LIPOPROTEIN PROFILE IN JUVENILE RHEUMATOID ARTHRITIS

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«صَدَقَ اللهُ العَظِيم»

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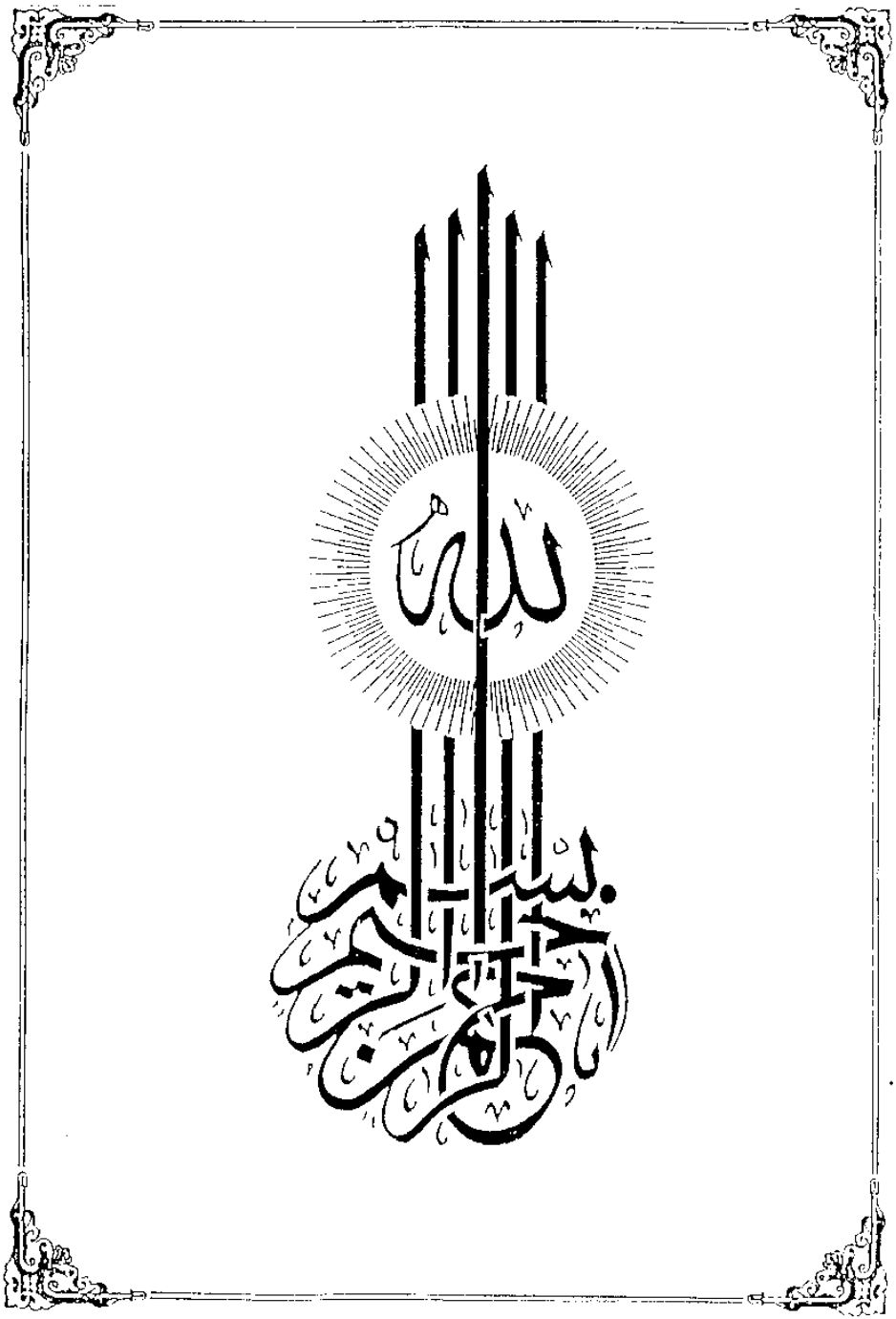

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List of Abbreviations

ANA	Antinuclear antibody
APO	Apolipoproteins
C3	Third component of complement
Chylo	chylomicron.
CNS	Central nervous system
CRP	C- reactive protein
DIC	Disseminated intravascular coagulopathy
ESR	Erythrocyte sedimentation rate
HDL(HDLs)	High density lipoproteins
HDL-C	High density lipoprotein cholesterol
IDL(IDLs)	Intermediate density lipoproteins
Ig	Immunoglobulin
JRA	Juvenile rheumatoid arthritis
LCAT	Lecithin cholesteral acyl transferase
LDL(LDLs)	Low density lipoproteins
LDL-C	Low density lipoprotein cholesterol
LPL	Lipoprotein lipase

NSAID	Non steroidal anti-inflammatory drug
PCAT	Phosphatidyl choline cholesterol acyl transferase
RA	Rheumatoid arthratis
RF	Rheumatoid factor
VLDL (VDLs)	Very low density lipoproteins

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Introduction & Aim of the Work

The metabolic changes in chronic inflammatory connective tissue diseases ought to be recognized not only because of their potentially tissue - damaging effect but also because treatment with anti-inflammatory and disease - modifying drugs may have metabolic side effects (Svenson et al., 1987).

Dyslipoproteinemia is a feature of certain rheumatic diseases including adult rheumatoid arthritis (Lorber et al., 1985). This may explain the increased mortality reported in patients with rheumatoid arthritis - compared to the general population - with cardiovascular disease on the top of the list of causes of mortality (Mutru et al., 1985).

Increased concentrations of total cholesterol, low density lipoprotein cholesterol (LDL - C) and apolipoprotein B (Apo B) have been found to be associated with an increased risk of cardiovascular disease. Moreover, low concentrations of high density lipoprotein - cholesterol (HDL - C) and apolipoprotein A_1 (Apo A_1) have been found to be risk factors for cardiovascular disease (Kottke et al., 1986).

This study is aimed to outline the lipoprotein and apolipoprotein patterns in patients with juvenile rheumatoid arthritis and their possible relation to disease activity and therapy.

Juvenile Rheumatoid Arthritis

Juvenile rheumatoid arthritis (JRA) is a disease or group of diseases characterized by chronic synovitis and associated with a number of extra - articular inflammatory manifestations.

A confusing number of names have been applied, including juvenile arthritis, Still's disease, juvenile chronic polyarthritis, and chronic childhood arthritis (Harris, 1990).

Juvenile rheumatoid arthritis is one of the more frequent chronic illnesses of childhood and an important cause of disability and blindness. It may not represent a single disease but a syndrome of diverse etiologies (Cassidy et al., 1986).

As a matter of fact, it is the most common of the collagen vascular diseases in children (Rennebohm, 1988).

❐ Etiology :

The etiology of JRA is unknown. Among possible causes are infection, autoimmunity, trauma, stress, and immunogenetic predisposition.

An inflammatory arthritis of humans has been observed with infections from both mycoplasma and viruses (rubella and parvovirus) (Schwarz et al., 1987). Certain viral illnesses of

childhood such as rubella may result in a self - limited arthritis; persistent rubella virus infection has been demonstrated in the synovia of children with JRA (Chantler et al., 1985).

Chronic inflammation may be perpetuated by immune complexes formed from auto antibodies such as antinuclear antibody (ANA) or rheumatoid factor (RF) induced by infections.

It is observed frequently that onset of JRA may follow physical trauma to an extremity such as fall or an ankle sprain. It is also well documented that psychological stress appears to be particularly common in families of children with JRA (Henoch et al., 1978).

❒ Genetic background :

There are very few reported instances in which JRA has been observed in more than one family member. Although the cases are few in number, it is striking that within any one family JRA tended to be of the same type of onset (Rosenberg and Petty, 1980). Early studies of Ansell et al., 1962 reported that female relatives of children with JRA showed an increased frequency of seronegative erosive polyarthritis and that male relatives had an increased prevalence of sacroiliac arthritis (Ansell, 1977). One further association bears attention, that is the occasion occurrence of JRA and adult rheumatoid arthritis (RA) in the same family. Rossen et al., 1980 studied four families with multiple cases of RA and JRA

and concluded that susceptibility to arthritis was influenced by a dominant gene with variable penetrance and expressivity.

Pathology:

I) Articular.

Rheumatoid arthritis is characterized by chronic non suppurative inflammation of the synovium. Affected synovial tissues are edematous, hyperemic and infiltrated with lymphocytes and plasma cells. Secretion of increased amounts of joint fluid results in effusion (Schaller, 1980).

Projections of thickened synovial membrane from villi, which protrude into joint spaces. Hyperplastic rheumatoid synovia, may spread over and become adherent to articular cartilage (Pannus formation). With continuing chronic synovitis and proliferation of synovia, articular cartilage and other joint structure may become eroded and progressively destroyed.

Many children with JRA never incur perminent joint damage despite prolonged synovitis(Harris, 1990). However, once joint destruction has commenced, erosions of subchondral bone, narrowing of the " joint space " (loss of articular cartilage), destruction or fusion of bones, and deformity, subluxation, or ankylosis of the joints may result. Tenosynovitis and myositis may be present (Williams and Ansell, 1985).