Editorial

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Novel Biomarker in the Diagnosis of Acute Aortic Dissection

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Acute aortic dissection (AAD) is caused by a tear in the intimal lining of the aorta that extends into the media of the wall [1]. Blood in the aortic media then pushes the dissection flap into the middle of the aorta, separating the true from the false lumen. AAD is a serious condition that can result in significant mortality and morbidity. Aortic dissection mortality increases by 1%–2% per hour after symptom onset. In many cases, immediate intervention is required. Thus, prompt diagnosis is mandatory.

Although imaging is the key tool in the diagnosis of AAD, the assessment of biomarkers is under investigation as a potential rapid diagnostic approach. The following are recognized as the most common biomarkers: white blood cell count, D-dimer, fibrinogen, and C-Reactive Protein (CRP). In particular, D-dimer has high sensitivity for the diagnosis of AAD [2] and is therefore useful for ruling out AAD. However, there are limitations, such as cases of false lumen occlusion and younger patients with AAD, where D-dimer levels remain normal. Additionally, the cutoff value for D-dimer varies depending on the laboratory analytical reagent, so it is necessary to check the cutoff value at each hospital.

This study by Higuchi and colleagues is the first report to analyze the kinetics of citrullinated fibrinogen (C-Fbg) in AAD [3]. C-Fbg is produced when proteins such as fibrinogen are converted by peptidylarginine deiminase in neutrophils during inflammation. It is expected that the pathogenesis of AAD is related to the mechanism of C-Fbg elevation. In this exploratory study, the authors aim to assess whether blood C-Fbg levels are significantly elevated in AAD and whether they differ from those observed in acute coronary syndromes. In this study, C-Fbg showed a significant correlation with D-dimer. It was also found that while D-dimer decreased in blood concentration over time, C-Fbg remained high. These findings suggested that C-Fbg might contribute to the early diagnosis of AAD.

Additionally, an interesting finding of this study is that C-Fbg did not correlate with CRP. C-Reactive Protein (CRP) is an acute-phase reactant produced by the liver in response to cytokines. CRP is an independent risk factor for vascular inflammation and a prognostic factor for cardiovascular events [4]. It has also been confirmed that C-Fbg increases in inflammatory diseases such as rheumatoid arthritis. Based on this report, clinical application to inflammatory aortic syndrome is expected.

References

1) LeMaire SA, Russell L. Epidemiology of thoracic aortic dissection. Nat Rev Cardiol 2011; 8(2): 103-13.
2) Yao J, Bai T, Yang B, Sun L. The diagnostic value of D-dimer in acute aortic dissection: a meta-analysis. J Cardiothorac Surg 2021; 16(1): 343.
3) Fujimura S, Higuchi Y, Izawa et al. Evaluation of serum citrullinated fibrinogen in patients with acute aortic dissection. LMI 2024; 3(2): 50-58.
4) Kaptoge S, Di Angelantonio E, Pennells L, et al. C-reactive protein, fibrinogen, and cardiovascular disease prediction. N Engl J Med 2012; 367(14): 1310-20.