ows compared with findings on coronary angiography (CAG), MRI, and intravascular ultrasound.

RESULTS: Linear shadows were detected in 11 CALs on high-resolution 2DE in 9 patients with Kawasaki disease. The outer diameters of CALs on 2DE (7.0 ± 2.1 mm) were larger than those on CAG (4.4 ± 1.6 mm), whereas the inner diameters between linear shadows (3.9 ± 1.6 mm) were almost equal to the diameters of CALs on CAG. There was a statistically significant positive correlation ($y = 0.99x - 0.10$; $r^2 = 0.77$) between the diameters of CALs on CAG and the inner diameters between linear shadows on 2DE. A thickened intima was revealed in the same regions that showed linear shadows on 2DE, in 7 of 11 lesions on MRI, and in all 4 lesions on which intravascular ultrasound was performed. In 3 patients who had been followed up over 3 years, linear shadows inside CALs on 2DE persisted, and the diameter between linear shadows was almost consistent with the diameter of CALs on CAG.

CONCLUSIONS: These results suggest that linear shadows inside CALs on 2DE would reflect the existence of a thickened intima. We expected that following up the changes of linear shadows inside CALs was useful for noninvasive evaluation of coronary arterial remodeling such as intimal hypertrophy or stenotic change.

LONG-TERM FOLLOW-UP RESULTS OF PERCUTANEOUS CATHETER INTERVENTION FOR CORONARY ARTERY LESIONS AFTER KAWASAKI DISEASE: MULTICENTER COLLABORATIVE STUDY

Submitted by Masahiro Ishii

Masahiro Ishii[a], Hiromi Muta[b], Yayoi Nakahata[a], Syouhei Ogata[a], Sumito Kimura[a], Mamoru Ayusawa[c], Motofumi Iemura[b], Syunichi Ogawa[d]

[a]Kitasato University, Tokyo, Japan; [b]Kurume University, Fukuoka-ken, Japan; [c]Nihon University, Tokyo, Japan; [d]Nihon Medical University, Tokyo, Japan

INTRODUCTION: The long-term prognosis in patients with Kawasaki disease (KD) after percutaneous coronary intervention (PCI) remains unclear.

OBJECTIVE: We sought to clarify the long-term results of PCI for KD.

METHODS: Patients who developed coronary stenotic lesions caused by KD and were treated with PCI were investigated. Restenosis or obstruction was diagnosed when the stenosis was ≥75% according to coronary angiography or ischemic change was observed by myocardial perfusion imaging.

RESULTS: A total of 55 stenotic lesions were reported in 49 patients in 5 institutions. The types of PCI included percutaneous transluminal coronary angioplasty (n = 22), stent implantation (n = 7), percutaneous transluminal coronary rotational ablation (PTCRA) (n = 22), and combination of PTCRA with stent implantation (n = 4). Median age at PCI was 14.5 years, and the median follow-up period in the PCI group was 6.3 years. of 55 stenotic lesions in the PCI group, 52 (95%) were dilated successfully by PCI. Immediate complications in the PCI group included neoaneurysm in 5 patients, transient bradycardia in 3 patients, and atrial fibrillation in 1 patient. Treatment for restenosis in the PCI group included re-PCI in 3 patients, coronary artery bypass grafting in 6 patients, and heart transplantation in 1 patient. No patient in the PCI group died. There was no difference in effectiveness among the 3 PCI devices (percutaneous transluminal coronary angioplasty versus stent implantation versus PTCRA: log-rank test, P = .3).

CONCLUSIONS: PCI for KD can be accomplished and can be effective in the long-term.

EXPOSURE TO TOBACCO SMOKE DECREASES ELASTICITY OF THE AORTA IN CHILDREN

Submitted by Katariina Kallio

Katariina Kallio[a], Eero Jokinen[b], Olli Simell[c], Olli Raitakari[d]

[a]Research Center of Applied and Preventive Cardiovascular Medicine and Departments of [c]Pediatrics and [d]Clinical Physiology, University of Turku, Turku, Finland; [b]Department of Pediatrics, University of Helsinki, Helsinki, Finland

INTRODUCTION: Attenuated arterial elasticity is one of the earliest markers of atherosclerosis.

OBJECTIVE: Our aim was to investigate the relationship of passive smoking and elastic properties of the aorta.

METHODS: We studied 11-year-old healthy children (n = 386) from the randomized, prospective atherosclerosis prevention trial (STRIP). Aortic elasticity was analyzed by using M-mode ultrasound imaging on the basis of the measurement of blood pressure and arterial diameter changes during diastole and systole. Aortic compliance (AC) and aortic stiffness index (SI) were calculated. Exposure to tobacco smoke was measured by using serum cotinine concentration, which was analyzed with gas chromatography.

RESULTS: Cotinine concentrations ranged from nondetectable (detection limit: 0.16 ng/mL) to 6.8 ng/mL. Cotinine values and aortic elasticity measures were similar between genders. Children were classified into 3 groups according to their cotinine concentration: the top-decile cotinine group (n = 39), the nondetectable cotinine group (n = 220), and the low cotinine group (n = 127). Conventional atherosclerosis risk factors were similar between the 3 cotinine groups. A decreasing trend in AC (P = .041) and an increasing trend in SI (P = .006) was observed across the cotinine groups with an

increasing level of tobacco smoke exposure. In addition, systolic and diastolic blood pressure and BMI were independent predictors of the aortic elasticity indices. In multivariable models, cotinine level ($P = .020$) and systolic blood pressure ($P < .001$) were inversely associated with AC and directly related to SI (cotinine level, $P = .005$; systolic blood pressure, $P = .0003$).
CONCLUSIONS: These data suggest that passive smoking is associated with decreased aortic elasticity in children, indicating early arterial changes.

RHEUMATIC FEVER IN THE NEW MILLENNIUM

Submitted by Alyaa Kotby
Alyaa Kotby
Pediatric Department and Pediatric Cardiology Clinic, Ain Shams University Faculty of Medicine, Cairo, Egypt

INTRODUCTION: During the past 2 decades the presentation of rheumatic fever (RF) has changed markedly from that of an acute florid illness to a more subtle subacute form that is frequently missed.
OBJECTIVE: With this work we attempted to describe the changing face of RF with its different patterns and time of presentation, particularly subclinical carditis.
METHODS: This work included 1732 patients with RF followed up in the pediatric department and pediatric cardiology clinic at Pediatric Hospital, Ain Shams University. Every patient was subjected to a thorough clinical examination, measurement of erythrocyte sedimentation rate and antistreptolysin O titer and C-reactive protein levels, a chest radiograph, electrocardiography, and echocardiography Doppler. Echocardiography was performed at the time of admission and repeated after 2, 4, and 6 weeks and 1 year after the attack. Diagnosis of RF was based on the revised Jones criteria.
RESULTS: Age at the first attack was <5 years for 10% of the patients, 5 to 10 years for 51%, 11 to 15 for 36%, and >15 for 3%; the male/female ratio was 1:1.34. Major clinical RF manifestations were carditis (60%), polyarthritis (56%), chorea (15%), erythema marginatum (0.12%), and subcutaneous nodules (0.12%). Seventy-two percent had carditis, after we combined clinical and echocardiographic criteria of cardiac affection 6 weeks after the attack. Pure arthritis was present in 41% of the patients, arthritis and carditis in 29%, and arthritis and subclinical carditis in 30%. One year after the initial attack the number of patients with echocardiographic features of valve affection remained the same. Pure chorea was present in 55% of the patients, chorea and carditis in 30%, and chorea and subclinical carditis in 25%. One year after the initial attack, 70% of the patients with chorea had echocardiographic features of valve affection. Chronicity of chorea is common with multiple relapses.
CONCLUSIONS: RF is not uncommon in children <5 years of age. Subclinical carditis should be anticipated and looked for at the right time in susceptible patients, particularly those with rheumatic arthritis and chorea. Multicenter studies should be carried out for the addition of the echocardiographic features of carditis to Jones' minor criteria for the diagnosis of RF. Diagnosis of carditis requires a high index of suspicion in at least 1 of 3 cases.

EXPERIMENTAL RESEARCH OF SIMVASTATIN IN REVERSING PULMONARY VASCULAR REMODELING IN VIVO AND IN VITRO

Submitted by Hanmin Liu
Hanmin Liu, Bin Liu, Yimin Hua, Xiaoqin Wang, Li Yu, Tongfu Zhou
West China Second University Hospital, Sichuan University, Chengdu, China

INTRODUCTION: Simvastatin was predicted to be a potential inhibitor to pulmonary vascular remodeling. This novel reversion induced by simvastatin has remained an uncertain mechanism.
OBJECTIVE: Our goal was to explore the role of simvastatin as a potential inhibitor of pulmonary vascular remodeling.
METHODS: We established a neointimal pulmonary hypertensive rat model receiving monocrotaline after pneumonectomy. Simvastatin was administered after the operation. Hemodynamic and vascular remodeling corresponding indices were detected. *GATA-6*, a gene transcription factor, was evaluated in vivo. Proliferation and the cellular cycle were assessed in cultured vascular smooth muscle cells (VSMCs). α-SM-actin, F-actin, and paxillin were detected to evaluate the phenotype changes.
RESULTS: Neointimal changes developed in 88.5% of right lung intraacinar arteries after pneumonectomy and monocrotaline administration. Mean pulmonary artery pressure, the right ventricle/(left ventricle + S) ratio, and media wall thickness significantly increased in rats that had pneumonectomy and were treated with monocrotaline but decreased significantly in simvastatin-treated rats. The expression of *GATA-6* markedly decreased in these rats and was significantly upregulated after receiving simvastatin. In vitro, the proliferation was significantly downregulated in VSMCs with simvastatin compared to that with platelet-derived growth factor. α-SM-actin increased significantly, and F-actin or paxillin was downregulated in simvastatin-treated rats.
CONCLUSIONS: Our data indicate that simvastatin is most likely a pulmonary vascular remodeling inhibitor, which may reverse the proliferation of VSMCs and phenotype changes. Simvastatin can also upregulate *GATA-6* expression in lung tissue.

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