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Determinant of increased type B natriuretic peptide (BNP) in acute phase of Kawasaki disease

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BACKGROUND: The aim of this study was to identify the determinant of increased type B natriuretic peptide (BNP) in patients with acute Kawasaki disease (KD).
METHODS: Subjects were 56 patients with acute KD and 21 febrile controls (FC) and they underwent multi-modal echocardiographic studies to determine functional parameters including LVEF, LVDd transformed into z-value (zLVDd), LV volume indexed to body surface area (LVVI), Tei-index, and E/e'. In addition to white blood cell count (WBC) and C-reactive protein (CRP), inflammatory cytokines including interleukin (IL)-1ß, IL-6, IL-8, IL-10, IL12p70, and tumor necrosis factor were determined in 47 of KD and 10 of FC. Echocardiographic and laboratory data were compared between KD and FC. Also, correlations between Log (BNP) and these data were determined.
RESULTS: There was no significant difference in age, days of illness at study, WBC, CRP, but log (BNP) in KD was significantly higher than that in FC (1.58 ± 0.64 vs. 1.01 ± 0.44, p=0.0003). Though, zLVDd (0.81 ± 1.00 vs. 0.22 ± 0.74), LVVI (58 ± 22 vs. 46 ± 12 ml/M2, p<0.03), E/e' (9.6 ± 2.3 vs. 8.4 ± 1.6, p<0.05) in KD were significantly higher than those in FC, there was no significant difference in LVEF and LV Tei-index. Among the inflammatory cytokines, only IL-6 in KD was significantly higher than that in FC (233 ± 241 vs. 46 ± 37 pg/ml, p<0.02). Among the parameters that correlated with Log (BNP) including zLVDd (r = 0.35, p < 0.005), LVVI (r = 0.33, p < 0.004), LVEF (r = -0.27, p < 0.002), E/e' (r = 0.30, p<0.03), WBC (r = 0.38, p < 0.01), CRP (r = 0.51, p = 0.0003), and IL-6 (r = 0.76, p < 0.0001), multiple stepwise regression analysis identified IL-6 as the single most significant predictor of Log (BNP) (β = 0.77, p < 0.0001).
CONCLUSION: In patients with acute Kawasaki disease, BNP is significantly increased with mildly dilated left ventricles and this increased BNP is associated with not only left ventricular systolic dysfunction but also inflammation itself represented by increased IL-6.