Name	: Mr. Aditya Bhosle	**Reg. No**	: 25601182
Age/Sex	: 26 Years / Male	**Reg. Date**	: 17-May-2025 11:12 AM
Ref. By	: Dr. Krishna Dave Vaidya	**Collected On**	: 17-May-2025 11:12 AM
Client Name	:	**Report Date**	: 17-May-2025 04:59 PM
Sample Source	: LAB	**Centre Location**	: Divine Makarpura

RENAL FUNCTION TESTS

Invitro tests for quantitative determination of renal substrates by COBAS INTEGRA , roche - USA

Parameter	Result	Unit	Biological Ref. Interval
URIC ACID *Uricase Colorimetry*	5.20	mg/dL	3.4 - 7.0

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Ref. By	: Dr. Krishna Dave Vaidya	**Collected On**	: 17-May-2025 11:12 AM
Client Name	:	**Report Date**	: 17-May-2025 05:38 PM
Sample Source	: LAB	**Centre Location**	: Divine Makarpura

SERUM INSULIN

Aa invitro quantitative determination of insulin by e 411 - roche , USA

Parameter	Result	Unit	Biological Ref. Interval
SERUM INSULIN (FASTING)	**36.78**	mU / ml	2.6 - 24.9

Electrochemiluminescence (ECLIA) , compititive assay

FACT FILE

Insulin is a peptide hormone with a molecular weight of approximately 6000 daltons. It is secreted by the B-cells of the pancreas and passes into circulation via the portal vein and the liver. Insulin is generally released in pulses, with the parallel glucose cycle normally about 2 minutes ahead of the insulin cycle. The insulin molecule consists of two polypeptide chains, the a-chain with 21 and the ß-chain with 30 amino acids. Biosynthesis of the hormone takes place in the ß-cells of the islets of Langerhans in the form of single-chain preproinsulin, which is immediately cleaved to give proinsulin. Specific proteases cleave proinsulin to insulin and C-peptide which pass into the bloodstream simultaneously. About half of the insulin, but virtually none of the C-peptide, is retained in the liver. Circulating insulin has a half-life of 3-5 minutes and is preferentially degraded in the liver, whereas inactivation or excretion of proinsulin and C-peptide mainly takes place in the kidneys. The action of insulin is mediated by specific receptors and primarily consists of facilitation of the uptake of sugar by the cells of the liver, fatty tissue and musculature; this is the basis of its hypoglycemic action. Serum insulin determinations are mainly performed on patients with symptoms of hypoglycemia.

They are used to ascertain the glucose/insulin quotients and for clarification of questions concerning insulin secretion, e.g. in the tolbutamide test and glucagon test or in the evaluation of oral glucose tolerance tests or hunger provocation tests. Although the adequacy of pancreatic insulin synthesis is frequently assessed via the determination of C-peptide, it is still generally necessary to determine insulin. For example, therapeutic administration of insulins of non-human origin can lead to the formation of anti-insulin antibodies. In this case, measurement of the concentration of serum insulin shows the quantity of free - and hence biologically active - hormone, whereas the determination of C-peptide provides a measure of the patient's total endogenous insulin secretion.

A disorder in insulin metabolism leads to massive influencing of a number of metabolic processes. A too low concentration of free, biologically active insulin can lead to the development of diabetes mellitus. Possible causes of this include destruction of the ß-cells (type I diabetes), reduced activity of the insulin or reduced pancreatic synthesis (type II), circulating antibodies to insulin, delayed release of insulin or the absence (or inadequacy) of insulin receptors. On the other hand, autonomous, non-regulated insulin secretion is generally the cause of hypoglycemia. This condition is brought about by inhibition of gluconeogenesis, e.g. as a result of severe hepatic or renal failure, islet cell adenoma, or carcinoma. Hypoglycemia can, however, also be facilitated intentionally or unintentionally (factitious hypoglycemia). In 3 % of persons with reduced glucose tolerance, the metabolic state deteriorates towards diabetes mellitus over a period of time. Reduced glucose tolerance during pregnancy always requires treatment. The clearly elevated risk of mortality for the fetus necessitates intensive monitoring.

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HOMEOSTATIC MODEL ASSESSMENT FOR INSULIN RESISTANCE - HOMA IR

Parameter	Result	Unit	Biological Ref. Interval
Fasting Glucose *Hexokinase gen.3*	81	mg/dL	74 - 106
Fasting Insulin *CHEMILUMINESCENCE*	**36.8**	µIU/ml	2.6 - 24.9
HOMA Index *Calculated*	**4.42**		0.5 - 2.0
Beta cell function (Homa B) *Calculated*	**337.8**	%	72 - 201
Insulin Sensitivity (Homa S) *Calculated*	22.6	%	
INTERPRETATION	Moderate insulin resistance		

FACT FILE
REFERENCE RANGE FOR HOMA INDEX

HOMA Inxex : 0.5 - 2.0 - Normal
HOMA Index : 2.1 - 2.9 - Early insulin resistance
HOMA Index : 3.0 - 5.0 - Moderate insulin resistance
HOMA Index : > 5.0 - Severe insulin resistance

Insulin resistance (IR) and beta-cell dysfunction are characteristic features of type 2 diabetes mellitus (T2DM). Insulin resistance is characterized by decrease in insulin mediated glucose disposal in insulin-sensitive tissue and increased hepatic glucose production whereas beta-cell dysfunction occurs when beta-cells are unable to compensate for the insulin resistance. Measurement of both of these parameters at diagnosis of T2DM can be a potential tool in evaluation, risk stratification and monitoring treatment of DM.
Homeostasis model assessment was first developed in 1985 by Matthews *et al.* It is a method used to quantify insulin resistance and beta-cell function from basal (fasting) glucose and insulin (or C-peptide) concentrations. HOMA is a model of the relationship of glucose and insulin dynamics that predicts fasting steady-state glucose and insulin concentrations for a wide range of possible combinations of insulin resistance and ß-cell function. Insulin levels depend on the pancreatic ß-cell response to glucose concentrations while, glucose concentrations are regulated by insulin-mediated glucose production via the liver. Thus, deficient ß-cell function will echo a diminished response of ß-cell to glucose-stimulated insulin secretion. Similarly, insulin resistance is reflected by the diminished suppressive effect of insulin on hepatic glucose production. The HOMA model has proved to be a robust clinical and epidemiological tool for the assessment of insulin resistance.

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