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Name: Sample Report	Present Weight: 90 kg
Age /Gender: 28 years / Female	Height: 168 cm
Co-morbidities: Pre-diabetes, PCOS	BMI: 31.9 kg/m ²
Current Medications: None	Waist: 42 inch

	Optimal	Caution	At Risk	High Risk
Glycemic Control & Insulin Resistance				
Systemic Inflammation				
Lipids & Cardio-metabolic Risk				
Hepatic / Metabolic Liver Status				
Endocrine & Hormonal Health				
Vitamin & Micronutrients				
Diet Quality & Eating Behaviours				
Adiposity Pattern				
Sleep Quality & Stress Levels				
Physical Activity & Fitness				

TOP 3 HEALTH PRIORITIES (HIGHEST RISK)

1. Improve glycemic control with target HbA1c of 5.3%, Fasting Insulin less than 6 μ U/mL HOMA-IR less than 2
2. Improve Lipid profile with target TG<150, HDL>50, LDL<100
3. Nutritional correctional of deficient vitamins and micro-nutrients

DOCTOR REMARKS

- Target weight goal: 65-70 kg in 12 month.
- Dietary and lifestyle intervention as first line of therapy for glycemic control and lipid profile improvement.
- 15 days monitoring of blood glucose levels - Post prandial - 2 hours
- Prescription for nutritional deficiency correction for Vitamin D, Vitamin B12 and Iron

NUTRITIONAL COACH REMARKS

Nutrition: Follow a low carb, balanced diet with target protein intake of 100 gm / day

Movement: Strength training 2-3x / week. Post meal walks.

Sleep/Stress: 8 hours of quality sleep and daily anti-stress protocol

RECOMMENDED PROGRAM TO ACHIEVE YOUR GOALS

The Good Advance Program - 6 months

(6 doctor sessions, 24 nutritional coach sessions, 2x CGM, 2x Blood work and more)

SAMPLE REPORT

TEST	RESULTS	UNIT	BIOLOGICAL REF RANGE	TEST REMARK
Complete Blood Count				
RBC Count <i>Electrical Impedance</i>	4.83	millions/cmm	4.5 - 6.5	
Haemoglobin <i>SLS Hemoglobin Method</i>	L 13.2	g/dL	13.5 - 18	
PCV <i>Pulse Light Detection Method</i>	43.1	%	40 - 54	
Mean Corpuscular Volume <i>Calculated</i>	89.2	fL	76 - 96	
Mean Corpuscular Hemoglobin <i>Calculated</i>	27.3	pg	27 - 32	
Mean Corpuscular Hb Concentration <i>Calculated</i>	30.6	g/dL	30 - 35	
Red Cell Distribution Width (RDW) <i>Calculated</i>	13.3	%	11.5 - 14	
Total Leucocyte Count(TLC) <i>Flowcytometry</i>	7,050	Cells/cmm	4000 - 11000	
Differential Counts				
Neutrophils <i>Flowcytometry</i>	46.4	%	40 - 75	
Lymphocytes <i>Flowcytometry</i>	43.3	%	20 - 45	
Monocytes <i>Flowcytometry</i>	6.5	%	2 - 10	
Eosinophils <i>Flowcytometry</i>	2.8	%	1 - 6	
Basophils <i>Flowcytometry</i>	1.0	%	0 - 1	
Absolute Counts				
Absolute Neutrophil Count <i>Calculated</i>	3270	Cells/cmm	2000-7000	
Absolute Lymphocyte Count <i>Calculated</i>	3050	Cells/cmm	1000-5000	
Absolute Monocyte Count <i>Calculated</i>	460	Cells/cmm	200-1000	
Absolute Eosinophil Count <i>Calculated</i>	200	Cells/cmm	20-500	
Absolute Basophil Count <i>Calculated</i>	70	Cell/cmm	20-100	
Platelet Count <i>Electrical Impedance</i>	2,78,000	Cells/cmm	150000 - 400000	

Note:(LL-VeryLow,L-Low,H-High,HH-VeryHigh,A-Abnormal)

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SAMPLE REPORT

Mean Platelet Volume (MPV) <i>Calculated</i>	9.8	fL	7.2 - 11.7
ESR <i>Automated (Opto-electronic unit)</i>	9	mm after 1hr	0 - 15
BIOCHEMICAL INVESTIGATIONS			
Plasma Glucose - F <i>HEXOKINASE/G-6-PDH</i>	89	mg/dL	Normal : 70 - 99 Impaired Fasting : 100 - 125 Diabetic : =>126
Insulin Fasting <i>CMIA</i>	15.90	μU/mL	2 - 25
Cholesterol <i>Enzymatic</i>	191	mg/dL	<200 - Desirable 200 - 239 - Borderline High > 240 - High "NCEP Guidelines ATP III".
Triglyceride <i>GlycerolPhosphate Oxidase</i>	H198	mg/dL	< 150 - Normal 150 - 199 - Borderline 200 - 499 - High > 500 - Very High "NCEP Guidelines ATP III".
HDL Cholesterol <i>Accelerator Selective Detergent</i>	L 30	mg/dL	< 40 - Low Level 40 - 60 - Average Level > 60 - High Level NCEP Guidelines ATP III.
LDL Cholesterol <i>Calculated</i>	H 121.40	mg/dL	0 - 100
VLDL <i>Calculated</i>	H 39.60	mg/dL	<30
Non-HDL Cholesterol <i>Calculated</i>	H161		< 130 Optimal 130-159 Near Optimal 160-189 Borderline high 190-219-High >or = 220- Very high
LDL/HDL Ratio <i>Calculated</i>	H 4.05		
Chol/HDL <i>Calculated</i>	H 6.37		< 3.5 - Low risk 3.5 - 5.0 - Normal risk > 5.0 - High risk
Amylase <i>Ethylidene Blocked-pNPG7</i>	59.0	U/L	28 - 100 U/L

PI note change in BRI.

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SAMPLE REPORT

BIOCHEMICAL INVESTIGATIONS

Lipase <i>Colorimetric, Quinone Dye</i>	29.00	U/L	< 60 U/L
Urea <i>Urease</i>	20.10	mg/dL	12.84 - 42.8
Uric Acid <i>Uricase</i>	5.70	mg/dL	3.7 - 7.7
Creatinine <i>Kinetic Alkaline Picrate</i>	0.90	mg/dL	0.6 - 1.2
Calcium <i>Arsenazo III</i>	8.70	mg/dL	8.4 - 10.2
Phosphorus Inorganic <i>Phosphomolybdate</i>	4.65	mg/dL	2.3 - 4.7
Vitamin B - 12 Level <i>CMIA</i>	216.0	pg/mL	187 - 883
High Sensitive CRP <i>Immunoturbidimetric</i>	0.05	mg/dL	0 - 0.5 mg/dL For Cardiac risk : < 0.1 Low risk 0.11 - 0.3 Average risk 0.31 - 0.5 High risk For Acute Inflammation/Infection > 1.0
Triiodothyronine (T3) <i>CMIA</i>	93.87	ng/dL	35 - 193
Thyroxine (T4) <i>CMIA</i>	6.34	µg/dL	4.87 - 11.72
TSH <i>CMIA</i>	0.65	µIU/mL	0.35 - 4.94
Cortisol 8 AM <i>CMIA</i>	15.70	µg/dL	2.9 - 17.3
Magnesium <i>Enzymatic</i>	2.24	mg/dL	1.6 - 2.6

Glycated Haemoglobin Estimation

HbA1C <i>HPLC</i>	H 5.80	%	Non Diabetic : Less than 5.7 % Pre Diabetic : 5.7 - 6.4 Diabetic : => 6.5 %
Estimated Avg Glucose (3 Mths) <i>Calculated</i>	119.76	mg/dL	

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SAMPLE REPORT

Interpretation :

HbA1C level reflects the mean glucose concentration over previous 8-12 weeks and provides better indication of long term glycemic control.

Levels of HbA1C may be low as result of shortened RBC life span in case of hemolytic anemia.

Increased HbA1C values may be found in patients with polycythemia or post splenectomy patients.

Patients with Homozygous forms of rare variant Hb(CC,SS,EE,SC) HbA1c can not be quantitated as there is no HbA.

In such circumstances glycemic control can be monitored using plasma glucose levels or serum Fructosamine.

The A1c target should be individualized based on numerous factors, such as age, life expectancy, comorbid conditions, duration of diabetes, risk of hypoglycemia or adverse consequences from hypoglycemia, patient motivation and adherence.

LIVER FUNCTION TEST

Bilirubin Total <i>Diazonium Salt</i>	0.83	mg/dL	0.2 - 1.2
Bilirubin Direct	0.24	mg/dL	0 - 0.5
Bilirubin Indirect <i>DIAZO REACTION</i> <i>Calculated</i>	0.59	mg/dL	0.1 - 1
S.G.P.T. <i>NADH (Without P-5-P)</i>	13.00	U/L	0 - 45
S.G.O.T. <i>NADH (Without P-5-P)</i>	19.00	U/L	11 - 34
Alkaline Phosphatase <i>Para-Nitrophenyl Phosphate</i>	74.00	U/L	40-150
Gamma Glutamyl Transferase <i>L-Gamma-glutamyl-3-carboxy-4-nitroanilide</i>	20.00	U/L	0 - 55
Proteins (Total) <i>Biuret</i>	6.89	g/dL	6.4 - 8.3
Albumin <i>Bromo Cresol Green</i>	4.23	g/dL	3.5-5.0
Globulin	2.66	g/dL	2.0 - 3.5
A/G Ratio	1.6		1.0 - 2.0


VITAMIN D

25OHCholecalciferol (D2+D3) <i>CMIA</i>	L 15.8	ng/mL	Deficient : < 20 ng/mL Insufficient : 20 - 30 ng/mL Sufficient : 30 - 100 ng/mL Toxicity : > 150 ng/mL
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SAMPLE REPORT

25-OH-VitD plays a primary role in the maintenance of calcium homeostasis. It promotes intestinal calcium absorption and, in concert with PTH, skeletal calcium deposition, or less commonly, calcium mobilization. Modest 25-OH-VitD deficiency is common; in institutionalised elderly, its prevalence may be >50%. Although much less common, severe deficiency is not rare either. Reasons for suboptimal 25-OH-VitD levels include lack of sunshine exposure, a particular problem in Northern latitudes during winter; inadequate intake; malabsorption (e.g. due to Celiac disease); depressed hepatic vitamin D 25-hydroxylase activity, secondary to advanced liver disease; and enzyme-inducing drugs, in particular many antiepileptic drugs, including phenytoin, phenobarbital, and carbamazepine, that increase 25-OH-VitD metabolism. Hypervitaminosis D is rare, and is only seen after prolonged exposure to extremely high doses of vitamin D. When it occurs, it can result in severe hypercalcemia and hyperphosphatemia.

INTERPRETATION

Levels <10 ng/mL may be associated with more severe abnormalities and can lead to inadequate mineralization of newly formed osteoid, resulting in rickets in children and osteomalacia in adults. In these individuals, serum calcium levels may be marginally low, and parathyroid hormone (PTH) and serum alkaline phosphatase are usually elevated. Definitive diagnosis rests on the typical radiographic findings or bone biopsy/histomorphometry.

Patients who present with hypercalcemia, hyperphosphatemia, and low PTH may suffer either from ectopic, unregulated conversion of 25-OH-VitD to 1,25 (OH)²-VitD, as can occur in granulomatous diseases, particularly sarcoidosis, or from nutritionally-induced hypervitaminosis D. Serum 1,25 (OH)²-VitD levels will be high in both groups, but only patients with hypervitaminosis D will have serum 25-OH-VitD concentrations of >80 ng/mL, typically >150 ng/mL.

Patients with CKD have an exceptionally high rate of severe vitamin D deficiency that is further exacerbated by the reduced ability to convert 25-OH-VitD into the active form, 1,25 (OH)²-VitD. Emerging evidence also suggests that the progression of CKD & many of the cardiovascular complications may be linked to hypovitaminosis D.

Approximately half of Stage 2 and 3 CKD patients are nutritional vitamin D deficient (25-OH-VitD, less than 30 ng/mL), and this deficiency is more common among stage 4 CKD patients. Additionally, calcitriol (1,25 (OH)²-VitD) levels are also overtly low (less than 22 pg/mL) in CKD patients. Similarly, vast majority of dialysis patients are found to be deficient in nutritional vitamin D and have low calcitriol levels. Recent data suggest an elevated PTH is a poor indicator of deficiencies of nutritional vitamin D and calcitriol in CKD patients. CAUTIONS Long term use of anticonvulsant medications may result in vitamin D deficiency that could lead to bone disease; the anticonvulsants most implicated are phenytoin, phenobarbital, carbamazepine, and valproic acid.

Electrolytes

Sodium <i>ISE, Indirect</i>	143.00	mmol/L	136 - 145
Potassium <i>ISE, Indirect</i>	4.10	mmol/L	3.5 - 5.1
Chloride <i>ISE, Indirect</i>	105.00	mmol/L	98 - 107
Bi Carbonate <i>Enzymatic</i>	27.00	mmol/L	22 - 29

Iron studies

Iron <i>Ferene Method</i>	77.00	µg/dL	65 - 175
Total Iron Binding Capacity <i>Calculated</i>	331.00	µg/dL	251 - 436
Unsaturated Iron Binding Capacity <i>Ferene Method</i>	H 254.00	µg/dL	69 - 240
Transferrin Saturation % <i>Calculated</i>	23.3	%	20 - 50

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SAMPLE REPORT

Interpretation:

Tests	Iron Deficiency anaemia Anaemia of Chronic disease	Anaemia of Chronic disease	Iron overload	Hemoglobinopathy (Especially Trait)
Serum Iron	Decreased	Decreased	Increased	Normal
Serum Total Iron Binding Capacity	Increased	Decreased or Normal	Increased or Normal	Normal
% Transferrin Saturation	Decreased	Decreased or Normal	Increased or Normal	Normal
Serum UIBC	Increased	Decreased or Normal	Decreased	Normal
Serum Ferritin	Decreased	Increased	Increased or Normal	Normal
Serum Soluble Transferrin receptor	Increased	Normal	Decreased	Normal
Serum Hepcidin	Normal	Increased	Normal	Normal

Clinical Pathology

Urine Routine Examination

Physical Examination

Appearance <i>Automated - Light Scattering</i>	Clear	Clear
Colour <i>Automated - Light Scattering</i>	Yellow	Straw to Yellow
Reaction (pH) <i>Indicator</i>	5.5	5-9
Specific gravity <i>Refractive Index</i>	1.019	1.000-1.030
Chemical Examination		
Protein <i>Reflectance Photometry (Protein Error of Principle indicator)</i>	Negative	mg/dL Negative
Glucose <i>Reflectance Photometry - Glucose Oxidase & Peroxidase</i>	Negative	mg/dL Negative

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SAMPLE REPORT

Clinical Pathology

Urine Routine Examination

Bile Pigments <i>Fouchet's test</i>	Negative	mg/dL	Negative
Urobilinogen <i>Coupling of Urobilinogen with stabilized Diazonium Salt</i>	Not increased	mg/dL	0-2 mg/dl
Ketones <i>Reflectance Photometry-Sodium Nitroprusside</i>	Negative	mg/dL	Negative
Nitrites <i>Griess Reaction</i>	Negative	mg/dL	Negative
Blood <i>Peroxidase</i>	Negative		Negative
Leucocyte <i>Granulocyte esterase</i>	Negative	Leu/ μ L	
Microscopic Examination			
Red Blood Cells <i>Phase Contrast Microscopy (Sedimentation based with AIEM)</i>	0.20	/HPF	0-2.3 cells/hpf
Pus Cells <i>Phase Contrast Microscopy (Sedimentation based with AIEM)</i>	0.90	/HPF	0-2.7 cells/hpf
Epithelial Cells <i>Phase Contrast Microscopy (Sedimentation based with AIEM)</i>	0	/HPF	0-1.1 cells/hpf
Hyaline Casts <i>Phase Contrast Microscopy (Sedimentation based with AIEM)</i>	H 0.60	/HPF	0-0.5 p/hpf
Pathological Casts <i>Phase Contrast Microscopy (Sedimentation based with AIEM)</i>	0	/HPF	0-0.3 p/hpf
Crystals			
Calcium oxalate Monohydrate <i>Phase Contrast Microscopy (Sedimentation based with AIEM)</i>	0	/HPF	0-1.4 p/hpf
Calcium oxalate Dihydrate <i>Sedimentation based with AIEM (Automatic Image Evaluation Module)</i>	0.10	/HPF	0-1.4 p/hpf
Triple phosphate <i>Phase Contrast Microscopy (Sedimentation based with AIEM)</i>	0	/HPF	0-1.4 p/hpf
Uric Acid <i>Phase Contrast Microscopy (Sedimentation based with AIEM)</i>	0	/HPF	0-1.4 p/hpf
Bacteria <i>Phase Contrast Microscopy (Sedimentation based with AIEM)</i>	11.70	/HPF	0-29.5 p/hpf

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SAMPLE REPORT**Clinical Pathology**

Urine Routine Examination

Yeast <i>Phase Contrast Microscopy (Sedimentation based with AIEM)</i>	0	/HPF	0-0.7 p/hpf
Amorphous Deposits <i>Sedimentation based with AIEM (Automatic Image Evaluation Module)</i>	0	/HPF	0-29.5 p/hpf

----- End Of Report -----

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