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**N-terminal pro-brain natriuretic peptide elevation in Kawasaki disease with clinically normal cardiac function: Relation with cytokines**

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Background: N-terminal pro-brain natriuretic peptide (NTproBNP) is often elevated in patients with Kawasaki disease (KD) that shows clinically normal cardiac function in acute phase, and is reported as a marker for diagnosis, as well as an index of heart failure. Cardiac function in acute phase of KD has not been investigated 3D speckle tracking.

Objectives: We sought the cause of NTproBNP elevation by investing the correlation NTproBNP with cytokines and 3D-GLS.

Methods: Forty-eight patients in our hospital in 2015 were studied. Age ranged from 3m to 11y. We measured NTproBNP, TNF $\alpha$ , its receptors, IL-6, IL-10 and indices of cardiac functions simultaneously before and 1 week after immunoglobulin treatment (IVIg).

Results: The mean plasma NTproBNP before/after IVIg were 750/132pg/ml, TNF $\alpha$  were 4.6/2.9pg/ml, sTNFR1 and 2 were 2827/1112pg/ml and 6617/3143pg/ml, IL-6 were 101/9pg/ml, IL-10 were 32/8 pg/ml. The cytokines after IVIg were significantly lower than before IVIg ( $P < 0.01$ ). 2DEF were 70.5/75.9%, 3DEF were 66.8/69.2%, GLS were -19.8/-21.3%. GLS was not correlated with NTproBNP, but 3DEEF was weakly correlated. Correlation between sTNFR's and NTproBNP were sTNFR( $r=0.57/0.48$ ), sTNFR2( $r=0.35/0.59$ ), and IL-6( $r=0.65/0.42$ ), which showed significant correlations.

Conclusion: Although correlations between NTproBNP and 3D cardiac function were not apparent, correlation of NTproBNP and TNF-family was distinct. NTproBNP can be a marker reflecting inflammation of KD even in a case without clinically significant cardiac dysfunction.