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(REVIEW ARTICLE)

The role of p53 mutant protein as a predictor of oral cavity malignancy: Narrative review

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Abstract

Background: The incidence of oral malignancy has a high prevalence in the world. The incidence of oral malignancies, commonly called OSCC because 90% of cancers are found in squamous cells when viewed histologically, exacerbated by the encouragement of risk factors, such as consuming alcohol, smoking, and other factors. Several recent studies have found that p53 mutant can be used as a predictor of oral malignancy.

Objective: To describe the function of mutant p53 as an indicator for the potential development of oral cancer.

Methods: This paper uses a literature review method. The literature sources used to collect references were Pubmed and Science Direct using the keywords "p53 mutant and oral malignancy."

Discussion: Based on the references that have been found, p53 mutant is found in several oral malignancies, such as HNSCC, OSCC, LSCC. The presence of p53 mutant can be detected using immunohistochemistry and PCR-SSCP. P53 mutant is also associated with other components in cancer progression. For example, p53 mutant contributes to the regulation of mi-R 182 in cancer. P53 mutant can be used as an assessment of cancer therapy. In cancer therapy using cisplatin, the high expression of p53 mutant makes it resistant to this therapy.

Conclusion: P53 mutant can be used as a predictor of oral malignancy. The function of p53 which should minimize the occurrence of cancer does not function in p53 mutant. Mutant p53 exerts a dominant-negative impact and acquires oncogenic properties that contribute to cancer progression.

Keywords: Oral Cavity Malignancy; P53 Mutant; Gain-Of-Function; Apoptosis; Good Health and Well Being

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1. Introduction

Cancer remains one of the most prevalent diseases worldwide. In 2020, there were 19,292,789 new cases reported globally, with 9,958,133 deaths. In Indonesia, 396,914 new cases and 234,511 cancer-related deaths were recorded[1]. The rising prevalence of cancer is attributed to increasing risk factors such as smoking, obesity, physical inactivity, and infections[2].

Oral cancer also shows a high global prevalence and is associated with a significant mortality rate. Globally, an estimated 377,713 new cases and 177,757 deaths were recorded in 2020. In Indonesia, 5,780 new cases were reported, with 3,087 deaths[3].

Cancer arises from disturbances and modifications in cellular functions, resulting from the buildup of genetic and epigenetic changes that cause genomic instability. Various risk factors can accelerate the progress of cancer development, including physical, chemical, and biological factors[4].

Physical factors that can cause cancer are exposure to ultraviolet, ionizing radiation, and electromagnetic fields. Epidemiological data indicate a link between minimal electromagnetic field exposure and breast cancer risk. Ionizing radiation is a carcinogen that has the ability to induce growth in all organs. This condition can cause cancer to occur spontaneously. Meanwhile, ultraviolet radiation is a common factor that often occurs in the environment. This radiation often affects the skin with dangerous signs[4].

Chemical factors that often cause cancer are the use of tobacco cigarettes, consuming alcohol, and other chemicals. Tobacco products used in cigarettes are a source of various carcinogens, such as nitrosamines, tar and other toxic factors. Active and passive smokers adversely affect health and increase the risk of cancer. The risk of cancer depends on the amount and type of alcohol consumed and other factors[4]. People who smoke heavily and frequently consume alcohol have a high risk of developing Oesophangeal Squamous Cell Carcinoma. Stephen et al. stated that active smokers who consume >15 g/day of alcohol have an 8 times higher risk of developing Oesophangeal Squamous Cell Carcinoma compared to non-smokers who consume 0-5 g/day of alcohol. Meanwhile, non-smokers who consume >15 g/day of alcohol have a 4-fold higher risk of developing Oesophangeal Squamous Cell Carcinoma compared to non-smokers who consume 0-5 g/day of alcohol[5].

Biological factors that are often associated with cancer are diet, physical activity, and infection. The development of the era has resulted in the development of types of food that are now increasingly found to contain toxic substances that are carcinogenic. Wrong diet can cause obesity, which is also suspected of being a cause of malignant tumors. Lack of physical activity is considered a cancer risk factor through mechanisms such as increased body weight[4]. Infectious agents are responsible for approximately 15% of cancer cases globally. These include viruses and bacteria, such as Human Papillomavirus (HPV), which is linked to cervical cancer, and Epstein-Barr Virus (EBV), which is associated with nasopharyngeal cancer. *Neisseria gonorrhoeae* bacteria are associated with prostate and bladder cancer, and *Chlamydia trachomatis* bacteria are associated with ovarian cancer[6].

Along with the development of technology, oncology has entered a new era by utilizing the immune response as a cancer biomarker. Previously, oral cancer patients could only be detected when they had entered an advanced stage through visible clinical signs. This condition resulted in delayed treatment because the cancer had entered an advanced stage which caused the patient to have a relatively low life expectancy. The existence of cancer biomarkers can be a solution to detect cancer early so that treatment can be carried out immediately which makes the patient's life expectancy higher[7].

Several cancer biomarkers have been discovered and used clinically to monitor the stage of cancer development. One of these biomarkers is carcinoembryonic antigen (CEA) which has been studied since 1965. Carcinoembryonic antigen (CEA) is a glycoprotein involved in cell adhesion, commonly expressed during fetal development. It is produced by fetal intestinal tissues and certain epithelial tumors, which explains why elevated levels can also be found in some non-malignant conditions, including inflammatory bowel disease. In healthy adults, CEA expression is typically suppressed, resulting in very low levels. However, abnormal CEA expression may occur in various cancers. Therefore, CEA is considered a valuable tumor marker for distinguishing diagnoses, tracking disease progression, and assessing therapeutic response[8].

The use of CEA as a tumor biomarker has limitations, particularly its low sensitivity and specificity, as CEA levels can vary widely across different types of cancer. In people who smoke, it is necessary to consider CEA levels because

smokers' CEA levels are higher than non-smokers. Age also affects CEA levels. In the elderly, CEA levels tend to be higher than in young people[9].

Mutant p53 is recognized as a critical biomarker in diagnosing and assessing the prognosis of cancer. Its elevated expression across various cancer types is often associated with increased disease severity. The mutation of the p53 gene disrupts its normal role in halting the cell cycle at the G1 phase and initiating apoptosis. This disruption leads to the continuous accumulation of damaged genes and uncontrolled cell proliferation, which are defining traits of cancer[10]. In cases of Head and Neck Squamous Cell Carcinoma (HNSCC), mutant p53 expression is associated with accelerated tumor progression and greater lymph node involvement. A study involving 20 patients with invasive HNSCC reported the absence of mutant p53 in clinically healthy mucosa, but its presence in dysplastic tissue. According to Zhou et al[11], these findings support the possibility that mutant p53 could serve as a prognostic marker for malignant transformation in the oral cavity. Therefore, the purpose of this study is to examine the predictive value of p53 mutations in the onset of oral cancer.

2. Methods

The keywords used in searching for references were "mutant p53 and oral malignancy" and "mutant p53 and oral malignancy." The sites used in searching for references were Pubmed and Science Direct which were collected in one folder with Mendeley. Inclusion criteria: references using English or Indonesian, references in the form of research, reviews, and can be accessed in full text, references published in the time span from 2016 to 2021. Exclusion criteria: references do not mention the theory of "mutant p53 as a predictor of oral malignancy" which is used as the basis for the study.

Definition of mutant p53: Continuous exposure to carcinogens can cause disruption of the p53 protein. p53 gene mutations produce a dysfunctional protein that loses its ability to regulate the cell cycle and trigger apoptosis. According to Irmawati et al[10], this state results in uncontrolled cell division and the ongoing formation of faulty genes, both of which are indicators of cancer.

Definition of oral cancer: Oral cancer refers to malignancies that develop in the lips and/or oral cavity, and it is predominantly classified histologically as Squamous Cell Carcinoma (SCC), accounting for approximately 90% of cases. Oral cancer is defined by the International Agency for Research on Cancer (IARC) as cancers that impact the salivary glands, floor of the mouth, lips, tongue, parotid glands, and gums[12].

3. Result

Table 1 illustrates the article selection process using specific keywords in PubMed and ScienceDirect. A total of 472 articles were initially identified, but 453 were excluded due to irrelevance to the topic of mutant p53 and oral cavity malignancy. Consequently, 19 articles were included in the final review.

Table 1 Overview of included studies on mutant p53 expression and its association with oral malignancy progression and therapeutic response

No.	Author, Year	Journal Name	Results	Theory	Conclusion
1.	(Escobar-Hoyos*et al.*, 2020)	Cancer Cell	Mutant p53 is the second most frequently found gene in PDAC with 40% mutations occurring. *Missense* and 30% deletion occur *frameshift.*	Mutant p53 in dysregulated RAS signaling in PDAC has the potential to lead to other malignancies.	The mechanisms of the two most commonly mutated oncogenes in human cancers, KRAS and mutant p53, may be therapeutically interconnected.
2.	(Gleber-Netto *et al.*, 2020)	Cancer	Tumors that have mutant p53 expression are at higher risk of developing	Cohort study in geographically diverse patients to determine mutant p53 as a risk stratification for OSCC.	There was an association between mutant p53 and OSCC in

			OSCC than tumors that have *wild*p53		two cohort studies which makes mutant p53 potentially useful as a biomarker.
3.	(Gohara*et al.*, 2021)	Molecular and Clinical Oncology	Of the 94 OSCC patients, there were 23 patients (23.4%) with positive Ap53Ab.	Wild-type p53 is quickly broken down in cells, resulting in a short half-life, whereas mutant p53 degrades more slowly, leading to its accumulation in the nucleus. This condition makes mutant p53 identifiable in immunohistochemistry.	Ap53Ab was re-evaluated as a tumor marker to better understand its potential utility.
4.	(Green *et al.*, 2018)	Annals of Oncology	Proteasome degradation, conformational restoration *wild*p53, autophagy can be used to suppress mutant p53.	Mutant p53 degradation has been considered as a therapeutic approach in cancer therapy.	The oncogenic properties of mutant p53, invasiveness and resistance to cancer therapy, can be modified for therapeutic benefit.
5.	(Hadzi-Mihailovic *et al.*, 2017)	Journal of B.O.U.N	p53 expression was found in 80% of OLP specimens detected using immunohistochemistry.	Overexpression of p53 is not only common in OSCC, but can also occur in dysplastic lesions. Low p53 expression in OLP on histological examination suggests that OLP can undergo malignant transformation.	P53 may be a useful biomarker in the diagnosis of OLP patients at risk for OSCC.
6.	(Irmawati *et al.*, 2020)	Malaysian Journal of Medicine and Health Sciences	Mutant p53 levels in group A was 2.58+0.8; group B was 14.75+1.57; and group C was 6.67+1.57.	This study demonstrates that moderate-intensity exercise can lower mutant p53 expression in group C compared to group B. However, the level of mutant p53 in group C is still higher than in group A. The expression observed in group A is attributed to passive exposure to cigarette smoke in the experimental animals.	Light-intensity exercise can stimulate the transcription of p53 mRNA, which subsequently leads to the translation and synthesis of wild-type p53.
7.	(Klinakis & Rampias, 2020)	EBioMedicine	The frequency of mutant p53 in metastatic tumors was lower (38.8%) than in primary tumors (71.5%).	Metastasis in Head and Neck Squamous Cell Carcinoma (HNSCC) involves a selective mechanism linked to p53 transactivation. Compared to primary tumors, metastatic HNSCC exhibits a lower incidence of mutant p53.	Mutant p53 levels in metastatic HNSCC were found to be lower than in primary HNSCC possibly due to metastases caused by HPV+ and EBV+ that do not contain p53 mutations.
8.	(Koehler *et al.*, 2016)	Annals of Oncology	10 of 19 patients were found to have mutant p53 expression and 6 of 19	Hypothetically, mutations in p53 contribute to tumor angiogenesis making tumors	Mutations of p53 or Rb1 are thought to contribute to the

			patients were found to have Rb1 expression.	more sensitive to antiangiogenic therapy.	angiogenesis of cancer cells, making them sensitive to antiangiogenic therapy.
9.	(Lindemann *et al.*, 2018)	Journal of Dental Research	13 of 14 patients had high mutant p53 expression. Meanwhile, 22 patients with *wild*p53 and 8 patients with low mutant p53 expression.	High expression of mutant p53 makes patients resistant to cisplatin therapy.	p53 mutations have been shown to be significantly predictive of response *platinum-based therapy*.
10.	(Nagata *et al.*, 2018)	Journal of Cutaneous Pathology	Mutant p53 tested positive in 80.5% of low-risk cases and 75% of high-risk cases.	Expression of p53 on immunohistochemistry is a classic indicator of malignant transformation. Mutant p53 detected in LSCC could signify extensive genetic damage and serve as a biomarker for malignant transformation.	PAb240 antibodies have the potential to be predictors of malignant transformation.
11.	(Rai *et al.*, 2017)	Annals of Diagnostic Pathology	36 of 60 oral cavity tumor samples had c-Myc and p53 expression, with 24 samples at an advanced stage.	There is overexpression of p53 and c-Myc in OSCC indicating that p53 and c-Myc are often dysregulated in HNSCC. This condition causes OSCC to develop faster by maintaining the cell proliferation process.	Overexpression of c-Myc and p53 may contribute to the development of OSCC and may be an indication of an aggressive tumor.
12.	(Sasaki *et al*., 2016)	Oncotarget	In cancer, p53 accumulation also influences ICAM2 induction.	Mutant p53 can reduce ICAM2 expression in cancer cells. Reduced ICAM2 expression has been linked to unfavorable outcomes in breast, lung, and bladder cancers.	p53-induced ICAM2 expression contributes to the suppression of cancer cell migration and invasion.
13.	(He *et al.*, 2021)	BMC Cancer	In the group with high mutant p53 expression, there was poor response to PD-1 blockade therapy.	P53 is a protein that is often mutated in HNSCC. These mutations can cause poor prognosis, making it an effective biomarker in evaluating prognosis and surveillance of HNSCC therapy response.	*Signature gene*combined with mutant p53 status may be a more effective method in evaluating prognosis and therapy response in HNSCC patients.
14.	(Singh *et al.*, 2016)	Mutation Research - Fundamental and Molecular Mechanisms of Mutagenesis	Of the 24 oral cancer samples, 11 samples contained mutant p53 only in cancer tissue, 6 patients contained mutant p53 only in healthy tissue, and 7 patients contained mutant p53 in both.	The variation of mutant p53 that occurs in certain tumors suggests that the environment plays a role in the mutation process.	Differences in geography and community habits lead to the discovery of p53 mutant variations. Evaluation of p53 mutant status can predict the early stage of oral cancer patients.

15.	(Tuna *et al.*, 2019)	Neoplasia	aUPD on chromosomes 4q, 5q, 9p, 9q, 13q, 17p and CDKN2A were more frequently found in patients with mutant p53 compared to wild p53 with HNSCC.	In cases of HNSCC, mutant p53 is more frequently found in smokers compared to nonsmokers, and is also more prevalent among HPV-negative patients than those who are HPV-positive	aUPD is associated with disease risk factors and the presence of mutant p53.
16.	(Wang *et al.*, 2016)	Journal of Oral Pathology and Medicine	15 of 71 patients with normal histology showed signs of recurrence.	P53 has limited value for predicting local recurrence consistent with other biomarkers. The combination of p16 and p53 may provide a biomarker with more sensitive predictive value.	Mutant p53 can be used as an early stage marker of OSCC and may be useful for predicting recurrence.
17.	(Wang *et al.*, 2017)	Archives of Oral Biology	In 19 HNSCC samples with high miR-182 regulation, 16 samples also had p53 overexpression and the remaining 3 samples had no p53 expression or were detected in low amounts.	High miR-182 upregulation is associated with p53 overexpression in HNSCC. Meanwhile, miR-182 overexpression directly facilitates the tumor growth and metastatic activity in HNSCC.	In HNSCC, elevated miR-182 expression in tumors is linked to p53 mutations.
18.	(Zaid *et al.*, 2018)	Asian Pacific Journal of Cancer Prevention	Of the 105 OSCC tissue samples, 78 samples (74.3%) showed p53 expression, 65.5% in non-smokers and 83.1% in smokers.	Mutant p53 is linked to shisha smoking in OSCC. People who smoke shisha have a higher risk of having mutant p53 than non-smokers.	Mutant p53 has been linked to shisha smoking in cases of oral squamous cell carcinoma (OSCC), premalignant lesions, and even in histologically normal oral mucosa.
19.	(Zhou *et al.*, 2019)	Journal of Molecular Cell Biology	P53 is the most commonly altered protein found in human cancers. Approximately 80% of its mutations result from single nucleotide changes.	Mutant p53 is considered an obstacle in cancer therapy due to impaired tumor-suppressive activity, interference with wild-type p53, and the development of cancer-promoting capabilities.	The oncogenic functions of mutant p53 are associated with advanced cancer stages and treatment resistance.

4. Discussion

Loss of function and changes in expression of p53 often occur in human cancers. Exposure to large amounts of carcinogens causes wild p53 to mutate into mutant p53. Missense mutations represent the most frequent type of p53 alteration, followed by nonsense mutations and, to a lesser extent, frameshift deletions. This condition causes p53 to lose its ability to regulate the cell cycle and trigger apoptosis. The gene will experience continuous damage and uncontrolled proliferation. If this condition is not stopped, it may lead to malignancy[10,13].

Prevalence states that 20-80% of p53 is mutated in HNSCC. Escobar-Hoyos et al[14], stated that mutant p53 is the second most frequently found gene in PDAC with 40% missense mutations and 30% frameshift deletions. Irmawati et al[10], stated that there are more than 50% mutant p53 in human tumors with 95% mutations spread throughout the exons encoding p53, especially in the middle which is the specific p53 binding region. Hadzi-Mihailovic et al[15], stated that overexpression of p53 not only occurs in OSCC, but also in dysplasia lesions. Meanwhile, there are more mutant p53 in smokers than non-smokers. Likewise, there are more mutant p53 in HPV-negative patients than in HPV-positive patients with HNSCC as stated by Tuna et al[16].

Mutant p53 is linked to a weakened poor prognosis, immune response, and resistance to both radiotherapy and chemotherapy. It is regarded as a key indicator in the transformation of dysplastic cells into malignant ones. According to Shi et al[13], Head and Neck Squamous Cell Carcinoma (HNSCC) accounts for over 40% of cancer-related deaths globally, with 41% of cases involving mutant p53. These findings highlight the role of mutant p53 in predicting the treatment and prognosis response of HNSCC. Additionally, a study by Nagata et al[17], assessed mutant p53 expression using immunohistochemistry in 40 Laryngeal Squamous Cell Carcinoma (LSCC) samples and found positive results in 80% of them. Based on WHO classification, mutant p53 was detected in 73.3% of mild dysplasia cases (11 out of 15), 88.8% of moderate dysplasia (16 out of 18), and 71.4% of severe dysplasia (5 out of 7). These results suggest that mutant p53 expression in LSCC epithelium may serve as an early marker of severe genetic damage that could lead to malignancy.

A study by Zhou et al[18], reported mutant p53 expression in 316 breast cancer patients, linking it to poor prognosis and resistance to adjuvant systemic therapy. This association between mutant p53, chemoresistance, and unfavorable clinical outcomes has also been observed in lung, gastric, colorectal, and hematological cancers. Thus, mutant p53 represents a significant challenge in effective cancer treatment. therapy due to its loss of function, dominant-negative effect, and oncogenic gain-of-function.

Another study by Wang et al[19], on 25 tissue samples from HNSCC patients and 10 tonsil tissue samples from healthy patients used immunohistochemistry to detect mutant p53. This study used microRNA 182 (mi-R 182) as a comparison. The results obtained were 17 of 25 HNSCC samples showed overexpression of p53. Meanwhile, around 19 HNSCC samples had high expression of mi-R 182, 16 samples also showed high expression of p53 and the remaining 3 samples did not show p53 expression. Both results indicate that overregulation of mi-R 182 is related to overexpression of p53 caused by mutant p53. Several other studies also mention that mutant p53 plays a role in controlling mi-R 182 expression.

A study by Singh et al[20], conducted in Gujarat, India, on 92 tissue samples (46 pairs, primary malignancy and surrounding normal tissue) from oral cancer patients using PCR-SSCP and sequencing to detect mutant p53 expression. The results obtained were that there were 51 mutations in tissue with primary malignancy and surrounding normal tissue in 24/46 samples (52.2%). Mutant p53 was detected exclusively in malignant tissue in 11 patients, while 6 patients showed its presence only in normal tissue. Additionally, 7 patients exhibited mutant p53 in both malignant and normal tissues. The frequency of mutant p53 in moderately differentiated tumors was around 58.3%, in advanced stage tumors around 55.6%, and in tumors with increased lymph nodes around 66.7%. Odds Ratio (OR) analysis also showed that patients with mutant p53 were at high risk of lymph node metastasis.

Research by Zaid et al[21], which was conducted to analyze p53 overexpression associated with shisha smoking in Syria and Lebanon in 105 tissue samples (52 smokers, 53 non-smokers) of OSCC, 96 premalignant tissue samples (48 smokers, 48 non-smokers), and 60 normal tissue samples analyzed using immunohistochemistry. In 105 OSCC tissue samples, 78 samples (74.3%) showed p53 expression, in 65.5% non-smokers and 83.1% smokers. In an analysis of 96 premalignant tissue samples, p53 expression was observed in approximately 23% of non-smokers and 41.7% of smokers. Among 60 normal tissue samples, 6.6% of non-smokers and 16.6% of smokers also showed p53 expression. These findings indicate that shisha smoking is associated with a higher frequency of p53 expression compared to non-smokers. Persistent exposure to this habit may lead to p53 mutations, potentially progressing to malignancy. Mutant p53 has been linked to the early stages of tumor development in the oral cavity.

According to a study by Lindemann et al[22], mutant p53 may act as a key indicator in predicting patient response to platinum-based chemotherapy. The study found that 13 out of 14 patients exhibiting high levels of mutant p53 showed resistance to cisplatin treatment. Conversely, patients with wild-type p53 (22 individuals) and those with low mutant p53 expression (8 individuals) were also identified as unresponsive to cisplatin therapy. These results indicate that mutant p53 expression can be detected with cisplatin chemotherapy. When there is resistance, it can be concluded that mutant p53 expression in the tissue is already high. When there is no response to therapy, it can be concluded that p53 expression in the tissue is relatively low or absent.

Another study conducted by Koehler et al[23], found that mutant p53 was found in most patients with a frequency of 53% (10/19). 22% (6/19) of patients had mutations in Rb1. Mutations of p53 or Rb1 are thought to play a role in the angiogenesis of cancer cells which makes them sensitive to antiangiogenic therapy.

Low expression of mutant p53 can be caused by the expression of large amounts of wild p53. Moderate intensity exercise can trigger transcription of p53 mRNA which can then lead to translation and synthesis of wild p53. Research by Irmawati et al[10], using *Mus musculus*. In this study, benzopyrene induction was carried out as much as 0.08 mg dissolved in 0.04 ml of oleum olivarum in groups B and C. Meanwhile, animals in group C were given continuous

exercise. The results obtained were that the expression of mutant p53 in group B was 14.75 + 1.57 while in group C it was 6.67 + 1.57. It can be inferred that light intensity exercise helps decrease the production of mutant p53.

Based on various references that have been obtained, it explains that mutant p53 is found in several oral cavity malignancies, such as HNSCC, OSCC, LSCC. Mutant p53 can be identified through immunohistochemistry and PCR-SSCP (single-strand conformational polymorphism). Additionally, it may be linked to other factors involved in cancer development. As in the study of Wang et al[19], using mi-R 182 as a comparison and the study of Koehler et al[23], which linked mutant p53 with Rb1 in cancer. Then, mutant p53 can be used as an assessment of cancer therapy. In cancer therapy using cisplatin, high expression of mutant p53 makes it resistant to the therapy as explained by Lindemann et al[22].

5. Conclusion

p53 mutant protein can be used as a predictor of oral cavity malignancy, because exist of this protein referred that the cell undergo changes into malignancy by the inhibition of mutation cell apoptosis.

Compliance with ethical standards

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Disclosure of conflict of interest

The authors declare that there is no conflict of interest regarding the publication of this article.

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