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HAEMOGRAM

EDTA whole Blood

TEST		RESULT	UNIT	BIOLOGICAL REF INTERVAL
HAEMOGLOBIN	:	13.8	gms/dl	13.5 - 18.0
RBC INDICES				
Total R.B.C. Count	:	4.93	millions/cu.mm	4.5 - 6.5
Packed Cell Volume	:	42.1	%	40 - 54
M. C. V.	:	85.4	cu.micron	78 - 96
M. C. H.	:	28.0	picogram	27 - 32
M. C. H. C.	:	32.8	g / dl	30 - 35
R. D. W.	:	12.9	%	11 - 15
TOTAL W. B. C. COUNT	:	6060	/cu.mm	4000 - 11000
DIFFERENTIAL COUNT				
Neutrophils	:	58.7	%	50 - 70
Lymphocytes	:	32.7	%	20 - 40
Eosinophils	:	2.0	%	01 - 04
Monocytes	:	6.1	%	02 - 06
Basophils	:	0.5	%	00 - 01
PLATELET COUNT	:	1.41	Lakh	1.5 - 4.5
E. S. R. (At 1st Hour) (Westergren's method)	:	14	mm	02 - 15

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C - REACTIVE PROTEIN serum

PRINCIPLE	:	Particle enhanced turbidimetric assay
CONCENTRATION	:	**2.5 mg / l**
Biological Ref Interval	:	Less than 6 mg / l
RESULT	:	**Negative**

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PLASMA GLUCOSE / BLOOD SUGAR -URINE SUGAR ESTIMATION

TESTS	RESULTS	UNITS	BIOLOGICAL REF INTERVAL
PLASMA GLUCOSE			
FBS (Fasting blood sugar)	105	mg / dl	60 - 110
FUS (Fasting urine sugar)	Absent		Absent
URINE ACETONE			

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Hb A1c - Fact file

Glycohaemoglobin is being used with increasing frequency to monitor long term blood glucose control and compliance in patient with diabetes mellitus. It provides an index of mean concentration of blood glucose (eAG - estimated average glucose) during the preceding two to three months. It complements more traditional measures of glucose control such as blood and urine glucose level.

The term " Glycohaemoglobin / Glycosylated haemoglobin / Hb A1c " refers to a series of minor haemoglobin components that are **stable adducts formed by haemoglobin with various sugars.** The reaction between haemoglobin and sugar is an example of a nonenzymatic condensation of glucose with free amino groups on the globin (NH2 terminal valine of B chain). The process is slow , continuous and irreversible.

Human erythrocytes are freely permeable to glucose , **Within each erythrocyte GHb is formed from haemoglobin at the rate which depends on the ambient concentration of glucose. Higher the prevailing ambient level of blood glucose , higher the GHb**

The level of Hb A1c at any point of time is contributed to by all circulating erythrocytes , from oldest (120 days) to the yongest RBCs. Hense Hb A1c is a **" weighted average " of glucose during the preciding three months** , mear preceding 30 days contributes substantiallly more to the level of Hb A1c than do glucose level 90 -120 days erlier..

There is very predictable relationship between Hb A1c and eAG (estimated average glucose / weighted average glucose).
A formula based on linear regression analysis sponsered by ADA , EASD , IDF is eAG (mg / dl) = (28.7 * Hb A1c) - 46.7 replacing the older one eAG (mg / dl) = (35.6*Hb A1c) - 77.3 recommended by DCCT .
Post lunch and bedtime glucose correlate well with Hb A1c (data of full 7 point glucose profile by capillary blood). Fasting glucose correlates less well and with increasing Hb A1c and average glucose (eAG) from 7 point profile , it underestimate both the values.

Any cause of shortened red cell survival will reduce exposure of red cells to glucose with consequent decrease in GHb values e.g. haemolytic anaemias like Hb S , Hb C Harlem , Hb E , Hb D . Chronic blood loss , Acute recent blood loss , etc.
A falsely low GHb test results are also be noted in c/o high Hb F samples . Thalassemia major , HPFH and othe conditions. (Hb F > 10 %) . Glycated Hb F is not detected by assay as it does not contain the glycated B chain that characterizes Hb A1c.

A falsely elevated GHb test results can be caused by " labile GHb " - an acutely generated reversible nonenzymatic linked glucose intermediary product present after heavy meal .
Few medical condition may cause falsely eleveted Hb A1c , like uremia , chronic excessive alcohol intake and hypertriglyceridemia.
Thalassemia minor causing imbalance between the synthesis or a and b chain causes falsely eleveted values.
Chronic iron deficiency anaemia in which there is an increased erythrocyte life span can raise GHb falsely.
Gestational diabetes may falsely increase or decrease Hb A1c
Studies even relate Hb A1c values with temperature / seasonal variation . Values are higher in cooler months and lower in warmer months (June - July).

IS SPECIFIC PATIENT PREPARATION IS NECESSORY ?

In any case , **no fasting sample is required at all . Avoid alcohol intake and heavy meal before the assay**
Hb A1c results are to be reported by clinical laboratories world wide in SI units (mmol / mol - no decimals) or derived NGSP units (% - one decimal). using IFCC - NGSP master equation,

An international expert committee that includes representatives from American Diabetes Association (ADA) , International

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Diabetes Federation (IDF) , and European Association for the Study of Diabetes (EASD) has **officially endorsed the Hb A1c as the Diagnostic test for the Diabetes . Cut off is >= 6.5**

WHY HB A1c IS CLINICALLY IMPORTANT

According to DCCT , a linear relationship is documented between the lowering Hb A1c / eAG and prevention of chronic complications . If Hb A1c is mainained at or about the upper level of normal , it can reduce app. 75 % retinal (eye) 60 % neuronal (nervous system) and 35 % renal (kidney) complications.

A reliable at-risk marker for assessing possible development of complications
An excellent mean to assess , encourage and reinforce individual patient compliance
An indicator for aditional diagnostic tests such as Urine Microalbumin
A meaningful Mean Blood Glucose (MBG) / estimated Average Glucose (eAG) relationship
An useful method to judge the efficacy and effectiveness of intervention strategies.

WHAT ARE THE ADA RECOMMENDATIONS TO PREVENT COMPLICATED DIABETES

* Perform the A1c test at least two times a year in patient who are meeting treatment goals and have stable glycemic control
* Perform the A1c test quarterly in patient whose therapy has changed or who are not meeting glycemic control.
* Use of point-of-care testing for A1c allow for timely decisions on therapy changes when needed.

GLYCEMIC GOAL IN ADULT BY ADA.

To reduce microvascular and neuropathic complications of type 1 and type 2 diabetic patients - **below or around 7 %**

REFERENCES (Data published by)

1. American Diabetic Association (ADA)
2. Internationa Diabetic Federation (IDF)
3. European Association for Study of Diabetes (EASD)
4. American Association of Clinical Endocrinologist
5. National Glycohaemoglobin Standardization Programme (NGSP)
6. Diabete Control and Complications Trial (DCCT)
7. A Journal of Disease metabolism

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Glycosylated Hb / Glycated Hb / Hb A1c

Method	Tina - quant
Interpretation (% Glyco Hb)	
> 8	Poor glycemic control
7 - 8	Fair glycemic control
6 - 6.9	Good glycemic control
< 6	Non-diabetic level / Near normal glycemia
Result	**5.5**
estimated Average Glucose (eAG)	**111.2 mg / dl**
Comment	

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BIOCHEMICAL ANALYSIS
(Serum)

APPEARANCE : CLEAR

TESTS	RESULTS	UNITS	Biological Ref Interval
S.G.P.T. (ALT) method : UV kinetic	22	U / L	< 40
S.G.O.T. (AST) method : UV kinetic	16	U / L	5 - 34
S. PROTEINS			
TOTAL PROTEINS method : BIURET colorimetric	6.43	gm / dl	6.0 - 7.8
ALBUMIN method : BCG colorimetric	3.78	gm / dl	Adult : 3.5 - 5.2 60 - 90yr : 3.5 - 4.6 >90yr : 2.9- 4.5
GLOBULINS	2.6	gm / dl	2.3 - 3.5
A / G RATIO	1.5	%	
SERUM URIC ACID method : Enzymatic	4.4	mg / dl	M 3.5 to7.2 F :2.6 to 6.0
S.CREATININE method : Jafferate	1.01	mg / dl	M : 0.7 TO 1.4 F : 0.6TO 1.1

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RENAL FUNCTION TESTS

TESTS	RESULTS	UNITS	BIOLOGICAL REF INTERVAL
BLOOD UREA	22	mg / dl	15 - 40
S. CALCIUM	8.0	mg / dl	8.4 - 11.0

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LIPID PROFILE serum
APPEARANCE : Clear

TESTS	RESULTS	UNITS	BIOLOGICAL REF INTERVAL
CHOLESTEROL	217	mg / dl	< 200
TRIGLYCERIDES	119	mg / dl	< 200
HDL CHOLESTEROL (Direct)	51.7	mg / dl	45 - 85
Non HDL CHOLESTEROL	165	mg / dl	
LDL CHOLESTEROL	142	mg / dl	Less than 130
VLDL	24	mg / dl	upto 34
CHOL : HDL RATIO	4.2	%	2.5 - 4.5

NCEP RISK CLASSIFICATION

TESTS	DESIRABLE	MODERATE RISK	HIGH RISK
LDL CHOLESTEROL	< 130	130 - 160	> 160
HDL CHOLESTEROL	> 60	35 - 59	< 35
TOTAL CHOLESTEROL	< 200	200 - 239	> 240
CHOL / HDL RATIO	3.3 - 4.4	4.4 - 11	> 11
LDL / HDL RATIO	0.5 - 3.0	3.0 - 6.0	> 6.0
TRIGLYCERIDE	< 200	200 - 400	400 - 1000
APO A / APO B RATIO	> 1.2	-	< 1.3
APOLIPORPOTEIN (a)	< 30	-	>

Desirable levels	: Non-HDL cholestrol
Less than 100	Ideal for people at high cardiac risk
100 to 130	Ideal for people at low cardiac risk
130 to 159	Ideal - near ideal for healthy population
160 to 189	Mildly/borderline elevated
190 to 219	Intermediuate / moderately elevated
More than 200	Severly elevated / very high

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SERUM ELECTROLYTES

TESTS	RESULTS	UNITS	NORMALS
S. SODIUM	**138**	mmol/L	135 - 145
S. POTASSIUM	**4.2**	mmol/L	3.5 - 5.5
S. CHLORIDE	**102**	mmol/L	98 - 108
S. BICARBONATE	**25**	mmol/L	21 - 31

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PSA Fact file

Prostate-specific antigen (PSA) is a glycoprotein produced by prostate gland , bulbourethral glands and urethral lining . PSA exists in serum in multiple forms : Complex to alpha-1-anti-chymotrypsin (PSA-ACT complex)unbounded (free PSA) and enveloped by alpha-2-macroglobulin (not detected by immunoassays).

PSA alone is neither specific nor sensitive as nonmalignant conditions like BPH , Acute renal failure prostatitis , prostatic infarct , following TURP , Niddle biopsy , and DRE may cause false high PSA causing low specificity of the total PSA and normal PSA (less than 4) in many organ confined malignancies causing low sensitivity of the assay.

App. 20 % Cases of BPH have PSA level between 4 to 10 ng / ml and 2 % have PSA level more than 10 ng / ml

Only 2 % of cancers can be detected by screening of healthy asymptomatic adult males .Cases detected by Digital Rectal Examination (DRE) and PSA screening may not be the same On the contrary DRE alone detects app. 50 % organ confined cancers , hence must be used in conjugation with DRE.

FREE V/S TOTAL PSA

Higher total PSA levels and lower percentages of free PSA are associated with high risk of prostate cancer when the total PSA is in the range of 4 to 10 ng / ml (gray zone) , a free to total PSA ratio less than 0.10 indicates 49 to 65 % risk of prostatic carcinoma. At the same time with free to total PSA ratio > 0.25 , risk of prostatic malignancy drastically reduces to 9 to 16 % depending on the age . So it is more important to measure free and total PSA in conjugation.

AGE SPECIFIC PSA LEVEL

Because of man's PSA level tends to increase with age , use of age specific PSA level may increases the accuracy of PSA test and avoids the unnecessary prostatic biopsies in older man , on the other hand it may delay the detection of prostatic carcinoma in middle age group so till date use of age specific PSA level is controversial .

age yrs	**PSA ng / ml**
less than 40	< 2.0
40 - 49	< 2.5
50 - 59	< 3.5
60 - 69	< 4.5
70 - 79	< 6.5
80 and more	< 7.2

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PSA VELOCITY

PSA Velocity is the rate of change in man's PSA level over a period of time expressed in ng / ml/ year. PSA **increase of more than 0.75 ng / ml / year** or more than 20 % of base line PSA indicates possible malignancy . It requires at least three redaings over 18 months period

PSA DENSITY

it is a ratio of total PSA to prostate gland volume measured by transrectal sonography.
Helps to distinguish BPH from Prostatic carcinoma , especially in gray zone (PSA 4 to 10 ng / ml)
Density more than 0.15 % favours malignancy

DIAGNOSIS , FOLLOW UP AND MONITORING

In c/o confirmed prostatic carcinoma

< 4 ng / ml will have organ confined disease
< 10 ng / ml , bone metastasis are rare
> 10 ng / ml , 50 % will have extracapsular disease
> 50 ng / ml , most will have lymphnode metastasis
> 100 ng / ml , bone metastasis are likely with s/s of 66 and 90 % respectivelly

Following radical surgery within 3 months PSA reaches to undetectabel level and following successful radiotherapy , it takes almost 6 months to reach normal level.

3rd Generation PSA / ultrasensitive PSA has sensitivity upto 0.003 ng / ml (American Metabolic Laboratory). It is the best guide for follow up cases to predict recurrence and disease therapy.
For highsensitive 3rd generation assay , normal range is 0 to 2.8 ng / ml.

In C/O prostatic carcinoma , 3rd Generation PSA is very much helpful in follow up after prostatectomy. It gives informations much earlier than 2nd generation PSA . Any rising trends in total PSA level measured 3 consicutive times within 3 to 6 wks sugggest possible recurrence , even with total PSA withiin normal limit.

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TUMOUR MARKER - TOTAL PROSTATE SPECIFIC ANTIGEN (TPSA)/ hybritech PSA

PRINCIPLE : CHEMILUMINESCENCE

NORMAL : < 4.0 ng / ml

RESULT : **7.40 ng / ml**

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TROPONIN I HIGH SENSITIVE (TNHS)

An automated quantitative assay for the determination of cardiac troponin I by , mini - VIDAS

TEST	RESULT	UNIT	Biological Ref.Interval
TROPONIN I HIGH SENSITIVE Enzyme linked fluorescent assay	8.4 Normal	ng / l	< 19 Normal healthy individual 19 to 99 Possible myocardial damage >= 100 Acute Myocardial Infarct

Troponin is a regulatory protein of the thin filament of striated muscle, and consists of three subunits I , T , and C.
Troponin I has a cardiac isoform which enables highly specific detection of myocardial injury. This isoform is rapidly released after acute myocardial infarction (AMI) and can be detacted in blood between 4th and 8th hour after the onset of chest pain , with a peak between 14th and 36th hours. Concentrations in blood remain high for 3 to 7 days.
Cardiac troponin is the biomarker of choice for detection of myocardial necrosis as it is more specific and sensitive than classic cardiac enzymes CK and CK MB .
The recommendation of the consensus committee of the European society for cardiology (ESC) and the American college of cardiology (ACC) specify that the diagnosis of myocardial necrosis can be made when level of cardiac troponin in the blood is greater than the 99th percentile of a healthy population in the clinical setting of acute ischemia .
Patients presenting an acute coronary syndrome and high concentrations of cardiac troponin I and /or CKMB are considered to be victims of myocardial infarction , whereas the diagnosis of unstable angina will be made if the concentrations of cardiac troponin land CK MB are situated in the reference range.
Several published guidelines agree that a single test for troponin on arrival of the patient in the hospital is insufficient. The collection of atleast 3 blood samples during the early triage period has been recommended.
Apart from its role in the diagnosis of AMI , the determination of cardiac troponin I is useful for assessing the effect of thrombolytic therapy and estimating the extent of necrosis.
For patients with acute coronary syndrom, troponin is a prognostic indicator and allows to stratify the risk of cardiac events and mortality.

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LIVER FUNCTION TESTS

TESTS		RESULTS	UNITS	Biological Ref Interval
S. BILIRUBIN	TOTAL	0.73	mg / dl	0.0 - 1.0
	DIRECT	**0.37**	mg / dl	0.0 - 0.2
	INDIRECT	0.36	mg / dl	0.0 - 0.8
S. ALKALINE PHOSPHATASE		85.2	U / L	40 - 129

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sf-1, narsinhdham complex,
sangam char rasta, harni road,
baroda-22. ph. : 2489092
E-mail : divinehcl@gmail.com

Healthcare Laboratory

Patient's Name	: Kanad Bandyopadhyay	Ref.No.	: HL-13550-21
Age/Sex	: 63 Years /Male	Reg. Date	: 15/11/2021 09:07
Referred by	: C/o. Dr At Doorstep	Collection. Time	: 15/11/2021 8 : 35

4011

URINE ANALYSIS

SPECIMEN : Fasting

PHYSICAL EXAMINATION	**RESULT**	**NORMAL**
Quantity (ml)	10	600 - 2500 / 24 hrs
Colour	Pale yellow	Pale yellow
Appearance	Clear	Clear
Reaction (PH)	6.0	5.0 - 8.5
Sp. Gravity	#####	1.000 - 1.030
CHEMICAL EXAMINATION		
Protein	Absent	0 - 0.1 gm / 24 hrs
Sugar (Glucose)	Absent	Absent
Ketone	Absent	Absent
Bilirubin	Absent	Absent
Urobilinogens	Normal	0 - 04 mg / 24 hrs
Bile Salts	Absent	Absent
Bile Pigments	Absent	Absent
Occult blood	Absent	Absent
MICROSCOPIC EXAMINATION / HPF		
Pus Cells	2 -- 3	0 - 1
Red Blood Cells	Absent	Absent
Epithelial Cells	2 -- 3	2 - 3
Casts (/ lpf)	Not seen	
Crystals	Not seen	

authorized signatory

verify by : mukesh j
reporting Verification Time : 15/11/2021 10:47:00
report print Date/Time :15/11/2021 15:32

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---: end of report :---