

Eosinophilic myopericarditis caused by toxocaracanis

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A 21-year-old woman visited emergency department due to epigastric pain, nausea, vomiting, headache and dizziness from 3 days ago. The patient took medications because of upper respiratory infection and myalgia 3 days ago, with no past medical history. BP was 80/60 mm Hg and PR was 100/minute. RR was 20/minute and BT was 36.6 [ordm]C. Brain CT showed no significant lesions with no focal neurologic deficits. Abdomen and pelvis CT showed right pleural effusion, pericardial effusion and focal ascites in pelvis. CBC count showed eosinophilia (eosinophil count 9800/[mu]L with 40% eosinophil) and cardiac enzyme was elevated (CK-MB = 22.6 ng/mL, troponin T-hs = 1.21 ng/mL). A TTE revealed decreased LV systolic function (EF = 44%) with moderate pericardial effusion (0.9 ~ 1.3 cm). She was diagnosed as hypereosinophilic myopericarditis and was hospitalized in intensive care. Despite of applying intravenous methylprednisolone, pericardial effusion was increased (2.3 ~ 2.4 cm), so pericardiocentesis was done. Pericardial fluid analysis appeared 90% of eosinophilia with high protein level (5294.20 mg/dL). As toxocaracanis antibody and sparganum antibody showed positive in serologic test, albendazole and paraziquantel were applied. Peripheral eosinophilia disappeared (eosinophil count 430/[mu]L) with 10mg of prednisolone. LV systolic function was improved (EF = 73%) and pericardial effusion was disappeared 1 month later after discharge. The patient visits out-patient department, taking 2.5mg of prednisolone without any discomfort.

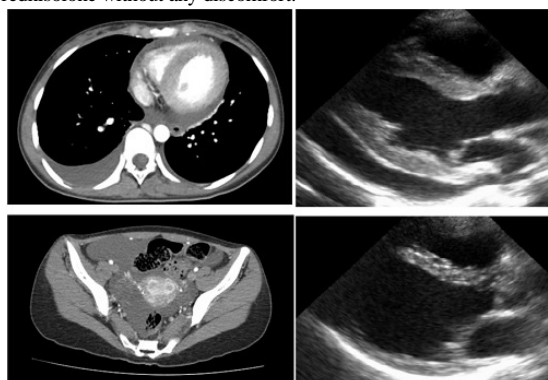


Figure 1. fluid collection in abdomen

Figure 2. TTE, pericardial effusion

Isolated systolic hypertension associates with carotid intima media, intima, media thickness

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Background and objectives: It is known that systolic BP (SBP) but not diastolic BP (DBP) is associated with carotid intima-media thickness. But it has not been studied well that which one is more associated with SBP between the carotid intima thickness (CIT) and the carotid media thickness (CMT). We evaluated the association between SBP and CMT, CIT and CMT. **Results:** Consecutive 1,760 subjects were enrolled, divided into 3 groups; isolated systolic HTN (ISH; SBP \geq 140 mmHg & DBP < 90 mmHg), isolated diastolic HTN (IDH; SBP < 140 mmHg & DBP \geq 90 mmHg), and systolic and diastolic HTN (SDH; SBP \geq 140 mmHg & DBP \geq 90 mmHg). ISH group (N = 151) has thicker CMT, CIT and CMT except Lt. CMT than controlled BP group (Lt. CMT = 0.74 \pm 0.16 mm vs. 0.69 \pm 0.17 mm, p = 0.002; Lt. CIT = 0.33 \pm 0.08 mm vs. 0.31 \pm 0.07 mm, p = 0.002; Lt. CMT = 0.42 \pm 0.12 mm vs. 0.39 \pm 0.13 mm, p = 0.051; Rt. CMT = 0.73 \pm 0.19 mm vs. 0.68 \pm 0.16 mm, p < 0.001; Rt. CIT = 0.32 \pm 0.07 mm vs. 0.30 \pm 0.06 mm, p = 0.013; Rt. CMT = 0.42 \pm 0.14 mm vs. 0.39 \pm 0.13 mm, p = 0.001). IDH group (N = 66) and SDH group (N = 169) had no significant difference of CMT, CIT and CMT than controlled BP group. The pulse pressure of ISH group was significantly higher than IDH group (67.9 \pm 10.9 mmHg vs. 40.1 \pm 6.4 mmHg, p < 0.001) and SDH group (67.9 \pm 10.9 mmHg vs. 56.6 \pm 10.8 mmHg, p < 0.001). This could be one reason that ISH group had thicker CMT, CIT and CMT than IDH and SDH group. **Conclusion:** ISH group was only correlated with increased CMT, CIT and CMT than controlled BP group. IDH and SDH group had not showed significant difference than controlled BP group.

