86

IS SYNDROME X RELATED TO ANGIOTENSIN-CONVERTING ENZYME GENE POLYMORPHISM?

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Insertion/deletion polymorphism of the angiotensin-converting enzyme (ACE) gene may be associated with coronary artery disease. Syndrome X (microvascular angina) patients have typical anginal symptoms, positive exercise tests but near normal coronary angiograms. We studied 34 patients with syndrome X, 45 patients with coronary artery disease (abnormal coronary angiograms or documented myocardial infarction), and 102 normal controls. DNA was extracted from peripheral leucocytes and amplified by PCR using specific primers. Insertion (I) or deletion (D) alleles were identified after electrophoresis of PCR products.

	n	DD	ID	II	D	I
Normal controls	102	22%	32%	46%	0.38	0.62
Coronary artery disease	45	22%	47%	31%	0.46	0.54
Syndrome X	34	35%*	50%	15%	0.60^{+}	0.40

*$\chi^2 = 10.6$, $p < 0.005$ vs. normal $^{+}\chi^2 = 10.6$, $p < 0.001$ vs. normal

The frequency of the D allele is increased in syndrome X patients compared to normal controls (odds ratio 2.5, 95% CI: 1.4-4.4). Our results raise the possibility that as the D allele is associated with increased ACE activity, increased angiotensin II levels may predispose a person without significant coronary artery stenosis to myocardial ischaemia.

87

PLASMA ADRENOMEDULLIN LEVELS IN CHRONIC AIRFLOW OBSTRUCTION IN MAN

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Adrenomedullin (AM) is a peptide first isolated from phaeochromocytoma and adrenal medulla. It is also found in the heart, lungs, kidneys and plasma. It is vasodilatory, natriuretic and *in vitro*, dilates bronchial smooth muscles. Since it is synthesised in the endothelium, it may also have a paracrine role. AM induces smooth muscle relaxation by activating adenylyl cyclase and by stimulating the release of nitric oxide. Its role in diseases of the airways is not clear. We used a specific radioimmunoassay (lower limit of detection 2 pg/tube, coefficient of variation 7%) to measure the immunoreactivity of human AM in the plasma of normal subjects and patients with stable asthma or acute exacerbation of chronic obstructive airways disease (COAD):

	n	mean ± standard error
normal	23	8.1 ± 1.0 pmol/l
asthma	20	13.4 ± 1.5 pmol/l
chronic obstructive airways disease	16	13.9 ± 1.5 pmol/l

Plasma AM level in patients with stable asthma and acute exacerbation of COAD were raised compared to controls ($p<0.01$). These results provide evidence that AM may play a role in the pathophysiology of chronic airflow obstruction.