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Commentary

Analysing the Consequences of Carcinogenicity in the Patients of Oral Mucosa Cancer

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DESCRIPTION

Depicts an early oral Squamous Cell Carcinoma (SCC) that was once thought to be an amalgam-related lichenoid reaction.

Most head and neck cancers are caused by mouth (oral) cancer, which is a common neoplasm globally. Theoretically, given that the mouth is simple for patients and healthcare professionals to access and inspect, provided they have appropriate illumination, it should be mostly preventable or detectable at an early stage.

Early oral cancer frequently has no symptoms, which delays diagnosis. Any single ulcerated lesion that persists for more than two to three weeks needs to be taken seriously, and a biopsy should be done. A diagnosing aid is the acronym RULE (red, ulcerated, lump, extending for 3 or more weeks).

In the underdeveloped world, oral SCC is very prevalent, mostly in elderly males. Human Papilloma Virus (HPV) infection has been linked to a continued rise in younger patients, particularly women, without established risk factors, especially in the oropharynx. Oral SCC appears to have a complex aetiology that is heavily influenced by lifestyle factors, primarily habits (especially tobacco smoking alone or in conjunction with betel use, alcohol use, and/or poor diet). Particularly in cases of oropharyngeal cancer, additional variables including infectious agents may potentially be at play (HPV). Certain cases of SCC are caused by immune disorders or immunosuppression, metabolic disorders involving carcinogens, or disorders involving DNA-repair enzymes. Lip cancer is predisposed by sun exposure.

The key signs of oral SCC are findings from the history and clinical examination by a qualified dentist (oral medicine specialist), however even when the clinical picture is congruent with oral SCC, the diagnosis must always be histologically validated with tissue biopsies.

Modern DNA technology has discovered chromosomal variations in oral SCC that are suggestive of the involvement of Tumour Suppressor Genes (TSGs), specifically in chromosomes 3, 9, 11, and 17. Functioning TSGs seem to support growth control, while their mutation can unbridle these control mechanisms.

In addition to harming TSGs, cancer may also harm other growth-controlling genes, primarily oncogenes (cell signalling genes), particularly those on chromosomes 11 (PRAD1 in particular) and 17. (Harvey ras [H-ras]). Modifications in these and other oncogenes may impair the regulation of cell development, which may ultimately result in the unchecked spread of cancer. One of the oncogenes that initially attracted the attention of molecular biologists with an interest in cancer, cell signalling, and cell growth control was H-ras. It has a role in cell signalling, along with the gene encoding the epidermal growth factor receptor.

Some patients have enzymes that metabolise carcinogens. Alcohol dehydrogenase oxidises ethanol to acetaldehyde, which is cytotoxic and produces free radicals and DNA hydroxylated bases; genotypes of alcohol dehydrogenase type 3 appear to be associated with oral SCC. Several environmental procarcinogens can be activated by cytochrome P450. Cytochrome P450 IIEI (CYP2E1) partially metabolises ethanol to acetaldehyde. Oral SCC may be predisposed to by mutations in specific TSGs, which may be linked to cytochrome P450 genotypes. The action of Glutathione S Transferase (GST) genotypes may be compromised; for instance, GSTM1's null genotype has a reduced ability to detoxify tobacco carcinogens. Oral SCC has been shown to be predisposed to by some GSTM1 and GSTP1 polymorphism genotypes as well as GSTM1 and GSTT1 null genotypes. Procarcinogens are acetylated by the N-acetyltransferases NAT1 and NAT2. At least in some groups, N-acetyl transferase NAT1*10 genotypes may be a genetic determinant of oral SCC.

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