ADVANCE DIAGNOSTICS CENTRE
C1-C2/17A, NEAR NIHARIKA TALKIES
KORBA- 495677
PH-09228333 MOBILE-9300888178

NAME	: MR ASHISH RATHORE	32 Years / Male		Reg No.	: 25544
Ref. By	: DR. SAHU K.N.(MD MED.)		Reg. Date	:	11/10/2022 10:24AM
Address	:		Collected At	:	MedZone Center

INVESTIGATION REPORT

CLINICAL BIOCHEMISTRY

TEST	RESULT	UNIT	BIOLOGICAL REF RANGE	TEST METHOD
C-Peptide				
Sample Type	: SERUM			
C-Peptide	: **0.83**	ng/mL	1.1 - 4.4	Electro CLIA

Connecting peptide is a single chain 31-amino acid (AA 33-63) connecting (C) polypeptide with a molecular weight of approx. 3021 daltons. In the process of biosynthesis of insulin the C-peptide is formed as a by-product together with insulin by the proteolytic cleavage of the precursor molecule proinsulin, stored in secretory granules in the Golgi complex of the pancreatic β-cells. Proinsulin in turn was cleaved from preproinsulin. C-peptide fulfills an important function in the assembly of the two-chain insulin (A- and B-chain) structure and the formation of the two disulfide bonds within the proinsulin molecule. Insulin and C-peptide are secreted in equimolar amounts and released into circulation via the portal vein. As half of the insulin, but almost none of the C-peptide is extracted in the liver, C-peptide has a longer half-life (about 35 min.) than insulin; 5 to 10 times higher concentration of C-peptide persist in the peripheral circulation, and these levels fluctuate less than insulin. The liver does not extract C-peptide, which is removed from the circulation by the kidneys and degraded, with a fraction excreted unchanged in the urine. The concentration in urine is about 20-50 fold higher than in serum. C-peptide concentrations are therefore elevated in renal disease. In the past, C-Peptide has been considered biologically inactive. However, recent studies have demonstrated that it is capable of eliciting molecular and physiological effects suggesting that C-peptide is in fact a bioactive peptide. There is evidence that C-peptide replacement, together with insulin administration, may prevent the development or retard the progression of long-term complications in type 1 diabetes. Measurements of C-peptide, insulin and glucose are used as an aid in the differential diagnosis of hypoglycemia (factitious hypoglycemia and hypoglycemia caused by hyperinsulinism) to ensure an appropriate management and therapy of the patients. To quantify the endogenous insulin secretion, C-peptide is measured basally, after fasting and after stimulation and suppression tests. Due to high prevalence of endogenous anti-insulin antibodies C-peptide concentrations reflect the endogenous pancreatic insulin secretion more reliably in insulin-treated diabetics than the levels of insulin itself. Measurements of C-peptide may therefore be an aid in the assessment of a residual β-cell function in the early stages of type-1 diabetes mellitus and for the differential diagnosis of latent autoimmune diabetes of adults (LADA) and type-2 diabetes. C-peptide measurements are also used to asses the success of islet transplantation and for monitoring after pancreatectomy. Urine C-peptide is measured when a continuous assessment of β-cell function is desired or frequent blood sampling is not practical (e.g. in children). C-peptide excretion in urine has been used to assess pancreatic function in gestational diabetes, and in patients with unstable glycemic control in insulin-dependent diabetes mellitus (IDDM). Although testing for C-peptide is not requested for the routine monitoring of diabetes, it is a valuable tool for the individual therapeutic decisions which are essential for an optimal long-term metabolic control. Elevated C-peptide levels may result from increased β-cell activity observed in hyperinsulinism, from renal insufficiency, and obesity. Correlation was also found between higher C-peptide levels and increasing hyperlipoproteinaemia and hypertension. Decreased C-peptide levels are observed in: starvation, factitious hypoglycemia, hypoinsulinism (NIDDM, IDDM), Addison's disease and after radical pancreatectomy.

METHOD: One-step sandwich and competitive FEIA

INSTRUMENT: TOSHO AIA-360 JAPAN

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INVESTIGATION REPORT

CLINICAL BIOCHEMISTRY

TEST	RESULT	UNIT	BIOLOGICAL REF RANGE	TEST METHOD
Insulin				
Sample Type	: SERUM			
Insulin	: 6.40	uU/mL	Fasting Insulin :2.6 - 24.9	E CLIA

ınsulin is a peptide hormone with a molecular weight of approximately 6000 D. 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 polypeptides, the alpha-chain with 21 and the beta-chain with 30 amino acids. Biosynthesis of the hormone takes place in the beta-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 amino acid sequence of insulin has remained surprisingly constant during evolution, with the result that prior to the development of genetically engineered human insulin it was possible to successfully use porcine or bovine insulin in the therapy of diabetes mellitus. 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 could 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. Too low a concentration of free, biologically active insulin can lead to the development of diabetes mellitus. Possible causes of this include destruction of the beta-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. The Roche Cobas Insulin assay employs two monoclonal antibodies which together are specific for human insulin.

METHOD: One-step sandwich and competitive FEIA

INSTRUMENT: TOSHO AIA-360 JAPAN

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Sample Registered On : 11/10/2022 10:24AM
Sample Received On : 11/10/2022 10:28AM
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Sample Barcode :

Checked By:gopal

Dr. VANDANA CHANDANI