Special Article

Adenomyosis: Problems in Pathological Diagnosis and Clinical Impact

Chutima Chavanisakun MD[1], Siriwan Tangjigamol MD[2]

[1] Department of Pathology, Faculty of Medicine Vajira Hospital, Navamindradhiraj University, Bangkok, Thailand
[2] Department of Obstetrics and Gynecology, Faculty of Medicine Vajira Hospital, Navamindradhiraj University, Bangkok, Thailand

Adenomyosis is a common benign gynecologic lesion which is characterized by abnormal endometrial mucosa in the muscular wall of the uterus. Pathological diagnosis of this condition is generally straightforward but there are few mimicking lesions that a pathologist may be reluctant to make a diagnosis, such as adenomyosis with atrophic and fibrotic stroma vs well differentiated endometrioid adenocarcinoma with myometrial invasion, adenomyosis with vascular involvement vs. low-grade stromal sarcoma, and cancer arising from adenomyosis or involving adenomyosis. The gynecologic pathologists, gynecologists and gynecologic oncologists should be aware of these mimicking lesions or conditions which may have some problems in diagnosis including the features that have clinical impact upon stage of cancer and treatment.

Keywords: Adenomyosis, Endometrioid adenocarcinoma, Endometrial stromal sarcoma

J Med Assoc Thai 2018; 101 (Suppl. 8): S211-S217
Website: http://www.jmatonline.com

Adenomyosis is a benign gynecologic condition which can be found in approximately 20% of female population, being most common in peri-menopausal or multiparous women[1-4]. The pathogenesis of the adenomyosis is still uncertain. Various theories had been described[5-7]. The first theory is an origination from the pleuripotential Mullerian remnant. The second theory is an invagination of the basal layer of endometrium to the myometrial lymphatic system. The third possibility is a deportation of bone marrow stem cell via vascular system to the myometirum. The last theory is an iatrogenic cause during a surgical procedure which may introduce an endometrial tissue into myometrium.

Adenomyosis, although benign, has many clinical considerations. It frequently causes symptoms of menorrhagia, pelvic pain and dysmenorrhea which may severely debilitate women affecting their work- or family-lives. Clinical diagnosis of adenomyosis is generally made by history, physical including pelvic examination and imaging studies e.g. ultrasound, computerized tomography scan or magnetic resonance imaging. However, other pelvic pathologies especially uterine tumors may have similar presentations and findings. Hence, a definite diagnosis is usually achieved by a pathologic examination of resected myometrial lesion (myomectomy) or uterus (hysterectomy) which is a more common surgical treatment especially in pre- and peri-menopausal woman who have completed family life[8].

Pathologic feature of adenomyosis is a presence of both glandular and stromal endometrial tissues in the muscular wall of the uterus. These displaced endometrial tissues break down and bleed during each menstrual cycle causing fibrotic reaction and progressive thickening of myometrial wall. The pathological diagnosis in most cases is straightforward. However, there are a few instances that a pathological diagnosis can be difficult. This review described some features of adenomyosis which has equivocal features and needed to be differentiated from other lesions especially malignant conditions.

Correspondence to:
Chavanisakun C. Department of Pathology, Faculty of Medicine Vajira Hospital, Navamindradhiraj University, Bangkok 10330, Thailand.
Phone: +66-81-5732313
E-mail: Chutima.c@nmu.ac.th

Adenomyosis with atrophic and fibrotic stroma

Adenomyosis with atrophic and fibrotic stromal component is usually found in postmenopausal

How to cite this article: Chavanisakun C, Tangjigamol S. Adenomyosis: Problems in Pathological Diagnosis and Clinical Impact. J Med Assoc Thai 2018;101;Suppl.8: S211-S217.

woman especially in breast cancer patient who has had tamoxifen[9]. This feature is important particularly in patients with endometrial cancer which is frequently found after breast cancer[10] when presence of scattered endometrial glands in the myometrium with inconspicuous stromal component may raise a possibility of endometrioid adenocarcinoma invading into myometrium. Useful features to differentiate adenomyosis from malignant tumor are atrophic change of endometrial gland (Figure 1A), lack of tissue reaction, and presence of typical adenomyosis in the remaining tissue[11,12]. In case of tamoxifen use, endometrial gland usually have cystic dilatation (Figure 1B) and tubal, mucinous or oxyphilic metaplasia of glandular epithelium[9]. In a difficult case with equivocal features, immunohistochemical study may aid in diagnosis. CD10 which is generally positive in stromal cell is not helpful because the stromal cells can be present in both adenomyosis and invasive foci of endometrioid carcinoma[13]. Interferon-induce transmembrane protein 1 (IFITM1) is more useful that it would be positive in stromal cells in adenomyosis but not in foci of cancer with myometrial invasion[14].

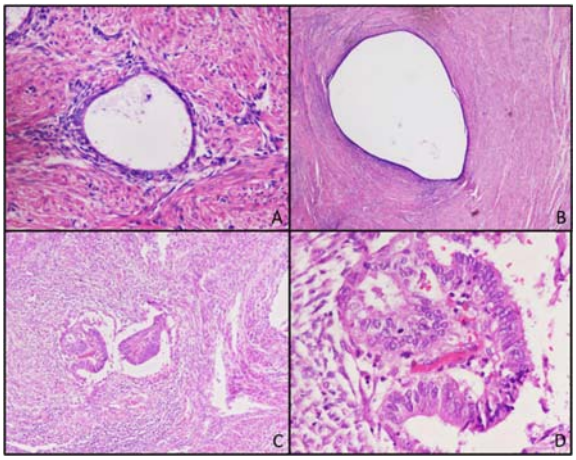

Figure 1. Adenomyosis in postmenopausal woman with stromal atrophy and fibrosis (A; 400x). Endometrial gland dilation in a patient with breast cancer after tamoxifen use (B; 100x). Myoinvasion of endometrioid adenocarcinoma with stromal desmoplasia (C; 200x). Nuclear atypia of the endometrioid adenocarcinoma (D; 400x).

Adenomyosis with vascular involvement

Adenomyosis in the myometrial vascular channels is relatively common. This histomorphology of vascular dissemination may support one of the theories of pathogenesis of endometriosis as described earlier[15-17].

The prevalence of vascular involvement in adenomyosis from previous reports ranged from 12% to 18%[18,19]. Features of vascular involvement of adenomyosis vary in many aspects in terms of: number of vascular involvement which may be found as a single, a few or multiple vessels involvement, location of adenomyotic foci which may attach to the vessel wall or lay free within the vascular lumen (Figure 2A), or the components which may have only stroma component (two third of cases) (Figure 2B) or both components (Figure 2C)[19].

The two conditions can be easily differentiated upon a gross inspection and histologic features. Adenomyosis generally shows coarse trabeculation without a definite mass whereas the endometrial stromal sarcoma usually presents with a polypoid mass. However, an adenomyosis can present with a mass formation, so called 'adenomyoma', when the histologic examination is required for a definite diagnosis.

Presence of endometrial gland with stromal component (usually with atrophic change) and absence of features which are usually found in endometrial stromal sarcoma e.g. tongue-like growth pattern, mitotic activity and extrauterine extension are features of typical adenomyosis[19]. However, when only

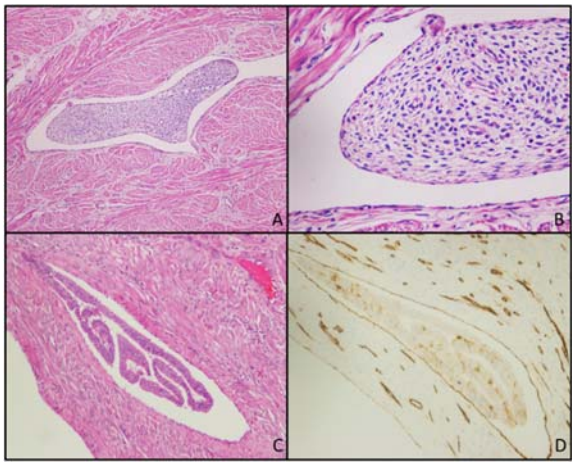

Figure 2. Adenomyosis with vascular involvement. Adenomyotic focus lay free within the vascular lumen (A; 200x). Only stromal component is shown (B; 400x). Glandular component with minimal stromal cell (C; 200x). Vascular channel highlighted by CD34 stain (D; 200x).

endometrial stroma tissue is present in the vessels especially in multiple foci, it might mislead to a diagnosis of low-grade endometrial stromal sarcoma [ESS]. On the contrary, some endometrial stromal sarcoma occasionally contain a large number of endometrioid-type glands which may mislead to a diagnosis of adenomyosis[19]. In difficult case, additional sections may be required for a definite diagnosis.

Histologic features which help to differentiate adenomyosis with vascular involvement from low-grade endometrial stromal sarcoma are summarized in Table 1.

Co-existing adenomyosis in endometrial adeno-carcinoma

Adenomyosis was reported in a hysterectomy specimen of endometrioid endometrial carcinoma in approximately 16% to 34%[20-26]. This wide range of prevalence may partly depend on the extent of each lesion and an awareness of a pathologist in reporting this benign lesion in a patient with malignant condition. Previous studies reported conflicting data regarding a prognosis of coexistence of adenomyosis and endometrial carcinoma. Some studies found that patients with concomitant adenomyosis and endometrioid endometrial carcinoma had poorer prognosis than those having cancer alone. These studies proposed that adenomyotic foci were precursor of endometrial carcinoma and these adenomyotic foci, subsequently in the course after cancer development, increased contact area for myometrial invasion as evidenced by an increased frequency for deep myometrial invasion (outer-half myometrial invasion) in stage I endometrioid adenocarcinoma[20,27,28]. However, this was not translated to a poorer survival[27]. This was explained that the extension of carcinoma from foci of adenomyosis to adjacent deep myometrium (outer-half myometrium) was actually an early myometrial invasion, so did not impact survival.

Other studies, with larger numbers of patients, reported a more favorable prognosis of the patient with coexistence of adenomyosis in endometrial carcinoma because it was commonly associated with early stage, smaller tumor size, lower grade tumor, superficial myometrial invasion, less lymphovascular space and lymph node involvement[21-25,29,30]. These more favorable pathologic features found in adenomyosis were explained by a few mechanisms. Rapid proliferation of endometrial stroma under stimulation of estrogen or inflammatory cytokines serves as a mechanical block of endometrial cancer invasion in the myometrium[30-32]. The other processes are via an increased secretion of several cytokines, such as, interferon [IFN]-α, IFN-γ, tumor necrosis factor [TNF]-α, and interleukin [IL]-10 resulting in anti-tumor progression[30-32]. High expression of ERβ which functions as tumor suppressor and low expression of glycodelin which has angiogenic role in tumorigenesis were also observed in endometrioid endometrial carcinoma with adenomyosis[30].

Endometrial carcinoma arising in adenomyosis

One condition of adenomyosis which has clinical merits is an endometrial carcinoma arising in adenomyosis. This entity is rarely found, being reported in about 1% in woman with endometrial carcinoma[33].

A mechanism for malignant transformation of adenomyosis is unclear. Few studies proposed a loss of heterozygosity in the DNA mismatch repair gene (hMSH2, hMLH1, p16 and GALT) as a possible

Table 1. Histologic features of adenomyosis with vascular involvement and low-grade endometrial stromal sarcoma

Pathologic features	Adenomyosis with vascular involvement	Endometrial stromal sarcoma
Gross feature	No mass formation (except adenomyoma)	Tumor mass
Microscopic features		
Endometrial gland	Present	Rarely present
Stroma	Atrophic change	Active proliferation
Growth pattern	Localized	Expansile tongue-like
Mitotic activity	Absent or low	Frequent
Lymphovascular involvement	Less frequent of multiple vessels involvement	Frequent
Extrauterine extension	None	Possible
Other areas	Typical adenomyosis elsewhere in the myometrium	None

(Modified from Meenakshi M, McCluggage WG. Vascular involvement in adenomyosis: report of a large series of a common phenomenon with observations on the pathogenesis of adenomyosis. Int J Gynecol Pathol 2010;29:117-21.)

etiology[34,35]. A more explicit example is carcinoma arising from ovarian endometriosis which is more frequently found. Based on molecular genetic studies, carcinoma arising in ovarian endometriosis was categorized in two groups: type I and type II. Type I which is associated with a benign precursor lesion and low-grade cancer (endometrioid, clear cell, and low-grade serous cancers) reveals KRAS, phosphatase and tensin homologue [PTEN], and phosphatidylinositol-4 5-bisphosphate 3-kinase catalytic subunit alpha [PIK3-CA] mutations[36]. On the other hand, type II which is associated with high-grade cancer shows TP53 and HER2 mutations[37,38]. Another study showed high frequency of genetic mutations in PTEN, KRAS and AT-rich interactive domain 1A gene [ARID1A] among endometriosis-associated ovarian cancer (of both endometrioid and clear cell histologies)[39]. There may be a possibility of genetic mutations in adenocarcinoma arising from adenomyosis[32,40,41]. However, limited data is available and more researches on this issue are needed.

Malignant transformation of adenomyosis is mostly found in the postmenopausal woman and is extremely rare in premenopausal women with normal endometrium[42]. History of tamoxifen was reported[40]. Clinical manifestation includes abnormal vaginal bleeding, slight fever and weight loss.

The most common histological subtypes of cancer arising in adenomyosis were endometrioid type and, to a lesser degree, serous, clear cell and poorly differentiated adenocarcinoma[32,40,41]. Histomorpho logy may show transitions between endometrial epithelium of adenomyosis, borderline malignancy (noninvasive), and invasive carcinoma. The diagnostic criteria of endometrial carcinoma arising in adenomyosis are: 1) the carcinoma must not be present in the normally situated endometrium or elsewhere in the pelvis; 2) the carcinoma must be seen to arise from the epithelium within the adenomyosis rather than invasion from another source; and 3) endometrial (adenomyotic) stromal cells must be seen to support a diagnosis of adenomyosis[43].

Several studies reported cancer arising in adenomyosis had poor prognosis with aggressive features, such as, high-grade tumor, deep myometrial invasion, advance stage and positive nodal metastasis[32,41,44-46]. This might be due to a direct invasion of cancer deep into myometrium or vascular channels in the myometrium from an absence of anatomic barrier in the basal layer of endometrium[32,47].

Endometrial carcinoma with adenomyosis involvement

It is generally accepted that endometrial carcinoma with adenomyosis involvement has no influence on staging and prognosis[42,43,45]. The problems of adenomyotic foci involved with cancer lie on the pathologic diagnosis and the measurement of depth of invasion.

Diagnostic features of adenomyosis with cancer involvement but without cancer invasion are smooth rounded contours and presence of: benign glands with intervening or surrounding endometrial stroma and absence of desmoplasia or inflammatory infiltration[23-25]. Myometrial invasion from adenomyosis has features of: jagged infiltrative contour, neoplastic epithelial cells surrounded by myometrium without intervening endometrial stroma, and desmoplastic stromal reaction[48].

In general, the depth of myometrial invasion is defined by the distance between endomyometrial junction and the deepest part of the invasive tumor clusters. Measuring the depth of cancer invasion into myometrium from adenomyotic foci is a challenge especially when invasive tumor cluster was found only in adenomyotic focus. Although a definite rule of measurement the depth of myometrial invasion of cancer in adenomyosis is still controversy. The criteria adopted by Ali et al[48] are generally used and has been included into College of American Pathologists [CAP] guideline protocol in 2016[49]. It was advised that the depth of invasion is the distance between the interface of adenomyosis and the farthest point of cancer away from adenomyotic focus[49]. Therefore, myoinvasion of the cancer from adenomyotic foci that situated in deep myometrium is not always considered as deep myometrial invasion. However, further study for this measurement and prognostic significance is needed for more information.

Conclusion

Adenomyosis is a common benign gynecologic lesion which is characterized by abnormal endometrial mucosa in the muscular wall of the uterus. Pathological diagnosis of this condition is generally straightforward but there are few mimicking lesions that a pathologist may be reluctant to make a diagnosis, such as, adenomyosis with atrophic and fibrotic stroma vs. well differentiated endometrioid adenocarcinoma with myometrial invasion, adenomyosis with vascular involvement vs low-grade stromal sarcoma, and cancer arising from adenomyosis or involving adenomyosis.

The gynecologic pathologists, gynecologists and gynecologic oncologists should be aware of these mimicking lesions or conditions which may have some problems in diagnosis including the features that have clinical impact upon stage of cancer and treatment.

What is already known on this topic?

Adenomyosis is a common benign gynecologic lesion. Pathologic feature of adenomyosis is a presence of both glandular and stromal endometrial tissues in the muscular wall of the uterus. These displaced endometrial tissues break down and bleed during each menstrual cycle causing fibrotic reaction and progressive thickening of myometrial wall. Pathological diagnosis of this condition is generally straightforward but there are few mimicking lesions and conditions that a pathologist may be reluctant to make a diagnosis.

What this study adds?

This review presented some mimicking lesions and some problematic conditions with pathological clues to make a diagnosis. These mimicking lesions and conditions include adenomyosis with atrophic and fibrotic stroma vs well differentiated endometrioid adenocarcinoma with myometrial invasion, adenomyosis with vascular involvement vs. low-grade stromal sarcoma, and cancer arising from adenomyosis or involving adenomyosis.

Potential conflicts of interest

The authors declare no conflict of interest.

References

1. Dietrich JE. An update on adenomyosis in the adolescent. Curr Opin Obstet Gynecol 2010;22:388-92.
2. Benagiano G, Brosens I. History of adenomyosis. Best Pract Res Clin Obstet Gynaecol 2006;20:449-63.
3. Benagiano G, Brosens I, Carrara S. Adenomyosis: new knowledge is generating new treatment strategies. Womens Health (Lond) 2009;5:297-311.
4. Benagiano G, Habiba M, Brosens I. The pathophysiology of uterine adenomyosis: an update. Fertil Steril 2012;98:572-9.
5. Garcia L, Isaacson K. Adenomyosis: review of the literature. J Minim Invasive Gynecol 2011;18:428-37.
6. Ferenczy A. Pathophysiology of adenomyosis. Hum Reprod Update 1998;4:312-22.
7. Sasson IE, Taylor HS. Stem cells and the pathogenesis of endometriosis. Ann N Y Acad Sci 2008;1127:106-15.
8. Taran FA, Stewart EA, Brucker S. Adenomyosis: Epidemiology, risk factors, clinical phenotype and surgical and interventional alternatives to hysterectomy. Geburtshilfe Frauenheilkd 2013;73:924-31.
9. McCluggage WG, Desai V, Manek S. Tamoxifen-associated postmenopausal adenomyosis exhibits stromal fibrosis, glandular dilatation and epithelial metaplasias. Histopathology 2000;37:340-6.
10. Tangjitgamol S, Khunnarong J, Srijaipracharoen S. Synchronous and metachronous malignancy in endometrial cancer patients treated in a tertiary care center of Thailand. J Gynecol Oncol 2015;26:293-302.
11. Silverberg SG. Problems in the differential diagnosis of endometrial hyperplasia and carcinoma. Mod Pathol 2000;13:309-27.
12. Reichert RA. Pathology of uterine corpus. In: Reichert RA, editor. Diagnostic gynecologic and obstetric pathology: An atlas and text. Philadelphia, PA: Lippincott Williams & Wilkins; 2012. p. 168-306.
13. Srodon M, Klein WM, Kurman RJ. CD10 imunostaining does not distinguish endometrial carcinoma invading myometrium from carcinoma involving adenomyosis. Am J Surg Pathol 2003;27:786-9.
14. Busca A, Djordjevic B, Giassi A, Parra-Herran C. IFITM1 Is Superior to CD10 as a marker of endometrial stroma in the evaluation of myometrial invasion by endometrioid adenocarcinoma. Am J Clin Pathol 2016;145:486-96.
15. Machado DE, Abrao MS, Berardo PT, Takiya CM, Nasciutti LE. Vascular density and distribution of vascular endothelial growth factor (VEGF) and its receptor VEGFR-2 (Flk-1) are significantly higher in patients with deeply infiltrating endometriosis affecting the rectum. Fertil Steril 2008;90:148-55.
16. Abrao MS, Podgaec S, Dias JA Jr, Averbach M, Garry R, Ferraz Silva LF, et al. Deeply infiltrating endometriosis affecting the rectum and lymph nodes. Fertil Steril 2006;86:543-7.
17. Noel JC, Chapron C, Fayt I, Anaf V. Lymph node involvement and lymphovascular invasion in deep infiltrating rectosigmoid endometriosis. Fertil Steril 2008;89:1069-72.
18. Sahin AA, Silva EG, Landon G, Ordonez NG, Gershenson DM. Endometrial tissue in myometrial

vessels not associated with menstruation. Int J Gynecol Pathol 1989;8:139-46.
19. Meenakshi M, McCluggage WG. Vascular involvement in adenomyosis: report of a large series of a common phenomenon with observations on the pathogenesis of adenomyosis. Int J Gynecol Pathol 2010;29:117-21.
20. Ismiil N, Rasty G, Ghorab Z, Nofech-Mozes S, Bernardini M, Ackerman I, et al. Adenomyosis involved by endometrial adenocarcinoma is a significant risk factor for deep myometrial invasion. Ann Diagn Pathol 2007;11:252-7.
21. Kucera E, Hejda V, Dankovcik R, Valha P, Dudas M, Feyereisl J. Malignant changes in adenomyosis in patients with endometrioid adenocarcinoma. Eur J Gynaecol Oncol 2011;32:182-4.
22. Bergeron C, Amant F, Ferenczy A. Pathology and physiopathology of adenomyosis. Best Pract Res Clin Obstet Gynaecol 2006;20:511-21.
23. Mittal KR, Barwick KW. Endometrial adenocarcinoma involving adenomyosis without true myometrial invasion is characterized by frequent preceding estrogen therapy, low histologic grades, and excellent prognosis. Gynecol Oncol 1993;49:197-201.
24. Jacques SM, Lawrence WD. Endometrial adenocarcinoma with variable-level myometrial involvement limited to adenomyosis: a clinicopathologic study of 23 cases. Gynecol Oncol 1990;37:401-7.
25. Hernandez E, Woodruff JD. Endometrial adenocarcinoma arising in adenomyosis. Am J Obstet Gynecol 1980;138:827-32.
26. Khunnarong J, Tangjitgamol S, Srijaipracharoen S. Other gynecologic pathology in endometrial cancer patients. Asian Pac J Cancer Prev 2016;17:713-7.
27. Taneichi A, Fujiwara H, Takahashi Y, Takei Y, Machida S, Saga Y, et al. Influences of uterine adenomyosis on muscle invasion and prognosis of endometrioid adenocarcinoma. Int J Gynecol Cancer 2014;24:1429-33.
28. Ismiil ND, Rasty G, Ghorab Z, Nofech-Mozes S, Bernardini M, Thomas G, et al. Adenomyosis is associated with myometrial invasion by FIGO 1 endometrial adenocarcinoma. Int J Gynecol Pathol 2007;26:278-83.
29. Gizzo S, Patrelli TS, Dall'asta A, DI Gangi S, Giordano G, Migliavacca C, et al. Coexistence of adenomyosis and endometrioid endometrial cancer: Role in surgical guidance and prognosis estimation. Oncol Lett 2016;11:1213-9.
30. Hertlein L, Rath J, Zeder-Goss C, Furst S, Bayer D, Trillsch F, et al. Coexistence of adenomyosis uteri and endometrial cancer is associated with an improved prognosis compared with endometrial cancer only. Oncol Lett 2017;14:3302-8.
31. Matsuo K, Cahoon SS, Gualtieri M, Scannell CA, Jung CE, Takano T, et al. Significance of adenomyosis on tumor progression and survival outcome of endometrial cancer. Ann Surg Oncol 2014;21:4246-55.
32. Machida H, Maeda M, Cahoon SS, Scannell CA, Garcia-Sayre J, Roman LD, et al. Endometrial cancer arising in adenomyosis versus endometrial cancer coexisting with adenomyosis: are these two different entities? Arch Gynecol Obstet 2017;295:1459-68.
33. Sampson JA. Endometrial carcinoma of the ovary, arising in endometrial tissue in that organ. Arch Surg 1925;10:1-72.
34. Martini M, Ciccarone M, Garganese G, Maggiore C, Evangelista A, Rahimi S, et al. Possible involvement of hMLH1, p16(INK4a) and PTEN in the malignant transformation of endometriosis. Int J Cancer 2002;102:398-406.
35. Koike N, Tsunemi T, Uekuri C, Akasaka J, Ito F, Shigemitsu A, et al. Pathogenesis and malignant transformation of adenomyosis (review). Oncol Rep 2013;29:861-7.
36. Pearce CL, Templeman C, Rossing MA, Lee A, Near AM, Webb PM, et al. Association between endometriosis and risk of histological subtypes of ovarian cancer: a pooled analysis of case-control studies. Lancet Oncol 2012;13:385-94.
37. Kurman RJ, Shih I. Molecular pathogenesis and extraovarian origin of epithelial ovarian cancer shifting the paradigm. Hum Pathol 2011;42:918-31.
38. Kurman RJ, Shih I. The origin and pathogenesis of epithelial ovarian cancer: a proposed unifying theory. Am J Surg Pathol 2010;34:433-43.
39. Wiegand KC, Shah SP, Al Agha OM, Zhao Y, Tse K, Zeng T, et al. ARID1A mutations in endometriosis-associated ovarian carcinomas. N Engl J Med 2010;363:1532-43.
40. Izadi-Mood N, Samadi N, Sarmadi S, Eftekhar Z. Papillary serous carcinoma arising from adenomyosis presenting as intramural leiomyoma. Arch Iran Med 2007;10:258-60.
41. Abushahin N, Zhang T, Chiang S, Zhang X, Hatch K, Zheng W. Serous endometrial

intraepithelial carcinoma arising in adenomyosis: a report of 5 cases. Int J Gynecol Pathol 2011;30:271-81.
42. Kazandi M, Zeybek B, Terek MC, Zekioglu O, Ozdemir N, Oztekin K. Grade 2 endometrioid adenocarcinoma arising from adenomyosis of the uterus: report of a case. Eur J Gynaecol Oncol 2010;31:719-21.
43. Colman HI, Rosenthal AH. Carcinoma developing in areas of adenomyosis. Obstet Gynecol 1959;14:342-8.
44. Puppa G, Shozu M, Perin T, Nomura K, Gloghini A, Campagnutta E, et al. Small primary adenocarcinoma in adenomyosis with nodal metastasis: a case report. BMC Cancer 2007;7:103.
45. Matsuo K, Gualtieri MR, Cahoon SS, Toboni MD, Machida H, Moeini A, et al. Contributing factors for menopausal symptoms after surgical staging for endometrial cancer. Menopause 2016;23:535-43.
46. Mori M, Furusawa A, Kino N, Uno M, Ozaki Y, Yasugi T. Rare case of endometrioid adenocarcinoma arising from cystic adenomyosis. J Obstet Gynaecol Res 2015;41:324-8.
47. Mehasseb MK, Bell SC, Brown L, Pringle JH, Habiba M. Phenotypic characterisation of the inner and outer myometrium in normal and adenomyotic uteri. Gynecol Obstet Invest 2011;71:217-24.
48. Ali A, Black D, Soslow RA. Difficulties in assessing the depth of myometrial invasion in endometrial carcinoma. Int J Gynecol Pathol 2007;26:115-23.
49. Movahedi-Lankarani S, Gilks CB, Soslow R, Otis CN, Oliva E; College of American Pathologists. Protocol for the examination of specimens from patients with carcinoma of the endometrium [Internet]. 2016 [cited 2018 Jul 13]. Available from: http://www.cap.org/ShowProperty?nodePath=/UCMCon/Contribution%20Folders/WebContent/pdf/cp-endometrium-16protocol-3300.pdf.