International Journal of Clinical Obstetrics and Gynaecology

ISSN (P): 2522-6614
ISSN (E): 2522-6622

www.gynaecologyjournal.com
2018; 2(3): 13-15
Received: 04-03-2018
Accepted: 05-04-2018

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Assessment of cases of endometrial hyperplasia in females

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Abstract
Background: Endometrium is capable of marked hyperplasia as a response to stimulus of prolonged and unopposed oestrogen. The present study was conducted to assess endometrial hyperplasia in females.
Materials & Methods: The present study was conducted on 45 females with history of menorrhagia. General information such as name, age, drug history of estrogen etc. was recorded. Per rectum examination was done in all patients. In all patients, transvaginal ultrasound (TVS) was doneto seeendometrial thickness and ovarian enlargement. After curettage, histopathology of the specimens was done.
Results: Age group 40-45 years had 22 patients, 45-50 had 14, 50-55 had 8 and 55-60 years had 6 patients. The difference was significant (P- 0.01). Type of hyperplasia was cystic hyperplasia (28), adenomatous hyperplasia (10) and atypical hyperplasia (7). The difference was significant (P- 0.05). Endometrial thickness on transvaginal ultrasound (TVS) was 5-6 mm seen in 7 cases, 7-8 mm seen in 12 cases and >10 mm seen in 26 cases. The difference was significant (P<0.05).
Conclusion: Author concluded that endometrial hyperplasia was most commonly seen in age group 40- 45 years. Most common type of hyperplasia was cystic hyperplasia.

Keywords: Cystic hyperplasia, endometrial hyperplasia, transvaginal ultrasound

Introduction
Endometrium is capable of marked hyperplasia as a response to stimulus of prolonged and unopposed oestrogen. Of the two systems used to classify EH, namely, the World Health Organization (WHO) and the endometrial intraepithelial neoplasia (EIN) classification systems, the EIN system is more objective than the WHO system, but the latter is more widely used [1].
Simple or cystic hyperplasia is a benign proliferation of endometrial glands that are irregular and dilated but do not display back to back crowding or cellular atypia. Complex adenomatous hyperplasia is a proliferation of endometrial glands with irregular outline architectural complexity and back to back crowding but no atypia [2].
The endometrioid adenocarcinoma is the most frequent histologic variant, accounting for 58-80% of the cases. Almost all cases of that histologic type are hormone-dependent and associated with obesity, exogeneous use of hormones, and elevated estrogen levels. The lesion is known to arise from an endometrial hyperplasia. The risk of progression of hyperplasia into endometrioid carcinoma is more closely related to the presence of cytologicatypia and to architectural crowding [3].
Epplein *et al*. [4] in their study of risk of complex and atypical endometrial hyperplasia in relation to anthropometric measures and reproductive history found that estrogen-only hormone therapy, which is a type of menopausal hormone therapy (MHT), is a risk factor for EH. Armstrong *et al*. [5] conducted a study of diagnosis and management of endometrial hyperplasia suggested that hormonal therapy is the major risk factor in women leading to endometrial hyperplasia. The present study was conducted to assess endometrial hyperplasia in females.

Materials & Methods
The present study was conducted in the department of Gynecology & Obstetrics. It comprised of 45 females with history of menorrhagia. All were informed regarding the study and written consent was obtained. Ethical clearance was taken before starting the study from the institutional ethical committee.
General information such as name, age, drug history of estrogen etc. was recorded. All were subjected to general physical and pelvic examination. Per rectum examination was done in all patients. Routine investigations such as blood group, Rh-factor, random blood sugar and urine

routine was performed. In all patients, trans vaginal ultrasound (TVS) was done to see for endometrial thickness and ovarian enlargement. Curettage and histopathology of the specimens was done. Results thus obtained were subjected to statistical analysis using chi- square test. P value less than 0.05 was considered significant.

Results

Table I: Age wise distribution of cases

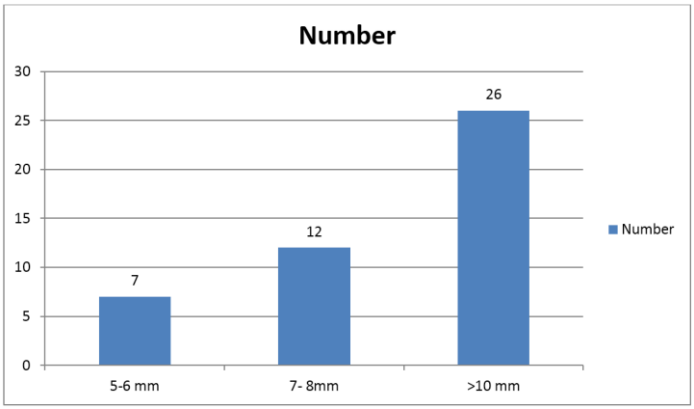
Age group (years)	Number	P value
40-45	22	0.01
45-50	14	
50-55	8	
55-60	6	

Table I shows that age group 40-45 years had 22 patients, 45-50 had 14, 50-55 had 8 and 55-60 years had 6 patients. The difference was significant (P- 0.01).

Table II: Type of endometrial hyperplasia

Type	Number	P value
Cystic hyperplasia	28	0.05
Adenomatous hyperplasia	10	
Atypical hyperplasia	7	
Total	45	

Table II shows that type of hyperplasia was cystic hyperplasia (28), adenomatous hyperplasia (10) and atypical hyperplasia (7). The difference was significant (P- 0.05).



Graph I: Endometrial thickness on transvaginal ultrasound (TVS)

Graph I shows that endometrial thickness on transvaginal ultrasound (TVS) was 5-6 mm seen in 7 cases, 7-8 mm seen in 12 cases and >10 mm seen in 26 cases. The difference was significant (P<0.05).

Discussion

Endometrial hyperplasia is a precursor of endometrial carcinoma. It accounts for 6% of new female cases and 3% of female cancer deaths. It is the most common malignancy of female reproductive tract. Studies have revealed that oestrogentherapy, obesity, diabetes early menarche and late menopause are amongst various risk factors for endometrial hyperplasia. The risk of progression of hyperplasia into endometrial carcinoma is more closely related to the presence of cytologic atypia and to architectural crowding. The morphologic distinction between atypical hyperplasia and well-differentiated carcinoma in an endometrial biopsy or in curetting may be problematic with a marked variation of inter-observer correlation [6].

In present study, 45 females were found to have endometrial hyperplasia. We found that age group 40-45 years had 22 patients, 45-50 had 14, 50-55 had 8 and 55-60 years had 6 patients. This is in agreement with Tavasoli *et al.* [7]

The simple cystic hyperplasia is a benign proliferation with the reported risk 0-1% of progression into carcinoma. In this study, type of hyperplasia was cystic hyperplasia (28), adenomatous hyperplasia (10) and atypical hyperplasia (7). Amera *et al.* [8] in their study found that out of 100 patients, 15patients were found to have endometrial hyperplasia, 10 patients simple cystic hyperplasia, 3patients had adenomotous hyperplasia, 2 patients had atypical hyperplasia, 8 patients had menorrhagia, 1 with polymenorrhagia, and 6 patients with polymenorrhoea. 13 patients were treated medically, 5 patients needed surgical treatment following medical treatment, 2 patients underwent total abdominal hysterectomy (TAH) and bilateral salpingooophorectomy (BSO) who were 51–53 years of age with atypical hyperplasia. Author suggested that endometrial hyperplasia is a pre-malignant condition; if treated in time, incidence can be reduced and early treatment can increase life expectancy and quality in women over age of 45 years.

Horn *et al.* [9] found that 86% of the patients presented with obesity, 23% had an exogeneous use of estrogens. The leading symptom was vaginal bleeding in 65.5% of patients. 91% had complex, 92% had atypical hyperplasia and 89% had endometrioidcarcinoma, representing an overall correlation of 90%. 2% of the cases with complex hyperplasia progressed into carcinoma and 10.5% into atypical hyperplasia. 52% of the atypicalhyperplasias progressed into carcinomas. In the case of progestogen treatment, 61.5% showed remission confirmed by re-curetting, compared with 20.3% of the cases without hormonal treatment.

We subjected patients to TVS and found that endometrial thickness was 5-6 mm seen in 7 cases, 7-8 mm seen in 12 cases and >10 mm seen in 26 cases. This is similar to Campbel *et al.* [10] Jin [11] in her study included 1,868 women with EH and 868 women with EC. The mean ages of the patients were 44.1- 0.4 years for those with EH and 52.7- 0.6 years for those with EC. The EH and EC incidence rates were 3.7%woman-years and 8% woman-years, respectively. The EH and EC incidence rates peaked when the women were in their late forties and fifties, respectively.

In a study by Terakawa *et al.* [12], 51 patients with endometrial hyperplasia were followed for 6 months, with repeated endometrial biopsy monthly for 3 months and at the end of 6 months. In 69% histology became normal. Jalil R [13] correlated the indications of abdominal hysterectomy with histopahological findings in her reports preoperative diagnosis, by D&C of endometrial hyperplasia was14.42%.

In the study of Ferenczy and Gelfand [14] it is stated that 10 out of 20 patients treated with progestogens for 'endometrial hyperplasia with cytologicatypia' had a persistence and five of them recurred. Additionally, five patients developed an adenocarcinoma during a mean follow uptime 5.5 years. Progestogens are the treatment of choice of endometrial hyperplasia because of their inhibitory effect on epithelial proliferation. They act by reducing estrogenic receptors and increasing their catabolism, stimulating the 17-b-hydroxyesteroid dehydrogenase and sulfotransferase enzymes and thereby diminishing the estrogenic dominant conditions that lead to endometrial abnormalities which occur in hyperestrogenism.

Conclusion

Author concluded that endometrial hyperplasia was most commonly seen in age group 40- 45 years. Most common type of hyperplasia was cystic hyperplasia.

References

1. Kurman RJ, Kamiriski PF, Norris HJ. The behavior of endometrial hyperplasia; A long term study of untreated endometrial hyperplasia in 170 patients. Cancer. 1985; 56:403-12.
2. Wentz WB. Progestin therapy in endometrial hyperplasia. Gynaecol Oncol. 1974; 2:362-7.
3. Mishal DR, Droegmuller-W, Merbot-AL. Abdominal uterine bleeding. Corhensive Gynaecology, 2011, 953-64.
4. Fayyaz S, Majeed SS. Audit of Gynaecological hysterectomies. J Postgrad Med Inst. 2001; 15(2):208–12.
5. Terakawa N, Kigawa J, Taketani Y, Yoshikawa H, Yajima A, Noda K *et al.* The behavior of endometrial hyperplasia: aprospective study. J Obstet Gynaecol Res. 1997; 23(3):223-30.
6. Menwissen JH *et al*. Endometrial Biopsy the female patient 1993; 4:19–23.
7. Tavasoli, Ben Yehuda OM, Kim YB, Leuchter RS. Does Hysteroscopy improve upon the sensitivity of dilatation and curettage in the diagnosis of endometrial hyperplasia or carcinoma? Gynaecol Oncol 1998; 68(1):4-7.
8. Amera, Tan KT, Pang MW. Ho TH. Endometrial hyperplasia and the risk of endometrial carcinoma. Singapore Med J. 1997; 38(1):11–5.
9. Horn, Reich O, Tamussino K, Bader AA, Pieber D, Schöll W *et al.* Concomitant endometrial Hyperplasia in patients with endometrial carcinoma. Gynaecol Oncol. 1998; 69(1):64-8.
10. Campbel, Colacurci N, De Placido G, Mollo A, Perino A, Cittadini E. Short term use of Goserlin Depot in the treatment ofdys functional uterine bleeding. Clin Exp Obstet Gynecol. 1995; 22:212-9.
11. Jin RJ. Not so benign endometrial hyperplasia: endometrial cancer after endometrial ablation. J Am Assoc Gynaecol Laprarosc. 1997; 4(4):507-11.
12. Terakawa, Kaku T, Tsukamoto N, Hachisuga T *et al*. Endometrial carcinoma associated with hyperplasia. Gynecol Oncol. 1996; 60:22-5.
13. Jalil, Skov BG, Broholm H, Engel U *et al.* Comparison of there producibility of the WHO classifications of 1975 and 1994 of endometrial hyperplasia. Int J Gynecol Pathol. 1997; 16:33-7.
14. Ferenczy and Gelfand. Endometrioidneoplasia retrogressive terminology. AmJ Surg Pathol. 2000; 24:754-5.