adha CLL2016
93RD ANNUAL SESSION
JUNE 8-14, 2016 / PITTSBURGH, PA

CE Course Handout

HPV: New Research, New Directions

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adha American Dental Hygienists' Association

American Dental Hygienists' Association, June 9, 2016

"The Human Papilloma Virus: New Research, New Directions"

Presenter – Jacquelyn L. Fried, RDH, BA, MS, Associate Professor and Director, Interprofessional Initiatives, University of Maryland School of Dentistry, Baltimore, Maryland; jfried@umaryland.edu

I. Introductory comments

   A. Course objectives

   B. Why is HPV important to us?

   C. Why has HPV Head and Neck Cancer (HNC) become so prominent?

   D. HPV HNCs in relationship to all HNCs

   E. Survival rates for HNCs – why unchanged?

      1. Looking

      2. Talking

II. History of HPV

   A. Identified in 80s

   B. Linked with genital cancers

   C. Today – known cause of HNCs

III. Characteristics of HPV

   A. More than 120 strains identified

   B. High risk and low risk types

   C. High risk cause malignancies

   D. 16 and 18 types – most commonly associated with cervical, anal, penile and HNCs

E. HPV 16 – present in 85-95% of HPV+ HNCs

F. Low risk types – may cause warts and intra-oral lesions

G. Clinical examples of HPV HNCs

H. Most prevalent oropharyngeal cancers (OPCs): tonsillar and base of tongue

IV. Terminology

A. HNCs – broad grouping

B. Sub-categories: OCs and OPCs

C. Two very different entities

V. Epidemiology of HNCs in U.S.

A. Prevalence- 60K individuals

B. Mortality - >12K

C. 8, 400 cases potentially caused by HPV

D. 3% of all cancers in U.S.

E. 25% of OPCs – HPV+

F. Of HPV+ - over 95% associated with HPV 16

G. Incidence rates – 228% growth in OPCs

H. Gender – males (3:1)

VI. Global epidemiologic data

A. Highest in developed western European countries

B. Growth rate of epidemic proportions

VII. HPV+ and HPV- (traditional carcinogen-induced)

A. Two separate diseases?

B. Case example – assessing risk factors

C. Differences

1. Populations affected
2. Clinical
3. Histopathological
4. Location
5. Tissue proclivity

VIII. Onset

A. Unknown

B. Peak prevalence – bi-modal

C. Ages – late 20s and 60s

D. First peak – sexual behavior

E. Second peak – suggested age changes related to immunological defenses; latency; stress events

F. Rationale for gender differences – sexual transmission and immunity

IX. From genital to oral infection

A. Genital infection precedes oral

B. Oral infection precedes HNC

C. Genital infection

1. most common STD
2. women – prevalence by virus type, year, age
3. ubiquitous but high clearance rate
4. persistent – most commonly associated with CA

5. histological progression of cervical CA

X. Oral infection

A. 7% of population infected

B. 3.7% high risk – 2K individuals

C. 14 fold increased risk for HPV+ HNCs

XI. Transmission routes

A. High risk sexual behaviors* - oral/genital; oral/anal

B. Prevalence of oral sex practices – note ages

C. Birth canal

D. Mother's milk

E. Auto-inoculation

F. Drinking straws?

G. Gillison – oral sex; not likely with kissing

H. D'Souza – the more oral sex, the greater the risk

I. Risks to current and future partners

J. Other associated risk behaviors

1. Age of sexual debut

2. #'s of partners

3. Lack of condom use

4. Oral sex with someone who has history of HPV+ Ca

K. Is HPV+ OSCC a sexually transmitted disease?

XII. Risk factors contributing to tumorigenesis

A. Environmental knowns and unknowns

B. Genetics – knowns and unknowns

C. Lifestyle behaviors

D. Tobacco use can exacerbate HPV associated HNCs

XIII. Life cycle/HPV behaviors

A. Little know about biologic mechanisms

B. May be cleared, latent, related to immune response, cause reinfection

C. Length of presence – may be persistent; become HNC

D. Serum marker is HPV 16 – E6

E. Behavior at cellular level –transformation from virus to host cancer

1. E6, E7 – early arrivers allow for replication and disrupt host tumor suppressors
2. Later arrivers – encode host proteins
3. E6 behavior – P53
4. E7 behavior - Rb

XIV. Theories re cancer development

A. Viruses cause cancer

B. Chronic inflammation

1. Role of oral hygiene (Thanh)
2. Periodontal disease as entry point (Tezal)

C. Bacteria

D. Saliva

E. If these theories hold, where does that put us?

XV. Detection

A. Patient symptomology/reporting

B. Tests for detecting HPV in oral mucosa

C. In-office detection devices – lack evidence for effectiveness

D. Technological detection devices

E. Salivary diagnostics

F. New developments in salivary and serum testing

G. Variation in findings

XVI. Diagnosis

A. Sophisticated assays:

1. IHC

2. ISH

3. PCR

B. Is finding HPV in the tumor good enough? NO!

C. E6 or E7 RNA must be found

D. Biopsy- gold standard

E. Specimen variation

XVII. Prevention

A. Risk behaviors that can be changed: tobacco, food choices, alcohol

B. Sexual behaviors???

C. Patient management and education – our roles

D. Challenges of discussing sensitive topics

1. Parental approval?

2. Discussing sex?

3. Reasonable segue ways

E. Advocacy outside of the employment setting – public health problem

1. Public speaking
2. Interprofessional topic

F. Vaccination recommendation

1. Australia data
2. Potential promise with oral HPV
3. Systemic vaccine – systemic effect

G. Marketed vaccines

1. Bi-valent
2. Quadra-valent
3. Nano-valent
4. Ages of administration: boys and girls between 9 and 11
5. Up to age 26

XVIII. Treatment

A. Depends on stage

B. Usually combination therapy (chemo-radiation)

C. Discussions of overtreatment re HPV+ neoplasms

D. Surgical interventions – highly disfiguring

XIX. Prognosis

A. Better with HPV+

B. Higher survival rate

1. Improved responses to therapy

2. Age factor – less entrenched co-morbidities
3. Different etiologic pathways
4. Supportive research data

XX. Benign lesions

A. Types

B. Treatment

C. Prognosis

XXI. Future possibilities

A. Unanswered questions

B. New technology

C. Vaccine for oral HPV?

XXII. Summary