Welcome To The Clinical Meet JANUARY 2018

Department Of Medicine

&

Department Of Microbiology

Dr D Y Patil Medical College and Research Hospital

PLEASE SILENCE YOUR PHONE

A Case Of Deep Jaundice

Dr. Karan

JR- III

Dept. Of Medicine

History

- A 44 year old male farmer by occupation came to OPD with complaints of abdominal pain, yellowish discoloration of eyes, reduced appetite and itching all over the body since 2 months.

- The abdominal pain was more in the epigastric region and used to increase with food intake but not with any change in posture. He also gave h/o passing pale coloured stools.

- There was no history of fever with chills / vomiting.

Past History

- He was diagnosed with sputum positive pulmonary tuberculosis in 2006 along with HIV infection.
- He completed treatment for tuberculosis and was regularly taking ART.
- Although his CD4 count on diagnosis was not available his CD4 count in 2015 was 37.
- His latest CD4 count was 169.

Treatment History

- The patient is on

  - Tab.Lamivudine 300 mg HS
  - Tab.Tenofovir 300 mg HS
  - Tab.Atazanavir with Ritonavir boosting(300 + 100) HS

for the past 4 years and Tab.Septran-DS OD since 2006.

Personal history

- Mixed diet.
- Normal bowel and bladder habits.
- No h/o addiction to alcohol / smoking / substance abuse.

On Examination

- The patient was
  - Afebrile
  - Deep Icterus +
  - Mild Pallor +
  - There was no lymphadenopathy.
- BP-110/60 mmhg
- PR-100/min
- RR-18/min

Systemic examination

- Per abdomen :
  - Abdomen was distended with no clinically demonstrable free fluid.
  - Dilated veins with flow away from umbilicus.
  - Liver was non-tender and firm in consistency a liver span of 20 cms.
  - Splenomegaly extending to 11cm below left costal margin.

Rest of the system examinations were normal.

Lab Investigations

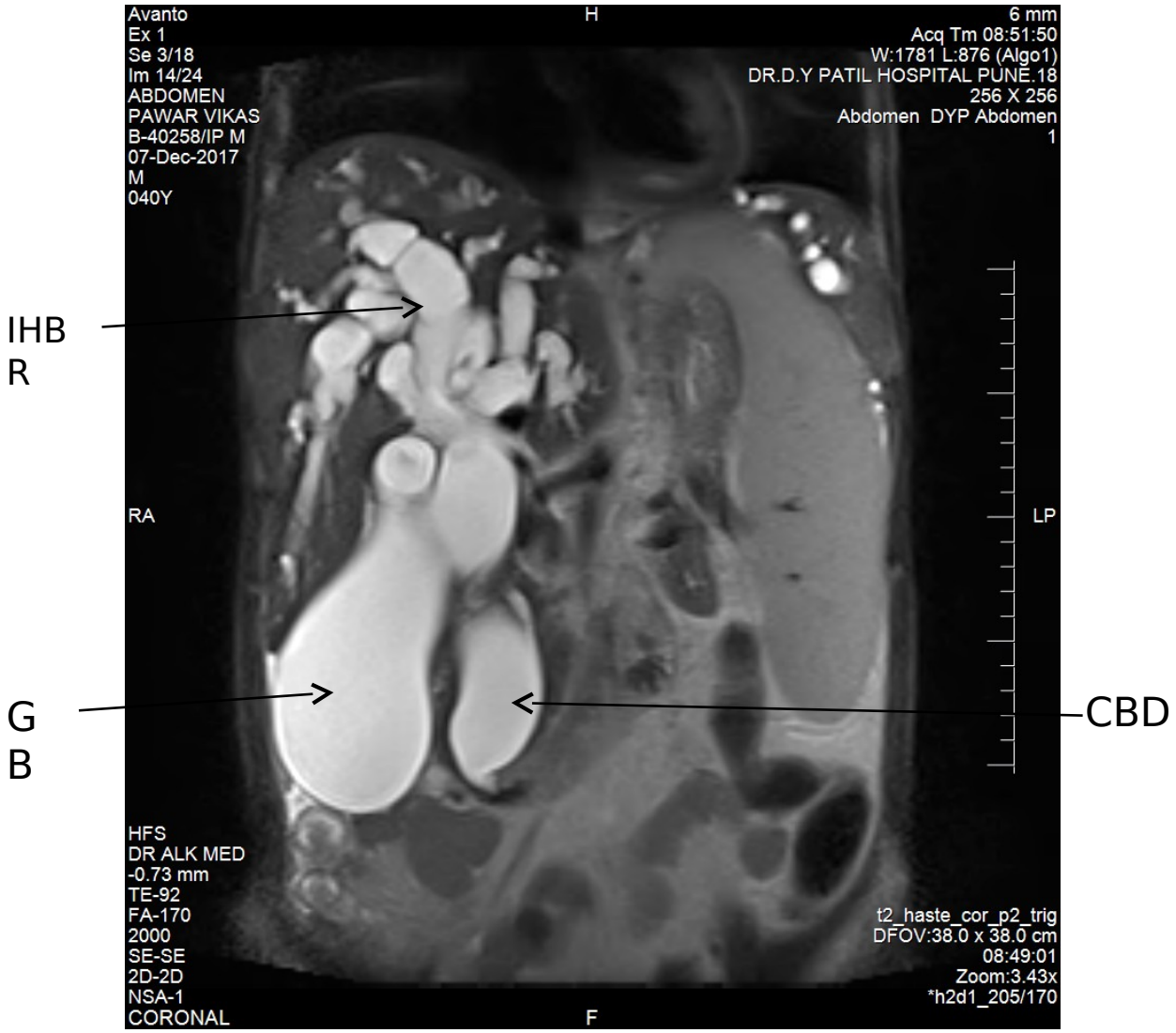
Investigation	Result
Hb	9.6 gm/ dl
TLC	7400/ cu.mm
Platelet count	2.6 lakh/ cu.mm
Total bilirubin	25.62 mg/dl
Direct bilirubin	20.63 mg/dl
ALP	1200 U/L
Gamma Glutamyl Transferase	249 U/L (10-40 U/L)

Imaging studies

- Ultrasound abdomen showed:
  - Grossly dilated CBD and pancreatic duct giving double duct sign, splenomegaly with dilated splenic vein, hepatomegaly with dilated IHBR.

- MRCP was done which showed:
  - Hepatosplenomegaly with gross dilatation of biliary tract with tapering of distal CBD just proximal to the ampula of vater - likely due to cholangitis stricture.
  - The gall bladder is well distended with normal walls. No pericholecystic pathology.
  - Pancreas is normal in size and signal intensity.
  - CBD measures 35mm (6mm). Pancreatic duct is dilated 5mm (3mm).

MRCP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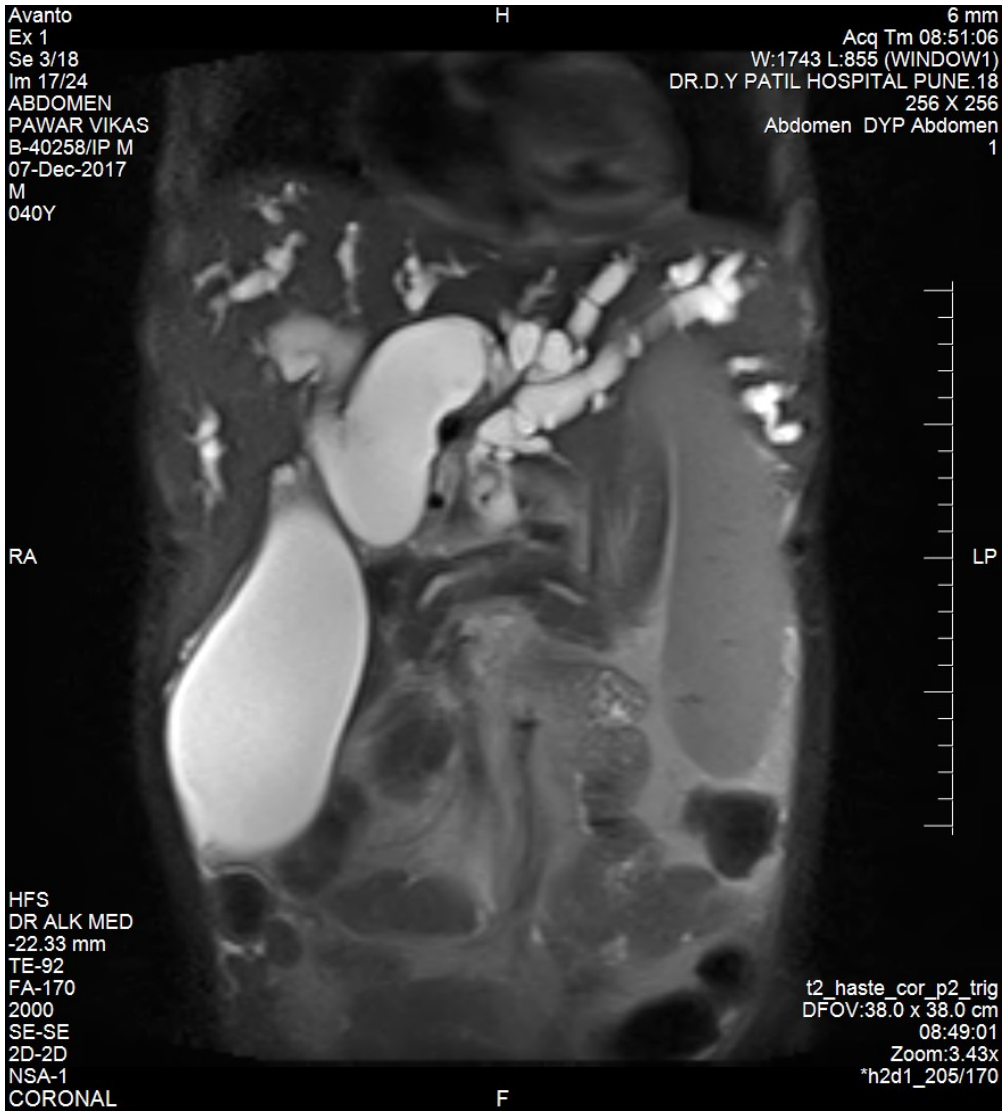
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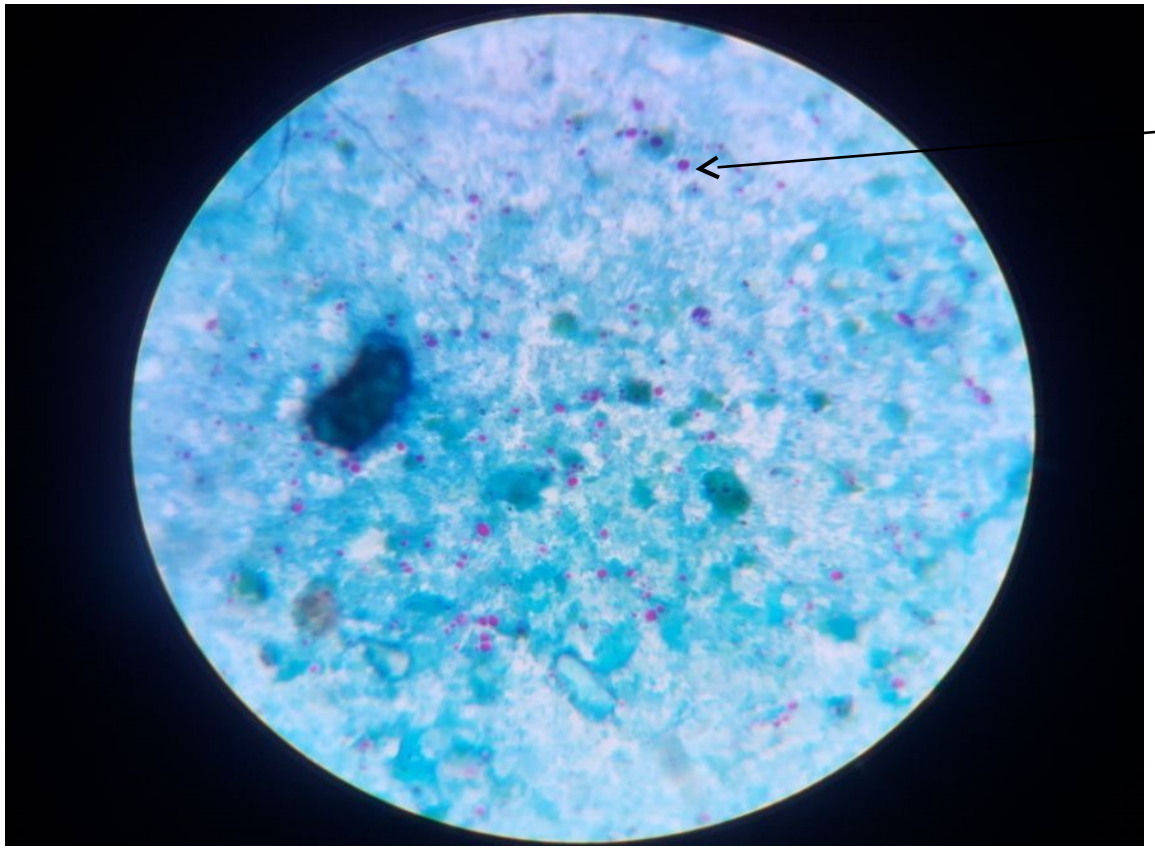
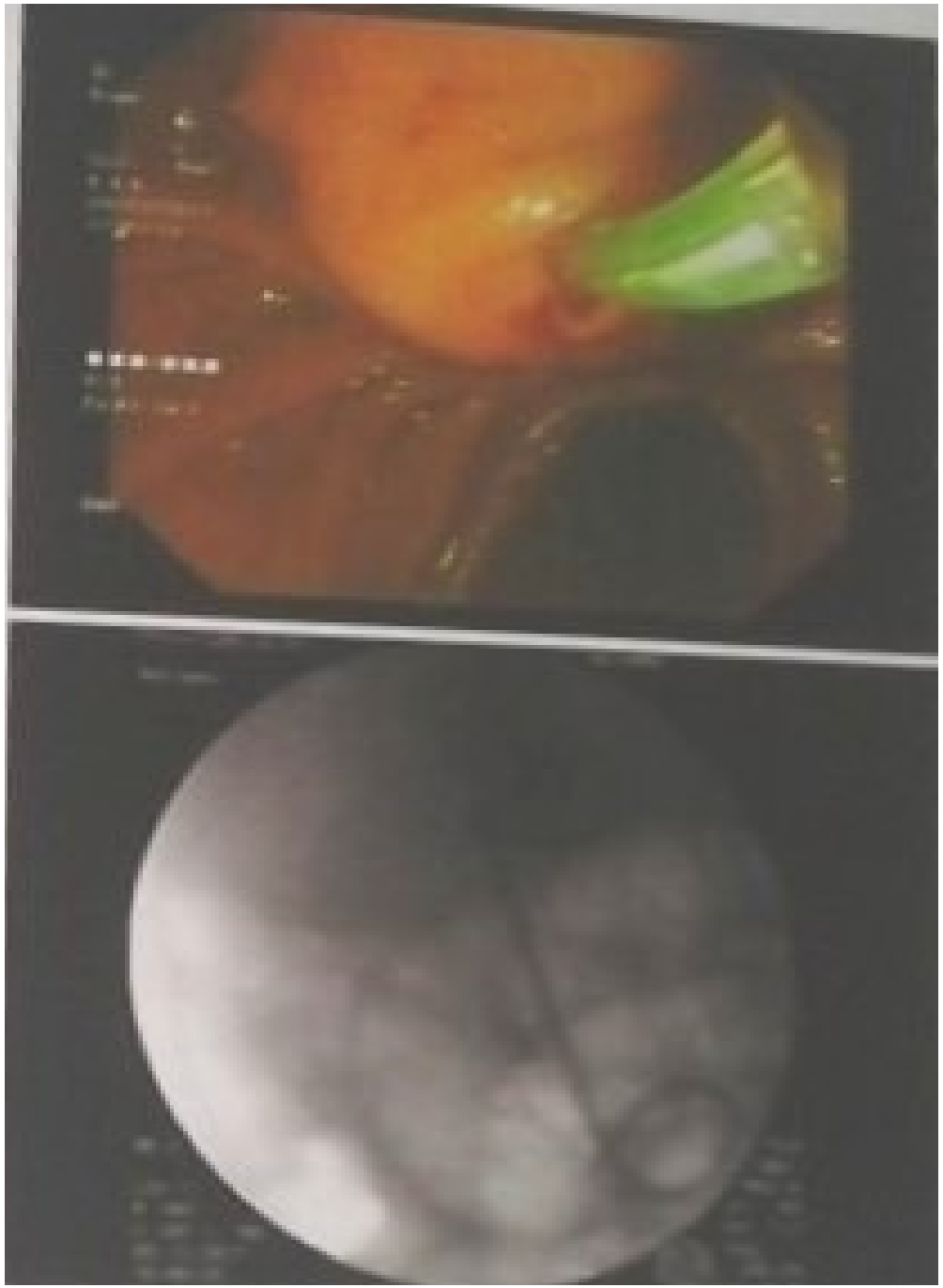
- As our patient had obstructive jaundice with no calculi or growth in the biliary tract we suspected AIDS cholangiopathy caused by opportunistic infection with cryptosporidium.
- His stool R/M confirmed the presence of Oocyst of Cryptosporidium (kinyoun stain).



- In view of stricture at distal end of CBD the patient underwent ERCP with therapeutic papillotomy with CBD stenting was done.

- Post procedure his bilirubin and ALP levels came down by 50% within 48 hours of ERCP and patient showed signs of clinical improvement in the form of reducing icterus and improved appetite.

- The patient was advised to continue the antiretroviral therapy on discharge and has been in regular follow up with us.



Summary

- This is a case of PLHIV with deep jaundice with raised alkaline phosphatase and stricture at the distal CBD which was released by a papillotomy with CBD stenting and stool showing oocyst of cryptosporidium hence our patient had AIDS cholangiopathy.

Discussion

- AIDS cholangiopathy is a biliary syndrome in AIDS patients, which was first described by Cello in 1989. It is diagnosed on clinical features, raised alkaline phosphatase, on ultrasound and ERCP/ MRCP investigation, evidence of cryptosporidium in stool.
- Cello described 4 different entities of cholangiographic abnormalities in AIDS cholangiopathy - papillary stenosis and cholangitis (most common presentation ~ 50%), papillary stenosis alone (30%), intrahepatic sclerosing cholangitis alone (10%), long extrahepatic bile duct stricture (10%).

Discussion

- Opportunistic infections of the biliary tree are believed to be the most common cause of AIDS cholangiopathy.
- The most commonly identified organisms are cryptosporidium and cytomegalovirus. Other opportunistic organisms are microsporidia, cyclospora, *Mycobacterium avium complex, Isospora belli.*
- Intestinal cryptosporidiosis appears to be a major feature of the disease and 10 - 16% of AIDS patients with intestinal cryptosporidiosis develop biliary symptoms.
- Infection of human intestine by cryptosporidium has been reported in immunocompetent and

References

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Thank you !!!

AN UNUSUAL MANIFESTATION IN PLHIV

Dr. Shalaka S. Shinde
JR - II
Medicine Department

- A 49 yrs old male patient , farmer by occupation , resident of Khed came with c/o :-

- Shortness of breath since 15 days
- Easy fatigability since 15 days
- Dry cough since 15 days
- Swelling of both lower limbs since 10 days
- **No history of :-**

  - Fever , joint pain , photosensitivity , hematuria , burning micturation , decreased urine output.

PAST HISTORY

- Per rectal bleeding one month back for 4-5 days (fresh per rectal bleed 4-5 drops after defecation , not mixed with stool)

- Not a k/c/o DM/HTN/TB/BA

- No history of previous blood transfusion.

GENERAL EXAMINATION

- Afebrile
- Pulse – 84/min
- BP- 150/90 mmHg
- Pallor + ,
- Pedal edema (pitting) + ,
- Knuckle pigmentation + ,
- Platynychia +

SYSTEMIC EXAMINATION

- P/A –

- Soft , no hepatomegaly
- Spleen palpable 4-5 cms below the left costal margin , non-tender

- RS – left basal fine creptations +

- CVS – S1S2 audible , no murmur

- CNS – No focal neurological deficit

LAB PARAMETERS

- HB – 6.1 gm/dl
- TLC-6500 (39/57/2/2)
- PLT COUNT – 2.8 cumm
- ESR- 48 (raised)
- MCV-65.3
- Retic count-2.2%
- RDW – 55.3 cumm
- PBS – Microcytic , hypochromic
- Direct Coombs Test – Negative

- LFT :

-BIL (T/D) – 0.49/0.22 mg/dl

-ALT – 15 U/L (0-40)

-AST – 24 U/L (5-35)

-ALP – 123 U/L (15-112)

-Serum protein – 7.44 g/dl

-Sr. Albumin – 2.86 g/dl

-Sr. Globulin – 4.1 g/dl (2.0-3.5)

-Sr. Uric acid – 9.8 gm/dl (3.4-7.0)

FLP :Sr TGL – 68 mg/dl

Cholesterol – 93 mg/dl

HDL – 31 mg/dl

LDL – 50.9 mg/dl

Sr Na^+ – 138 mm/l

Sr K^+ – 4.0 mm/l

Blood urea – 46

- HbA1c – 4.7 % (normal)
- Urine R/M – Albumin : 2+ Negative

Sugar : nil

Count – 320 cells

RBCs : >50/hpf

AFB – negative

Blood : 2+

c/s – No growth

Pus cells : 0-1

Epi cells : 0-1

Casts : Absent

HIV – Positive (ELISA)

HbsAg/HCV - Negative

CD4

Sputum

Sputum

- TFT :

  TSH – 19.9 µ IU/ml (N- 0.3-5.5)

  T3 – 105 ng/dl (N - 60-200)

  T4 – 6.2 µg/dl (N - 4.5-12)
- C3 – 76.50 mg/dl (N : 90-180 mg/dl)
- C4- 4.10 mg/dl (N: 10-14 mg/dl)
- Sr. Protien Electrophoresis : Hypergammaglobulinemia noticed , Monoclonal band not seen.
- ANA – 0.62 (Neg < 0.8)
- ANA Blot – PM-Scl , Jo-1 : weakly positive
- c-ANCA , p-ANCA – Negative

- **Iron profile** :-
  - Sr. iron – 22 µg/dl (70-180)
  - TIBC – 374 µg/dl (225-535)
  - % transferrin saturation – 6 % (13-45)
  - Ferritin – 19.3 mg/ml (22-322)

- **Bone marrow aspiration** – Mild erythroid hyperplasia with normoblastic maturation.

- **ECG** – ‘T’ wave inversion in I, avL , V4 , V5 , V6
- **CXR** – Inhomogenous opacity in left lower zone.
- **2D Echo** – Mild concentric LVH , hypertensive changes , good LV function , EF – 60%
- **HRCT thorax** – Patchy areas of consolidation in postero-basal segments of lower lobe of left lung field.
- **USG (A/P)** – Splenomegaly 14.5 cms

  RK – 11.6*4.6 LK – 10.3*5.9

PROVISIONAL DIAGNOSIS :-

- In view of the history , examination and investigations , a provisional diagnosis of :-

" HIV WITH IRON DEFICIENCY ANEMIA WITH PROTIENURIA UNDER EVALUATION WITH HYPOTHYROIDISM WITH HYPERTENSION WITH LOWER RESPIRATORY TRACT INFECTION "

- **Nephrology opinion** :-

-Renal biopsy was advised with keeping in mind the diagnosis of :-

Nephritic syndrome (? cause)

- **RENAL BIOPSY REPORT :-**
- 2 /11 Glomerular global sclerosis
- Rest of the glomeruli show

  - Segmental endocapillary proliferation rich in lymphocytes

  - Segmental duplication of peripheral capillary wall along with moderate mesangial proliferation.
- Immunofluorescence :- revealed 6 glomeruli

- IgG – 2+ IgA-Neg

- C3- 3+ IgM-Neg

FINAL DIAGNOSIS

" HIV INDUCED IMMUNE MEDIATED MEMBRANO-PROLIFERATIVE GLOMERULONEPHRITIS WITH HYPOTHYROIDISM"

TREATMENT

- He was also started with (ART) - TLE regimen :-
  - Tenofovir 300 mg
  - Lamivudine 300 mg
  - Efavirenz 600 mg
- Inj. Ceftriaxone 1gm BD for 7 Days

Outcome

- The patient is currently doing well and is on regular follow up.
- RFT - WNL
- Urine R/M - Reduced protein and RBCs.

DISCUSSION

- Patients with HIV infection are at increased risk for both acute kidney injury and chronic kidney disease.
- Some risk factors are specific to HIV causing renal disease :-

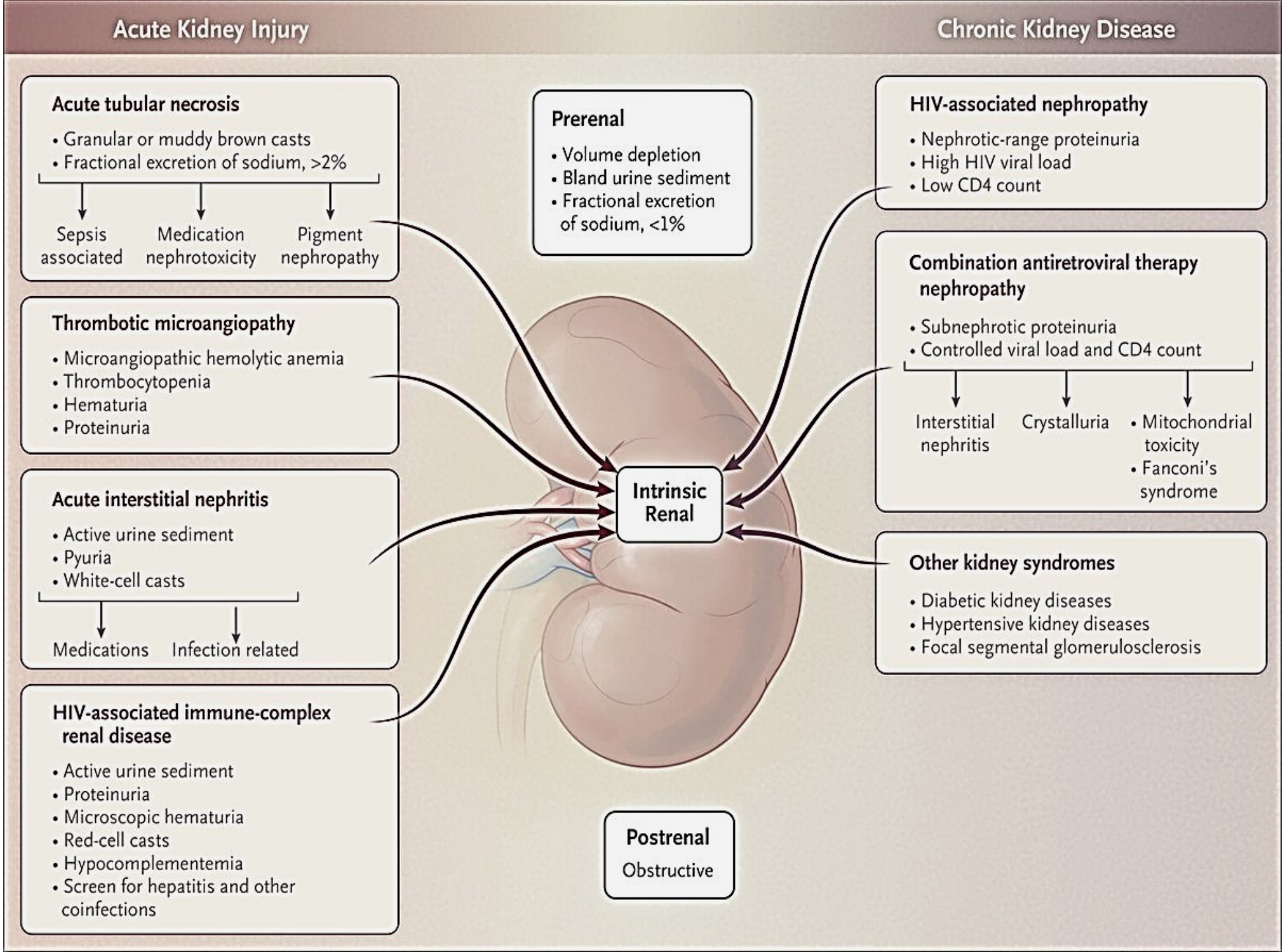
✓ low CD4 count

✓ high viral load

✓ co-infection with hepatitis B and C virus

TAKE HOME MESSAGE

- Untreated HIV infection as well as ART are associated with kidney disease.
- ART is a double edged sword: although it can lead to improvement in the life expectancy of person with HIV infection, it can also increase clinical uncertainty regarding changes in renal function in this population.


Acute Kidney Injury
Chronic Kidney Disease
Acute tubular necrosis
• Granular or muddy brown casts
• Fractional excretion of sodium, >2%
Sepsis associated
Medication nephrotoxicity
Pigment nephropathy
Thrombotic microangiopathy
• Microangiopathic hemolytic anemia
• Thrombocytopenia
• Hematuria
• Proteinuria
Acute interstitial nephritis
• Active urine sediment
• Pyuria
• White-cell casts
Medications
Infection related
HIV-associated immune-complex renal disease
• Active urine sediment
• Proteinuria
• Microscopic hematuria
• Red-cell casts
• Hypocomplementemia
• Screen for hepatitis and other coinfections
Prerenal
• Volume depletion
• Bland urine sediment
• Fractional excretion of sodium, <1%
Intrinsic Renal
Postrenal
Obstructive
HIV-associated nephropathy
• Nephrotic-range proteinuria
• High HIV viral load
• Low CD4 count
Combination antiretroviral therapy nephropathy
• Subnephrotic proteinuria
• Controlled viral load and CD4 count
Interstitial nephritis
Crystalluria
• Mitochondrial toxicity
• Fanconi's syndrome
Other kidney syndromes
• Diabetic kidney diseases
• Hypertensive kidney diseases
• Focal segmental glomerulosclerosis

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2. Kimmel PL, Barisoni L, Kopp JB. Pathogenesis and treatment of HIV-associated renal diseases: lessons from clinical and animal studies, molecular pathologic correlations, and genetic investigations. Ann Intern Med 2003; 139: 214-26.
3. Franceschini N, Napravnik S, Eron JJ Jr, Szczech LA, Finn WF. Incidence and etiology of acute renal failure among ambulatory HIV-infected patients. Kidney Int 2005; 67: 1526-31.
4. Chawla LS, Eggers PW, Star RA, Kimmel PL. Acute kidney injury and chronic kidney disease as interconnected syndromes. N Engl J Med 2014; 371: 58-66.

THANK YOU

A Case of Breathlessness

Dr Vishal Asrani
Jr II
Department of medicine

Chief complaint

46 yr old female presented with complaint of

Progressive breathlessness on exertion

Fever , intermittent, low to moderate grade

1month

Swelling over feet and facial puffiness

She also had

- Joint pain in small hand joints & early morning stiffness for 30 minutes, increasing in cold weather since 2 months.

- Reddening of cheeks especially on exposure to sunlight intermittently over last 6 months.

No H/O

Chest pain, syncope and palpitations

Cough , oliguria

Weight loss , loss of appetite or jaundice

Tuberculosis , diabetes ,hypertension

General Examination

- Patient was afebrile
- Pulse rate – 120 beats /min, regular,low volume
- BP – 80/60 mm Hg right upper limb supine position
- Tachypneic, RR = 30/min

- JVP – Increased 10 cm
- Bilateral pitting edema in lower limbs till knee
- Mild pallor, no clubbing, cyanosis, lymphadenopathy, icterus
- Reddish macular malar rash was seen sparing the nasolabial folds

Systemic Examination

- CVS- muffled heart sounds
- RS – Bilateral vesicular breath sounds no adventitious sounds
- CNS – conscious and oriented and no neurological deficit
- P/A – soft and no organomegaly

Musculoskeletal examination

Tender joints – 1st 2nd 3rd PIP in right hand

1st 2nd 3rd 4th PIP in left hand

both wrists

Swollen joint - 1st 2nd PIP in right hand

1st 2nd PIP in left hand

•Laboratory Investigations:-

HAEMOGLOBIN	**9.2 gm/dl**
TLC	5200/cmm
PLATELETS	80000/cmm
ESR	22 mm fall in 1 hr
Polymorphs	72%
Lymphocytes	20%
Eosinophil	2%
Monocytes	6%
Urine microscopy	**No proteinuria**

Serum electrolyte

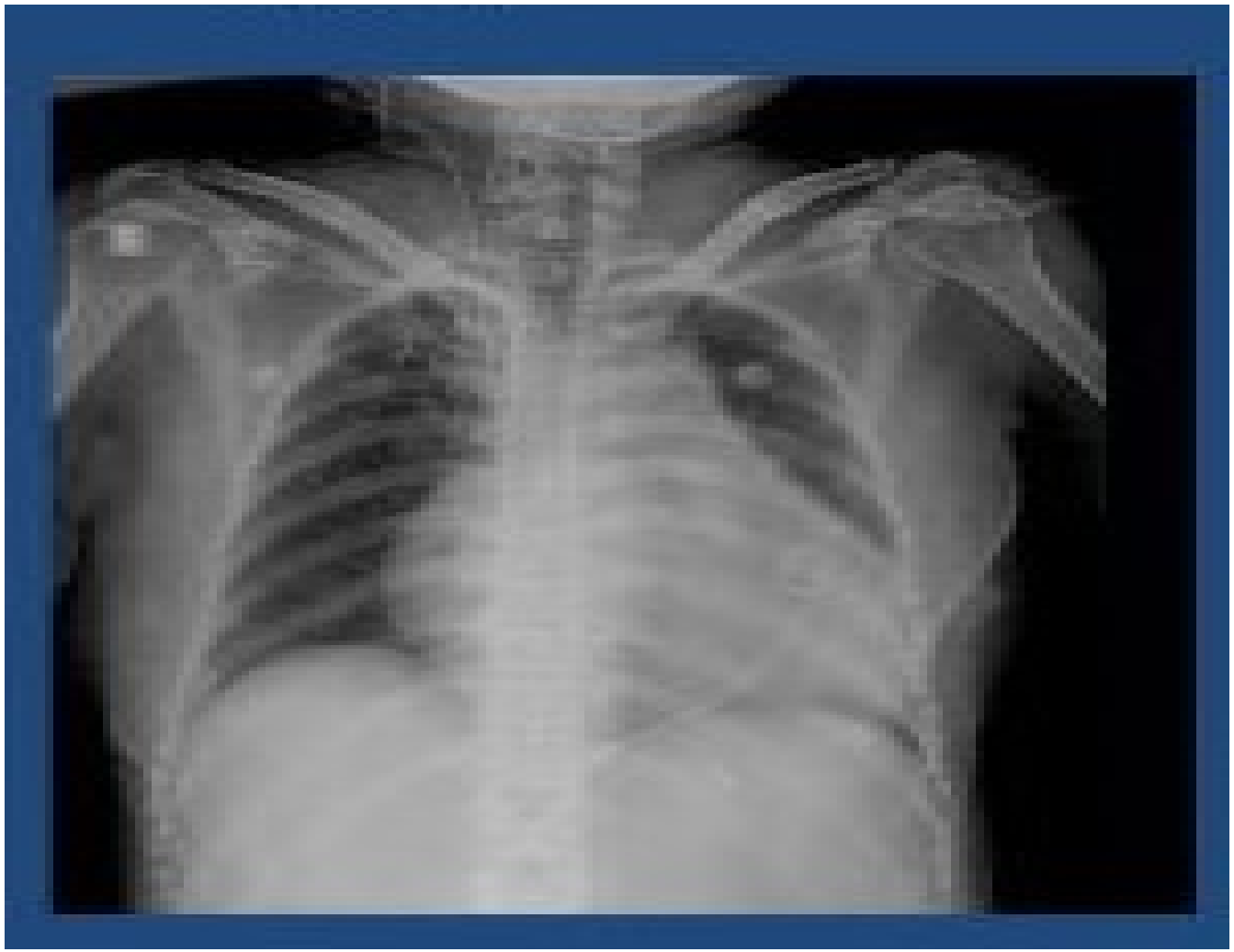
liver function tests

Normal

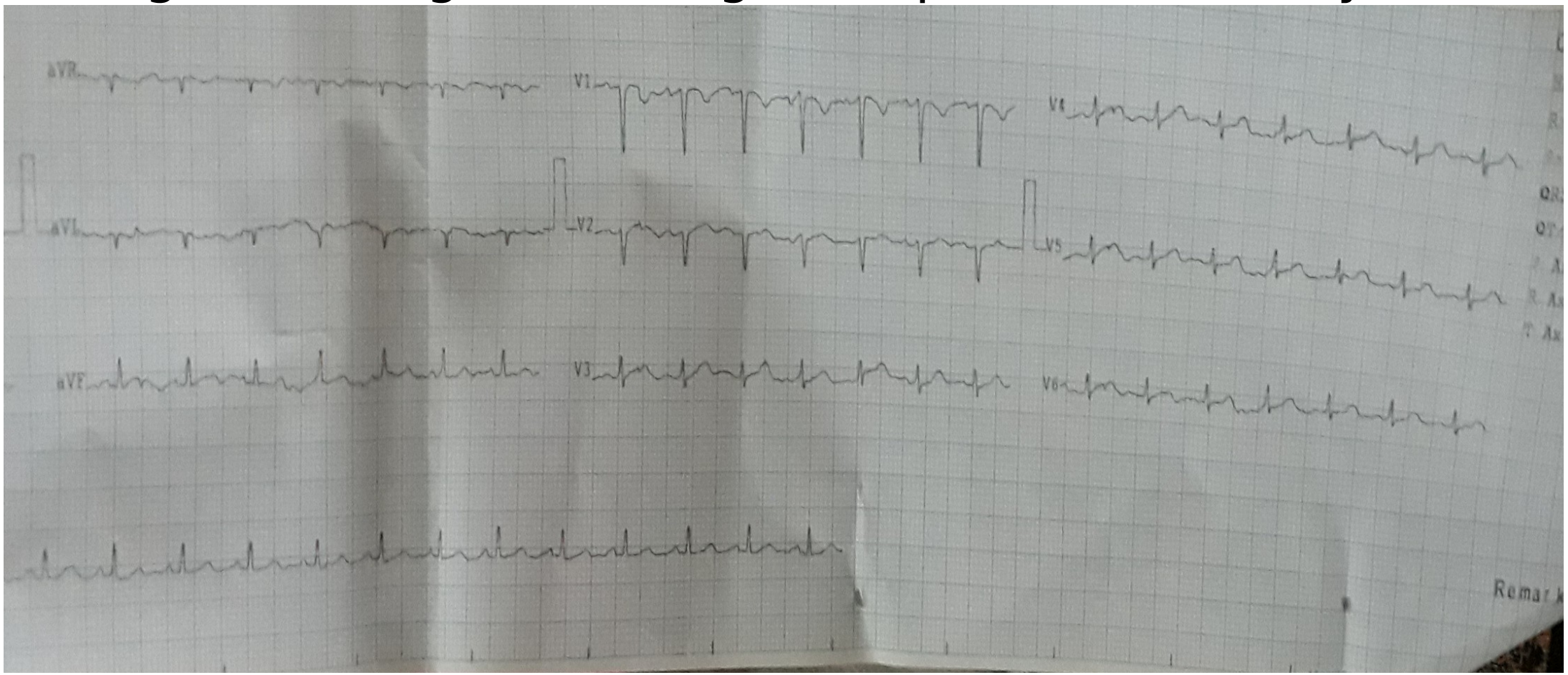
renal function tests

thyroid function tests

Chest Xray - shows cardiomegaly



Ecg – showing low voltage complexes and tachycardia



2D – Echo - Showed large pericardial effusion

with tamponade with a

Diastolic

Right Ventricular free wall

Pericardiocentesis with pig tail catheter insertion was done

Fluid was hemorrhagic–

RBC – 1.5 million/cumm

Cell counts

Total Cells- 1480 / cumm

Glucose - 141

Protein - 5.6 gm%

Polymorphs - 28%

lymphocytes- 68%

Macrophages – 04%

Fluid ZN stain was negative

Cytology – was negative for malignant cells

Fluid ADA – 50 IU/L(BORDERLINE POSITIVE FOR TUBERCULOSIS)

Patient was treated with AKT and the AKT was stopped after ruling out tuberculosis

- **ANA(ANTI NUCLEAR ANTIBODY)** - positive **ANA by ELISA – 5.4 OD ratio**

- **ANA blot was** positive for

  U1RNP(Ribo nucleo protein)

  Anti SM (strongly positive)

  Nucleosome

FINAL DIAGNOSIS

- On the basis of clinical correlation of features such as presence of
  Pericardial effusion(serositis)
  Thrombocytopenia
  Malar skin rash
  positive immunological markers for SLE

The final diagnosis was

SYSTEMIC LUPUS ERYTHROMATOSUS with Cardiac Tamponade as the initial presentation

Patient was started ---

Tab prednisolone 1mg/kg/day :60mg/day

Tab azathioprine – 50 mg OD

Tab hydroxychloroquine 200 mg BD and

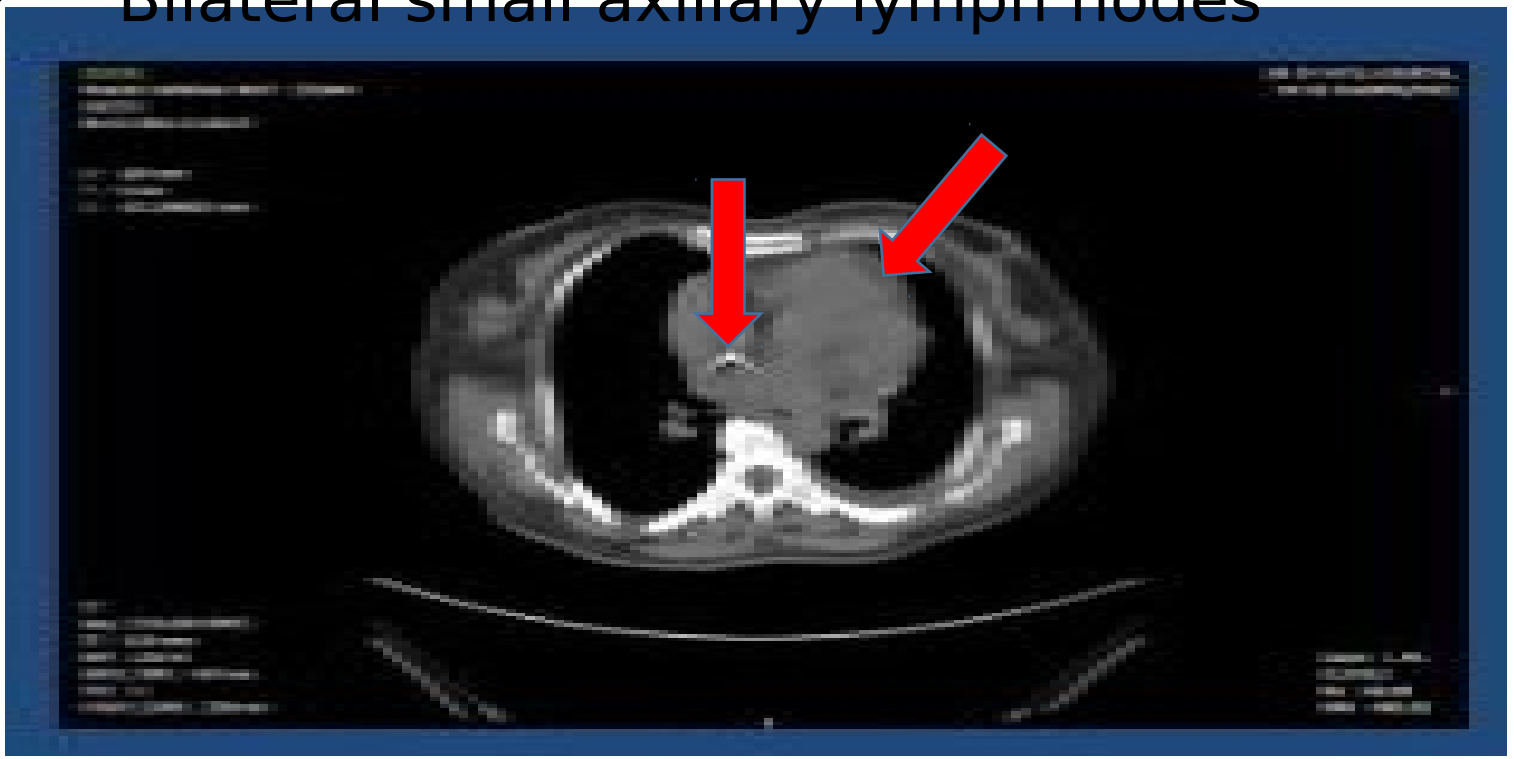
Tab torsemide 40 mg BD

Pericardial fluid was drained daily till no fluid came through the catheter

Serial 2D ECHO showed minimal pericardial effusion with normal cardiac function.

HRCT Thorax was also done which showed

- Bilateral mild pleural effusion
- Mild pericardial effusion with radiopaque drain in situ
- Bilateral small axillary lymph nodes



- Pigtail Catheter was removed . Patient responded well to the treatment and was discharged on the above treatment

At present:

Patient has no dyspnea and oedema.

The malar rash has resolved fully.

Joint pains have significantly reduced

Her platelet count on follow up was 1,56,000/cmm

2D Echo is normal.

DISCUSSION

- Pericarditis may occur in approximately 25% of SLE patients.

  Pericardial effusions may be asymptomatic and are usually mild.

- **Cardiac tamponade, especially as an initial form of presentation and also throughout the disease, is unusual**. occuring in about 1 – 2 % of patients with SLE according to literature
- The main differentials are :

  Tuberculosis

- Patient may present with fever, dyspnoea, and congestive heart failure. Clinical features of left ventricular dysfunction, non-specific ST-T wave changes, and decreased ejection fraction are found in >80% of patients

- Pericardial fluid in SLE is exudative and can be hemorrhagic.

- Pericardiocentesis is life saving in cardiac tamponade .

- Medium to high dose steroid therapy ,azathioprine , hydroxychloroquine is the treatment of choice for the underlying SLE

Thank you

An Unusual Case of Abnormal Behaviour and Seizures

DR. Shweta Deshmukh
DEPARTMENT OF MEDICINE
MEDICINE JR II

- A 15 year old male patient presented to OPD with complaints of :

- Generalized tonic clonic seizures (3 episodes) 20 days back
- Abnormal behaviour and Irrelevant talk since 20 days

- Patient was apparently alright a month back .
- Patient had fever, mild grade, relieved on taking medication and not associated with headache and vomiting which lasted for 2 days and recovered completely.
- After a week, patient developed 3 episodes of GTCS on same day.
- Following admission in our hospital , patient had abnormal behaviour like he used to do self muttering and had visual hallucinations and also auditory hallucinations in the form of shooting someone along with the sounds of gunshots like that in a video game (battle field) which he used to play at home and he

• Past history :

- No similar complaints in the past.
- No History of DM / TB / HTN / Seizure Disorder/head injury.
- Sleep- Disturbed .
- He was an average student in school before the symptoms appeared.
- No other significant personal and family history

On examination :

Afebrile

Pulse- 86/min

BP- 120/80mmof hg

RR-18/min

Spo2- 99%on RA

· Systemic examination :

- **CNS**:

➢Conscious.

➢orientation to place and time disturbed along with past memory .

➢ Patient was elated and had euphoric mood.

➢He also had auditory and visual hallucinations . **MMSE : 21.**

➢Cranial nerves , motor system and sensory system examination was normal.

- CVS: S1S2 heard
- RS: AEBE
- PA: Soft and Non tender

Investigations :

Investigation	Values
CBC	WNL
RFTS	WNL
LFTS	WNL
Sr. electrolytes	WNL
Sr. proteins	WNL
Sr. ca , ph , Mg	WNL
Urine R/M	WNL
BSL ®	

Investigations	Values
HIV	Negative
HBsAg	Negative
HCV	Negative
Dengue	Negative
Widal	Negative
RMT	Negative
Sr. VDRL	Negative
Blood culture	Negative

- **MRI brain** : No Abnormality **USG(AP):**normal
- **Slit lamp examination :** no KF ring .
- **CSF Examination :**

• **R/M :-**

1. Sugars: 73 (NR- 40 to 80 dL)

2. Proteins**: 92.7 (NR- 15 to 45 mg/dL)**

3. Cells**: 20 Lymphocytic cells (no RBCs) (NR- 0 to 5 cells)**

•.**C/S :-** No Growth

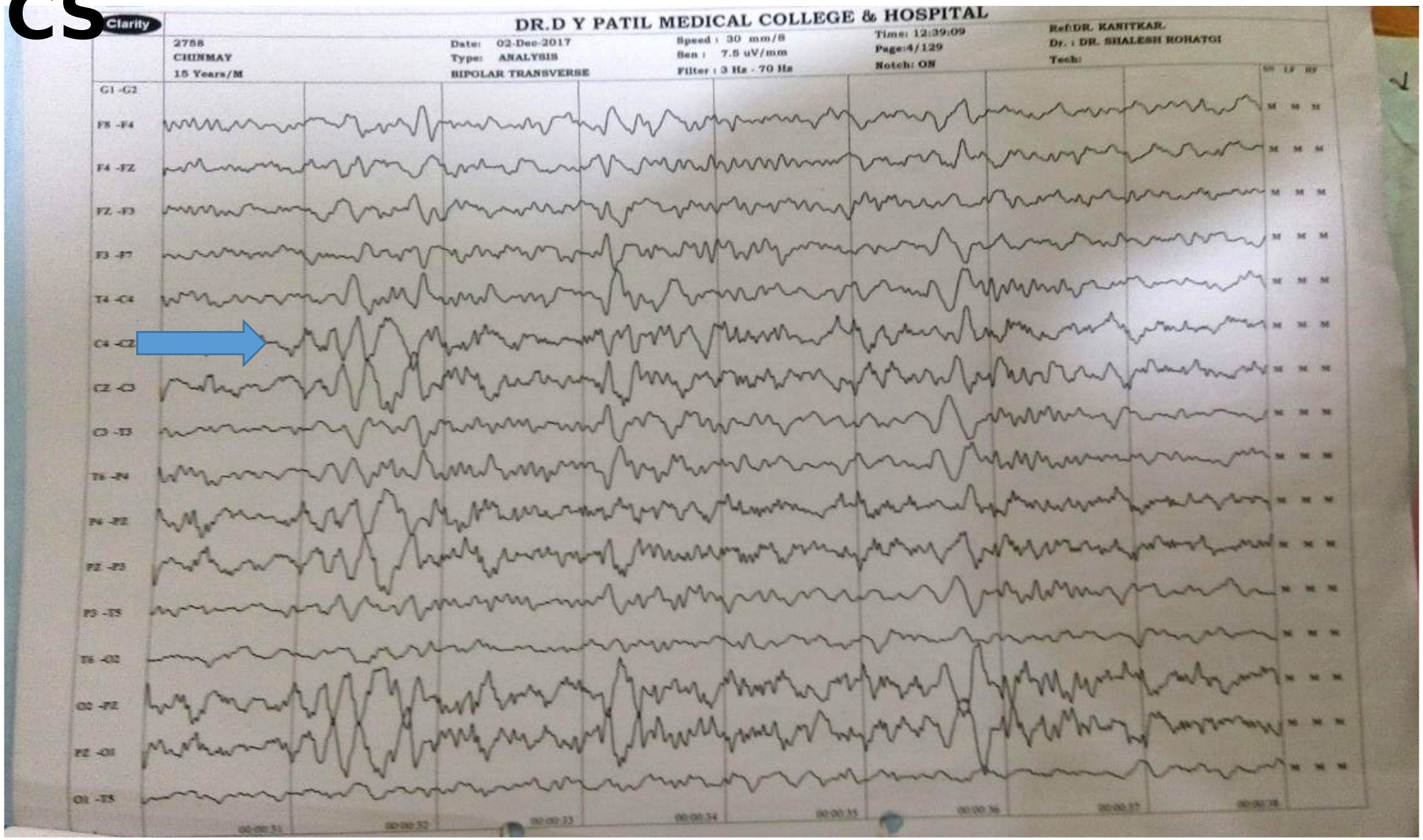
Differential diagnosis :

- After looking at clinical and CSF picture we made a differential diagnosis of and accordingly further work up planned :

- ? Viral encephalitis (infectious cause)
- ? Autoimmune encephalitis (immune cause)
- ? Hashimotos encephalitis
- ? Idiopathic

- CSF : HSV PCR : not detectable (most common viral cause)
- TFT and ANTI TPO antibodies : negative
- EEG : s/o **Records of Generalized Seizures**.

EEG showing sharp waves s/0 records of GTCS



- Then we thought of ? **Autoimmune CAUSE**

- **ANTI NMDA (N-methyl D-aspartate Receptor) Antibodies:** Strongly **Positive.**

Hence made a diagnosis of

Autoimmune NMDR encephalitis.

Treatment given :

- On admission we started on antiepileptics , tab phenytoin 100 mg BD and tab lacosamide 50mg BD and tab haloperidol HS.
- Then after diagnosis we started with inj methyl prednisolone 500 mg for 5 days .
- And also IVIg 15 gm OD for 3days given and oral prednisolone 40mg od we continued.
- Now patient is showing slow progressive improvement in his abnormal euphoric behaviour.

Autoimmune encephalitis :

- Autoimmune encephalitis is a group of neuropsychiatry disorders that causes sub acute deficits of memory and cognition ,often followed by suppressed level of consciousness or coma.

- Appropriate autoantibody testing can confirm specific diagnoses, although this is often done in parallel with exclusion of infectious and other causes.

- There are many autoimmune antibodies eg: Voltage gated K+ channel antibodies, GAD-65, Anti LG 1 ,anti GABA antibodies.

- Among these anti NMDR antibodies are most common in young age group a/w tumours(40-50%) (teratomas of ovaries),many cases are not a/w tumours

- Anti NMDR encephalitis has characteristic clinical symptoms of psychosis and memory impairment early along with abnormal movement, seizures, and depressed levels of consciousness emerging later.
- The response to immunotherapy is good but may take many months to reach its full effects .
- The incidence is **2-3 / 1,00,000** cases of encephalitis.
- 40% cases are due to infections, 40% cases are idiopathic , **only 20% cases are immune mediated.** (With **the largest being Anti NMDA**)

Take Home Message:

- Autoimmune encephalitis is a difficult clinical diagnosis due to the similarities in the clinical and imaging findings of many forms of autoimmune and infectious encephalitis. Hence if a patient is presenting with impaired memory, cognition and seizures, then we should also think of autoimmune encephalitis .

- If a clear autoimmune cause for the symptoms is established, treatment with IVIg and steroids is the main stay. Immunotherapy should be continued till resolution of symptoms and disappearance of anti-NMDR antibodies.

References

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Thank you

A Rare Form of Epilepsy

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Jr II

- 14 year old boy from the district of Yelandur , Karnataka, came with c/o
  - 4 episodes of tonic limb movements followed by loss of consciousness for 10-15 mins since last 3 months,
  - last episode were 4 days prior to hospitalization.
  - Episodes were associated with
    - uprolling of the eyes,
    - frothing from the mouth and

- No tongue bite , clonic movements or head trauma.
- On further interrogation , patient had seizure episodes during hot water bath.
- No similar episodes during any other routine activities.
- No history of fever preceding the seizure episode.

Past History

- No developmental delays.
- Normal birth history.
- No history of febrile seizures in the past
- No history of head trauma
- No similar complaints in the family

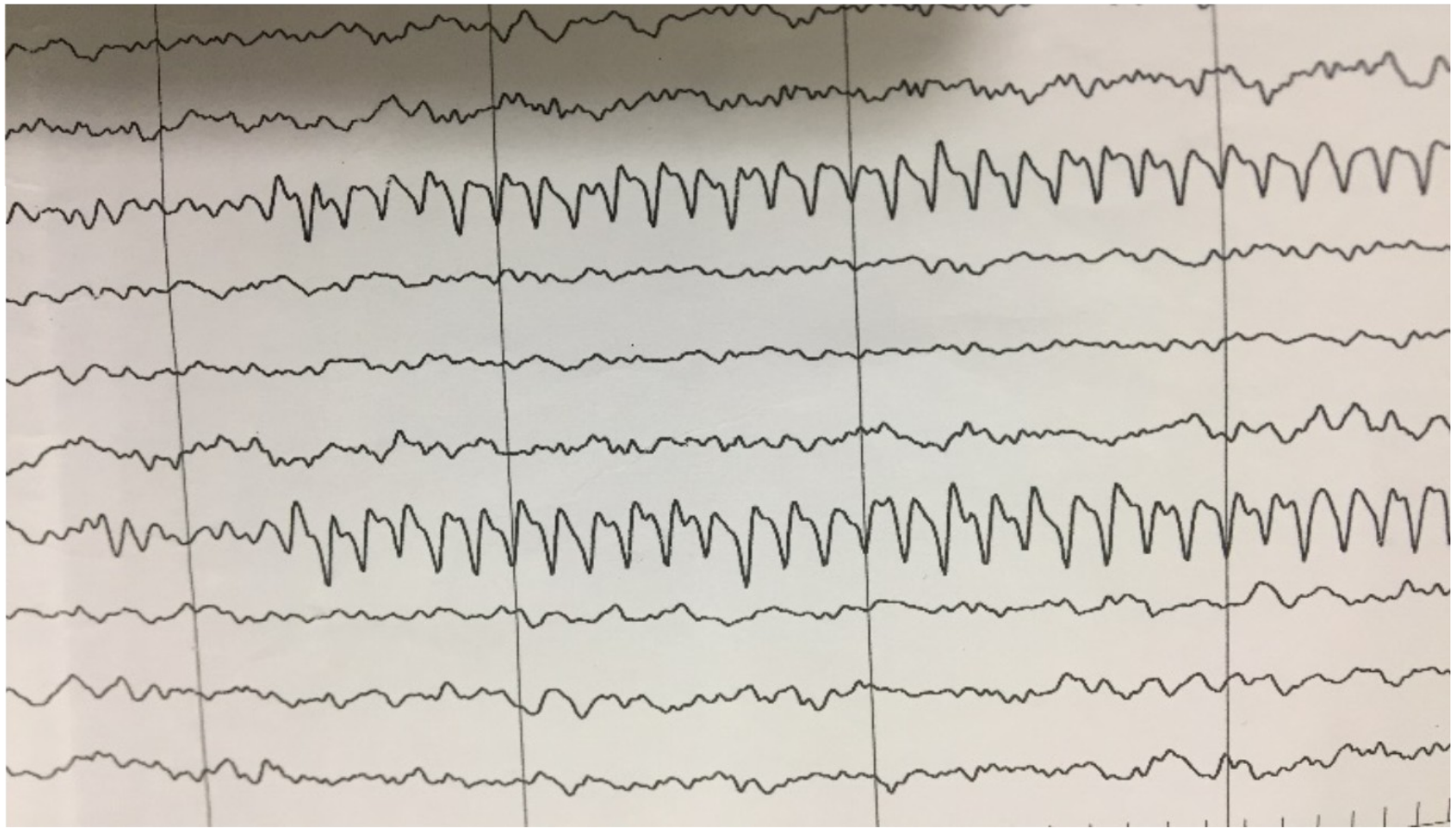
Examination

- Both General and systemic examination was normal.
- No neurological deficit.

Investigations

All laboratory investigations were normal

- EEG - Suggestive of Right hemispherical dysrhythmia.
- MRI Brain – Normal Study.



From the history , geographical birth location of the patient , and investigations , diagnosis of “Hot water epilepsy” was made.

Treatment

- Patient was advised to have lukewarm water bath and to take Tab Clobazam 5 mg 1 hour prior to the bath intermittently.

- On follow up -

No seizure episode were noted during hospitalization.

Discussion

- Reflex epilepsy is a condition in which seizures can be provoked by external stimulus or internal mental process.
- It may manifest as partial or generalised seizures.

- Triggering factors can be
  1. Visual stimuli
  2. Somatosensory – light touch , tapping , immersion in hot water
  3. Auditory stimuli
  4. Movement induced reflex – nonketotic hypergylcemia
  5. Complex actions and mental processes
     Reading , eating , micturation , walking , laughing.

Seizures triggered by immersion in hot water were first described in 1945 from New Zealand, After this, there were isolated case reports from all round the world:
Australia, United States of America, Canada,United Kingdom, and Japan.

A large number of patients with this type of epilepsy have been reported from India

- Indian patients are typically boys , with onset of 13 years of age who are reported to have complex partial or GTCS seizures during ritual bathing when jugs of hot water (>45 C) are poured over the head.

- A large population study (BURN) in and around the Bangalore, reported that HWE accounts for 6.9% of all epilepsies in this community, with prevalence of 60 per 100,000 which was published as an epidemiologic study from Yelandur , a rural area near Mysore

- Classification proposed by the International League Against Epilepsy (ILAE) task force in 2001 includes HWE under the reflex epilepsies

- HWE patients probably have an aberrant thermoregulatory system and are extremely sensitive to the rapid increase in temperature occurring during hot water head baths, which precipitates seizures.

- Familial HWE cases with more than one affected member have been noted in 7–15% of Indian

- Interictal scalp EEG is usually normal, but 15–20% might show diffuse abnormalities.

- Neuroimaging has been unremarkable in most but focal cortical malformations has been noted.

- Treatment includes prophylactic clobazam and avoidance of sudden exposure of head to the large volume of hot water.

- Antiepileptic therapy is only indicated when above measures failed and when patient continues to have

Take Home Message

Epilepsy has vast number of etiological factors hence
it is important to route out the cause of the epilepsy rather then just starting the patients on the antiepileptic measures as antiepileptics are not indicated in all types of epilepsies.

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Thank you!

An Interesting Case of Quadriparesis

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Department of Medicine

History

- A 30 year old female came with chief complaints
- Weakness both lower limbs- 5months
- Difficulty in neck holding- 5 months
- Weakness of both upper limbs- 3 months
- weakness was mainly proximal. Had difficulty in getting up in bed
- Insidious onset and progressive

- No history suggestive of of sensory symptoms, bowel and bladder involvement, cranial nerve involvement , involuntary movements

- She gave history of weight loss of 5 to 6 kgs in 5 months she also noticed a painless swelling in left lower abdomen, insidious in onset, gradually increasing in size.

- No history of vaginal bleeding or discharge.

- No h/o skin rash, joint pains, ulcers
- No significant past or family history
- Obstetrics History -G2 P0 L0 A2
- Menstrual history- normal
- Impression – gradually progressive quadriparesis with truncal weakness and abdominal mass under evaluation.

On examination

General examination:

No skin rash or lesions.

Temperature, pulse ,respiratory rate and BP normal.

Systemic examination:

- Per abdomen examination

  - left iliac region swelling,
  - a 10*5cm mass
  - well defined margins, surface smooth, hard ,not moving with respiration, no local rise of temperature,fixed, no bruit.

CNS Examination:

- Normal higher functions and cranial nerves
- **Motor:**
  - Weakness of neck extension
  - Truncal weakness
  - Hypotonia in all limbs,
  - Power in all the limbs
    - Proximally was grade 1,
    - Distally power was 4
  - Deep tendon reflexes were diminished in all limbs.
  - Planters were flexors.
- **Sensory system-** Normal
- No signs of meningeal irritation

- Other Systems

  Cardiovascular and Respiratory system – Normal.

- **Clinical diagnosis-**
- Polymyositis with abdominal mass? Malignancy
- Cause- ?Paraneoplastic

INVESTIGATIONS

CBC,LFT,RFT-NORMAL

SEROLOGY-HIV, HbSAg & Anti HCV - Negative

CSF-NORMAL

ESR-60mm/hr (0-29mm/hr)

CA125-1589u/ml (0-35)

CPK NAC-609IU/L (38-176)

AFP-1.85IU/ml (0.5-5.5)

CEA-3.3ng/ml (<0.5)

BETA HCG-1.2MIU/ml(<10)

ANA BLOT-NEGATIVE

IMAGING

- **USG ABDO PELVIS-**

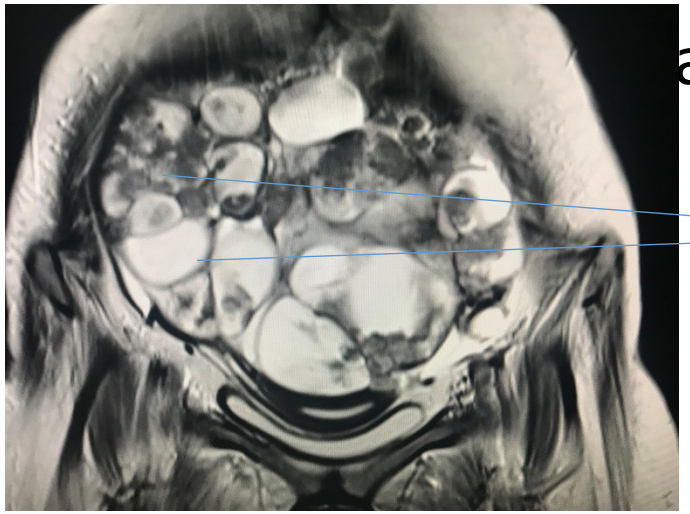
S/O-b/l cystic ovaries with increased vascularity ? Neoplastic lesion.

- **CECT ABDO PELVIS PLAIN AND CONTRAST:**

S/O carcinoma ovary with L3 vertebral metastasis,4 to 10 ribs metastasis.

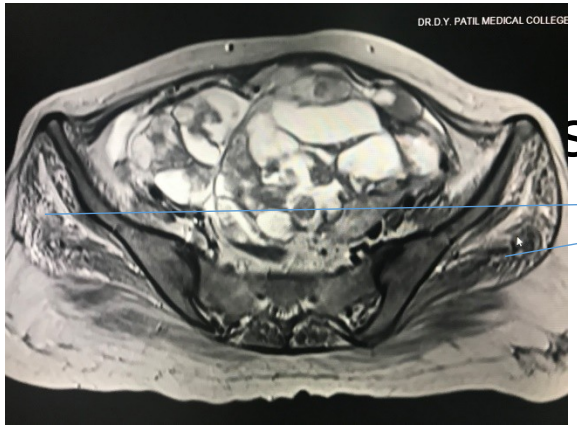
- **MRI ABDO PELVIS:**

Both ovaries are separately not visualized -s/o-Neoplastic mass arising from ovary



ary needs consideration.

Bilateral multiple solid and cystic lesions – carcinoma ovary



s and muscles in upper thigh appear hyperintense – due to

Hyperintensity in bilateral gluteal muscles

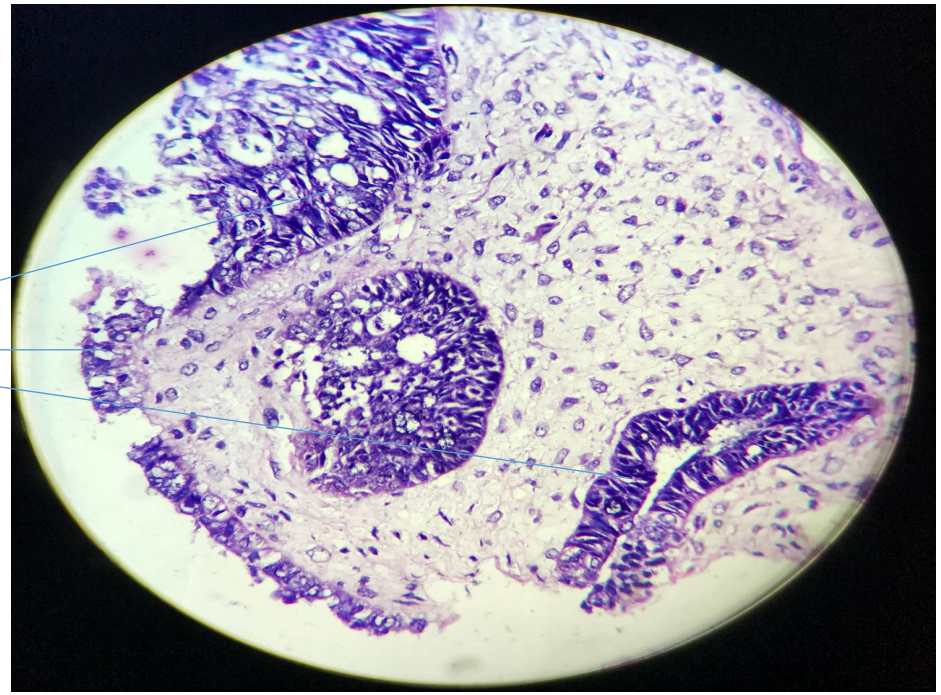
Neuro-electrophysiology

- **NCV -** Normal
- **EMG-** Increased insertional activity
  - Increased spontaneous acivity
  - Low motor unit actional potentials
  - S/O- inflammatory muscle disease.

Biopsy

- CT Guided Left Ovarian Mass Biopsy – S/O Mucinous Cystadenocarcinoma.

simple non-stratified columnar cells resembling gastric intestinal epithelium



- Muscle Biopsy Of Left Vastus Lateralis was S/O- Myositis.

TREATMENT DURING HOSPITALISATION

- Physiotherapy

- Tab Prednisolone 50 mg od.
- Tab Azathioprine 50 mg od.

- Was referred to gynecologist and oncophysician

Discussion

- **Causes of inflammatory muscle disorders**

  - Primary polymyositis
  - Dermatomyositis
  - Polymyositis assoctaed with malignancy
  - Polymyositis assoctaed with connective tissue disorder
  - Inclusion body myositis
  - Idiopathic.

- The hallmark of these disorders is muscle weakness.specially proximal and truncal weakness , neck muscle weakness.

- DM and PM are associated with malignancies only in a minority of cases.

- The risk appears to be higher in women.

- The most common tumors are cancer of the breast, lung, ovary, stomach, and non-Hodgkin lymphoma.

- PATHOPHYSIOLOGY-

  Antigens targeted for an immune response are expressed both in the inciting tumor and the affected neuronal tissue.

  Histidyl t-RNA synthetase- as an epitope

- TREATMENT:

  Removal of tumour and immunusuppresion with steroids.

Take home message

All the women with polymyositis and dermatomyosotis should undergo cancer screening.

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Thank you

Case of Pancytopenia with Recurrent Jaundice

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JR II

Case:-

- 59 year old male came to our OPD with complaints of

  - Generalized weakness
  - Breathlessness
  - yellowish discoloration of urine and eyes.
  - Loose stools on and off

Since 2 months

Past history

- History of recurrent episodes of yellowish discoloration sclera and urine over the past 4 years which were present for few days and subsided on treatment.
- Bleeding per rectum since 4 years.
- History of 4 blood transfusions over the past 4 years.
- History of hemorrhoidectomy 2 years back.

- Chronic alcoholic for 6 years. Abstinence since 10 years.
- No previous medical documents were available.

On examination

- Conscious, oriented, Averagely nourished
- Pulse : 100/min
- BP : 110/70 mm Hg
- Afebrile
- Pallor +++
- Icterus +
- No Cyanosis,Clubbing,Lymphadenopathy,Edema

- S/E :
  - CVS: normal
  - RS: normal
  - P/A: soft, non tender, no Hepato-Splenomegaly, no free fluid
  - CNS: normal

Laboratory investigations on admission:

- Hb: 4.0 gm%
- PBS: macrocytic normochromic
- RBC indices

  PCV- 12.0%

  MCV-118.8fl

  MCH- 31.8pg

  MCHC-33.3g/dl
- TLC: 2400/mm^3
  - P- 65%, L- 30%, E- 01%, M- 04%
- Platelet count: 60,000/mm^3

LIVER FUNTION TESTS

- S.Bilirubin: 2.3mg%
  - Direct: 0.6mg%
  - Indirect : 1.7mg%
- S. ALT(SGPT): 15 IU/L
- S. AST(SGOT): 41 IU/L
- S. ALP: 51 IU/L
- PT/ INR: 1.2
- S. Protein: 6.0 gm%
  - S. Albumin: 3.7gm%
  - S. Globulin: 2.3gm%

- Blood Urea: 29 mg %
- S. Creatinine: 0.9 mg%

- S. Calcium: 9.7 mg %
- S. Phosphorous: 4.5 mg%
- S. Uric Acid: 8.2 mg%

- S. Amylase: 60 IU/L
- S. Lipase: 36 IU/L

- Sr. B12 levels – 183 pg/mL (191-663 pg/ml)
- Serum folate – 3.5 ng/ml (3.63- 26.6 ng/ml)

- S. Sodium: 137 mmol/l
- S. Potassium: 4.2 mmol/l
- S. Magnesium: 2.0 mg%
- Random Blood Sugar : 107 mg%
- TFT – within normal limits.
- HIV: non-reactive
- HBsAg: negative
- Anti HCV: negative
- HB electrophoresis was normal

Provisional Diagnosis-

- Pancytopenia with Mild Unconjugated Hyperbilirubinemia was evaluated further.

- Fasting lipid profile:
  - S. Cholesterol: 82 mg%
  - S. Triglycerides: 76 mg%
  - S. HDL: 19 mg%
  - S. LDL: 43 mg%

- Stool for OB: negative
- Stool for fat globules: positive
- Urinary D xylose test (25gm) – 3gm

- Bone marrow aspiration showed hypercellular marrow with erythroid hyperplasia.

  Giant metamyelocytes, megakaryocytes seen.

- OGD scopy suggestive of lax Lower oesophagial sphincter, grade B reflux esophagitis and sliding hiatus hernia

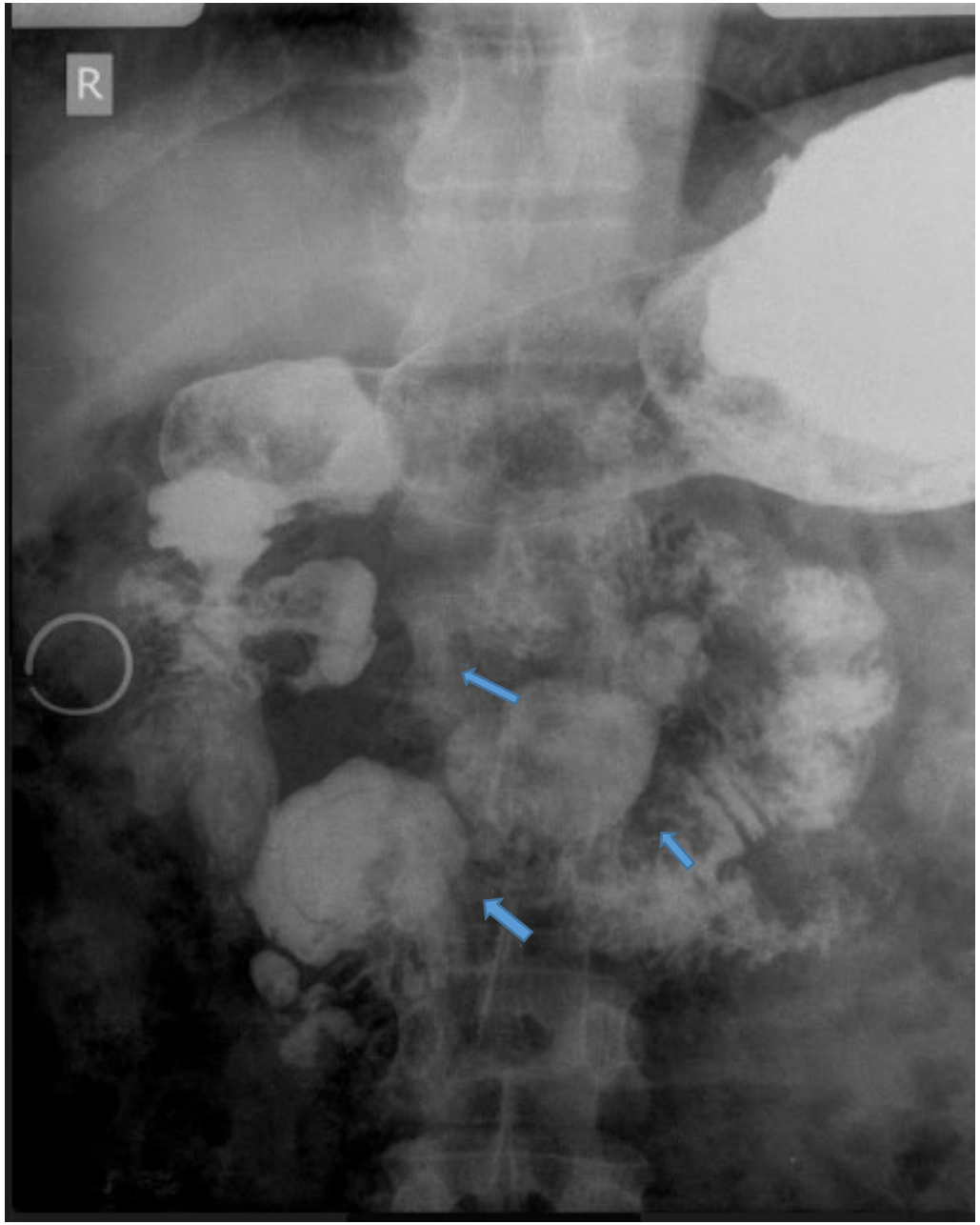
- **USG Abdomen & Pelvis:**

  - Right kidney shows raised echogenicity with partial to complete loss of CMD and entire kidney shows multiple cysts largest of size 54x52mm.
  - Rest normal
  - Portal doppler normal

- **CT Abdomen & Pelvis with contrast:**

  - Mild IHBR dilatation and dilated CBD secondary to duodenal diverticulum from second part causing compression of lower CBD.
  - Small sized right kidney with loss of CMD due to chronic obstructive changes caused by a staghorn calculus and 2 calculi at upper and middle pole. Non excretion of contrast from right kidney. 2 cystic lesions, largest measuring 7.4x5.8cm

- Barium meal follow through:
  - Outpouching suggestive of diverticulum arising from medial wall of second part of duodenum, superior aspect of third part of duodenum and proximal jejunum.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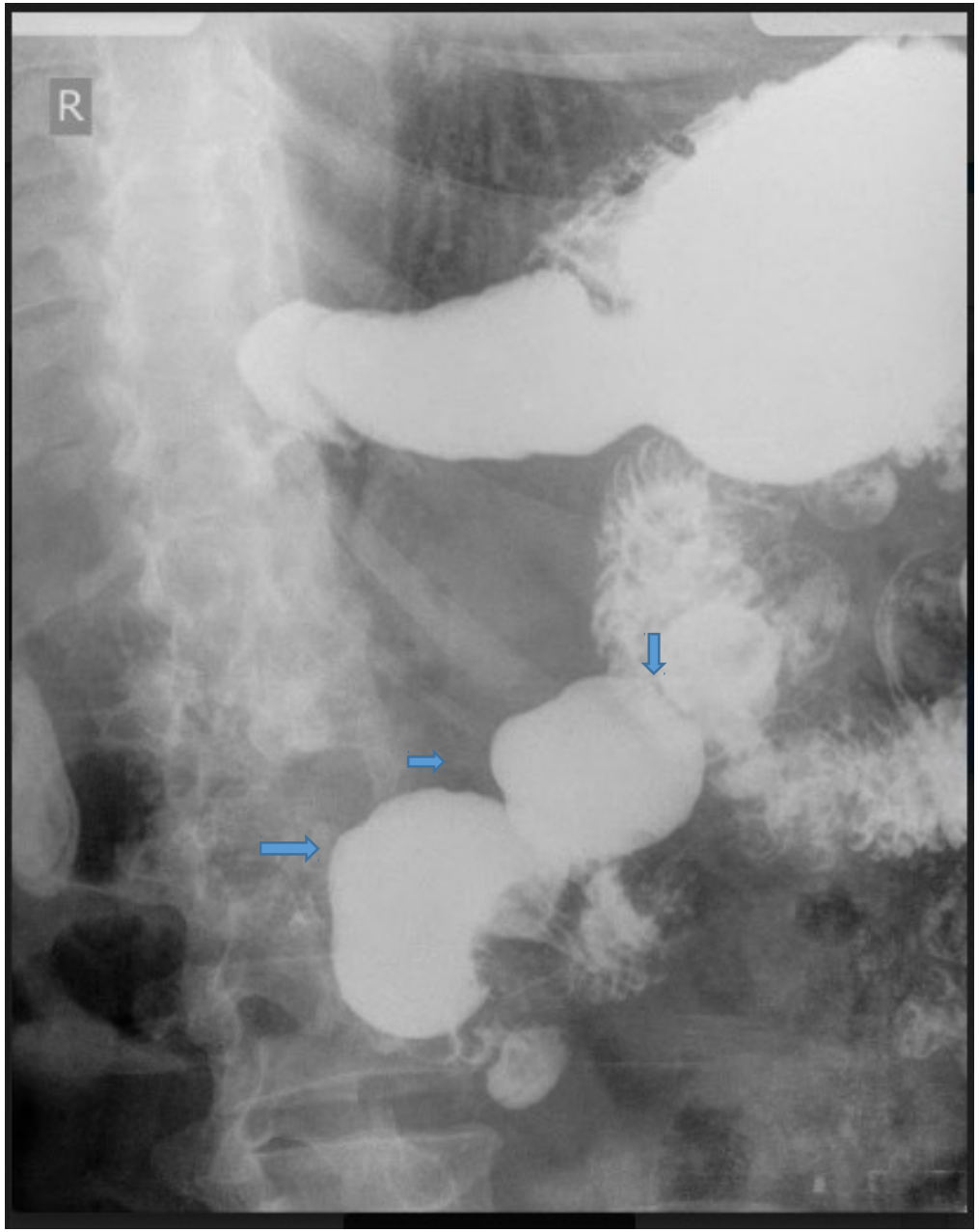
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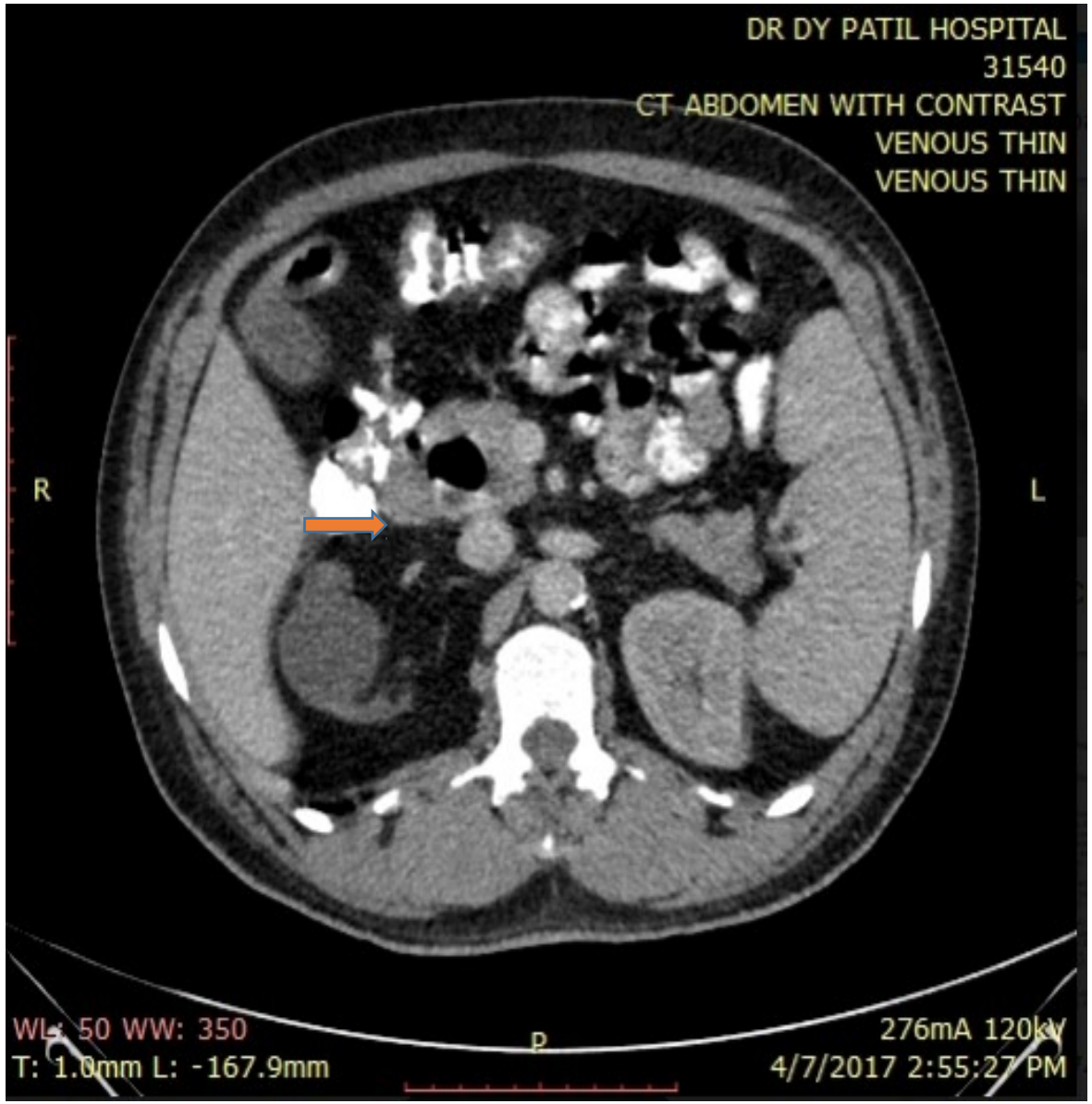
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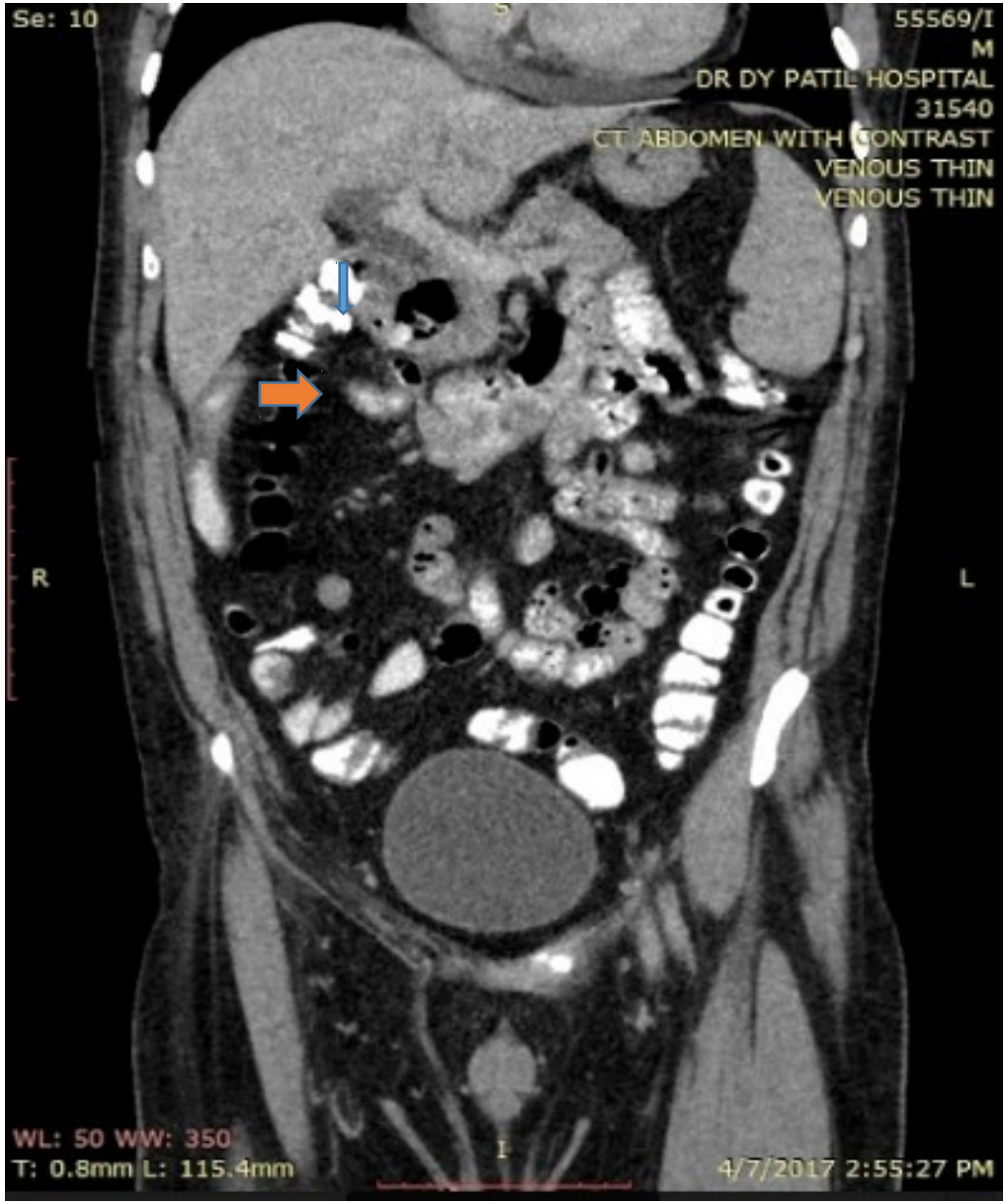
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- Patient was treated with
  - Inj. Cyanocobalamin 1000 mcg OD for one week, followed by alternate days for one week, and later on once weekly for one month.
  - Advised to continue Inj. Cyanocobalamin 1000mcg once a month for 6 months.
  - Folate supplementation was given
  - Tab. Doxycycline 100mg BD for 3weeks.
  - Tab. Rifaxamine 550mg BD for 3weeks
- He showed improvement after treatment

- Hb – 6.4g%
- TLC – 10600/cumm
- Platelet count – 2.4 lakh/cumm

- Reticulocyte count – 5%
- Serum LDH – 246 IU/L

Final Diagnosis

Severe Megaloblastic Anemia due to Malabsorption caused by bacterial overgrowth in extensive duodenal and jejunal diverticulosis.

Discussion

- The cause of diverticula is largely unknown. Many patients have an underlying intestinal motility disorder.
- Periodic elevated intraluminal pressures can lead to herniation through areas of weakness at the mesenteric border where blood vessels penetrate the muscularis.
- Patients with multiple duodenal diverticula may develop bacterial overgrowth.
- Malabsorption may result from associated bacterial overgrowth.

- Bacterial overgrowth syndromes comprise a group of disorders with diarrhea, steatorrhea, and macrocytic anemia whose common feature is the proliferation of colonic-type bacteria within the small intestine.

- This bacterial proliferation is due to stasis caused by impaired peristalsis

- Patients with jejunal diverticula usually are asymptomatic unless bacterial overgrowth within the diverticula is sufficient to cause vitamin B12 deficiency, by uptake of the vitamin by the bacteria, or malabsorption resulting from bacterial deconjugation of bile salts and impaired lipid digestion.

- “Often the diagnosis of bacterial overgrowth is suspected clinically and confirmed by response to treatment.”[1]

- The administration of broad-spectrum antibiotics usually constitutes effective treatment that suppresses bacterial flora.[2]
- Diverticula of the duodenum are often found near the ampulla of Vater, but rarely case obstruction of the bile duct.
- If obstruction occurs it is partial and jaundice intermittent.
- Duodenal diverticula are typically diagnosed on upper GI X-rays. They are easily missed on endoscopy unless a side-viewing endoscope is used.

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Thank you

A CASE OF QUADRIPARESIS

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Department of Medicine

Clinical History

- A 27 yr male, shopkeeper by occupation presented with chief complaints of
  - High grade fever with chills - 3 days
  - Weakness of all 4 limbs - 1 day
  - Bodyache - 1 day

No h/o burning micturition, cough, headache, altered behaviour, trauma, diarrhoea, rigorous exercise.

No addictions.

Mixed diet

Past/Personal/Family History – Not Significant

General examination on admission

- Temperature 100°F
- Blood Pressure – 130/70mm of Hg
- Pulse rate – 110/min
- Blanching rash present all over the body
- Single breath count of 50
- SPO2 97% on room air

Systemic examination

Higher mental functions, speech & cranial nerves	Normal
Tone	Reduced in all 4 limbs
Power	3/5 at all joints in all ranges of motion Hand grip reduced by 60% in both hands
Deep tendon reflexes	Biceps, Triceps, Supinator, Knee jerk - absent Ankle reflex present bilaterally
Plantars	Both flexors
Sensory system	Normal
Signs of cerebellar involvement	None
CVS, RS, P/A	All normal

Differentials on admission

- **Guillian Barre syndrome**
- **Electrolyte imbalance induced quadriplegia (Hypokalemic paralysis)**
- **Acute transverse myelitis**

Investigations
Day 1

INVESTIGATIONS	DAY 1
Hb	13.9 gm%
TLC	2600/cumm
DLC	Polymorphonuclear cells 60 % Lymphocytes 40% Eosinophils 5% Monocytes 5%
PLATELET COUNT	37,000/cumm
Peripheral blood smear	Normocytic normochromic, no parasites seen

INVESTIGATIONS	DAY 1
SODIUM	135meq/L
POTASSIUM	2.2meq/L
CALCIUM	9.9g/L
S.Bil	WNL
AST	523U/L
ALT	346U/L
ALP	137U/L
RFT	WNL
ABG	WNL

Other investgations

- Dengue Ns1 Ag & IgM Ab – Positive
- Chikungunya - Negative
- HIV/HBsAg - negative
- Thyroid Function Test – WNL
- CPK total – 533 IU/L (15-190)
- Urine for haemoglobin and myoglobin – negative
- Urine routine microscopy – WNL
- Urinary potassium was – WNL
- Stool routine microscopy – WNL
- Nerve Conduction Study – WNL
- ECG, Chest X-ray, USG of abdomen/pelvis - WNL

Treatment and course of hospital stay

Treatment given

- 40 meq KCl was given in 500 ml N.S. over 6hrs on the first day which resulted in spontaneous improvement in the muscle weakness within 6 hours.
- Syp. Potassium chloride 2tsf three times daily.
- 1.5 litres of i.v. fluids per day
- Tab. Paracetamol 500mg TID

Response to treatment

- S. Potassium had risen to 5.0 meq/l after 24 hours
- Platelet count increased progressively to above 1lakh/cumm over 9 days.

Final Diagnosis

Dengue fever with hypokalemic paralysis

Discussion

- Hypokalemia is a well documented electrolyte imbalance in dengue fever (prevalence -14% to 28%)
- Majority of the patients have mild hypokalemia (not below 3 meq/L)[1] but hypokalemic paralysis is uncommon
- In a 4 year study done on 489 dengue patients in King George Medical College, Lucknow, the incidence of dengue associated hypokalemic paralysis (DHP) was found to be around 3.7% [2]

Possible Mechanisms of DHP

- Redistribution of potassium into the cells
- Transient renal tubular abnormalities leading to increased urinary potassium loss
- Increased catecholamine levels in response to stress of the infection

- Hypokalemic periodic paralysis (HPP) was unlikely because
  - This was the first episode presenting at age of 27 years without any significant past history
  - Was not precipitated by increased exercise or heavy meals rich in carbohydrates.
  - There was also no positive family history suggestive of HPP.

NEUROLOGICAL COMPLICATIONS OF DENGUE

Related to neurotropic effect of the virus	Related to the systemic complications of dengue infection	Post-infection
Encephalitis	Encephalopathy	Acute disseminating encephalomyelitis
Meningitis	Stroke(both hemorrhagic and ischemic)	Encephalomyelitis
Myositis	Hypokalemic paralysis	Myelitis
Rhabdomyolysis	Papilledema	Neuromyelitis optica
Myelitis		Optic neuritis
		Guillain Barre syndrome
		Phrenic neuropathy
		Long thoracic

TAKE HOME MESSAGE

Dengue can present with hypokalemia and rarely it can cause hypokalemic paralysis

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Thank You