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Coexistence and Correlation of Adenomyosis, DIE and Ovarian Endometrioma Size: Insights from MR

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Abstract

Introduction: The coexistence of ovarian endometriomas, adenomyosis, and deep infiltrating endometriosis (DIE) is frequently observed in endometriosis. This study aimed to investigate the relationship between endometrioma size and the presence of adenomyosis and DIE on pelvic MRI, as well as the association between adenomyosis and DIE, and to assess whether endometrioma size could serve as a marker for these conditions.

Methods: This retrospective single-center study included 118 women with ovarian endometriomas who underwent pelvic MRI between January 2019-January 2023. The presence of adenomyosis and DIE was recorded based on standardized MRI criteria. The largest endometrioma diameter was measured for each patient. Statistical analyses included Kruskal–Wallis tests for group comparisons, point-biserial correlations to assess the relationship between endometrioma size and adenomyosis/DIE, and Chi-square tests to evaluate the association between adenomyosis and DIE.

Results: Ovarian endometrioma size was not significantly associated with adenomyosis or DIE. Median endometrioma diameters did not differ significantly between patients with and without adenomyosis or DIE (all $p>0.05$). In contrast, there was a strong association between adenomyosis and DIE: adenomyosis was present in 27 of 74 women with DIE (36.5%) compared with only 4 of 44 women without DIE (9.1%). Chi-square testing confirmed this relationship ($p=0.001$), and symmetric measures indicated a moderate positive correlation ($r=0.301$, $p=0.001$).

Conclusion: Ovarian endometrioma size does not appear to predict adenomyosis or DIE; however, the coexistence of adenomyosis and DIE is significant and clinically relevant. Identification of adenomyosis on MRI should prompt careful evaluation for concurrent DIE, as this combination may reflect a more severe endometriosis phenotype.

Key words: Endometriosis; deep infiltrating endometriosis; adenomyosis; ovarian endometrioma; magnetic resonance imaging; pelvic imaging.

Introduction

The presence of endometrial glands and stroma outside the uterine cavity is a definition of endometriosis, which is a chronic gynecologic condition linked to estrogen. Ovarian endometrioma (OE), deep infiltrating endometriosis (DIE), and superficial peritoneal endometriosis are the three main subtypes of the disease (1). DIE is characterized as the presence of an endometriosis implant penetrating more than 5 mm below the peritoneal surface. It is regarded as the most aggressive subtype of endometriosis and often affects the broad ligaments, rectovaginal septum, torus uteri, bladder, rectum or sigmoid colon, and uterosacral ligaments (2). In previous studies, fertility, persistent pelvic pain, and significant surgical complexity are all found to be closely linked to DIE, which frequently necessitates a multidisciplinary approach (3, 4). Up to 50% of women with DIE have ovarian endometrioma, which is the most prevalent type of endometriosis. According to earlier research, endometrioma coexistence is indicative of more severe and widespread pelvic disease, with a higher incidence of intestinal and posterior compartment involvement (2, 5, 6). While endometrioma size has been shown as a potential indicator of disease burden, several reports have demonstrated that ovarian endometrioma diameter alone does not reliably predict DIE severity, suggesting that other possible factors must be considered (3, 8). The presence of ectopic endometrial tissue within the myometrium characterizes adenomyosis, another hormone-dependent condition that causes myometrial cysts, thickening of the junctional zone, and uterine enlargement. Most authors agree that junctional zone thickness ≥12 mm is

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indicative of adenomyosis (7). Its prevalence is unclear, ranging from 15% to 20%. Although adenomyosis and endometriosis, especially DIE, were once thought to be distinct conditions, new research shows a strong and intricate relationship between them (1, 9, 10). Up to 50% of women with DIE have adenomyosis, according to magnetic resonance imaging (MRI)-based studies, and this condition has been associated with worse surgical outcomes, refractory pelvic pain, and more severe symptoms (8). Despite these correlations, little is known about how adenomyosis, DIE, and ovarian endometrioma interact. Few studies have examined whether measurable characteristics, like endometrioma size, correlate with the likelihood of adenomyosis or DIE, as well as the correlation between adenomyosis and DIE. The majority of research has concentrated on either the coexistence rates or the effect of adenomyosis on surgical complications. Clarifying these connections could improve preoperative risk assessment and enhance counseling, imaging interpretation, and surgical planning. The purpose of this study was to assess the correlation between DIE, adenomyosis, and endometrioma size on pelvic MRI. In particular, we evaluated whether adenomyosis and DIE show a significant correlation and whether endometrioma size predicts the presence of either condition. This study advances our understanding of the overlapping pathophysiology of these conditions by combining thorough MRI assessment. It may also help radiologists and clinicians to develop a more thorough evaluation for endometriosis patients.

Material and Methods

This retrospective study was approved by our institutional Biomedical Research Ethics Committee (approval number: 2024.053.IRB2.026, date: 19.03.2024) and conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent for imaging and the anonymized use of data for research purposes was obtained from all patients at the time of their examinations as per our institution policy. The institutional imaging archive was reviewed to identify patients who underwent pelvic MRI between January 2019 and January 2023. All patients with diagnosis of ovarian endometrioma based radiological data were initially considered for inclusion. Patients with suboptimal MRI examinations that prevented implant assessment and patients without clinical examination or transvaginal ultrasound documentation were excluded. All MRI examinations were carried out using a specific pelvic phased-array coil on a 1.5T or 3T scanner (MAGNETOM Aera or Skyra, *Siemens Healthineers, Erlangen, Germany*). In order to minimize bowel motion artifacts, anti-peristaltic medications were administered, the patient's bladder was emptied about half an hour before scanning. When tolerated by the patient, vaginal gel was applied to enhance visualization of the vaginal fornices and implants; however, it was not used in all cases. The MRI protocol included small FOV axial, sagittal, and oblique coronal T2-weighted images, axial T2-weighted fat-saturated image, axial and sagittal T1-weighted fat-saturated and as well as T1-weighted axial non-fat saturated images, post-contrast T1-weighted fat-saturated sequences in both axial and sagittal planes. In order to rule out any potential pelvic malignancy, we regularly used diffusion-weighted imaging in our pelvic MRI examination. Image interpretation was performed by two radiologists with five and twenty years of experience in diagnostic radiology. Both readers were blinded to clinical data and reached a consensus for all evaluations. Ovarian endometrioma was diagnosed when a cystic ovarian lesion demonstrated homogeneous high signal intensity on T1-weighted images with no signal loss on fat-saturated T1-weighted sequences and mixed or low signal intensity (T2 shading) on T2-weighted images, consistent with blood-containing cysts. For each patient, the largest endometrioma was identified, and its longest diameter was measured. The presence of adenomyosis was recorded when MRI indicated junctional zone thickening $\geq$ 12 mm or junctional zone thickness exceeding 50% of the myometrium. DIE was diagnosed when solid endometriotic implants infiltrating deeper than 5 mm beneath the peritoneal surface were present. The diagnosis was based on the presence of irregular or nodular T2-hypointense areas consistent with abundant fibrosis or smooth muscle proliferation. On T1-weighted or fat-saturated T1-weighted sequences, these lesions demonstrated variable signal intensity depending on the presence or absence of blood products. The uterosacral ligaments, rectovaginal septum, torus uterinus, vaginal fornices, bladder, and bowel wall were systematically evaluated for these findings. All statistical analyses were performed using IBM SPSS Statistics version 28.0 (IBM Corp., Armonk, NY, USA). Distribution for continuous variables was assessed with the Shapiro–Wilk test. Since most measurements were non-normally distributed, non-parametric tests were applied. The Kruskal–Wallis test was used to compare age and endometrioma diameter across

Table 1: Median age (years) stratified by DIE and adenomyosis status

Parameters	Adenomyosis Absent	Adenomyosis Present	p value
DIE Absent	31.5 years	37 years	0.227
DIE Present	36 years	35 years	

DIE: deep infiltrating endometriosis

Table 2: Correlation and group comparison results for endometrioma diameter and adenomyosis DIE

Median endometrioma diameter (mm)	Adenomyosis Absent	Adenomyosis Present	p value
DIE Absent	37 mm	40,5 mm	0.812
DIE Present	42 mm	41 mm	

DIE: deep infiltrating endometriosis

Table 3: Cross-tabulation of patients by adenomyosis and DIE status

Parameters	Adenomyosis Absent	Adenomyosis Present	p value
DIE Absent	40	4	0.001
DIE Present	47	27	

DIE: deep infiltrating endometriosis

four groups defined by the presence or absence of adenomyosis and DIE. Point-biserial correlation coefficients were calculated to examine the relationships between endometrioma diameter and the presence of adenomyosis and DIE. The association between adenomyosis and DIE was evaluated using the chi-square test, supplemented with continuity correction, Fisher's exact test, and likelihood ratio. Symmetric measures, including Pearson's r and Spearman correlation, were computed to assess the strength and direction of associations. A p-value < 0.05 was considered statistically significant.

Results

A total of 125 patients with ovarian endometriomas were initially included. After excluding seven patients (four due to poor image quality and three due to missing clinical or ultrasound data), 118 patients were included in the final analysis. Among these, 74 (62.7%) had coexisting DIE and 31 (26.3%) had MRI findings consistent with adenomyosis. Notably, adenomyosis and DIE coexisted in 27 patients (22.9%), meaning that 87.1% of patients with adenomyosis also had DIE. Importantly, the prevalence of adenomyosis was significantly higher in women with DIE (36.5%, 27/74) than in those without DIE (9.1%, 4/44)." Four subgroups were examined in terms of age distribution: (1) no adenomyosis / no DIE, (2) adenomyosis only, (3) DIE only, and (4) both adenomyosis and DIE. Patients without adenomyosis or DIE had median ages of 31.5 years, those with adenomyosis but no DIE had median ages of 37 years, those with DIE but no adenomyosis had median ages of 36 years, and those with both adenomyosis and DIE had median ages of 35 years. According to statistical analysis, there was not a significant age difference between the four groups (p = 0.227) (Table 1). Ovarian endometrioma diameter was also compared between these four groups. Adenomyosis-only patients had a median endometrioma diameter of 40.5 mm, DIE-only patients had a median diameter of 42 mm, patients with both adenomyosis and DIE had a median endometrioma diameter of 41 mm, and the group without either condition had a median diameter of 37 mm. The Kruskal–Wallis test indicated there was no statistically significant differences between the four groups (p = 0.812) (Table 2). The potential association between the size of ovarian endometriomas and the presence of adenomyosis or DIE was also evaluated by using point-biserial correlation analyses. It showed that there is a small and statistically insignificant correlation (r = 0.028, p = 0.765) between the diameter of ovarian endometriomas and the occurrence of adenomyosis. Likewise, there was a small and statistically insignificant correlation between endometrioma diameter and DIE (r = 0.060, p = 0.517). Taken together, all these findings collectively indicate that endometrioma size is not a predictor of adenomyosis or DIE in our patient

cohort. In contrast, a clear and statistically significant relationship was found between presence of adenomyosis and DIE. The chi-square analysis shows a significant association between these two conditions (p = 0.001) (Table 3). As additional tests, the continuity correction (p = 0.002), likelihood ratio (p = 0.001), and Fisher's exact test (p = 0.001) supported our result, and all indicated a highly significant relationship. Additionally, symmetric measures further confirmed the same finding. Pearson's r and ordinal Spearman correlation both yielded a moderate positive correlation of 0.301 (p = 0.001), meaning that patients with adenomyosis were significantly more likely to also have DIE. Crosstab analysis showed the distribution of adenomyosis and DIE across the study cohort. The crosstab's adjusted residuals supported the statistical significance of the association by confirming that patients with adenomyosis were underrepresented in the DIE-negative group (adjusted residual = -3.3) and overrepresented in the DIE-positive group (adjusted residual = +3.3).

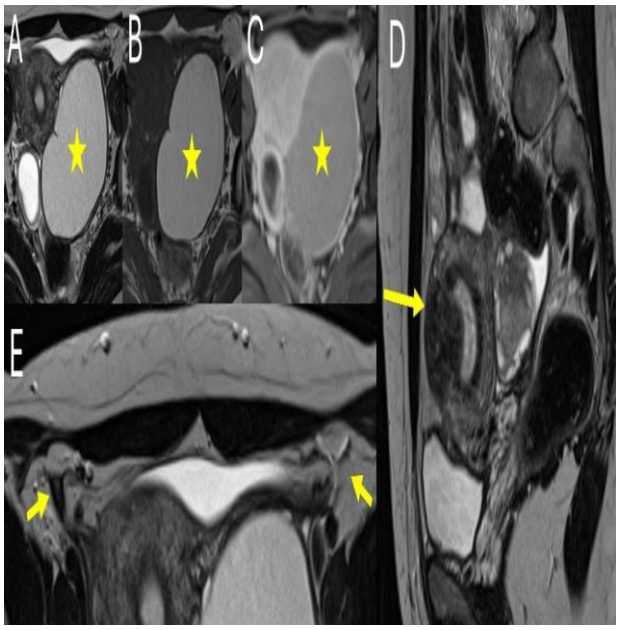

Figure 1: MRI findings of a patient with a large ovarian endometrioma, focal adenomyosis, and deep infiltrating endometriosis (DIE). (A) Axial T2-weighted image demonstrates large left ovarian endometrioma (asterisks). (B) Axial pre-contrast T1-weighted image shows the hyperintense signal of the endometrioma (asterisks). (C) Axial fat-suppressed post-contrast T1-weighted image confirms the presence of endometrioma (asterisks) without significant enhancement. (D) Sagittal T2-weighted image depicts focal adenomyosis in the anterior uterine wall (arrow). (E) Axial T2-weighted image shows bilateral broad ligaments; the right broad ligament is thickened and nodular with a DIE implant (arrow on the right side), while the contralateral side is unremarkable (arrow on the left side).

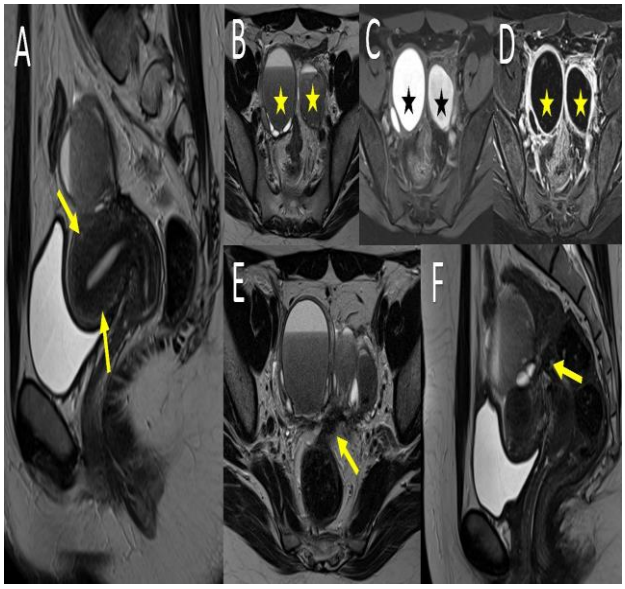

Figure 2: MRI findings of a patient with bilateral ovarian endometriomas, kissing ovaries, diffuse adenomyosis, and deep infiltrating endometriosis (DIE). (A) Sagittal T2-weighted image shows increased junctional zone thickness indicating diffuse adenomyosis (arrows). Axial T2-weighted image (B) and axial fat-suppressed pre-contrast image (C) present bilateral endometrioma (asterisks). (D) The subtraction axial image indicates no significant enhancement. Axial (E) and sagittal (F) T2-weighted image shows DIE implant located on the uterocervical junction tethering both ovaries and the sigmoid colon to this site (arrows).

Discussion

In this retrospective MRI-based study of 118 women with ovarian endometriomas, we found that endometrioma size was not significantly associated with the presence of either adenomyosis or DIE. Point-biserial correlation analysis demonstrated only negligible relationships between the largest ovarian endometrioma diameter and the presence of adenomyosis or DIE. Likewise, comparisons of median ovarian endometrioma size across groups defined by adenomyosis and DIE status did not yield significant differences. In contrast, a statistically significant association between adenomyosis and DIE was identified: adenomyosis was present in 27 of 74 women with DIE, compared with only 4 of 44 women without DIE (p = 0.001), and symmetric measures indicated a moderate positive correlation. Taken together, these findings suggest that while ovarian endometrioma size alone does not predict disease phenotype, the coexistence of adenomyosis and DIE is highly significant and may reflect shared pathophysiological mechanisms. In our study population, we identified a statistically significant association between adenomyosis and the presence of DIE, with 36.5% of patients with DIE also exhibiting

adenomyosis (Figure 1 and 2). This prevalence is comparable to the 42.76% reported by González et al. (9), who found that adenomyosis was significantly associated with more severe forms of endometriosis, including stage IV disease and rectosigmoid involvement. Their study also highlighted a higher frequency of debilitating symptoms, such as dysmenorrhea and deep dyspareunia, in patients with both conditions. While González et al. (9) did not observe a significant association between adenomyosis and ovarian endometriomas, they reported a clear correlation between adenomyosis and rectosigmoid or rectocervical DIE, supporting our finding of a meaningful relationship between adenomyosis and DIE regardless of endometrioma presence or size. Additionally, Larsen et al. (10) found adenomyosis in 34.6% of patients with endometriosis, further reinforcing its frequent coexistence. Although they reported no correlation between adenomyosis and rectovaginal DIE specifically, they did observe a trend toward more extensive adenomyotic wall invasion in women with advanced disease. These observations align with our results, suggesting that adenomyosis and DIE may represent interrelated phenotypes within the broader endometriosis spectrum, potentially reflecting a more aggressive or widespread disease process in affected individuals.

In our study of 118 women with ovarian endometriomas, 62.7% (n = 74) had coexisting DIE and 26.3% (n = 31) had adenomyosis. Despite the frequent coexistence of these pathologies, we found no statistically significant correlation between the size of the ovarian endometrioma and the presence of either DIE or adenomyosis. This finding contrasts with the assumption that larger endometriomas may indicate more extensive disease. In alignment with our results, Kwok et al. (11) demonstrated no significant difference in the number or distribution of DIE lesions between patients with ovarian endometriomas ≤50 mm and those with lesions >50 mm. Similarly, Kondo et al. (12) found no difference in DIE prevalence between endometrioma size groups (<40 mm vs. ≥40 mm), suggesting that endometrioma diameter is not a reliable marker for predicting the extent or severity of deep infiltrating lesions. On the other hand, they indicated higher DIE prevalence among patients with ovarian endometriomas when compared to those without. Perelló et al. (4) did also report significantly higher DIE prevalence among patients with ovarian endometriomas when compared to those without, reinforcing the idea that while endometrioma presence may serve as a marker for DIE, its size may not carry prognostic significance. Therefore, our results support that the size of an ovarian endometrioma may not be a relevant indicator for evaluating DIE risk. In parallel, we also did not observe a significant relationship between ovarian endometrioma diameter and the presence of adenomyosis. In our cohort, 26.3% (31/118) of patients with endometriomas had coexisting adenomyosis, yet statistical analysis did not reveal a meaningful correlation. Similarly, Alborzi et al. (13) reported no significant associations between ovarian endometrioma, uterosacral or rectocervical DIE, and the presence of adenomyosis. These findings reinforce the idea that although adenomyosis frequently coexists with endometrioma, its severity or distribution may not be predicted by the size or presence of ovarian endometriomas.

Study Limitations: This study has several limitations. First, it has a retrospective single-center design that may cause selection bias, as the patient cohort may not fully represent the broader population. Second, although MRI interpretation was performed in consensus by two experienced radiologists, interobserver variability was not formally assessed. Third, the sample size, while sufficient to demonstrate the significant association between adenomyosis and DIE, may still be underpowered to detect smaller effects. Fourth, the DIE diagnosis relied on MRI and clinical suspects in this presented study, and not all cases had histopathological confirmation. Fifth, MRI examinations were performed using both 1.5T and 3T scanners, which may introduce minor variations in image quality and signal characteristics. Lastly, clinical factors like symptoms and reproductive problems were not assessed because of the radiological nature of the study. External validation in larger, multicenter, prospective cohorts is warranted to confirm the generalizability of our findings.

Conclusion

This MRI-based study concludes by showing that there is a significant correlation between adenomyosis and DIE, even though the size of an ovarian endometrioma does not predict the presence of either condition. A careful evaluation of DIE implants when adenomyosis is presented on MRI is needed. Our study results underline the necessity of a thorough, multidisciplinary approach to patient evaluation and management and imply that adenomyosis may function as an imaging marker for DIE, which is a more complex endometriosis phenotype. To better highlight the relationship between ovarian endometriomas,

DIE, and adenomyosis, larger, prospective, multicenter studies with clinical symptoms and histopathological confirmation are needed.

Ethical approval: Ethics Committee permission was obtained from our institutional Biomedical Research Ethics Committee with the decision number 2024.053.IRB2.026 on 19.03.2024.

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Author contributions: Concept (HOA, ADK, UK), Design (HOA, ADK, UK), Data Collection and/or Processing (HOA, ADK, MSS), Analysis and/or Interpretation (HOA, ADK, MSS), Literature review (HOA, ADK), Writing – original draft (HOA), Writing – review and revision (ADK), Software and visualization support (MSS)

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