Voice Of APLAR heumatology

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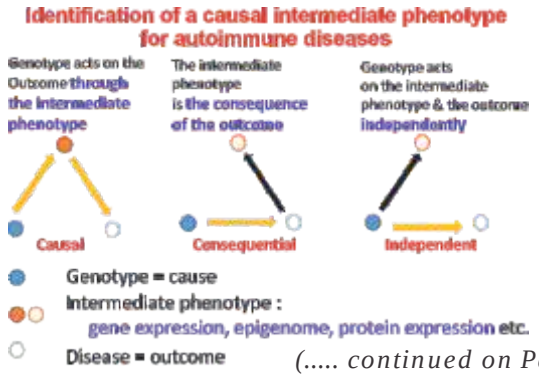
Genetic and functional genetics of autoimmune diseases

Kazuhiko Yamamoto

The majority of autoimmune diseases, including rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE), are multi-factorial diseases that develop through the interaction of several factors, such as genetic and environmental factors. A limited number of disease susceptibility genes such as those of the major histocompatibility complex have been known to exist for several decades. After these eras, genome-wide association studies (GWAS)



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"November 4, 2017

MUMBAI Physician Receives Top Honor from American College of Rheumatology

DR. PRAKASH PISPATI was honored with the designation of Master by the American College of Rheumatology (ACR) during the 2017(Nov.3) ACR/ARHP Annual Meeting in San Diego. Recognition as a Master is one of the highest honors that the ACR bestows on its distinguished members.

The designation of Master is conferred on ACR members, age 65 or older, who have made outstanding contributions to the field of rheumatology through scholarly achievement and/or service to their patients, students, and the rheumatology profession. Honorees have devoted their careers to furthering rheumatology research and improving clinical standards in the treatment of rheumatic diseases..."

ACR President Elect David Daikh, Prakash Pispati, ACR President Sharad Lakhanpal

Nailfold Capillaroscopy : A practical BEDSIDE tool

Group Captain V Vasdev

Raynaud's phenomenon (RP) is one of the common symptoms encountered in Rheumatology practice. The biggest challenge for a clinician is to differentiate between the primary and the secondary RP. There are a few clinical clues and investigations that may help a Rheumatologist to differentiate between the two as depicted in Table 1.

Out of all these features, Nailfold capillaroscopy (NFC) has an exceptionally high negative

Table 1 - Differentiating features primary vs secondary Raynaud's

Features	Primary RP	Secondary RP
Prevalence	Common	Uncommon
ANA positivity	Frequent	Infrequent
Association with SCTD	Yes	No
Complications (Digital ulcers/ necrosis)	Yes	No
Characteristic NFC changes	Yes	No

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From the Editor's Desk

E-in-C

The Mysterious Triangle:

HLA B27 SI Joints AS

LONDON .... right behind the Big Ben is this renowned historic Westminster Hospital. Rheumatologist Professor Derrick Brewerton was enjoying lunch and gossip with Dr. David James who had just returned from Africa.(see below). Painstaking research followed unraveling the mysterious triangular affair between HLA B27, the sacroiliac joints vis-à-vis Ankylosing Spondylitis and 'cousins' the Spondarthropathies. May I present to you this fascinating story rather unusually. Afterall a picture is worth a thousand words, isn't it? The 'mysterious triangle' between *HLA B27*, *Sacroiliac joints* and Ankylosing Spondylitis can be as intriguing as the 'Bermuda triangle'. When at a short sabbatical at Oxford some 15 years back, I was a witness to a brainstorming session between rheumatologists and geneticists. Two of them had returned from Africa wondering why AS is a rare disease there. They postulated that subsets of HLA B27 gene presented

This Week's Citation Classic

CC/NUMBER 29
JULY 21, 1980

Brewerton D A, Caffrey M, Hart F D, James D C 0, Nicholls A & Sturrock R D.
Ankylosing spondylitis and HL-A 27. *Lancet* 1:904-7, 1973.
[Westminster Hospital, London, England]

The inherited antigen now known as HLA B27 was found in 72 of 75 patients with the rheumatic disease ankylosing spondylitis and in three of 75 matched controls. The same antigen was also present in 31 of 60 first-degree relatives. [The *SCI*® indicates that this paper has been cited over 385 times since 1973.]

Derrick Brewerton
Westminster Hospital
Horseferry Road
London SW1P 2AP
England

June 12, 1980

"It was lunch in the common room at Westminster Hospital on a hot summer day in 1971. As a rheumatologist I had been fascinated for many years by the enigma of ankylosing spondylitis, which is primarily a

was obvious. Although it now seems strange, we made a joint decision in 1972 that James and Caffrey would write a brief (laboratory oriented) report for Nature,[1] while I wrote a

Bluestone and Terasaki. Their article in the *New England journal of Medicine*[2] was published between our *Nature* and *Lancet* reports, all three appearing within a few weeks. Subsequently, James, Bluestone, and I shared the Robecchi (European) and Geigy (International) prizes for arthritis research. I won the Bose prize of the Royal College of Physicians.

"Before writing the *Lancet* article, we had already established that HLA B27 was also strongly associated with acute anterior uveitis (iritis), seronegative limb arthritis, Reiter's disease, and the spondylitis in patients with psoriasis, ulcerative colitis, and Crohn's disease. In the article we hinted that this might

reports, all three appearing within a few weeks. Subsequently, James, Bluestone, and I shared

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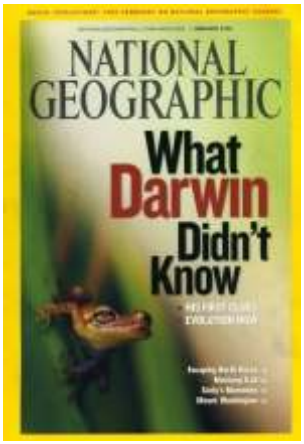
clues eg. probably 2705 makes Caucasians vulnerable and 2701 makes Africans resistant to AS.... Since then we know a lot, time is not away when genetic manipulation may make human species less vulnerable to AS.

As a medical student we were taught four cardinal principles were paramount while examining a patient :

Inspection, Palpation, Percussion, Auscultation. Try it on SI joints in a patient with low back pain. You will be foxed because SI joints cannot be seen, touched or heard .... only indirect tests clinical, laboratory and imaging to assist never foolproof.

The tiny pair of SI joints, what are they actually, structurally? ; Synovial, cartilaginous or fibrous ? This question I often pose to medical audiences, rarely to get a right answer. *Gray's Anatomy* in minute detail will tell you that SI joints are synovial for a start, turning cartilaginous after 35 – 40 years of age wisely permitting lithotomy position during parturition in women, then over time turning fibrous. Check it out !The deep-seated pair of SI joints are hard to reach by invading microorganisms directly sometimes via blood stream but mostly by remotely controlled cytokines triggered by abnormal microbial flora in the gut, the larynx, the skin, the lungs, the urinary / the reproductive tracts ... We blame such diverse microbes as Gram negative, Gram positive, acid fast microorganisms, viruses, even parasites ! So we have different subsets under one generic term *Reactive Arthritis*.



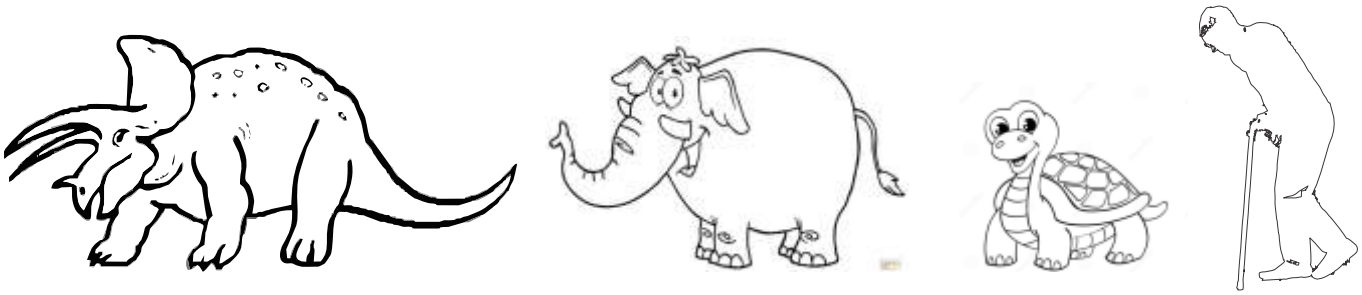
Strangely, commonly used tests such as Rheumatoid Factor, Anti-CCP, ANA and subsets, ANCA, ACLAs APLAs etc. are strictly all *negative*. So the definition *Seronegative Spondyloarthritis*. As new biomarkers are discovered / invented, the prefix *Seronegative* may become minimal or cancelled out.

Indeed I find this 'love triangle' between HLA B27, SI joints and Ankylosing Spondylitis mysterious. No wonder the theme alongside complex collagen vascular diseases dominates most Rheumatology congresses .

National Geographic to mark 150th Birth Anniversary of Charles Darwin had a specially designed cover page. Inside, It predicted that gene - environment interaction can be modulated to predict, prevent diseases protecting us hopefully. Is this happening? Pose this question to the new breed of genetic bioengineers.... the future is upon us. Till then, am afraid we have got to bear the burden of backache probing its backyard.

Prakash Pispati, M.D., F.R.S.M. (Lon.), M.Sc (Med.), FICP
Editor-in-Chief : Voice of APLAR
Master – American College of Rheumatology
Master, Honorary Member and Past President – Asia Pacific League of Associations for Rheumatology
Director of Rheumatology, Mentor - Jaslok Hospital & Research Centre, Mumbai, India
Sr. Consultant Rheumatologist - Saifee Hospital, Mumbai, India
Past President – Indian Rheumatology Association

Snippet...



60,000 kg 11,000 kg 914 kg 65 kg

Are quadrupeds adept at weight bearing? Are we bipeds poor in our backs ?

Count Leo Tolstoy seemed bothered likewise so he guessed, *"Man is the only animal that walks on two legs, drinks when not thirsty and makes love in all four seasons."*

How true, isn't the human zoo a biologist's nightmare ?.....*Prakash Pispati,* E-in-C

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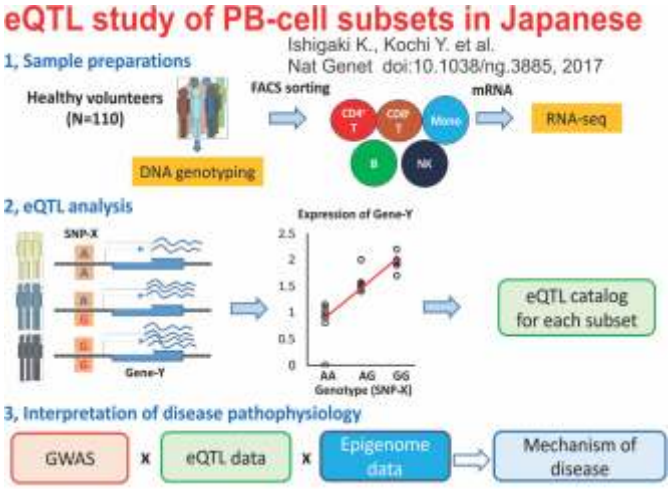

have been used for more than 10 years to identify susceptibility genes for several autoimmune diseases. These findings have contributed to our understanding of the pathogenesis of these diseases. As the analysis of susceptibility genes has progressed, it has become apparent that many disease susceptibility gene variants are involved at the expression level of genes, which is called expression Quantitative Trait Locus (eQTL). Furthermore, expression of genes related to disease pathogenesis is cell-specific, with involvement of epigenetic mechanisms. Genetic information exists before the onset of disease, and thus has a causal relationship to the disease. Therefore, the analysis of genomic function in human autoimmune research is essential, with regard to understanding the pathological mechanisms as well as having applications for drug discovery.

Kazuhiko Yamamoto
Laboratory for Autoimmune Diseases
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Can patients with ANCA associated vasculitis be treated with complement blockade?

A synopsis of invited talk in BMJD 2017 Annual Congress, Gold Coast, Australia, August 31st to September 3rd 2017

The histopathological hallmark of ANCA-associated vasculitis (AAV) is "pauci-immune" necrotizing crescentic glomerulonephritis, characterized on renal histology by little or no glomerular staining for immunoglobulins and complement. Therefore, it was previously assumed that the complement system is not involved in the pathogenesis of AAV. However, increasing evidence suggests that activation of the complement system, via the alternative pathway, might play a role in the development of AAV. In a mouse model of AAV induced by anti-MPO IgG, C5-/- mice developed no disease, indicating complement activation is vital in the development of AAV. C4-/- mice developed disease comparable with wild-type mice, indicating that neither the classic nor the lectin complement activation pathways are required for disease induction. In contrast, B-/- mice developed disease, indicating that alternative pathway complement activation is required for induction of AAV.

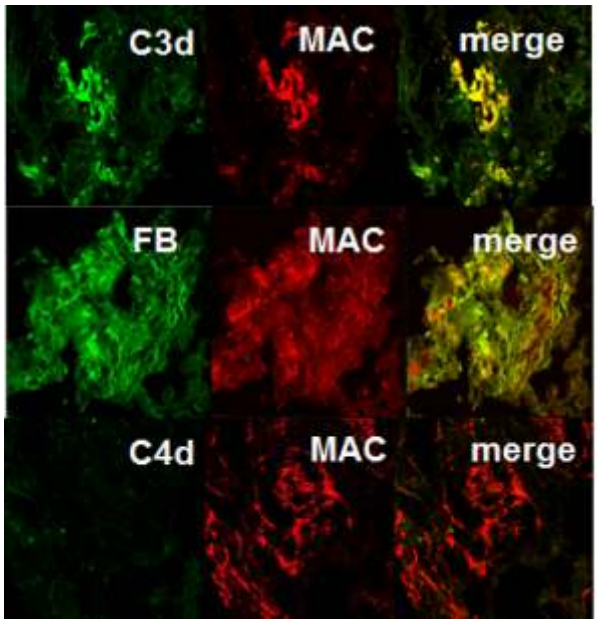

Fig 1. Co-localization of complement components in glomeruli of MPO-ANCA positive patients with microscopic polyangiitis (MPA) using direct immunofluorescence assay on frozen renal biopsy tissue. The colocolization of C3d and the complement activation end product membrane attacking complex (MAC) indicated that complement was activated in ANCA associated vasculitis; Colocolization of factor B (FB) and MAC indicated that alternative pathway was involved, while no colocolization of C4d and MAC indicated that neither the classical pathway nor the lectin pathway were involved in ANCA disease.

Xing GQ et al. J Clin Immunol 2009;29:282-91

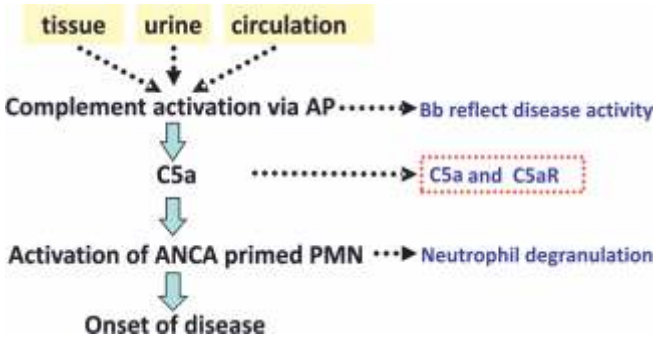

Fig 2. Evidence from circulation, kidney tissue and urine demonstrated that complement activation via alternative pathway in ANCA disease. The interaction between complement activation product C5a and its receptor C5aR may paly an important role in the pathogenesis of ANCA disease.

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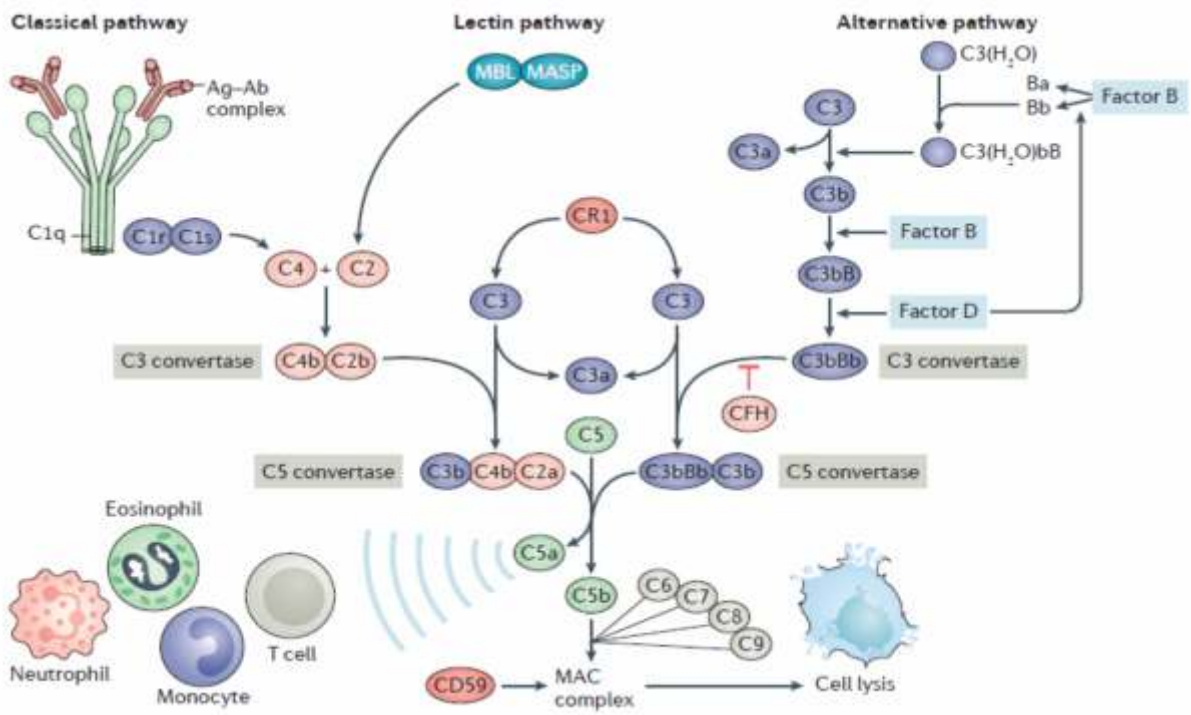

Fig 3. The three pathways of complement activation: the classical, mannose-binding lectin and alternative pathways.
Three pathways of complement activation exist: the classical, mannose-binding lectin (MBL) and alternative complement pathways. Activation of the classical pathway begins with the binding of immune complexes to C1q leads to the activation of its C1r and C1s serine protease subunits. Then, C1s activates C4 and C2, which subsequently results in the formation of the classical pathway C3 convertase, C4bC2a. The alternative pathway is initiated by hydrolysis of C3. Factor D cleaves the C3b-bound factor B, resulting in the formation of C3bBb, the C3 convertase. The lectin pathway is activated through the binding of MBL and mannose-associated serine protease 1 (MASP). MASP can cleave and activate C4 and C2 to form C4bC2a. Activation of any abovementioned pathway leads to activation of C3. Activation of C3 generates C3b and C3a, a chemo-attractant factor. Sufficient activation of C3 leads to the activation of C5. C5 activation leads to the formation of C5a and C5b–9.
Chen M, Jayne DR, Zhao MH. Nat Rev Nephrol. 2017 Jun;13(6):359-367.

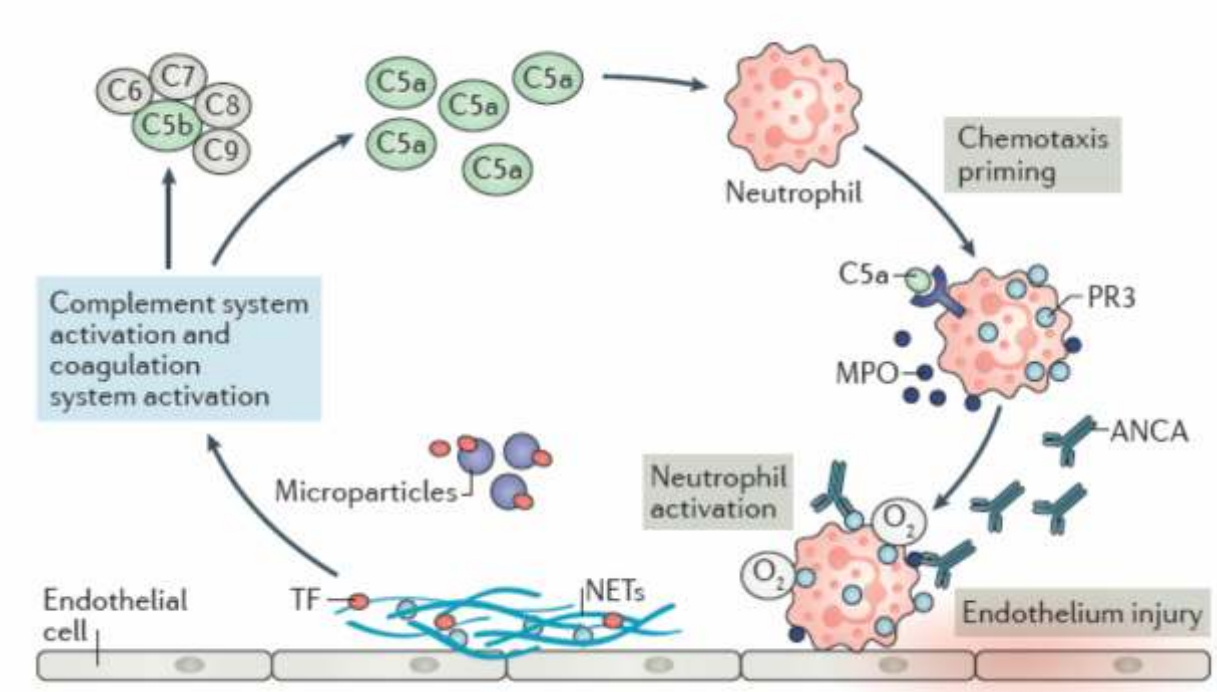

Fig 4. Proposed working model for the interaction of anti-neutrophil cytoplasmic antibody (ANCA), neutrophils and complement activation in the pathogenesis of ANCA-associated vasculitis. Neutrophils are primed by cytokines, such as C5a or TNF, which leads to the translocation of ANCA antigens from cytoplasm to the cell surface. ANCAs can further activate primed neutrophils to undergo a respiratory burst and degranulation, and release tissue factor (TF)-bearing microparticles and NETs. Activated neutrophils can lead to the injury of endothelial cells, activation of the coagulation system, and activation the alternative complement pathway via their cell membranes, microparticles and NETs. Activation of the alternative complement pathway and NETs in turn leads to the generation of C5a, which amplifies the inflammatory response through enhanced neutrophil recruitment and priming of neutrophils for ANCA-mediated activation.
Chen M, Jayne DR, Zhao MH. Nat Rev Nephrol. 2017 Jun;13(6):359-367

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In human studies, we found that vasculitic lesions in affected glomeruli stained for complement factors C3d, Bb and C5b-9; our further studies identified that levels of the unique alternative complement pathway factor Bb, measured in serum, urine and renal histology, closely associated with disease activity.

Recent studies indicated that the interaction between C5a and C5a receptor (C5aR) on neutrophils composes an amplification loop and thus, plays a central role in the pathogenesis of AAV. In human studies, circulating level of C5a in active AAV was significantly higher, compared with AAV in remission. In vitro studies indicated that C5a could dose-dependently prime neutrophils for ANCA-induced respiratory burst and degranulation. The generation of C5a could also lead to infiltration and degranulation of more neutrophils at sites of complement activation resulting in the development of inflammation.

Targeting the complement system is emerging as a novel treatment approach for AAV. Eculizumab, a humanized recombinant monoclonal antibody against C5, was used in an AAV patient with success. The recently released CLEAR study, a multicenter phase II randomized double-blinded placebo-controlled trial led by EUVAS, demonstrated the efficacy and safety of CCX168 (avacopan), a small molecule inhibitor of C5aR, in patients with AAV. This study showed that CCX168 could replace corticosteroid treatment with a more rapid onset of effect and lower adverse effects. A parallel phase II study of CCX168 on AAV conducted in North America, CLASSIC, showed similar results.

In conclusion, activation of the complement system via the alternative pathway plays a major role in the pathogenesis of AAV. Blocking complement activation, especially inhibition of C5aR might be a promising therapeutic option for patients with AAV.

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(..... continued from Page 1) ....Nailfold Capillaroscopy predictive value and good positive predictive value for early distinction between primary and secondary RP. Sadly, NFC remains a grossly underutilized investigation despite its proven role in Rheumatology practice. Presently, the gold standard tool for NFC is a nailfold video capillaroscope (NVC). NVC is an expensive equipment with limited availability in developing countries. Despite the obvious advantages and the undisputed diagnostic value, NFC using NVC largely remains underutilized, mainly due to cost factors, lack of expertise and availability issues. The silver lining is that several other instruments such as Ophthalmoscope, dermatoscope, USB microscope and stereomicroscope can also be effectively used for NFC albeit with few limitations.

IMPORTANCE OF NFC IN RHEUMATOLOGY

The consensus emphasizes that all patient with RP should undergo a NFC examination to assist in characterizing the clinical and evolving pro?le. Besides, there is a myriad of information that can be obtained by NFC in various Systemic connective tissue disorders (SCTD) and other systemic diseases with microangiopathy, such as Coronary artery disease and Diabetes Mellitus. One of the best advantages for clinicians can have with NFC is its high negative predictive value for CTD (>90%) in subjects with RP. On the other hand, its positive predictive value is only about 50%, but this is higher than any other single screening test including autoantibodies. NFC changes have also been included as a part of the recently updated classification criterion for systemic sclerosis by the European League Against Rheumatism (EULAR) in 2013. NFC changes can be very useful in differentiating Dermatomyositis(DM) and polymyositis (PM). In DM, there are florid NFC changes as against minimal changes in PM. Scleromyositis is an overlap syndrome combining features of SSc and DM/PM, associated with the positivity for anti-PM/Scl antibodies, more benign course than SSc, and normal capillaries on NFC. However, gross NFC abnormalities may predict emergence of typical SSc. Antiphospholipid syndrome (APS) and small-vessel vasculitides can both present with NFC changes in form of multiple microhaemorrhages and hemosiderin deposits, with morphologically normal capillaries.

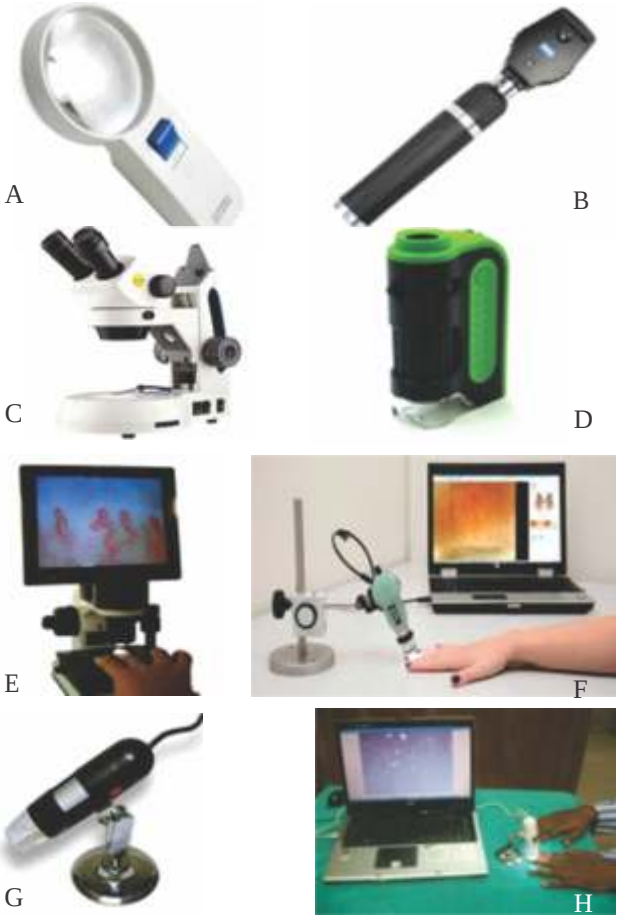

Fig 1. Instruments for NFC A- Magnifying glass; B- Ophtalmoscope; C- Stereomicroscope; D- Hand held microscope; E- Nailfold video capillaroscope; F- Capillaroscope ; G & H- USB digital microscope

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Table 2 - Analysis of various instruments for nailfold capillaroscopy

TECHNIQUE	ADVANTAGES	DISADVANTAGES
Magnifying glass (with inbuilt light source)	Ease of use and availability Cost factor	Low magnification 5-10 X Can visualize only gross capillary dilatation
Ophthalmoscope	Easy availability	Low magnification 10 - 20 X Can visualize only gross NFC changes Difficult to use for operator Poor reproducibility
Dermatoscope	Easy availability in dermatology practice	Same as Ophthalmoscope
Hand held microscope	Ease of use and availability Cost effective Small size Good magnification 20-40 X Acceptable resolution and sensitivity for major NFC changes	No option for storing images Cannot be used for subtle NFC changes or research activities
Stereomicroscope	Moderate cost	Cumbersome
	Good resolution & magnification	Not available in most OPDs Specialized training required Additional camera and fibreoptic light source required Time consuming
Nailfold video capillaroscope	Ideal tool for research activities Magnification upto 600 X Excellent image quality reproducibility	Prohibitive cost Availability Needs specialized training Time consuming Cannot be used bedside or in OPDs
USB digital microscope	Ease of use & availability Cost factor, Small size Quick operation Magnification upto 200 X Good resolution and sensitivity Ideal for bedside NFC/ OPD care	Not an ideal tool for research purposes.

INSTRUMENTS FOR NFC

NVC is considered the gold standard for carrying out NFC. The major drawbacks for use of NVC for NFC are its cost, time factor, need for special training and its non suitability for use in the OPD services. Various factors especially cost and specialized training is a major hurdle in popularizing this extremely important investigation.

We recommend simple, inexpensive instruments like USB microscope, Capillaroscope and handheld microscope for clinicians in OPD practice. However, NVC is ideally suited for research activities as well as analyzing subtle abnormalities and complex calculation of various indices. The major advantages and disadvantages of each technique are summarized in Table 2.

METHODOLOGY

However, for screening purposes in a busy OPD, examination of ring and little finger is enough. We analyse the stored images for linear capillary density, capillary tortuosity, variability in capillary length, number of enlarged capillaries, avascular areas and presence of disorganized architecture. We classify the observed changes in NFC as normal, scleroderma pattern or non specific abnormalities. 'Scleroderma pattern' is characterized by the presence of dilated capillaries,

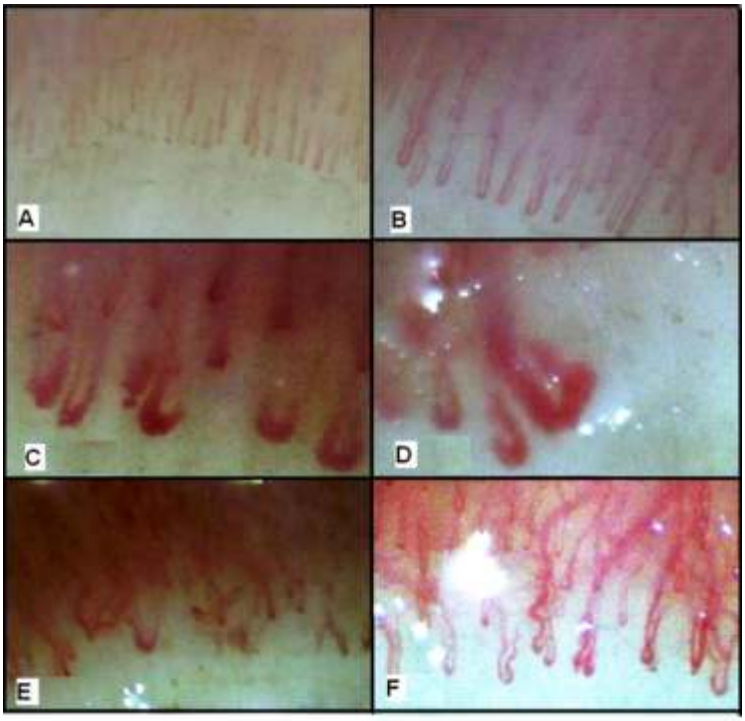

Fig 2. Characteristic nailfold capillaroscopy findings in subjects using USB digital microscope A & B– Normal NFC at 30 X and 100 X, respectively. C - Dilated capillary loops with reduced capillary density in systemic sclerosis. D – Non specific dilatation and tortuosity in SLE E – Completely disorganized architecture in advanced systemic sclerosis. F – measurement of capillary density by photographing milimetre scale alongwith capillaries.

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Can you treat systemic lupus erythematous when you can't see or recognize a target?

Tommy Cheung

Treat to target strategy has proven very effective in may chronic diseases eg RA. Therfore it is reasonable to consider this treatment approach in SLE. Treatment of SLE should aim at ensuring long-term survival, preventing organ damage and optimising health related quality of life, by controlling disease activity, minimising comorbidities and drug toxicity. The treatment target of SLE should be remission of systemic symptoms and orgam manifestations.

Clinicians should continue to treat and monitor patients with SLE aggresively. Patients with renal lupus, treat to target strategy sgould be implemented according to the Joint EULAR/ERA-EDTA recommendations for the management of lupus nephritis.Complete renal presponse should be the ultimate therapeutic goal, which is defined as urine protein less than 0.5 gm per day and normalization of renal function.

In addition to tight control of disease activity, other startegies should be implemented to prevent disease flare as it is a major contributing factor for damage accrual. Use of immunosuppressive agents should be considered for all patients as it is effective in preventing major flares and reducing cardiovascular morbidities. Although serological activity is associated with the risk of disease flare, it is currently not rcommended to intensify treatment in patients without clinical activity because the use of immunosupressive agents, in particular glucocorticoid, can lead to furthet organ damage.

Researches are underway to evaluate the optimal approach and target for implementing this new concept. In the near future, identification of accurate biomarkers of disease activity and development of more efficacious but yet safe therapeutic agents can further contribute to successful implemntation of treat-to-target in SLE.

(Synopsis of detailed presentation at Conference of Bone, Muscle, and Joint Disease (BMJD) – APLAR Symposium, Gold Coast Australia, 2017).

Tommy Cheung
Clinical Assistant Professor
Division of Clinical Pharmacology
Department of Medicine, LKS Faculty of Medicine
The University of Hong Kong

BREAKING NEWS

APLAR Hon Treasurer Dr Debashish Danda installed President IRA 2017 -2019

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welcomes *YOUR*

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Lets have your e-mail / WhatsApp number for future VOA issues

E-in-C...prakashpispati@gmail.com

....*continued from page 7* ....Nailfold Capillaroscopy

megacapillaries and decreased capillary density with vascular deletion areas. Non specific capillary loop abnormalities include mild capillary dilatation, variability of loop length and mildly reduced capillary density. Some of the images and typical abnormalities recorded with simple USB microscope are shown in Figure 2.

CONCLUSION

Nailfold capillaroscopy (NFC) is a simple and non-invasive technique for the analysis of microvascular abnormalities seen in various SCTD, especially in SSc group of disorders. NFC despite its immense potential remain grossly underutilized owing to certain shortcomings of "Gold standard" equipment. Our innovative, cost effective techniques for NFC using handheld microscope, USB digital microscope and capillaroscope are ideally suited for clinicians in a busy OPD practice. All this comes at a fraction of a price of NVC and ideal for resource poor countries. The only disadvantage is that it is less than ideal technique for modern research activities.

Group Captain V Vasdev
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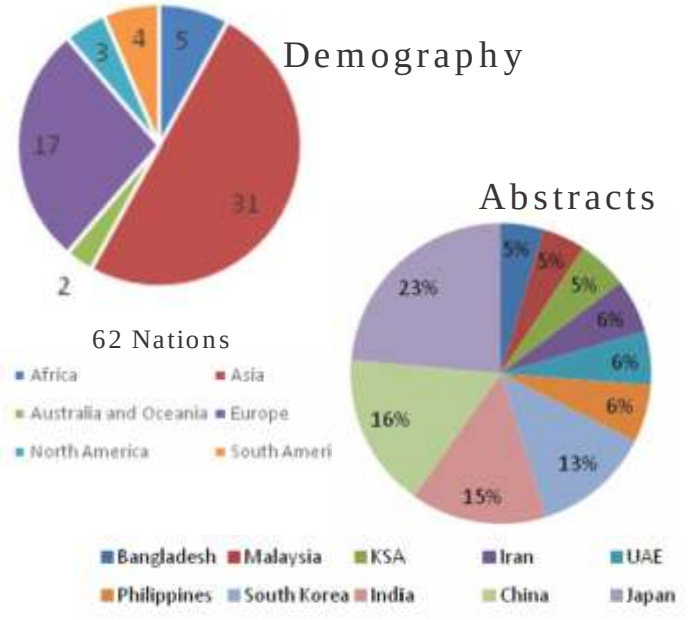
Wonderful APLAR Congress - Dubai 2017



Closing ceremony: VOA first anniversary

EXCERPTS FROM OFFICIAL REPORT FROM LEBANON

The scientific programme comprised of :

- 110 Faculty Members from 23 Different Countries
- 6 Plennary Lectures
- 32 Scientific Sessions
- 6Special Sessions
- 12 Industry Sponsored Symposia
- 40 Oral abstract presentations
- 480 Poster presentations

VIEWPOINT OF A MEDICAL STUDENT:

The 19th edition of APLAR was held at the World Trade Center, Dubai. It kicked off on the 17th of October, 2017 with a Welcome and introductory speeches on the future aims and cares to be offered for rheumatology patients, both young and old, by Chairman Emirates Rheumatology Society, Dr. Waleed AlShehhi and APLAR President Dr. Kazuhiko Yamamoto at the glittering Congress inaugural.

The much awaited Great APLAR debate by Dr. Waleed AlSheehi and Dr. John Cush took place in the post lunch session of the last day. It started as a fun filled session as Dr. AlSheehi started the debate asking us to always remember the man in white and the man with the blue tie by which he projected himself as an equivalent to Mahatma Gandhi and his opponent to Donald Trump !

Dr Yuva from India presented a poster about lupus nephritis along with her guide Dr. Subramanian Nallasivan, and Dr Divya who presented posters about Biosimilars in rheumatology and Clinical profile in Pediatric rheumatology as well.

The grand finale was the closing ceremony marking the 1st anniversary of Voice of Aplar, replete with cake cutting by the Chiarman Dr Waleed AlShehhi and President of APLAR Prof Yamamoto with all the esteemed Ex Com members on the stage... thanks to E-in-C of VOA Prakash Pispati, efficient APLAR and event managers.

PAKISTAN SOCIETY FOR RHEUMATOLOGY

22nd Annual International Conference

April 6th-8th, 2018

Pearl Continental Hotel, Lahore

Organized by

Division of Rheumatology

FMH College of Medicine and Dentistry

Abstract Submission
Last Date: 28 February, 2018
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FIRST ANNOUNCEMENT

19th MSR-SSR Workshops in Rheumatology 2018

ROAD TO SUCCESS in RHEUMATOLOGY

3rd - 5th August 2018

One World Hotel Petaling Jaya, Selangor, Malaysia

Organised by:

MSR Malaysian Society of Rheumatology

Singapore Society of Rheumatology

With participation of

Ministry of Health Malaysia

Online information / registration:
http://msr-ssr2018.com

RA Remission Study

Special Interest Group – RA, APLAR – a brief report

With the advent of newer technologies, Rheumatoid arthritis is being diagnosed earlier and earlier now. There is also the concept of Very Early Rheumatoid Arthritis. The ACR came around with the new criteria in 2010 precisely so that we could diagnose them early. The stakes are high with the availability of biologics, and now, small molecule therapy. These are costly drugs and it only makes economic sense to try and get our subjects of RA into remission, or possibly, cure, as early as possible to justify their use.

There are different definitions of remission ranging from clinical to ultrasound to molecular ones (1). The concept of molecular remission or low disease activity (equivalent to minimal residual disease) comes from our oncology colleagues. With treat to target strategies for RA and the routine use of outcome measures, it should only be a matter of time and picking up early disease that our subjects should all be in remission ! But is it indeed so ? What is happening in the real life world ?

Remission in RA has been studied prospectively and otherwise and there is already published data to it. While most of it comes from the West (2), there is some from the Asian subcontinent also (3,4). At APLAR 2017 this year, in Dubai, we therefore got together to form a 'Special Interest Group' in RA and representatives from different countries from Asia participated in the meet to look into what we could achieve together. As a first, under the guidance of Prof Zhan-Guo Li we will be looking at the cross sectional data on what proportion of our real life OPD subjects of RA are in remission with details of what treatment they have been on and other variables. We plan to collate data from the different countries by the end of March 2018.

As the pressure for pushing our subjects earlier into remission mounts, and rightly so, we hope to capture what we are doing and as a follow up some time in a later study how to improve upon it so that together, as a fraternity, we are able to see our subjects free of their disease, on (remission) or, more optimistically, off (cure) drugs.

As we go about doing this, one is but reminded of the famous saying *'No one can whistle a symphony, it takes a whole orchestra to play it'*.

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"Dear friends and colleagues, Please come to Kaohsiung, Taiwan for the 20th APLAR Congress in 2018. We have prepared a rich program with exciting social events. Kaohsiung's sunshine and cuisine are just amazing. Make sure you will be there."

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