



East Delhi Physicians Association

Ist Quarterly News Bulletin

President EPDA Dr. Paras Gangwal

Secretary
 Dr. Swathi Jami

Editors
 Dr. Aman Rohatgi
 Dr. Ashok Kumar





## From EDITORS DESK ------



### Greetings to all esteemed members of the EDPA

We are excited to bring you the 1st edition of the EDPA E-Newsletter, containing insightful updates and case presentations from our dedicated professionals. In this edition our focus is on the unique cases that have been shared during our monthly presentations. Here are some highlights:

- 1. **Common presentation of a rare disorder 24 Hydroxylase Deficiency:** This presentation sheds light on a rare disorder that manifests with common symptoms, emphasizing the importance of early recognition and accurate diagnosis for effective management.
- 2. **Case of CBD Stricture:** Exploring a challenging case of Common Bile Duct (CBD) stricture, our experts delve into diagnostic dilemmas and therapeutic strategies, providing valuable insights for clinicians dealing with similar cases.
- 3. **Case of Hypokalemic Periodic Paralysis:** The presentation of a case involving Hypokalemic Periodic Paralysis brings attention to this rare neuromuscular disorder, offering valuable lessons in diagnosis and treatment.
- 4. **Case of Allergic Bronchopulmonary Aspergillosis:** Through this case study, our clinicians navigate the complexities of allergic bronchopulmonary aspergillosis, highlighting diagnostic challenges and therapeutic interventions for optimal patient care.
- 5. **Thrombotic Thrombocytopenic Purpura:** Our experts dissect a challenging case of Thrombotic Thrombocytopenic Purpura, unraveling its clinical manifestations, diagnostic approach, and management strategies for improved patient outcomes. These presentations exemplify the collaborative spirit of our community and underscore the importance of sharing knowledge and experiences to enhance patient care. We extend our gratitude to all contributors and look forward to continued engagement and learning in the upcoming presentations.

Warm regards,

Dr. Aman Rohatgi Dr. Ashok Kumar *Executive Editors* EDPA Newsletter





## PRESIDENT'S MESSAGE



#### Dear Esteemed Members of the East Delhi Physician's Association,

As we embark on the momentous occasion of our association's 25th Silver Jubilee Year, I, Dr. Paras Gangwal, President of the East Delhi Physician's Association, extend my warmest congratulations to each and every one of our esteemed members. With over 350 dedicated MD Physicians, our association stands as a beacon of academic excellence and professional camaraderie.

This year marks a significant milestone in our journey, and it is with great pride that I invite all members to actively participate in the myriad activities planned by the EDPA throughout the year. Your involvement and enthusiasm are crucial in ensuring the grand success of our endeavors and in elevating the reputation of our esteemed association to new heights.

In addition to our academic pursuits, I urge each member to prioritize their health and well-being. As physicians, we understand the importance of nutrition, healthy habits, and longevity. I encourage you all to lead by example and incorporate healthy behaviors into your daily lives. Remember, a healthy body and mind are essential for us to continue serving our patients and community effectively.

Furthermore, I would like to express my heartfelt gratitude to the core team of the East Delhi Physician's Association for their invaluable guidance and support to the current executive members. Your wisdom and experience have been instrumental in steering our association towards continued success.

I must commend and praise our current Secretary, Swathi Jami, for her remarkable contributions. Despite being newly enrolled in the EDPA, Swathi has undertaken tremendous tasks and has swiftly evolved into a proficient advocate for EDPA's academic pursuits and activities. Her dedication and commitment are truly commendable, and I have no doubt that she will continue to excel in her role.

I am also delighted to welcome the new upcoming executives under the leadership of Dr. Pankaj Chaudhary. With Swathi Jami assuming the role of Super Secretary for the EDPA Silver Jubilee Year, I am confident that our association is in capable hands. To our new executives, we as current executives offer our guidance and encouragement to carry out more activities aimed at public education. Rest assured, you have the full support and guidance of the core team behind you at every step of the way.

Together, let us work tirelessly towards the advancement of our association and the betterment of our community. With unity, determination, and a shared vision, we will undoubtedly achieve great things in the year ahead.

Warm regards,

**Dr. Paras Gangwal** *President* East Delhi Physician's Association





## SECRETARY'S MESSAGE

#### Dear Members,

Welcome to our association's first quarterly bulletin of the year, where we proudly highlight the outstanding presentations from our esteemed members during the first quarter.

Within these pages, you'll find a rich tapestry of abstracts, each representing the expertise and dedication of our members in delivering exemplary healthcare. As we delve into these presentations, let us celebrate the diversity of knowledge and experiences that enrich our association.

Your active participation not only enhances our collective learning but also strengthens the fabric of our association. Together, we continue to push the boundaries of medical practice and elevate the standard of care for our patients.

Thank you for your unwavering commitment to excellence and for your invaluable contributions to our association.

Warm regards,

**Dr Swathi Jami**, Secretary, EDPA





## EDITOR ADVISOR'S MESSAGE



The monthly scientific CME of EDPA is great academic exercise. The case presentation is of high academic excellence with enlightened content. It can be compared to any postgraduate medical presentation of post department of any corporate hospital or top 5 medical Institution of the country

This e bulletin is a collection of monthly CME cases discussed in the last one academic year. It demonstrates the vision of the Chairman scientific committee and the determination and commitment of teachers who involve initiate and inspire the postgrade students to come forward and present cases .The variety of cases presented by Super specialists/ Physicians is evident of knowledge, clinical armatorium and professionalism in the scientific committee. I am sure the Bulletin will serve as a reference / Guide to the postgraduate students and physicians alike. Producing something of this magnitude is like having a baby. The gestation period is long, the labour is painful but the result is great.

Two Erudite Editor's DrAman&DrAshok have completed the task untimely with prudence&Pragmatism.

I must acknowledge the contribution commitment courage and conviction of our smiling secretary Swati Jami in compiling the articles so diligently

Long live EDPA

Dr Anil Chaturvedi







### **TTP Abstract**

Dr. Neeru Agarwal

### Introduction

Thrombotic thrombocytopenia purpura (TTP) is a rare form of thrombotic microangiopathy (TMA) characterized by microangiopathic hemolytic anaemia (MAHA), severe thrombocytopenia, and ischemic end-organ damage resulting from the formation of platelet-rich thrombi in the microvasculature. The pathophysiology of TTP is based on a severe ADAMTS 13 deficiency ( activity < 10%). Although the exact cause of TTP remains unclear, previous studies have suggested that autoimmune diseases and other various infections including Human immunodeficiency virus infection (HIV), Hepatitis C (HCV) and Helicobacter pylori, can induce TTP.5 However, TTP caused by simultaneous infection with hepatitis A and E has been rarely reported. We have encountered a case of TTP caused by a simultaneous infection with hepatitis A and E in a male patient.

### Case

A 23-year-old male was admitted to our hospital with complaints of persisting fever, associated with nausea and vomiting for 7 days and jaundice of unknown cause for 5 days. On the way to the hospital, the patient also developed one episode of tonic-clonic movement with uprolling of eyes, likely a seizure activity. There was a history of bathing in Ganga river water and intake of roadside food 1-2 weeks before the onset of illness.

Upon arrival in the emergency room, the patient's condition deteriorated, so the patient was advised ICU admission. In the ICU patient had a fall in oxygen saturation and blood pressure with low GCS (E2V3M4) for which he was intubated and taken on inotropic support.

Upon admission, physical examination revealed a Glasgow Coma Scale score of E3V4M5, and vital signs were as follows: body temperature: 97.5 °F, Blood pressure: 110/80 mmHg (while receiving Noradrenaline @ 10ml/hr), pulse: 112/min, respiratory rate: 24/min, oxygen saturation (SpO2): 97% (on ventilator support). ABG revealed severe metabolic acidosis with serum bicarbonate levels of 10.2 meq/l. In view of severe metabolic acidosis, anuria and deranged kidney function test, the patient was advised for urgent SLED (Sustained Low Efficiency Dialysis)

Based on laboratory results, the patient was diagnosed with acute viral hepatitis caused by hepatitis E and hepatitis A, severe renal dysfunction with anuria and thrombocytopenia. Despite giving 2 sessions of haemodialysis and 3 units of PRBC (Packed Red Blood Cell) transfusion, the patient's condition did not improve significantly.





### Table 1 : Laboratory work up

| S. No | TEST                                      | RESULT                      |
|-------|---|-----------------------------|
| 1.    | HbsAg                                     | Non Reactive                |
| 2.    | Anti HCV                                  | Negative                    |
| 3.    | HAV IgM                                   | 6.56 ( <b>Positive</b> )    |
| 4.    | Anti HEV IgM                              | 4.6 ( <b>Positive</b> )     |
| 5.    | HIV Assay ( I and II )                    | Non Reactive                |
| 6.    | Scrub Typhus IgM                          | Negative                    |
| 7.    | Leptospira Antibody Panel                 | Negative                    |
| 8.    | Typhidot                                  | Negative                    |
| 9.    | Malaria Antigen                           | Negative                    |
| 10.   | ANA Immunofluorescence                    | 1 : 40 (Negative)           |
| 11.   | Coombs Test Direct                        | Negative                    |
| 12.   | G 6 PD Deficiency Assay                   | 21.4 (7 – 20.5 U/g)         |
| 13.   | C 3 Complement                            | 70 (79 – 152 mg/dl)         |
| 14.   | C 4 Complement                            | 13 (16-38 mg/dl)            |
| 15.   | Clostridium Difficle Toxin A &<br>B Stool | Negative                    |
| 16.   | Serum Procalcitonin                       | 10.91 ( < 0.5 ng/ml)        |
| 17.   | Blood Culture                             | Staphylococcus Haemolyticus |
| 18.   | Spot Urine Protein Creatinine<br>Ratio    | 1.68 g/g                    |
| 19.   | ADAMTS 13 Activity                        | 31.70 % ( 60.6 – 130.6 % )  |



In view of progressively falling haemoglobin, hemolysis workup was done that showed LDH - 2495 U/L, Reticulocyte count - 8.5% and serum haptoglobin was – less than 30 MG/dL. Peripheral smear showed marked

anisopoikilocytosis, admixed with elliptocytes, tear drop cells, target cells, polychromatophils, spherocytes, fragmented red cells, and few macrocytes. Furthermore, the patient's ADAMTS13 activity was low (31.70%). All these reports tilted the diagnosis towards TTP.

| S.No. | PARAMETER                | FINDING  |  |  |  |
|-------|--------------------------|--|--|--|--|
| 1.    | S. LACTATE DEHYDROGENASE | 2495 U /L  |  |  |  |
|       | (N : 98 – 192 U/L)       |  |  |  |  |
| 2.    | S. RETICULOCYTE COUNT    | 8.5 %  |  |  |  |
|       | (N: 0.5 – 2.5 %)         |  |  |  |  |
| 3.    | S. HAPTOGLOBIN           | < 30 MG/dL   |  |  |  |
|       | (N: 30 – 200 MG/dL)      | A  |  |  |  |
| 4.    | PERIPHERAL SMEAR         | Marked anisopoikilocytosis, admixed with elliptocytes, |  |  |  |
|       | 1                        | tear drop cells , target cells , polychromatophils ,   |  |  |  |
|       |                          | spherocytes, fragmented red                            |  |  |  |
|       |                          | cells, few macrocytes.                                 |  |  |  |

### Table 2 : Laboratory evidence of ongoing haemolysis

Following the additional diagnosis of TTP, the patient was given plasma exchange of 35ml/kg/day × 10 times, steroids in the form of pulse methylprednisolone for 3 doses (500 mg solumedrol for 3 doses) followed by a maintenance dose of steroids. Gradually patient's condition improved, his hemoglobin stopped dropping, bilirubin started falling and he was extubated on the 7th day of admission, after the 4th session of plasma exchange. However, his urine output did not pick up, so he underwent multiple sessions of plasma exchange followed by dialysis. After 10 sessions of plasma exchange, when there was no evidence of hemolysis, plasma exchange was stopped.

### **EDPA** NEWSLETTER



The patient was shifted to the ward on day 13 of admission. The first dose of injection rituximab 500 mg was given to the patient after the last plasma exchange and he was advised rituximab 500 mg once a week for 4 consecutive weeks. 21 days after admission, the patient's condition improved significantly and was discharged with advice to be on maintenance haemodialysis thrice per week.

On further follow up of the patient, patient started producing urine. His dialysis frequency was reduced as per the need from thrice to twice per week haemodialysis. After 4 weeks of discharge, patient was producing around 3 litres of urine per day. His dialysis catheter was removed and his dialysis was stopped.

| Parameter                          | Day 1 | Day 6 | Day 11 | Day 16 | Day 21 | Two weeks<br>after<br>discharge |
|------------------------------------|-------|-------|--------|--------|--------|---------------------------------|
| Hb<br>(N : 13 – 17 g/dl)           | 7.6   | 7.5   | 6.8    | 6.9    | 6.4    | 7.4                             |
| WBC<br>(N:4000-10,000)             | 12.1  | 18.1  | 16.1   | 19.2   | 12     | 7.6                             |
| RBC<br>(N: 4.5 – 5.5<br>million/L) | 3.59  | 2.88  | 2.82   | 2.89   | 2.78   | 3.33                            |
| PLATELETS<br>(N:150-410<br>/L)     |       |       | 1      | V      |        |                                 |
| T. BIL<br>(N : 0.2 – 1.2<br>mg/dL) | 11.3  | 10.5  | 8.5    | 7.1    | 3.25   | 2.24                            |
| D. BIL<br>(N : 0 – 0.3mg/dL)       | 10.9  | 8.4   | 7.1    | 5.3    | 2.9    | 1.97                            |
| ALBUMIN<br>(N : 3.5 – 5.2<br>g/dL) | 3     | 3.1   | 3.3    | 3.3    | 3.48   | 4.14                            |

### Table 3 : Trend of laboratory reports



| ALT                      | 1178 | 784  | 68.8 | 41   | 39.5 | 40.2 |
|--------------------------|------|------|------|------|------|------|
| (N : 0 – 40<br>IU/L)     |      |      |      |      |      |      |
|                          |      |      |      |      |      |      |
| AST                      | 347  | 214  | 69.6 | 53   | 48.1 | 44.4 |
| (N : 0 – 40<br>IU/L)     |      |      |      |      |      |      |
| РТ                       | 22.2 | 21.3 | 17.8 | 15.9 | 14.1 | 11.8 |
| (N : 9.8 – 12.9<br>sec)  |      |      |      |      |      |      |
| CREATININE               | 10.5 | 5.8  | 6.2  | 5.8  | 3.47 | 1.4  |
| (N : 0.7 – 1.2<br>mg/dL) |      |      |      |      |      |      |

### Discussion

To the best of our knowledge, this is the fifth reported case of TTP associated with hepatitis E, and the second case involving a severely affected male patient who was diagnosed with TTP using an ADAMTS13 test. In the present case, the patient in the ICU received close monitoring and comprehensive treatment, including ADAMTS13 testing, assessment of liver function, kidney function, cardiac function and multiple blood transfusions. During this period, the patient also required ventilatory support for several days. After multiple sessions of plasma exchange and weekly rituximab administration, the patient exhibited progressive improvement and remained in good health two weeks after discharge. The patient also resumed his previous urine output and was off dialysis after 15 days of discharge. The association between hepatitis E infection and severe TTP remains ambiguous, possibly implicating an immune-mediated mechanism and warranting further investigation.

### Conclusion

Patients with severe liver dysfunction, severe kidney dysfunction and progressive fall of haemoglobin with evidence of hemolysis should be promptly admitted to the ICU for comprehensive treatment and continuous monitoring of relevant indicators. The importance of ADAMTS13 testing in patients with hepatitis and thrombocytopenia cannot be ignored as these can be a manifestation of TTP that altogether needs a different and aggressive approach.







# Common presentation of a rare disorder- 24 hydroxylase deficiency

### Dr. Himanshu Sharma

Nephrolithiasis is a common presenting complaints in our practice. Recurrence of nephrolithiasis warrants All patients should undergo a focused history, radiologic imaging and biochemical stone analysis with further evaluation for other metabolic causes.

One of the rare conditions that presents with hypercalcaemia and recurrent renal calculi is 24 hydroxylase enzyme deficiency. The 24-hydroxylase enzyme breaks down the active form of vitamin D, called 1,25-dihydroxyvitamin D3 or calcitriol, to an inactive form when the vitamin is no longer needed. The enzyme also breaks down 25-hydroxyvitamin D (also known as calcidiol), which is the form of vitamin D that is stored in the body.

A deficiency of this enzyme's function impairs the breakdown of calcitriol. The resulting excess of calcitriol increases calcium absorption into the bloodstream, causing hypercalcemia. Dysregulation of calcium absorption in the kidneys leads to hypercalciuria, nephrocalcinosis, and nephrolithiasis.

24 hydroxylase deficiency should be considered as a differential diagnosis especially in patients with family history of renal calculi.









### **CBD** Stricture

Dr. Kunal Das

26 year female one month after laparoscopic cholecystectomy presented with jaundice & right upper abdominal pain with tenderness in right upper quadrant of the abdomen on examination. Laboratory data on arrival showed hyperbilirubinemia and elevation of biliary enzymes.

Diagnostic imaging modalities including abdominal ultra-sonogram & MRCP suggested a stone in distal CBD with proximal obstructive biliopathy.

Jaundice subsided after ERCP which revealed distal CBD stone (could not be retrieved) with distal CBD stricture and CBD stenting was done and patient improved. Later when she came for stent removal, cholangiogram still revealed a distal CBD stricture and a fully covered self-expandable metallic stent (FCSEMS) was placed. Later when she came for FC SEMS stent removal, cholangiogram still revealed a distal cbd stricture.

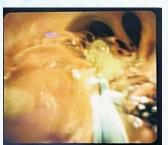
Suspicious of the stricture a spy glass cholangioscopy was done which revealed small impacted stone in the distal CBD which we are able to successfully remove it.

Spy glass Cholangioscopy is a novel and important option for treatment of CBD stone and in evaluation of indeterminate biliary strictures apart from other uses. In this we reported a case where we innovatively used the spy glass cholangioscopy in resolution of distal CBD stricture and hence avoided surgery.

**Keywords:** CBD stone, CBD stricture, ERCP (endoscopic retrograde cholangiopancreatography), SPY GLASS cholangioscopy.







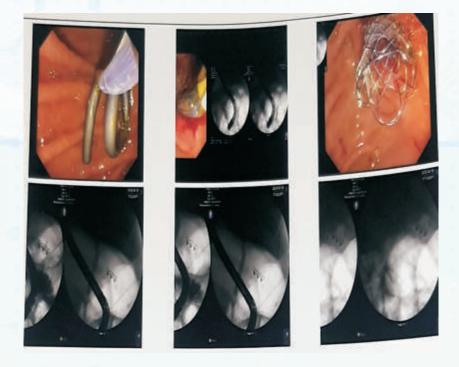
Spy glass cholangioscopy images revealing a small stone.

### EDPA NEWSLETTER





MRCP revealing choledocholithiasis (10mm Calculus in distal 1/3rd of CBD), CBD dilated & IHBRD with proximal obstructive biliopathy.



ERCP images showing distal CBD benign biliary stricture and FC SEMS stent insitu.







### Case Report of Hypokalemic Periodic Paralysis

Dr. Pankaj Nand Chaudhary

### SJOGREN'S SYNDROME PRESENTING AS HYPOKALEMIC PARALYSIS SECONDARY TO DISTAL RTA

### Prepared by -

Dr. Aleena Ivan Theodore, Dr. P.N. Choudhary, Dr. Ashok Grover, Dr. Neha Sharma

### ABSTRACT

Hypokalemic periodic paralysis is a rare muscular disorder characterized by episodic muscle weakness that can lead to respiratory failure. This disorder is a common manifestation of renal tubular acidosis. Renal tubular acidosis can occur associated with various systemic disorder such as Sjogrens syndrome and thyroid disorders.

### **INTRODUCTION**

Distal RTA is a known cause of hypokalemia, which may rarely be severe enough to present as hypokalemic periodic paralysis. Sjogrens syndrome presenting for the first time with hypokalemic paralysis due to distal RTA in a patient with no sicca symptoms is even rarer.

### CASE

A 29 year old female presented to emergency room with c/o weakness in all 4 limbs which was insidious in onset and it gradually progressed starting from the upper limbs to involve the lower limb as well. She also had pain along with weakness gradually the patient was not able to stand or walk. Not able to get up from sitting position. Not able to hold things in hand or comb hair. No h/o any fever, no h/o any difficulty in swallowing, no breathing difficulty, no difficulty in moving neck or facial muscles, no bladder bowel involvements. On physical examination. Patient was moderately built and nourished. No pallor, icterus, clubbing, cyanosis,lymphadenopathy or edema. Vitals -BP 120/70 mm Hg, PR- 82 bpm, T 97.8F, RR 16/min JVP not elevated. On CNS examination patient was conscious oriented to time place person, following commands.CRANIAL NERVE EXAMINATION - was within normal limits. Bulk- normal,Tone – U/L / LL hypotonia,Power – B/LUL- 2/5 LL-2/,Coordintaion – not able to assess. Reflexes Biceps - 1+, triceps –1+, supinator- 1+,Ankle – 0,Abdominal reflex- present,Knee reflex - 1+,plantar- B/L flexor







.Sensory system examination – was within normal limits . No signs of meningeal irritation. Rest of the systemic examination was within normal limits . Patient was admitted and initial routine lab tests were send, we got a call from lab informing that her potassium is - 1.8. ABG showed severe metabolic acidosis .PH- 7.168 / pco2- 18.8 / hco3- 6.9 / Na- 141/ k- 2.3 / Cl- 132 – Anion gap- 2. Gradually weakness increased and patient had difficulty in even moving neck and chewing – RT insertion was done. Nephrology , neurology and rheumatology opinion was taken and was advised to send ANA and vasculitis profile , urine spot electrolytes were send .In v/o low potassium patient was started on with Inj KCL at 10 MEQ / HR along with oral potassium citrate. Urine electrolytes send was – WNL, thyroid profile was sent – WNL. ANA came to be positive. Vasculitis panel- showed ->SS-A /Ro60 , SS-A /Ro52 /SS- B /La positive .Patient gradually started to improve after giving potassium supplementation and power of both right and left UL and LL were 5/5 after 3 days and she was discharged.

The patient was diagnosed with Hypokalemic periodic paralysis secondary to Distal RTA associated with Sjogrens syndrome.

#### DISCUSSION

Hypokalemic periodic paralysis is a common manifestation of renal tubular acidosis . Renal tubular acidosis is defined as the inability of the renal tubules to acidify urine. Under conditiond of normal or near normal GFR. There are 4 types of RTA - type 1, 2, 3, and 4. Type 1 is also called distal RTA, it is the most common type . Distal RTA can be caused by primary or secondary cause. The most common acquired secondary caused of distal RTA is autoimmune disease sjogrens syndrome , SLE, Rheumatoid arthritis, hypothyroidism . ANA comes positive and patient will be positive for SS-A /Ro60, SS-A /Ro52 /SS- B /La antibodies in case of sjogrens syndrome .Sjogrens syndrome is a chronic autoimmune disorder characterized by lymphocyte infiltration in salivary and lacrimal glands .Extra glandular involvement includes neurological, renal, hepatic, cutaneous, respiratory and vascular . The prevalence of RTA is 4.5 – 9 % in sjogrens syndrome. The most common electrolyte abnormality in distal RTA is hypokalemia occurring in 28-53% of patients.

#### **CONCLUSION**

Mild asymptomatic renal disease is common in Sjogrens Syndrome, although hypokalemic paralysis and at times respiratory failure can be initial presentation. It is imperative to keep the possibility of Sjogrens syndrome while evaluating a patient with hypokalemic paralysis.

#### REFERENCES

- Muthukrishnan J, Dawra S, Marwaha V, Narayanan CS. Sjögren's syndrome presenting as hypokalemic paralysis. Med J Armed Forces India. 2015 Jul;71(Suppl 1):S172-4. doi: 10.1016/j.mjafi.2013.11.005.Epub 2014 Jan 25.PMID: 26265821; PMCID: PMC4529555.
- Louis-Jean S, Ching PR, Wallingford A. Distal Renal Tubular Acidosis in Sjögren's Syndrome: A Case Report. Cureus. 2020 Oct 15;12(10):e10962. doi: 10.7759/cureus.10962. PMID: 33083163; PMCID: PMC7567320.







### Allergic Bronchopulmonary Aspergillosis[ABPA]

Dr. Pankaj Nand Chaudhary

**Prepared by** – Dr. P.N. Choudhary Dr. Ashok Grover Dr. Kriti Mishra

Allergic bronchopulmonary aspergillosis (ABPA) is a hypersensitivity reaction to Aspergillus fumigatus, primarily affecting individuals with underlying lung conditions such as asthma or cystic fibrosis. Here, we present a case report of a patient diagnosed with ABPA, detailing the clinical presentation, diagnostic workup, management, and outcome.

### **INTRODUCTION:**

Allergic bronchopulmonary aspergillosis (ABPA) is a complex pulmonary disorder characterized by an exaggerated immune response to Aspergillus fumigatus antigens. It predominantly occurs in patients with underlying lung diseases such as asthma and cystic fibrosis. Despite being recognized for several decades, ABPA remains a challenging condition to diagnose and manage due to its variable clinical presentation and overlap with other respiratory conditions. We report a case of ABPA to highlight its clinical features, diagnostic challenges, and therapeutic strategies.

### **CASE PRESENTATION:**

56years old female was referred to the internal medicine OPD from ophthalmology OPD in view of uncontrolled blood sugars , as the patient was planned for surgical procedure in view of diabetic retinopathy.Patient was a known case of type 2 DM and was on multiple OHAs.Patient old prescription showed diagnosis of COPD and was managed at home with nebulizers by her husband who is an alternative medicine practitioner. On further questioning Patient c/o breathlessness on exertion and mild cough with expectoration.On General physical examination -Pallor + , no icterus/ no clubbing / no lymphadenopathy /no cyanosis /no edema.The patient was afebrile , had heart rate of 102/min ,respiratory rate of 22 breaths per min ,blood pressure of 112/72 MMHG and O2 saturation of 94% on room air. On respiratory examination , there was equal chest expansion on both side of chest , no tracheal deviation and spine was central. No localised tenderness over the chest. On auscultation patient had occasional rhonchi.Other systemic examinations were unremarkable.In view of uncontrolled blood sugars patient was admitted and was started on with initial symptomatic treatment. As the patient was





planned for surgery , all routine investigations were send including chest x ray.Haematological investigations revealed HB-9.4 g% ,Platelets count –367k ,CRP—5.3,Total leucocyte counts 9600/mm3 , Neutrophils 51.7 % , Lymphocytes 27.9% , Eosinophils 10%.Chest x ray showed Homogeneous opacity noted in the right upper / mid zones Soft tissue density area noted in the right cardio phrenic angle.High resolution computed tomography [HRCT] showed Hyperdense secretion noted in the posterior segmental bronchus of right upper lobe with collapse / consolidation of the posterior segment of right upper lobe.Discrete and confluent nodular opacities with tree in bud appearance noted in the superior segment of right lower lobe.No pleural / fissural thickening seen. No pleural / pericardial effusion.INVESTIGATIONS--Total IgE Levels: >5000,Galactomannan : POSITIVE[0.57],Aspergillus-specific IgE: POSITIVE [ 40.4,normal-0-0.34] ,Aspergillus Antibody , IgG : POSITIVE [32.59 , >11 NTA –POSITIVE]Serological testing for Aspergillus-specific IgE and IgG antibodies was positive, confirming the diagnosis.

#### **TREATMENT AND OUTCOME:**

The patient was initiated on a multidisciplinary treatment approach involving oral corticosteroids, inhaled bronchodilators, and antifungal therapy with itraconazole. Additionally, environmental control measures were emphasized to minimize exposure to Aspergillus spores. Over the following weeks, the patient's symptoms improved significantly, with resolution of wheezing and reduction in dyspnea. Serial monitoring of serum IgE levels and peripheral eosinophil counts guided the tapering of oral corticosteroids.

#### **DISCUSSION:**

This case underscores the importance of considering ABPA in the differential diagnosis of patients with refractory asthma, particularly in the presence of characteristic radiological findings such as central bronchiectasis and mucoid impaction. Early recognition and prompt initiation of treatment are essential to prevent disease progression and minimize long-term complications. Longitudinal follow-up is crucial to assess treatment response and adjust therapeutic interventions accordingly.

#### **CONCLUSION:**

ABPA is a challenging pulmonary disorder characterized by immune-mediated hypersensitivity to Aspergillus fumigatus antigens. Clinicians should maintain a high index of suspicion for ABPA in patients with uncontrolled asthma, especially in the presence of compatible radiological findings and elevated serum IgE levels.

#### REFERENCE

- Reference 1: Shah A, Panjabi C. Allergic aspergillosis of the respiratory tract. European Respiratory Review. 2014; 23: 8.http://err.ersjournals.com/content/23/131/8.abstract
- Reference 2: Agarwal R, Garg M, Aggarwal AN, Saikia B, Gupta D, Chakrabarti A. Serologic allergic bronchopulmonary aspergillosis (ABPA-S): Long-term outcomes. Respiratory Medicine. 2012; 106:942-947.https://doi.org/10.1016/j.rmed.2012.03.001





The East Delhi Physicians Association's Annual Conference, EDPACON 2023, held on 17th December 2023 at Le Meridien, New Delhi, was a resounding success. With the theme **"Advances & Innovations in Medicine & Allied Specialties**" the conference brought together leading healthcare professionals to discuss cutting-edge developments in the field. Renowned speakers shared insights on emerging technologies, breakthrough treatments, and interdisciplinary collaborations, fostering a dynamic exchange of ideas. Attendees gained valuable knowledge and networking opportunities, enhancing their practice and patient care. EDPACON 2023 showcased the commitment of East Delhi Physicians Association to advancing healthcare through continuous learning and innovation. Here are the few Glimpses of the EDPACON 2023....

















### East Delhi Physicians Association

E-mail : eastdelhiphysiciansassociation@gmail.com Website : www.edpadelhi.com