Assessment – case base learning

Level- Post Graduates- Dept of Dermatology

Topic- Erythroderma

Answer all the questions. **Time: 20 min**

Each correct response carries 1 mark. No negative marking

1. **Shore nails are seen in**
   a) **Psoriatic erythroderma** b) **Eczemas**
   c) **Malignancy associated erythroderma** d)**Drug induced erythroderma**

2. **List 3 important components of Sezary syndrome**.....

.....

3. **Name the characteristic finding seen in lymph nodes in dermatopathic lymphadenopathy**.....

4. **Name 2infectious causes of erythroderma**.....

5. **What is red man syndrome?**

.....

.....

6. **Name two drugs associated with drug induced erythroderma**

.....

7. **What are the components of erythroderma desquamativum/leiner's Disease** .....

.....

8. **Enlist four important complications of erythroderma**.....

.....

9. **What is "nose sign" seen in erythroderma?**

.....

10. **"Deck chair" sign is classically seen in which condition?**.....

Childhood vitiligo

- Vitiligo is an acquired pigmentary disorder
- Occurring irrespective of age, sex and race
- Cosmetic concern it arouses in the psyche of patients and their family members because of the stigma associated with it

- Present anytime in life, including the neonatal period and childhood
- 50%the disease onset is before 20 years of age
- 25% of the cases, it starts before the age of 10 years
- Childhood vitiligo differs from the adult disease –

✓ Female preponderance is observed
✓ Segmental presentation is more common
✓ Associated other autoimmune or endocrine disorders are rarer

- "segmental" and "non-segmental" types
- Generalized (vitiligo vulgaris, universal vitiligo)
- Localized (focal, mucosal, acrofacial, acral)

- Genetic susceptibility
- Various susceptibility loci (autoimmunity susceptibility gene) for vitiligo are AIS1 (chromosome 1), AIS2 (chromosome 7), AIS3 (chromosome 8) and SLEV1 (chromosome 17)
- "transporter associated with antigen processing protein-1 (TAP-1)" gene is suggestive of the possible role of MHC class I antigen in antimelanocyte autoimmune response.

- NACHT-leucine-rich-repeat protein-1 (NALP1) gene (chromosome 17p13)
- Mutation in the autoimmune regulator (AIRE) gene (chromosome 21q22.3) results in a rare recessive disorder, "autoimmune polyendocrinopathy candidiasis ectodermal dystrophy syndrome (APECED)"
- Link to NALP1 and AIRE gene may explain the association of vitiligo to other autoimmune disorders and presence of circulating autoantibodies in these patients

- Various hypotheses prevail regarding the pathogenesis of NSV
- Autoimmune mechanism
- Autocytotoxic hypothesis
- Aberration of cellular immunity with melanocyte destruction
- Inhibition of melanogenesis
- Aberration of vitamin D3 metabolism

DEBATE

TOPICAL STEROIDS

- Mid-potent topical corticosteroids are the first-line therapy for children with localized vitiligo
- Available studies report a 45-60% response rate to topical steroid in childhood vitiligo

Cho S, Kang HC, Hahm JH. Characteristics of vitiligo in Korean children. Pediatr Dermatol 2000;17:189-93.

Halder RM. Childhood vitiligo. Clin Dermatol 1997;15:899-906.

Palit A, Inamdar AC. Childhood vitiligo , symposium vitiligo. Ind J Dermatol Venerol Leprol 2012; 78: 30-41

- Repigmentation in vitiligo has been observed with the use of topical steroids, especially clobetasol propionate, betamethasone valerate, and even with new molecules like fluticasone and mometasone.

Bleehen SS. The treatment of vitiligo with topical corticosteroids Light and electronmicroscopic studies. Br J Dermatol. 1976;94:43–50.

Kumari J. Vitiligo treated with topical clobetasol propionate. Arch Dermatol. 1984;120:631–5.

Thappa DM. Vitiligo. Indian J Dermatol Venereol Leprol. 2002;68:227–8.

- The clobetasol propionate and betamethasone -17-valerate have been used in different concentrations with success rates by many workers

Kandil E. Treatment of vitiligo with 0.1% betamethasone-17-valerate in isopropyl alcohol: A double blind trial. Br J Dermatol 1974;91:457-460.

Bleehen SS. The treatment of vitiligo with topical corticosteroids. Br J Dermatol 1976;94 (suppl 12):4-50.

Clayton A. A double blind trial of 0.05% clobetasol propionate in the treatment of vitiligo. Br J Dermatol 1977;96:71-73.

- Mechanism of the beneficial effects of costicosteroids in cases of vitiligo remains uncertain. The finding of an increased incidence of autoantibodies in patients with vitiligo, suggests a possible autoimmune basis for the disease. Topical steroids may locally suppress the immunologic changes allowing inactive melanocytes to repopulate affected skin sites

Janak Kumari. Vitiligo treated with topical clobetasol propionate. Arch Dermatol 1984;120:631-635.
Brostoff J, Bor S, Feiwel M. Autoantibodies in patients with vitiligo. Lancet 1969;2:177-178.

- A prospective study showed that: 13 of 23 children (57%) with vitiligo (mean age: 7.9 years, mean duration of vitiligo 1.3 years) treated with a medium strength topical steroid (prednicarbate 0.25%) twice a day for at least four months had 50% or greater repigmentation to all involved skin areas

Cho S, Kang HC, Hahm J. Characteristics of vitiligo in Korean Children. Pediatr Dermatol 2000;17:189-193.

- A prospective study of 12 children with vitiligo (mean age: 13.1 years) showed that 10 children had a mean of 95% repigmentation after a combined treatment of topical corticosteroids in the morning and calcipotriene ointment in the afternoon for an average of 4.5 months (range: 2-7 months)

Travis L, Silverberg N. Calcipotriene and corticosteroid combination therapy for vitiligo. Pediatr Dermatol 2004;21:495-498

- In a double-blind randomized placebo-controlled trial of topical tacrolimus (0.1%) versus topical clobetasol propionate (0.05%) in childhood vitiligo (age 2-16 years), the efficacy of both the therapeutic agents was comparable and no significant adverse effect was recorded in either group

Ho N, Pope E, Weinstein M, Greenberg S, Webster C, Krafchik BR. A double-blind randomized placebo-controlled trial of topical tacrolimus 0.1% versus clobetasol propionate 0.05% in childhood vitiligo. Br J Dermatol 2011;165:626-32

- Forty-five children with stable vitiligo were selected. The age range of the patients was from 2-14 years
- Patients were advised to apply mometasone furoate 0.1% ointment once a day for six months or till complete repigmentation whichever was earlier
- Study revealed complete improvement in 88% of facial vitiligo, 63% of vitiligo of trunk and 61% of vitiligo of extremities after 6 months therapy without any local or systemic side effects

Masuria BL, Batra A, Kotiwala RK, Khuller R, Sing. Topical mometasone furoate for the treatment of childhood vitiligo. Ind J Dermatol Venerol Leprol 1999 ; 65: 219-21

- In an open comparative trial of mometasone cream (0.1%, once-daily application) and pimecrolimus cream (1%, twice-daily application) in the treatment of localized childhood vitiligo, the repigmentation rates were 65% and 42%, respectively, at the end of 3 months
- **Mometasone cream** was found to be **equally effective in all body parts** whereas **pimecrolimus** was effective only **over the face**

Köse O, Arca E, Kurumlu Z. Mometasone cream versus pimecrolimus cream for the treatment of childhood localized vitiligo. J Dermatolog Treat 2010;21:133-9.

- TCIs are slower to exert beneficial effect compared to topical corticosteroid
- Best response is observed on the thinnest areas of the skin (eyelids)
- Calcineurin inhibitors are not yet approved for the treatment of vitiligo and are not recommended for use in children <2 years

Tamesis ME, Morelli JG. Vitiligo treatment in childhood: A state of the art review. Pediatr Dermatol 2010;27:437-45.

Gelmetti C, Frasin A, Restano L. Innovative therapeutics in pediatric dermatology. Dermatol Clin 2010;28:619-29.

SYSTEMIC STEROIDS

- Rapidly progressive generalized vitiligo in older children and adolescents may be treated with a short course of systemic steroid

Palit A, Inamdar AC. Childhood vitiligo , symposium vitiligo. Ind J Dermatol Venerol Leprol 2012; 78: 30-41

- Better way to avert the side effects is administering oral betamethasone as a single morning dose (0.1 mg/kg body weight) on two consecutive days in a week (oral mini-pulse therapy) for 12 weeks, and thereafter reducing the dosage by 1 mg/month for the next 3 months

Rath N, Kar HK, Sabhnani S. An open labeled, comparative clinical study on efficacy and tolerability of oral minipulse of steroid (OMP) alone, OMP with PUVA and broad / narrow band UVB phototherapy in progressive vitiligo. Indian J Dermatol Venereol Leprol 2008;74:357-60.

- Different pulse therapies with systemic steroids have been devised to minimize these side effects, of which the oral minipulse (OMP) therapy has been used particularly in vitiligo

Kanwar AJ, Dhar S, Dawn G. Oral minipulse therapy in vitiligo. Dermatology. 1995;190:251–2.

Pasricha JS, Khaitan BK. Oral minipulse therapy with betamethasone in vitiligo patients having extensive or fast spreading disease. Int J Dermatol. 1993;31:753–77.

Radakovic-Fijan S, Furnsinn-Friedl AM, Honigsmann H, Tanew A. Oral dexamethasone pulse treatment of vitiligo. J Am Acad Dermatol. 2001;44:814–7.

- Pasricha *et al* have studied the effect of oral mini-pulse therapy (OMP) in children and adults with extensive or fast-spreading vitiligo, and have observed a 26-50% response in 25%, 51-75% response in 7.5% and >75% response in 15% of patients

Pasricha JS, Khaitan BK. Oral mini-pulse therapy with betamethasone in vitiligo patients having extensive or fast-spreading disease. Int J Dermatol 1993;32:753-7.

- Majid *et al* have used methylprednisolone OMP in combination with topical fluticasone for 6 months in 400 children with progressive vitiligo. Complete halt of progression was noted in >90% of the children after initiation of therapy, and >65% of children had well to excellent repigmentation at the end of the study period
- Two-thirds of patients the repigmentation was graded as good to excellent
- In 41 patients (13.2%) the lesions resolved completely within the six months study period (100% repigmentation) and they did not require any further therapy for their vitiligo

Majid I, Masood Q, Hassan I, Khan D, Chisti M. Childhood vitiligo: Response to methylprednisolone oral minipulse therapy and topical fluticasone combination. Indian J Dermatol 2009;54:124-7.

- Rath *et al* have compared the efficacy of combined phototherapy (PUVA, NB-UVB and broad band UVB) and OMP with OMP alone in 86 patients (aged 10-50 years) with progressive vitiligo. Combination therapy was found to be superior to OMP alone, the most effective being with NB-UVB, followed by PUVA and broad band UVB

Rath N, Kar HK, Sabhnani S. An open labeled, comparative clinical study on efficacy and tolerability of oral minipulse of steroid (OMP) alone, OMP with PUVA and broad / narrow band UVB phototherapy in progressive vitiligo. Indian J Dermatol Venereol Leprol 2008;74:357-60

- Systemic photochemotherapy with psoralen-ultraviolet A (PUVA) is contraindicated in small children and can be used only beyond 12 years of age

Tamesis ME, Morelli JG. Vitiligo treatment in childhood: A state of the art review. Pediatr Dermatol 2010;27:437-45

- NB-UVB phototherapy in children - there may be an increased chance of developing long-term skin cancers because of the higher number of expected years of life in children
- In resource-poor countries, the facility of phototherapy is available mostly in tertiary health care centers. Hence, it may not be easily accessible to families from remote localities. Moreover, multiple hospital visits confer loss of school days of children and working days of parents, reducing compliance to treatment

Dogra S, De D. Update on photo (chemo) therapy in childhood dermatoses. In: Inamadar AC, Palit A, editors. Advances in pediatric dermatology. 1st ed. New Delhi: Jaypee Publishers; 2011. p. 146-60.

- Surgical procedures are not performed in very young children- segmental or stable focal lesions in younger children extends proportionate to their body growth
- Success of many surgical procedures depends upon the post-operative immobility of the operated part, which becomes difficult to maintain in young children
- Restrictive factors for surgical procedures are inability to treat larger area and the risk of Koebnerization of the donor site.

Palit A, Inamdar AC. Childhood vitiligo , symposium vitiligo. Ind J Dermatol Venerol Leprol 2012; 78: 30-41

- Cultured melanocyte transplantation is a relatively tedious technique requiring specialized set up, trained staff and a preparation time of 6-8 weeks
- Cost may be a restrictive factor for poor families in availing this treatment modality

Gupta S, Kumar B. Epidermal grafting in vitiligo: Influence of age, site of lesion and type of disease on outcome. J Am Acad Dermatol 2003;49:99-104.

- Non-cultured autologous epidermal transplantation
- Cosmetic camouflage -? children

THANK YOU

FATHER MULLER MEDICAL COLLEGE, MANGALORE

DEPARTMENT OF FORENSIC MEDICINE & TOXICOLOGY

From,

03-5-2016

Dr. Nagesh K.R

Professor & Head,

Dept. of Forensic Medicine, FMMC, Mangalore.

To,

The Dean,

FMMC, Mangalore.

Respected Sir,

Subject: Report on Case Based Learning Session –regarding.

Referring to the above subject, a session on Case Based Learning (CBL) was conducted for MBBS 3rd term students on 23-4-2016 between 3.30 pm to 4.30 pm on Medical Jurisprudence topic. The students were divided into 3 groups of 6 students each. Dr. Hareesh Gouda conducted the session as facilitator and Dr. Nagesh K.R, Dr. Varun Pai were present during the session. Students actively participated in the session. Feedback from the students was taken about the session. Students were informed that such sessions will be conducted in future for some of the suitable topics.

The case scenario and feedback checklist has been enclosed.

This is for your kind information.

Yours sincerely,

(Dr. Nagesh K.R)

CC to: The Convener, MEU, FMMC, Mangalore.

Encl.:

1. Case scenario.
2. Feedback checklist.

Case Scenario	Learning Objectives
A 10 year old boy, George, from Bangalore studying in a residential school at Mangalore, falls from a height of about 7 feet while playing in the school. He develops severe pain and swelling of middle part of right leg. His class teacher Ms. Susan informs George's father about the incident and takes him to Fr Muller Medical College Hospital, Mangalore for the treatment. In casualty, on duty orthopaedician Dr. Simon examines George after obtaining consent from Ms. Susan. The X ray of right leg showed comminuted fracture (multiple fractures) of right tibia. Dr. Simon informs Ms. Susan that immediate surgery is required to treat the fracture. After obtaining the written informed consent from the class teacher Dr. Simon operates on George and corrects the fracture by putting intra-medullary nail.. *(Note to students: Intra-medullary nail fixation of fractured fragments is the accepted method of treatment for comminuted fracture of shaft of long bone)* Question 1. Can Ms. Susan give consent for examination and surgery of George ? Question 2. Can George give consent for examination and surgery ? Question 3. Why consent is required for medical examination ? Question 4. What type of consent has to be taken for surgery ? Question 5. Can RMP treat a patient in an emergency without consent ?	By the end of this discussion, the student should know, 1. Loco parentis. 2. Consent in medical examination. 3. Consent and types. 4. Emergency doctrine.
Next day, George's father reaches the hospital and disputes with Dr. Simon for doing surgery without his consent. He warns Dr. Simon of filing negligence suit against him. He also warns the class teacher Ms. Susan to face legal consequences for giving consent for surgery. And, he gets George discharged against Dr. Simon's advice. However, Dr. Simon tells George's father that complete bed rest is required for George for 3 weeks and to take the medicines prescribed by him. George's father takes his son to Bangalore and consults Dr. Prakash. Dr. Prakash is a General Practitioner with MBBS degree and has his clinic near to George's house.	By the end of this discussion, the studer should know, 1. Duties of registered Medic Practitioner. 1. Legal responsibility of RMP cases of discharge again medical advice. 2. Medical negligence. 3. Consumer Protection Act.

Dr. Prakash after examining George tells his father that intra-medullary fixation of fracture was not at all required in this case and he would have treated only by cast. Also makes comment that Dr. Simon does not know anything about treating such type of fracture and he operates such cases only to charge more fees. He also tells George's father that such intramedulary nails cause many complications in future. Dr. Prakash gives suggestion of filing medical negligence case against Dr. Simon for unnecessary medical expenditure. Question 1. Can Dr. Simon discharge George on his father's request ? Question 2. What are the duties of a RMP ? Question 3. Is Dr. Simon negligent in treating George ? Question 4. In a case of medical negligence, Inwhich court the patient can file the case?	

FEED BACK

Sl No	Criteria	Agree	Neutral	Disagree
1.	Case scenario is appropriate to explain the learning objectives			
2.	CBL created interest in learning			
3.	CBL helped in understanding the topic properly			
4.	CBL brought in more interaction			
5.	CBL provides opportunity to express			
6.	CBL session was conducted in a systematic manner			
7.	Facilitator encouraged the interaction			
8.	CBL can be used along with lectures			
9.	Lecture method would be better to teach the current topic			
10.	Teach other topics as well by CBL			

Feed back on Case Based Learning (CBL) Session conducted on 23-4-16 for MBBS III term students

Topic: Medical Jurisprudence

Sl No	Criteria	Agree	Neutral	Disagree
1.	Case scenario is appropriate to explain the learning objectives	✓		
2.	CBL created interest in learning	✓		
3.	CBL helped in understanding the topic properly	✓		
4.	CBL brought in more interaction	✓		
5.	CBL provides opportunity to express	✓		
6.	CBL session was conducted in a systematic manner	✓		
7.	Facilitator encouraged the interaction	✓		
8.	CBL can be used along with lectures	✓		
9.	Lecture method would be better to teach the current topic			✓
10.	Teach other topics as well by CBL		✓	

Topic: Ventilator Management of COPD patient with unanticipated difficult airway.

Date: 24/01/2018

Presenter: Dr. Shubha. P.

Moderator: Dr. Hilda. S.

69 year old male who is a k/c/o COPD admitted with acute exacerbation of COPD to MICU. Put on NIV. But in view of deterioration on Non invasive ventilation, invasive ventilation was planned. Unanticipated difficult intubation. After 3 attempts, intubated successfully using gum elastic bougie @ 7.5 mm ETT. Connected to ventilator on SIMV mode. Responded well and ABG and clinical condition improved following intubation & mechanical ventilation. Weaning started 24 hours later and successfully weaned on day 3. patient's condition improved over the course of MICU stay and was shifted out of the ICU after 5 days.

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Topic :- DELAYED RECOVERY

Moderator :- Dr. SUDHIR HEGDE.

Speaker :- Dr VEERESH.

Date :- 26/7/17.

45yr female S/P thoracotomy 15yr back came for Lap hysterectomy [illegible]. pt was in steep head down position. Intra op vitals were stable. adequate relaxants given intraop. ~~post op~~ operation ended in 2hr. Last dose of relaxant was 15 min. waited for another 20 min. But [illegible] [illegible]. [illegible] spontaneous efforts. [illegible] pt reversed of NM blockade. pt was still drowsy. Pt extubated in [illegible] BP ↑. pt developed resp distress. pt was reintubated & given [illegible] Lasix, mannitol & connected to ventilator for [illegible] pt recovered. pt weaned off ventilator on next day (POD1).

Sl. No	Name	Designation	Signature
1	Dr KISHAN SHETTY	Assoc Prof	[signature]
2.	Dr Sudhir hegde	Prof.	[signature]
3.	Dr. Rithesh Joseph	SR.	[signature]
4.	DR. KARL SA RIBEIRO	ASSIST PROF	[signature]
5	Dr GEORGE JACOB M	SR	[signature]
6	Dr Bharath [illegible]	AP	[signature]
7.	DR. SHAILAJA . S	Assoc. Prof	Shailaja . S
8 -	DR. MAHABALA	Professor	[signature]
9.	DR. PRIYANKA	III YR RESIDENT	Priyanka
10.	Dr Pragna	Pg.	[signature]
11	Dr Vishal	PG	[signature]
12	Dr. Parmanand Reddy	PG	[signature]
13.	Dr. HEZIL DEEPIKA SALDANHA	PG	[signature]
14	Dr. Preethi D'souza	PG	[signature]
15.	Dr. Aruna Salian	PG	[signature]
16	Dr Veeresh	PG	[signature]

Members on leave

1 Dr. Vishnu K

2 Dr. Gurumurthy

3 Dr. Rashmi Menon

4. Dr. Suni Abraham

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Topic :- Primi° with 31 weeks with Secondaries with breast, Scalp, with breathlessness posted for Emg Caesarian Section

Date 14·09·2017

Presenter : Dr. Roshni°

Moderator : Dr. Harshavardhan

Summary: A 26 yr old female patient with 31 weeks for POG came with chief complaints of lump in the left breast and both axillae. Incidentally on 30/10/17 was found to have a lesion in the scalp and on FNAC was diagnosed to have infiltrating Ca Lt breast ē metastasis. Pt general condition deteriorated after three days and was shifted to HDU due to Tachypnoea, tachycardia and bl pitting and suspected pulmonary embolism.
In view of deteriorating patient condition, and to save the baby emergency LSCS was done & spinal anaesthesia under high risk and death on table condition - Intraop events → uneventful. Pt was shifted to post op and 10 WD was transfused in view of anaemia. Post op Day 3. Pt deteriorated further and was declared dead on 8/11/17 due to LRM. Cause of death → Pulmonary Embolism

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Topic : Unexplained persistent hypotension following spinal Anaesthesia

Date :- 14.09.2017

Presenter : Dr. Sony

Moderator :- Dr. Harshavardhan / Dr. Antony

A 47 Y/F came for wound debridement for Non healing ulcer of (L) ankle. ISAB Intraops under aseptic condition 26 G Quincke Babcock was inserted into L_3-L_4 space with 2.5cc of 0.5% Bupivacaine (hy) after confirming free flow of CSF in (L) lateral position. pt was given for 3 mins Later there was sudden Bradycardia with (HR-35/min) given Atropine 0.6mg IV was given. pt suddenly developed tachycardia (HR-150) and started complain of chest pain. given Ranitidine 50mg IV was given. along with given Ondansetron 5mg IV. Now HR=90 B.P-110/80 SpO2= 97%. But still complain of chest pain. after 10 minutes Surgery was over and shifted to post op Vitals HR-120 BP-90/60 SpO2=100 pt was started on tenofovir. after 4 days vitals stable and shifted to ward

Topic :- Sudden deterioration in the post operative period.

Date : 10/11/17

Presentor : Dr Veena

Moderator : Dr Rishi [illegible]

84 yr old female, bed ridden c # IT (L) femur. posted for PFN. Pt diagnosed to have mild AS c AR (c Calcific AV). EF 57%. Pt was taken up c risk by Cardiac opinion + planned for Spinal Anaesthesia. Intra op period was uneventful. Bupivacaine adjuvant was added to spinal. Sx ended in 2hr. Shifted to post op c stable vitals.

At 4:45 pm pt had sudden (coffee ground) vomiting & aspirated + collapsed. Pt was resuscitated. Once ROSC, pt was connected to ventilator & on inotropes. ABG showed acidosis. Corrections were given for the same. Pt had ? myoclonus at 7:30 pm. Next morning again collapsed pt was declared dead at 8:45 am on 8/11/17.

Cause of death :- ? Aspiration → Asphyxial death
- ? Pulm embolism

Case Based Learning

A 35 year old female Sandhya was a banker by occupation. She complained of unsteadiness while walking. She also felt weak to carry out her daily routine activities. She was a strict vegan.

1. What are the causes of unsteadiness?
2. At the end of history, what condition do you think Sandhya is suffering from?

On examination, she was pale, rhomberg's sign was positive. She had bilateral extensor planter response with absent ankle jerk . Her laboratory investigations revealed anemia, macrocytic RBCs with hypersegmented neutrophils. A specific blood test was asked based on these reports. She was started on injections for her clinical condition. On follow up, she felt better and her symptoms had significantly improved.

1. What is your diagnosis?
2. What are the common causes for this condition?
3. Mention other causes of macrocytic RBCs.
4. How will you treat her?
5. How will you assess the response to treatment?

OBJECTIVES:

At the end of the session, the student should be able to:

1. Enumeate the causes for Megaloblastic anemia.
2. Discuss the clinical features of Megaloblastic anemia
3. List the differential diagnosis
4. Discuss the management

CASE BASED LEARNING

A 35 year old gentleman Mr. Raju, hailing from Bantwal, working in a poultry farm presents to you with complaints of fever of 3 weeks duration. Fever is associated with generalized myalgia, loss of appetite and weight loss. He also complains of left hip joint pain due to which he has difficulty in walking. On taking a detailed history, patient reports of having fever with myalgia one and half month ago , which lasted for 2 weeks and subsided. Following this he was afebrile for 1 week after which he again started developing fever spikes.

<u>On examination</u> :

PR – 100/min , BP – 120/80 mm Hg
RR -22/min Temparature – 100^{o} F
On system examination , liver palpable 2 cm below right costal margin , non tender, firm in consistency .

Investigation :

HB - 12 gm%
TC - 8,800/ cu.mm
Neutrophils - 45%
Lymphocytes - 50%
Eosinophils - 4%
Monocytes - 1%
Basophils - 0%
ESR - 30mm/hr

Liver function test

T. bilirubin – 1mg/dl, unconjugated bilirubin – 0.8 mg/ dl conjugated bilirubin - 0.2 mg/dl, AST -42 IU/l , ALT – 44 IU/l , ALP – 100 IU /l , S. Total protein - 7.2 gm/dl , S. Albumin – 4 gm/dl, S. Globulin - 3.2gm dl

1. What are the deferential diagnosis to be considered
2. What are the investigations to be performed to confirm the diagnosis
3. What are the other clinical Manifestations of this condition and how do you evaluate
4. Drugs used in treatment of this clinical condition

Case Based Learning

A 23 year old Mohan, a hostel resident presented with 5 days history of fever, abdominal pain and vomiting. On examination mild icterus + tender hepatomegaly +

A) What possibilities would you like to consider?
B) Mention causes of tender hepatomegaly.

His lab report revealed total count 12,000/mm^3 normal renal parameters, lever function tests showed TB :3.5mg%, DB :2 mg%, TP:6.5 g/dl, Serum albumin: 4g/dl, globulin 2.5g/dl, AST :2500IU, ALT :3000 IU

PT normal.

a) What do you conclude based on the above reports?
b) What further tests would you like to ask for?
c) Mention other causes of marked elevation of liver enzymes(>500 IU)
d) What are the complications of the condition?
e) How will you treat this condition?

Objectives :

At the end of the session the student should be able to

1. Enumerate the causes for acute hepatitis
2. List the clinical features of acute hepatitis
3. Enumerate the relevant investigations
4. Discuss the treatment for acute hepatitis
5. List the preventive measures for acute hepatitis

The above topic of acute hepatitis was taught to 8th term students on 13.04.2016 as per the above protocol. At the end of the session the students found it better than the traditional didactic lectures. I planned to implement case based learning for my theory classes in future.

A 18 year old girl presented with paraplegia, ataxia, fever and fatigue which had started a few months back and which was getting worse since the past 2 weeks. Examination of the sensory system showed loss of pain, touch, temperature, vibration and joint position sense. The limbs were found to be spastic. Peripheral smear showed the presence of macrocytic anemia with hyper segmented neutrophils. Work up revealed that the patient had features suggestive of tuberculosis involving the ileocecal junction.

1)what is your diagnosis for the patient and substantiate?
2)Enumerate various causes that can cause the nutritional deficiency in question?
3)What is the schillings test and describe in detail?
4) How do you treat the condition?
5)what are the differential diagnoses for the same?

A forty year old female presented with history of severe pain in the left forearm and ankle following a trivial injury. The patient had a swelling in the left leg and also complained that she had pain in the same leg and on movement of the ankle since the past 2 years. Radiography showed the presence of pathological fractures in the radius and ulna and an expansile lytic lesion in the region of the ankle. X ray skull was suggestive of a "pepper pot" appearance. Radiograph of the hands showed osteopenia with expansile lytic lesions in the metacarpals. USG neck showed a 1.5x1.5 hypoechoic lesion on the posterior aspect of the thyroid gland. Labs revealed Calcium-12mg/dl, Alkaline phosphatase 727u/l, Serum parathyroid hormone -1265pg/ml.

1) What is your diagnosis for the patient?
2) What are the differential diagnoses for the patient?
3) How can we best treat the patient?
4) Describe the complications of hypocalcemia and hypercalcemia?
5) Describe calcium metabolism in detail?
6) Describe the treatment modalities available for the treatment of osteoporosis?

A 20-year-old female patient was admitted to the hospital for persistent vomiting, diarrhea, and nausea. She had Crohns disease since age 9 and was on chronic total parenteral nutrition (TPN). Two months before admission, multivitamin infusion (MVI) was discontinued in the TPN because of the shortage of its supply. An oral multivitamin tablet was substituted instead. Patient presented with altered sensorium,had abnormal eye movements and an unsteady gait. Fat stores and muscle tissue were decreased. Her respiratory rate was 24/min. Her blood pressure, while she was standing, was 105/65 mmHg. Liver tests were normal. The serum lactate concentration was substantially increased. Magnetic resonance imaging (MRI) showed several areas of abnormally high signal on T2- weighted images in the brainstem, thalamus, and mamillary bodies.

1) what is your possible diagnosis for the patient and substantiate?
2) Why are the fat stores and muscle tissue decreased in this patient?
3) Which metabolic enzyme is affected with regards to the vitamin in question?How and why?
4) What are the cardiovascular manifestations?
5) How do you treat the condition?