Accessory or ectopic breast tissue persisting from embryologic development is found in about 2-6% of females and 1-3% of males. Hormonal stimulation at the time of menarche, pregnancy and lactation all directly influence the proliferation of breast tissue proper and at the ectopic site. Patients with accessory breast tissue present in the reproductive age group usually. Presentation in menopause is rare. We report a case of 51 years old post menopausal female who presented with the complaint of 5 x 7cm lump in right inguinal region. Histopathology examination revealed it to be an ectopic breast tissue in the inguinal area. Interestingly fibrocystic changes were also seen in the tissue. The patient was cured of the symptoms after surgical removal of the lump.

KEY WORDS: Ectopic Breast Tissue, Fibrocystic Disease, Post Menopausal Age

INTRODUCTION

Polymastia, accessory or ectopic breast tissue is the remnants of mammary streaks. The incidence of accessory or ectopic breast tissue is about 2-6% in females and 1-3% in males. However near about 67% of such conditions are seen in either the thoracic or abdominal portions of milk line. While 20% of the cases they are found in axillary regions. Other common ectopic sites includes the posterior neck, face, chest, shoulder, buttock, vulva, hip, posterior and/or lateral thigh and perineum. Rarely bilateral accessory breasts are also reported. They can either consist of functional or non functional components of a complete or partial breast tissue.

In view of all this, the current case report has been written to highlight the fact that accessory breast tissue with fibrocystic disease must be considered amongst the differential diagnosis of inguinal swellings.

CASE REPORT

Fifty one years old female patient presented with the complaint of having soft and mobile mass in right inguinal region since three years. For the last two months the size of mass has been increasing. She had a menopause three years back. There is no significant medical or surgical history. On examination of specific area overlying normal skin was soft to firm, mobile and non-tender mass with a size of 09 x 09 cm was noticed. The excision biopsy with tissue histopathology was advised. The gross examination revealed soft to firm nodule of 09 x 09cm. It was well circumscribed, grey white in colour. Its cut section was solid with dilated spaces and whitish fluid oozed out. This is shown in Fig -1.

On microscopic examination benign breast tissue was identified along with fat. The breast tissue was composed of normal tubules and ducts of breast; dilated cystic spaces lined by benign ductal epithelial cells were seen. These spaces were filled by proteinaceous material and macrophages. Apocrine metaplasia was also seen in few ducts (Fig-1 & 3). Thus, the diagnosis of inguinal breast tissue with fibrocystic disease was confirmed.

**FIGURE–1: GROSS APPEARANCE OF INGUINAL MASS. SOLID CUT SURFACE WITH CYSTIC SPACES.**

**FIGURE–2: ECTOPIC BREAST TISSUE COMPOSED OF BENIGN LOOKING DUCTS.**
REFERENCES


DISCUSSION

The intrauterine development of breast tissue in a fetus is from the ectodermal ridges (milk lines) having extension from the axillae to the inguinal regions. These regress spontaneously during the process of embryogenesis except for the pair at the pectoral region which forms mammary glands or tissues in adults.

The presence of specific symptoms like the discomfort, pain and milk secretion is directly under the influence of hormonal stimulation. Just like the normal breast tissue, they have the potential to give rise to benign proliferative processes or malignant carcinomas of breast tissue.

The presentation of accessory breast tissue mimics lymphadenopathy, lipoma, sebaceous cyst, vascular malformation, hidradenitis, or malignancy. In order to pick up the proper diagnosis options like needle or excision biopsy must be considered in early course of disease. Awareness regarding the timely diagnosis is important because these tissues harbours the same potential of developing a malignancy as an ordinary one. Malignant transformation in fibrocystic disease is rare however about 5% of these cases involve the type of changes that would be considered a risk factor for developing breast cancer. Therefore the surgical resection is necessary.

Due to the deficiency of literature review we cannot correlate the presence of accessory inguinal breast tissue with fibrocystic disease in post-menopausal age. However, it should be considered amongst the differential list in specific age group.

CONCLUSION

Accessory breast tissue with fibrocystic disease must be considered amongst the differential diagnosis of inguinal swellings.
Endometriosis is a common gynecologic problem. Malignant transformation of endometriosis, though a rare event, is well documented. Synchronous endometrial and ovarian carcinoma either primary or metastatic is also uncommon. Here we report a case of endometriosis with concurrent ovarian and uterine endometroid carcinoma.

**KEY WORDS:** Endometriosis, Malignant Transformation, Endometrioid Carcinoma, Synchronous Carcinoma

## CASE REPORT

Fifty years old female presented with abnormal uterine bleeding. On investigation her uterus was enlarged and a solid mass was present in one of the ovaries. She underwent total abdominal hysterectomy with bilateral salpingoophrectomy. The specimen received in the laboratory consisted of uterus with cervix measuring 8x6x5cm, ovaries 3x3 and 2x2 cm each and tubes, each measuring 5x0.5cm. A white, necrotic growth was present in the uterine cavity measuring 4x3cm, extending 0.5cm into the uterine wall. One ovary contained white, necrotic growth while the other had small cysts (Fig-1). A piece of omentum measuring 4x3x0.5cm was unremarkable on cut section. A well differentiated endometroid carcinoma was diagnosed in the uterus and right ovary (Fig-2, 3), along with right ovarian endometriosis (Fig-4). Adenomyosis was present in the uterus. The tumor in the ovary and uterus were histologically similar. Both consisted of well differentiated back to back neoplastic glands, scant stroma, invasion and squamous nests. Uterine tumor involved less than 1/3rd of the myometrium. Sections from fallopian tubes and omentum were unremarkable. The case was diagnosed as

- Endometroid carcinoma Rt. ovary (well differentiated)
- Rt. Ovarian endometriosis
- Endometroid carcinoma uterus (well differentiated) involving less than 1/3rd of myometrium

**FIGO Stage:** Ovarian carcinoma T1, Uterine carcinoma T1b
the endometrium. There are significant implications in regard to therapy and prognosis.

Ulbright and Roth et al, proposed pathologic criteria for differentiation between primary and metastatic ovarian carcinoma in 1985 which was further elaborated by Scully et al (Table I & II). Features supporting a primary uterine carcinoma includes either a multinodular ovarian pattern (major criterion) or two or more of the following minor criteria:

- Small (less than 5 cm) ovary (ies),
- Bilateral ovarian involvement,
- Deep myometrial invasion, vascular invasion
- Tubal lumen involvement.

Synchronous lesions are favored when the endometrial carcinoma is minimally invasive and small. An endometrial primary with metastasis to the ovary is most likely when the ovarian tumors are multiple and less than 5 cm in greatest dimension, and when deep myometrial invasion and vascular invasion are present. In our case uterine endometrial carcinoma is well differentiated and histologically resembled the ovarian counterpart. But it was superficial and no vascular or deep myometrial invasion was seen. There was no bilateral involvement of ovaries and carcinoma was present in the Rt. ovary which measured about 3x3cm. No multinodular involvement or tubal involvement was seen. Based on these
observations we assume that the carcinogenic process which led to cancer formation in the ovary from endometriosis, also affected the endometrial glands of the uterus leading to carcinoma. According to theory of secondary mullerian system the epithelium of female genital tract and peritoneal surface have shared molecular receptors responding to carcinogenic stimulus leading to the development of multiple primary malignancies synchronously.19

Early stage and low histologic grade are also the characteristics of synchronous endometrial and ovarian tumors, especially in subtype of endometrioid histology, the prognosis of synchronous tumors is better than metastatic lesions.16,15 In our case both the ovarian and the uterine tumor were histologically low grade and low stage.

Along with the above mentioned gross and histologic criteria, other special techniques can be used to differentiate metastases from primary lesions.15,17 Concomitant tumors with exactly the same morphology arising in ovary and endometrium, especially the endometrioid carcinoma, will show the same immunohistochemical expression patterns.18 But Halperin et al reported that 62.5% of synchronous primary endometrial and ovarian cancers can be classified by detection of ER and PR content and that 31.3% of synchronous primary endometrial and ovarian cancers can be detected by Bcl-2, which is positive in uterine cancer metastatic to ovary.19 Molecular profiling in synchronous endometroid and ovarian cancers may aid in determining a differential diagnosis.15-17 A recent investigation of loss of heterozygosity suggested that a higher rate of loss of heterozygosity was detected in patients with synchronous lesions than in patients with single tumors.20 Somatic mutations of the β-catenin and PTEN genes are the most common genetic abnormalities identified in ovarian endometroid carcinomas. Compared with uterine endometroid carcinoma, ovarian endometroid carcinoma have a similar frequency of β-catenin abnormalities but lower rate of microsatellite instability and PTEN alterations.20,21

CONCLUSION

Synchronous ovarian and uterine cancer is very rare presentation. Careful gross and microscopic examinations can aid to differentiate and identify synchronous ovarian and uterine cancer in a patient. Axillary studies can also add to the diagnosis. This workup has significance in patient care and therapy as well as identifying deeper aspects of tumorigenesis in such cases.

REFERENCES


