TEST PATIENT

TEST PHYSICIAN

NutriPATH

GUa d`Y'HYgh'BUa Y Sex::

DR JOHN DOE 111 CLINIC STF 99H

7@=B=7 GI 6I F6 J=7 " \$\$\$ 111 H9GH ROAD TEST SUBURB

P: 1300 688 522

E: info@nutripath.com.au A: PO Box 442 Ashburton VIC 3142

@AB =8: 00000000 UR#:0000000

DUHY Collected: 00-00-0000

	НА	EMATOLO	GY	
BLOOD - CITRAT	Result	Range	Units	
FIBRINOGEN	3.1	2.0 - 4.5	g/L	
	BIC	OCHEMIST	TRY	
BLOOD - SERUM	Result	Range	Units	
CHOLESTEROL	6.6 *H	0.0 - 5.5	mmol/L	
TRIGLYCERIDES	1.5	0.2 - 1.5	mmol/L	
HDL(Protective)	1.6	> 1.2	mmol/L	
LDL(Atherogenic)	<i>4.4</i> *H	0.5 - 3.5	mmol/L	
LDL/HDL RATIO (Risk Factor)	2.8	0.0 - 3.2		
Lipoprotein (a)	273 *H	0.0 - 75.0	nmol/L	
Apolipoprotein B	1.32 **H	0.50 - 1.30	g/L	
Apolipoprotein A-1	1.70	1.10 - 2.05	g/L	
RATIO (APO B / APO A-1)	0.78	0.35 - 1.15		
HOMOCYSTEINE	11.0	5.0 - 12.0	umol/L	
HIGH SEN CRP	0.90	0.00 - 10.00	mg/L	

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Lipid Profile Comment

CHOLESTEROL COMMENT:

For secondary prevention, total cholesterol Treatment Target is <4.0 mmol/L Triglycerides Treatment Target <2.0 mmol/L HDL Treatment Target Value >1.0 mmol/L

LDL-CHOLESTEROL COMMENT:

As there is an elevated LDL level, we suggest a Liposcreen (LDL Subractions) Test to determine the presence of small, dense (highly atherogenic) LDLs which are a primary cause of Coronary Artery Disease (CAD).

The LDL subtypes are not detectable through conventional Lipid Profiles.

LIPOPROTEIN(a) ELEVATED:

Consists of an LDL bound to Apolipoprotein component. Causes atherothrombogenesis and strongly associated with peripheral and coronary events.

Consider the following possible causes:

Genetic predisposition, Excessive intake of partially hydrogenated oils/fats, low-fibre, low vegetable-based diet, Hypothyroidism, Post-Menopausal elevation, Diabetes, particularly with central obesity, Chronic renal insufficiency, Simvistatin Therapy, Compounded likelihood of CVD if also high LDL and/or total Cholesterol.

Consider the following actions:

Aerobic Exercise, Dietary modification, 1 g TID Niacin OR inositol hexaniacinate (non-flush if availalable), CoQ10, L-lysine, proline, HRT if indicated, Magnesium, Coronary vasodilator therapy - as elevated Lp(a) may impair normal vasodilation mechanisms.

Vitamin C, L-Lysine and Vitamin E are also beneficial.

Increased HDL levels appear to reduce the threat posed by high levels of Lp(a).

Lp(a) COMMENT:

For Lp(a) levels > 0.30 g/L the relative risk of MI is 1.75 compared to patients with Lp(a) below this level. Lp(a) is an acute phase reactant and the level is elevated in acute illness.

APOLIPOPROTEIN B ELEVATED:

Apolipoprotein B levels increase during pregnancy, hypercholesteremia, LDL receptor defect, bile obstruction, hyperlipemia type II, and nephrotic syndrome. Suspect: Elevated LDL, Hyperlipoproteinemia type 2a or 2b, Hyper-beta-lipoproteinemia, Arterial Stenosis (High Apo B can be associated with carotid or coronary stenosis). Consider the following actions: Treat as for elevated Cholesterol and Triglycerides, 1 g TID Niacin OR inositol hexaniacinate (non-flush if availalable), use Psyllium and other water soluble fibres, vegetable-based diet including soy products, Zinc supplementation and Anti-oxidants.

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General Chemistry Comment

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ELEVATED HOMOCYSTEINE LEVEL:

Hyperhomocysteinemia is a risk factor for arterial and venous thrombosis.

Highest 25th percentile of Homocysteine levels showed 3 fold greater MI risk than the lowest 25th percentile.

Homocysteine is elevated in B12, B6 and folate deficiency as well as renal impairment. A fasting specimen is required as homocysteine is affected by diet.

In the Methylation process, Homocysteine levels may be lowered by one of the following;

- 1. Conversion to Methionine to SAMe (via TMG or methylB12)
- 2. Conversion to Cystathionine to Glutathione (via Vit B6)
- 3. Conversion to Tetrhydrofolate to 5MTHF (via VitB2, VitB6)

GLUCOSE (FASTING)

3.5 - 6.05.3

mmol/L

Tests ordered: FIB,FGLU,CVP,IMPEI,CFee