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Case Report

A CASE OF METABOLIC SYNDROME

Rabia Khatoon *MMed (FamMed, UM)*, Khoo Ee Ming *MRCGP, FAMM*
Department of Primary Care Medicine, University of Malaya

Address for correspondence: Dr Rabia Khatoon, Department of Primary Care Medicine, University of Malaya, 50603 Kuala Lumpur. Tel: 03-79492602, HP: 012-6985026, Fax: 03-79577941, Email: akim@streamyx.com

ABSTRACT

This case report illustrates a 40-year-old woman who presented with chest discomfort that was subsequently diagnosed to have metabolic syndrome. Metabolic syndrome is a common condition associated with increased cardiovascular morbidity and mortality. As primary care providers, we should be detect this condition early, intervene and prevent appropriately before complications occur.
Keywords: metabolic syndrome, cardiovascular disease, diagnosis, management
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INTRODUCTION

Metabolic syndrome or Syndrome X is a cluster of risk factors associated with morbidity and mortality of cardiovascular diseases (CVD). Albeit common, it has not received the attention it has warranted, and has often been overlooked. As primary care providers, we should actively look out for these patients so that early detection, intervention and prevention can be undertaken. Described below is a Chinese lady with metabolic syndrome.

CASE SUMMARY

Madam LSC, a 40-year-old housewife, presented with chest discomfort of one-week duration to the primary care clinic of a tertiary hospital. She had no past history of diabetes mellitus (DM) or hypertension (HT). She had premature menopause at the age of 37 years. Her mother has diabetes mellitus and hypertension. She was an ex-smoker and had lived a very sedentary lifestyle. Physical examination showed an obese woman with a body weight of 82kg, height 165cm, with a BMI of 32 kg/m^2. Her pulse rate was 78/min; blood pressure (BP) was 160/110 mmHg on both sides of the arms. She had acanthosis nigricans on her neck but the rest of the general and systemic examinations were normal. Her random blood sugar was 8.9mmol/l as measured by glucometer.

Further tests were ordered to assess her cardiovascular (CV) risks, premature menopause as well as possible cardiovascular target organ damage. She was treated with non-steroidal anti-inflammatory drugs for her costochondritis.

On review a week later, her results were: Fasting blood sugar (FBS) 8.3 mmol/L, triglyceride (Tg) 2.4mmol/L, total cholesterol (TC) 8.3 mmol/L, LDL-cholesterol (LDL-C) 6.07 mmol/L, HDL-cholesterol (HDL-C) 0.98 mmol/L, serum creatinine and electrolytes were normal, liver function test (LFT) showed mildly raised AST and ALT, HbA1C was 8.46%. Her thyroid function tests, serum cortisol, oestradiol, progesterone, testosterone and DHEAS were normal. Her FSH was 57.1mU/L and LH was 27.9 mU/L, and these were in the menopausal range. She had macroalbuminuria. Her electrocardiogram, chest radiograph and Pap smear results were normal. Her blood pressure remained persistently high with a reading of 160/106 mmHg. She had all the features of metabolic syndrome with premature menopause.

She was started on metformin 850mg twice a day, simvastatin 10 mg at night, amlodipine 5 mg daily and enalapril 2.5 mg was added later. She was referred to a gynaecologist for further evaluation of premature menopause, and dietitian for dietary advice. Non-pharmacological measures (weight reduction, exercise, low salt intake, behavioral modification and diet), adherence to therapy, regular follow-up and potential side effects of medications were addressed.

Over the next eight months she was regularly followed up by the author and her weight had reduced to 74 kg. She was taking high fibre, low fat and low carbohydrate diet, and had started jogging. Her post lunch random blood sugar (RBS) was 6.0mmol/l and BP was 120/70 mmHg. Repeated fasting biochemical results showed FBS 6.5 mmol/L, HbA1C 6.69%, TC 6.1 mmol/L, LDL-C 3.2 mmol/L, HDL-C 1.02 mmol/L, Tg 3.1 mmol/L and the liver enzymes remained the same. She was congratulated for being successful at reducing her weight and was encouraged to

continue her measures. Her simvastatin dosage was increased to 20 mg and she was referred to an ophthalmologist for evaluation of early diabetic retinopathy. She was further advised about foot care and proper footwear.

Table 1. WHO and IDF definitions of metabolic syndrome

	WHO (1998)	IDF (2004)
Criteria for diagnosis	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 > 1.7 mmol/L (150 mg/dL) and/or HDL-C < 0.9 mmol/L (35 mg/dL) in men and < 1.0 mmol/L (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/m^2 and/or waist/hip ratio > 0.9 in men and >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 ≥5.6 mmol/L (100 mg/dL), or previously diagnosed type 2 DM (If FBS> 5.6 mmol/L, an oral glucose tolerance test [OGTT] is strongly recommended, but is not necessary to define presence of the syndrome.)
Other	Microalbuminuria (overnight urinary albumin excretion rate > 20 mcg/min or 30 mg/g Cr)	

DISCUSSION

Metabolic syndrome affects approximately 25% of adults and is particularly increasing due to central obesity.[1] A few criteria were used to define metabolic syndrome but we describe the more commonly adopted definition by the World Health Organization (WHO) and the much discussed recent criteria defined by the International Federation of Diabetes Mellitus (IDF criteria)[2] [see table 1].

Essentially, metabolic syndrome revolves around the management of a cluster of chronic diseases such as diabetes mellitus, hypertension, dyslipidaemia, and obesity. There is no single treatment for patients with metabolic syndrome but rather early detection and management of these chronic diseases and modification of the risk factors. All these chronic conditions are 'bread and butter' problems encountered in general practice and yet it is difficult to achieve optimal control. The most important modifiable risk factors include smoking and patient should be advised to stop it.[3]

Insulin resistance

There is general agreement that insulin resistance is the underlying cause of metabolic syndrome. Insulin resistance and the resulting hyperinsulinaemia have been implicated in the development of glucose intolerance and the progression of type 2 diabetes mellitus, hypertension, polycystic ovarian syndrome, hypercoagulability and vascular inflammation as well as eventual development of CVD.[4] Recently IDF has proposed central obesity as an important component of metabolic syndrome because it is highly correlated with other components of metabolic syndrome and is easily measured using waist circumference.[5]

Weight loss

One of the important aspects of management for metabolic syndrome is weight reduction. A realistic goal for weight reduction is to reduce body weight by 7-10% over a period of 6-12 months.[3] This is achieved by encouraging patient to focus on exercise and improve their personal level of activity. Great benefit occurs when sedentary persons incorporate moderate intensity exercises into their lifestyle.[1] Regular physical training and endurance exercise training

can induce body fat loss and a mobilization of abdominal and visceral adipose tissue can increase insulin sensitivity and improve the atherogenic lipoprotein profile.[6] However, the goals set must be realistic and achievable, and should be adjusted according to the patient's level of acceptance and compliance.[7] DASH (dietary advice to stop systolic hypertension) diet should be emphasized and this includes fruits, vegetables, low fat dairy products, whole grains, fish, polyunsaturated and monounsaturated fats.[8] There is a need to develop a national comprehensive plan to prevent and treat the obesity epidemic as it is closely related to metabolic syndrome. [9]

Hypertension

The other important aspect of management is to optimize patient's blood pressure.[10] The ADA[10] and JNC 7 [10] recommend the goal of blood pressure for a patient with diabetes mellitus to be less than 130/80 mmHg. Angiotensin converting enzyme (ACE) inhibitors, which can prevent microvascular, and macrovascular complications as well as the progression of albuminuria[10,] are preferred therapeutic agent unless contraindicated otherwise.

Glycaemic control

Good glycaemic control is important in the management of patient with metabolic syndrome. The goal for HbA1C level is less than 7%. UKPDS 33 had demonstrated a 25% reduction in the risk of microvascular complications in type 2 diabetic patients who had achieved intensive glycaemic control.[10] It is also important to identify patients who have impaired glucose tolerance (IGT). One to three quarters of patients with IGT will develop diabetes mellitus within a decade from the time of diagnosis of IGT. The annual progression rates from IGT to diabetes range from 1-10%.[10] The Da Qing IGT and Diabetes study showed that diet and exercise led to a significant decrease in the incidence of diabetes mellitus over a 6-year period among those with IGT.[11] The Diabetes Prevention Program also showed that lifestyle intervention was more effective than therapeutic intervention, and the incidence of diabetes mellitus is reduced by 58% in those receiving lifestyle intervention compared with 31% in those receiving Metformin in patients with IGT.[12] It is therefore important to advise patient to modify their lifestyle early to prevent diabetes mellitus from developing.

Dyslipidemia

Lipid lowering is central to the reduction of morbidity and mortality in patients with diabetes mellitus. The goals of therapy in diabetic patients are to achieve LDL-C <2.59 mmol/L, and HDL-C $\geq$1.03 mmo/L and Tg < 1.69 mmo/L.[10] Statins are preferred agent for dyslipidemia in diabetics as they improve the prognosis and reduce the risk of recurrent coronary events in these patients as shown in the Scandinavian Simvastatin Survival Study (4S),[10] Heart Protection Study (HPS)[8] and Cholesterol and Recurrent Events trial (CARE).[10]

Role of aspirin

Aspirin should be considered in those patients with at least a 10% risk of a coronary event over 10 years.[13] It reduces the raised plasminogen activator inhibitor and fibrinogen that is commonly found in patients with metabolic syndrome.[5]

Management of microalbuminuria

Microalbuminuria, a strong independent risk factor for cardiovascular events, results from endothelial dysfunction and oxidative stress in metabolic syndrome. Treatment with ACE inhibitors, delays mortality in patients with diabetes with microalbuminuria . This benefit occurs regardless of whether patients are hypertensive.[14]

Insulin sensitizers

Thiazolidinediones decrease hyperglycaemia by improving glucose uptake in muscles and adipose tissue and reducing glucose production. It also decreases triglyceride level and raise high-density lipoprotein cholesterol level. It helps to reduce microalbuminuria and blood pressure.[15]

Assessment of CVD risk

Metabolic syndrome confers a 2-fold increase in the relative risk for CVD events in individuals without established type 2 diabetes mellitus.[16] For individuals with the metabolic syndrome who do not have established CVD or type 2 diabetes mellitus, the absolute 10-year CVD risk is best assessed by Framingham risk scoring.[17] Framingham risk scoring system is designed to estimate risk in adults aged 20 and over who do not have heart disease or diabetes mellitus. It is used to determine individual's 10-year risk of developing CHD (myocardial infarction and coronary death). The risk factors used in assessment include age, total cholesterol, HDL cholesterol, systolic blood pressure, treatment for hypertension and cigarette smoking.[17] A person's 10-year risk status will determine the intensity of therapy for each risk factor and, in particular, whether drug therapy should be initiated.[16]

In this patient, there were few reasons for the success of her management. This patient was highly motivated, responsible and compliant to her treatment and health advices. She was well informed about her conditions and its consequences and was involved in her management plan and decision making process. Continuity of care was also a key element in successfully managing this patient.

CONCLUSION

Metabolic syndrome is common and family physicians need to be aware that they are handling a group of patient with high cardiovascular risks. This case report have highlighted the role of family physician in the management of patients with metabolic syndrome that portrays all principles of family medicine such as the importance of continuity of care, the coordination of patient care, prevention of diseases, modification of disease risk factors, and the importance of patient education for increased awareness of the problems so as to instil autonomy in the patient to take care of their own illness.

REFRENCES

1. Nelson MR. Managing metabolic syndrome and multiple risk factors. *Aust Fam Physician.* 2004;33(4):201-4 [PubMed]
2. Reaven GM. Metabolic syndrome. Is this diagnosis necessary? *Am J Clin Nutr.* 2006;83(6):1237-47 [PubMed]
3. Turnbull C, Woodward N, Croxson S. Current diagnosis and management of diabetes. *The Practitioner.* 2004;24:8362-8 [PubMed]
4. De Artola I Jr. Metabolic syndrome early identification and a proactive approach are warranted. *Am Fam Physician.* 2004;69(8):1868-70 [PubMed]
5. Kwok LO, Karen SLL, Bernard MYC. The Metabolic syndrome. *Medical Progress.* 2006;33(7):325-8
6. Despres JP. Our passive lifestyle, our toxic diet, and the atherogenic /diabetogenic metabolic syndrome: can we afford to be sedentary and unfit? *Circulation.* 2005;112(4):453-5 [PubMed]
7. Carapetis M, Phillips P. Eat less, walk more. *Aust Fam Physician.* 2002;31:1065-71 [PubMed]
8. Deen D. Metabolic syndrome: time for action. *Am Fam Physician.* 2004; 69:2875-82 [PubMed]
9. Mokdad AH, Serdula MK, Dietz WH, *et al*. The continuing epidemic of obesity in the United States. *JAMA.* 2000;284:1650-1 [PubMed]
10. Beckman TJ. Regular screening in type 2 diabetes. A mnemonic approach for improving compliance, detecting complications. *Postgrad Med.* 2004;115:19-27 [PubMed]
11. Pan XR, Li GW, Hu YH, *et al*. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and Diabetes Study. *Diabetes Care.* 1997;120(40):537-44 [PubMed]
12. Knowler WC, Barrett-Connor E, Fowler SE, *et al.* Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. Diabetes prevention program. *N Engl J Med.* 2002; 346:393-406 [PubMed]
13. Wagh A, Stone NJ. Treatment of metabolic syndrome. *Expert Rev Cardiovasc Ther.* 2004;2(2):213-28 [PubMed]
14. Strippoli GF, Craig M, Deeks JJ, *et al.* Effects of angiotensin converting enzyme inhibitors and angiotensin II receptor antagonists on mortality and renal outcomes in diabetic nephropathy: systematic review. *BMJ.* 2004;329:828-39 [PubMed]
15. Scott CL. Diagnosis, prevention, and intervention for the metabolic syndrome. *Am J Cardiol.* 2003;92(1A suppl):35i-42i [PubMed]
16. Grundy SM, Cleeman JI, Daniels SR, *et al.* Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. *Circulation.* 2005;112:2735-52 [PubMed]
17. Third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III): final report. *Circulation.* 2002;106(25): 3143-3421 [PubMed]

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