

Scientific Paper Presentation Schedule of MAPICON 2026			
Poster Paper Presentation Schedule: 17.9.2026 (Thursday) Day 1.			
Morning Session: 9.00am to 10.10am			
No	Time	Name	College
PP-1	9.00am to 9.10am	Anshul Nagpal	GGSMC&H, Faridkot
PP-2	9.10am to 9.20am	Harsumeet Kaur Brar	GGSMC&H, Faridkot
PP-3	9.20am to 9.30am	Himanshu Garg	GGSMC&H, Faridkot
PP-4	9.30am to 9.40am	Himanshu Soni	GGSMC&H, Faridkot
PP-5	9.40am to 9.50am	Kalkatte Yogesh Shivajirao	GGSMC&H, Faridkot
PP-6	9.50am to 10.00am	Pankaj Swami	GGSMC&H, Faridkot
PP-7	10.00am to 10.10am	Prashant Sagar	GGSMC&H, Faridkot
Oral Paper Presentation Schedule: 17.9.2026 (Thursday) Day 1.			
Evening Session: 5.00pm to 5.20pm			
OP-1	5.00pm to 5.10pm	Daksh Kotru	MMIMSR, Mullana
OP-2	5.10pm to 5.20pm	Mitali Aggarwal	MMIMSR, Mullana
Poster Paper Presentation Schedule: 17.9.2026 (Thursday) Day 1.			
Evening Session: 5.20pm to 6.10pm			
PP-8	5.20pm to 5.30pm	Pooja Batra	MMIMSR, Mullana
PP-9	5.30pm to 5.40pm	Rupal Sharma	MMIMSR, Mullana
PP-10	5.40pm to 5.50pm	Sachit Khurana	MMIMSR, Mullana
PP-11	5.50pm to 6.00pm	Saumya Jolly	MMIMSR, Mullana
PP-12	6.00pm to 6.10pm	Sonia Saini	MMIMSR, Mullana
Oral Presentation Schedule: 18.9.2026 (Friday) Day 2.			
Morning Session: 9.00am to 9.20am			
OP-3	9.00am to 9.10am	Taranjot Kaur	MOH, Ludhiana
OP-4	9.10am to 9.20am	Gurjot Singh Gill	MGMHC&H, Kishanganj
Poster Paper Presentation Schedule: 18.9.2026 (Friday) Day 2.			
Morning Session: 9.20am to 10.00am			
PP-13	9.20am to 9.30am	Taranjot Kaur	MOH, Ludhiana
PP-14	9.30am to 9.40am	Komalpreet Kaur	
PP-15	9.40am to 9.50am	Gurjot Singh Gill	MGMHC&H, Kishanganj
PP-16	9.50am to 10.00am	Mansi	MOH, Ludhiana.
Paper Presentation Schedule: 19.9.2026 (Saturday) Day 3.			
Morning Session: 9.00am to 10.10am			
PP-19	9.00am to 9.10am	Armaandeep Kaur	AIIMS, Bathinda
PP-20	9.10am to 9.20am	Armaandeep Kaur	AIIMS, Bathinda
OP-5	9.20am to 9.30am	Shubhneet kaur	AIMSR, Bathinda
PP-21	9.30am to 9.40am	Harshman	AIMSR, Bathinda
PP-17	9.40am to 9.50am	Radhika	GGSMC&H, Faridkot
PP-18	9.50am to 10.00am	Amarinder Singh	GGSMC&H, Faridkot
PP- 22	10.00am to 10.10am	Aakash Bansal	GGSMC&H, Faridkot
PP = Poster Presentation, OP = Oral Presentation			



**13th Annual Conference of the
Association of Physicians of India**
(Malwa Branch)

MAPICON 2026

17th - 20th September 2026

Hotel Ginger, Opp. Lake No. 3, G.T. Road, Bathinda

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Scientific Programme & Abstract Book

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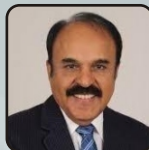
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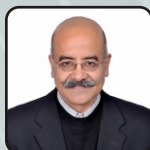
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Scientific Programme, 17-09-2026, Thursday (Day-1)

9:00am to 10:00am : Registration & Tea

9:00am to 10:20am : Scientific Paper Presentations

General Session

Moderator: K. K. Nohria

10.20am to 10.40am	Chander Shekhar Kakkar	Malaria: An Overview
10.40am to 11.00am	Selvakumar Naganathan	Liver Transplantation: Current Status in India
11.00am to 11.20am	Sumit Pal Singh Chawla	Approach and Management of Ascites
11.20am to 11.40am	Gurpreet S Wander	Indian Hypertension Guidelines: What is New in 2026?
11.40am to 12.00pm	B K Aggarwal	Thrombocytopenia in Pregnancy- Common Causes and Management
12.00pm to 1.00pm	Inauguration	

General Session

Moderator: M.R. Gupta

1.00pm to 1.20pm	Puneet Saxena	Role of Glucagon in Management of Obesity
1.20pm to 1.40pm	Arvind Gupta	Remission of Metabolic Disorders with Yoga and Lifestyle: Is It Possible?
1.40pm to 2.00pm	Ravinder Garg	Glycaemic Variability: What, Why & How?
2.00pm to 3.00pm	Lunch	

General Session

Moderator: Ravinder Maheshwari

3.00pm to 3.20pm	Salil Jaura	DOACs in Clinical Practice: Evidence to Excellence
3.20pm to 3.40pm	Amit Taneja	Antibiotic Resistance and Antibiotic Stewardship
3.40pm to 4.00pm	Parmod Kumar Goyal	Landmark and Recent Court Judgments Relevant to Medical Professionals: A Practical Overview
4.00pm to 4.20pm	Rajni Jindal	Recent Advances in IVF
4.20pm to 4.40pm	Monika Gupta	Preparation of Human Bones for Osteology Teaching: A Stepwise Protocol
4.40pm to 5.00pm	Aashana Goel	Oral Health in Children
5.00pm to 6.20pm	Scientific Paper Presentations	

Scientific Programme, 18-09-2026, Friday (Day-2)

9:00am to 10:00am : Registration & Tea

9:00am to 10:00am : Scientific Paper Presentations

Cardiology Session

Moderator: K. K. Nohria

10.00am to 10.20am	Madan Mohan Bansal	Management of Chronic Stable Angina
10.20am to 10.40am	Rakendra Singh	Coronary Artery Disease in Young
10.40am to 11.00am	Rajesh Jindal	Management of Pulmonary Artery Hypertension: An Update
11.00am to 11.20am	Arvind Bansal	Prevention of Sudden Cardiac Death in Young
11.20am to 11.40am	Sonu Sharma	Management of Resistant Hypertension
11.40am to 12.00pm	Usha G	Hypertensive Disorders of Pregnancy

General Session

Moderator: Ashok Goyal

12.00pm to 12.20pm	Hari Kishan Boorugu	Overview of Liver Disorders In Pregnancy
12.20pm to 12.40pm	Abhishek Garg	Headache in General Practice : An Overview
12.40pm to 1.00pm	Shifali Gupta	Relevance of Genetic Testing in Medical Practice
1.00pm to 1.20pm	Arun Bansal	Psychological Well-Being: An Ignored Entity
1.20pm to 1.40pm	Deepak Garg	Current Trends in Limb Salvage Surgery for Bone Cancer
1.40pm to 2.00pm	Puneet Kumar	Mildly Raised LFT in OPD: Ignore, Monitor or Investigate?
2.00pm to 3.00pm	Lunch	

Research Society for the Study of Diabetes in India (RSSDI) Session

Moderator: K. K. Nohria

3.00pm to 3.20pm	Ankit Chhabra	The Ozempic Edge in T2DM
3.20pm to 3.40pm	Manveer Gupta	Essentials of Wellness in Elderly
3.40pm to 4.00pm	Rohit Aggarwal	Type-5 diabetes / MRDM
4.00pm to 4.20pm	Vipin Talwar	Medical management of Obesity Beyond Glp-1 Agonists
4.20pm to 4.40pm	Naveen Mittal	One Hour Postprandial/Post Glucose Challenge: Why it is Important ?

Scientific Programme, 19-09-2026, Saturday (Day-3)

9:00am to 10:00am : Registration & Tea

9:00am to 10:00am : Scientific Paper Presentations

General Session

Moderator: K. K. Goyal

10.00am to 10.20am	Neha Gupta	Advances in Systemic Cancer Treatment
10.20am to 10.40am	Sandeep Suri	Altered Chrono Biology & Diabetes
10.40am to 11.00am	Anisha Sharma	Aesthetic Dermatology: Beyond Fillers and Toxins
11.00am to 11.20am	Anmol Sharma	Stem Cell Therapy in Knee OA: Hype or Hope?
11.20am to 11.40am	Hari Om Aggarwal	Rational Understanding and Prevention of Osteoporosis

Clinical Cardio Diabetic Society of India (CCDSI) Session

Moderator: Vitull K Gupta

11.40am to 12.00pm	Inauguration of Mid Term CCDSI Conference	
12.00pm to 12.20pm	A N Rai	Interesting Clinical Cases presenting as Prolonged QT Interval
12.20pm to 12.40pm	Ashish K Saxena	Cardio Protection in Diabetes
12.40pm to 1.00pm	Ajay Kumar Sinha	Past, Present and Future of AF management
1.00pm to 1.20pm	Abhijeet Kumar	What Happens When You Stop Taking GLP-1 RA's ?
1.20pm to 1.40pm	Mohammed Shafat Imam Siddiqui	MASLD in Diabetes Practice
1.40pm to 2.00pm	Deepak Das	Prediabetes: Who Progresses and Who Reverts?
2.00pm to 3.00pm	Lunch	

Innovative Physicians Forum (IPF) Session

Moderator: M.R. Gupta

3.00pm to 3.20pm	Ritu	Update on Heart Failure Management in T2DM
3.20pm to 3.40pm	Ved Prakash Kalra	Cancer of Oesophagus: An Overview
3.40pm to 4.00pm	Jugal Kishor Sharma	Autonomic Dysfunction in Diabetes
4.00pm to 4.20pm	Anil Manchanda	Black and White of Adipose Tissue
4.20pm to 4.40pm	Puneet Khanna	Pulmonary Complications of Obesity
4.40pm to 5.00pm	Parvinder Kaur	Oral Health & Diabetes

Scientific Programme, 20-09-2026, Sunday (Day-4)

9:00am to 10:00am : Registration & Tea

General Session

Moderator: Vitull K Gupta

10.00am to 10.20am	Shaurya Goyal	MASLD in India: An Overview
10.20am to 10.40am	Baldev Aulakh	Robotic Revolution in Uro-Oncology, Urology & Transplantation
10.40am to 11.00am	Parshant Aggarwal	Effective Communication: The Key to Better Care
11.00am to 11.20am	Bishav Mohan	Innovation in Public Health: Humble Beginning
11.20am to 11.40am	Rajoo Singh Chinna	Fatty Liver Disease: What to Do?
11.40am to 12.00pm	Virendra Singh	Alcoholic Liver Disease: An Overview
12.00pm to 12.40pm	Felicitation Ceremony	

Lipid Association of India (LAI) Session

Moderator: K.K. Nohria

12.40pm to 1.00pm	Raman Puri	ASCVD Risk Calculator for Indian Population
1.00pm to 1.20pm	Charanjit Singh	Lifestyle Modification in Dyslipidemia Management
1.20pm to 1.40pm	Vimal Mehta	Management of Dyslipidemia in ACS
1.40pm to 2.00pm	Mani Kant Singla	Management of Diabetic Dyslipidemia
2.00pm to 2.20pm	Jagroop Singh	Dengue Fever : An Overview
2.20pm to 3.00pm	Lunch	

AIIMS Session

Moderator: Vitull K Gupta

3.00pm to 3.20pm	Avneet Garg	Allergic Bronchopulmonary Aspergillosis (ABPA): An Overview
3.20pm to 3.40pm	Vaibhav Saini	Initiative on Cochlear Implants : An Overview
3.40pm to 4.00pm	Rajiv Gupta	CABG Vs. PTCA
4.00pm to 4.20pm	Monica Kakkar	AI in Lab: A Revolutionary Inclusion
4.20pm to 4.40pm	Rakesh Kakkar	National Guidelines for Snakebite Management
4.40pm to 5.00pm	Anupinder Thind	Essential Communication skills for Healthcare Professionals in Healthcare Management
5.00pm	Delegate Certificate Distribution, Valedictory Function & Vote of Thanks	

Scientific Paper Presentation Schedule

Poster Paper Presentation Schedule: 17.9.2026 (Thursday) Day 1 Morning Session: 9.00am to 10.10am			
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OP-4	9.10am to 9.20am	Gurjot Singh Gill	MGMHC&H, Kishanganj
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PP-14	9.30am to 9.40am	Komalpreet Kaur	
PP-15	9.40am to 9.50am	Gurjot Singh Gill	MGMHC&H, Kishanganj
PP-16	9.50am to 10.00am	Mansi	MOH, Ludhiana
Oral Paper Presentation Schedule: 19.9.2026 (Saturday) Day 3 Morning Session: 9.50am to 10.00am			
OP-5	9.30am to 9.40am	Shubhneet Kaur	AIMSR, Bathinda
PP-17	9.40am to 9.50am	Radhika	GGSMC&H, Faridkot
PP-18	9.50am to 10.00am	Harshman	AIMSR, Bathinda

ORAL PRESENTATIONS

ABSTRACTS

OP-1 The Hemoglobin Gradient: Anemia as a Clinical Marker of Hepatic Decompensation in Liver Cirrhosis

Daksh Kotru, Yogesh Garg, Bimal K. Agrawal. Dept. of General Medicine,
MM Institute of Medical Science and Research, MM Deemed to be University,
Mullana-133207, Haryana

Background: Anemia in cirrhosis is multifactorial, reflecting gastrointestinal blood loss, hypersplenism, chronic inflammation, nutritional deficiency, and marrow suppression. Its relationship with hepatic functional reserve may provide useful clinical information.

Objective: To assess the prevalence, morphological and iron profile of anemia in cirrhosis and its association with Child-Pugh class, splenomegaly, and etiology.

Methods: A cross-sectional hospital-based study included 100 adults with cirrhosis. Hemoglobin, RBC indices, peripheral smear, iron profile, transferrin saturation, ferritin, and Child-Pugh score were assessed. CKD, malignancy, pregnancy, pre-existing hematological disease, and prior anemia treatment were excluded.

Results: Anemia was present in 77% of patients: mild 15%, moderate 40%, and severe 22%. Mean hemoglobin was 9.99 +/- 2.99 g/dL. Macrocytosis was most frequent (31%), followed by microcytic (28%) and normocytic (17%) patterns. Mean transferrin saturation was low at 14.6 +/- 8.1%, despite mean ferritin of 215.1 +/- 218.9 ng/mL. Hemoglobin declined progressively across Child-Pugh classes A, B, and C: 10.67, 9.50, and 7.83 g/dL respectively ($p < 0.01$). Greater anemia severity was associated with splenomegaly ($p = 0.002$) and alcoholic etiology ($p = 0.038$).

Conclusion: Anemia severity shows a graded association with hepatic decompensation. Hemoglobin may serve as a simple, low-cost adjunctive marker of worsening liver function, while ferritin alone may inadequately characterize iron status in cirrhosis.



OP-2 Clinical Profile of Patients Presenting with Upper GI Bleed at a Tertiary Care Hospital

Mitali Aggarwal, Bimal K Agrawal, Nayankumar Amrutiya. Dept. of General Medicine, MM Institute of Medical Science and Research, MM Deemed to be University, Mullana-133207, Haryana

Background: Upper gastrointestinal bleeding (UGIB) is a potentially life-

threatening emergency with variceal and non-variceal causes. Its clinical and endoscopic profile varies with underlying risk factors and patient population.

Objective: To study the clinical profile, etiological spectrum, laboratory parameters, ultrasonographic findings, and endoscopic characteristics of patients presenting with UGIB at a tertiary care hospital.

Methods: A prospective observational study was conducted in 100 adults presenting with hematemesis and/or melena at MMIMSR Mullana from November 2024 to May 2026. Patients with lower gastrointestinal bleeding or bleeding disorders were excluded. Clinical evaluation, laboratory investigations, abdominal ultrasonography, and upper gastrointestinal endoscopy were performed. Data were analysed using SPSS version 31.

Results: Males constituted 77% of patients, with ≥ 60 years being the largest age group (31%). Melena was the commonest presentation (73%), followed by hematemesis (44%). Chronic liver disease (CLD) was the predominant comorbidity (71%); alcohol use and smoking were present in 41% and 40%, respectively. Mean haemoglobin was 8.99 ± 2.16 g/dL, platelet count $112.12 \pm 99.43 \times 10^3/\mu\text{L}$, and INR 1.52 ± 0.49 . Ultrasonography showed CLD and ascites in 71% each, with moderate ascites being most frequent (38%). Endoscopy revealed esophageal varices in 71%, including large varices in 41%; 36% had variceal bleeding requiring EVL. Gastric and duodenal ulcers were found in 16% and 10%, respectively.

Conclusion: UGIB predominantly affected males and was strongly associated with CLD and portal hypertension, with esophageal varices being the leading endoscopic finding. Early stabilization and timely endoscopic evaluation are essential for optimal management.



OP-3 Incremental Prognostic Value of the Clinical Frailty Scale beyond APACHE II and SOFA in Critically Ill Adults

Taranjot Kaur. 2nd year Resident, Dept. of General Medicine,
Mohandai Oswal Hospital, Ludhiana, Punjab

Background: APACHE II and SOFA quantify acute physiological derangement and organ dysfunction but may not capture pre-existing physiological reserve. The Clinical Frailty Scale (CFS) may provide complementary prognostic information.

Objective: To evaluate the incremental prognostic value of CFS for in-hospital mortality beyond APACHE II and SOFA.

Methods: We analysed 70 critically ill adults from a prospective ICU cohort. CFS, APACHE II and SOFA were assessed at ICU admission. In-hospital mortality was the primary outcome. Multivariable logistic regression assessed the association of CFS with mortality after adjustment for APACHE II and SOFA. Model discrimination was evaluated using AUROC, with incremental performance assessed by likelihood-ratio testing. Bootstrap

resampling was used to assess model performance.

Results: Twenty-five patients (35.7%) died during hospitalization. Median APACHE II, SOFA and CFS scores were 15, 8.5 and 4, respectively. Non-survivors had higher APACHE II (17 vs 14; $p=0.025$) and SOFA scores (10 vs 8; $p=0.043$). AUROC increased from 0.653 for APACHE II plus SOFA to 0.688 after addition of CFS (Δ AUROC 0.035). CFS had an adjusted OR of 1.16 per one-point increase (95% CI 0.89–1.51; $p=0.275$). Addition of CFS did not significantly improve model fit ($\chi^2=1.215$; $p=0.270$).

Conclusion: CFS demonstrated modest incremental prognostic value beyond APACHE II and SOFA, although the improvement was not statistically significant. Larger prospective studies are warranted.



OP-4 Type 3c Diabetes Mellitus Secondary to Chronic Calcific Pancreatitis: Ten-Patient Retrospective Case Series from a Tertiary Care Teaching Hospital in Eastern India

Gurjot Singh Gill, Taranjot Singh, P S Karmakar.
Mata Gujri Medical College & Hospital, Kishanganj

Background: Type 3c diabetes mellitus (T3cDM), or pancreatogenic diabetes, is an under-recognized form of secondary diabetes resulting from exocrine pancreatic disease. Clinical overlap with type 2 diabetes mellitus (T2DM) frequently results in misclassification and delayed appropriate management. Indian data on T3cDM remain limited.

Objective: To describe the demographic, clinical, radiological, metabolic, complication, and treatment profiles of patients with T3cDM secondary to chronic calcific pancreatitis.

Methods: This retrospective case series included 10 consecutive patients diagnosed with T3cDM secondary to chronic calcific pancreatitis were studied. Clinical, laboratory, imaging, ophthalmological, treatment, and outcome data were reviewed. T3cDM was diagnosed in patients with diabetes and structural pancreatic abnormalities consistent with chronic pancreatitis after exclusion of other common causes.

Results: Ten patients (7 males, 3 females) with a median age of approximately 40 years (range, 23–55 years) were included. Chronic abdominal pain and poor glycaemic control were predominant presentations. Imaging demonstrated pancreatic calcification, atrophy, and/or main pancreatic duct dilatation in all patients. HbA1c ranged from 4.8% to 15.8%, demonstrating marked glycaemic heterogeneity. Peripheral neuropathy and diabetic retinopathy were the most frequent complications. Exocrine pancreatic insufficiency requiring enzyme replacement was observed in several patients. Insulin was the primary treatment in all patients, with selected patients receiving oral antihyperglycaemic agents.

Conclusion: T3cDM remains frequently under-recognized and misclassified as T2DM. In diabetic patients with recurrent abdominal pain, pancreatic

calcification, glycaemic variability, or exocrine dysfunction, pancreatic imaging should be considered to facilitate accurate classification and comprehensive endocrine and exocrine management.



OP-5 Outcomes of Non-Invasive Ventilation in Patients of Acute Respiratory Failure in Respiratory Care Unit of a Tertiary Care Hospital of Malwa Region in North India

Shubhneet Kaur, 2nd year PG Resident, Adesh University
of Medical Sciences, Bathinda

Background: Acute respiratory failure (ARF) is a life-threatening condition characterized by inadequate oxygenation and/or ventilation. Non-invasive ventilation (NIV) provides positive-pressure ventilatory support without endotracheal intubation and can improve gas exchange while reducing the need for invasive mechanical ventilation. Outcomes may vary according to the type and etiology of respiratory failure.

Aim: To evaluate the effectiveness and outcomes of NIV in patients with acute respiratory failure and compare outcomes between Type I and Type II respiratory failure.

Methods: This hospital-based prospective longitudinal study was conducted in the Department of Respiratory Medicine, Adesh Hospital, AIMSR, Bathinda. Sixty patients aged >18 years with acute respiratory failure due to primary pulmonary pathology requiring NIV were included, with 30 patients each having Type I and Type II respiratory failure. Patients received BiPAP-based NIV and were monitored clinically and by serial arterial blood gas analysis at 0, 4, 12, 24, 48 and 72 hours. HACOR score was assessed at one hour. NIV success, intubation, mortality, duration of NIV and hospital stay were evaluated.

Results: Pneumonia was the predominant cause of Type I respiratory failure (43.3%), whereas COPD exacerbation predominated in Type II respiratory failure (56.7%). Overall NIV success was 75%. NIV success was significantly higher in Type II than Type I respiratory failure (86.7% vs. 63.3%, $p=0.038$), with lower NIV failure requiring intubation (13.3% vs. 36.7%). In Type II patients, PaCO_2 decreased from 67.1 ± 12.9 mmHg at baseline to 48.4 ± 7.7 mmHg at 24 hours. Hospital stay was significantly shorter in Type II patients (7.1 ± 3.0 vs. 9.0 ± 4.1 days, $p=0.047$).

Conclusion: NIV was effective in acute respiratory failure, with particularly favourable outcomes in Type II respiratory failure, demonstrating higher success, lower intubation requirement and shorter hospitalization.

POSTER PRESENTATIONS

ABSTRACTS

**PP-1 Tubercular Meningitis with Transverse Myelitis:
A Neurological Conundrum**

Anshul Nagpal, Junior Resident, Dept. of General Medicine,
Guru Gobind Singh Medical College and Hospital, Faridkot

Background: Transverse myelitis is an inflammatory disorder of the spinal cord characterized by motor, sensory, and autonomic dysfunction. Tuberculosis is a rare but important cause, particularly in association with tuberculous meningitis. Early recognition and treatment are essential to prevent permanent neurological disability.

Case Presentation: A 22-year-old male presented with fever for 20 days, urinary retention for 14 days, headache and inability to bear weight for 3 days, followed by altered sensorium. Examination revealed decreased higher mental functions, positive Kernig's and Brudzinski's signs, bilateral lower-limb hypotonia with power 0/5 and absent deep-tendon reflexes. Cerebrospinal fluid analysis showed lymphocytic predominance, markedly elevated protein, low glucose, positive globulin and raised ADA levels. CSF CBNAAT was positive, confirming tuberculosis. Anti-tubercular therapy was initiated, with improvement in fever and consciousness; however, paraparesis persisted. MRI spine was suggestive of transverse myelitis involving the C7–D5 region. Intravenous methylprednisolone followed by oral steroids resulted in significant improvement in lower-limb tone and power.

Conclusion: Tuberculosis should be considered as an important cause of transverse myelitis, especially in patients with tuberculous meningitis. Early diagnosis, appropriate anti-tubercular therapy and timely corticosteroid treatment may improve neurological recovery and reduce permanent disability.



**PP 2 Hypokalemic Paralysis as a Presenting Manifestation of
Primary Hyperaldosteronism: A Case Report**

Harsumeet Kaur Brar, 2nd Year PG Resident, Dept. of General Medicine, Guru
Gobind Singh Medical College and Hospital, Faridkot

Background: Primary hyperaldosteronism (PA) is a potentially curable cause of secondary hypertension. Hypokalemic paralysis is an uncommon manifestation of severe potassium depletion and may be the initial clue to Primary Hyperaldosteronism.

Case Presentation: A 35-year-old previously healthy man, not known to have hypertension, presented with acute-onset bilateral lower-limb weakness for one day. The weakness was flaccid and ascending, with difficulty standing and walking. There was no history of vomiting, diarrhoea, diuretic use or previous similar episodes. On examination, blood pressure was 180/110 mmHg and lower-limb power was 2/5, with reduced tone and diminished deep-tendon reflexes. Investigations revealed severe hypokalemia (K^+ 1.8 mEq/L) with metabolic alkalosis (pH 7.50) and elevated spot urinary potassium (34.90 mEq/L), indicating renal potassium wasting. Plasma aldosterone was 49.8 ng/dL, with suppressed plasma renin activity (0.32 ng/mL/hr) and elevated aldosterone-renin ratio (155.625). CECT abdomen revealed a 9 × 9 mm left adrenal adenoma, with a normal right adrenal gland. Intravenous potassium replacement resulted in significant improvement in muscle weakness.

Conclusion: Hypokalemic paralysis may be the first manifestation of Primary Hyperaldosteronism, even in a patient without previously diagnosed hypertension. The combination of hypertension, severe hypokalemia, metabolic alkalosis and renal potassium wasting should prompt evaluation for mineralocorticoid excess.



PP-3 Alpha Lipoic Acid and Insulin Autoimmune Syndrome:

A Rare Association

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Background: Insulin autoimmune syndrome (IAS) is a rare cause of spontaneous hypoglycemia characterized by hyperinsulinemia and circulating insulin autoantibodies (IAA), usually in individuals without prior exposure to exogenous insulin. Alpha-lipoic acid (ALA), a commonly used antioxidant supplement, has been implicated as a trigger of IAS.

Case Presentation: A 40-year-old woman presented with recurrent fasting and postprandial hypoglycemic episodes associated with neuroglycopenic and autonomic symptoms. She had no history of exogenous insulin use. During hypoglycemia, plasma glucose was 32 mg/dL with markedly elevated insulin (1,100 μ U/mL) and C-peptide (29.7 ng/mL), with positive insulin autoantibodies (>100 U/mL). Evaluation for other causes of hypoglycemia, including endogenous hyperinsulinism, was unrevealing. Thyroid peroxidase antibodies were positive, while other autoimmune, endocrine, hepatic, renal, and infectious evaluations were non-contributory. A detailed medication and supplement history revealed regular use of ALA for approximately four months.

Outcome: ALA was discontinued, dietary modification was advised, and frequent low-carbohydrate meals were recommended. The patient subsequently experienced spontaneous resolution of hypoglycemic episodes, with normalization of insulin autoantibody titers on follow-up.

Conclusion: This case highlights ALA as a potential trigger of insulin autoimmune syndrome. IAS should be considered in patients with unexplained hyperinsulinemic hypoglycemia, particularly when insulin levels are disproportionately high and insulin autoantibodies are positive. Careful review of over-the-counter medications and dietary supplements is essential, as withdrawal of the offending agent may lead to complete resolution.

PP-4 Pancreatic Tuberculosis mimicking Malignancy in a PLHA with HCV Co-infection

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Background: Pancreatic tuberculosis (TB) is a rare form of extrapulmonary tuberculosis that frequently masquerades as pancreatic adenocarcinoma clinically and radiologically. It presents a significant diagnostic challenge, particularly in immunocompromised individuals.

Case Presentation: A 40-year-old male—a known Person Living with HIV/AIDS (PLHA) on antiretroviral therapy (ART) and newly diagnosed, treatment-naïve hepatitis C virus (HCV) reactive—presented with a 2–3 month history of gradual, progressive, non-radiating, moderate epigastric pain. He reported a 12–15 year history of chronic smoking and occasional alcohol use. Abdominal examination was unremarkable. Laboratory evaluations revealed elevated inflammatory markers (ESR: 78mm/1st hr), (CRP: 48 mg/L), (CD4 count: 206 cells/mm³) and a positive QuantiFERON-TB Gold test. Abdominal ultrasonography (USG), CECT, and MRCP identified an ill-defined, hypodense mass in the pancreatic body and tail (4.6cm x 8.2cm x 6.6cm) without major vascular encasement, accompanied by peripancreatic necrotic lymphadenopathy. Ultrasound-guided biopsy of the lesion demonstrated caseating granulomas with surrounding lymphocytic infiltration, confirming tuberculosis.

Management & Outcome: The patient was successfully managed with a standard 6-month antitubercular therapy (ATT) regimen (2 months HRZE followed by 4 months of HRE). Follow-up MRCP performed 8 months after initiating ATT demonstrated significant regression of both the pancreatic mass (2.6cm x 5.3cm x 3.2cm) and the associated necrotic lymphadenopathy.

Conclusion: Pancreatic TB should be maintained in the differential diagnosis of pancreatic masses in endemic regions and immunocompromised patients. Histopathological confirmation via image-guided biopsy is pivotal to establish a diagnosis and prevent unnecessary major surgical interventions.

PP-5 Segmental Zoster Paresis in an Uncontrolled Type 2 Diabetic Patient Presenting With Wrist Drop: A Rare Complication of Herpes Zoster

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Background: Segmental zoster paresis (SZP) is a rare neurological complication of herpes zoster, characterized by focal motor weakness corresponding to the affected dermatome or myotome. Its presentation may mimic cerebrovascular, infectious, cervical spine, or autoimmune disorders, making early recognition important.

Case Presentation: A 65-year-old man with uncontrolled type 2 diabetes mellitus presented with an erythematous and purpuric rash involving the left forearm and palm for approximately two weeks. He received a 10-day course of valacyclovir as an outpatient. On follow-up, he developed left arm pain that progressed to severe burning pain in the left hand, persistent purpuric lesions, and left wrist drop. Examination revealed sensory deficits and marked weakness of the left upper limb, with 0/5 power in wrist flexion and extension, 1/5 in elbow flexion and extension, and 4/5 in shoulder flexion and extension. Diffuse hand swelling, dorsal forearm lesions, and petechiae were also noted.

Investigations: CT cervical spine showed no acute pathology or dislocation. CT brain, cervical MRI, CSF analysis, ultrasonography, and electrophysiological studies were performed. Laboratory investigations and inflammatory markers were unremarkable. The clinical presentation and investigations supported SZP.

Treatment and Outcome: An additional 10-day course of valacyclovir, corticosteroids with tapering, analgesics, and gabapentin was administered for neuropathic pain.

Conclusion: SZP should be considered in patients with herpes zoster and acute focal paresis. Early recognition enables timely treatment and may prevent unnecessary investigations.



PP-6 Hemodynamically Unstable Ventricular Tachycardia: A Rare Primary Presentation of Acute Coronary Syndrome

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Background: Ventricular tachycardia (VT) is a potentially life-threatening arrhythmia that may occur in the setting of acute myocardial ischemia. Hemodynamically unstable VT as the initial presentation of acute coronary syndrome (ACS) is uncommon.

Case Report: A 70-year-old man with no history of diabetes, hypertension, alcohol use, or smoking presented with sudden-onset breathlessness and cold, clammy extremities. He was found to have severe hypotension (SBP 40 mmHg) and tachycardia (HR 194/min), consistent with hemodynamically

unstable VT. Immediate electrical cardioversion with 100 J restored hemodynamic stability and sinus rhythm. Although the patient had no history of chest pain or discomfort, subsequent evaluation revealed ST-segment elevation in the inferolateral leads, elevated CK-MB and troponin-I, and echocardiographic evidence of reduced left ventricular systolic function (LVEF 32%) with regional wall-motion abnormalities. Coronary angiography demonstrated significant 90% proximal left circumflex artery stenosis with TIMI II distal flow. Percutaneous transluminal coronary angioplasty with stent placement was performed, resulting in restoration of TIMI III flow and sustained sinus rhythm. The post-procedure course was uneventful.

Conclusion: Hemodynamically unstable VT can rarely be the primary presentation of ACS, even in the absence of typical chest pain. Prompt recognition, immediate cardioversion, and evaluation for an underlying ischemic etiology are essential. Correction of the underlying coronary pathology remains the cornerstone of definitive management.



PP-7 Addison's Disease with Idiopathic Intracranial Hypertension and Neuropsychiatric Manifestations

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Background: Addison's disease (primary adrenal insufficiency) is a rare endocrine disorder characterized by impaired adrenal production of cortisol and aldosterone. Although fatigue, weight loss, hypotension, electrolyte abnormalities, and hyperpigmentation are well-recognized manifestations, neuropsychiatric symptoms and idiopathic intracranial hypertension (IIH) are uncommon presentations.

Case Presentation: A 19-year-old female presented with severe headache, vomiting, altered sensorium, marked irritability, and psychotic behavior. She had progressive generalized skin hyperpigmentation for 7–8 months, associated with fatigue, anorexia, weight loss, salt craving, and behavioral changes. On examination, she was thin built, hypotensive (84/60 mmHg), and had established bilateral papilloedema. MRI brain was normal. Investigations revealed hyponatremia (125 mEq/L), markedly low serum cortisol (0.15 µg/dL), and elevated ACTH (98 pg/mL), supporting primary adrenal insufficiency. Cerebrospinal fluid opening pressure was 24 mm H₂O.

Outcome: Intravenous hydrocortisone was administered along with adequate hydration. The patient showed rapid improvement in irritability within 6 hours and became conscious and oriented within 24 hours of treatment. Vision remained normal, and neurological examination was unremarkable.

Conclusion: This case highlights the rare association of Addison's disease with idiopathic intracranial hypertension and neuropsychiatric manifestations. Progressive hyperpigmentation, hypotension, electrolyte abnormalities, and unexplained behavioral changes should prompt

consideration of adrenal insufficiency. Early recognition and prompt steroid replacement can lead to rapid clinical improvement and prevent potentially serious complications.

PP-8 Refractory Hypocalcaemia Due to Idiopathic Hypoparathyroidism with Severe Left Ventricular Systolic Dysfunction

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Background: Hypoparathyroidism is an uncommon cause of severe hypocalcaemia. Prolonged calcium deficiency may impair myocardial excitation-contraction coupling and rarely produce reversible left ventricular systolic dysfunction.

Case presentation: A 45-year-old woman with known dilated cardiomyopathy and a left ventricular ejection fraction of 28% presented with muscle cramps and twitching for 15 days. On examination, her vital signs were stable, with no clinical evidence of heart failure; however, Trousseau's sign was positive. Laboratory evaluation revealed profound hypocalcaemia (total serum calcium: 2.0 mg/dL), hyperphosphataemia (6.7 mg/dL), hypomagnesaemia (1.4 mg/dL), and severe vitamin D deficiency (3.01 ng/mL), spot urinary calcium was 6.35 mg/dL. Further testing demonstrated a suppressed intact parathyroid hormone level of 3.01 pg/mL, with preserved renal function. Despite treatment with intravenous calcium gluconate, oral calcium, vitamin D, and magnesium supplementation, her serum calcium remained markedly low. Persistent hypocalcaemia with suppressed parathyroid hormone and hyperphosphataemia, in the absence of previous neck surgery or any identifiable secondary cause, supported the diagnosis of idiopathic hypoparathyroidism. Subcutaneous teriparatide 20 µg was therefore added to conventional therapy. Thereafter, serum calcium levels increased progressively, reaching 8.4 mg/dL by day 24, with corresponding clinical improvement. The patient remained stable at discharge, while her cardiac dysfunction was managed with guideline-directed medical therapy. The clear temporal improvement following teriparatide initiation indicated that parathyroid hormone deficiency was the principal cause of the refractory hypocalcaemia.

Conclusion: Idiopathic hypoparathyroidism should be considered in patients with refractory hypocalcaemia, particularly when low parathyroid hormone and hyperphosphataemia coexist. Severe hypocalcaemia may present with profound myocardial dysfunction. When standard calcium and vitamin D replacement is inadequate, teriparatide may facilitate biochemical correction. Early endocrine evaluation and sustained calcium normalization are essential to reduce the risks of arrhythmia, heart failure, and other potentially serious complications.

PP-9**Charcot Neuroarthropathy in a Patient with
Type 3c Diabetes Mellitus: A Case Report**

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Background: Type 3C diabetes mellitus, also known as pancreatogenic diabetes, results from pancreatic exocrine and endocrine dysfunction. Chronic hyperglycaemia & diabetic peripheral neuropathy can lead to serious musculoskeletal complications including Charcot neuroarthropathy (CNO).

Case Presentation: 40 year old male patient, k/c/o type 3C diabetes mellitus since 11 years on Inj Mixtard 14U s/c twice daily, presented with multiple episodes of vomiting, abdominal pain moderate to severe intensity and restlessness since 1 day. Vital signs include PR-80/min, BP-130/70mmhg, RR-22/min, SpO2-99% on room air, RBS-440mg/dl, ABG was suggestive of metabolic acidosis. Physical examination was suggestive of epigastric tenderness. Local examination of left foot suggestive of swelling and deformity at ankle joint with grade 1 ulcer over lateral aspect of left foot measuring around 5*4cm. Management and Outcome: Patient was started on insulin infusion and diabetic ketoacidosis was managed. X-ray bilateral ankle and foot was done suggestive of marked joint disorganisation. Bilateral lower limb doppler suggestive of atherosclerotic changes in left superficial femoral artery, monophasic waveform in left posterior tibial artery and subcutaneous edema. Custom therapeutic footwear to accommodate deformity and regular dressing of ulcer was done. He exhibited steady clinical improvement with resolution of metabolic abnormalities.

Discussion And Conclusion: The Eichenholtz classification describes 3 stages of disease progression: pre-Charcot (prodromal), developmental/destruction, coalescence and consolidation. Adverse outcomes in CNO were frequent and occurred early after diagnosis, with high rates of hospitalisation, amputation and death. These findings support CNO as marker of systemic vulnerability and highlights the need for prioritised surveillance and multidisciplinary management in routine clinical care.

**PP-10 Aseptic Meningoencephalitis as the Initial Presentation of
MOG Antibody-Associated Disease - A Case Report**

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Background: Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is a distinct inflammatory demyelinating disorder of the central nervous system. Although optic neuritis, myelitis, acute disseminated encephalomyelitis and cortical encephalitis are well-recognized presentations, meningoencephalitis is an uncommon phenotype that may mimic infectious meningitis.

Case Report: A 27-year-old male presented with severe headache, intermittent vomiting for 30 days and fever for 15 days. Examination revealed partial disorientation, exaggerated deep tendon reflexes, positive Babinski sign and meningeal signs. Cerebrospinal fluid (CSF) showed lymphocytic pleocytosis (210 cells/mm³), elevated protein (129 mg/dL) and low glucose (40 mg/dL), suggestive of tubercular meningitis. He was treated empirically with antibiotics followed by antitubercular therapy, but showed minimal clinical improvement. Microbiological investigations remained negative. Initial MRI brain demonstrated T2/FLAIR hyperintensities involving the pons, medulla and cervicomedullary junction. Repeat imaging after 9 days showed an increase in the number and size of lesions. Serum MOG-IgG was subsequently found to be positive. Intravenous methylprednisolone pulse therapy resulted in marked clinical improvement, followed by oral prednisolone.

Discussion: MOGAD can rarely present as aseptic meningoencephalitis and closely mimic infectious meningitis. Lack of response to antimicrobial therapy and negative microbiological investigations should prompt evaluation for autoimmune demyelination. Our case also highlights radiological lag, wherein MRI abnormalities progressed despite clinical improvement following immunotherapy. Dynamic MRI changes during an acute MOGAD attack may therefore not parallel the clinical course, emphasizing that treatment response should primarily be assessed clinically rather than by early radiological resolution.



PP 11 Severe Acute Non-Cardiogenic Pulmonary Edema (NCPE) and Acute Hypoxemic Respiratory Failure Following Intravenous “Chitta” Exposure

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Background: Pulmonary complications of opioid and polysubstance intoxication may be overlooked when altered sensorium dominates the initial presentation. Drug-associated non-cardiogenic pulmonary edema (NCPE) is recognized, although aspiration and other causes of acute lung injury may overlap clinically.

Case Presentation: A 20-year-old previously healthy male presented with altered sensorium following intravenous exposure to a street preparation locally termed “chitta,” with vomiting and an episode of hematemesis. He subsequently developed severe hypoxemia with RR 32/min and SpO₂ 50% on room air. Examination revealed pinpoint poorly reactive pupils and bilateral rhonchi/crepitations. ABG showed PaO₂ 43.8 mmHg and SaO₂ 31%, requiring intubation and mechanical ventilation. Chest radiography demonstrated extensive bilateral central/perihilar air-space opacities suggestive of pulmonary edema. Echocardiography showed preserved

biventricular function with LVEF 60.2%, while blood and respiratory cultures were sterile. Serial radiographs showed progressive resolution with rapid clinical improvement and successful liberation from mechanical ventilation. Active HCV infection was additionally detected with HCV RNA 3.4×10^6 IU/mL.

Discussion: The temporal association, profound hypoxemia, bilateral infiltrates, preserved cardiac function, negative infectious evaluation and rapid reversibility favored a drug-associated acute lung injury/NCPE phenotype. Historical literature demonstrates both rapidly reversible opioid-associated pulmonary edema and uncommon prolonged courses in which aspiration was considered contributory.

Conclusion: Severe hypoxemia in drug intoxication should prompt evaluation for pulmonary involvement rather than attribution to CNS depression alone.

PP-12 A Clinical Diagnostic Enigma: A Case of Adult-Onset Still's Disease (AOSD) Masquerading as Sepsis

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Background: Adult-Onset Still's Disease (AOSD) is an uncommon autoinflammatory disorder marked by quotidian high fevers, evanescent rash, arthralgia, and systemic involvement. Without definitive serological markers, it frequently mimics severe bacterial sepsis, occult malignancies, or systemic autoimmune disorders. Marked leukocytosis and extreme hyperferritinemia are critical diagnostic clues.

Aim: To evaluate the diagnostic approach, application of the Yamaguchi criteria, and treatment response in an adult presenting with prolonged pyrexia and severe hyperferritinemia mimicking sepsis.

Methods: A 40-year-old female presented with a 15-day history of high-grade fever up to 104°F, chills, diaphoresis, sore throat, joint stiffness, and an itchy maculopapular rash. Comprehensive workup included serial septic panels (blood cultures, procalcitonin), endemic infection screens (malaria, scrub typhus, leptospirosis, Mantoux), autoimmune assays (ANA, RF), serum ferritin, inflammatory markers, and chest-abdomen CECT imaging.

Results: Workup revealed significant neutrophilic leukocytosis (TLC 19,100/ μ L, 92% neutrophils), anemia (Hb 8.8 g/dL), elevated CRP (233 mg/L), ESR (125 mm/hr), acute kidney injury (creatinine 1.50 mg/dL), and severe hyperferritinemia (>2,000 ng/mL). Microbial cultures and autoimmune serology were non-reactive. CECT revealed hepatosplenomegaly, generalized reactive lymphadenopathy, and minimal pleural effusion. Meeting 4 major and 4 minor Yamaguchi criteria, AOSD was diagnosed. The patient was started on oral prednisolone (20 mg/day

tapered) and naproxen, achieving complete defervescence within 72 hours, alongside normalization of leukocyte counts, rash clearance, and renal recovery.

Conclusion: AOSD requires prompt recognition in cases of prolonged fever of unknown origin with disproportionate hyperferritinemia. Early application of Yamaguchi criteria allows timely corticosteroid therapy, avoiding catastrophic complications like Macrophage Activation Syndrome.



PP-13 Liver on Trial: Urinary Ascites Masquerading as Hepatorenal Syndrome–AKI

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Background: Ascites with acute kidney injury (AKI) in a patient with alcohol exposure may readily lead to a diagnosis of hepatorenal syndrome (HRS-AKI). However, diagnostic anchoring on a hepatic etiology can obscure uncommon and reversible mimics.

Case: A 68-year-old man with no known chronic liver or kidney disease presented with progressive abdominal distension following an episode of dysuria. He had a two-year history of intermittent urinary retention, benign prostatic hyperplasia and chronic cystitis. Examination revealed gross ascites. Investigations showed severe AKI with preserved urine output. Ultrasonography demonstrated normal-sized kidneys with preserved corticomedullary differentiation. Ascitic fluid was straw-coloured, with a high serum-ascites albumin gradient and low protein, initially suggesting portal hypertensive ascites. However, preserved hepatic synthetic function, normal platelet count, serum albumin and liver morphology, with no other evidence of chronic liver disease, were discordant with HRS. Despite albumin and terlipressin therapy, renal function failed to improve. This prompted diagnostic reconsideration. Ascitic fluid creatinine was markedly elevated, with an ascites-to-serum creatinine ratio >5 , raising suspicion of urinary ascites. CT cystography confirmed a ~ 1.9 -cm urinary bladder wall rent with intraperitoneal urinary leakage. Following urethral catheterization and conservative management, serum creatinine normalized within 48 hours.

Conclusion: Urinary ascites is an uncommon but important mimic of HRS-AKI. In patients with unexplained ascites and AKI, particularly when hepatic parameters are discordant or urinary symptoms are present, ascitic fluid creatinine can provide the crucial diagnostic clue and redirect management towards the underlying urological pathology.

Beyond the Lymph Node: The Spectrum of Rosai-Dorfman Disease

Komalpreet Kaur

Background: Rosai-Dorfman disease (RDD), or sinus histiocytosis with massive lymphadenopathy, is a rare histiocytic disorder typically presenting with massive cervical lymphadenopathy. Extranodal involvement occurs in 40–45% of cases and can closely mimic lymphoproliferative disorders, infectious diseases, and malignancies.

Case Presentation: An 18-year-old female presented with a six-month history of fever, cough, generalized body aches, abdominal distension, and progressive lymphadenopathy. Physical examination revealed generalized non-tender, firm lymphadenopathy, cutaneous hyperpigmented plaques, moderate hepatomegaly, and massive splenomegaly. Investigations showed pancytopenia, normochromic normocytic anemia, and an elevated ESR. Abdominal ultrasound confirmed hepatosplenomegaly and abdominal lymphadenopathy. Fine-needle aspiration cytology (FNAC) of a cervical lymph node revealed lymphoid cells, scattered large histiocytes, and diagnostic emperipolesis, confirming RDD. Treatment with systemic corticosteroids led to significant clinical improvement and rapid reduction in lymph node size.

Conclusion: RDD should be considered in the differential diagnosis of patients presenting with generalized lymphadenopathy and systemic extranodal manifestations. Recognizing key cytological features such as emperipolesis enables prompt diagnosis and avoids misclassification as a hematologic malignancy.



Evaluation of an Interesting Case of Left Upper Limb Distal Amyotrophy

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Background: Distal upper limb amyotrophy in young individuals poses a diagnostic challenge, with differentials including anterior horn cell disorders, motor neuropathies, and cervical cord pathologies. We report an adolescent male with isolated asymmetric distal upper limb wasting diagnosed as Hirayama disease following detailed clinicoradiological evaluation.

Case Presentation: A 19-year-old right-handed male presented with two months of progressive weakness and wasting of the left hand and forearm, causing difficulty with writing, buttoning, typing, gripping, and lifting weights. There was no sensory involvement, neck trauma, gait or sphincter disturbance, toxin exposure, or family history of neuromuscular disease. Examination revealed marked distal amyotrophy involving the interossei, thenar, hypothenar, and forearm flexors, with relative brachioradialis sparing, producing characteristic oblique amyotrophy. Distal power was reduced, while tone and deep tendon reflexes were preserved without

pyramidal signs. Sensory and other neurological examinations were normal. Nerve conduction studies showed reduced motor amplitudes with preserved sensory responses, while electromyography demonstrated chronic denervation involving C8–T1 myotomes.

Discussion: Dynamic cervical MRI revealed anterior displacement of the posterior dural sac during flexion, enlargement of the posterior epidural space, and dynamic lower cervical cord compression, confirming Hirayama disease. The combination of unilateral distal wasting, preserved sensation, absence of upper motor neuron signs, and C8–T1 denervation should prompt consideration of this condition. Important differentials include amyotrophic lateral sclerosis, spinal muscular atrophy, multifocal motor neuropathy, syringomyelia, and cervical myelopathy. Hirayama disease results from flexion-induced anterior dural displacement causing repeated cervical cord compression and anterior horn cell injury, predominantly at C7–T1 levels.

Conclusion: Hirayama disease should be considered in young patients presenting with unilateral distal upper limb amyotrophy and preserved sensation. Recognition of oblique amyotrophy and characteristic dynamic MRI findings is crucial for early diagnosis. Timely cervical immobilization may halt progression and prevent irreversible neurological disability.



PP-16 Navigating Anatomical Challenges: Pacemaker Implantation in a Patient of Postoperative Complex Congenital Heart Disease

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Background: Pacemaker implantation after complex congenital heart disease (CHD) repair is challenging when abnormal venous pathways and cardiac anatomy limit conventional fluoroscopic guidance. We report postoperative anatomy in which multimodality imaging enabled successful transvenous pacing.

Case: A 19-year-old boy with mirror-image dextrocardia, left isomerism with a large atrial septal defect, near-common atrium, bilateral superior vena cavae without an intercommunicating vein, and previous surgery developed complete heart block with a narrow-QRS escape rhythm of 35/min after a second cardiac surgery. At 8 years, he underwent atrial septal defect closure, systemic atrioventricular valve repair, and rerouting of systemic and pulmonary venous pathways. Subsequently, worsening breathlessness led to a second surgery comprising rerouting of the right superior vena cava and coronary sinus, residual atrial septal defect closure, and systemic atrioventricular valve repair. Pre-procedural echocardiography, CT angiography, catheterization data, and venography delineated the anatomy. Left axillary venous access was selected. The ventricular lead was positioned at the apical interventricular septum of the morphologic right ventricle after unstable right ventricular outflow tract positioning, with left ventricular angiography confirming placement. The atrial lead was fixed in the left-sided

atrial appendage. Adequate pacing parameters were achieved without complications.

Conclusion: In complex postoperative CHD, successful transvenous pacing depends on precise reconstruction of patient-specific anatomy. Integrating multimodality imaging enabled rational access selection and accurate septal lead placement, supporting systematic pre-procedural planning for pacemaker implantation in complex CHD.

PP-17 **GBS or CIDP? When the Diagnosis Changes with Time:**

A Case of Acute-Onset CID

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Background: Guillain–Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy characterized by rapidly progressive symmetrical weakness. Acute-onset CIDP (A-CIDP) may initially mimic GBS but is distinguished by persistent progression or relapse beyond the acute phase.

Case Presentation: An 18-year-old male developed bilateral lower-limb followed by upper-limb weakness and breathlessness five days after acute diarrheal illness. Nerve conduction studies suggested peripheral demyelinating neuropathy. He was initially treated with IVIG and corticosteroids. CECT chest revealed aspiration pneumonitis, and he was discharged on home BiPAP support. Fifteen days later, worsening breathlessness and deterioration after initial improvement raised suspicion of A-CIDP. He received IV methylprednisolone, azathioprine, and three sessions of plasmapheresis. Despite initial improvement, his condition deteriorated with duodenal obstruction and bilateral pneumothorax. He was subsequently referred to a higher centre, where ventilatory support and low-dose steroids resulted in clinical improvement.

Conclusion: The patient had an acute progressive demyelinating polyradiculoneuropathy following diarrheal illness, initially managed as GBS, but showed persistent clinical deterioration despite IVIG, steroids, azathioprine, and plasmapheresis.

The prolonged and refractory course raises suspicion for acute-onset CIDP (A-CIDP) or another immune-mediated demyelinating neuropathy, warranting further electrophysiological and immunological evaluation.

PP-18 **When Seeing Isn't Enough: Failed Bougie Passage despite an Excellent Video laryngoscopic View**

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Background: Video laryngoscopy (VL) improves visualization of the glottic inlet. However, an adequate glottic view does not always ensure successful tracheal intubation, particularly when the larynx is markedly anterior and bougie delivery is unfavorable.

Aim: To highlight failed bougie passage despite video laryngoscopic visualization of the glottis in a patient with a markedly anterior larynx and emphasize tube-delivery difficulty.

Methods: A 52-year-old male scheduled for elective laparoscopic cholecystectomy underwent general anaesthesia. Airway assessment revealed Mallampati III, mouth opening >3 fingerbreadths, thyromental distance approximately 3 fingerbreadths, and normal neck movements. Direct laryngoscopy produced a Cormack–Lehane grade III view with a markedly anterior larynx. Video laryngoscopy provided visualization of the laryngeal inlet; however, bougie advancement remained unsuccessful despite optimization and external/cricoid manipulation. A further attempt also failed. During repeated attempts, heart rate decreased from approximately 52/min to 48/min, while SpO₂ remained around 97%. Further attempts were abandoned. Bag-mask ventilation was maintained, neuromuscular blockade was reversed with sugammadex 200 mg IV, and the patient recovered with adequate spontaneous ventilation.

Results: Despite improved glottic visualization with VL, tracheal access could not be achieved because of difficulty directing the bougie toward the markedly anterior larynx. Further attempts were avoided, and the planned surgery was deferred.

Conclusion: A good video laryngoscopic view does not necessarily indicate easy intubation. Difficult airway management includes both visualization and tube delivery. When bougie advancement fails despite an adequate view, repeated forceful attempts should be avoided, oxygenation prioritized, and an alternative airway strategy considered.



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