Infective Endocarditis in Cancer Patients: Incidence, Mechanisms and Approach

Wilhelm P. Mistiaen* 
Department of Physiotherapy, Faculty of Medicine & Health Sciences, University of Antwerp, Belgium

EDITORIAL

Infective endocarditis (IE) has a high mortality rate on short-term, even after appropriate medical or surgical treatment. The presence a second potentially life-threatening disease as midterm such as cancer could pose serious therapeutic dilemmas. The association between both conditions was described in the 1950 and its findings were confirmed in the following decades [1-3]. The questions to be answered are: how often does this association occur and how can be dealt with in an appropriate way? Many case reports, and short series were published. There are also some more contemporary genomic and cell biological studies available. However, prospective case control series and nationwide surveys are rare. Between 1990 and 2010, cancer has been consistently present at a level of 11%-12% in elderly Medicare patients diagnosed with IE [4]. In patients with IE caused by Streptococcus galloyticus, more than half had also colorectal cancer or polyps. This is far more compared to the general asymptomatic population [1]. Such high figures have been confirmed in other series [3,5-8]. Increase of hepatobiliary and pancreatic as well as some other cancers have also been detected [6,8,9]. This is especially true within the first three months after an episode of infective endocarditis. This cancer rate drops however in the following years [9], which is a strong indicator for its association with IE.

The digestive system is the third most common portal of entry, after the skin and the oral cavity [10]. This is corroborated by the observation that the incidence of colorectal cancer in patients with IE caused by Streptococcus galloyticus is much higher compared to other types of cancer [11]. In patients with cirrhosis, an increased permeability of the gut wall also might play a role in bacteremia [12]. An oral portal of entry is important in patients with hematologic disease, especially when this condition is complicated by gingivitis and periodontitis. Inflammation and ulceration caused by chemotherapy increases the likelihood of bacterial entry into the circulation [13]. Strains of Streptococcus viridans are important for this type of malignancies. In patients with colorectal neoplasias, Streptococcus galloyticus might just be a bystander [5,9,14], because the vicinity of tumor tissue could offer a competitive advantage. However, this is still uncertain. These microorganism also could have oncogenic capacities because of its ability to activate cytokines, COX-2 mediated inflammation, neo-angiogenesis, and further degeneration by disabling apoptosis [8,12]. This relation is clouded by the fact that carriage of Streptococcus galloyticus is unstable in time. In some patients, this microorganism has been identified in an earlier stage while it seemed absent in a later period [2,5].

The mechanism of virulence could be related to bacterial pili, with stronger binding capacity to collagen and formation of biofilms in tumors and damaged valves [1,2,14]. There is also a possible role of a polysaccharide capsule in aiding to avoid the immune system. This allows crossing of the epithelial barrier without provoking epithelial interleukin response and enhances also the ability of the microorganism to form a biofilm around collagen in diseased heart valves [6]. Similar findings were made for Streptococcus infantarius and Streptococcus pasteurianus. Furthermore, Streptococcus infantarius spp. coli is capable to invade tissues and to survive within macrophages. These findings stress the importance of biological typing of Streptococci in IE [1,2,6,15] but this is not always performed [5]. Another mechanism for IE to develop is immune suppression [10], especially in patients with hematologic disease. These mechanism include neutropenia, insufficient phagocytosis, abnormal B and T cell function, and chemotherapy-related immune suppression. An age effect or use of alcohol could be other explanations for decrease in immunity [11]. Remarkably, low platelets, as result of chemotherapy may be protective. In contrast, thrombophilia might result in formation of sterile vegetation which might serve as precursor for IE [8].

Few data are available concerning the outcome in patients with IE and cancer. Overall survival is diminished. Colorectal cancer-specific survival is not, however. Infective endocarditis leads to changes in oncologic treatment but not to worse oncologic outcome [11]. Age adjusted Charlson comorbidity index is also a predictor for all-cause hospital mortality and of long-term survival after infective endocarditis. With increasing of the index, the presence of cancer also increased [2% if 2 or less, 13% if between 3 and 5, and 21% if the index is more than 5]. The main “drivers” of age adjusted Charlson comorbidity index are cancer, diabetes and heart failure. Treatment strategy should be guided by the abovementioned findings. Colonoscopy is recommended in all patients with bacteremia or IE by Streptococcus galloyticus.
REFERENCES


