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CLINICAL CASE REPORT OF METABOLIC SYNDROME

General Medicine

(Dr) Mohammed Muzammil* Medical Student (MBBS), Shadan Institute of Medical Sciences. *Corresponding Author

Dr. Mohammed Riyaz Esani Diabetes and Multispeciality Centre, Consultant Physician.

Dr. Mohammed Abdul Azeez Resident, Esani Diabetes and Multispeciality Centre.

ABSTRACT

A 51 years old female, presented with discomfort in chest since one-week of duration. She was having on and off history of generalized weakness, fatigability, increased frequency of urination. She had no past history of hypertension or diabetes, she attained menopause 1 year back. Her lifestyle is sedentary. Physical examination: obese woman. Height 161 cm, weight 84 kg, BMI of 32.4 kg/m2 (Class I obesity). Pulse rate: 98/min; regular, normal volume, no radio-radial, nor radio-femoral delay. Blood pressure: 160/110 mmHg in right and left upper limb. She was having black velvety hyperpigmentation of the skin especially on the neck possibly acanthosis nigricans, apart from this patch, rest of the systemic examination is normal. Her random blood sugar was 210 mg/dL as measured by Glucometer. Patient was diagnosed as metabolic syndrome. Patient was counselled and explained she is having diabetes mellitus, hypertension, and she has all the signs of metabolic syndrome. Patient was started on life style modification, diet modification, Pharmacotherapy, lipidemic drugs.

KEYWORDS

Metabolic Syndrome, Insulin Resistance, Diabetes Mellitus, Hyperlipidemia, Hypertension, Obesity

Clinical Case:

A 51 years old female, presented with discomfort in chest since one-week of duration. She was having on and off history of generalized weakness, fatigability, increased frequency of urination.

She had no past history of hypertension or diabetes, she attained menopause 1 year back. Her lifestyle is sedentary. Physical examination: obese woman. Height 161 cm, weight 84 kg, BMI of 32.4 kg/m2 (Class I obesity). waist circumference: 92cm, hip Circumference: 96cms, waist hip ratio: 0.96. Pulse rate: 98/min; regular, normal volume, no radio-radial, nor radio-femoral delay. Blood pressure: 160/110 mmHg in right and left upper limb.

She was having black velvety hyperpigmentation of the skin especially on the neck possibly acanthosis nigricans, apart from this patch, rest of the systemic examination is normal. Her random blood sugar was 210 mg/dL as measured by Glucometer.

Further tests were ordered to assess her cardiovascular (CV) risks, premature menopause as well as possible cardiovascular target organ damage.

INVESTIGATIONS

- Fasting blood sugar: 144 mg/dL, Post prandial blood sugar: 260, HbA1C was 8.32%.
- Lipid profile: Triglyceride:241, TC: 320, LDLC: 235, HDL-C:38
- Liver function test showed mildly raised AST (54) and ALT (38),
- Serum Insulin 25.6 (Range: 3.0 - 25.0 mU/L)
- Renal function test normal,
- Thyroid function tests normal.
- Serum cortisol: normal
- Serum oestradiol: normal
- DHEAS, testosterone, progesterone: normal.
- FSH 57.2 mU/L, LH 27.8 mU/L(menopausal range)
- Urine picture: protein present, glucose positive, no leukocyte, macroalbuminuria,
- Electrocardiogram: mild LVH with stain pattern, echocardiography: normal
- Chest radiograph normal
- Pap smear results normal
- U/s abdomen and pelvis: Mild Hepatomegaly (Grade 1), No lesion in pancreas and rest was within normal limits.

She had all the features of metabolic syndrome as per various guidelines criteria.

She was initiated on following medication, as per standard care of guidelines

- Dietary modification, Life style modification was emphasized,
- Metformin 500 mg morning and evening one tablet a day,
- Atorvastatin 10 mg at night, for dyslipidaemia
- Telmisartan 40 mg once a day for hypertension

Over next 10 days later the patient reviewed her fasting blood sugar 131 mg/dl, Plbs: 226 mg/ dL blood pressure was normal (124mm of Hg systolic and Diastolic 72mm of hg). Her medication dose of metformin was increased to 1000 mg morning and evening. Patient was reviewed after 8 weeks she had reduced 3.8 kg of weight with the diet modification with pharmacotherapy. She was on low carbohydrate diet, low fat and high fiber. The Fasting blood glucose: 105mg/dl, Post prandial blood glucose: 162mg/dl, pulse 90/ min, BP: 120/70 mmHg. On review the investigation results are as follows; HbA1C 6.8%, TC 194mg/dl, LDL-C 124 mg/dl, HDL-C 36 mg/dl, Tg 274 mg/dl and the liver enzymes normalised.

DISCUSSION:

There are many different factors that contribute to the development of Metabolic syndrome. However, as initially proposed by Reaven, insulin resistance is thought to play a central role in connecting the different components of Metabolic syndrome and adding to the syndrome's development. Elevated free fatty acids (FFA) and abnormal adipokine profiles can both cause and result in insulin resistance and can manifest as Metabolic syndrome.[1-2]

Metabolic syndrome is burning topic presently, majority of adults are affected by metabolic syndrome, it constitutes nearly by 25% of cases, and increasing trend is seen in patients with central obesity. Various criteria have been given by IDF, WHO,[3-4] other diabetic societies all over the world.

Definitions Of Metabolic Syndrome:

	WHO (1998)	IDF (2004)
Criteria	Type 2 DM or Impaired Glucose Tolerance (IGT) and any 2 of the following factors	Central obesity plus any 2 of the other following factors
Hypertension	BP > 140/90 mmHg and/or currently on antihypertensive therapy	Systolic BP ≥130 or diastolic BP ≥85 mmHg, or currently on antihypertensive therapy

Dyslipidemia	Tg > 150 mg/dL and/or HDL-C < 35 mg/dL in men and <(40 mg/dL) in women	Tg > 1.7 mmol/L, *or* treatment for this lipid abnormality or HDL-C < 1.03 mmol/L (40 mg/dL) in men and < 1.29 mmol/L (50 mg/dL) in women or treatment for this lipid abnormality.
Obesity	BMI > 30 kg/m2 and/or waist/hip ratio: > 0.9 in men >0.85 in women	Central obesity (waist circumference ≥94 cm for men and ≥80 cm for Europeans women.
Glucose	Type 2 DM or IGT	Fasting plasma glucose ≥ 100 mg/dL, or previously diagnosed type 2 DM

Insulin Resistance:

The most common underlying cause for the metabolic syndrome is insulin resistance. In patient with insulin resistance, the insulin levels are elevated and it is well known that it leads to impairment in glycemic levels and worsening the diabetes is seen.[4]

Insulin resistance is most simply defined by its end organ effects; a decreased ability of insulin to suppress lipolysis and hepatic glucose production, as well as facilitate glucose uptake from peripheral tissues. There are numerous factors thought to mediate insulin resistance and its adverse effects in Metabolic syndrome. Despite its widespread appreciation in metabolic disease, insulin resistance is not fully understood and remains a core area of investigation and research.

Randle et al. first demonstrated that an elevation in FFA in the diaphragm and heart was associated with an increase in oxidation of fatty acid and impaired glucose utilization via the Randle cycle effect, increased FFAs and fatty acid oxidation lead to increased intracellular glucose content and decreased glucose uptake. As FFA levels increase, the capacity of the adipose tissue to take up and store FFAs can be exceeded. When this occurs, FFAs accumulate in tissues with limited ability for lipid storage, such as the liver and skeletal muscle. This phenomenon is called as ectopic deposition of fat and it is being strongly associated with insulin resistance. in obesity and Metabolic syndrome there is an Increased FFAs which can lead to insulin resistance via several different mechanism.[4-9]

Adipose tissue is also considered as an active endocrine organ that releases adipokines, which are bioactive mediators that have an effect on metabolism. It has been demonstrated by many studies that individuals with Metabolic syndrome have an abnormal adipokine level which is known to produce it effect on insulin sensitivity[10]

The metabolic syndrome is a product of the complex intertwining of inflammation and insulin resistance; with its relationship to both of these, the gut microbiota has been demonstrated to have a strong influence on metabolic diseases.[11-15]

Weight Loss:

Patient is advice to make a goal to reduce the weight by 5-10% in duration of 1 year. This can help in achieving in proper diet, physical activity, and exercise. Physical exercise can cause the loss of body fat and mobilization of visceral and abdominal adipose tissue which increases the sensitivity of insulin and also decrease in the Cholesterol levels which is atherogenic. The recommended diet should include < 200 mg/day of cholesterol, < 7% saturated fat, with total fat comprising 25-35% of calories, low simple sugars and increased fruits, vegetables and whole grains. Smoking cessation should be instituted in all patients with metabolic syndrome. Additionally, low dose aspirin is recommended in cases of moderate to high cardiovascular risk where no contraindication to aspirin therapy exists.

Currently available FDA-approved pharmacotherapy for obesity includes, orlistat, locaserin, buproprion/naltrexone and liraglutide 3.0 mg. Further, individuals with morbid obesity (BMI> 40 kg/m2 or >35 kg/m2 with comorbidities) may be candidates for bariatric surgery (126). Bariatric surgery has been demonstrated to be an effective treatment of obesity with improvements in weight, T2DM, hypertension, hyperlipidemia, and sleep apnea.

Hypertension:

Recommended goal of blood pressure in diabetes mellitus patient is less than 130/80 mmHg. Drug of choice is ARB or ACE inhibitors; it is proved to prevent microvascular, and macrovascular complications as well as the progression of albuminuria.

Drug therapy for dyslipidemia is generally approached with the use of HMG Co-A reductase inhibitors (statins). The primary objective in CVD risk reduction is to lower LDL-C values and the drug of choice for these purposes is statins, which have been shown not only to lower LDL-C, but also to modestly raise HDL-C and lower triglycerides. The second targets in lipid improvement to reduce CVD risk are HDL-C and triglycerides. Niacin is effective at raising HDL-C as well as lowering triglycerides and LDL-C. Fibrates are effective at lowering triglycerides but do not have the beneficial effects on HDL-C and LDL-C. Omega-3 polyunsaturated fatty acids (n-3 PUFA) in fish oil can also be used to lower triglycerides with recent data from the REDUCE-IT trial.

Glycaemic Control:

An insulin-sensitizing agent, such as metformin, is typically used at the start of hyperglycemia treatment in patients with metabolic syndrome. Some literature suggests that metformin may help to reverse the pathophysiologic changes of metabolic syndrome. This includes when it is used in combination with lifestyle changes or with peroxisome proliferator-activated receptor agonists, such as fibrates and thiazolidinediones (eg, pioglitazone), each of which may produce favourable metabolic alterations as single agents in patients with metabolic syndrome The glitazones (pioglitazone) specifically reduce hyperglycemia at the small risks of weight gain and increased heart failure. However, they decrease FFA levels, lessen insulin resistance, reduce triglycerides, and increase HDL.

CONCLUSION:

The metabolic syndrome is considered as a collection of multiple related risk factors which can predispose an individual to the development of T2DM and CVD. It affects a large number of people worldwide and its prevalence is increasing over the years. The diagnostic criteria for metabolic syndrome are given to provide a purpose of consistency in clinical care management. Insulin resistance has both metabolic and mitogenic effects which can result in the development of hyperglycemia and T2DM, hypertension, dyslipidemia, NAFLD, PCOS, OSA, sexual dysfunction, and rarely cancer. In patients with metabolic syndrome, lifestyle modification is imperative in decreasing the CVD risk and treating many of the associated conditions. Treatment of the individual conditions is often also required.

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