Hiranandani
HOSPITAL
(A Fortis Network Hospital)

Hiranandani Fortis Hospital
Mini Seashore Road,
Sector 10 - A, Vashi,
Navi Mumbai - 400 703.
Tel. : +91-22-3919 9222
Fax : +91-22-3919 9220/21
Email : vashi@vashihospital.com

BMI CHART

Date: 12/3/24

Name: Hiren Parmar Age: 32 yrs Sex: M / F

BP: 130/80 Height (cms): 5.11 Weight(kgs): 78.5 BMI: 23

SPO2 - 98%.
Pulse - 82

WEIGHT lbs	100	105	100	115	120	125	130	135	140	145	150	155	160	165	170	175	180	185	190	195	200	205	210	215
kgs	45.5	47.7	50.50	52.3	54.5	56.8	59.1	61.4	63.6	65.9	68.2	70.5	72.7	75.0	77.3	79.5	81.8	84.1	86.4	88.6	90.9	93.2	95.5	97.7
HEIGHT in/cm	Underweight					Healthy					Overweight					Obese				Extremely Obese				
5'0" - 152.4	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42
5'1" - 154.9	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	36	37	38	39	40
5'2" - 157.4	18	19	20	21	22	22	23	24	25	26	27	28	29	30	31	32	33	33	34	35	36	37	38	39
5'3" - 160.0	17	18	19	20	21	22	23	24	24	25	26	27	28	29	30	31	32	32	33	34	35	36	37	38
5'4" - 162.5	17	18	18	19	20	21	22	23	24	24	25	26	27	28	29	30	31	31	32	33	34	35	36	37
5'5" - 165.1	16	17	18	19	20	20	21	22	23	24	25	25	26	27	28	29	30	30	31	32	33	34	35	35
5'6" - 167.6	16	17	17	18	19	20	21	21	22	23	24	25	25	26	27	28	29	29	30	31	32	33	34	34
5'7" - 170.1	15	16	17	18	18	19	20	21	22	22	23	24	25	25	26	27	28	29	29	30	31	32	33	33
5'8" - 172.7	15	16	16	17	18	19	19	20	21	22	22	23	24	25	25	26	27	28	28	29	30	31	32	32
5'9" - 176.2	14	15	16	17	17	18	19	20	20	21	22	22	23	24	25	25	26	27	28	28	29	30	31	31
5'10" - 177.8	14	15	15	16	17	18	18	19	20	20	21	22	23	23	24	25	25	26	27	28	28	29	30	30
5'11" - 180.3	14	14	15	16	16	17	18	18	19	20	21	21	22	23	23	24	25	25	26	27	28	28	29	30
6'0" - 182.8	13	14	14	15	16	17	17	18	19	19	20	21	21	22	23	23	24	25	25	26	27	27	28	29
6'1" - 185.4	13	13	14	15	15	16	17	17	18	19	19	20	21	21	22	23	23	24	25	25	26	27	27	28
6'2" - 187.9	12	13	14	14	15	16	16	17	18	18	19	19	20	21	21	22	23	23	24	25	25	26	27	27
6'3" - 190.5	12	13	13	14	15	15	16	16	17	18	18	19	20	20	21	21	22	23	23	24	25	25	26	26
6'4" - 193.0	12	12	13	14	14	15	15	16	17	17	18	18	19	20	20	21	22	22	23	23	24	25	25	26

Doctors Notes:

Signature

Hiranandani Healthcare Pvt. Ltd.
Mini Sea Shore Road, Sector 10 -A, Vashi, Navi Mumbai - 400703
Board Line: 022 - 39199222 | Fax: 022 - 39199220
Emergency: 022 - 39199100 | Ambulance: 1255
For Appointment: 022 - 39199222 | Health Checkup: 022 - 39199300
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CIN : U85100MH2005PTC154823
GST IN: 27AABCH5894D1ZG | PAN NO: AABCH5894D

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UHID	**13026337**	**Date**	**12/03/2024**		
Name	**Mr Hiren Parmar**	**Sex**	**M**	**Age**	**32**
OPD	**Opthal**	**Health Check-Up**			

Drug allergy: → Not known.
Sys illness: → No
Habit: → No.

Cln. No.

HO No

RE → 6/6.
LE → 6/6.

RE → Phree 6/6.
LE → Phree 6/6
RE N6
NV → CC N6

IOP → RE → 14.8.
→ LE → 15.3

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UHID	**13026337**	**Date**	**12/03/2024**		
Name	**Mr Hiren Parmar**	**Sex**	**M**	**Age**	**32**
OPD	**Dental**	**Health Check-Up**			

O/E - Stains +
- Calculus +
- Impacted $\bar{c}$ $\frac{8|8}{8|8}$

~~Rx~~

Drug allergy:
Sys illness:

Treatment
Ald - (1) Scaling Grade I
(2) OPG (Xray)
(3) Extraction $\bar{c}$ $\frac{-|-}{8|8}$

Dr. Trupti

Fortis

MC-5837

agilus diagnostics

PATIENT NAME : MR.HIREN KARSHANBHAI PARMAR **REF. DOCTOR :**

CODE/NAME & ADDRESS : C000045507 FORTIS VASHI-CHC -SPLZD FORTIS HOSPITAL # VASHI, MUMBAI 440001	ACCESSION NO : **0022XC002288** PATIENT ID : FH.13026337 CLIENT PATIENT ID: UID:13026337 ABHA NO :	AGE/SEX :32 Years Male DRAWN :12/03/2024 09:04:00 RECEIVED : 12/03/2024 09:10:02 REPORTED :12/03/2024 14:23:45

CLINICAL INFORMATION :

UID:13026337 REQNO-1675211
CORP-OPD
BILLNO-150124OPCR014358
BILLNO-150124OPCR014358

Test Report Status **Final**	Results	Biological Reference Interval	Units
HAEMATOLOGY - CBC			
CBC-5, EDTA WHOLE BLOOD			
BLOOD COUNTS, EDTA WHOLE BLOOD			
HEMOGLOBIN (HB) METHOD : SLS METHOD	14.7	13.0 - 17.0	g/dL
RED BLOOD CELL (RBC) COUNT METHOD : HYDRODYNAMIC FOCUSING	4.91	4.5 - 5.5	mil/µL
WHITE BLOOD CELL (WBC) COUNT METHOD : FLUORESCENCE FLOW CYTOMETRY	5.64	4.0 - 10.0	thou/µL
PLATELET COUNT METHOD : HYDRODYNAMIC FOCUSING BY DC DETECTION	294	150 - 410	thou/µL
RBC AND PLATELET INDICES			
HEMATOCRIT (PCV) METHOD : CUMULATIVE PULSE HEIGHT DETECTION METHOD	45.9	40.0 - 50.0	%
MEAN CORPUSCULAR VOLUME (MCV) METHOD : CALCULATED PARAMETER	93.5	83.0 - 101.0	fL
MEAN CORPUSCULAR HEMOGLOBIN (MCH) METHOD : CALCULATED PARAMETER	29.9	27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION(MCHC) METHOD : CALCULATED PARAMETER	32.0	31.5 - 34.5	g/dL
RED CELL DISTRIBUTION WIDTH (RDW) METHOD : CALCULATED PARAMETER	**11.1 Low**	11.6 - 14.0	%
MENTZER INDEX METHOD : CALCULATED PARAMETER	19.0		
MEAN PLATELET VOLUME (MPV) METHOD : CALCULATED PARAMETER	9.4	6.8 - 10.9	fL
WBC DIFFERENTIAL COUNT			

Dr. Akshay Dhotre, MD
(Reg,no. MMC 2019/09/6377)
Consultant Pathologist

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Patient Ref. No. 22000000908195

Diagnostics Report

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NEUTROPHILS METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	50	40.0 - 80.0	%
LYMPHOCYTES METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	**42 High**	20.0 - 40.0	%
MONOCYTES METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	6	2.0 - 10.0	%
EOSINOPHILS METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	2	1 - 6	%
BASOPHILS METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	0	0 - 2	%
ABSOLUTE NEUTROPHIL COUNT METHOD : CALCULATED PARAMETER	2.82	2.0 - 7.0	thou/µL
ABSOLUTE LYMPHOCYTE COUNT METHOD : CALCULATED PARAMETER	2.37	1.0 - 3.0	thou/µL
ABSOLUTE MONOCYTE COUNT METHOD : CALCULATED PARAMETER	0.34	0.2 - 1.0	thou/µL
ABSOLUTE EOSINOPHIL COUNT METHOD : CALCULATED PARAMETER	0.11	0.02 - 0.50	thou/µL
ABSOLUTE BASOPHIL COUNT METHOD : CALCULATED PARAMETER	**0.00 Low**	0.02 - 0.10	thou/µL
NEUTROPHIL LYMPHOCYTE RATIO (NLR) METHOD : CALCULATED	1.2		

MORPHOLOGY

RBC METHOD : MICROSCOPIC EXAMINATION	PREDOMINANTLY NORMOCYTIC NORMOCHROMIC
WBC METHOD : MICROSCOPIC EXAMINATION	NORMAL MORPHOLOGY
PLATELETS METHOD : MICROSCOPIC EXAMINATION	ADEQUATE

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Interpretation(s)
RBC AND PLATELET INDICES-Mentzer index (MCV/RBC) is an automated cell-counter based calculated screen tool to differentiate cases of Iron deficiency anaemia(>13) from Beta thalassaemia trait
(<13) in patients with microcytic anaemia. This needs to be interpreted in line with clinical correlation and suspicion. Estimation of HbA2 remains the gold standard for diagnosing a case of beta thalassaemia trait.
WBC DIFFERENTIAL COUNT-The optimal threshold of 3.3 for NLR showed a prognostic possibility of clinical symptoms to change from mild to severe in COVID positive patients. When age = 49.5 years old and NLR = 3.3, 46.1% COVID-19 patients with mild disease might become severe. By contrast, when age < 49.5 years old and NLR < 3.3, COVID-19 patients tend to show mild disease.
(Reference to - The diagnostic and predictive role of NLR, d-NLR and PLR in COVID-19 patients ; A.-P. Yang, et al.; International Immunopharmacology 84 (2020) 106504
This ratio element is a calculated parameter and out of NABL scope.

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HAEMATOLOGY			
ERYTHROCYTE SEDIMENTATION RATE (ESR),EDTA BLOOD			
E.S.R METHOD : WESTERGREN METHOD	**22 High**	0 - 14	mm at 1 hr
GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD			
HBA1C	4.4	Non-diabetic: < 5.7 Pre-diabetics: 5.7 - 6.4 Diabetics: > or = 6.5 Therapeutic goals: < 7.0 Action suggested : > 8.0 (ADA Guideline 2021)	%
METHOD : HB VARIANT (HPLC) ESTIMATED AVERAGE GLUCOSE(EAG) METHOD : CALCULATED PARAMETER	79.6	< 116.0	mg/dL

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)

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Patient Ref. No. 2200000090819

Diagnostics Report

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

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.
GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD-**Used For:**

1. Evaluating the long-term control of blood glucose concentrations in diabetic patients.
2. Diagnosing diabetes.
3. Identifying patients at increased risk for diabetes (prediabetes).

The ADA recommends measurement of HbA1c (typically 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 determine whether a patients metabolic control has remained continuously within the target range.
1. eAG (Estimated average glucose) converts percentage HbA1c to md/dl, to compare blood glucose levels.
2. eAG gives an evaluation of blood glucose levels for the last couple of months.
3. eAG is calculated as eAG (mg/dl) = 28.7 * HbA1c - 46.7

HbA1c Estimation can get affected due to :
1. Shortened Erythrocyte survival : Any condition that shortens erythrocyte survival or decreases mean erythrocyte age (e.g. recovery from acute blood loss,hemolytic anemia) will falsely lower HbA1c test results.Fructosamine is recommended in these patients which indicates diabetes control over 15 days.
2.Vitamin C & E are reported to falsely lower test results.(possibly by inhibiting glycation of hemoglobin.
3. Iron deficiency anemia is reported to increase test results. Hypertriglyceridemia,uremia, hyperbilirubinemia, chronic alcoholism,chronic ingestion of salicylates & opiates addiction are reported to interfere with some assay methods,falsely increasing results.
4. Interference of hemoglobinopathies in HbA1c estimation is seen in

a) Homozygous hemoglobinopathy. Fructosamine is recommended for testing of HbA1c.
b) Heterozygous state detected (D10 is corrected for HbS & HbC trait.)
c) HbF > 25% on alternate paltform (Boronate affinity chromatography) is recommended for testing of HbA1c.Abnormal Hemoglobin electrophoresis (HPLC method) is recommended for detecting a hemoglobinopathy

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Patient Ref. No. 22000000908195

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IMMUNOHAEMATOLOGY

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD

ABO GROUP METHOD : TUBE AGGLUTINATION	TYPE B		
RH TYPE METHOD : TUBE AGGLUTINATION	POSITIVE		

Interpretation(s)
ABO GROUP & RH TYPE, EDTA WHOLE BLOOD-Blood group is identified by antigens and antibodies present in the blood. Antigens are protein molecules found on the surface of red blood cells. Antibodies are found in plasma. To determine blood group, red cells are mixed with different antibody solutions to give A,B,O or AB.

Disclaimer: "Please note, as the results of previous ABO and Rh group (Blood Group) for pregnant women are not available, please check with the patient records for availability of the same."

The test is performed by both forward as well as reverse grouping methods.

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BIOCHEMISTRY			
LIVER FUNCTION PROFILE, SERUM			
BILIRUBIN, TOTAL	0.73	0.2 - 1.0	mg/dL
METHOD : JENDRASSIK AND GROFF			
BILIRUBIN, DIRECT	0.18	0.0 - 0.2	mg/dL
METHOD : JENDRASSIK AND GROFF			
BILIRUBIN, INDIRECT	0.55	0.1 - 1.0	mg/dL
METHOD : CALCULATED PARAMETER			
TOTAL PROTEIN	7.7	6.4 - 8.2	g/dL
METHOD : BIURET			
ALBUMIN	3.9	3.4 - 5.0	g/dL
METHOD : BCP DYE BINDING			
GLOBULIN	3.8	2.0 - 4.1	g/dL
METHOD : CALCULATED PARAMETER			
ALBUMIN/GLOBULIN RATIO	1.0	1.0 - 2.1	RATIO
METHOD : CALCULATED PARAMETER			
ASPARTATE AMINOTRANSFERASE(AST/SGOT)	17	15 - 37	U/L
METHOD : UV WITH P5P			
ALANINE AMINOTRANSFERASE (ALT/SGPT)	**46 High**	< 45.0	U/L
METHOD : UV WITH P5P			
ALKALINE PHOSPHATASE	90	30 - 120	U/L
METHOD : PNPP-ANP			
GAMMA GLUTAMYL TRANSFERASE (GGT)	44	15 - 85	U/L
METHOD : GAMMA GLUTAMYLCARBOXY 4NITROANILIDE			
LACTATE DEHYDROGENASE	124	85 - 227	U/L
METHOD : LACTATE -PYRUVATE			
GLUCOSE FASTING,FLUORIDE PLASMA			
FBS (FASTING BLOOD SUGAR)	95	Normal : < 100 Pre-diabetes: 100-125 Diabetes: >/=126	mg/dL
METHOD : HEXOKINASE			

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CORP-OPD
BILLNO-150124OPCR014358
BILLNO-150124OPCR014358

Test Report Status **Final**	Results	Biological Reference Interval	Units
KIDNEY PANEL - 1			
BLOOD UREA NITROGEN (BUN), SERUM			
BLOOD UREA NITROGEN METHOD : UREASE - UV	6	6 - 20	mg/dL
CREATININE EGFR- EPI			
CREATININE METHOD : ALKALINE PICRATE KINETIC JAFFES	**0.76 Low**	0.90 - 1.30	mg/dL
AGE	32		years
GLOMERULAR FILTRATION RATE (MALE) METHOD : CALCULATED PARAMETER	122.47	Refer Interpretation Below	mL/min/1.73m2
BUN/CREAT RATIO			
BUN/CREAT RATIO METHOD : CALCULATED PARAMETER	7.89	5.00 - 15.00	
URIC ACID, SERUM			
URIC ACID METHOD : URICASE UV	**3.4 Low**	3.5 - 7.2	mg/dL
TOTAL PROTEIN, SERUM			
TOTAL PROTEIN METHOD : BIURET	7.7	6.4 - 8.2	g/dL

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Fortis

MC-5837

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PATIENT NAME : MR.HIREN KARSHANBHAI PARMAR **REF. DOCTOR :**

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ALBUMIN, SERUM			
ALBUMIN METHOD : BCP DYE BINDING	3.9	3.4 - 5.0	g/dL
GLOBULIN			
GLOBULIN METHOD : CALCULATED PARAMETER	3.8	2.0 - 4.1	g/dL
ELECTROLYTES (NA/K/CL), SERUM			
SODIUM, SERUM METHOD : ISE INDIRECT	139	136 - 145	mmol/L
POTASSIUM, SERUM METHOD : ISE INDIRECT	4.57	3.50 - 5.10	mmol/L
CHLORIDE, SERUM METHOD : ISE INDIRECT	103	98 - 107	mmol/L

Interpretation(s)

Interpretation(s)
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 yellow discoloration in jaundice.**Elevated levels** results from increased bilirubin production (eg, hemolysis and ineffective erythropoiesis), decreased bilirubin excretion (eg, obstruction and hepatitis), and abnormal bilirubin metabolism (eg, hereditary and neonatal jaundice). Conjugated (direct) bilirubin is elevated more than unconjugated (indirect) bilirubin in Viral hepatitis, Drug reactions, Alcoholic liver disease Conjugated (direct) bilirubin is also elevated more than unconjugated (indirect) bilirubin when there is some kind of blockage of the bile ducts like in Gallstones getting into the bile ducts, tumors &Scarring of the bile ducts. Increased unconjugated (indirect) bilirubin may be a result of Hemolytic or pernicious anemia, Transfusion reaction & a common metabolic condition termed Gilbert syndrome, due to low levels of the enzyme that attaches sugar molecules to bilirubin.

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AST is an enzyme found in various parts of the body. AST is found in the liver, heart, skeletal muscle, kidneys, brain, and red blood cells, and it is commonly measured clinically as a marker for liver health. AST levels increase during chronic viral hepatitis, blockage of the bile duct, cirrhosis of the liver,liver cancer,kidney failure,hemolytic anemia,pancreatitis,hemochromatosis. AST levels may also increase after a heart attack or strenuous activity.ALT test measures the amount of this enzyme in the blood.ALT is found mainly in the liver, but also in smaller amounts in the kidneys,heart,muscles, and pancreas.It is commonly measured as a part of a diagnostic evaluation of hepatocellular injury, to determine liver health.AST levels increase during acute hepatitis,sometimes due to a viral infection,ischemia to the liver,chronic hepatitis,obstruction of bile ducts,cirrhosis.

ALP is a protein found in almost all body tissues.Tissues with higher amounts of ALP include the liver,bile ducts and bone.Elevated ALP levels are seen in Biliary obstruction, Osteoblastic bone tumors, osteomalacia, hepatitis, Hyperparathyroidism, Leukemia, Lymphoma, Pagets disease,Rickets,Sarcoidosis etc. Lower-than-normal ALP levels seen in Hypophosphatasia,Malnutrition,Protein deficiency,Wilsons disease.

GGT is an enzyme found in cell membranes of many tissues mainly in the liver,kidney and pancreas.It is also found in other tissues including intestine,spleen,heart, brain and seminal vesicles.The highest concentration is in the kidney,but the liver is considered the source of normal enzyme activity.Serum GGT has been widely used as an index of liver dysfunction.Elevated serum GGT activity can be found in diseases of the liver,biliary system and pancreas.Conditions that increase serum GGT are obstructive liver disease,high alcohol consumption and use of enzyme-inducing drugs etc.

Total Protein also known as total protein,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 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

GLUCOSE FASTING,FLUORIDE PLASMA-**TEST DESCRIPTION**
Normally, the glucose concentration in extracellular fluid is closely regulated so that a source of energy is readily available to tissues and sothat no glucose is excreted in the urine.
Increased in:Diabetes mellitus, Cushing' s syndrome (10 – 15%), chronic pancreatitis (30%). Drugs:corticosteroids,phenytoin, estrogen, thiazides.
Decreased in :Pancreatic islet cell disease with increased insulin,insulinoma,adrenocortical insufficiency,hypopituitarism,diffuse liver disease, malignancy(adrenocortical,stomach,fibrosarcoma),infant of a diabetic mother,enzyme deficiency diseases(e.g.galactosemia),Drugs-insulin,ethanol,propranolol;sulfonylureas,tolbutamide,and other oral hypoglycemic agents.
NOTE: While random serum glucose levels correlate with home glucose monitoring results (weekly mean capillary glucose values),there is wide fluctuation within individuals.Thus, glycosylated hemoglobin(HbA1c) levels are favored to monitor glycemic control.
High fasting glucose level in comparison to post prandial glucose level may be seen due to effect of Oral Hypoglycaemics & Insulin treatment,Renal Glyosuria,Glycaemic index & response to food consumed,Alimentary Hypoglycemia,Increased insulin response & sensitivity etc.
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 EGFR- EPI-- Kidney disease outcomes quality initiative (KDOQI) guidelines state that estimation of GFR is the best overall indices of the Kidney function.
- It gives a rough measure of number of functioning nephrons .Reduction in GFR implies progression of underlying disease.
- The GFR is a calculation based on serum creatinine test.
- Creatinine is mainly derived from the metabolism of creatine in muscle, and its generation is proportional to the total muscle mass. As a result, mean creatinine generation is higher in men than in women, in younger than in older individuals, and in blacks than in whites.
- Creatinine is filtered from the blood by the kidneys and excreted into urine at a relatively steady rate.
- When kidney function is compromised, excretion of creatinine decreases with a consequent increase in blood creatinine levels. With the creatinine test, a reasonable estimate of the actual GFR can be determined.
- This equation takes into account several factors that impact creatinine production, including age, gender, and race.
- CKD EPI (Chronic kidney disease epidemiology collaboration) equation performed better than MDRD equation especially when GFR is high(>60 ml/min per 1.73m2).. This formula has less bias and greater accuracy which helps in early diagnosis and also reduces the rate of false positive diagnosis of CKD.

References:

National Kidney Foundation (NKF) and the American Society of Nephrology (ASN).
Estimated GFR Calculated Using the CKD-EPI equation-https://testguide.labmed.uw.edu/guideline/egfr
Ghuman JK, et al. Impact of Removing Race Variable on CKD Classification Using the Creatinine-Based 2021 CKD-EPI Equation. Kidney Med 2022, 4:100471. 35756325
Harrison"s Principle of Internal Medicine, 21st ed. pg 62 and 334
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.

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

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.

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Test Report Status **Final**	Results	Biological Reference Interval	Units
BIOCHEMISTRY - LIPID			
LIPID PROFILE, SERUM			
CHOLESTEROL, TOTAL	151	< 200 Desirable 200 - 239 Borderline High >/= 240 High	mg/dL
METHOD : ENZYMATIC/COLORIMETRIC,CHOLESTEROL OXIDASE, ESTERASE, PEROXIDASE			
TRIGLYCERIDES	83	< 150 Normal 150 - 199 Borderline High 200 - 499 High >/=500 Very High	mg/dL
METHOD : ENZYMATIC ASSAY			
HDL CHOLESTEROL	44	< 40 Low >/=60 High	mg/dL
METHOD : DIRECT MEASURE - PEG			
LDL CHOLESTEROL, DIRECT	91	< 100 Optimal 100 - 129 Near or above optimal 130 - 159 Borderline High 160 - 189 High >/= 190 Very High	mg/dL
METHOD : DIRECT MEASURE WITHOUT SAMPLE PRETREATMENT			
NON HDL CHOLESTEROL	107	Desirable: Less than 130 Above Desirable: 130 - 159 Borderline High: 160 - 189 High: 190 - 219 Very high: > or = 220	mg/dL
METHOD : CALCULATED PARAMETER			
VERY LOW DENSITY LIPOPROTEIN	16.6	</= 30.0	mg/dL
METHOD : CALCULATED PARAMETER			
CHOL/HDL RATIO	3.4	3.3 - 4.4 Low Risk 4.5 - 7.0 Average Risk 7.1 - 11.0 Moderate Risk > 11.0 High Risk	
METHOD : CALCULATED PARAMETER			

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LDL/HDL RATIO	2.1	0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate Risk >6.0 High Risk	

METHOD : CALCULATED PARAMETER

Interpretation(s)

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CLINICAL PATH - URINALYSIS

KIDNEY PANEL - 1

PHYSICAL EXAMINATION, URINE

COLOR	PALE YELLOW		
METHOD : PHYSICAL			
APPEARANCE	CLEAR		
METHOD : VISUAL			

CHEMICAL EXAMINATION, URINE

PH	6.0	4.7 - 7.5
METHOD : REFLECTANCE SPECTROPHOTOMETRY- DOUBLE INDICATOR METHOD		
SPECIFIC GRAVITY	<=1.005	1.003 - 1.035
METHOD : REFLECTANCE SPECTROPHOTOMETRY (APPARENT PKA CHANGE OF PRETREATED POLYELECTROLYTES IN RELATION TO IONIC CONCENTRATION)		
PROTEIN	NOT DETECTED	NOT DETECTED
METHOD : REFLECTANCE SPECTROPHOTOMETRY - PROTEIN-ERROR-OF-INDICATOR PRINCIPLE		
GLUCOSE	NOT DETECTED	NOT DETECTED
METHOD : REFLECTANCE SPECTROPHOTOMETRY, DOUBLE SEQUENTIAL ENZYME REACTION-GOD/POD		
KETONES	NOT DETECTED	NOT DETECTED
METHOD : REFLECTANCE SPECTROPHOTOMETRY, ROTHERA'S PRINCIPLE		
BLOOD	NOT DETECTED	NOT DETECTED
METHOD : REFLECTANCE SPECTROPHOTOMETRY, PEROXIDASE LIKE ACTIVITY OF HAEMOGLOBIN		
BILIRUBIN	NOT DETECTED	NOT DETECTED
METHOD : REFLECTANCE SPECTROPHOTOMETRY, DIAZOTIZATION- COUPLING OF BILIRUBIN WITH DIAZOTIZED SALT		
UROBILINOGEN	NORMAL	NORMAL
METHOD : REFLECTANCE SPECTROPHOTOMETRY (MODIFIED EHRLICH REACTION)		
NITRITE	NOT DETECTED	NOT DETECTED
METHOD : REFLECTANCE SPECTROPHOTOMETRY, CONVERSION OF NITRATE TO NITRITE		
LEUKOCYTE ESTERASE	NOT DETECTED	NOT DETECTED
METHOD : REFLECTANCE SPECTROPHOTOMETRY, ESTERASE HYDROLYSIS ACTIVITY		

Rekha. N

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Diagnostics Report

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MICROSCOPIC EXAMINATION, URINE			
RED BLOOD CELLS	NOT DETECTED	NOT DETECTED	/HPF
METHOD : MICROSCOPIC EXAMINATION			
PUS CELL (WBC'S)	2-3	0-5	/HPF
METHOD : MICROSCOPIC EXAMINATION			
EPITHELIAL CELLS	0-1	0-5	/HPF
METHOD : MICROSCOPIC EXAMINATION			
CASTS	NOT DETECTED		
METHOD : MICROSCOPIC EXAMINATION			
CRYSTALS	NOT DETECTED		
METHOD : MICROSCOPIC EXAMINATION			
BACTERIA	NOT DETECTED	NOT DETECTED	
METHOD : MICROSCOPIC EXAMINATION			
YEAST	NOT DETECTED	NOT DETECTED	
METHOD : MICROSCOPIC EXAMINATION			
REMARKS	URINARY MICROSCOPIC EXAMINATION DONE ON URINARY CENTRIFUGED SEDIMENT		

Interpretation(s)

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SPECIALISED CHEMISTRY - HORMONE			
THYROID PANEL, SERUM			
T3	153.8	80.0 - 200.0	ng/dL
METHOD : ELECTROCHEMILUMINESCENCE IMMUNOASSAY, COMPETITIVE PRINCIPLE			
T4	8.31	5.10 - 14.10	µg/dL
METHOD : ELECTROCHEMILUMINESCENCE IMMUNOASSAY, COMPETITIVE PRINCIPLE			
TSH (ULTRASENSITIVE)	1.000	0.270 - 4.200	µIU/mL
METHOD : ELECTROCHEMILUMINESCENCE,SANDWICH IMMUNOASSAY			

Interpretation(s)

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SPECIALISED CHEMISTRY - TUMOR MARKER

PROSTATE SPECIFIC ANTIGEN, SERUM

PROSTATE SPECIFIC ANTIGEN	0.489	0.0 - 1.4	ng/mL

METHOD : ELECTROCHEMILUMINESCENCE,SANDWICH IMMUNOASSAY

Interpretation(s)
PROSTATE SPECIFIC ANTIGEN, SERUM-- PSA is detected in the male patients with normal, benign hyperplastic and malignant prostate tissue and in patients with prostatitis.
- PSA is not detected (or detected at very low levels) in the patients without prostate tissue (because of radical prostatectomy or cystoprostatectomy) and also in the female patients.
- It a suitable marker for monitoring of patients with Prostate Cancer and it is better to be used in conjunction with other diagnostic procedures.
- Serial PSA levels can help determine the success of prostatectomy and the need for further treatment, such as radiation, endocrine or chemotherapy and useful in detecting residual disease and early recurrence of tumor.
- Elevated levels of PSA can be also observed in the patients with non-malignant diseases like Prostatitis and Benign Prostatic Hyperplasia.
- Specimens for total PSA assay should be obtained before biopsy, prostatectomy or prostatic massage, since manipulation of the prostate gland may lead to elevated PSA (false positive) levels persisting up to 3 weeks.
- As per American urological guidelines, PSA screening is recommended for early detection of Prostate cancer above the age of 40 years. Following Age specific reference range can be used as a guide lines.
- Measurement of total PSA alone may not clearly distinguish between benign prostatic hyperplasia (BPH) from cancer, this is especially true for the total PSA values between 4-10 ng/mL.
- Total PSA values determined on patient samples by different testing procedures cannot be directly compared with one another and could be the cause of erroneous medical interpretations. Recommended follow up on same platform as patient result can vary due to differences in assay method and reagent specificity.

References-
1. Burtis CA, Ashwood ER, Bruns DE. Teitz textbook of clinical chemistry and Molecular Diagnostics. 4th edition.
2. Williamson MA, Snyder LM. Wallach's interpretation of diagnostic tests. 9th edition.

****End Of Report****
Please visit www.agilusdiagnostics.com for related Test Information for this accession

Dr. Akshay Dhotre, MD
(Reg,no. MMC 2019/09/6377)
Consultant Pathologist

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Navi Mumbai, 400703
Maharashtra, India
Tel : 022-39199222,022-49723322,
CIN - U74899PB1995PLC045956
Email : -

Patient Ref. No. 22000000908195

Diagnostics Report

Fortis

MC-5837

agilus diagnostics

PATIENT NAME : MR.HIREN KARSHANBHAI PARMAR **REF. DOCTOR :**

CODE/NAME & ADDRESS : C000045507 FORTIS VASHI-CHC -SPLZD FORTIS HOSPITAL # VASHI, MUMBAI 440001	ACCESSION NO : **0022XC002365** PATIENT ID : FH.13026337 CLIENT PATIENT ID: UID:13026337 ABHA NO :	AGE/SEX :32 Years Male DRAWN :12/03/2024 12:01:00 RECEIVED : 12/03/2024 12:01:55 REPORTED :12/03/2024 13:06:11

CLINICAL INFORMATION :

UID:13026337 REQNO-1675211
CORP-OPD
BILLNO-150124OPCR014358
BILLNO-150124OPCR014358

Test Report Status **Final**	Results	Biological Reference Interval	Units
BIOCHEMISTRY			
GLUCOSE, POST-PRANDIAL, PLASMA			
PPBS(POST PRANDIAL BLOOD SUGAR) METHOD : HEXOKINASE	70	70 - 140	mg/dL

Interpretation(s)
GLUCOSE, POST-PRANDIAL, PLASMA-High fasting glucose level in comparison to post prandial glucose level may be seen due to effect of Oral Hypoglycaemics & Insulin treatment, Renal Glyosuria, Glycaemic index & response to food consumed, Alimentary Hypoglycemia, Increased insulin response & sensitivity etc.Additional test HbA1c

****End Of Report****
Please visit www.agilusdiagnostics.com for related Test Information for this accession

Dr. Akshay Dhotre, MD
(Reg,no. MMC 2019/09/6377)
Consultant Pathologist

View Details View Repo

Patient Ref. No. 22000000908272

PERFORMED AT :
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Maharashtra, India
Tel : 022-39199222,022-49723322,
CIN - U74899PB1995PLC045956
Email : -

32 Years

HIREN PARMAR
Male

3/12/2024 10:54:46 AM

HC

Normal

Rate	74	. Sinus rhythm.....normal P axis, V-rate 50- 99
		. Consider left atrial enlargement.....wide or notched P waves
PR	151	. Borderline T wave abnormalities.....T/QRS ratio < 1/20 or flat T
QRSD	90	
QT	373	
QTc	414	

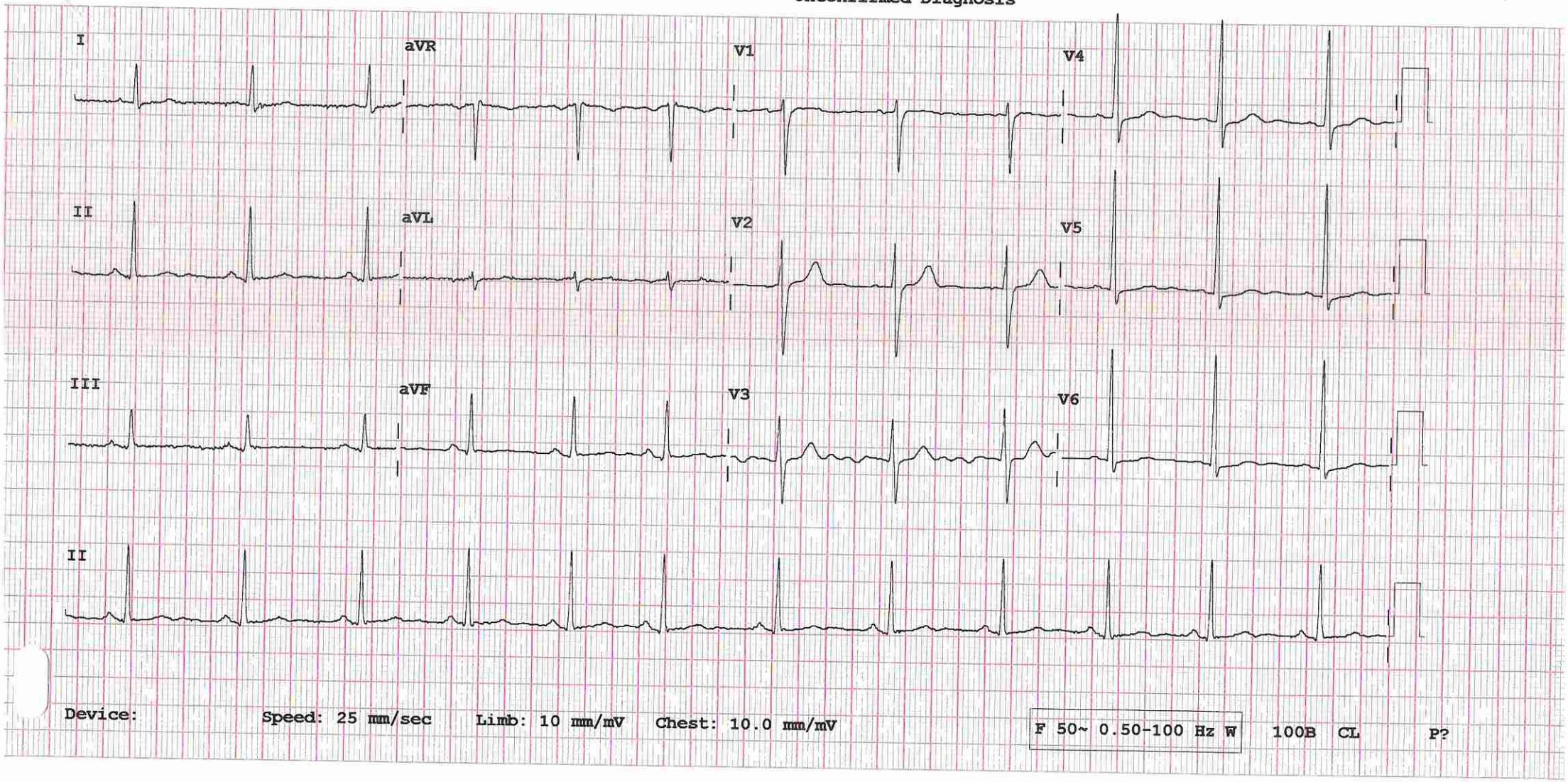
--AXIS--

P	82
QRS	68
T	41

12 Lead; Standard Placement

- BORDERLINE ECG -

Unconfirmed Diagnosis



Device:

Speed: 25 mm/sec Limb: 10 mm/mV Chest: 10.0 mm/mV

F 50~ 0.50-100 Hz W 100B CL P?

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CIN: U85100MH2005PTC 154823
GST IN : 27AABCH5894D1ZG
PAN NO : AABCH5894D

Hiranandani HOSPITAL (A Fortis Network Hospital)

DEPARTMENT OF NIC

Date: 12/Mar/2024

Name: Mr. Hiren Karshanbhai Parmar
Age | Sex: 32 YEAR(S) | Male
Order Station : FO-OPD
Bed Name :

UHID | Episode No : 13026337 | 14615/24/1501
Order No | Order Date: 1501/PN/OP/2403/30541 | 12-Mar-2024
Admitted On | Reporting Date : 12-Mar-2024 14:45:50
Order Doctor Name : Dr.SELF .

ECHOCARDIOGRAPHY TRANSTHORACIC

FINDINGS:

- No left ventricle regional wall motion abnormality at rest.
- Normal left ventricle systolic function. LVEF = 60%.
- No left ventricle diastolic dysfunction. No e/o raised LVEDP.
- Trivial mitral regurgitation.
- No aortic regurgitation. No aortic stenosis.
- Trivial tricuspid regurgitation. No pulmonary hypertension. PASP = 25 mm of Hg.
- Intact IVS and IAS.
- No left ventricle clot/vegetation/pericardial effusion.
- Normal right atrium and right ventricle dimension.
- Normal left atrium and left ventricle dimension.
- Normal right ventricle systolic function. No hepatic congestion.
- IVC measures 14 mm with normal inspiratory collapse.

M-MODE MEASUREMENTS:

LA	29	mm
AO Root	24	mm
AO CUSP SEP	19	mm
LVID (s)	27	mm
LVID (d)	41	mm
IVS (d)	10	mm
LVPW (d)	10	mm
RVID (d)	30	mm
RA	31	mm
LVEF	60	%

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Hiranandani HOSPITAL (A Fortis Network Hospital)

DEPARTMENT OF NIC

Date: 12/Mar/2024

Name: Mr. Hiren Karshanbhai Parmar
Age | Sex: 32 YEAR(S) | Male
Order Station : FO-OPD
Bed Name :

UHID | Episode No : 13026337 | 14615/24/1501
Order No | Order Date: 1501/PN/OP/2403/30541 | 12-Mar-2024
Admitted On | Reporting Date : 12-Mar-2024 14:45:50
Order Doctor Name : Dr.SELF .

DOPPLER STUDY:

E WAVE VELOCITY: 0.8m/sec.
A WAVE VELOCITY:0.5 m/sec
E/A RATIO: 1.4

	PEAK (mmHg)	MEAN (mmHg)	V max (m/sec)	GRADE OF REGURGITATION
MITRAL VALVE	N			Trivial
AORTIC VALVE	05			Nil
TRICUSPID VALVE	25			Trivial
PULMONARY VALVE	2.0			Nil

Final Impression :

- No RWMA.
- Trivial MR and TR. No PH.
- Normal LV and RV systolic function.

DR. PRASHANT PAWAR
DNB(MED), DNB (CARD)

DR.AMIT SINGH,
MD(MED),DM(CARD)

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Hiranandani HOSPITAL (A Fortis Network Hospital)

Date: 12/Mar/2024

DEPARTMENT OF RADIOLOGY

Name: Mr. Hiren Karshanbhai Parmar
Age | Sex: 32 YEAR(S) | Male
Order Station : FO-OPD
Bed Name :

UHID | Episode No : 13026337 | 14615/24/1501
Order No | Order Date: 1501/PN/OP/2403/30541 | 12-Mar-2024
Admitted On | Reporting Date : 12-Mar-2024 11:31:21
Order Doctor Name : Dr.SELF .

X-RAY-CHEST- PA

Findings:

Both lung fields are clear.

The cardiac shadow appears within normal limits.

Trachea and major bronchi appears normal.

Both costophrenic angles are well maintained.

DR. YOGINI SHAH
DMRD., DNB. (Radiologist)

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PAN NO : AABCH5894D

Hiranandani HOSPITAL (A Fortis Network Hospital)

Patient Name	:	Hiren Karshanbhai Parmar	Patient ID	:	13026337
Sex / Age	:	M / 32Y 7M 4D	Accession No.	:	PHC.7667538
Modality	:	US	Scan DateTime	:	12-03-2024 11:50:24
IPID No	:	14615/24/1501	ReportDatetime	:	12-03-2024 11:58:23

USG – WHOLE ABDOMEN

LIVER is normal in size and echogenicity. No IHBR dilatation. No focal lesion is seen in liver. Portal vein appears normal in caliber.

GALL BLADDER is physiologically distended. Gall bladder reveals normal wall thickness. No evidence of calculi in gall bladder. No evidence of pericholecystic collection.
CBD appears normal in caliber.

SPLEEN is normal in size and echogenicity.

BOTH KIDNEYS are normal in size and echogenicity. The central sinus complex is normal. No evidence of calculi/hydronephrosis.
Right kidney measures 10.8 x 3.8 cm.
Left kidney measures 10.7 x 5.0 cm.

PANCREAS is normal in size and morphology. No evidence of peripancreatic collection.

URINARY BLADDER is normal in capacity and contour. Bladder wall is normal in thickness. No evidence of intravesical calculi.

PROSTATE is normal in size & echogenicity. It measures ~ 11.5 cc in volume.

No evidence of ascites.

Impression:

- **No significant abnormality is detected.**

DR. KUNAL NIGAM
M.D. (Radiologist)