Management and Clinical Outcomes of Endometrial Hyperplasia during a 13-Year Period in Songklanagarind Hospital

Nathapol Sirimusika MD*, Krantarat Peeyananjarassri MD*,
Yuthasak Suphasynth MD*, Virach Wootipoom MD*,
Kanet Kanjanapradit MD**, Alan Geater PhD***

** Department of Obstetrics and Gynecology, Faculty of Medicine, Prince of Songkla University, Songkhla, Thailand*
*** Department of Pathology, Faculty of Medicine, Prince of Songkla University, Songkhla, Thailand*
**** Epidemiology Unit, Faculty of Medicine, Prince of Songkla University, Songkhla, Thailand*

Background: *Endometrial hyperplasia has long been considered a precursor of endometrial cancer but there is no consensus regarding its management.*
Objective: *To identify management practices and evaluate outcomes of treatments for women diagnosed with endometrial hyperplasia (EH).*
Material and Method: *The medical records of endometrial hyperplasia at Songklanagarind Hospital between January 2000 and December 2012 were retrospectively reviewed.*
Results: *Two hundred ninety seven patients were diagnosed with endometrial hyperplasia during the study period. Four patients who did not come for treatment and could not be contacted were excluded. Therefore, 293 patients were included in the study. Simple hyperplasia (SH) was the most common diagnosis accounting for 79.2% of all cases, followed by complex hyperplasia (CH) 13.0%, complex atypical hyperplasia (CAH) 5.8%, and simple atypical hyperplasia (SAH) 2.0%. Seventy-eight percent (18/23) of the patients with atypical endometrial hyperplasia were treated by hysterectomy compared with 9.6% (26/270) of patients without atypia. Of the patients diagnosed with atypical EH, 30.4% (7/23) were associated with endometrial carcinoma. Overall, 6% (12/201) of the women who had initial non-hysterectomy management and had additional tissue taken to assess response, had persistent disease, and 1% (2/201) had progressive disease. Eleven patients (5.9%), who had an initial complete regression during the non-hysterectomy management, experienced a recurrence to EH and 2.1% (4/187) were found to have recurrence to endometrial cancer.*
Conclusion: *The majority of patients with atypical hyperplasia were managed by initial hysterectomy. The high risk of concomitant endometrial cancer supports this choice of treatment. In the non-atypical EH, the initial non-hysterectomy management was common but EH recurrence and progression to endometrial cancer after the initial regression occurs often. Therefore, long-term follow-up should be advised.*

Keywords: *Endometrial hyperplasia, Clinical outcomes, Endometrial carcinoma*

J Med Assoc Thai 2014; 97 (3): 260-6
Full text. e-Journal: *http://www.jmatonline.com*

Endometrial hyperplasia is a precursor to endometrial carcinoma[1], which is the most common female gynecological malignancy in Western countries[2] and the fourth most common female gynecological malignancy in Thailand[3]. It is generally classified as simple or complex with or without atypia, based on the architectural complexity of endometrial glands and nuclear cytology[4]. Known risk factors for endometrial hyperplasia are related to an excess of estrogen level relative to progesterone. Therefore, progestin is used to treat endometrial hyperplasia[5,6]. A number of previous systematic reviews or meta-analysis reported on the outcomes of progestin therapy[7-10]. However, few studies have evaluated clinical outcomes of endometrial hyperplasia in Thailand[11]. Hence, the present retrospective study aimed to evaluate the clinical outcomes and identify the management practices of endometrial hyperplasia treatment during a 13-year period in Songklanagarind Hospital, a referral hospital in Southern Thailand.

Correspondence to:
Peeyananjarassri K, Department of Obstetrics and Gynecology, Faculty of Medicine, Prince of Songkla University, Hatyai, Songkhla 90110, Thailand.
Phone: 089-468-1754
E-mail: krantarat@yahoo.com

Material and Method

Study design and population

After Institutional Review Board approval, the present retrospective study was conducted. Pathological test result as well as inpatient and outpatient record databases were reviewed for data on

all patients diagnosed with endometrial hyperplasia between January 1, 2000 and December 31, 2012. We excluded patients who were incidentally diagnosed with endometrial hyperplasia post hysterectomy, and those lost follow-up, could not be contacted after diagnosis.

Clinical data included age at diagnosis, body mass index (BMI), menopausal status, obstetrical history, medical history of diabetes, hypertension, presenting symptoms, and history of hormonal therapy were obtained. The method of initial diagnosis was recorded along with data related to endometrial biopsy, dilatation and curettage, hysteroscopic biopsy, and type of endometrial hyperplasia. Initial management, drug, dose, regimen and route of medical administration, treatment duration, method, and timing of interval endometrial re-evaluation were reviewed from the patients' medical records. The histopathological diagnoses were also reviewed by experienced gynecological pathologists as part of our routine clinical practice for most cases to confirm diagnosis and were classified according to the WHO criteria. After diagnosis, the patients were counseled and offered either the hysterectomy or the non-hysterectomy treatment (expectant management, medical treatment, hysteroscopic resection) according to histological diagnosis, patient prevalence to preserve uterus or fertility, and additional tumors or persistent symptoms. For patients with medical treatment, all received hormonal therapy, such as medroxyprogesterone acetate (MPA), norethisterone (NET), oral contraceptive pills (OCPs), depot-medroxyprogesterone acetate (DMPA), or menopausal hormone therapy. Follow-up in the non-hysterectomy group was at the discretion of the physician; nevertheless, in general, the endometrial biopsies of these were re-evaluated after the initial management and those found to have persistent or progressive disease on the follow-up biopsy were underwent surgery or, if fertility was a concern, surveillance was continued. The follow-up interval comprised the length of time from the initial diagnosis to the last known status including the first occurrence of endometrial carcinoma, hysterectomy, death, last pathological specimen diagnosis or clinical history on censoring date May 31, 2013, which was obtained by reviewing the patients' records, contacting the patients or their gynecologists.

Outcome measures

To assess the treatment response, the follow-up histological results were defined as regression, persistence, or progression based on the comparison of the diagnosed sample with the follow-up specimen. Complete regression was defined as no evidence of endometrial hyperplasia or cancer cells in the follow-up specimens. Partial regression was defined as the presence of endometrial hyperplasia less severe than the histological result at the baseline. A higher grade lesion or worsening of atypia or endometrial carcinoma was classified as progression. Persistent disease was indicated if the last biopsy showed the same histological finding as of the entry diagnosis. Recurrence was defined as an initial completed regression and failure to remain in regression with the evidence of at least one follow-up biopsy showing hyperplasia or carcinoma. The patient was considered to have clinical remission even though no additional tissue samples after treatment were available, but she has been completed clinical follow-up and was asymptomatic or no abnormal uterine bleeding.

Results

Two hundred ninety seven medical records of patients diagnosed with endometrial hyperplasia during the 13-year study period were assessed. None was diagnosed incidentally following hysterectomy but four patients did not return for treatment and could not be contacted, thus, were excluded. The remaining 293 patients were enrolled in the present study.

The clinical characteristics of the patients are presented in Table 1. The median age at diagnosis was 47 years (range 27-86 years), median parity was 2 (range 0-8), and average BMI was 26.8 kg/m^2. Of the 293 patients, 80.5% were multiparous, 52.9% were premenopausal, and 22.8% were obese (BMI >30 kg/m^2). All of the patients had abnormal uterine bleeding as their presenting symptoms. One patient with simple hyperplasia was diagnosed with breast cancer and had exogenous used of tamoxifen for three years. None had family history of bowel, breast, ovary, or endometrial cancer.

The endometrial tissue sampling for initial diagnosis was obtained via outpatient endometrial biopsy in 192 (65.5%) cases, dilatation and curettage in 98 (33.5%), hysteroscopic biopsy in two (0.7%), and transcervical mass biopsy in one (0.3%) case. Simple hyperplasia (SH) was the most common diagnosis accounting for 79.2% of all cases followed by complex hyperplasia (CH) 13.0%, complex atypical hyperplasia (CAH) 5.8%, and simple atypical hyperplasia (SAH) 2.0%.

Table 1. Patient demographics and clinical characteristics

Demographics and clinical characteristics	n (%) n = 293
Age (years), median (range)	47 (27-86)
Parity	
Nulliparous	57 (19.5)
Multiparous	236 (80.5)
Menopausal status	
Premenopausal	155 (52.9)
Perimenopausal	104 (35.5)
Postmenopausal	34 (11.6)
Other medical conditions	
None	188 (64.2)
Diabetes	6 (2.0)
Hypertension	27 (9.2)
Hypertension and diabetes	21 (7.2)
BMI (kg/m^2)	
<18.5	4 (1.4)
18.5-24.9	118 (40.3)
25-29.9	103 (35.2)
30-34.9	47 (16.0)
≥35	20 (6.8)

BMI = body mass index

The initial management of the patients diagnosed with endometrial hyperplasia is shown in Table 2. Overall, 44 (15.0%) subjects underwent initial hysterectomy and 249 (85.0%) received non-hysterectomy treatment. Of all the patients, 2.7% were asymptomatic after curettage and consequently underwent expectant management. The majority of the patients on medical treatment received oral medroxyprogesterone acetate (MPA) at a dosage and regimen that were determined at the discretion of the treating physician. However, of the 227 patients, 97.4% received MPA 10 mg daily, 84.6% received MPA for 12 to 14 days per month, and 92.5% over a period of three to six months. The majority (78.3%) of patients with atypical endometrial hyperplasia received hysterectomy in contrast to those without evidence of cytological atypia (9.6%). Of those given non-hysterectomy management, 201 (80.7%) had additional endometrial sampling after treatment, 34 (13.7%) experienced clinical remission, and 14 (5.6%) were lost to follow-up after the initiation of treatment. All of the patients who were lost to follow-up had been diagnosed with simple hyperplasia, 12 received MPA, one was on expectant management, and one received oral contraceptive pills. Of those who had additional tissue taken after non-hysterectomy treatment, 187 (93.0%) had complete regression, 12 (6.0%) had persistent disease, and two (1.0%) had progression (the details are shown in Table 3). Of the patients who received non-hysterectomy treatment and had complete regression, 63.1% (118/187) had the clinical follow-up schedule with a gynecologist at Songklanagarind Hospital, and two-thirds of them (79/118) continued receiving hormonal treatment after complete regression to maintain a normal endometrium. The median follow-up interval in the non-hysterectomy treatment group was 56.4 months (range 1.4-132.1 months). Of the patients with complete regression, 5.9% (11/187) experienced recurrent endometrial hyperplasia and 2.1% (4/187) were found to have endometrial cancer 6-73 months after complete regression.

Of the 44 patients who had initial hysterectomy, seven (15.9%) were found to have endometrial cancer via hysterectomy specimen and all of them had been

Table 2. Initial treatment of 293 patients diagnosed with endometrial hyperplasia

Treatment	Classification of endometrial hyperplasia, n (%)			
	SH (n = 232)	CH (n = 38)	SAH (n = 6)	CAH (n = 17)
Expectant management	7 (3.0)	1 (2.6)	-	-
Progestin therapy				
MPA	199 (85.8)	24 (63.2)	3 (50.0)	1 (5.9)
NET	6 (2.6)	-	1 (16.7)	-
Oral contraceptives	3 (1.3)	-	-	-
DMPA	1 (0.4)	-	-	-
Menopausal hormone therapy	1 (0.4)	-	-	-
Endometrial resection	1 (0.4)	1 (2.6)	-	-
Hysterectomy	14 (6.0)	12 (31.6)	2 (33.3)	16 (94.1)

SH = simple hyperplasia; CH = complex hyperplasia; SAH = simple atypical hyperplasia; CAH = complex atypical hyperplasia; MPA = medroxyprogesterone acetate; NET = norethisterone; DMPA = depot medroxyprogesterone acetate

diagnosed with CAH. Five of seven patients were found to have endometrial carcinoma at initial hysterectomy within 12 weeks of diagnosis and without interval treatment. Concerning the remaining of two patients, one was lost to follow-up for about one year before hysterectomy was performed, and the other suffered from uncontrolled asthma requiring eight months of treatment before undergoing hysterectomy. The indications for initial hysterectomy are shown in Table 4.

Overall, there were 293 patients with endometrial hyperplasia including 12 (4.1%) associated with endometrial cancer. The risk was higher in the group of patients with atypical EH (30.4%).

Discussion

The present study observed that endometrial hyperplasia affected patients at a median age of 47 years and was associated with abnormal uterine bleeding. These findings are comparable to the exiting epidemiological data[12]. Our study found that the majority of patients (78.3%) with atypical EH at Songklanagarind Hospital were treated by hysterectomy, whereas non-hysterectomy treatment was more common in those without atypia (90.4%). In general, our findings are comparable to those of the study of Clark et al[13]. In their analyses of 281 patients, Clark et al, reported that 77.1% of patients with atypical EH were treated by hysterectomy, whereas non-hysterectomy treatment was more common (70.2%) in those without atypia. Opting for conservative management in patients with EH depends on several factors, e.g., patient's age, desire for future fertility, medical comorbidities, and presence of cytological atypia. In the present study, the high rate of hysterectomy in atypical EH cases was due to fear of disease progression. This was confirmed by the finding that 30.4% of patients with atypical EH had endometrial carcinomas in subsequent hysterectomy specimens and all of them had been diagnosed with CAH. The overall rate of endometrial carcinoma of the patients with atypical EH in our study was comparable to the rates reported in other studies[14-16]. There is a consensus recommendation that total hysterectomy is the current standard of care for patients with atypical endometrial hyperplasia who are not planning future pregnancies and are fit for surgery, providing definitive assessment of a possible concurrent carcinoma, and effectively treating premalignant lesion[17]. We observed that 18 of 201 patients (9.0%) who received non-hysterectomy treatment had persistent or progressive disease in the median follow-up interval of 56.4 months, which is less than the results of the Clark et al[13] and Horn et al study[18]. It might be due to the differences in numbers in the spectrum that our study revealed a high rate of SH 79.2%, compared to those of the studies of Clark et al and Horn et al, which reported that the majority of patients had CH. In the present study, 2.5% (5/198)

Table 3. Histological outcomes after completion of initial non-hysterectomy treatment according to type of endometrial hyperplasia

Type of EH Total n = 201	Histological outcomes after initial non-hysterectomy treatment					
	Normal, n (%)	SH, n (%)	CH, n (%)	SAH, n (%)	CAH, n (%)	Cancer, n (%)
SH (n = 174)	163 (93.7)	9 (5.2)	1 (0.6)	-	-	1 (0.6)
CH (n = 24)	22 (91.7)	-	2 (8.3)	-	-	-
SAH (n = 2)	2 (100)	-	-	-	-	-
CAH (n = 1)	-	-	-	-	1 (100)	-

EH = endometrial hyperplasia; SH = simple hyperplasia; CH = complex hyperplasia; SAH = simple atypical hyperplasia; CAH = complex atypical hyperplasia

Table 4. Indication for initial hysterectomy

Indication for hysterectomy	SH, n (%)	CH, n (%)	SAH, n (%)	CAH, n (%)
Additional ovarian tumor	8 (57.1)	4 (33.3)	-	-
Fear of disease progression	2 (14.3)	8 (66.7)	2 (100)	16 (100)
Additional uterine mass with persistent AUB	4 (28.6)	-	-	-

SH = simple hyperplasia, CH= complex hyperplasia, SAH = simple atypical hyperplasia
CAH = complex atypical hyperplasia, AUB = abnormal uterine bleeding

of patients with non-atypical endometrial hyperplasia, who received initial non-hysterectomy treatment, had progressive disease to cancer during the follow-up period, which is a comparable finding to those of other studies that have reported a low malignant potential (1%-3%) in non-atypical EH[1]. Progestogen therapy is the most commonly used form of treatment for endometrial hyperplasia without atypia because of its extremely low risk of progression to cancer[1,19-21] and the low proportion of concurrent endometrial cancer[22-24]. The goal of treatment is to prevent progression to cancer and control abnormal uterine bleeding. MPA was the progestogen typically used in the present study because of the highest level of clinical experience at our institution. We observed that almost all of the physicians prescribed MPA 10 mg daily for 12 to 14 days per month over three to six months. There was a series of 376 patients with varying degrees of endometrial hyperplasia treated with a progestin for 7, 10, or 13 days a month for three to six months; complete regression was reported in 81, 98, and 100% of patients, respectively[25]. Progestogen has an inhibitory effect on epithelial proliferation. It acts by reducing estrogenic receptors and increasing estrogen catabolism, stimulating the 17- hydroxysteroid dehydrogenase and sulfotransferase enzymes and thereby diminishing the estrogenic dominant conditions that lead to endometrial hyperplasia which occurs in hyperestrogenism. In the present study, 93% (187/201) of the patients who received non-hysterectomy treatment experienced initial complete regression. Of these, 63.1% had been scheduled for follow-up after initial tissue regression and two-thirds of these received progesterone medication as maintenance treatment. At the present, there is still no consensus on the optimal non-hysterectomy management, the appropriate follow-up surveillance, or maintenance treatment. However, in the present study, 5.9% of the endometrial hyperplasia patients with initial complete regression experienced recurrent endometrial hyperplasia, and 2.1% were found to have endometrial cancer. Therefore, long-term follow-up should be advised. Finally, as a retrospective study, data bias can be possible therefore further prospective study on the most beneficial medication regimens, methods of follow-up evaluation, and the role of maintenance treatment after initial regression should be undertaken.

Conclusion

In the present study, the majority of patients with atypical hyperplasia were managed by initial hysterectomy and the high risk of concomitant endometrial cancer supports this choice of treatment. In the non-atypical EH, the initial non-hysterectomy management was common but EH recurrence and progression to endometrial cancer after the initial regression occurs often. Therefore, long-term follow-up should be advised.

What is already known on this topic?

Endometrial hyperplasia was precursor to endometrial carcinoma. From the previous studies found the progestogen treatments was more efficient but unknown definitive consensus of maintenance medication treatment and follow-up times after completed regression for prevention and detective the recurrence of disease and progression to endometrial carcinoma.

Form two decade studies found that role of conservative treatment in high severe type such as atypical related but in study found high concurrence endometrial carcinoma. This data should be advised for risk and benefit of non-hysterectomy treatment and risk of cancer related especially atypical related type.

What this study adds?

The recommendation of management in suggested that the hysterectomy should be advised and performed in atypical hyperplasia especially in complex atypical hyperplasia due to high incidence of concurrent endometrial carcinoma.

In addition, our study found recurrence in endometrial hyperplasia or progression to endometrial carcinoma after completed regression of disease especially patient with persistence risk factor for endometrial carcinoma. Therefore, long-term follow-up in non-hysterectomy treatment is suggested.

Acknowledgement

The present study was supported by Department of Obstetrics and Gynecology, Faculty of Medicine, Prince of Songkla University.

Potential conflicts of interest

None.

References

1. Kurman RJ, Kaminski PF, Norris HJ. The behavior of endometrial hyperplasia. A long-term study of "untreated" hyperplasia in 170 patients. Cancer 1985; 56: 403-12.
2. Jemal A, Siegel R, Xu J, Ward E. Cancer statistics,

2010. CA Cancer J Clin 2010; 60: 277-300.
3. Attasara P, Buasom R. The most common cancer: female. Hospital-Based Cancer Registry 2011; 27: 2-3.
4. Silverberg SG, Kurman RJ, Nogales F, Mutter GL, Kubik-Huch RA, Tavassoli FA. Tumors of the uterine corpus. In: Tavassoli FA, Devilee P, editors. Pathology and genetics of tumours of the breast and female genital organs. World Health Organization classification of tumours. Lyon, France: IARC Press; 2003: 217-32.
5. Armstrong AJ, Hurd WW, Elguero S, Barker NM, Zanotti KM. Diagnosis and management of endometrial hyperplasia. J Minim Invasive Gynecol 2012; 19: 562-71.
6. Montgomery BE, Daum GS, Dunton CJ. Endometrial hyperplasia: a review. Obstet Gynecol Surv 2004; 59: 368-78.
7. Gunderson CC, Fader AN, Carson KA, Bristow RE. Oncologic and reproductive outcomes with progestin therapy in women with endometrial hyperplasia and grade 1 adenocarcinoma: a systematic review. Gynecol Oncol 2012; 125: 477-82.
8. Gallos ID, Shehmar M, Thangaratinam S, Papapostolou TK, Coomarasamy A, Gupta JK. Oral progestogens vs levonorgestrel-releasing intrauterine system for endometrial hyperplasia: a systematic review and metaanalysis. Am J Obstet Gynecol 2010; 203: 547.e1-10.
9. Gallos ID, Yap J, Rajkhowa M, Luesley DM, Coomarasamy A, Gupta JK. Regression, relapse, and live birth rates with fertility-sparing therapy for endometrial cancer and atypical complex endometrial hyperplasia: a systematic review and metaanalysis. Am J Obstet Gynecol 2012; 207: 266.e1-12.
10. Baker J, Obermair A, Gebski V, Janda M. Efficacy of oral or intrauterine device-delivered progestin in patients with complex endometrial hyperplasia with atypia or early endometrial adenocarcinoma: a meta-analysis and systematic review of the literature. Gynecol Oncol 2012; 125: 263-70.
11. Rattanachaiyanont M, Angsuwathana S, Techatrisak K, Tanmahasamut P, Indhavivadhana S, Leerasiri P. Clinical and pathological responses of progestin therapy for non-atypical endometrial hyperplasia: a prospective study. J Obstet Gynaecol Res 2005; 31: 98-106.
12. Reed SD, Newton KM, Clinton WL, Epplein M, Garcia R, Allison K, et al. Incidence of endometrial hyperplasia. Am J Obstet Gynecol 2009; 200: 678. e1-6.
13. Clark TJ, Neelakantan D, Gupta JK. The management of endometrial hyperplasia: an evaluation of current practice. Eur J Obstet Gynecol Reprod Biol 2006; 125: 259-64.
14. Pennant S, Manek S, Kehoe S. Endometrial atypical hyperplasia and subsequent diagnosis of endometrial cancer: a retrospective audit and literature review. J Obstet Gynaecol 2008; 28: 632-3.
15. Trimble CL, Kauderer J, Zaino R, Silverberg S, Lim PC, Burke JJ, et al. Concurrent endometrial carcinoma in women with a biopsy diagnosis of atypical endometrial hyperplasia: a Gynecologic Oncology Group study. Cancer 2006; 106: 812-9.
16. Antonsen SL, Ulrich L, Hogdall C. Patients with atypical hyperplasia of the endometrium should be treated in oncological centers. Gynecol Oncol 2012; 125: 124-8.
17. Trimble CL, Method M, Leitao M, Lu K, Ioffe O, Hampton M, et al. Management of endometrial precancers. Obstet Gynecol 2012; 120: 1160-75.
18. Horn LC, Schnurrbusch U, Bilek K, Hentschel B, Einenkel J. Risk of progression in complex and atypical endometrial hyperplasia: clinicopathologic analysis in cases with and without progestogen treatment. Int J Gynecol Cancer 2004; 14: 348-53.
19. Lacey JV, Jr., Ioffe OB, Ronnett BM, Rush BB, Richesson DA, Chatterjee N, et al. Endometrial carcinoma risk among women diagnosed with endometrial hyperplasia: the 34-year experience in a large health plan. Br J Cancer 2008; 98: 45-53.
20. Lacey JV Jr, Sherman ME, Rush BB, Ronnett BM, Ioffe OB, Duggan MA, et al. Absolute risk of endometrial carcinoma during 20-year follow-up among women with endometrial hyperplasia. J Clin Oncol 2010; 28: 788-92.
21. Tabata T, Yamawaki T, Yabana T, Ida M, Nishimura K, Nose Y. Natural history of endometrial hyperplasia. Study of 77 patients. Arch Gynecol Obstet 2001; 265: 85-8.
22. Salman MC, Usubutun A, Boynukalin K, Yuce K. Comparison of WHO and endometrial intraepithelial neoplasia classifications in predicting the presence of coexistent malignancy in endometrial hyperplasia. J Gynecol Oncol 2010; 21: 97-101.
23. Yang YF, Liao YY, Peng NF, Li LQ, Xie SR, Wang RB. Prediction of coexistent carcinomas risks by subjective EIN diagnosis and comparison with

WHO classification in endometrial hyperplasias. Pathol Res Pract 2012; 208: 708-12.
24. Chen YL, Cheng WF, Lin MC, Huang CY, Hsieh CY, Chen CA. Concurrent endometrial carcinoma in patients with a curettage diagnosis of endometrial hyperplasia. J Formos Med Assoc 2009; 108: 502-7.
25. Gambrell RD Jr. Progestogens in estrogen-replacement therapy. Clin Obstet Gynecol 1995; 38: 890-901.

การดูแลรักษาและผลการรักษาโรคเยื่อบุมดลูกหนาตัวผิดปกติประสบการณ์ 12 ปี ในโรงพยาบาลสงขลานครินทร์

ณัฐพล ศิริมุสิกะ, กรัณฑรัตน์ ปิยนันท์จรัสศรี, ยุทธศักดิ์ ศุภสินธุ์, วิรัช วุฒิภูมิ, คเณศ กาญจนประดิษฐ์, อลัน กีเตอร์

ภูมิหลัง: *โรคเยื่อบุมดลูกหนาตัวผิดปกติเป็นรอยโรคที่สามารถพัฒนาเป็นมะเร็งของเยื่อบุโพรงมดลูกได้ แต่ในปัจจุบันยังไม่มีข้อตกลงที่เป็นมาตรฐานเกี่ยวกับการดูแลรักษาผู้ป่วยกลุ่มนี้*

วัตถุประสงค์: *ศึกษาแนวทางการดูแลรักษาและผลการรักษาของผู้ป่วยที่ได้รับการวินิจฉัยโรคเยื่อบุมดลูกหนาตัวผิดปกติ*

วัสดุและวิธีการ: *ศึกษาย้อนหลังจากการทบทวนแฟ้มประวัติผู้ป่วยที่ได้รับการวินิจฉัยโรคเยื่อบุมดลูกหนาตัวผิดปกติ จากผลชิ้นเนื้อพยาธิวิทยา ตั้งแต่เดือนมกราคม พ.ศ. 2543 ถึง เดือนธันวาคม พ.ศ. 2555*

ผลการศึกษา: *ผู้ป่วยที่ได้รับการวินิจฉัยโรคเยื่อบุมดลูกหนาตัวผิดปกติจำนวน 297 ราย มี 4 ราย ที่ไม่ได้มาติดตามการรักษาตั้งแต่ต้นและไม่สามารถติดต่อผู้ป่วยได้ เหลือผู้ป่วยเข้ามาในการศึกษา 293 ราย ชนิดของโรคเยื่อบุมดลูกหนาตัวผิดปกติที่พบมากที่สุด คือ simple hyperplasia (SH) ร้อยละ 72.9 รองลงมา คือ complex hyperplasia (CH) ร้อยละ 13.0, complex atypical hyperplasia (CAH) ร้อยละ 5.8, และ simple atypical hyperplasia (SAH) ร้อยละ 2.0 ตามลำดับ ร้อยละ 78 (18/23 ราย) ของผู้ที่ได้รับการวินิจฉัยเป็น atypical hyperplasia ได้รับการรักษาด้วยการตัดมดลูก ซึ่งต่างจากกลุ่มที่เป็น non-atypical hyperplasia ที่พบเพียงร้อยละ 9.6 (26 ใน 270 ราย) ผู้ป่วยที่เป็น atypical endometrial hyperplasia พบว่ามีรอยโรคมะเร็งเยื่อบุโพรงมดลูกร่วมด้วย สูงถึงร้อยละ 30.4 (7 ใน 23 ราย) สำหรับกลุ่มที่ได้รับการรักษาด้วยการไม่ตัดมดลูก พบว่ามีรอยโรคคงเดิมหลังการรักษา ร้อยละ 6 (1 ใน 201 ราย) และรอยโรครุนแรงขึ้น ร้อยละ 1 (2 ใน 201 ราย) ในกลุ่มที่ได้รับการรักษาด้วยการไม่ตัดมดลูกและรอยโรคหายแล้ว พบว่ามีรอยโรคเยื่อบุมดลูกหนาตัวผิดปกติกลับเป็นซ้ำ ร้อยละ 5.9 (11 ใน 187 ราย) และร้อยละ 2.1 (4 ใน 187 ราย) มีรอยโรครุนแรงขึ้นและได้รับการวินิจฉัยเป็นมะเร็งเยื่อบุโพรงมดลูก*

สรุป: *ผู้ป่วยส่วนใหญ่ที่เป็นเยื่อบุมดลูกหนาตัวผิดปกติชนิด atypical hyperplasia ได้รับการตัดมดลูก และพบรอยโรคมะเร็งเยื่อบุโพรงมดลูกร่วมด้วยสูงในผู้ป่วยกลุ่มนี้ สำหรับในกลุ่ม non-atypical hyperplasia ส่วนใหญ่ได้รับการรักษาด้วยการไม่ตัดมดลูก และเมื่อติดตามการรักษาพบรอยโรคกลับเป็นซ้ำ หรือ กลายเป็นมะเร็งเยื่อบุโพรงมดลูกภายหลังได้ ดังนั้นจึงแนะนำให้มีการติดตามผู้ป่วยโรคเยื่อบุมดลูกหนาตัวผิดปกติหลังการรักษาเพื่อเฝ้าระวังภาวะนี้*