The Libyan International Medical University

Faculty of Basic Medical Science

Systemic lupus erythematous versus rheumatoid arthritis in

(Joint Pain)

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Abstract:

Systemic lupus erythematous (SLE) and rheumatoid arthritis (RA) are both autoimmune diseases. In fact, the two diseases are sometimes confused because they share many symptoms. The most obvious similarity between RA and SLE is joint pain**.**

Introduction:

Systemic lupus erythematous is an autoimmune disease involving multiple organs, characterized by a vast array of autoantibodies, particularly antinuclear antibodies (ANAs), in which injury is caused mainly by deposition of immune complexes and binding of antibodies to various cells and tissues.

Injury to the skin, joints, kidney, and serosal membranes is prominent, but virtually every organ in the body may be affected.

The prevalence of SLE is variable, dependent on ethnic origin and ranges from 40–200 per 100,000 of population. SLE is more common in those of African and Asian ancestry than in Europeans.

It is predominantly a disease of females, with a female to male ratio of 9:1. Non-European patients with SLE tend to have a younger onset of disease and greater incidence of serious organ involvement, reflective of a more severe clinical phenotype.

Current treatment regimens consist of antimalarial drugs, corticosteroids, conventional disease-modifying anti-rheumatic drugs, cyclophosphamide and biologics.

However, conventional therapies fail to adequately suppress disease activity in a significant proportion of patients and newer targeted therapies are being developed to address this unmet need.[6,7] While there is no single causative agent, the disease arises from a complex interplay of genetic, epigenetic and environmental factors.

Genome wide association studies have identified over 60 risk loci involved in the susceptibility of SLE.[8] Genetic factors alone are insufficient to explain the onset of

SLE and there is likely to be an interaction with environmental factors for the disease to develop in a genetically susceptible individual.

Environmental triggers include ultraviolet light, demethylating drugs and viruses. Sunlight is the most common trigger for a flare of disease, especially cutaneous manifestations.

Epstein-Barr virus infection may be an environmental risk factor for the development of SLE, especially in juveniles. Autoantibodies in the sera can precede the development of clinical features of lupus by many years.

Chronic Epstein-Barr virus infection may cause interferon alpha production, which is a feature of SLE.

Many drugs, especially those that undergo acetylation such as hydralazine and procainamide, can cause drug-induced lupus, which is usually self-limiting and regresses on withdrawal of the drug.

The majority of these patients do not develop SLE. [9,10] Rheumatoid arthritis is a chronic inflammatory disorder of autoimmune origin that principally attacks the joints, producing a nonsuppurative proliferative and inflammatory synovitis.

RA often progresses to the destruction of the articular cartilage and, in some cases ankylosis (adhesion) of the joints. Extraarticular lesions may occur in the skin, heart, blood vessels, and lungs.

The prevalence in the United States is approximately 1%, and it is three times more common in women than in men. The peak incidence is in the third through fifth decades of life. [1,2]

Aim of the study:

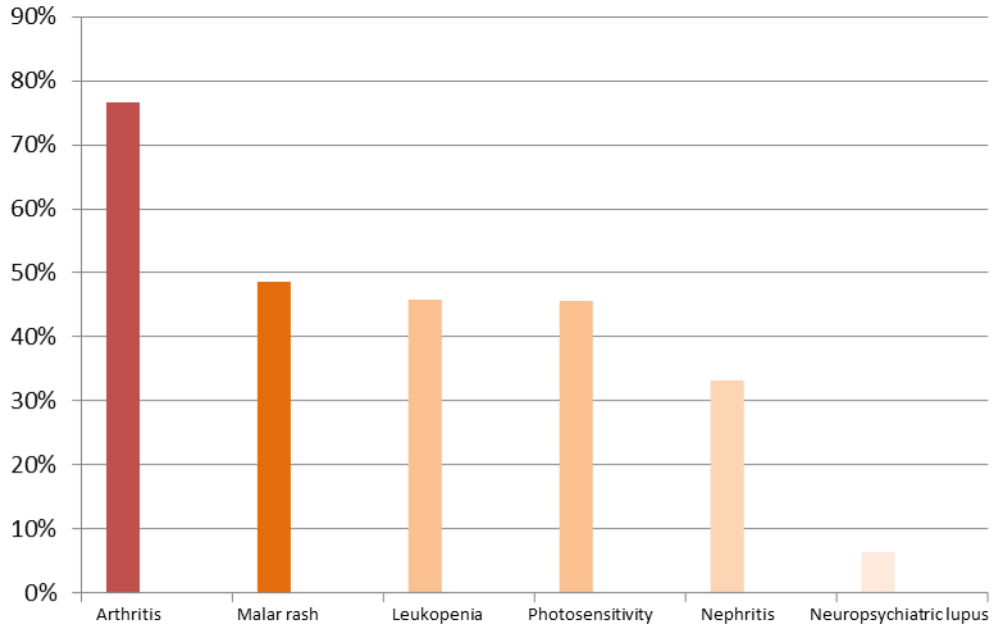
This report aim to differentiate between SLE and RA via the symptoms and laboratory tests.

Materials and Methods:

The data of this report was collected from published studies on internet, and redesigned in more clear way to demonstrate the differences between SLE and RA.

Results:

An Egyptian population sample of 1109 SLE patients, of whom 114 (10.3%) were males and 995 were females (89.7%) shows the following findings :



The following table shows the laboratory findings of SLE and RA[2]

Disease	Specificity of Autoantibody	% Positive	Disease Associations
Systemic lupus erythematosus (SLE)	Double-stranded DNA	40–60	Nephritis; specific for SLE
	U1-RNP	30–40	
	Smith (Sm) antigen (core protein of small RNP particles)	20–30	Specific for SLE
	Ro (SS-A) nucleoprotein	30–50	Congenital heart block; neonatal lupus
	Phospholipid-protein complexes (anti-PL)	30–40	Anti-phospholipid syndrome (in ~10% of SLE patients)
	Multiple nuclear antigens ("generic ANAs")	95–100	Found in other autoimmune diseases, not specific
Rheumatoid arthritis	CCP (cyclic citrullinated peptides); various citrullinated proteins	60–80	Specific for rheumatoid arthritis
	Rheumatoid factor	60–70	Not specific

Discussion:

A study population comprised 603 incident RA subjects (mean age 58 years, 73% female) with a mean follow-up time of 15 years.

By 25 years after RA incidence, ≥4 SLE features were observed in 15.5% of the RA subjects.

[3] As in other autoimmune diseases, genetic predisposition and environmental factors contribute to the development, progression, and chronicity of the both diseases.

In rheumatoid arthritis an evidence suggests that the anti-citrullinated protein antibodies (ACPA) in combination with a T cell response to the citrullinated proteins contribute to disease chronicity.

Approximately 30% of RA patients do not have ACPA in the blood. About 80% of patients have serum IgM or IgA autoantibodies that bind to the Fc portions of their own IgG.

These autoantibodies are called rheumatoid factor and may also deposit in joints as immune complexes, although they are not uniformly present in all patients with RA and can be found in patients without the disease.

It is estimated that 50% of the risk of developing RA is related to inherited genetic susceptibility. The HLA class II locus is associated with ACPA-positive RA.

Evidence suggests that an epitope on a citrullinated protein, vinculin, mimics an epitope on many microbes and is the target of CD4+ T cells when presented by predisposing HLA-DQ alleles.

A second gene linked to RA, PTPN22, encodes a protein tyrosine phosphatase that is postulated to inhibit T cell activation. SLE is a highly variable multisystem disease, and its diagnosis relies on a constellation of clinical, serologic, and morphologic findings. It may be acute or insidious in its onset. Often, the patient is a young woman with some or all of the following features: a butterfly rash on the face; fever; pain without deformity in one or more joints; pleuritic chest pain; and photosensitivity.

In many patients, however, the presentation is subtle and puzzling, taking forms such as fever of unknown origin, abnormal urinary findings, or joint disease masquerading as rheumatoid arthritis or rheumatic fever. Generic ANAs, detected by immunofluorescence assays, are found in virtually 100% of patients, but these are not specific for SLE. Renal involvement may produce a variety of findings, including hematuria, red blood cell casts, proteinuria, and nephrotic syndrome. In others, neuropsychiatric manifestations including

psychosis or convulsions, or coronary artery disease may be prominent. Infections also are common, presumably because of the immune dysfunction that underlies SLE as well as treatment with immunosuppressive drugs. [1,2] A study found that Secondary antiphospholipid syndrome was present in 11.5% of the patients.[4] 54% of the patients with SLE displayed H-ANA, 22% S-ANA, 11% HS-ANA, 9% N-ANA, 1% C-ANA, 2% oANA and 1% were never IF-ANA positive. [5]

Conclusion:

It is very difficult to differentiate between SLE and RA, because they share the same main manifestation (arthritis).The presence of malar rash make the disease easy to be diagnosed. To perform the diagnosis of patient with arthritis we need to check the antibodies that elevate in these diseases specially the specific antibodies (ANAs ,CCP).

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