



Metabolic Syndrome Associated with Subclinical Hypothyroidism and Vit D Insufficiency - A Case Report and Discussion

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Author's contribution

The sole author designed, analysed, interpreted and prepared the manuscript.

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Case Report

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ABSTRACT

Metabolic syndrome (MetS) associated with hypothyroidism, was reported in literature. Like-wise association between hypothyroidism and Vit. D deficiency was reported. Hypothyroidism, Vit. D deficiency in DM2 patients was also reported, in the literature. But the occurrence of MetS, hypothyroidism and Vit. D deficiency, together occurring in a case was unreported. The author presented such triple association in a male patient aged 54 years. While Met S itself was a risk factor for CVD (cardiovascular vascular disease) and so were independently, the hypothyroidism and hypovitaminosis D, the triple association would have cumulative risk for CVD. So, it would be profitable to screen all MetS cases with hypothyroidism, for Vit. D deficiency also, in view of the possible cumulative risk. Literature was reviewed as to the relationship of MetS to hypothyroidism and Vit D deficiency, as well as the changing diagnostic criteria of MetS and it's pathogenesis.

Keywords: *Metabolic syndrome; hypothyroidism; vitamin D; insulin resistance; central obesity; dyslipidaemias.*

1. INTRODUCTION

Metabolic syndrome (MetS), a group of five risk factors, (vide infra) increased the likelihood of developing heart disease, diabetes and stroke. The term “metabolic syndrome” was used in 1977 by Herman Haller [1] who was studying the risk factors associated with atherosclerosis. In 1977 and 1978, Gerald B. Phillips developed the concept that MetS was a risk factors for myocardial infarction Since 1998, the criteria for MetS had been changing. The world widely accepted definition of MetS was given below. A brief review of criteria proposed by various agencies were reviewed.

NCEP ATP III definition, ((as modified by AHA (American Heart Association))

Diagnosis of metabolic syndrome was made by the presence of any 3 or more of the following criteria.

- Increased blood pressure (greater than 130/85 mmHg)
- High blood sugar levels. FBS greater than 100 mg/dl.
- Excess fat around the waist(waist measurement above 40 cm, 35 F)
- High triglyceride levels(above 100 mg / dl.)
- Low levels HDL(less than 35 mg/dl)

Changing criteria and concepts since 1998 of mets

1998 WHO criteria of MetS: 1998, WHO laid down demonstrating ‘insulin resistance (IR) central to the diagnosis of MetS, in addition to any of the two criteria listed above. IR was deemed if FBS was raised above normal (100 mg/ dl) or IGT was present on hours OGT test (greater than 140 mg/dl) or Hyperinsulinemic euglycaemic clamp studies or elevated homeostatic model assessment of insulin resistance (HOMA-IR) value, which was proportional to the product of the fasting insulin and fasting glucose level. WHO definition used waist-to-hip ratio or body-mass index for assessing obesity and micro albumin ria as an additional diagnostic criteria, both of which were later removed by later definitions.

1999 The European Group for the Study of Insulin Resistance (EGIR) criteria: In 1999 it proposed a modification to the WHO definition (Balkau and Charles, 1999) Ref no:?. Like the

WHO, insulin resistance was mandatory in addition to the other criteria laid by WHO. To site,obesity criteria was simplified by suggesting of waist circumference. insulin resistance is defined by a fasting plasma insulin value that was greater than the 75th percentile.

2005 The International Diabetes Foundation (IDF), published new criteria for metabolic syndrome (Zimmet et al.) [2] Although it included the same general criteria as the other definitions, it requires that obesity, but not necessarily insulin resistance, uses the MetS .

2009 the Harmonising criteria : IDF, National Heart, Lung, and Blood Institute, American Heart Association, World Heart Federation, International Atherosclerosis Society and International Association for the Study of Obesity [3] jointly adopted the revised NCEP criteria, where abdominal obesity was not a necessary component.

2010, WHO Expert Consultation [4] warned that MetS was a concept that focuses attention on complex multifactorial health problems but had limited practical utility as a management tool.

2. CASE REPORT

A 59 year old male patient was a known case of hypertension and was on amlodipine + telmisartan 40 mg combination tablet in the morning and Tab bisoprolol 25 mg at night. With medication his BP was fluctuating with maximum around 140/90 mmHg, more at night than in the morning. He was diabetic and was on Diamicon MR (40 mg) + Metformin 1000t mg combination tablet at breakfast and Metformin 1000 mg at dinner. He is a case of hypothyroidism his TSH was 7.16 µg/dl with with 25 mg of tab thyronorm. He was a patient of dyslipidaemia with high TG and low HDL. He was on statin 20 mg OD. He was under regular care of a cardiologist whose treatment stated above was being followed. Thyroid antibody test was done to r/o autoimmune Thyroid its (Hashimotos disease type) and was found negative. His serum D3 level was low. All the reports were attached. He was a regular tennis player and a busy executive. He underwent a prophylactic stent operation, when he felt uneasy while playing tennis in 2014 and the angiogram showed a critical block in one of the branches of left coronary artery. He was free of any complaints except for the flocculating blood

sugar levels and B.P. He was in for titration of his antidiabetic, antihypertensive and thyroid drugs together with contemplated supplementation of a fibrate and Vit. D, for the raised TGs and low serum D3 levels, respectively.

3. DISCUSSION

The interconnected pathophysiology of the components of MetS: IR was considered central to the pathogenesis of MetS while the vascular endothelial dysfunction was the final common pathway. Central obesity was considered as the initiator by some, while both were considered to be equally important by the others. As seen above, the 2010 WHO declaration that abdominal obesity as not an important factor, leaves IR as the central point of MetS. The foregoing account makes this role of IR clear in the genesis of MetS

IR and Hyperglycaemia/DM2: It was said "Insulin resistance was a powerful predictor of T2D and hyperinsulinemia was a surrogate marker for insulin resistance". In non-diabetic patients conferred 5 fold increased risk of developing diabetes.

IR caused hyperglycaemia by

- 1) Poor uptake of glucose by peripheral tissues (liver, skeletal muscle and adipose tissue).
- 2) Reduced glycogenesis.
- 3) Increased glycogenolysis
- 4) Increased gluconeogenesis
- 5) Increased lipolysis resulting in increased circulating FFA
- 6) Decreased adiponectin
- 7) Increased leptin resistance

IR and Atherogenic Dyslipidaemias:

IR caused -

1. Increased synthesis of TGs. by increased lipolysis, increased FFA levels which are substrates for T G synthesis.
2. Increased VLDL synthesis: In the liver by increased production and decreased clearance.
 - a) Increased FFAs stabilize the production of apoB, the major lipoprotein of very-low-density lipoprotein (VLDL) particles, resulting in more production
 - b) Insulin normally degrades apo B through PI3K-dependent pathways so IR directly increases VLDL production.

c) Insulin regulated the activity of lipoprotein lipase, the major mediator of VLDL clearance.

3. Increased LDL
4. Decreased HDL

IR and Visceral Adiposity:

IR caused visceral adiposity and visceral adiposity can cause IT by

- 1) Pro inflammatory cytokines like TNF Alfa and IL-6 etc.
- 2) Decreased production of adiponectin.
- 3) Activation of Renin-Angiotensin mechanism. This could also cause hypertension, of the criteria for diagnosing MS.

The final pathway:

Vascular Dysfunction: Endothelium was affected, by oxidative stress, hyperglycaemia, advanced glycation products, FFAs, inflammatory cytokines or adipokines. The result was endothelial dysfunction, which results in the reduced bioavailability of NO in the vasculature. Ip3k pathway was inhibited, whereas the MAP kinase pathway was not. This lead to a change in the balance between these two parallel pathways. Inhibition of the PI3K-Akt pathway lead to a reduction in endothelial nitric oxide (NO) production, resulting in endothelial dysfunction, and a reduction in GLUT4 translocation, leading to decreased skeletal muscle and fat glucose uptake. The MAP kinase pathway was unaffected in MS, so there is continued ET-1 production, expression of vascular cell adhesion molecules and mitogenic stimulus to vascular smooth muscle cells. In these ways, insulin resistance leads to vascular abnormalities that predispose to atherosclerosis.

- 1) MetS vs Hypothyroidism
- 2) Study in India by Shantha, et al. [1] 5. found subclinical hypothyroidism in 21.9% and overt hypothyroidism in 7.4% metabolic syndrome patients Similarly, a study by Meher, et al. [5] showed a high prevalence of subclinical hypothyroidism (22%) and overt hypothyroidism (4%) in the metabolic syndrome patients.
- 3) The effects due to metabolic syndrome and hypothyroidism might be compounded to increase risk for CVD.
- 4) A study by Gyawali, et al. in Kavre district of central Nepal reported thyroid

dysfunction in 31.84% of metabolic syndrome patients, the most common dysfunction was subclinical hypothyroidism (29.32%) followed by overt hypothyroidism (1.67%) and subclinical hyperthyroidism (0.83%) [6].

- 5) Positive association was also reported, between a higher TSH level within the euthyroid reference range and the prevalence of the metabolic syndrome [7].
- 6) A study in Korea indicated that higher levels of TSH may predict the metabolic syndrome in the study subjects, suggesting that the influence of thyroid function on metabolic abnormality extended into subjects without metabolic syndrome [8].
- 7) In the present study, been observed that sub clinically hypothyroid patients have higher systolic BP, diastolic BP, fasting blood glucose and triglycerides compared to euthyroid patients [9].
- 8) There was positive relation between TSH and LDL cholesterol, whereas negative relation between TSH and HDL cholesterol [10].
- 9) There were contrasting reports about the association between various metabolic syndrome parameters and thyroid function. In a study in Nigeria, metabolic syndrome was significantly associated with higher free T4 levels. [11] In However, in a study in Turkey, TSH was not related with any metabolic syndrome parameters. [12].
- 10) Thyroid function is associated with certain components of metabolic syndrome (waist circumference and HDL cholesterol).

Hypothyroidism and CVS risk:

- 11) Thyroid function significantly affected lipoprotein metabolism as well as some cardiovascular disease (CVD) risk factors, thus influencing overall CDV risk [13].
- 12) Hypothyroid patients had increased lipoprotein (a) Lp(a) levels, which are associated with increased CVD risk [14].

Hypothyroidism and Dyslipidaemias: Thyroid dysfunction could have an important effect on lipid profile. Screening for the later was cost effective [15].

A study in India, subclinical hypothyroidism was significantly associated with metabolic syndrome and a linear association was observed between

TSH levels and total cholesterol, triglycerides, LDL, and HDL cholesterol levels across the metabolic syndrome group [16].

LDL and T3;

Diiodothyronine (T₃) upregulated DT receptors by controlling the LDL receptor gene activation. This T₃-mediated gene activation I was done by the direct binding of T₃ to specific thyroid hormone responsive elements (TREs) [17]. Furthermore, T₃ controls the sterol regulatory element-binding protein-2 (SREBP-2), which in turn regulated the LDL receptor's gene expression [18]. T₃ has also been associated with protecting LDL from oxidation [19].

13) Thyroid and HDL

Thyroid hormones could influence HDL metabolism by:

- 1) Increased cholesteryl ester transfer protein (CETP) activity, which exchanges cholesteryl esters from HDL₂ to the very low density lipoproteins (VLDL) and TGs to the opposite direction [20].
- 2) Stimulation of the lipoprotein lipase (LPL), by the thyroid hormone, which catabolizes the TG-rich lipoproteins and the hepatic lipase (HL), which hydrolyses HDL₂ to HDL₃ and contributed to the conversion of intermediate-density lipoproteins (IDL) to LDL and in turn LDL to small dense LDL (sdLDL) [21].
- 3) Up-regulation by T₃ of apolipoprotein AV (ApoAV), which plays a major role in TG regulation [22]. Indeed, increased levels of ApoAV have been associated with decreased levels of TGs. [23] Proposed mechanisms for this effect included the decrease of hepatic VLDL-TG production and the increase of plasma LPL levels and activity, resulting in increase of lipoprotein remnant generation due to enhanced LPL-mediated lipolysis of VLDL-TG.
- 4) A greater clearance of lipoprotein core remnants, caused by increased hepatic uptake due to an enhanced affinity for the LDL receptor, has also been ascribed to Apo AV

DM2 vs Hypothyroidism

- 1) There was increased risk of 1.4 times in progression of prediabetics to diabetics.
- 2) Hypothyroidism caused hyperglycaemia.

- 3) HbA1C was increased (Anantarapu S, et al. [24].

Vit D and DM2: Many studies demonstrated an inverse relationship between serum 25(OH) D and diabetes, metabolic syndrome, insulin resistance and beta cell function [25].

Vit D and Hypothyroidism: Patients with Hashimoto's thyroiditis, an autoimmune thyroid disease had lower vitamin D levels. (5) studies have reported the prevalence of vitamin D deficiency in Hashimoto's disease (92%) was significantly higher than in healthy controls (63%) [26]. Furthermore, Mackawy, et al. [27] concluded that the patients with hypothyroidism suffered from hypovitaminosis D and there was a positive significant correlation between serum level of vitamin D with thyroid hormones and a negative significant correlation with TSH levels and suggested that the deficiency of serum levels of vitamin D was significantly associated with the degree and severity of hypothyroidism. There were two explanations for this association. First, the low levels of vitamin D may be due to poor absorption of vitamin D from the intestine. Second, the body might not activate vitamin D properly.

MetS and Vit D: Many researchers point to vitamin D₃ deficiency as a factor in the pathogenesis of hypertension (vitamin D₃ inhibited renin and endothelin synthesis and the proliferation of smooth muscle cells), MetS, and diabetes (development of insulin resistance). It had hence been suggested that vitamin D₃ deficiency raised the risk of cardiovascular disease [28].

Vit D and visceral adiposity: Vit.D deficiency was closely related to visceral obesity [29].

Vit D deficiency vs other diseases: Vit D deficiency was associated with various diseases such as cardiovascular disease, cancer, infection and adiposity as well as osteoporosis. meta-analysis of 28 studies demonstrated that higher serum 25(OH) D levels were associated with a 55% reduction in diabetes, a 51% decreased risk of the metabolic syndrome and a 33% lower risk of cardiovascular disease (CVD) [30].

Vit D deficiency, hypothyroidism and DM2 association was reported by a study conducted by Abdulbari Bener, et al. [31].

Vitamin D and CVD risk

- 1) Cross-sectional associations between vitamin D status and CVD. Risks provided by the National Health and Nutritional Examination Surveys (NHANES) (1988–1994, 2000–2004) [32] conducted in the United States. NHANES 1988–1994: NHANES 2000–2004: survey revealed a) vitamin D deficiency was associated with increased prevalence of self-reported coronary heart disease, heart failure, and peripheral vascular disease. b) Prevalence of peripheral arterial disease is also increased comparing lowest quartile of 25 (OH) D to highest quartile of 25 (OH) D.
- 2) Hensrud D, Endrick I, et al. [33] reported that individuals with vitamin D deficiency (25(OH)D <20 ng/mL) had higher prevalence of self-reported angina, myocardial infarction, and heart failure compared with individuals with higher levels of vitamin D.
- 3) Judd, et al. [34] determined, in non-hypertensive individuals from NHANES 1988–1994, that optimal vitamin D status (>32 ng/mL) provided a 20% reduction in the rate of blood pressure rise with age.

Mahmoud Barbarawi,; Babikir Kheiri, PGDiP Yazan Zayed, et al. [35] "meta-analysis of randomized clinical trials that included more than 83 000 participants, vitamin D supplementation was not associated with reduced risks of major adverse cardiovascular events, myocardial infarction"

4. CONCLUSION

A case of MetS associated with hypothyroidism and Vit D deficiency in a male patient is reported. Reporting of such triple association is considered a rarity. The cumulative CVD risk due the triple association is stressed. Screening for MetS should also include hypothyroidism and Vit D is stressed

CONSENT

As per international standard or university standard, patient's consent has been collected and preserved by the authors.

ETHICAL APPROVAL

As per international standard, ethical approval has been collected and preserved by the author.

COMPETING INTERESTS

Author has declared that no competing interests exist.

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APPENDIX

Patients Reports

1) Fasting Plasma Sugar (FBS)

Patient Name: MR. XXX
Age / Sex: 59 years / Male
Referred by: DR YYY
Sample Collection Time: Aug 30, 2019, 08:23 a.m.
Method: Hexokinase
136 mg / dL (Range 70 - 110)

2) Glycosylated Haemoglobin (GHB/HBA1C)

Name : Mr XXX
Age/Gender: 59 Y/M Collected : 16/Aug/2019 11:35AM
Ref Doctor: YYY
Glycosylated Haemoglobin HbA1c 8.5%
Normal: <5.7
Pre-Diabetes: 5.7-6.4
Diabetes: 6.5

3) Lipid Profile,

SERUM Total Cholesterol
112 mg/dL
Desirable: < 200
Borderline High: 200 - 239
High: > 240
CHOD-POD
HDL Cholesterol 29.22 mg/dL Low: < 40
High: > 60
Direct Measurement
with Dextran
Sulphate
Total Triglycerides 309 mg/dL Desirable Level: 150
Borderline: 150-199
High: 200-499
Very High: 500
Enzymatic-GPO POD
VLDL Cholesterol 61.8 mg/dL ≤ 30 CALCULATED
LDL Cholesterol 20.98 mg/dL <100: Optimal CALCULATED
Non - HDL Cholesterol 82.78 mg/dL <130
Chol / HDL Ratio 3.83 Low Risk : 3.3-4.4
CALCULATED
HDL/LDL Cholesterol Ratio 1.39
LDL/HDL Ratio 0.7

4) Thyroid Profile Report

Name: Mr XXX
Age/Gender: 59 Y/M Collected: 16/Aug/2019 11:35AM
Ref Doctor: YYY
DEPARTMENT OF BIOCHEMISTRY-ROUTINE
ZOYLO ADVANCED HEALTH CHECKUP
Patient Name: MR. XXX

Age / Sex: 59 years / Male

Referred by: DR YYY

Sample Collection Time: Aug 30, 2019, 08:23 a.m.

Serum T3

Method: ECLIA

1.34 ng/mL 0.80-2.0

T4

Method: ECLIA

7.16 µg/dl 5.1-14.1

TSH

Method: ECLIA

7.11 µIU/ml 0.3-5.0

END OF REPORT

5) Ant thyroid Antibody Report

Department of Biochemistry-routine

Zoylo Advanced Health Checkup

Anti-TG 27.21 IU/mL <120 IU/ml-Normal Range

120-180 IU/ml- Borderline

>180 IU/ml-Positive

Anti- TPO 13.61 IU/mL <40 IU/ml-Normal Range

40-60 IU/ml-Borderline >60 IU/ml-Positive

6) Vitamin D Report

Zoylo Digihealth Pvt Ltd. Department of Biochemistry-special

Zoylo Advanced Health Checkup

Name: Mr. XXX

Age/Gender: 59 Y M

Collected: 16/Aug/2019

Ref Doctor: YYY

TEST: 25 (OH) VITAMIN-D,

SERUM .

RESULT:

7) 25 (OH) Vitamin-D 22.20 ng/mL

Deficiency: < 10.0

Sufficiency: 30- 100

Insufficiency: 10- 30

Toxicity > 100

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