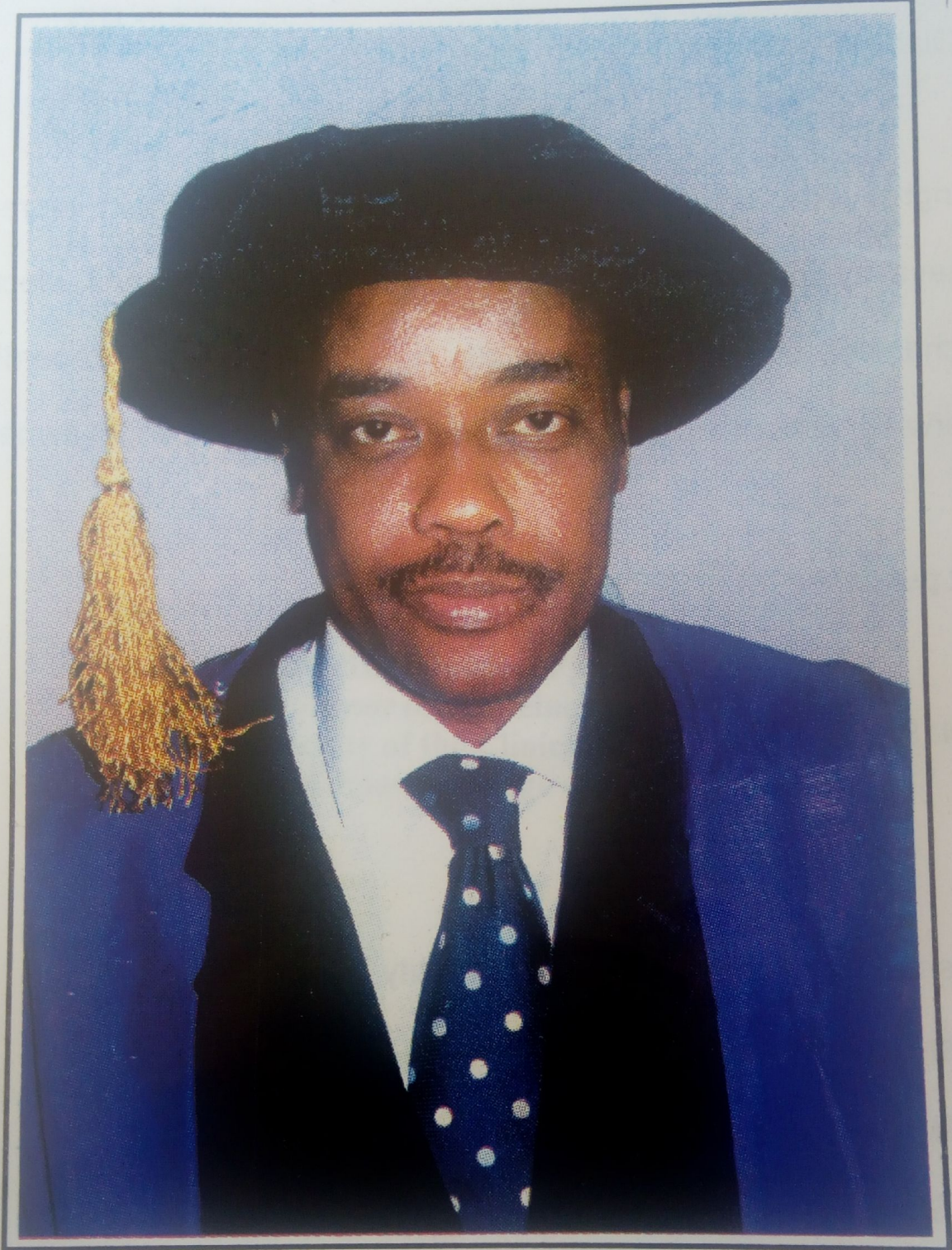


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**OF ARTHRITIS, RHEUMATISM AND JOINT  
FAILURE: THE MYSTERY OF THE BODY AT  
WAR WITH ITSELF**

**BY**

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**35TH INAUGURAL LECTURE  
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AGO-IWOYE**

**3rd May, 2005.**

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# OF ARTHRITIS, RHEUMATISM AND JOINT FAILURE: THE MYSTERY OF THE BODY AT WAR WITH ITSELF

The Vice-Chancellor  
Deputy Vice-Chancellor  
Registrar  
Other Principal Officers of the University  
Provosts and Deans  
Colleagues, and friends from other Universities and Institutions  
Our Royal Fathers  
Gentlemen and Ladies of the Press  
Distinguished Ladies and Gentlemen  
Great Great OOUTES

## PRE-AMBLE

It gives me great pleasure to deliver the 35th Inaugural lecture of the University, the very first from the Department of Medicine.

I am sure most people are aware that the field of Medicine has so many specialties. Each organ of the body has specialists whose lifetime are spent elucidating and managing that particular field. For every specialty, there are physicians and there are surgeons. The physician treats by the use of medications, the surgeon brings out his knife at any slight provocation.

Physicians specialize in different aspects of Internal Medicine such as Cardiology, Gastro-enterology, Neurology, Nephrology, Pulmonology.

I have specialized in the little known specialty of Internal Medicine called Rheumatology.

My surgical colleagues are called Orthopaedic Surgeons. I have spent the past 23 years diagnosing and managing conditions seen

in the specialty. I have also researched on various aspects of arthritis and rheumatism.

The Vice-Chancellor, ladies and gentlemen, HERE IS MY STORY:

Rheumatology is variously defined as:

- A. the medical specialty that deals with the prevention, treatment and rehabilitation of different types of arthritis and musculo-skeletal disorders.
- B. Specialty of medicine that deals with diagnosis and management of non-traumatic diseases of the musculo-skeletal system connective tissues.

The latter definition is to make a distinction between rheumatology and orthopaedic surgery, the latter dealing mostly with the surgical management of the musculo-skeletal system.

The musculo-skeletal system consists of the bones, muscles (which is the meat we eat) the tendons, ligaments otherwise called 'Ishan' in Yoruba. A most important aspect of the musculo-skeletal system is the joints. This is the interphase or interlocking of bones such that movement is effected. We move around, using the joints of the arms, legs and the back.

The musculo-skeletal system effect movement or motion, an essential characteristic of a living thing. Other characteristics of living things include nutrition, excretion, respiration, reproduction. All these other characteristics can only be achieved by motion or movement. The musculo-skeletal system is primus inter pares of these characteristics.

This has even been said in the Holy Bible -

*"For the word of God is quick and powerful and sharper than any two*

*edged sword, piercing even to the dividing asunder of soul and spirit, and of the JOINTS and MARROW and is a discerner of the thoughts and intents of the heart" Hebrew 4:12.*

But then, the musculo-skeletal system has had mention in the Bible even before then. A look at the old testament of the Bible (King James version) Genesis Chapter 2 verse 23 "And Adam said This is now bone of my bones and flesh of my flesh; she shall be called woman because she was taken out of man" This is the first mention of any structure of the human body in the Bible!!

Then again, Colossians 2:19 *And not beholding the head from which all the body by JOINTS and BANDS having nourishment ministered and knit together to increaseth with the increase of God" Translate bands to ligaments and tendons!!*

This is also effectively reflected again in Ephesians 4:16. Another Biblical reference is Hebrews 4:12.

Quite a lot of important people in the past have been known to suffer from different types of arthritis. These include Christopher Columbus, Constantine ix Monomachus, Mary Queen of Scots, Erasmus, Benjamin Franklin, Renoir Sir Arthur Conan Doyle in the story "Missing Three Quarters" describes the opulent Lord Mount-James, one of the richest men in England as being able to 'chalk his billiard cue with his knuckles'. He was most probably referring to gouty arthritis with tophi

**MISCONCEPTIONS AND MISGIVINGS** - There are a lot of misconceptions, misgivings and misapplication of the disorder arthritis or rheumatism. Such as:

1. Arthritis is arthritis - Not so. There are actually more than 200 types or causes of arthritis. Making a diagnosis of arthritis is like saying a person has headache. It is a



manifestation of a disease and not the disease itself. We know headaches could be caused by disorders as mild as malaria or as serious as cancer, meningitis, stroke.

2. Arthritis is a disease of the old. **WRONG.**

Arthritis affects persons as young as 3 or as old as 100. Because there are different types, it is recognized that a particular type seen in middle aged to elderly persons called **OSTEOARTHRITIS**. But we can have Juvenile Arthritis affecting children aged less than 16 years! Other types of arthritis pick on different age groups.

3. Arthritis cannot be cured. So it must be endured. **WRONG.**

While it is true that certain types of arthritis cannot be cured by a once and for all 'magic bullet', it is also true that the pain can be effectively managed in most cases.

4. Arthritis occurs when you drink cold water or expose yourself to cold. **WRONG.** The pain of arthritis is not originated by cold but it can be worsened by it.

### **WHAT IS ARTHRITIS? WHAT IS RHEUMATISM?**

Though these terms are used interchangeably and though the two conditions cause joint pains, they are however different in their presentation and cause.

When we refer to **ARTHRITIS**, we refer to the disorder of the primary structures that determine joint functions such as bones, cartilage and synovial membranes (the structure that produces lubricating fluid-synavial fluid).

When supporting structures of the joint such as tendons, ligaments and capsule (Ishan) are affected, these are called **RHEUMATISM** (Soft Tissue rheumatism: Non-articular rheumatism)

While most types of arthritis are generalised, most types of rheumatism are localized in a particular joint. But whether arthritis or rheumatism, the complaints are usually the same. They include joint pains, joint swelling, joint stiffness, warm to touch and occasional deformity.

The disturbance in the joint causing all these complaints is INFLAMMATION. This is the body reaction to assault on the joint by any provoking factor. Inflammation is actually a process meant to protect the joint but sometimes this goes awry such that the body continues to react even when the noxious stimuli are gone!!

Arthritis and rheumatism constitute the generic name rheumatic or rheumatological diseases. There are more than 200 types or causes of these rheumatic diseases.

But a few pertinent questions need to be asked about rheumatic diseases.

### HOW MANY PEOPLE HAVE THE DISEASE?

Rheumatic diseases have been reported in all countries, cultures and cities. It is well recognized that community based studies provide the most accurate record of the incidence and prevalence of the disease. Such studies can either be determined at a particular time as a reflection of new cases measured. The term prevalence relates to existing disease.

Most studies of incidence of rheumatic diseases especially in rural communities have given an incidence of 30-38%. We have reported an incidence of 33% in a rural farming population of Aiyetoro-Gbedde, a village in the then Kwara state but now in Kogi state. The total population there is about 8,500 (Adelowo, 1984).

Many of those studied had reported absence from work for at least two weeks.

## WHO IS SUSCEPTIBLE TO THE DISEASE?

There seems to be sex, age and ethnic differences in frequencies of certain rheumatic diseases. Connective diseases (Auto Immune Diseases) for instance are commoner among Afro-Americans. Osteoarthritis is commoner among females. Gout and Spondyloarthropathy are commoner in males. Osteoarthritis is commoner among middle aged and elderly persons while Juvenile Arthritis is exclusive to those under 16 years.

## WHERE DOES THE DISEASE OCCUR?

Differences in disease rates between countries in regions may be due to genetic (including racial) and/or environmental factors. Possible environmental factors include diet, socio-economic factors, sunlight, bacteria or viruses peculiar to an area. For example Rheumatoid Arthritis occurs with similar frequency in most Caucasian populations. It however has a higher prevalence among certain groups of American Indians and among Scandinavian countries.

Migrations studies have indicated in certain cases as to whether the differences are environmental or genetic. If a migrating people have the same frequency of disease in their native and newly adopted countries, this suggests that genetic factors are more common.

A word of caution here. While the criteria needed to diagnose a disease may be peculiar to that population, another set of criteria may sometimes be needed in a different setting. For instance, rheumatoid factor while being present in Caucasians with rheumatoid arthritis, is usually absent among Africans. When erythrocyte sedimentation rate (ESR) is used instead as is done in South America, a bigger haul of patients with rheumatoid arthritis is seen.

### **WHEN DOES THE DISEASE OCCUR?**

Clusters of cases in a particular season of the year or sporadic time-space clusters suggest that all or most of the cases in the cluster may have been exposed to a causative agent from a common source. Lyme disease, a form of arthritis caused by a type of bacteria is a good example. This form of arthritis is seen in certain persons clustering at a place and within a certain period. For instance, between 1970 and 1985, there was a marked fall in the incidence of Rheumatoid arthritis in women in the U.K. This coincided with the increasing use of contraceptive pills and there is evidence to suggest the two are linked.

### **WHY DOES THE DISEASE OCCUR?**

Here I refer to the parable of the sower in the Bible when the seed falls on thorns there is no development. Whereas when it falls on fertile soil, it germinates and grows.

The evolution of disease is similar. There is a lot of interplay between the seed (environmental factor) and the soil (genetic factor). This has been proven in such conditions as rheumatoid arthritis and lupus where there are genetic factors such as presence of certain Histocompatibility complex and Antigen, (HLA) and possibility of infection by bacteria, viruses or even influence of dietary factors.

### **WHAT HAPPENS TO PEOPLE WITH THE DISEASE?**

Deformities, death, disillusionment, are the outcome in many cases. It has been suggested that the reason certain rheumatic conditions are not seen in Africans is because they die before such manifestations appear!

### **THE ESCALATING BURDEN OF ARTHRITIS**

Arthritis has been called the 'primary crippling disease' of the world. In 1990, 37 million Americans one in every seven were reported to

suffer from one form of arthritis of the other. One in every three families has a sufferer an estimated 27 million.

Europeans have arthritis too. In many studies, arthritis is the most frequently occurring condition among the elderly, far above hypertension, cataracts, heart diseases and diabetes. About 51% of persons aged over 65 years have arthritis.

Arthritis limits major functions such as working, self-care and routine household activities in about 3% of Americans.

The economic burden is no less. The cost of musculo-skeletal conditions is as high as 2.5% of the U.S. gross national product. Musculo-skeletal conditions account for 315 million physician visits annually and 8.26 million hospital admissions. About half the absolute cost of musculo-skeletal conditions is due to direct medical care and the rest is due to indirect costs. The average total annual cost of rheumatoid arthritis and lupus for an individual patient is about 80% of U.S per capita income. Unfortunately there are no such data available in Nigeria.

### **WHAT DATA ARE AVAILABLE IN NIGERIA?**

The earliest report on the various types of arthritis and rheumatism among Nigerians were by Greenwood, working in University College Hospital, Ibadan (1969, 1970). Fifteen years later, Adelowo in (FMCP dissertation, 1982; Nigerian medical practitioners (1985) reported 138 patients seen. The diagnostic groups are -

**TABLE 1: FREQUENCY OF TYPES OF ARTHRITIS IN UCH**

|   |      |
|---|------|
| Osteoarthritis (Degenerative Joint Disease) | 82.7 |
| Rheumatoid Arthritis                        | 9.5% |
| Tuberculous Arthritis                       | 2.2% |
| Gout  | 1.4% |
| Reiter's Disease                            | 1.4% |
| Scleroderma                                 | 1.4% |
| Psoriatic Arthritis                         | 0.7% |
| Rheumatic fever                             | 0.7% |

In a recent review of the first 1000 patients seen in a rheumatology clinic, forty three (43) different types of arthritis and rheumatism were documented (Adelowo, Oguntona). This is made up of the following, amongst others:

**TABLE 2: FREQUENCY OF TYPES OF ARTHRITIS IN A RHEUMATOLOGY CLINIC**

|  |       |
|--|-------|
| Osteoarthritis   | 28.7% |
| Soft Tissue Rheumatism                                 | 11.5% |
| Back pain syndrome (including Spondylosis)             | 13.6% |
| Rheumatoid Arthritis                                   | 10.1% |
| Cervical spondylosis                                   | 7.9%  |
| Gout   | 6.5%  |
| Systemic Lupus Erythematosus                           | 5.9%  |
| Neuropathic pain (pain of nerve Origin from arthritis) | 2.6%  |
| Shoulder pain syndrome                                 | 3.9%  |
| Fibromyalgia   | 2.6%  |
| Juvenile chronic Arthritis                             | 1.4%  |

Other types such as polymyalgia rheumatical, avascular necrosis, plantar fasciitis, Carpal Tunnel syndrome and others constitute the remainder.

We do recognize that as the level of practice goes up from the community at large or primary health care, to secondary health care to tertiary health care, uncommon conditions begin to appear. The auto-immune diseases of rheumatoid arthritis, Systemic Lupus, Erythmatosus, Scleroderma, Juvenile Chronic arthritis, are more often diagnosed at the tertiary level. These cases were infrequently reported among Africans giving room to assumption that these cases are not seen. The same statement was said of Hypertension until Akinkugbe debunked this. The same was said of Psychiatric illness until Lambo debunked it.

We know that these conditions are not uncommon. What you do not search for, you do not see! Our message to doctors is that many of these cases are not being diagnosed because most of our doctors were never taught about these conditions during their medical training. Luckily enough our medical college is the exception!

## **PAIN AND ASSESSMENT**

Pain is the major complaint that brings a patient to the doctor. It has driven people into the hands of doctors more often than any other symptoms in human history. Some of the adverse decisions taken that alter human history were taken when kings and leaders were in pain!

It is the complaint that a rheumatologist is confronted with in almost 100% of cases of arthritis. Yet, pain is extra-ordinarily difficult to describe, understand or share, often leaving sufferers frustrated,

isolated and distraught. As such, it is very important for arthritis sufferers to be able to establish a meaningful dialogue with their doctors to convey exactly how they feel.

We express pain variously and communicate it, silently in shivers, gazes, winces and in the stretching, twisting, writhing movements of the body; verbally as shrieks, screams or whimpers, sighs, moans, groans. People who do not perceive pain lack the protective responses to noxious stimuli, such as biological withdrawal reflex when the hand is hit by a door. Such persons suffer multiple injuries and shortened life span. Pain is thus a gift nobody wants. But it is needed to protect us from further suffering, damage and even further pain. In times past, pain signified punishment, torture and damnation. You have sinned so you deserve punishment!

Pain is subjective and is always expressed as a first person. We should not doubt or question a person's expression of pain, otherwise, we question his very identity.

A doctor is programmed to inquire further when a person presents with pain. Hence questions like 'where is the pain?', 'what does it feel like?', 'how do you describe the pain?', 'is the pain constant?', 'when was the worst pain ever?', 'what makes the pain better?', 'what aggravates the pain?'. Such searching questions only help to elucidate the cause and chart out the management. But some pains are not amenable to such questions or the answers offered appear complex and indecipherable.

Such pain which may persist for many months (and even years) despite a variety of treatments can cause enormous distress and disturbance of sleep, emotion, thought and social life. Such pain is termed chronic pain. Arthritis pain is one of such.

Doctors are sometimes 'skeptics' when the description of chronic

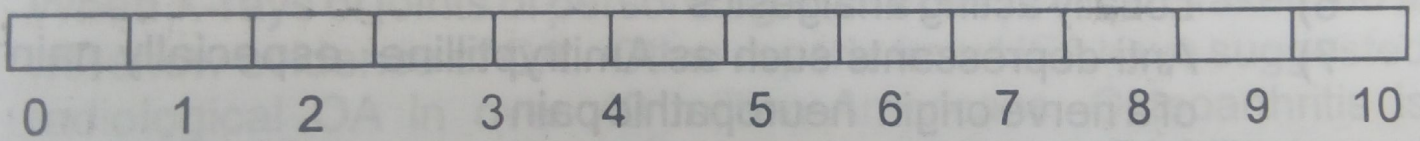


Other assessments were functional status (scale 0-3). Investigation and patients judgements (Scale 0-5).

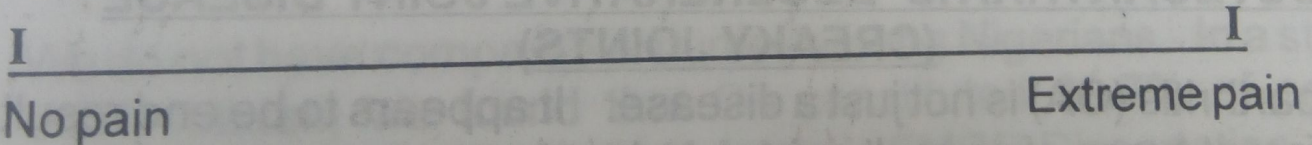
The eventual aims of these is to use figures that can be subjected to statistics.

A similar method was adopted in another paper comparing these two drugs in patients with soft tissue rheumatism (Adelowo et al. Nigerian Quarterly Journal of Medicine, 1998)

Other methods of assessment such as visual analogue scale, Graphic rating scale have also been used in other trials (Adelowo O. O. and Omotosho A. B. The Nigerian Journal of medical Research, 1997; Adelowo O. O., Nigerian Medical Journal, 1998): Adelowo O. O. current Therapeutic Research, 1985). Other methods include the WOMAC (Western Ontario Macmasters Universities) which is a composite of pain and functional assessment based on series of questions. Others include Likert Scale none, mild, moderate, severe, extreme: Numerical rating scale



Visual analogue scale



There is a trend towards assessment of functions and quality of life. This is borne out in such assessments as Health Assessment Questionnaire (HAQ); Arthritis Impact Measurement Scales (AIMS) Systemic Lupus Activity Measure (SLAM); Systemic Lupus Erythematosus Activity Index (SLEAI), Rapid Assessment of

Disease Activity in Rheumatology (RADAR) and others.

In addition to all these, it is very important that patients express their assessment of the treatment (patient's global assessment) as well as physician's global assessment.

### WHAT ARE THE ORIGINS OF PAIN IN ARTHRITIS?

Virtually any structure around the joint could cause pain. These could come from the muscle, raised pressure in the bone as a result of inflammation, synovial membrane inflammation, stretching of capsule of the joint, stretch of ligaments, tendon insertion, elevation of periosteum.

### WHAT MEDICATIONS HAVE BEEN USED?

- 1) Non-steroidal anti-inflammatory drugs
- 2) Simple analgesics Paracetamol
- 3) Steroids
- 4) Colchicine
- 5) Narcotic analgesics
- 6) Locally acting analgesics
- 7) Anti-depressants such as Amitryptilline especially pain of a nerve origin neuropathic pain
- 8) Anti-convulsives neuropathic pain

### OSTEOARTHRITIS DEGENERATIVE JOINT DISEASE (CREAKY JOINTS)

Osteoarthritis (OA) is not just a disease. It appears to be end result of different types of assault, injury or damage to the joint. All forms of arthritis, if it persists long enough will end up as osteoarthritis. It has also been called 'Joint Failure' since essentially the changes, taking place are such that the joints are unable to perform their function. It is also being labeled 'creaky joint' to indicate the sound emanating from such joints during motion. Although it has been called a disease of 'wear' and 'tear' and as such suggesting a

pessimistic and hopeless case because the changes in the joint proceed inexorably downhill to joint destruction. New information however indicates that the change may be reversible.

Osteoarthritis is one form of arthritis that has been found in vertebrates, amphibian, fishes and birds and in all mammalian species.

Because OA is most common in aging patients, it is often proposed that the disease is an intrinsic part of the aging process. This 'wear' and 'tear' theory also stimulated by this observation assumes a decreasing capacity with age of the articular cartilage to resist mechanical stress. New information reveals that most of the symptoms are not just consequent on the 'wear' and 'tear' but that the body in its attempt at repair 'over-responds' leading to osteophyte formation which further hinders movement causing pain.

### EPIDEMIOLOGY

When X-rays of joints of persons aged over 55 years are taken, 80% will show evidence of OA. Other reports from USA have suggested radiological OA in over 40 million Americans. Osteoarthritis is second to only heart diseases as a cause of work disability in men over 50 years of age.

We do not have comparable studies among Nigerians. In a study in UCH, Ibadan, we found that OA constituted 82.7% of patients coming to an Arthritis clinic (Adelowo, FMCP Dissertation, 1982; Adelowo, Nigerian Medical practitioner, 1985).

There is a wide variation in the joints involved. Involvement of the hands is particularly common among Caucasians and American Indian population. Studies from Nigeria (Adelowo, WAMJ, 1986) have shown that hand involvement is minimal as shown below.

Knee involvement is the commonest.

**TABLE 3: JOINTS AFFECTED IN PATIENTS WITH OSTROARTHRITIS**

| JOINTS      | FEMALE (%) | MALE (%)  | TOTAL (%)  |
|-------------|------------|-----------|------------|
| Knee        | 179 (66.3) | 36 (44.4) | 215 (61.2) |
| Lumbosacral | 51 (18.9)  | 16 (19.8) | 67 (19.)   |
| HIP         | 11 (4.1)   | 7 (8.6)   | 18 (5.1)   |
| Ankle       | 7 (2.6)    | 9 (11.1)  | 16 (4.6)   |
| Hands       | 7 (2.6)    | 5 (6.2)   | 12 (3.4)   |
| Cervical    | 6 (2.2)    | 3 (3.7)   | 9 (2.6)    |
| Shoulders   | 6 (2.2)    | 5 (6.2)   | 11 (3.1)   |
| Wrist       | 3 (1.1)    | 0 (%)     | 3 (0.9)    |

Osteoarthritis affects middle-aged to elderly usually 45 years and above, though we do see cases as from 40 years. Our experience in UCH, Ibadan (Adelowo, WAMJ) gave an age range of 35 - 78 years (mean  $53.2 \pm 9.0$ )

The condition is commoner in females than males (from our series the ratio of female to male is almost 3:1).

### **RISK FACTORS FOR OA**

The universally recognized risk factor include obesity, heredity, joint hypermobility, smoking, other joint diseases, trauma to the joint, occupational, sports and leisure physical activity, diabetes.

We have reported the high prevalence of OA among market women, especially those who sit on low stools and display their wares on high table (Adelowo, FMCP dissertation, 1982).

The extreme bending of the knees result in damage to the supporting structures of the joints as ligaments, tendons.

We have also observed the high association of OA with Benign Hypermobility Syndrome (Adelowo O. O., Nigerian Medical Practitioner, 1998). An increasing association is that of Osteoarthritis and high heel shoes, especially the stiletto shoes. These shoes cause re-distribution of forces across the joints and instability leading to OA. Also, as many Nigerians get involved in professional football, it is not unexpected that OA of the ankles and knees will be reported more frequently. Anyway, the good news is that they would have enough money for their treatment!

## **DIET AND ARTHRITIS**

### **CRYSTAL (BAD SALT) ARTHRITIS**

Probably the most painful form of arthritis seen is Gouty arthritis. This is arthritis resulting from the deposition of a 'bad' type of salt uric acid in the joint. It is called the 'disease of kings' and 'king of diseases'. But it is more of "disease of plenty".

The typical story is that of a man who goes out socializing at a pepper soup joint. He consumes a lot of beer, meat and returns home. He is woken up at night by an intense pain usually on the foot and knee ( in Nigerians) and practically notices the joint swelling up right in his 'eye'. He may have had previous episodes that got better on their own or with pain killers. By the time he gets to the hospital, he is very sick, feels feverish and is in excruciating pain. He would ask the doctor to just 'cut off the offending limb'!

Gout arthritis can either present as acute, or chronic form. It is due to deposition of crystals of uric acid in the joints. Uric acid is the end product of break down of protein of the nucleus nucleoprotein. It is most commonly seen in men above 30 and occasionally, in women after menopause. Gout can either be primary in which there is no identifiable cause or it may be secondary to other causes such as alcohol, obesity, excessive meat consumption, kidney diseases, leukemia and drugs.

While most textbooks describe occurrence of gout mostly in the big toe, our findings indicate that the bigger joints such as the foot, ankle and knees are more commonly affected in Nigerians (Adelowo O. O., NMP, 1985). Recent studies have shown a frequency of 6.5% among patients seen with arthritis in a clinic.

Gouty arthritis can also co-exist with other types of arthritis especially Osteoarthritis. There is a rare association with rheumatoid arthritis. We have previously reported this uncommon association (Adelowo O. O., Scand J. Rheumatology 1986). Our case was one of the 12 cases reported worldwide up to then.

### **BACK PAIN**

Back pain is one of the commonest presentation in an arthritis clinic. Back pain by definition includes pain in any part of the back from the neck to the sacrum/coyx. Low back pain is felt in the lower back and this is probably the commonest area where back pain is felt.

Most people will have back pain at one time or the other in their life time. However, over 50% of such persons will improve after 1 (one) week while more than 90% are better at 8 weeks. The remaining 7-10% continue to experience symptoms for longer than 6 months. The economic burden is quite high. In the U.S., 10% of cases with chronic low back pain account for 57% of the total expenditures on work related sickness payments. Many risk factors described for

back pain involve occupational or psychologic characteristics. Occupational factors include jobs that require lifting heavy objects or sitting crouched up at desks. Workers involved in heavy duty labour who are aged over 45 years have a 2.5 times greater risk of absence from work secondary to back pain compared to workers aged 24 years or younger.

There are many causes of back pain ranging from mild causes such as strain and sprain or potentially fatal causes as cancer either in the bones themselves or as spread from other structures such as the pancreas, thyroid, intestine, lungs.

There are universal causes of back pain. Men have back pain when they make babies. Women have back pain when they give birth to made babies!!

Most back pains are of mechanical origin in 90% of cases. Mechanical back pain is pain consequent to overuse of normal anatomic structure or pain secondary to trauma or deformity of an anatomic structure. Other causes have been implicated in back pain. They include infections, sickle cell disease, osteoarthritis, osteoporosis, spinal stenosis, gout, cancer of the bone as well as cancer of the pancreas, colon, prostate, ovary, uterus. Mechanical back pains are self limiting. They will resolve on their own or with pain killers and physical therapy. As such, they do not need further investigations, and x-rays taken in such cases do not show any abnormalities.

The dilemma facing a doctor is how to distinguish mechanical, innocuous cases from severe causes as cancer. The so called 'red-flag' signs that will prompt a doctor to investigate back pains further are:

- 1) Association with fever, loss of weight, loss of appetite and other constitutional disorders.

- 2) Back pain starting in a 50 year old for the first time.
- 3) Back pain associated with increasing weakness of the limbs.
- 4) Unrelenting back pain, worse at night.
- 5) Back pain not responding to pain killers.
- 6) Back pain associated with other systemic features.
- 7) Back pain associated with urinary or faecal incontinence.
- 8) Back pain associated with deformities at the back.

However, cases with 'red flag' signs need further investigations including X-rays, and many times other imaging investigations as CT and MRI Scan. These will usually highlight both bony and non-bony structures of the back.

Another aspect of back pain is the resultant pressure on nerves in some cases with intervertebral disc problem. This usually manifests as sensory features such as pins, needles, numbness, pain or perception of electric shock on the hands, feet, leg and arms. This condition is called a NEUROPATHIC pain and is due to the effect of the arthritis disc on the nerves that cross it.

Neuropathic pain is felt well away from the side of the pressure at the back and the sufferer may even have forgotten about the neck or back pain.

Neuropathic pain is one of the most challenging types of pain to treat. There are however new medications that are being used.

In a recent review of 1000 patients seen in a rheumatology clinic, all causes of low back pain accounted for 103 of the cases seen i.e. 10.3%. Neck pain accounted for 79 cases i.e. 7.9% out of these, 26 patients (2.6%) had neuropathic pain (Adelowo, Oguntona)



## NON-ARTICULAR RHEUMATISM (SOFT TISSUE RHEUMATISM)

Many patients presenting at a rheumatology clinic will have complaints relating to the soft tissue i.e. those non-bone, supporting structures of the joints. These include capsule of the joint, ligaments, tendon, muscles and bursae. The pain from these structures are similar to those of arthritis except that they are mostly localized and are present on moving the joint. The pain occurrence has a unique occurrence mostly at night. In a published report in African Journal of Medicine & Medical Sciences, we have reported the following categories (Adelowo, Odusan, 1997)

**TABLE 4: DIAGNOSTIC TYPES OF SOFT TISSUE RHEUMATISM**

|                           |       |
|---------------------------|-------|
| Capsulitis                | 32.4% |
| Rotator cuff syndrome     | 14.7% |
| Achilles tendinitis       | 11.8% |
| Fibromyalgia              | 8.8%  |
| Bicipitalis Tendinitis    | 8.8%  |
| do Quevuaim tenosynovitis | 8.8%  |
| Plantar Fasciitis         | 8.8%  |
| Carpal Tunnel Syndrome    | 8.8%  |

These 68 cases represent 58.8% of the 165 new cases seen in a rheumatology clinic at OSUTH over a period of 2 years. Our recent experience in another rheumatology clinic showed 11.5% of the

1000 cases seen over a 5 year period. The frequency of the various diagnostic groups amongst others, is as below:

**TABLE 5: DIAGONOSTIC TYPES OF SOFT TISSUE RHEUMATISM IN A RHEUMATOLOGY CLINIC**

|                        |        |
|------------------------|--------|
| Hypermobility syndrome | 26.9   |
| Fibromyalgia           | 23.48% |
| Polymyalgia Rheumatica | 9.6%   |
| Carpal Tunnel syndrome | 10.4%  |
| Planter Fasciitis      | 9.6%   |
| Trigger Finger         | 3.4%   |

Others are Bursitis, Epicondylitis, etc. A peculiar characteristics of most of these pain syndromes is that they occur mostly at night and may be associated with sleep disorders.

One of the most baffling and topical disorders in rheumatology is the FIBROMYALGIA syndrome. This condition probably represents the frontier between the mind and the body. Its characteristics include:

- a) Pain all over the body with no specified locations.
- b) Pain occurring on both halves of the body.
- c) Pain occurring below and above the waist.
- d) Pain occurring at the back.

e) Tender areas on the body when examined by a doctor.

Such pains are also usually associated with stiffness of the body, headache, pins and needles, irritable bowels, sleep disorders, depression.

We initially thought 'it was all in the mind', but recent studies on Magnetic Resonance Imaging (MRI) has shown that these persons have abnormal blood flow to their thalamus. Theories propagated for this condition include abnormal interplay of pain mediators in the central nervous system; a heightened reaction to sensory stimulus which in normal persons should not cause pain. That is they have a low threshold to pain. Another theory is the 'gate' theory in which repeated impulse in the pain pathway heads to a weakening of the pathway such that all impulses whether pain or touch is interpreted as pain. A narrow pathway becomes expressway for pain!

Their management includes combination of pain killers, muscle relaxants, anti-depressives. They also benefit from psychologist evaluation. This is one condition that treatment is generally unsatisfactory and many persons carry the scar for years.

### **JUVENILE CHRONIC ARTHRITIS**

Arthritis in children! Not many people, doctors inclusive, realize that children can present with arthritis. We are so used to associating arthritis with old persons.

By definition, Juvenile Arthritis occur in children aged 16 years and

below. It is variously called Juvenile Chronic Arthritis (JCA) Juvenile Rheumatoid Arthritis (by the Americans). Of recent, a new terminology, Juvenile Idiopathic Arthritis has been suggested by the International League of Association of Rheumatology (ILAR) Task Force. This is yet to catch on.

### **EPIDEMIOLOGY**

An incidence of 5 - 18 and a prevalence of 30-150 per 100,000 children under the age of 16 are found in Europe and on the American continent. In a field study of more than 46,000 children in Turkey, researchers found chronic arthritis in 64/100,000. Of note, only 12 of the 30 defined children had previously been diagnosed by a physician.

It is not surprising that many of these cases will be missed in the Nigerian population since rheumatology is hardly ever taught in any of Nigerian medical schools. Our university has the distinction that rheumatology is a strong aspect of curriculum of our medical students.

### **PATHOGENESIS "ORIGIN OF THE DISEASE"**

Genetic cause seems to play an important role. HLA DRB1, DQB1 have been identified in cases with eye involvement Uveitis

There may also be an imbalance in cytokines; these are proteins that perpetuate inflammation and progression in patients with arthritis. It has been speculated that a lower production of one of these cytokines, IL-10, could lead to more severe form of the disease.

Another theory is that of immunologic abnormalities leading to overproduction of auto-antibodies such as Anti-Nuclear Antibody

(ANA), Anti Neutrophil Cytoplasmic Antibodies (ANCA), Anti Cardiolipin Antibodies (ACA).

Endocrine abnormalities such as Prolactin elevation have also been implicated.

### **CLINICAL FEATURES**

There are three main features of this disease

1) Presence of arthritis (2) Growth retardation (3) Eye involvement Uveitis

There are three major types of Juvenile Chronic Arthritis

1) Systemic - In these cases, seen in children as young as 3 years, there is presence of fever, anaemia, rashes, enlargement of spleen and liver, increased white blood cell count.

2) Pauci-articular - This is the commonest and most dreaded because of the increased association of eye involvement. In this type, less than three joints are involved more usually joints of the knees and hip.

There are two sub-types

a) Anti-nuclear antibody positive - commoner in female and usually associated with eye involvement such as cataracts, glaucoma and blindness.

b) Anti-nuclear antibody negative - seen in older children, especially males. This type is usually associated with back pain.

3) Polyarticular - Three or more different types of joints are affected. There are two sub-types:

a) Rheumatoid factor negative - this runs a milder course and does not usually lead to early joint destruction.

b) Rheumatoid factor positive - this runs a downhill course and is seen in older children. It presents just like rheumatoid arthritis in adults.

In a recent study in patients seen in our clinic, fourteen (1.4%) cases were documented (Adelowo, Oguntona). Age range is 7 years to 16 years. Male/Females were 1:1. Almost all the cases had delay in presentation ranging from 2 years to 8 years with a mean of 5 years. The distribution of the types are :

|                            |     |
|----------------------------|-----|
| (Systemic Still's disease) | - 1 |
| Pauci articular            | - 8 |
| Poly articular             | - 5 |

Rheumatoid factors was positive in only 1 case.

Antinuclear antibodies were positive in only 2 cases. It would seem that our own patients do not have the abnormal antibodies seen among Caucasians.

## **TREATMENT**

There has been a paradigm shift over the past few years. We are introducing Disease Modifying Anti-rheumatic drugs (DMARD) early in the course of illness. We are also giving less corticosteroids than we used to. The DMARDs have gone a long way in limiting the inexorable deformities, disability, and stunted growth that were invariably seen years ago. Methotrexale is the gold standard. But other DMARD such as Chlorambucil, Sulfasalazine and sometimes Cyclophosphamide injections have been used.

New agents that target specific molecule in the inflammatory cascade-anti-tumour necrosis factors are being used early in the course of illness.

Non steroidal Anti-inflammatory drugs such as Ibuprofen, Naproxene are also used to control pain but these do not control the disease.

# AUTO-IMMUNE DISEASES - THE BODY AT WAR WITH ITSELF

## THE IMMUNE SYSTEM

The concept and manifestations of diseases with the generic name 'auto-immune' diseases constitute a mystery in medicine. Auto-immune means the body is developing immunity or reaction against itself. The immune system which consists of white blood cells of the types "T" and "B" lymphocytes are programmed to react against foreign and strange particles such as bacteria, viruses and other microbes. These white blood cells constitute the defence (military) system of the body. Just like in any armed forces, different categories of these white cells are arranged in "companies" "battalions", "divisions". Each of these battalions have specific types of ammunition just like armoured tanks, artillery, rifles, machine guns. These they deploy in the epic battle against these invading germs. Many times, these soldiers (white blood cells) overcome the invaders and we are free of infection. However, at other times, the white cells are overcome and we breakdown in infections.

"T" cells effect cell mediated defence mechanism against viral, fungal and mycobacteria agent. Specific T cells called "effector" cells are able to kill host cells which have been infected with virus by producing certain humoral substances (chemical weapons) called lymphokines. Those lymphokines initiate inflammation and enhance the concentration and killing ability of the immune competent cells.

The "B" cells (Humoral antibodies) are the defence mechanisms against bacteria infection. They bind to and neutralize bacteria toxins. They also bind to surface of bacteria and cause direct disintegration of bacteria usually with the help of "appetizers" called Complements. B cells also bind to bacteria allowing for more efficient scavenging (phagocytosis) by macrophages.

It is also the same white blood cells that are deployed to kill off any organ or tissue transplanted from another person. It is well known that though the skin or structures of persons look alike, they contain protein structures called antigens that are peculiar to each person. When such foreign particles are transplanted to another person, the recipient develops 'antibodies' to kill off the particles.

It is being recognized that almost all diseases if they exist long enough will develop features of auto-immunity. There have been theories to suggest that chronic diseases are perpetuated because of element of auto-immunity.

### AUTO-IMMUNITY

Auto-immune diseases could be organ specific such as with thyroid-Hashimoto thyroiditis, primary myxoedema thyrotoxicosis.

|          |   |                            |
|----------|---|----------------------------|
| Stomach  | - | pernicious anaemia         |
| Adrenal  | - | Addison's disease          |
| Pancreas | - | Insulin dependent diabetes |

It could however be systemic in which many organs are affected at the same time, though a particular organ may be more commonly affected in a particular disease. These diseases variously called Connective Tissue Diseases, Collagen Diseases constitute the 'holy grail' of rheumatology. It is the aspect of rheumatology that challenges the diagnostic and management acumen of rheumatologists. *They are auto-immune diseases whose major product, immune complexes are the culprits in the damage of organs.*

The immune complexes are the 'sludge' produced by the interaction of the antigen and the antibody and complements. It represents the "damaged buildings", "killed persons" in war. The collateral damage



in war! The persistence of these collateral damaged structures constitute the product that initiates and perpetuates inflammation which leads to further damage.

Connective Tissue Diseases include:

- a) Rheumatoid arthritis
- b) Systemic Lupus Erythematosus
- c) Scleroderma
- d) Polymyositis
- e) Dermatomyositis
- f) Juvenile Chronic Arthritis

Other allied conditions which are classified as vasculitides include Polyarteritis nodosa, polymyalgia rheumatica, Behcet disease, Kawasaki disease, Panniculitis.

These conditions are recognized as immune complex diseases. Once deposition of immune complex in tissue has occurred, certain cells called mast cells and phagocytes (scavenger cells) are activated. They secrete chemical weapons in their attack leading to inflammatory response. This is usually enhanced through the generation of soluble mediators (messengers or appetizers) called complements. Like when vultures are attracted to carcasses, other cells called polymorphs and mononuclear phagocytes cause release of additional inflammatory messengers (mediators) attracting more cells which secrete various chemicals causing more mess and destruction of cells.

Try as these scavenger cells may, they are unable to remove these immune complexes (sludge) and this leads to prolonged tissue damage.

### **CHARACTERISTICS OF CONNECTIVE TISSUE DISEASES**

1. They are multisystemic, that is many systems are affected at the same time.
2. They affect mostly females.
3. They usually have genetic basis - high concurrence in identical twins.
4. They are characterized by HLA antigen.
5. They are characterized by elevation of acute phase proteins such as ESR.
6. They are characterized by presence of auto-antibodies.
7. They respond to steroids and immuno suppressives.

### **WHAT FACTORS CAUSE AUTO-IMMUNE DISEASES**

#### **"HARAKIRI" APOPTOSIS OR PROGRAMMED CELL DEATH**

The human body behaves like the human society. There is always death in the presence of life and vice versa. So also in the human body, new cells are being formed while old cells are dying. Were this not to be so, the human body may not be able to maintain its shape because of a differential between cell formation and cell death.

Apoptosis or programmed cell death is the means by which this equilibrium is preserved in the human body. Such programming which is controlled by our genes results in death of injured, infected or mutated cells. Nature extols survival of the fittest. These deficient cells opt for "Harakiri" Just like the Japanese Samurai of old. "It is better to die than to be unwanted". Yorubas will say "*Iku ya ju esin lo*" Cell suicide is the result.

These suicidal cells will constitute a mess to the body were they allowed to exist. Just like the animal world scavengers, so also these cells 'stink' get to other healthy cells and scavengers called phagocytes. The dying cells have markers (receptors) on them that say 'eat me'. The phagocytes are attracted to these 'eat me' cells and do exactly that; a form of cannibalism. These cannibal cells should not create a mess of their own. They should eat up cleanly and leave a reaction called inflammation.

However in auto-immune diseases, there seems to be defects in these scavenging job such that the dying cells accumulate so much and these scavengers are overwhelmed, thereby resulting in inflammation. Another possibility is that as these dying cells accumulate, some of the contents in the nuclei may seep out and induce the immune competent cells to generate attacking system called auto-antibodies. The interaction will result in inflammation.

### **IT IS ALL IN OUR STARS**

It is well known that auto-immune diseases result because certain persons have genes in their body which just makes them liable. These genes have been well recognized on white blood cells and they are called Human Leucocyte Antigen (HLA). Rheumatoid Arthritis is for instance seen in those with HLA, DRI & HLADR4. SLE is seen in those with HLADR2, DR3, B.8 Spondyloarthropathies are associated with HLA, B.27

It is all in our stars.

### **'LACK OF APPETISER' THEORY**

For the microbe to be easy and 'delicious' to ingest, there are appetizers in the blood called complements. Deficiency or disorders in some of these complements can result in abnormal

function of auto-antibodies such as in SLE.

### **OUR HUSBAND HAS GONE MAD AGAIN**

With respect to that literary giant, Ola Rotimi. The antibodies which are the 'soldiers' in the body will normally attack any unfamiliar or strange cell or bacteria. That is the essence of our fighting infection from these microbes.

The antibodies are familiar with self cells and do not attack them. In auto-immune diseases, these antibodies seem to have gone crazy and now attack self cells.

A band of these immune competent cells appear to carry out a coup against the body. Compare our Nigerian soldiers.

Those immune competent cells lose their self recognition properties possibly because of aging, tiredness from so many battles or alteration by drugs or irradiation.

### **MIMICRY 'THE FRIENDLY FIRE THEORY'**

Who is not familiar with the friendly fire incidences during the Gulf War and the Iraq expedition. Sometimes the microbe takes on the toga of the body mimicry. In the bid to attack the microbe, the artillery is fired on to normal self cells. Viruses are particularly notorious for adapting cells to produce proteins that are suitable for itself but which becomes foreign to the body.

### **SEROLOGY**

There are a lot of markers in the blood of individuals with auto-immune diseases. These are the artilleries of the epic battle going on. They are called auto-antibodies and are measured in the blood by different methods including Enzyme linked immuno assay (ELISA); gas chromatography; Indirect Immuno-fluorescent method; Latex agglutination, sheep cell agglutination.

Systemic Lupus Erythematosus has the largest array of these auto-antibodies. These include Anti-Nuclear antibody (ANA), Anti-

DNA, Extractable Nuclear Antigen, Rheumatoid factors, Anti-Cardiolipin Antibodies. The substrate for some of these antibodies called immunoglobulins could also be elevated in auto-immune diseases.

We have determined the immunoglobulin levels in patients with arthritis and these have been variously reported in both African Journal of Medical Sciences (Oyemade, Salimonu, Adelowo) as well as West African Journal of Medicine (Adelowo, Salimonu, Oyemade, 1987)

Other reports have highlighted the significance of Anti-DNA antibodies in Sera of Nigerians with particular reference to auto-immune diseases (Adelowo, Salimonu, Arinola African Journal of Medicine and Medical Sciences 1998)

Recent studies (Adelowo, Oguntona) have shown the very high titre of Anti-Nuclear Antibodies (ANA) in Nigeria patients with SLE. We have a patient who has one of the highest ever recorded titre of ANA a 16 year old girl with a titre of 1:102,000 (normal is 1:40).

### **SYSTEMIC AUTO-IMMUNE DISEASES**

Systemic Auto-immune Diseases (Connective Tissue Diseases) can affect any organ or structure in the body where there are Connective Tissues those bunch of cells (tissue) that connect one structure of the body to another, such as the skin to the underlying structure or the covering of the lungs. Organs that have been known to be affected include especially the joints and the skin. Other organs are the kidney, lungs, heart, brain, liver, eye; that is, virtually any organ.

Most of these diseases are due to the deposition of immune complexes (sludge) in the blood vessels and elsewhere. These immune complexes are capable of generating inflammation and deposition of all sorts of white cells such as lymphocytes,

macrophages, phagocytes. The release of different mediators (chemicals) in this process result in damage to different organs.

Most types of Connective Tissue Diseases have been identified among Nigerians including:

- a) Rheumatoid arthritis with manifestation commonly in the joints.
- b) Systemic Lupus Erythematosus - manifestation commonly in the joints and skin but affecting virtually all organs of the body.
- c) Scleroderma - with manifestation on the skin.
- d) Polymyositis - manifestation in the muscles.
- e) Sjogren's syndrome - manifestation in secretory glands such as saliva, tear drops, digestive secretions in the intestine.
- f) Anti-phospholipid syndrome - with manifestation in the pregnant uterus resulting in recurrent abortion.

### RHEUMATOID ARTHRITIS

This is an auto-immune disease affecting mostly females in age group 30 and above. It usually presents with a crippling destructive arthritis characterized by ill-health, arthritis of joints of the hands, elbows, knees, ankles and feet. There is associated stiffness in the joints on waking up in the mornings, small knobby swelling at the elbows and elsewhere. However, the damage going on in the joints may also affect the lungs, heart, eyes, kidneys.

Rheumatoid factors, a blood measurement of mediator of inflammation may be detected in the blood. Diagnostic X-ray changes may be seen, though infrequent among Nigerians.

In a study reported in Nigerian Medical Practitioner (1985), Adelowo gave a frequency of 9.5% in patients seen in a rheumatology clinic. In a recent study (Adelowo, Oguntona), rheumatoid arthritis accounted for 10.1% of the 1000 cases of arthritis seen in an arthritis clinic.

Rheumatoid factor which is seen in about 60-80% of patients are said to be detected in quite a small percentage of 14% among Nigerians (Greenwood, 1968).

In our recent report, however, Rheumatoid factors were observed in about 40% of the cases seen. Is there a change in incidence of this important factor? Possibly yes. It is also suggested that they may be due to the fact that these tests were done on an urban population of patients. It has been confirmed that the incidence of rheumatoid factor is higher among the urban population.

There has been a paradigm shift in the management of these cases. Up to 20 years ago, most of our patients ended up with marked deformities of the joints and disability. Then we used to treat them with kid gloves medicine or Non-steroidal anti-inflammatory drugs. These drugs control the pain and swelling but the disease rumbles underneath. We are now using Disease Modifying Drugs (DMARD) earlier in the disease. Such drugs as Methotrexate, Sulfasalazine, Chlorambucil have contributed tremendously in reducing deformities and even early deaths in these patients.

Newer agents that target specific steps in the inflammatory cascade are now being prescribed. Such agents are drugs targeted at alpha-tumour factors and anti-Interleukin 1. There are obviously interesting times ahead.

# SYSTEMIC LUPUS ERYTHEMATOSUS (LUPUS)

## THE HYDRA-HEADED DISEASE, THE CHAMELEONIC DISEASE

Probably the most challenging disease any rheumatologist has to face is Systemic Lupus Erythematosus. It is the ultimate disease affecting virtually any organ or structure in the body. It is the ultimate disease. It is a hydra-headed disease. No sooner do you treat one aspect than another one appears. It presents as a chameleon changing its presentation from day to day. It affects the skin, joints, eyes, lungs, heart, kidneys, brain, nerves, intestine and even the uterus carrying a baby.

Twenty years ago, a diagnosis of SLE was a definite death sentence. However with improved diagnostic skills, early detection, better management of disease and complications, we are upbeat that 80% of cases will still be alive 10 years down the line.

Lupus is virtually a disease of blacks and Hispanics. It affects almost exclusively women of child bearing age mostly. In the USA, the prevalence is 1:1000; Annual incidence is 68/100,000. In Los Angeles area, it affects one in every 250 women of child bearing age.

SLE had been said to be rare among Black Africans. Our findings do not support this. In a study of 1000 cases of arthritis seen in a rheumatology clinic, 52 (5.2%) had confirmed SLE. I believe we are missing or overlooking these cases. We have previously reported cases in the Nigerian Medical Practitioner (Adelowo, O. O. et al, 1994)

The diagnosis is made using American College of Rheumatology Criteria. In our series, the commonest presentation is joint pain and swellings. Others include rashes on the face, skin rashes



elsewhere, pleurisy, kidney presentation, convulsion, blood abnormalities such as low white blood cells (Leucopenia), low red blood cells (anaemia); low platelet count (thrombocytopenia).

One diagnostic finding in our series is the markedly elevated Erythrocyte sedimentation rate about 100 (seen in 39% of cases) and above 50 (in 84% of cases).

Another landmark finding is the markedly elevated auto-antibodies, anti-Nuclear antibodies seen in almost all the cases. There are other auto-antibodies seen, as Lupus has the largest array of auto-antibodies.

Treatment should be started early to avoid the renal and central nervous system complication seen early in blacks. Very powerful drugs such as steroids, and immuno-suppressives like Cyclophosphamide, Azathioprine, Cyclosporin are indicated.

### **RARE CONNECTIVE TISSUE DISEASES**

Other rare Connective Tissue Diseases seen among Nigerians include Scleroderma (Adelowo, Central African Journal of Medicine; 1985); Anti-Phospholipid Syndrome (Nigerian Medical Practitioner).

### **OTHER TYPES OF ARTHRITIS**

Other types of arthritis that have been reported among Nigerian include:

- 1) Psoriatic Arthritis (Adelowo O. O. Nigerian Medical Practitioner, 1987).
- 2) Diffused Idiopathic Skeletal hyperostosis (Adelowo O. O.

- WAMJ 1994).
- 3) Arthritis of Inflammatory Bowel Disease (Adelowo, Williams, Kuku, 1998).
  - 4) Avascular Necrosis with Arthritis (Adelowo O. O., Nwosu A. O. Nigerian Medical Practitioner, 1999).
  - 5) HIV and Arthritis (Adelowo O. O. et al, Nigerian Medical Practitioner, IN PRESS).

### **DIALYSIS**

This is not part of rheumatology as my colleagues will recognize! In a unique study at a private hospital, we had looked at the possibility of a Chronic Peritoneal Dialysis programme in a private hospital (Adelowo O. O., - Oladiran B African Journal of Medicine and Medical Sciences, 1994). There had not been any previous report of such programme in any hospital in Nigeria. Previous reports had studied acute peritoneal dialysis.

Our study, using Oreopoulos chronic dialysis catheter showed that an Intermittent Peritoneal Dialysis programme may be more suitable than Chronic Ambulatory Peritoneal Dialysis. This may be an alternative to the more expensive Haemodialysis.

### **RHEUMATOLOGY: WHAT'S HAPPENING, WHAT'S ON, WHAT'S UP**

Language changes from generation to generation. The cliché used to be What's happening and then What's on? Whatever it is, the question is still- What's new?!

There are new developments in rheumatology like in all other specialties of medicine. New frontiers are being overcome, new drugs and modalities of treatment developed. A few examples:

## OSTEOARTHRITIS

This is a condition in which the degeneration of joint cartilage progresses inexorably. There is hitherto no known agent that will stop this progression. However, there are new possibilities in a treatment modality called VISCOSUPPLEMENTATION. Here an important constituent of the lubricating fluid in the joint called synovial fluid, (hyaluronic acid) is injected into the joint. These injections into the joints of the knee enhance the lubrication of the joint, reducing friction and this may eventually slow down the joint degeneration. This modality is being used more and more especially for patients who refuse or are unsuitable for surgery. Our own experience in the twenty or so patients injected are quite encouraging. Thus this treatment appears to be the best medical modality for osteoarthritis of the knee.

Ultimately however, the most enduring treatment will be replacing the degenerated cartilage. Genetic manipulation to stimulate new cartilage cells to grow and repair the worn out cartilage is being tried among animals. Human trials may be just a few years away.

Another research is the cultivation of cartilage cells in the laboratory and transferring these into the joint.

Pain killers that do not irritate the stomach, COX-2 inhibitors, have been developed. There are a few problems with them, but they still offer the safest treatment for relieving pain of many forms of arthritis. They are not necessarily more effective but are definitely safer as far as the gastro-intestinal tract is concerned.

## AUTO-IMMUNE DISEASES

When the body is at war with itself, a ceasefire must be established before cessation of hostilities!

One way out is to send the "rebel soldiers" to sleep with immunosuppressives. Unfortunately these drugs will also suppress (send to sleep) normal cells! There are agents being developed that target only the "rebellious" cells without affecting the "obedient" cells.

It is also possible to intervene even after the antibodies have interacted with the cells of the body. This can be done at different phases of this interaction. As such, there are drugs that attack specific molecules of the inflammatory cascade. The agent that has brought hope and relief to so many include the agents called Anti-tumor necrosis factor alpha (ANTI TNF $\alpha$ ). Those available include Etanercept, Infliximab, Adalimumab). These drugs are increasingly being used for patients not responding to the standard immunosuppressives. A lot of patients with different auto-immune diseases have walked again and their quality of life have improved.

Other agents being increasingly used are Anti-Interleukin 1 (Anakinra).

### MY CONTRIBUTION TO THE UNIVERSITY

I have been in the University system, on and off for the past 22 years. I rose from the post of Lecturer 1 to Senior Lecturer at University of Ilorin in 1985. I have had an inter-regnum in private medical practice and public service. I returned to the university recognizing that is the place I have my fulfillment as a human being. I rose to the level of reader and to a full professor of medicine in 2001.

At every level, I have served on various committees of the university, I was Head of Department of Medicine and culminating in my present post of Dean of the Faculty of Clinical Sciences.

As Head of medicine, I have had the support of my colleagues in introducing Problem Based Learning. This is fun and a detailed way of learning for students and teachers alike. As opposed to didactic learning where the teacher regurgitates 'stuff', students are challenged with hypothetical but practical questions. They will search for the answers and will bring these to the class for discussion. Authorities in Medical Education have found out that this is the best way to learn.

As Dean, Faculty of Clinical Sciences, we have introduced a few things.

- 1) We had the first Faculty retreat programme in 2004. Our Vice-Chancellor was the chairman. Students and staff discussed issues on medical education including Objective Structured Clinical Examination (OSCE); Problem Based Learning, Computer and Internet application in Medical Education.
- 2) Our Faculty organizes regular Faculty Lectures. We invite role models who are not necessarily medical doctors to discuss issues outside medicine but relevant to well-being of and future of our students. We have had a past President of Nigeria Bar Association to talk to us on "Law and the Society"; Managing Director of a Discount House to discuss "Investment Opportunities in Nigeria"; a motivational speaker to discuss "Equipping yourself for success". We have even invited a topmost female Engineer to talk about "Home and Career: a balancing act". Recently an emeritus

professor of medicine gave a talk on "Medicine Past, Present and Future". We believe we need to equip our students for the future. They should be well rounded and grounded.

- 3) We have informal programmes with medical students tagged 'Express yourself', where students are encouraged to discuss freely on current topical or ethical issues. We have discussed "Euthanasia", "Abortion". It is a village square discussion!
- 4) We have sourced for funds, from friends of the Faculty, which have financed such projects as:
  - a. providing air-conditioners for our medical library.
  - b. furnishing a reading room for medical students in their hostel.
  - c. furnishing a tutorial room for medical students' use in the hospital operating theatre.
- 5) We recognize that staff welfare is important. We have bought many air-conditioners for use in academic staff offices.
- 6) We have sourced for funds to provide curtains in our Glass House theatre, thus allowing use of audio-visuals.
- 7) We devote an afternoon every 3 months to effect students/faculty adviser interaction.
- 8) We hold regular meetings with our medical students' association and class representatives to explain policies in the Faculty and exchange views.

## CONTRIBUTIONS TO THE NATION

- 1) I have served at the highest legislative level by my appointment as one of the 46 men and women that drafted the Constitution of Nigeria in 1987 - 1988. I was the only non-legal practitioner in the all important Legal Drafting Committee.
- 2) I was also a nominated member of the Constituent Assembly (1988-1989)
- 3) I write a regular Health Education column in Saturday Guardian titled "Questions you had always wanted to ask your doctor".

## CONTRIBUTIONS TO MEDICAL PROFESSION

1. First Nigerian Professor of Rheumatology.
2. Vice Chairman NMA (1984-1985).
3. Vice President, African League of Associations of Rheumatology (1989-1996).
4. External Examiner, Universities of Ibadan, LAUTECH, Ilorin, Ile-Ife, Jos, Lagos.
5. Examiner, Fellowship of Post-graduate Medical College, FWACP.
6. Faculty Secretary, Faculty of Internal Medicine, NPMCN.
7. Author of Post graduate curriculum for Rheumatology FWACP, FMCP.

## RECOMMENDATIONS

1. Establishment of more units of rheumatology in Teaching hospitals and Specialist hospitals.
2. Training more rheumatologists.
3. Increasing awareness of the scope of rheumatic diseases in

- the community.
4. People should avoid precipitating causes of certain types of arthritis.
  5. Doctors to be encouraged to refer patients with arthritis early to rheumatologists.
  6. Increased funding of our hospitals.
  7. Increased participation by private organisations in funding projects in our universities and hospitals.

### **ACKNOWLEDGEMENT**

I had always wanted to be a doctor. I was told by my parents that while others played, I played doctor. A long stick was my stethoscope. I got into rheumatology with my eyes wide open for three reasons:

- a) I had always wanted to do something different from others.
- b) I had listened to a lecture on rheumatic diseases and I was enchanted by the scope
- c) I had a dear grandmother who suffered from arthritis. I thought I could help an old woman.

My first and loudest acknowledgement goes to the Almighty God, the ever Merciful, Compassionate, Gracious, Loving Father who by His grace caused me to be created and to have travelled this far on this journey called life. He has showered untold blessings on me. He is God that allows you a detour in life but brings you back on your road of destiny.

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- 1) Tolulope Abimbola
- 2) Fopefoluwa Kikelomo
- 3) Fogofoluwa Olufemi

I leave you with the lyrics of a record by Simon and Garfunkel .....In the clearing stands a boxer and a fighter by his trade and he carries the reminders of every glove that laid him and cut him, till in his anger and his shame, I am leaving, I am leaving **BUT THE FIGHTER STILL REMAINS.**

The Vice-Chancellor, Sir, Ladies and Gentlemen, **THAT IS MY STORY and THAT IS MY SONG.** Thank you.

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