Reg No - 4785

ARTHRITIS RHEUMATISM & PAIN CLINIC

DR P D RATH

MD, FACR, FRCP (Edin), FRCP (Glasgow)
FNIMS, FRCM, GCPR (USA, AUS)
DIPLOMA MSK ULTRASOUND (UCAM, SPAIN)
POST GRADUATE CERTIFICATE IN RHEUMATOLOGY
JOHN HOPKINS UNIVERSITY (USA)

DIRECTOR & HEAD OF DEPTT RHEUMATOLOGY

MAX SUPER SPECIALITY HOSPITAL
SAKET, SMART, PANCHSHEEL (NEW DELHI)

MEMBER BRITISH SOCIETY OF RHEUMATOLOGY
MEMBER EUROPEAN LUPUS SOCIETY
MEMBER NATIONAL OSTEOPOROSIS SOCIETY (UK)
AESCULOP FELLOWSHIP (BRAUN) INTERVENTIONAL PAIN MANAGEMENT

SPECIALIST IN
RHEUMATOID ARTHRITIS
OSTEOARTHRITIS
PSORIATIC ARTHRITIS
ANKYLOSING SPONDYLITIS
SLE
SCLERODERMA
GOUT
OSTEOPOROSIS
CHILDHOOD ARTHRITIS

CONSULTANT AT
MAX HEALTH CARE, NEW DELHI
SAKET
PANCHSHEEL
MAX SMART

BP 100/70
[illegible]
SpO2 98%

(W) – Shalini Singh. Age [illegible]

D Rt [illegible]

UTRA
-ve

ll. Allegra 180 (W) x 5 dys
ll. [illegible] x 3 dys
4) Inj [illegible]
Inj [illegible]
s.c [illegible]
F/U (MON)

ll. Inj Folitrax [illegible]

ll. Folic [illegible]

2. [illegible]

~~Inj [illegible]~~

4) Diet chart

CBC ESR
SGOT SGPT
[illegible]

Dr. P. D. Rath
MD, FACR, FRCP (Edin), FRCP
FNIMS, FRCM, GCPR (UWA,
DIPLOMA MSK USG (UCAM,
Director & Head Of Department
DMC REG No. 22141

CAUTION
RISK OF INFECTIONS/SIDE EFFECTS ON THESE MEDICATIONS EXPLAINED IN DETAIL

ARTHRITIS RHEUMATISM & PAIN CLINIC

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SAKET
PANCHSHEEL
MAX SMART

Shalini Singh. 41 F

DRA Refractory

See-Oct 'ees.

CBC
ESR
SGOT
SGPT
Sr Creatin

1. Inj Folitrax 25mg S.C
2. Folitrax 5mg daily
3. Hair fall B.D.
4. Inj Enbrel 50mg S.C.
5. Hcos 3gm BD
6. Shelcal DS B.D.
7. Dycotrin 50mg

3 Months

Dr. P. D. Rath
MD, FACR, FRCP (Edin), FRCP (Glasgow)
FNIMS, FRCM, GCPR (UWA, AUS)
DIPLOMA MSK USG (UCAM, SPAIN)
Director & Head Of Department - Rheumatology
DMC REG No. 22141

CAUTION
1. SIDE EFFECTS/SIDE EFFECTS
2. MEDICATIONS EXPLAINED

Reg 4785

ARTHRITIS RHEUMATISM & PAIN CLINIC

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SAKET
PANCHSHEEL
MAX SMART

BP 140/90
PR 107/min
SpO2 98%

Shalini Sable. 41 F
Ⓓ RA Refractory 18/11/23

1. Iron-HD BD.
11 Nucoxia 90mg SOS
2. Inj Enbrel 50mg SC/week
3. Inj Folitrax 20mg SC/week (WED)
4. Folvite 5mg MON/Thu/SAT
11. HCQS 300mg Ⓓ
0-0 1. S Tocal NS BD.
0-0 11. Dycain 50mg BD.
Ⓓ 4. Supracal NO Ⓔ
11 Upre D3 60K / week x 8 wk.
0-0 11 Cranpac-D BD.
11 PAN-D Ⓜ

Ⓓ 3 months
CBC ESR CRP SGOT
S. Prot Creatinine

CLINIC : 1477, Sector-37, Noida UP-201301
Timing : Mon, Fri 10:00 am to 12:30 pm. Tue, Thu, Sat 6:00pm-9:00 pm
For Appointment Call : 0120-4316153, 4303937, 9818457413

MAX SAKET : Mon, & Fri - 4:00-7:00 pm, Wed - 4:00-6:00pm, Tue,Thu, Sat - 11:00-4:00 pm
MAX PANCHSHEEL : Wed-11:00 am - 1:00 pm
MAX SMART : Mon, Fri, - 3:00-4:00 pm, Wed-2:00-4:00 pm

AKSHAYAM HEALTH SERVICES
I TREAT, HE CURES

MAX Lab

Laboratory Investigation Report

Patient Name	: Mrs. Shalini Singh	Centre	: 3046 - Akshayam Health Services
Age/Gender	: 41 Y 0 M 0 D /F	OP/IP No	: /
Max ID/Mobile	: ML01984160/6395945511	Collection Date/Time	: 09/May/2022 03:05PM
Lab ID	: 2810052200035	Receiving Date	: 09/May/2022
Ref Doctor	: Dr P D Rath	Reporting Date	: 10/May/2022
Passport No.	:		

Serology Special

Customized Package LTBI

Test Name	Result	Unit	Bio Ref Interval
Quantiferon Test (TB Gold), (IGRA), (Gamma Interferon For TB), Special Tube			
ELISA			
Quantiferon TB-Gold	Negative		
Antigen Tube - Nil Tube	0.01	IU/mL	< 0.35

Interpretation :

* **Positive** Quantiferon TB Gold result should be correlated with medical evaluation and diagnostic examination for active tuberculosis disease (e.g. AFB smear, culture, chest X-ray etc).

* **Negative** Quantiferon TB Gold result rules out Mycobacterium tuberculosis infection. A false negative result is possible if the patient is immunocompromised or has other comorbid conditions which affect immune function.

* **Indeterminate** results may be related to the suppressed immune status of the individual being tested. An indeterminate result should be repeated using a new blood specimen.

This assay can not differentiate between Latent infection and Active Tuberculosis.

Kindly correlate with clinical findings

*** End Of Report ***

Dr. Poonam. S. Das, M.D
Principal Director
Max Lab & Blood Bank Services

Dr. Bansidhar Tarai, M.D
Associate Director
Microbiology & Molecular Diagnostics

Dr. Madhuri Somani, M.D. , D.N.B
Consultant - Microbiology

Conditions of reporting: 1. The tests are carried out in the lab with the presumption that the specimen belongs to the patient name as identified in the bill/test request form. 2. The test results relate specifically to the sample received in the labeled and are assumed to be collected and transported per specific instructions given by the physicians/laboratory. 3. The reported results are for the information and interpretation by the referring doctor only. 4. Some tests are referred to other laboratories to provide a wider test menu to the customer. 5. Max Healthcare shall in no event be liable for accidental damages loss, or destruction of specimen which is not attributable to any direct and mala fide act or omission of Max Healthcare or its employees. Liability of Max Healthcare for deficiency of services, or other errors and omissions shall be limited to fee paid by the patient for the relevant laboratory services.

MAX Lab

Laboratory Investigation Report

Patient Name	: Mrs. Shalini Singh	Centre	: 3046 - Akshayam Health Services
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Ref Doctor	: Dr.P.D Rath	Reporting Date	: 09/May/2022
Passport No.	:		

Serology
Customized Package LTBI

Test Name	Result	Unit	Bio Ref Interval
Rapid Card Test - Hepatitis B Surface Antigen, (HBsAg) , Serum			
Immunochromatography			
Rapid Card HBsAg	Negative		

Comment

Interpretation

This is only a screening test.
All reactive samples should be confirmed by 'HBsAg confirmatory test or HBV DNA PCR'.
A non – reactive does not exclude the possibility of exposure to infection with HBV (window period)
False positive results can be obtained due to the presence of other antigens or elevated levels of RF factor.

Advise: Confirmatory test 'HBsAg Confirmatory Quantitative' test followed by 'HBV DNA quantitative PCR'

Test Name	Result	Unit	Bio Ref Interval
Rapid Card Test - HCV, Serum			
Immunochromatography			
HCV Card Test	Negative		

Comment **Interpretation**

It is only a screening test. All reactive samples should be confirmed by HCV quantitative PCR
A non reactive result does not exclude the possibility of exposure to or infection with HCV.
Patients with auto immune liver disease may show falsely reactive results

Kindly correlate with clinical findings

*** End Of Report ***

Dr. Saloni Sehgal (MBBS, MD)
Principal Consultant &
Head Microbiology & Infection Control

Dr. Neera Kaushik
Senior Microbiologist

Max Lab Limited (A Wholly Owned Subsidiary of Max Healthcare Institute Ltd.)
Plot No. NH-01 Sector-19, Noida, Uttar Pradesh-201305
(CIN No. U85100DL2021PLC381826)

SIN No:B2B546785, Test Performed at :794 - Max Hospital - Vaishali, W-3, Sector-1, Vaishali, Ghaziabad-201012, U.P

Helpline No: 7982 100 200 www.maxlab.co.in feedback@maxlab.co.in

Booking Centre :3046 - Akshayam Health Services, 1477 Sec-37, Noida, 8929622560

The authenticity of the report can be verified by scanning the Q R Code on top of the page

Pathkind

NATIONAL REFERENCE LAB
PATHKIND DIAGNOSTICS PVT. LTD.
Plot No. 55 - 56, Udyog Vihar, Phase 4, Gurugram - 122015
E-Mail: care@pathkindlabs.com | Website: www.pathkindlabs.com
Customer Care: 75000 75111

Client

Pathkind Collection Center (Bhud Road)
Najeempura, Bhud Road
Bhood, Distt. Bulandashar, UP-203001, C-9720787466

Processed By
Pathkind Diagnostics Pvt. Ltd.
Plot No. 55-56, Udhyog Vihar Ph-IV, Gurugram - 122015

Name	: **Mrs. SHALINI SINGH**	Billing Date	: 14/01/2023 10:55:17
Age	: 42 Yrs	Sample Collected on	: 14/01/2023 11:00:43
Sex	: Female	Sample Received on	: 14/01/2023 12:30:36
P. ID No.	: P1105200005123	Report Released on	: 15/01/2023 11:35:58
Accession No	: **11052210131629**	Barcode No.	: 994298584, 992789579
Referring Doctor	: SELF		
Referred By	: Dr Rajesh Saxena C/O Pathkind CC Bhud Road	Ref no.	:

Report Status - Final

Test Name	Result	Biological Ref. Interval	Unit
High-risk conditions Any one of following: 1. ASCVD (CAD/PAD/TIA or stroke) 2. Homozygous familial 3. hypercholesterolemia 4. Diabetes with ≥2 major ASCVD risk factors*/target organ damage	(recommended) LDL-C goal of ≤30 mg/dl (optional) CAD with ≥1 of following: 1. Diabetes without target organ damage/≤1 major 2. ASCVD risk factors 3. Familial hypercholesterolemia 4. ≥3 major ASCVD risk factors 5. CKD stage 3B and 4 6. ≥2 major ASCVD risk factors with ≥1 moderate 7. non-conventional risk factor# 8. Lp(a) ≥50 mg/dl 9. Coronary calcium score ≥300 HU 10. Extreme of a single risk factor 11. PAD 12. H/o TIA or stroke 13. Non-stenotic carotid plaque	CAD with ≥1 of following: 1. Diabetes + polyvascular disease/≥2 2. major ASCVD risk factors*/target organ 3. damage 4. Recurrent ACS (within 12 months) 5. despite on LDL-C goal 6. Homozygous familial 7. Hypercholesterolemia	

The LDL-C goal of ≤30 mg/dl must be pursued after detailed risk-benefit discussion between physician and patient.

Clinical judgment to be used in decision making if the patient has disease/risk factors not covered in the table, eg. peripheral arterial disease or cerebrovascular disease.

*Major ASCVD risk factors: 1. Age- male ≥45 years, female ≥55 years, 2. Family h/o premature CAD- male <55 years, female <65 years, 3. Smoking/tobacco use, 4. Systemic hypertension, 5.Low HDL (males <40 mg/dl and females <50 mg/dl).

#Moderate non-conventional risk factors: 1. Coronary calcium score 100-299 HU, 2. Increased carotid intima-media thickness, 3. Lp(a) ≥20-49

Dr. Aarti Khanna Nagpal
DNB (Pathology)
Senior Consultant

The Test/s marked with (#) is are not accredited by NABL

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69, Ground Floor, Ambedkar Road, Ghaziabad-201001

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Referred By	: Dr Rajesh Saxena C/O Pathkind CC Bhud Road	Ref no.	:	

Report Status - Final

Test Name	Result	Biological Ref. Interval	Unit
	SEROLOGY		
C-Reactive Protein (CRP), Quantitative *Sample: Serum* *Method: Spectrophotometery*	4.79	0 - 5	mg/L
	HEALTHKIND ADVANCE		
	BIOCHEMISTRY		
HbA1C (Glycosylated Hemoglobin)			
HbA1c *Sample: Whole Blood EDTA* *Method: High Perfomance Liquid Chromatography (HPLC)*	5.0	Non Diabetic : < 5.7 % Prediabetic Range : 5.7 - 6.4 % Diabetic Range : >= 6.5 % Goal of Therapy :<7.0 % Action suggested :>8.0 %	%
Mean Plasma Glucose *Sample: Whole Blood EDTA* *Method: Calculated*	96.8	< 116	mg/dL
Liver Function Extended Panel			
Lactate Dehydrogenase (LDH) *Sample: Serum* *Method: Spectrophotometery*	191	<223	U/L
Gamma-Glutamyl Transferase (GGT) *Sample: Serum* *Method: Spectrophotometery*	14	<42	U/L
Phosphorus *Sample: Serum* *Method: Spectrophotometry-Phosphomolybdate Reduction*	3.3	2.6 - 4.5	mg/dL
Iron Studies **Sample: Serum** *Method: Method: Spectrophotometry-Ferrozine*			
Iron *Sample: Serum* *Method: Spectrophotometery*	**25 L**	37 - 145	µg/dL

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Report Status - Final

Test Name	Result	Biological Ref. Interval	Unit
(UIBC) Unsaturated Iron Binding Capacity *Sample: Serum*	**394 H**	110 - 370	µg/dL
Total Iron Binding Capacity (TIBC) *Sample: Serum* *Method: Calculated*	419	228 - 428	µg/dL
% Saturation *Sample: Serum* *Method: Calculated*	**6 L**	20 - 50	%
Ferritin *Sample: Serum* *Method: ECLIA*	**4.63 L**	15 - 150	ng/mL
Thyroid Profile Total			
Total T3 (Triiodothyronine) *Sample: Serum* *Method: ECLIA*	1.10	0.8 - 2	ng/mL
Total T4 (Thyroxine) *Sample: Serum* *Method: ECLIA*	9.91	5.1 - 14.1	µg/dL
TSH 3rd Generation *Sample: Serum* *Method: ECLIA*	4.150	0.27 - 4.2	µIU/mL
Vitamin Profile			
Vitamin D 25 - Hydroxy *Sample: Serum* *Method: ECLIA*	44.1	Deficiency < 20 Insufficiency 20 - 30 Sufficiency 30 - 100 Toxicity > 100	ng/mL
Vitamin B12 *Sample: Serum* *Method: ECLIA*	**769 H**	191 - 663	pg/mL

C-Reactive Protein (CRP), Quantitative

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Report Status - Final

Test Name	Result	Biological Ref. Interval	Unit

Clinical Significance :
"C-reactive protein (CRP) is a trace protein which rises in acute inflammation. After onset of an acute phase response, the serum CRP concentration rises rapidly within 6-12 hours and peaks at 24-48 hours and extensively. Very high CRP levels are associated with severe trauma and infection (sepsis)."

HbA1C (Glycosylated Hemoglobin)

Clinical Significance :
Hemoglobin A1c (HbA1c) level reflects the mean glucose concentration over the previous period (approximately 8-12 weeks) and provides a much better indication of long-term glycemic control than blood and urinary glucose determinations. American Diabetes Association (ADA) include the use of HbA1c to diagnose diabetes, using a cutpoint of 6.5%. The ADA recommends measurement of HbA1c 3-4 times per year for type 1 and poorly controlled type 2 diabetic patients, and 2 times per year for well-controlled type 2 diabetic patients) to assess whether a patient's metabolic control has remained continuously within the target range. Falsely low HbA1c results may be seen in conditions that shorten erythrocyte life span. and may not reflect glycemic control in these cases accurately.

Lactate Dehydrogenase (LDH)

Clinical Significance :
Lactate dehydrogenase (LD) levels are raised in megaloblastic anemia, untreated pernicious anemia, Hodgkin's disease, abdominal and lung cancers, severe shock, and hypoxia, myocardial infarction (MI), pulmonary infarction, pulmonary embolism, leukemia, hemolytic anemia, infectious mononucleosis, progressive muscular dystrophy, liver disease, and renal disease.

Phosphorus

Clinical Significance :
Serum phosphorus levels are low in case of shift of phosphate from extracellular to intracellular space, renal phosphate wasting, loss from the gastrointestinal tract, and loss from intracellular stores. Serum Phosphorus levels rise when the kidneys have an inability to excrete phosphate, increased intake or a shift from of phosphate from the tissues into the extracellular fluid.

Iron

Clinical Significance :

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Test Name	Result	Biological Ref. Interval	Unit

Serum Iron is normal or low in iron deficient anaemia, pregnancy, patients taking oral contraceptive medications,in chronic inflammatory and malignancies. Serum Iron is high in hereditary hemochromatosis and in iron overload states.

Total Iron Binding Capacity (TIBC)

Clinical Significance :
Transferrin is the primary plasma iron transport protein but accounts for 25% to 30% saturation with iron. The additional amount of iron that can be bound is the unsaturated iron-binding capacity (UIBC). The total iron-binding capacity (TIBC) can be indirectly determined using the sum of the serum iron and UIBC. TIBC levels are usually low when serum Iron levels are high and vice versa.

Ferritin

Clinical Significance :
Decreased levels of serum Ferritin is associated with increased risk for developing iron deficiency which in turn cn lead to anaemia. Increased levels of serum ferritin is associated with iron overload conditions(like hereditary hemochromatosis), common liver disorders, neoplasms, acute or chronic inflammation and hereditary hyperferritinemia-cataract syndrome.

Total T3 (Triiodothyronine)

Clinical Significance :
Thyroid hormones, T3 and T4, which are secreted by the thyroid gland, regulate a number of developmental, metabolic, and neural activities throughout the body. The thyroid gland synthesizes 2 hormones - T3 and T4. T3 production in the thyroid gland constitutes approximately 20% of the total circulating T3, 80% being produced by peripheral conversion from T4. T3 is more potent biologically. Total T3 comprises of Free T3 and bound T3. Bound T3 remains bound to carrier proteins like thyroid-binding globulin, prealbumin, and albumin). Only the free forms are metabolically active. In hyperthyroidism, both T4 and T3 levels are usually elevated, but in some rare cases, only T3 elevation is also seen. In hypothyroidism T4 and T3 levels are both low. T3 levels are frequently low in sick or hospitalized euthyroid patients.

Total T4 (Thyroxine)

Clinical Significance :
Total T4 is synthesized in the thyroid gland. About 0.05% of circulating T4 is in the free or biologically active form. The remainder is bound to thyroxine-binding globulin (TBG), prealbumin, and albumin. High levels of T4 (and FT4) causes hyperthroidism and low levels lead to hypothyroidism.

TSH 3rd Generation

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Report Status - Final

Test Name	Result	Biological Ref. Interval	Unit

Clinical Significance :
TSH levels are elevated in primary hyporthyroidism and low in primary hyperthyroidism. Evaluation of TSH is useful in the differential diagnosis of primary from secondary and tertiary hypothyroidism. In primary hypothyroidism, TSH levels are elevated, while in secondary and tertiary hypothyroidism, TSH levels are low or normal. High TSH level in the presence of normal FT4 is called subclinical hypothyroidism and low TSH with normal FT4 is called subclinical hyperthyroidism. Sick, hospitalized patients may have falsely low or transiently elevated TSH. Significant diurnal variation is also seen in TSH levels.

Guidelines for TSH levels in pregnancy, as per American Thyroid Association, are as follows:

PREGNANCY TRIMESTER	BIOLOGICAL REFERENCE INTERVAL	UNIT
FIRST TRIMESTER	0.100 - 2.500	µIU/mL
SECOND TRIMESTER	0.200 - 3.000	µIU/mL
THIRD TRIMESTER	0.300 - 3.000	µIU/mL

Vitamin D 25 - Hydroxy

Clinical Significance :
The 25-hydroxy vitamin D test is used to detect bone weakness or other bone malfunctions or disorders that occur as a result of a vitamin D deficiency.Those who are at high risk of having low levels of vitamin D include people who don't get much exposure to the sun, older adult, people with obesity, babies who are breastfed only, post gastric bypass surgery, Crohn's disease and other intestinal malabsorption conditions. Hypervitaminosis D usually occurs due to over intake of Vitamin D supplementation.

Vitamin B12

Clinical Significance :
Vitamin B12 is necessary for hematopoiesis and normal neuronal function. It requires intrinsic factor (IF) for absorption. Vitamin B12 deficiency may be due to lack of IF secretion by gastric mucosa (eg, gastrectomy, gastric atrophy) or intestinal malabsorption (eg, ileal resection, small intestinal diseases). Vitamin B12 deficiency results in macrocytic anemia, glossitis, peripheral neuropathy, weakness, hyperreflexia, ataxia, loss of proprioception, poor coordination, and affective behavioral changes.

Dr. Gulzar Ali
MD (Pathology)
Pathologist

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hood, Distt. Bulandshar, UP-203001, C-9720787466

Name	: **Mrs. SHALINI SINGH**	Billing Date	: 14/01/2023 10:55:17
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Sex	: Female	Sample Received on	: 14/01/2023 12:30:36
P. ID No.	: P1105200005123	Report Released on	: 15/01/2023 11:35:58
Accession No	: **11052210131629**	Barcode No.	: 994298582
Referring Doctor	: SELF		
Referred By	: Dr Rajesh Saxena C/O Pathkind CC Bhud Road	Ref no.	:

Report Status - Final

Test Name	Result	Biological Ref. Interval	Unit
HEALTHKIND ADVANCE			
HAEMATOLOGY			
Complete Blood Count (CBC)			
Haemoglobin (Hb) *Sample: Whole Blood EDTA* *Method: Photometric measurement*	**8.0 L**	12 - 15	gm/dL
Total WBC Count / TLC *Sample: Whole Blood EDTA* *Method: Impedance*	9.0	4 - 10	thou/µL
RBC Count *Sample: Whole Blood EDTA* *Method: Impedance*	3.9	3.8 - 4.8	million/µL
PCV / Hematocrit *Sample: Whole Blood EDTA* *Method: RBC Pulse height*	**28.3 L**	36 - 46	%
MCV *Sample: Whole Blood EDTA* *Method: Calculated*	**74.0 L**	83 - 101	fL
MCH *Sample: Whole Blood EDTA* *Method: Calculated*	**20.7 L**	27 - 32	pg
MCHC *Sample: Whole Blood EDTA* *Method: Calculated*	**28.2 L**	31.5 - 34.5	g/dL
RDW (Red Cell Distribution Width) *Sample: Whole Blood EDTA* *Method: Calculated*	**16.8 H**	11.9 - 15.5	%
DLC (Differential Leucocyte Count) *Method: Flowcytometry/Microscopy*			
Neutrophils *Sample: Whole Blood EDTA* *Method: VCS Technology & Microscopy*	66	40 - 80	%

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Report Status - Final

Test Name	Result	Biological Ref. Interval	Unit
Lymphocytes *Sample: Whole Blood EDTA* *Method: VCS Technology & Microscopy*	31	20 - 40	%
Eosinophils *Sample: Whole Blood EDTA* *Method: VCS Technology & Microscopy*	01	1 - 6	%
Monocytes *Sample: Whole Blood EDTA* *Method: VCS Technology & Microscopy*	02	2 - 10	%
Basophils *Sample: Whole Blood EDTA* *Method: VCS Technology & Microscopy*	00	0 - 2	%
Absolute Neutrophil Count *Sample: Whole Blood EDTA*	5940	2000 - 7000	/µL
Absolute Lymphocyte Count *Sample: Whole Blood EDTA*	2790	1000 - 3000	/µL
Absolute Eosinophil Count *Sample: Whole Blood EDTA*	90	20 - 500	/µL
Absolute Monocyte Count *Sample: Whole Blood EDTA*	**180 L**	200 - 1000	/µL
Absolute Basophil Count *Sample: Whole Blood EDTA*	**00 L**	20 - 100	/µL
DLC Performed By *Sample: Whole Blood EDTA*	EDTA Smear		
Platelet Count *Sample: Whole Blood EDTA* *Method: Impedance*	280	150 - 410	thou/µL
MPV (Mean Platelet Volume) *Sample: Whole Blood EDTA* *Method: Calculated*	10.5	6.8 - 10.9	fL

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Report Status - Final

Test Name	Result	Biological Ref. Interval	Unit
Sample: Whole Blood EDTA **Erythrocyte Sedimentation Rate (ESR)** *Sample: Whole Blood EDTA* *Method: Capillary Photometry*	**28 H**	< 12	mm 1st Hour
BIOCHEMISTRY			
Fasting Plasma Glucose *Sample: Fluoride Plasma - F* *Method: Hexokinase*	**115 H**	74 - 99	mg/dL
Lipid Profile Direct			
Total Cholesterol *Sample: Serum* *Method: Spectrophometry-Esterase/CO/Peroxidase*	180	Desirable Level : < 200 Borderline : 200 - 239 High Risk : >/= 240	mg/dL
Triglycerides *Sample: Serum* *Method: GPO-PAP*	139	Desirable : < 150 Borderline High : 150 - 199 High : 200 - 499 Very High : >/= 500	mg/dL
HDL Cholesterol *Sample: Serum* *Method: Immunoinhibition-direct measure*	56	Low : < 40 Optimal : 40 - 60 High : > 60	mg/dL
VLDL Cholesterol *Sample: Serum* *Method: Calculated*	27.8	Desirable 10 - 35	mg/dL
Non HDL Cholesterol *Sample: Serum*	124	< 130	mg/dL
Total Cholesterol / HDL Ratio *Sample: Serum* *Method: Calculated*	**3.21 L**	Low Risk : 3.3 - 4.4 Average Risk : 4.5 - 7.0 Moderate Risk : 7.1 - 11.0 High Risk : > 11.0	
LDL / HDL Ratio *Sample: Serum* *Method: Calculated*	1.79	Low Risk : 0.5 - 3.0 Moderate Risk : 3.1 - 6.0	

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Report Status - Final

Test Name	Result	Biological Ref. Interval	Unit
		High Risk : > 6.0	
Liver Function Extended Panel			
Bilirubin Total *Sample: Serum* *Method: Dichloroaniline*	0.4	0 - 1.1	mg/dL
Bilirubin Direct *Sample: Serum* *Method: Dichloroaniline*	0.2	0 - 0.2	mg/dL
Serum Bilirubin (Indirect) *Sample: Serum* *Method: Calculated*	0.20	< 0.90	mg/dL
SGOT / AST *Sample: Serum* *Method: UV with P5P*	23	0 - 27	U/L
SGPT / ALT *Sample: Serum* *Method: UV with P5P*	25	0 - 33	U/L
Alkaline Phosphatase (ALP) *Sample: Serum* *Method: IFCC*	72	0 - 98	U/L
Total Protein *Sample: Serum* *Method: Spectrophotometry Bluret*	8.1	6.4 - 8.3	g/dL
Albumin *Sample: Serum* *Method: BROMOCRESOL GREEN(BCG)*	3.7	3.49 - 4.75	g/dL
Globulin *Sample: Serum* *Method: Calculated*	**4.4 H**	1.9 - 3.7	g/dL
Albumin Globulin A/G Ratio *Sample: Serum* *Method: Calculated*	**0.8 L**	1 - 2.1	

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Report Status - Final

Test Name	Result	Biological Ref. Interval	Unit
Kidney Function Test			
Blood Urea Nitrogen (BUN) *Sample: Serum* *Method: Spectrophotometry-Urease / GLDH*	12.15	7 - 18.69	mg/dL
Urea *Sample: Serum* *Method: Calculated*	26.00	15 - 40	mg/dL
Creatinine *Sample: Serum* *Method: Jaffe's Reaction*	0.89	0.6 - 1.1	mg/dL
BUN Creatinine Ratio *Sample: Serum* *Method: Calculated*	14	10 - 20	
Uric Acid *Sample: Serum* *Method: Uricase-Peroxidase*	5.4	2.6 - 6	mg/dL
Sodium *Sample: Serum* *Method: ISE-Direct*	141	136 - 145	mmol/L
Potassium *Sample: Serum* *Method: ISE-Direct*	4.4	3.5 - 5.1	mmol/L
Chloride *Sample: Serum* *Method: ISE-Direct*	**109 H**	97 - 107	mmol/L
# Calcium *Sample: Serum* *Method: Phosphonazo III*	8.7	8.6 - 10	mg/dL
SEROLOGY			
Hepatitis B Surface Antigen (HBsAg) Rapid Card *Sample: Serum* *Method: Immunochromatography*	Non Reactive	Non Reactive	

This Test/s marked with (#) is/are not accredited by NABL

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Test Name	Result	Biological Ref. Interval	Unit

CLINICAL PATHOLOGY

Urine Routine & Microscopic Examination

Method: Reflectance Photometry

Physical Examination

Test Name	Result	Biological Ref. Interval	Unit
Colour *Sample: Urine* *Method: Physical Examination*	Yellow	Pale Yellow	
Appearance *Sample: Urine* *Method: Physical Examination*	Clear	Clear	
Specific Gravity *Sample: Urine* *Method: pKa change of pretreated polyelectrolytes*	1.025	1.003 - 1.035	
pH *Sample: Urine* *Method: Double indicator principle*	5.5	4.7 - 7.5	

Chemical Examination

Test Name	Result	Biological Ref. Interval	Unit
Glucose *Sample: Urine* *Method: Glucose oxidase/peroxidase*	Not Detected	Not Detected	
Protein *Sample: Urine* *Method: Protein-error-of-Indicators principle*	Not Detected	Not Detected	
Ketones *Sample: Urine* *Method: Rothera's*	Not Detected	Not Detected	
Blood *Sample: Urine* *Method: Peroxidase*	Not Detected	Not Detected	
Bilirubin *Sample: Urine* *Method: Azo dye*	Not Detected	Not Detected	

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Test Name	Result	Biological Ref. Interval	Unit
Urobilinogen *Sample: Urine* *Method: PABa & phenazopyridine*	Normal	Normal	
Nitrite *Sample: Urine* *Method: Diazonium compound*	Not Detected	Not Detected	
Microscopic Examination *Method: Microscopy*			
Pus Cells *Sample: Urine*	1-2	0 - 5	/hpf
RBC *Sample: Urine*	Not Detected	Not Detected	/hpf
Epithelial Cells *Sample: Urine*	2-4	0 - 5	/hpf
Casts *Sample: Urine*	Not Detected	Not Detected	/hpf
Crystals *Sample: Urine*	Calcium Oxalate	Not Detected	/hpf
Bacteria *Sample: Urine*	Not Detected	Not Detected	/hpf
Remarks *Sample: Urine*			

Remarks : Microscopic Examination is performed on urine sediment
Haemoglobin (Hb)

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Hemoglobin is the iron containing protein molecule in red blood cells that carries oxygen from the lungs to the body's tissues and returns carbon dioxide from the tissues back to the lungs. Decrease in Hemoglobin levels results in anaemia and very high Hemoglobin levels results in hemochromatosis.

PCV / Hematocrit

Clinical Significance :
Hemoglobin is the iron containing protein molecule in red blood cells that carries oxygen from the lungs to the body's tissues and returns carbon dioxide from the tissues back to the lungs. Decrease in Hemoglobin levels results in anaemia and very high Hemoglobin levels results in hemochromatosis. Hematocrit or Packed cell volume (PCV) is the proportion of blood volume occupied by red blood cells and is typically about three times the hemoglobin concentration.

Platelet Count

Clinical Significance :
Platelets or thrombocytes are a cellular component of blood whose function is to stop bleeding by clumping or clotting blood vessel injuries. Low platelet count, also known as Thrombocytopenia, can be either due to less production or increased destruction of platelets. High platelet count or Thrombocytosis can be due to unregulated production, secondary to congenital, reactive or neoplastic conditions.

Complete Blood Count (CBC)

Clinical Significance :
CBC comprises of estimation of the cellular componenets of blood including RBCs, WBCs and Platelets. Mean corpuscular volume (MCV) is a measure of the size of the average RBC, MCH is a measure of the hemoglobin cointent of the average RBC and MCHC is the hemoglobin concentration per RBC. The red cell distribution width (RDW) is a measure of the degree of variation in RBC size (anisocytosis) and is helpful in distinguishing between some anemias. CBC examination is used as a screening tool to confirm a hematologic disorder, to establish or rule out a diagnosis, to detect an unsuspected hematologic disorder, or to monitor effects of radiation or chemotherapy. Abnormal results may be due to a primary disorder of the cell-producing organs or an underlying disease. Results should be interpreted in conjunction with the patient's clinical picture and appropriate additional testing performed.

Erythrocyte Sedimentation Rate (ESR)

Clinical Significance :
The erythrocyte sedimentation rate (ESR) is a simple but non-specific test that helps to detect inflammation associated with conditions such as

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infections, cancers, and autoimmune diseases.

-

Total Cholesterol

Clinical Significance :
Serum cholesterol is elevated in hereditary hyperlipoproteinemias and in other metabolic diseases. Moderate-to-markedly elevated values are also seen in cholestatic liver disease. Increased levels are a risk factor for cardiovascular disease. Low levels of cholesterol may be seen in disorders like hyperthyroidism, malabsorption, and deficiencies of apolipoproteins.

Triglycerides

Clinical Significance :
Triglycerides are partly synthesized in the liver and partly derived from the diet. Increased serum triglyceride levels are a risk factor for atherosclerosis. Hyperlipidemia may be inherited or may be due to conditions like biliary obstruction, diabetes mellitus, nephrotic syndrome, renal failure,certain metabolic disorders or drug induced.

HDL Cholesterol

Clinical Significance :
High-density lipoprotein (HDL) is an important tool used to assess risk of developing coronary heart disease.Increased levels are seen in persons with more physical activity. Very high levels are seen in case of metabolic response to medications like hormone replacement therapy.Raised levels are also seen in case of chronic intoxication with alcohol, heavy metals or industrial chemicals.Low HDL cholesterol correlates with increased risk for coronary heart disease (CHD). Very low levels are seen in Tangier disease, cholestatic liver disease and in association with decreased hepatocyte function.

Lipid Profile Direct

Proposed LDL-C goals in very high risk and extreme risk group patients by the Lipid Association of India.

Very High Risk group(VHRG)	Extreme Risk group	
	Category A	Category B
LDL-C goal of <50 mg/dl High-risk conditions	LDL-C goal of <50 mg/dl (recommended) LDL-C goal of ≤30 mg/dl (optional)	LDL-C goal of ≤30 mg/dl CAD with ≥1 of following:

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Test Name	Result	Biological Ref. Interval	Unit
Any one of following: 1. ASCVD (CAD/PAD/TIA or stroke) 2. Homozygous familial 3. hypercholesterolemia 4. Diabetes with ≥2 major ASCVD risk factors*/target organ damage	CAD with ≥1 of following: 1. Diabetes without target organ damage/≤1 major 2. ASCVD risk factors 3. Familial hypercholesterolemia 4. ≥3 major ASCVD risk factors 5. CKD stage 3B and 4 6. ≥2 major ASCVD risk factors with ≥1 moderate 7. non-conventional risk factor# 8. Lp(a) ≥50 mg/dl 9. Coronary calcium score ≥300 HU 10. Extreme of a single risk factor 11. PAD 12. H/o TIA or stroke 13. Non-stenotic carotid plaque	1. Diabetes + polyvascular disease/≥2 2. major ASCVD risk factors*/target organ 3. damage 4. Recurrent ACS (within 12 months) 5. despite on LDL-C goal 6. Homozygous familial 7. Hypercholesterolemia	

The LDL-C goal of ≤30 mg/dl must be pursued after detailed risk-benefit discussion between physician and patient.

Clinical judgment to be used in decision making if the patient has disease/risk factors not covered in the table, eg. peripheral arterial disease or cerebrovascular disease.

*Major ASCVD risk factors: 1. Age- male ≥45 years, female ≥55 years, 2. Family h/o premature CAD- male <55 years, female <65 years, 3. Smoking/tobacco use, 4. Systemic hypertension, 5.Low HDL (males <40 mg/dl and females <50 mg/dl).

#Moderate non-conventional risk factors: 1. Coronary calcium score 100-299 HU, 2. Increased carotid intima-media thickness, 3. Lp(a) ≥20-49 mg/dl, 4. Impaired fasting glucose, 5. Increased waist circumference, 6. Apolipoprotein B ≥ 110 mg/dl, 7. hsCRP ≥2 mg/L.

Bilirubin Total

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Sex	: Female	Sample Received on	:	14/01/2023 12:30:36
P. ID No.	: P1105200005123	Report Released on	:	15/01/2023 11:35:58
Accession No	**: 11052210131629**	Barcode No.	:	992789579, 994298582, 994298583, 994298584
Referring Doctor	: SELF			
Referred By	: Dr Rajesh Saxena C/O Pathkind CC Bhud Road	Ref no.	:	

Report Status - Final

Test Name	Result	Biological Ref. Interval	Unit

Clinical Significance :
"Total Bilirubin is one of the most commonly used tests to assess liver function. A number of inherited and acquired diseases affect bilirubin production, metabolism, storage and excretion and causes hyperbilirubinemia resulting in jaundice.Hyperbilirubinemia may be due to increased bilirubin production (eg, hemolysis and ineffective erythropoiesis), decreased bilirubin excretion (eg, obstruction and hepatitis), and abnormal bilirubin metabolism (eg, hereditary and neonatal jaundice). Unconjugated hyperbilirubinemia is seen in newborn andd known as physiological jaundice. Elevated unconjugated bilirubin in the neonatal period may result in brain damage (kernicterus).Crigler-Najjar syndromes type I and type II are also associated with elevated levels of indirect bilirubin.Both conjugated and unconjugated bilirubin are increased in hepatitis and space-occupying lesions of the liver; and obstructive lesions such as carcinoma of the head of the pancreas, common bile duct, or ampulla of Vater."

Bilirubin Direct

Clinical Significance :
"Direct bilirubin is a measurement of conjugated bilirubin.Jaundice can occur as a result of increased bilirubin production (eg, hemolysis and ineffective erythropoiesis), decreased bilirubin excretion (eg, obstruction and hepatitis), and abnormal bilirubin metabolism (eg, hereditary and neonatal jaundice). Inherited disorders in which direct bilirubin levels are increased are seen in Dubin-Johnson syndrome and Rotor syndrome,idiopathic neonatal hepatitis and biliary atresia. The most commonly occurring form of jaundice of the newborn called physiological jaundiceis due to increase in levels of indirect bilirubin.Both conjugated and unconjugated bilirubin are increased in hepatocellular diseases such as hepatitis and space-occupying lesions of the liver, bstructive lesions such as carcinoma of the head of the pancreas, common bile duct, or ampulla of Vater."

SGOT / AST

Clinical Significance :
"Elevated aspartate aminotransferase (AST) values are seen most commonly in parenchymal liver diseases. Values can be elevated from 10 to 100 times the normal range, though commonly 20 to 50 times elevations are seen. AST levels are raised in infectious hepatitis and other inflammatory conditions affecting the liver along with ALT, though ALT levels are higher. The ALT:AST ratio which is normally <1 is reversed in these conditions and becomes >1. AST levels are usually raised before clinical signs and symptoms of disease appear. AST and ALT also rise in primary or metastatic carcinoma of the liver, with AST usually being higher than ALT.Elevated AST values may also be seen in disorders affecting the heart, skeletal muscle and kidney, such as myocardial infarction, muscular dystrophy, dermatomyositis, acute pancreatitis and crushed muscle injuries."

SGPT / ALT

This Test/s marked with (#) is/are not accredited by NABL

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Report Status - Final

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Clinical Significance :
Elevated alanine aminotransferase (ALT) values are seen in parenchymal liver diseases characterized by a destruction of hepatocytes. Values are at least 10 times higher the normal range and may reach up to 100 times the upper reference limit. Commonly, values are seen to be 20 - 50 times higher than normal. In infectious hepatitis and other inflammatory conditions affecting the liver, ALT levels rise more than aspartate aminotransferase (AST), and the ALT/AST ratio, which is normally <1, is reversed and becomes >1. ALT levels usually rise before clinical signs and symptoms of disease appear.

Alkaline Phosphatase (ALP)

Clinical Significance :
Alkaline Phosphatase levels can be elevated in both liver related as well as bone related conditions. ALP levels are raised (more than 3 fold) in extrahepatic biliary obstruction (eg, by stone or by cancer of the head of the pancreas) than in intrahepatic obstruction, and is directly proportional to the level of obstruction. Levels may rise up to 10 to 12 times the upper limit of normal range and returns to normal on surgical removal of the obstruction. ALP levels rise together with GGT levels and If both GGT and ALP are elevated, a liver source of the ALP is likely. Among bone diseases, ALP levels rise in Paget disease (up to 25 fold),osteomalacia,rickets,primary and secondary hyperparathyroidism and osteogenic bone cancer. Elevated ALP is seen in children following accelerated bone growth. Also, a 2 to 3fold elevation may be observed in women in the third trimester of pregnancy, although the interval is very wide and levels may not exceed the upper limit of the reference interval in some cases.

Total Protein

Clinical Significance :
High levels of Serum Total Protein is seen in increased acute phase reactants in inflammation, late-stage liver disease, infections,multiple myeloma and other malignant paraproteinemias.n. Hypoproteinemia is seen in hypogammaglobulinemia, nephrotic syndrome and protein-losing enteropathy.

Albumin

Clinical Significance :
"Hypoalbuminemia can be caused by impaired synthesis due to liver disease (primary) or due to diminished protein intake (secondary), increased catabolism due to tissue damage and inflammation; malabsorption of amino acids; and increased renal excretion (eg, nephrotic syndrome).Hyperalbuminemia is seen in dehydration."

Blood Urea Nitrogen (BUN)

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Report Status - Final

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Clinical Significance :
Increased blood urea nitrogen (BUN) may be due to prerenal causes (cardiac decompensation, water depletion due to decreased intake and excessive loss, increased protein catabolism, and high protein diet), renal causes (acute glomerulonephritis, chronic nephritis, polycystic kidney disease, nephrosclerosis, and tubular necrosis) and postrenal causes (eg, all types of obstruction of the urinary tract, such as stones, enlarged prostate gland, tumors).

Creatinine

Clinical Significance :
Serum creatinine is inversely correlated with glomerular filtration rate (GFR). Increased levels of Serum Creatinine is associated with renal dysfunction.

Sodium

Clinical Significance :
Serum Sodium estimation is performed to assess acid-base balance, water balance, water intoxication, and dehydration.

Potassium

Clinical Significance :
Potassium (K+) is the major intracellular cation. It regulates neuromuscular excitability, heart contractility, intracellular fluid volume, and hydrogen ion concentration. High levels of serum Potassium is seen in acute renal disease and end-stage renal failure due to decreased excretion.Levels are also high during the diuretic phase of acute tubular necrosis, during administration of non-potassium sparing diuretic therapy, and during states of excess mineralocorticoid or glucocorticoid.

Chloride

Clinical Significance :
"Chloride (Cl) is the major extracellular anion and it has an important role in maintaining proper body water distribution, osmotic pressure, and normalanion-cation balance in the extracellular fluid compartment. Chloride is increased in dehydration, renal tubular acidosis, acute renal failure, metabolic acidosis associated with prolonged diarrhea and loss of sodium bicarbonate, diabetes insipidus, adrenocortical hyperfuction,salicylate

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intoxication and with excessive infusion of isotonic saline or extremely high dietary intake of salt. Hyperchloremia acidosis may be a sign of severe renal tubular pathology. Chloride is decreased inoverhydration, chronic respiratory acidosis, salt-losing nephritis, metabolic alkalosis, congestive heart failure, Addisonian crisis, certain types of metabolic acidosis, persistent gastric secretion and prolonged vomiting, aldosteronism, bromide intoxication,

Calcium

Serum Calcium levels are used to monitor and diagnose a wide range of diseases of bone, kidney, parathyroid gland, or gastrointestinal tract. Calcium levels may also reflect abnormal vitamin D or protein levels. Hypocalcemia or low serum calcium levels is associated with absent or decreased function of the parathyroid glands, impaired vitamin-D synthesis, low dietary intake and chronic renal failure. Hypercalcemia is due to increased mobilization of calcium from the skeletal system or increased intestinal absorption.It is usually seen in case of primary hyperparathyroidism (pHPT) or bone metastasis of carcinoma of the breast, prostate, thyroid gland, or lung.

Hepatitis B Surface Antigen (HBsAg)

Clinical Significance :
Hepatitis B surface antigen (HBsAg) is the first serologic marker appearing in the serum at 6 to 16 weeks following exposure to HBV. In acute infection, HBsAg usually disappears in 1 to 2 months after the onset of symptoms. Persistence of HBsAg for more than 6 months in duration indicates development of either a chronic carrier state or chronic HBV infection.

In case of negative results:
Please note that while rapid test is a sensitive and reliable screening test, it should not be used as a sole criterion for diagnosis. It is recommended to use molecular testing (PCR) for confirmation.

In case of positive results:
The test has been performed on two different rapid technologies. Please note that while rapid test is a sensitive and reliable screening test, it should not be used as a sole criterion for diagnosis. It is recommended to use molecular testing (PCR) for confirmation.

Urine Routine & Microscopic Examination