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Risk Factors for Endometrial Hyperplasia and Cancer in Patients with Abnormal Uterine Bleeding in Al-Zahrawi Hospital

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ABSTRACT

Aim and Background: The study aims to determine the risk factors for endometrial hyperplasia and endometrial cancer in a patient with abnormal uterine bleeding and thus early diagnostic investigations to detect the disease and limit its development. *Method*: The study sample includes women attending AL-zhrawi Hospital with a complaint of abnormal uterine bleeding and who fulfilled the entry criteria. The women in the sample were divided according to the result of the pathological autopsy into The first group "control group": it is the group that showed normal endometrioses by histopathology and The second group "cases group": it is divided into two categories: the first category it is the group that showed the presence of endometrial cancer through histopathology.

Results: The mean age in the endometrial cancer group was 58.6 years \pm 6.5 years, the BMI value was expressed as the arithmetic mean of values for each group, and the highest mean in the cancer group was 28.75 kg/m². The differences between the endometrial and control groups and the two hyperplastic groups The control is statistically significant with a P value of 0.001, and 60% of endometrial cancer patients were in menopause, and this is associated with hormonal changes accompanying the age of transition to menopause, and 45% of them were not giving birth. 35% of patients with cancer were found in this study. Endometriosis has diabetes, and this value was statistically significant compared to the control group with (P<0.05), and arterial hypertension was recorded in 80% of our patients, and it was statistically significant (P<0.05), and family history was found. of endometrial cancer in 50% of endometrial cancer patients, and half of the cases were in the mother and the other half in the sister.

Conclusion: In this study, according to the set conditions, 123 women suffering from abnormal uterine bleeding were recruited. The number of diagnosed endometrial cancer cases was 20, and each of the following factors was statistically significant for the development of endometrial cancer: advanced age - high BMI- early puberty - anuria female childbirth the presence of a family history of endometrial cancer diabetes mellitus High arterial pressure, while the following factors were not statistically significant: Increased number of births - Intensity of vaginal bleeding.

Keywords: Cancer, Endometrial hyperplasia

INTRODUCTION

Abnormal uterine bleeding has multiple patterns and various descriptive terms. It is a common health problem for women, and it constitutes 20% of women's clinic visits [1]. Causes include anatomical changes, hormonal abnormalities, infections, systemic infections, medications, and pregnancy complications (Table 1) [2]. It can affect women of all ages, and the most important factors involved in it are age and fertile status. In adult women, the cause is more related to pregnancy and sexually transmitted diseases, with a lower incidence of anovulatory cycles. The incidence of bleeding associated with fibroids is increased and uterine polyps with age [2]. Perimenopause is due to poor function of the thalamic-pituitary-ovarian axis more frequent, and the bleeding rate is attributed to complications of pregnancy and sexually transmitted diseases, With age, the risk of bleeding increases due to benign and malignant neoplastic lesions [2]. Bleeding after menopause can be of normal origins, such as vaginal or uterine atrophy Polyps. In addition, malignant neoplasms, especially endometrial cancer, are more common this age group. Estrogen-secreting ovarian cancer can cause endometriosis Uterus with abnormal uterine bleeding [2].

Pathogenesis

The endometrium consists of two specific regions: the functional layer and the basal layer. The basal layer is located in direct

contact with the myometrium, under the functional layer. And it's less It is influenced by hormones and serves as a repository for postmenstrual remodeling. In contrast, the functional layer defines the uterine cavity, undergoes dramatic changes during the cycle, and sheds during menstruation [2]. Blood supply to the uterus through the uterine and ovarian arteries, from which the arcuate arteries arise. to perfuse the muscle, and these in turn are divided into radial arterioles that extend within the muscle at a right angle from the arcuate arterioles.

Muscular, which is somewhat insensitive to hormonal changes, and spiral arterioles irrigate the layer functional and end with a subepithelial lattice [2]. At the end of each menstrual cycle, progesterone levels drop and trigger the release of proteolytic MMP enzymes (matrix metalloproteinases). These enzymes break down the mesenchyme and vascular structure of the functional layer. Subsequent hemorrhage and sloughing constitute the menstruation that occurs [2]. Platelet aggregation and thrombus formation control blood loss, and in addition, the remaining endothelial arterioles contract under the influence of astringent media, which reduces bleeding. Disruption of the previous balance leads to the occurrence of abnormal uterine bleeding, some of which are irregular in quantity and some in timing or duration [3].

Initial evaluation for abnormal bleeding includes a detailed clinical history, physical examination, cytology, pelvic echo, and blood tests. The aim is to try to establish a cause for the bleeding Before moving on to the more invasive procedures [3].

Hyperplasia of the endometrium

Glandular hyperplasia of the endometrium is generally a benign lesion. Hyperplasia is classified according to the World Health Organization into four types: simple without atypia, complex without atypia, simple with atypia, and complex with atypia. Because of the association of hyperplasia with hyperestrogenism, atypical hyperplasia is considered a preneoplastic lesion[4].

Hyperplasia without atypical

This type of hyperplasia is microscopically an accumulation of glandular in the stroma without nuclear abnormalities. This type is usually asymptomatic and is incidentally identified in uterine samples. Simple hyperplasia without atypical is large glands with irregular external borders. Follow-up of this pattern without treatment for 15 years shows the development of endometrial cancer in 1% of cases, while it relapses spontaneously in 80% of patients [5].

As for the complex hyperplasia without atypical, it shows the appearance of complex crowded glands with papillae that include the lumen. These lesions are regressed by the effect of progestin therapy in 85% of patients. cases, but the development of cancer occurs in 3%-5% of cases in the absence of treatment [5].

Hyperplasia without atypical

In Latin America, Yellow Fever remains a persistent threat. Between 1980 and 2012, 150 outbreaks of this entity have been reported in 26 African countries, with more than 200 000 cases occurring globally. From December to February 2017, an outbreak of Yellow Fever affected Brazil, with 1345 suspected cases, 295 confirmed cases, and 215 deaths.

Atypical hyperplasia

It is characterized histologically by a profuse swarming of the endometrial glands, which are demarcated by enlarged cells. The nuclear-cytoplasmic index increases, which reflects increased nuclear activity. The nuclei become irregular and the chromatin aggregates in the form of coarse granules with the dominance of nucleoli [5]. They are considered pre-neoplastic lesions, and the development of cancer is 10% in cases of simple hyperplasia, and 30% in complex hyperplasia. Most of the lesions relapse with progestin therapy, but with a high rate of recurrence upon discontinuation of treatment [5]. A progressive study of the Gynecological Tumors Group (GOG) recorded that women with untreated atypical hyperplasia, diagnosed with biopsy before surgery, had attached endometrial cancer in 42.6% of cases after a hysterectomy. As for women with an endometrial biopsy result less than atypical hyperplasia, cancer was found in 18.9% of the cases. Excised womb samples [5].

Endometrial cancer

Endometrial cancer is the most common malignancy of the female reproductive system and accounts for about half of all gynecological cancers in the United States. About 41,200 new cases and 7,350 deaths associated with this cancer were registered in 2006 [6]. It is the fourth most common type of cancer after breast, lung, and bowel, and the eighth leading cause of death from malignant tumors in women. Overall, approximately 2%-3% of women will develop endometrial cancer during their lifetime [6]. In recent years, specific factors have increased interest in the diagnosis and treatment of endometrial cancer. These factors include low incidence and death of cervical cancer in developed countries, higher life expectancy, and hormonal therapy. Postmenopausal women, early diagnosis [3]. Availability of easy diagnostic tools and a clearer understanding of precancerous lesions of the endometrium. It led to an increase in the number of women diagnosed with endometrial cancer. Endometrial cancer appears early and is generally treatable without radical surgery or radiation. However, deaths from endometrial cancer currently exceed those from cervical cancer in the United States, and in developing countries lack medical and economic resources plays a role. A role in delaying its discovery and treatment and thus increasing deaths due to it [7,8]

It mainly occurred In postmenopausal women, and virulence increased with age demonstrating the role of estrogen in the development of most uterine cancers, and thus any factor that increases unopposed estrogen exposure increases the risk of endometrial cancer [3]. In recent decades, a better definition of histopathology, the pattern of spread, and clinical and pathologic factors affecting prognosis has been developed, and the treatment of endometrial cancer has been developed from preoperative pelvic irradiation followed by hysterectomy depending on the clinical stage to specific treatment using hysterectomy as initial treatment and followed by treatment. After surgery, depending on the surgical and pathological findings, there is still a need for more analysis and investigations to determine whether this approach will translate to improved survival rates and reduced mortality [3].

Clinical characteristics

Endometrial cancer usually occurs in women in the sixth and seventh decades of life, with a median rate of 60 years. About 90% of women with endometrial cancer have genital bleeding or missing as a single symptom, and most women recognize the importance of this symptom and ask for Medical consultation within 3 months [9]. Some women suffer from pelvic heaviness or discomfort, which points to uterine enlargement or the spread tumor outside the uterus Bleeding should not occur due to cervical stenosis, especially in elderly women. It is accompanied by hemothorax or uterine suppuration presenting with purulent vaginal discharge, and these findings are often associated with a bad prognosis [9].

Less than 5% of women diagnosed with endometrial cancer are asymptomatic, and screening for endometrial cancer is usually carried out in the absence of symptoms as a result of screening for an abnormal pap smear, or evaluation for abnormal findings on pelvic imaging for another reason, if there is a Malignant cell on the neck smear suggest that the disease is advanced [9]. Peri- and post-menopausal bleeding should be well investigated and taken seriously Seriousness and no importance of quantity and continuity [2].

Risk factors

Several risk factors for the development of uterine malignancies have been identified, most of which are associated with prolonged endometrial stimulation, and this risk is increased by hypo fertility and irregular menstrual cycles as a result of anovulatory cycles (pro-longed exposure to estrogen without adequate progesterone), This is associated with early puberty, late menopause, and childlessness (Table 2) [3]. The risk factors for developing endometrial hyperplasia are similar to the risk factors for developing endometrial cancer [10,11]. Studies have found an increase in the incidence of endometrial cancer among the generations born in the United States of immigrants of Asian, Chinese, and Japanese descent, and this has been attributed to the change in the surrounding environmental and societal conditions [12]. Other medical conditions, such as high blood pressure and hypothyroidism, are associated with endome-trial cancer, but the causal relationship has not been established [3].

The age

The chance of developing endometrial cancer increases with age, and 75% of cases occur in people over 55 years old [3]. Younger women with endometrial cancer. They have a better prognosis than older women [13]. Increased patient age is independently associated with disease recurrence [14].

Genetic and familial factor

Women with Hereditary Nonpolyposis Colorectal Cancer (HNPCC) or Lynch syndrome with an inherited mutation in the MLH1-MSH2 repair genes MSH6 have a 40%-60% lifetime risk of developing endometrial cancer [15]. It found an association between the occurrence of endometrial cancer and the presence of a family history of endometrial cancer among the female relatives of the patient. Family history of endometrial cancer [16,17].

Puberty

Early puberty has been identified as a specific risk factor for the development of endometrial cancer due to exposure to non-opposite long-term estrogen in the anovulatory cycle, and studies recorded significant differences in the incidence of puberty before the age of 12 years between two groups of endometrial cancer and a control group [16,18].

Obstetric condition

The incidence of endometrial cancer was found to be higher among nulliparous women [19]. Andarieh and her colleague's (2016) study record reported that the incidence of nulliparous women was significantly higher in the endometrial cancer group, with a statistical difference between them (P<0.001), and a previous study found, the incidence of endometrial cancer was lower among neonates [20]. Infertile women have a higher risk of endometrial cancer due to their exposure to the medication. Prescribed in the treatment of infertility and have been reported to play a role in carcinogenesis endometriosis [16].

Non-opposite estrogens

The association between treatment with exogenous estrogen is not adverse in postmenopausal women. The incidence of endometrial cancer was originally suggested in the early 1970s when a 20% -35% increase in the incidence of endometrial cancer was observed in Western Caucasian women when using estrogen therapy [21], this risk exists when using unopposed estrogen therapy at low or high doses [22]. This risk appears to increase with increasing doses of estrogen used. Grady and colleagues reviewed 14 case-control studies examining estrogen dose and relative risk of endometrial cancer, and 11 of these studies showed an increase in relative risk with increasing doses of conjugated equine estrogens [23]. Other factors that lead to long-term estrogen exposure, such as functional ovarian neoplasia, and polycystic ovary syndrome, are also associated with an increased risk of endometrial cancer. HRT without progestins increases the risk of endometrial cancer from 4 months -8 months. This risk increases with higher doses and with prolonged use, and the initial risk can be reduced with the use of progestin. It was noted that the use of anti-estrogen tamoxifen for the treatment of breast cancer was associated with a 2 month-3 month risk of developing endometrial cancer [3]. Contraceptive pills, by providing a sufficient amount of progestin, are considered a protective factor against the development of endometrial cancer in the uterus [24].

Risk factors of menopause

Considering that higher or above physiological doses of estrogens increase requirements. Progesterone does not adequately counteract the developing actions of estrogen on the endometrium, so the perimenopausal transition period can represent a 'risk factor' especially for unopposed estrogen action. Several studies have demonstrated an increase in estrogen level and urinary excretion during the perimenopausal period with an association with low and irregular progesterone levels following menstrual abnormalities [25]. The hormonal environment during perimenopause appears to support insufficiently adverse endometrial development, especially with the onset of irregular periods [25].

Epidural

The occurrence of menorrhea after the age of 52 years increases the risk of endometrial cancer 2.4 times compared to women who experience menorrhagia before the age of 49 years of age (possibly due to prolonged exposure to progesterone-deficient menstrual cycles) [3].

Obesity

The most common risk factor for endometrial cancer is obesity, and no other cancer shows the same This was strongly associated with obesity [26]. Obesity increased in line with the increase in life expectancy. It is responsible for the high incidence of endometrial cancer [27]. More than a third of women with endometrial cancer in the UK are obese [28]. 17 epidemiological studies out of 18 epidemiological studies showed that the frequency of overweight and obesity was significantly higher in cases than in controls [29]. Most of the theories put forward to explain the increased risk of endometrial cancer relied on elevated levels of circulating estrogen through the conversion of androstenedione to estrone in the peripheral adipose tissue, and deficient circulating levels of Sex Hormone Binding Globulin (SHBG) [3]. In addition, increased adiposity leads to increased insulin resistance and a pro-inflammatory environment, which has been linked with subcutaneous carcinoma. uterus [30]. Obesity is also an important risk factor for endometrial cancer in premenopausal women. A study recorded the average weight of premenopausal women with endometrial cancer, as 198 pounds, compared to 173 pounds for women over 45 years old [31]. The lower SHBG levels associated with premenopausal obese women compared with normal-weight women may be responsible for the higher levels of free estrogen [32]. Insufficient progesterone levels in the luteal phase may be an important contributing factor to the increased risk of endometrial cancer in premenopausal women, but a recent study indicated that leptin may be a possible cause of ovulation disruption and artificial steroids [33].

Diabetes miles

Several case studies found a two-fold increase in the risk of endometrial cancer sugars [34,35]. Thirteen epidemiological studies, published between 1958 and 1990, showed an increase in the incidence of diabetes in cases more than in controls, but only 3 studies showed a statistically significant difference, and the percentage of women with endometrial cancer who reported a scribin' deficiency varied 6% -23% [36]. I conducted a case-control study on 123 cases of diabetic patients and 2291 witnesses, and I found that Diabetic women have a higher risk of endometrial cancer, OR=1.86 [37]. There is a serious debate about how hyperinsulinemia, or hypergly-cemia, affects the development of carcinoma endometrium in diabetic women, it was assumed that insulin plays a role as an inducing factor on mitosis in the endometrium by amplifying the effects of IGF in the endometrial development [38]. IGF compounds, especially IGF-1, play a role in the metabolism mediating estrogen-induced endometrial development via autoregulatory mechanisms, and in addition 18 Therefore, it was observed that insulin decreases the binding of progesterone to its receptors, and weakens the antagonistic action for reproduction by anti-estrogens [39,40].

Exercises

The role of exercise in protecting against endometrial cancer remains unclear, 10 case studies out of 11 case studies suggested that moderate exercise was associated with a decrease in the risk of endometrial cancer, but this decrease may be through association with other health factors such as normal weight. and healthy diet.

Diet

The role of dietary factors in the development of endometrial cancer has been the subject of interest for decades, particularly in light of the wide differences in incidence between women living in Western or Asian countries. Several studies evaluated the role of diet in endometrial cancer and found that the consumption of whole grains, fresh fruits, and vegetables was associated with a decrease in the risk of endometrial cancer, and there was interest in whether the vegetarian diet could improve hormonal status in women Ladies. A study was conducted to determine the role of the Mediterranean people's diet in the development of endometrial cancer, and it was found that eating a Mediterranean diet rich in vegetables leads to a decrease in the risk of endometrial cancer and that eating a diet rich in meat stimulates a pro-inflammatory response that leads to an increase in CRP, that chronic clinical inflammation leads to insulin resistance, which In turn, it is responsible for stimulating cell proliferation and initiating programmed cell death. Asian women living in Asia have a 1/15 risk of endometrial cancer Compared to Caucasian women living in the West, and as a result, there was wide interest in the Asian diet as a possible protective factor. This diet is higher in fiber and vegetable food, lower in fat, and includes a large portion of legumes as the main source of protein. In a large case-control study, Goodman and colleagues analyzed the consumption of legumes (such as beans and soy) in the diet, and the researchers found that High consumption of beans and bean products Soy is associated with a decreased risk of endometrial cancer 0.45=OR.

MATERIALS AND METHODS

Purpose of the study

Determination of risk factors for uterine hyperplasia of the breast in bleeding patients. Uterine abnormalities, veneration of the condition. Diagnostic investigations, PCR, to detect the disease and limit its development.

Study justifications

He announced endometrial cancer among the most common malignancies that cause major opposition and deaths in women, uterine cancer, and more cancers of the female reproductive system, which are common and with a continuous increase.

The studied sample

The sample of the study includes women who were referred to Al-Zahrawi Hospital with a complaint of abnormal uterine bleed-ing, and who met the entry criteria. The women in the sample were divided according to the result of the histopathology into: The first group, the "control group", is the group that showed normal endometriosis with histopathology. The second group, "the group of cases": Is divided into two categories: The first category is the category that showed hyperplasia of the endometrium by pathological anatomy. The second category: is the category that showed the existence of Endometrial carcinoma histopathology.

Entry criteria

- The woman is over 35 years old.
- Presence of abnormal uterine bleeding.

Exclusion criteria

- Coagulopathies.
- Pregnancy.
- Intrauterine devices.
- Uterine fibroids, endometrial polyps, endometriosis, malignancies of the cervix, and ovarian cysts.

Sample size

Rather, the full sample size is 123 patients, distributed as follows: 20 patients with endometrial cancer, 69 patients with endometrial hyperplasia, and 34 women (witnesses).

Place of study

Al-Zah Arwa Hospital.

Study type

Cross-sectional case-control.

Study method

The study is a cross-sectional case-control study and includes adamantine women who had severe and recurrent uterine lesions with a risk of pathologic anatomy of the endometrium.

Data collection and analysis

A new collection of the payment data of the students who checked the conditions of the study using a questionnaire designed for that. The clinical examination and the results of the pathological anatomy were approved against the documents, and the results of laboratory analyzes were "pregnancy test, blood count, Urea, creatinine, liver enzymes, bleeding and clotting times". By counting all the data, encoding them, entering them into the computer, and making a statistical analysis against specific tables and representing them graphically. The statistical analysis was carried out using the SPSS-17 program, and the variable was considered to have a statistical value Significant for P-values less than or equal to 0.05 (Table 3).

Age(%)	41_50	2(10)	P value 0,001	Family history of endometrial	10(50)	Mother(%)	5(50)	
	51_60	10(50)		cancer(%)		Sister(%)	5(50)	
	61_70	7(35)		Diabetes mellitus(%)	Have diabetes		7(35)	P value
	71_76	1(5)			Non diabetics		13(65)	0,04

Table 1. Endometrial cancer risk factors (n=20).

	27	1(5)		Arterial	Having high arterial pressure	16(80)	P value 0,007
Body Mass	28	10(50)	P value	hypertension(%)	Normal arterial pressure	4(20)	
Index (%)	(%) 29 7(35) 0,001 Intensity of		mild	10(50)			
	30	2(10)		gynecological bleeding(%)	Medium	6(30)	
	10	3(15)			Heavy	4(20)	
Bubonty (9/)	11	10(50)	P value 0,001	- Previous Births(%)	0	9(45)	
Puberty(%)	12	6(30)			3	4(20)	
	13	1(5)			4	5(25)	P value
The occurrence of menopause(%)	There is menopause	12(60)	P value		5	1(5)	0,002
	There is no menopause	8(40)	<0,05		6	1(5)	

 Table 2. Risk factor of endometrial hyperplasia(n=69)

	36-40	4(5,79)	Family history			
	41-50	42(60,87)	of endometrial	12(17,4)	Mother(%)	5(50)
Age(%)	51-60	17(24,64)	hyperplasia(%)			
	61-70	5(1,45)	\mathbf{D} = \mathbf{b} = \mathbf{c} = \mathbf{c} = \mathbf{U} = \mathbf{c} = 0	Have diabetes		23(33,3)
	71-76	1(1,45)	Diabetes mellitus(%)	Non diabetic		46(66,7)
	25	5(7,25)	Arterial	Having high arterial pressure		39(56,5)
Dody Moos Index	28	42(60,87)	hypertension(%)	Normal arterial pressure		30(43,5)
Body Mass Index (%)	29	17(24,64)		mild		7(10,14)
(70)	30	4(5,79)	Intensity of			
	32	1(1,45)	gynecological	Medium		43(62,32)
	9	4(5,79)	bleeding(%)	Heavy		19(27,54)
	10	5(7,25)				
Puberty(%)	11	42(60,87)		3		4(5,8)
	12	17(24,64)		4		41(59,45)
	15	1(1,45)		5		15(21,75)
The occurrence of	There is menopause	10(14,49)	Previous Births(%)	6		3(4,35)
	There is menopause	10(14,49)		7		2(2,9)
menopause(%)	There is no	59(85,51)			10	1(1,45)
	menopause	59(05,51)		12		1(1,45)

 Table 3. Abnormal Uterine Bleeding(n=34)

	36 40	5(14,71)			
	41_50	18(52,94)		Have diabetes	4(11,8)
Age(%)	51_60	9(26,47)	Diabetes mellitus(%)		
	61_70	1(2,94)			
	71_76	1(2,94)		Non diabeticsd	20(88,2)
	22	1(2,94)		Having high arterial pressure	14(41,2)
	25	18(52,94)	Arterial hypertension(%)	Normal arterial pressure	20(58,8)
Body Mass Index (%)	26	9(26,47)		Mild	6(17,65)
(70)	27	5(14,71)			
	28	1(2,94)	Intensity of gynecological bleeding(%)	Medium	22(64,71)
	12	21(61,74)	biccuilig(70)	Heavy	6(17,65)
$\mathbf{D}_{\mathbf{u}}\mathbf{b}_{\mathbf{o}}\mathbf{v}\mathbf{t}_{\mathbf{v}}(0/0)$	13	12(35,28)			0(17,03)
Puberty(%)	14	1(2.04)	Previous Births(%)	3	5(14,71)
	14	1(2,94)		4	18(52,94)

			5	9(26,47)
The occurrence of	There is menopause	6(17,65)	6	1(2,94)
menopause(%)	There is no menopause	28(82,35)	7	1(2,94)

RESULTS & DISCUSSION

Endometrial cancer is common cancer that causes a significant proportion of deaths due to Tumors despite its early symptoms of abnormal uterine bleeding. The determining factors A risk for the development of uterine malignancies in patients with abnormal uterine bleeding constitutes an important point in the Early investigation, thus reducing morbidity And mortality. In this study, and according to the established conditions, 123 women suffering from uterine Bleeding have recruited anomaly. The number of diagnosed cases of endometrial cancer was 20. Each of the following factors was statistically significant in the development of Endometrial cancer: Advanced age-high BMI-early puberty-anorexia - childbirth-the presence of a Family history of endometrial cancer-diabetes high arterial pressure. While the following factors were not statistically significant: The increase in the number of Births, the severity of vaginal bleeding. Previous studies have reported that the risk factors for the development of endometrial Hyperplasia are similar to Risk factors for the development of endometrial cancer. All of the patients in our study had abnormal uterine bleeding "menstrual irregularities".

According to the conditions of the study, Soliman and colleagues' study recorded Their study 188 in the menstrual cycle Patient from 1989-2003 in the United States A Disorder in 39% of endometrial cancer patients. The mean age in the endometrial cancer group was 6.58 years \pm 5.6 years versus 28.49 years \pm 42.7 years for hyperplastic patients and 09.48 years \pm 18.8 years for the control, the difference was Important medical reports about cancer for cancer patients, which is consistent with the Publications endometriosis is a disease of the 6th and 7th decades. BMI was higher in cancer and hyperplastic groups compared to the control group with Differences Statistically significant, which is what previous studies recorded about the Important role of obesity and overweight in the development of endometrial malignancies, which later led to work on studying the role of diet in preventing it. The important factor in the development of neoplastic events in the endometrium is Prolonged exposure to estrogen that is not opposed by progesterone, and the Accompanying factors are early puberty, late menstruation, and childlessness. The study Recorded an important association of these factors with the occurrence of endometrial Cancer, and 60% of endometrial cancer patients were in menopause, and this is accompanied by hormonal changes accompanying the age of transition to menopause. 45% of them were childless. Previous international studies recorded similarly high rates of childless women with significant differences: the study of Yamazawa and colleagues (50%). Tran and associates study (51%), and Soliman and colleagues study (59%).

The incidence of puberty was significantly earlier reported in patients with endometrial Cancer and in hyperplastic patients compared to the control, which is consistent with previous findings. In this study, 35% of endometrial cancer patients were found to have diabetes, and this Value was statistically significant compared to the control group with (P<0.05). This association was recorded in previous similar studies, and Soliman and colleagues Recorded the presence of diabetes in 23% of young endometrial cancer patients, and this is Consistent with the increased risk of endometrial cancer in diabetes. As for arterial hypertension, it was recorded in 80% of our patients, and it was statistically significant (P<0.05), and since its position as an independent risk factor was not identified in A significant way, few studies included it in the results. Soliman and his colleagues recorded A significant percentage of 23% in patients with endometrial cancer at an early age, and These values give high arterial pressure importance in endometrial cancer.

CONCLUSION

A family history of endometrial cancer was found in 50% of endometrial cancer patients, half Of the cases were in the mother and the other half were in a sister. This high frequency of Familial history strongly suggests a genetic role in the development of endometrial cancer, Although the descriptive study pattern did not allow for the identification of specific genetic Syndromes or defects. This is supported by the results of previous studies, and Andarieh's. The study recorded a significant difference (P=0.001) between the group of endometrial cancer.

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