causing cervical distortion with extreme displacement of the cervical canal which could interfere with semen deposition and migration [5-6]. Also myomas can interfere with semen deposition and migration [7].

The clinical effect is related to their local mass effect and pressure on adjacent organs, excessive menstrual flow, repeated abortions and infertility. Other effects are constipation, hydronephrosis, endometriosis, anemia due to menorrhagia, and dysmenorrheal due to degeneration torsion or possibly an association with adenomyosis [8].

Knowledge of the predisposing factors may provide information of the cause of these tumors and their preventive measures.

Ultrasound has been the preferred imaging modality for the diagnosis of fibroid because of its cost effectiveness, availability, freedom from ionizing radiation and ease of examination.

Uterine fibroids display a spectrum of sonographic appearance including focal uterine enlargement, distortion of the endometrial complex or uterine serosal contour depending on the location, well define margins, maybe calcified, internal degenerations, hemorrhage and necrosis may also be seen. They could also be echogenic, hypoechoic or isoechoic relative to the myometrium. Calcified myomas may produce a posterior acoustic shadowing.

This study sought to determine the risk of developing uterine fibroid with obesity, delayed child birth and nulliparity in a population of women in South-South Nigeria.

Method

Ultrasound scans were performed in 242 randomly selected women of reproductive age (20-45). Reproductive history was obtained from them to ascertain their parity status and the age at which the parous subjects had their first children.

Subjects were divided into groups five (5) based on age and age at first birth as follows. Group 1 (20-25yrs), Group 2 (26-30yrs), Group3 (31-35yrs), Group4 (36-40yrs), Group5 (41-45yrs). The number of patients with fibroid for each age range was recorded and compared with the number of patient presenting for ultrasound examination at the time of study without fibroid. The number of cases of fibroid for all the age range were also compared to determine age range at which nulliparity and first child birth as well as rate of fibroid occurrence was highest.

Another randomly selected set of 180 women who presented for ultrasound scan were used to study the relationship between body mass index and uterine fibroids.

The height and weight of subjects were measured and used to calculate body mass index (BMI = weight (Kg) /Height^2 (m). They were classified into four (4) groups depending on whether they have low (BMI < 18.5kg/m^2), normal (BMI=18.5-24.5Kg/m^2) or high body mass index/overweight (BMI=25-29.5Kg/m^2) and whether they were obese (BMI above 30Kg/m^2). Data was collated and sample percentages calculated. Correlation analysis was used for statistical analysis.

Result

Results show a weak positive correlation ($r = +1$; $p<0.05$) between uterine fibroid and nuliparity or delayed child birth at 31 – 35 years of age. 64.9% of patients aged 36 – 45 years had one or more fibroid nodules ($r=+0.9$; $p<0.05$).

No statistically significant association was observed between low, normal and high body mass index with uterine fibroid. However, a significant association ($r=+0.85$; $p<0.05$) was observed between obesity and body mass index (Figures 1 &2). The influence of nulliparty and delayed child birth on fibroid development was more than obesity.

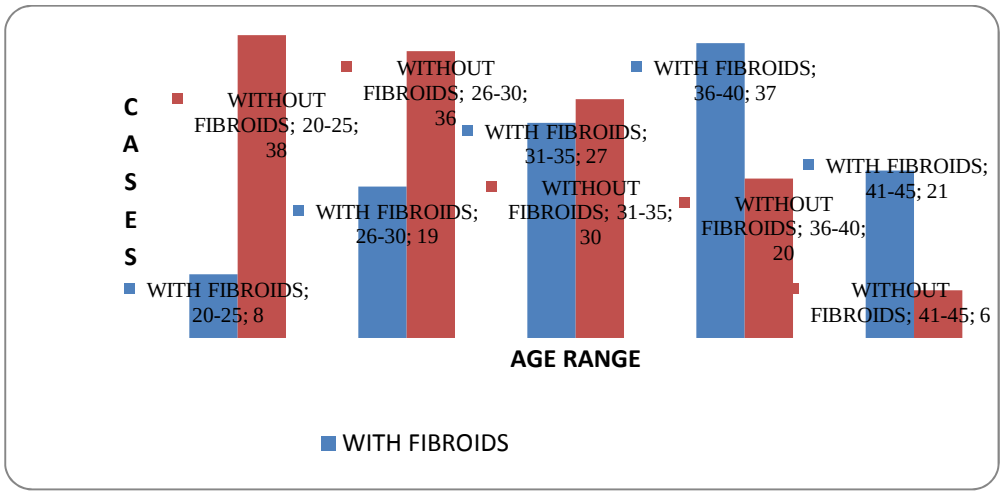

Figure 1: Distribution of cases of uterine fibroid with age at nulliparity and first birth.

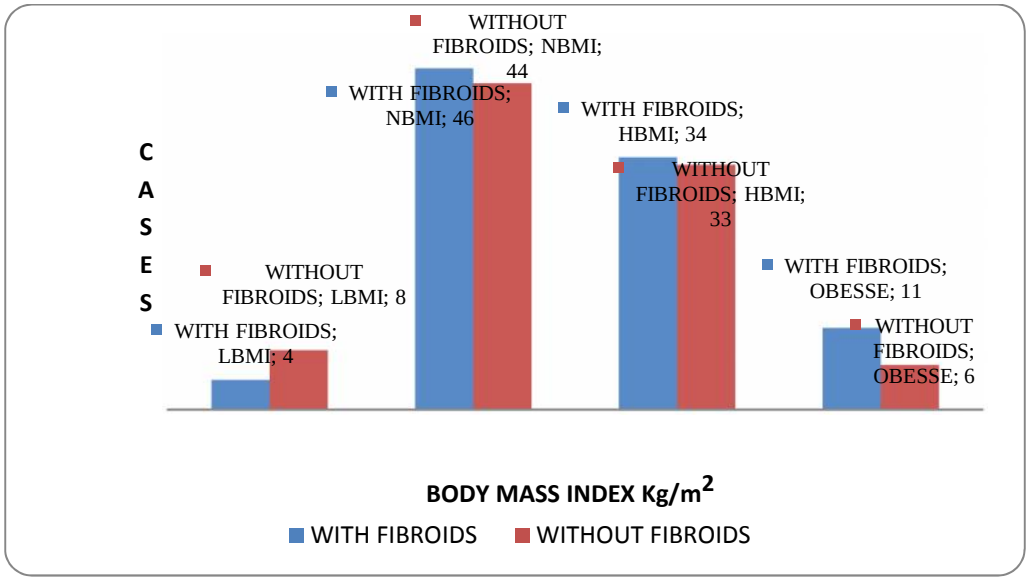

Figure 2: Distribution of cases of uterine fibroid with BMI.

Discussion

Uterine leiomyomas are a very interesting condition peculiar to not only Nigerian women but in women the world at large. Several research efforts yielded positive result concerning the common risk factors. However, from our study there was no clear relationship between nulliparity and fibroid development at

ages 20 to 30 ($r= -0.24$; $p<0.05$), weak negative correlation.

The number of ultrasound diagnosed cases of uterine fibroids slightly increased ($r= +0.1$; $p<0.05$), weak positive correlation from 31-35years.

The percentage incidence of uterine fibroid was much higher at 36-45 years ($r= +0.9$; $p<0.05$), strong positive correlation. 50% of the women under this age were nulliparous (Figure 1). This observation is consistent with earlier report by Shikora et al [9] that increase in the incidence of fibroid is due to delayed child birth. It also conforms to the observation of Marshall et al [2,10], who documented that the risk of uterine leiomyoma is reduced to half for every child delivered. This they attributed to decreased estrogen stimulation of the uterine smooth muscles by pregnancy in early parity.

There was increased incidence of ultrasound confirmed uterine fibroid in women aged 36-45. The possible explanation for this finding is that pregnancy and lactation reduce the period of exposure to unopposed estrogen whereas nulliparity and reduced fertility may be characterized by unopposed estrogen and progesterone stimulation of the uterine smooth muscle cells.

In an attempt to elucidate the effect of body mass index on development of uterine fibroid, it was observed that there was no statistically significant difference between low, normal and high body mass index with uterine fibroid in the population studied. However, a significant number of cases of uterine fibroids (64%) were recorded in obese women ($r= +0.85$; $P<0.05$). It should be noted however that the sample size is relatively small (Figure 2). This observation agrees with some published reports by Ross et al. [11] that the risk of uterine fibroid increases 21 % for 10kg weight gain and 6% increase in risk for each unit increase in BMI. Shikora et al [9] in a study in Boston Massachusetts also stated that 51% of hysterectomy/ myomectomy confirmed cases of uterine fibroids were overweight and 16% gross obese.

Overall nulliparity and delayed child birth (36-45) years of age appear to be the most potential risk factors in the population studied (Figure 1). The effect of these risk factors for uterine leiomyoma may achieve significance through their contribution to either initiation or promotion phases of the disease. Although their impact appears to be related to their effect on estrogen stimulation, other mechanisms may be involved.

The effect of age may reflect more opportunity for cells whose growth is not regulated to be produced or alternatively a prolonged period for growth under the influence of hormones during the reproductive years. If the likelihood of fibroid development and growth accelerate during late reproductive years, hormonal factors associated with pre-

menopause may be implicated as stimulants or the increase may be a cumulative effect of many years of estrogen and progesterone stimulation.

Obesity as a risk factor for uterine leiomyoma may also be related to hormonal factors associated with the female reproductive cycle. A significant increase occur in conversion of circulating adrenal androgens to estrone through aromatization by adipose tissues but other pathologic pathways may be involved [12].

Considering the increasing incidence of uterine fibroid in Nigeria [1], there is little or no doubt that the initiating conditions may be the same for all women. The presence of fibroid offers no known advantage to affected women but rather considerable morbidity in many cases.

From the foregoing, it could be said that the high incidence of uterine leiomyoma in our society today is due to late reproductive years which ultimately allow prolonged uterine smooth muscle stimulation and proliferation of its smooth muscle cells by factors which could probably be beneficial for gestation. This is in contrast to the low incidence rates of the past where women reproducing quiet at an early age accompanied by more demanding physical conditions and prolonged breast feeding [13]. In such cases, the effect of estrogen is presumed to be directed towards nurturing a gestation rather than the uterine smooth muscle cells. There were also fewer menstrual periods due to higher number of children from women thus reducing the opportunity for cells of the uterine smooth muscles to grow in to fibroids [14].

Changes in modern life style such as shift to higher fat, low fibre diet, delayed child birth due to economic constraints and the potential effect of environmental estrogen could be responsible for the increased risk of developing uterine fibroid at present.

Conclusion

On the basis of this present study, it could be said that nulliparity, delayed child birth and obesity are potential risk factors for uterine leiomyoma in South – South Nigeria. Further research efforts may provide better understanding of other risk factors such as family history and oral contraceptive use and increase the awareness of people about the disease. Knowledge gained from such study will help in providing solutions that will reduce the incidence of the disease.

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