



Volume 3



Volume 2



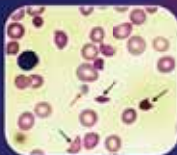
Volume 1



# Progress in Medicine 2016

(Medicine Update 2016)

Volume 26 2016



K K Pareek • Gurpreet S Wander



# Progress in Medicine 2016

(Medicine Update 2016)



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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.

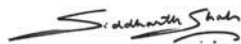
Every physician must have a commitment to the highest standard of excellence in the practice of medicine and in the generation and dissemination of knowledge. In a fast developing medical science, no student or physician owns a library of monograph and journals that covers the whole of internal medicine. 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 unrust 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

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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.



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# Acknowledgments

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The guidance of our mentor Dr Siddharth N Shah is always simple, spiritual and supportive. His commitment to academics and passion to achieve perfection in scientific pursuits whether it is sessions, books or guidelines has been guiding us to move a few steps in that path of his. Professor YP Munjal is responsible for the unification of the *Progress in Medicine 2016* and the *Medicine Update 2016* since it is originally his advice to us to bring this in collaboration for the convenience of the delegates and other physicians especially the postgraduate students. Dr Amal K Banerjee and Dr VK Chopra are responsible for the elaborate and extensive cardiology coverage. Professor YK Chawla inspired a big team of consultants from our Alma mater PGIMER, Chandigarh, to contribute for this book. Sanjay Jain my good friend from PGI coordinated this.

Professor SK Sharma, Head, Department of Medicine, AIIMS, has been kind to respond positively to all our requests. We have no words to express our gratitude to him since he has arranged talks, CPC, clinical case presentations and workshops. His team has contributed in all these key knowledge sharing areas. Jyotirmoy Pal and Banshi Saboo helped in framing the program. It has been a great learning experience to work with them.

Our past presidents and senior API functionaries including Sandhya Kamath, Shashank Joshi, A Muruganathan, Milind Nadkar and Rajesh Upadhyay guided us in various stages for bringing out this book. The future torch bearers including BR Bansode, Rohini Handa and Mangesh Tiwaskar helped us knit the knots.

The foreign faculty from US, Australia and England was kind to contribute the chapters in time. My two teachers in cardiology Dr Navin C Nanda and Dr Inder S Anand have always been encouraging and inspiring. My friends Prakash P Punjabi, Gursharan Singh and Jay Chatterjee from England are responsible for a significant contribution from England to apprise us of the recent developments.

The APICON Ludhiana 2014 team has been helpful, scientifically this time, which includes my good friends Rajinder Bansal; Narender Pal Jain; our Principal Sandeep Puri, Head, Department of Medicine; Dinesh Gupta; Harminder S Pannu; Mohanjeet Kaur, and Vitull K Gupta.

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The team from Kota has helped Dr KK Pareek in arranging the CME and the *Progress in Medicine 2016* (Part I of this three volumes collective output). The contribution of GD Ramchandani and Girish Mathur cannot be expressed in words. In fact, SK Goyal, Kota; RK Goyal, Ajmer; S Maheshwari, Ajmer; and LA Gauri, Bikaner, India, are responsible for simplifying many issues in the compilation of this book by their scientific contribution.

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## VOLUME 1: PROGRESS IN MEDICINE 2016

### SECTION 1

### DIABETES

- 1. Can We Change the Natural History of Type 2 Diabetes Mellitus?** 3  
*V Mohan, A Amutha*
- 2. Diabetes is a Vasculopathy: Not Lone Endocrinopathy** 8  
*Rajeev Chawla*
- 3. Heart Disease in Diabetes: Indian Pattern** 14  
*YP Munjal, Bhagya N Pandit, Anmol S Pannu*
- 4. Childhood Onset Diabetes** 18  
*Ranjit Unnikrishnan*
- 5. Brittle Diabetes: Current Approach** 21  
*SV Madhu, Sundaram Gopalakrishnan*
- 6. Diabetes Educators: A New Paradigm for Management of Indian Diabetics** 24  
*Shilpa Joshi*
- 7. Home Monitoring in Diabetes** 27  
*RK Goyal*
- 8. Going Beyond HbA1c: Ambulatory Glucose Profiling** 29  
*Prasanna Kumar KM*
- 9. Diet and Diabetes** 32  
*Pramod K Sinha, Mandip S Bhangoo*
- 10. Sulfonylureas: A Reappraisal of their use in Clinical Care** 37  
*Krishna G Seshadri*
- 11. Role of Newer Antidiabetic Agents in Indian Diabetic Patients** 40  
*Anuj Maheshwari*
- 12. Glycemic Variability and Role of Alpha-glucosidase Inhibitors** 45  
*Barshi Saboo, Ravinder Garg, Geeti Puri Arora*
- 13. Dipeptidyl Peptidase-4 Inhibitors** 49  
*Ajay Kumar*
- 14. Newer Hypoglycemic Agents for Type 2 Diabetes** 52  
*Harbir K Rao, Rajinder S Gupta, Simar Rajan Singh*
- 15. When to Initiate and how to Intensify Insulin Therapy in Type 2 Diabetes** 56  
*Sanjeev Phatak*
- 16. Peripheral Neuropathy in Diabetes** 62  
*R Rajasekar, M Nataraj*
- 17. Diabetic Foot** 65  
*Vijay Viswanathan, Anitha Rani*
- 18. Management of Hyperglycemia in Critical Care Setting** 67  
*Apurba Mukherjee, Giridhari Kar*
- 19. Prevalence of Noncommunicable Diseases in India: Results from the ICMR—INDIAB Study (Phase I)** 72  
*Ranjit Mohan Anjana, Tanvir Kaur, Rajendra Pradeepa*

20.	<b>Mobile Technologies in Managing Diabetes</b> <i>Viral N Shah, Narendral Jain</i>	77
21.	<b>Evolution of Path-breaking Technologies in Diabetes</b> <i>Joathydev Kesavadev</i>	83
22.	<b>Saroglitazar, a Novel Agent: Role in Diabetic Dyslipidemia</b> <i>Bipin Kumar Sethi, V Sri Nagesh, Ayesha Vaseen, Jayant Kelwade</i>	88
23.	<b>Bionic Pancreas</b> <i>Shalini Jaggi, Rajeev Chawla, Aastha Chawla</i>	91

## SECTION 2 RHEUMATOLOGY

24.	<b>Laboratory Investigations and Interpretations in Rheumatic Diseases</b> <i>Paramjeet Singh, Ghan Shyam Pangtey, Amit Kansal</i>	97
25.	<b>Hyperuricemia: Relevance in Clinical Practice</b> <i>Vikram Londhey, Tarun Satija</i>	103
26.	<b>Asymptomatic Hyperuricemia: Not so Benign</b> <i>Renu Saigal</i>	107
27.	<b>Recent Guidelines for Management of Gout</b> <i>URK Rao</i>	113
28.	<b>Rheumatoid Arthritis: Recent Advances and an Indian Perspective</b> <i>Milind Y Nadkar, Abhinav Anand</i>	116
29.	<b>Biologics In Rheumatoid Arthritis</b> <i>Gauri Liyakat Ali, Sharanbasu, Asim Khan, Ambreen Liyakat</i>	119
30.	<b>Low-dose Methotrexate: A Widely Misunderstood Drug</b> <i>Anand N Malaviya</i>	128
31.	<b>Art and Science of Using Corticosteroids in Rheumatic Disease</b> <i>Rohini Handa</i>	137
32.	<b>Changing Concepts in Spondyloarthropathy</b> <i>Kishore Babu, B Lakshmi Narasimha Rao, G Narasimulu</i>	141
33.	<b>Evidence-based Medical Management of Osteoarthritis</b> <i>RN Sarkar, K Bhattacharyya</i>	144
34.	<b>Lupus Nephritis: The Current Scenario</b> <i>Siddharth Jain, Uma Kumar</i>	148
35.	<b>Vasculitis versus Pseudovasculitis: A Challenge to Clinicians</b> <i>Arup Kumar Kundu, Anupam Maity</i>	155
36.	<b>Systemic Vasculitis: An Enigma Unfolding</b> <i>Aman Sharma</i>	160
37.	<b>Immunoglobulin G4-related Disease: Cause or Effect?</b> <i>Harvinder Luthra</i>	167
38.	<b>Ocular Inflammatory Diseases: What a Physician should Know?</b> <i>Yojana Gokhale, Eesha Shukla, Akruati Desai</i>	172
39.	<b>Biosimilars: The Future of Biological Drugs</b> <i>Praveen Hissaria, Manisha Ginde</i>	179

## SECTION 3 ONCOLOGY

40.	<b>New Diagnostics and Screening Methods for Cancer</b> <i>TVSVGK Tilak, Amit Dhiman</i>	189
41.	<b>Immunological Aspects of Cancer</b> <i>Jayanta Chatterjee, Sadaf Ghaem-Maghami</i>	194

42.	<b>Management of Infections in Immunocompromised Patients</b> <i>Sanjeet Dadwal</i>	199
43.	<b>Chronic Myeloid Leukemia: An Overview</b> <i>Hemant Malhotra</i>	204
44.	<b>An Approach to a Solitary Pulmonary Nodule</b> <i>Madhuchanda Kar, Angira Dasgupta</i>	210

#### SECTION 4 NEPHROLOGY

45.	<b>Prevention and Management of Acute Kidney Injury in Critical Ill</b> <i>NP Singh, Anish Kumar</i>	219
46.	<b>Cardiorenal Syndrome: An Overview</b> <i>Ananda Bagchi, Avijit Bhattacharya, Aradhya Sekhar Bagchi</i>	224
47.	<b>Hypertension and the Kidney</b> <i>MK Mani</i>	229
48.	<b>How to Handle the CKD Epidemic in India?</b> <i>PP Varma, R Nair</i>	234
49.	<b>Practical Approach to Glomerular Diseases</b> <i>Ratan Jha, Kiran Chandra Patro, Shekhar Shiradhonkar</i>	237
50.	<b>Diabetic Kidney Disease: Indian Perspective</b> <i>Arvind Gupta, Rajeev Gupta</i>	243
51.	<b>Management of Hyperkalemia in Chronic Kidney Disease</b> <i>Asif Hasan, Muhammad Uwais Ashraf, Sekhar Chakravarty</i>	247
52.	<b>Recurrent Urinary Tract Infections in the Elderly</b> <i>Y Sathyaranayana Raju, Y Rahul</i>	250
53.	<b>Frontier in Nephrology: Onconephrology</b> <i>Tejas Patel</i>	253

#### SECTION 5 HEMATOLOGY

54.	<b>Anemia: A Clinical Approach</b> <i>PS Ghalaut, Anubha Garg, Arvind Chahal, Varun Yadav</i>	261
55.	<b>Total and Differential Leukocyte Count: Changes in Disease States</b> <i>Rumma Manchanda</i>	265
56.	<b>Aplastic Anemia: Current Concepts in Management</b> <i>Velu Nair</i>	268
57.	<b>Thrombocytopenia: Rational Approach</b> <i>SV Ramanamurthy, DJK Chakravarty</i>	276
58.	<b>Interpretation of CBC and Thrombocytopenia with Special Reference to ITP</b> <i>Rekha Bhat, Swati Pai</i>	282
59.	<b>Heparin-induced Thrombocytopenia</b> <i>BL Bhardwaj, Harnoor S Bhardwaj, Kanchan Saini</i>	286
60.	<b>Thrombophilia: When and how to Investigate/Manage?</b> <i>Charu Jani, Kanjaksha Ghosh</i>	288
61.	<b>Blood Component Therapy: Which, when and how much?</b> <i>Mathew Thomas</i>	294
62.	<b>Classification of RBC Disorders</b> <i>Sudhir Mehta, Shaurya Mehta</i>	297

**SECTION 6****ENDOCRINOLOGY**

- |     |  |     |
|-----|--|-----|
| 63. | <b>Subclinical Hypothyroidism: To Treat or Not to Treat</b><br><i>Ankit Shrivastav, Abhijit Swami</i>                            | 303 |
| 64. | <b>Subclinical Thyroid Disorder: To Treat or Not to Treat</b><br><i>DC Sharma</i>  | 306 |
| 65. | <b>Primary Hyperparathyroidism (Indian Scenario): Trends Over Past Two Decades</b><br><i>Muthukrishnan J, Naveen Mittal</i>      | 310 |
| 66. | <b>Polycystic Ovary Syndrome with Physicians</b><br><i>Minal Mohit, Sanjiv Maheshwari</i>  | 315 |
| 67. | <b>Polycystic Ovarian Syndrome</b><br><i>Samar Banerjee, Suman Puri</i>  | 319 |
| 68. | <b>Hyperprolactinemia: When to Suspect and How to Manage?</b><br><i>Piya Ballani Thakkar</i>                                     | 326 |
| 69. | <b>Andropause, Prevalence and Presentation: Role of Testosterone in Clinical Practice</b><br><i>Rakesh Sahay, Sandeep Sharma</i> | 330 |
| 70. | <b>Male and Female Sexual Dysfunctions in India</b><br><i>Deepak Jumani, Shweta Gupta</i>  | 334 |

**SECTION 7****CLINICAL SYMPTOMS AND SIGNS**

- |     |   |     |
|-----|---|-----|
| 71. | <b>Constipation: Evaluation and Management</b><br><i>VG Mohan Prasad</i>  | 343 |
| 72. | <b>Constipation in Clinical Practice: Causes and Management</b><br><i>Tejinder Singh, Praneet Wander</i>                                | 350 |
| 73. | <b>Approach to a Patient with Chronic Diarrhea</b><br><i>Varun Mehta, Govind K Makharia</i>   | 353 |
| 74. | <b>Vertigo: Clinical Approach and Management</b><br><i>Mohan Kameswaran, Sunil Goyal</i>  | 359 |
| 75. | <b>Hemoptysis: Diagnostic and Therapeutic Approach</b><br><i>Jai Kishan Karahyla</i>  | 364 |
| 76. | <b>Dry Cough: Presentation, Causes and Management Algorithm</b><br><i>AA Mahashur, R Banka</i>  | 372 |
| 77. | <b>Noncardiac Chest Pain</b><br><i>Rakesh Kalapala, Tarun Aggarwal</i>  | 377 |
| 78. | <b>Headache in Medicine OPD</b><br><i>Sanjay Prakash, K Ravishankar</i>   | 381 |
| 79. | <b>A Case-based Approach to a Dizzy Patient</b><br><i>Salil Gupta, Deepak Goel</i>  | 386 |
| 80. | <b>Diplopia: Clinical Approach and Management</b><br><i>Vivek Lal, Monika Singla</i>  | 390 |
| 81. | <b>Insomnia: A Disorder or a Symptom?</b><br><i>Manvir Bhatia, Abdul Muniem, BS Bal</i>   | 396 |
| 82. | <b>Cutaneous Manifestations of Systemic Diseases: Some Spotters</b><br><i>Eva Tsele, Sangeeta Punjabi</i>                               | 403 |
| 83. | <b>Physical Signs in Chest Examination: Clinical Relevance in Present Day</b><br><i>Basanta Hazarika, Santokh Singh, Nayanjyoti Bez</i> | 410 |

84.	Some Lessons for Internists on Bedside Clinical Cardiology for Valvular Heart Disease <i>Jaganmohan A Tharakan, SP Abhilash</i>	415
85.	Vignettes in Respiratory Medicine: Some Interesting Cases with Clinical Lessons <i>DP Singh, Sudhir Kumar</i>	422
86.	Pearls in Cardiology: Some Interesting Cases with Lessons <i>Neeraj Pandit, Girish Narayan Mishra</i>	426

## SECTION 8 INVESTIGATIONS

87.	Procalcitonin Hope or Hype <i>Sameer Gulati, Bhupendra Gupta</i>	433
88.	A Clinical Utility of Serum Galactomannan Test <i>Shankha Shubhra Sen, Apu Adhikary</i>	437
89.	Polymerase Chain Reaction: Where is it Relevant as a Diagnostic Test? <i>Gandharba Ray, L Ravi Kumar, Deepinder Chhina</i>	440
90.	Clinical Utility of Getting CRP done in Infections, Inflammation and Healthy High-risk Individuals <i>Nitin Sinha</i>	444
91.	Diagnostic Value of Sputum Induction in Respiratory Disorders: Present Status, Future Course <i>RS Bhatia, Rajbir Singh, Akash Batta</i>	448
92.	Urinary Albumin and Creatinine Ratio versus 24-hour Urinary Protein Estimation <i>Vivek Kumar, Harbir S Kohli</i>	451
93.	Role of Tumor Markers in Clinical Practice <i>Gaurav Prakash, Anupriya Kaur, Sanjay Jain</i>	454
94.	Fibroscan: Potential Role in Clinical Practice <i>GS Lamba, Monika Jain, RM Chhabra</i>	459
95.	Antithyroid Peroxidase: Clinical Utility <i>Hemant Thacker</i>	463
96.	D-dimer: Where to Send and how to Interpret? <i>Sudhir Bhandari</i>	466
97.	Clinical Utility of Serum Ferritin <i>Asha N Shah</i>	469
98.	Chest Radiography (X-ray and CT Scan) for Internists <i>Udas Chandra Ghosh, Soma Saha, Kavita Saggar, Rajdeep Singh</i>	472
99.	From Basic Principles in Echocardiography to Newer Modalities <i>HP Singh, AP Singh, Gursewak S Gill</i>	479
100.	Role of Stress ECG Testing in Diagnosis and Prognostication of Coronary Artery Disease <i>SB Gupta, Sudhir Verma, Samman Verma</i>	485
101.	Role of Echocardiography for Assessment of LV Function <i>Rakesh Gupta, Parth Sthapak, A Sankaranarayanan, Garima Arora</i>	491
102.	Echocardiographic Evaluation for Valvular Heart Disease: A Guide for the Need of Intervention/Surgery <i>Shravan NS Turaga, Saima H Khan, Rohit Tandon, Navin C Nanda</i>	501
103.	Echocardiography in Ischemic Heart Disease: Clinical Utility <i>SK Parashar, Mahmoud Elsayed, Jugal R Chahwala, Parth K Parikh</i>	512
104.	Myocardial Viability Testing in Patients with IHD: Test Utility and Limitations <i>Arif Wahab, Vipul N Roy, Naresh Goyal</i>	519



## VOLUME 2: MEDICINE UPDATE 2016-1

## SECTION 9 FOOD NUTRITION AND LIFESTYLE

- |   |     |
|---|-----|
| 105. Diet and Healthy Aging   | 527 |
| <i>Sonia Arora, Krishma Virk, Vitull K Gupta</i>  |     |
| 106. Nutritional Supplements in India: How Safe are they?                               | 534 |
| <i>Ruby Sound, Shashank R Joshi</i>   |     |
| 107. B <sub>12</sub> Deficiency in India: Clinical Relevance and Management             | 539 |
| <i>Pradip Joshi, KS Kajal</i>   |     |
| 108. Adulteration and Safety Issues in Packaged Food Industry: The Unpalatable Truth    | 545 |
| <i>Sandhya Kamath</i>   |     |
| 109. Food Allergies   | 550 |
| <i>Maria A Lagarde, Yogesh Govil</i>  |     |
| 110. Science of Happiness and Spiritualism  | 554 |
| <i>Ashok Panagariya</i>   |     |
| 111. Sahaja Yoga Meditation: A Simple, Fast Way to Reduce Stress and Enhance Well-being | 559 |
| <i>Sandeep Rai, Madhur Rai</i>  |     |
| 112. Role of Yoga and Meditation for Lifestyle Diseases                                 | 561 |
| <i>Mohit D Gupta, Girish MP, Parth Sthapak</i>  |     |

## SECTION 10 METABOLIC

- |   |     |
|---|-----|
| 113. Metabolic Syndrome and Cardiovascular Risk: Indian Perspective                         | 571 |
| <i>Gurmukh S Sainani, Namrata Shindekar</i>   |     |
| 114. Recent Lipid Guidelines: Lessons for us  | 577 |
| <i>Mohan Bhargava, Ravi Prakash, Prachal Bhargava</i>                                       |     |
| 115. New Guidelines for Management of Dyslipidemia: Are they Relevant to Indian Population? | 581 |
| <i>JPS Sawhney, Saurabh Bagga, Simran Sawhney, Dapo Iluyomade</i>                           |     |
| 116. Statins and Cancer Prevention  | 586 |
| <i>Vikas Khurana, Shikhar Khurana</i>   |     |
| 117. Novel Lipid-lowering Therapeutics  | 589 |
| <i>SN Narasingan</i>  |     |
| 118. PCSK9 Inhibitor: A New Molecule for Dyslipidemia                                       | 595 |
| <i>Veerappa Kothiwale, C Prudhvi</i>  |     |
| 119. PCSK9 Inhibitors: Potential as Future Lipid-lowering Agents!                           | 599 |
| <i>PC Manoria, Pankaj Manoria, Piyush Manoria, SK Parashar</i>                              |     |
| 120. Osteoporosis in India: Prevention and Management                                       | 605 |
| <i>Satyabrata Ganguly, Bidyut K Das</i>   |     |
| 121. Obesity in India: Prevalence, Implications and Management                              | 610 |
| <i>Nihar Mehta, Vishal Vanani</i>   |     |
| 122. Obesity Determinants: Genetics or Epigenetics  | 616 |
| <i>Soumitra Ghosh</i>   |     |
| 123. A Decade of Experience in Bariatric Surgery in Indian Patients                         | 619 |
| <i>Shashank Shah, Poonam Shah, Ashish Ahuja</i>   |     |

## SECTION 11 HYPERTENSION

- |   |     |
|---|-----|
| 124. Role of Inflammation in Hypertension: Its Therapeutic Implications | 627 |
| <i>Sanjay Jain, SK Pondaiah</i>   |     |
| 125. Blood Pressure Variability: Importance in Management of CVD        | 630 |
| <i>M Chennaiappan</i>   |     |

126. Home Blood Pressure Monitoring: Clinical Utility in India <i>A Muruganathan</i>	636
127. Ambulatory Blood Pressure Monitoring <i>Mangesh Tiwaskar, Shilpa Tiwaskar, Parveen Dhingra</i>	641
128. Treatment of Hypertension in 2016: Role of Beta-blockers <i>Satya Narayan Routray, Chander Bowry, Dipak Ranjan Das</i>	648
129. Treatment of Hypertension in 2016: Role of Diuretics <i>T Govindan Unni</i>	651
130. Management of Hypertension in 2016: Role of Calcium Channel Blockers <i>Kamlesh Tewary, AK Das</i>	656
131. Treatment of Hypertension in 2016: Role of ACE-Inhibitors <i>Abhay N Rai, Mritunjay K Singh</i>	660
132. Treatment of Hypertension in 2016: Role of Angiotensin Receptor Blockers <i>BR Bansode</i>	664
133. Rational Combination Therapies for Managing Hypertension <i>NK Soni, VB Jindal</i>	667
134. Gazing the Crystal Ball: Future of Hypertension <i>BB Thakur, Smita Thakur</i>	670
135. Indian Hypertension Guidelines: Evolution and Key Lessons <i>Siddharth Shah</i>	677

**SECTION 12****INFECTIOUS DISEASES****Malaria**

136. Recent Change in the Behavior of <i>Plasmodium</i> <i>Debasis Chakrabarti, Swaroop K Baruah</i>	683
137. Changing Clinical Scenario of Malaria <i>Amar Pazare</i>	688
138. Current Guidelines on Management of Malaria in India <i>Manoj K Mohapatra, Pradip K Mohanty</i>	690

**Dengue**

139. Clinical Presentation and Systemic Manifestations of Dengue Fever <i>RR Chaudhary, KK Dwivedi, Narinder Sharma</i>	697
140. Dengue Fever: Prevalence, Profile and Management in India <i>Manab K Ghosh, Bibhuti Saha, Sandeep Puri</i>	701

**Typhoid**

141. Typhoid Fever: Presentation and Management Strategies in 2016 <i>Uttam K Paul, Smarajit Banik</i>	706
142. Typhoid Fever: Overview with Recent Advances <i>Surendra Daga</i>	712

**Swine Flu**

143. Clinical Presentation of Swine Flu: An Overview <i>Sanjiv Maheshwari, Swapnil Samadhiya</i>	715
144. H1N1 (Swine Flu) in India: Issues in Diagnosis, Management and Prophylaxis <i>Falguni S Parikh, Sweta Shah</i>	718

**Encephalitis**

145. Cryptococcal Meningitis <i>Dolanchampa Modak</i>	722
146. Japanese Encephalitis <i>Bhupen Barman, KG Lynrah, Pranjal Phukan</i>	726

147. Acute Febrile Encephalopathy <i>Trupti H Trivedi, U Sundar</i>	730
148. Rickettsial Fever in India (Scrub Typhus) <i>Shantanu K Kar, Bhagirathi Dwivedi, Jayanta K Panda</i>	737
149. Scrub Typhus: Prevalence and Profile in India <i>Prasanta K Bhattacharya</i>	744
150. Overview of Ebola Virus <i>K Shankar, K Taruni</i>	748
151. Kala-azar: Prevalence, Presentation and Management in India <i>Shyam Sundar, Jaya Chakravarty</i>	752
152. Brucellosis <i>Rajiv Raina, JPS Gill</i>	756
153. Systemic Antifungal Treatment: Current Principles and Practice <i>Ajai K Garg, AK Agarwal, GD Sharma</i>	761
154. Hemophagocytic Lymphohistiocytosis <i>Dinesh Gupta, Ram Singh, Anita Tahlan</i>	768
155. Neglected Tropical Diseases: Burden in Asian Countries <i>Partha S Karmakar, Lokeshwar Singh</i>	773
156. Adult Vaccination in India <i>RK Singal, Vivek Pal Singh</i>	778

### SECTION 13 SEPSIS AND ANTIBIOTICS

157. Clinical Approach to a Patient with Sterile Pyuria <i>Mohanjeet Kaur, Mary John</i>	787
158. MRSA, VRE, NDM: Prevalence, Prevention and Management in Indian Scenario <i>Saurabh Srivastava, Dheerendra Kuber, Bornali Borua</i>	792
159. Diabetes and Sepsis: Indian Scenario <i>Jayant K Panda, Manoranjan Behera</i>	798
160. Evaluation and Management of Severe Sepsis and Septicemic Shock <i>MPS Chawla, Amit Bery</i>	805
161. Current Management of Sepsis <i>Narendra Rungto, Pramod Sarwa</i>	813
162. Antimicrobial Stewardship in a Resource Limited Setting Like India <i>Akashdeep Singh, Gurpreet S Wander, Sangram Singh</i>	817
163. Newer Antibiotics in India: Dosages and Routes of Administration <i>Dinesh Khullar, Rahul Grover</i>	820
164. Inhaled Antibiotics <i>Vasu Vardhan, Anil Abbot, Puneet Saxena</i>	825
165. Penems: Where do they Stand? <i>Nitin Abhyankar, Sandeep Kaushal</i>	829
166. War against Antibiotic Resistance: What can be Done <i>Jyoti Goyal, RK Mani</i>	833

### SECTION 14 TOXICOLOGY

167. Organophosphorus Poisoning in India <i>Virendra Kumar Goyal</i>	841
168. Health Effects of Toxic Gases in India <i>Gurusaran Sidhu, Ankit Walia, Abhitej Sidhu</i>	845

169. Poisonings and Extracorporeal Therapy: Role of a Nephrologist <i>Manisha Sahay</i>	851
170. Current Concepts in Management of Snakebite: A Case-based Discussion <i>Vinay Rampal, V Palaniappan</i>	857
171. Aluminum Phosphide Poisoning in Northern Central India <i>SB Siwach</i>	862
172. Hair Dye Poisoning <i>Neelima Singh, Sushma Trikha, Priyam Singh</i>	866
173. Corrosive Poisoning: Prevalence and Management <i>Yalaka Rami Reddy, Ajay Kumar, Rakesh Kochhar</i>	868

## SECTION 15 HUMAN IMMUNODIFICIENCY VIRUS

174. HIV Infected—Where do I go? Services for HIV Infected Persons in the Country <i>BB Rewari, Vimlesh Purohit</i>	875
175. HIV-TB: Deadly Synergy <i>PK Aggrawal</i>	880
176. HIV and Tuberculosis <i>Sanjeev Sinha, BB Rewari</i>	884
177. HIV in Critical Care <i>Atul K Patel, Sanjay Pujari</i>	890
178. Combination Antiretroviral Therapy <i>BB Rewari, Sudhir Chawla</i>	893
179. National ART Guidelines <i>Subhasish K Guha, Dipanjan Bandyopadhyay</i>	899
180. Nutrition in HIV/AIDS: A Clinician's Perspective <i>S Bhagyabati Devi, T Jeeten K Singh, Ksh Birendra Singh</i>	905

## SECTION 16 TUBERCULOSIS

181. Tuberculosis Mimics <i>Alaka Deshpande</i>	913
182. Lymph Node Tuberculosis: Conflicts and Controversies <i>Jalil Chowdhury, Sandeep Chhabra, Aminder Singh</i>	918
183. Xpert MTB/RIF in the Diagnosis of Tuberculosis: Challenging the Gold Standard <i>Abhenil Mittal, Abhishek Sharma, Mikashmi Kohli, SK Sharma</i>	923
184. Musculoskeletal Manifestations of Tuberculosis <i>Harpreet Singh, Sameer Arora</i>	931
185. Abdominal Tuberculosis: What's New? <i>Vishal Sharma, Rajoo Singh Chinna, Atul Sachdev</i>	934
186. Tubercular Meningitis: From Controversy to Consensus <i>Kameshwar Prasad, Deepti Vibha</i>	940
187. Current Concepts in Treatment of Tuberculosis: RNTCP, WHO and Indian Guidelines <i>Prashant Prakash, Prabhat Agarwal</i>	944
188. Management of ATT Hepatotoxicity <i>PK Sharma, Navjyot Kaur, Parmod Mittal</i>	953
189. Diabetes Mellitus and Tuberculosis: Double Trouble <i>Alladi Mohan, J Harikrishna, Prabath Kumar</i>	958
190. Nontuberculous Mycobacterial Infection: Growing Problem in Community <i>Harsh Toshniwal, Vipul Shah, Manoj Shevkani, Kalpak Bhatt</i>	962

**SECTION 17 RESPIRATORY SYSTEM**

- |   |      |
|---|------|
| 191. Health Effects of Air Pollution                                      | 969  |
| <i>SK Jindal, Anil Vij</i>  |      |
| 192. Health Effects of Smokeless Tobacco Products                         | 973  |
| <i>Inderpaul S Sehgal, Digambar Behera</i>                                |      |
| 193. Bronchoscopy: Where it Helps?  | 978  |
| <i>Parvaiz A Koul</i>   |      |
| 194. Advances in Asthma Management  | 985  |
| <i>Devasahayam J Christopher, T Balamugesh</i>                            |      |
| 195. Recent Trends in Management of Bronchial Asthma                      | 991  |
| <i>Mohd Sabir, SK Kochar, VK Tundwal</i>                                  |      |
| 196. Severe Asthma: Recognition and Management                            | 998  |
| <i>Nishtha Singh, Virendra Singh, Kulwani S Bhatia</i>                    |      |
| 197. Asthma-COPD Overlap Syndrome: What's in a Name?                      | 1001 |
| <i>Puneet Saxena, Vishal Chopra</i>                                       |      |
| 198. Chronic Obstructive Pulmonary Disease Update 2016                    | 1006 |
| <i>Aloke G Ghoshal, Avik Ghoshal, Indranil Halder</i>                     |      |
| 199. Cor Pulmonale  | 1011 |
| <i>Akashdeep Singh, Gurpreet S Wander, Raghav Mohan</i>                   |      |
| 200. Nonspecific Interstitial Pneumonia                                   | 1018 |
| <i>Deepak Talwar, Karamvir Goyal, Arjana Talwar</i>                       |      |
| 201. Idiopathic Pulmonary Fibrosis: New Hopes with Newer Therapies        | 1023 |
| <i>Ajay Handa, Akashdeep Singh</i>  |      |
| 202. Interstitial Lung Disease: Recent Trends in its Management           | 1028 |
| <i>PS Shanker</i>   |      |
| 203. Algorithmic Approach for the Management of Pleural Effusion          | 1033 |
| <i>Agam Vora, Navneet Singh</i>   |      |
| 204. Obstructive Sleep Apnea in India: Prevalence, Profile and Management | 1038 |
| <i>Ranjit Kumar Singh</i>   |      |
| 205. Pulmonary Rehabilitation   | 1043 |
| <i>AK Janmeja, Deepak Aggarwal, Anil Kashyap</i>                          |      |

**SECTION 18 NEUROLOGY**

- |   |      |
|---|------|
| 206. Headache Management: From Plants to Implants                 | 1049 |
| <i>Sumit Singh, VN Mishra</i>                                     |      |
| 207. First Seizure: How to Approach?                              | 1053 |
| <i>Mrinal Kanti Roy</i>   |      |
| 208. Temporal Lobe Epilepsy: Clinical Presentation and Management | 1057 |
| <i>V Nagaraajan</i>   |      |
| 209. Do we Really Require Newer Antiepileptic Drugs?              | 1061 |
| <i>Bhupendra Chaudhary, Binay Karak</i>                           |      |
| 210. Status Epilepticus   | 1064 |
| <i>Gagandeep Singh, Parampreet Kharbanda, Monika Singla</i>       |      |
| 211. Current Guidelines for the Management of Stroke              | 1069 |
| <i>Abhijeet K Kohat, Shirish M Hastak, Subhash Kaul</i>           |      |
| 212. Recent Advances in the Management of Acute Ischemic Stroke   | 1075 |
| <i>MV Padma Srivastava</i>  |      |

213. <b>Cerebral Venous Sinus Thrombosis: What a Physician Must Know?</b>	1080
<i>UK Misra, J Kalita, V Bansal</i>	
214. <b>Stroke Mimics</b>	1087
<i>PK Maheshwari, A Pandey</i>	
215. <b>Carotid Artery Disease: When and how to Treat?</b>	1090
<i>Dheeraj Khurana, Atul Mathur, Biplob Das, Sarju Ralhan</i>	
216. <b>Muscular Dystrophy: A Clinical Approach</b>	1096
<i>Satish V Khadilkar, NA Nadkarni</i>	
217. <b>Clinical Approach to Movement Disorders</b>	1101
<i>Vinay Goyal, Birinder S Paul</i>	
218. <b>Recent Advances in the Management of Demyelinating Neuropathies: Indian Perspective</b>	1105
<i>Anup K Bhattacharya, Bhaskar Ghosh</i>	
219. <b>Demyelinating Central Nervous System Disorders in India: An Update</b>	1111
<i>Lekha Pandit</i>	
220. <b>Autoimmune Encephalitis: Channelopathy/Synaptopathy/Circuitopathy</b>	1117
<i>Alexander Mathew</i>	
221. <b>Motor Neuron Disease: Indian Scenario</b>	1123
<i>Sudesh Prabhakar, Roopa Rajan</i>	
222. <b>From Early to Advanced Parkinson's Disease: How Treatment Approach Changes?</b>	1128
<i>U Meenakshisundaram, UM Sreenivas</i>	
223. <b>Approach to Patient with Coma</b>	1133
<i>B Vengamma</i>	
224. <b>Raised Intracranial Pressure without Focal Neurological Signs</b>	1139
<i>Sumeet Singla, AK Agarwal, Payal Jain</i>	
225. <b>Role of Decompressive Craniectomy in Malignant Middle Cerebral Artery Infarction</b>	1143
<i>Rajinder Bansal, Ashwani Chaudhary, Hanish Bansal</i>	

## SECTION 19 GASTROENTEROLOGY

226. <b>Irritable Bowel Syndrome: What is New in Diagnosis and Treatment?</b>	1149
<i>Uday C Ghoshal</i>	
227. <b>Treatment Modalities in Gastroesophageal Reflux Disease</b>	1155
<i>Manish S Bhatnagar, Nidhi Bhatnagar</i>	
228. <b>Inflammatory Bowel Disease in India: Profile, Pattern and Management</b>	1159
<i>Ajit Sood, Vandana Midha</i>	
229. <b>IBDOME: IBD from Primary Phenotypes to Immune-mediated Disorders</b>	1163
<i>Shishira Bharadwaj, Gursimran S Kochhar, Bo Shen</i>	
230. <b>Crohn's Disease: An Update</b>	1172
<i>Charles N Bernstein</i>	
231. <b>Role of Probiotics in Gastrointestinal Diseases</b>	1179
<i>Sethu Babu</i>	
232. <b>Development of GI Endoscopy: A Snapshot</b>	1183
<i>Nageshwar Reddy</i>	
233. <b>Management of Acute Pancreatitis</b>	1189
<i>Divya Yadav, Prabhleen Chahal</i>	
234. <b>Clinical Presentation and Management of Chronic Pancreatitis</b>	1193
<i>Puneet Chhabra, Surinder S Rana, Deepak K Bhasin</i>	
235. <b>Endovascular Management of Mesenteric Ischemia</b>	1198
<i>Praveg Goyal, Smit Shrivastava, Saroj K Sinha, Rajesh Vijayvergiya</i>	

**SECTION 20 HEPATOLOGY**

236. NASH—Newer Trends in Management: A US Perspective <i>Tavankit Singh, Gursimran S Kochhar, Arthur J McCullough</i>	1205
237. NASH—Newer Trends in Management: An Indian Perspective <i>Rajesh Upadhyay, Anupam Prakash</i>	1208
238. NASH for Clinicians <i>Deepak Amarapurkar</i>	1213
239. Hepatitis B: Prevalence, Presentation and Management in South Asians <i>Anjan Saikia, Mamun Al Mahtab</i>	1220
240. Physician's Role in Eradicating Hepatitis C <i>Amrisha Sahney, SK Sarin</i>	1224
241. Management of Acute Liver Failure <i>Mithun Sharma, Sandeep Sidhu</i>	1233
242. Acute Liver Failure in India: How is it Different from the West? <i>Bhaskar Nandi, Omesh Goyal</i>	1241
243. Guidelines for Decompensated Alcoholic Liver Disease: UK Perspective <i>N Summers, S Ramakrishnan</i>	1246
244. Recent Advances in Medical Management of Portal Hypertension <i>Sandeep Satsangi, YK Chawla</i>	1251
245. Organ Transplantation in India: Medicolegal and Ethical Issues <i>Ashok K Taneja, Ravindra Gupta, Hardev Ramandeep Ginn, Akshat Taneja</i>	1256
246. Liver Transplantation in India: Social and Medical Aspects and Current Status <i>Mohamed Rela, Gomathy Narasimhan</i>	1261
247. Approach to Obstructive Jaundice: Role of Endoscopic Ultrasound and ERCP <i>Nageshwar Reddy</i>	1265

**VOLUME 3: MEDICINE UPDATE 2016–2****SECTION 21 GENERAL CARDIOLOGY**

248. Epidemiology of Heart Disease in India <i>Rajeev Gupta, Kulwant Singh</i>	1271
249. Management of Congenital Heart Diseases in Adults <i>S Ramakrishnan, Rajiv Kumar</i>	1277
250. Acute Rheumatic Fever: Application of Revised Jones Criteria in Indian Context <i>IB Vijayalakshmi</i>	1284
251. Pulmonary Arterial Hypertension (Primary and Secondary): Current Classification and Indian Perspective <i>Jagdish Hiremath, Kaushik Sheth</i>	1289
252. Infective Endocarditis in Developing Country <i>Ravi S Math, Shyam S Kothari</i>	1294
253. Management of Acute Pulmonary Embolism: Recent Guidelines <i>Ankur Gupta, Manojkumar Rohit</i>	1303
254. Diseases of the Aorta (Dissection, Aneurysm...): Therapeutic Options Available in Our Country <i>Sanjay Mehrotra, VS Prakash</i>	1307
255. Challenges of Takayasu Arteritis in India <i>Sanjay Tyagi, Manotosh Panja, Prattay Guha Sarkar, Swati Tyagi</i>	1314

256. Anemia and Cardiac Diseases in India: Prevalence, Consequence and Management	1323
<i>Yash Paul Sharma, Prafull Sharma, Anita Sharma, Kanupriya Vashishth</i>	
257. Venous Diseases: Role of Pharmacotherapy and Interventions	1329
<i>Budnur C Srinivas, Bhupinder Singh</i>	
258. Interventional Procedures to Salvage Visceral Bleeding Emergencies	1335
<i>Sanjiv Sharma, Bishav Mohan</i>	
259. Impact of Recent Advances in Technology (Devices and Stents) on Practice of Cardiology	1341
<i>Mathew Samuel Kalarickal, Vijay Kumar S</i>	
260. Novel Oral Anticoagulant Drugs: Where and How to Use?	1351
<i>Subhash Varma, Uday Yanamandra, Vikas Suri</i>	

## SECTION 22 ISCHEMIC HEART DISEASE

261. Chronic Stable Angina: Medical Therapy versus Revascularization	1361
<i>VK Bahl, Neeraj Parakh</i>	
262. Pharmacological Management of Refractory Angina	1366
<i>VK Katyal, Abhishek Goyal, Ashima Katyal</i>	
263. PCI versus CABG: In Search of Best Treatment for Ischemic Heart Disease	1372
<i>Prakash P Punjabi, Sajiram Sarvananthan, Jaspal S Kooner</i>	
264. Bioabsorbable Scaffolds Stents: Current Role in Coronary Interventions and Future Perspective	1378
<i>Praveen Chandra, Niraj Gupta</i>	
265. Use of Chest Pain Protocols to Improve Patient Care	1383
<i>Richard A Chazal</i>	
266. Acute Coronary Syndrome: Management Protocol in Primary and Tertiary Care Setup	1388
<i>Tejas Patel, Sanjay Shah</i>	
267. Dual Antiplatelet Therapy in Various Cardiovascular Disorders: When and how Long to use?	1394
<i>Santanu Guha, Dipankar Ghosh Dastidar, Nandita Ghosh Dastidar</i>	
268. What have we Learned from Acute Coronary Syndrome Registries in India?	1402
<i>PP Mohanan, MN Krishnan</i>	
269. Thrombolytic Agents: Which, when and how to use in Acute MI?	1410
<i>I Sathyamurthy, K Jayanthi</i>	
270. Ischemic Preconditioning	1415
<i>VN Mishra, Sameer Vyas, Ishan Mishra</i>	
271. Thrills of Primary Angioplasty in Acute Myocardial Infarction	1421
<i>Lekha A Pathak, Salil Shirodkar, Sachin K Patil, Ronak Ruparelia</i>	
272. Pharmacoinvasive Therapy: A New Paradigm in Management of STEMI in India	1427
<i>Sreenivas K Arramraju, Vuppaladadhaim Hariram</i>	
273. Protocol-based STEMI Management in India	1434
<i>Suma M Victor, Thomas Alexander, Ajit Mulasari S</i>	
274. Management of STEMI in Resource-limited Settings	1438
<i>Lekha A Pathak, Salil Shirodkar, Ronak Ruparelia</i>	

## SECTION 23 HEART FAILURE, CARDIOMYOPATHY AND ARRHYTHMIA

275. Heart Failure: Prevalence, Pattern and Profile in India	1445
<i>Ajay Bahl, KK Talwar</i>	
276. Biomarkers in Heart Failure: An Update	1449
<i>Amal Kumar Banerjee</i>	
277. A Step Care Management of Systolic Heart Failure	1454
<i>Saumitra Ray</i>	



278. Metabolic Syndrome, Occult Cardiovascular Disease and Diastolic Heart Failure: A Triple Jeopardy <i>HK Chopra, Manmohan Singh</i>	1462
279. Angiotensin Receptor Nephrylysin Inhibition: A New Paradigm in Cardiovascular Disease Management <i>Kamal K Sethi, Subrato K Datta</i>	1470
280. Latest Advances in the Management of Heart Failure with Preserved Ejection Fraction <i>Shibba T Chhabra, Inder S Anand</i>	1473
281. Profile and Pattern of Cardiomyopathies in India <i>Ajay Bahl</i>	1479
282. Hypertrophic Cardiomyopathy: Current Concepts <i>PB Jayagopal, Aftab Khan, Jain T Kallarakal, Debabrata Bera</i>	1482
283. Cardiac Sarcoidosis/Tuberculosis: Not so Uncommon cause of VT/Cardiomyopathy in our Country <i>Daljeet K Saggi, C Narasimhan</i>	1489
284. Role of Heart Rate in Cardiovascular Diseases <i>Sunil Sathe, Anish Chandarana</i>	1493
285. Ventricular Ectopy: Is it Benign or a Predictor of Heart Failure and Death? <i>Praveg Goyal, Rajesh Vijayvergiya</i>	1496
286. Syncope: Causes and Clinical Evaluation <i>Niraj Yadav, Anoop K Gupta</i>	1499
287. Algorithm for Syncope Management <i>IVMR Tammi Raju, Nandan Putti, B Hygriv Rao</i>	1504
288. Sudden Cardiac Death: Challenges and Opportunities <i>Sumeet S Chugh</i>	1510
289. Newer Investigations for Diagnosis of Rhythm Abnormalities: External Loop Recorder and Internal Loop Recorder <i>Nayani Makkar, Balbir Singh</i>	1515
290. Single Chamber, Dual Chamber and Rate Responsive Pacemakers: Which one in what Indication? <i>Sandeep Thakkar, Arun K Chopra</i>	1518
291. Advances in Pacing Modalities: MR Compatibility, Atrial Fibrillation Prevention and the Leadless Pacemakers <i>Varita Arora</i>	1523
292. Role of Complex Devices in Cardiac Disorder: AICD, Cardiac Resynchronization Therapy and Combo Devices <i>Archana Rao, Raj Pradhan</i>	1528
293. Atrial Fibrillation: Current Treatment Options <i>SB Gupta</i>	1531
294. Warfarin Therapy: Issues in Initiation, Monitoring and Follow-up in day-to-day Practice <i>Sandeep Bansal</i>	1536
295. What All is Possible in Electrophysiology and where do We Stand in India? <i>Anitha Sathishkumar, Naved Aslam, Ulhas Pandurangi, M Khalilullah</i>	1540

**SECTION 24****CRITICAL CARE**

296. Communication with Family of Intensive Care Patients <i>Vivek Kumar, Vikas Loomba</i>	1547
297. Standards of Quality Assurance in Indian ICU <i>Rajesh Pande, Jigeshu Devatia</i>	1551

298. Scoring System in Critically Ill: Does it Help in Prognostication?	1554
<i>Shivakumar Iyer, Jignesh Shah</i>	
299. Advances in Hemodynamic Monitoring in Intensive Care Unit in India	1559
<i>KC Misra, Venkat Raman K</i>	
300. Optimizing Nutrition in Critically Ill	1563
<i>Ramakrishnan N, Sriram K</i>	
301. Practical Approach for Management of Electrolyte Disturbances in ICU	1567
<i>Sundaram Arulhraj, Aarathy Kannan, Ajinkya Borhade, Sachin Babhalsure</i>	
302. Do's and Don'ts in Hyponatremia	1573
<i>Bikramiyoti, Ankur Gupta</i>	
303. Approach to Management of Hyponatremia	1577
<i>Sanjay Pandya</i>	
304. Optimizing Pharmacotherapy in the Critically Ill Patient	1582
<i>Dilip R Karnad, Gauri Saroj</i>	
305. Anaphylaxis: How Best to Manage	1586
<i>Siddharth Marda</i>	
306. Infections in ICU: Indian Scenario	1590
<i>AP Misra, Rajesh Mahajan</i>	
307. Simplified Algorithm for ABG Interpretation	1595
<i>Pradip Dalvi, Sumit Ray, Sameer Jog</i>	
308. Arterial Blood Gas Analysis for Physicians	1600
<i>Atul Bhasin</i>	
309. Acid-base Problems in Diabetic Ketoacidosis	1604
<i>Dinesh Gupta, Gursimran Kaur, Akriti Gupta</i>	
310. What a Physician should Know about Mechanical Ventilation?	1607
<i>Rajesh Chawla, Updesh Sidhu, Aakanksha Chawla</i>	
311. Mechanical Ventilation, Difficulty in Weaning: How to Approach?	1613
<i>AP Singh, Gurinder Mohan</i>	
312. Ventilator-associated Pneumonia	1618
<i>A Bhagwati</i>	
313. Critical Illness Polyneuropathy	1623
<i>Gurinder Mohan, AP Singh, Ranjeet Kaur</i>	
314. Continuous Renal Replacement Therapy in ICU Settings	1627
<i>Vivek Gupta, Etienne Macedo, Ravinder L Mehta</i>	
315. Extracorporeal Membrane Oxygenation: A New Therapeutic Modality—Where We are in India	1634
<i>Poonam Malhotra, Venkat Goyal, Pranay Oza</i>	
316. Fungal Infections in the ICU	1639
<i>Chandana Reddy, BK Aggarwal</i>	

## SECTION 25

## MEDICAL DISORDERS IN WOMEN AND PREGNANCY

317. Hypertensive Disorders in Pregnancy: Newer Mechanisms	1649
<i>C Venkata S Ram</i>	
318. Thyroid Disorders in Pregnancy	1653
<i>Lalit K Meher, Sujoy Ghosh</i>	
319. Rheumatic Diseases in Pregnancy: Common Conundrums Facing Physicians	1661
<i>May Ching Soh</i>	

320. **Obstetric Critical Care: Challenges and Approach** 1669  
*Niteen D Karnik, Anish Gupta*
321. **Complex Medical Disorders in Pregnancy** 1675  
*Mandish K Dhanjal*
322. **Drug Doses Adjustment in Pregnancy** 1682  
*Deepak Gupta, Pooja Tandon, Gurleen Wander*
323. **Gynecological Cancers: Presentation and Evaluation from a Physician's Perspective** 1686  
*Jayanta Chatterjee, Sadaf Ghaem-Maghami*
324. **National Guidelines for Diagnosis of Gestational Diabetes Mellitus** 1691  
*V Seshiah, V Balaji, Madhuri S Balaji*

**SECTION 26 JOURNAL CLUB**

325. **How to Read and Interpret a Journal Article: Biostatistics for the Clinician** 1699  
*Tiny Nair*
326. **Lessons Learnt from Significant Trials in NEJM in Last Three Years: Translating into Practices in India** 1703  
*Anupam Prakash, Nirupam Prakash, Nupur Jain*
327. **Lessons Learnt from Significant Trials in Gastroenterology in Last Three Years** 1707  
*Vikram Bhatia, Vishal Sharma, Vineet Ahuja*
328. **Lessons Learnt from Significant Recent Trials in Cardiology in Last Three Years** 1712  
*S Nagendra Boopathy, Manish Bansal, K Sarat Chandra*
329. **Recent Advances in Neurology Based on Significant RCTs in Last Three Years** 1718  
*Apoorva Pauranik*
330. **Landmark Trials in Cardiology** 1725  
*Saurabh Mehrotra, Sundeep Mishra*

**SECTION 27 GERIATRICS**

331. **Preventive Medicare for the Elderly** 1733  
*Mahesh Marda*
332. **Urinary Incontinence in Elderly** 1736  
*Jyotirmoy Pal, Swati Kumar, Anindya Sarkar*
333. **Adult Vaccination** 1742  
*Jyotirmoy Pal, Sattick Siddanta, Subhodeep Gupta, Praneet Wander*
334. **Frailty: What Physicians should Know?** 1748  
*Astha Koolwal, Ashish Goel*
335. **Geriatric Syndromes** 1753  
*Dinesh Gupta, Gursimran Kaur, Akriti Gupta*
336. **Dementia In India: Prevalence and Management** 1759  
*Suvarna Alladi, Jaydip R Chaudhuri*

**SECTION 28 GENERAL (SOFT SKILLS AND SOCIAL ASPECTS)**

337. **What Is Health?** 1767  
*BM Hegde*
338. **Art and Science of Medicine 2016** 1770  
*Shashank R Joshi*
339. **Importance of Communication Skills for Doctors** 1775  
*KK Pareek, Girish Mathur, GD Ramchandani*

340.	<b>Lost Art of Bedside Manners</b> <i>Anil Chaturvedi</i>	1780
341.	<b>Challenges Faced by Medical Profession Today</b> <i>Ajay Kumar</i>	1782
342.	<b>Legal Issues in Medicine: Present Day Challenges for Indian Physicians</b> <i>KK Aggarwal</i>	1788
343.	<b>Business Strategy Primer for Physicians</b> <i>Amit K Ghosh, Karthik Ghosh</i>	1797
344.	<b>Developing an Emergency Medicine Department in an Indian Nursing Home or Hospital</b> <i>Sarabmeet S Lehl, Angel Rajan Singh, Monica Gupta</i>	1803
345.	<b>Air Ambulance Services in India: Current Status, Problems and Future Prospects</b> <i>Munish Prabhakar, Geeta Kampani, Rajesh Kakkar</i>	1809
346.	<b>Evidence-based Medicine</b> <i>Vitull K Gupta, Meghna Gupta, Gurpreet S Wander</i>	1813
347.	<b>Collaborative Research and the Practicing Clinician</b> <i>Subramanian Shankar</i>	1818
348.	<b>Drug Interactions in Day-to-Day Clinical Practice: Which One's We Must Keep in Mind</b> <i>Shankar Hotchandani, Chirag Mistry, Kaza Ahluwalia, Uday Bansal</i>	1821
349.	<b>Preoperative Cardiovascular Evaluation and Perioperative Cardiovascular Medical Management</b> <i>Michael A Mikhail, Arya B Mohabbat, Amit K Ghosh</i>	1827
350.	<b>Some Recent Issues: E-Cigarettes, Fecal Transplant, Bronchial Thermoplasty, Medical Marijuana</b> <i>Tanu Shweta Pandey</i>	1832
351.	<b>All Things Flow, Nothing Abides</b> <i>BM Hegde</i>	1837
352.	<b>The American Medical Association, its Strategic Plan, and the Impact on Health and Health Care in the US</b> <i>Andrew W Gurman</i>	1839

**SECTION 29****THE FUTURE (GENETICS, IT)**

353.	<b>From Genome to Genetic Medicine: Opportunities and Challenges</b> <i>Narinder K Mehra, Deepali K Bhat, Neeraj Kumar, Ajay K Baranawal</i>	1847
354.	<b>Cardiovascular Disease in South Asians: Risk Factors, Genetics and Environment</b> <i>Dharambir K Sanghera, Sunita Dodani</i>	1853
355.	<b>Genetics in Chronic Medical Disorders</b> <i>Suman Kapur</i>	1859
356.	<b>Medicine in the Future: Genomes and Smartphones</b> <i>Navjit Dullet, Praneet Wander</i>	1861
357.	<b>Risk Management in Primary Care (Role of Information Technology)</b> <i>Gursharan Singh, Gurwinder S Virk</i>	1865
358.	<b>Smartphones in Medicine: Augmenting Bedside Clinical Skills and Beyond ...</b> <i>SV Kulkarni, Sagar Sinha, Adhar Kulkarni, Aditya S Kulkarni</i>	1868
359.	<b>Should Smartphones or Tablets be Used in Front of Patients?</b> <i>Amitabh Sagar, Harmeet S Dhooria</i>	1874
360.	<b>Digital Medicine in United States: Implications for Indian Healthcare</b> <i>Praneet Wander, Ashish Atreja</i>	1879

**SECTION 30 MISCELLANEOUS**

- |  |             |
|--|-------------|
| <b>361. How to Combat the Threat of Diabetes, Coronary Artery Disease and Stroke in India?</b>       | <b>1887</b> |
| <i>Ashok K Das</i>   |             |
| <b>362. Diuretics in Hypertension</b>  | <b>1890</b> |
| <i>Kunal Kothari</i>   |             |
| <b>363. Hypertension Guidelines: From Confusion to Clarity, Thanks to SPRINT</b>                     | <b>1895</b> |
| <i>C Venkata S Ram</i>   |             |
| <b>364. Rationale and Importance of Combination Drug Therapy in Hypertension</b>                     | <b>1899</b> |
| <i>Amit A Bharadiya, C Venkata S Ram</i>   |             |
| <b>365. Invasive Fungal Infections: Current Concepts</b>   | <b>1905</b> |
| <i>Deven Juneja, Ankur Gupta, Pritam Gupta</i>   |             |
| <b>366. Chronic Kidney Disease: Epidemiology and Solutions</b>                                       | <b>1912</b> |
| <i>Anupam Bhel, Gaurav Sagar, Sanjiv Jasuja</i>  |             |
| <b>367. Dengue Fever</b>   | <b>1917</b> |
| <i>Shibendu Ghosh, Prabuddha Mukhopadhyay, Saumen Bhat</i>   |             |
| <b>368. Insulin Therapy in Diabetes Mellitus: When, Which and How?</b>                               | <b>1922</b> |
| <i>Sudhir Bhandari</i>   |             |
| <b>369. Early Aggressive Management of Type 2 Diabetes</b>   | <b>1926</b> |
| <i>Shashank R Joshi, Saurabh Arora</i>   |             |
| <b>370. Controversies in the Treatment of Acute Pancreatitis</b>                                     | <b>1930</b> |
| <i>Rakesh Tandon</i>   |             |
| <b>371. How to Interpret Bacterial Load and MIC in Culture Report</b>                                | <b>1935</b> |
| <i>Smarajit Banik, Sanjay Kumar Mallick</i>  |             |
| <b>372. Ultrasonography in Chest Diseases</b>  | <b>1939</b> |
| <i>BNBM Prasad, Sudhir Saxena, Monika Goyal</i>  |             |
| <b>373. Common Pitfalls in Day-to-Day Management of Diabetes Mellitus</b>                            | <b>1944</b> |
| <i>Siddharth N Shah</i>  |             |
| <b>374. Role of Dual Peroxisome Proliferator-activated Receptor Agonist in Diabetic Dyslipidemia</b> | <b>1948</b> |
| <i>Jayanta K Panda, Prakash C Panda, Smita K Panda</i>   |             |
| <b>375. Myths and Pearls in the Management of Rheumatoid Arthritis</b>                               | <b>1951</b> |
| <i>Sukumar Mukherjee, Somnath Bhar</i>   |             |
| <b>376. Management of Hepatic Encephalopathy</b>   | <b>1956</b> |
| <i>Rajesh Upadhyay, VK Gupta, Shubhra Gupta, Praneet Wander</i>                                      |             |
| <b>377. Large Vessel Vasculitis</b>  | <b>1961</b> |
| <i>Milind Y Nadkar, Madhur Maheshwari</i>  |             |

*"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.<sup>1</sup> 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.<sup>2</sup> 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.<sup>3</sup> This helps in decreasing the number of so called unclassifiable vasculitis and even has been validated by us in Indian population.<sup>4</sup> Subsequently, the revised CHCC nomenclature system was proposed in 2012.<sup>5</sup> 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.<sup>6</sup> This clearly shows that the quest for ideal classification and diagnostic criteria is a "work in progress".<sup>7</sup> 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.<sup>8</sup>

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.<sup>9</sup> 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.<sup>2</sup> 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).<sup>10</sup> 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 of 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



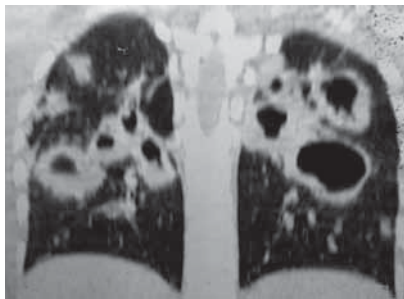
**Fig. 2 Collapse of the nasal bridge in GPA**  
Source: Reproduced from Sharma A. Textbook of Systemic Vasculitis, 1st edition. New Delhi: Jaypee Brothers Medical Publishers (P) Ltd; 2015.



**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.

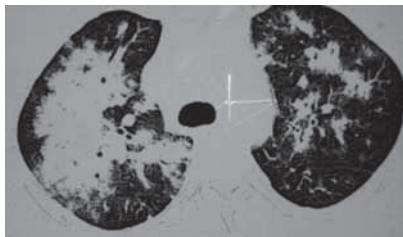


**Fig. 1 Nasal septal perforation in GPA**  
Source: Reproduced from Sharma A. Textbook of Systemic Vasculitis, 1st edition. New Delhi: Jaypee Brothers Medical Publishers (P) Ltd; 2015.



**Fig. 4 Multiple cavitating nodules in GPA**  
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**Fig. 5** CT scan showing diffuse alveolar hemorrhage

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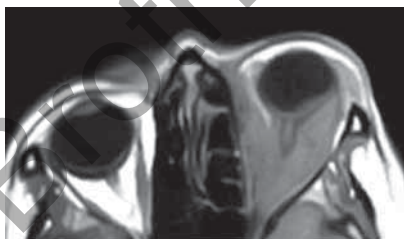
**Fig. 6** Necrotizing scleritis in GPA

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

crescentic glomerulonephritis presenting as rapidly progressive renal failure.<sup>11</sup> The outcomes are dependent upon the histopathological subtype.<sup>12</sup> The presentation based upon ANCA positivity and negativity are also being compared these days.<sup>13</sup> 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.<sup>14</sup> 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



**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.

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 paucimmune 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.<sup>11</sup> 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.

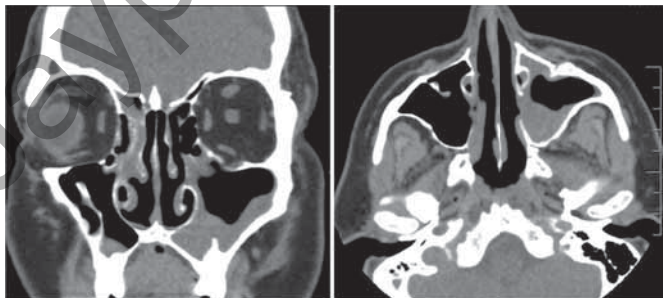
Dominant ANCA pattern seen in these patients is p-ANCA and MPO. C-ANCA and PR3 positivity is uncommon. Anti-lysosomal-associated membrane protein 2 (LAMP2) is proposed to be pathogenic and its serial monitoring may be of value, but as of now, it is of academic interest only. The treatment is on the lines 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 ( $> 1,500/\text{mm}^3$ ) 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.

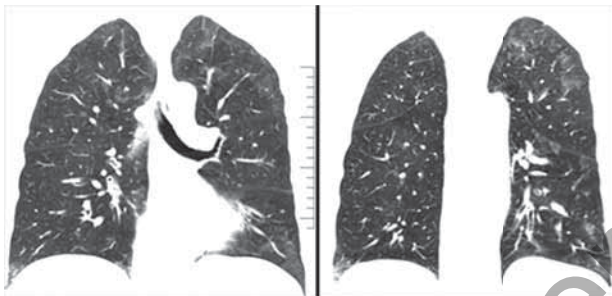
The three main features on histopathology include 1) vessel wall infiltration (mainly with eosinophil) with fibrinoid necrosis, 2) extravascular eosinophilic infiltrates and 3) extravascular necrotizing granulomas. The diagnosis is based upon suggestive clinical finding supported by histopathology wherever 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**

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



**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,<sup>15</sup> 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.<sup>16</sup>

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