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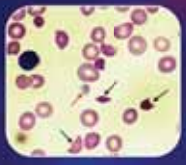

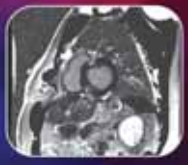

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Mr Shiv Narayan Pareek — *Dr Harjit Singh*

for all the values they taught us

Our Spouse

Shashi Pareek — *Simran Kaur*

for being so tolerant and supportive all the life

Our Children

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Shipra and Prashant — *Praneet*

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And above all

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who taught us Medicine

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Foreword

It is a privilege and honor to write a Foreword for the *Medicine Update 2016*. This voluminous publication of API encompasses the scientific proceedings of APICON 2016. The updates are sought after not only by the delegates but also by the postgraduate students preparing for examinations. Medicine is ever changing. Yesterday's concepts and therapies are old today and today's are tomorrow's old. The explosion of information is mind-boggling. It is impossible for a practicing physician or even an avid reader to keep updated across the breadth of the information on his/her own, despite its accessibility on the Internet, which is a good aid to gather information on a specific area, but not on everything; it is too much time consuming and labor intensive. That is what an expert does for the helpless physicians by capsuling the matter in an article.

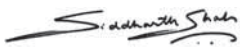
Every physician must have a commitment to the highest standard of excellence in the practice of medicine and in the generation and dissemination of knowledge. In a fast developing medical science, no student or physician owns a library of monograph and journals that covers the whole of internal medicine. The publication of *Medicine Update 2016* is a modest attempt in achieving this goal by including conventional subjects of internal medicine useful to the practicing physicians and postgraduates along with topics which are infrequently discussed and understood but no less important.

Dr Gurpreet S Wander, President Elect and Chairman—Scientific Committee in this *Medicine Update 2016*, has aptly covered the theme of this scientific meeting "Translating Evidence to Practice—Indian Perspective" by emphasizing on the recent development in the field of medicine keeping in mind, Indian context. Besides including basic clinical medicine, he has covered newer modes of therapy and advances in drugs and devices. To make it more comprehensive, he has included sections on journal scan and analysis of articles from prominent journals in the last 3 years. For the postgraduates and clinicians, there are chapters on soft skills including medical ethics, legal aspects, evidence-based medicine and communication skills. Indian and Foreign faculties have been included which would be difficult to access from a single source. Experts from ICMR, Past Presidents of API and Deans of ICP, and AIIMS and PGIMER faculties have contributed their lifetime experience. This has been possible only with untiring day-to-day efforts of Dr Wander (Wonder).

Dr KK Pareek, Dean of Indian College of Physicians has created an excellent CME program which has been covered by the progress in clinical medicine. This is eagerly looked upon by postgraduates of the country. He has covered many topics of internal medicine avoiding overlap of the scientific program. The delegates will look forward to his address on "Professionalism and Communication Skills in Medical Practice". He has also arranged six workshops which will give opportunity to the delegates for interactive learning. This is the first time that the proceedings of the conference are brought out in three volumes and as a joint effort. We look forward to a fruitful CME and Scientific program.

Over the years, I have experienced that the scientific program of APICON is increasingly becoming crowded making it difficult for delegates to attend the topics of their choice. The *Medicine Update* volumes are becoming bigger and bigger. I am sure the future will see publication in electronic format for the conference to really address the latest advances made in the field of medicine. *Medicine Update* serves another important function. It is a source of "who is who" and "of what" information. One knows whom to contact when in distress.

To quote great physician William Osler, "To unrest from nature the secrets which have perplexed philosophers in all ages, to track to their sources the causes of disease, to correlate vast stores of knowledge, that they may be quickly available for the prevention and cure of diseases."



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Preface

We live in a rapidly evolving and changing world today. The pattern, profile and presentation of diseases is changing; the patients expectations, knowledge and perception of the disease and the profession is changing; the clinic and the hospital facade, facilities and functioning is different and so is the clinician's access, aptitude and approach to information. It is only appropriate that in this ever-changing combination of situations and circumstances, we keep updating ourselves.

Let us submit that the practice of medicine is more demanding, under closer audit and with higher expectations. But then we are also empowered with easy access to high-end databases, simplified practice guidelines, more informative and less invasive imaging and laboratory methodologies and thus can serve this expectant, little less tolerant and certainly more informed patient in a better manner than we could earlier.

We, in this profession, need to develop, evolve and then cater according to the changes in the society. The way any person shops, communicates, travels, books tickets and pays bills has changed to his/her convenience and so the medical profession will need to serve this person in a more convenient, comfortable, acceptable and compassionate manner. The experience will have to be made, if not more enjoyable but at least less insipid, formal, scary and sordid.

We need to know how others in our profession are upgrading themselves, making systems amiable and suitable for these changing situations, so that we can also adopt their methods to adapt to this situation. Many earlier impressions, personal experiences and opinions and the dogmas and axioms of yesteryears have been challenged and proven wrong in the present-day era of evidence-based medicine with large registries and double blind randomized and at times even sham controlled clinical trials. The update and the meeting try to fulfill this purpose to an extent.

The superspecialty of Internal Medicine is all embracing and absorbing. We all sub-specialize in some areas, but our patients want care beyond systems since disease do not behave in a segregated and separate manner as we the caregivers do. Hence, the need for all specialists to go through sections in the book and sessions in the meeting of other sub-specialties. Learn and know what is new in other fields and then use for the benefit of the patients. The tubular vision needs to change to a wider thought process, perspective and horizon.

People all across the universe get similar diseases and ailments. However, the environmental differences, dietary changes, differing activity levels and genetic variations make the disease pattern different in various races and areas. We need to imbibe new knowledge with Indian flavor and values. The experts have laid emphasis on Indian pattern and presentations and so, this figures repeatedly in the titles of the topics.

The books (*Progress in Medicine 2016* and *Medicine Update 2016*) appear in three volumes with 30 sections and 377 chapters. It covers recent advances in various sub-specialties of medicine. Special sections on clinical symptoms and signs and investigations have been kept for highlighting importance of good history taking and planning relevant investigations. A section on journal club highlighting last three years' landmark trials. There is resurgence of many infectious diseases in our country which need a fresh look. Noncommunicable diseases are on the rise in our country. Newer drugs, management strategies have been added. Special sections on geriatrics and medical disorders of women are there. Tuberculosis is still a challenge. HIV is fast picking up in our country. Sepsis, especially in ICUs, is an issue and needs to be discussed. Genetics, information technology and molecular biology are fields the present-day physician needs to know. The section on soft skills and social aspects is important, for the developing young clinical physicians.

The Indian physician is moving forward and we will see more and more guidelines by Indian experts for disease management. Our role in international trials is increasing and presence in international meetings is even more. The desire and endeavor of API in empowering Indian physicians is creative, committed, continuous and significant. This book is a small effort in that large motive and movement. It has been a pleasure to coordinate and work on this book. We enjoyed compiling it for you and hope you like it and derive some useful information and future perspective.

KK Pareek **Gurpreet S Wander**

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KK Pareek **Gurpreet S Wander**

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CHAPTER

36 Systemic Vasculitis: An Enigma Unfolding

Aman Sharma

"It's not what you look at that matters, it's what you see"

–Henry David Thoreau

ABSTRACT

Systemic vasculitides are characterized by inflammation of the blood vessel wall. These are group of disorders with various different subtypes. Each subtype has a predilection for involvement of vessels of different sizes, along with a unique pattern of organ involvement. There have been various efforts to classify these according to the size of the involved vessels with the revised Chapel Hill nomenclature system being the most commonly used criteria, but the quest for an "ideal" classification and diagnostic criteria continues. The understanding about pathogenesis of these disorders has increased in recent times along with increase in the therapeutic armamentarium. But these are still an under-recognized entity in India with only few centers reporting about these diseases. The key to the diagnosis is keeping them in the differential diagnosis of all patients, especially those with simultaneous noncontiguous multisystem involvement. For example, possibility of antineutrophil cytoplasm antibody (ANCA) associated vasculitis should be considered in all patients with pulmonary symptoms or upper airway involvement or collapse nasal bridge or asthma and RPRF. Similarly, Takayasu's arteritis should be considered in young hypertensive patients with constitutional symptoms or neurological manifestations. Giant cell arteritis (GCA) is very rare in India. There are various other vasculitic disorders. Once we are able to do the patterns recognition, then diagnosing them is easy and treatment gratifying since many of these disorders if left untreated can be fatal.

INTRODUCTION

Systemic vasculitis as the name suggests, is characterized by the inflammation of the blood vessel wall resulting in organ dysfunction due to ischemia, hemorrhage or necrosis.[1] These are a group of complex and mostly multisystem disorders with each subtype having a unique pattern of clinical presentation. Once a physician is able to do that pattern recognition, the diagnosis is not difficult. The most important factor in making a diagnosis is keeping vasculitis in the differential diagnosis.

EPIDEMIOLOGY

The reliable epidemiological data is not available from India. The most common described vasculitis from India is Takayasu's arteritis. GCA is rare with about 75 total cases published from India.[2] Granulomatosis with polyangiitis (GPA) has been reported more commonly from North India. There are significant geographical variations.

CLASSIFICATION

Though the earliest descriptions of vasculitis were in the 19th century, with clinical description of Henoch-Schönlein purpura (HSP) by Henoch and Schönlein, and pathological description of classical polyarteritis nodosa by Kussumaul and Maier, the understanding increased significantly in the last century leading to improved diagnosis thus resulting in better patient outcomes. Since many of these disorders presented with overlapping clinical features, it was important to have uniform criteria for research. The first effort to classify was done by Zeek who gave the pathological description of some subtypes in 1950s. American College of Rheumatology (ACR) gave its criteria in 1990 and subsequently the Chapel Hill Consensus Conference (CHCC) nomenclature system gave definitions in 1994. A new entity of microscopic polyangiitis (MPA) which was previously clubbed with classical polyarteritis nodosa was recognized by CHCC. However, ANCA was not incorporated in these criteria. Subsequently,

Richard Watt's team gave a consensus methodology algorithm for diagnosis of vasculitis incorporating various soft pointers and ANCA positivity.[3] This helps in decreasing the number of so called unclassifiable vasculitis and even has been validated by us in Indian population.[4] Subsequently, the revised CHCC nomenclature system was proposed in 2012.[5] I would like to clarify that these are all classification criteria or nomenclature systems and not "diagnostic criteria" and patient may still have vasculitis even if he does not fulfill any classification criteria and these disorders may manifest with lesser known or unknown presentations.[6] This clearly shows that the quest for ideal classification and diagnostic criteria is a "work in progress".[7] An ACR European League Against Rheumatism (EULAR) study is presently ongoing to develop the diagnostic criteria in which a few Indian centers are participating and results are likely to come in coming years.[8]

I would briefly describe about the common vasculitic disorders, as details of clinical presentation and management are outside the purview of this chapter and those interested to know more are encouraged to read the textbooks suggested before the references. The citations in the write up are mostly limited to our personal experience.

IMPORTANT INDIVIDUAL VASCULITIC DISORDERS

Large Vessel Vasculitis

Takayasu's arteritis and GCA are the types of large vessel vasculitis.

Takayasu's Arteritis

This is the most commonly described vasculitic disorder from various different centers from all over India. There is inflammation of the aorta and its branches with description of five subtypes depending upon the angiographic pattern of involvement. There is an acute stage in which there is fever, musculoskeletal symptoms and weight loss. Due to nonspecific symptoms, the diagnosis is very rarely made at this stage. Most of the patients are diagnosed at a later stage, when the initial vessel inflammation has resulted in narrowing or stenosis of the vessels. This is a disease of the young individuals, often females, who present with symptoms of ischemia of ocular, cerebral, renovascular, cardiopulmonary and peripheral vascular systems. This may present with absence of upper limb pulses, thus is also known as "pulseless disease". The imaging modalities play an important role in diagnosis. Computed tomography and magnetic resonance angiography can delineate the extent of involvement. Though positron emission tomography-computed tomography (PET-CT) scan is an upcoming modality, increased uptake on PET however is not specific for Takayasu's arteritis and cannot be differentiated from uptake of atherosclerotic involvement of aorta.[9] Unlike various other vasculitic disorders, a biopsy is not feasible to confirm the diagnosis. Steroids and immunosuppression are the backbone of management. It is often difficult to differentiate whether symptoms are due to disease activity or disease damage. Disease activity and damage indices have been developed and validated by Indian Rheumatology Association Vasculitis Group and these can help in this differentiation.

Giant Cell Arteritis

This is a vasculitis disorder of the elderly which is very rare in India.[2] The total number of published cases till now is only 75. This should be considered in an elderly with recent onset headache, jaw claudication, features of polymyalgia rheumatica and visual impairment. There is tenderness over the superficial temporal artery and scalp. Erythrocyte sedimentation rate (ESR) is usually very high along with elevated C-reactive protein (CRP). "Halo sign" on the ultrasound examination of superficial temporal arteries is very sensitive with high specificity. Temporal artery biopsy should be done, but negative result cannot exclude the diagnosis because the involvement is patchy with intervening normal areas. Once a diagnosis is considered, the treatment should be started immediately to prevent the vision loss as once it occurs, it is irreversible. Corticosteroids are the mainstay. Other drugs, like methotrexate may be required in some patients. Anti-interleukin 6 monoclonal antibodies (tocilizumab) have been used in treatment of some patients of large vessel vasculitis recently.

Medium Vessel Vasculitis

Classical polyarteritis nodosa (PAN) and Kawasaki disease are the two medium vessel vasculitides. Kawasaki disease is a vasculitis of childhood and would not be described here.

Classical Polyarteritis Nodosa

This has become a rare disorder after CHCC identified MPA as a different entity in 1994. This is one of the vasculitides with a probable etiology due to causal association with hepatitis B virus infection. This subtype is now clubbed in a different group as vasculitis with a probable etiology in CHCC 2012. The patients can present with constitutional symptoms (fever, weight loss).[10] The other common features are hypertension, neurological involvement especially in the form of mononeuritis multiplex and cardiac involvement in form of cardiomyopathy. These patients can have gastrointestinal involvement presenting with postprandial abdominal angina. Testicular pain is a unique and often ignored presentation of PAN. There may be hepatic, renal or splenic infarction and angiography may show microaneurysms. Five factor score (FFS), based upon various organ system involvement namely

central nervous system (CNS), cardiac, gastrointestinal, proteinuria and renal dysfunction has been used to predict the prognosis in these patients. Once thought to be a "single shot illness", the recent studies suggest that even the patients without poor prognosis factors (FFS = 0) can have relapses as frequently as patients of MPA.

Small Vessel Vasculitis

There are two subgroups:

1. Antineutrophil cytoplasmic antibody (ANCA) associated vasculitis (AAV) and
2. Immune complex vasculitis.

ANCA-associated Vasculitis

There are three types of ANCA associated vasculitides:

1. Granulomatosis with polyangiitis (GPA, previously known as Wegener's granulomatosis)
2. Microscopic polyangiitis
3. Eosinophilic granulomatosis with polyangiitis (EGPA, previously known as Churg-Strauss syndrome).

Granulomatosis with polyangiitis (GPA, previously known as Wegener's granulomatosis): In India, this is the most common AAV, reported predominantly from North part. The dominant clinical presentation is in the form involvement of upper airways, lungs and kidneys. The upper airway involvement and granulomatous involvement of lungs differentiates it from MPA. The upper airway involvement is in the form of nasal crusting, nasal bleed, nasal and oral ulcers, recurrent sinusitis, saddle nose deformity and occasionally bony destruction with sinus formation (Figs 1 to 3). The lung involvement may vary from asymptomatic lung nodules with or without cavitation to life-threatening diffuse alveolar hemorrhage (Figs 4 and 5). The kidney involvement is mostly in the form of pauci-immune

Fig. 2 Collapse of the nasal bridge in GPA

Source: Reproduced from Sharma A. Textbook of Systemic Vasculitis, 1st edition. New Delhi: Jaypee Brothers Medical Publishers (P) Ltd; 2015.

Fig. 3 Ulcer in the buccal cavity in GPA

Source: Reproduced from Sharma A. Textbook of Systemic Vasculitis, 1st edition. New Delhi: Jaypee Brothers Medical Publishers (P) Ltd; 2015.

Fig. 1 Nasal septal perforation in GPA

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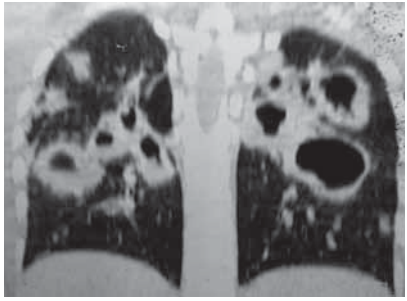

Fig. 4 Multiple cavitating nodules in GPA

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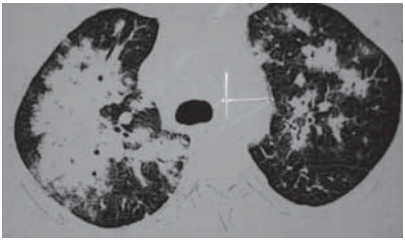

Fig. 5 CT scan showing diffuse alveolar hemorrhage
Source: Reproduced from Sharma A. Textbook of Systemic Vasculitis, 1st edition. New Delhi: Jaypee Brothers Medical Publishers (P) Ltd; 2015.

Fig. 6 Necrotizing scleritis in GPA
Source: Reproduced from Sharma A. Textbook of Systemic Vasculitis, 1st edition. New Delhi: Jaypee Brothers Medical Publishers (P) Ltd; 2015.

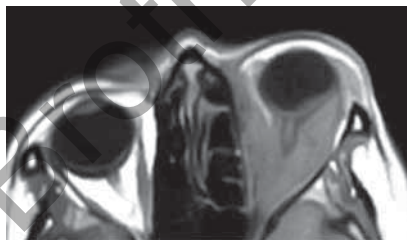

Fig. 7 Pseudotumor of the orbit
Source: Reproduced from Sharma A. Textbook of Systemic Vasculitis, 1st edition. New Delhi: Jaypee Brothers Medical Publishers (P) Ltd; 2015.1.

crescentic glomerulonephritis presenting as rapidly progressive renal failure.[11] The outcomes are dependent upon the histopathological subtype.[12] The presentation based upon ANCA positivity and negativity are also being compared these days.[13] There can be involvement of the eyes in the form of necrotizing scleritis (Fig. 6), peripheral ulcerative keratitis and orbital pseudotumor (Fig. 7). These may result in scleromalacia perforans and corneal melt. Varieties of skin lesions are also seen and these may vary from palpable purpura, nodules, ulcers and cutaneous infarcts. Neurological involvement usually in the form of mononeuritis multiplex and CNS involvement is rare.[14] In the absence of a diagnostic criteria, the diagnosis is based upon clinical setting supported by histopathology and ANCA positivity. The international consensus statement recommends use of both indirect immunofluorescence (IIF) and ELISA as around 5% patients have positive ELISA only while IIF is negative. Though two main antigen targets are (1) proteinase (PR3) and (2) myeloperoxidase (MPO), predominant ANCA positivity in GPA is PR3 and only small subset has MPO positivity. Three patterns are observed of IIF, cytoplasmic ANCA (C-ANCA), perinuclear ANCA (p-ANCA) and atypical ANCA. C-ANCA is the common IIF pattern associated with GPA and it may be p-ANCA in a small subset.

Imaging plays an important role in diagnosis and may also help in suggesting a site for biopsy. The CT PNS may show bone destruction and sinusitis. High-resolution computed tomography (HRCT) may show nodules, GGOs and small fibrotic bands. There have been studies evaluating the role of PET-CT in AAV, but would require further validation before they can be recommended for use in daily clinical practice.

It is important to assess whether the symptoms are due to disease activity or due to disease damage as unnecessary increase in immunosuppression is going to increase the morbidity related to enhanced immunosuppression. There are various validated tools for assessing disease activity and damage in these patients. The approach to treatment is largely same for all AAVs. There is a categorization of disease into subtypes depending on disease severity. These are localized, early systemic (without organ or life-threatening), generalized (renal, other organ threatening and S creatinine < 5.6 mg/dL), severe (renal, other organ failure and S creatinine > 5.6 mg/dL) and refractory (unresponsive to steroids and cyclophosphamide). There is an initial phase of remission induction followed by remission maintenance. Broadly the drug of choice for remission induction is cyclophosphamide unless the disease is very mild or localized where methotrexate may be used. Rituximab is an alternate option in patients with contraindications or toxicity to cyclophosphamide. Once remission is achieved, the remission can be maintained with drugs, like azathioprine, methotrexate and mycophenolate mofetil. Recent studies have shown that low dose rituximab use in maintenance therapy has lower relapse rates as compared to azathioprine. Plasma exchange can be used in patients with RPRF and S creatinine more than 5.6 mg/dL and diffuse alveolar hemorrhage. Treatment of relapsing and refractory disease is a challenge.

Microscopic polyangiitis: Microscopic polyangiitis was recognized as a distinct entity only at the CHCC meeting in 1994. This was considered to be a part of PAN before that. This is one of the three AAVs. This is characterized by necrotizing vasculitis, with no immune deposits and no granuloma formation. There is no upper airway involvement.

The clinical presentation is in the form of constitutional symptoms and renal involvement. The renal involvement is in the form of rapidly progressive glomerulonephritis (RPGN). There is microscopic hematuria and proteinuria. Kidney biopsy shows pauci-immune glomerulonephritis. There may be oliguria or anuria and need for dialysis at presentation. There can be renal and pulmonary presenting as renopulmonary syndrome. Pulmonary involvement may be in the form of alveolar hemorrhage. Pulmonary fibrosis may be seen in about one-third of the patients. Mononeuritis multiplex is the dominant neurological manifestation and is seen commonly.[11] The other manifestations may be in the form of skin rash, like purpura, ulcers or gangrene; ocular involvement, like conjunctivitis, episcleritis, scleritis or retinal vasculitis. Heart and gastrointestinal tract are involved rarely.

Dominant ANCA pattern seen in these patients is p-ANCA and MPO. C-ANCA and PR3 positivity is uncommon. Anti-lysosomal-associated membrane protein 2 (LAMP2) is proposed to be pathogenic and its serial monitoring may be of value, but as of now, it is of academic interest only. The treatment is on the lines of other AAVs as described above in description of GPA.

Eosinophilic granulomatosis with polyangiitis: This was formerly known as Churg-Strauss syndrome. Though this is clubbed in the group of AAVs, the ANCA positivity is seen in only 30–40% patients. Even the main manifestations, like eosinophilia and asthma and, main pathogenic mechanisms differ from MPA and GPA. This is one of the rarest forms of AAV. The diagnostic criteria given by Lanham in 1980 were too broad and nonspecific and are not used in day-to-day practice. There are three prototype phases of this disease. There is a prodromal phase of allergic manifestations and asthma. The second phase is characterized by blood eosinophilia (> 1,500/mm^3) and tissue eosinophilia and the third phase of vasculitis. The three phases may not occur sequentially and may even overlap. The common clinical manifestations are in the form of constitutional symptoms, rhinitis (Fig. 8), polyposis and asthma. There are lung infiltrates. Nodules and alveolar hemorrhage is rare (Fig. 9). Skin and peripheral nervous system involvement is also common. The skin manifestations may vary from palpable purpura, cutaneous nodules, livedo reticularis, ulcerations and infarcts (Figs 10 and 11). Peripheral nervous system involvement is common. Cardiac involvement may occur in 10–50% patients. Cardiomyopathy has been associated with increased mortality in these patients. Other uncommon manifestations can be gastrointestinal involvement in form of abdominal pain, diarrhea and vomiting, eosinophilic granulomatous colitis or mesenteric vasculitis; renal involvement and ocular involvement.

The three main features on histopathology include 1) vessel wall infiltration (mainly with eosinophil's) with fibrinoid necrosis, 2) extravascular eosinophilic infiltrates and 3) extravascular necrotizing granulomas. The diagnosis is based upon suggestive clinical finding supported by histopathology wherever possible. The treatment depends upon type and severity of the disease. Immunosuppression and antiasthma measure are the mainstay of treatment.

There are various other subgroups of vasculitides, but description of all of those subtypes is outside the preview of this chapter and these would only be enumerated here.

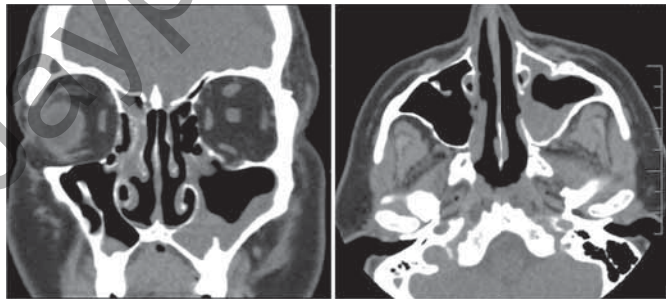

Fig. 8 Sinus CT of patient with EGPA showing bilateral nonerosive sinusitis

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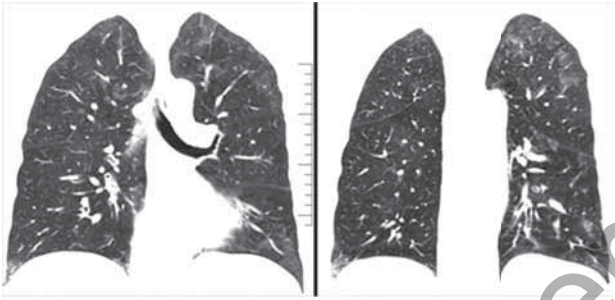

Fig. 9 Chest CT with EGPA showing multiple patchy ground glass opacities
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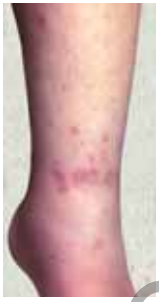

Fig. 10 Purpuric macular skin rash in a patient with EGPA
Source: Reproduced from Sharma A. Textbook of Systemic Vasculitis, 1st edition. New Delhi: Jaypee Brothers Medical Publishers (P) Ltd; 2015.

Fig. 11 Erythematous skin lesions of the scalp in patients with EGPA
Source: Reproduced from Sharma A. Textbook of Systemic Vasculitis, 1st edition. New Delhi: Jaypee Brothers Medical Publishers (P) Ltd; 2015.

Immune Complex Small Vessel Vasculitis

The various immune complex vasculitides are anti-GBM disease, cryoglobulinemic vasculitis, IgA vasculitis (previously Henoch-Schönlein purpura), hypocomplementemic urticarial vasculitis.

Variable Vessel Vasculitis

The various subtypes are Behçet's disease and Cogan syndrome.

Single Organ Vasculitis

The various subtypes are cutaneous leukocytoclastic angiitis, cutaneous arteritis, primary angiitis of CNS, isolated arteritis and other single organ vasculitides.

Vasculitis Associated with Systemic Disease

These are vasculitic disorders associated with systemic diseases, like lupus vasculitis, rheumatoid vasculitis, sarcoid vasculitis and vasculitis associated with other connective tissue diseases.

Vasculitis Associated with Probable Etiology

Various vasculitic disorders with probable etiology are hepatitis C virus (HCV) associated cryoglobulinemic vasculitis, hepatitis B virus (HBV) associated polyarteritis nodosa,[15] syphilis associated aortitis, drug associated immune complex vasculitis, drug associated ANCA associated vasculitis and cancer associated vasculitis.

CONCLUSION

To conclude, I would like to say that these complex disorders can have myriad presentations and there can be also various mimics of these disorders. Unlike the commonly held myth, these are seen in India, but there is a need to increase the awareness and recording outcomes in a structured way in India.[16]

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