P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

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NAME	**: Baby. VERONICA**		
AGE/ GENDER	: 11 YRS/FEMALE	**PATIENT ID**	: 1573272
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 122412040020**
REFERRED BY	:	**REGISTRATION DATE**	: 04/Dec/2024 03:23 PM
BARCODE NO.	: 12505997	**COLLECTION DATE**	: 04/Dec/2024 03:25PM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE	**REPORTING DATE**	: 04/Dec/2024 04:16PM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

HAEMATOLOGY

COMPLETE BLOOD COUNT (CBC)

Test Name	Value	Unit	Biological Reference interval
RED BLOOD CELLS (RBCS) COUNT AND INDICES			
HAEMOGLOBIN (HB) *by CALORIMETRIC*	**11.6**^{L}	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	4.36	Millions/cmm	3.50 - 5.50
PACKED CELL VOLUME (PCV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	**33.8**^{L}	%	35.0 - 49.0
MEAN CORPUSCULAR VOLUME (MCV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	**77.6**^{L}	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	**26.5**^{L}	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	34.2	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	12.5	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	36.4	fL	35.0 - 56.0
MENTZERS INDEX *by CALCULATED*	17.8	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX *by CALCULATED*	22.16	RATIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CELLS (WBCS)			
TOTAL LEUCOCYTE COUNT (TLC) *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	8850	/cmm	4000 - 12000
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	**48**^{L}	%	50 - 70
LYMPHOCYTES	40	%	20 - 45

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Test Name	Value	Unit	Biological Reference interval
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
EOSINOPHILS	**8^{H}**	%	1 - 6
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
MONOCYTES	4	%	3 - 12
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
BASOPHILS	0	%	0 - 1
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT	4248	/cmm	2000 - 7500
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE LYMPHOCYTE COUNT	**3540^{L}**	/cmm	800 - 4900
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE EOSINOPHIL COUNT	**708^{H}**	/cmm	40 - 440
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE MONOCYTE COUNT	354	/cmm	80 - 880
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE BASOPHIL COUNT	0	/cmm	0 - 110
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.			
PLATELET COUNT (PLT)	283000	/cmm	150000 - 450000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELETCRIT (PCT)	0.27	%	0.10 - 0.36
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
MEAN PLATELET VOLUME (MPV)	10	fL	6.50 - 12.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET LARGE CELL COUNT (P-LCC)	70000	/cmm	30000 - 90000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET LARGE CELL RATIO (P-LCR)	24.7	%	11.0 - 45.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET DISTRIBUTION WIDTH (PDW)	16.2	%	15.0 - 17.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

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Test Name	Value	Unit	Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

IRON PROFILE

IRON: SERUM *by FERROZINE, SPECTROPHOTOMETRY*	85.7	µg/dL	37.0 - 145.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM *by FERROZINE, SPECTROPHOTOMETERY*	195.55	µg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC) :SERUM *by SPECTROPHOTOMETERY*	281.25	µg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM *by CALCULATED, SPECTROPHOTOMETERY (FERENE)*	30.47	%	15.0 - 50.0
TRANSFERRIN: SERUM *by SPECTROPHOTOMETERY (FERENE)*	**199.69**^{L}	mg/dL	200.0 - 350.0

INTERPRETATION:-

VARIABLES	*ANEMIA OF CHRONIC DISEASE*	*IRON DEFICIENCY ANEMIA*	*THALASSEMIA α/β TRAIT*
SERUM IRON:	Normal to Reduced	Reduced	Normal
TOTAL IRON BINDING CAPACITY:	Decreased	Increased	Normal
% TRANSFERRIN SATURATION:	Decreased	Decreased < 12-15 %	Normal
SERUM FERRITIN:	Normal to Increased	Decreased	Normal or Increased

IRON:

1.Serum iron studies is recommended for differential diagnosis of microcytic hypochromic anemia.i.e iron deficiency anemia, zinc deficiency anemia,anemia of chronic disease and thalassemia syndromes.

2. It is essential to isolate iron deficiency anemia from Beta thalassemia syndromes because during iron replacement which is therapeutic for iron deficiency anemia, is severely contra-indicated in Thalassemia.

TOTAL IRON BINDING CAPACITY (TIBC):

1.It is a direct measure of protein transferrin which transports iron from the gut to storage sites in the bone marrow.

% TRANSFERRIN SATURATION:

1.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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Test Name	Value	Unit	Biological Reference interval

SPECIAL INVESTIGATIONS

INSULIN GROWTH FACTOR - 1/SOMATOMEDIN-C

INSULIN GROWTH FACTOR (IGF) - 1 SOMATOMEDIN-C: SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	121	ng/mL	118.0 - 448.0

INTERPRETATION:

1.Insulin-like growth factor- I (IGF- I) bioactivity is regulated by genetic and non-genetic factors like growth hormone, nutrition and insulin.
2.The rate of development of microalbuminuria (MA), an important early marker of diabetic nephropathy, has been related not only to factors such as age at diagnosis, sex and blood pressure, but also with the activity of the growth hormone–insulin-like growth factor- I (GH–IGF- I) axis.
3.Poor glycaemic control in type I diabetes, the most important factor for diabetic complications, is associated with elevated GH secretion and serum IGF binding protein (IGFBP)-1 levels, as well as reduced serum IGF- I levels.
4.In addition, derangements of the GH–IGF- I axis have been associated with hyperfiltration and MA in type I diabetes.
5.The mechanism behind this imbalance in the GH–IGF- I axis in type 1 diabetes has been suggested to be due to relatively low portal insulin levels resulting from s.c. administration of insulin.
6.Complete correction of the GH–IGF- I axis only seems possible with portal administration of insulin.
7.In the type I, II diabetes, GH / IGF- I axis is abnormal, GH increased, IGF- I reduced.
8.In type I diabetes, liver resistant GH, leading the liver IGF- I concentrations decreased.
9.At the same time, more IGFBP-I are generated, IGFBP-I can play a role in binding to and inhibit IGF- I .
10.This reduction of IGF- I cause the feedback of growth hormone's decrease.
11.Increased release of GH will lead to high blood sugar by antagonizing the function of insulin.
12.At the same time, the reduction of IGF- I also led to j growth retardation of juvenile or young with type I diabetes.
13.In poorly controlled type II diabetes, there will be also a high release of GH, antagonising the effect of peripheral tissues' insulin.
14.In any kind of diabetes, IGF- I can improve the control of blood sugar and reduce the serum GH's insulin-resistance in addition, IGF- I is important factor to adjust the function of bone cell and metabolism

INCREASED

1.gigantism
2.acromegaly
3.pregnancy.

DECREASED

1.growth hormone deficiencies
2.hypopituitarism.

NOTE:

IGF-1 may be normal in 5-10 % cases of acromegaly and 10-20 % cases of dwarfism.

*** End Of Report ***

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