02. Adiponectin

by Lukman Hakim

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Editorial

Adiponectin and Proto-oncogene MYC in Prostate Cancer: How Far Are We with the Evidence?

The GLOBOCAN data 2018 shows that prostate cancer (PCa) is the most common malignancy among males, and represents the 5th leading death of cancer in men.[1] Although some risk factors have been identified, the clear etiology of this disease remains unclear.

Obesity, adiposity, and high body mass index have found to be associated with PCa and its biochemical recurrence, but the underlying mechanism remains unknown.[2,3] Adipocyte is a secretory organ that produces several hormones, cytokines, and growth factors called adipokines. Among these adipokines, adiponectin (APN) has been robustly studied and found to be associated with PCa.[4-6]

Studies on the association between APN and PCa have resulted in contradictory results. Some studies have proven its association, whereas others failed.[7,8] Among those who succeeded, some confirmed positive correlations, whereas others have negatively reported.[9-11] One of the rational explanations behind these conflicted evidence was proven to be correlated with the genetic polymorphisms, leading to various risks associated with PCa. The AdipoQ rs2241766 allele and AdipoR1 rs10920531 were associated with higher risk of PCa; meanwhile, the AdipoR1 rs2232853 variant was associated with a lower risk.[12]

The current predominance of evidence has shown an inverse correlation between APN and prostate malignancy. Therefore, an alteration of APN may potentially be used as a marker for early detection, to predict metastasis and a guidance for targeted therapy.[13]

Moreover, the proto-oncogene MYC is a family of regulator genes that code for transcription factors. It consists of c-MYC (also refers to MYC), I-MYC, and n-MYC. The proto-oncogene MYC has been thought to be responsible for the regulation of cellular metabolism, proliferation, and apoptosis. Its amplification occurs in 10%–30% of localized PCa, and more than 50% of advanced tumors have been linked to poorer prognosis.[14-17]

Studies on MYC as a biomarker for PCa have been intensively conducted, mainly using immunohistochemistry technique to identify the expression of MYC and correlate them with the clinicopathological aspects of PCa. Various MYC antibodies, cellular staining localization (nucleus or cytoplasmic), and scoring systems have been adopted, leading to various conclusions related to PCa.[18] A study by Pettersson *et al.* (2018) on MYC at the protein and mRNA level has shown that neither MYC protein overexpression nor MYC mRNA overexpression is a strong prognostic marker following radical prostatectomy.[17] Some studies have reported a positive association between MYC expression and clinicopathological factors of PCa, whereas others have negatively reported or even proven to be unassociated.[19-21] The short half-life of MYC protein, the different role between MYC protein and MYC gene amplification, and the possibility of different gene amplification rather than 8q/8q24 are all the possible explanations to these contradictory evidence.

Looking back to the current evidence of APN and proto-oncogene MYC in their relation to the clinicopathological factors of PCa, further studies need to be conducted focusing on the exact association, prior to its application in the clinical setting.

Lukman Hakim*

Department of Urology, Faculty of Medicine at Airlangga University, Dr. Soetomo Hospital, Surabaya, Indonesia

Address for correspondence:
Dr. Lukman Hakim,
Department of Urology, Faculty of Medicine at Airlangga University/
Dr. Soetomo Hospital, Mayjend Moestopo 6-8, Surabaya 60286, Indonesia.
E-mail: lukman-h@fk.unair.ac.id

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PAGE 1

PAGE 2