Risk Factors for Self-Reported Uterine Fibroids: A Case–Control Study

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ABSTRACT

To identify risk factors for uterine fibroids, a case–control design was used to analyze data from control subjects enrolled in the Cancer and Steroid Hormone Study. Case patients were 201 women who reported a history of uterine fibroids, and control subjects were 1503 women without fibroids, individually matched by age to case patients. Reporting of fibroids was more frequent among premenopausal women, women who had frequent Papanicolaou (Pap) smears, women who used oral contraceptives and had infrequent Pap smears, and women with higher education. Reporting of fibroids was less frequent among women with a lower body mass index who were current or long-time smokers. (*Am J Public Health.* 1996;86:858–862)

Introduction

Uterine leiomyomas (fibroids) are common, but since they may not present specific symptoms, they often remain undetected.[1] Fibroids contribute to substantial health care expenditures[2] and surgical treatment.[3] In the United States, more than 650 000 women undergo hysterectomy each year at a cost of $3 billion annually[4]; fibroids account for more than 175 000 (26.9%) of these hysterectomies.[5] Although we know little about their etiology, estrogen[6,7] stimulates and progesterone[7] inhibits fibroid growth. Premenopausal status,[8,9] younger age at menarche,[9] higher education,[8,9] and obesity in one (but not another) study[9] have been associated with a higher risk of fibroids, while such risk has been inversely related to parity,[8,9] cigarette smoking,[8] and oral contraceptive use in some[8] but not all studies.[9,10] Another study reported that use of oral contraceptives and intrauterine devices was positively associated with fibroids while other studies indicated that oral contraceptives may not cause but may reflect the enhanced detection of fibroids.[11,12]

We assessed risk factors for fibroids, including some indicators of access to medical care, in a case–control study.

Methods

A case–control design was used to analyze data from women selected as noncancer, community-based comparison subjects for the Cancer and Steroid Hormone Study, a national population-based case–control study of breast, ovarian, and endometrial cancers.[13–15] Women 20 to 54 years of age were identified between December 1, 1980, to December 31, 1982, by Waksberg's[16] method of selecting random telephone numbers of households in geographically defined areas (Atlanta, Ga; Connecticut; Detroit, Mich; Iowa; New Mexico; San Francisco, Calif; Seattle, Wash; and Utah); these women were then interviewed at home by trained interviewers. The questionnaire elicited information on demographic and behavioral characteristics, reproductive and contraceptive histories, use of health care, and other health conditions. Women were asked if they had ever been told by a physician that they had fibroids and, if yes, the year of the diagnosis.

For an incident case–control study aimed at reducing the recall bias due to reporting fibroids, a case patient was defined as any woman who reported having had an initial physician diagnosis of fibroids between 1978 and 1982. Women with an oophorectomy were excluded. Control subjects were selected from among women who had not had a hysterectomy and had not been diagnosed with fibroids. Five to eight control subjects were matched to individual case patients by age (±4 months).

An index date for each case patient and her corresponding control subject was defined as the midpoint of the year in which the case of fibroids was diagnosed. The cumulative duration of oral contraceptive use (in months) was determined from the beginning of the reported oral contraceptive exposure to the index date. Parity, total months of breast-feeding, smoking status, and duration of smoking (in pack-years) were also determined to the index date. Menopausal status was determined by asking about the time since the last menstrual period and the presence of menopausal symptoms. Women were classified as perimenopausal if their reported last menstrual period was within the 6 months prior to the index date but they were experiencing menopausal symptoms. Menopause was defined as the spontaneous cessation of the menstrual period occurring at least 6 months before the index date, and surgical menopause was defined as the cessation of menstrual

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periods resulting from an operation performed before the index date.[17] Other potential risk factors included race, education, adult body mass index (defined as weight [in grams] divided by the square of height [in centimeters]), age at menarche, and frequency of Papanicolaou (Pap) smears. Information on the frequency of Pap smears was obtained at the interview by asking women, "Before 1 year ago or before hysterectomy, how often did you have a Pap smear—that is, a test for cervical cancer?"

Oral contraceptive use was dichotomized into less than 3 months and 3 months or longer, and smoking was dichotomized into never smoked and ever smoked. Additionally, smoking status was categorized into never smokers, ex-smokers, and current smokers; smoking levels, into 0, 0 to 7, 8 to 24, and 25 or more pack-years; and years of education, into less than 12, 12, and 13 or more years. These factors were entered in the logistic regression model as indicator variables. Age at menarche was categorized into 8 to 11, 12 to 14, and more than 14 years, and it showed a consistent trend in the preliminary analysis. To reduce the number of terms in the logistic regression models, these categories were scored as integers (0, 1, and 2) and entered into models as a single scored variable. Parity was dichotomized into 0 and 1 or more, and breast-feeding was dichotomized into nonbreast-fed and breast-fed (ever breast-fed). Women were classified as premenopausal and postmenopausal, which included a few perimenopausal women. Pap smear frequency was classified as fewer than 1 Pap smear every 5 years and 1 or more Pap smears every 5 years. Body mass index was dichotomized at the median value of all women in the analysis. Body mass index categories were also analyzed by terciles and quartiles, but this produced no important difference in the odds ratios (ORs) compared with the odds ratios obtained using the dichotomous variable. Race was classified into African American and others, including White and other races.

Conditional logistic regression was used to derive both odds ratios as estimates of relative risk and corresponding 95% confidence intervals (CIs).[18,19] Since preliminary assessment did not show any confounding between geographic areas and race, Pap smears, menopause, smoking, body mass index, or breast-feeding, geographic areas were not included for further analysis. Collinearity was found between breast-feeding and parity, so

TABLE 1—The Relationship between Selected Demographic and Behavioral Characteristics and Uterine Fibroids, Estimated from a Conditional Logistic Regression Noninteractive Model

Characteristics	Case Subjects	Control Subjects	Odds Ratio[a]	95% Confidence Interval
Menopausal status				
Postmenopausal	21	250	1.0	Referent
Premenopausal	172	1191	3.5	1.7, 7.2
Pap smears				
<1 per 5 y	11	151	1.0	Referent
≥1 per 5 y	188	1350	1.9	0.9, 1.4
Age at menarche, y				
≤11	17	76	1.0	Referent
12–14	162	1238	0.9	0.5, 1.4
>14	22	187	0.8	0.3, 1.9
$\chi^2_{\text{linear trend}} = 0.36$ ($P = .567$)				
Education, y				
<12	20	213	1.0	Referent
12	172	542	1.1	0.9, 1.5
≥13	109	748	1.4	0.8, 2.3
$\chi^2_{\text{linear trend}} = .97$ ($P = .320$)				
Breast-feeding				
None	88	665	1.0	Referent
Yes	87	667	0.9	0.6, 1.2
Race				
Other races	185	1358	1.0	Referent
African American	16	145	0.6	0.3, 1.2
Body mass index				
Lean	94	756	1.0	Referent
Heavy	106	742	1.0	0.7, 1.5
Cigarette smoking				
Never smoked	97	675	1.0	Referent
Ever smoked	104	815	0.8	0.5, 1.1
Oral contraceptive use				
<3 months	74	582	1.0	Referent
≥3 months	108	804	1.0	0.7, 1.6

[a]Adjusted for menopausal status, frequency of Pap smears, age at menarche, education, breast-feeding, race, body mass index, smoking, and oral contraceptive use. Excluded unknown values include 8 case patients and 62 control subjects for menopausal status, 2 case patients and 2 control subjects for Pap smears, 2 control subjects for age at menarche, 26 case patients and 170 control subjects for breast-feeding, 1 case patient and 5 control subjects for body mass index, 13 control subjects for smoking, and 19 case patients and 117 control subjects for oral contraceptive use.

TABLE 2—The Relationship between Oral Contraceptive Use and Uterine Fibroids, by Frequency of Pap Smears, Estimated from a Conditional Logistic Regression Model with Interactive Terms

Oral Contraceptive Use	<1 Pap Smear per 5 Years			≥1 Pap Smear per 5 Years		
	Case Subject	Control Subject	OR[a] (95% CI)	Case Subject	Control Subject	OR[a] (95% CI)
<3 months	5	103	1.0 (referent)	68	478	4.6 (1.3, 15.4)
≥3 months	6	38	5.0 (1.1, 24.0)	102	766	4.3 (1.3, 13.8)

Note. OR = odds ratio, CI = confidence interval. Interaction for Pap smear frequency and levels of oral contraceptive use, $P = .0370$.

[a]Adjusted by conditional logistic regression for menopausal status, smoking (pack-years), body mass index, duration of breast-feeding, and age at index date. Excluded values include 1 case patient and 1 control subject whose Pap smear frequency was unknown, and 19 case patients and 117 control subjects whose history of contraceptive was unknown.

TABLE 3—The Relationship between Smoking Status and Uterine Fibroids, by Body Mass Index, Estimated from a Conditional Logistic Regression Model with Interaction

Smoking Status	Low Body Mass Index			High Body Mass Index		
	Case Subject	Control Subject	OR[a] (95% CI)	Case Subject	Control Subject	OR[a] (95% CI)
Never smokers	47	313	1.0 (referent)	50	350	1.0 (referent)
Ex-smokers	19	113	0.9 (0.5, 1.9)	15	122	0.6 (0.3, 1.4)
Current smokers	28	329	0.3 (0.2, 0.5)	41	267	1.3 (0.8, 2.1)
χ^2_{trend}	11.25 (P = .0008)			2.37 (P = .124)		

Note. Low body mass index = median or less (≤2.22), high body mass index = greater than median (>2.22). OR = odds ratio, CI = confidence interval. Interaction for BMI and smoking (P = .0009).

[a]Adjusted by conditional logistic regression for Pap smear frequency, oral contraceptive use, duration of breast-feeding, menopausal status, race, and age at index date. Excluded values include 9 control subjects whose smoking histories were unknown, and 1 case patient and 3 control subjects whose body mass index values were unknown.

TABLE 4—The Relationship between Smoking Levels and Uterine Fibroids, by Body Mass Index, Estimated from a Conditional Logistic Regression Model with Interaction

Smoking Level (in Pack-Years)	Low Body Mass Index			High Body Mass Index		
	Case Subject	Control Subject	OR[a] (95% CI)	Case Subject	Control Subject	OR[a] (95% CI)
0	47	317	1.0 (referent)	50	356	1.0 (referent)
0–7[b]	18	122	0.7 (0.4, 1.5)	16	130	0.6 (0.3, 1.4)
8–24[c]	17	191	0.5 (0.2, 0.9)	18	133	1.0 (0.5, 2.1)
25–182[d]	12	121	0.4 (0.1, 0.9)	22	115	1.8 (0.9, 2.0)
χ^2_{trend}	8.106 (P = .0044)			2.356 (P = .1253)		

Note. Low body mass index = median or less (≤2.22), high body mass index = greater than median (>2.22). OR = odds ratio, CI = confidence interval. Interaction for body mass index and smoking (P = .0104).

[a]Adjusted by conditional logistic regression for Pap smear frequency, oral contraceptive use, duration of breast-feeding, menopausal status, race, and age at index date. Excluded values include 13 control subjects whose smoking histories were unknown, and 1 case patient and 5 control subjects whose body mass index values were unknown.

[b]Mean ± 1 SD smoking (in pack-years) = 3.1 ± 2.3.

[c]Mean ± 1 SD smoking (in pack-years) = 16.3 ± 4.9.

[d]Mean ± 1 SD smoking (in pack-years) = 38.5 ± 15.1.

each of these variables was put in the model separately. Multiplicative interaction was assessed by examining separate two-factor cross-product terms for oral contraceptive use and smoking, each with the covariables. Significant interaction terms were included in the model. Likelihood ratio tests were used to assess the significance of variables, including interaction, in the regression models.[20] Statistical testing for trends in smoking, education, and age at menarche was based upon the significance of the regression coefficients.[21]

Results

We identified 201 women with self-reported fibroids and 1503 control subjects. Mean age (years) ± 1 SD was 43.2 ± 5.8 for case patients and 42.8 ± 15.1 for control subjects. Geographic distribution of case patients and control subjects, respectively, was as follows: 26 and 218 from the Atlanta geographical area, 41 and 380 from Connecticut, 58 and 353 from Detroit, 21 and 233 from Iowa, 6 and 52 from New Mexico, 29 and 192 from San Francisco, 14 and 45 from Seattle, and 6 and 30 from Utah. We found a mean difference of 1.1 years for case patients and 1.2 years for control subjects between the Cancer and Steroid Hormone Study interview date and the index date.

Premenopausal women and women with frequent Pap smears were more likely to report fibroids than were postmenopausal women and women with less frequent Pap smears (Table 1). Odds ratios for reporting fibroids increased from 1.0 to 1.1 and 1.4 for less than 12, 12, and 13 or more years of education, respectively. Odds ratios for reporting fibroids slightly decreased from 1.0 to 0.9 and 0.8 for up to 11, 12 to 14, and more than 14 years of age at menarche, respectively. Smoking and breast-feeding were negatively associated with reported fibroids, but not significantly. Although the finding was based upon a small number of cases, African-American women tended to report fibroids less often than other women. Parity was negatively related to the reporting of fibroids (OR = 0.8, 95% CI = 0.5, 1.4); oral contraceptive use and body mass index were not associated with it at all.

Reporting of fibroids by oral contraceptive use differed by frequency of Pap smears (P = .0370) (Table 2). Among women with less frequent Pap smears, the reporting of fibroids increased with oral contraceptive use (OR = 5.0, 95% CI = 1.1, 24.0), whereas among women with frequent Pap smears, reporting of fibroids was more than four times greater for both oral contraceptive users (OR = 4.3, 95% CI = 1.3, 13.8) and nonusers (OR = 4.6, 95% CI = 1.3, 15.4) than it was for women with less frequent Pap smears and no use of oral contraceptives.

Reporting of fibroids by smoking status differed by body mass index (P = .0009) (Table 3). Among lighter women, the reporting of fibroids decreased in current smokers (OR = 0.3, 95% CI = 0.2, 0.5). Among heavier women, smoking was not associated with self-reported fibroids. We also found effect modification between pack-years of smoking and body mass index (P = .0104) (Table 4). Among lighter women, reporting of fibroids decreased with increasing level of smoking (P = .0044). Among heavier women, the reporting of fibroids increased slightly, but this was not statistically significant.

Discussion

Our findings are consistent with earlier reports that fibroids are more frequent in premenopausal women,[8,9,22] in more educated women,[8,9] in women with early age at menarche, and in nulliparous women.[9] Our finding that oral contraceptive use is not associated with reported fibroids is also in line with an earlier report[9]; however, among women who reported less frequent Pap smears, the

positive association between reporting fibroids and using oral contraceptives might have been due to a detection bias, given that long-term oral contraceptive users probably have more frequent medical visits, which could lead to the detection of fibroids. This finding is in line with some reports[11,12] that indicate that oral contraceptive use may enhance the diagnosis of fibroids owing to detection bias. Conversely, women with infrequent Pap smears who have not used oral contraceptives probably have had less opportunity for fibroid detection. Increased reporting of fibroids with frequent Pap smears among both users and nonusers of oral contraceptives would suggest that gynecological surveillance owing to frequent Pap smears may enhance the detection of fibroids.

Smoking has been reported to reduce the risk of fibroids,[8] to cause earlier menopause,[22] and to decrease the risk of endometrial cancer.[23–26] These effects may occur through a smoking-associated lowering of endogenous estrogens.[27] This mechanism may underlie the less frequent reporting of fibroids among menopausal women, among lighter women who smoke, and among lighter women with the highest level of pack-year smoking. On the other hand, the elevated production of estrogen in the adipose tissue[28] of heavier women may outweigh the estrogen-reducing effect of smoking. The higher frequency of fibroids in women with high body mass index and the highest level of pack-year smoking could also be the result of detection bias: these women are more prone to suffer from complications of overweight and heavy smoking and thus might have had more medical visits leading to the detection of fibroids.

Although the race–fibroids association was adjusted for Pap smears and other possible markers of access to medical care, the reason for lower reporting of fibroids among African-American women remains unknown. This finding differs from that of previous studies,[2,8] which show a higher frequency of fibroids among African-American women; we do not understand the reason for this discrepancy, but less reporting of fibroids by African-American women could be owing to a lower use of hospitals by this group, as was found for hypertension and diabetes.[29] This finding needs further study.

This study had several noteworthy features. First, it involved a population-based sampling scheme in many different geographic areas. Second, it involved a relatively large sample size and included racially and economically diverse populations. Third, the subjects and interviewers were blinded to any possible hypothesis related to uterine fibroids.

At the same time, this analysis has several limitations. The Cancer and Steroid Hormone Study was not designed to examine fibroids. The case definition was based solely on self-reported information; thus, a positive response to the question of reporting an initial physician diagnosis of fibroids is probably not a sensitive indicator of the true presence of fibroids. Fibroids are often asymptomatic and access to medical care is variable, so some women in the case and control groups may have been incorrectly classified, which could have biased our results. Although it was not feasible to document the reported fibroids for each woman through medical records, a separate validation study of self-reported infertility, number of ovaries, and dates of benign breast biopsy or ovarian surgery showed reasonable agreement for these self-reported gynecologic data,[15] providing indirect evidence that the reporting of fibroid diagnoses by the women in this study is sufficiently accurate to be used in this analysis.

Difficulties in establishing the onset of fibroids imply that some exposures (oral contraceptive use and smoking) could have occurred after the fibroids developed but before they were recognized clinically. Moreover, since the difference between the interview date and the index date was 1.1 years for case patients and 1.2 years for control subjects, we used Pap smear information obtained at the interview date. These difficulties may have led to a misclassification of exposure, which, if it occurred equally among case patients and control subjects, would tend to bias our results toward the null.

Our findings were that markers of access to medical care—frequent Pap smears, oral contraceptive use, and higher education—contribute to the detection of fibroids. Early detection of fibroids may alter the trends in management of this condition (leading to more myomectomies and less hysterectomies), which may reduce the health care expenditures associated with fibroids. However, further epidemiological studies of fibroids are necessary, and failure to account for such correlates of diagnostic evaluation could result in misleading inferences. □

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Call for Papers for the World Association for Psychosocial Rehabilitation Conference

The US branch of the World Association for Psychosocial Rehabilitation (WAPR), in collaboration with the American Public Health Association, has issued a call for abstracts for its upcoming sixth annual conference on psychosocial rehabilitation, "Tertiary Prevention: Psychosocial Rehabilitation Reduces Relapse." The meeting will be held in New York City, November 16 and 17, 1996.

WAPR invites researchers and health and mental health practitioners to submit proposals for paper presentations, panels, workshops, and institutes. Abstracts of at least 250 words may be submitted on any of the following topics: social skills training; cognitive/behavioral therapies; patient and family education; organization of psychosocial rehabilitation, including managed care models; psychosocial rehabilitation and psychopharmacology; strategies for work with specific ethnic and culturally diverse populations; organizing and advocacy for the mentally ill; and other topics related to the improvement of the quality of life of persons with mental illness. Poster presentations and exhibits will also be accepted, for which there is no charge unless sponsored by a profit-making entity.

Abstracts should be sent to the Annual Conference Planning Committee, c/o Zebulon Taintor, MD, President, WAPR–US, 19 E 93rd St, New York, NY 10128. The deadline for receipt of abstracts is *July 25, 1996.* For more information, please call Celeste Wallin, Executive Director, at (212) 996-7149.