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CAN SEVERE HYPERTRIGLYCERIDEMIA CAUSE ANGINA IN THE ABSENCE OF SIGNIFICANT CORONARY ARTERY LESIONS?

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ABSTRACT

Hyperlipidaemia is considered one of the most common risk factors for ischemic heart disease. Severely elevated triglyceride levels may lead to hyperviscosity which may contribute to endothelial dysfunction, tissue ischemia and chylomicronaemia. This case report features a 39-year-old, non-smoker male with a past medical history of hypertension and no family history of coronary artery disease, who presented to the clinic with angina. The electrocardiogram and echocardiogram were normal. Laboratory tests were normal except for severely elevated triglyceride levels. A dobutamine stress echocardiogram was positive, so the patient underwent coronary angiography which revealed normal coronary arteries with slow flow in all of the coronary arteries (TIMI 2 flow grade). The patient was then discharged on high-intensity statin, fenofibrate, isosorbide mononitrate, nebivolol, and was given lifestyle modification instructions. After 3 months the patient came back to the clinic for follow-up. He was in good general condition and symptom free. Laboratory tests (including lipid profile) were within normal limits.

KEYWORDS

Ischemic heart disease, hypertriglyceridemia, dobutamine stress echocardiography

LEARNING POINTS

- Understanding the effects of severely elevated triglyceride levels on coronary endothelial function.
- Prevention of harmful effects resulting from severely elevated triglyceride levels.

INTRODUCTION

Despite advances in drug and interventional therapy, ischemic heart disease is still a principal cause of morbidity and mortality in Western society[1]. Hyperlipidaemia is considered one of the most common risk factors for ischemic heart disease[2]. Elevated plasma triglyceride concentration is a common biochemical finding, but the evidence for the benefit of treating this lipid disturbance remains less robust

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than that for treating elevated low-density lipoprotein-cholesterol. Therefore, current prevention guidelines focus on low-density lipoprotein (LDL) cholesterol lowering as the primary target of therapy, primarily by statins[3]. Elevated triglyceride levels are associated with increased long-term risk of ischemic heart disease. However, short-term complications of severely elevated triglyceride levels may result from hyperviscosity which may contribute to endothelial dysfunction, tissue ischemia and chylomicronaemia[4].

CASE DESCRIPTION

A 39-year-old, non-smoker male with a past medical history of hypertension and no family history of coronary artery disease presented to the clinic with pressure-like chest pain on minimal exertion, and dyspnoea grade 3 on the modified Medical Research Council (mMRC) dyspnoea scale. Results of physical examination included: blood pressure 138/79 mmHg, arterial oxygen saturation (SaO_2) 98%, respiratory rate 20 breaths per minute, heart rate 85 bpm, body mass index (BMI) 39 kg/m^2. The rest of the physical examination was normal. Electrocardiogram (ECG) and echocardiogram were normal. The laboratory tests results are shown in *Table 1*.

Test	Result
White blood cells, WBC (×10^9/l)	5.2
Haemoglobin (g/dl)	14.2
Mean corpuscular volume, MCV (fl)	85
Mean corpuscular haemoglobin, MCH (pg/cell)	32
Platelets (×10^9/l)	300
Creatinine (mg/dl)	0.9
Blood urea nitrogen, BUN (mmol/l)	4
Glucose fasting (mg/dl)	95
Sodium, Na (mEq/l)	142
Potassium, K (mmol/l)	4.3
International normalized ratio, INR	1.2
Partial thromboplastin time, PTT (sec)	29
Alanine aminotransferase, ALT (U/l)	28
Aspartate aminotransferase, AST (U/l)	27
Troponin I (ng/ml)	0.01 (negative)
Total cholesterol (mg/dl)	412
Triglycerides (mg/dl)	1245
Low-density lipoprotein, LDL (mg/dl)	110
High-density lipoprotein, HDL (mg/dl)	53
Amylase (U/l)	65
Lipase (U/l)	73
Plasma viscosity (mPa/s)	1.98 (normal range 1.50-1.72)

Table 1. Laboratory test results.

Chest X-ray showed clear lungs with no visible nodules, tumours or masses. Abdominal ultrasound did not show any abnormality in the liver, pancreas, or gallbladder, and there was no free fluid in the abdomen. The patient had intermediate pre-test probability of coronary artery disease, so we decided to perform a non-invasive test of the coronary arteries. The patient was unable to exercise due to extreme obesity, so we could not perform an exercise stress ECG. Therefore, we performed a dobutamine stress echocardiogram which was positive. The patient then underwent coronary angiography which revealed normal coronary anatomy with absence of coronary artery plaques and no significant coronary lesions. However, there was slow flow in all of the coronary arteries (TIMI 2 flow grade). The patient was then discharged on high-intensity statin, fenofibrate, isosorbide mononitrate, nebivolol, and was given lifestyle modification instructions.

After 3 months of treatment the patient came back to the clinic. The patient was in good general condition, with no chest pain and no dyspnoea on exertion.

DISCUSSION

We report the case of a patient with angina likely caused by triglyceride-mediated hyperviscosity. Hypertriglyceridemia is strongly associated with metabolic syndrome, and because the incidence of metabolic syndrome and obesity has increased in the recent years, the incidence of hypertriglyceridemia is likely to increase too[5]. Most cases of hypertriglyceridemia are not severe enough to cause acute symptoms or complications, but some cases when the levels of triglycerides are very high (more than 1000 mg/dl) it may cause acute symptoms or complications due to hyperviscosity[6,7].

Inokuchi and colleagues reported a case of 56-year-old male who presented with a sudden coma and it was noted that he had very high levels of serum triglycerides with a high plasma viscosity. The patient then improved gradually with administration of intravenous fluids and reduction of triglycerides levels[8]. Wiltchik and colleague reported a case of 54-year-old female who had tonic-clonic seizure and then was brought to the emergency department in an unresponsive state. Her lipid profile showed that triglyceride levels were very high. The patient then underwent plasmapheresis with improvement of triglyceride levels and mental state[9]. Zhou and colleagues reported a case of 58-year-old male with metastatic rectal cancer treated with capecitabine who presented to the emergency department with angina. The patient then underwent coronary

angiography which showed no significant coronary lesions. His lipid profile showed very high levels of triglycerides. The patient then received fenofibrate therapy with improvement of triglyceride and, cholesterol levels and angina[10]. Rosenson and colleagues reported that elevated triglyceride levels can raise the plasma viscosity which may decrease blood flow to target organs[11].

Therefore, we think that in our patient severe hypertriglyceridemia decreased the coronary blood flow and thus caused angina. This was consistent with slow coronary blood flow in coronary angiography. Hypertriglyceridemia can result from obesity, familial disease, poorly controlled diabetes mellitus, and alcohol misuse. The only one of these risk factors, our patient had was extreme obesity (BMI 39 kg/m^2).

Our patient adhered to a strict diet and exercised daily and took all prescribed medications, so after 3 months his BMI was 32 kg/m^2 and triglyceride levels were significantly lower, and he had no remaining symptoms.

CONCLUSION

In our case we noted that severely elevated triglyceride levels may lead to hyperviscosity which may contribute to coronary endothelial dysfunction that may cause angina. Large studies on the relationship between severely elevated triglyceride levels and angina are needed.

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