**From:** "ROOT" <root@sctimst.ac.in>
**To:** "ROOT" <root@sctimst.ac.in>
**Date:** 28/10/2025 11:44 AM
**Subject:** CPC Clinical Protocol 29.10.2025

The next Wednesday CPC will be held on **October 29, 2025** at **08.00 hours** (IST) in **Lecture Theatre 1**, Nehru Hospital, PGIMER, Chandigarh under the Chairmanship of **Prof. Sanjay Jain.**

The session will also be available on the Webex platform. Kindly follow the link below to join.

https://pgitelemed.webex.com/pgitelemed/j.php?MTID=md00288711fdbf94321a0819943e426a2

In case you join in thru WebEx, kindly **ensure that your microphone and camera are switched off** and **PLEASE DO NOT SHARE YOUR SCREEN**.

The Clinical handout of the case to be discussed is attached herewith.
The clinical protocol will be discussed by ***Dr. Raja Ramachandran, Department of Nephrology.*** Radiology will be presented by ***Dr. Nidhi Prabhakar.*** Autopsy pathology will be presented by ***Dr. Ritambhra Nada.***



Yours sincerely,

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Regional Resource Centre, North
Department of Telemedicine
PGIMER, Chandigarh

# CPC: 29th Oct 2025

| | | | | | |
|---|---|---|---|---|---|
| **NAME** | Ms XXXXX | **Age** | 16 years | **Gender** | Female |
| **C.R. No.** | XXXXXXXX | **Hospital stay** | 12 days | **Treating unit** | Nephrology (Prof HS Kohli) |
| **Clinical discussant** | Dr Raja Ramachandran | **Radiology Discussant** | Dr Nidhi Prabhakar | **Pathology discussant** | Prof Ritambhra Nada |

**Chief Complaints**
Cough x 6 days, loose stools x 6 days and fever x 2-3 days

***Background History***:
The patient, a 16-year-old female, was diagnosed with childhood-onset nephrotic syndrome in 2012 at 4½ years of age. She had a frequently relapsing course, receiving multiple courses of steroids along with azathioprine until 2017. In 2017, she presented to the Advanced Pediatric Center, PGIMER, where she was started on levamisole for frequently relapsing nephrotic syndrome, but relapses persisted.

In **2018**, diagnosed with late steroid-resistant nephrotic syndrome (biopsy suggestive of minimal change disease with immature glomeruli), and she was initiated on tacrolimus with low-dose steroids, which were continued for two years, with a few relapses managed with short steroid courses. She was lost to follow-up for two years, during which she stopped all medications and self-medicated with steroids intermittently. In **February 2022**, she developed left-leg cellulitis, requiring hospitalisation and antibiotics. A month later, she had another relapse, for which she received prednisolone 60 mg daily, achieving remission by day 12. She again defaulted on follow-up and continued 15 mg every other day for nearly a year without supervision. In **May 2023**, she presented to the Adult Nephrology unit with another relapse. She was restarted on prednisolone 1 mg/kg/day but failed to achieve remission after 12 weeks (**late steroid-resistant**). Subsequently, tacrolimus 1 mg twice daily was added; however, no response was achieved after six months, and serum albumin remained low (1–1.2 g/dL). Genetic testing in March 2024 was negative for monogenic steroid-resistant nephrotic syndrome.

This admission, she presented with diffuse abdominal distension for one week without abdominal pain. She also reported a dry cough for six days, loose stools (6–7 episodes/day), and low-grade intermittent fever for 2–3 days. Over the past 10 days, she had developed worsening pedal oedema and oliguria (<500 mL/day). There is no history of altered mental status, seizures, or syncope.

**Past/Personal/Family history**:
No history of DM/HTN/ Blood transfusion / Major Surgery/ TB/COPD/Asthma. Non-smoker, Non-alcoholic. No contact history of TB. No history of similar illness in any of the other family members.

**Examination**
The patient was conscious, oriented, and afebrile at presentation. Blood pressure was 102/70 mmHg, pulse 90/min and regular, respiratory rate 22/min, and $SpO_2$ 97% on room air. She had pallor but no icterus, cyanosis, clubbing, or lymphadenopathy. Pedal oedema was present, and jugular venous pressure was not elevated. Examination of the oral cavity was normal. She was noted to be short-statured with facial plethora and malar erythema.

On systemic examination, the cardiovascular system revealed normal S1S2 with no added sounds or murmurs. Respiratory examination showed bilateral coarse crepitations over the infra-axillary and infra-scapular areas. The abdomen was soft and non-tender, with no hepatosplenomegaly or ascites, and abdominal striae were

present. The central nervous system examination showed that the patient was alert, oriented, and had no focal neurological deficits.

**Investigations**

| Parameters | 12/03/24 | 13/03/24 | 15/03/24 | 16/03/24 | 19/03/24 | 21/03/24 | 23/03/24 |
|---|---|---|---|---|---|---|---|
| Hb | 7.9 | | | 7.7 | 8.4 | 8.5 | 7.1 |
| TLC | 13.8 | | | 7.73 | 12.8 | 14.4 | 18.8 |
| DLC | | | | | | 85/9/1/4/1 | |
| Platelet count | 130 | | | 170 | 96 | 50 | 32 |
| Na/K | | 106/3.68 | 109/3.23 | 109.6/2.9 | 124.2/3 | 125/3.45 | 127.2/4.4 |
| Urea/Cr | | 70.2/0.93 | 70/0.79 | 66.6/0.63 | 47.7/.05 | 51/0.74 | 65.3/1.17 |
| Bil (T/B) | | 0.18/0.06 | 0.27/0.06 | 0.27/0.13 | 0.3/0.13 | 0.20/0.07 | 0.24/0.08 |
| AST/ ALT | | 44.8/19.5 | 27/18 | 17.2/12 | 21.7/12.3 | 61/23.8 | 283.7/100 |
| ALP | | 141 | 114 | - | 109 | 112 | - |
| TP/SA | | 3/1.15 | 2.8/1.06 | 3/1.72 | 2.82/1.74 | 2.5/1.35 | 2.68/<1 |
| PT/APTT/INR | | 17/30.4/1.44 | | | > 2min | 35/56/3.5 | >2min/65/- |
| CRP/LDH | | 50/694 | | 56.4 | - | 29.35/1040 | 31.7/1247 |
| Procalcitonin | | | | | 90 | - | - |
| Ca/P/Mg | | 6.5/4.5/1.4 | | 6.8/3.9/1.2 | 7.3/2.2/0.98 | 6.9/3/1.2 | 6/4.5/1.2 |
| C3/C4 | | - | | | - | 79/14 | 60/7.5 |

**CECT chest and abdomen (21/03/24):** Imaging revealed a focal mass in the right atrium, likely representing a thrombus. There were patchy ground-glass opacities in the posterobasal and laterobasal segments of the right lower lobe, along with eccentric filling defects in the segmental branches of both lower pulmonary arteries, consistent with pulmonary thromboembolism. The right-sided cardiac chambers were dilated, with reflux of contrast into the inferior vena cava and hepatic veins, suggestive of right heart strain. A mild pericardial effusion and mild left pleural effusion were also noted. Additionally, there was diffuse circumferential bowel wall thickening, diffuse subcutaneous edema with fat stranding, and bilaterally enlarged kidneys with mildly reduced nephrographic density, indicative of systemic congestion and renal involvement.

**USG Abdomen (13/03/24):** Hepatomegaly and diffuse thickening of the subcutaneous tissue in the anterior abdominal wall. Moderate ascites and left moderate pleural effusion. **2D ECHO (22/03/23):** mild to moderate TR, RVSP- 20 + RAP, Right atrial mass 1.8 cm globular protruding into the RV during atrial systole -? Thrombus ? RA myxoma, Mild MR, LVEF Normal. **DVT scan:** No evidence of DVT

**Others:** Blood cultures taken on 16th and 20th March 2024 were sterile. The iron profile (16/03/24) showed a serum iron of 40.4 µg/dL, TIBC of 98.9 µg/dL, transferrin saturation of 40.8%, and a serum ferritin level of 349.7 ng/mL. Special stains (stool) for typical and atypical organisms were negative. Stool routine microscopy and culture showed no abnormalities or pathogenic growth, while faecal calprotectin was mildly elevated at 60 µg/mg, and stool for Clostridium difficile toxin assay was negative.

**Urine routine examination** on 20/03/24 revealed proteinuria (+++) amounting to approximately 100 mg/dL; urine culture was sterile.

**Cerebrospinal fluid analysis** (13/03/24) revealed a total cell count of 42 cells/mm$^3$ with 20.6% neutrophils and 79.4% lymphocytes, and RBC count of 21,069/mm$^3$, with no organisms identified. Sputum Gram stain (22/03/24) showed only commensal flora, with no pathogenic bacteria.

# CPC: 29th Oct 2025

| Date | pH | PCO2 | PO2 | Bicarbonate | Sodium | Potassium | Calcium |
|---|---|---|---|---|---|---|---|
| **14.03.24** | 7.459 | 32.2 | 38.9 (77%) | 22.3 | 103 | 3.03 | 0.88 |
| **24.03.24** | 7.434 | 32 | 29.9 (58%) | 21 | 106 | 3.47 | 0.92 |

**Course and management**

A 16-year-old female with a history of steroid- and calcineurin-resistant nephrotic syndrome (delayed non-responder) presented with complaints of decreased urine output and loose stools. She was on warfarin for nephrotic syndrome with severe hypoalbuminemia (serum albumin 1 g/dL). On admission, urine output was markedly reduced (100 mL/12 h), though serum creatinine was normal (0.46 mg/dL), and she had severe hyponatremia. A working diagnosis of nephrosarca with hypervolemic hyponatremia was made, and she was started on diuretics (Torsemide and metolazone) with intravenous albumin, leading to gradual improvement in urine output. Because of diarrhea, stool work-up was sent, and she was empirically treated with metronidazole and ciprofloxacin, following which stool frequency reduced from 10–15 to 4–5 episodes/day; all stool investigations were negative. During hospitalization, she developed a fever on 19 March, with procalcitonin 90 ng/mL, and was started on piperacillin-tazobactam, after which the fever subsided. Her INR became supratherapeutic, prompting temporary discontinuation of warfarin. On 21st March, she again developed a fever spike and new-onset left lower-zone crepitations. CECT chest and abdomen revealed a right atrial thrombus, bilateral segmental pulmonary artery emboli, dilated right cardiac chambers, mild pericardial and pleural effusions, circumferential bowel wall thickening, bulky kidneys, and features of congestive hepatopathy. Echocardiography confirmed a 1.8 cm right-atrial thrombus with mild-to-moderate tricuspid regurgitation. Although hemodynamically stable, anticoagulation could not be restarted due to falling platelet counts and rising INR. Antibiotics were escalated to meropenem and vancomycin due to fever. On 24th March 2024, the patient developed a sudden cardiac arrest at 8:30 a.m., presumed secondary to pulmonary thromboembolism. CPR was initiated as per ACLS protocol, but there was no return of spontaneous circulation, and she was declared dead at 9:59 a.m.

**Impression**

Childhood-Onset Nephrotic Syndrome
Biopsy proven- Minimal Change disease
Delayed non-responder
Hyponatremia- hypervolemic
Right Atrial Thrombus with Pulmonary Thromboembolism
Sepsis (? HAP) with Coagulopathy