Age/Gender : 41 Y/Female Registration No. : 768630

Patient ID: 182404240005Registered Time: 24/Apr/2024 10:02AMBarcodeNo: 624300Collection Time: 24/Apr/2024 01:28PMReferred By: SEJAL CLINICAL LABReceiving Time: 24/Apr/2024 01:28PMPanel: CHD TricityReporting Time: 24/Apr/2024 01:46PM

HAEMATOLOGY

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Dr V Ahmad Consultant Microbiologist Dr Shailja Puri MD. Pathology Dr. Anmol Taneja MD. Pathology

NAME : ASHU UPNEJA Age/Gender : 41 Y/Female Registration No. Patient ID : 182404240005 Registered Time : 24/Apr/2024 10:02AM : 624300 Collection Time : 24/Apr/2024 01:28PM BarcodeNo Referred By : SEJAL CLINICAL LAB Receiving Time : 24/Apr/2024 01:28PM

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	<u>HAEMA</u>	<u>TOLOGY</u>		
Test Name	Value	Unit	Bio Ref.Interval	
ABSOLUTE LEUKOCYTES (WBC) COUNT				
ABSOLUTE NEUTROPHIL COUNT by Calculated	3.64	x10^3Cells/μL	2.0-7.0	
ABSOLUTE LYMPHOCYTES COUNT by Calculated	2.02	x10^3Cells/μL	1.0-3.0	
ABSOLUTE EOSINOPHIL COUNT by Calculated	0.33	x10^3Cells/μL	0.02-0.50	
ABSOLUTE MONOCYTE COUNT	0.52	x10^3Cells/µL	0.20-1.00	

CLINICAL NOTES

by Calculated

A complete blood count (CBC) is used to evaluate overall health and detect wide range of disorders, including anemia, infection and leukemia. There have been some reports of WBC and platelet counts being lower in venous blood than in capillary blood samples ,although still within these reference ranges.

POSSIBLE CAUSES OF ABNORMAL PARAMETERS:-

High RBC, Hb, or HCT - dehydration, polycythemia, shock, chronic hypoxia Low RBC, Hb, or HCT - anemia, thalassemia, and other Hemoglobinopathies Low MCV - microcytic anemia

High MCV - macrocytic anemia, liver disease

Low WBC - sepsis, marrow hypoplasia

: CHD Tricity

High WBC - acute stress, infection, malignancies

Low platelets - risk of bleeding High platelets - risk of thrombosis

Notes

Panel

Macrocytic Anemia/Dimorphic Anemia can have low platelet count.

Microcytic Anemia/Leucocytosis can have Reactive thrombocytosis.

For microcytic indices a Mentzer index of less than 13 suggests that the patient may have thalassemia trait, and an index of more than 13 suggests that the patient may have iron deficiency.

Reference ranges are from Dacie and Lewis Practical Hematology 12th edition(2016) Reference ranges may vary between laboratories. CBC Test processed on fully automated analyser(6 Part Differential SYSMEX XN-330)

This device performs hematology analysis according to th Hydrodynamic Focusing(DC method), Flurosence Flow cytometry Method(using a semiconductor laser) and SLS-Hb method.

Note: The result obtained relate only to the sample given/received & tested. A single test result is not always indicative of a disease, it has to be correlated with clinical data for interpretation.



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HAEMATOLOGY

Test Name Value Unit Bio Ref.Interval

ESR (WESTERGREN's METHOD)

ESR [WESTERGREN] **43** mm/1st 0 - 15

Sedimentation
Note:-

1. Test conducted on EDTA whole blood at 37°C.

 $2.\,$ ESR readings are auto- corrected with respect to Hematocrit (PCV) values.



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HAEMATOLOGY

	<u>HAEMA I</u>	<u>OLOG1</u>		
Test Name	Value	Unit	Bio Ref.Interval	
	н	bA1C		
Hb A1C, GLYCOSYLATED Hb	5.40	%	Non-Diabetic < 6.0 Good Control 6.0-7.0 Weak Control 7.0-8.0 Poor control > 8.0	
Estimated Average Glucose Expected Values:	108.28	mg/dL	, 55. 555.7 5.5	
Non Diabetic	< 6.0	%		
Goal	< 7.0	%		
Action Suggested	> 8.0	%		

Comment:-

Glycosylated Hb is a normal adult Hb which is covalently bounded to a glucose molecule. Glycosylated Hb concentration is dependent on the average blood glucose concentration and is stable for the life of the RBC (120 days). Glycohaemoglobin serves as suitable marker of metabolic control of diabetics. Its estimation is unaffected by diet, insulin, exercise on day of testing and thus reflects average blood glucose levels over a period of last several weeks /months. There is a 3 - 4 week time before percent Glycohaemoglobin reflects changes in blood glucose levels.



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NAME : ASHU UPNEJA

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Panel : CHD Tricity Reporting Time : 24/Apr/2024 02:40PM

BIOCHEMISTRY

Test Name	Value	Unit	Bio Ref.Interval
	<u>LIPID PROFILE</u>	Ī	
Specimen: Serum			
TOTAL CHOLESTEROL Enzymatic(CHO-POD)	143.80	mg/dL	Desirable < 200 Borderline 200 - 239 High > 240
TRIGLYCERIDES, Serum GPO-POD	82.50	mg/dL	Normal 150 Border line high 150-199 High 200-490
HDL-CHOLESTEROL , Serum Direct measure	44.00	mg/dL	40-60
LDL CHOLESTEROL,Serum Calculated	83.30	mg/dL	Optimal<100 Near or Above Optima-I00-129 Borderline High 130-159 High 160 - 189
NON HDL CHOLESTEROL Calculated	99.80	mg/dL	Optimal<130 Near or Above Optima-I30-159 Borderline High 160 - 189 High 190 - 219
VLDL ,Serum Calculated	16.50	mg/dL	0.0- 30.0
TOTAL LIPIDS Calculated	370.10	mg/dL	350.00-700.00
TOTAL CHOLESTEROL /HDL RATIO ,Serum	3.27		< 4.97
LDL / HDL CHOLESTEROL RATIO Calculated	1.89		1.5-3.5
TRIGLYCERIDES/HDL RATIO	1.88	RATIO	3.00-5.00

ALERT!!! 10-12 hours fasting is mandatory for lipid parameters. If not, values might fluctuate.

CLINICAL NOTES:-Lipid profile is initial screening tool for abnormalities in lipids. The results of this test can identify certain genetic diseases & can determine approximate risks for cardiovascular disease, certain forms of pancreatitis. Hypertriglyceridemia is indicative of insulin resistance when present with low HDL & elevated LDL, while elevated TG is risk factor for coronary artery disease, especially when low HDL is present. TG of 500mg/dL or more can be concerning for development of pancreatitis.



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BIOCHEMISTRY

Test Name	Value	Unit	Bio Ref.Interval
			_
	LIVER FUNCTION TES	ST(LFT)	
BILIRUBIN TOTAL	1.01	mg/dL	0.1-1.2
DIRECT BILIRUBIN(CONJUGATED), Serum	0.16	mg/dL	< 0.20
INDIRECT BILIRUBIN(Unconj.),Serum	0.85	mg/dL	0.20-0.80
TOTAL PROTEIN, Serum	7.70	g/dL	6.6-8.3
ALBUMIN,SERUM BCG	4.36	g/dL	3.5-5.0
GLOBULIN,Serum	3.34	gm/dL	2.3-3.5
A/G RATIO Calculated	1.31		1.0 - 2.3
SGOT (AST) ,Serum	19.50	U/L	0-50
SGPT (ALT), Serum	13.20	U/L	0-50
SGOT/SGPT RATIO	1.48		
ALKALINE PHOSPHATASE ,Serum	70.6	U/L	30-120
GAMMA GT ,Serum	26.70	U/L	5 - 64

COMMENT:-

These are group of tests that can be used to detect the presence of liver disease, distinguish among different types of liver disorders, gauge the extent of known liver damage, and monitor the response to treatment. Most liver diseases cause only mild symptoms initially, but these diseases must be detected early. Some tests are associated with functionality (e.g., albumin), some with cellular integrity (e.g., transaminase), and some with conditions linked to the biliary tract (gamma-glutamyl transferase and alkaline phosphatase). Conditions with elevated levels of ALT and AST include hepatitis A,B,C, paracetamol toxicity etc. Several biochemical tests are useful in the evaluation and management of patients with hepatic dysfunction. Some or all of these measurements are also carried out (usually about twice a year for routine cases) on those individuals taking certain medications, such as anticonvulsants, to ensure that the medications are not adversely impacting the person's liver. Reference ranges are from Teitz fundamental of clinical chemistry 8th ed (2018) Reference ranges vary between laboratories.



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 NAME
 : ASHU UPNEJA

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BIOCHEMISTRY

Test Name	Value	Unit	Bio Ref.Interval
	KFT AND ELECTRO	<u>YTES</u>	
UREA Urease	22.1	mg/dL	17.0 -43.0
CREATININE, Serum	0.79	mg/dL	0.60-1.40
BLOOD UREA NITROGEN UREASE	10.33	mg/dl	9 - 20
URIC ACID , Serum	3.60	mg/dL	2.4-5.7
CALCIUM , Serum	9.82	mg/dL	8.6 - 10.6
PHOSPHORUS , Serum Phosphomolybdate	2.94	mg/dL	2.5 - 4.5
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO CALCULATED	13.08	RATIO	10.0 - 20.0
UREA/CREATININE RATIO	27.97		
eGFR Calculated	85.37	mL/min/1.73m2	
ELECTROLYTES			
SODIUM (SERUM)	139.5	mmol/L	137-145
POTASSIUM (SERUM)	3.99	mmol/L	3.5-5.1
CHLORIDE ISE Direct	102.60	mmol/L	98-107

Kidney function tests are group of tests that can be used to evaluate how well the kidneys are functioning. Creatinine is a waste product that comes from protein in the diet and also comes from the normal wear and tear of muscles of the body. In blood, it is a marker of GFR in urine, it can remove the need for 24-hour collections for many analytes or be used as a quality assurance tool to assess the accuracy of a 24-hour collection Higher levels may be a sign that the kidneys are not working properly. As kidney disease progresses, the level of creatinine and urea in the blood increases. Certain drugs are nephrotoxic hence KFT is done before and after initiation of treatment with these drugs.

Low serum creatinine values are rare; they almost always reflect low muscle mass. Apart from renal failure Blood Urea can increase in dehydration and GI bleed.

Note: The result obtained relate only to the sample given/received & tested. A single test result is not always indicative of a disease, it has to be correlated with clinical data for interpretation.



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NAME : ASHU UPNEJA

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BIOCHEMISTRY

Test Name	Value	Unit	Bio Ref.Interval	
	<u>IRON</u>	PROFILE		
Iron	31.20	ug/dL	60-180	
TOTAL IRON BINDING CAPACITY	246.10	ug/dL	240-450	
Calculated		J		
UIBC	214.90	ug/dL	155-355	
NiTRO-PSAP				
TRANSFERRIN NEPHELOMETRY	174.73	mg/dL	178 - 354	
TRANSFERRIN SATURATION	12.68	%	15.0-50.0	

Comment:

Most body iron is found in hemoglobin. The serum measurement of iron is useful in the differential diagnosis of anemia, iron deficiency anemia, thalassemia, possible sideroblastic anemia, and iron poisoning. Total iron-binding capacity in serum, representing transferrin concentration in iron-binding capacity, is a useful index of nutritional iron status.

Iron deficiency anemia is characterized by a decreased serum Fe, increased TIBC or transferrin, and a decreased transferrin saturation. Serum TIBC is increased in iron deficiency. Serum TIBC is decreased in anemia of chronic disease.

Iron is an essential trace mineral element which forms an important component of hemoglobin, metallocompounds and Vitamin A. Deficiency of iron, leads to microcytic hypochromic anemia. The toxic effects of iron are deposition of iron in various organs of the body and hemochromatosis.

Total Iron Binding capacity (**TIBC**) is a direct measure of the protein Transferrin which transports iron from the gut to storage sites in the bone marrow. In iron deficiency anemia, serum iron is reduced and TIBC increases.

Transferrin Saturation occurs in Idiopathic hemochromatosis and Transfusional hemosiderosis where no unsaturated iron binding capacity is available for iron mobilization. Similar condition is seen in congenital deficiency of Transferrin



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Panel

NAME : ASHU UPNEJA Age/Gender : 41 Y/Female Registration No. Patient ID : 182404240005 : 24/Apr/2024 10:02AM Registered Time : 624300 Collection Time : 24/Apr/2024 01:28PM BarcodeNo Referred By : SEJAL CLINICAL LAB Receiving Time : 24/Apr/2024 01:28PM

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Test Name	Value	Unit	Bio Ref.Interval	
	THYROI	<u>D PROFILE</u>		
T3 ,Serum	1.14	ng/mL	0.87 - 1.78	
T4 ,Serum	6.52	ug/dl	3.06 - 12.54	
TSH by CLIA	2.30	μIU/ml	0.34-5.60	

Comments:

T3 is physiologically more active than T4 & plays an important role in maintaining euthyroidism.

T3 circulates in free form (0.3 %) and in bound form (99.7%).

: CHD Tricity

T4 is predominantly bound to carrier protein - thyroid binding globulin (TBG-99.9%).

T4 assay aids in diagnosis of hyperthyroidism - primary or secondary hypothyroidism & thyroid hormone resistances.

T4 there must also be associated with the other three of the thyroid assessment, such as TSH & T3 as well as with the clinical examina on to the patient TSH levels are subject to circadian variation, reaching peak levels between 2am to 4am and at a minimum between 6pm to 10pm. The varia on is of the order of 50%; hence time of the day has influence on the measured serum TSH concentrations. Significant numbers of parents particularly those above 55 years of age have a serum TSH level between $4.68 \& 10 \mu IU/ml$.

This borderline elevation may be due to presence of SUBCLINICAL HYPOTHYROIDISM. Thyroid profile and an -thyroid (an TPO & TG) an bodies es ma on is suggested in all such cases.

Very low serum TSH values are observed in patients who are being treated for hypothyroidism. In such patients Serum Free T3 & Free T4 estimation may also be performed.

INCREASED LEVELS:

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

DECREASED LEVELS:



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IMMUNOLOGY

Test Name Value Unit Bio Ref.Interval

- 1. Toxic multi-nodular goitre & Thyroiditis.
- 2. Over replacement of thyroid harmone in treatment of hypothyroidism.
- 3. Autonomously functioning Thyroid adenoma
- 4.Secondary pituatary or hypothalmic hypothyroidism
- 5.Acute psychiatric illness
- 6.Severe dehydration.
- 7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.

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 (eg: phenytoin , salicylates).
- 3. Serum T4 levles 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 hypothroidism, pregnancy, phenytoin therapy.



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IMMUNOLOGY

Test Name Value Unit Bio Ref.Interval

VITAMIN D (25 OH)

VITAMIN D(25 OH) ,Serum 25.10 ng/mL < 20 Deficiency

20- 30 Insufficiency
30-100 Sufficienty
>100 Toxicity

Expected Values

Deficiency <20 ng/mL
Insufficiency 20-<30 ng/mL
Sufficiency 30-100 ng/mL
Toxocity >100.0

Note: It should be taken into cosideration that differences in Vitamin D (25-OH) levels may exist with respect to gender, age, season, geographical latitude and ethnic groups

Comments

Vitamin D Total assay is used as an aid in the assesment of Vitamin D sufficiency in adults.

Vitamin D is acquired either by exposure to sunlight or ingestion of food containing vitamin D. It is metabolized to vit D, 25 hydroxy in the liver in the first step by vit D,25-hydroxylase system. A small amount of it further gets metabolized by hydroxylation in kidney to vit D 1,25 dihydroxy. Since vit D, 25 hydroxy is the predominant circulating form of Vit D in normal population, it is considered to be the most reliable index of vit D status.

Vitamin D is essential for bone health. In children, severe deficiency leads to bone-malformation, known as rickets. Milder degrees of insufficiecy are believed to cause reduced efficiency in the utilization of dietary calcium.

The measurement of 25-OH-D is becoming increasingly important in the management of patients with various disorders of calcium metabolism associated with Rickets, neonatal hypocalcemia, pregnancy, nutritional and renal osteodystrophy, hypoparathyroidism, and postmenopausal state. Increased levels are found in Vit D intoxication.

Decreased levels are detected in Rickets, osteomalacia, secondary hyperparathyroidism, malabsorption of vit D (e.g. liver diseases, cholestasis), and diseases that increase Vit D metabolism (viz. Tuberculosis, sarcoidosis, primary hyperparathyroidism).



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IMMUNOLOGY

Test Name Value Unit Bio Ref.Interval

VITAMIN B12

VITAMIN B12 LEVEL ,Serum 536.00 pg/mL 180-914

CLIA

Comment

Vitamin B12 belongs to the corrin family it is a cofactor for the conversion of methylemalonyl coenzyme A to succinyl coenzyme A, synthesis of methionine from homocysteine and formation of myelin. It is required along with folate for DNA synthesis. The major source of vitamin B12 for human beings is meat, while herbivorous animals get their requirement from contaminated vegetable matter and coprophagy. Megaloblastic anaemia can be due to cobalamin & or folic acid deficiency.

Vitamin B12 or Cyanocobalamin, is a complex corrinoid compound containing four pyrrole rings

that surround a single cobalt atom. Humans obtain vitamin B12 exclusively from animal dietary sources, such as meat, eggs and milk.

Clinical and laboratory findings for Vitamin B12 deficiency include neurological abnormalities,

decreased serum B12 levels and increased excretion of methylmalonic acid. The impaired DNA synthesis

associated with Vitamin B12 deficiency causes macrocytic anaemias. These anaemias are characterized by abnormal maturation of erythrocyte precursors in the Bone-Marrow, which results in the presence of magaloblasts and in decreased erythrocyte survival.

Pernicious anaemia is a macrocytic anaemia caused by Vitamin B12 deficiency that is due to lack of intrinsic factor. Low Vitamin B12 intake, gastrectomy, diseases of small intestine, malabsorption and trans-cobalamin deficiency can also cause Vitamin B12 deficiency.

Increased Levels :-

- 1. Renal failure
- 2. Liver disease

Decreased Levels :-

- 1. Megaloblastic anemia
- 2. Vegetarianism

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IMMUNOLOGY

Test Name Value Unit Bio Ref.Interval

TESTOSTERONE TOTAL

Age Min Max Unit Male 0-49 years 1.63 (47.01) 34.0 (980.56) nmol/l (ng/dl) Male >50 years 4.41 (127.18) 35.38 (1020.36) nmol/l (ng/dl) Female 0-49 years 0.25 (7.21) 2.75 (79.3) nmol/l (ng/dl) Female >50 years 0.30 (8.65) 1.28 (36.9) nmol/l (ng/dl)	TESTO by CLIA	STERONE TO	TAL ,Serum	36.90 ng/dl		7.21-79.30
Male >50 years 4.41 (127.18) 35.38 (1020.36) nmol/l (ng/dl) Female 0-49 years 0.25 (7.21) 2.75 (79.3) nmol/l (ng/dl)		Age	Min	Max	Unit	
Female 0-49 years 0.25 (7.21) 2.75 (79.3) nmol/l (ng/dl)	Male	0-49 years	1.63 (47.01)	34.0 (980.56)	nmol/l (ng/dl)	
	Male	>50 years	4.41 (127.18)	35.38 (1020.36)	nmol/l (ng/dl)	
Female >50 years $0.30 (8.65)$ $1.28 (36.9)$ nmol/l (ng/dl)	Female	0-49 years	0.25 (7.21)	2.75 (79.3)	nmol/l (ng/dl)	
	Female	>50 years	0.30 (8.65)	1.28 (36.9)	nmol/l (ng/dl)	

Comments:

Testosterone is secreted in females by the ovary and formed indirectly from androstendione in adrenal glands. In males it is secreted by the testes. It circulates in blood, bound largely to sex hormone binding globulin (SHBG). Less than 1% of the total testosterone is in the free form. The bioavailable fractions include the free form and that "weakly bound" to albumin (40% of the total in men and 20% of total in women) & bound to cortisol binding globulin (CBG). It is the most potent circulating androgenic hormone. The total testosterone bound of SHBG fluctuates ,since SHBG levels are effected by medication, disease, sex steroids and insulin.

INCREASED LEVELS

Precocious puberty (Males)

Congenital Adrenal Hyperplasia

Polycystic ovarian disease

Ovarian tumours

Androgen resistance

DECREASED LEVELS

Delayed puberty (Males)

Kindly correlate clinically.

*** End Of Report ***



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