DIAGNOSTIC REPORT

agilus diagnostics

PATIENT NAME : SANJAY KUMAR SAHU		**REF. DOCTOR :** DR. SADAR HOSPITAL
CODE/NAME & ADDRESS : CR00000045 BPL PATIETNS SADAR HOSPITAL, BOKORO, SADAR HOSPITAL, BOKORO, SECTOR - 1, BOKORO STEEL CITY, BOKORO 827001 7260813496	ACCESSION NO : **0707XG000733** PATIENT ID : SANJM011082707 CLIENT PATIENT ID: ABHA NO :	AGE/SEX :41 Years Male DRAWN : RECEIVED : 12/07/2024 12:46:39 REPORTED : 12/07/2024 17:26:45

Test Report Status **Final**	Results	Biological Reference Interval	Units

HAEMATOLOGY - CBC

CBC WITH ESR (CBC+PS+ESR) EDTA WHOLE BLOOD/SMEAR

BLOOD COUNTS,EDTA WHOLE BLOOD

HEMOGLOBIN (HB)	**7.3 Low**	13.0 - 17.0	g/dL
RED BLOOD CELL (RBC) COUNT	**2.75 Low**	4.5 - 5.5	mil/µL
WHITE BLOOD CELL (WBC) COUNT	**3.40 Low**	4.0 - 10.0	thou/µL
PLATELET COUNT	**89 Low**	150 - 410	thou/µL

RBC AND PLATELET INDICES

HEMATOCRIT (PCV)	**22.6 Low**	40 - 50	%
MEAN CORPUSCULAR VOLUME (MCV)	**82.0 Low**	83 - 101	fL
MEAN CORPUSCULAR HEMOGLOBIN (MCH)	**26.6 Low**	27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC)	32.4	31.5 - 34.5	g/dL
RED CELL DISTRIBUTION WIDTH (RDW)	**15.0 High**	11.6 - 14.0	%
MENTZER INDEX	29.8		
MEAN PLATELET VOLUME (MPV)	9.5	6.8 - 10.9	fL

WBC DIFFERENTIAL COUNT

NEUTROPHILS	63	40 - 80	%
LYMPHOCYTES	28	20 - 40	%
MONOCYTES	05	2 - 10	%
EOSINOPHILS	04	1 - 6	%
BASOPHILS	0	< 1 - 2	%
ABSOLUTE NEUTROPHIL COUNT	2.14	2.0 - 7.0	thou/µL
ABSOLUTE LYMPHOCYTE COUNT	**0.95 Low**	1.0 - 3.0	thou/µL
ABSOLUTE MONOCYTE COUNT	**0.17 Low**	0.2 - 1.0	thou/µL
ABSOLUTE EOSINOPHIL COUNT	0.14	0.02 - 0.50	thou/µL
ABSOLUTE BASOPHIL COUNT	0	0.0 - 0.1	thou/µL
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	2.3		

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Dr.Sanjeew Kumar
Consultant - Pathologist &
Laboratory Head

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DIAGNOSTIC REPORT

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	ACCESSION NO : **0707XG000734**	AGE/SEX : 41 Years Male
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	CLIENT PATIENT ID:	RECEIVED : 12/07/2024 12:50:54
	ABHA NO :	REPORTED : 12/07/2024 17:54:56

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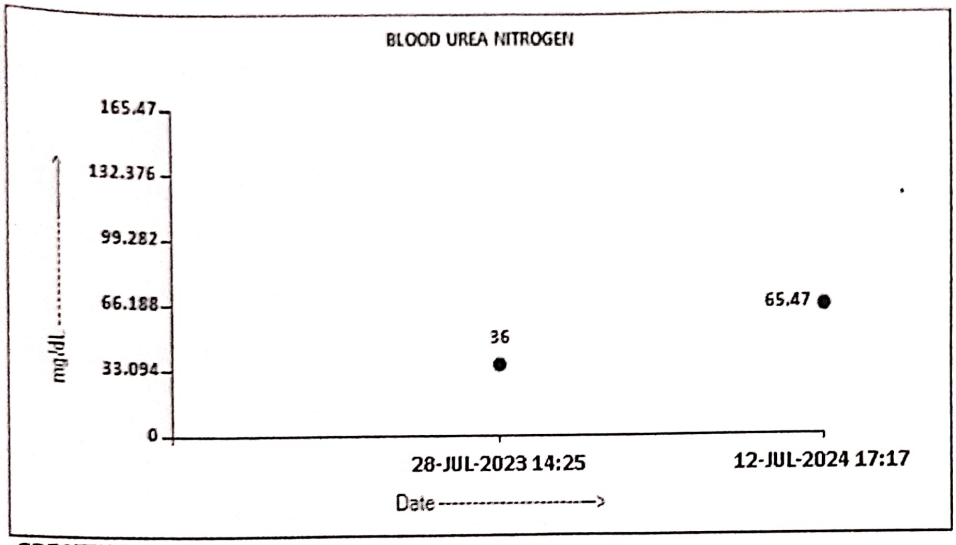
BIOCHEMISTRY

KIDNEY FUNCTION TEST

BLOOD UREA NITROGEN (BUN), SERUM

BLOOD UREA NITROGEN	**65.47 High**	6 - 22	mg/dL



CREATININE, SERUM

CREATININE	**7.31 High**	0.6 - 1.4	mg/dL

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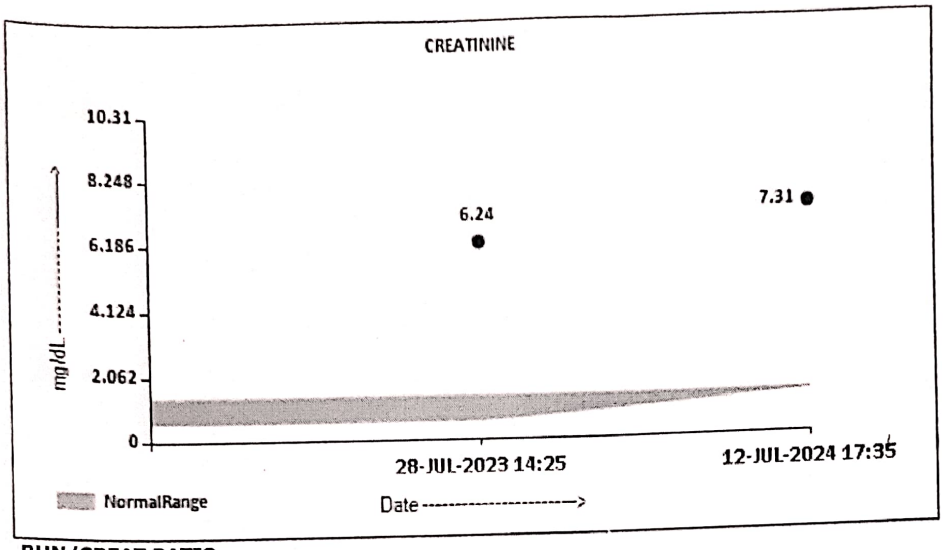

BUN/CREAT RATIO			
BUN/CREAT RATIO	8.96	5.0 - 15.0	
URIC ACID, SERUM			
URIC ACID	5.8	3.6 - 7.2	mg/dL
TOTAL PROTEIN, SERUM			
TOTAL PROTEIN	6.8	6.0 - 8.3	g/dL
ALBUMIN, SERUM			
ALBUMIN	4.4	3.2 - 5.0	g/dL
GLOBULIN			
GLOBULIN	2.4	2.0 - 4.1	g/dL

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CALCIUM, SERUM			
CALCIUM	**7.8 Low**	8.4 - 10.4	mg/dL
ELECTROLYTES (NA/K/CL), SERUM			
SODIUM, SERUM	**133.8 Low**	137 - 145	mmol/L
POTASSIUM, SERUM	4.28	3.6 - 5.0	mmol/L
CHLORIDE, SERUM	106.1	98 - 107	mmol/L

Interpretation(s)

Sodium	Potassium	Chloride
Decreased in: CCF, cirrhosis, vomiting, diarrhea, excessive sweating, salt-losing nephropathy, adrenal insufficiency, nephrotic syndrome, water intoxication, SIADH. Drugs: thiazides, diuretics, ACE inhibitors, chlorpropamide, carbamazepine, anti depressants (SSRI), antipsychotics.	**Decreased in:** Low potassium intake, prolonged vomiting or diarrhea, RTA types I and II, hyperaldosteronism, Cushing's syndrome, osmotic diuresis (e.g., hyperglycemia), alkalosis, familial periodic paralysis, trauma (transient). Drugs: Adrenergic agents, diuretics.	**Decreased in:** Vomiting, diarrhea, renal failure combined with salt deprivation, over-treatment with diuretics, chronic respiratory acidosis, diabetic ketoacidosis, excessive sweating, SIADH, salt-losing nephropathy, porphyria, expansion of extracellular fluid volume, adrenalinsufficiency, hyperaldosteronism, metabolic alkalosis. Drugs: chronic laxative, corticosteroids, diuretics.
Increased in: Dehydration (excessivesweating, severe vomiting or diarrhea), diabetes mellitus, diabetesinsipidus, hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice, oral contraceptives.	**Increased in:** Massive hemolysis, severe tissue damage, rhabdomyolysis, acidosis, dehydration, renal failure, Addison' s disease, RTA type IV, hyperkalemic familial periodic paralysis. Drugs: potassium salts, potassium- sparing diuretics, NSAIDs, beta-blockers, ACE inhibitors, high-dose trimethoprim-sulfamethoxazole.	**Increased in:** Renal failure, nephrotic syndrome, RTA, dehydration, overtreatment with saline, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis, hyperadrenocorticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, salicylates.
Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose.	**Interferences:** Hemolysis of sample, delayed separation of serum, prolonged fist clenching during blood drawing, and prolonged tourniquet placement. Very high WBC/PLT counts may cause spurious. Plasma potassium levels are normal.	**Interferences:** Test is helpful in assessing normal and increased anion gap metabolic acidosis and in distinguishing hypercalcemia due to hyperparathyroidism (high serum chloride) from that due to malignancy (Normal serum chloride)

LIVER FUNCTION PROFILE, SERUM			
TOTAL PROTEIN	6.8	6.0 - 8.3	g/dL
ALBUMIN	4.4	3.2 - 5.0	g/dL

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GLOBULIN	2.4	2.0 - 4.1	g/dL
ALBUMIN/GLOBULIN RATIO	1.8	1.0 - 2.1	RATIO
ASPARTATE AMINOTRANSFERASE(AST/SGOT)	20	0 - 45	U/L
ALANINE AMINOTRANSFERASE (ALT/SGPT)	14	0 - 45	U/L
ALKALINE PHOSPHATASE	**181 High**	41 - 137	U/L
GAMMA GLUTAMYL TRANSFERASE (GGT)	**61 High**	0 - 50	U/L
LACTATE DEHYDROGENASE	**486 High**	200 - 450	U/L
GLUCOSE RANDOM, PLASMA			
RBS(RANDOM BLOOD SUGAR)	153	Non-Diabetic: < 200 Diabetic: > or = 200 "In individuals with symptoms of hyperglycemia or hyperglycemic crisis."	mg/dL

Interpretation(s)
BLOOD UREA NITROGEN (BUN), SERUM-**Causes of Increased** levels include Pre renal (High protein diet, Increased protein catabolism, GI haemorrhage, Cortisol, Dehydration, CHF Renal), Renal Failure, Post Renal (Malignancy, Nephrolithiasis, Prostatism)
Causes of decreased level include Liver disease, SIADH.
CREATININE, SERUM-**Higher than normal level may be due to:**
• Blockage in the urinary tract, Kidney problems, such as kidney damage or failure, Infection, or reduced blood flow, Loss of body fluid (dehydration), Muscle problems, such as breakdown of muscle fibers, Problems during pregnancy, such as seizures (eclampsia)), or high blood pressure caused by pregnancy (preeclampsia)
Lower than normal level may be due to:• Myasthenia Gravis, Muscuophy
URIC ACID, SERUM-**Causes of Increased levels:**-Dietary(High Protein Intake,Prolonged Fasting,Rapid weight loss),Gout,Lesch nyhan syndrome,Type 2 DM,Metabolic syndrome **Causes of decreased levels-**Low Zinc intake,OCP,Multiple Sclerosis
TOTAL PROTEIN, SERUM-is a biochemical test for measuring the total amount of protein in serum.Protein in the plasma is made up of albumin and globulin.
Higher-than-normal levels may be due to: Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma,Waldenstroms disease.
Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage),Burns,Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome,Protein-losing enteropathy etc.
ALBUMIN, SERUM-Human serum albumin is the most abundant protein in human blood plasma. It is produced in the liver. Albumin constitutes about half of the blood serum protein. **Low blood albumin levels (hypoalbuminemia) can be caused by:** Liver disease like cirrhosis of the liver, nephrotic syndrome, protein-losing enteropathy, Burns, hemodilution, increased vascular permeability or decreased lymphatic clearance,malnutrition and wasting etc.
CALCIUM, SERUM-**Common causes of decreased value of calcium (hypocalcemia)** are chronic renal failure, hypomagnesemia and hypoalbuminemia.
Hypercalcemia (increased value of calcium) can be caused by increased intestinal absorption (vitamin D intoxication), increased skeletal reabsorption (immobilization), or a combination of mechanisms (primary hyperparathyroidism). Primary hyperparathyroidism and malignancy accounts for 90-95% of all cases of hypercalcemia.
Values of total calcium is affected by serum proteins, particularly albumin thus, latter's value should be taken into account when interpreting serum calcium levels. The following regression equation may be helpful.
Corrected total calcium (mg/dl)= total calcium (mg/dl) + 0.8 (4- albumin [g/dl])*
because regression equations vary among group of patients in different physiological and pathological conditions, mathematical corrections are only approximations. The possible mathematical corrections should be replaced by direct determination of free calcium by ISE. A common and important source of preanalytical error in the measurement of calcium is prolonged tornIquet application during sampling. Thus, this along with fist clenching should be avoided before phlebotomy.
LIVER FUNCTION PROFILE, SERUM-
Bilirubin is a yellowish pigment found in bile and is a breakdown product of normal heme catabolism. Bilirubin is excreted in bile and urine, and elevated levels may give

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HAEMATOLOGY

CBC WITH ESR (CBC+PS+ESR) EDTA WHOLE BLOOD/SMEAR

ERYTHROCYTE SEDIMENTATION RATE (ESR),EDTA BLOOD

E.S.R	**64 High**	0 - 14	mm at 1 hr

Interpretation(s)
ERYTHROCYTE SEDIMENTATION RATE (ESR),EDTA BLOOD-**TEST DESCRIPTION :-**
Erythrocyte sedimentation rate (ESR) is a test that indirectly measures the degree of inflammation present in the body. The test actually measures the rate of fall (sedimentation) of erythrocytes in a sample of blood that has been placed into a tall, thin, vertical tube. Results are reported as the millimetres of clear fluid (plasma) that are present at the top portion of the tube after one hour. Nowadays fully automated instruments are available to measure ESR.

ESR is not diagnostic; it is a non-specific test that may be elevated in a number of different conditions. It provides general information about the presence of an inflammatory condition.CRP is superior to ESR because it is more sensitive and reflects a more rapid change.

TEST INTERPRETATION
Increase in: Infections, Vasculities, Inflammatory arthritis, Renal disease, Anemia, Malignancies and plasma cell dyscrasias, Acute allergy Tissue injury, Pregnancy, Estrogen medication, Aging.
Finding a very accelerated ESR**(>100 mm/hour)** in patients with ill-defined symptoms directs the physician to search for a systemic disease (Paraproteinemias, Disseminated malignancies, connective tissue disease, severe infections such as bacterial endocarditis).
In pregnancy BRI in first trimester is 0-48 mm/hr(62 if anemic) and in second trimester (0-70 mm /hr(95 if anemic). ESR returns to normal 4th week post partum.
Decreased in: Polycythermia vera, Sickle cell anemia

LIMITATIONS
False elevated ESR : Increased fibrinogen, Drugs(Vitamin A, Dextran etc), Hypercholesterolemia
False Decreased : Poikilocytosis,(SickleCells,spherocytes),Microcytosis, Low fibrinogen, Very high WBC counts, Drugs(Quinine, salicylates)

REFERENCE :
1. Nathan and Oski's Haematology of Infancy and Childhood, 5th edition;2. Paediatric reference intervals. AACC Press, 7th edition. Edited by S. Soldin;3. The reference for the adult reference range is "Practical Haematology by Dacie and Lewis,10th edition.

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EIA - INFECTIOUS SECTION			
HEPATITIS C ANTIBODIES, SERUM			
HEPATITIS C ANTIBODIES	NON REACTIVE	NON REACTIVE	

Interpretation(s)
HEPATITIS C ANTIBODIES, SERUM-**Hepatitis C Virus (HCV)** is a blood borne flavivirus. It is one of the most important causes of post-blood transfusion as well as community acquired non-A non-B hepatitis and chronic liver failure. Although the majority of infected individuals may be asymptomatic, HCV infection may develop into chronic hepatitis, cirrhosis and/or increased risk of hepatocellular carcinoma.

Notes & Limitations: HCV antibody is typically not detected until approximately 14 weeks after infection (or 5 weeks after appearance of the first biochemical marker of illness) and is almost always detectable by the late convalescent stage of infection. A negative result may also be observed due to loss of HCV antigen, years following resolution of infection. Infants born to hepatitis C infected mothers may have delayed seroconversion to anti-HCV. Hence a negative result should be evaluated cautiously with respect to clinical findings. It is to be noted that absence of HCV antibodies after 14 weeks of exposure is strong evidence against HCV infection.Presence of HCV antibodies does not imply an active Hepatitis C infection but is indicative of both past and/or recent infection .It has been reported that as many as 90% of individuals receiving intravenous commercial immunoglobulin test falsely positive for HCV antibody. Also,patients with autoimmune liver disease may show a false positive HCV antibody result. Hence it is advisable to confirm a positive antibody result with a supplemental test. A positive result when followed by a positive supplemental test (i.e. HCV-RNA-PCR) suggests active hepatitis C infection.

****End Of Report****

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EIA - INFECTIOUS SECTION

HEPATITIS B SURFACE ANTIGEN, SERUM

HEPATITIS B SURFACE ANTIGEN	NON REACTIVE	NON REACTIVE

Interpretation(s)
HEPATITIS B SURFACE ANTIGEN, SERUM-**Hepatitis B** is caused by infection with HBV, a enveloped DNA agent that is classified as hepadnavirus. This test detects the presence of viral surface antigen i.e HBsAg also known as "Australia antigen" in serum sample and is indicative of HBV infection, either acute or chronic.
Test Utility: HBsAg is the first serologic marker appearing in the serum 6-16 weeks following hepatitis B viral infection. In typical HBV infection, HBsAg will be detected 2-4 weeks before the liver enzyme levels (ALT) become abnormal and 3-5 weeks before patient develops jaundice. In acute cases HBsAg usually disappears 1-2 months after the onset of symptoms. Persistence of HBsAg for more than 6 months indicates development of either a chronic carrier state or chronic liver disease.The presence of HBsAg is frequently associated with infectivity. HBsAg when accompanied by Hepatitis Be antigen and/or hepatitis B viral DNA almost always indicates infectivity.
Limitations: For diagnostic purposes, results should be used in conjunction with patient history and other hepatitis markers for diagnosis of acute or chronic infection. If the antibody results are inconsistent with clinical evidence, additional testing is suggested to confirm the result.HBsAg detection will only indicate the presence of surface antigens in the serum and should not be used as the sole criteria for diagnosis, staging or monitoring of HBV infection. This test may be negative during "window period" i.e. after disappearance of anti-HBsAg antibody.The current assay being a highly sensitive test may yield a small percentage of false positive reports. Hence all HBsAg positive specimens should be confirmed with an assay based upon Neutralisation of Human anti Hepatitis B Surface antibody.

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CAMP 2 BOKARO

Registration No : 20230024214
Visit No : 7/ Last Visit Date : 04/04/2024 12.00 AM / Token No : 72
Room No : Main Building A,OPD Block,Ground,G.Medicine OPD 9

Dr. Sanjay Kumar
Medicine OPD

Name : **Mr. Sanjay Kumar Sahu**
Sex/Age : **41Y 2M 18D / M**
Department : Medicine

Registration Amount : **Rs. 5**
Mobile No : **9693861075**
Address :BALIDIH,(JHARKHANDI)
Date of Registration : 01/07/2024 01.32 PM

Patient Type : General
Guardian Name : LT K SAHU(Father)

MLC Patient : NO

Last Complete Collection Date/Amount : 14/07/2023 04.32 PM/Rs. 5

Dr

HIV
HCV
HBsAg
CBC
LFT
S. Creatinine
LFT
S. Urea
, FBS

A 1C/C/O - PPD Diabetes

Report for Blood Examⁿ :-

HIV - Non-Reactive

12/7/24

Prepared By: Ms. Kumari Priti

Date Time: 01/07/2024 01.32 PM

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मुस्कान हॉस्पीटल
एण्ड रिसर्च सेन्टर

Dr. S. C. Munshi
MBBS, DCH, MD (Paeds)
Consultant Paediatrician & Neonatologist
Time : 9:30 am to 01:30 pm
(Sunday Off)

Dr. Irfan Ansari
MBBS, MS (Gen. Surgery)
Consultant Laparoscopic & Cancer Surgeon
Time : 10:30 am to 02:30 pm
(Friday Evening Off)

Dr. Md. Shahnawaj Anwar
MBBS, MD (Med.)
Consultant Physician
Cardiologist & Diabetologist
Time : 11:00 am to 02:30 pm
07:00 pm to 08:00 pm
(Sunday Evening Off)

Dr. Manoj Kr. Srivastava
MBBS, AFMC (PUNE)
Child Specialist, General Physician & Surgeon
Time : 11:30 am to 02:00 pm

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Facilities Available :

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- Variceal Band Ligation.
- Sclerotherapy
- Colonoscopy
- ERCP.

Eye Department :
- Phaco Surgery & OCT etc.
- Ben Franklin Optical Point

Neuro Surgery Deparment :
- OPD.

Name : Sanjay Kr. Sahu
Age/Sex: 47/M
Weight

MUSKAN Hospital &Research Centre

Date : 15 JUL 2024

CKD on MHD

BP- 160/100 mmhg

HR- 88/m

Chest - B/L NBC
CVS - S1S2
P/A Soft

F/U Case of CKD

MHD Weekly

Rx
Nicardia XL 30mg 1—1—1 TDS
Arkamin 0.1mg 1—1—1
Prazopress XL 5mg 1—x—1
Vit E
Folate 5mg OD
Nodosis 500mg BD
Glycimid MR 30mg OD
Zyloric 40mg OD
Inj Wepox 10000 ml S/C weekly

BUN - 65.42
Creat - 7.39
RBS - 153
Hb - 7.2g

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