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Patient with extremely high combined hyperlipidemia in primary care practice – a case study

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Summary

This case report describes the history of a patient who, for several years, repeatedly complained to her family physician regarding her abdominal problems. She was diagnosed with pancreatic necrosis and erosive gastritis. Within a few years the patient developed diabetes. Control laboratory tests revealed triglycerides levels about 8000 mg/dl, total cholesterol exceeding 900 mg/dl, LDL exceeding 500 mg/dl, with significantly elevated liver enzymes. *Geriatria 2010; 4: 289-291.*

Keywords: combined hyperlipidemia, hypertriglicerydemia, hypolipemic treatment

Streszczenie

Poniższy opis przypadku ukazuje historię pacjentki, która przez kilka lat zgłaszała się do swojego lekarza pierwszego kontaktu z powodu dolegliwości żołądkowo-jelitowych. Pacjentce zdiagnozowano martwicę trzustki i nadżerkowe zapalenie błony śluzowej żołądka. W ciągu kilku następnych lat u pacjentki rozwinęła się cukrzyca. Kontrolne badania laboratoryjne ujawniły triglicerydy na poziomie 8000 mg/dl, całkowity cholesterol przekraczający 900 mg/dl, LDL przekraczający 500 mg/dl oraz znacząco podwyższone poziomy enzymów wątrobowych. *Geriatria 2010; 4: 289-291.*

Słowa kluczowe: hiperlipidemia mieszana, hypertriglicerydemia, leczenie hipolipemizujące

Case report

A 57-year-old patient complained to her family physician her feelings of general malaise, upper abdominal pain and diarrhea, preceded by constipation. She denied any prior hospitalizations. Two days prior to visiting her physician, the patient's abdominal pain was managed pharmacologically by the emergency medical service, due to suspected nephrolithiasis. On the physical examination the abdomen was bloated with marked, perceptible tenderness in the right lower quadrant, with symptoms of peritonitis present. The abdominal ultrasound examination revealed small deposits in both kidneys and a slight widening of the calycopelvic system of the left kidney. The patient was referred to the hospital. In the surgical ward, pancreatic necrosis was diagnosed and the patient was operated on urgently. The post-operative period was without complications and the patient, in a good general condition, was discharged with recommendations to undergo care with a surgical consulting unit and primary care practice.

During the following months, the patient complained of joint pains. She was referred to a rheumatologist, who diagnosed osteoarthrosis, cervical vertebra scoliosis and spondylosis. The patient underwent rehabilitation; she had been taking NSAIDs for a few years prior, due to osteoarthrosis and several injuries that she had experienced.

Within the next few years the patient admitted to her family physician that she had experienced several bouts of abdominal complaints. The patient suffered

from recurrent abdominal pains, nausea or persistent vomiting, belching, and diarrhea alternating with constipation. The patient's condition improved with conservative treatment. Concurrently, she was diagnosed with diabetes mellitus type 2; insulin therapy (glargine) was initiated. The patient was referred to a metabolic disease consulting unit, where she had basic laboratory tests performed. The tests were repeated due to received abnormalities. In the second test biochemical abnormalities were: TG 8070 mg/dl, total cholesterol 705 mg/dl, LDL 385 mg/dl, HDL 117 mg/dl. The patient underwent treatment in the metabolic diseases consulting unit.

After two years, the patient was admitted to her primary care practitioner, she was suffering from diarrhea and biliary vomiting for a week. On examination, the abdomen was soft and tender in the epigastrium. The patient declined hospitalization and was managed pharmacologically. Due to persistent vomiting she was hospitalized one week later. She was referred for abdominal ultrasound examination and consultation by a gastroenterologist. She was diagnosed with acute gastritis, deformation and dysfunction of the pylorus; the test results for Helicobacter pylori were positive. Standard eradication therapy was instituted. Within two weeks following the eradication, the patient was referred to the hospital due to increasing complaints. Biochemical abnormalities were: AspAT 367 U/l, alanine transferase 135 U/l, GGTP 3947 IU/l, amylase 594 IU/l, total cholesterol 916 mg/dl, HDL 15 mg/dl, LDL 547 mg/dl, TG 7241 mg/dl, OB 40 mm. Urine testing abnormalities: Urine gravity: 1015, proteins: 25, ketones 5. The abdominal ultrasound examination revealed post-inflammatory lesions with small calcifications in the pancreas. The patient was diagnosed with exacerbation of chronic gastroduodenitis. After a week, the patient was discharged for follow-up treatment with her primary care practitioner, and examination for metabolic disorders.

Pharmacotherapy introduced by the general practitioner (40 mg atorvastatin and micronized fenofibrate 267 mg, daily) led to improvement and partial normalization in lipid management. Basic laboratory tests indicated: total cholesterol 258 mg/dl, TG 567 mg/dl.

During the following year the patient complained to her family physician several times of her continuing abdominal ailments. She was treated with standard pharmacotherapy. The patient's latest laboratory tests revealed her total cholesterol was 301 mg/dl, LDL 160 mg/dl, HDL 34 mg/dl, TG 563 mg/dl.

Discussion

ATP III continues to identify elevated LDL-C as the primary target of cholesterol-lowering therapy [1]. According to the ATP III algorithm diabetes is a CHD risk equivalent. All persons with CHD or CHD risk equivalents can be called high risk. Normal lipid concentrations are more atherogenic in diabetic patients than non-diabetic patients due to changes in the composition of lipid particles [2]. Even in diabetic people without any of these risk factors, the cardiovascular death rate is higher than in a non-diabetic cohort without other risk factors. Together with the number of risk factors, the cardiovascular death rate increases in both the diabetic and non-diabetic patients, but the increase is much greater in the diabetic group [2]. In the UKPDS study in 3055 type 2 diabetic patients followed up for 7.9 years, 11% of patients suffered a myocardial infarction or developed angina. None of these patients had any signs of cardiovascular disease at baseline. The investigators identified the following modifiable risk factors in this population: raised LDL-C, low HDL-C, raised blood pressure, hyperglycemia and smoking. They also calculated the anticipated risk reduction attributable to modification of these risk factors [2]. According to the findings, the lowering of LDL-C is a very attractive target for the reduction of coronary heart disease in type 2 diabetes [2].

The goal for LDL-lowering therapy in high-risk patients is an LDL-C level <100 mg/dL. For all high-risk patients with LDL-C levels ≥100 mg/dL, LDL-lowering dietary therapy should be initiated. When baseline LDL-C is ≥130 mg/dL, an LDL-lowering drug should be started simultaneously with dietary therapy [3].

Statin therapy can safely reduce the 5-year incidence of major coronary events, coronary revascularisation, and stroke by about one fifth per mmol/L reduction in LDL-C, largely irrespective of the initial lipid profile or other presenting characteristics. The absolute benefit relates chiefly to an individual's absolute risk of such events and to the absolute reduction in LDL-C achieved. These findings reinforce the need to consider prolonged statin treatment with substantial LDL-C reductions in all patients at high risk of any type of major vascular event.

Less definitive information exists for hypertriglyceridemia. The latest findings indicate that elevated TG levels constitute an independent risk factor for CHD, apart from other lipid/nonlipid risk factors. The

scientific literature suggests that elevated levels of TG are associated with increased CHD risk, but because of their biologic variability and their association with other risk factors (e.g., low HDL-C, insulin resistance, diabetes, hypertension), the benefit of TG lowering requires further investigation in randomized intervention trials[4-7].

Effective treatments are available for lowering TG levels. As indicated by the results of primary and secondary prevention trials, reductions in TG levels may be associated with significant reduction in the combined outcome of risk of death from CHD and nonfatal MI or stroke [7].

Fibrates are first-line therapy for primary hypertriglyceridemia and mixed hyperlipidemia, and are useful in the management of individuals with very high TG levels who are at risk for pancreatitis [7].

Conclusions

The presented case shows hyperlipidemia possibly to be a secondary disorder, developing as a result of diabetes and pancreatitis. Due to extremely high levels of lipid parameters, pharmacological treatment was introduced simultaneously with lifestyle modifications. Regarding the past medical history in this particular patient, metabolic adjustment, including hypoglycemic and lipid-lowering treatment, seems to be the main goal of therapy prescribed by primary care physician [8]. Diabetes, pancreatitis and probable liver steatosis limit both the assortment and dosages of medications. Nevertheless, chronic, combined usage of statins and fibrates led to partial normalization of the patient's lipid management.

Hyperlipidaemia, an important cardiovascular risk factor, should be actively tested for in patients with other risk factors and chronic diseases. This is an area of concern for primary care physicians. Patients with coexisting disorders are particularly susceptible to the consequences of dyslipidaemia. Conversely, ideal lipid levels are significantly more difficult to achieve in this group. Ongoing scrutiny will be required for new treatment strategies, as they are evaluated in clinical practice. The above case confirms that even in combined hyperlipidaemia with excessively high lipid levels, optimisation of lipid management can be acquired with standard and accepted pharmacotherapy.

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References

1. 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) NIH Publication No. 02-5215, September 2002.
2. Taskinen MR. Diabetic dyslipidemia. Atherosclerosis Suppl 2002;3:47-51.
3. Grundy SM, Cleeman JI, Merz CN, Brewer HB Jr, Clark LT, Hunninghake DB, et al. NCEP Report. Implications of Recent Clinical Trials for the National Cholesterol Education Program Adult Treatment Panel III Guidelines. Circulation 2004; July 13. http://www.circulationaha.org DOI: 10.1161/01.CIR.0000133317.49796.0E
4. Goldberg IJ. Hypertriglyceridemia: impact and treatment; Endocrinol Metab Clin North Am 2009;38:137-49.
5. Laforest L, Souchet T, Moulin P, Ritleng C, Desamericq G, Le Jeunne P, et al. Prevalence of low high-density lipoprotein cholesterol and hypertriglycerydaemia in patients treated with hypolipidaemic drugs. Arch Cardiovasc Dis 2009;102:43-50. Epub 2009 Jan 25.
6. Mamcarz A. Hipertriglicerydemia – czynnik ryzyka ChNS; Puls Medycyny 2006; 15(138).
7. Jacobson TA, Miller M, Schaefer EJ. Hypertriglyceridemia and Cardiovascular Risk Reduction. Clinical Therapeutics 2007;29:763-77.
8. Fernández Calvo MA, Fernández Calvo O, Charlín Pato G. Tratamiento de las dislipemias en atención primaria. FAP 2006;4:85-89.