ICoLA 2019
The 8th International Congress on Lipid &Atherosclerosis(ICoLA)
The 58th Conference of the Korean Society of Lipid & Atherosclerosis
September 5(Thu.) ~ 7(Sat.), 2019, Conrad Hotel Seoul, Republic of Korea

Clonal Hematopoiesis; Is It a New Risk Factor for Metabolic Dysfunction?

Changhee JUNG

Department of Internal Medicine, Asan Medical Center, University of Ulsan College of Medicine, Korea

chjung0204@gmail.com

Abstract

The accumulation of somatic mutations in hematopoietic stem/progenitor cells (HSPCs) is known to be an inevitable consequence of the process of aging. Some of these random mutations confer a competitive advantage to the mutant cells, leading to clonal expansion. This phenomenon is called as age-related clonal hematopoiesis (CH). The genes that were most commonly mutated in CH were *DNMT3A, TET2* and *ASXL1*.

A number of studies have associated with CH with an increase in all-cause mortality. Although the presence of CH was associated with the increased risk of hematologic cancer, this only affected 0.5% to 1% of mutation carriers each year and did not explain the marked increase in all-cause mortality. Instead, the increased all-cause mortality was attributable to increased risk of cardiovascular disease (CVD). Based on these epidemiological data, several groups tried to elucidate the possible molecular mechanism underlying the presence of CH and CVD such as atherosclerosis, myocardial infarction and heart failure.

In addition to the causal link between CH and CVD, a modest and significant association between CH and type 2 diabetes was observed. In this talk, I'd like to introduce the possible causal link between the loss of function in *DNMT3A*, the most frequently mutated gene in CH, and metabolic dysfunction including adipose tissue inflammation in mouse.

Keywords

Clonal hematopoiesis, DNMT3A, Type 2 diabetes, Metabolic dysfunction, Adipose tissue inflammation