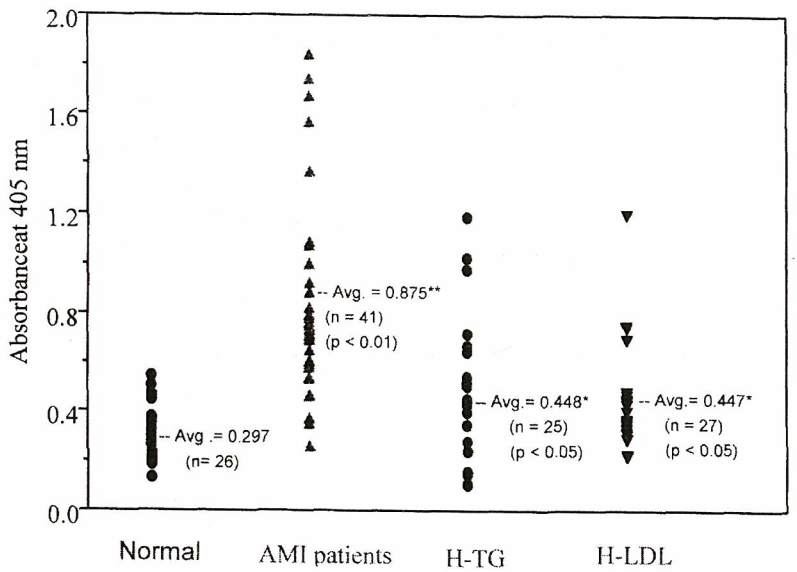

Fig. 3. Comparison of the anti-ox-LDL antibody expression in 4 different groups including normal subjects, AMI patients, high-triglyceride subjects and high-LDL subjects. The antibody titers in AMI patients were 195% higher than in normal subjects, and the titers in high-triglyceride subjects and in high-LDL subjects were also 50% and 50% higher, respectively, than in normal subjects.

= 0.875) were 195% higher than those in normal subjects (average O.D. = 0.297), which is statistically significant ($p < 0.01$). The autoantibody titers in high triglyceride subjects (average O.D. = 0.448) and high LDL subjects (average O.D. = 0.447) were 50% and 50% higher, respectively, than those in normal subjects, which is also statistically significant ($p < 0.05$) (Fig. 3).

DISCUSSION

Atherosclerosis has long been a medical problem in humans. Acute myocardial infarction is the leading cause of death in Western countries, and also in Asian countries in past decades. It has become apparent that coronary atherosclerotic lesions with less severe angiographic disease are associated with rapid progression to severe stenosis or total occlusion and that these lesions may account for acute coronary syndrome such as unstable angina or acute myocardial infarction.[3] All these problems begin from the atherosclerotic plaques which become disrupted and cause a thrombosis. The atherosclerotic plaques that are prone to rupture characteristically have a thin fibrous cap, a large lipid core, activated smooth muscle cells, monocytes/macrophages, and T lymphocytes.[19] But what triggers the plaques to rupture remains unclear. Oxidized LDL has been the main issue in atherogenesis and plague rupture. Products of oxidized LDL was reported to be toxic and chemotactic for circulating monocytes.[2] LDL oxidation may play a quantitatively important role in atherogenesis. Ox-LDL is also immunogenic and can elicit autoantibody formation and activate T cells.[2] The autoantibodies to many epitopes of oxidized LDL are found in atherosclerotic lesion of humans.[20] The autoantibody titer to an epitope of oxidized LDL, MDA-LDL, was reported to be a highly significant predictor of the progression of carotid intimal-medial thickness in a group of middle-aged Finnish males,[21] and elevated plasma levels of MDA-modified LDL may suggest plaque instability.[22]

Chlamydia infection was also suspected in the pathogenesis of acute myocardial infarction as well as in atherosclerosis.[23] Circulating immune complex was reported to be a strong and independent risk factor for development of myocardial infarction.[24] Ryan et al. also reported increased autoantibody titers in patients