MIMSR MEDICAL COLLEGE, LATUR

DEPARTMENT OF BIOCHEMISTRY

NOTICE

All 1st MBBS Students are informed that ECE (Early Clinical Exposer) will be conducted on dated 17/01/2020 between 11Am to 12 noon. Attendance for the same will be observed seriously.

Topic :- Diabetes Mellitus

Speaker:- Dr. Gajanan Gondhali (Asso. prof) Medicine

Prof & Head

Dept. of Biochemistry

MIMSR Medical College, Latur


Temparature : 23.19°C
Accuracy : 62.222
time : 2020-01-17 11:16:09
longitude : 76.5796639
latitude : 18.4298912
Vishwanathpuram,Latur,Maharas


Temparature : 23.19°C
Accuracy : 42.025
time : 2020-01-17 11:14:38
longitude : 76.5796976
latitude : 18.4298594
Vishwanathpuram,Latur,Maharas


Temparature : 23.19°C
Accuracy : 60.0
time : 2020-01-17 11:18:11
longitude : 76.5796976
latitude : 18.4298594
Vishwanathpuram,Latur,Maharas


Temparature : 23.19°C
Accuracy : 60.0
Time : 2020-01-17 11:16:37
longitude : 76.5796976
latitude : 18.4298594
Vishwanathpuram,Latur,Maharas

MIMSR MEDICAL COLLEGE, LATUR
DEPARTMENT OF BIOCHEMISTRY

Date:- 27/01/2020

Activity :- QUIZ Competition

Date:- 03 /02/2020, **Time** :- 11.00 am to 01.00 pm

Venue:- Dept. of Anatomy Lecture Hall.

Participating Candidate Roll No.:- 01, 19, 33, 41, 55, 68, 81, 97, 119, 121, 135, 141.

Prof & Head
Dept. of Biochemistry
Professor and Head
Department of Biochemistry
M.I.M.S.R. Medical College,
LATUR - 413 512.

Dean
MIMSR M.C. Latur
Dean
M.I.M.S.R. Medical College,
& Y.C.R. HOSPITAL,
LATUR - 413 531.


Biochemistry Quiz
Temparature : 28.47°C
Accuracy : 110.0
time : 2020-02-03 12:34:19
longitude : 76.5797569
latitude : 18.4294805
Vishwanathpuram,Latur,Maharash


Temparature : 30.86°C
Accuracy : 33.185
time : 2020-02-03 13:04:22
longitude : 76.5795241
latitude : 18.4300684
Vishwanathpuram,Latur,Maharash
79th
Birthday Celebration of
Hon. Prof. Dr. Vishwanath D. Karad Sir
Quiz
for
MBBS
Thank you All!!!!!!!


Henry
Gray
28.47°C
Accuracy : 622.93
time : 2020-02-03 11:36:04
longitude : 76.5797569
latitude : 18.4294805


Andreas
Vesalius
Temperature : 28.47° C
Accuracy : 545.709
time : 2020-02-03 11:38:51
longitude : 76.5797569
latitude : 18.4294805
Hatte Nagar,Latur,Maharashtra


Round I
War Of Nerves
Quiz
Temparature : 28.47°C
Accuracy : 98.4
time : 2020-02-03 12:08:20
longitude : 76.5797569
latitude : 18.4294805
Vishwanathpuram,Latur,Maharash


Temparature : 30.86°C
Accuracy : 158.211
time : 2020-02-03 13:07:51
longitude : 76.5798345
latitude : 18.429855
Vishwanathpuram,Latur,Maharas
Birthday Celebra
Hon. Prof. Dr. Vishwanath
Quiz
for
1st MBB


Temparature : 30.86°C
Accuracy : 102.607
time : 2020-02-03 13:09:49
longitude : 76.5798667
latitude : 18.4297584
Vishwanathpuram,Latur,Maharash
Hon. Prof.

MIMSR MEDICAL COLLEGE , LATUR.
DEPARTMENT OF PATHOLOGY,

No.MIMSR/Patho/CPC/1731/ /2017 **Date :- 24/01/2017**

It is to inform you that, C.P.C. meeting will be held on 31/01/2017 at 3.00 pm in Pathology lecturer hall, copy of case history (Clinical Protocol is attached herewith.

Kindly, circulate this protocol to the all Teacher's, P.G. Student's Interns & Students of your department.

Thanking you,

In-charge
C.P.C. Activity
Dr. A.S.Acharya

H.O.D.
Dept. of Pathology
The Head
Department of Pathology
MIMSR Medical College, LATUR

Copy to :-

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MIMSR MEDICAL COLLEGE, LATUR.

Department of pathology

C.P. C.

CIRCULAR

Date :- 24/01/2017

Sr. No.	Name of Department	Receivers Sign
1	Principal	
2	Anatomy	
3	Physiology	
4	Biochemistry	
5	Microbiology	
6	Pharmacology	
7	FMT	
8	PSM	
9	ENT	
10	Ophthalmology	
11	OBGY	
12	Pediatrics	
13	Orthopedic	
14	Surgery	
15	Medicine	
16	Skin & VD	
17	Radiology	
18	PSY	
19	Anesthesia	

MIMSR MEDICAL COLLEGE, LATUR.
DEPARTMENT OF PATHOLOGY.

C.P.C. PRESENTATION.

Venue :- Pathology Lecturer Hall, Date 31/01/2017, Tuesday, at 3.00 pm.

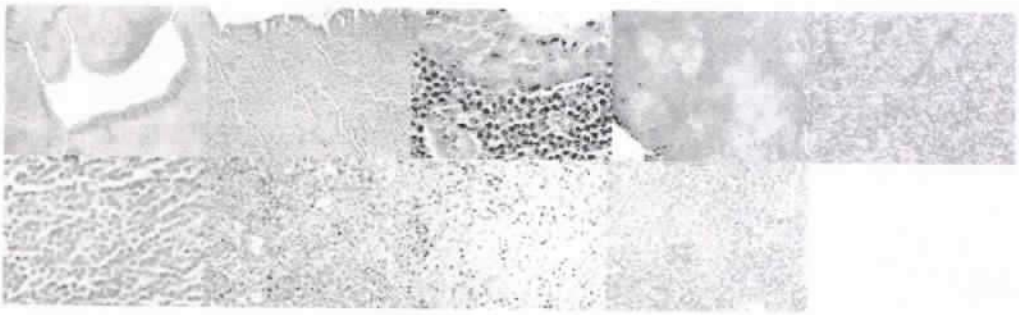
PATIENT HISTORY:

This is a 79-year old male with a cervical, axillary, inguinal, and abdominal lymphadenopathy whose case was sent for consultation. The patient was status-post excision of a right neck mass. This case was signed out as an "atypical lymphoid proliferation suspicious for lymphoma involving a portion of the parotid gland."

GROSS DESCRIPTION:

From the referring institution was of two pink-tan oval portions of soft tissue, 2.0 X 1.2 X 1.0 cm and 3.5 X 1.8 X 1.6 cm. The smaller specimen was serially sectioned revealing solid pink-tan tissue. The larger specimen was serially sectioned revealing a central abscess filled with yellow-green exudate.

MICROSCOPIC DESCRIPTION



What is your clinical diagnosis ? :-

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CIRCULAR

Date :- 20/09/2017
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Sr. No.	Name of Department
1	Principal
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MIMSR MEDICAL COLLEGE, LATUR

DEPARTMENT OF PATHOLOGY

C.P.C PRESENTATION

PATIENT HISTORY

The patient was a 31 year-old female that moved to Pittsburgh from California one year back. She has a history of episodic pain involving arms, legs and abdomen (pain usually starts at night or in the morning when she wakes up). In July of 2008 she developed pain crisis at child delivery. After her move to Pittsburgh, in May 2009, she was admitted to a hospital with nausea and vomiting. A CT scan was consistent with pneumonia. She had a favourable course after being treated wih Azithromycin. Three months later, in August 2009 she presented to the same hospital complaining of fever and productive cough (green sputum). Once again, her diagnosis was pneumonia and she improved after iv Moxifloxacin. Her clinical scenario prompted an extensive work-up, including a CBC and peripheral blood smear.

PATIENT WORK-UP

CBC

• WBC	14.6 K/cmm	4.8-11.0
• RBC	3.9 M/cmm	4.2-5.4
• Hgb	10.7 g/dL	12-16
• MCV	82.2 fl	81-99
• PLT	258 K/cmm	130-400
• Anisocytosis	1+	0-4+
• Target cells	1+	0-4+

Peripheral blood smear

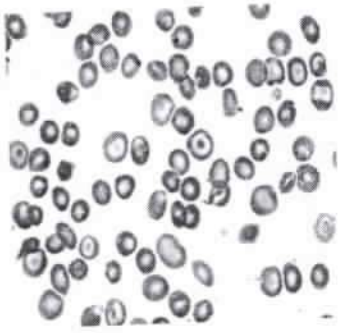

Figure 1. Peripheral blood reveals a large number of target cells and folded forms

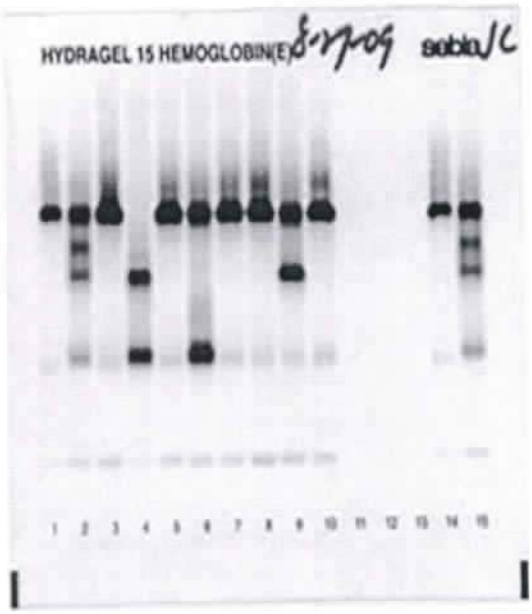

Figure 3: Alkaline hemoglobin electrophoresis

1. - Helena control hemoglobin A-A2
2. - Sebia control hemoglobin A, F, S, C
3. - normal
4. - our patient
5. - normal
6. - hemoglobin C trait (Hb AC)
7. - normal
8. - normal
9. - hemoglobin S trait (Hb AS)
10. - normal

Hemoglobin concentrations

Test name	Result	Reference range
Hgb A	6.9 %	96.5- 100.0
Hgb F	canc	0.0-2.0
Hgb C	39.8 %	
Hgb S	53.3%	
Hgb A2	canc	0.0-3.5
SC preparation	POSITIVE	NEG

MIMSR MEDICAL COLLEGE , LATUR.
DEPARTMENT OF PATHOLOGY,

No.MIMSR/Patho/CPC/1731/ /2017 **Date :- 20/09/2017**

It is to inform you that, C.P.C. meeting will be held on 26/09/2017 at 3.00 pm in Pathology lecturer hall, copy of case history (Clinical Protocol is attached herewith.

Kindly, circulate this protocol to the all Teacher's, P.G. Student's Interns & Students of your department.

Thanking you.

In-charge
C.P.C. Activity
Dr. A.S.Acharya

H.O.D.
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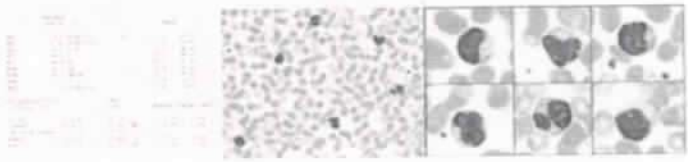
MIMSR MEDICAL COLLEGE, LATUR.
DEPARTMENT OF PATHOLOGY.

C.P.C. PRESENTATION.

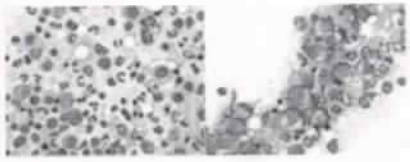
Venue :- Pathology Lecturer Hall, Date 01/08/2017, Tuesday, at 3.00 pm.

PATIENT HISTORY

An 80-year old male with a past medical history of dyslipidemia, hypertension, gout and prostate cancer was evaluated for peripheral blood lymphocytosis.

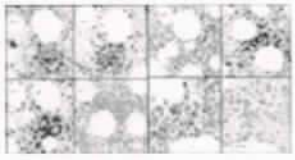


The complete hemogram (Fig. 1) and peripheral blood smear (Fig. 2) demonstrated absolute lymphocytosis, including many large granular lymphocytes (Fig. 3), some intermediate in size with abnormal nuclear contours. Based on a 100-cell lymphocyte count, 66% of all lymphocytes were large granular lymphocytes. Absolute large granular lymphocyte count = 6,461 per microliter.



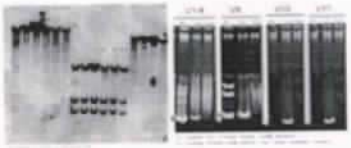
A bone marrow aspirate showed hypercellular marrow with a normal differential (Figs. 4 and 5) on biopsy and touch imprints (Fig. 6) showed an increased number of large granular lymphocytes.

Bone marrow biopsy (Fig. 7) and particle preparation (Fig. 8) showed 30-40% cellularity with a diffuse increase in small lymphocytes that focally form small, ill-defined aggregates, with a normal M:E ratio, complete maturation in all lineages and adequate megakaryocytes



Immunohistochemical stains performed on bone marrow biopsy showed an increase in CD3, CD2, CD5, CD7 and CD8 positive T cells forming ill defined aggregates and also a few scattered CD56, CD57, TIA1 & Granzyme B positive cells (Figs. 9 and 10)

Flow cytometry studies (Fig. 11) performed on the bone marrow demonstrated 46% bright CD45+ (lymphocyte) events and 3% CD14+ monocytes. T and NK cell marker analysis (Fig. 12) shows a prominent population (26-30% of total) of NK-like T-cells with CD2+, CD3+, CD5+, CD7 (dim+), CD56+, CD16/57+, CD4-, CD8+, T-cell receptor alpha-beta+ immunophenotype (highlighted in blue on Fig.12).



Cytogenetic studies showed a normal male karyotype 46,XY [20], molecular studies showed that Southern Blot (Fig. 13, positive bands [white arrow]) and PCR (Fig. 14, positive bands in V1-8 & V9 [rose arrows]) were positive for clonal β chain and γ chain T-cell receptor gene rearrangements, respectively.

MIMSR MEDICAL COLLEGE, LATUR.

Department of pathology

C.P. C.

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Date :- 25/07/2017

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MIMSR MEDICAL COLLEGE , LATUR.
DEPARTMENT OF PATHOLOGY,

No.MIMSR/Patho/CPC/1731/ /2017 **Date :- 25/07/2017**

It is to inform you that, C.P.C. meeting will be held on 01/08/2017 at 3.00 pm in Pathology lecturer hall, copy of case history (Clinical Protocol is attached herewith.

Kindly, circulate this protocol to the all Teacher's, P.G. Student's Interns & Students of your department.

Thanking you,

In-charge
C.P.C. Activity
Dr. A.S.Acharya

H.O.D.
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MIMSR MEDICAL COLLEGE, LATUR.
DEPARTMENT OF PATHOLOGY.

C.P.C. PRESENTATION.

Venue :- Pathology Lecturer Hall, Date 20/06/2017, Tuesday, at 3.00 pm.

PATIENT HISTORY

The patient is a 55-year-old man who presented to the ED with gradual worsening of weakness. The patient reports that he had been fatigued for one month, having to rest constantly after work, and feeling exhausted with minimal exertion. He also reported feeling lightheaded with rising, as well as having epigastric abdominal pain, which was not associated with food. Four days prior to the presentation, the patient began to have black, tarry stools. He was evaluated in his primary care physician's office the day prior, and was found to have hemoglobin of 6.5 g/dL. His prior baseline from two weeks ago was 15.3 g/dL. He was subsequently referred to the ED.

The patient has a history of pancolonic ulcerative colitis diagnosed in 1979 and now in remission. He has not been on medication for 17 years, but he did receive treatment with prednisone around time of diagnosis, then sulfasalazine. He had undergone recent surveillance EGD and colonoscopy a month ago, which was unchanged from previous studied. He also has gastroparesis, not symptomatic for several years. He is on ASA 81 mg. He takes a PPI for GERD which is well-controlled.

In the ED, his vital signs were: pulse: 121, blood pressure: 127/85, respiratory rate: 20, and oxygen saturation: 99% on room air. Physical examination was remarkable for generalized pallor, mild tachycardia, and diastasis of rectus (also known as abdominal separation). Nasogastric lavage was negative, but the fetal occult blood test was positive. Pertaining labs are listed below

	ED Labs	Reference Range
Hematology:		
WBC	9.4	3.8-10.8 THOUS MCL
RBC	3.07	4.20-5.80 MILL MCL
Hgb	6.9	13.2-17.1 G DL
HCT	22.4	38.5-50.0 %
MCV	73.1	80.0-100.0 FL
MCH	22.6	27.0-33.0 PG
MCHC	30.9	32.0-36.0 G DL
RDW	21.1	11.0-15.0 %
Chemistry:		
Iron	<10	45-182 ug dL
Total Iron Binding Capacity	298	250-420 ug dL
Ferritin	63	10-282 ng mL

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1	Principal	
2	Anatomy	
3	Physiology	
4	Biochemistry	Ganesh.
5	Microbiology	
6	Pharmacology	
7	FMT	
8	PSM	
9	ENT	
10	Ophthalmology	
11	OBGY	Jadhavar Manisha Manisha
12	Pediatrics	sabale
13	Orthopedic	Aghav
14	Surgery	
15	Medicine	
16	Skin & VD	
17	Radiology	
18	PSY	
19	Anesthesia	

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DEPARTMENT OF PATHOLOGY,

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Thanking you,

In-charge
C.P.C. Activity
Dr. A.S.Acharya

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The Head
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MIMSR Medical College, LATUR

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MIMSR Medical College, LATUR

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Department of pathology

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Date :- 14/04/2017

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1	Principal	
2	Anatomy	
3	Physiology	
4	Biochemistry	
5	Microbiology	
6	Pharmacology	
7	FMT	
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The Head
Department of Pathology
MIMSR Medical College

MIMSR MEDICAL COLLEGE , LATUR.
DEPARTMENT OF PATHOLOGY,

No.MIMSR/Patho/CPC/1731/ /2017 **Date :- 14/04/2017**

It is to inform you that, C.P.C. meeting will be held on 18/04/2017 at 3.00 pm in Pathology lecturer hall, copy of case history (Clinical Protocol is attached herewith.

Kindly, circulate this protocol to the all Teacher's, P.G. Student's Interns & Students of your department.

Thanking you,

In-charge
C.P.C. Activity
Dr. A.S.Acharya

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MIMSR Medical College, LATUR

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MIMSR MEDICAL COLLEGE, LATUR.
DEPARTMENT OF PATHOLOGY.

C.P.C. PRESENTATION.

Venue :- Pathology Lecturer Hall, Date 18/04/2017, Tuesday, at 3.00 pm.

PATIENT HISTORY

An 82 year old female with a medical history of mitral regurgitation and prolapse and moderate coronary artery disease presented to the emergency department with increased dyspnea on exertion. She underwent coronary artery bypass graft and mitral valve repair surgery. A post-operative CBC demonstrated a mildly elevated white blood cell count, normocytic anemia with occasional ovalocytes and acanthocytes and thrombocytopenia. A peripheral blood smear was sent for pathology review.

LABORATORY

The post-operative complete blood count and differential revealed the following:

Results Assessed	Patient Value	Normal Value
WBC	10.7	(3.8-10.6)x10^9/L
RBC	3.04	(3.73-4.89) x10^9/L
Hgb	9.4	(11.6-14.6) g/dL
Hct	27.4	(34.10-43.3)%
MCV	90.2	(82.6-97.4) fL
MCH	31.0	(27.8-33.4) pg
MCHC	34.3	(32.7-35.5) g/dL
RDW	15.1	(11.8-15.2)%
Mean platelet volume	9.9	(6.8-10.4) fL
Poly	63	(44-77)%
Lymphocytes	20	(13-44)%
Monocytes	11	(4-13)%
Eosinophils	6	(0-6)%
ABS Poly	6.74	(2.24-7.68) fL
ABS Lymphocytes	2.14	(0.80-3.65) pg
ABS Monocytes	1.18	(0.30-0.90) g/dL
ABS Eosinophils	0.64	(0.00-0.40) x10^9/L
Ovalocytes	Present	
Acanthocytes	Present	
Platelets	66	(156-369) x10^9/L

Morphologic review of the peripheral blood smear revealed neutrophils surrounded by platelets as well as prominent phagocytosis of the platelets by neutrophils (see image). This phenomenon was not observed in association with other cells (i.e. red blood cells, eosinophils, monocytes or lymphocytes). Due to the association of the platelets with neutrophils, the automated instrument count was not accurately reflecting the platelet count.

MIMSR MEDICAL COLLEGE, LATUR.

Department of pathology

C.P. C.

CIRCULAR

Date :- 16/02/2018

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4	Biochemistry	
5	Microbiology	
6	Pharmacology	
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16.2.18

In-charge
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No.MIMSR/Patho/CPC/1731/ /2018 **Date :- 16/02/2018**

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Kindly, circulate this protocol to the all Teacher's, P.G. Student's Interns & Students of your department.

Thanking you,

16.2.18

In-charge
C.P.C. Activity
Dr. A.S.Acharya

H.O.D.
Dept. of Pathology

The Head
Department of Pathology
MIMSR Medical College, LATUR

Copy to :-
1 Principal,
2 All dept.

MIMSR MEDICAL COLLEGE, LATUR
DEPARTMENT OF PATHOLOGY
C.P.C PRESENTATION

VENUE:- PATHOLOGY LECTURE HALL, DATE 20/02/2018

CLINICAL HISTORY

The patient is a 22 year old Saudi Arabian male, who has been living in the United States for three years. He initially presented 2 days prior to admission to the emergency department of an outside hospital with cough, night sweats, anorexia, severe fatigue, and an unintentional 90 lbs weight loss over the 6 months prior to presentation. He is a previous smoker (1 pack/day for 8 years), but quit several months ago due to his chronic cough.

He denies any sick contacts, fever, or chills. A purified protein derivative (PPD) skin test recently performed at an outside institution was negative.

The patient had lived in Tennessee when he initially arrived in the US, and has lived in Pennsylvania for the last year. Since his move to Pennsylvania, he has travelled to the Middle East twice to visit family. Most recently, he has not left his apartment in two months due to his incapacitating fatigue.

RADIOLOGY FINDINGS

Chest radiography was significant for bilateral reticular infiltrates and bilateral apical cavitary lesions (Figure 1).

Chest computed tomography demonstrates prominent apical cavitary lesions bilaterally (Figures 2 and 3).

HISTOLOGY FINDINGS

Biopsies taken from the right middle lung lobe demonstrate a necrotizing pneumonia (Figure 4), with foci of epithelioid granulomas (Figures 5 and 6). An acid fast stain performed on the biopsy highlights one acid fast bacillus (Figure 7).

LABORATORY RESULTS

An acid fast stain performed on a smear from bronchoalveolar lavage fluid was positive for bacilli, and culture grew organisms identified as *Mycobacterium tuberculosis* complex by DNA probe. A Quantiferon TB Gold test was positive. Urine antigen testing for Histoplasma was negative.

ADDITIONAL HISTORY

The patient was initially placed on a RIPE (Rifampin, Isoniazid, Ethambutol, Pyrazinamide) regimen for treatment as an inpatient. Upon further consideration, the patient and his family decided that he should return home to the Middle East to complete treatment. To allow for sooner travel, rapid sequence-based molecular drug sensitivity testing was requested at CDC. Prior to discharge, however, the patient's liver enzymes increased dramatically (ALT: 429 IU/L, AST: 534 IU/L), and all anti-tubercular therapy was stopped pending the sensitivity results, which were not yet available. He was discharged to home without any medication, as he was living alone at the time.

FINAL DIAGNOSIS ? :

MIMSR MEDICAL COLLEGE, LATUR.

Department of pathology

C.P. C.

CIRCULAR

Date :- 23/01/2018

Sr. No.	Name of Department	Receivers Sign
1	Principal	Vinod B. Jogdan
2	Anatomy	M. K. Sirsat
3	Physiology	
4	Biochemistry	Pundkar G. D. Ganesh
5	Microbiology	M. D. Kamble
6	Pharmacology	
7	FMT	
8	PSM	R. B. Karad
9	ENT	Charge Bio
10	Ophthalmology	R. A. Shinde
11	OBGY	Manisha
12	Pediatrics	
13	Orthopedic	
14	Surgery	
15	Medicine	
16	Skin & VD	D. D. Suvarge
17	Radiology	V. B. Kaul
18	PSY	
19	Anesthesia	

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DEPARTMENT OF PATHOLOGY,

No.MIMSR/Patho/CPC/1731/ /2018 Date :- 23/01/2018

It is to inform you that, C.P.C. meeting will be held on 30/01/2018 at 3.00 pm in Pathology lecturer hall, copy of case history (Clinical Protocol is attached herewith.

Kindly, circulate this protocol to the all Teacher's, P.G. Student's Interns & Students of your department.

Thanking you,

In-charge
C.P.C. Activity
Dr. A.S.Acharya

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No.MIMSR/Patho/CPC/1731/ /2018 Date :- 23/01/2018

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MIMSR MEDICAL COLLEGE, LATUR.
DEPARTMENT OF PATHOLOGY.

C.P.C. PRESENTATION.

Venue :- Pathology Lecturer Hall, Date 30/01/2018, Tuesday, at 3.00 pm.

PATIENT HISTORY:

The patient is a 32 year old male who presented to the emergency department complaining of progressive fatigue and shortness of breath on exertion over a two to three month period. He denied any infection, bleeding or bruising. He reported consumption of one case of beer per day for ten years, but in the last four years had reduced his intake to one six-pack per day. Past medical history is significant for multiple gunshot wounds to the abdomen in the late 90s requiring resection of small bowel and colon, including the terminal ileum and cecum. This was followed by repair of a large ventral hernia in 2001. No outpatient medications and no known drug allergies. Physical exam showed mild scleral icterus, lungs clear to auscultation, heart regular rate and rhythm without murmurs, rubs, or gallops, abdomen with midline scar, liver palpable 2 cm below the left costal margin, and spleen not enlarged.

A bone marrow specimen was sent to pathology accompanied by peripheral blood showing the following:

Peripheral Blood (Complete Blood Cell Count/Differential):

	Patient Value	Normal Range (Male)
WBC	5.2 x 10^9/L	3.8 - 10.6
RBC	2.61 x 10^{12}/L	4.13 - 5.57
Hemoglobin	9.1 g/dL	12.9 -16.9
Hematocrit	25.7%	38.0 - 48.8
MCV	98.2 fL	82.6 - 97.4
MCH	34.8 pg	27.8 - 33.4
MCHC	35.5 gm/dL	32.7 - 35.5
RDW	14.2%	11.8 - 15.2
PLT	20 x 10^9/L	156 - 369

	Percentage of Cells	Absolute Number	Normal Range for Absolute
POLYS	68%	3.54	2.24 - 7.68
LYMPHS	19%	0.99	0.80 - 3.65
ATYPICAL LYMPHS	2%	0.10	
MONOS	3%	0.16	0.30 - 0.90
EOS	5%	0.26	0.00 – 0.40
BASO	2%	0.10	0.00 – 0.06
META	1%		
nRBC/100WBC	12		

However, **Laboratory Values on Admission** included the following:

	Patient Value	Normal Range (Male)
WBC	2.6 x 10^9/L	3.8 - 10.6
Hemoglobin	2.7 g/dL	12.9 -16.9
Hematocrit	7.7%	38.0 - 48.8
MCV	129.7 fL	82.6 - 97.4
RDW	46.7%	11.8 - 15.2
PLT	48 x 10^9/L	156 – 369
Reticulocytes	9.2%	0.8 – 2.0
Abs Retic	0.052 x 10^{12}/L	0.018 – 0.158
LDH	6338 IU/L	<170
TBILI	8.8 mg/dL	0.3 – 1.5
DBILI	<0.1	
Iron	159 µg/dL	65 – 165
Saturation	64%	25 – 50%
TIBC	247 µg/dL	250 – 420
Ferritin	135 ng/mL	10 – 282
B12	63 pg/mL	211 – 911
Folate	13.2 ng/mL	>5.4
RBC Folate	805 ng/mL	293 – 809

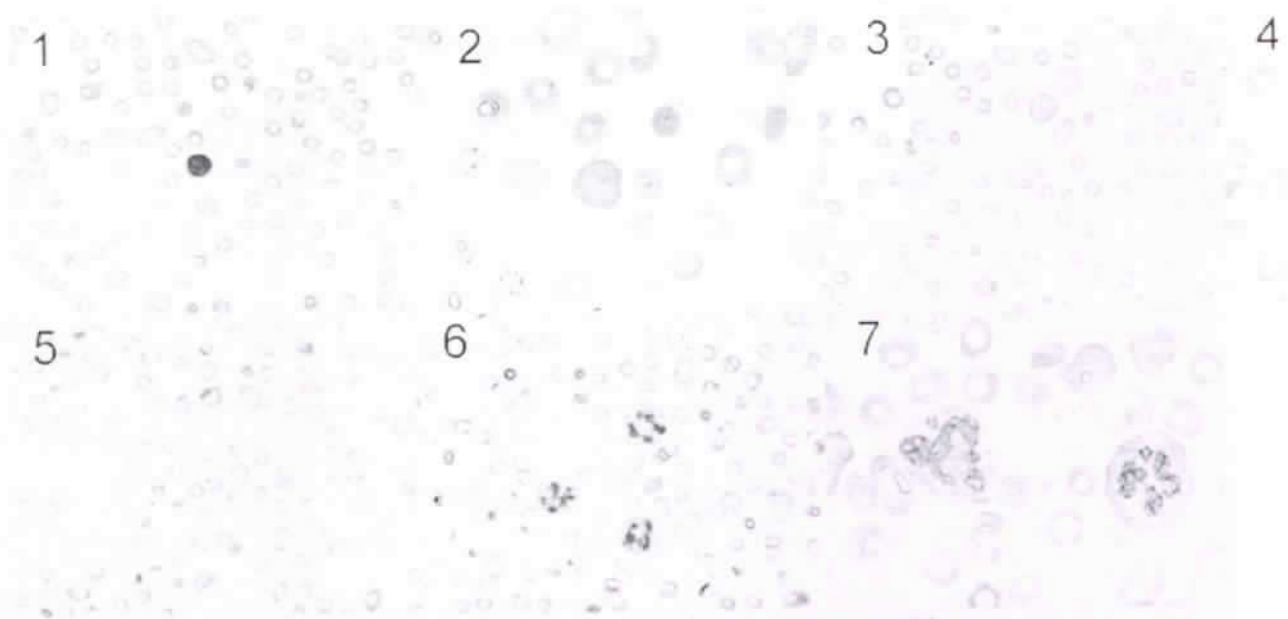
Bone Marrow Differential on aspirate smear:

Bone Marrow Differential	Patient Value	Adult Mean	Normal Range
Blast	0.7%	1.0	0.0 - 2.0
Promyelocyte	2.0%	3.0	2.0 - 4.0
Myelocyte	8.0%	12.0	8.0 - 16.0
Metamyelocyte	5.3%	17.0	10.0 - 25.0
Band	8.3%	12.0	9.0 - 18.0
PMN	10.0%	9.0	7.0 - 14.0
Eos Myelo/Meta	1.3%	2.0	1.0 - 4.0
Eos Band	0.3%	1.0	0.0 - 3.0
Eos Seg	2.3%	1.0	1.0 - 2.0
Basophil	0%	0.0	0.0 - 0.2
Monocytes	0%	1.0	0.0 - 2.0
Pronormoblasts	10.7%	1.0	0.0 - 1.0
Normoblasts	45.7%	24.0	16.0 - 32.0
Lymphocytes	4.3%	16.0	11.0 - 23.0
Plasma Cells	1.0%	2.0	0.0 - 3.0
Other	0%		
Myeloid/Erythroid Ratio	0.7	2.4	1.5 – 3.3

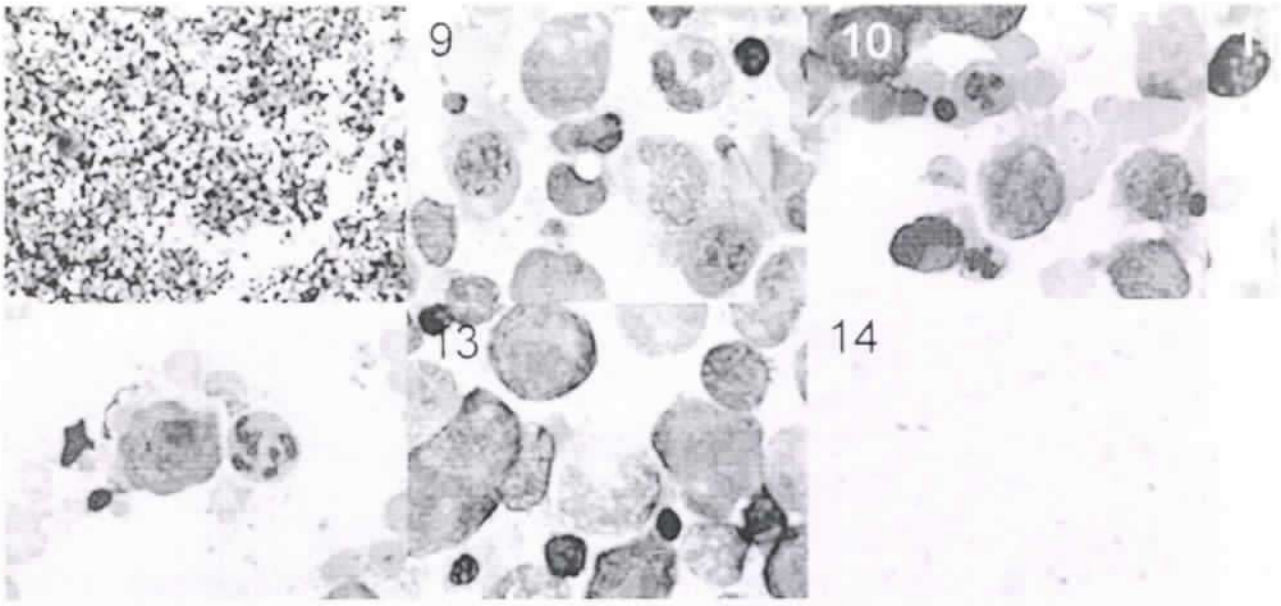
Bacterial and fungal cultures from bone marrow were negative. Parvovirus and HIV were negative.

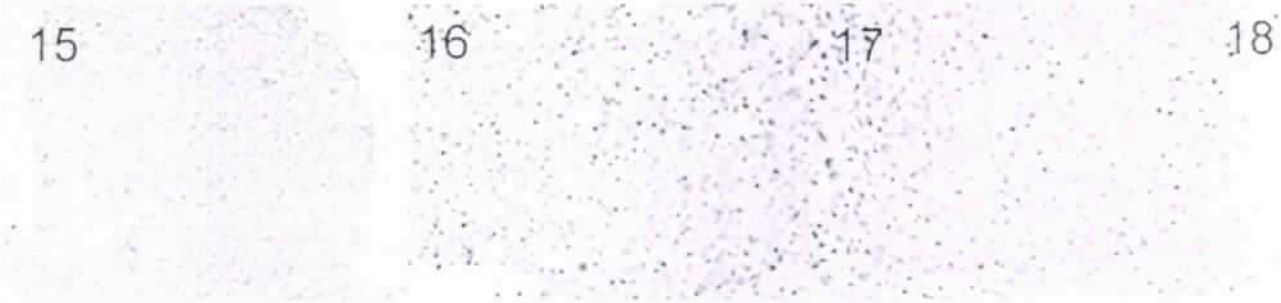
MICROSCOPIC DESCRIPTION:

Peripheral blood erythrocytes show marked anisocytosis (Image 1), basophilic stippling (Image 2), polychromasia (Image 3), nucleated forms (Image 4), and macroovalocytes (Image 5). Hypersegmented granulocytes are frequent (Images 6 and 7).



The marrow is markedly hypercellular, >95% cellularity (Image 15). Erythroid precursors show marked megaloblastic features with large nuclei and open chromatin (Images 9 and 12). Dyserythropoiesis is prominent, including binucleate forms and budding nuclei (Images 10 and 11). Myeloid precursors demonstrate marked megaloblastic features and dysplastic forms, including hypersegmented nuclei and giant metamyelocytes (Images 12 and 13). Megakaryocytes are present in decreased numbers. Stainable iron is present and slightly increased (Image 14). No ringed sideroblasts are identified. Erythroid precursors are large and immature-appearing on biopsy (Images 16 and 17). The decreased myeloid/erythroid ratio is evident on the aspirate smear and biopsy (Image 8, aspirate; Image 17, biopsy with PAS). Mild reticulin fibrosis is present (Image 18).





FLOW CYTOMETRY:

Normal with heterogeneous cellular populations and CD34 positive blasts comprising less than 1.5% of total events analyzed. Evaluation for evidence of paroxysmal nocturnal hemoglobinuria was negative.

CYTOGENETICS:

Normal, 46 X,Y.

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Department of pathology

C.P. C.

CIRCULAR

Date :- 06/04/2018

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3	Physiology	
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6	Pharmacology	
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No.MIMSR/Patho/CPC/1731/ /2018 Date :- 06/04/2018

It is to inform you that, C.P.C. meeting will be held on 10/04/2018 at 3.00 pm in Pathology lecturer hall, copy of case history (Clinical Protocol is attached herewith.

Kindly, circulate this protocol to the all Teacher's, P.G. Student's Interns & Students of your department.

Thanking you,

In-charge 6.4.18
C.P.C. Activity
Dr. A.S.Acharya

H.O.D.
Dept. of Pathology
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Department of Pathology
MIMSR Medical College, LATUR

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No.MIMSR/Patho/CPC/1731/ /2018 Date :- 06/04/2018

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In-charge 6.4.18
C.P.C. Activity
Dr. A.S.Acharya

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MIMSR Medical College, LATUR

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MIMSR MEDICAL COLLEGE, LATUR
DEPARTMENT OF PATHOLOGY
C.P.C PRESENTATION

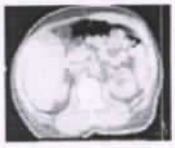
VENUE:- PATHOLOGY LECTURE HALL, DATE 06/04/2018

PATIENT HISTORY

A 66 year old married, Caucasian male with a history of HIV/AIDS presents to the emergency department with a chief complaint of back pain for the past 3 weeks and hemoptysis for the past few days. The pain is sharp, in the left side of his lower back, and does not radiate or cause weakness or numbness. The pain is exacerbated with movement and minimally relieved with pain medication. The patient denies fevers, night sweats, and weight loss. His HIV is managed with Atripla. His HIV RNA levels are undetectable. His family history is significant for his mother having pancreatic cancer, his father having prostate cancer, and his brother acquiring tuberculosis at the age of 14. The patient is a retired maintenance man and lives at home. He has a 15 pack year smoking history. He denies alcohol and intravenous drug use. The physical exam is significant for tachycardia and tenderness over the left rib cage and flank. A straight leg test is positive at 30 degrees, reproducing the patient's flank pain.

IMAGING

A CT scan of the chest, abdomen (Figure 1), and pelvis reveal lytic lesions involving the vertebrae, bilateral iliac wings, and sternum. There is a non-displaced fracture of the left posterior tenth rib. A CT scan of the head reveals lytic lesions of the calvarium.



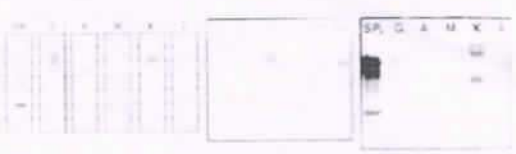
LABS

Table 1 shows baseline lab values. The patient is anemic, thrombocytopenic, and has renal insufficiency. His lactate dehydrogenase is also elevated. Immunology studies reveal decreased levels of IgG and IgM as well as increased β2 microglobulin levels and elevated free Kappa (κ) light chain levels. Free Lambda (λ) light chain levels are within the normal range.

Table 1:

Chemistry	
Patient Values	Normal Values
WBC: 4.6 x 10^3 /L	3.8 – 10.6 x 10^3/L
Hgb: 6.7 g/dL	12.9 – 16.9 g/dL (male)
Hct: 19.3%	38.0 – 48.8%
Platelets: 45 x 10^3/L	150 – 369 x 10^3/L
Sodium: 137 mmol/L	136 – 146 mmol/L
Potassium: 4.4 mmol/L	3.5 – 5.0 mmol/L
Bicarbonate: 24 mmol/L	21 – 31 mmol/L
Calcium: 9.4 mg/dL	8.4 – 10.5 mg/dL
BUN: 42 mg/dL	8.0 – 26 mg/dL
Creatinine: 3.7mg/dL	0.5 – 1.4 mg/dL
Lactate Dehydrogenase: 185	171
Immunology	
Patient Values	Normal Values
IgA: 169	82 – 453
IgG: 701	751 – 1560
IgM: 22	40 – 274
β2 Microglobulin: 8.47 mg/L	< 2.70 mg/L
Free κ: 5600 mg/L	3.3 – 19.4 mg/L
Free λ: 19.1 mg/L	5.7 – 26.3 mg/L
κ/λ (calculated): 293.19	0.26 – 1.65

Figure 2 shows the serum protein electrophoresis results along with immunofixation. Serum protein electrophoresis shows a definitive band in the gamma region that corresponds with the band in the κ column on immunofixation. There are no bands within the IgG, IgA, or IgM immunofixation columns. These results rule out IgGκ, IgAκ, and IgMκ. IgE and IgD are not routinely tested for in our labs, but when a κ band without an associated heavy chain is seen, IgD and IgE immunofixation is performed to test for IgDκ and IgEκ. Immunofixation results for IgDκ and IgEκ are exhibited in Figure 3. A definitive κ band is present. There are no bands in the IgD or IgE columns, ruling out IgDκ and IgEκ. These results suggest free κ chains in the blood. Urine protein electrophoresis and immunofixation results are shown in Figure 4. Urine protein electrophoresis shows large amounts of protein in the urine, with a majority of proteins lying in the gamma region of the gel. κ bands are present, signifying free κ chains in the urine. Serum free κ and λ levels are quantified, and a κ /λ) ratio is calculated, yielding a value of 293.19 (Normal range: 0.26 - 1.65).



A bone marrow biopsy is performed and reveals reduced trilineage hematopoiesis and hypercellular bone marrow involved by neoplasm. Kappa-restricted plasma cells comprise approximately 90% of cells.

Final Diagnosis -?

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Department of pathology

C.P. C.

CIRCULAR

Date :- 21/06/2018

Sr. No.	Name of Department	Receivers Sign
1	Principal	
2	Anatomy	
3	Physiology	
4	Biochemistry	
5	Microbiology	
6	Pharmacology	
7	FMT	
8	PSM	
9	ENT	
10	Ophthalmology	
11	OBGY	
12	Pediatrics	
13	Orthopedic	
14	Surgery	
15	Medicine	
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17	Radiology	
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DEPARTMENT OF PATHOLOGY,

No.MIMSR/Patho/CPC/1731/ /2018 Date :- 21/06/2018

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Kindly, circulate this protocol to the all Teacher's, P.G. Student's Interns & Students of your department.

Thanking you,

In-charge
C.P.C. Activity 21.6.18
Dr. A.S.Acharya

H.O.D.
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The Head
Department of Pathology
MIMSR Medical College, LATUR

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No.MIMSR/Patho/CPC/1731/ /2018 Date :-21/06/2018

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MIMSR MEDICAL COLLEGE, LATUR.
DEPARTMENT OF PATHOLOGY.

C.P.C. PRESENTATION

Venue :- Pathology Lecturer Hall, Date 26/06/2018, Tuesday, at 3.00 pm.

PATIENT HISTORY

Chief Complaint

I feel awful.

HPI

The patient is a 22 year old female with no significant past medical history. Four months prior to admission, she started to notice weight gain, fatigue, facial swelling, increased thirst, and increased frequency of urination. She was seen at an outside institution where she was found to have hypokalemia and an elevated morning cortisol of 54 µg/dL. Additional workup revealed an elevated urinary free cortisol (greater than 1000 µg/day) and an elevated morning cortisol (56 µg/dL) after a 1 mg dexamethasone suppression test. Abdominal CT revealed a liver mass 6 cm in greatest dimension that was thought to be a hemangioma. She was started on amiloride and potassium and referred to endocrinology at UPMC. When she presented at the clinic a few days later, she was found to be unstable and transferred to the Emergency Department, then subsequently admitted to the hospital.

Family History

Non contributory

Vital Signs

Temperature: 36.8 °C
Blood pressure: 132/72 mm Hg
Pulse: 166 per minute
Respiratory rate: 28 per minute

Physical Exam

Notable for central adiposity, Cushingoid facies, a large dorsal fat pad, hirsutism, striae, and proximal muscle weakness.

Initial Studies

Test	Patient Value	Reference Range
Glucose	623 mg/dL	70-99 mg/dL
Sodium	129 mmol/L	136-145 mmol/L
Potassium	4.3 mmol/L	3.5-5.0 mmol/L
Chloride	96 mmol/L	98-107 mmol/L
Bicarbonate	20 mmol/L	21-31 mmol/L
BUN	28 mg/dL	9-20 mg/dL
Creatinine	0.7 mg/dL	0.5-1.4 mg/dL

Test	Patient Value	Reference Range
WBC	19.3×10^3/µL	$3.8\text{-}10.6 \times 10^3$/µL
Hemoglobin	14.6 g/dL	11.6-14.6 g/dL
Hematocrit	41.6%	34.1-43.3%
Platelets	249×10^3/µL	$156\text{-}369 \times 10^3$/µL

Interim Hospital Course

The patient was initially treated with IV fluids and insulin. The patient's pulse decreased to 110 per minute and her blood glucose improved. The patient also noted that she was feeling much better. Next, the following tests were performed:

Test	Patient Value	Reference Range
Cortisol (AM)	30 µg/dL	7.35 µg/dL
Adrenocorticotrophic Hormone (ACTH)	161 pg/mL	9.46 pg/mL

She had an MRI of the pituitary, which revealed an equivocal hypodense area on the left side, suggesting a possible microadenoma.

A high dose (8mg) dexamethasone suppression test was performed and cortisol was found to be 116 µg/dL in the morning, indicating non-suppressible cortisol production.

Imaging Studies

MRI Abdomen

A large (6.0x5.4x4.0cm) enhancing liver mass (Image 1) and bilateral adrenal hyperplasia.

Interim Hospital Course

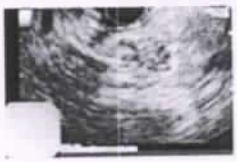
On the second day, she developed emesis with a coffee ground appearance and melena. Her hemoglobin dropped from 14.6 g/dL to 5.1 g/dL. She underwent emergent esophagogastroduodenoscopy (EGD) to evaluate the source of the bleeding and was found to have multiple non-bleeding cratered duodenal ulcers (Image 2).

The patient required multiple blood transfusions, but was eventually stabilized. Additional workup revealed the following results:

Test	Patient Value	Reference Range
Gastrin	1599 pg/mL	100 pg/mL
Chromogranin A	142 ng/mL	1.9-15 ng/mL
Corticotropin-Releasing Hormone (CRH)	20 pg/mL	12 pg/mL

Endoscopic Ultrasound

Mass in the pancreatic body measuring 13 mm by 13 mm in maximal cross-sectional diameter with the endoscopic appearance of a neuroendocrine tumor (Image 3). The lesion was biopsied.



Cytology Report for the Biopsy

Diagnosis: Neuroendocrine Neoplasm

Comment: The specimen is focally cellular and comprised of intermediate to large sized cells with high N:C ratio and granular chromatin pattern. A panel of immunohistochemical studies performed on the cellblock material reveals the following results with regard to the neoplastic process:

Synaptophysin: Positive
ACTH: Positive in rare isolated cells
AE1/AE3: Positive
CEA-m: Negative

MIMSR MEDICAL COLLEGE, LATUR.

Department of pathology

C.P. C.

CIRCULAR

Date :- 21/08/2018

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5	Microbiology	
6	Pharmacology	
7	FMT	
8	PSM	
9	ENT	
10	Ophthalmology	
11	OBGY	
12	Pediatrics	
13	Orthopedic	
14	Surgery	
15	Medicine	
16	Skin & VD	
17	Radiology	
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DEPARTMENT OF PATHOLOGY,

No.MIMSR/Patho/CPC/1731/ /2018 **Date :- 21/08/2018**

It is to inform you that, C.P.C. meeting will be held on 28/08/2018 at 3.00 pm in Pathology lecturer hall, copy of case history (Clinical Protocol is attached herewith.

Kindly, circulate this protocol to the all Teacher's, P.G. Student's Interns & Students of your department.

Thanking you,

In-charge
C.P.C. Activity
Dr. A.S.Acharya

H.O.D.
Dept. of Pathology
The Head
Department of Pathology
MIMSR Medical College, LATUR

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DEPARTMENT OF PATHOLOGY,

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In-charge
C.P.C. Activity
Dr. A.S.Acharya

H.O.D.
Dept. of Pathology
The Head
Department of Pathology
MIMSR Medical College, LATUR

Copy to :-
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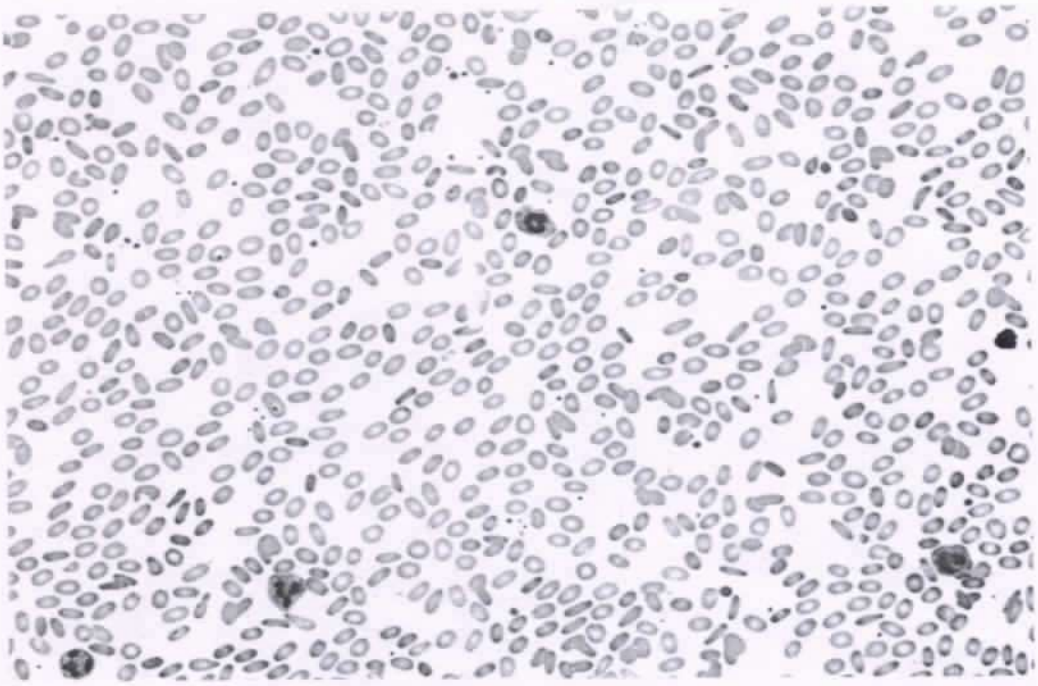
2 **All dept.**

PATIENT HISTORY

The patient is a 33 year old female who presented for a routine clinical evaluation during a twin pregnancy. The patient was asymptomatic otherwise and appeared to be in good health. The patient's gestational age was 22 weeks at clinical presentation. Routine laboratory investigation of the peripheral blood showed an absolute neutrophilia along with



the following clinical laboratory values:

LABORATORY VALUES

	Patient value	Normal range
Hemoglobin	10.6 gm/dL	12.3 -15.3 gm/dL
Hematocrit	29.4 %	35.9 – 44.6 %
WBC count	12,400 x 10^9/L	4,400 – 11,300 x 10^9/L
RBC count	2.9 x 10^{12}/L	4.6 x 10^{12}/L
MCV	101.4 fL	80.0 - 96.1 fL
MCHC	36.4 pg	27.5 – 33.2 pg

Microscopic images of the peripheral blood smear are as follows:

MICROSCOPIC EXAMINATION

Figure 1. Peripheral blood smear (50X). Wright Giemsa stain.

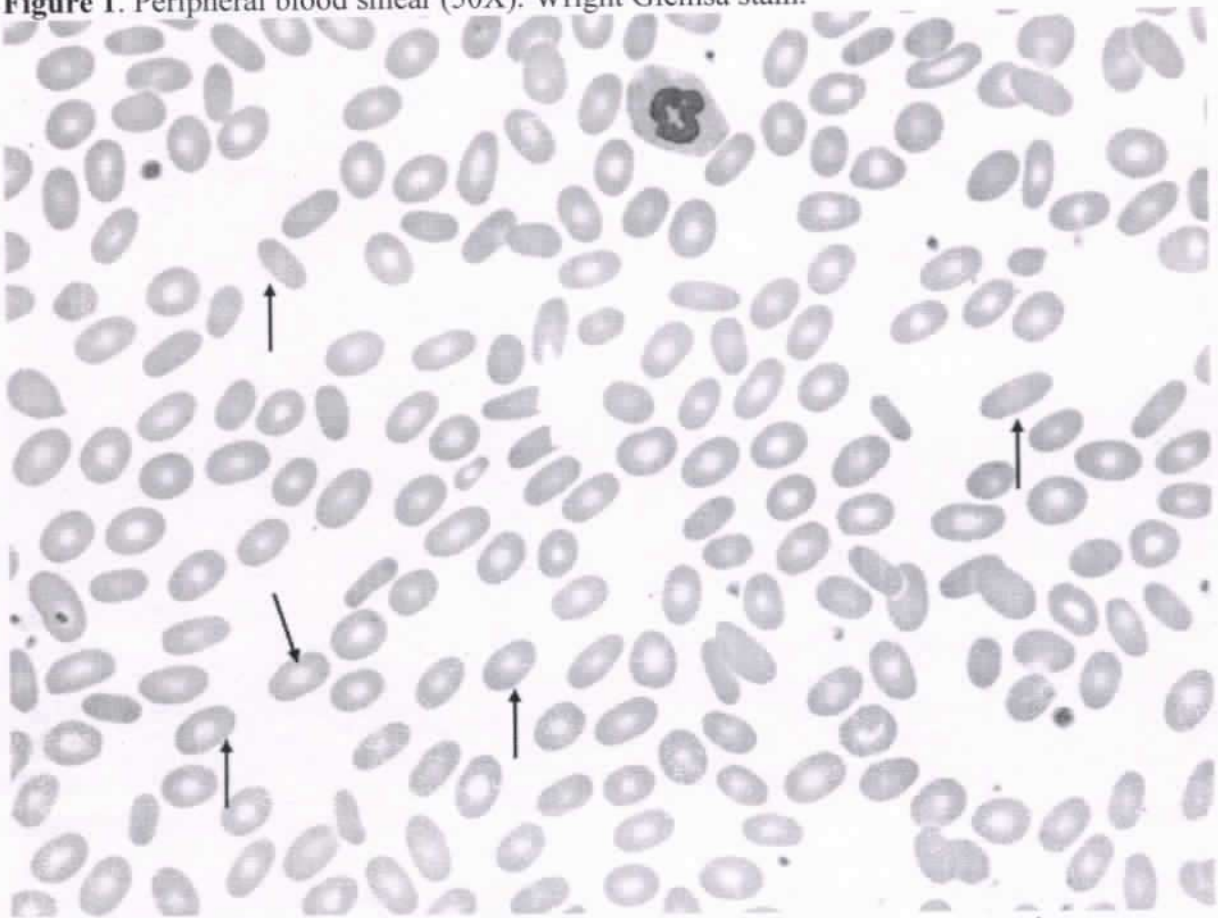


Figure 2. Peripheral blood smear (100X). Arrows point towards the representative abnormal red blood cells.

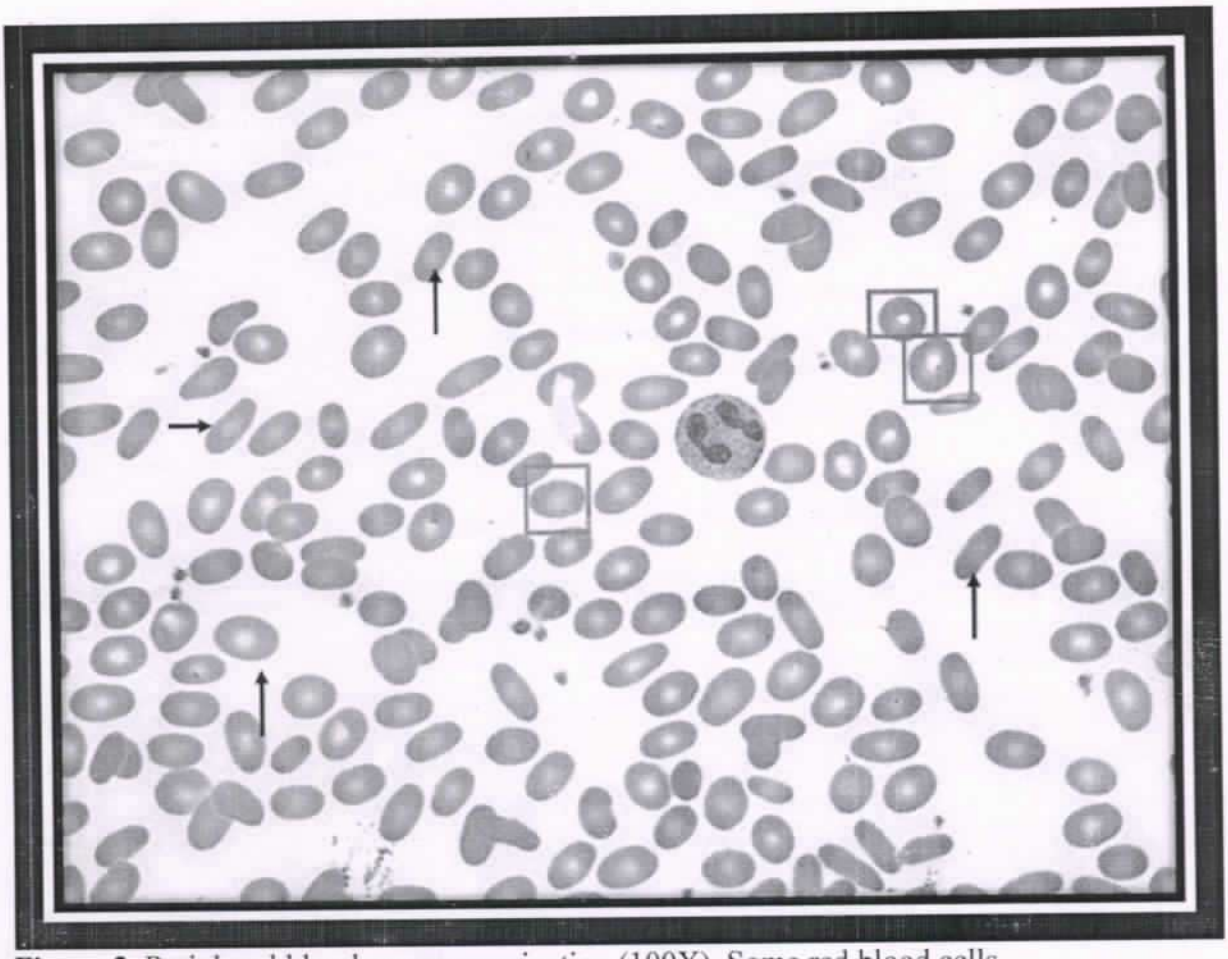


Figure 3. Peripheral blood smear examination (100X). Some red blood cells demonstrate basophilic stippling of the cells (red squares around the cells); arrows point to abnormal red blood cells characteristic of the pathology.

Final Diagnosis -- ?

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CIRCULAR

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No.MIMSR/Patho/CPC/1731/ /2018 **Date :- 19/10/2018**

It is to inform you that, C.P.C. meeting will be held on 23/10/2018 at 3.00 pm in Pathology lecturer hall, copy of case history (Clinical Protocol is attached herewith.

Kindly, circulate this protocol to the all Teacher's, P.G. Student's Interns & Students of your department.

Thanking you,

In-charge
C.P.C. Activity
Dr. A.S.Acharya

H.O.D.
Dept. of Pathology

The Head
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MIMSR Medical College, LATUR

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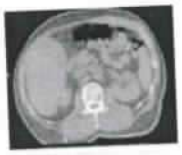
PATIENT HISTORY

. PATIENT HISTORY

A 66 year old married, Caucasian male with a history of HIV/AIDS presents to the emergency department with a chief complaint of back pain for the past 3 weeks and hemoptysis for the past few days. The pain is sharp, in the left side of his lower back, and does not radiate or cause weakness or numbness. The pain is exacerbated with movement and minimally relieved with pain medication. The patient denies fevers, night sweats, and weight loss. His HIV is managed with Atripla. His HIV RNA levels are undetectable. His family history is significant for his mother having pancreatic cancer, his father having prostate cancer, and his brother acquiring tuberculosis at the age of 14. The patient is a retired maintenance man and lives at home. He has a 15 pack year smoking history. He denies alcohol and intravenous drug use. The physical exam is significant for tachycardia and tenderness over the left rib cage and flank. A straight leg test is positive at 30 degrees, reproducing the patient's flank pain.

IMAGING

A CT scan of the chest, abdomen (Figure 1), and pelvis reveal lytic lesions involving the vertebrae, bilateral iliac wings, and sternum. There is a non-displaced fracture of the left posterior tenth rib. A CT scan of the head reveals lytic lesions of the calvarium.



LABS

Table 1 shows baseline lab values. The patient is anemic, thrombocytopenic, and has renal insufficiency. His lactate dehydrogenase is also elevated. Immunology studies reveal decreased levels of IgG and IgM as well as increased β2 microglobulin levels and elevated free Kappa (κ) light chain levels. Free Lambda (λ) light chain levels are within the normal range.

Table 1:

Chemistry	
Patient Values	Normal Values
WBC: 4.6 x 10^3 /L	3.8 – 10.6 x 10^2/L
Hgb: 6.7 g/dL	12.9 – 16.9 g/dL (male)
Hct: 19.3%	38.0 – 48.8%
Platelets: 45 x 10^3/L	156 – 369 x 10^3/L
Sodium: 137 mmol/L	136 – 146 mmol/L
Potassium: 4.4 mmol/L	3.5 – 5.0 mmol/L
Bicarbonate: 24 mmol/L	21 – 31 mmol/L
Calcium: 9.4 mg/dL	8.4 – 10.5 mg/dL
BUN: 42 mg/dL	8.0 – 26 mg/dL
Creatinine: 3.7mg/dL	0.5 – 1.4 mg/dL
Lactate Dehydrogenase: 185	< 171
Immunology	
Patient Values	Normal Values
IgA: 169	82 – 453
IgG: 701	751 - 1560
IgM: 22	40 - 274
β2 Microglobulin: 8.47 mg/L	< 2.70 mg/L
Free κ: 5600 mg/L	3.3 – 19.4 mg/L
Free λ: 19.1 mg/L	5.7 – 26.3 mg/L
κ / λ (calculated): 293.19	0.26 – 1.65

Figure 2 shows the serum protein electrophoresis results along with immunofixation. Serum protein electrophoresis shows a definitive band in the gamma region that corresponds with the band in the κ column on immunofixation. There are no bands within the IgG, IgA, or IgM immunofixation columns. These results rule out IgGκ, IgAκ, and IgMκ. IgE and IgD are not routinely tested for in our labs, but when a κ band without an associated heavy chain is seen, IgD and IgE immunofixation is performed to test for IgDκ and IgEκ. Immunofixation results for IgDκ and IgEκ are exhibited in Figure 3. A definitive κ band is present. There are no bands in the IgD or IgE columns, ruling out IgDκ and IgEκ. These results suggest free κ chains in the blood. Urine protein electrophoresis and immunofixation results are shown in Figure 4. Urine protein electrophoresis shows large amounts of protein in the urine, with a majority of proteins lying in the gamma region of the gel. κ bands are present, signifying free κ chains in the urine. Serum free κ and λ levels are quantified, and a κ /λ) ratio is calculated, yielding a value of 293.19 (Normal range: 0.26 - 1.65).

A bone marrow biopsy is performed and reveals reduced trilineage hematopoiesis and hypercellular bone marrow involved by neoplasm. Kappa-restricted plasma cells comprise approximately 90% of cells.

What is your clinical diagnosis ? :-

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No.MIMSR/Patho/CPC/1731/ /2019 **Date :- 09/02/2019**

It is to inform you that, C.P.C. meeting will be held on 12/02/2019 at 3.00 pm in Pathology lecturer hall, copy of case history (Clinical Protocol is attached herewith.

Kindly, circulate this protocol to the all Teacher's, P.G. Student's Interns & Students of your department.

Thanking you,

In-charge
C.P.C. Activity
Dr. A.S.Acharya

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MIMSR MEDICAL COLLEGE, LATUR.
DEPARTMENT OF PATHOLOGY.

C.P.C. PRESENTATION.

Venue :- Pathology Lecturer Hall,
Date 12/02/2019, **Tuesday, at 3.00 pm.**

PATIENT HISTORY:

- The patient is a 30-year old man with a 30-pound weight loss in one month, poor appetite, and episodes of abdominal and back pain. He has had progressive pancytopenia over 1 month.

Complete Blood Count

	Patient Value	Normal Range (Male)
WBC	3.1 x10E-9 L	3.8 - 10.6
RBC	2.76 x10E-12 L	4.13 - 5.57
Hgb	8.4 g/dl	12.9 - 16.9
Hct	23.9 %	38.0 - 48.8
MCV	86.6 fl	82.6 - 97.4
MCH	30.6 pg	27.8 - 33.4
MCHC	35.3 gm/dl	32.7 - 35.5
RDW	15.4 %	11.8 - 15.2
PLT	18 x10E-9 L	156 - 369

Peripheral Blood Differential

Cell type	Percentage	Abs. No.	Normal Range
POLYS	10 %	0.31	2.24 - 7.68
BANDs	8 %	0.25	0.10 – 0.80
LYMPHS	70 %	2.17	0.80 - 3.65
ATYPICAL LYMPHS	1 %	0.03	0.03
MONOS	5 %	0.16	0.30 – 0.90
BLASTS	5 %	0.16	
META	1 %	0.03	

- **PERIPHERAL BLOOD FILM**
- RED BLOOD CELL MORPHOLOGY: Mild anisocytosis, mild polychromasia, and rare nucleated red blood cells (Figure 1).

- WHITE BLOOD CELL MORPHOLOGY: Blasts are medium to large with scant to moderate cytoplasm, fine chromatin, round to irregular nuclei, and small nucleoli.

Some blasts display fine granules, and some appear monocytic and could be classified as promonocytes

- PLATELETS: Decreased.
- **BONE MARROW**

Bone Marrow Differential	Patient Value	Normal Range
Blast	82.3 %	0.0 - 2.0
Promyelocyte	0.7 %	2.0 - 4.0
Myelocyte	0.3 %	8.0 - 16.0
Metamyelocyte	1.3 %	10.0 - 25.0
Band	1.7 %	9.0 - 18.0
PMN	0.7 %	7.0 - 14.0
Eos Myelo Meta	0 %	1.0 - 4.0
Eos Band	0 %	0.0 - 3.0
Eos Seg	0 %	1.0 - 2.0
Basophil	0 %	0.0 - 0.2
Monocytes	4.7 %	0.0 - 2.0
Pronormoblasts	0 %	0.0 - 1.0
Normoblasts	5.0 %	16.0 - 32.0
Lymphocytes	3.3 %	11.0 – 23.0
Plasma Cells	0%	0.0 - 3.0
Other	0%	
Meyloid Erythroid (ratio)	N A	1.5 – 3.3

Total = of cells counted: 300

- The marrow aspirate smears are paucispicular with some necrotic material, but display intact cells in some areas (Figures 3 and 4). The bone marrow biopsy is approaching 100% cellular in some areas, but also shows large areas of coagulative necrosis (Figures 5 and 6). The aspirate smears and intact areas on the biopsy display numerous blasts with a similar morphology to those observed in the peripheral blood (Figures 3, 4 and 7). Normal hematopoiesis is almost completely effaced. Only very rare maturing granulocytes and erythroid precursors are seen. Megakaryocytes appear absent. No ringed sideroblasts are identified on the iron stain done on a touch imprint, but erythroid precursors are rare.

- What is your diagnosis?

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It is to inform you that, C.P.C. meeting will be held on 09/04/2019 at 3.00 pm in Pathology lecturer hall, copy of case history (Clinical Protocol is attached herewith.

Kindly, circulate this protocol to the all Teacher's, P.G. Student's Interns & Students of your department.

Thanking you,

In-charge
C.P.C. Activity
Dr. A.S.Acharya

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MIMSR Medical College, LATUR

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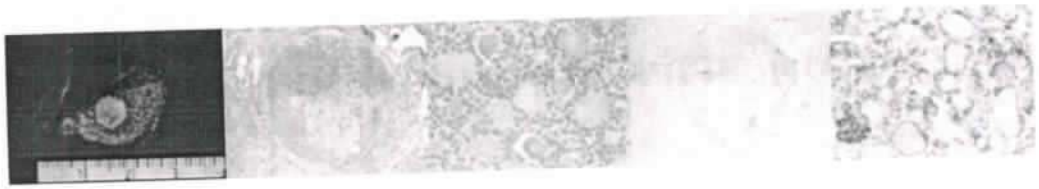
A 63-year-old woman with a thyroid nodule.

HISTORY -

The patient is a 63 year old woman with elevated calcium who, on work-up for hyperparathyroidism, was found to have a single nodule within the superior pole of the left lobe of her thyroid, measuring 0.5 cm, as well as three hypoechoic nodules of the right lobe, measuring up to 2.0 cm. The patient underwent surgical parathyroid exploration and total thyroidectomy after fine needle aspiration of the dominant right lobe nodule demonstrated a "follicular neoplasm or lesion." However, the remainder of this case will focus on a smaller right lobe nodule that was not biopsied.



Grossly, a 0.5 x 0.5 x 0.5 cm right superior pole nodule was found to be well-circumscribed, thinly encapsulated, solid, tan and soft (Figure 1, Gross appearance of thyroid nodule).

Histologic sections demonstrated a well-circumscribed, encapsulated lesion with cells showing enlarged nuclei, with an open chromatin pattern, prominent nucleoli, some with nuclear grooves and some nuclear overlap (Figures 2, H&E, low power and 3, H&E, high power). Immunohistochemical staining demonstrated positivity for HBME-1 (Figures 4, HBME-1, low power and 5, HBME-1, high power).

FINAL DIAGNOSIS - ?

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It is to inform you that, C.P.C. meeting will be held on 11/06/2019 at 3.00 pm in Pathology lecturer hall, copy of case history (Clinical Protocol is attached herewith.

Kindly, circulate this protocol to the all Teacher's, P.G. Student's Interns & Students of your department.

Thanking you,

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It is to inform you that, C.P.C. meeting will be held on 13/08/2019 at 3.00 pm in Pathology lecturer hall, copy of case history (Clinical Protocol is attached herewith.

Kindly, circulate this protocol to the all Teacher's, P.G. Student's Interns & Students of your department.

Thanking you,

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Dr. A.S.Acharya

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MIMSR Medical College, LATUR

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Thanking you,

In-charge
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Dr. A.S.Acharya

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Case- An 82 year old female with thrombocytopenia

Hematopathology

Contributed by Brian K. Theisen, MD and Raymond E. Felgar MD, PhD

PATIENT HISTORY

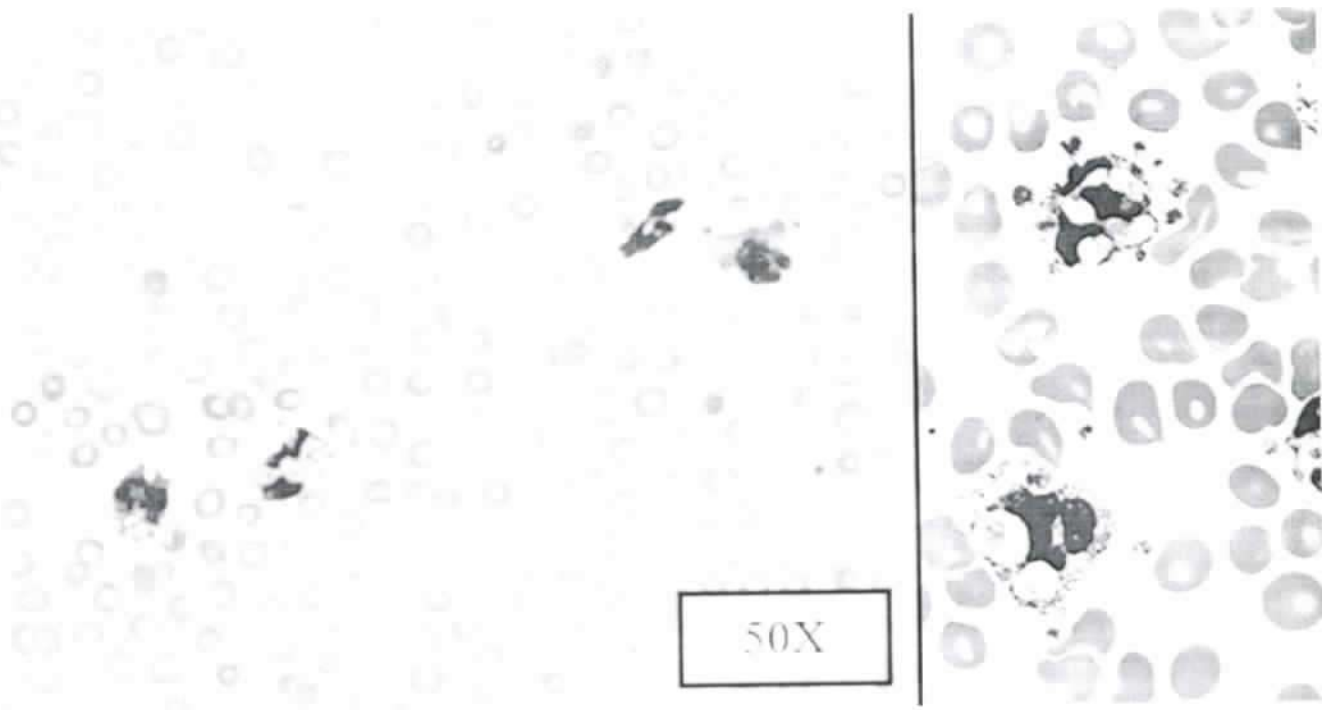
An 82 year old female with a medical history of mitral regurgitation and prolapse and moderate coronary artery disease presented to the emergency department with increased dyspnea on exertion. She underwent coronary artery bypass graft and mitral valve repair surgery. A post-operative CBC demonstrated a mildly elevated white blood cell count, normocytic anemia with occasional ovalocytes and acanthocytes and thrombocytopenia. A peripheral blood smear was sent for pathology review.

LABORATORY

The post-operative complete blood count and differential revealed the following:

Results Assessed	Patient Value	Normal Value
WBC	10.7	(3.8-10.6) x10^3 L
RBC	3.04	(3.73-4.89) x10^3 L
Hgb	9.4	(11.6-14.6) g dL
Hct	27.4	(34.10-43.3)%
MCV	90.2	(82.6-97.4) fL
MCH	31.0	(27.8-33.4) pg
MCHC	34.3	(32.7-35.5) g dL
RDW	15.1	(11.8-15.2)%
Mean platelet volume	9.9	(6.8-10.4) fL
Poly	63	(44-77)%
Lymphocytes	20	(13-44)%
Monocytes	11	(4-13)%
Eosinophils	6	(0-6)%
ABS Poly	6.74	(2.24-7.68) fL
ABS Lymphocytes	2.14	(0.80-3.65) pg
ABS Monocytes	1.18	(0.30-0.90) g dL
ABS Eosinophils	0.64	(0.00-0.40) x10^3 L
Ovalocytes	Present	
Acanthocytes	Present	
Platelets	66	(156-369) x10^3 L

Morphologic review of the peripheral blood smear revealed neutrophils surrounded by platelets as well as prominent phagocytosis of the platelets by neutrophils (see image). This phenomenon was not observed in association with other cells (i.e. red blood cells, eosinophils, monocytes or lymphocytes). Due to the association of the platelets with neutrophils, the automated instrument count was not accurately reflecting the platelet count.



12

Date:- 03/07/2019.

MIMSR Medical College & YCR Hospital, Latur
Department of Pediatrics
Interdepartmental Meeting Circular

An Interdepartmental meeting has been arranged on 6th July 2019, from 11am to 1pm, for discussion on the case of Piyusha Chate 6 and 1/2 years old female child with steroid resistant nephrotic syndrome.

Kindly greet the meeting and give your highlights on the case.

Thank you,

Prof. & HOD
Dept. of Pediatrics
MIMSR Medical College, Latur.

Copy to:-

1) Dept. of Pharmacology.
2) Dept. of Pathology ✓

noted.

Date:- 04/09/2019.

MIMSR Medical College & YCR Hospital, Latur
Department of Pediatrics
Interdepartmental Meeting Circular

We have arranged interdepartmental meeting on 7th Sep. 2019 Saturday from 11am to 1pm, on patient Kirti Sonawane, 9 years old girl with Recurrent UTI with Vesico-ureteric Reflux (VUR).

Kindly greet the meeting and give your highlights.

Thank you,

Prof. & HOD
Dept. of Pediatrics
MIMSR Medical College, Latur.

Copy to:-

1) Dept. of Radiology.
2) Dept. of Pathology. ✓
3) Dept. of Pharmacology.

noted

Date:- 31/07/2019.

MIMSR Medical College & YCR Hospital, Latur
Department of Pediatrics
Interdepartmental Meeting Circular

An Interdepartmental meeting has been arranged on 3rd August 2019, from 11am to 1pm, for discussion on the case of Radha Jadhav 7 and 1/2 years old female child with Acute rheumatic fever with Mitral Regurgitation.

Kindly greet the meeting and give your highlights on the case.

Thank you,

Prof. & HOD
Dept. of Pediatrics
MIMSR Medical College, Latur.

Copy to:-

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2) Dept. of Radiology

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No.MIMSR/Patho/CPC/1731/ /2019 **Date :- 12/10/2019**

It is to inform you that, C.P.C. meeting will be held on 15/10/2019 at 3.00 pm in Pathology lecturer hall, copy of case history (Clinical Protocol is attached herewith.

Kindly, circulate this protocol to the all Teacher's, P.G. Student's Interns & Students of your department.

Thanking you,

In-charge
C.P.C. Activity
Dr. A.S.Acharya

H.O.D.
Dept. of Pathology

Copy to :-
1 **Principal,**
2 **All dept.**

MIMSR MEDICAL COLLEGE , LATUR.
DEPARTMENT OF PATHOLOGY,

No.MIMSR/Patho/CPC/1731/ /2019 **Date :- 12/10/2019**

It is to inform you that, C.P.C. meeting will be held on 15/10/2019 at 3.00 pm in Pathology lecturer hall, copy of case history (Clinical Protocol is attached herewith.

Kindly, circulate this protocol to the all Teacher's, P.G. Student's Interns & Students of your department.

Thanking you,

In-charge
C.P.C. Activity
Dr. A.S.Acharya

H.O.D.
Dept. of Pathology

Copy to :-
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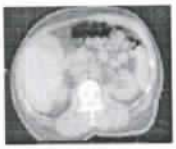
2 **All dept.**

PATIENT HISTORY

A 66 year old married, Caucasian male with a history of HIV/AIDS presents to the emergency department with a chief complaint of back pain for the past 3 weeks and hemoptysis for the past few days. The pain is sharp, in the left side of his lower back, and does not radiate or cause weakness or numbness. The pain is exacerbated with movement and minimally relieved with pain medication. The patient denies fevers, night sweats, and weight loss. His HIV is managed with Atripla. His HIV RNA levels are undetectable. His family history is significant for his mother having pancreatic cancer, his father having prostate cancer, and his brother acquiring tuberculosis at the age of 14. The patient is a retired maintenance man and lives at home. He has a 15 pack year smoking history. He denies alcohol and intravenous drug use. The physical exam is significant for tachycardia and tenderness over the left rib cage and flank. A straight leg test is positive at 30 degrees, reproducing the patient's flank pain.

IMAGING

A CT scan of the chest, abdomen (Figure 1), and pelvis reveal lytic lesions involving the vertebrae, bilateral iliac wings, and sternum. There is a non-displaced fracture of the left posterior tenth rib. A CT scan of the head reveals lytic lesions of the calvarium.



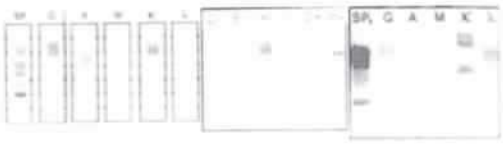
LABS

Table 1 shows baseline lab values. The patient is anemic, thrombocytopenic, and has renal insufficiency. His lactate dehydrogenase is also elevated. Immunology studies reveal decreased levels of IgG and IgM as well as increased β2 microglobulin levels and elevated free Kappa (κ) light chain levels. Free Lambda (λ) light chain levels are within the normal range.

Table 1:

Chemistry	
Patient Values	**Normal Values**
WBC: 4.6 x 10^3 /L	3.8 – 10.6 x 10^3/L
Hgb: 6.7 g/dL	12.9 – 16.9 g/dL (male)
Hct: 19.3%	38.0 – 48.8%
Platelets: 45 x 10^3/L	156 – 369 x 10^3/L
Sodium: 137 mmol/L	136 – 146 mmol/L
Potassium: 4.4 mmol/L	3.5 – 5.0 mmol/L
Bicarbonate: 24 mmol/L	21 – 31 mmol/L
Calcium: 9.4 mg/dL	8.4 – 10.5 mg/dL
BUN: 42 mg/dL	8.0 – 26 mg/dL
Creatinine: 3.7mg/dL	0.5 – 1.4 mg/dL
Lactate Dehydrogenase: 185	< 171
Immunology	
Patient Values	**Normal Values**
IgA: 169	82 – 453
IgG: 701	751 - 1560
IgM: 22	40 - 274
β2 Microglobulin: 8.47 mg/L	< 2.70 mg/L
Free κ: 5600 mg/L	3.3 – 19.4 mg/L
Free λ: 19.1 mg/L	5.7 – 26.3 mg/L
κ / λ (calculated): 293.19	0.26 – 1.65

Figure 2 shows the serum protein electrophoresis results along with immunofixation. Serum protein electrophoresis shows a definitive band in the gamma region that corresponds with the band in the κ column on immunofixation. There are no bands within the IgG, IgA, or IgM immunofixation columns. These results rule out IgGκ, IgAκ, and IgMκ. IgE and IgD are not routinely tested for in our labs, but when a κ band without an associated heavy chain is seen, IgD and IgE immunofixation is performed to test for IgDκ and IgEκ. Immunofixation results for IgDκ and IgEκ are exhibited in Figure 3. A definitive κ band is present. There are no bands in the IgD or IgE columns, ruling out IgDκ and IgEκ. These results suggest free κ chains in the blood. Urine protein electrophoresis and immunofixation results are shown in Figure 4. Urine protein electrophoresis shows large amounts of protein in the urine, with a majority of proteins lying in the gamma region of the gel. κ bands are present, signifying free κ chains in the urine. Serum free κ and λ levels are quantified, and a κ /λ) ratio is calculated, yielding a value of 293.19 (Normal range: 0.26 - 1.65).



A bone marrow biopsy is performed and reveals reduced trilineage hematopoiesis and hypercellular bone marrow involved by neoplasm. Kappa-restricted plasma cells comprise approximately 90% of cells.

Final Diagnosis -- Infection of *Mycobacterium tuberculosis*

Sequence Based Susceptibility Results

Locus	Drug	Result	Likelihood of unknown mutation*
rpoB	Rifampin	No mutation	3%
inhA	Isoniazid	No mutation	14%
katG	Isoniazid	No mutation	14%
embB	Ethambutol	No mutation	21%
pncA	Pyrazinamide	No mutation	14%
gyrA	Quinolones	No mutation	20%
rrs	Kanamycin Amikacin Capreomycin	No mutation	9%
eis	Kanamycin	No mutation	13%
tlyA	Capreomycin	No mutation	45%

*Based on an examination of 500 isolates at CDC

All tested loci were negative for mutations. The isolate was also sent for culture based susceptibility testing. Once the patient's liver tests returned to normal, he was gradually re-started on rifampin, isoniazid, ethambutol, and pyrazinamide; he was also placed on moxifloxacin. He had a difficult clinical course, resulting in a repeat admission approximately three weeks after discharge due to poor appetite, weight loss, and fatigue. He was then discharged to short-term rehabilitation for nutritional support.

Subsequent biochemical testing performed on the isolate for niacin and nitrate reduction were positive, indicating that this isolate is indeed *Mycobacterium tuberculosis*. Phenotypic testing revealed that the patient's isolate was susceptible to rifampin, isoniazid, ethambutol, and pyrazinamide. The Department of Homeland Security indicated that the patient would be required to complete two weeks of effective antituberculous therapy and document three negative AFB smears before he would be permitted to board an airplane.

DISCUSSION

Mycobacterium tuberculosis is still the leading single infectious cause of death throughout the world (1, 2). Within the United States, TB is rare, but has been noted in immigrants and individuals travelling from TB endemic areas. Resistance to anti-tuberculous medications occurs in two predominant forms: MDR (Multi-drug resistant) TB is resistant to two of the primary treatment agents, rifampin and isoniazid (3). XDR (Extensively drug resistant) TB demonstrates resistance at the MDR level, plus resistance to fluoroquinolones (such as ciprofloxacin) and an injectable agent (e.g. aminoglycosides).

In the US, MDR TB is believed to account for approximately 1.1% of new cases (4). However, resistance to single agents is more common, with approximately 8% of new cases resistant to Isoniazid (1), and approximately 1.8% resistant to Rifampin (5). Thus, it is important to assess new cases of tuberculosis for resistance to anti-tuberculous agents. However, due to the slow growing nature of TB, drug sensitivity testing (DST) can take up to 6 weeks to complete depending on the method used (4). This is an inordinate amount of time to wait prior to starting appropriate therapy, so all patients are started on empiric treatment pending the results of sensitivity studies. Molecular drug sensitivity testing is now starting to be available, and can dramatically decrease turn-around times in these cases.

All cases of tuberculosis are reported to the County and State health departments, who also report these cases to the Centers for Disease Control (CDC). There is a quarantine process in effect whereby these patients may be placed on a Do Not Board list (jointly managed by CDC and the Department for Homeland Security), and prevented from obtaining a boarding pass or boarding an aircraft in this country. There are explicit criteria for placement on this list, including if the patient is infectious, non-compliant or unaware of their diagnosis and has expressed intention to travel (6). These lists are accessible by both the airlines and Customs and Border Protection, who may enforce such provisions. In addition, if it is believed that the patient previously traveled while infectious, flight manifests can be requested from the airlines, and the local state and county health departments will attempt to arrange for skin testing for other passengers on the flight. In the case of TB, only passengers sitting in the same row as the index case, as well as passengers two rows in front and two rows behind, will be notified (7).

Genetic mechanisms of drug resistance in TB are complex, but are thought to be primarily due to chromosomal mutations, and are not plasmid mediated (1, 2). These changes occur rather infrequently, with fewer new mutations than would be expected by random variation (3). Given the relatively stable chromosomal mutations that are seen, molecular assays for microbial classification and drug sensitivity have been developed. Many of these tests allow for rapid (within 2 hours) identification of *Mycobacterium tuberculosis* and determination of preliminary sensitivity patterns. Some of these new tests are even available at the point of care, with minimal technical training required (8). These assays also allow for the separation of *Mycobacterium tuberculosis* from other atypical mycobacteria, which can be pertinent in guiding empiric therapy (4). Additionally, these tests are more sensitive than traditional techniques and allowed for the detection of AFB smear negative disease in up to 90% of cases in one study (9).

Reference laboratory molecular testing for anti-tubercular drug sensitivity is also available on multiple platforms. Recent studies have demonstrated the viability of the Ion Torrent platform for next generation sequencing, with 100% concordance with phenotypic sensitivity studies, and the ability to characterize mixed strain populations (10, 11). Sanger sequencing is still the gold standard for most of these reference type assays, with focused examination of multiple genetic loci required in order to detect clinically significant mutations. Importantly, the absence of mutation in any of these assays is NOT confirmation of drug susceptibility, as all mechanisms of drug resistance have not yet been elucidated (12). This can cause false negative results if the specific locus is not sequenced. The converse is often true, however, whereby detection of mutation signifies resistance to the selected drug. For these reasons, culture based sensitivity studies still must be performed to ensure that sequence-based testing has not failed to detect an unknown mutation. Current

indications for sequence based sensitivity testing include: strains with a high risk of rifampin resistance (based on patient origin or contacts), patients known to have adverse reactions to certain anti-tuberculous drugs, patients where drug resistance will have a high public health impact (such as daycare workers, nurses, and our present patient), mixed or non-viable cultures, and isolates which fail to grow on drug susceptibility testing (12).

Detection of rifampin resistance is most pertinent in many cases due to its usage as a first-line therapeutic agent. Fortunately, the majority (>95%) of rifampin resistant isolates have a single point mutation in an 81bp region of the rpoB gene, enabling this assay to be very sensitive. Mutations in this region alter protein structure so as to prevent rifampin binding (12). Isoniazid resistance is also of concern, as it often occurs concurrently with rifampin resistance (8), and is a primary treatment modality for tuberculosis. There are two important genetic loci examined for isoniazid resistance, including inhA, which encodes the promoter region of the drug target (mutations here lead to overproduction and resultant drug saturation) and katG (mutations at this locus prevent activation of the isoniazid prodrug) (12). Additional genes are also analyzed in many comprehensive molecular sensitivity panels (see above results for entire panel, with likelihood of unknown mutation causing false negative result) (12).

Molecular drug sensitivity testing for tuberculosis provides rapid turnaround with relatively high specificity for the tested agent. Culture based sensitivity must still be completed to ensure genotype-phenotype correlation, but these new assays allow for faster testing, treatment, and cure of this hard to culture and sometimes difficult to treat microorganism.

Acknowledgments

The authors would like to thank Jeffrey Driscoll, PhD at the Centers for Disease Control and Prevention, National Center for Hepatitis, HIV, STD and TB Prevention for his assistance with this case, and subsequent review of this case report.

REFERENCES

1. Ramaswamy SV, Reich R, Dou SJ, Jasperse L, Pan X, Wanger A, Quitugua T, Graviss EA. Single nucleotide polymorphisms in genes associated with isoniazid resistance in *Mycobacterium tuberculosis*. Antimicrob Agents Chemother 2003; 47(4): 1241-1250.
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3. McGrath M, Gey van Pittius NC, van Helden PD, Warren RM, Warner DF. Mutation rate and the emergence of drug resistance in *Mycobacterium tuberculosis*. J Antimicrob Chemother 2013 Sept 26 [Epub ahead of print].
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MIMSR MEDICAL COLLEGE, LATUR.

Department of pathology

C.P.C.

CIRCULAR

Date :- 7/12/2019

Sr. No.	Name of Department	Receivers Sign
1	Principal	
2	Anatomy	
3	Physiology	
4	Biochemistry	
5	Microbiology	
6	Pharmacology	
7	FMT	
8	PSM	
9	ENT	
10	Ophthalmology	
11	OBGY	
12	Pediatrics	
13	Orthopedic	
14	Surgery	
15	Medicine	
16	Skin & VD	
17	Radiology	
18	PSY	
19	Anesthesia	

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DEPARTMENT OF PATHOLOGY,

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The Head
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MIMSR Medical College, LATUR

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The Head
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MIMSR Medical College, LATUR

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PATIENT HISTORY

The patient is a 55-year-old man who presented to the ED with gradual worsening of weakness. The patient reports that he had been fatigued for one month, having to rest constantly after work, and feeling exhausted with minimal exertion. He also reported feeling lightheaded with rising, as well as having epigastric abdominal pain, which was not associated with food. Four days prior to the presentation, the patient began to have black, tarry stools. He was evaluated in his primary care physician's office the day prior, and was found to have hemoglobin of 6.5 g/dL. His prior baseline from two weeks ago was 15.3 g/dL. He was subsequently referred to the ED.

The patient has a history of pancolonic ulcerative colitis diagnosed in 1979 and now in remission. He has not been on medication for 17 years, but he did receive treatment with prednisone around time of diagnosis, then sulfasalazine. He had undergone recent surveillance EGD and colonoscopy a month ago, which was unchanged from previous studied. He also has gastroparesis, not symptomatic for several years. He is on ASA 81 mg. He takes a PPI for GERD which is well-controlled.

In the ED, his vital signs were: pulse: 121, blood pressure: 127/85, respiratory rate: 20, and oxygen saturation: 99% on room air. Physical examination was remarkable for generalized pallor, mild tachycardia, and diastasis of rectus (also known as abdominal separation). Nasogastric lavage was negative, but the fetal occult blood test was positive. Pertaining labs are listed below:

	ED Labs	Reference Range
Hematology:		
WBC	9.4	3.8-10.8 THOUS MCL
RBC	3.07	4.20-5.80 MILL MCL
Hgb	6.9	13.2-17.1 G DL
HCT	22.4	38.5-50.0 %
MCV	73.1	80.0-100.0 FL
MCH	22.6	27.0-33.0 PG
MCHC	30.9	32.0-36.0 G DL
RDW	21.1	11.0-15.0 %
Chemistry:		
Iron	<10	45-182 ug dL
Total Iron Binding Capacity	298	250-420 ug dL
Ferritin	63	10-282 ng mL

Following examination and GI consult, he was admitted on to the floor for further work up of his GI bleeding. Packed red blood cells were ordered for transfusion with goal hemoglobin of greater than 8 g/dL. Seventy milliliters within the transfusion of the first packed red blood cell, the patient became hypotensive and diaphoretic with chill/rigor and dyspnea. Pre and post vital signs were:

Vital Signs	Baseline	At Reaction
Temperature	97.6	98.0
Blood Pressure	110 71	62 52
Pulse	105	101
Respiration	23	26
O2SAT	99%	88%

A condition C was called.

FINAL DIAGNOSIS

Final Diagnosis -- Severe allergic reaction to plasma proteins

---

FINAL DIAGNOSIS

Severe allergic reaction to plasma proteins (most likely IgA)

DISCUSSION

Introduction

The immunoglobulin IgA is the most abundant immunoglobulin in the human body. It is found in tissues and in secretions especially from the GI tract and the respiratory tract in the form of saliva, tears, breast milk, but has very low levels in serum. This reflects its role in mucosal immunity and the development of tolerance. Approximately 5 to 15 g of IgA (66mg/kg/day) [1] is produced daily in an adult and this is more than any other immunoglobulin. IgA exists as two isoforms, IgA1 and IgA2. In the circulation, IgA exists as a monomer. In secretions it is present in dimeric form, and the complex also includes a J chain and a secretory piece (Figure 1). All of these components are necessary in order for secretory IgA to preserve its function[1]. IgA2 after forming a dimer has a shorter hinge than IgA1, and is therefore thought to confer more resistance to bacterial proteolytic enzymes[1]. There is preliminary evidence indicating that normal levels of IgA vary based on certain demographic characteristics such as age, ethnicity, gender, and body habitus[1].

Clinical Significance of IgA deficiency

Primary antibody deficiency (PAD) is defined as a reduction or absence of one or more immunoglobulin without a contributory disorder or cause. IgA deficiency (IgAD) is the most common PAD. The definition of IgAD is a measurement of <7 to <5 mg/dL (the range is selected depending upon the lowest local laboratory detectable testing limit) in patients over the age of 4 years[2]. Age 4 is used to avoid premature diagnosis of transient deficiency that may be present in younger children with delayed IgA development[2]. Although the prevalence of IgAD varies significantly in different areas and among races, it has been estimated to 1:328 in the United States[3]. IgA deficiency has expanded since its

first description in 1964 in two healthy subjects, it has become known that IgA deficiency can be both clinically manifested and associated with a myriad of other diseases. It has also been found that among patients initially diagnosed with autoimmune diseases the prevalence of IgA deficiency is increased. However, the reciprocal is also true: patients initially diagnosed with IgA deficiency have been found to have a higher prevalence of autoimmune diseases. Most individuals identified as IgAD are asymptomatic and identified incidentally during laboratory evaluation for celiac disease, allergy, or autoimmune disease by medical specialists working in rheumatology, hematology, internal medicine, and allergy testing[4]. Although incidental diagnosis of IgAD may not be of clinical significance to the specialists seeking disease diagnosis, it can be associated with future implications for blood transfusion administration and with the increased risks of development of recurrent sinopulmonary infections, gastrointestinal infections and disorders, and autoimmune diseases[4].

IgAD transfusion reaction

Allergic blood transfusion reactions are one of the most common adverse transfusion events, but there are no reliable estimate regarding the incidence of IgA allergic reaction. The first reported case of anaphylactic transfusion reaction associated with IgAD was published more than 40 years ago[5]. The pathophysiology of IgA-mediated anaphylaxis in humans has not been clearly elaborated. Given that mast cells, IgE, FceR, and histamine are generally considered major players in anaphylaxis reactions, IgG-mediated systemic anaphylaxis was recently demonstrated in the murine system involving FccRs, basophils and platelet activating factor (PAF) as major players (Figure 2). In this reaction, PAF rather than histamine was the major chemical mediator that induced systemic anaphylaxis[6]. This study showed that both IgG and IgE mechanisms are involved in IgA-mediated anaphylaxis. Although it remains uncertain whether this pathway is present in humans, there is supportive evidence for this mechanism.

Signs and symptoms of IgA anaphylactic reactions include hives, rash, pruritis, angioedema, dyspnea, stridor, wheezing, hypotension, syncope, arrhythmia, shock, cramps, diarrhea, and vomiting[7]. These signs and symptoms are common to any anaphylactic reaction and do not differentiate the etiology of the reaction.

Although pre-transfusion medications such as acetaminophen and diphenhydramine are not always effective[6], most transfusion reactions are easily treated. When urticaria occurs, diphenhydramine may be administered. Severe urticarial reactions may require treatment with methylpredonisolone or predonine. Once a severe reaction develops or anaphylaxis occurs, prompt action should be taken to maintain oxygenation levels and stabilize hypotension. Epinephrine may be administered intramuscularly or subcutaneously. In case the patient is unconscious or in shock, epinephrine may be given intravenously. If bronchospasm is present, respiratory symptoms may not respond to epinephrine, and adding a beta II agonist or aminophylline may be required. The three approaches that have been employed to transfuse IgA-deficient patients with anti-IgA are: (1) autologous blood transfusion, (2) transfusion with fully washed red blood cell (RBC) or platelet (PLT) components, or (3) transfusion components from IgA-poor blood donors[3].

Miscellaneous facts

Gonorrhea patients can have low IgA levels/function due to the ability of the bacteria to cleave IgA into its Fab and Fc fragments. Other bacterial such as pneumococcus may also have this ability.

CASE CONCLUSION The patient's symptoms resolved after treatment with solumedrol and Benadryl. The patient received 2 units of washed red blood cells several hours later without any adverse reactions reported. Laboratory investigation for hemolysis was negative. Since the patient had a transfusion history of receiving 4 units of unwashed red blood cells without allergic reaction in 10/2000, an IgA level was measured and determined to be less than 7 mg/dL (normal range: 82 - 453 mg/dL). It was recommended that the patient receive washed cellular blood components and be premedicated with antihistamines and steroids for future transfusions. The patient subsequently underwent EGD and was found to have a large ulcerated jejunal mass proven to be a large B-cell lymphoma.

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MIMSR/YCRH/Surg./ 15 /2020. Date : 13.02.2020

MIMSR Medical College & YCR Hospital, Latur.

Department of Surgery

To,
The HOD
Department of Pathology
MIMSR Medical College,Latur.

Subject :- About interdepartmental meeting & Clinico –Pathological conference.

Respected Madam

As per University Curriculum we need to arrange CPC and Interdepartmental Meeting.

In CPC Meeting PG Students will present cases by rotation. If cases are not available due to lack of clinical postmortems, it could be supplemented by published CPC's

In Interdepartmental Meeting interesting cases may be chosen and presented by the PG's and discussed by them as well as the senior staff of surgery department. The staff of pathology department would then show the slides and present final diagnosis. In this session the advanced IHC techniques, the burgeoning markers other recent developments can be discussed.

We will conduct this meeting on second Wednesday of every month. Time 04 To 05 pm.

For Records.

29/2/2020

Prof. & HOD
Department of Surgery
Professor & HOD
Dept. of Surgery
M.I.M.S.R. Medical College,
LATUR-413 531

COPY TO

* Dean office

*

M.I.M.S.R.,M.C.LATUR	
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Dean/Principal	
Registrar	
C.A.F.O.	
O.S.	
H.R.	
Section	Pathology
I.W.No.	128

13.02.2020

Keep Record in Dept. of Patho

weightage for internal assessment (See Checklist II of Internal Assessment). A timetable for the subject with names of the student and the moderator should be scheduled at the beginning of every year.

4)Student Symposium: Recommended as an optional multi disciplinary programme. The evaluation may be similar to that described for subject seminar.

5)Ward Rounds: Ward rounds may be service or teaching rounds.

a)Service Rounds: Postgraduate students and Interns should do ward rounds every day for the care of the patients. Newly admitted patients should be worked up by the PGs and presented to the seniors the following day.

b)Teaching Rounds: Every unit should have 'grand rounds' for teaching purpose. A diary should be maintained for day to day activities by the students.

Entries of (a) and (b) should be made in the Log book.

6)Clinico-Pathological Conference: Recommended once a month for all post graduate students. Presentation be done by rotation. If cases are not available due to lack of clinical postmortems, it could be supplemented by published CPCs.

7)Inter Departmental Meetings: Strongly recommended particularly with departments of Pathology and Radio-Diagnosis at least once a week. These meetings should be attended by post graduate students and relevant entries must be made in the Log Book.

Pathology: A dozen interesting cases may be chosen and presented by the post graduate students and discussed by them as well as the senior staff of Surgery department. The staff of Pathology department would then show the slides and present final diagnosis. In these sessions the advance immuno-histochemical techniques, the burgeoning markers other recent developments can be discussed.

Radio-diagnosis: Interesting cases and the imaging modalities should be discussed.

8)Teaching Skills:: Post graduate students must teach under graduate students (Eg. medical, nursing) by taking demonstrations, bed side clinics, tutorials, lectures etc.

Assessment is made using a checklist by surgery faculty as well students. Record of their participation be kept in Log book. Training of post graduate students in Educational Science and Technology is recommended.

9)**Continuing Medical Education Programmes** (CME) : At least 2 state level CME programmes should be attended by each student in 3years.

10)**Conferences:** Attending conferences is optional. However it should be encouraged.

11)**Dissertation** Every candidate pursuing MS degree course is required to carry out work on a selected research project under the guidance of a recognized post graduate teacher. The results of such a work shall be submitted in the form of a dissertation.

MIMSR MEDICAL COLLEGE, LATUR.

Department of pathology

C.P.C.

CIRCULAR

Date :- 8/2/2020

Sr. No.	Name of Department	Receivers Sign
37	Principal	
38	Anatomy	
39	Physiology	
40	Biochemistry	
41	Microbiology	
42	Pharmacology	
43	FMT	
44	PSM	
45	ENT	
46	Ophthalmology	
47	OBGY	12/5/20.
48	Pediatrics	
49	Orthopedic	
50	Surgery	
51	Medicine	
52	Skin & VD	
53	Radiology	
54	PSY	
19	Anesthesia	

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CLINICAL HISTORY

A 21-year-old female with a history of elevated blood pressure as a child and right nephrectomy for pyelonephritis. In 2003, she underwent an excision of a bladder mass.

PATHOLOGICAL FINDINGS

Gross examination revealed tan-brown mass 4.5 cm in greatest dimension with a central 2.5 cm wide and 0.5 cm deep ulcer. The histologic sections showed large, epithelioid cells in a nested pattern separated by a delicate network of fibrous stroma and vasculature. The tumor invaded into the deep muscularis propria with negative surgical margins. No angiolymphatic invasion, mitotic figures, or areas of necrosis were identified. One of eight lymph nodes was found to contain metastatic carcinoma, replacing the native tissue.

The neoplastic cells show strong, diffuse expression of synaptophysin and chromogranin and no expression of pankeratin. The sustentacular cells are highlighted with a S100 stain.

H&E sections of bladder mass.

H&E sections of the lymph node

Selected images of the immunohistochemistry performed of the lesion.

Table 2 Summary of Molecular Genetic Testing(Adapted from Gene Reviews[4])			
Gene (Syndrome)	**Proportion of Hereditary PGL/PCC Attributed to Mutations in this Gene**	**Molecular Testing Method**	**Mutations Detected**
SDHA (PGL5)	0.6-3%	Sequence analysis	Sequence variants
SDHB (PGL4)	22%-38% 12-20% of skull base and neck PGL 24%-44% of chest, abdomen, pelvic PGL/PCC	Sequence analysis Deletion/duplication	Sequence variants Partial and whole gene deletions
SDHC (PGL3)	4%-8%	Sequence analysis Deletion/duplication	Sequence variants Partial and whole gene deletions
SDHD (PGL1)	30% 40%-50% of skull base and neck PGL 15% of chest, abdomen, pelvic PGL/PCC	Sequence analysis Deletion/duplication	Sequence variants Partial and whole gene deletions
SDHAF2 (PGL2)	Unknown	Sequence analysis	Sequence variants

After further testing, this patient was found to have a mutation in the SDH gene with a B subunit mutation (SDHB). The SDHB mutation is an autosomal dominant mutation with approximately one-third of the patients having a family history. They are associated with extra-adrenal sympathetic paragangliomas. The risk of malignancy is 34-97% and is the highest among the SDH mutations. Treatment should include prompt resection of the neoplasm to remove excess catecholamine secretion and as a result of their tendency to metastasize.

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No.MIMSR/Patho/CPC/1731/ /2020 **Date :- 13/6/2020**

It is to inform you that, C.P.C. meeting will be held on 16/6/2020 at 3.00 pm in Pathology lecturer hall, copy of case history (Clinical Protocol is attached herewith.

Kindly, circulate this protocol to the all Teacher's, P.G. Student's Interns & Students of your department.

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In-charge
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Dr. A.S.Acharya

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CLINICAL HISTORY

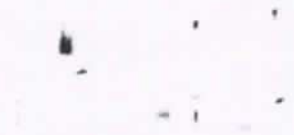
An 80-year old male with a past medical history of dyslipidemia, hypertension, gout and prostate cancer was evaluated for peripheral blood lymphocytosis.

The complete hemogram (Fig. 1) and peripheral blood smear (Fig. 2) demonstrated absolute lymphocytosis, including many large granular lymphocytes (Fig. 3), some intermediate in size with abnormal nuclear contours. Based on a 100-cell lymphocyte count, 66% of all lymphocytes were large granular lymphocytes. Absolute large granular lymphocyte count = 6,461 per microliter.

A bone marrow aspirate showed hypercellular marrow with a normal differential (Figs. 4 and 5) on biopsy and touch imprints (Fig. 6) showed an increased number of large granular lymphocytes.

Bone marrow biopsy (Fig. 7) and particle preparation (Fig. 8) showed 30-40% cellularity with a diffuse increase in small lymphocytes that focally form small, ill-defined aggregates, with a normal M:E ratio, complete maturation in all lineages and adequate megakaryocytes

Immunohistochemical stains performed on bone marrow biopsy showed an increase in CD3, CD2, CD5, CD7 and CD8 positive T cells forming ill defined aggregates and also a few scattered CD56, CD57, TIA1 & Granzyme B positive cells (Figs. 9 and 10)



Flow cytometry studies (Fig. 11) performed on the bone marrow demonstrated 46% bright CD45+ (lymphocyte) events and 3% CD14+ monocytes. T and NK cell marker analysis

marrow, liver, and spleen. Rheumatoid arthritis, the presence of autoantibodies, circulating immune complexes and hypergammaglobulinemia are commonly associated, especially in cases of T-cell origin.

The predominant lymphocytes in PB and BM films are LGL with moderate to abundant cytoplasm and fine or coarse azurophilic granules. The granules in the LGL often exhibit a characteristic ultrastructural appearance described as parallel tubular arrays and contain a number of proteins that play a role in cell mediated cytotoxicity such as perforin, TIA-1 and granzyme B, as well as a number of serine esterases and granzyme M.

Proliferations of large granular lymphocytes are a relatively frequent finding in peripheral blood samples (6), including viral infections, autoimmune disorders, and following bone marrow transplantation and chemotherapy. The problem of differentiating T-LGL leukemia from a benign large granular lymphocytic proliferation is difficult based just on the morphology of the lymphocytes, since they do not often have distinguishing cytologic features. The extent of BM involvement is variable and LGL usually comprise less than 50% of the cellular elements with interstitial/intrasinusoidal infiltrates which are difficult to identify by morphologic review.

T-LGL leukemia is typically a disorder of mature CD3, CD8 and T-cell receptor aß positive cytotoxic T cells, hence immunohistochemistry helps in highlighting interstitial clusters of LGL cells that are positive for CD8, CD 57, CD 3, TIA-1 and granzyme B (5). However, the presence of a small clonal T- large granular lymphocytes is not synonymous with T-cell malignancy, since minor T-cell clones can be found in oligoclonal immune reactions, especially in the elderly and immunosuppressed individuals Therefore other criteria such as an increase in blood LGL population, demonstration of T-cell clonality, and demonstration of a distinct peripheral blood T-cell population by flow cytometry are needed (4, 7, 8).

The above case highlights some of the distinctive characteristics and difficulties associated with the diagnosis of T-LGL leukemia. In our patient , there was lymphocytosis with absolute LGL count of 6.46 X 10^9 /L along with immunohistochemistry on the bone marrow biopsy demonstrating clusters of CD 3, CD8, CD57, TIA-1 and Granzyme B positive cells and flow cytometry supporting these findings. Since similar populations of NK like T-cells may also be seen in normal individuals and in certain reactive situations, T-cell clonality studies may be critical in estabilishing the diagnosis, as illustrated here. However, T-cell clonality alone should not be used as a sole diagnostic criterion, since small clonal populations may also be seen in certain non-neoplastic settings.

It also highlights the fact that it is important to examine the peripheral blood smear where it is frequently possible to pick up the large granular cells and a diagnosis of a large granular lymphocyte leukemia can be considered, which might otherwise be overlooked in a bone marrow biopsy specimen.

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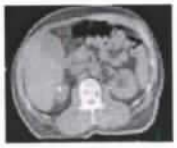
PATIENT HISTORY

A 66 year old married, Caucasian male with a history of HIV/AIDS presents to the emergency department with a chief complaint of back pain for the past 3 weeks and hemoptysis for the past few days. The pain is sharp, in the left side of his lower back, and does not radiate or cause weakness or numbness. The pain is exacerbated with movement and minimally relieved with pain medication. The patient denies fevers, night sweats, and weight loss. His HIV is managed with Atripla. His HIV RNA levels are undetectable. His family history is significant for his mother having pancreatic cancer, his father having prostate cancer, and his brother acquiring tuberculosis at the age of 14. The patient is a retired maintenance man and lives at home. He has a 15 pack year smoking history. He denies alcohol and intravenous drug use. The physical exam is significant for tachycardia and tenderness over the left rib cage and flank. A straight leg test is positive at 30 degrees, reproducing the patient's flank pain.

IMAGING

A CT scan of the chest, abdomen (Figure 1), and pelvis reveal lytic lesions involving the vertebrae, bilateral iliac wings, and sternum. There is a non-displaced fracture of the left posterior tenth rib. A CT scan of the head reveals lytic lesions of the calvarium.



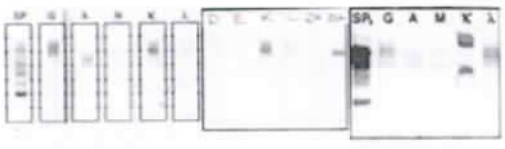
LABS

Table 1 shows baseline lab values. The patient is anemic, thrombocytopenic, and has renal insufficiency. His lactate dehydrogenase is also elevated. Immunology studies reveal decreased levels of IgG and IgM as well as increased β2 microglobulin levels and elevated free Kappa (κ) light chain levels. Free Lambda (λ) light chain levels are within the normal range.

Table 1:

Chemistry	
Patient Values	**Normal Values**
WBC: 4.6 x 10^3 /L	3.8 – 10.6 x 10^3/L
Hgb: 6.7 g/dL	12.9 – 16.9 g/dL (male)
Hct: 19.3%	38.0 – 48.8%
Platelets: 45 x 10^3/L	156 – 369 x 10^3/L
Sodium: 137 mmol/L	136 – 146 mmol/L
Potassium: 4.4 mmol/L	3.5 – 5.0 mmol/L
Bicarbonate: 24 mmol/L	21 – 31 mmol/L
Calcium: 9.4 mg/dL	8.4 – 10.5 mg/dL
BUN: 42 mg/dL	8.0 – 26 mg/dL
Creatinine: 3.7mg/dL	0.5 – 1.4 mg/dL
Lactate Dehydrogenase: 185	< 171
Immunology	
Patient Values	**Normal Values**
IgA: 169	82 – 453
IgG: 701	751 - 1560
IgM: 22	40 - 274
β2 Microglobulin: 8.47 mg/L	< 2.70 mg/L
Free κ: 5600 mg/L	3.3 – 19.4 mg/L
Free λ: 19.1 mg/L	5.7 – 26.3 mg/L
κ / λ (calculated): 293.19	0.26 – 1.65

Figure 2 shows the serum protein electrophoresis results along with immunofixation. Serum protein electrophoresis shows a definitive band in the gamma region that corresponds with the band in the κ column on immunofixation. There are no bands within the IgG, IgA, or IgM immunofixation columns. These results rule out IgGκ, IgAκ, and IgMκ. IgE and IgD are not routinely tested for in our labs, but when a κ band without an associated heavy chain is seen, IgD and IgE immunofixation is performed to test for IgDκ and IgEκ. Immunofixation results for IgDκ and IgEκ are exhibited in Figure 3. A definitive κ band is present. There are no bands in the IgD or IgE columns, ruling out IgDκ and IgEκ. These results suggest free κ chains in the blood. Urine protein electrophoresis and immunofixation results are shown in Figure 4. Urine protein electrophoresis shows large amounts of protein in the urine, with a majority of proteins lying in the gamma region of the gel. κ bands are present, signifying free κ chains in the urine. Serum free κ and λ levels are quantified, and a κ /λ) ratio is calculated, yielding a value of 293.19 (Normal range: 0.26 - 1.65).



A bone marrow biopsy is performed and reveals reduced trilineage hematopoiesis and hypercellular bone marrow involved by neoplasm. Kappa-restricted plasma cells comprise approximately 90% of cells.

Final Diagnosis -- Multiple Myeloma

FINAL DIAGNOSIS

Multiple Myeloma

DISCUSSION

Multiple myeloma is a bone marrow plasma cell neoplasm that has an associated M-protein in serum and/or urine, and has end organ damage characterized by hypercalcemia, renal insufficiency, anemia, and / or lytic lesions of the bone. A bone marrow biopsy with evidence of clonal plasma cells is required for diagnosis. Multiple myeloma accounts for 1% of all malignant tumors and 10-15% of hematopoietic neoplasms. Ninety percent of cases occur in adults over the age of 50 with a median age of 70[1]. HIV positive patients with multiple myeloma tend to be younger (approximately 33 years of age), and there is a 4.5 fold increased risk of developing multiple myeloma. The propensity for HIV patients to develop myeloma may be related to immune dysregulation, elevated serum interleukin-6 levels, chronic antigenic stimulation from infections including HIV, disorganization of the bone marrow microenvironment, and uncontrolled proliferation of Epstein-Barr virus-driven infected B lymphocytes. HIV patients tend to develop aggressive plasma cell tumors, large malignant effusions, hyperviscosity syndrome, and multiple extramedullary plasmacytomas[7]. This patient presented with lytic lesions to the spine, sternum, pelvis and calvarium. He was normocalcemic but had anemia and renal insufficiency as demonstrated by his elevated blood urea nitrogen and creatinine levels. These findings along with a bone marrow biopsy of Kappa-restricted plasma cells comprising 90% of the specimen, strongly suggest the diagnosis of multiple myeloma is well founded.

Most cases of multiple myeloma are monoclonal IgG (50%). IgA monoclonal gammaglobulins account for 20%. Light chains account for 20% of cases, and monoclonal IgD, IgE, IgM, or biclonal gammaglobulins account for less than 10% of cases; non-secretory cases account for 3%[1]. Sixty to seventy percent of multiple myeloma patients have both free light chains and a serum monoclonal (M) protein[12]. The identification of κ bands in serum protein electrophoresis and immunofixation makes this case an example of a light chain-only multiple myeloma. The combination of serum protein electrophoresis and immunofixation studies makes the detection of M protein sensitivity equal to approximately 97%. Serum protein electrophoresis has a sensitivity of only 82% while serum immunofixation alone is 93% sensitive[2]. This patient's serum protein electrophoresis identified a monoclonal protein in the gamma region. To identify the κ clonality of the gammaglobulin, immunofixation was needed.

The International Myeloma Working Group identified several uses for serum free light chain analysis in multiple myeloma. When combined with serum protein electrophoresis and serum immunofixation, serum free light chain assay is sufficient to screen for most pathological monoclonal plasmaproliferative disorders. The serum free light chain assay also plays an important role in prognosis[3]. Van Rhee and colleagues showed that patients

with serum free light chain values greater than 75 mg/dL at baseline had inferior 24-month overall survival and event free survival than patients with lower levels of serum free light chains[13].

The free light chain ratio (κ/λ) is a calculation to determine if there is a monoclonal proliferation of plasma cells producing free κ or λ light chains. Normal κ /λ ratio values range from 0.26 to 1.65. Any value above 1.65 indicates increased production of λ free light chains while values below 0.26 indicate a proliferation of ? free light chains[3,5]. Kyrtsonis and colleagues demonstrated that elevated κ /λ or λ/κ values were associated with a poor prognosis. In their study, baseline serum free light chain ratio values correlated with creatinine, lactate dehydrogenase and the percentage of bone marrow infiltration. Their work also displayed improved three and five year survival rates among patients with low serum free light chain ratios (94% and 82% respectively) when compared to individuals with high baseline serum free light chain ratios (58% and 30%)4. This patient's κ /λ was 293.19, which indicated a proliferation of free κ light chains. In healthy individuals, free light chains are cleared rapidly through the renal glomeruli and are metabolized in the proximal tubules; little protein escapes into the urine. Large quantities of free light chains are required to overcome the kidney's ability to metabolize approximately 10 to 30 g of free light chain daily[3]. Table 1 shows that the serum free κ level in our patient was 5600 mg/L, which was greater than 200x the normal limit. These levels overwhelmed the kidney, causing free κ light chains to spill into the urine as seen in Figure 4.

Other factors that portend a poorer prognosis include light chain multiple myeloma, a creatinine ≥ 2 mg/dL, a C-reactive protein (CRP) ≥ 4 mg/L, an elevated lactate dehydrogenase, β2 microglobulin ≥ 5.5 mg/L, a bone marrow infiltration of ≥50%, spontaneous bone fractures, a hemoglobin less than 10 g/dL, and a platelet count less than 100 x 10^9/L[4]. Unfortunately our patient displayed almost all of these criteria (CRP was not reported), and therefore will more likely have a poorer outcome.

Patients can also be stratified into standard risk and high risk categories as done by Rajkumar and colleagues. High risk patients have any one of the following features: deletion 13 or hypodiploidy on metaphase cytogenetic studies, deletion 17p- or immunoglobulin heavy chain (IgH) translocations t(4;14) or t(14;16), or plasma cell labeling index of 3% or higher. High risk patients have lower survival (2-3 years) versus average risk multiple myeloma patients, who survive 6-7 years[2]. Therefore, fluorescence in situ hybridization (FISH) and/or cytogenetic testing should be performed in all patients[10]. FISH and cytogenetics testing on this patient were pending at the time of writing.

In young patients with multiple myeloma the standard of care is high dose Melphalan followed by autologous stem cell transplant. Older patients receive oral Melphalan. Newer treatments such as Bortezomib can be considered in high risk patients[2]. Optimum treatment for the treatment of multiple myeloma in HIV patients has not been well described[6]. Dezube et al reviewed the outcomes of HIV-related plasma cell dyscrasias. Their paper showed varying results. In terms of survival, one of the better results was a 45 year old male treated with radiotherapy, Vincristine, Adriamycin, and Dexamethasone (VAD), who had a relapse of myeloma at 6 years [6,7]. Another myeloma case died at 1 week after treatment with Melphalan and Prednisone [6,8]. One patient had undergone an autologous stem cell transplantation and VAD but died 13 months later [6,9]. The authors

made two important points. The first was the importance of highly active anti-retroviral therapy (HAART). Patients with HIV and multiple myeloma, who were on HAART therapy lived at least one year. The second important point was the properties of Thalidomide and their implications for treatment of HIV multiple myeloma patients. Thalidomide is non-myelosuppressive, stimulates CD4 and CD8 activity, directly inhibits HIV replication, and does not affect the pharmacokinetics of HAART[6].

In summary, multiple myeloma is a plasma cell neoplasm that arises in the bone marrow and causes end organ damage to patients. HIV patients are more likely to develop multiple myeloma and tend to be younger than non-HIV multiple myeloma patients. It is important to thoroughly work up patients with multiple myeloma. Work up includes serum protein electrophoresis, serum protein immunofixation, serum free light chain levels and κ/λ ratio, and FISH with cytogenetics. The prognosis is determined by serum free light chain levels, FISH and cytogenetics results, physical signs, and multiple lab values. Standard of care treatment is Melphalan with or without autologous stem cell transplant. Treatment of multiple myeloma in HIV patients is less clear. Thalidomide has implications in treatment due to its prohibition of viral replication, increased CD4 and CD8 activity, and no interference with concurrent HAART therapy.

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CLINICAL PRESENTATION

The patient is a 3 year old male who initially presented with seizures at age 18 months and was diagnosed with a Chiari malformation; this was successfully surgically repaired. During the pre-operative assessment, however, he was found to have isolated hepatomegaly. Since that time, his parents have noted increasing fatigue, leg pain, intermittent abdominal pain, and a possible two pound weight loss. Repeated blood sugar measurements revealed glucose levels in the 30s and 40s in the morning and early afternoon. He has no history of jaundice, or pruritus. There is no family history of similar symptoms (see Figure 1).

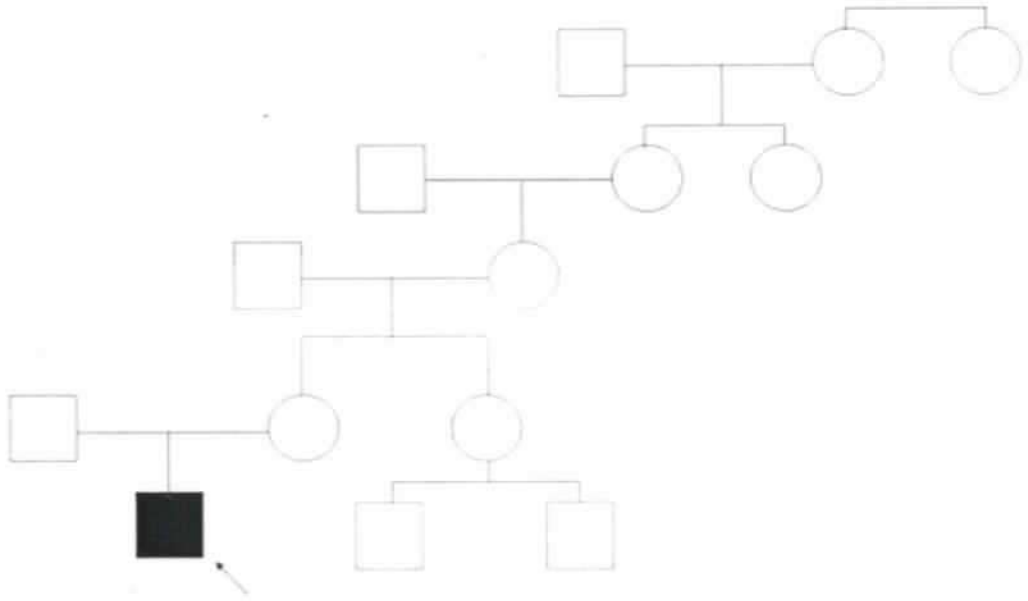

Figure 1: Pedigree

Physical exam was significant for hepatomegaly, with the liver edge palpable 3 cm below the costal margin. There is no splenomegaly, ascites, peripheral edema, or cutaneous evidence of chronic liver disease. Physical exam was otherwise unremarkable.

Laboratory studies were as follows:

AST	50 IU/L
ALT	30 IU/L
GGT	16 IU/L
Alkaline Phosphatase	221 IU/L (H)
Albumin	4.5 g/dL
Bilirubin	0.2 mg/dL
PT	15.9 sec
ESR	33 mm/hr (H)
Alpha-1-anti-trypsin	103 mg/dL
Hepatitis C RNA	Negative
Anti-smooth muscle antibody	Negative
Liver, Kidney Microsomal Antibody	Negative
Glycogen Storage Disease Type VI	Negative by PCR

Abdominal MRI revealed hepatomegaly with no focal abnormalities.

To further define the pathologic process, a liver biopsy was performed and demonstrated:

HEPATOCELLULAR BALLOONING WITH INTRACYTOPLASMIC LIPID AND GLYCOGEN ACCUMULATION

---- MILD PORTAL AND PERI-PORTAL FIBROSIS

As glycogen storage disease (GSD) type VI was previously excluded, the clinical team requested that we start testing for type IX GSD. Testing for phosphorylase B kinase, alpha subunit (PHKA2), with reflex testing for the beta subunit (PHKB) was requested. 3ml of blood in EDTA was received for testing.

Out of further clinical concern, prior to the return of the PHKA2 results, further molecular testing for GSD III was ordered. Testing for fructose 1,6 bisphosphatase (FBP1) , Aldolase B (ALDOB), and Amylo-alpha-1, 6-glucosidase, 4-alpha-glucanotransferase (AGL) was also performed.

MOLECULAR RESULTS

Final Diagnosis -- Glycogen storage diseases

MOLECULAR RESULTS

PHKA2 Gene sequencing: POSITIVE for a hemizygous deletion in exon 33 of c.3648_3649delAA, which results in a frameshift and the development of a stop codon 26 codons downstream (p.Arg1217SerfsStop26).

AGL Gene Sequencing: Heterozygous Variant of Unknown Significance of c.1481 G>A. This causes a single amino acid substitution (p.Arg494His).

ALDOB sequence and deletion/duplication analysis: NEGATIVE.

FBP1 Sequence Analysis: NEGATIVE.

DISCUSSION

Glycogen storage diseases result from the inability to properly metabolize glycogen. Glycogen is a carbohydrate which is able to be stored and easily mobilized to maintain appropriate blood sugar levels during periods of fasting. This molecule is most abundant in the liver and skeletal muscle; this localization contributes to many of the observed clinical symptoms. Symptoms of glycogen storage diseases include muscle pain, cramps, exercise intolerance, and easy fatigability. Signs of fasting hypoglycemia, ketosis, and poor weight gain, with or without hepatomegaly, can also be seen[1]. Glycogen storage diseases have a

prevalence of approximately 1/10,000, although their frequency is believed to be underestimated due to the lack of efficient molecular methods[2].

There are numerous pathways involved in glycogen metabolism, and defects in any of them can cause different types of glycogen storage disease, as demonstrated in this graphic (Figure 2):

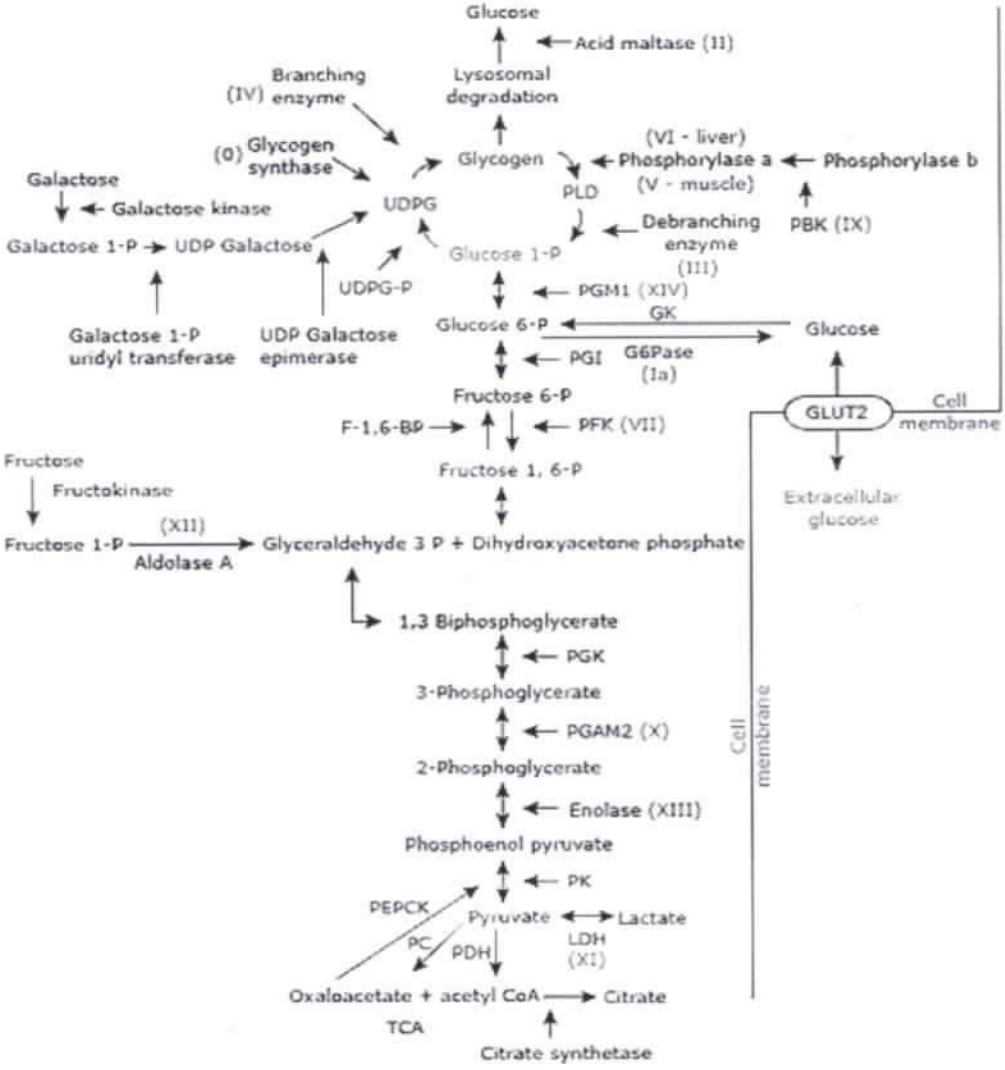

Figure 2: *Adapted from: Griggs R, Mendell J, Miller R. Metabolic myopathies. In: Evaluation and Treatme Griggs R, Mendell J, Miller R (Eds), FA Davis Co., Philadelphia 1995. p.247.*[1]

The mutation seen in the presented case involves a two nucleotide deletion in the PHKA2 gene. This gene encodes for the alpha subunit of the liver isozyme of phosphorylase kinase B[3]. The phosphorylase kinase (PHK) enzyme is important in the breakdown of glycogen to Glucose-1-P and dysfunction can result in GSD Type IX. PHKA2 is encoded on the X-chromosome, and as such GSD-IX is also referred to as X-linked glycogenosis (XLD)[4, 5]. The PHKA2 enzyme consists of 4 subunits, and the alpha subunit is a 138kDa protein that has an inhibitory role within the PHK complex[6, 7]. This gene is usually the first sequenced when there is concern for GSD-IX, as it is dysfunctional in 75% of cases[4].

Patients with type IX GSD often have excessive glycogen in the liver and can present with short stature, hepatomegaly, and sometimes ketotic hypoglycemia[4]. They often show signs of transaminase and triglyceride elevation, but cirrhosis is very rare[4]. Enzymatic studies assaying for the activity of the PHKA enzyme can be performed in vitro, however these often show negative results even when the in vivo enzyme is dysfunctional[4, 6, 8]. Molecular studies are ordered to rule out this disease and to prevent more invasive studies (such as liver biopsy) [6, 7, 8, 9]. This case demonstrates a deletion of two adenine nucleotides at position 3648-3649 of exon 33 in the PHKA2 gene, which results in an amino acid change and subsequent frameshift. In this patient, the frameshift actually results in a longer protein, with a carboxy terminal sequence change as follows (the altered amino acid sequence is in bold):

Wild Type (1235 AA)	TMTYLTRAVA SYLQELLPNS GCQMQ
Mutant (1241 AA)	TMTYLTSSGF LPAGIVAQFG LPDAIGSHLE T

This amino acid sequence is obviously significantly altered from the above wild type, and the function is likely to be impacted by these changes.

Most frameshifts result in the formation of an early stop codon, and subsequent truncation of the protein. Truncated proteins tend to get degraded within the cell, and thus result in loss of enzymatic activity in vivo[6, 7]. This specific microdeletion (and subsequent frameshift) has been reported in a patient from Poland, and resulted in mild fasting intolerance in addition to hepatomegaly[10]. As most PHKA2 gene mutations are found in specific families, with few uniformly present in populations, it is relatively surprising that this microdeletion has been previously reported[8]. Also of note, as demonstrated by the pedigree on the prior screen, there have been very few males born in this family. As this mutation is X-linked, and since there are no de novo mutations reported in the literature, it seems likely that this mutation has been carried in the females in this family over a long period of time before revealing itself in this patient.

The second variant identified in this patient, c.1481G>A of the AGL gene, results in an Arginine to Histidine amino acid change at position 494. The AGL gene encodes for the glycogen debranching enzyme (GDE), which is required for glycogen breakdown and subsequent phosphorylation[11, 12]. Partial or total lack of this enzyme results in GSD Type III, which is autosomal recessive, and felt to be rare[11]. In patients lacking this enzyme, glycogen accumulates in the liver, heart, and muscle11. Clinical manifestations include hepatomegaly, hypoglycemia, short stature, and sometimes myopathy and cardiomyopathy[12]. Mutations in AGL are variable, and many are confined to single families[9]. This protein change is predicted to be "probably damaging" by the PolyPhen-2 protein prediction software, with the SIFT software program predicting this variant as "not tolerated." This variant has also been previously identified, but only in the context of a patient with two other known damaging mutations[13]. Thus, the clinical significance of this variant is unclear. Given the patient's more mild clinical presentation, it is likely that this variant is not significantly contributing to his disease.

This case demonstrates the difficulty in interpreting multiple genetic variants in the same patient, especially when these genetic changes involve similar pathways and present with similar symptoms. Performing reflex testing to ensure that only the appropriate testing is performed, in an order agreed upon by the clinical team, may help alleviate some of these

redundant and potentially confusing results. However, given the future of personalized medicine, multiple variants of unclear significance may be revealed in patients, even if other pathogenic mutations have already been identified. Thus, the interpretation of these results will likely continue to prove difficult, with further genomic study hopefully helping to identify rare benign SNPs as opposed to truly pathogenic alleles.

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It is to inform you that, C.P.C. meeting will be held on 13/04/2021 at 3.00 pm in Pathology lecturer hall, copy of case history (Clinical Protocol is attached herewith.

Kindly, circulate this protocol to the all Teacher's, P.G. Student's Interns & Students of your department.

Thanking you,

In-charge
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Dr. J.B.Patil

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PATIENT HISTORY

The patient is a 55-year-old man who presented to the ED with gradual worsening of weakness. The patient reports that he had been fatigued for one month, having to rest constantly after work, and feeling exhausted with minimal exertion. He also reported feeling lightheaded with rising, as well as having epigastric abdominal pain, which was not associated with food. Four days prior to the presentation, the patient began to have black, tarry stools. He was evaluated in his primary care physician's office the day prior, and was found to have hemoglobin of 6.5 g/dL. His prior baseline from two weeks ago was 15.3 g/dL. He was subsequently referred to the ED.

The patient has a history of pancolonic ulcerative colitis diagnosed in 1979 and now in remission. He has not been on medication for 17 years, but he did receive treatment with prednisone around time of diagnosis, then sulfasalazine. He had undergone recent surveillance EGD and colonoscopy a month ago, which was unchanged from previous studied. He also has gastroparesis, not symptomatic for several years. He is on ASA 81 mg. He takes a PPI for GERD which is well-controlled.

In the ED, his vital signs were: pulse: 121, blood pressure: 127/85, respiratory rate: 20, and oxygen saturation: 99% on room air. Physical examination was remarkable for generalized pallor, mild tachycardia, and diastasis of rectus (also known as abdominal separation). Nasogastric lavage was negative, but the fetal occult blood test was positive. Pertaining labs are listed below:

	ED Labs	Reference Range
Hematology:		
WBC	9.4	3.8-10.8 THOUS/MCL
RBC	3.07	4.20-5.80 MILL/MCL
Hgb	6.9	13.2-17.1 G/DL
HCT	22.4	38.5-50.0 %
MCV	73.1	80.0-100.0 FL
MCH	22.6	27.0-33.0 PG
MCHC	30.9	32.0-36.0 G/DL
RDW	21.1	11.0-15.0 %
Chemistry:		
Iron	<10	45-182 ug/dL
Total Iron Binding Capacity	298	250-420 ug/dL
Ferritin	63	10-282 ng/mL

Following examination and GI consult, he was admitted on to the floor for further work up of his GI bleeding. Packed red blood cells were ordered for transfusion with goal hemoglobin of greater than 8 g/dL. Seventy milliliters within the transfusion of the first packed red blood cell, the patient became hypotensive and diaphoretic with chill/rigor and dyspnea. Pre and post vital signs were:

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It is to inform you that, C.P.C. meeting will be held on 08/06/2021 at 3.00 pm in Pathology lecturer hall, copy of case history (Clinical Protocol is attached herewith.

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PATIENT HISTORY

CLINICAL HISTORY

The patient is a 22 year old Saudi Arabian male, who has been living in the United States for three years. He initially presented 2 days prior to admission to the emergency department of an outside hospital with cough, night sweats, anorexia, severe fatigue, and an unintentional 90 lbs weight loss over the 6 months prior to presentation. He is a previous smoker (1 pack/day for 8 years), but quit several months ago due to his chronic cough.

He denies any sick contacts, fever, or chills. A purified protein derivative (PPD) skin test recently performed at an outside institution was negative.

The patient had lived in Tennessee when he initially arrived in the US, and has lived in Pennsylvania for the last year. Since his move to Pennsylvania, he has travelled to the Middle East twice to visit family. Most recently, he has not left his apartment in two months due to his incapacitating fatigue.

RADIOLOGY FINDINGS

Chest radiography was significant for bilateral reticular infiltrates and bilateral apical cavitary lesions (Figure 1).

Chest computed tomography demonstrates prominent apical cavitary lesions bilaterally (Figures 2 and 3).

HISTOLOGY FINDINGS

Biopsies taken from the right middle lung lobe demonstrate a necrotizing pneumonia (Figure 4), with foci of epithelioid granulomas (Figures 5 and 6). An acid fast stain performed on the biopsy highlights one acid fast bacillus (Figure 7).

LABORATORY RESULTS

An acid fast stain performed on a smear from bronchoalveolar lavage fluid was positive for bacilli, and culture grew organisms identified as *Mycobacterium tuberculosis* complex by DNA probe. A Quantiferon TB Gold test was positive. Urine antigen testing for Histoplasma was negative.

ADDITIONAL HISTORY

The patient was initially placed on a RIPE (Rifampin, Isoniazid, Ethambutol, Pyrazinamide) regimen for treatment as an inpatient. Upon further consideration, the patient and his family decided that he should return home to the Middle East to complete treatment. To allow for sooner travel, rapid sequence-based molecular drug sensitivity testing was requested at CDC. Prior to discharge, however, the patient's liver enzymes increased dramatically (ALT: 429 IU/L, AST: 534 IU/L), and all anti-tubercular therapy was stopped pending the sensitivity results, which were not yet available. He was discharged to home without any medication, as he was living alone at the time.

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CLINICAL HISTORY:

A caucasian man in his 50s presented to the emergency department with increasing exertional dyspnea, generalized weakness, lethargy, and anemia. The patient underwent orthotopic cardiac transplant approximately six months earlier for ischemic cardiomyopathy secondary to a previous myocardial infarction. The anemia was first noted a few weeks postoperatively.

A bleeding duodenal ulcer was discovered approximately one month after the transplant. Endoscopic repair was performed and the patient received antibiotic treatment for Helicobacter pylori. The anemia did not resolve, although repeat endoscopy one month after the repair demonstrated that the ulcer was healing. The continuing anemia was attributed to an elevated tacrolimus (ProGraf®, FK507) level; tacrolimus was subsequently discontinued, and cyclosporin (Neoral®) was then used as the patient's primary immunosuppressant. The medication changes were made approximately one month prior to the presentation at the emergency department.

The patient's medications included cyclosporin (Neoral®), mycophenolate mofetil (CellCept®), valganciclovir (Valcyte®), atorvastatin (Lipitor®), pantoprazole (Protonix®), trimethoprim-sulfamethoxazole, levothyroxine, and prednisone.

PERIPHERAL BLOOD COUNTS AND MORPHOLOGIC FINDINGS:

CBC with differential

	Patient Value	Normal Range
WBC	2.3 x 10^9/L	3.8 – 10.6 x 10^9/L
RBC	2.38 x 10^{12}/L	4.13 – 5.57 x 10^9/L
Hemoglobin	7.4 g/dL	12.9 – 16.9 g/dL
Hematocrit	20.7 %	38.0 – 48 %
MCV	87.1 fL	82.6 – 97.4 fL
MCH	31.1 pg	27.8 – 33.4 pg
MCHC	35.7 g/dL	32.7 – 35.5 g/dL
RDW	13.7 %	11.8 – 15.2 %
Platelets	254 x 10^9/L	156 – 369 x 10^9/L
Reticulocytes	0.008 x 10^{12}/L	0.018 – 0.158 x 10^{12}/L
Reticulocytes	0.3 %	0.8 – 2.0 %

	Patient Value (Percentage)	Patient Value (Absolute)	Normal Range (Absolute)
PMNs	58 %	1.33 x 10^9/L	2.24 – 7.68 x 10^9/L
Bands	1 %	0.02 x 10^9/L	0.10 – 0.80 x 10^9/L
Lymphocytes	23 %	0.53 x 10^9/L	0.80 – 3.65 x 10^9/L
Atypical Lymphocytes	10 %	0.23 x 10^9/L	
Monocytes	6 %	0.14 x 10^9/L	0.30 – 0.90 x 10^9/L
Eosinophils	2 %	0.05 x 10^9/L	0.00 – 0.40 x 10^9/L

Rare plasmacytoid lymphocytes were identified; review of the peripheral blood smear was otherwise unremarkable.

What is your clinical diagnosis ? :-

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C.P.C. PRESENTATION.

Venue :- Pathology Lecturer Hall, Date 08/10/2021, Friday, at 3.00 pm.

The case History :-

60 years old lady presented with colicky abdomen pain of 6 days duration. She was a diabetic on treatment.

An USG & CT done for suspected cholescystitis showed a well defined lobulated mass measuring 7.7X6X5.5cm, involving body & tail of pancreas.

A clinical diagnosis pancreatic Ca. was made & distal pancatectomy with splenectomy was done.

No regional L.N. was noted.

What is your Diagnosis? :

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C.P.C. PRESENTATION.

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What is your Diagnosis? :

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It is to inform you that, C.P.C. meeting will be held on 10/12/2021 at 3.00 pm in Pathology lecturer hall, copy of case history (Clinical Protocol is attached herewith.

Kindly, circulate this protocol to the all Teacher's, P.G. Student's Interns & Students of your department.

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It is to inform you that, C.P.C. meeting will be held on 10/12/2021 at 3.00 pm in Pathology lecturer hall, copy of case history (Clinical Protocol is attached herewith.

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Venue :- Pathology Lecturer Hall, Date 10/12/2021, Friday, at 3.00 pm.

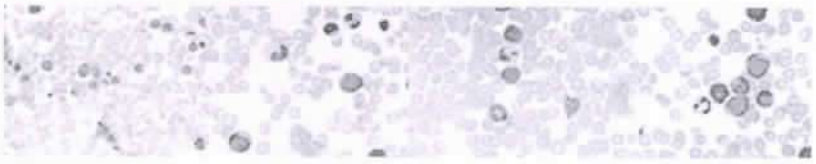
CLINICAL HISTORY: The patient was a 12- day old female with who was diagnosed with Downs Syndrome. She was noted to have mild cyanosis with symptoms of mild hypoxemia. Her initial blood count demonstrated severe anemia and thrombocytopenia. A bone marrow smear and aspirate was subsequently ordered. All her other blood work including coagulation studies were normal.

PERIPHERAL BLOOD COUNTS AND MORPHOLOGIC FINDINGS:

CBC with differential

	Patient Value		Normal Range
Blasts	73%		<5%
WBC	138.9 x 10^9/L		5.0 – 21.0x 10^9/L
RBC	3.18 x 10^{12}/L		4.5 – 9.57 x 10^9/L
Hemoglobin	11.7 g/dL		12.9 – 16.9 g/dL
Hematocrit	33.2 %		37.0 – 42 %
MCV	104.1 fL		88.6 – 126.0 fL
MCH	31.1 pg		27.8 – 33.4 pg
MCHC	35.1g/dL		32.7 – 35.5 g/dL
RDW	18.8 %		11.5 – 15.2 %
Platelets	95x 10^9/L		150 – 450 x 10^9/L
Eosinophils	2 %	0.05 x 10^9/L	0.00 – 0.40 x 10^9/L

PERIPHERAL BLOOD:



As illustrated in Figures 1-4, the peripheral smear demonstrated many blasts with a small amount of blue cytoplasm and prominent vacuoles. The nuclei are enlarged, with clumped chromatin and some folds. There was also some toxic granulation of neutrophils.

BONE MARROW ASPIRATE:

The bone marrow smear showed a cellular aspirate. There were large clusters of cells with enlarged nuclei, clumped chromatin and scant blue cytoplasm similar to the peripheral blood. There were in addition decreased levels of erythroid precursors and an absence of megakaryocytes.

DIAGNOSIS - ?

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Venue :- Pathology Lecturer Hall, Date 07/01/2022, Friday, at 3.00 pm.

Case of an ovarian mass, Pathology,Radiology and Surgery department
Clinical history: Patient 17 year old lady
came to the opd with complains of abdominal pain.
Ultrasonography report: Large pelvic mass anterior to uterus (?) ovarian mass.

CT abdomen and pelvis: Large well defined solid cystic lesion seen in pelvis at midline probably arising from
left ovary of size 12.5* 8 cm with few internal enhancing septa with early loss of surrounding fat planes suggestive of ovarian neoplastic etiology more likely cystadenocarcinoma>cystadenoma.
Mild to moderate free fluid in the abdomen.
HCG Beta Subunit, Serum: Below,1.2 mIU/mL,AFP: 1.15 ng/ml

Gross appearance of specimen
(Left side ovarian mass) received
- Ovarian mass measuring 10*6*5.5cm.
- External surface was smooth bossolated with congestion.

Cut section shows solid and cystic areas. Solid areas are homogeneous and yellow in colour.
Multiloculated cysts are noted.
Cysts were filled with hemorrhagic fluid.

Microscopy
- Tumour composed of cells arranged in diffuse sheets with scattered interspersed follicle also known as 'Call-Exner bodies'

These follicles are of varying size and shapes containing secretions.

Tumour cells have round, oval vesicular nuclei and abundant eosinophilic cytoplasm.

DIAGNOSIS - ?

MIMSR MEDICAL COLLEGE, LATUR.

Department of pathology

C.P.C.

CIRCULAR

Date :- 04/03/2022

Sr. No.	Name of Department	Receivers Sign
1	Principal	
2	Anatomy	
3	Physiology	
4	Biochemistry	
5	Microbiology	
6	Pharmacology	
7	FMT	
8	PSM	
9	ENT	
10	Ophthalmology	
11	OBGY	
12	Pediatrics	
13	Orthopedic	
14	Surgery	
15	Medicine	
16	Skin & VD	
17	Radiology	
18	PSY	
19	Anesthesia	

MIMSR MEDICAL COLLEGE , LATUR.
DEPARTMENT OF PATHOLOGY,

No.MIMSR/Patho/CPC/1731/ /2022 **Date :- 04/03/2022**

It is to inform you that, C.P.C. meeting will be held on 11/03/2022 at 3.00 pm in Pathology lecturer hall, copy of case history (Clinical Protocol is attached herewith.

Kindly, circulate this protocol to the all Teacher's, P.G. Student's Interns & Students of your department.

Thanking you,

In-charge
C.P.C. Activity
Dr. J.B.Patil

H.O.D.
Dept. of Pathology

The Head
Department of Pathology
MIMSR Medical College, LATUR

Copy to :-

1 **Principal,**
2 **All dept.**

MIMSR MEDICAL COLLEGE , LATUR.
DEPARTMENT OF PATHOLOGY,

No.MIMSR/Patho/CPC/1731/ /2022 **Date :- 04/03/2022**

It is to inform you that, C.P.C. meeting will be held on 11/03/2022 at 3.00 pm in Pathology lecturer hall, copy of case history (Clinical Protocol is attached herewith.

Kindly, circulate this protocol to the all Teacher's, P.G. Student's Interns & Students of your department.

Thanking you,

In-charge
C.P.C. Activity
Dr. J.B.Patil

H.O.D.
Dept. of Pathology

The Head
Department of Pathology
MIMSR Medical College, LATUR

Copy to :-

1 **Principal,**

2 **All dept.**

Venue :- Pathology Lecturer Hall, Date 11/03/2022, Friday, at 3.00 pm.

The case History :-

The patient is a 30-year old man with a 30-pound weight loss in one month, poor appetite, and episodes of abdominal and back pain. He has had progressive pancytopenia over 1 month.

Complete Blood Count

	Patient Value	Normal Range (Male)
WBC	3.1 x10E+9/L	3.8 - 10.6
RBC	2.76 x10E+12/L	4.13 - 5.57
Hgb	8.4 g/dl	12.9 - 16.9
Hct	23.9 %	38.0 - 48.8
MCV	86.6 fl	82.6 - 97.4
MCH	30.6 pg	27.8 - 33.4
MCHC	35.3 gm/dl	32.7 - 35.5
RDW	15.4 %	11.8 - 15.2
PLT	18 x10E+9/L	156 - 369

Peripheral Blood Differential

Cell type	Percentage	Abs. No.	Normal Range
POLYS	10 %	0.31	2.24 - 7.68
BANDs	8 %	0.25	0.10 – 0.80
LYMPHS	70 %	2.17	0.80 - 3.65
ATYPICAL LYMPHS	1 %	0.03	0.03
MONOS	5 %	0.16	0.30 – 0.90
BLASTS	5 %	0.16	
META	1 %	0.03	

What is your clinical diagnosis ? :-


बैंक ऑफ़ इंडिया
Bank of India


ग्रामीण दंत रुग्णालय
MAEER'S PUNE
Maharashtra Institute of Dental
Sciences and Research Centre, Latur
WORLD
AIDS
DAY


MIT HEALTH CAMPUS, LATUR
INTERNATIONAL


INTERNATIONAL
Women Day
2020
Dr. Vishwanath D. Karad


MIMSR
MIMSR Medical College, MIDSR Dental College
International Women Day - 2021


Alice


राष्ट्रीय कुष्ठरोग निर्मुलन कार्यक्रम


HAPPY TEACHER'S DAY!
MIMSR MEDICAL COLLEGE, LATUR


HERPER


GUYTON
08


BLOOD DONATION
CAMP


"Workshop on Basic Surgical Skills"


त्वचा कोरडी ठेवा
ZOCON
Fungal Skin Infections


त्वचा कोरडी ठेवा
4
SKIN


PEDIATRICS
8
9
बालरोग विभाग
"जागतिक
स्तनपान सप्ताह"


PEDIATRICS
8
9


MIMSR MEDICAL COLLEGE &
YCR HOSPITAL, LATUR
CME on
MEASLES & RUBELLA
VACCINATION CAMPAIGN
organised jointly by
Dept. of Community Medicine & Dept. of Pediatrics


माईर पुणेचे
एम.आय.एम.एस.आर. वैद्यकीय महाविद्यालय
श्रीमती सरस्वती कराड रक्तपेढी, लातूर
भव्य रक्तदान शिबीर
स्थळ : श्री सरस्वती कराड रक्तपेढी, लातूर
दिनांक : २८/०१/२०१८ गुरुवार
वेळ : स. ९ ते ३


MAEER MIT WORLD PEACE SOCIETY, PUNE'S
MIMSR MEDICAL COLLEGE, &
Y.C.R. HOSPITAL, LATUR
MIMSR
Department Of Medical Education & Department Of Anatomy
Date : 29/09/2018


224
149


WEL-COME
GUEST LECTURE


Cavernous Sinus
SPANDAN 2K16


GLUCONEOGENESIS
THE CORI CYCLE
BLOOD


POSTER PRESENTATION

MIMSR MEDICAL COLLEGE, LATUR
DEPARTMENT OF BIOCHEMISTRY
Poster Presentation

Date:- 17/01/2020

Activity :- SDL (Self Directed Learning)
Date:- 31/01/2020, **Time :-** 11 am to 01 pm
Venue:- Dept. of Biochemistry

Sr. No	Topic Name	Group Name
1	Glycogen Metabolism, Regulation	Group **F** Roll No .(51 to 60)
2.	HMP Shunt & its Significance	Group **M** Roll No (121 to 130)
3.	Vitamin D	Group **J** Roll No (91 to 100)
4.	ETC & Oxidative Phosphorylation	Group **N** Roll No (131 to 140)
5.	Comptitive Inhibition	Group **I** Roll No (81 to 90)
6.	DNA Replication	Group **B** Roll No (17 to 20)
7.	Transcripition	Group **L** Roll No (111 to 120)
8.	Vitamin C	Group **O** Roll No (141 to 150)
9.	rDNA Technology	Group **C** Roll No (21 to 30)
10.	Blotting Technics	Group **H** Roll No (71 to 80)
11.	Blood Sugar Regulations	Group **E** Roll No (41 to 50)
12.	Gluconeogenesis & Coris cycle	Group **K** Roll No (101 to 110)
13.	Sickle Cell anemia	Group **A** Roll No (01 to 10)
14.	Vitamin A	Group **D** Roll No (31 to 40)
15.	Thallasemia	Group **G** Roll No (61 to 70)

Prof & Head
Dept. of Biochemistry
Professor and Head
Department of Biochemistry
M.I.M.S.R. Medical College,
LATUR - 413 512.

Dean
M.I.M.S.R. Medical College,
& Y.C.R. HOSPITAL,
LATUR - 413 531.


THALESSEMIA
Temperature : 26.01 °C
Accuracy : 29.54
Date : 2020-01-31 12:06:04
longitude : 76.57969976
latitude : 18.4298564


LIBRARY CUM
SEMINAR HALL
Temparature : 26.61 °C
Accuracy: 100.0
time : 2020-01-31 11:38:15
longitude : 76.57750245
latitude : 18.4244341
Vishwanathpuram,Latur,Maharash


Dept. of Biochemistry
GLUCONEOGENESIS
THE CORI CYCLE
BLOOD
Temparature : 26.61°C
Accuracy : 20.798
time : 2020-01-31 11:13:33
longitude : 76.579699
latitude : 18.4298578
Vishwanathpuram,Latur,Maharash


Village Legal Care
and Support Centre
ग्राम कायदेविषयक सेवा
आणि आधार केंद्र
THALESSEMIA
Accuracy : 25.878
time : 2020-01-31 12:14:10
longitude : 76.5797017
latitude : 18.4298691
Vishnuandpuram,Latur,Maharash
Temparature : 26.61 °C


Village Legal Care
and Support Centre
ग्राम कायदेविषयक सेवा
आणि आधार केंद्र
Research Laboratory
THALESSEMIA


Temparature : 26.61 °C
Accuracy : 26.16
time : 2020-01-31 12:45:47
longitude : 76.57796976
latitude : 18.4298594
Vishwanathpuram,Latur,Maharash

MIMSR MEDICAL COLLEGE, LATUR.
DEPARTMENT OF PATHOLOGY

Date :- 22/11/2018

To,

H.O.D.
Department of Pathology,
Govt. Medical College,
Latur.

Sub :- Regarding invitation for CME participation.

Respected Sir,

The department of Pathology, MIMSR Medical College, Latur is organizing a **CME on "Basics in Surgical Pathology" on 27th November 2018**. I, on the behalf of Pathology dept. would like to invite all teaching staff members & P.G. Student's to participate in this CME.

CME Programme:-

Morning session (10.00 am to 1.00pm)

Surgical Pathology grossing techniques – for P.G.Studetns.

Speakers:

1. Dr.Manisha Biradar,Asst.Prof.(GMC,Latur)
2. Dr.J.B.Patil, Asso.Prof.
3. Dr.S.N.Kanthikar,Asso.Prof.
4. Dr.R.B.Badanale,Asst.Prof.
5. Dr.P.B.Chege,Asst.Prof.

Afternoon session (2.30pm to 5.00pm)

Lecture 1 :- **Biopsy - A vision of life**
Dr. N.V. Kulkarni, Professor, Dept. of Surgery

Lecture 2 :- **Challenges in rural Oncopathology practice**
Dr. R.S.Ayachit, Professor, Dept. of Pathology

Note :-
Free Registration.
All teaching staff members are invited for the afternoon sessions,while all the P.G.Students are invited for both the sessions.

Venue: Pathology lecture hall, MIMSR, Latur,1st floor college building.

o/c

Dr. Mrs.S.N.Kulkarni
Prof. & Head
Dept. Of Pathology
MIMSR Medical College,
Latur

The Head
Department of Pathology
MIMSR Medical College, LATUR

(1) Dept of Microbiology

(2) Dept of pharmacology 23/11/18

(3) Dept. of Physiology

(4) Dept. of Anatomy Dr. M.S. Ugale

(5) Dept of FMT V-C Solanki

(6) Dept. of Biochemistry

(7) Dept. of PSM

(8) Dept of Medicine.

9) Dept of skin

10) Dept of OBG

11) Dept of Paeds

12) Dept. of ENT

13) Dept. of ophthalmology -

14) Dept of Ortho

15) Dept of Surgery

16) Dept of TB chest

17) Dept of oral medicine

18) Dept. of oral maxillofacial surgery - Dr Amol Deipatrick 23/11/18.

19) Dept of oral pathology - Dr. Smita Chawane

20) Dental Principal office - 23/11/18. Dr. Vishwanath P.

21) Physiotherapy Office -

22) Matron office -

23) Dept. of Anaesthesia

24) Dept. of Radiology

MIMSR MEDICAL COLLEGE, LATUR.
DEPARTMENT OF PATHOLOGY

Date :- 22/11/2018

All the HOD's, teaching staff members & P.G. Students working in MIMSR Medical College & Y.C.R. Hospital are hereby invited to participate in the **CME Programme on "Basics in Surgical Pathology" on 27th November 2018** in Pathology Lecture hall from 9.00am to 5.00pm.

Morning session :- (10.30 am to 1.30.pm)

Surgical Pathology grossing techniques – For Pathology P.G. Students

Speakers:
1. Dr. Manisha Biradar,Asst.Prof.(GMC,Latur)
2. Dr. J B.Patil, Asso.Prof.
3. Dr. S.N.Kanthikar,Asso.Prof.
4. Dr. R.B.Badanale,Asst.Prof.
5. Dr. P.B.Chege,Asst.Prof.

Afternoon session :- (2.30 to 5.00pm.)

Lecture 1 :- **Biopsy - A vision of life**
Dr.N.V. Kulkarni, Professor, Dept. of Surgery

Lecture 2 :- **Challenges in rural Oncopathology practice**
Dr. R.S. Ayachit, Professor, Dept. of Pathology

Note :-

Free Registration.
All teaching staff members are invited for the afternoon session.
The lectures are very informative & important for P.G. Students.
So kindly make them compulsory.

Venue: Pathology lecture hall, MIMSR, Latur,1st floor college building.

Dr.Mrs.S.N.Kulkarni
Prof. & Head
Dept. Of Pathology
MIMSR Medical College,
Latur

The Head
Department of Pathology
MIMSR Medical College, LATUR

The Eminent SPEAKERS are:-

Dr.N.V.Kulkarni
Professor, Surgery
MIMSR Medical College,
Latur

Dr.R.S.Ayachit
Professor,Pathology
MIMSR Medical College,
Latur

Dr.Manisha Biradar
Asst.Prof.Pathology
Govt. Medical College,
Latur

Sr. No.	Time	Topic	Speaker
1	9 to 9.30am	Registration & breakfast	
2	9.30 to 10.00am	Inauguration	
3	10 to 10.30am	Introduction – Basics in Surgical Pathology	Dr.S.N.Kulkarni, Prof. & HOD
4	**10.30 to 1.30 pm**	**Surgical Pathology – Grossing techniques**	
a.	10.30 to 11.30 pm	**Head & neck** - Mandible, Tongue, Neck dissection	Dr.R.B.Badanale,Asst.Prof.
b.	11.30 to 12.00 pm	Breast	Dr.M.V.Biradar.Asst.Prof.
c.	12.00 to 12.30 pm	GIT-Colon	Dr.S.N.Kanthikar.Asso.Prof.
d.	12.30 to 1.00 pm	Kidney & urinary bladder	Dr.P.B.Chege,Asst.Prof.
e.	1.00 to 1.30 pm	Male genital system - Testis	Dr.J.B.Patil.Asso.Prof.
5	**1.30 to 2.30 pm**	**Lunch**	
6	2.30 to 3.30 pm	Biopsy – a vision of life	Dr.N.V.Kulkarni.Professor
7	3.30 to 4.30 pm	Challenges in rural Oncopathlogy practice	Dr.R.S.Ayachit.Professor
8	4.30 to 5.00 pm	Questions & answer session	
9	5.00pm onwards	Vote of thanks	Dr.S.N.Kanthikar.Asso.Prof.

ORGANISED BY :-
Department of Pathology,
MIMSR Medical College,
Latur.

CME PROGRAMME

CME on " Basics in Surgical Pathology"

27th November 2018

Sr. No.	Time	Topic	Speaker
1	9 to 10 am	Registration & breakfast	Registration & breakfast
2.	10 to 10.15am	Inauguration	-
-	-	**SESSION – 1st**	-
3	10.15 to 1.00pm	Grossing techniques live demonstration & hands on training	Dr.J.BV.Patil,Asso.Prof. Dr.S.N.Kanthikar,Asso.Prof. Dr.Manisha Biradar, Asst.Prof.(GMC,Latur) Dr.R.B.Badanale,Asst.Prof. Dr.P.B.Chege,Asst.Prof.
4	1.00 to 2.00 pm	Lunch	-
-	-	**SESSION – 2nd**	-
5	2.00 to 2.40pm	Lecture - 1st	Dr.N.V.Kulkarni, Professor Dept. of Surgery
6	2.40 to 3.20pm	Lecture – 2nd	Dr.R.S.Ayachit, Professor Dept. of Pathology
7	3.20 to 3.30pm	Tea break	-
8	3.30 to 4.30pm	Slide seminar	Dr.R.S.Ayachit, Professor Dept. of Pathology
9	4.30 to 5.00pm	Question & Answer session	-
10	5.00pm	Vote of thanks	-

Organizing Committee

Dept .of Pathology

MIMSR Medical College, Latur

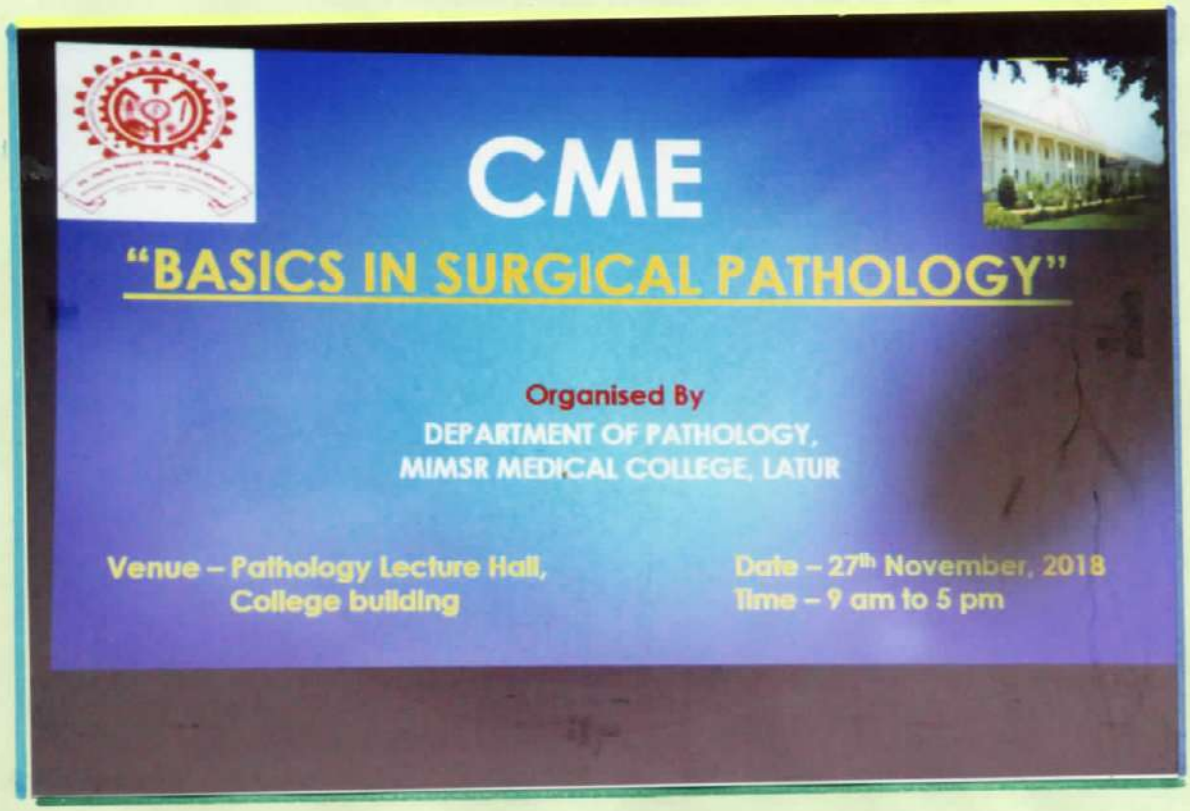
Organisres :- Dr.S.N.Kulkarni(Prof. & Head), Dr.R.V.Kulkarni(Professor), Dr.R.S.Ayachit(Professor), Dr.S.B.Ingle(Professor), Dr.J.B.Patil(Asso.Professor), Dr.A.S.Acharya(Asso.Professor), Dr.S.N.Kanthikar(Asso.Professor), Dr.K.S.Dhumure(Asst.Prof.), Dr.M.V.Kalyane(Asst.Prof.), Dr.R.B.Badanale(Asst.Prof.) Dr.P.B.Chege(Asst.Prof.) & Dr.M.A.Sonwane.

MIMSR MEDICAL COLLEGE, LATUR.
DEPARTMENT OF PATHOLOGY

CME on " Basics in Surgical Pathology"

27th November 2018

Sr.No.	Name of the Delegates	Mobile No	Sign
1.	Dr. Manisha V. Biradar	8983506164	Manisha
2.	Dr. Shewale Manjiri M.	9623017843	Manjiri
3.	Dr. Neeraja J. Nitsure	9730036228	Nitsure
4.	Dr. Vishakha J. Dakhore	9552888299	Vish
5.	Dr. Uttara Mookerji	7756015537	Uttara
6.	Dr. Jayamala Kamble	9892280967	Kamble
7.	Dr. Shrirayu R. Suryawanshi	9404564033	
8.	Dr. Sambodhi C. Killarikar	8275454419	Killarikar
9.	Dr. Surendra. B. Madke	8888392041	
10	Dr Raju. A Ramteke	8788960693	
11.	Dr. Priyanka Tile	8169329205	
12.	Dr. Ankita Chaudhari	7507201856	
13	Dr. Roshni. D. Bomble	8830153769	
14	Dr. Kapil S. Dhumane	9545083197	
15.	Dr. Megha M. Kasture	8898449338	
16.	Dr. Prajakta Vishwakarma	9403259432	
17.	Dr. Shubhangi Khule	9421270772	
18.	Dr. Noopur Patil	9920843059	
19	Dr. Shubhangi Khule	9421270772	
20	Dr Acharya A.S.	9404657705	
21	Dr. Shivraj Kanthikar	9764777213	
22	Dr. Prashant B. Chege	9028528792	
23	Dr. Rujuta Ayachit	9422072623	
24	Dr. Mirat Ghelani	9004685572	
25.	Dr Jayashree patil	9538162007	
26	Dr. R.V. Ionikdoni	9423078577	
27)	Dr. M.P. Zille	9890514092	
28	Dr. Narsinha V. Kulkarni	9422656396	
29)	Dr Kardile Rekha	9604681807	
30)	Dr BHOYAR A.P.	9403418883	
31)	Dr GUPTA K.C.	9403019493	
32)	Dr Asad	9224842084	
33)	Dr. Keval R. Dhone	9763258898	
34	Dr. M. A. Sonwane	9422968442	
35	Dr. S.N. Kulkarni	9881099851	
36	Dr. R.P. Gundawar	9503376899	


CME
"BASICS IN SURGICAL PATHOLOGY"
Organised By
DEPARTMENT OF PATHOLOGY,
MIMSR MEDICAL COLLEGE, LATUR
Venue – Pathology Lecture Hall,
College building
Date – 27th November, 2018
Time – 9 am to 5 pm


CME
"BASICS IN SURGICAL PATHOLOGY"


CHALLENGES IN ONCOPATHOLOGY
RURAL PRACTICE
Dr. Rujuta Ayachit
MD Path, PDCC Oncopath


CME
"BASICS IN SURGICAL PATHOLOGY"

APPLICATION ATTESTATION FORM (AAF) STS 2019

STS Reference ID: 2019-02033
Name of the Student: PRAGGYA YAADAV
Name of the Guide:
Name of Medical/Dental College: MAHARASHTRA INSTITUTE OF MEDICAL SCIENCES & RESEARCH, LATUR
Title of the STS Proposal: MOBILE PHONES: A CONTEMPORARY ECOSYSTEM FOR BACTERIAL GROWTH- OUR EXPERIENCE FROM RURAL MAHARASHTRA

Certificate to be signed by the Student

I certify that I am an MBBS/BDS student and am here by providing true information in the online application form for STS 2019 best to my knowledge. I am submitting only one application for STS 2019. In the event any information is found to be false, my studentship may be cancelled. I also certify that the research proposal is an original work prepared under the guidance of my Guide. I confirm that I have not committed 'plagiarism' in preparing this proposal. I understand that after evaluation of my proposal, I may or may not be selected and I shall abide by the decision of ICMR.

If selected, I shall follow all instructions provided on ICMR website for carrying out the research, preparation and submission of STS report. I also understand that if I am unable to complete my project & submit the report before the last date, no certificate or stipend will be awarded to me. I have gone through all the Instructions and Terms & Conditions for STS 2019 provided on ICMR website and will abide by them.

Signature of Student: Praggya Yaadav Name of the Student: PRAGGYA YAADAV
Date: 21/01/2019

Certificate to be signed by the Guide

I agree to accept the applicant Mr./Ms. PRAGGYA YAADAV studying in MBBS/BDS-I/II/III/IV (tick appropriate). I certify that he/she is not an intern or student of other courses and I will offer him/her all facilities and guidance for carrying out STS research. I also certify that the proposal is an original submission prepared by the student under my guidance. I confirm that neither me and nor my student have committed 'plagiarism' in preparing this proposal. I am forwarding only one STS 2019 student application. If my student is selected, I shall provide required facilities to enable early completion of research work, so that the report is submitted before the last date.

Signature of Guide:

Name: DR. ASHA PICHARE
Designation: PROFESSOR & HEAD OF DEPARTMENT
Department: MICROBIOLOGY

Attested By

Signature of Head of Department
Professor & Head
Department of Microbiology
M.I.M.S.R. Medical College
LATUR-413531

(Name in Block letters with seal)
DR. ASHA PICHARE

Signature of Head of Medical/Dental College
DEAN
M.I.M.S.R. Medical College
LATUR-413531

(Name in Block letters with seal)
DR. N.P. JANADAR

ill form completely & check it before submission. F

ICMR STS – REPORT 2019

Reference ID: 2019-02033

1. TITLE: Mobile Phones: A Contemporary Ecosystem for Bacterial Growth- Our Experience from Rural Maharashtra

2. INTRODUCTION:

Mobile phones are the most widely used gadgets today and have been integrated into our daily lives. Today, India has about 1.18 billion mobile phone users. [1] With advancement in technology, mobile phones have become a part and parcel of our daily life for the people of all disciplines including medicine . Phones are used to dispense laboratory and imaging results, patient data, photographic images, which are being used by physicians during bedside rounds, in order to engage clinicians, residents, and students. HCWs access pharmaceutical knowledge and literature by mobile phones, which facilitates learning and clinical performance. [2] Mobile phones can be highly loaded with tens of thousands of microbes living on each square inch area and represent an often overlooked reservoir for several enteric diseases [3] Mobiles can be brought in ICU's, post-operative wards and operation theatres etc. by enabling vibratory mode. [4] However, they are rarely cleaned and are often touched during or after examination of patients and handling of specimens without proper hand washing. [5] Thus, unhygienic ways of handling mobile phones make them a leading reservoir of an array of pathogenic microorganisms which can

prove fatal to the patients.[6] It can also be a potential hazard to the patient's family or to the doctor's family or to the doctor themselves.[7] They are also widely used in contaminated areas such as toilets, hospitals and kitchens, which are loaded with microorganisms and can transfer on the cell phone [8] and it becomes an exogenous source of nosocomial infection among hospitalized patients.

Also, scientists at the University of Arizona in the United States of America have found out that cell phones carry 10 times more bacteria than most toilet seats [9, 10] and can be even dirtier than the bottom of your shoe ![11, 12]

This study was undertaken to analyze the carrying rate of bacteria on mobile phones of health care personnel in clinical and nonclinical departments and effectiveness of disinfection by 70% Isopropyl alcohol.

3. REVIEW OF LITERATURE

A mobile phone is a device that can make and receive telephone calls over a radio link whilst moving around a wide geographic area [13]. Today, mobile phones have become an indispensable part of our life. Although they are usually stored in bags or our pockets, they are handled frequently and held close to the face [14]. Modern mobile phones (smart phones) are operated by tapping our finger on the glass touch screen.

A mobile phone can spread infectious diseases by its frequent contact with hands [15]. Hands are important for many aspects of personal, occupational and clinical hygiene [16]. Hands of health care workers have long been

known to be the potential source of infection. There is much evidence that contaminated fomites or surfaces play a key role in the spread of bacterial infections [17].

The sources of infection can be divided into two main groups: exogenous and endogenous [18]. Endogenous infections occur when the infectious agent comes from the patient's own body, usually from his/her own normal flora. The exogenous infection, on the other hand, develops from bacteria outside the body, which is the case most of the time they can be human, animal, or environmental in origin[19].

The combination of constant handling and the heat generated by the phones create a prime breeding ground for all sorts of microorganisms. The human surface tissue is constantly in contact with environmental microorganisms and becomes readily colonized by certain microbial species [20]. Hand washing is a process which removes soil and transient microorganisms off the hands. Hence, this simple process has long been a mainstay of any control measure for reducing nosocomial infections [21].

The WHO five moments to wash the hands effectively is widely used. It is a precautionary measure in order to avoid hospital acquired infections and cross transmissions. Also, alcohol-based products may be used for disinfecting hands[22]. Re-contamination of hands with mobiles occurs due the use of the hands for recording the pulse rate or the measurement of blood pressure or searching regarding medicines and treatment on internet or by simply keeping the phone on the patient's bed during rounds[23]. A well-practiced infection control plan– that encompasses hand hygiene is effective for the prevention of such nosocomial infections [24,25].

Unfortunately, studies continue to report unacceptably low hand washing compliance rates amongst health workers, despite the simplicity of hand washing procedure, [26].

Hence, accessories like mobile phones used by the health care workers for the betterment of the patient are inevitably contaminated with multi drug resistant organisms and a simple procedure of disinfections of mobile phones with 70% iso propyl alcohol has proven to be very effective [5].

4. AIMS AND OBJECTIVES

a) To analyze the carrying rate of bacteria on mobile phones of health care personnel.
b) To compare growth of bacteria on mobile phones used by doctors and paramedical staff of clinical and non-clinical health care departments.
c) To assess the effectiveness of disinfection by 70% Isopropyl alcohol

5. MATERIAL AND METHODS

a. MATERIALS:

1. Cotton Swabs
2. Sterile Saline
3. Blood Agar
4. MacConkey Agar
5. Grams Stain kit

6. Biochemical Tests: e.g. Coagulase Test, Catalase Test, Sugar Fermentation Test, IMViC Test, etc.
7. 70% Isopropyl Alcohol

b. METHODOLOGY:

1. **Study Setting:** Department of Microbiology of the concerned medical college and hospital

2. **Study Design**: Cross-Sectional Observational Study

3. **Sampling Method:** Stratified Random Sampling Method

4. **Study Duration**: 2 Months from 1st June'19 to 6th August'19

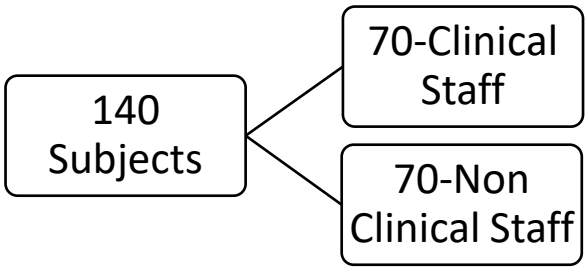
5. **Study Population:** Doctors and Paramedical staff

6. **Inclusion Criteria**:
   - Study subject having touch screen mobile.
   - Willing to participate in the study after written informed consent.

7. **Exclusion Criteria**:
   - Study subjects who do not have touch screen mobile phones.
   - Those who are not willing to participate.

8. **Informed Consent:** Informed written consent was taken from every participant in the study prior to taking mobile swabs

9. **Number of Samples:** 140



c. METHOD OF DATA COLLECTION:

A total of 140 cell phones belonging to health care personnel from both clinical and non-clinical departments of a rural hospital in Maharashtra were screened for bacterial isolates after written consent was taken from them.

(a) Sterile swabs soaked in sterile saline water were used for swabbing the mobile phones.

(b) This was followed by disinfection of cell phones with 70% isopropyl alcohol.

(c) After allowing it to dry for 10 minutes, repeat swabs were taken from the cell phones.

(d) These swabs were brought to the department of Microbiology immediately, where they were subjected to culture on blood agar and MacConkey agar.

(e) After incubation for about 24 hours at 37 degree Celsius, the growth obtained was identified on the basis of colony characters and morphology by gram staining and various biochemical tests following standard procedures[27].

6. OBSERVATIONS AND RESULTS

A total of 140 mobile phones were swabbed and cultured after due consent.

Table 1. Total Mobile phones Showing Positive & Negative Growth

TOTAL SAMPLES	POSITIVE GROWTH	NEGATIVE GROWTH
140	88 (62.86%)	52 (37.14%)

Out of total 140 mobile phones, growth was obtained from 88 (62.86%) mobile phones. *(Table 1)*

Table 2: Mobile phones Showing Positive & Negative Growth in Clinical and Non-Clinical Departments

Sr. No.	Department	Total Samples	Positive Growth	Negative Growth
1	Clinical	70	57 (81.42%)	13 (18.58%)
2	Non-Clinical	70	31 (44.28%)	39 (55.72%)

Out of 70 mobile phones swabbed from each department (clinical and non-clinical) growth was obtained from 57 (81.42%) mobiles of clinical departments and 31(44.28%) mobiles from non-clinical departments. *(Table 2)*

Table 3. Microorganisms Isolated from Mobile Phones

Sr.No.	ISOLATES	CLINICAL	NON-CLINICAL	Total
1	*Coagulase Negative Staphylococci (CoNS)*	24(27.28%)	13 (14.77%)	37 (42.05%)
2	*Staphylococcus aureus*	20 (22.72%)	14 (15.91%)	34 (38.63%)
3	*Pseudomonas aeruginosa*	8 (9.10%)	3 (3.41%)	11 (12.50%)
4	*E. coli*	3(3.41%)	-	3 (3.41%)
5	*Micrococcus*	2 (2.27%)	1 (1.13%)	3 (3.41%)
	Total	57 (64.78%)	31(35.22%)	88 (100%)

Overall isolation rate was more from clinical departments, i.e, 57 (64.78%) than non-clinical departments, i.e, 31(35.22%)

Coagulase Negative Staphylococcus (CoNS) was found on 37 (42.05%) mobiles. It was most commonly found followed by ***Staphylococcus aureus*** on 34 (38.63%) mobiles and ***Pseudomonas aeruginosa*** on 11 (12.50%) both from clinical and non-clinical departments.

E.coli was found on 3 (3.40%) mobile phones of the doctors and residents of the Clinical Department. They regularly perform clinical work in the wards and Intensive Care Unit (ICU) of the hospital.

Micrococci were found on 3 (3.40%) mobile phones.

All the above isolates obtained were more in percentage from the clinical departments, than the non-clinical departments.

The organisms that were isolated are tabulated in *Table 3 and Fig 1*.

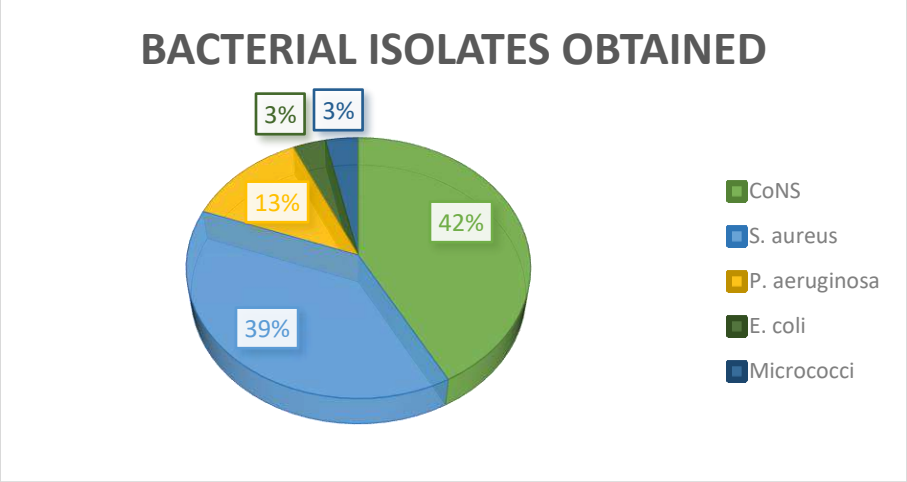


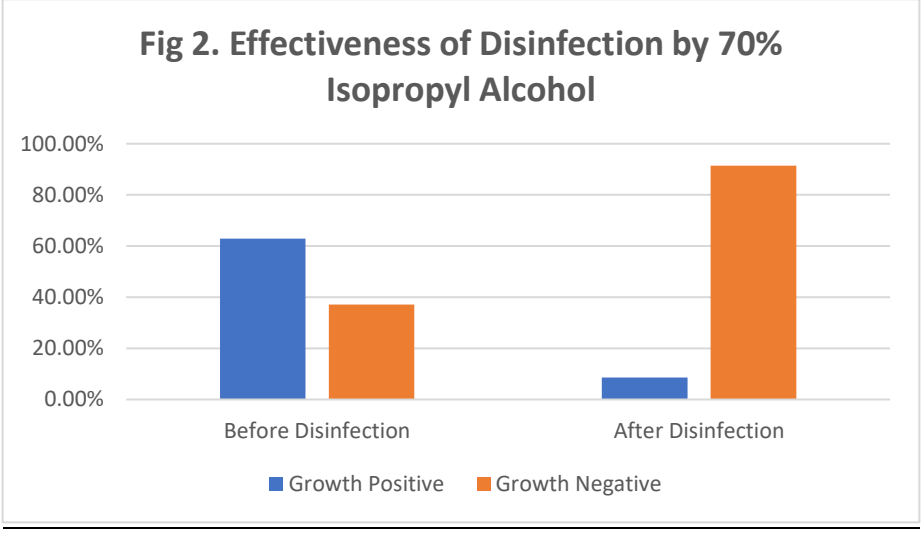
Fig 1: Total percentage of bacterial isolates obtained

Table 4. Results after Disinfection of Mobile Phones with 70 % Isopropyl Alcohol

Sr.No.	MOBILE PHONES	GROWTH POSITIVE	GROWTH NEGATIVE	TOTAL (n)
1	Before Disinfection	88 (62.86 %)	52(37.14 %)	140
2	After Disinfection	12(8.58 %)	128(91.42%)	140

After disinfection with 70% isopropyl alcohol only 12 (8.58%) mobile phones showed positive growth. There was a decrease in the bacterial carriage rate from 62.86% to 8.58%.

Thus, proving that the efficacy of disinfection is 91.42%. *(Table 4 and Fig 2)*



7. DISCUSSION:

Due to increase of mobile phones at affordable prices, they have become universally accepted accessories. The heat generated by them contributes to the bacteria harbouring on them to multiply at alarming levels. Thus, to live a healthy life, a standard of living must be maintained in terms of hygiene.

In this study, out of total 140 mobiles that were swabbed, growth was obtained in 88(62.857%) mobile phones *(Table 1).*

Similar observations were made by Chinchal Panchal *et al* [28], which showed positive growth in 65(65%) mobile phones.

This study is in contrast with the findings of another study by Usha Arora *et al* [5] which showed positive growth in 65(40.62%) mobile phones.

This might be because of more frequent usage of mobile phones by health care workers in our institution.

Out of 70 mobile phones swabbed from each clinical and nonclinical department, growth was obtained in 57 (81.42%) of clinical departments and 31(44.28%) of non-clinical departments. *(Table 2)*

Similar observations were made by Chinchal Panchal *et al* [28], which showed positive growth in 46(92%) mobiles of the clinical departments and positive growth in 14(56%) mobiles of the non-clinical departments.

It was observed that mobile phones from the health care personnel working in clinical departments showed a higher bacterial carriage rate as compared to the health care personnel of the non-clinical side as they are regularly involved in clinical work of the hospital like being in contact with patients who may harbor a variety of diseases, organisms. They also visit the Intensive Care Unit (ICU) and Operation Theater often. These bacteria can be readily transferred to the critically ill patients who already have a low immunity.

Out of the total organisms isolated, CoNS was present on 37(42.05%) mobiles. It was the most common organism isolated followed by *Staphylococcus aureus* which was present on 34(38.63%) mobiles. *(Table 3 and Fig 1)*

This finding correlates with the results of the study by Usha Arora *et al*[5] who also found CoNS as the commonest isolate showing an isolation rate of 27(41.53%) followed by *Staphylococcus aureus* on 22 (33.8%) mobiles. Similar observations have also been obtained by Surender Kaur *et al*[29] in which CoNS was present on 30 (42.8%) mobiles and *Staphylococcus aureus* was on 17 (24.28%) mobiles.
It is known that organisms like *Staphylococcus aureus* and *Coagulase Negative Staphylococcus* resist drying and thus, can survive and multiply rapidly in the warm environments of mobile phones.

Pseudomonas aeruginosa was isolated from 11(12.50%) mobile phones in our hospital. Higher isolation rate, from 21(18%) mobiles was noted by S. E. Amala *et al*[30].

Pseudomonas aeruginosa defiles the activities of many antiseptic and germicides used in disinfection and is therefore, is an important agent of hospital acquired infections. *Pseudomonas* is metabolically versatile, ubiquitous in both terrestrial and aquatic environs [31].The presence of this organism on mobile phones of medical personnel calls for serious public health attention

E. coli was found on 3(3.40%) Clinicians mobile phones. Higher isolation rate of 51(22.90%) was noted by Ketaki Ghatole et al [32]

They were mainly isolated from mobile phones of health care personnel performing surgeries or handling acutely ill patients and therefore they could transfer this to the patients.

Low carriage rate of the above two bacteria, i.e, *Pseudomonas* and *E. coli*, in our hospital may be due to good sterilization and disinfection measures followed at our hospital.

In this study, *micrococcus sp*. was found on 3(3.40%) mobiles.

Similar study by Usha Arora *et al*[5] shows isolation in 7(4.37%) mobiles.

These bacteria are located in various places such as water, soil and are part of normal skin microbial flora, frequently found on devices which are not adequately cleaned or disinfected [33]. Generally, *micrococcus sp*. is

considered on-haring bacteria and have not been reported as a nosocomial infection agent[34].

The efficacy of disinfection is 91.42% in our study. *(Table 4 and Fig 2)* Similar findings were noted by Usha Arora *et al*[5], showing efficacy of disinfection of 96.87%.

Another study by Ketaki Ghatole *et al* [32] showed efficacy of disinfection as 96% suggesting that alcohol-based solutions like 70% Isopropyl alcohol are effective in disinfection of mobile phones and can reduce carriage rate of bacteria and transmission of infections across patients and health care workers.

8. CONCLUSION

From this study, it can be concluded that Clinical health care personnels have a higher bacterial carriage rate of 57(81.42%) on their mobile phones as compared to the Non-Clinical side 31(44.28%).
It was observed that simple procedures like disinfection with 70% Isopropyl alcohol decreased the bacterial carriage rate to 8.58% from 62.86% before disinfection.
Therefore, the efficacy of disinfection is 91.42%.

Thus, mobiles that are carried around by health care professionals in the hospital may serve as mechanical vectors for the transmission of bacteria to

the patients and even to their family members. Restriction or prohibition of such devices is impractical. Therefore, tactics to prevent nosocomial transmission is a must.

9. SUMMARY

Our study was carried out at a medical college and hospital in rural Maharashtra over a period of 2 months from 1st June'19 to 6th August'19. This study was undertaken to analyze and compare the carrying rate of bacteria on mobile phones of health care personnel of clinical and non-clinical departments and test the effectiveness of disinfection by 70% Isopropyl alcohol.

In our study 140 mobiles were swabbed and cultured.

Positive growth was obtained from 88 mobile phones and hence, the carrying rate of bacteria on mobile phones was found to be 62.86%.

In the clinical department positive growth was obtained from 57(81.42%) mobiles while from the non-clinical departments positive growth was obtained from 31(44.28%) mobiles.

The commonest isolate obtained from the mobiles of both the departments were CoNS in 37(42.05%) followed by *Staphylococcus aureus* in 34(38.63%), *Pseudomonas aeruginosa* in 11(12.50%), *E. coli* in 3(3.41%) and *Micrococci* in 3(3.41%).

The bacterial carriage rate decreased from 62.86% to 8.58% after using 70% Isopropyl alcohol. Therefore, the efficacy of disinfection was nearly 92% after using 70% Isopropyl alcohol.

Thus, mobile phones can aid in the transmission of nosocomial infections to the immunodeficient patients and others.

To prevent this, simple procedures like disinfection of the mobile phone with 70% Isopropyl alcohol can prove effective in controlling nosocomial transmissions.

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