Acute Pancreatitis and Hyperchylomicronemia

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Hyperchylomicronemia is known to lead a life-threatening acute pancreatitis. The major metabolic basis is a deficiency of lipoprotein lipase or apolipoprotein C-II, both of which play a critical role in the metabolism of triglyceride-rich lipoproteins. We presented two secondary chylomicronemic cases: One was already published elsewhere as autoimmune hyperchylomicronemia, in which several episodes of acute pancreatitis were demonstrated. The other was a case of unknown origin, in which both lipoprotein lipase and apolipoprotein C-II were preserved. The case had no history of acute pancreatitis, although it demonstrated coronary heart disease and atherosclerotic major artery disease. From these observation, we speculated cases with no abnormality of lipoprotein lipase or apolipoprotein C-II are less frequently suffered from acute pancreatitis, especially in Japan where fat consumption is less than that in western country. *J Atheroscler Thromb*, 1998 ; 4 : 162-163.

Key words : Chylomicron, Pancreatitis, Lipoprotein lipase, Apolipoprotein C-II, Hypertriglyceridemia, Atherosclerosis

Introduction

Primary hyperchylomicronemia is known as a cause of acute pancreatitis. On contrary, it is sometimes observed that there is no history or sign of acute pancreatitis even in a case with severe hyperchylomicronemia. There is no clear understanding for these phenomenon.

We presented here two hyperchylomicronemic cases and discussed the critical points for the development of acute pancreatitis in hyperchylomicronemic case from a clinical point of view.

Case presentation

Case 1

The 27 years-old female case was published elsewhere

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(1). In brief, the case was a secondary deficiency of lipoprotein lipase and hepatic triglyceride lipase due to the presence of autoantibodies against these enzymes. Graves' disease and idiopathic thrombocytopenic purpura were also accompanied. The case represented several episodes of acute pancreatitis with severe abdominal pain and had received emergency operation three times. The detected highest level of serum triglyceride was 4,550 mg/dl. The case was a typical secondary hyperchylomicronemic case which caused acute pancreatitis.

Case 2

The second case was 57 year-old male. He visited for the treatment of type IV and type V hyperlipidemia. Diabetes mellitus was not present. Hyperlipidemia was first pointed out at 43 years-old of age. There was no history nor sign of acute pancreatitis, although the highest triglyceride level was around 5,000 mg/dl and cholesterol was 700 mg/dl. The main problem was an atherosclerotic coronary artery disease and arteriosclerosis obliterance. He received arterial bypass operation from common iliac artery to left femoral artery for the treatment of abrupt femoral artery occlusion three years ago. Significant coronary stenosis was also observed at the time and

percutaneous transluminal coronary angioplasty was operated. When he visited our hospital serum cholesterol was 199 mg/dl, triglyceride was 1,063 mg/dl, HDL-c was 24 mg/dl and lipoprotein (a) was 9.4 mg/dl under the treatment of 20 mg of pravastatin and 400 mg of bezafibrate. Lipoprotein lipase was 217 ng/ml in postheparin plasma which is within normal range and apolipoprotein C-II was preserved. Apolipoprotein E phenotype was E4/4 on isoelectric focusing analysis. A major mechanism of this hypertriglyceridemic case might be overproduction of triglyceride-rich lipoprotein rather than a decrease in the metabolism.

Discussion

Brunzell and Biermann reported that serum triglyceride level of acute pancreatitis demonstrated biphasic distribution (2). The lower peak is around several hundred mg per dl and it may come from the secondary phenomenon

from acute pancreatitis. The higher peak is around several thousands mg per dl and it may be the cause of acute pancreatitis. However, we presented a case with a very high triglyceride level without any sign nor history of acute pancreatitis. We speculate that acute pancreatitis does not occur even in a severe hyperchylomicronemic state in case that the critical steps to metabolize triglyceride-rich lipoprotein were preserved such as lipoprotein lipase and apolipoprotein C-II.

References

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