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Sclerosing Adenosis : A Dignostic Pitfall in Breast Carcinoma

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ABSTRACT

Sclerosing adenosis (SA) is a proliferative lesion that is commonly found in benign breast biopsies. The identification of increased cancer risk for patients with SA suggests that tumorigenic alterations may be associated with the pathogenesis of this lesion. We here report a case of old female who presented with a breast lump. Trucut biopsy proved it to be a case of sclerosing adenosis, later on lumpectomy evidence of mixed Ductal and Lobular carcinoma.

Key words : *Benign breast disease, Breast carcinoma, Sclerosing adenosis, Ductal carcinoma, proliferative breast disease.*

INTRODUCTION

Sclerosing adenosis (SA) is a common benign breast lesion demonstrating increased numbers of distorted lobules accompanied by stromal fibrosis. The presence of SA stratified risk in subsets of women defined by age, involution status, and family history. However, SA does not further

stratify risk in women diagnosed with other forms of proliferative breast disease, either with or without atypia. SA conveys an approximate doubling of breast cancer risk. Its role in breast carcinogenesis remains undefined; its presence may aid in risk prediction for women after a breast biopsy. The identification of increased cancer risk

for patients with SA suggests that tumorigenic alterations may be associated with the pathogenesis of this lesion.¹

The clinical, radiological and histopathological properties of sclerosing adenosis may resemble the masses that are usually associated with malignancy, which is the factor responsible for the clinical significance of the disease.² We reported sclerosing adenosis on trucut biopsy of breast lump which revealed intraductal carcinoma with foci of lobular carcinoma and DCIS in same lesion.

CASE REPORTS

Forty seven years old female presented with history of lump in right breast for 5 months. No history of local trauma, pain and discharge. No family history of breast cancer was present. No similar episode was present. On local examination, swelling was firm to hard measuring 3x2 cms. Swelling was not freely mobile.

On mammography, thickened fibroglandular tissue seen in upper quadrant of breast at 10 to 11 o'clock position measuring approximate 3x2.8 cms showing no significant flow on color Doppler- ?fibroadenosis (BIRAD III). Accessory breast seen in bilateral axillary region.

Fine needle aspiration of lump was done. On smear examination, only fibroadipose tissue was obtained even after repeated aspiration. Biopsy was advised for this lesion. Trucut biopsy sample measuring 1x0.8 cms, was sent to our pathology department. On H&E examination a diagnosis of sclerosing adenosis was made. Later lumpectomy

was done for breast lesion for rule out breast cancer. On H& E examination of representative areas of lump showed infiltrating ductal carcinoma in overall background of sclerosing adenosis. A few foci of lobular carcinoma and DCIS are also identified. On immunohistochemistry, tumor was negative for ER, PR and Her-2-neu markers.

DISCUSSION

Sclerosing adenosis (SA) is a proliferative lesion that is commonly found in benign breast biopsies. The identification of increased cancer risk for patients with SA suggests that tumorigenic alterations may be associated with the pathogenesis of this lesion. It is a histologically complex entity that consists of enlarged and distorted lobules, containing duplicated and crowded acini, with prominent myoepithelium and stromal fibrosis. Although the cellular composition can be difficult to appreciate due to architectural distortion, the acini in SA include abundant myoepithelial, in addition to luminal epithelial cells. The process is usually accompanied by stromal fibrosis.

As such, SA combines proliferation of epithelial, myoepithelial, and mesenchymal cells, which differs from the more homogeneous proliferation of luminal epithelial cells seen in usual ductal hyperplasias. This concurrent aberration of epithelial and mesenchymal compartments in SA appears to set the stage for a higher likelihood of subsequent malignancy. There are likely phenotypic changes in these cells resulting from

microenvironmental signals that stimulate progression to more advanced stages of BBD or carcinoma.¹

It has been shown that myoepithelial cells in DCIS show decreased expression of genes involved in normal cell function and increased expression of genes that stimulate proliferation, migration, invasion, and angiogenesis; similarly, DCIS-associated mesenchymal cells can drive progression to invasive disease through deposition and modification of extracellular matrix molecules and through recruitment of other stromal components.¹ Increased breast cancer risk associated with SA suggests that key pretumorigenic alterations are already occurring in some of these patients. Identification of those alterations associated with progression to cancer could advance risk prediction for women with SA, and provide¹

It could be associated with microcysts, apocrine metaplasia, luminal histiocytes and pseudopapillomas, called glomeruloid structure.² It poses a diagnostic confusion with infiltrating lobular carcinomas and tubular carcinomas even in histological sections, but is identified by the relative preservation of overall lobular architecture, the compressed glands, lack of atypia, the retention of two cell layers, and the confirmation of the presence of myoepithelial cells by immunohistochemistry. However, in tissue sections, especially core biopsies, the identification of myoepithelial cells and basement membrane by immunohistochemistry is really useful in difficult cases. Although core needle

biopsy provides a definite diagnosis in most cases, a coexisting cancer may be missed due to sampling error. It is important to distinguish sclerosing adenosis from other benign lesions due to the fact that it is associated with small but definite risk of invasive carcinoma and the risk increases if it is associated with atypical hyperplasia.^{2,3}

Relative risk of cancer associated with SA is 1.3-1.9 in various studies which is similar to fibroadenoma and intraductal papilloma. Relative risk is more than 4 with atypical hyperplasia associated with benign breast disease.⁴⁻⁶

Lobular carcinoma in situ (LCIS) is another relatively frequent condition found with sclerosing adenosis, and in this case, differentiating the lesion from invasive carcinoma is much more problematic.²

This may produce 2 patterns that resemble invasive lobular carcinoma. First, if the LCIS is florid, marked expansion of the underlying sclerosing adenosis may cause fusion of the lobules, obscuring the surrounding myoepithelium, intervening stroma, and acinar architecture, thereby producing a low magnification appearance of a near-solid sheet of invasive tumor cells. A second pattern that may mimic stromal invasion at high magnification is the appearance of narrow cords and compressed tubules involved by LCIS within stroma. Without appreciating the low magnification architecture of underlying sclerosing adenosis, this pseudo-infiltrating pattern could be mistaken for invasive lobular carcinoma, To distinguish LCIS in

sclerosing adenosis from invasive cancer, the same approach is advised as for distinguishing sclerosing adenosis from invasive cancer: recognition of overall smooth lobular contours of the proliferation; swirling, streaming distribution of acini; recognition of myoepithelium, either on H & E stain or myoepithelial immunostaining. Often the basement membrane around each acini still can be seen even if the myoepithelium is compressed beyond recognition. Presence of adjacent uninvolved sclerosing adenosis is another clue. These features may not be easily appreciated in a core needle biopsy and so myoepithelial immunostaining is prudent before diagnosing invasive lobular carcinoma if there is any hint of sclerosing adenosis in the biopsy.⁷

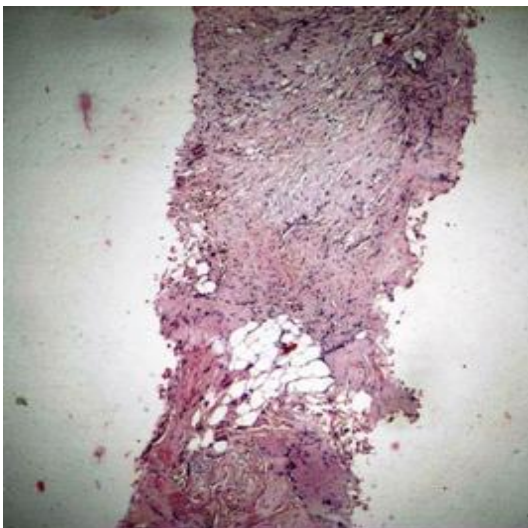


Fig.1 Microscopic view of trucut biopsy (H&E, 100x)

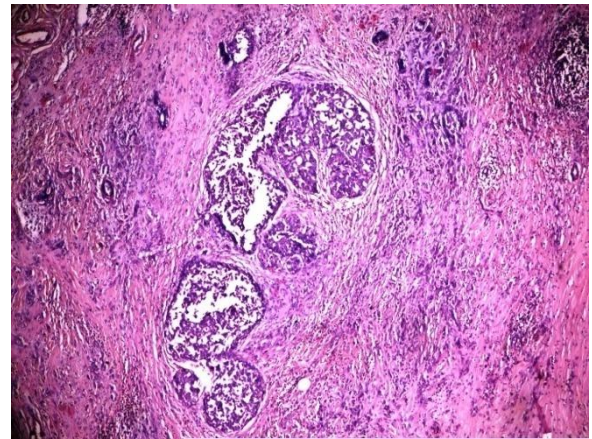


Fig.2 Microscopic view showing DCIS foci (H&E, 40x)

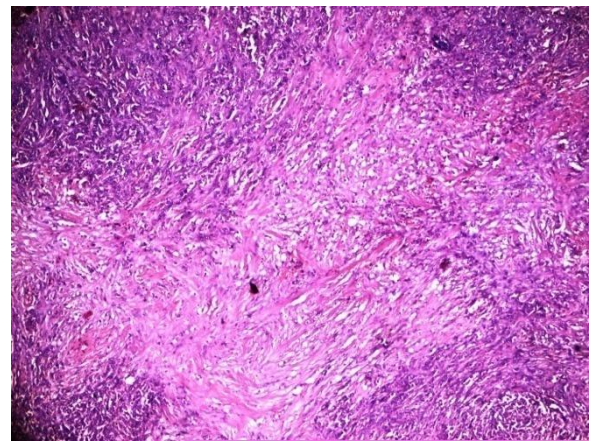


Fig.3 Microscopy shows Lobular Carcinoma foci (H&E, 40x)

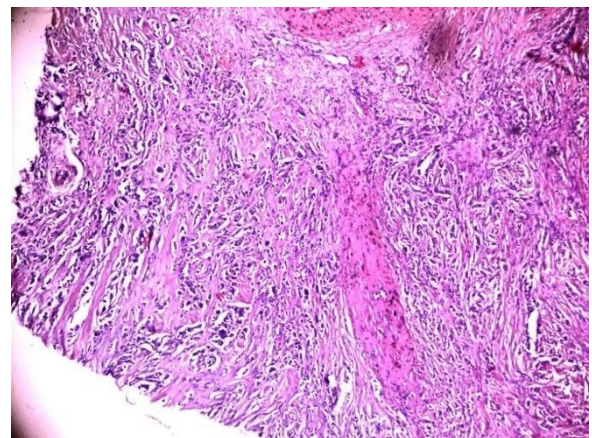


Fig.4 Microscopy shows Infiltrating ductal carcinoma (H&E, 40x)

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