P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

0171-2532620, 8222896961 pkrjainhealthcare@gmail.com

NAME	**: Baby. VERONICA**		
AGE/ GENDER	: 11 YRS/FEMALE	**PATIENT ID**	: 1573272
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 122408070001**
REFERRED BY	:	**REGISTRATION DATE**	: 07/Aug/2024 08:38 AM
BARCODE NO.	: 12504020	**COLLECTION DATE**	: 07/Aug/2024 08:39AM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE	**REPORTING DATE**	: 07/Aug/2024 12:48PM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

Test Name	Value	Unit	Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE FASTING (F)

GLUCOSE FASTING (F): PLASMA *by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)*	85.13	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0

INTERPRETATION

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A fasting plasma glucose level below 100 mg/dl is considered normal.
2. A fasting plasma glucose level between 100 - 125 mg/dl is considered as glucose intolerant or prediabetic. A fasting and post-prandial blood test (after consumption of 75 gms of glucose) is recommended for all such patients.
3. A fasting plasma glucose level of above 125 mg/dl is highly suggestive of diabetic state. A repeat post-prandial is strongly recommended for all such patients. A fasting plasma glucose level in excess of 125 mg/dl on both occasions is confirmatory for diabetic state.

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

NAME	**: Baby. VERONICA**		
AGE/ GENDER	: 11 YRS/FEMALE	**PATIENT ID**	: 1573272
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 122408070001**
REFERRED BY	:	**REGISTRATION DATE**	: 07/Aug/2024 08:38 AM
BARCODE NO.	: 12504020	**COLLECTION DATE**	: 07/Aug/2024 08:39AM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE	**REPORTING DATE**	: 07/Aug/2024 01:30PM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

Test Name	Value	Unit	Biological Reference interval

ENDOCRINOLOGY

THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	1.231	ng/mL	0.35 - 1.93
THYROXINE (T4): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	8.02	µgm/dL	4.87 - 13.20
THYROID STIMULATING HORMONE (TSH): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	0.925	µIU/mL	0.50 - 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 trilodothyronine (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 (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.

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**

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

NAME	**: Baby. VERONICA**		
AGE/ GENDER	: 11 YRS/FEMALE	**PATIENT ID**	: 1573272
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 122408070001**
REFERRED BY	:	**REGISTRATION DATE**	: 07/Aug/2024 08:38 AM
BARCODE NO.	: 12504020	**COLLECTION DATE**	: 07/Aug/2024 08:39AM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE	**REPORTING DATE**	: 07/Aug/2024 01:30PM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

Test Name			Value	Unit	Biological Reference interval
6 - 12 Months	0.74 - 2.40	6 - 12 Months	7.10 – 16.16	6 – 12 Months	0.70 - 7.00
1 - 10 Years	0.92 - 2.28	1 - 10 Years	6.00 - 13.80	1 – 10 Years	0.60 - 5.50
11- 19 Years	0.35 - 1.93	11 - 19 Years	4.87- 13.20	11 – 19 Years	0.50 – 5.50
> 20 years (Adults)	0.35 - 1.93	> 20 Years (Adults)	4.87 - 12.60	> 20 Years (Adults)	0.35– 5.50
RECOMMENDATIONS OF TSH LEVELS DURING PREGNANCY (µIU/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, idonie 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 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.
8.Pregnancy: 1st and 2nd Trimester

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

0171-2532620, 8222896961 pkrjainhealthcare@gmail.com

NAME	**: Baby. VERONICA**		
AGE/ GENDER	: 11 YRS/FEMALE	**PATIENT ID**	: 1573272
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 122408070001**
REFERRED BY	:	**REGISTRATION DATE**	: 07/Aug/2024 08:38 AM
BARCODE NO.	: 12504020	**COLLECTION DATE**	: 07/Aug/2024 08:39AM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE	**REPORTING DATE**	: 10/Aug/2024 10:26AM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

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)*	**103^{L}**	**ng/mL**	**111.0 - 551.0**

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 ***

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)