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Viva Voce in ORAL PATHOLOGY

(With Explanatory Answers)

Forewords
K Sadashiva Shetty
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2 Benign and Malignant Tumors of the Oral Cavity

1. **Define neoplasia.**
   Willis in 1952 defined neoplasia as an abnormal mass of tissue, the growth of which exceeds and is uncoordinated with that of the normal tissues and persists in the same excessive manner after cessation of the stimuli, which evoked the change.

2. **What are carcinomas?**
   Carcinomas are malignant tumors of epithelial origin.

3. **What are sarcomas?**
   Sarcomas are malignant tumors of mesenchymal (connective tissue) origin.

4. **Define metastasis.**
   Metastasis is defined as spread of tumor by invasion in such a way that discontinuous mass/masses are formed at the site of lodgement.

5. **What is differentiation?**
   Differentiation is the extent of morphological and functional resemblance of the tumor cells to the corresponding cells of origin.

6. **What is anaplasia?**
   Anaplasia is the lack of differentiation.

7. **Enumerate few differences between benign and malignant tumors.**
   Following are few important differences between benign and malignant tumors.

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Sl No	Feature	Benign tumors	Malignant tumors
1.	Tumor boundary and size	Well circumscribed/ encapsulated, usually small	Poorly circumscribed and irregular, usually large
2.	Secondary changes like hemorrhage and ulceration	Occur less often	Occur more often
3.	Differentiation	Usually well differentiated	Well to poorly differentiated
4.	Malignant cytological features like nuclear hyperchromatism, pleomorphism, altered nucleo-cytoplasmic ratio	Absent	Present
5.	Metastasis	Absent	Frequently present

8. What is hamartoma?
Hamartoma is a mass of disorganized mature cells of tissues native to that particular site.

9. What is choristoma?
Choristoma is ectopic rest of normal tissue.

10. Name few benign epithelial tumors of oral cavity.
Following are few benign epithelial tumors of oral cavity:
a. Squamous papilloma
b. Keratoacanthoma
c. Oral nevi.

11. Name few malignant epithelial tumors of oral cavity.
Following are few malignant epithelial tumors of oral cavity:
a. Squamous cell carcinoma
b. Verrucous carcinoma
c. Spindle cell carcinoma.

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12. Name few benign mesenchymal tumors of oral cavity.
Following are few benign mesenchymal tumors of oral cavity:
a. Fibroma
b. Peripheral and central ossifying fibroma
c. Lipoma
d. Giant cell tumor (osteoclastoma)
e. Osteoma
f. Chondroma.

13. Name few malignant mesenchymal tumors of oral cavity.
Following are few malignant mesenchymal tumors of oral cavity:
a. Fibrosarcoma
b. Malignant fibrous histiocytoma
c. Liposarcoma
d. Kaposi's sarcoma
e. Osteosarcoma
f. Chondrosarcoma
g. Ewing's sarcoma.

14. What is the etiology of squamous papilloma?
Squamous papillomas are associated with human papillomavirus (HPV) infection. HPV types 6 and 11 are usually detected in the neoplastic tissues of papilloma.

15. Which are the intraoral sites of occurrence of squamous papilloma?
Squamous papilloma are most commonly found on tongue, lips, buccal mucosa, gingiva and palate.

16. What is the clinical presentation of lesion in squamous papilloma?
Squamous papilloma clinically appears as exophytic, soft, usually pedunculated growth with numerous finger-like surface projections giving a characteristic cauliflower-like appearance to the lesion.

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17. What is the difference between oral squamous papilloma and verruca vulgaris?
Verruca vulgaris or common wart is the skin analogue of oral squamous papilloma. It is highly contagious and spreads to other unaffected areas by autoinoculation, whereas oral squamous papilloma is not contagious.

18. What is papillomatosis?
Multiple papillomas may coalesce together forming a large lesion known as papillomatosis.

19. Name few skin disorders associated with papillomatosis.
Following are few skin disorders associated with papillomatosis:
a. Cowden's syndrome (multiple hamartoma and neoplasia syndrome)
b. *Acanthosis nigricans*
c. Focal dermal hypoplasia syndrome.

20. What are koilocytes?
These are virus altered epithelial cells, which demonstrate pyknotic and crenated nuclei, often surounded by a clear zone (perinuclear clearing). These cells are seen in lesions caused by HPV, like squamous papillomas, condyloma acuminatum, focal epithelial hyperplasia.

21. What is self-healing carcinoma?
Keratoacanthoma is also known as self-healing carcinoma because of clinical and pathological resemblance to squamous cell carcinoma.

22. Keratoacanthoma is associated with which syndrome?
Keratoacanthoma is associated with Muir-Torre syndrome.

23. What are the clinical stages of keratoacanthoma?
Following are the clinical stages of keratoacanthoma:
a. Proliferative stage
b. Maturation stage
c. Resolving stage.

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24. What is the clinical presentation of keratoacanthoma?
Keratoacanthoma clinically appears as dome shaped, sessile nodule with central plug of keratin.

25. Which is the most common location of keratoacanthoma?
Most common location of keratoacanthoma is outer edge of vermilion border of both upper and lower lips.

26. What is pseudoepitheliomatous hyperplasia?
Pseudoepitheliomatous hyperplasia is a common exuberant oral epithelial response in which the rete pegs are extended deeply into the underlying connective tissue in irregular fashion.

27. Name few oral lesions exhibiting pseudoepitheliomatous hyperplasia.
Following are few oral lesions exhibiting pseudoepitheliomatous hyperplasia:
a. Keratoacanthoma
b. Granular cell tumor of tongue
c. Papillary hyperplasia of palate
d. Blastomycosis.

28. What is a nevus?
A nevus is defined as congenital, hamartomatous developmental tumor-like malformation of the skin or mucous membrane.

29. Classify nevi.
Nevi are classified as follows:
a. Congenital nevi
b. Acquired nevi: It is further classified as
   - Intradermal/intramucosal nevi
   - Junctional nevi
   - Compound nevi
   - Blue nevi
   - Spitz nevi (spindle cell/epitheloid cell).

30. Which are the two most common types of intraoral nevi?
Intramucosal nevi are the most common type of intraoral nevi, blue nevi being the second most common type.

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31. Which is the most common intraoral site of nevi?
Hard palate is the most common intraoral site of nevi.

32. What is the origin of nevus cell?
Nevus cell is neuroectodermal in origin.

33. Enumerate the differences between nevus cell and a melanocyte.
Following are the important differences between nevus cell and a melanocyte:

Nevus cell	Melanocyte
1. Lacks dendritic processes	1. Dendritic processes are present
2. Retains melanosomes	2. Transfers melanosomes to adjacent keratinocytes
3. Tends to form groups called as nests/thèques	3. Do not form nests

34. What is the location of nevus cells in intramucosal nevi?
Nevus cells are located within the connective tissue in the intramucosal nevi.

35. What is the location of nevus cells in junctional nevi?
Nevus cells are located at the junction of the epithelium and connective tissue in junctional nevi.

36. What is the location of nevus cells in compound nevi?
Nevus cells are located both in the basal layer of the epithelium and underlying adjacent connective tissue in compound nevi.

37. What is Abtropfung effect?
Abtropfung effect is also known as dropping off effect, seen in junctional nevi. It is the crossing of the nevus cells across the junction of epithelium and connective tissue, growing down into the underlying connective tissue.

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38. What are the types of nevus cell?
Following are the types of nevus cells:
- Type A: Epithelioid
- Type B: Lymphocyte like
- Type C: Spindle shaped.

39. What are the stages of development of nevi?
Following are the stages of development of nevi:
Junctional nevi ⟶ Compound nevi ⟶ Intradermal nevi.

40. Which nevi have maximum tendency to undergo malignant transformation?
Junctional nevi have maximum tendency to undergo malignant tranformation into malignant melanoma.

41. What is the importance of spitz nevi?
Spitz nevi share the histopathological features of malignant melanoma.

42. Why blue nevi appear blue?
In blue nevi, the pigmented spindle-shaped melanocytes, are located deep in the dermis, parallel to the surface epithelium and hence they reflect light of only short wavelength (like blue light), which imparts blue color to these nevi. This phenomenon is known as Tyndall effect.

43. Define a premalignant lesion.
WHO defines a premalignant lesion as "a morphologically altered tissue in which cancer is more likely to occur than its apparently normal counterpart."

44. Enumerate few oral premalignant lesions.
Following are few oral premalignant lesions:
a. Leukoplakia
b. Erythroplakia
c. Stomatitis nicotina.

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45. Define a premalignant condition.
WHO defines a premalignant condition as "the generalized state of the body, which is associated with a significantly increased risk of cancer."

46. Enumerate few oral premalignant conditions.
Following are few oral premalignant conditions:
a. Oral submucous fibrosis
b. Erosive lichen planus
c. Syphilitic glossitis (luetic glossitis)
d. Sideropenic dysphagia.

47. What is dysplasia?
Dysplasia means disordered cellular arrangement. It is characterized by cellular proliferation and cytological changes of the epithelial cells.

48. Enumerate the various features of epithelial dysplasia.
Epithelial dysplastic features can be divided as architectural and cytological features.
Architectural features include:
a. Loss of epithelial stratification
b. Loss of polarity of basal cells
c. Basal cell hyperplasia
d. Increased number of mitotic figures
e. Dyskeratosis (premature keratinization in single cell/individual cell keratinization
f. Bulbous rete ridges
g. Keratin pearl and epithelial pearl formation.
Cytological features include:
a. Nuclear pleomorphism (abnormal variation in nuclear shape)
b. Anisonucleosis (abnormal variation in nuclear size)
c. Cellular pleomorphism (abnormal variation in cell shape)
d. Anisocytosis (abnormal variation in cell size)
e. Increased nuclear size

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f. Increased nuclear-cytoplasmic ratio
g. Nuclear hyperchromatism
h. Poikilokaryosis (division of nucleus without the division of cytoplasm)
i. Atypical mitotic figures
j. Increased number and size of nucleoli.

49. Define leukoplakia.

Leukoplakia is defined as "a white patch or plaque in the oral cavity, which cannot be scraped off or stripped off easily, which cannot be characterized clinically or pathologically as any other disease and is not associated with any physical or chemical agents except the use of tobacco."

50. Enumerate few white lesions of oral cavity.

Following are few white lesions of oral cavity:
a. Leukoplakia
b. Candidal leukoplakia (hyperplastic candidiasis)
c. Oral hairy leukoplakia (**Greenspan lesion**)
d. Smoker's palate.

Note: Greenspan lesion is oral hairy leukoplakia, whereas **Grinspan syndrome** is association of lichen planus with diabetes mellitus and hypertension.

51. What is cryptogenic leukoplakia?

Idiopathic leukoplakia (non-tobacco associated) are also known as cryptogenic leukoplakia.

52. What are the etiological factors of leukoplakia?

Following are few etiological factors of leukoplakia:
a. Tobacco (single most important factor)
b. Alcohol
c. Viruses (like human papilloma, herpes simplex)
d. Chronic candidiasis
e. Chronic irritation
f. Dietary deficiency (like vitamin A, B complex deficiency)

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53. Which are the most frequently affected sites in leukoplakia?

Buccal mucosa and commissural areas are the most frequently affected sites in leukoplakia.

54. What is the clinical classification of leukoplakia?

Leukoplakia is classified clinically as:

a. Homogeneous leukoplakia
b. Ulcerated leukoplakia
c. Speckled leukoplakia or erythroleukoplakia or nodular leukoplakia.

55. Compare the clinical features of the three types of leukoplakia.

Homogeneous leukoplakia	Ulcerated leukoplakia	Speckled leukoplakia
Usually asymptomatic	Associated with pain or burning sensation	Associated with pain or burning sensation
White patch with flat or corrugated surface	Ulcer is seen in the center of predominantly white lesion	Mixed red and white lesion exhibiting multiple keratotic nodules on the erythematous base

56. Which type of leukoplakia carries maximum risk of malignant transformation?

Speckled leukoplakia exhibits maximum risk of malignant transformation.

57. How much is the malignant transformation rate of leukoplakia?

According to WHO, malignant transformation rate of leukoplakia is about 3%–6%.

58. How is epithelial dysplasia graded histopathologically?

Histopathologically epithelial dysplasia is graded as follows:

a. Mild dysplasia: Dysplastic changes restricted only to basal and parabasal layers

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b. Moderate dysplasia: Dysplastic changes involving basal to midportion of the spinous layer of the epithelium
c. Severe dysplasia: Dysplastic changes involving most of the thickness of the epithelium.

59. What is carcinoma in situ?

Carcinoma in situ is the most severe form of epithelial dysplasia, with the dysplastic changes involving the entire thickness of the epithelium, from basal layer to surface of mucosa. It is also known as intraepithelial carcinoma.

Note: In carcinoma in situ, there is no invasion of the epithelial cells into the underlying connective tissue as the basement membrane is intact.

60. What is proliferative verrucous leukoplakia?

Proliferative verrucous leukoplakia is a unique form of leukoplakia of unknown etiology, characterized by extensive multiple keratotic plaque with roughened suface projections. Proliferative verrucous leukoplakia is basically a clinical term. These lesions have aggressive behavior with a high malignant transformation rate.

61. What is erythroplakia?

Erythroplakia is a red patch or plaque in the oral mucosa, which cannot be characterized clinically or pathologically as any other disease and which has no apparent cause.

62. Which are the most common sites of involvement in erythroplakia?

Floor of mouth, tongue and soft palate are the most common sites of involvement in erythroplakia.

63. Which is the premalignant lesion with the highest malignant transformation rate?

Erythroplakia is the premalignant lesion with the highest malignant transformation rate.

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64. What is the cause of erythroplakic lesion appearing as red?

In erythroplakia the epithelial thickness is reduced and there is lack of keratinization. Due to these factors the underlying microvasculature of the connective tissue is seen through, imparting the red color to the lesion.

65. What is tobacco pouch keratosis?

Tobacco pouch keratosis is the grayish white fissured lesion seen commonly in the mandibular vestibule in the area of chronic tobacco placement.

66. What are chevrons and in which lesion is it seen?

Chevrons are parakeratin pointed projections above or within superficial epithelial layer.
They are seen in tobacco pouch keratosis.

67. What is leukedema?

Leukedema is a common mucosal alteration that represents a variation of the normal condition rather than a true pathological change. It appears as faint white diffuse filmy lesion with mild wrinkling of the mucosa. It commonly affects buccal mucosa bilaterally. It is characterized histologically by intracellular edema within the spinous layer.

68. How to differentiate between leukedema and leukoplakia?

Leukedema faints or disappears upon stretching of the mucosa, whereas leukoplakia persists.

69. What is the difference between smoker's palate and palatal changes associated with reverse smoking?

Palatal changes associated with reverse smoking are precancerous, whereas smoker's palate does not exhibit malignant transformation.

70. Reverse smoking is commonly practiced in which state of India?

Reverse smoking is commonly practiced among females of Andhra Pradesh state.

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This manual provides quick revision of Oral Pathology for theory and viva-voce examinations.

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- The language used is simple and lucid
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- Appendices provided at the end of the book are extremely useful for the last-minute revision
- Apart from undergraduate students, this manual will also be helpful for students taking PG entrance examinations.



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