Ductal carcinoma in situ (DCIS)

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Ductal carcinoma in situ (DCIS) is a malignant clonal proliferation of cells growing to breast ducts, with no evidence of invasion into the surrounding stroma. The recognision of DCIS as a specific disease occurred during the first half of the 20th century; until then it was rare, accounting for only 1-2% of newly diagnosed breast cancers. Currently, it comprises approximately 20-25% of breast cancer cases. Very few cases of DCIS present as a palpable mass; most are diagnosed by mammography, usually as clustered microcalcifications. DCIS may also present as pathologic nipple discharge, with or without a mass. The frequency of diagnosis of DCIS has greatly increased with the wide use of digital mammography.

It is generally accepted that DCIS is not one entity but an heterogeneous group of lesions at clinical, radiological, histological and genetical level. This heterogeneity adds complexity to the Clinician's and Pathologist's role, in managing accurately the patients, in macroscopic specimen handling, in microscopic diagnosis and in prognostic feature assessment. The microscopic heterogeneity of DCIS has lead to the development of a number of systems for classification based on architectural growth pattern, nuclear morphology and on the presence or absence of necrosis. The type of architectural pattern, the high nuclear grade, the presence of luminal necrosis, the size/extent of the lesion, the young age of patient and the symptomatic clinical presentation have been reported to correlate with the likelihood of local reccurrence. The above pathological prognostic parameters as well as the distance to excision margins/margin status should all be included in the histopathology report.

Current thinking is that most invasive breast carcinomas (IBCs) evolve through a nonobligatory series of "stages" over long periods of time, probably decades in most cases. In order, the stages include ductal hyperplasia, atypical ductal hyperplasia and DCIS. DCIS is considered as a direct precursor of most IBCs. Many types of evidence support this hypothesis: histologically, most IBCs are accompanied by DCIS,

immunohistochemically and genetically DCIS and IBCs share many abnormalities, especially when they are detected in the same breast. The similarities extend even to global gene expression profiles, where DCIS has been classified in molecular subtypes, identical to the molecular subtypes of IBDs. Recently, a lot of research has been done concerning the cellular and molecular mechanisms by which the stromal cells must cooperate with the tumor cells of the in situ lesions for invasion.

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