when carefully searched for in all but one patient. ANA and RF were found in 13 of 17 and "scleroderma pattern" was noted in 9 of 13 patients on nailfold capillaroscopy. Taut skin developed later in 57% of pts following extracutaneous presentation by 1 month to 7 yrs (mean 2.3 yrs).

Conclusion Clues to the diagnosis of ssSSc can be classified as: 1) background conditions – unclassified system disorder, Raynaud's, polyarthritis 2) evolving visceropathy of obscure provenience – dysphagia, malabsorption, pulmonary fibrosis, heart failure, nephropathy 3) corroborative laboratory and clinical features – hyper/hypopigmentation, telangiectasis, calcinosis, typical nailfold capillaroscopy 4) confirmatory evidence – visceral biopsy, appearance of taut skin.

FRI0206 PLASMA HOMOCYSTEINE LEVELS IN PATIENTS WITH SYSTEMIC SCLEROSIS

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Background

Objectives Homocysteine is a nonessential amino acid containing sulfur. Homocysteine has a toxic effect on the endothelium suggesting that it may take part in the etiopathogenesis of the diseases with vascular impairment. Since complex etiopathogenesis of systemic sclerosis (SSc) involves endothelial injury and activation, elevated plasma homocysteine levels might be expected in this disease. Standing from this point, we investigated plasma homocysteine levels in patients with limited (ISSc) and diffuse (dSSc) systemic sclerosis and in healthy controls.

Methods Serum samples were collected from 59 (31 limited and 28 diffuse; F/M:56/3 male) patients with systemic sclerosis attending Ege University Rheumatology department, and 27 age and sex matched healthy controls were included in the study. Homocysteine was measured by high-performance liquid chromatography. Hyperhomocysteinemia may occur due to diabetes mellitus, hyperlipidemia and vitamin B12, folic acid deficiencies. Therefore, patients with these conditions excluded from the study.

Results Statistical analysis was performed using one way analysis of variance test. All data are presented as mean $\pm$ SD. A p value <0.05 was accepted as statistically significant.

Plasma homocysteine levels in patients with diffuse SSc (16.31 ± 5.916 mmol/L) and limited SSc (13.27 ± 8.352 mmol/L) were significantly higher than healthy controls (6.34 ± 1.540 mmol/L). When we compared the two subgroups of SSc, plasma homocystein levels in diffuse SSc, was significantly higher than limited SSc ($F = 19.22$ p < 0.001).

Conclusion Numerous clinical studies have been published on the association between homocysteine and vascular diseases. It has been shown to be cytotoxic to endothelium, to induce a vascular-endothelial cell activator, and to promote smooth muscle cell proliferation. Our finding of

Significantly higher plasma homocysteine levels in the both subgroups of SSc implicate the contribution of hyperhomocysteinemia to endothelial damage in this disease.

FRI0207 ASSESSMENT OF TRIO AS THE CANDIDATE GENE FOR FAMILIAL CHONDROCALCINOSIS CCAL2

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Background Chondrocalcinosis is usually caused by the deposition of calcium pyrophosphate crystals on articular cartilage. There is a clear relationship between this crystal deposition and excess extracellular triphosphate and pyrophosphate. TRIO, a novel multidomain transmembrane protein which promotes the exchange of guanine diphosphate for guanine triphosphate in the extracellular space is an excellent positional candidate gene for CCAL2.

Objectives

Methods A positional candidate gene strategy was employed as a valid approach to identify the putative disease locus for CCAL2. The Online Mendelian Inheritance in Man OMIM cytogenetic map was used to identify TRIO as a possible candidate gene within the critical region on chromosome 5p. Mutation analysis required the design of PCR primers to amplify the coding DNA from human genomic DNA in affected individuals. The coding DNA sequence for TRIO was known¹ but information on the intron-exon splicing sequences was unavailable. Sotgun sequence obtained from a P1-derived artificial chromosome (PAC) 514p4 from a PAC contig of the critical region had shown homology with TRIO using the homology search algorithms BLASTN. The intron-exon junctions were identified from this PAC and other adjacent PACs on the contig, using a combination of vectorette PCR and the Genome Priming System (New England Biolabs inc) followed by Big Dye Terminator sequencing on an 377 ABI genetic analyser.

Results From the TRIO coding DNA sequence of 9 kilobases over 55 exons were identified. Both of the functional guanine exchange factor (GEF) domains (18 exons) have been fully sequenced but no mutations have yet been identified in affected members from two large families.

Conclusion TRIO is a novel multidomain transmembrane protein which is localised within the CCAL2 region on chromosome 5p. It is likely to influence the availability of extracellular triphosphate and thus pyrophosphate through the function of the active GEF domains. This gene however has no mutations within the coding regions for both the GEF domains in two large kindreds with familial chondrocalcinosis.

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FRI0208 COEXPRESSION OF TH1 AND TH2-ASSOCIATED CHEMOKINE RECEPTORS IN T CELLS INFILTRATING EARLY FIBROTIC SKIN IN A SLE PATIENT

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Background Fibrotic skin changes in systemic sclerosis (SSc) and allied conditions characteristically are preceded by an