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ASSESSMENT OF LIFESTYLE MODIFICATIONS FOR DIABETES MELLITUS

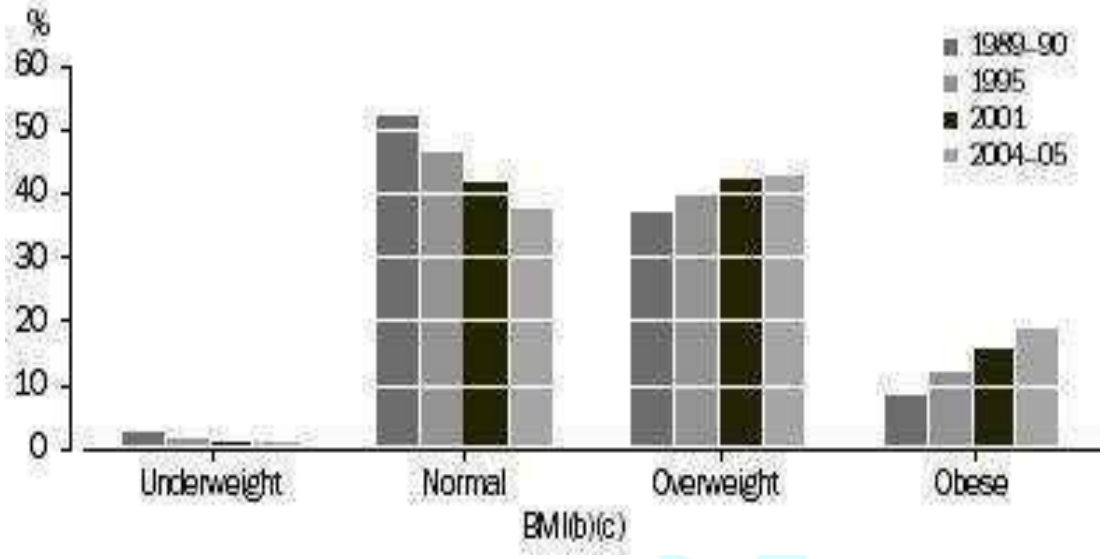
Dr. Kolanedi.Anupama Priyadarshini[1], B.Purandeshwari[2], A.Naga namratha reddy[2], V.Guru Keerthi[2], K.Sravani[2], K.Dedeepya[2],T.Lakshmi Vasanthi[2], K.Naga Phani kumar[2]

1. Asst professor, Department of Pharmacy Practice, SIMS College of pharmacy, mangaladas nagar, guntur, pin-522509
2. Department of Pharmacy Practice, SIMS College of pharmacy, mangaladas nagar, guntur, pin-522509.

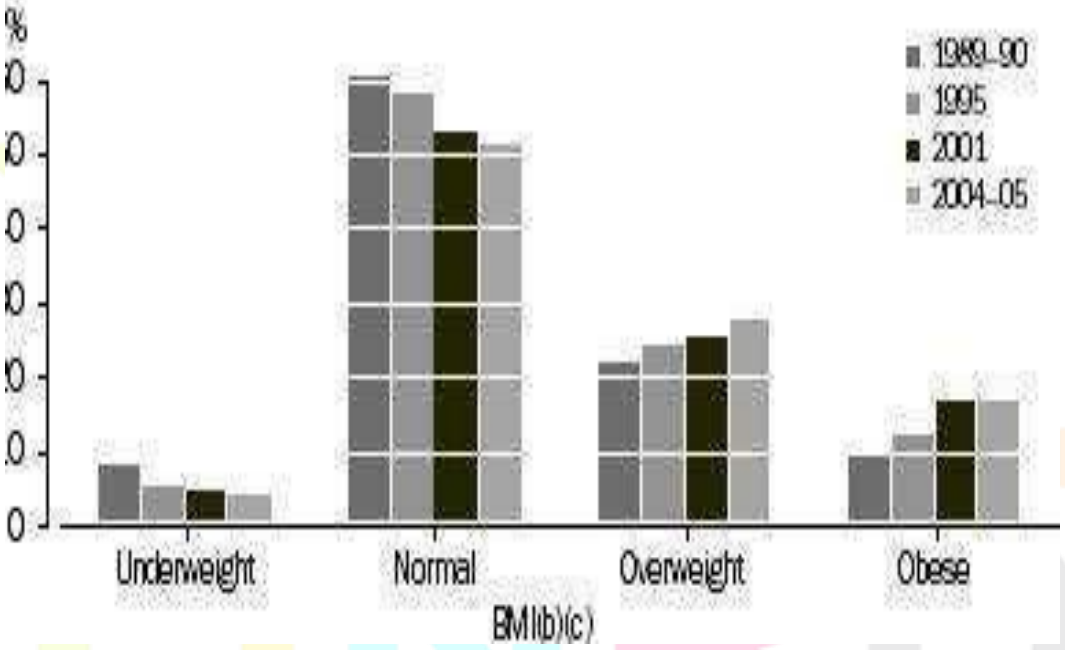
Introduction:

The prevalence of diabetes, especially type 2 diabetes and hypertension are significantly increased with the prevalence of obesity . Type 2 diabetes, hypertension frequently associated with type 2 diabetes, and obesity are important risk factors for cardiovascular morbidity and mortality and cardiac- and renal complications. Hyperglycemia as well as hyperinsulinemia in type 2 diabetes is a cardiovascular risk by itself . Type 2 diabetes, hypertension and obesity are characterized by stimulation of the renin-angiotensinaldosterone system (RAAS), elevated sympathetic activity and insulin resistance. Importantly, these characteristics, themselves, are one of the cardiovascular risks. Therefore, pharmacological and non-pharmacological treatments for type 2 diabetes should be selected from favourable effects on stimulated RAAS, elevated sympathetic nervous system activity, insulin resistance and leptin resistance.

Weight loss is recommended to delay and prevent type 2 diabetes in obesity, and for the treatment. Lifestyle modification such as a caloric restricted diet, reducing sedentary behaviour and an increase in exercise form the basis of all therapy. Weight loss treated with lifestyle modification including calorie restriction and/or exercise causes normalization of stimulated RAAS, sympathetic activation, insulin resistance, and hyperleptinemia, which are usually observed in type 2 diabetes and obesity. Recently, Straznicky *et al*. and Masuo *et al*have shown the low caloric diet and exercise have different effects on insulin resistance, the RAAS, and sympathetic nervous activity in obese hypertensive subjects, although similar weight loss was observed between both interventions. Straznicky *et al.* reported that exercise had stronger effects of normalized the RAAS stimulation, sympathetic activation and insulin resistance compared to diet only, whereas Masuo *et al.* showed mild calorie restriction and mild exercise has different mechanisms on weight loss (normalization on sympathetic



(a) **Body mass index(a),** Men

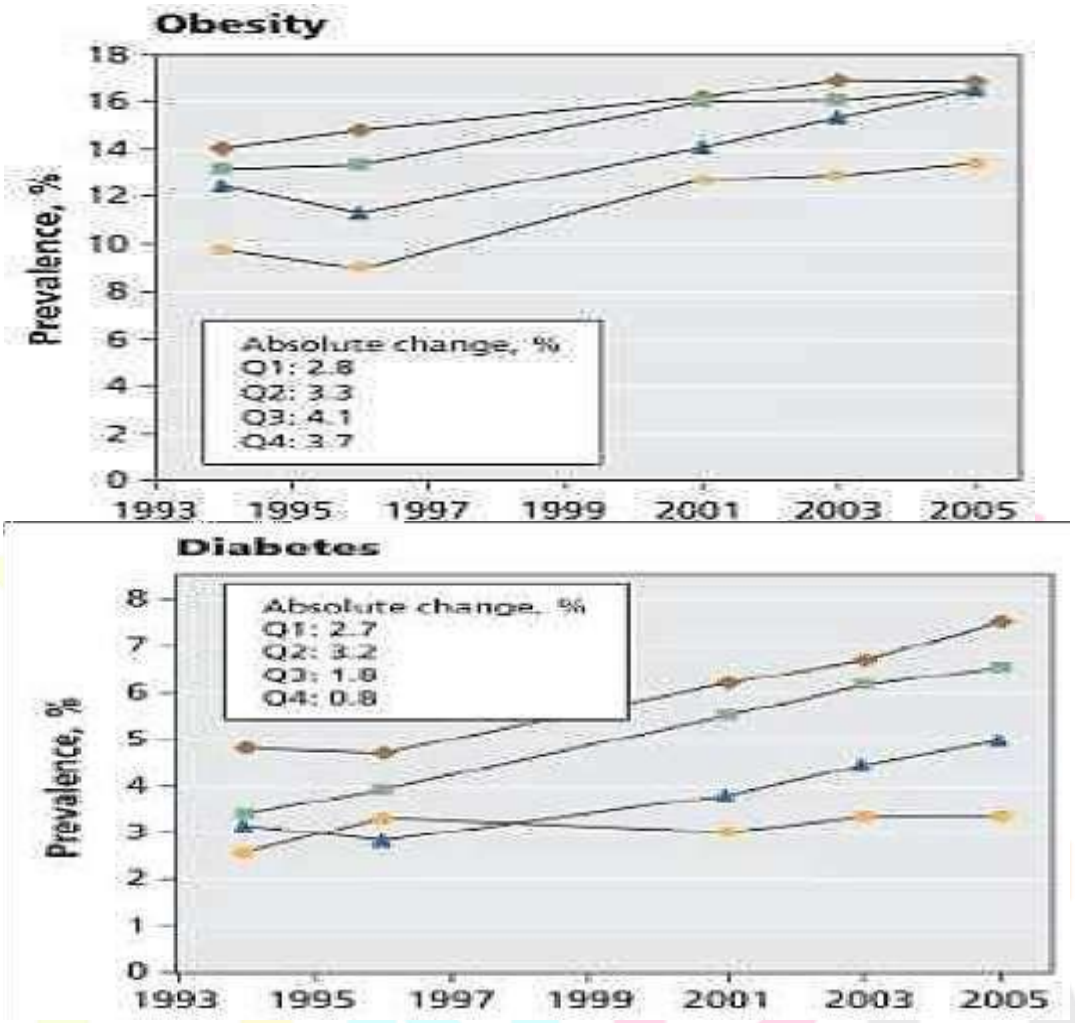


(b) **Body Mass index(a),** Women

activation for mild calorie restriction, and normalization on insulin resistance for exercise). The observations, however, demonstrate that a combination therapy for weight loss with a low caloric diet and exercise is recommended for weight loss due to stronger suppression of insulin resistance and sympathetic activation, which both are known as strong risk factors for cardiovascular events. Although few studies have observed changes in body weight, blood pressure, neurohormonal changes over a long duration such as 2 years, observed more than 30% individuals who initially succeeded to significantly lose weight, had rebound weight gain over 2 years. Understanding mechanisms underlying both type 2 diabetes and obesity may help to achieve weight loss and maintenance of weight loss and the stricter blood glucose goal. Maintenance of weight loss is another key factor to reduce cardiovascular risks in

type 2 diabetes in obesity

In addition, most hypertensive patients with diabetes and obesity are very resistant to controlling hypertension and frequently require two or more types of medications to achieve blood pressure goals. Similarly, diabetic patients, especially type 2 diabetic patients with obesity, need higher dose of anti-diabetic medications such as metformin or insulin. However, pharmacological treatments for hypertension anddiabetes with weight loss could reduce pharmacological treatment .



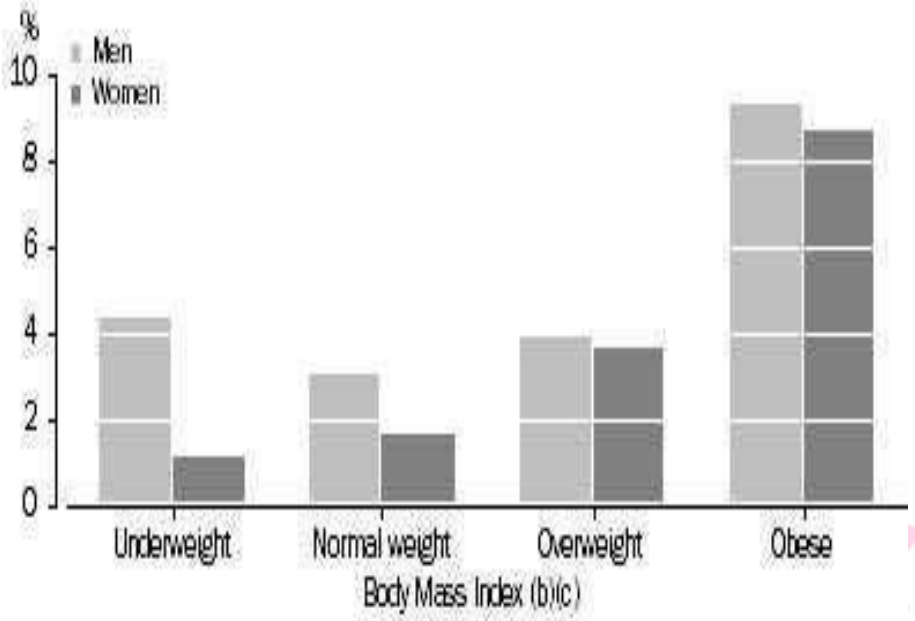
The purpose of this review is to provide, *i)* the importance of lifestyle modifications to delay and prevent type 2 diabetes, *ii*) Lifestyle modification to reduce cardiovascular risks in type 2 diabetes, and *iii)* weight loss for the better pharmacological control on type 2 diabetes and hypertension, which frequently co- exist with type 2 diabetes. *iv)* The mode of weight loss influences different physiological pathways, with calorie restriction and exercise program. *v*) Different mechanisms may contribute to reductions in blood pressure and cardiovascular risks associated with weight loss with the relevant physiological mechanisms at play being dependent on the mode of weight loss.

2. Type 2 diabetes versus Type 1 diabetes

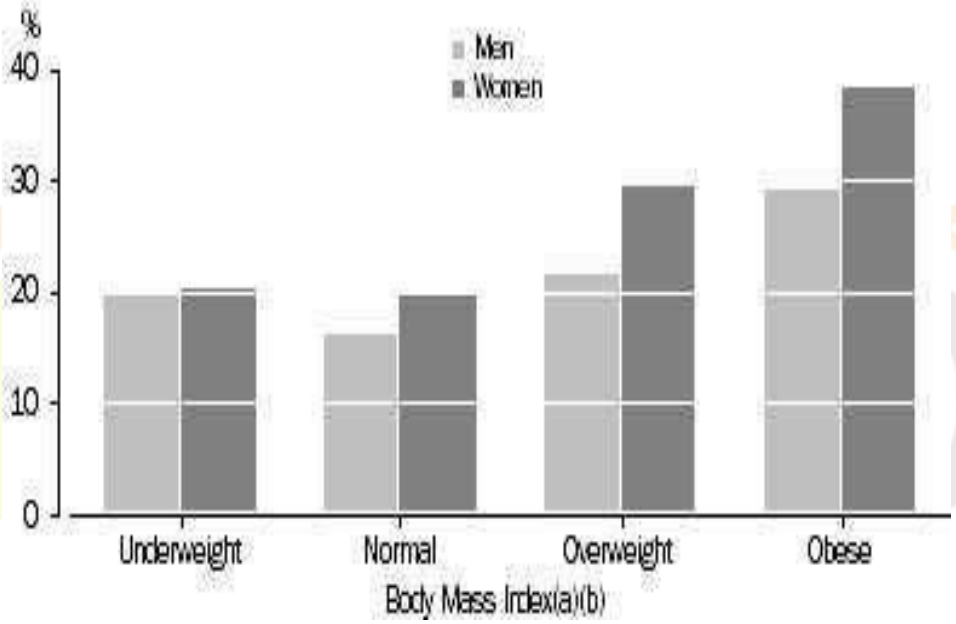
Prevalence of diabetes has increased markedly over the last 20 years in parallel with obesity (Figures 2 and 3) [1, 2]. As of 2010 there are approximately 285 million people with the disease compared to around

30 million in 1985. Long-term complications from high blood sugar can include heart disease, strokes, renal failure, diabetic retinopathy, and diabetic neuropathy.

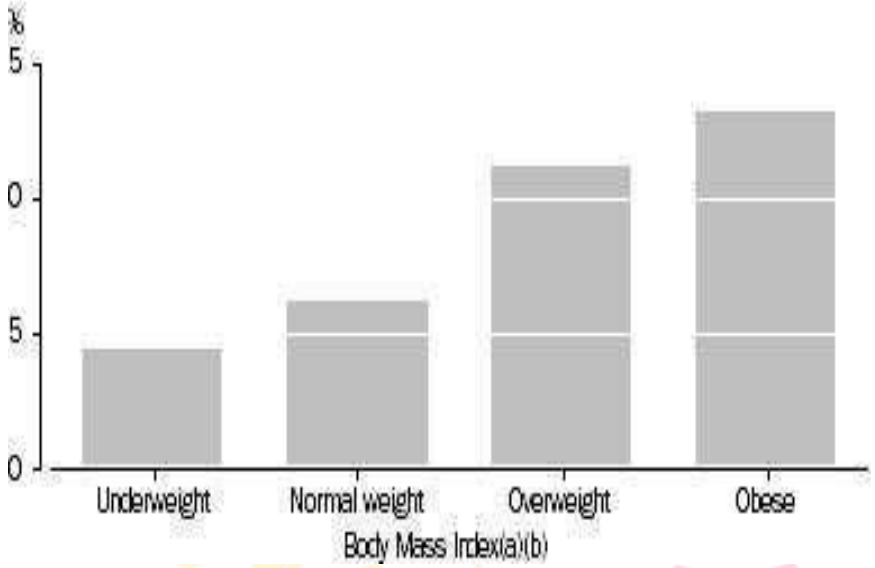
Type 2 Diabetes



Prevalence of Cardiovascular Diseases



Prevalence of hyperlipidemia



Diabetes mellitus includes type 2 diabetes (formerly noninsulin dependent diabetes), type 1 diabetes (formerly insulin dependent diabetes), and gestational diabetes. These 3 types of diabetes have different characteristics and progress Ninety percent of diabetic patients are type 2 diabetes and the other 10% are due primarily to diabetes mellitus type 1 and gestational diabetes.

2.1. Diabetes mellitus type 2 (Formerly noninsulin-dependent diabetes mellitus (NIDDM))

Type 2 diabetes is the most common form of diabetes, affecting 90% of all patients with diabetes. This type of diabetes is characterised by metabolic disorder with insulin resistance and relative insulin deficiency [10]. This is in contrast to type 1 diabetes, in which there is an absolute insulin deficiency due to destruction of islet cells in the pancreas [9, 11]. Obesity is thought to be the primary cause of type 2 diabetes in people who are genetically predisposed to the disease, and obesity has been found to contribute to approximately 55% of case of type 2 diabetes [12].

The disease is strongly genetic in origin but lifestyle factors such as excess weight, inactivity, high blood pressure and poor diet are major risk factors for its development. Symptoms may not show for many years and, by the time they appear, significant problems may have developed. People with type 2 diabetes are twice as likely to suffer cardiovascular disease. The classic symptoms are excess thirst, frequent urination, and constant hunger.

Type 2 diabetes is initially managed by increasing exercise and dietary modification. If blood glucose levels are not adequately lowered by these measures, medications such as metformin or insulin may be needed. In those on insulin, there is typically the requirement to routinely check blood sugar levels.

2.2. Type 1 diabetes (Insulin-dependent diabetes)

Type 1 diabetes is an auto-immune disease targeting on the insulin-producing beta cells in the pancreas. This type of diabetes, also known as juvenile-onset diabetes, accounts for approximately 10% of all people with the disease. In the majority of cases this type of diabetes appears before the patient is 40 years old, triggered by environmental factors such as viruses, diet or chemicals in people genetically predisposed. Patients with type 1 diabetes will require insulin therapy regularly, and should follow a careful diet and exercise plan.

2.3. Gestational diabetes mellitus

Gestational diabetes, or glucose intolerance, is first diagnosed during pregnancy through an oral glucose tolerance test. Between 5.5 and 8.8% of pregnant women develop gestational diabetes in Australia and 2 to 10 percent of all pregnancies in USA. The hormones produced during pregnancy increase the amount of insulin needed to control blood glucose levels. If the body can‟t meet this increased need for insulin, women can develop gestational diabetes during the late stages of pregnancy.

While the glucose intolerance usually returns to normal after the birth, the mother has a significant risk of developing permanent diabetes while the baby is more likely to develop obesity and impaired glucose tolerance and/or diabetes later in life . Risk factors for gestational diabetes include a family history of diabetes, increasing maternal age, obesity, lack of sleep and being a member of a community or ethnic group with a high risk of developing type 2 diabetes. Self-care and dietary changes are essential in treatment.

RAMESH HOSPITALS

H-[illegible]

Laboratory Test Report

DEPARTMENT OF BIOCHEMISTRY

Patient Name	: Mrs. [illegible]	Age / Sex	: [illegible] Y(s)/Female
Bill Date	: [illegible]-2020 [illegible] AM	UMR No.	: GN-[illegible]2030
Sampled Date/Time	: [illegible]-2020 [illegible] AM	Bill No.	: [illegible]
Report Date	: [illegible]-2020 [illegible] AM	Result No	: [illegible] / [illegible]
Ref By	: Dr. [illegible]	Org. Name	: [illegible] SERVICES
		Specimen	: [illegible] PLASMA

Parameters	Results	Units	Biological Ref. Interval	Method
FASTING BLOOD SUGAR	[illegible]	mg/dL	: [illegible] - [illegible] mg/dL	GOD - POD
HbA1c	[illegible]	%	Non diabetic level : [illegible] - [illegible] % Diabetic control : [illegible] - [illegible] % Poor control : [illegible] - [illegible] %	HPLC

*** End Of Report ***

[illegible]

BIO-CHEMIST

NOTE: * Indicates abnormal test result ---- # indicates critical test results.

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[illegible]

DIABETES & ENDOCRINE CLINIC

[illegible], POGATHOTA, NELLORE - 524 001 (A.P.) Ph. : [illegible]

Dr. S. SATYANARAYANA MURTHY, B.Sc., M.B.B.S., D.DIAB. [illegible]

Dr. M.V. RAMA MOHAN, M.D., D.M.

Name: I.LAKSHMAMMA

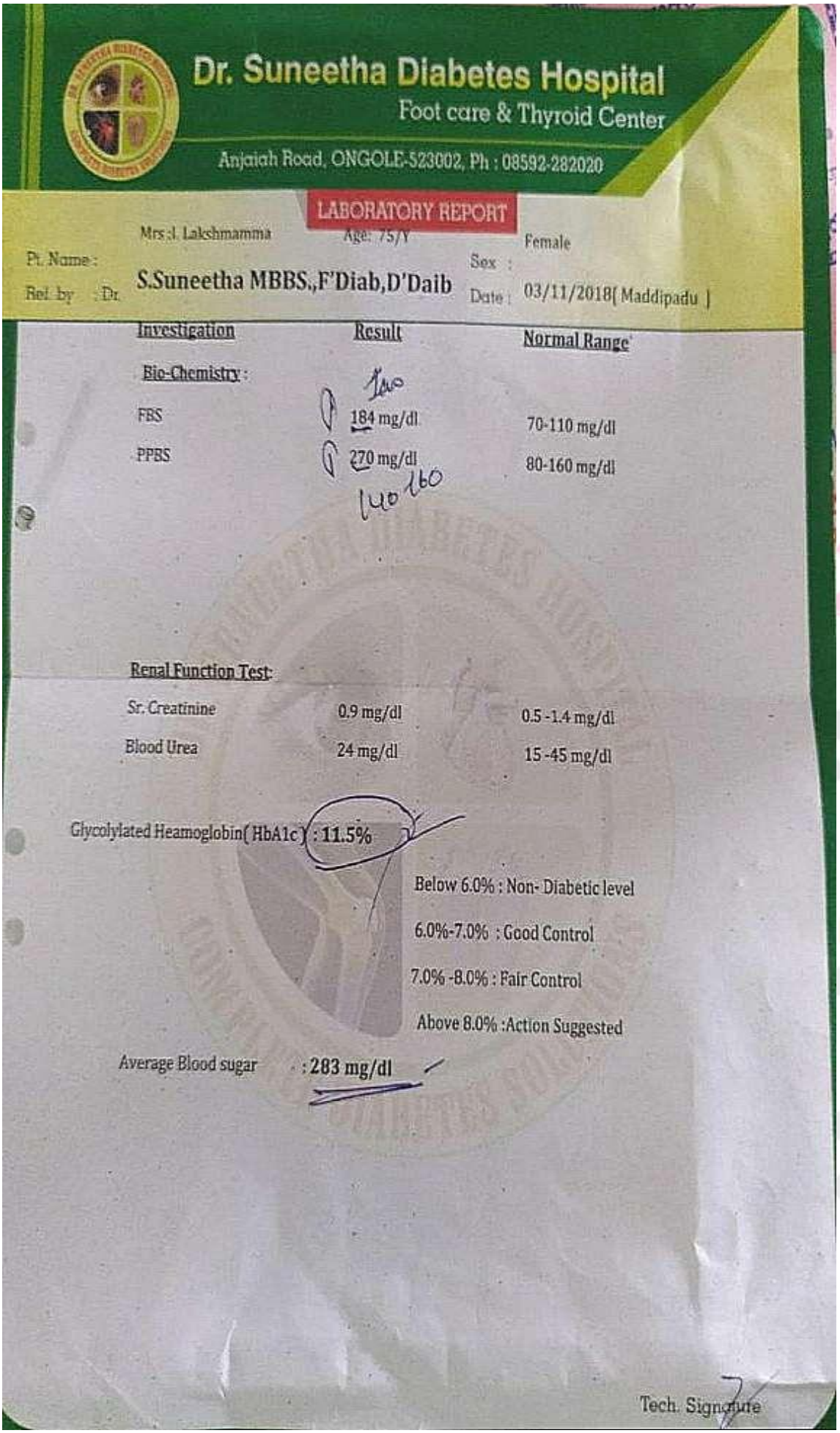
Age: 72 Years

Sex: Female

S.No.: 60

Date: 23-01-2017

TEST NAME	RESULT	REF. RANGE
Fasting Blood Sugar	152 mg/dl	80 - 100 mg/dl
Post Prandial Blood Sugar	278 mg/dl	100 - 160 mg/dl
Blood Urea	18.0 mg/dl	15 - 40 mg/dl
Serum Creatinine	0.65 mg/dl	0.5- 1.4 mg/dl
Total Cholesterol	201 mg/dl	120 - 200 mg/dl
Triglycerides	199 mg/dl	60-150 mg/dl



Dr. Suneetha Diabetes Hospital

Foot care & Thyroid Center

Anjaiah Road, ONGOLE-523002, Ph : 08592-282020

LABORATORY REPORT

Pt. Name : Mrs.J. Lakshmamma Age: 75/Y Sex : Female

Ref. by : Dr. S.Suneetha MBBS.,F'Diab,D'Daib Date : 03/11/2018(Maddipadu)

Investigation	Result	Normal Range
Bio-Chemistry :		
FBS	184 mg/dl	70-110 mg/dl
PPBS	270 mg/dl	80-160 mg/dl

Renal Function Test:

Sr. Creatinine	0.9 mg/dl	0.5 -1.4 mg/dl
Blood Urea	24 mg/dl	15 -45 mg/dl

Glycolylated Heamoglobin(HbA1c) : 11.5%

Below 6.0% : Non- Diabetic level

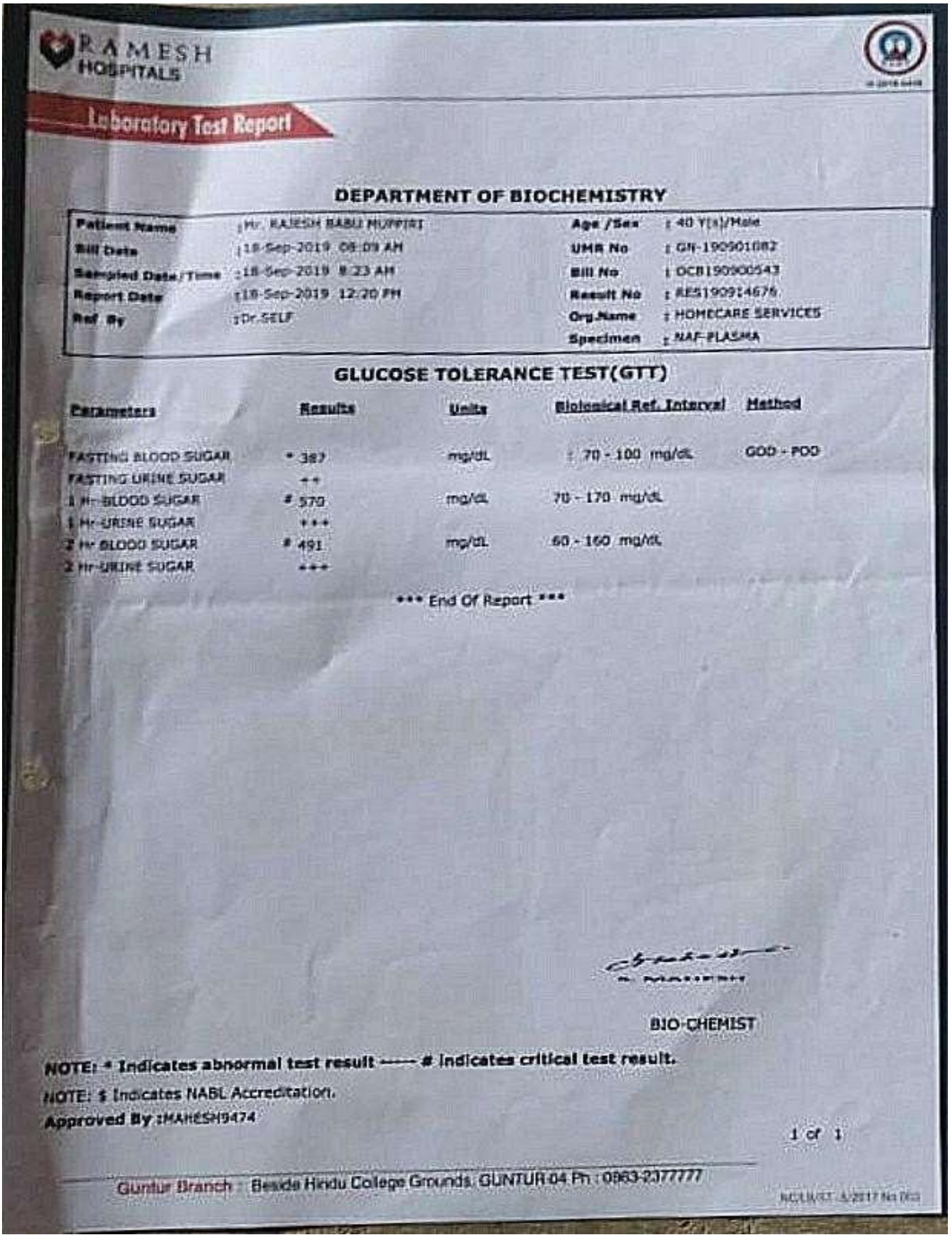
6.0%-7.0% : Good Control

7.0% -8.0% : Fair Control

Above 8.0% :Action Suggested

Average Blood sugar : 283 mg/dl

Tech. Signature



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Laboratory Test Report

DEPARTMENT OF BIOCHEMISTRY

Patient Name	:Mr. RAJESH BABU MUPPIRI	Age /Sex	: 40 Y(s)/Male
Bill Date	:18-Sep-2019 08:09 AM	UHR No	: GN-190901082
Sampled Date/Time	:18-Sep-2019 8:23 AM	Bill No	: OCB190900543
Report Date	:18-Sep-2019 12:20 PM	Result No	: RES190914676
Ref By	:Dr.SELF	Org.Name	: HOMECARE SERVICES
		Specimen	: NAF-PLASMA

GLUCOSE TOLERANCE TEST(GTT)

Parameters	Results	Units	Biological Ref. Interval	Method
FASTING BLOOD SUGAR	* 387	mg/dL	: 70 - 100 mg/dL	GOD - POD
FASTING URINE SUGAR	++			
1 Hr-BLOOD SUGAR	# 570	mg/dL	70 - 170 mg/dL	
1 Hr-URINE SUGAR	+++			
2 Hr BLOOD SUGAR	# 491	mg/dL	60 - 160 mg/dL	
2 Hr-URINE SUGAR	+++			

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RAMESH HOSPITALS

Laboratory Test Report

DEPARTMENT OF BIOCHEMISTRY

Patient Name	: Mrs. HEMA NALINI JUGUNTA	Age /Sex	: 67 Y(s)/Female
Bill Date	: 01-Nov-2019 12:40 PM	UMR No.	: GN-181102030
Sampled Date/Time	: 01-Nov-2019 12:42 PM	Bill No.	: B-GN191100117
Report Date	: 01-Nov-2019 01:44 PM	Result No	: RES191100611 / BIOOSE500174
Ref By	: DR.BHARANI BELLAM MD (GENERAL MEDICINE), DM (ENDOCRINOLOGY)	Org.Name	:
		Specimen	: NAF-PLASMA

Parameters	Results	Units	Biological Ref. Interval	Method
RANDOM BLOOD SUGAR	104	mg/dL	60 - 140 mg/dL	GOD-POD
HbA1c	6.5	%	Non diabetic level : 4.3 - 6.3 % Diabetic control : 6.4 - 7.9 % Poor control : 8.0 - 9.0 %	HPLC

*** End Of Report ***

M-0901

S. MAHESH

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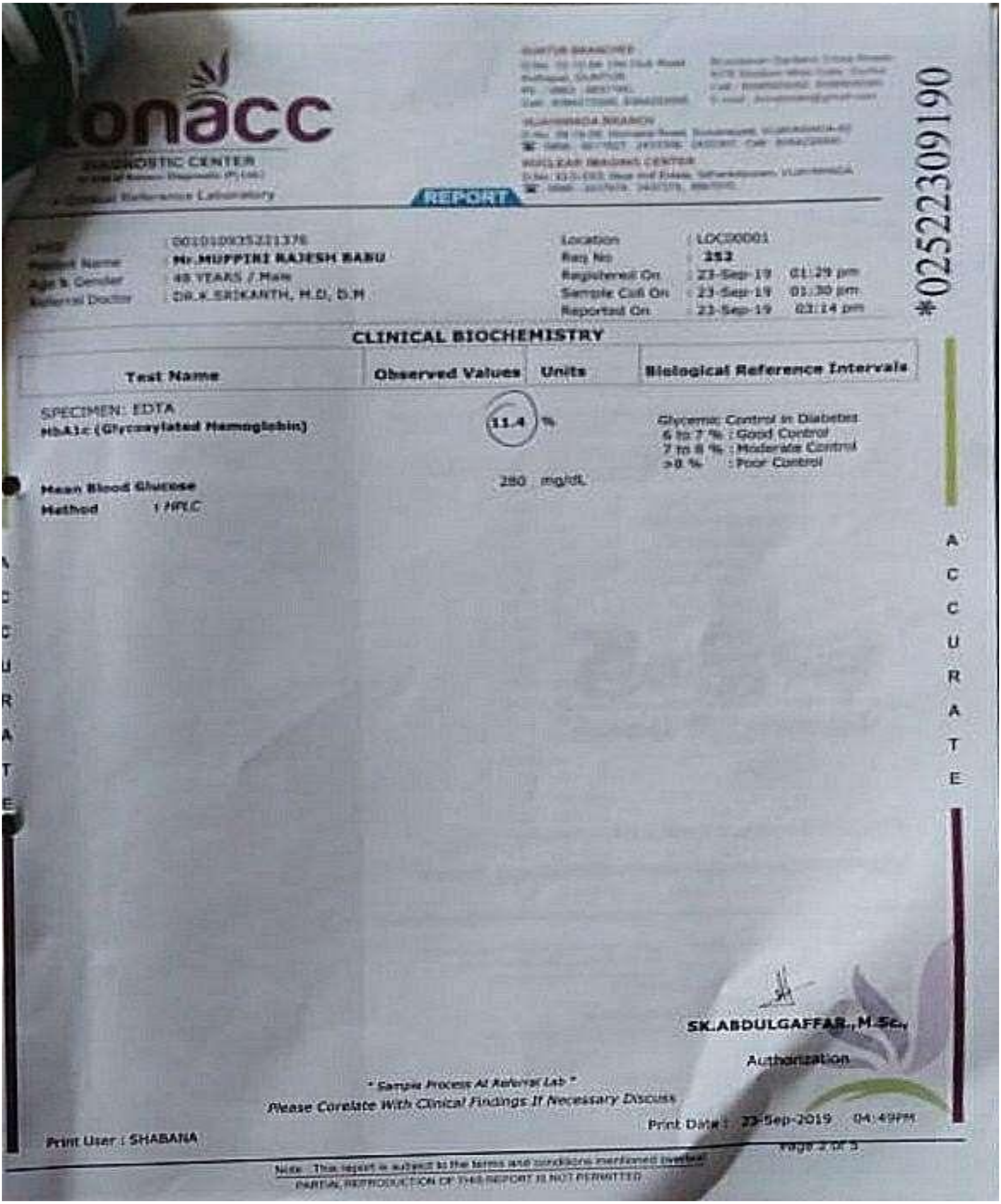

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DIAGNOSTIC CENTER

REPORT

*025222309190

	00101092522 1376	Location	: LOC00001
Patient Name	Mr.MUPPIRI RAJESH BABU	Req No	: 252
Age & Gender	48 YEARS / Male	Registered On	: 23-Sep-19 01:29 pm
Referral Doctor	DR.K.SRIKANTH, M.D, D.M	Sample Coll On	: 23-Sep-19 01:30 pm
		Reported On	: 23-Sep-19 03:14 pm

CLINICAL BIOCHEMISTRY

Test Name	Observed Values	Units	Biological Reference Intervals
SPECIMEN: EDTA			
HbA1c (Glycosylated Hemoglobin)	11.4	%	Glycemic Control in Diabetes 6 to 7 % : Good Control 7 to 8 % : Moderate Control >8 % : Poor Control
Mean Blood Glucose	280	mg/dL	
Method : HPLC			

ACCURATE

SK.ABDULGAFFAR., M.Sc.,

Authorization

* Sample Process At Referral Lab *

Please Corelate With Clinical Findings If Necessary Discuss

Print Date : 23-Sep-2019 04:49PM

Print User : SHABANA

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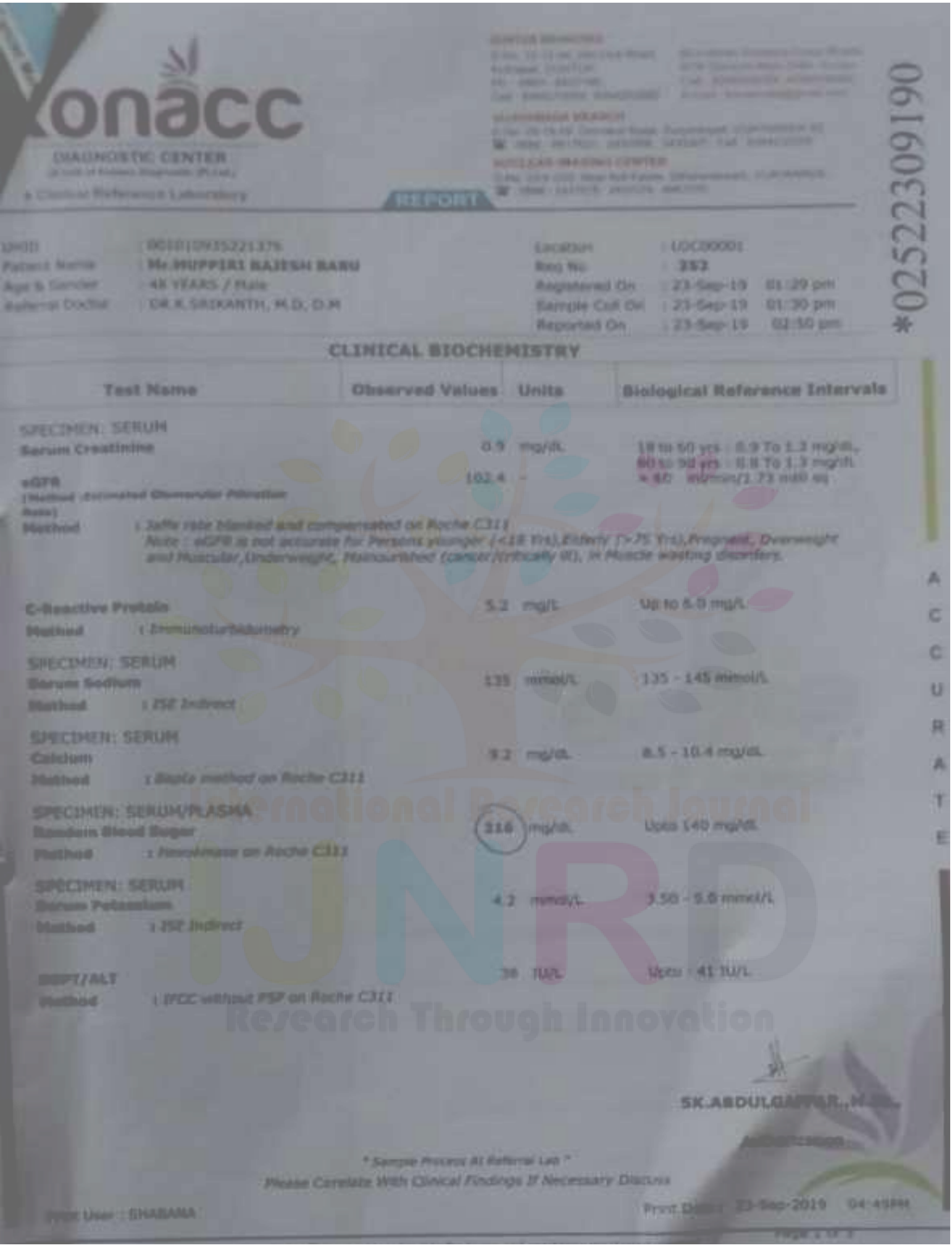
Vonacc
DIAGNOSTIC CENTER
Clinical Reference Laboratory

REPORT

0252223091900

UHID	: 0010109352213?6	Location	: UDC00001
Patient Name	: Mr.MUPPIRI RAJESH BABU	Reg No	: 352
Age & Gender	: 48 YEARS / Male	Registered On	: 23-Sep-19 01:29 pm
Referral Doctor	: DR.K.SRIKANTH, M.D, D.M	Sample Coll On	: 23-Sep-19 01:30 pm
		Reported On	: 23-Sep-19 02:50 pm

CLINICAL BIOCHEMISTRY

Test Name	Observed Values	Units	Biological Reference Intervals
SPECIMEN: SERUM			
Serum Creatinine	0.9	mg/dL	18 to 60 yrs : 0.9 To 1.3 mg/dL, 60 to 90 yrs : 0.8 To 1.3 mg/dL
eGFR (Method :Estimated Glomerular Filtration Rate)	102.4	-	> 60 ml/min/1.73 m^2^ sq
Method : *Jaffe rate blanked and compensated on Roche C311* *Note : eGFR is not accurate for Persons younger (<18 Yrs),Elderly (>75 Yrs),Pregnant, Overweight and Muscular,Underweight, Malnourished (cancer/critically ill), in Muscle wasting disorders.*			
C-Reactive Protein	5.2	mg/L	Up to 6.0 mg/L
Method : *Immunoturbidimetry*			
SPECIMEN: SERUM			
Serum Sodium	135	mmol/L	135 - 145 mmol/L
Method : *ISE Indirect*			
SPECIMEN: SERUM			
Calcium	9.2	mg/dL	8.5 - 10.4 mg/dL
Method : *Bapta method on Roche C311*			
SPECIMEN: SERUM/PLASMA			
Random Blood Sugar	316	mg/dL	Upto 140 mg/dL
Method : *Hexokinase on Roche C311*			
SPECIMEN: SERUM			
Serum Potassium	4.2	mmol/L	3.50 - 5.0 mmol/L
Method : *ISE Indirect*			
SGPT/ALT	38	IU/L	Upto : 41 IU/L
Method : *IFCC without P5P on Roche C311*			

ACCURATE

SK.ABDUL GAFFAR, M.Sc.,
Biochemist

" Sample Process At Referral Lab "
Please Correlate With Clinical Findings If Necessary Discuss

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Print Date : 23-Sep-2019 04:49PM
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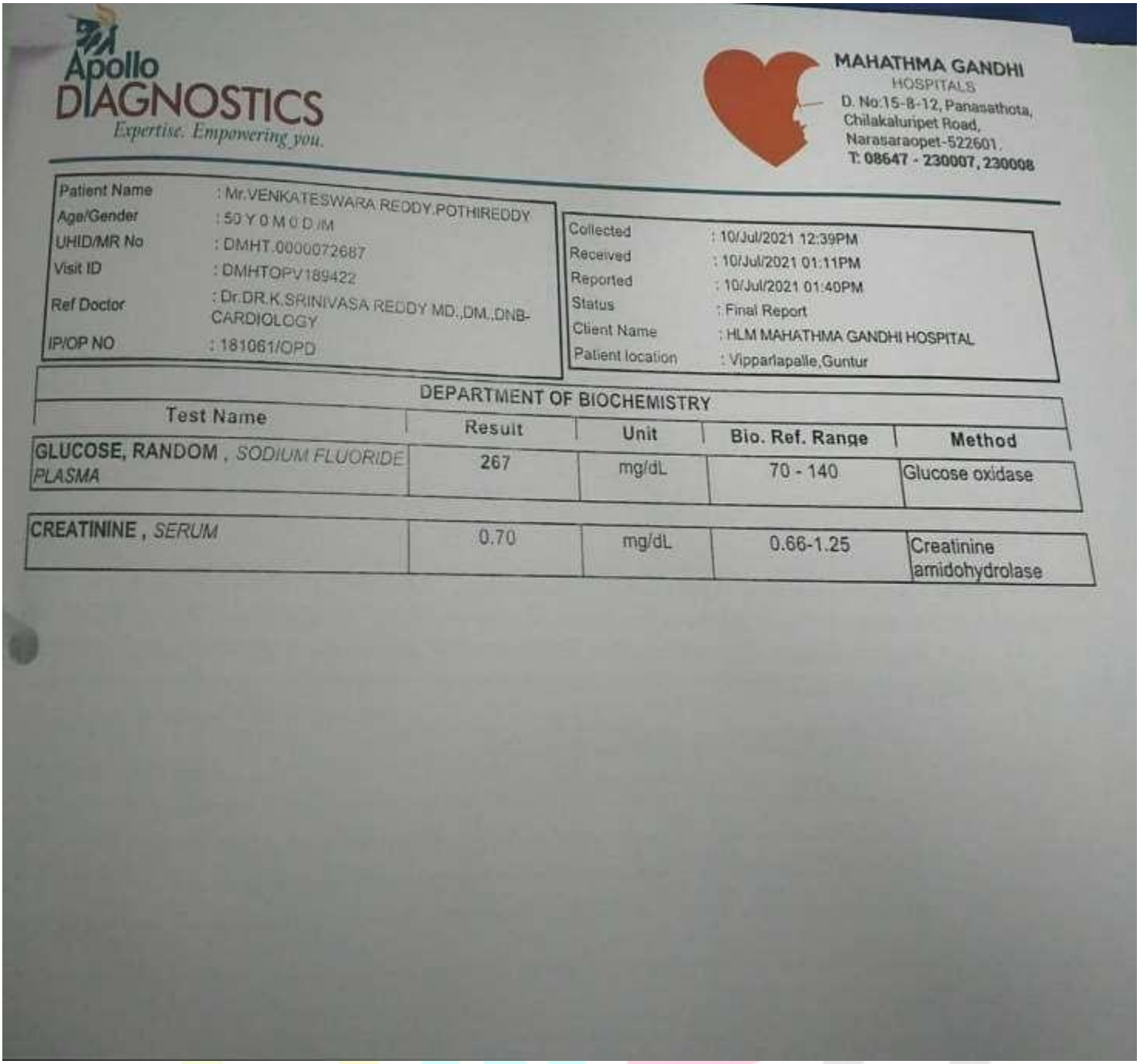

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D. No:15-8-12, Panasathota,
Chilakaluripet Road,
Narasaraopet-522601.
T: 08647 - 230007, 230008

Patient Name	: Mr.VENKATESWARA REDDY.POTHIREDDY	Collected	: 10/Jul/2021 12:39PM
Age/Gender	: 50 Y 0 M 0 D /M	Received	: 10/Jul/2021 01:11PM
UHID/MR No	: DMHT.0000072687	Reported	: 10/Jul/2021 01:40PM
Visit ID	: DMHTOPV189422	Status	: Final Report
Ref Doctor	: Dr.DR.K.SRINIVASA REDDY MD.,DM.,DNB-CARDIOLOGY	Client Name	: HLM MAHATHMA GANDHI HOSPITAL
IP/OP NO	: 181061/OPD	Patient location	: Vipparlapalle,Guntur

DEPARTMENT OF BIOCHEMISTRY

Test Name	Result	Unit	Bio. Ref. Range	Method
GLUCOSE, RANDOM , *SODIUM FLUORIDE PLASMA*	267	mg/dL	70 - 140	Glucose oxidase
CREATININE , *SERUM*	0.70	mg/dL	0.66-1.25	Creatinine amidohydrolase

RAMESH HOSPITALS

Laboratory Test Report

DEPARTMENT OF BIOCHEMISTRY

Patient Name	: Mr. GURAVAIAH KONA	Age /Sex	: 44 Y(s)/Male
Bill Date	: 02-Jul-2018 08:49 AM	UMR No.	: GN-180700046
Sampled Date/Time	: 02-Jul-2018 9:00 AM	Bill No.	: B-GN180700075
Report Date	: 02-Jul-2018 11:57 AM	Result No	: RES180700728 / BIOOSE290879
Ref By	: DR.CARDIOLOGY CONSULTATION	Org.Name	:
		Specimen	: Blood

Parameters	Results	Units	Biological Ref. Interval	Method
[illegible]41c	7.1	%	Non diabetic level : 4.3 - 6.3 % Diabetic control : 6.4 - 7.9 % Poor control : 8.0 - 9.0 %	HPLC

POST BREAK FAST BLOOD SUGAR(PPBS)

Post Prandial Blood Sugar	* 182	mg/dl	60 - 170 mg/dl	GOD-POD

*** End Of Report ***

M-0901

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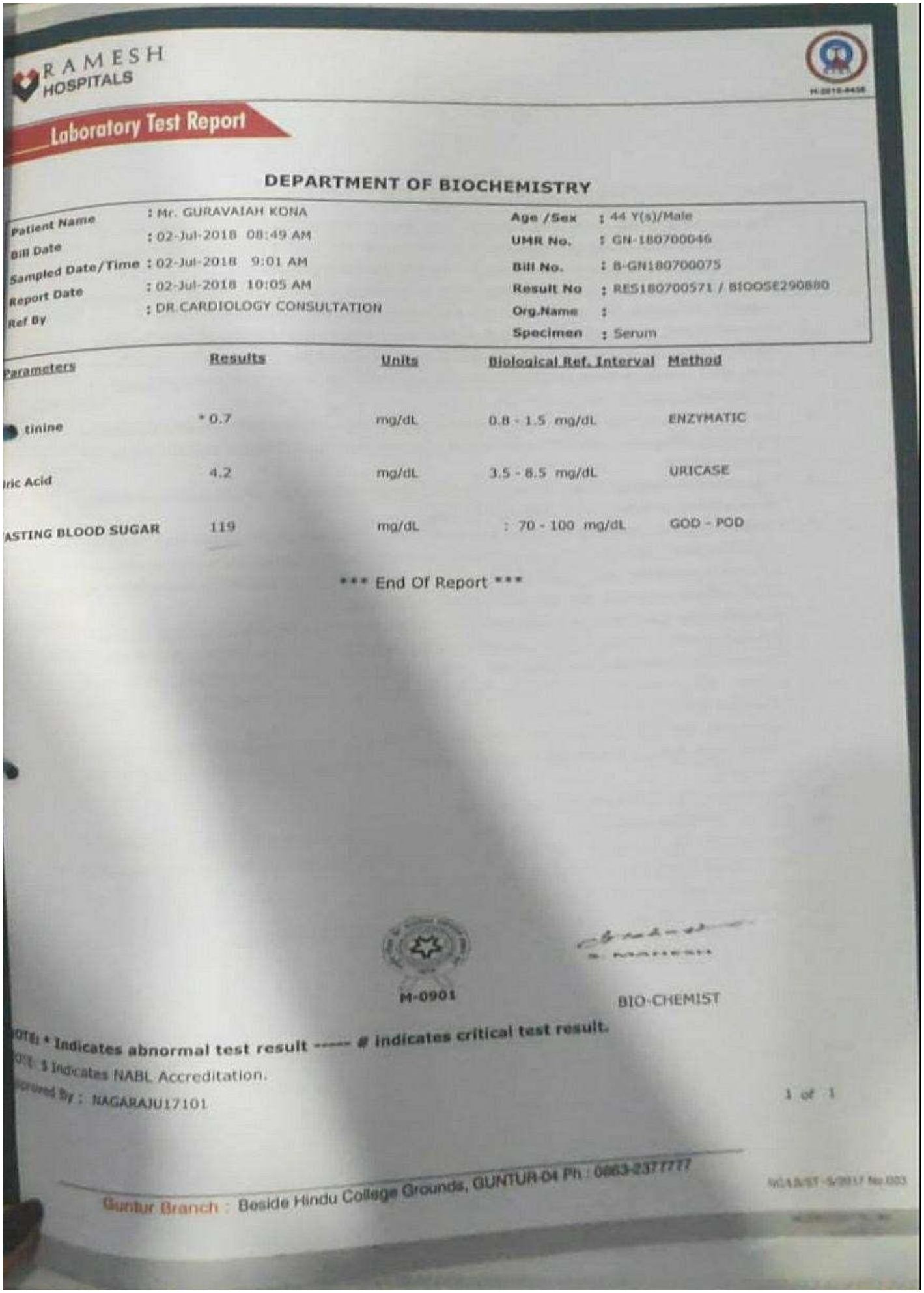

RAMESH HOSPITALS

Laboratory Test Report

DEPARTMENT OF BIOCHEMISTRY

Patient Name	: Mr. GURAVAIAH KONA	Age /Sex	: 44 Y(s)/Male
Bill Date	: 02-Jul-2018 08:49 AM	UMR No.	: GN-180700046
Sampled Date/Time	: 02-Jul-2018 9:01 AM	Bill No.	: B-GN180700075
Report Date	: 02-Jul-2018 10:05 AM	Result No	: RES180700571 / BIOOSE290880
Ref By	: DR.CARDIOLOGY CONSULTATION	Org.Name	:
		Specimen	: Serum

Parameters	Results	Units	Biological Ref. Interval	Method
tinine	* 0.7	mg/dL	0.8 - 1.5 mg/dL	ENZYMATIC
ric Acid	4.2	mg/dL	3.5 - 8.5 mg/dL	URICASE
ASTING BLOOD SUGAR	119	mg/dL	: 70 - 100 mg/dL	GOD - POD

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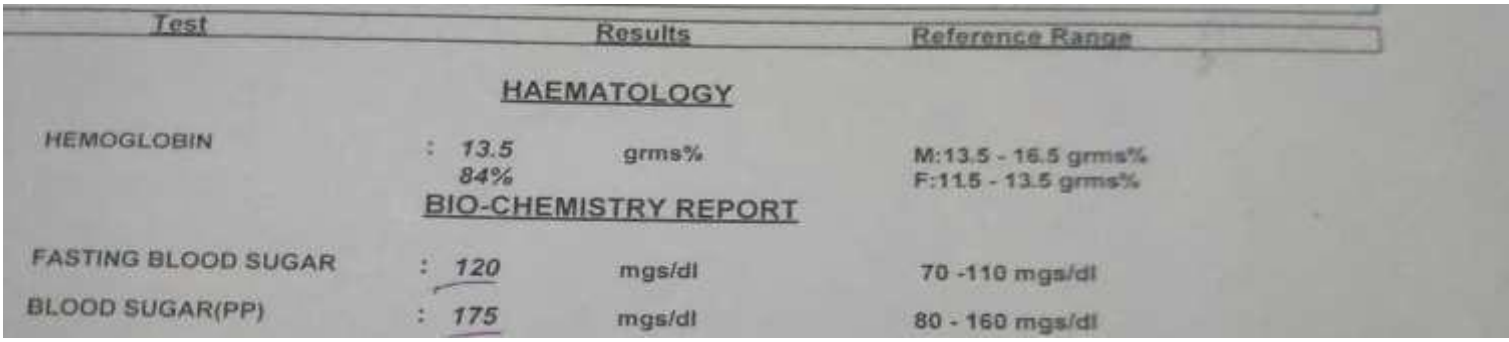


Test		Results	Reference Range
HAEMATOLOGY			
HEMOGLOBIN	:	13.5 grms%	M:13.5 - 16.5 grms%
		84%	F:11.5 - 13.5 grms%
BIO-CHEMISTRY REPORT			
FASTING BLOOD SUGAR	:	120 mgs/dl	70 -110 mgs/dl
BLOOD SUGAR(PP)	:	175 mgs/dl	80 - 160 mgs/dl

NABL Accredited Laboratory

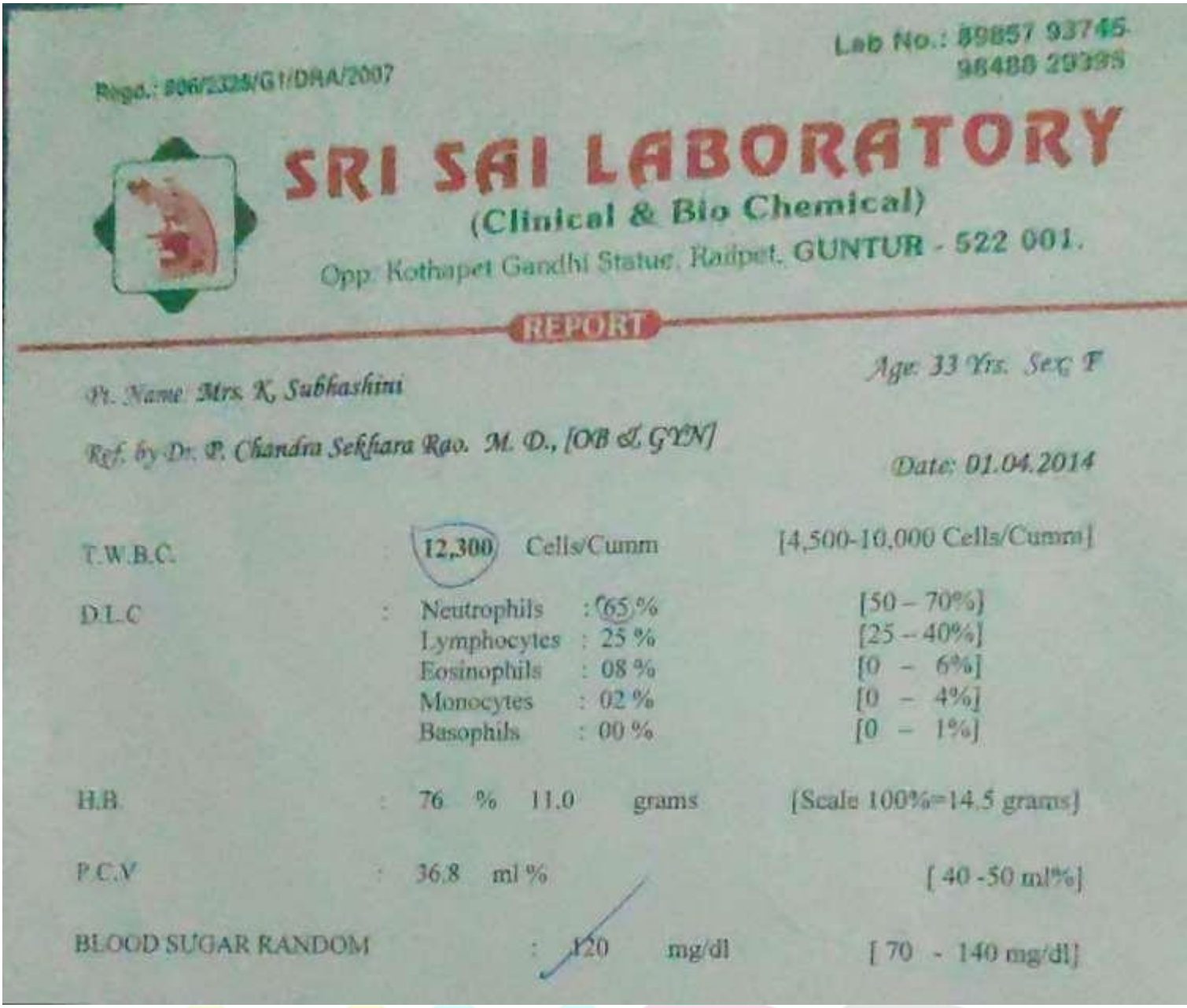
Name	: Mrs. J. HEMANALINI		
PID No.	: L2L3517151	Register On	: 16/10/2020 12:15 PM
SID No.	: 75525043	Collection On	: 16/10/2020 12:22 PM
Age / Sex	: 67 Year(s) / Female	Report On	: 16/10/2020 3:34 PM
Type	: OP	Printed On	: 16/10/2020 3:39 PM
Ref. Dr	: C/O RAVI		

Investigation	Observed Value	Unit	Biological Reference Interval
HAEMATOLOGY			
Haemoglobin *(EDTA Blood/Photometry – Cell counter)	**10.94**	g/dL	12.5 - 16.0
BIOCHEMISTRY			
Glucose Fasting (FBS) *(Plasma - F/Glucose oxidase/Peroxidase)	**328**	mg/dL	70 - 110
INTERPRETATION: Factors such as type, quantity and time of food intake, Physical activity, Psychological stress, and drugs ca influence blood glucose level.			
Glycosylated Haemoglobin (HbA1c)			
HbA1C *(Whole Blood/HPLC-Ion exchange)	**13.5**	%	Normal: 4.0 - 5.6 Prediabetes: 5.7 - 6.4 Diabetic: >= 6.5
INTERPRETATION: Detection Limit :3.8-18.5%			
Estimated Blood Glucose *(Blood/Derived)	340.75	mg/dl	
INTERPRETATION: NOTE : Conditions like Uremia, hypertriglyceridemia,hyperbilirubinemia, Alcohol, Aspiria, Aplastic Anaemi and presence of HbF can give false elevated values. Severe nephropathy, acute or chronic blood loss, hemolytic anaemia, pregnancy etc., can cause falsely low HbA1c. GHB measurement is not appropriate when there has been a change in diet or treatment within 6 weeks.			
IMMUNOASSAY			
THYROID PROFILE / TFT			
T3 (Triiodothyronine) - Total *(Serum/ Chemiluminescent Immunometric Assay (CLIA))	**0.43**	ng/mL	0.8 - 1.6
T4 (Thyroxine) - Total *(Serum/ Chemiluminescent Immunometric Assay (CLIA))	9.87	µg/dL	4.2 - 12.0
TSH (Thyroid Stimulating Hormone) * (Serum/Chemiluminescence)	1.95	µIU/mL	0.4 - 4.2

NAGESHWARARAO.M.SC BIOCHEMISTRY
BIOCHEMIST

DR BHANUPRIYA KAKARALA,MD
Consultant Pathologist
Reg. No. : APMC/FMR/74647
APPROVED BY

VERIFIED BY



Regd.: 806/2328/G1/DRA/2007

Lab No.: 89857 93745
96488 29333

SRI SAI LABORATORY

(Clinical & Bio Chemical)

Opp: Kothapet Gandhi Statue, Ratpet, GUNTUR - 522 001.

REPORT

Pt. Name: Mrs. K. Subhashini — Age: 33 Yrs. Sex: F

Ref. by Dr. P. Chandra Sekhara Rao. M. D., [OB & GYN] — Date: 01.04.2014

T.W.B.C.	12,300 Cells/Cumm		[4,500-10,000 Cells/Cumm]
D.L.C	Neutrophils	: 65 %	[50 – 70%]
	Lymphocytes	: 25 %	[25 – 40%]
	Eosinophils	: 08 %	[0 – 6%]
	Monocytes	: 02 %	[0 – 4%]
	Basophils	: 00 %	[0 – 1%]
H.B	: 76 % 11.0	grams	[Scale 100%=14.5 grams]
P.C.V	: 36.8 ml %		[40 -50 ml%]
BLOOD SUGAR RANDOM	: 120	mg/dl	[70 - 140 mg/dl]

CONCLUSION

The prevalence of diabetes, especially type 2 diabetes and hypertension are significantly increased due, at least in part, to the increased prevalence of obesity. Type 2 diabetes is frequently associated with obesity, and is an important risk factor for cardiovascular morbidity and mortality and cardiac- and renal complications. Type 2 diabetes, hypertension and obesity are characterized by stimulation of the renin-angiotensin-aldosterone system (RAAS), elevated sympathetic activity and insulin resistance. Importantly, these characteristics, themselves, confer cardiovascular risk. Therefore, treatments for type 2 diabetes should be selected from favourable effects on stimulated RAAS, elevated sympathetic nervous system activity, insulin and leptin resistance.

Weight loss is recommended as the first line of treatment for type 2 diabetes and hypertension associated with type 2 diabetes in obesity. Lifestyle modification such as a caloric restricted diet, reducing sedentary behaviour and increases in exercise form the basis of all therapy. Weight loss treated with lifestyle modification including calorie restriction and/or exercise causes normalization of stimulated RAAS,

sympathetic activation, insulin resistance, and hyperleptinemia. Recently, Masuo *et al*. and Straznicky *et al.* have shown that low caloric diet and exercise have different effects on insulin resistance, the RAAS, and sympathetic nervous activity in obese hypertensive subjects, even though similar weight loss was observed. statement from American Heart Association reported that high-protein diet and low carbohydrate diet are not recommended diets due to increases in cardiovascular risk.

Gastric bypass and adjustable gastric banding are the two most commonly performed bariatric procedures for the treatment of morbid obesity or obesity which is resistant to lifestyle modification such as a low caloric diet plus exercise. Weight loss by bariatric surgery leads to improvement or normalization of glucose metabolisms from multiple mechanisms including caloric restriction, changes in the enteroinsular axis, alterations in the adipoinsular axis, release of nutrient-stimulated hormones from endocrine organs, stimulation from the nervous system, and psychosocial aspects including a dramatic improvement in quality of life.

Understanding the mechanisms underlying type 2 diabetes in obesity may help to achieve weight loss and maintenance of weight loss and resultant better control on type 2 diabetes, and delay and prevent the onset of type 2 diabetes or reduce complications.

This review provides information regarding, *i)* the importance of lifestyle medication on type 2 diabetes in obesity, *ii)* different effects of lifestyle modifications on weight loss and neurohormonal parameters between diet and exercise, and *iii)* the mode of weight loss and how it influences different physiological pathways. Different mechanisms may contribute to control in blood glucose levels and blood pressure and cardiovascular risks associated with weight loss with the relevant physiological mechanisms at play being dependent on the mode of weight loss.

BIBLIOGRAPHY

[1] American Diabetes Association. Evidence-Based Nutrition Principles and Recommendations for the Treatment and Prevention of Diabetes and Related Complications. Diabetes Care January s60.Toji C, Okamoto N, Kobayashi T, Furukawa Y, Tanaka S, Ueji K, et al. Effectiveness of diet versus exercise intervention on weight reduction in local Japanese residents. Environ Health Prev Med.

[2] Masuo K, Kawaguchi H, Mikami H, Ogihara T, Tuck ML. Serum uric acid and plasma norepinephrine concentrations predict subsequent weight gain and bloodpressure elevation. Hypertension.

[3] Straznicky NE, Eikelis N, Nestel PJ,Dixon JB, Dawood T, Grima MT, et al. Baseline sympathetic nervous system activity predicts dietary weight loss in obese metabolic syndrome subjects. J Clin Endocrinol Metab.

[4] Ribeiro MM, Silva AG, Santos NS, Guazzalle I, Mates LN, Tronbetta IC, et al. Diet and exercise training restore blood pressure and vasodilatory responses during physiological maneuvers in obese children. Circulation.

[5] Trombetta IC, Batalha LT, Rondon MU, et al. Gly16 + Glu27 beta2-adrenoceptor polymorphisms cause increased forearm blood flow responses to mental stress and handgrip in humans. J Appl Physiol.

[6] Tonacio AC, Trombetta IC, Rondon MU, Laterza MC, Frazzatto E, Alves MJ, et al. Effects of diet and exercise training on neurovascular control during mental stress in obese women. Braz J Med Biol .

[7] Santarpia L, Contaldo F, Pasanisi F. Body composition changes after weight-loss interventions for overweight and obesity. Clin Nutr.

[8] Dumortier M, Brandou F, Perez-Martin A, Fedou C, Mercier J, Brun JF. Low intensity endurance exercise targeted for lipid oxidation improves body composition and insulin sensitivity in patients with the metabolic syndrome. Diabetes Metab.

[9] Poirier P, Pespres JP. Exercise in weight management of obesity. Cardio Clin.

[10] Skilton MR, Mikkilä V, Würtz P, Ala-Korpela M, Sim KA, Soinuinen P, et al. Fetal growth, omega-3 (n-3) fatty acids, and progression of subclinical atherosclerosis: preventing fetal origins of disease? The Cardiovascular Risk in Young Finns Study. Am J Clin Nutr.

[11] Mozaffarian D, Abdollahi M, Campos H, Houshiarrad A, Willett WC. Consumption of trans-fats and estimated effects on coronary heart disease in Iran. Eur J Clin Nutr.

[12] Whitman SC, Rateri DL, Szilvassy SJ, Yokoyama W, Daugherty A. Depletion of natural killer cell function decreases atherosclerosis in low-density lipoprotein receptor null mice. Arterioscler Thromb Vasc Biol.

[13] Teegala SM, Willett WC, Mozaffarian D. Consumption and health effects of trans fatty acids: a review. J AOAC .

[14] Larsson SC, Virtamo J, Wolk A. Dietary fats and dietary cholesterol and risk of stroke in women. Atherosclerosis.

[15] Chowdhury R, Stevens S, Gorman D, Pan A, Wamakula S, Chowdhury S, et al. Association between fish consumption, long chain omega 3 fatty acids, and risk of cerebrovascular disease: systematic review and meta-analysis. BMJ

[16] Oh K, Hu FB, Manson JAE, Stampfer MJ, Willett WC. Dietary Fat Intake and Risk of Coronary Heart Disease in Women: 20 Years of Follow-up of the Nurses' Health Study. Am. J. Epidemiol.

[17] Storlien LH, Baur LA, Kriketos AD, Pan DA, Cooney GJ, Jenkins AB, et al. Dietary fats and insulin action. Diabetologia

[18] Halton TL, Willett WC, Liu S, Manson JE, Albert CM, Rexrode K, et al. Low- Carbohydrate-Diet Score and the Risk of Coronary Heart Disease in Women. N EnglJ Med.