Human Viruses, bacteria and cancers

Luigi Santacroce

University of Bari, Italy

Actually, several studies have established a relationship between microorganisms and chronic conditions such as atherosclerosis, neurologic disorders, cancer, and obesity. The link between microorganisms and increasing numbers of diseases never before envisioned as having microbial etiology opens fascinating scientific, medical, and public health perspectives. Apart from bacteria as Helicobacter pylori, experimental and epidemiologic data show a causative role for viruses, particularly in cervical and cancer of the liver, than viruses must be thought of as one of the most important risk factor for cancer development in humans. As a consequence, today we will be sure that a lot of cancers have aetiologies with infectious agents as necessary factors. Several DNA tumor viruses encode viral oncoproteins which will directly transform the cells. In vitro tests (i.e., using the NIH/3T3 cell stocks) allowed to clarify that the nonviral tumors have endogenous activated oncogenes. Generally, tumor viruses, after the infection of their host, determine mild disease conditions or no, or cause non-neoplastic diseases (e.g., HBV). This natural condition is simply one among the explanations why it’s so difficult to spot the viral agents as causal factors for human cancers. HPV is one among the foremost recent virus focused as liable for cancers aside from cervical. The clinical scenarios of HPV infection depend from the location of the lesion and therefore the virus serotype. In fact, HPV DNA was detected in 100% of cervical carcinomas, 40% in tumors of the penis, also as vulvar and vaginal, in 90% in anal carcinomas, 12% in oropharyngeal carcinomas and 3% of cancers of the mouth. Viruses may contribute to the event of human tumors both indirectly, inducing immunosuppression or modifying the host cell genome without persistence of viral DNA, and directly inducing oncoproteins or by altering the expression of host cell proteins at the location of viral DNA integration.

One of the maximum recognized chronic human bacterial infection is Helicobacter pylori (H. pylori), about 50% of populations were infected. It's one of spiral, flagellated, motile gram –ve bacteria forming urease enzyme in stomach for survival. There is many varies between nations as regard the prevalence of H. pylori; the prevalence varies between 7 and 33% as declared by European studies. In developing countries H. pylori is usually acquired in childhood before the age of 10 years, while the age related increase in occurrence presented in developed countries.

H. pylori have a critical view inside the occurrence of pathogenesis of many upper GIT pathologies such as gastritis, peptic ulcer disease (PUD) and gastric cancer. Many researches detected that H. pylori have the ability for affecting extra-intestinal organs producing diseases like liver and bile tract disease, ear and eye diseases, cardiovascular disease, diabetes mellitus, immunological and haematological, gynecological and pulmonary pathologies.

In developed countries, the rate of occurrence of allergic airway diseases like asthma has increased over years. Till now, the reasons for this increased prevalence still unknown. There is a suggested association between allergic asthma and lots of factors including changes in smoking habits, exposure to infections transmitted orofecally and food born, having a furry animals, number of siblings, the level of income and education and presence of particulates in diesel exhaust. Besides, there is a crucial relationship between infection in childhood, especially with Helicobacter pylori and allergic diseases.

Various pro-inflammatory substances such as cytokines and eicosanoids have released from the gastric mucosa due to its colonization by H. pylori. Therefore, a pathogenetic link between H. pylori infection and disease characterized by activation of inflammatory mediators and/or induction of autoimmunity might exist. The diagnosis of H. pylori infection can be done by detecting IgG antibodies to H. pylori in blood, urea breath test by drinking 13C-labeled or 14C-labelled urea, H. pylori–specific antigens in stool and endoscopic biopsy from the pre-pyloric region of the gastric mucosa by many techniques.

The aim of this study is to detect the possible relationship between is to detect the possible relationship between clinical and functional severity of bronchial asthma and H. pylori infection for better controlling of the disease.

A cross sectional Study administered at the outpatient clinic of chest department, Zagazig University Hospitals, during the amount from February 2015 to December 2015. One hundred and twenty asthmatic patients were selected randomly from bronchial asthma patients attended to outpatient clinic. They were agreed to do spirometry and H. pylori tests. They were (91 females) with mean age ± SD (36.96 ± 5.09) years and (29 males) with mean age ± SD (38.86 ± 4.29) years & with age range from (26–44) years.

The more degree of severity of asthma, the less +ve H. pylori cases, with a highly statistically significant difference between +ve and –ve H. pylori as regard severity of bronchial asthma. There was very highly statistically significant difference between mild, moderate, severe persistent asthma as regard H. pylori IgG. (P value < 0.001). Mild asthma were most +ve H.
pylori (58.3%), while moderate and severe cases were (31.3%) and (10.4%) respectively.