

LIPOPROTEIN PROFILE IN JUVENILE RHEUMATOID ARTHRITIS

Thesies
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List of Contents

	Page
□ Introduction & Aim of the Work	*
□ Review of Literature	
* Juvenile Rheumatoid Arthritis	
★ Etiology	1
★ Genetic Background	1
★ Pathology	3
★ Classification Criteria	5
★ Clinical Manifestations	5
★ Course & Prognosis	16
★ Laboratory Investigations	17
★ Treatment	23
* Plasma Lipoproteins	
※ Lipoprotein Function	26
※ Classes of Lipoproteins	26
※ Plasma Lipid & Lipoprotein Levels	32
※ Apolipoproteins	35
※ Lipoprotein Disturbances in Children	37
□ Subjects & Methods	40
□ Results	51
□ Discussion	89
□ Recommendations	103
□ Summary	104
□ References	106
□ Arabic Summary	

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 arthrtis
RF	Rheumatoid factor
VLDL (VDLs)	Very low density lipoproteins

List of Tables

I Tables in Review of Literature		Page
Table (1)	Criteria for the classification of JRA	4
Table (2)	Classification of the types of onset of JRA	6
Table (3)	Acute phase proteins	18
Table (4)	Drug therapy for JRA	24 - 25
Table (5)	Plasma cholesterol and triglyceride levels in childhood and adolescence	34
 II Tables of Results		
Table (6)	Inborn errors of lipoprotein metabolism	38
Table (7)	Acquired disorders of lipoprotein metabolism	39
Table (8)	Clinical & laboratory data of the control group	52
Table (9)	Clinical & laboratory data of rheumatoid patients	54 - 55
Table (10)	Serum lipids in rheumatoid patients versus controls	57
Table (11)	Serum lipids in rheumatoid patients in activity versus controls	61
Table (12)	Serum lipids in rheumatoid patients in remission versus Controls	63

Table (13)	Serum lipids & ESR in rheumatoid activity versus remission	65
Table (14)	Serum lipids in rheumatoid patients receiving corticosteroids versus controls.	67
Table (15)	Serum lipids in the group of rheumatoid patients receiving NSAID versus controls	69
Table (16)	Serum lipids in rheumatoid patients under both Corticosteroids & NSAID versus Controls	71
Table (17)	Serum lipids in rheumatoid patients not receiving anti - rheumatic therapy versus Controls	73
Table (18)	Serum lipids & ESR in the group of rheumatoid factor positive versus rheumatoid factor negative patients.	78

List of Figures

	Page
Figure (1) Over view of lipoprotein inter relations	32
Figure (2) Serum HDL in rhumatoid patients versus controls	58
Figure (3) Serum Apo A ₁ in rheumatoid patients versus controls	58
Figure (4) Serum Apo B in rheumatoid patients in activity versus controls	62
Figure (5) Serum HDL in rheumatoid patients in remission versus controls	64
Figure (6) Serum Apo B/Apo A ₁ ratio in rheumatoid patients in remission versus controls	64
Figure (7) Serum HDL in rheumatoid patients under various anti - rheumatic drugs versus control group	74
Figure (8) Serum LDL in rheumatoid patients under varoius anti - rheumatic drugs versus control group	74
Figure (9) Serum triglycerides in rheumatoid patients under various anti - rheumatic drugs versus control group	75

Figure (10)	Serum Apo B in rheumatoid patients under various anti - rheumatic drugs versus control group	75
Figure (11)	Mean ApoB/ApoA ₁ ratio in rheumatoid factor positive versus negative patients	79
Figure (12)	Serum Apo B/Apo A ₁ and age in rheumatoid patients not under therapy	80
Figure (13)	Serum LDL/ HDL and age in rheumatoid patients not under therapy	81
Figure (14)	Serum cholesterol /HDL and Age in rheumatoid patients not under therapy	81
Figure (15)	Serum Apo B/Apo A ₁ and body weight in the whole rheumatoid patients	82
Figure (16)	Serum LDL/HDL ratio and body weight in the rheumatoid children not under therapy	83
Figure (17)	Serum cholesterol /HDL ratio and body weight in the rheumatoid children not under therapy	83
Figure (18)	Serum ApoB/ApoA ₁ ratio and height in the rheumatoid patients not under therapy	84
Figure (19)	Serum LDL/ HDL ratio and height in the rheumatoid patients not under therapy	85

Figure (20)	Serum cholesterol / HDL ratio and height in the rheumatoid patients not under therapy	85
Figure (21)	Serum triglycerides and ESR in rheumatoid patients in activity	86
Figure (22)	Serum LDL/HDL ratio and ESR in rheumatoid patients	87
Figure (23)	Serum cholesterol / HDL ratio and ESR in rheumatoid patients	88

List of Plates

Plate (1)	Plate for apolipoprotein A ₁ estimation	56
Plate (2)	Plate for apolipoprotein B estimation	60

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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 A₁ (Apo A₁) 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 occasional 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:

1) 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 permanent 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).