



P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

☎ 0171-2532620, 8222896961 ✉ pkrjainhealthcare@gmail.com

NAME : Mrs. MANJEET
AGE/ GENDER : 48 YRS/FEMALE
COLLECTED BY :
REFERRED BY :
BARCODE NO. : 12507633
CLIENT CODE. : P.K.R JAIN HEALTHCARE INSTITUTE
CLIENT ADDRESS : NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA

PATIENT ID : 1801611
REG. NO./LAB NO. : 122503220006
REGISTRATION DATE : 22/Mar/2025 09:29 AM
COLLECTION DATE : 22/Mar/2025 10:07AM
REPORTING DATE : 22/Mar/2025 01:05PM

Test Name	Value	Unit	Biological Reference interval
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HAEMATOLOGY COMPLETE BLOOD COUNT (CBC)

RED BLOOD CELLS (RBCS) COUNT AND INDICES

HAEMOGLOBIN (HB) <i>by CALORIMETRIC</i>	13.1	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	4.2	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	37.4	%	37.0 - 50.0
MEAN CORPUSCULAR VOLUME (MCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	89	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	31.3	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	35.1	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	12.3	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	42.1	fL	35.0 - 56.0
MENTZERS INDEX <i>by CALCULATED</i>	21.19	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX <i>by CALCULATED</i>	26.16	RATIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0

WHITE BLOOD CELLS (WBCS)

TOTAL LEUCOCYTE COUNT (TLC) <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	4880	/cmm	4000 - 11000
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DIFFERENTIAL LEUCOCYTE COUNT (DLC)

NEUTROPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	40 ^L	%	50 - 70
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LYMPHOCYTES <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	48 ^H	%	20 - 40
EOSINOPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	7 ^H	%	1 - 6
MONOCYTES <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	5	%	2 - 12
BASOPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	0	%	0 - 1
<u>ABSOLUTE LEUKOCYTES (WBC) COUNT</u>			
ABSOLUTE NEUTROPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	1952 ^L	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	2342 ^L	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	342	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	244	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	0	/cmm	0 - 110
<u>PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.</u>			
PLATELET COUNT (PLT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	152000	/cmm	150000 - 450000
PLATELETCRIT (PCT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	0.19	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	12 ^H	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	64000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	42.3	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	16.5	%	15.0 - 17.0

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD




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CLINICAL CHEMISTRY/BIOCHEMISTRY

LIPID PROFILE : BASIC

CHOLESTEROL TOTAL: SERUM <i>by CHOLESTEROL OXIDASE PAP</i>	235.33 ^H	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM <i>by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)</i>	100.17	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (DIRECT): SERUM <i>by SELECTIVE INHIBITION</i>	60.22	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	155.08 ^H	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
NON HDL CHOLESTEROL: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	175.11 ^H	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTEROL: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	20.03	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	570.83	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	3.91	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0




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LDL/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2.58	RATIO	MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0 LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.66 ^L	RATIO	3.00 - 5.00

INTERPRETATION:

- Measurements in the same patient can show physiological & analytical variations. Three serial samples 1 week apart are recommended for Total Cholesterol, Triglycerides, HDL & LDL Cholesterol.
- As per NLA-2014 guidelines, all adults above the age of 20 years should be screened for lipid status. Selective screening of children above the age of 2 years with a family history of premature cardiovascular disease or those with at least one parent with high total cholesterol is recommended.
- Low HDL levels are associated with increased risk for Atherosclerotic Cardiovascular disease (ASCVD) due to insufficient HDL being available to participate in reverse cholesterol transport, the process by which cholesterol is eliminated from peripheral tissues.
- NLA-2014 identifies Non HDL Cholesterol (an indicator of all atherogenic lipoproteins such as LDL, VLDL, IDL, Lp(a), Chylomicron remnants) along with LDL-cholesterol as co-primary target for cholesterol lowering therapy. Note that major risk factors can modify treatment goals for LDL & Non HDL.
- Additional testing for Apolipoprotein B, hsCRP, Lp(a) & LP-PLA2 should be considered among patients with moderate risk for ASCVD for risk refinement




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KIDNEY FUNCTION TEST (BASIC)

UREA: SERUM by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)	15.71	mg/dL	10.00 - 50.00
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETRY	1.01	mg/dL	0.40 - 1.20
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	7.34	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	7.27 ^L	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	15.55	RATIO	
URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE	3.29	mg/dL	2.50 - 6.80




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INTERPRETATION:

Normal range for a healthy person on normal diet: 12 - 20

To Differentiate between pre- and postrenal azotemia.

INCREASED RATIO (>20:1) WITH NORMAL CREATININE:

1. Prerenal azotemia (BUN rises without increase in creatinine) e.g. heart failure, salt depletion, dehydration, blood loss) due to decreased glomerular filtration rate.
2. Catabolic states with increased tissue breakdown.
3. GI hemorrhage.
4. High protein intake.
5. Impaired renal function plus .
6. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, burns, surgery, cachexia, high fever).
7. Urine reabsorption (e.g. ureterocolostomy)
8. Reduced muscle mass (subnormal creatinine production)
9. Certain drugs (e.g. tetracycline, glucocorticoids)

INCREASED RATIO (>20:1) WITH ELEVATED CREATININE LEVELS:

1. Postrenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).
2. Prerenal azotemia superimposed on renal disease.

DECREASED RATIO (<10:1) WITH DECREASED BUN :

1. Acute tubular necrosis.
2. Low protein diet and starvation.
3. Severe liver disease.
4. Other causes of decreased urea synthesis.
5. Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid).
6. Inherited hyperammonemias (urea is virtually absent in blood).
7. SIADH (syndrome of inappropriate antidiuretic hormone) due to tubular secretion of urea.
8. Pregnancy.

DECREASED RATIO (<10:1) WITH INCREASED CREATININE:

1. Phenacimide therapy (accelerates conversion of creatine to creatinine).
2. Rhabdomyolysis (releases muscle creatinine).
3. Muscular patients who develop renal failure.

INAPPROPRIATE RATIO:

1. Diabetic ketoacidosis (acetoacetate causes false increase in creatinine with certain methodologies, resulting in normal ratio when dehydration should produce an increased BUN/creatinine ratio).
2. Cephalosporin therapy (interferes with creatinine measurement).



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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

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ENDOCRINOLOGY

THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	1.33	ng/mL	0.35 - 1.93
THYROXINE (T4): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	9.58	µgm/dL	4.87 - 12.60
THYROID STIMULATING HORMONE (TSH): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	3.24	µIU/mL	0.35 - 5.50

3rd GENERATION, ULTRASENSITIVE

INTERPRETATION:

TSH levels are subject to circadian variation, reaching peak levels between 2-4 a.m and at a minimum between 6-10 pm. The variation is of the order of 50%. Hence time of the day has influence on the measured serum TSH concentrations. TSH stimulates the production and secretion of the metabolically active hormones, thyroxine (T4) and triiodothyronine (T3). Failure at any level of regulation of the hypothalamic-pituitary-thyroid axis will result in either underproduction (hypothyroidism) or overproduction (hyperthyroidism) of T4 and/or T3.

CLINICAL CONDITION	T3	T4	TSH
Primary Hypothyroidism:	Reduced	Reduced	Increased (Significantly)
Subclinical Hypothyroidism:	Normal or Low Normal	Normal or Low Normal	High
Primary Hyperthyroidism:	Increased	Increased	Reduced (at times undetectable)
Subclinical Hyperthyroidism:	Normal or High Normal	Normal or High Normal	Reduced

LIMITATIONS:-

1. T3 and T4 circulates in reversibly bound form with Thyroid binding globulins (TBG), and to a lesser extent albumin and Thyroid binding Pre Albumin so conditions in which TBG and protein levels alter such as pregnancy, excess estrogens, androgens, anabolic steroids and glucocorticoids may falsely affect the T3 and T4 levels and may cause false thyroid values for thyroid function tests.
2. Normal levels of T4 can also be seen in Hyperthyroid patients with : T3 Thyrotoxicosis, Decreased binding capacity due to hypoproteinemia or ingestion of certain drugs (e.g.: phenytoin, salicylates).
3. Serum T4 levels in neonates and infants are higher than values in the normal adult, due to the increased concentration of TBG in neonate serum.
4. TSH may be normal in central hypothyroidism, recent rapid correction of hyperthyroidism or hypothyroidism, pregnancy, phenytoin therapy.

TRIIODOTHYRONINE (T3)		THYROXINE (T4)		THYROID STIMULATING HORMONE (TSH)	
Age	Refferance Range (ng/mL)	Age	Refferance Range (µg/dL)	Age	Reference Range (µIU/mL)
0 - 7 Days	0.20 - 2.65	0 - 7 Days	5.90 - 18.58	0 - 7 Days	2.43 - 24.3
7 Days - 3 Months	0.36 - 2.59	7 Days - 3 Months	6.39 - 17.66	7 Days - 3 Months	0.58 - 11.00
3 - 6 Months	0.51 - 2.52	3 - 6 Months	6.75 - 17.04	3 Days - 6 Months	0.70 - 8.40
6 - 12 Months	0.74 - 2.40	6 - 12 Months	7.10 - 16.16	6 - 12 Months	0.70 - 7.00




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1 - 10 Years	0.92 - 2.28	1 - 10 Years	6.00 - 13.80
11- 19 Years	0.35 - 1.93	11 - 19 Years	4.87- 13.20
> 20 years (Adults)	0.35 - 1.93	> 20 Years (Adults)	4.87 - 12.60
RECOMMENDATIONS OF TSH LEVELS DURING PREGNANCY (μ U/mL)			
1st Trimester			0.10 - 2.50
2nd Trimester			0.20 - 3.00
3rd Trimester			0.30 - 4.10

INCREASED TSH LEVELS:


- 1.Primary or untreated hypothyroidism may vary from 3 times to more than 100 times normal depending upon degree of hypofunction.
- 2.Hypothyroid patients receiving insufficient thyroid replacement therapy.
- 3.Hashimotos thyroiditis
- 4.DRUGS: Amphetamines, iodine containing agents & dopamine antagonist.
- 5.Neonatal period, increase in 1st 2-3 days of life due to post-natal surge

DECREASED TSH LEVELS:

- 1.Toxic multi-nodular goiter & Thyroiditis.
- 2.Over replacement of thyroid hormone in treatment of hypothyroidism.
- 3.Autonomously functioning Thyroid adenoma
- 4.Secondary pituitary or hypothalamic hypothyroidism
- 5.Acute psychiatric illness
- 6.Severe dehydration.
- 7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.
- 8.Pregnancy: 1st and 2nd Trimester




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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM 40.7 ng/mL
by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

DEFICIENCY: < 20.0
INSUFFICIENCY: 20.0 - 30.0
SUFFICIENCY: 30.0 - 100.0
TOXICITY: > 100.0

INTERPRETATION:

DEFICIENT:	< 20	ng/mL
INSUFFICIENT:	21 - 29	ng/mL
PREFERRED RANGE:	30 - 100	ng/mL
INTOXICATION:	> 100	ng/mL

- Vitamin D compounds are derived from dietary ergocalciferol (from plants, Vitamin D2), or cholecalciferol (from animals, Vitamin D3), or by conversion of 7- dihydrocholecalciferol to Vitamin D3 in the skin upon Ultraviolet exposure.
- 25-OH--Vitamin D represents the main body reservoir and transport form of Vitamin D and transport form of Vitamin D, being stored in adipose tissue and tightly bound by a transport protein while in circulation.
- Vitamin D plays a primary role in the maintenance of calcium homeostasis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid hormone (PTH).
- Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.

DECREASED:

- Lack of sunshine exposure.
- Inadequate intake, malabsorption (celiac disease)
- Depressed Hepatic Vitamin D 25- hydroxylase activity
- Secondary to advanced Liver disease
- Osteoporosis and Secondary Hyperparathyroidism (Mild to Moderate deficiency)
- Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.

INCREASED:

- Hypervitaminosis D is Rare, and is seen only after prolonged exposure to extremely high doses of Vitamin D. When it occurs, it can result in severe hypercalcemia and hyperphosphatemia.

CAUTION: Replacement therapy in deficient individuals must be monitored by periodic assessment of Vitamin D levels in order to prevent hypervitaminosis D

NOTE:- Dark coloured individuals as compare to whites, is at higher risk of developing Vitamin D deficiency due to excess of melanin pigment which interfere with Vitamin D absorption.




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P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

☎ 0171-2532620, 8222896961 ✉ pkrjainhealthcare@gmail.com

NAME : Mrs. MANJEET
AGE/ GENDER : 48 YRS/FEMALE
COLLECTED BY :
REFERRED BY :
BARCODE NO. : 12507633
CLIENT CODE. : P.K.R JAIN HEALTHCARE INSTITUTE
CLIENT ADDRESS : NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA

PATIENT ID : 1801611
REG. NO./LAB NO. : 122503220006
REGISTRATION DATE : 22/Mar/2025 09:29 AM
COLLECTION DATE : 22/Mar/2025 10:07AM
REPORTING DATE : 22/Mar/2025 06:26PM

Test Name	Value	Unit	Biological Reference interval
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VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

1353^H pg/mL 200.0 - 1100.0


INTERPRETATION:-

INCREASED VITAMIN B12	DECREASED VITAMIN B12
1.Ingestion of Vitamin C	1.Pregnancy
2.Ingestion of Estrogen	2.DRUGS:Aspirin, Anti-convulsants, Colchicine
3.Ingestion of Vitamin A	3.Ethanol lgestion
4.Hepatocellular injury	4. Contraceptive Harmones
5.Myeloproliferative disorder	5.Haemodialysis
6.Uremia	6. Multiple Myeloma

- Vitamin B12 (cobalamin) is necessary for hematopoiesis and normal neuronal function.
 - In humans, it is obtained only from animal proteins and requires intrinsic factor (IF) for absorption.
 - The body uses its vitamin B12 stores very economically, reabsorbing vitamin B12 from the ileum and returning it to the liver; very little is excreted.
 - 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 frequently causes macrocytic anemia, glossitis, peripheral neuropathy, weakness, hyperreflexia, ataxia, loss of proprioception, poor coordination, and affective behavioral changes. These manifestations may occur in any combination; many patients have the neurologic defects without macrocytic anemia.
 - Serum methylmalonic acid and homocysteine levels are also elevated in vitamin B12 deficiency states.
 - Follow-up testing for antibodies to intrinsic factor (IF) is recommended to identify this potential cause of vitamin B12 malabsorption.
- NOTE:**A normal serum concentration of vitamin B12 does not rule out tissue deficiency of vitamin B12. The most sensitive test for vitamin B12 deficiency at the cellular level is the assay for MMA. If clinical symptoms suggest deficiency, measurement of MMA and homocysteine should be considered, even if serum vitamin B12 concentrations are normal.




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Test Name	Value	Unit	Biological Reference interval
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CLINICAL PATHOLOGY

URINE ROUTINE & MICROSCOPIC EXAMINATION

PHYSICAL EXAMINATION

QUANTITY RECIEVED <small>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</small>	30	ml	
COLOUR <small>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</small>	PALE YELLOW		PALE YELLOW
TRANSPARANCY <small>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</small>	CLEAR		CLEAR
SPECIFIC GRAVITY <small>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</small>	1.02		1.002 - 1.030

CHEMICAL EXAMINATION


REACTION <small>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</small>	ACIDIC		
PROTEIN <small>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</small>	NEGATIVE (-ve)		NEGATIVE (-ve)
SUGAR <small>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</small>	NEGATIVE (-ve)		NEGATIVE (-ve)
pH <small>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</small>	5.5		5.0 - 7.5
BILIRUBIN <small>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</small>	NEGATIVE (-ve)		NEGATIVE (-ve)
NITRITE <small>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</small>	NEGATIVE (-ve)		NEGATIVE (-ve)
UROBILINOGEN <small>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</small>	NOT DETECTED	EU/dL	0.2 - 1.0
KETONE BODIES <small>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</small>	NEGATIVE (-ve)		NEGATIVE (-ve)
BLOOD <small>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</small>	NEGATIVE (-ve)		NEGATIVE (-ve)
ASCORBIC ACID <small>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</small>	NEGATIVE (-ve)		NEGATIVE (-ve)

MICROSCOPIC EXAMINATION

RED BLOOD CELLS (RBCs)	NEGATIVE (-ve)	/HPF	0 - 3
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Test Name	Value	Unit	Biological Reference interval
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
PUS CELLS	4-5	/HPF	0 - 5
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
EPITHELIAL CELLS	3-4	/HPF	ABSENT
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
CRYSTALS	NEGATIVE (-ve)		NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
CASTS	NEGATIVE (-ve)		NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
BACTERIA	NEGATIVE (-ve)		NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
OTHERS	NEGATIVE (-ve)		NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
TRICHOMONAS VAGINALIS (PROTOZOA)	ABSENT		ABSENT
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			

*** End Of Report ***




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