

Editorial



Usefulness of Plasma Pentraxin 3 Levels in Acute and Chronic Inflammatory Diseases

Sang Hoon Han

Division of Infectious Disease, Department of Internal Medicine, Yonsei University College of Medicine, Seoul, Korea

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Address for Correspondence:

Sang Hoon Han, MD, PhD

Division of Infectious Disease, Department of Internal Medicine, Yonsei University College of Medicine, 50-1 Yonsei-ro, Seodaemun-gu, Seoul 03722, Korea.

E-mail: shhan74@yuhs.ac

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ORCID iDs

Sang Hoon Han

<https://orcid.org/0000-0002-4278-5198>

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► See the article “Association between Plasma Pentraxin 3 Levels and Bone Mineral Density in Elderly Koreans: the Dong-gu Study” in volume 33, e165.

Long pentraxin 3 (PTX3) is an acute phase reactant and one of the pattern recognition receptors (PRRs) binding a wide range of pathogen/damage associated molecular patterns (PAMP/DAMP).¹ It plays an important role in the innate immune system, like C-reactive protein (CRP), which is a short pentraxin synthesized in the liver and as an opsonin-activating classical complement pathway.^{2,3} The PTX3 is produced at a local inflammatory site by numerous cells including vascular endothelial cells, lung or tubular epithelial cells, mesangial cells, smooth muscle cells, synoviocytes, differentiating adipocytes, myeloid cells (especially neutrophil), monocytes, dendritic cells, and tumor cells.³ Because the PTX3 basically interacts with C1q of the first component of the classical complement cascade and can induce acute or chronic inflammation through heterogeneous cell types in many organ sites, it has the pleomorphic biological functions of a variety of medical fields including infectious disease including sepsis caused by bacteria, fungi or viruses (activation of microbes recognition and prompt innate immunity), cardiovascular diseases (endothelial dysfunction), metabolic diseases/obesity, wound healing or tissue remodeling (fibrin degradation and matrix deposition), autoimmune inflammatory rheumatic diseases (recognition of self vs. non-self), and cancers (anti-angiogenic or pro/anti-tumorigenic functions).²⁻⁶ The PTX3 has the different effects of promoting or inhibiting cancers according to the tumor cell type.⁴

In the current issue of *Journal of Korean Medical Science*, Lee et al.⁷ analyzed the relation of plasma PTX3 levels to bone mineral density (BMD) indicating osteoporosis in 1,440 individuals among a prospective ongoing large urban cohort. Similar with previous reports, the PTX3 levels had the significant positive correlation with CRP and white blood cell count.³ In spite of relative small coefficient values (r), the PTX3 was a significant independent factor related to BMD with negative correlation. These findings might suggest that the high PTX3 levels could play a protective role of osteoporosis development. The basic in vitro and animal study for the effect of PTX3 on osteoclasts and osteoblasts activities evaluating bone formation homeostasis will be warranted to verify the author's hypothesis. In this report, the women population did not show the association between PTX3 and BMD. The previous studies reported that the PTX3 can have a potential effect on fertility by several mechanisms.⁴ In addition, the PTX3 can induce specific hormones such as thyroid-stimulating hormone and insulin.⁴ Based on further epidemiologic investigation with a large population of women participants, the measurement of various hormone levels could confirm and explain this phenomenon.

Even though the PTX3 levels can be affected by complex processes and pathways including cytokines, chemokines, growth factors, and particular promoter genes,⁴ the measurement of plasma PTX3 levels is still fascinating, because it has enough worth as the understanding of disease pathogenesis, diagnostic or prognostic surrogating biomarker, and therapeutic candidate in various diseases according to specific conditions. However, the several prospective multicenter studies on large scale and comprehensive review including meta-analysis should be performed to apply it to usual clinical practice for certain diseases.

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