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ORIGINAL ARTICLE

Endometriosis in the African American woman—racially, a different entity?

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Abstract Endometriosis has been identified in up to 10% of women in some reports; however, few studies have evaluated African American women. The purpose of this study was to localize the implantation sites of endometriosis in urban Detroit female patients. This study was a retrospective chart analysis of patients with laparoscopes for endometriosis at St. John Detroit Riverview Hospital in Detroit, Michigan. All women had concomitant disease involving the uterus and multiple genital structures. In total, 93% had uterine implants, 62% had ovarian implants, 51% had posterior cul-de-sac disease, and 44% had fallopian tube involvement. Forty-five percent had abdominal wall spread, 8% with large bowel implants, and 13% with small bowel involvement. Fifty percent had uterosacral implants, 2% had bladder involvement, 2% had perihepatic involvement, and 4% had omental implants. African American women appear to have a predilection for uterine implants of endometriosis, which may be due to genetic, environmental, or previously presented theories. Further study of urban African American females is necessary to investigate the departure from typical sites of endometriotic implant localization.

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Background

Endometriosis, which is the pathologic localization of endometrial glands and stroma at non-endometrial and myometrial locations, will forever be linked to Dr. Sampson and his well-known theory of retrograde menses as its etiology in 1921. Since then, the study of endometriosis has evolved immensely. However, one area where research of endometriosis still needs to improve is in the study of this disease process in the urban African American female.

In reviewing the current literature, there are only two articles that are devoted to endometriosis and the African American woman. Both of those articles were written by Dr. Donald Chatman in 1975 and 1976. His focus was to prove the chief complaint of abdominal pain in the African American female was not typically pelvic inflammatory disease, as was so often labeled in the Emergency Departments, but was indeed endometriosis [1, 2]. A recent report however showed that among women presenting for infertility care, African American women were significantly more likely to have salpingitis than non-African Americans; however, in this report, endometriosis was only identified in 2.6% of 77 African American women [3].

In Chatman's papers, he states that the implants he found were visualized in the typical locations including the uterosacral ligaments, cul-de-sac, and ovarian surfaces. We propose that although African American women have typical implantation sites, they may have a higher preponderance of

atypical sites including the uterus. This may be explained by genetic factors, environmental influences, or previous presented theories.

Materials and methods

This retrospective chart review was conducted at St. John Detroit Riverview Hospital in Detroit, MI. The study was approved by the Institutional Review Board of St. John Detroit Riverview. The target population included African American women with a known diagnosis of endometriosis who had a laparoscopy performed by one of us (GS) over the time period of 1995–2002. These included women undergoing surgery indicated because of pelvic pain, masses, and/or infertility. This represents all women whose charts were able to be located from an archival storage facility. The basis for the diagnosis of endometriosis was laparoscopic pictures, which were available for review in 56% of the charts, while in the remaining patients, the locations of the endometriotic implants were taken directly from the chart. Histologic confirmation was not required.

The study parameters included were age, parity, age of menarche, age of diagnosis of severe pain, number of emergency room visits, race, surgical history, treatment history, and pelvic adhesions. There were 44 patients identified who met these criteria.

Findings

Among the population group of African American women, the age range for the participants was 17–57, with a mean age of 38.9 years. The mean age of menarche was 11.9 years in the 31 women with a documented age for menarche. The mean age of severe pain was 31 years of age. The average gravida and para were G2.4 and P 1.9. CA-125 was obtained in ten of the patients; two were found to be elevated. As far as prior therapies, 8 had previously received Provera, 17 had received Lupron, 5 had received oral contraceptives, and 1 patient had received Danocrine. Three women had documented cases of pelvic inflammatory disease.

Regarding the surgical management history, these women had a variety of procedures. Twenty-one patients had a total of 32 laparoscopies. Eighteen had undergone hysterectomies, five had myomectomies, and three had prior adnexal surgery. Additionally, one woman underwent an endometrial ablation procedure, while another had undergone hysteroscopy.

The proportion of patients found to have uterine implants was overwhelming, occurring in 93% of subjects. The women who were found to have uterine implants also had adhesions that affected other pelvic organs. It was found that 39% of patients had posterior uterine implants only, 2% had anterior implants only, 20% had both anterior and posterior uterine implants, and 32% had uterine implants that were not designated as anterior or posterior. Thus, a total of 93% had uterine endometriotic implants. Additionally, there were 62% of patients with ovarian implants, 51% with posterior cul-de-sac disease implants, and 44% with fallopian tube involvement. Forty-five percent had abdominal wall spread, 8% with large bowel implants, and 13% with small bowel involvement. Fifty percent had uterosacral implants, 2% with bladder involvement, 2% with perihepatic involvement, and 4% with omental implants. There was one patient with a prior history of endometriosis that had no endometriotic lesions identified at the time of her laparoscopy.

Discussion

In this retrospective study, the objective goal was to examine the sites involved with endometriosis. Traditionally, the most typical site for implantation of endometriosis has been the dependent portions of the pelvis, including the uterosacral ligaments, posterior cul-de-sac, and ovaries. While these sites were frequently involved in this study population, it was overwhelmingly found that the most common site for implants was the uterus. There was a greater predilection of the implants to attach to the posterior uterus vs. the anterior uterus. This suggestion of a possible racial difference in characteristics of endometriosis is consistent with a prior report of an increased incidence in Asian women as compared to Caucasian women [4], as well as the more recent report of endometriosis in 17 African American women in whom the rate of diagnosis was 40% lower than Caucasian women [5].

Establishing this pattern of occurrence raises the question as to why this may occur. There may be a variety of reasons why, but first, it is important to review the history of implantation theories. The embryonic rest theory developed by Von Recklinghausen and Russell in 1890 proposed that a nonspecific stimulus activated cells of mullerian origin at rest to differentiate into endometrium. In 1919, Meyer postulated that endometriosis develops from metaplasia of coelomic epithelium, from which the mullerian duct is derived in embryonic development. Sampson in 1921 further attempted to explain the genesis of endometriosis by proposing the theory of retrograde menstruation. It is well accepted today that an amalgamation of these, and other theories, represents the multiple origins of endometriosis.

Although the origin of endometriosis in African American women may be the same, there are multiple possible

reasons for this discrepancy. First would be genetic-based variation, although candidate genes responsible for the difference are not identified or readily apparent. The dominant site of implantation in our population was not. One possibility could be hypothesized as an immunologic difference. For example, women with endometriosis have been documented to have increased number of peritoneal leukocytes. These leukocytes secrete growth factors and cytokines that promote proliferation of endometriosis. Natural killer cells (NKC) have also been intensely studied as a part of this immune deregulation in women with endometriosis [6]. It has been shown that NKCs are suppressed in women with endometriosis, which would affect the body's ability to adequately remove ectopic endometrium. The two main areas that may influence the African American female disease state are environment and immunology.

Alternatively, differences seen may not represent a genetic variation but rather be a function of socioeconomic or environmental issues of Detroit. In major metropolitan areas such as Detroit, African Americans predominately live in an urban setting rather than a rural environment. Living primarily in an urban setting presents its own unique social issues that reflected in the environment. Issues such as clean water, availability of adequate nutrition, and accessibility to health care are all of concern.

The impact of adequate nutrition is a significant one in Detroit. It was recently named one of the most overweight cities in the USA. To be classified as obese, the body mass index (BMI) must be greater than 30; in this study, 36% were obese.

Women with higher BMIs tend to have higher levels of estrogen, as a function of aromatization of androgens into estrogens in adipose tissue. One hypothesis is that there may be a correlation with uterine endometriotic implants and African American with obesity secondary to the estrogen receptors in the uterine tissue [7]. The higher the BMI, the more estrogen.

However, obesity does not explain all of the cases of endometriosis in this population. Another possible issue would include the exposure to environmental toxicants. One of the major environmental toxins that have been studied is 2,3,7,8-tetrachlorodibenzo-p-dioxin, otherwise known as dioxin. Dioxins are a part of the polychlorinated diaromatic hydrocarbons, which are unwanted by-product of many industrial and combustion processes. This toxin degrades slowly, builds up in the food chain, and is ingested by humans. It is known that dioxin can inhibit ovarian progesterone synthesis and can have antiestrogenic effects.

A study by Bois et al. [8] reviewed residents in a town in Italy who were exposed to a chemical plant explosion that involved dioxin. His study was conducted in response to a study by Rier et al. [9] that looked at dioxin exposure in monkeys, and found that monkeys exposed to high levels of dioxin were prone to develop endometriosis. Bois et al. [9] revealed that dioxin is stored in adipose tissue and concluded that this particular Italian community had an increased risk of developing endometriosis. Thus, in general, humans may develop endometriosis based on the level of exposure to dioxin, or perhaps other organochlorine compounds. However, the relationship of dioxin to endometriosis remains controversial, as exemplified by the recent report of Guo et al. [10], questioning the existence of significant evidence supporting such a link.

Detroit is known as the motor city secondary to its vast industry of auto production. There are also a multitude of other industries present in the Detroit area. In our study based on African American population, there is potential exposure to dioxins or other agents. Exposure may be via ingestion of contaminated water, air, and/or food supply. Since dioxin is indeed stored in fat, the hypothesis for uterine implants may be the same as above for obesity.

Environmental factors may also influence the immunological system. Dysregulation within the immune system may promote the growth of endometriosis. Halme et al. [11] showed that women with endometriosis have an increased number of macrophages, which could have altered responses to retrograde construction products, although the specific mechanism for promotion of endometriotic lesions is unclear.

Gazvani et al. [12] discussed a strong role for the system's immune cells and mediators in the pathogenesis of endometriosis. Our population may have an altered immune system secondary to the urban environment in which they were raised in and/or currently live. The resulting deregulated immune system may contribute to the higher propensity of uterine implants in this group of women [3].

We acknowledge the limitations of this study include the limited number of participants that comprised the population [13], the lack of histological confirmation to make the diagnosis of endometriosis, the lack of a comparable Detroit Metropolitan population of non-African American women, and the non-controlled factor of incomplete ability to obtain charts from an archival storage facility. Nonetheless, we believe the observation of an apparent difference in the location of endometriotic implants in African American women, as compared to previously described populations, warrants future systematic examination.

Conclusion

In the end, these African American women appear to have a higher predisposition to uterine endometriotic implants. Reasons may include environmental, lifestyle, and/or immunological factors or a combination of the various

factors. This study reveals the need to have further investigation into this important subject.

Statement of Responsibility George H. Shade, M.D. provided clinical care for these patients and contributed to the initial concept of the study, data analysis, interpretation, writing and critical review of the manuscript.

Mieke Lane, D.O. conducted the chart review and contributed to the initial concept of the study, data analysis, interpretation, writing and critical review of the manuscript.

Michael P. Diamond, M.D. contributed to the initial concept of the study, data analysis, interpretation, writing and critical review of the manuscript.

Declaration of interest The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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