# Effect of Pravastatin in Treatment of Hypercholesterolemia in Non-insulin-dependent Diabetes Patients

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**Abstract**

To evaluate the efficacy of pravastatin in treatment of hypercholesterolemia in non-insulin-dependent diabetes patients, a 6-month trial of once daily 10 mg-pravastatin was studied in 30 patients with fairly, well controlled non-insulin-dependent diabetes mellitus. For 27 patients who completed the study, serum total cholesterol and low-density lipoprotein cholesterol were significantly lower 16.7±2.3 and 20.2±3.3% from 286.7±5.9 and 198.6±7.2 mg/dl at before to 232.6±7.9 and 147.8±6.4 mg/dl at 4th week and 237.8±6.4 and 155.0±5.8 mg/dl at 24th week after treatment with pravastatin, respectively ($P<0.00001$). Serum triglyceride level was decreased from 194.7±16.8 mg/dl to 175.0±16.8 mg/dl at 4th week and 176.6±14.1 mg/dl at 24th week and serum high-density lipoprotein cholesterol level was slightly increased from 45.4±2.8 mg/dl to 48.2±2.5 mg/dl at 4th week and 47.5±2.6 mg/dl at 24th week of pravastatin treatment, respectively ($P>0.05$). There was no serious adverse effect except acute hepatitis in one patient who recovered spontaneously after drug withdrawal. Once daily 10 mg-pravastatin is effective in the treatment of hypercholesterolemia in patients with non-insulin-dependent diabetes mellitus.

Atherosclerotic or coronary heart disease (CHD) is a common cause of morbidity and mortality in patients with non-insulin-dependent diabetes mellitus (NIDDM). Diabetes mellitus itself can increase the risk of developing CHD 2-3 fold[1]. This risk accelerates if patients also have other associated risk factors such as hypertension, hyperlipidemia which commonly coexist in NIDDM patients. Increased serum triglyceride, decreased serum high-density lipoprotein (HDL) cholesterol and increased serum total cholesterol levels are common lipid abnormalities found in NIDDM, the prevalence of which may be as high as 50 per cent in some studies[1,2]. In Thailand, studies from several large referral centers revealed the high prevalence of hypertriglyceridemia (≥200 mg/dl) of ~30-40% and hypercholesterolemia (≥250 mg/dl) of ~10-45% in NIDDM patients with the prevalence of CHD of ~10%[3,4]. Of these, only the minority had been treated with lipid-lowering agents. However, poor

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glycemic control in the majority of NIDDM patients in these studies could contribute in some degree to such a high prevalence. The prevalence of hypercholesterolemia in Thai NIDDM patients is about the same as those non-diabetics especially in urban areas but the prevalence of hypertriglyceridemia in diabetics is much higher[5,6]. These results are consistent with the studies from Western countries.

Hypercholesterolemia is well established to be a strong risk factor for atherosclerosis, the incidence of which seems to be directly related with serum cholesterol especially low-density lipoprotein (LDL) cholesterol levels[7,8]. Treatment of hypercholesterolemia, by whatever means, can decrease the morbidity and mortality of or probably prevent the development of CHD[9]. Several agents have been shown to be effective in the treatment of hypercholesterolemia, however, only bile-acid sequestrants and the 3-hydroxy-3-methylglutaryl coenzyme A (HMG CoA) reductase inhibitors are the most potent in terms of reducing serum cholesterol levels[10]. Because of its effectiveness, few adverse effects and simple administration, HMG CoA reductase inhibitors have been widely and increasingly used for the treatment of hypercholesterolemia in the past few years. The objective of this study is to evaluate the efficacy and safety of pravastatin, a HMG CoA reductase inhibitor, in short-term treatment of hypercholesterolemia in Thai NIDDM patients.

## PATIENTS AND METHOD

NIDDM patients (defined by the criteria of the National Diabetes Data Group)[11] who had stable fasting plasma glucose (FPG) in the past two out-patient clinic visits of <200 mg/dl with HbA1c of <10% and had high serum cholesterol levels which was considered to be high risk for the development of CHD were invited to participate in the study. High risk serum cholesterol levels were followed by the guidelines of the second report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation and treatment of high blood cholesterol in adults[12].

The patients were excluded from the study if they had one or more of the following; 1) fasting triglyceride level of >500 mg/dl 2) pregnancy, lactation or may become pregnant 3) recent history of myocardial infarction or cerebrovascular accidents or other severe medical or surgical illnesses 4) diseases or conditions which could affect serum lipid levels such as acute or chronic liver disease, acute or chronic renal failure, untreated hypothyroidism, nephrotic syndrome, obstructive liver disease, alcoholism or heavy drinkers. Drugs known to directly or indirectly disturb serum lipids were prohibited during the study unless they were crucial and their dosage was consistent throughout the study period. Patients who had been on other lipid-lowering agents and fulfilled the inclusion criterias for the study were requested to stop such drugs for at least a month (wash-out period).

The study was an open study consisting of a 3-month pre-treatment dietary therapy and a 6-month pravastatin therapy. Dietary therapy complied with step I diet recommended by the NCEP and should be maintained throughout the study. Monotherapy with pravastatin sodium (Mevalotin$^{R}$) in oral single daily dose of 10 mg after evening meal or bedtime was started if hypercholesterolemia still persisted after 3 months of dietary therapy. Patients were seen at -12, -6, 0, 4, 8, 16, 24 weeks. Body weight, blood pressure (in sitting position after 15-minutes rest) and heart rate were recorded at each clinic visit including FPG and fasting serum lipids. HbA1c was measured at -12, 0, 8, 16, 24 weeks, serum transaminases (SGOT, SGPT) and serum creatine phosphokinase (CPK) were monitored at 0, 8, 16 and 24 weeks. Adverse drug reactions as well as drug compliance were recorded at each clinic visit. Dropouts and discontinued cases were followed-up through letter to clarify their reasons.

The study was approved by the hospital ethic committee and each patient gave written informed consent before beginning the study.

### Laboratory analysis

Serum lipids which included total cholesterol, triglyceride and high-density lipoprotein (HDL)-cholesterol were analysed by enzymatic method using automated machine Hitachi model 704; low-density lipoprotein (LDL)-cholesterol was calculated using Friedewald equation[13]. FPG, SGOT, SGPT, CPK were measured with the same automated machine. Immunoturbidity method by commercial kit (Boehringer Mannheim, Germany) was used in the analysis of HbA1c with the normal value of 3.9-5.7%.

### Statistical analysis

Student paired *t*-test with or without re-

**Table 1. Laboratory parameters (expressed as mean±SE) of NIDDM patients before and during treatment with pravastatin.**

| | week | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | -12 | -6 | 0 | 4 | 8 | 16 | 24 | p value |
| FPG (mg/dl) | 161.8±8.8 | 161.1±11.7 | 151.2±6.4 | 157.6±7.1 | 158.0±5.8 | 176.0±8.2* | 165.8±7.3 | >0.05 |
| HbA1c (%) | 8.1±0.6 | - | 8.2±0.4 | - | 8.1±0.1 | 7.9±0.5 | 8.5±0.4 | >0.05 |
| Total chol. (mg/dl) | 295.0±8.8 | 280.9±7.1 | 286.7±5.9 | 232.6±7.9 | 241.5±5.8 | 244.1±7.6 | 237.8±6.4 | <0.00001 |
| LDL-chol. (mg/dl) | 203.2±7.5 | 190.2±7.1 | 198.6±7.2 | 147.8±6.4 | 158.3±5.5 | 158.5±6.0 | 155.0±5.8 | <0.00001 |
| HDL-chol. (mg/dl) | 47.5±2.9 | 48.6±2.5 | 45.4±2.8 | 48.2±2.5 | 50.0±2.7 | 48.1±2.5 | 47.5±2.6 | >0.05 |
| Triglyceride (mg/dl) | 221.2±19.1 | 209.3±19.0 | 194.7±16.8 | 175.0±16.8 | 180.6±15.2 | 187.6±16.3 | 176.6±14.1 | >0.05 |

*p = 0.02 between week 0 *vs* week 16, FPG = Fasting plasma glucose

peated measures analysis of variance was used. P value of <0.05 denotes statistical significance. Data were expressed as mean±SE unless otherwise indicated.

## RESULTS

There were 30 patients enrolled in this study; 19 had hypercholesterolemia and 11 had combined hypercholesterolemia and hypertriglyceridemia. Seven patients had been treated with other hypolipidemic agents prior to being recruited in this study (3 bezafibrate, 3 gemfibrozil, 1 simvastatin).

Of 30 patients, 27 (18 women, 9 men) completed the study protocol. Their mean age was 58.5±7.1 (S.D.) years with body mass index of 25.9±3.3 (S.D.) kg/m$^2$. All except two patients had taken pravastatin regularly; the drug was not taken for 2 weeks in one patient and 4 weeks in another. As shown in Table 1, serum total cholesterol and LDL-cholesterol levels were not changed during non-drug therapy but lowered significantly after pravastatin was started. Cholesterol-lowering effect of pravastatin was demonstrable at the 4th week and consistent throughout the study. Serum total cholesterol and LDL-cholesterol levels were significantly decreased from 286.7±5.9 and 198.6±7.2 mg/dl at before to 232.6±7.9 and 147.8±6.4 mg/dl at the 4th week and 237.8±6.4 and 155.0±5.8 mg/dl at the 24th week after pravastatin therapy, respectively (P<0.00001). At the end of the 24th week, pravastatin decreased serum total cholesterol 16.7±2.3% (95%CI:12.3-21.1) and LDL-cholesterol 20.2±3.3% (95%CI:13.6-26.7). Serum triglyceride levels were decreased 2.8±6.4% from 194.7±16.8 to 176.6±14.1 mg/dl and serum HDL-cholesterol levels were increased 9.6±6.2% from 45.4±2.8 to 47.5±2.6 mg/dl ; the magnitude of changes of which were not significantly different (P>0.05). Overall diabetes control was stable throughout the study indicated by stable HbA1c levels.

There was no alteration in serum SGOT, SGPT and CPK levels during pravastatin therapy in these 27 patients. Transient, mild myalgia without elevation of serum CPK level occurred in two patients. The study was prematurely terminated in three patients; one developed acute hepatitis at the 16th week of therapy with elevation of serum SGOT of 219 U/L, SGPT 221 U/L and CPK 490 U/L with normal serum bilirubin and alkaline phosphatase levels. The hepatitis viral study as well as serological studies of leptospirosis and rickettsial infection were all negative. The abnormal liver and muscle enzymes returned to normal promptly after pravastatin withdrawal. One patient who had had a previous history of urticaria developed mild urticarial rashes after the 8th week of therapy which disappeared after drug withdrawal. The other one complained of having chest tightness immediately after swallowing the first tablet of pravastatin. Both of these latter two patients refused to try another round of medication. There were no statistically significant changes from baseline in body weight, systolic and diastolic blood pressure as well as pulse rate.

## DISCUSSION

Diabetes mellitus increases the risk of CHD. This risk is greatest in those with multiple risk factors. Diabetic patients who have hypercholesterolemia should be placed on lipid-lowering agents unless the desired cholesterol level can be achieved with diet and exercise. To what extent should serum cholesterol level be lowered depends

on the number of risk factors and the presence or abscence of CHD. It is recommended that diabetic patients who have one or more additional risk factors, for instances, hypertension, current cigarette smoking, family history of premature CHD and have no evidence of CHD should keep serum LDL-cholesterol <130 mg/dl. However, if patients have already had CHD, LDL-cholesterol should be brought down under 100 mg/dl regardless of other risk factors[12,14].

Pravastatin and other drugs in the HMG CoA reductase inhibitor class, for example, simvastatin, lovastatin, fluvastatin act by inhibiting HMG CoA reductase which is the rate-limiting enzyme in cholesterol biosynthesis. The decrease in hepatic cholesterol stimulates the production of LDL receptors on the surface of liver cells resulting in the increase of LDL uptake and the reciprocal decrease in circulating LDL levels[15]. Pravastatin in daily dose of 10-40 mg can decrease serum total cholesterol in the range of 15 to 25% and LDL-cholesterol in the range of 22 to 33% in non-diabetic individuals with primary hypercholesterolemia, serum triglyceride decreases modestly and serum HDL-cholesterol is unchanged or slightly increased in some studies[16-22]. The degree of responses in diabetics seems to be similar to non-diabetics[23-25]. Study of pravastatin treatment in Thai non-diabetic patients with type II hyperlipoproteinemia showed that, with a daily dose of 10 mg (5 mg twice a day), pravastatin could respectively decrease serum total and LDL-cholesterol 26.4 and 33.8% and serum triglyceride was decreased 14.2% with no change in serum HDL-cholesterol[26]. By using the same dose and regimen of pravastatin, Deerochanawong et al[24] and Inoue et al[25] reported a similar degree of responses of serum total cholesterol, LDL-cholesterol and triglyceride in Thai and Japanese NIDDM patients, respectively. In spite of the same dose of pravastatin, the hypocholesterolemic and hypotriglyceridemic effects in our patients were less than those reports mentioned above. Since glycemic control can affect serum lipid levels, it is plausible that the poorer responses may partly be due to poorer glycemic control in our patients given the higher FPG and HbA1c levels in our study. The dosage regimen of pravastatin either once or twice a day administration has similar effects of lowering serum total cholesterol and LDL-cholesterol[27]. However, there are some studies showing that administration of lovastatin, one of HMG CoA reductase inhibitors, twice a day is somewhat more effective than once a day administration, at the same total daily dosage[28,29].

The adverse effects of HMG CoA reductase inhibitors are few and mild in nature[18,20-22,30]. Although long-term drug safety evaluation is lacking, the adverse effects apparently do not increase with time for a period of up to 5 years of treatment[22]. The most commonly reported adverse drug-related effects are confined to the gastrointestinal system which included abdominal pain, constipation, nausea, flatulence. These adverse effects are infrequent, mild and do not require discontinuation of therapy. Insomnia has been reported in ~1% of patients. The most concerned complications are hepatic and muscular adverse effects. Mild and transient elevations of serum transaminases were observed in less than 5% and symptomatic hepatitis was extremely rare. Myalgia without significant elevation of serum CPK was infrequently reported and usually resolved spontaneously without drug withdrawal. Rhabdomyolysis or severe elevation of serum CPK >10 times of normal was reported in patients who used HMG CoA reductase inhibitor combined with gemfibrozil or niacin[31,32]. Therefore, it is suggested that the combined use of HMG CoA reductase inhibitor with fibrate or niacin should be cautious. Although the risk of developing liver impairment and myopathy is minimal, it is recommended that liver and muscle enzymes should be followed, at least initially, in patients who have been treated with HMG CoA reductase inhibitors. Of 5 patients who had adverse drug effects in our study, 2 had transient, mild myalgia, 1 had clinical hepatitis with 5 times elevation of serum transaminases levels, 1 had skin rash and 1 had chest tightness which might not be a drug-related adverse effect. Nevertheless, compared with other studies, the adverse effects from pravastatin in our study is quite high. This is possibly a by-chance incident. Pravastatin claims to have hepatocyte-selective property which is not found in other HMG CoA reductase inhibitors[33]. It is not transported into other cells; therefore, it is unlikely to affect cholesterol metabolism in tissues other than the liver. However, the clinical significance of this property of pravastatin is unknown since the adverse effects of pravastatin are apparently not different from others.

Recommendations for treatment of dyslipidemia in diabetes are based on the results of intervention trials conducted in non-diabetic patients which showed that lowering serum cholesterol levels in hypercholesterolemic patients with or without CHD not only resulted in decreasing the occurrence, recurrence as well as the morbidity and mortality from CHD but also regressed atherosclerotic lesions in coronary arteries[20,22, 34-36]. The regression of atherosclerosis was also seen in carotid arteries as well[21,37]. Whether the benefit of lowering cholesterol levels can be applied to diabetic population at large is unproven. However, given the incidence of CHD and other atherosclerotic complications are increased and being a major cause of death in NIDDM, it is reasonable to treat dyslipidemia in diabetics as aggressive as in non-diabetics in order to prevent or decrease such complications.

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# ผลการรักษาภาวะไขมันคลอเรสเตอรอลในเลือดสูงด้วยยาพราวาสตาตินในผู้ป่วยเบาหวานชนิดไม่พึ่งอินสุลิน

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คณะผู้วิจัยได้ทำการศึกษาถึงประสิทธิภาพของยาพราวาสตาตินขนาด 10 มก. ในการรักษาภาวะไขมันคลอเลสเตอรอลในเลือดสูงในผู้ป่วยเบาหวานชนิดไม่พึ่งอินสุลินที่มีการควบคุมเบาหวานดีพอสมควรจำนวน 30 ราย เป็นระยะเวลา 6 เดือน พบว่าในจำนวนผู้ป่วย 27 รายที่รับประทานยาครบ 6 เดือน มีระดับคลอเลสเตอรอลและ LDL-คลอเลสเตอรอลในเลือดลดลงร้อยละ 16.7±2.3 และ 20.2±3.3 โดยลดลงจาก 286.7±5.9 และ 198.6±7.2 มก./ดล. ก่อนเริ่มรักษา เป็น 232.6±7.9 และ 147.8±6.4 มก./ดล.หลังจากสัปดาห์ที่ 4 และ 237.8±6.4 และ 155.0±5.8 มก./ดล.หลังจากสัปดาห์ที่ 24 ของการรักษาด้วยยาพราวาสตาติน ตามลำดับ ($P<0.00001$) ระดับไขมันไตรกลีเซอไรด์ลดลงจาก 197.4±16.8 มก./ดล.ก่อนเริ่มรักษา เป็น 175.0±16.8 มก./ดล.หลังจากสัปดาห์ที่ 4 และ 176.6±14.1 มก./ดล. หลังจากสัปดาห์ที่ 24 ของการรักษา ระดับไขมัน HDL-คลอเลสเตอรอลเพิ่มขึ้นเล็กน้อยจาก 45.4±2.8 มก./ดล. เป็น 48.2±2.5 มก./ดล. และ 47.5±2.6 มก./ดล.หลังจากสัปดาห์ที่ 4 และ 24 ของการรักษา ตามลำดับ ยาพราวาสตาตินไม่มีผลต่อการลดลงของระดับไขมันไตรกลีเซอไรด์และการเพิ่มขึ้นของระดับ HDL-คลอเลสเตอรอล อย่างมีนัยสำคัญทางสถิติ ($P>0.05$) ไม่พบผลข้างเคียงที่รุนแรงที่เกิดจากการรักษายกเว้นผู้ป่วยหนึ่งรายเกิดตับอักเสบเฉียบพลันซึ่งสามารถหายได้เองหลังจากหยุดยา ยาพราวาสตาตินในขนาด 10 มก. รับประทานวันละครั้ง ได้ผลในการลดระดับไขมันคลอเลสเตอรอลในผู้ป่วยเบาหวานชนิดไม่พึ่งอินสุลินที่มีระดับไขมันคลอเลสเตอรอลในเลือดสูง

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