Section: Pathology

Original Research Article

ISOLATED FALLOPIAN TUBE ENDOMETRIOSIS: A CLINICO-PATHOLOGICAL STUDY FROM A TERTIARY CARE CENTRE

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ABSTRACT

Background: Endometriosis is a chronic oestrogen-induced condition in which endometrium-like tissue occurs beyond the uterine cavity. Tubal endometriosis is an unusual presentation characterised by endometrial glands and stroma within the fallopian tube, most often detected incidentally. It is generally asymptomatic but may cause pelvic pain or infertility. The exact cause of the condition still remains unclear. The objective is to evaluate the clinico-pathological features of isolated fallopian tube endometriosis in hysterectomy and salpingectomy/salpingo-oophorectomy specimens received for benign gynaecological disorders.

Materials and Methods: Hysterectomy specimens with bilateral salpingectomy or salpingo-oophorectomy received for benign gynaecological disorders at a tertiary care centre were evaluated. The fallopian tubes were examined on haematoxylin and eosin-stained sections, and immunohistochemistry with CD10 was performed in selected cases for diagnostic confirmation.

Results: Fourteen cases of isolated tubal endometriosis were identified in women aged 35–45 years presenting with pelvic pain, dysmenorrhoea, menorrhagia, or heavy menstrual bleeding. Endometrial glands and stroma were confined to the mucosal lining of one or both fallopian tubes, consistent with mucosal or intraluminal tubal endometriosis, without gross abnormality or ovarian or peritoneal involvement. Associated findings included hydrosalpinx, salpingitis isthmica nodosa, leiomyomas and adenomyosis.

Conclusion: Isolated tubal endometriosis is an uncommon, frequently incidental finding that may be under-recognised in routine gynaecological specimens. Careful microscopic evaluation of the fallopian tubes is essential for accurate diagnosis and for understanding its association with reproductive dysfunction.

Keywords: Tubal endometriosis, Fallopian tube, Endometrial glands, Hysterectomy, Salpingectomy.

INTRODUCTION

Fallopian tubes, also called oviducts, are thin muscular tubes that facilitate sperm transport, fertilisation, and the movement of gametes and early embryos through the uterus.[1] Fallopian tubes are a ubiquitous part of routine surgical histopathology, as they are usually removed during surgery due to low-risk surgeries for conditions related to benign gynaecological disorders.[2] Endometriosis is a chronic, oestrogen-responsive condition defined by the presence of ectopic endometrial-type glands and

stroma located outside the uterine cavity. It affects approximately 10% of women in the course of their reproductive life and is frequently accompanied by pelvic pain, dysmenorrhoea, dyspareunia, inappropriate uterine bleeding, and infertility. Despite extensive research, the exact aetiopathogenesis remains incompletely understood.

The most widely accepted hypothesis is the retrograde menstruation, which proposes that viable endometrial fragments reflux through the fallopian tubes into the pelvic cavity, where they implant and proliferate. Other underlying factors, such as hormone imbalance, immune compromise, and inflammation pathways, are believed to influence the persistence and progression of the disease.[3] Alternative mechanisms, including lymphatic or haematogenous dissemination and coelomic metaplasia, have also been proposed. Based on these theories, mesothelial cells lining the peritoneum, pleura, or ovary can undergo metaplastic transformation into tissue of a more endometrial nature.[4,5]

When ectopic endometrial tissue involves the fallopian tube, the condition is termed tubal endometriosis. This entity presents with pelvic pain or infertility, or remains asymptomatic and is incidentally detected. Tubal endometriosis may be histologically characterised by superficial serosal implants, mucosal infiltration, or lesions of the residual tubal stump associated with salpingectomy.[1] Tubal endometriosis occurs predominantly in women during their reproductive years, usually between the ages of 20 and 50, and is rarely detected after menopause.[6] The endometriotic lesions also do occur in other sites such as the ovaries and pelvic peritoneum, abdominal wall, bowels, bladder, cervix and vagina.[7]

This study aims to identify the occurrence of fallopian tube endometriosis among patients who are evaluated for benign gynaecological indications. The aim is to review the existing literature, identify knowledge gaps, and highlight the need for more effective diagnostic and management strategies for tubal endometriosis.

MATERIALS AND METHODS

This observational study was conducted in the Department of Pathology at Chettinad Hospital and Research Institute. The study included hysterectomy specimens with bilateral salpingo-oophorectomy and hysterectomy specimens with bilateral salpingectomy received for benign gynaecological conditions during the study period. Detailed clinical history, operative findings, and gross pathological features were all recorded. All specimens were fixed in 10% formalin, processed routinely, and stained with haematoxylin and eosin for microscopic evaluation. Histopathological examination of the fallopian tubes was done to identify the presence of endometrial glands and stroma within the tubal mucosa. Cases showing endometriotic glands and stroma confined to the fallopian tubes, as shown in [Figures 1–4], without ovarian or peritoneal endometriosis were included in the study. Immunohistochemistry with CD10 was done in selected cases where confirmation was required.

RESULTS

Fourteen cases of isolated tubal endometriosis were identified during the study period in women aged between 35 and 45 years who presented with complaints of heavy menstrual bleeding, dysmenorrhoea, and pelvic pain. Most patients had a prior history of vaginal delivery and had no significant contraceptive history. Clinical examination findings were unremarkable, while radiological evaluation commonly demonstrated a bulky uterus with intramural or subserosal leiomyomas, and features of adenomyosis were noted in a few cases. The patients underwent total abdominal hysterectomy with bilateral salpingo-oophorectomy (TAH with BSO) or total abdominal hysterectomy with bilateral salpingectomy for benign gynaecological conditions. Intraoperatively, there was no evidence of pelvic endometriosis, ovarian involvement, adhesions, or peritoneal deposits in any of these cases. Gross examination of the fallopian tubes revealed patent lumina without obvious endometriotic lesions, while the uterus frequently showed multiple leiomyomas with a characteristic tan-white whorled appearance and associated adenomyosis. Histopathological examination consistently demonstrated endometrial glands and stroma confined to the mucosal lining of one or both fallopian tubes, consistent with mucosal or intraluminal tubal endometriosis. Bilateral involvement was observed in the majority of cases, whereas a few cases showed unilateral right- or left-sided involvement. Associated findings included adenomyosis, leiomyomas, hydrosalpinx, and salpingitis isthmica nodosa in isolated instances. The ovaries and peritoneum were free of endometriosis in all cases, supporting the diagnosis of isolated tubal endometriosis.

Table 1: Case-wise distribution of clinical and pathological findings in fallopian tube endometriosis

Number	Age	Symptoms	Surgery	Associated Pathology	Laterality
Case 1	38/F	Pelvic pain	TAH with B/L salpingectomy	Adenomyosis	Left fallopian tube
Case 2	42/F	Dysmenorrhoea	TAH with B/L salpingo-oophorectomy	Multiple leiomyomas; salpingitis isthmica nodosa	Right fallopian tube
Case 3	40/F	Pelvic pain; menorrhagia	TAH with B/L salpingectomy	Salpingitis isthmica nodosa	Left fallopian tube

Case 4	40/F	Menorrhagia	TAH with B/L salpingo-oophorectomy	Adenomyosis; leiomyomas	B/L fallopian tube
Case 5	45/F	Pelvic pain	TAH with B/L salpingectomy	Hydrosalpinx	B/L fallopian tube
Case 6	36/F	Menorrhagia	TAH with B/L salpingectomy	Adenomyosis	B/L fallopian tube
Case 7	43/F	Heavy menstrual bleeding	TAH with B/L salpingo-oophorectomy	Adenomyosis; leiomyomas; salpingitis isthmica nodosa	Left fallopian tube
Case 8	37/F	Menorrhagia	TAH with B/L salpingectomy	Leiomyomas	B/L fallopian tube
Case 9	41/F	Pelvic pain	TAH with B/L salpingo-oophorectomy	Adenomyosis; hydrosalpinx	B/L fallopian tube
Case 10	44/F	Menorrhagia	TAH with B/L salpingectomy	Adenomyosis	B/L fallopian tube
Case 11	43/F	Heavy menstrual bleeding	TAH with B/L salpingo-oophorectomy	Leiomyomas; salpingitis isthmica nodosa	Left fallopian tube
Case 12	41/F	Menorrhagia	TAH with B/L salpingectomy	Leiomyomas	B/L fallopian tube
Case 13	40/F	Pelvic pain	TAH with B/L salpingectomy	Hydrosalpinx	B/L fallopian tube
Case 14	39/F	Pelvic pain	TAH with B/L salpingectomy	Adenomyosis	Right fallopian tube

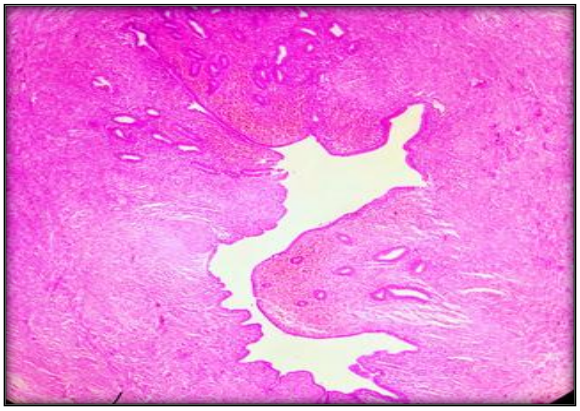

Figure 1: Photomicrograph showing endometriotic foci in the mucosa of the fallopian tube (H&E, ×10)

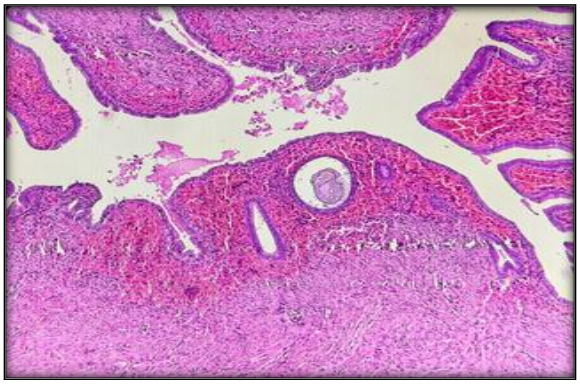

Figure 2: Photomicrograph showing endometriotic foci in the mucosa of the fallopian tube (H&E, ×20)

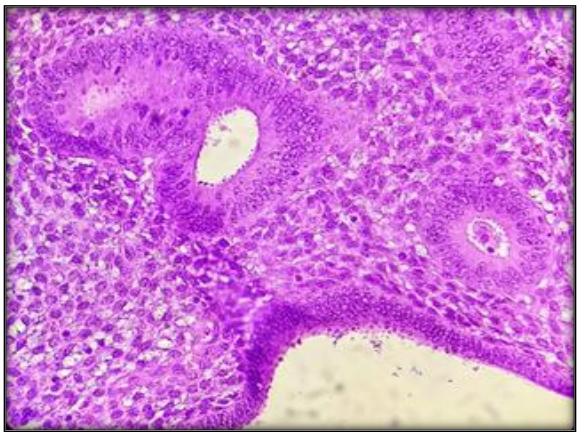

Figure 3: Endometrial glands and stroma within the tubal mucosa (H&E, ×40)

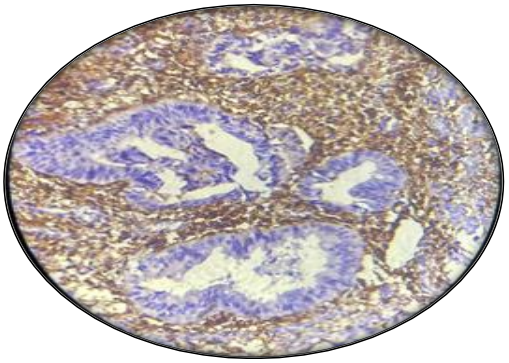

Figure 4: CD10 immunohistochemistry positive in endometriotic stroma (×40)

DISCUSSION

Endometriosis is now recognised as a heterogeneous disorder that may involve multiple organ systems and display a wide spectrum of clinical and pathological manifestations.[8] Though infertility, dyspareunia, and chronic pelvic pain are common in affected women, many women do not have symptoms and are incidentally diagnosed.[3,8] Dr. John Albertson Sampson laid the groundwork for understanding the pathogenesis of endometriosis, which would come to influence new conceptual frameworks focused on this disease.[9]

The retrograde menstruation hypothesis suggests that menstrual efflux may flow retrograde through the fallopian tubes, allowing viable endometrial cells to enter the proximal oviduct, where they can implant and proliferate, leading to tubal obstruction and endometriosis.[1] Nevertheless, as retrograde menstruation occurs frequently physiologically and only a subset of women develop endometriosis, additional modifying factors are thought to modulate disease progression. This may involve genetic risk factors, alterations in immune surveillance, hormonal changes or hormone pathways, and environmental exposures. In addition, new evidence suggests that endometrial stem or progenitor cells, dysregulation of matrix metalloproteinases and their tissue inhibitors, and increased local oestrogen production are central in stimulating the disease. Different pathogenetic explanations—including lymphovascular spread,

coelomic metaplasia, iatrogenic implantation, and embryonic cell rests—support the idea that endometriosis is more a combination of overlapping mechanisms than a single aetiologic pathway.[10] The steroid hormones of ovary which includes oestradiol and progesterone that regulates the endometrium through oestrogen and progesterone receptors. Oestradiol plays a major role in the growth and proliferation of endometrial tissue during the proliferative phase of the menstrual cycle.[11]

Endometriosis may pose significant diagnostic challenges, especially in uncommon anatomical sites.[12] Morphologically, the condition presents in various forms, with ovarian and peritoneal endometriosis being the most commonly encountered, followed by deep infiltrating endometriosis. Peritoneal lesions are found in 15–50% of women undergoing laparoscopic examination and are located around or just below the peritoneal region. Ovarian endometriosis (also known as endometriotic or "chocolate" cysts) develops in 2–10% of women of childbearing age and affects nearly half of women evaluated for infertility. Deep infiltrating endometriosis is characterised by involvement of structures outside the peritoneal surface (bladder, ureters, bowel, vagina, or uterosacral ligaments). Yet, there is limited understanding of the specific pathogenic mechanism underlying this aggressive form.[13]

Brosens histopathological taxonomy for 1993 divided endometriosis into three groups. The mucosal type corresponds to ovarian endometrial cysts; the peritoneal type comprises lesions with variable morphology, ranging from early glands proliferating into fibrotic plaques; and the glandular type consists mainly of fibromuscular tissue, which relates to deeply infiltrating disease.[13] Tubal endometriosis, defined as the infiltration of endometrial glands and stroma along the fallopian tube regardless of the underlying pathogenetic mechanism, is mostly confirmed by a combination of surgical findings and histological evaluation.[8] In the present case series, all cases demonstrated mucosal-limited involvement without serosal, ovarian, or peritoneal disease, suggesting a localised form of tubal endometriosis.

The fallopian tube tissue may contain ectopic endometrium, and a variety of developmental changes may result, including polypoid lesions, adenomyosis-like changes resembling salpingitis isthmica nodosa, and intraluminal endometriosis.[1] The histologic diagnosis depends on the detection of endometrial glands and stroma that can display proliferative, secretory, inactive, or hyperplastic structures.[9] Luminal obstruction, fibrosis, adhesions, and hydrosalpinx, all sequelae of chronic inflammation, can lead to tubal dysfunction caused by endometriosis. Persistent inflammation in the fallopian tube may cause intraluminal obstruction at different sites along its length. Obstruction at the fimbrial end results in narrowing and clubbing of the tubal fimbriae, leading to fluid accumulation within the lumen and formation of hydrosalpinx.[14] Because the two processes often co-occur, it is difficult to determine their relative contributions to tubal injury. Altogether, tubal involvement has been described in approximately 30% of women with endometriosis.[15]

CD10 has been shown to have a significant role in cellular proliferation and cell–cell adhesion. In the human endometrium, it contributes to the regulation of local tissue activity by promoting the enzymatic breakdown of biologically active peptides, including endothelin-1. CD10 expression is a recognised feature of both normal and endometriotic stromal cells, and its expression increases under the influence of progestins during decidualisation.[16]

Recent research indicates that inflammatory factors could affect stem cell behaviour within the fallopian tubes. Despite this, the precise role of inflammation in tubal and endometrial stem cell populations remains an area of active research. It is now generally accepted that endometriosis is a chronic inflammatory disease characterised by immune activation and cytokine release, mediated by fibroblast proliferation and subsequent fibrosis and adhesion formation, leading to pain and organ dysfunction.[3] However, histological characterisation of endometriosis helps guide accurate pathologic diagnosis, especially when concomitant or otherwise unclear phenotypes are present in gynaecological samples.[8]

CONCLUSION

The current study provides evidence that tubal endometriosis may be under-recognised in routine hysterectomy and salpingectomy specimens for benign non-pregnancy-related gynaecological conditions and warrants careful histological examination. Emerging data show that involvement of the endometrial lining and tubal mucosa in disease pathogenesis leads to the generation of lesions in which normal tubal function is disrupted, and clinical manifestations are likely to occur.

Understanding the cellular and molecular basis of these processes would be needed to clarify the aetiological mechanisms of disease, thus enabling a more finely tuned and specific therapeutic approach to be designed. The fallopian tube and endometrium have a consistent embryologic origin and cellular features. They are therefore compatible and appear to act as an active mediator of both the onset and progression of endometriosis. This highlights the need for heightened clinical and pathological awareness of tubal involvement and for further work to elucidate the effects of different types of tubal endometriosis on tubal physiology, reproductive function, and fertility outcomes.

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