frequent mitotic figures. On immunohistochemistry, the cells are positive for CD117, vimentin and BCL2. Wnt-APC beta-catenin pathway is implicated in the pathogenesis of these tumours with 72% of cases in a study showing stromal nuclear staining by beta-catenin [3]. They are usually benign and only few of them undergo malignant transformation and p53 gene mutation is the most common abnormality seen in this condition. Stromal p53 expression has almost consistently been reported to increase significantly with phyllodes tumour grade [4]. In a study frequent gain of 1q, deletion/copy-neutral LOH in chromosome 13 along with +7p and -9p were found in in borderline and malignant tumors using array CGH. The study found that in the borderline/malignant cases with 9p deletion, the deletion localized to 9p21.3, the site of p16INK4a [5]. Figure 1 shows a summation of the correlations of immunohistochemical/genetic changes in the progression of phyllodes tumors [5].

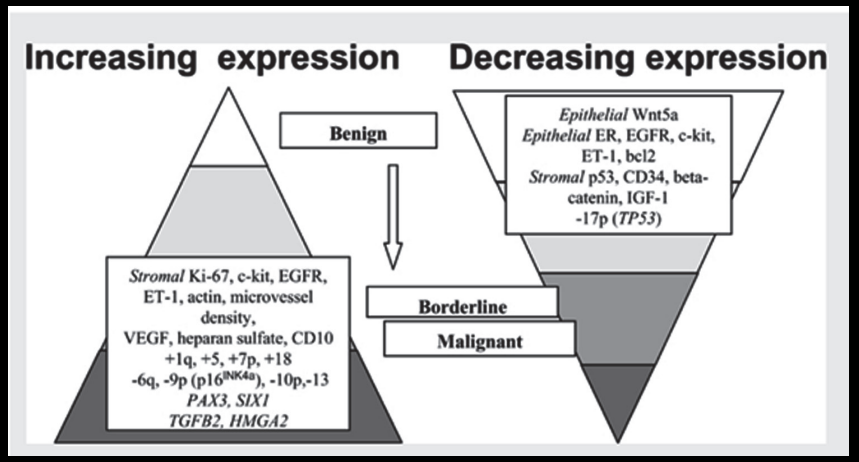

Fig 1

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