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
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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

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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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