

INCIDENCE OF CYTOMEGALOVIRUS INFECTION IN NSCLC CYTOLOGY

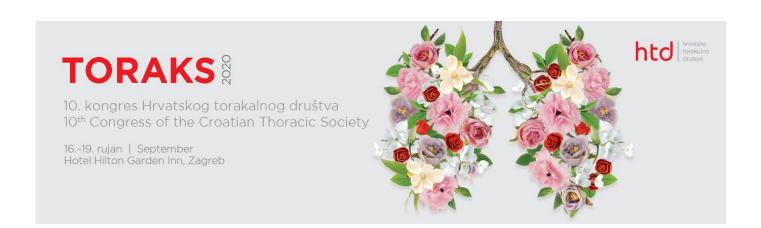
HARABAJSA S.¹, Šefčić H.², Vrabec Branica B.¹, Šimić V.¹, Milavić M.³, Židovec Lepej S.⁴, Badovinac S.⁵, Jakopović M.^{5,6}, Smojver-Ježek S.^{1,6}, Korać P.²

- ¹ University Hospital Centre Zagreb, Zagreb, Croatia

 Department of Pathology and Cytology, Division of Pulmonary Cytology Jordanovac
- ² Faculty of Science, University of Zagreb, Zagreb, Croatia Department for Biology, Division of Molecular Biology
- ³ School of Medicine, University of Zagreb, Zagreb, Croatia Institute of Pathology
- ⁴ University Hospital for Infectious Diseases Zagreb, Zagreb, Croatia Division of Immunology and Molecular Diagnostics
- ⁵ University Hospital Centre Zagreb, Zagreb, Croatia Department of Respiratory Diseases Jordanovac
- ⁶ School of Medicine , Zagreb, Croatia University of Zagreb

Objective:

Aim: Presence of human cytomegalovirus DNA (HCMV) in lung cancer cells may alter the activity of



cellular proto-oncogenes or tumor suppressor genes which further can result in cancer development as well as modulation of response to the cancer treatment. Aim of this study was to determine the incidence of HCMV infection in non-small cell lung cancer (NSCLC) cytology.

Methods: This study included 67 NSCLC cytological smears and their DNA isolates from newly diagnosed lung cancer patients hospitalized at the Department of Respiratory Diseases Jordanovac, University Hospital Centre Zagreb. The cytological smears were from samples obtained during bronchoscopy, fine needle aspirations and pleural effusions. The DNA was extracted from NSCLC cytological smears stained by May Grünwald Giemsa staining. The two years retrospective analysis included 34 NSCLC with EGFR gene mutations and 33 NSCLC without EGFR gene mutations. Specific fragments for glycoprotein B (gB) and immediate-early (MIE) gene of HCMV were amplified by polymerase chain reaction method.

Results: Among 34 NSCLC with EGFR mutations were 14 males (41,2%) and 20 females (58,8%). Among 33 NSCLC without EGFR mutations were 13 males (39,4%) and 20 females (60,6%). The median age of both groups was 69 years. Among NSCLC with EGFR mutations were 24 non-smokers, 8 smokers (including ex-smokers), and two with no smoking status. Among NSCLC without EGFR mutations were 23 smokers (including ex-smokers) and 10 non-smokers. The HCMV MIE gene was detected in: 13 (38,2%) samples of NSCLC with EGFR gene mutations, in one (12,5%) smoker and 11 (45,8%) non-smokers, four (28,6%) males and nine (45,0%) females. The HCMV MIE gene was not detected in samples without EGFR mutations. The HCMV gB gene was detected in: four (13.3%) samples of NSCLC with EGFR gene mutations and in one (3,1%) sample without EGFR gene mutations, in three (37,5%) smokers and one (4,2%) non-smoker, two (14,3%) males and two (10,0%) females. More frequent HCMV infection was determined on the basis of MIE gene detection in NSCLC with EGFR gene mutations (p <0.001). There was no statistically significant association of HCMV (MIE and gB) infection with age, gender, and smoking status in NSCLC with EGFR gene mutations.



Conclusion: Our results indicate more frequent HCMV (*MIE*) infection in NSCLC with *EGFR* gene mutations. More extensive research is needed to determine the incidence of HCMV infection in NSCLC cytology and its possible role in NSCLC pathogenesis.