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Original Contribution

# Non-random adenomatoid tumours of the female genital system: A comparative clinicopathologic analysis of 14 cases

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ABSTRACT

The aim of this study was to evaluate adenomatoid tumours (AT) clinicopathologically in the female genital tract and compare the histomorphological features of ATs according to their uterine or tuba-ovarian location.

Cases of AT were excised and collected from female genital tracts between the years of 2010–2017. Cases were evaluated depending on their clinical findings, localisation and pathological properties.

There were 14 cases of AT. Ten cases were uterine, and 4 cases were adnexal tumours. The diagnostic ratio of uterine ATs was 64.3%, and of tuba-ovarian ATs was 21.4% ($P > 0.05$). The size of the largest tumour was 6 cm. Two of the uterine and one of the ovarian cases had a macrocyst; 2 uterine and one ovarian case had a microcyst; and 6 uterine had a combined microcystic/trabecular pattern. Uterine cases showed a higher number of smooth muscle component, signet-ring cells and infiltrative nature compared with other cases ($P < 0.05$). All uterine cases were infiltrative.

Most of ATs of the female genital system were small in size and incidentally diagnosed in our cases but rarely detected as an adnexal mass forming lesion which mimics a malignancy. A comparative clinicopathologic analysis of these cases should be considered with the histomorphological and immunohistochemical features for an accurate differential diagnosis.

## 1. Introduction

Adenomatoid tumours (AT) are rare, benign, mesothelium-based tumours in which the most characteristic histomorphological features are the dilated acinar structures in various sizes distributed in the stroma [1]. It was first described by Sakaguchi in 1916 as “adenomyomatoma” [2]. Several hypotheses have been put forward about the origin of the tumour cells, as they may originate from mesothelial cells, primitive Müllerian pluripotent mesenchymal cells or coelomic epithelium [1]. Golden and Ash introduced the descriptive term “adenomatoid tumour” in 1945 [3,4]. However, subsequent studies have shown that the tumour cells originate from the mesothelium histologically, immunophenotypically and ultrastructurally, as is now accepted, and are additionally termed as benign mesothelioma [5-7].

ATs are most commonly located in the female and male genital tract [3]. Extragenital regions are rarer, and there are cases reported at the locations of the mediastinum, pleura, adrenal gland, heart, liver, pancreas, omentum, appendix and other visceral organs in the literature [1,8]. Histologically, AT may be observed in several patterns such as angiomatoid, solid and cystic patterns. AT can have various morphologies as well as various combinations of these main patterns [3,5].

In the ovary, ATs often give an incidental finding but occasionally present with symptoms referable to a pelvic mass. Most ATs are small and located in the hilum of ovaries. They are usually solid but may also be multicystic [9,10]. Tubal ATs are also subserosal tumours of mesothelial origin and usually discovered incidentally. They mostly measure up to 1–2 cm in size, are located beneath the tubal serosa and they are rarely bilateral. Lastly, uterine ATs are also incidental findings [11]. Multifocal/diffuse tumours have been reported in immunosuppressed patients [12]. Most of these tumours are located in the outer myometrium and they are usually solitary, small (often < 4 cm) and solid but rarely can be diffuse, multifocal, large (> 10 cm) or predominantly cystic [11-14]. They have relatively ill-defined borders (when compared to leiomyomas) with a nodular, grey-white, firm cut surface [11].

In this study, we evaluated 14 AT cases clinicopathologically in the female genital tract and compared the diagnostic ratio and the

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histomorphological features of tumours according to the uterine or tuba-ovarian location.

## 2. Materials and methods

### 2.1. Patients

During 2010–2017, we identified 14 cases of AT diagnosed in the female genital system, collected from the archives of the Pathology Department.

Each case was re-examined by two pathologists and all diagnoses were reconfirmed with haematoxylin & eosin stained slides. Clinical findings, localisations, macroscopic features, microscopic features and additional disease states of the 14 study cases were recorded.

Macroscopically, the lesions varied from between 0.2 and 6 cm in size. 7 cases were detected incidentally on randomly sampled areas of fallopian tube or uterine myometrium.

### 2.2. Pattern definitions

Considering several pattern structures of ATs, we determined five basic different morphological patterns in our study: cystic, microcystic, microcystic/trabecular, solid/trabecular and retiform/adenoid.

The pattern of microcystic development has previously been described as an angiomatoid pattern, characterised by dilate gland-like cystic structures that resemble lymphatic spaces [3]. These gland-like structures are usually lined with a monolayer flattened epithelium. More rarely, it may be lined with hobnail-like or epithelioid cells. Among these cystic structures, smooth muscle fibres and rarely nerve fibres can be observed.

The combined microcystic/trabecular pattern was previously described as adenomatoid [3]. In this histopathological pattern, the cystic spaces are smaller, crowded and tumour cells can form trabecular-like structures and vacuoles, and thread-like bridging strands are generally visible in the cytoplasm of lining cells.

The macrocystic pattern is characterised by small cystic spaces as well as large cysts that can be seen macroscopically [3]. These cysts are covered with flattened mesothelial cells.

In a solid/trabecular pattern, mesothelial cells proliferate as trabecular structures or solid nests.

The adenoid pattern formerly known as retiform pattern consists of branched microcysts and duct-like structures with irregular margins in a dominant fibrous stroma. The lining epithelium is usually flattened but rarely large epithelial cells can also be seen [3].

### 2.3. Immunohistochemistry

Immunohistochemical staining using antibodies against pan-cytokeratin (PanCK), cytokeratin-7 (CK7), cytokeratin-20 (CK20), calretinin, CD10, CD31, CD34, CD68, vimentin, inhibin and WT-1 was performed on one representative section per case on 4 mm-thick formalin-fixed, paraffin-embedded sections mounted on charged slides. All cases were stained in parallel with appropriate positive and negative controls. Staining intensity was semi-quantitatively scored as negative (0, < 5% cells stained), focally positive (1 +, 5–10% cells stained), positive (2 +, 10–50% cells stained), or diffusely positive (3 +, > 50% cells stained), and a mean intensity (MI; range 0–3) was calculated for uterine and tuba-ovarian sites. In addition to an assessment of clinicopathological features, morphological differences of ATs in uterine versus tuba-ovarian sites, if any, were compared including features previously described as characteristic of AT.

### 2.4. Statistical analysis

GraphPad Version 3.062003 was used for statistical analysis. All the descriptive values are reported as mean ± SD and median (minimum-maximum) values. Patients' age and tumour sizes were tested for a normal distribution using the Kolmogorov–Smirnov test. For two non-normally distributed dependent variables, a corresponding non-parametric test Wilcoxon test was used. A Chi-square Test was used to compare dependent qualitative data. The Yates Continuity Correction test (Yates' corrected Chi-square) was used to compare independent qualitative data. $P < 0.05$ levels were considered significant.

### 2.5. Ethics statements

All procedures performed in studies involving human participants were in accordance with the 1964 Helsinki declaration and its later amendments or comparable ethical standards and approved by the Local Clinical Research Ethics Committee (GOKAEK-2020/7.21. 2020/105). Each patient signed an informed consent form, allowing the use of the tissue fragments in scientific studies.

## 3. Results

In the present study, there were 14 AT cases of the female genital system, including 10 cases (71.4%) located in the uterine and 4 cases (28.6%) located in tuba-ovarian site [Table 1] of the tuba wall.

### 3.1. Clinicopathological features

For the patients with uterine ATs, the mean age was 48.4 ± 6.7 [35.0–59.0] and the mean tumour size was 2.7 ± 1.2 [0.5–4.0] [Table 1]. The diagnostic ratio of uterine ATs among all cases was 64.3%. All these patients were operated upon due to myoma uteri in five of the cases, an endometrial polyp in one case, cervical carcinoma in one case, a uterine prolapse in one case, a pelvic mass in one, and lastly an ovarian mass in one. In the patient who underwent surgery for an ovarian mass, AT was found incidentally in the myometrium [Table 1].

4 cases of uterine ATs were subserosal and 6 cases were intramural. Macroscopic cut surfaces in 9 cases were solid/nodular, round, firm, greyish white and whorled. A multilocular cystic morphology was present only in case. Microscopically, two of the uterine AT cases had a macrocystic [Fig. 1], 2 had a microcystic [Fig. 2] and 6 had a combined microcystic/trabecular pattern [Fig. 3]. A smooth muscle component was observed in 9 of the uterine AT cases. Signet-ring cell formation was present in 6 cases [Fig. 3]. Lymphoid aggregate structures were observed in two cases. All cases were infiltrative, and no mitosis, necrosis or atypia was observed [Table 2].

**Table 1**
Clinicopathological features of the adenomatoid tumours of the female genital tract.

| Site | Cases n (%) | Incidental n (%) | Age (years) X ± SD [range] | Size (cm) X ± SD [range] |
|---|---|---|---|---|
| Uterine | 10 (71.4) | 9 (64.3) | 48.4 ± 6.7 [35–59] | 2.7 ± 1.2 [0.5–4.0] |
| Tuba-ovarian | 4 (28.6) | 3 (21.4) | 50.0 ± 15.6 [30–68] | 1.9 ± 2.8 [0.2–6.0] |
| Total | 14 (100) | 12 (85.7) | 48.9 ± 9.4 [30–68] | 2.5 ± 1.7 [0.2–6.0] |
| | *P* value | 0.469 | 0.778 | 0.240 |

X ± SD: mean ± standard deviation.

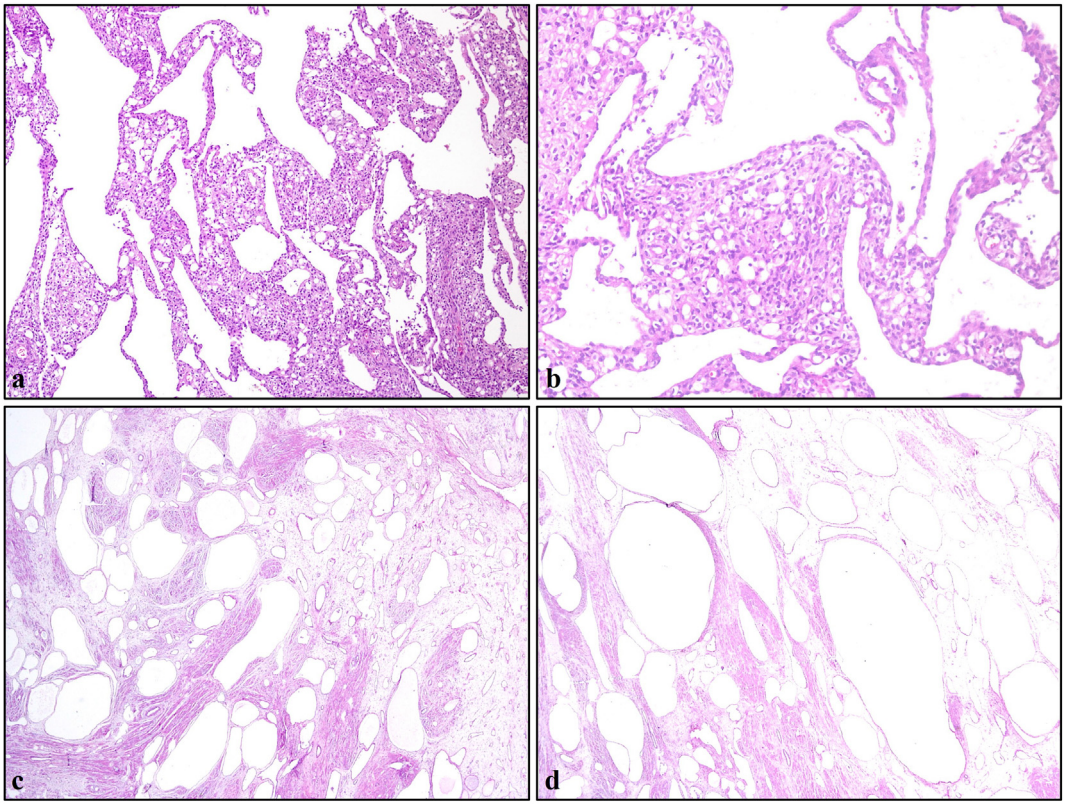


**Fig. 1.** Macrocystic patterns of adenomatoid tumour case; characterised by small cystic spaces as well as large cysts surrounded by flattened mesothelial cells (H&E; a: ×100, b: ×200, c: ×100, d: ×200).

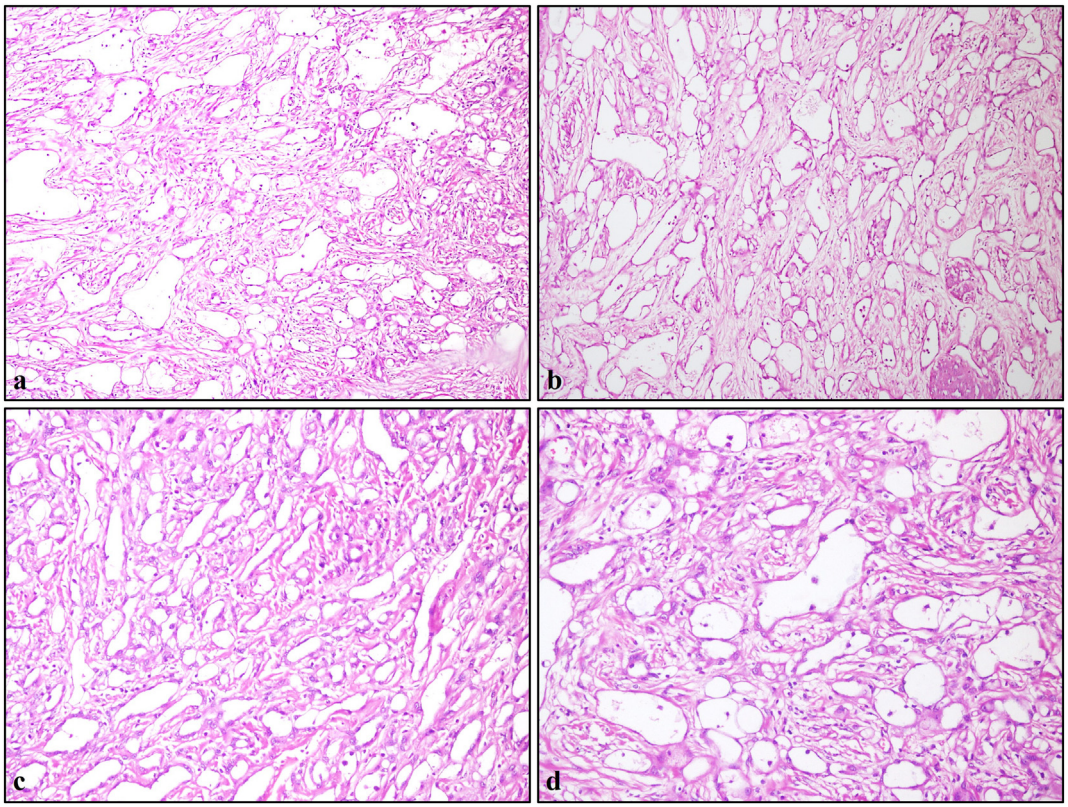


**Fig. 2.** Microcystic patterns of adenomatoid tumour case; characterised by dilate gland-like cystic structures that resemble lymphatic spaces, lined with a monolayer flattened epithelium. (H&E; a: ×100, b: ×200, c: ×200, d: ×400).

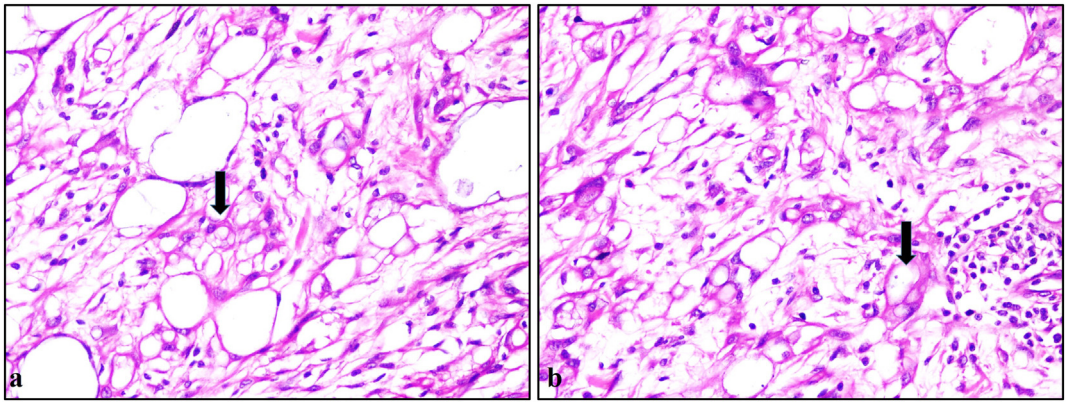


**Fig. 3.** Microcystic/trabecular pattern of adenomatoid tumour case; lined with hobnail-like or epithelioid cells or signet ring cells (black arrow) (H&E; a: ×400, b: ×400).

The mean age of the four patients with tuba-ovarian ATs was 50.0 ± 15.6 [30–68] and the mean tumour diameter was 1.9 ± 2.8 [0.2–6.0]. There was no significant difference in patients' age and size of tumours among the tumour locations [Table 1]. The diagnostic ratio of tuba-ovarian ATs among all cases was 21.4% and this was not significantly different from the uterine ATs. Two cases with tubal ATs had undergone operations due to unrelated causes of tumours such as endometrial polyps and urinary incontinence, and the AT was detected incidentally in resection materials. There was no macroscopically detected lesion in the case of tubal AT since the tumour diameter was 0.2 cm. In the other tubal AT case, a solid nodule with a greyish white colour was seen on the tubal serosal area [Figs. 4 and 5] [Table 2]. In ovarian AT cases, an ovarian mass was detected with dysmenorrhea, and the other case was detected incidentally in the resection material due to uterine prolapse. One of the ovarian AT cases was also incidentally detected in routine sampling while the other case had distinctive cystic structures [Table 2].

Microscopically, an adenoid/retiform pattern was observed in two tubal AT cases [Fig. 6]; one had a macrocystic and the other had a microcystic pattern. Accompanying smooth muscle was observed in one tuba-ovarian AT case while lymphoid aggregates were observed in two cases. None of the cases revealed any signet ring cell formation, infiltrative feature, prominent mitotic activity, necrosis and atypia [Table 2].

### 3.2. Comparison of histomorphological features

Among the histomorphological features of ATs, uterine cases showed a higher number of smooth muscle component, signet-ring cells and infiltrative nature compared with tuba-ovarian AT cases ($p$ = 0.015, 0.04 and 0.0002, respectively). There was no significant difference for lymphoid aggregates among uterine and tuba-ovarian AT cases [Table 3].

### 3.3. Immunohistochemistry

All of the AT cases revealed a diffuse positivity (Mean intensity = 3.0 ± 0 for all) in PanCK, CK-7, calretinin staining, and a positivity (Mean intensity = 2.0 ± 0) in WT-1 staining [Table 4]. None of them showed any reactions against CK-20, CD10, CD31, CD68 or vimentin antibodies. Only one of the ovarian cases showed a focal positivity for CD34 and inhibin. The rest of the AT cases had no staining for CD34 and inhibin.

## 4. Discussion

Adenomatoid tumours are benign neoplasms of mesothelial origin that can be seen in the female genital tract, more commonly involving the uterus and fallopian tubes than the ovary. It is the most common benign tumour of the fallopian tube [13]. Adenomatoid tumours, mostly diagnosed incidentally, pose a diagnostic challenge as they can histologically mimic malignant neoplasms, such as carcinoma, adenocarcinoma and mesothelioma [14-16]. The rare presence of AT cases suggests that macroscopic sampling may be inadequate or that the cases may not be noticed due to the unsuspecting benign appearance of the histological images, leading to the incidental diagnosis of the tumour [17]. In the present study, 14 AT cases were examined clinicopathologically, and the diagnostic ratio and the histomorphological features of tumours were compared according to the uterine or tuba-ovarian location.

The uterus is the most common localisation for ATs among the organs of the female genital system [15]. Of the 14 cases in our study, 71.4% were uterine and 28.6% were tuba-ovarian tumours. Most of our AT cases were operated for other reasons, such as endometrial polyps, myoma uteri, cervix carcinoma, uterine prolapse, and ATs were incidentally detected in these cases. The most common pattern seen among our AT cases with the uterine residents was the gross cystic appearance. Retiform/adenoid and solid/trabecular patterns were not observed in uterine cases. The retiform/adenoid pattern was seen more frequently in tuba-ovarian ATs.

In the literature, lymphoid aggregates are more common in the male genital system [5]. In another study, it was argued that the lymphoid aggregates were observed in the periphery of the tumour, and this could be a clue for diagnosis [18]. In our study, these aggregates were observed in 4 (28.6%) of 14 cases, all of which were located peripherally. There was no statistically significant difference among uterine and tuba-ovarian cases in terms of the incidence of lymphoid aggregates.

Previous studies have investigated whether AT shows the morphological changes according to the localisation and whether there is a smooth muscle structure in the normal histology of that region [3]. The smooth muscle component is frequently seen among uterine AT cases [5]. However, it is unclear and even controversial whether smooth muscle is a component of the tumour. Some authors, who agree on smooth muscle being the component of the tumour, use the term leiomyoma-adenomatoid tumour, while others agree that smooth muscle tissue is myometrium [6,7]. In our study, a smooth muscle component was observed in 10 (71.4%) cases; nine of these were uterine tumours and one case was a tubal tumour. Therefore, the incidence of smooth muscle component was considerably higher in uterine AT cases than in tuba-ovarian cases.

Vacuolated cells in ATs resembling signet-ring cells are described among many features of these tumours [19]. In our study, the signet-ring cell morphology was observed in 6 uterine cases (42.9%) but not in tuba-ovarian cases. Necrosis, atypia and mitosis were not observed in

**Table 2**
Macroscopic and histological features of adenomatoid tumours in the female genital tract.

| Case no | Site | Macroscopy | Size (cm) | Growth pattern | Smooth muscle component | Signet-ring cells | Lymphoid aggregates | Infiltrative | Mitosis | Necrosis | Atypia |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Uterine/subserosal | Solid/nodular | 3.5 | Macrocystic | + | − | − | + | − | − | − |
| 2 | Uterine/intramural | Solid/nodular | 3.5 | Combined microcystic/trabecular | + | + | − | + | − | − | − |
| 3 | Uterine/subserosal | Solid/nodular | 3.5 | Combined microcystic/trabecular | + | + | + | + | − | − | − |
| 4 | Uterine/intramural | Solid/nodular | 3.5 | Combined microcystic/trabecular | + | + | + | + | − | − | − |
| 5 | Uterine/intramural | Solid/nodular | 4.0 | Combined microcystic/trabecular | + | + | − | + | − | − | − |
| 6 | Uterine/intramural | Multilocular cystic | 2.15 | Macrocystic | + | − | − | + | − | − | − |
| 7 | Uterine/intramural | Solid/nodular | 1.3 | Microcystic | + | + | − | + | − | − | − |
| 8 | Uterine/subserosal | Solid/nodular | 2.0 | Combined microcystic/trabecular | + | − | − | + | − | − | − |
| 9 | Uterine/intramural | Solid/nodular | 0.5 | Microcystic | + | − | − | + | − | − | − |
| 10 | Uterine/subserosal | Solid/nodular | 3.0 | Combined microcystic/trabecular | − | − | − | + | − | − | − |
| 11 | Tuba/tuba wall | No lesion | 0.2 | Retiform/adenoid | + | − | + | − | − | − | − |
| 12 | Tuba/serosa | Solid/nodular | 1.0 | Retiform/adenoid | − | − | + | − | − | − | − |
| 13 | Over | Cystic | 6.0 | Macrocystic | − | − | − | − | − | − | − |
| 14 | Over/serosa | Cystic | 0.3 | Microcystic | − | − | − | − | − | − | − |
| | | | | Total | 10 | 6 | 4 | 10 | 0 | 0 | 0 |

any of the cases.

Evaluating the records of AT cases diagnosed in our hospital, it was noted that some of the patients were not followed, raising a limitation of the present study. However, recurrence was not detected in other follow-up cases. Very detailed knowledge of the clinical aspects could not be obtained, but our results supported the benign nature of the AT.

Based on a quite extensive histological spectrum of ATs, the differential diagnosis involves many benign or malignant tumours. Malignant mesothelioma (MM) should also be considered in the differential diagnosis due to the mesothelial origin, the localisation site, the origin of tumour cells and histological features of ATs. It is impossible to reach a diagnosis only based on the localisation and cell origins. However, ATs are small clean-margined lesions, histologically having some unsuspected nuclear features. Rarely having a diffuse pattern, they can infiltrate the peripheral organs. On the other hand, MM is usually larger in size and mostly represents a peritoneal surface involvement. However, it should not be forgotten that MM may also contain areas with an unsuspected appearance similar to AT. Even immunohistochemical markers of MM including PanCK, CK-7, calretinin and WT-1 reveal similar positive staining patterns, and CK-20, CD10, CD31, CD68 and vimentin show similar negative staining with AT, as in cases of the present study. These markers are helpful to differentiate the diagnosis from vascular or muscular or metastatic tumours. However, these mesothelial markers did not function as an immunohistochemical discriminator, hence, some studies have been done with Pax-8 immunohistochemistry. One of these reported that no expression was seen in the MM while Pax-8 expression was observed in AT cases [3].

ATs with a cystic pattern can often be confused with the multilocular peritoneal inclusion cysts. Furthermore, the fact that the tumour is localised on the serosal surface and does not form a solid mass should suggest these inclusion cysts in the differential diagnosis. In the present study, two ovarian tumours showed these cystic features.

Another differential diagnosis of mesothelial origin is the benign papillary mesothelioma. It is a polypoid peritoneal lesion that generates small papillary projections toward the surface with a thin fibrovascular core. For ATs, no papillary projections are observed on the surface [20], as for our study cases.

Although ATs have histological features of a benign structure, the tumours that have a trabecular or trabecular/microcystic pattern, as in six cases of the present study, may be confused with the adenocarcinoma. The infiltrative nature, the distinctive malignant cytologic atypia and architectural structure of the adenocarcinoma, as well as the absence of mesothelial marker expression helps with the differential diagnosis.

As stated before, ATs are most commonly located in the uterus and detected incidentally. As they macroscopically share similar morphology with the leiomyoma and may microscopically include a distinctive smooth muscle component, it is possible to misdiagnose as a leiomyoma if a careful sampling and detailed microscopic examination are not performed. However, the cystic/glandular structures with variable sizes observed in ATs are absent in leiomyomas.

In the differential diagnosis of ATs, the lymphangioma, epithelioid haemangioendothelioma and yolk sac tumours should also be considered. It is sufficient to use the endothelial and mesothelial immunohistochemical markers to discriminate the lymphangiomas and epithelioid haemangioendotheliomas. However, CD34, CD31 and factor VIII are endothelial markers that can be used. In the present study, CD31 immunostaining was negative for all AT cases but CD34 showed a focal positivity only in one of ovarian cases. If the AT is observed in a young patient, it may be confused with the yolk sac tumour. These tumours can histologically represent a microcystic or reticular pattern with flattened or cuboidal cells as in AT cases. But their primitive nuclear appearance and AFP expression help for a differential diagnosis. In the present study, none of the tumour markers were elevated in the bloods of our AT cases, neither was the nuclear atypia, such as that

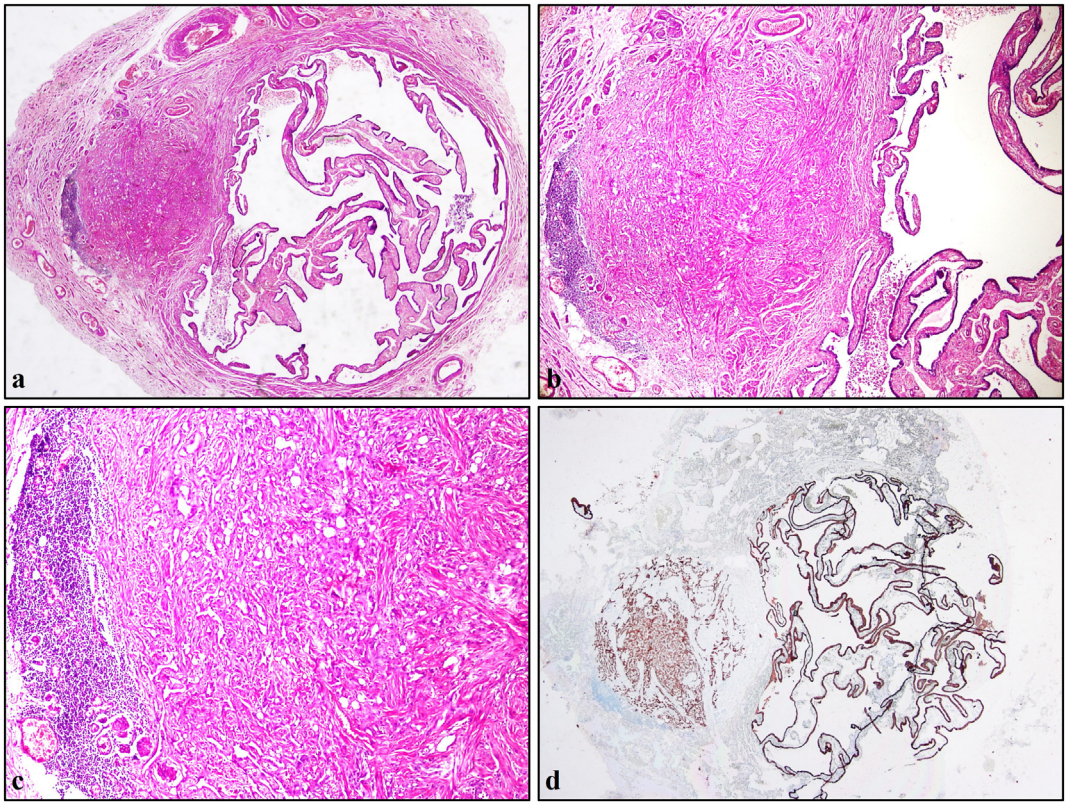


**Fig. 4.** Adenomatoid tumour located beneath the tubal serosa; note variably sized tubules lined by flattened to cuboidal appearing cells (a: H&E ×20, b: H&E ×40, c: H&E ×100, d: pan-cytokeratin ×20).

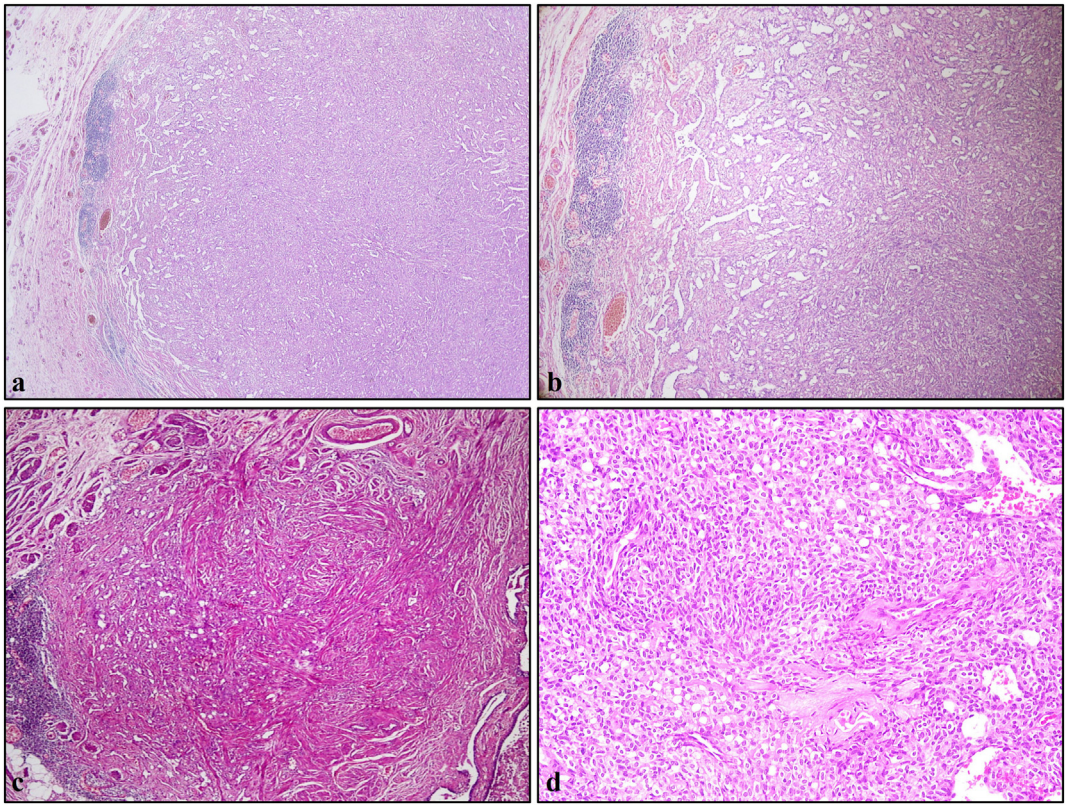


**Fig. 5.** Solid/trabecular pattern of adenomatoid tumour case; characterised by mesothelial cells proliferating as trabecular structures or solid nests (H&E; a: ×20, b: ×40, c: ×40, d: ×100).

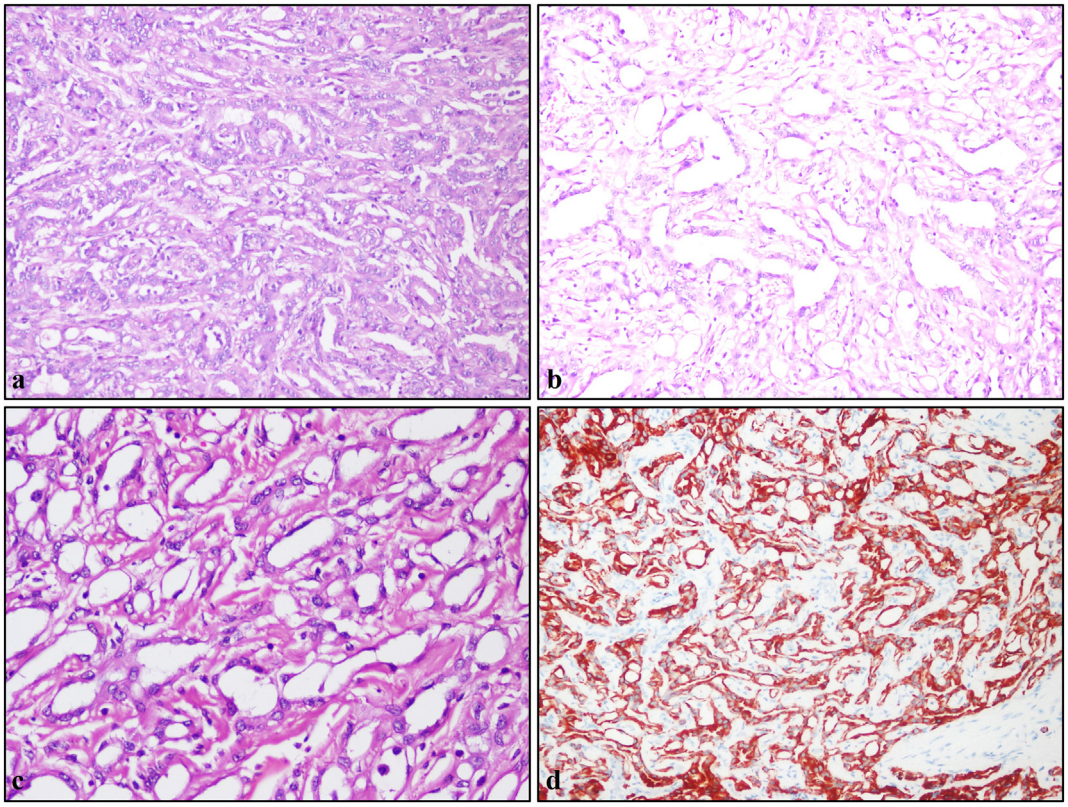


**Fig. 6.** Adenoid/retiform pattern of adenomatoid tumour case; characterised by branched microcysts and duct-like structures with irregular margins in a dominant fibrous stroma. Large epithelial cells (a) and flattened lining epithelium (b, c, d) are observed (a: H&E ×200, b: H&E ×400, c: H&E ×400, d: pan-cytokeratin ×200).

**Table 3**
Comparison of histomorphological features of adenomatoid tumours in the female genital tract.

| Site | Smooth muscle component n (%) | Signet-ring cells n (%) | Lymphoid aggregates n (%) | Infiltrative n (%) |
|---|---|---|---|---|
| Uterine | 9 (64.3) | 6 (42.9) | 2 (14.3) | 10 (71.4) |
| Tuba-ovarian | 1 (7.1) | 0 (0) | 2 (14.3) | 0 (0) |
| Total | 10 (71.4) | 6 (42.9) | 4 (28.6) | 10 (71.4) |
| P value | 0.015* | 0.04* | 0.262 | 0.0002* |

* $P < 0.05$ by Chi-square test.

**Table 4**
Immunohistochemical results of adenomatoid tumours, both in uterine and tuba-ovarian sites.

| Antibodies | Positive n (%) | Mean intensity |
|---|---|---|
| PanCK | 14 (100) | 3.0 ± 0 |
| CK-7 | 14 (100) | 3.0 ± 0 |
| CK-20 | 0 (0) | 0 ± 0 |
| Calretinin | 14 (100) | 3.0 ± 0 |
| CD10 | 0 (0) | 0 ± 0 |
| CD31 | 0 (0) | 0 ± 0 |
| CD34 | 1 (7.1) | 0.07 ± 0.27 |
| CD68 | 0 (0) | 0 ± 0 |
| Vimentin | 0 (0) | 0 ± 0 |
| Inhibin | 1 (7.1) | 0.07 ± 0.27 |
| WT-1 | 14 (100) | 2.0 ± 0 |

PanCK: pan-cytokeratin, CK: cytokeratin, WT-1: Wilms' tumour 1.

observed in yolk sac tumours, detected. There was a large AT with size of 6 cm, suggesting that the size of tumour may gain importance in the differential diagnosis of cystic malign epithelial tumours, although this diagnosis is rare.

In summary, most of the ATs of the female genital system are incidental, and a comparative clinicopathologic analysis of these cases should be considered with the histomorphological and immunohistochemical features for an accurate differential diagnosis.

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## Declaration of competing interest

None.

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