Human papillomaviruses (HPVs) in lung cancer: A causative trigger or just a co-incidence?

Keywords: Human papillomaviruses HPV Lung cancer

Editor,

We read with interest the study by Kato et al. [1], which detected HPV DNA in 17% of the examined 42 lung tumors, suggesting that HPV presence in lung cancer may be significantly related to EGFR mutations.

Lung cancer is a worldwide leading cause of mortality strongly associated with the smoking habit since 1900s [2]. However, during the last decade literature reporting the presence of HPVs in lung cancer has expanded rapidly, supporting a viral etiology for a subset of patients developing lung cancer [3,4]. In the recent systematic review performed by Syrjänen, who analyzed 100 studies, covering 7381 lung cancer cases from different geographical regions, 22.4% of lung cancer samples were HPV-positive [4]. The presence of HPVs in lung cancer samples provides evidence of their potential oncogenic role as a causative trigger in lung carcinogenesis.

However, the presence of HPVs in bronchoalveolar lavage (BAL) samples collected from patients without lung cancer adds controversial evidence about the real role of these viruses in humans (unpublished data). Further to this data, our recent findings demonstrating the presence of HPVs in the lower respiratory tract of asymptomatic children proposes that infection with HPVs may occur early in childhood and has no clinical outcome. In our recent study [5] determining the prevalence of HPVs in bronchoalveolar lavage (BAL) samples obtained from asymptomatic immunocompetent children, HPV DNA was detected in 6 out of 71 children. The presence of HPVs in the lower respiratory tract of asymptomatic adults and children supports the co-incidence scenario. Nevertheless, the question remains whether the presence of HPVs in lung tissue trigger lung carcinogenesis or it is a simple coincidence. This requires further prospective investigation to elucidate the actual epidemiologic condition.

Conflict of interest statement

The authors do not have any actual or potential conflicts of interest to declare.

References


George Sourvinos * Ioannis N. Mammis Stavros Derrdas Demetrios A. Spandidos

Department of Clinical Virology, Faculty of Medicine, University of Crete, Heraklion, Crete, Greece

* Corresponding author. Tel.: +30 2810 394 835; fax: +30 2810 394 835. E-mail address: sourvino@med.uoc.gr (G. Sourvinos)

25 October 2012
doi:10.1016/j.lungcan.2012.10.014