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Editorial on MicroRNA Dysfunction in Cervical Pre-cancer Development

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Editorial

Early detection of precancerous lesions and comprehension of risk factors are critical in preventing cervical cancer. Cervical cancer incidence and death rates in the United States have decreased by more than half since the mid-1970s, owing in part to increase screening, which allows cervical changes to be detected before they become cancerous. Although the human papillomavirus (HPV) vaccine protects women against cervical cancer, exposure to HPV before vaccination and HPV infection types not covered by the vaccine result in low vaccination effectiveness. Many women are only protected by bivalent and quadrivalent HPV vaccines, and they may engage in sexual activities prior to receiving the vaccine. The Pap test remains the primary method of protecting them from cervical cancer [1-3].

Cervical cancer develops in a multifactorial, multi-step process that begins with the transformation of normal cervical epithelium into cervical intraepithelial neoplasia (CIN) or squamous intraepithelial lesion (SIL), a non-invasive and pre-malignant lesion that progresses to invasive cervical cancer. The term SIL is used to describe the cytology test result, whereas CIN is used to report cervical biopsy results. The Bethesda system categorises SIL into two levels: low-grade SIL (LSIL) and high-grade SIL (HSIL) (HSIL). HSIL is considered the most severe pre-cancerous stage, while LSIL is considered less serious. LSIL cervical cells show karyomegaly, perinuclear halo, and binucleation (koilocytes); in contrast, HSIL cervical cells show intense binucleation and dyskaryosis in basal parabasal cells with little cytoplasm, hyper chromatic nuclei, and prominent nuclei.

Squamous cell carcinoma (SCC) would eventually develop from SIL. Squamous cell carcinoma is the most common type of cervical cancer, accounting for 70-80 percent of cases. Biopsy results, which classify CIN into CIN1, CIN2, and CIN3, describe the actual changes in the cells, as opposed to the cytology test, which is only a screening test. CIN1 is a low-grade CIN (LG-CIN), whereas CIN2 and CIN3 are high-grade CINs (HG-CIN). Early detection and treatment of LG-CIN and LSIL would reduce the incidence of cervical cancer significantly. As a result, the current review will concentrate on the cervical pre-cancer stage rather than the later cervical cancer stage. Two systematic review articles published in 2015 and 2017 investigated miRNA dysregulation during cervical cancer progression. A meta-analysis of studies published before October 2014 was included in He's study.

When CIN1 was compared to normal tissues, a total of 12 miRNAs were found, 7 of which were down-regulated and 5 of which were up-regulated. Because only mild cellular and nuclear abnormalities were found in CIN1 tissue, and the abnormalities are usually transient, a small number of miRNAs [4,5] are expected to be dysregulated. Some miRNAs have been found to be dysregulated on multiple occasions. Up-regulation of miR-21 on cervical cancer progression was found in five studies in Pardini's review. Nine miRNAs were found to be up-regulated in three studies, and eight miRNAs were found to be up-regulated in two studies. In terms of under-expressed miRNAs, miR-218 was linked to cervical cancer progression in six studies, while miR-375 and miR-203 were linked in four, and six miRNAs were linked in three.

When normal progresses to HSIL, miR-let-7b expression decreases gradually. In addition to Chen's study, two miR-let-7 family members, miR-let-7e-3p and miR-let-7b, were found to be down regulated throughout the progression of cervical cancer. MiR-let-7 is made up of a total of 12 members. MiR-let-7 is a well-known tumour suppressor. The fourth study looked at the relationship between HPV viral protein E6, p53, and miR-22. MiR-22 was discovered to be one of p53's direct transcriptional targets in synovial cells. In cervical cancer cells, the oncoprotein E6 suppressed the p53 protein. The binding of E6-E6AP with p53 caused p53 degradation. The study used clinical tissue and cell models to depict the E6-p53-miR22 pathway.

Conflict of Interest

None.

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