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# Location, color, size, depth, and volume may predict endometriosis in lesions resected at surgery

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**Objective:** To correlate the diagnosis of endometriosis in lesions excised at laparoscopy with pathologic diagnosis.

**Design:** Prospective study.

**Setting:** U.S. government research hospital.

**Patient(s):** Women with chronic pelvic pain thought to be due to endometriosis.

**Intervention(s):** Excision of lesions suspicious for endometriosis.

**Main Outcome Measure(s):** Histologic examination of lesions for color, width, depth, and location of endometriosis. Lesion colors were grouped as black, red, white, mixed color, or endometriomas.

**Result(s):** Sixty-five women with a surgical diagnosis of endometriosis had minimal (n = 22), mild (n = 25), moderate (n = 9), or severe disease (n = 9) according to the revised American Fertility Society classification. Endometriosis was confirmed in all but seven patients with minimal and one with severe disease. Twelve other patients did not have endometriosis. Of 314 lesions excised, 189 (61%) were endometriotic. Black or red lesions were less often histologically confirmed to be endometriosis than were white lesions, mixed-color lesions or endometriomas. Lesions > 5 mm wide or deep were more likely to be endometriosis than were narrower or shallower implants. Endometriomas deeper than 1 cm were histologically confirmed to be endometriosis, and 50% of peritoneal windows contained endometriosis.

**Conclusion(s):** White lesions, mixed-color lesions, endometriomas, and larger lesions by depth or width were more likely to be histologically confirmed endometriosis than were smaller, black, or red lesions. (Fertil Steril® 2002;78:743–9. ©2002 by American Society for Reproductive Medicine.)

**Key Words:** Endometriosis, color, surgery, histology

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Most gynecologists easily recognize powder burn or black lesions as endometriosis, yet nonpigmented or "subtle" implants that are vesicular, red, white, or peritoneal defects also may be endometriosis (1–3).

The positive predictive value of visualizing endometriosis has been reported to be as low as 45% (4). Murphy et al. reported endometriosis in samples without visible lesions taken from the cul-de-sac (5), and other investigators who have sampled normal-appearing peritoneum in the cul-de-sac or other sites common to endometriosis have detected histologically proven endometriosis in 2% to 66% of cases, depending on the technique used to identify lesions (6–8). Improvements in laparoscopy, such as high magnification and better optics, and teaching clinicians to identify more subtle endometriosis implants have led to higher detection rates (1).

While it may be possible to diagnose endometriosis by inspection, it is important to confirm the diagnosis histologically if one believes that surgery is necessary to make a diagnosis or treat endometriosis. This is especially true because many women with severe chronic pelvic pain thought to be due to endometriosis have only minimal or mild endometriosis and very few lesions (9, 10) and because many women with endometriosis have no symptoms (11, 12). Moreover, the location and stage of endometriosis lesions does not necessarily correlate with the severity of pain from endometriosis (13).

It is also important to exclude other pelvic diseases, such as endosalpingiosis (14–16), cancer, or pelvic infection, the lesions of which may look similar to endometriosis but may require different treatments. The variable clinical appearance of endometriosis, yet its similarity to other diseases, makes histologic verification rather than visualization necessary to confirm the diagnosis.

We sought to better understand the clinical characteristics of histologically proven endometriosis lesions. Our goals were to develop criteria that would predict histologic confirmation of endometriosis and to determine the accuracy of visualization of lesions for making a diagnosis, since many gynecologists do not routinely perform biopsy before undertaking surgical ablation or destruction of apparent lesions.

## MATERIALS AND METHODS

We studied women with chronic pelvic pain undergoing surgery as part of a clinical trial of a potential new treatment for endometriosis. All women had had pelvic pain for at least 6 months and were otherwise healthy, with regular menstrual cycles (25–37 days). None had been treated for endometriosis with surgical or hormonal therapy in the 6 months before study entry. In addition, no participant had taken birth control pills for at least 3 months before entry. The institutional review boards of the National Institute of Child Health and Human Development and Georgetown University Medical Center approved this clinical study.

All women entered into the study underwent laparoscopy at Georgetown University Hospital or the Clinical Center at the National Institutes of Health. Surgery was performed by one or both of the authors who were gynecologic surgeons (P.S. or C.W.). At laparoscopy, the goal was to remove all visible implants that might be endometriosis. All lesions suspicious for endometriosis were excised by using a contact neodymium:yttrium-aluminum-garnet laser (Surgical Laser Technologies, The Oaks, PA) after careful, systematic inspection of the peritoneal surfaces throughout the pelvis and the abdomen.

Pigmented (black, blue, or brown) and nonpigmented (white, clear, or red) lesions were excised. Peritoneal defects were also excised in toto regardless of whether they contained visible lesions. Endometriomas were removed by stripping the cyst wall from the ovary or peritoneal structures. If the appendix was believed to be chronically inflamed (on the basis of periappendiceal adhesions) or appeared to be involved with endometriosis, it was removed using an endoscopic gastrointestinal anastomosis device.

Charred areas observed occasionally in women with a history of previous surgery were not excised because they were presumed to be residual carbonization secondary to previous ablative therapies. Similarly, areas in which the peritoneum appeared thin but not recessed were presumed to be areas of reperitonealization after previous surgery and were not excised. Adhesions were lysed, but not necessarily resected and sent to pathology. Adhesions sent to pathology were not considered to be endometriotic lesions. Implants deep in the rectovaginal septum that obliterated the cul-de-sac or appeared to be transmural to the bowel wall were not resected ($n = 4$).

At the time of surgical excision, the location, width, depth, and color of each lesion were recorded. Location was categorized as the uterosacral ligaments, ovarian fossa, bladder peritoneum, ovaries, broad ligaments, round ligaments, side walls, cul-de-sac, bowel, or appendix. Where appropriate, the side of the pelvis involved was noted. Width was measured as the diameter across an implant in two dimensions and averaged. If more than one implant was noted within 0.5 cm of another one, the distance across all of the lesions was measured. Depth was determined on the basis of a single measurement. Volume was calculated using the formula of surface area multiplied by depth. If an implant was more than 5 mm across and 5 mm deep, it was divided, and part was sent to pathology and part was reserved for additional research studies. If a portion was set aside for research, the specimen was always examined under magnification as it was divided so that a sample of the identified implant was sent for pathologic examination.

All implants were classified as endometriomas or peritoneal lesions and were considered separately. Peritoneal lesions were grouped by color as red, black, or white. Red lesions included those that were clear, pink, or red. Black lesions included those that were black, blue, or brown. White lesions were considered to be scars. Implants described only as yellow were included with white lesions. Yellow lesions seen with any other color were grouped with the other color: red or black. Lesions with colors from more than one category were considered to be mixed color. The color and size of endometriosis found in peritoneal defects or windows was recorded.

A pathologist reviewed hematoxylin and eosin stained slides from formalin-fixed, paraffin-embedded specimens for evidence of endometriosis. If no endometriosis was seen on initial review, three slides taken from different levels of the paraffin block were reviewed. Pathologic descriptions consistent with endometrial glands and stroma were considered endometriosis. Descriptions of hemosiderin-laden macrophages alone were considered to be suspicious for endometriosis but not included in the analysis. Endosalpingiosis was defined as ciliated epithelium without endometrial stroma (16). The surgical diagnosis of endometriosis was made for a patient if lesions suspicious for endometriosis were observed and excised. The histologic diagnosis of endometriosis was made for a patient if at least one excised lesion was histologically positive.

The extent of endometriosis noted at surgery was described by using the revised American Fertility Society classification system (17). American Fertility Society scores

were determined from lesion width and depth (determined during excision), presence and size of ovarian endometriomas, presence and nature of tuboovarian adhesions, and status of the posterior cul-de-sac.

For analysis, the presence or absence of endometriosis was considered according to different lesion characteristics. Continuous variables, such as lesion dimension, were compared by using *t*-tests and were grouped into categories for comparison by $\chi^2$ test or $\chi^2$ for trend. Nominal variables, such as color or location, were compared by using the $\chi^2$ test. These characteristics were also used in a logistic regression model to analyze the prediction of histologically confirmed endometriosis. $P<.05$ was considered significant.

## RESULTS

Women underwent up to two surgeries during their participation in the study: The first procedure was to diagnose and confirm endometriosis, and the second was to look for recurrent or persistent endometriosis. Seventy-seven women had an initial surgery and 14 had repeated surgery. The mean age of the women was 32 years (range, 19–45 years). Seventy-five percent were white, 17% were black, 5% were Hispanic, and 3% were Asian. Sixty-four percent were nulligravid.

At the first surgery, 12 women were not believed to have endometriosis on visual inspection but had pelvic infection (n = 4), pelvic adhesions (n = 4), inguinal hernia (n = 2), tubal dilation (n = 1), or no pelvic findings but had Crohn's disease (n =1). Most women with endometriosis had minimal (n = 22) to mild (n = 25) disease, and fewer had moderate (n = 9) or severe endometriosis (n = 9). Those found to have stage III or IV were more likely than those with no endometriosis, stage I or stage II endometriosis to have had a history of endometriosis ($P=.01$ by $\chi^2$ for trend) (Fig. 1).

At the second surgery, the visual extent of endometriosis among women with previous histologic diagnosis of endometriosis was similar to that seen among the patients at the first surgery. Most women had minimal (n = 4) or mild (n = 5) disease, and only one each had moderate or severe endometriosis. Three women had no visible evidence of endometriosis at the time of the second look.

For first and second surgeries combined, the mean number of areas excised as suspicious for endometriosis was 2.7 for those with minimal disease, 4.7 for those with mild disease, 5.8 for those with moderate disease, and 4.7 for those with severe disease. Endometriomas were present in 78% of patients with severe disease, 50% of those with moderate disease, and 7% of those with mild disease, defining the stage of endometriosis in 64% of moderate or severe cases.

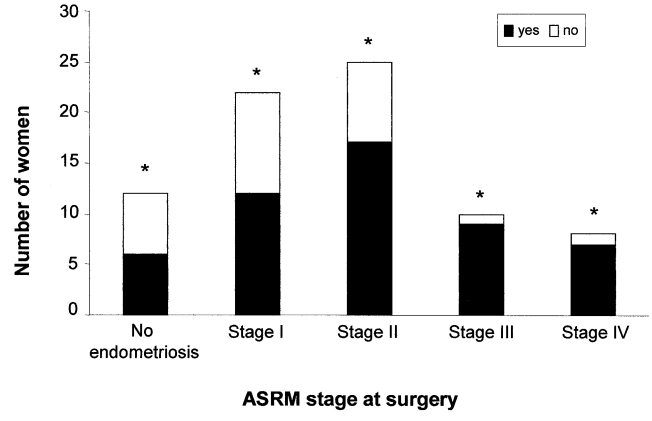
Of 314 lesions excised, 189 (61%) were histologically confirmed endometriosis. Of the lesions suspicious for endometriosis that were not histologically confirmed, 73% were designated fibroconnective tissue; 12% were ovary tissue; 6% were suspicious but not diagnostic of endometriosis; 7% were chronic inflammation, chronic hemorrhage or increased vascularity; 2% were endosalpingiosis; and 1% were fallopian tube tissue. No malignant lesions were observed. One patient had endometriosis in four of six lesions also diagnosed histologically as endosalpingiosis. Incidental findings included adhesions (n = 6), chronic inflammation of the appendix (n = 2), struma ovarii of the ovary (n = 1), peritubal cysts (n = 3), and corpus luteum or ovarian cyst (n = 2).

Of all implants and endometriomas, 63% were in the cul-de-sac, left and right ovarian fossa, or on the uterosacral ligaments (Table 1). Twelve percent of implants were on the bladder, and 11% were on the ovary. Half of ovarian lesions were endometriomas and half were superficial lesions. Clinically diagnosed endometriomas were more likely than superficial lesions to be confirmed as endometriosis (88% vs. 28%; $P=.0005$). Very few endometriotic lesions were noted on the colon (n = 7) or appendix (n = 3). The pelvic location of lesions did not influence the detection of endometriosis.

Figure 2 shows the total number of lesions and proportion that were histologically confirmed as endometriosis by color. Most lesions were black or red, but these colors correctly predicted endometriosis only 47% and 55% of the time, respectively. In contrast, white lesions were endometriosis 66% of the time. Endometriomas and lesions that were mixed color were most likely to be histologically confirmed, with endometriosis diagnosed 84% and 75% of the time, respectively. Thus, white lesions, those that were a mixture

**FIGURE 1**

Previous surgical diagnosis of endometriosis. *$P=.01$ by $\chi^2$ test for trend for likelihood of previous surgical diagnosis, by severity of disease.




*Stratton. Surgical vs. histological endometriosis. Fertil Steril 2002.*

## TABLE 1

Pelvic location of endometriosis.

| Location | No. (%) all lesions | Lesions with histologically confirmed endometriosis (%) |
|---|---|---|
| Cul-de-sac | 80 (26) | 64 |
| Ovarian fossa | 70 (22) | 67 |
| Uterosacral ligaments | 45 (14) | 62 |
| Bladder peritoneum | 38 (12) | 45 |
| Ovary | 34 (11) | 56 |
| Side wall | 17 (6) | 58 |
| Colon or appendix | 10 (3) | 70 |
| Uterus, fallopian tube | 10 (3) | 70 |
| Round or broad ligament | 5 (2) | 20 |
| Overall | 309 (100) | 60 |

*Stratton. Surgical* vs. *histological endometriosis. Fertil Steril 2002.*

of colors, or endometriomas predicted endometriosis more commonly than did lesions that were black or red ($P=.001$, $\chi^2$ test). If all implants were considered as black, red, or white regardless of other colors seen in the lesion, the rate of histologic diagnosis of endometriosis improved to 57% for black, 61% for red, and 72% for white lesions.

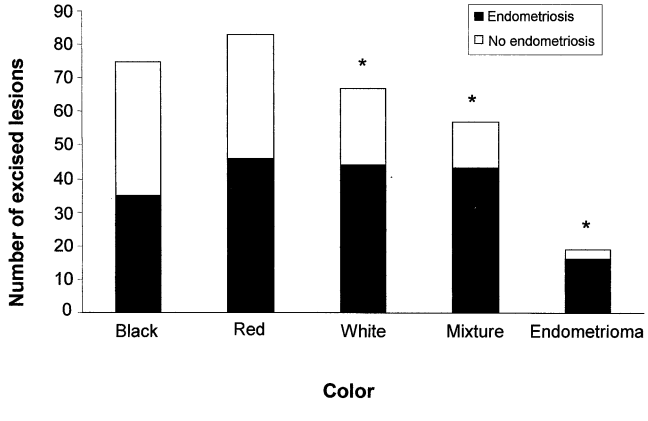
Most of the peritoneal endometriotic lesions were small and superficial; 87% were less than 1 cm wide and 47% were less than 5 mm wide. The greater the diameter of the lesion, the more likely it was histologically positive for endometriosis ($P=.003$, $\chi^2$ for trend) (Fig. 3). Similarly, histologically confirmed endometriosis lesions had a larger average width than did those without endometriosis (mean width [±SD], 7.9 ± 0.6 mm vs. 5.6 ± 0.7 mm; $P=.0004$). When lesions wider than 1 cm with one large confluent lesion were compared with those containing multiple smaller lesions, the rate of histologic confirmation was similar (data not shown).

Peritoneal lesions were almost always (97%) less than 1 cm deep, and 60% were less than 5 mm deep. The deeper the lesion, the more likely it was to be histologically confirmed endometriosis ($P<.0001$, $\chi^2$ for trend) (Fig. 4). Similarly, the mean depth of histologically confirmed endometriosis was significantly greater than that of unconfirmed lesions (mean 4.8 ± 0.3 mm vs. 3.4 ± 0.3 mm; $P<.0001$).

Overall, endometriotic lesions that could be confirmed histologically were significantly wider and deeper than were

## FIGURE 2

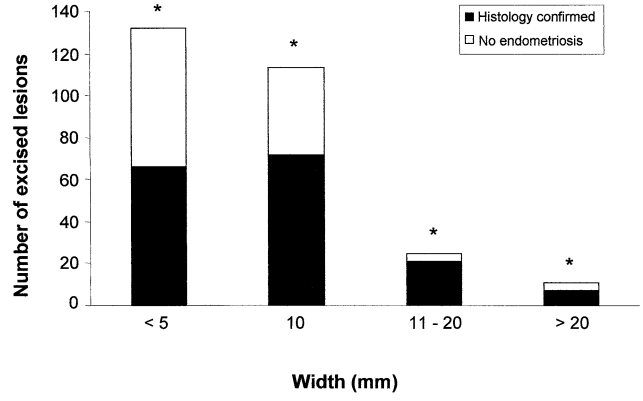
Color of endometriotic lesions. *$P=.001$ by $\chi^2$ test for histologically confirmed endometriosis, by color category.




*Stratton. Surgical* vs. *histological endometriosis. Fertil Steril 2002.*

## FIGURE 3

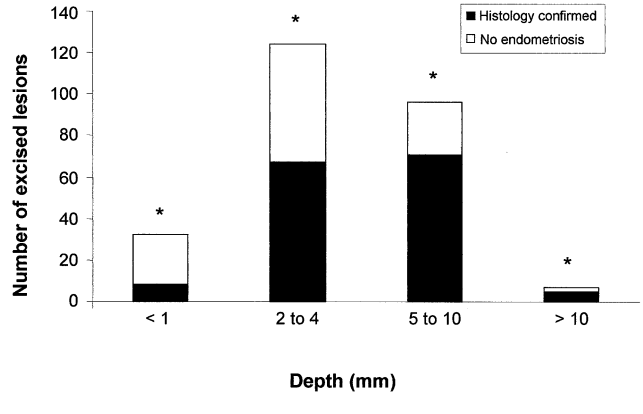
Width of endometriotic lesions. *$P=.003$ by $\chi^2$ test for trend for likelihood of histologically confirmed endometriosis, by width of lesion.




*Stratton. Surgical* vs. *histological endometriosis. Fertil Steril 2002.*

## FIGURE 4

Depth of endometriotic lesions. *$P<.0001$ by $\chi^2$ test for trend for likelihood of histologically confirmed endometriosis, by depth of lesion.




*Stratton. Surgical* vs. *histological endometriosis. Fertil Steril 2002.*

lesions that were negative on histologic examination. Histologically proven endometriotic lesions had a twofold greater mean volume than did nonendometriotic lesions ($P<.0001$).

Endometriomas ranged in size from 5 mm to 9 cm, and all were benign. Those deeper than 10 mm were always histologically confirmed as endometriosis and were significantly more likely than smaller lesions to be endometriosis ($P<.02$). Histologically proven endometriotic lesions were 50 times larger than those in which endometriosis could not be confirmed.

Fifty percent (7 of 14) of peritoneal defects were positive for endometriosis, but no particular color was predictive of endometriosis (data not shown). Of 11 women with peritoneal windows, 8 had endometriosis within the peritoneal window or at another pelvic location.

Endometriosis in smaller lesions sent for pathologic examination was compared with the rate of microscopic confirmation when only part of a larger lesion was examined. Overall, lesions that were divided were significantly larger than were lesions that were not divided (mean width, $7.9 \pm 0.6$ mm vs. $4.9 \pm 0.8$; $P<.0001$). Lesions that were divided were more likely to be positive than were those that were not divided (64% vs. 49%; $P=.02$). The proportion of lesions in each color category that were histologically confirmed to be endometriosis was similar regardless of whether the lesion was divided (data not shown).

When size and color were considered together, depth ($P=.03$), mixed color ($P=.01$), and white color ($P<.05$) predicted histologic confirmation of endometriosis by logistic regression. Location was not predictive of histologic confirmation of endometriosis.

## DISCUSSION

We report on surgical and histologic findings of apparent endometriotic lesions in 77 women with chronic pelvic pain who were thought to have a high likelihood of endometriosis before surgery. Endometriosis was diagnosed if at least one pigmented or nonpigmented implant was confirmed as endometriosis by histologic examination.

Our rate of detection of endometriosis is similar to rates reported by others after excision of all types of endometriosis lesions (1, 4). This suggests that surgeons who excise endometriosis should look for subtle lesions as well as pigmented ones.

As others have reported, we found that mixed-color lesions (called "puckered-pigmented lesions" in other reports [(1, 4, 18)]) and endometriomas were most likely to be confirmed as endometriosis on the basis of histologic examination (1, 4). We were surprised that white lesions were more likely to be endometriosis than black or red ones, which were more common and easier to see. This is contrary to the findings of Walter et al. (4) and Martin et al. (1). Perhaps the lower detection rate in those other studies was due to inclusion of adhesions with white lesions. We considered white peritoneal lesions or scarred areas separately from adhesions between pelvic areas or organs. In most cases, these adhesions were lysed but not excised. Of the few adhesions that were excised ($n = 6$), none were confirmed to contain endometriosis.

We found endometriosis in various pelvic locations, with most found in the cul-de-sac or on the uterosacral ligaments and ovarian fossa. Implant location did not predict histologic confirmation of endometriosis. Most peritoneal endometriotic lesions were superficial and small, measuring less than 1 cm in any direction. It is not surprising that larger lesions, as measured by depth or width, were more likely to be endometriosis than were smaller, shallower lesions. Rates of detection were similar between large lesions and areas containing many small lesions.

Our cohort included a large proportion of women with a history of surgically diagnosed endometriosis (51 of 77), perhaps because we work from a tertiary referral center and are specifically interested in pelvic pain from endometriosis. Since we did not review previous medical records, operative reports, or pathology reports, we did not know the previous extent of endometriosis, nor did we attempt to confirm the diagnosis before the initial surgery. We found that women with moderate to severe endometriosis were more likely to have had a previous surgical diagnosis of endometriosis. This may reflect the fact that persistent or recurrent endometriosis is more common in women with more extensive disease.

Of women with lesions suspicious for endometriosis at surgery, more than 72% (47 of 65) had minimal or mild endometriosis. All women with mild disease had histologically confirmed endometriosis, but one third with minimal disease (7 of 22) did not. One additional woman with severe disease had negative results on biopsy, perhaps because her endometriosis was below the peritoneal surface in a frozen pelvis. Our high false-negative rate among women with minimal disease is similar to findings of other investigators. Walter et al. (4) reported that 12 of 37 women with visible evidence of endometriosis did not have histologic confirmation. Others have reported that many women with chronic pelvic pain from endometriosis have minimal to mild disease (9, 10), leading to the observation that the extent of disease is not correlated with the amount of pelvic pain.

In our cohort, 12 women had no evidence of endometriosis at surgery but were found to have other potential causes of pelvic pain; most had pelvic adhesions or evidence of a pelvic infection. This finding is similar to that of Walter et al. (4), who reported that 7 of 44 women with chronic pelvic pain had no visual or histologic evidence of endometriosis. Half of these women had a previous diagnosis of endometriosis. In addition, the frequency distribution of endometriosis extent by American Society for Reproductive Medicine

stage was similar for initial and second-look surgical procedures, suggesting that endometriosis does not progress rapidly (19).

Our findings are important in that surgeons who do not excise lesions or biopsy lesions suspected to be endometriosis for biopsy may mislabel women as having endometriosis when they do not. On the basis of our data, this would be expected to occur one third of the time in patients with minimal disease. Thus, not confirming the diagnosis may lead to inappropriate surgical or medical treatments.

Our study has limitations. First, we did not perform random biopsies of normal-appearing peritoneum to look for endometriosis. Because our goal was to relieve pain by excising areas suspicious for endometriosis, we did not determine whether microscopic endometriosis was found in pelvic regions in which endometriosis is common. Other researchers have found low rates of endometriosis in normal-appearing peritoneum (4–8).

Second, because we excised all suspicious areas as well as areas that were believed to be endometriosis, the false-negative rate that we report may be higher than expected and our detection rate might be falsely low. Similarly, the lower detection rate may have been lower in smaller lesions because of sampling error.

Third, we may have unintentionally increased the false-negative rate of endometriosis by dividing larger lesions to set aside tissue for research purposes. Divided lesions were both larger and more likely to be confirmed as endometriosis. Since our rate of biopsy-confirmed endometriosis is not significantly lower than rates reported by others (1, 4) and we carefully divided specimens to send representative areas for pathologic examination, the false-negative rate may not be significantly increased by dividing the specimens. However, the only way to know whether this false-negative rate is artificially high as a result of division of larger specimens would be to send all reserved tissue for pathologic diagnosis. Such a study would be costly and logistically complex.

Our study also had many strengths. We had good statistical power owing to the large sample. Ninety-one surgical procedures were performed specifically to treat endometriosis, and more than 300 implants were excised from 65 women in whom endometriosis was surgically diagnosed. Second, the detailed characteristics of the implants excised were collected systematically. These data were recorded at the time of surgery on a previously constructed document for the specific purpose of collecting such data. Third, one surgeon (P.S.) was present at all surgical procedures, and the other (C.W.) was present at almost 80% of the study procedures, thereby reducing interobserver bias. Finally, our results provide data on the characteristics of endometriosis lesions that have not been studied or reported in such detail previously.

In conclusion, neither color, nor size, nor location are effective factors that predict the presence of endometriosis at the time of laparoscopic visualization. For women with minimal disease, histologic examination is often negative and the diagnosis of endometriosis cannot be confirmed. Thus, if laparoscopic surgery is going to performed for the specific purpose of confirming a diagnosis of endometriosis, surgeons must obtain tissue samples of each apparent lesion for histologic evaluation. Otherwise, considering a patient to have endometriosis on the basis of surgical visualization may not be as accurate as making the diagnosis on the basis of clinical suspicion alone (20). These data indicate that a histologic marker for endometriosis is needed in cases of negative findings during laparoscopy.

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