Well differentiated clinging carcinoma and atypical ductal hyperplasia are synonim.

Azzopardi in 1979 (1)described a type of in situ duct carcinoma that he named “clinging” to indicate that neoplastic cells are “attached” to the duct walls. One or two layers of neoplastic cells line the ducts in a mural fashion. Azzopardi (1) stated that this form of growth constitute the first neoplastic change that can be appreciated at light microscopy. He also implied that the neoplastic process might figure a continuous progression from normal epithelium to frank malignancy but most of the neoplastic progression develops at submicroscopic level. In conclusion he stated that clinging carcinoma was the earliest morphological recognizable sign of malignancy. De Potter et al.(2) later found that there are two types of Clinging Carcinoma (C.C.), i.e. one with monotonous , regular nuclei and one with pleomorphic nuclei. This latter form displayed cells positive with Erb-c2 and the implication was that pleomorphic C.C. was the precursor of poorly differentiated DCIS, while the form with monotonous nuclei was the precursor of well differentiated DCIS. Tavassoli (4) has shown that C.C., named DIN Ic, has the same pattern of LOH as invasive duct carcinoma and therefore C.C. also at molecular level appears to be a neoplastic process.

Page et al. in 1985 (5) introduced the term of atypical duct hyperplasia (ADH) , indicating a lesion that was bearing a risk of developping a duct carcinoma 4 times higher than a normal patient. The precise morphological definition was never definetely given and it was modified in the years till recent times in which by admission of David Page himself, most of the lesions regarded as atypical duct hyperplasia recognize the phenotype of CC with monotonous nuclei.

The similarity of the two lesions is also evident at molecular level as Lakhani et al.(3) have found similar LOH changes between ADH and in situ and invasive carcinomas.

It has also been found that the relative risk of subsequent carcinoma is very similar between ADH and CC ,i.e. in the order of 2-4 times the risk of developping a subsequent carcinoma.

It is proposed that all these lesions (i.e. CC-ADH-DIN Ic) are different names of the same entity , they have to be followed up and no specific treatment is necessary.

Reference List

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