HPV Related Oral Cancer Screening: Different Presentation, Different Protocol

A Peer-Reviewed Publication  
Written by Cris Duval, RDH

Abstract
Dental professionals, making assumptions about the two distinct pathways of oral cancer, often forgo comprehensive oral cancer screenings, which affects patients’ prognosis and creates malpractice liability. Absent comprehensive screenings, HPV-related oral cancer is diagnosed late-stage, when morbidity is highest. To curb late diagnosis we must heed current research. HPV-related oral cancer, which is rapidly on the rise, is different in its clinical presentation from tobacco/alcohol-related oral cancer. Thus, we cannot rely on what we learned in dental school, risk profiling, or traditional tools to screen for HPV-related oral cancer. We must change our approach, by implementing comprehensive screenings that incorporate extra/intraoral tactile screenings, visual screenings of all tissues, and asking appropriate, revealing questions.

Educational Objectives:
At the end of this self-instructional educational activity, the participant will be able to:
1. Discuss why patients with HPV-related oral cancer do not fit the profile of patients with tobacco/alcohol-related oral cancer.
2. Identify obstacles that thwart early discovery.
3. Discuss the clinical presentation of HPV-related oral cancer.
4. Determine that adjunctive screening tools do not help identify HPV-related lesions.
5. Implement the use of appropriate, revealing questions during the screening process.

Author Profile
Cris Duval, RDH provides clinical care in Seattle, WA. Cris provides lectures and has authored articles on dental hygiene, periodontal disease, and oral cancer. She serves as the Northwest Liaison for the Oral Cancer Foundation. She can be reached at cris@acld.com.

Author Disclosure
Ms. Duval is the owner of Duval Consulting, coaching hygiene teams to higher levels of productivity and patients to higher levels of health and wellness.

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Dental professionals, making assumptions about the two distinct pathways of oral cancer, often forgo comprehensive oral cancer screenings, which affects patients’ prognosis and creates malpractice liability. Absent comprehensive screenings, HPV-related oral cancer is diagnosed at a later stage, when morbidity is highest. To curb late diagnosis, we must heed current research. HPV-related oral cancer, which is rapidly on the rise, is different in its clinical presentation from tobacco/alcohol-related oral cancer. Thus, we cannot rely on what we learned in dental school, risk profiling, or traditional tools to screen for HPV-related oral cancer. We must change our approach, by implementing comprehensive screenings that incorporate extra/intraoral tactile screenings, visual screenings of all tissues, and asking appropriate, revealing questions.

In 2007, a 33 year old patient named Eric called his dentist’s office with a chief complaint of pain and swelling on his lower right side. The receptionist taking the call asked if he still had his wisdom teeth; Eric responded positively. When he presented for examination the doctor confirmed the presence of an infection and recommended extraction of tooth #32. Two months after completion of the procedure Eric was still experiencing severe pain.

It is important that oral healthcare professionals do not assume the cause of a patient’s signs and symptoms until they have been examined. A common assumption is that facial pain and swelling is caused by an infection around a wisdom tooth or an abscess. Another assumption is the unlikely nature of finding anything to the contrary in such a young person.

The assumptions noted above are correct in the vast majority of cases. Although Eric presented with signs and symptoms consistent with an infected third molar, that was not the correct diagnosis. Ultimately it was determined that he had oral cancer.

When it comes to providing care, making assumptions about a patient’s condition may lead to an improper or missed diagnosis which hurts the patient and leaves you and your practice susceptible to legal action:
A patient may successfully sue for malpractice if a dentist does not satisfy the accepted standard of care. It is possible to argue that negligence could be proven if a dentist fails to incorporate an oral cancer screening as part of his examination protocol. A delay in diagnosis which compromises the patient’s health, and which allows a localized, focal lesion to prosper into a later stage malignancy resulting in additional morbidity, or worse the death of the patient, certainly would bring severe legal consequences.¹

Just because a patient does not seem to fit the profile of someone who has oral cancer does not mean that an oral cancer screening doesn’t need to be provided during an examination.²

Oral health professionals, must bear in mind that patients make assumptions, too. If a patient calls the office and states, “I think I have a problem with my molar”, it should not be assumed that the patient’s self-diagnosis is correct. Further examination is required.

Essentially, everyone participates in the assumption process, and the result is delayed care at best, tragedy at worst. By avoiding assumptions, many consequences can potentially be averted.

You must conduct a comprehensive oral cancer screening on every patient at every visit even if it is likely that oral cancer is a remote possibility.

Albeit fundamental to the diagnostic process, intake calls have the potential to perpetuate assumptions. First, the patient assumes the physical cause of his pain and swelling, then the person taking the intake call assumes the medical cause of the patient’s symptoms, then the doctor assumes that both the intake call and the morning huddle were conducted without bias. Consequently, a diagnosis may be made before the patient arrives at the office.

This tendency is particularly common in cases of oral cancer. Because instances of tobacco/alcohol-related oral cancer have been historically rare, oral health professionals at all stages of the diagnostic process often summarily dismiss oral cancer as a possibility.³ Their train of thought could be summarized as follows: “I haven’t found one instance of oral cancer in my 25 years as a practicing dentist/hygienist; therefore, I cannot possibly find it now in this patient.”

This train of thought, which constitutes an assumption, is not valid regardless of when and by whom it occurs. Oral cancer is on the rise in all age groups regardless of tobacco and alcohol use due to the spread of a particular strain of human papilloma virus, HPV-16,³⁶⁷ Indeed, “HPV may contribute to the development of approximately 20% to 30% of all oral cancer cases” worldwide⁶ and 40 to 80% of all
oral cancer cases in the U.S.1 At this point, the fact that oral cancer used to be a statistical rarity can no longer dictate or guide diagnostic processes.

**Oral cancer is no longer “an old-person’s disease.”**

It is imperative that a paradigm shift is adopted by dental professionals since there has been a paradigm shift in what are now known to be two distinct, separate causes of oral cancer. Tobacco/alcohol use, which has been traditionally identified as the leading risk factor for development of oral cancer, still does predispose patients to oral cancer.10 Infection with HPV-16; however, has resulted in a significant increase in new cases.11 The prevalence of tobacco/alcohol-related oral cancer has dropped over the past several years, consistent with a decrease in tobacco use; however, HPV-related oral cancer is increasing.12 Indeed, the fastest growing segment of the oral cancer population is non-smokers between the ages of 25-50.13

Because there are two, distinct and separate causes of oral cancer,14 there are now two, distinct and separate profiles of patients with oral cancer; those with tobacco/alcohol-related oral cancer and those with HPV-related oral cancer. In essence, it cannot be assumed that a patient who does not fit the profile for tobacco/alcohol-related oral cancer does not have oral cancer; for that patient may in fact have HPV-related oral cancer.

The previously referenced patient named Eric was asked whether he drank alcohol or smoked cigarettes, Eric’s dentist assumed that he did not fit the profile of someone with oral cancer, because he doesn’t smoke15 and is young.16 Eric’s responses to the dentist’s questions seemed to confirm the dentist’s belief that he did not fit the profile and thus did not have oral cancer.17 Eric did have oral cancer, and because it was diagnosed late-stage, he suffered life-changing consequences.18

As oral health professionals, we consider it self-evident that post-treatment stage-one patients live longer and better than post-treatment stage-four patients.19 Thus, we strive to discover the presence and type of oral cancer as early as possible.20 Indeed, when oral cancer is found early there is an 80-90% survival rate, while when found late, the survival rate drops to 20-30%.21 It is absolutely critical to our patient’s oral and systemic health to “break the cycle of late diagnosis,” and this in turn requires a commitment to avoiding assumptions.

**Don’t make assumptions!**

Breaking the cycle of late diagnosis can be achieved by staying well informed, keeping up to date with current research and listening to the experts. For HPV-related oral cancer (in contradistinction to tobacco/alcohol-related oral cancer), dental professionals cannot rely on the information provided in dental or hygiene school.

**Yesterday's information will not carry the profession today.**

A paradigm shift in the approach to examination and diagnosis of oral cancer is necessary due to the following:

1) HPV-related oral cancer is distinct and separate from tobacco/alcohol-related oral cancer.
   a) Because professional education predominately focused on tobacco/alcohol-related oral cancer, rather than HPV-related oral cancer, the clinician’s knowledge may be lacking.
   b) Based on educational background and clinical experiences with respect to tobacco/alcohol-related oral cancer, dental professionals may be predisposed to making assumptions about HPV-related oral cancer.

2) The profession’s understanding of HPV-related oral cancer is nascent, which has sparked recent interest among researchers. As a result, new information is coming out on a daily basis

Oral health professionals must be flexible and willing to change our approach to oral cancer screenings based on today’s information. Keeping current on data and conclusions from the HPV-experts is critical to patients’ survival.

Current information is based on empirical research and deductive reasoning.26 Recent data tells us that HPV-related oral cancer is more elusive than, and thus different from, tobacco/alcohol-related oral cancer, because of both its clinical presentation and its clinical pathway.27 Unlike tobacco/alcohol-related oral cancer lesions which occur in the anterior floor of the mouth and the lateral borders of the tongue,28 HPV lesions are buried deep within the tissues, possibly below the basement membrane barrier at the base of the tongue, in the tonsillar area, and in the oropharynx.29

For this reason, HPV-related oral cancer cannot be detected with traditional tools: “No currently available adjunctive device using tissue auto fluorescence, tissue reflectance, or brush cytology, is capable of assisting in the early discovery of these disease states, as pre-cancers.”30 In short, even advanced adjunctive screening tools that fluoresce tissue to the basement membrane barrier – but not below it – do not help detect HPV-related lesions.

Moreover, HPV can cause oral cancer and produce no visible signs until the disease is very advanced.31 HPV-related oral cancer does not manifest pre-cancerous lesions, which historically have been heralded as the early warning signs of the disease process.32
Most Americans will have some version of HPV in their lifetimes. The HPV virus infects at least 50% of all people who have had sex at some time in their lives. It is “likely that changes in sexual behaviors of young adults over the last few decades and which are continuing today are increasing the spread of HPV and the oncogenic versions of it.”

While distressing, these statistics are not surprising, because there are 130 strains of HPV viruses. Admittedly, the majority of these strains are thought to be harmless. Out of the hundreds of strains of HPV, only a handful are oncogenic. Healthcare professionals are most concerned with HPV-16, which causes cervical cancer in women. The virus also causes oral cancer in men and women in a 3 to 1 ratio.

HPV-16 has an interesting and complex genetic makeup. The human papilloma virus is a double-stranded DNA virus that infects the epithelial cells of skin and mucosa. The virus also has an unpredictable and non-linear disease process. Admittedly, data about the lifestyle of HPV-16 is not conclusive at this point in time.

Unlike tobacco and alcohol-related oral cancer, HPV-16 has not been tracked for a sufficient period of time to fully understand its effects over time. The existence of HPV-related oral cancer was not known until the year 2000, when Dr. Maura L. Gillison first published a research article on the casual association between HPV and oral cancer. Consequently, there is significant data on HPV-16’s lifecycle, but inconclusive data on HPV-16’s biological and genetic structure, but inconclusive evidence is still lacking.

Current knowledge includes details of HPV-16’s lifecycle, with respect to the virus’ transmission/reproduction, capacity for infection, as well as, capacity to predispose persons to cancer. These details include the following:

- “Transmission of the virus occurs [through skin-to-skin contact] when [certain] areas come into contact with a virus, allowing it to transfer between epithelial cells.” The moist epithelial surfaces (squamous cells) include all areas covered by skin and/or mucosa such as the mouth, throat, tongue, tonsils, vagina, penis, and anus. Because of the nature of those surfaces through which the virus most readily passes, we now know that those who engage in sexual contact with 5 or more conventional or oral sex partners are most at risk. However, although “it is established now that sexual contacts, both conventional and oral, are a means of transferring the HPV virus, it is still poorly understood what other transfer pathways may exist.

- Following transmission of the virus between two persons’ epithelial cells, the receiving person may become infected with HPV-16 depending on his or her body’s ability to mount an adequate immune response.

- “Once the virus integrates its DNA genome within the host cell nucleus, it dysregulates expression of the oncoproteins, E6 and E7,” which are proteins with very strong binding capabilities that interfere with the host cell’s transcription cycle. This dysregulation allows HPV to reproduce quickly and in great numbers leading to uncontrolled reproduction of viral cells and possibly oral cancer.

- These damaged cells then constitute HPV-related lesions. Infection with a high risk HPV virus does not mean that the patient develops oral cancer, because development of HPV-related oral cancer is contingent upon that person’s immune system. In fact, “only 1% of those infected have a lack of an immune response to the HPV-16 strain. Most people’s immune systems will clear the infection before a malignancy has the opportunity to occur.” That said, even if the infected person does not have an HPV-related infection and/or develop HPV-related oral cancer, he/she may have a latent form of the virus, which could last for decades.

Use of tobacco and alcohol is the obvious leading risk factor for the development of tobacco/alcohol-related oral cancer, but not for the development of HPV-related oral cancer.

Actually, there are a considerable number of risks factors for HPV-related oral cancer. Because HPV-related oral cancer develops via transmission of HPV-16 over moist epithelial cells, sexual contact, including not limited to open mouth kissing and genital-oral contact, is a risk factor for HPV-related oral cancer. Age of the patient is a risk factor for HPV-related oral cancer, for the sexual behaviors of youth are changing, leading to an increase in the number of cases in younger populations. This stands in stark contrast to the number of cases of tobacco/alcohol-related oral cancer, which takes more time to develop, and thus, older populations are more at risk than younger populations. As mentioned above, gender of the patient is also pertinent; men are more likely than women to develop HPV-related oral cancer by a 3-to-1 ratio.

Other risk factors not yet conclusively shown to participate in the development of oral cancer include: 1) lichen planus and other auto-immune, inflammatory diseases of the mouth, 2) periodontal disease, whereby an area of trauma becomes an avenue of viral infection, 3) diets lower in fruits and vegetables, because without them a person’s immune system may not be properly supported, and 4) genetic predispositions, such as inherited defects in genes that contribute to DNA repair, such that damaged
cells are not repaired or killed off by the immune system before development of oral cancer. These risk factors, especially when existing in tandem with each other, help identify at risk patients.

It is important to keep in mind that, of those diagnosed with oral cancer, 7% have no currently identifiable cause of that cancer. The fact that this 7% subgroup exists suggests that there are other risk factors that have not yet been identified. There is much that we still do not know about oral cancer.

The experts are very concerned about our ability to successfully identify those patients who may be experiencing the signs and symptoms of oral cancer, because HPV-related oral cancer is so elusive.

Because HPV-related oral cancer cannot be easily detected, it is generally diagnosed in its later stages; either in the 3rd or 4th stage of the disease when morbidity is highest. To avoid tragedy and prepare patients for health and wellness, the dentist and/or hygienist must conduct oral cancer screenings on every patient at every visit. A comprehensive oral cancer screening includes screening for both tobacco/alcohol-related oral cancer and HPV-related oral cancer. This is imperative because, in the U.S., one person dies from oral cancer every hour of every day.

This is what a comprehensive oral cancer screening looks like...

1) Extra-oral visual and tactile examination, including but not limited to palpation of the neck;
2) Intra-oral visual and tactile examination;
3) Use of adjunctive screening tools, i.e. tissue auto fluorescence;
4) Asking patients appropriate and revealing questions and waiting for their responses;
5) Patient education, particularly provision of information on the benefits of vaccinating against HPV.

Extra- and intra-oral visual and tactile screening readily assist in the screening process for tobacco/alcohol-related oral cancer, but also help find more elusive HPV-related oral cancer.

For extra-oral visual and tactile screening, recommendations include palpation of the same structures currently accepted as the standard of care, along with palpation for almond-sized lumps in the cervical chain of lymph nodes. Such almond-sized lumps could be indicative of an HPV-16 infection, particularly if there is any degree of asymmetry between the two sides of the neck:

Palpation of the neck has never been more important. Unfortunately this is where many first presentations occur, along with recognition by the patient that something is amiss. A hard/firm, enlarged, painless, fixed node in the neck that has been present for over 21 days should be sampled by fine needle aspiration biopsy at minimum.

For intra-oral visual and tactile screening, recommendations include listening for slurred speech, looking for asymmetry in the tonsillar area (i.e. one tonsil is markedly larger than the other one), palpation of the tongue for any buried lumps, and/or observing whether a stuck-out tongue tracks to the side. Any of these symptoms could indicate the presence of a tumor buried deep within the tissues of the posterior mouth.

Adjunctive screening tools add to the diagnostic protocol and potential detection of lesions. Tools that auto fluoresce tissue “excite” the mitochondria all the way to the basement membrane. When tissue auto fluorescence is used, healthy mitochondria fluoresce back at the clinician a bright, apple-green color. Mitochondria that have undergone any pathological changes appear noticeably darker. Auto fluorescence is insufficient for the screening of HPV-related oral cancer because HPV-related oral cancer does not typically present surface lesions. Auto fluorescence is efficacious for the screening of tobacco/alcohol-related oral cancer, and thus, the tool must be utilized during a comprehensive screening.

Current screening protocol, which includes these first three steps, is particularly good for early discovery of tobacco/alcohol-related oral cancer, but insufficient for early detection of HPV-related oral cancer absent the introduction of two additional steps.

Because the majority of the people diagnosed with oral cancer do not use tobacco, screening both tobacco/alcohol-related and HPV-related oral cancer factors must be included in patient examinations. Since there are few effective tools for detection of both types of oral cancer, amending and restating the model of oral cancer screening needs to occur. In short, because the tools currently available are marginally useful for the screening of HPV-related oral cancer, screening for this disease dictates that clinicians focus upon and hone their communication skills during the screening process.

Clinicians need to pose questions that are specifically designed for early-detection of HPV-related oral cancer. Asking the right questions and waiting for an answer are critical.
A patient's symptoms and possible condition(s) become evident in the patient's answers to your questions.

Recent data from oral cancer experts underscores the need for more pointed questioning and patient education during an oral cancer screening. Today's oral cancer screening and identification of patients at risk hinge on the quality of the questions asked during patients' visits. When you ask quality questions, you increase the likelihood of early discovery.

We simply can't make clear distinctions without the use of questions! No distinctions! No decisions! No actions! No wonder questions are such a powerful force. Without questions we would not – could not – take any action!

~Bobb Biehl

The key is to ask open-ended questions which will elicit answers revealing considerable detail.

“When you use that approach and pay attention to the answer you're getting, you don't have to establish your credibility; the work that you do for your clients does so for you.”

This approach, in turn, creates consistency in the screening protocol.

To adequately screen for HPV-related oral cancer and rule out other conditions, the following questions should be included in the protocol:

- Can you tell me about any swelling or pain that you have experienced in your face, mouth, neck, tonsils, or throat areas?
- Can you tell me about any painless, non-moving, firm bumps on your neck?
- Can you tell me about any mouth sore that you have had lasting more than 2 weeks?
- Can you tell me about any oral bleeding that you have experienced?
- Can you tell me about any changes in the way things have recently tasted?
- Can you tell me about any changes in the way your voice sounds?
- Can you tell me about any changes to the surface of your mouth?
- Can you tell me about any problems that you have experienced eating or swallowing?
- Can you tell me about any recent changes in your weight?
- Can you tell me about any numbness or tingling in your face?
- Can you tell me about any recent changes in your vision?
- Can you tell me about your vaccination history?

A meaningful answer to any of these questions may indicate that the patient has an underlying viral infection with HPV, and thus, the need for further investigation.

In the past, before these types of questions were posed, oral health professionals were making inaccurate connections between the symptoms experienced (e.g. ear pain) and the cause of those symptoms (e.g. oral cancer). For example, if a patient acknowledges ear pain, one should follow-up with questions about the duration and intensity of that pain to rule out other possible causes (e.g. allergies, colds, and infections).

The majority of dental healthcare professionals believe they are providing thorough cancer screening. Experts indicate that there is more that can be done to provide a comprehensive oral cancer examination.

We must implement a more comprehensive oral cancer screening.

“The HPV positive group is the fastest growing segment of the oral cancer population.” One hundred people are newly diagnosed with oral cancer every day. This statistic paints a clear picture that more needs to be done. Because of the sharp increase of new HPV-related oral cancer cases, a simple visual and tactile examination for tobacco/alcohol-related oral cancer is no longer sufficient to save our patients' lives.

Oral health professionals sometime struggle with communicating new information to their patients. In addition to relying on the list of questions provided above, a good way to overcome this barrier is to start dialogue with patients by using the phrase “We now know….” This phrase feels safe and naturally leads to an atmosphere of sharing information.

It is important to remember the big picture: saving as many lives as possible. Screening for oral cancer is all about saving lives and increasing public awareness. Awareness must be increased and this can be accomplished one patient at a time.

The best way to increase public awareness is get individual patients behind your vision of changing what we now know about oral cancer.

Encourage patients to talk with their loved ones and friends. The following verbiage may prove to be beneficial. “When you leave here today and go back home or to work, please share with the people you love that you had a thorough oral cancer screening. Ask them if their dentist/hygienist screens them for oral cancer. If it is not being done, encourage them to request it. Tell your friends and
relatives how much you care about them by telling them about HPV-oral cancer.”

**Asking your loved ones questions can save a life.**

**Increasing public awareness can save thousands of lives.**

**References**


23. Asking your loved ones questions can save a life.

Increasing public awareness can save thousands of lives.


41. Hill, Brian. Phone interview. 1 March 2013.


43. Hill, Brian. Phone interview. 1 March 2013.


68. A full discussion of intra and extra-oral tactile and visual screening methods falls outside of the scope of this article. For further information, please refer to the literature and videos provided on the Oral Cancer Foundation’s website at www.oralcancerfoundation.org. Additional information on HPV-related oral cancer is also available on the National Institute of Health’s website at www.nidcr.nih.gov/oralhealth/topics/oralcan/oralcan.htm, and the Center for Disease Control’s website available at www.cdc.gov/oralhealth/topics/cancer.htm.
69. “The dentist should begin by observing the face, head and neck, with particular emphasis placed on the vermilion of the lips. The systematic intraoral examination should include all mucous membrane and gingival surfaces, with emphasis placed on the lateral border of the tongue, floor of the mouth, and pharynx, which are prime sites for oral cancer. The dentist then should perform bimanual, digital palpation to determine if there are any normal enlargements of facial or cervical lymph nodes or of salivary glands.” Marder, Michael Z., D.D.S. “Oral cancer screenings: Dental practice overview.” Marder, Michael Z., D.D.S. “Oral cancer screenings: Dental practice overview.” Web. 23 March 2013. <http://www.oralcancerfoundation.org/dental/pdf/legal_responsibilities.pdf>.

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Notes
Questions

1. What percentage of the people who are or have been sexually active will be infected with the HPV virus?
   a. 25%
   b. 45%
   c. 50%
   d. 35%

2. Where do HPV-related oral cancers most commonly occur?
   a. In the anterior floor of the mouth and tongue
   b. In the tonsil or base of the tongue, in the tonsillar area, and in the oropharynx
   c. On the hard palate
   d. On the lips

3. Which HPV strain is most commonly associated with oral cancer?
   a. HPV-18
   b. HPV-31
   c. HPV-40
   d. HPV-16

4. What is the fastest growing segment of the oral cancer population?
   a. Non-smokers between ages 25-50
   b. Smokers between ages 25-50
   c. Smokers between ages 50-75
   d. Non-smokers between ages 50-75

5. How often does someone die of HPV-related oral cancer?
   a. Every week
   b. Every hour of every day
   c. Every two years
   d. Every two years

6. Which of the following should not be a risk factor for HPV-related oral cancer?
   a. Genetic predisposition
   b. Engaging in oral sex
   c. Periodontal disease
   d. Airborne bacteria

7. Which of the following would not be indicated by pain / swelling around a molar?
   a. A HPV-related lesion
   b. An impacted molar
   c. An abscess at the apex of a tooth
   d. A surface cavity

8. Why are tissue auto-fluorescence screening tools insufficient to screen for HPV-related lesions?
   a. Tissue auto-fluorescence screening tools can only excite mitochondria in the cell down to the basement membrane layer, but HPV-related lesions generally are much deeper.
   b. Tissue auto-fluorescence is not a viable screening tool for either HPV-related oral cancer or tobacco/alcohol-related oral cancer.
   c. The efficacy of tissue auto-fluorescence tools for the screening of HPV-related oral cancer has not been documented.
   d. Tissue auto-fluorescence tools require extensive user training.

9. Where do tobacco/alcohol-related oral cancers most commonly occur?
   a. In the anterior floor of the mouth and the lateral borders of the tongue
   b. At the base of the tongue, in the tonsillar area, and in the oropharynx
   c. On the hard palate
   d. On the gingiva

10. What percentage of people develop oral cancers from no currently identified cause?
    a. 15%
    b. 2%
    c. 7%
    d. 10%

11. What percentage of those who visit a dentist/hygienist regularly report having had an oral cancer screening?
    a. 15%
    b. 75%
    c. 35%
    d. 50%

12. At what stage is HPV-related oral cancer most commonly diagnosed?
    a. First stage
    b. Second stage
    c. Third stage
    d. Third or fourth stage

13. Why are there more instances of HPV-related oral cancer than tobacco/alcohol-related oral cancer today?
    a. Less people are smoking, so it just seems like there are less people with tobacco/alcohol-related oral cancer than HPV-related oral cancer.
    b. Tobacco products are safer today, resulting in less tobacco-related oral cancer.
    c. The prevalence of tobacco/alcohol-related oral cancer has no bearing on the prevalence of HPV-related oral cancer.
    d. Infection with HPV-16 is “easier to get” than tobacco/alcohol-related oral cancer.

14. What is the survival rate when oral cancer is found early (i.e. during stage I)?
    a. 75-80%
    b. 65-70%
    c. 40-50%
    d. 80-90%

15. In addition to cervical cancer, with which type of cancer is HPV-16 associated?
    a. Breast cancer
    b. Brain cancer
    c. Colon cancer
    d. Oral cancer

16. How is HPV-16 predominantly transferred?
    a. Skin-to-skin contact
    b. An airborne pathogen
    c. Touching contaminated surfaces
    d. Blood transfusions

17. Which of the following is more likely to contract HPV-related oral cancer?
    a. Adult men
    b. Women aged 65+
    c. Children
    d. Adult women

18. Which of the following is not an obstacle to oral cancer diagnosis?
    a. Assumptions made by patients and oral health professionals
    b. Summarily dismissing oral cancer as a possibility because the patient does not “fit the profile” for tobacco/alcohol-related oral cancer
    c. Not keeping up with current research
    d. Listening to the oral cancer experts’ recommendations

19. What is the survival rate when oral cancer is found late (i.e. stage IV)?
    a. 20-30%
    b. 40-60%
    c. 60-70%
    d. 70-80%

20. Which of the following should be noted during an intake phone call?
    a. “The patient complains of pain and swelling around the third molar, so he probably has an impacted wisdom tooth.”
    b. “The patient complains of pain and swelling around the area of the third molar.”
    c. “The patient has a cavity.”
    d. “The patient needs a root canal because he says that he thinks that his tooth is dying.”

21. Which of the following are presented by HPV-related oral cancer?
    a. Pre-cancer lesions
    b. Early symptoms
    c. Visible lesions
    d. Extra-oral abnormalities

22. How many viral strains are there of the human papilloma virus?
    a. More than 100
    b. More than 300
    c. Less than 50
    d. More than 50

23. What type of disease process does HPV-related oral cancer have?
    a. Simple
    b. Non-linear
    c. Well-understood
    d. Predictable

24. Which of the following should not be included in a comprehensive oral cancer screening?
    a. Extra-oral visual and tactile screening, including but not limited to palpation of the neck
    b. Intra-oral visual and tactile screening
    c. Use of adjunctive screening tools, i.e. tissue auto-fluorescence
    d. Only asking the patient about alcohol and tobacco use

25. How many conventional or oral sex partners must one have had to be at risk for developing HPV-related oral cancer?
    a. 10
    b. 1
    c. 0.5
    d. 25

26. Once infected with a high risk HPV virus, what will happen to that person?
    a. That person will develop oral cancer, but his or her prognosis may be poor if the dental professional screens for cancer early enough.
    b. That person will not develop oral cancer.
    c. That person’s immune system may fight off the infection, so that he/she will not develop oral cancer.
    d. That person will develop a terminal form of cancer.

27. What percentage of those infected with HPV-16 have a sufficiently healthy immune response to kick out the virus?
    a. 80%
    b. 53%
    c. 75%
    d. 99%

28. When did Dr. Maura L. Gillion first publish a research article on the casual association between HPV and oral cancer?
    a. 1968
    b. 2007
    c. 2000
    d. 1984

29. To avoid tragedy and prepare patients for total health and wellness, when should the hygienist conduct oral cancer screenings?
    a. Every two years
    b. Every year
    c. Every patient at every visit
    d. Once there has already been some indication of a HPV viral infection

30. On what does today’s comprehensive oral cancer screening and identification of patients at risk hinge?
    a. Visual and tactile screening
    b. Visual and tactile screening and the quality of the question asked
    c. Visual and tactile screening, the quality of the question asked, and relying upon what I learned in dental/hygiene school
    d. Visual and tactile screening, the quality of the question asked, and the use of auto-fluorescence screening tools
For immediate results, go to www.ineedce.com to take tests online. Answer sheets can be faxed with credit card payment to PennWell Corp. For Questions Call 216.398.7822

Educational Objectives
1. Discuss why patients with HPV-related oral cancer do not fit the profile of patients with tobacco/alcohol-related oral cancer.
2. Identify obstacles that thwart early discovery.
3. Discuss the clinical presentation of HPV-related oral cancer.
4. Determine that adjunctive screening tools do not help identify HPV-related lesions.
5. Implement the use of appropriate, revealing questions during the screening process.

Course Evaluation
1. Were the individual course objectives met?  Yes No
   Objective #1: Yes No
   Objective #2: Yes No
   Objective #3: Yes No

   Please evaluate this course by responding to the following statements, using a scale of Excellent = 5 to Poor = 0.
2. To what extent were the course objectives accomplished overall? 5 4 3 2 1 0
3. Please rate your personal mastery of the course objectives. 5 4 3 2 1 0
4. How would you rate the objectives and educational methods? 5 4 3 2 1 0
5. How do you rate the author’s grasp of the topic? 5 4 3 2 1 0
6. Please rate the instructor’s effectiveness. 5 4 3 2 1 0
7. Was the overall administration of the course effective? 5 4 3 2 1 0
8. Please rate the usefulness and clinical applicability of this course. 5 4 3 2 1 0
9. Please rate the usefulness of the supplemental webliography. 5 4 3 2 1 0
10. Do you feel that the references were adequate?  Yes No

   11. Would you participate in a similar program on a different topic?  Yes No
12. If any of the continuing education questions were unclear or ambiguous, please list them.

   13. Was there any subject matter you found confusing? Please describe.

   14. How long did it take you to complete this course?

   15. What additional continuing dental education topics would you like to see?

PLEASE PHOTOCOPY ANSWER SHEET FOR ADDITIONAL PARTICIPANTS.

CANCELLATION/REFUND POLICY
Any participant who is not 100% satisfied with this course can request a full refund by contacting PennWell in writing.

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