Clofibrate - A Retrospective Utilization Review

Rita J. Cherni

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Introduction

The medical literature is replete with discussions of hyperlipidemias and their therapies (Table I). Reasons for treating hyperlipidemias normally presented are: 1) To prevent the abdominal pain and pancreatitis associated with some types of hyperlipidemias; 2) To cause a regression of the xanthomatous skin manifestations of some hyperlipidemias; and 3) To prevent further progression of atherosclerosis (1-5). Therapy undertaken to treat xanthomas and the abdominal complications of hyperlipidemia has been shown to be effective (1-6). Hyperlipidemia therapy is most commonly initiated for the third reason, to decrease the rate of development of atherosclerosis and the incidence of associated complications. There is strong evidence implicating some hyperlipidemias, particularly hypercholesterolemia, as a significant risk factor in coronary artery disease (2-4, 7-12). The value of lowering lipid levels in decreasing morbidity and mortality from coronary artery disease has not been proven (2, 4, 6, 8, 12, 14). In fact, recent studies have failed to show a significant decrease in mortality in drug treated patients (9,13,15,16).

As therapeutic benefit has not been proven and as drug therapy is associated with serious adverse reactions (13), attention has been focused on rational treatment approaches which would lower serum lipid levels without subjecting patients to undue dietary restriction or drug toxicity. Several authors have presented similar guidelines for hyperlipidemia therapy (1-6, 10-12, 17). These are best summarized by

TABLE I. The Hyperlipidemias[1] - Distinguishing Features and Treatments (2-4, 12, 14)

Type of Hyperlipidemia	Increased Plasma Lipoprotein	Distinguishing Features	Dietary Treatment	Drug Treatment of Choice
I	Chylomicrons	C normal or increased; TG increased; detected in early childhood; rare hyperlipidemia; xanthomas; abdominal complications	Restricted fat	No drug treatment
IIa	Low Density Lipoprotein	C increased; TG normal; detected in infancy or early childhood; common hyperlipidemia; xanthomas; increased CAD risk	Low C; low saturated fat; increased polyunsaturated fat	Cholestyramine
IIb	Low Density and Very Low Density Lipoproteins	C increased; TG increased; detected in infancy or early childhood; if severe, IIa features; milder forms appear like Type IV; common hyperlipidemia; increased CAD risk	Low C; low saturated fat; increased polyunsaturated fat; reduce to and maintain ideal weight; moderate alcohol restriction	Cholestyramine and/or Nicotinic acid Maybe Clofibrate
III	Abnormal Intermediate Lipoprotein	C increased; TG increased; detected in early adulthood; relatively uncommon; glucose intolerance; xanthomas; premature PVD	Low C; reduce to and maintain ideal weight; high protein; controlled fat; limited alcohol	Clofibrate
IV	Very Low Density Lipoprotein	C normal or increased; TG increased; detected in adulthood; common hyperlipidemia; glucose intolerance in 50% of cases; obesity; increased risk of CAD	Controlled carbohydrate; reduce to and maintain ideal weight; moderately restricted C; limited alcohol	Clofibrate
V	Chylomicrom Very Low Density Lipoproteins	C normal or increased; TG increased; detected in early adulthood; relatively uncommon; glucose intolerance; xanthomas; abdominal complications	Restricted fat; reduce to and maintain ideal weight; controlled carbohydrate; high protein; moderately restricted C; alcohol not recommended	Nicotinic acid

[1] C - Cholesterol
TG - Triglycerides
CAD - Coronary Artery Disease
PVD - Peripheral Vascular Disease

the noted worker in the field of hyperlipidemia research, Donald Fredrickson, in a "A Physician's Guide to Hyperlipidemia" (14). Excerpts from this article appear in the third edition of the cardiology text, The Heart (18). Many of Fredrickson's ideas also appear in the earlier second edition of this same text (19).

Fredrickson (14) considered diet to be the most important component of all hyperlipidemia therapies. Before prescribing any diet, at least two cholesterol and triglyceride measurements should be obtained as a baseline. Drug therapy should not be undertaken without knowledge of what diet alone can do. A drug should probably not be used unless diet fails to lower cholesterol and triglycerides below 300 mg% and low density lipoprotein below 200 mg%. At least a 20% decrease in lipids should be sustained to warrant continuation of the drug.

At the Veterans Administration Hospital (VA) in Madison, Wisconsin, it was noted that a significant number of patients had been receiving clofibrate (Atromid - S^{R} - Ayerst), a widely used hypolipidemic agent, without proper follow-up. This, together with the questionable efficacy of hypolipidemic drugs in reducing morbidity and mortality from coronary artery disease, prompted the investigators to undertake a clofibrate utilization review to assess the extent of the problem. The study was designed around the hyperlipidemia therapy guidelines as set down by Fredrickson (14). The purpose of the study was to assess the appropriateness of clofibrate prescribing and follow-up.

Methodology

A retrospective review was conducted on the medical care records of patients taking clofibrate. The patients' names were obtained from the VA Hospital pharmacy department, which provided a list of all new and refill clofibrate prescriptions over two months' time. As patients can only obtain a one month's supply of medication, it was felt that a list covering two consecutive months would identify most patients taking the drug. The investigators' objective was to review all data pertaining to a patient's hyperlipidemia management. These data, which included coronary artery disease risk factors, hyperlipidemic type, age, cholesterol and triglycerides values, body weight, dietary measures and drug therapy, were collected on a "Patient Data Sheet" (Fig. I). The "comments" section of this sheet provided space for other relevant information including height, dates of myocardial infarction and cardiovascular surgery, family history of hyperlipidemia or coronary artery disease, presence of xanthomas, a description of the patient's physique and the treatment of his problem prior to admission to the VA Hospital.

Each patient's medical care record was reviewed intensely to determine the scope of hyperlipidemia intervention. The reviewer examined outpatient and inpatient narratives, laboratory results, physician orders, consults, nursing notes, treatments and information sent from other hospitals. The investigators were particularly interested in documentation of a dietician's interaction with a patient. According to the dietary service at the VA Hospital, if a patient is

seen by a dietician, it is always documented in the medical record. Attention was also focused on the circumstances surrounding the prescribing of clofibrate. The date of drug initiation and dosage were noted. An attempt was made to determine what prompted the physician to prescribe clofibrate. This information, when available, was found in the narrative section of the medical record.

The team of investigators included a University of Wisconsin Hospitals pharmacy resident (R.J.C.), a VA cardiologist, and a VA clinical pharmacy practitioner. The pharmacy resident was responsible for data collection. All three individuals participated in establishing rational hyperlipidemia therapeutic criteria and evaluating the information derived from the patient medical records. Data were evaluated using the Clofibrate Utilization Assessment Sheet (Fig. II). The rational therapeutic criteria (Table II) were largely based on Fredrickson's hyperlipidemia therapy guidelines.

TABLE II. Rational Hyperlipidemia Therapy Criteria[1]

1. Dietary management is the keystone of therapy for all hyperlipidemias.
   a) Before any diet is prescribed at least two cholesterol and triglyceride levels should be obtained within 1 to 4 weeks of each other as a baseline.
   b) A dietary trial of at least two months' duration or until ideal weight[2] is reached should be prescribed.
   c) The patient should be seen by a dietician when a diet is prescribed.
   d) A dietetic trial should be monitored by monthly cholesterol and triglyceride determinations and be thoroughly evaluated before drugs are prescribed.
2. Adequate baseline determinations should be performed before any hypolipidemic agent is employed.
3. If use of a hypolipidemic agent is warranted, the agent prescribed should be appropriate for the patient's hyperlipidemic type.
4. Drugs should not be used unless diet fails to lower cholesterol and triglycerides below 300 mg%.
5. If a drug fails to decrease cholesterol and/or triglycerides by at least 20% below base level, its continuation is not warranted.
6. The initiation of therapy with a hypolipidemic drug in a

patient requires adequate follow-up.

a) The effectiveness of a drug should be assessed with at least one cholesterol and triglyceride determination within 8 to 12 weeks after initiation of therapy.

b) If drug therapy is continued beyond the first 8 to 12 week period, periodic cholesterol and triglyceride levels should be performed.

c) Drug therapy should be accompanied by ongoing dietary therapy.

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[1] Based on hyperlipidemia guidelines from Fredrickson's "A Physician's Guide to Hyperlipidemia" (14).

[2] Ideal body weights derived from Metropolitan Life Insurance tables using the patient's height and assuming a medium frame (20).

Hyperlipidemia typing was done by the VA Hospital clinical laboratories. Some patients had been diagnosed as exhibiting more than one hyperlipidemic type during their clinical courses. The type present at the time of clofibrate initiation was noted. Patients having no recorded types prior to drug treatment were designated as "not typed". Individuals classified as "normal" were also recorded. For purposes of this study, in cases where use of a drug was warranted, clofibrate was considered to be an acceptable choice for types IIb, III, or IV. It was not considered to be an acceptable choice for types II, "normal," or "not typed."

The investigators considered an acceptable dietary trial to have occurred if a specific diet was prescribed at least two months prior to clofibrate therapy and if a patient had been counseled by a dietician. A dietician's note in the medical record or a dietary consult which is an official physician request for the specialized services another department can offer a patient, was considered adequate proof that a dietician had been involved in a patient's care. Adequate evidence of ongoing dietary therapy after clofibrate had been prescribed was a dietetic note in the medical record, a dietary consult, or a statement by a physician that he counseled a patient regarding diet.

Adequate baseline determinations for dietary therapy were considered to be at least two sets of cholesterol and triglyceride determinations performed within 1 - 4 weeks of each other. Low density lipoprotein cholesterol is not routinely calculated at the VA and,

therefore, not included as part of the rational therapy criteria. It was assumed laboratory determinations were performed under the conditions deemed optimal by many authors (1-5, 12, 14):

1. The patient should not be on a diet of any sort, particularly a weight-loss diet.
2. The patient should be at a steady weight.
3. The laboratory should regularly control its analyses by comparison with acceptable standards.
4. Cholesterol and triglyceride determinations should be done no sooner than 4 to 8 weeks following an acute illness or traumatic episode, e.g., myocardial infarction.
5. Sampling should be preceded by a 12 to 16 hour fast.

Fifty-eight ambulatory patients, fifty-seven males and one female, were included in the review. The average age at the time of clofibrate initiation was 51.2 years with a range of 31 to 78 years. Of the fifty-eight medical care records reviewed, forty-one courses of therapy were fully evaluated using the Clofibrate Utilization Assessment Sheet. Each course of therapy had been initiated and continued by VA personnel. Of the remaining patients, some had been prescribed clofibrate in the late sixties when hyperlipidemia therapy guidelines were not generally available. Others had been treated by their local physicians and then referred to the VA, which assumed responsibility for their follow-up. There were seventeen patients in this group. Two components of follow-up were evaluated in these

patients: 1) Whether concomitant dietary therapy occurred, and 2) Whether periodic cholesterol and triglyceride determinations were performed.

TABLE III. Incidence in the Patient Population Studied Of Factors Associated With Or Known To Exacerbate Hyperlipidemias[1].

Risk Factor	No. of Patients	Percentage Of Patients
Smoker	24	41.4%
History of Smoking	23	39.7%
Obesity	37	63.8%
Alcohol Use	23	39.7%
Diabetes	11	19.0%
Hypothyroidism	1	1.7%
Family History of Coronary Artery Disease	32	55.2%
Renal Disease	1	1.7%
Hypertension	12	20.7%

[1] Fifty-eight Patients

TABLE IV. Incidence of Hyperlipidemic Types Within The Patient Population Studied

Shown are the percentages of patients included in each hyperlipidemic classification. Patients considered to be normal or who were not typed are also included. Types are presented exactly as stated in the medical record.

Hyperlipidemic Type	No. of Patients	Percentage Of Patients
II	1	1.7%
IIa	1	1.7%
Borderline II	1	1.7%
Normal to Borderline II	1	1.7%
IIb	3	5.2%
IIb or IV	1	1.7%
III	1	1.7%
IV	28	48.3%
Borderline IV	3	5.2%
Normal to Borderline IV	1	1.7%
Not typed	12	20.7%
Normal	5	8.6%

[1]Fifty-eight patients

Results

A total of fifty-eight patients underwent an average of 45.6 months of clofibrate therapy. At the conclusion of the review, forty-nine (84.4%) patients were still taking the drug. Of the medical records reviewed, ten (17.2%) contained in the narrative section a notation relating to the date the drug had been started and the factors that had prompted the physician to use it. In nine (15.5%) patients the dose prescribed was lower than that generally recommended. A weight gain was experienced by twenty-nine (50.0%) patients, twenty-six of whom were obese when clofibrate therapy was started.

Of the forty-one therapeutic regimens which were fully evaluated, forty (97.6%) patients received therapy containing at least one component which did not meet rational therapy criteria (Table V). Only one (2.4%) individual's management satisfied all six criteria (Table VI). One (2.4%) person's regimen met five criteria while nine (22.0%) met four. Of those remaining, nine (22.0%) subjects fulfilled three requirements, nine (22.0%) fulfilled two, six (14.6%) met only one, and six (14.6%) received therapy which met none of the standards.

Inadequate dietary therapy was a significant shortcoming occurring in thirty-five (85.4%) patients (Table Va). There had been no dietary trial before clofibrate in twenty-five (61.0%) individuals. An additional fifteen (36.6%) subjects had not been seen by a dietician either previous to drug therapy or at the time of its prescribing. Adequate information had not been obtained for nine (22.0%) patients

who had undergone a dietary trial. For ten (24.4%) patients, a dietary trial had been prescribed but had not been evaluated for effectiveness before drug treatments.

Clofibrate was prescribed for twenty-nine (70.7%) patients in the absence of sufficient baseline information. Most of these patients had only one or no cholesterol and triglyceride determinations performed prior to initiation of drug therapy. Only twelve (29.3%) patients had at least two blood samples drawn within 1 to 4 weeks of each other.

Clofibrate was not considered to be the drug of choice for fourteen (34.1%) patients. Of this group, five (12.2%) had been categorized as having normal blood lipid levels. Another six (14.6%) had not been typed prior to clofibrate initiation. The remaining three (7.3%) were type II, type IIa, and borderline type II, respectively.

Cholesterol and triglyceride levels had both been less than 300 mg% in twenty-five (61.0%) patients when the drug was prescribed. Clofibrate use was continued beyond the 8 to 12 week trial period in thirteen (31.7%) patients whose cholesterol or triglyceride levels had decreased by less than 20%. In fact, cholesterol levels in four (9.8%) cases and triglyceride levels in two (4.9%) individuals rose more than 5% above base level. Both lipids were elevated 5% or more in six (14.6%) patients.

One or more components of follow-up in thirty-four (82.9%)

patients were inadequate, as shown in Table Vb. A group of twenty-eight (68.3%) patients received the drug, but the efficacy of clofibrate therapy was not assessed after 8 to 12 weeks. Periodic cholesterol and triglyceride determinations were not done in twenty-eight (68.3%) people. Follow-up diet consults were ordered for only twelve (29.3%) patients. There was an indication in seventeen (41.5%) medical records that a physician may have counseled a patient. Of the seventeen patients whose therapy could not be completely evaluated due to insufficient data, six (35.3%) individuals showed no evidence of having periodic cholesterol and triglyceride determinations performed. Diet consults were ordered for nine (52.9%) during clofibrate therapy. There were indications in ten (58.8%) medical records, that a physician may have given a patient dietary advice.

TABLE V. Inappropriate Therapy

Summary of results from the clofibrate utilization review.

Therapeutic Criterion Violated	No. of Patients	Percentage Of Patients[1]
Inadequate Trial Using Dietary Therapy Prior to Drug Treatment	35	84.4%
Clofibrate Therapy Initiated with Inadequate Baseline Information	29	70.7%
Clofibrate Not the Drug of Choice for Hyperlipidemic Type	14	34.1%
Cholesterol and Triglycerides Less than 300mg% Prior to Clofibrate Treatment	25	61.0%
Clofibrate Continued with Less than a 20% Decrease in Cholesterol and/or Triglycerides Below Base Level	13	31.7%
Inadequate Followup of Therapeutic Response to Clofibrate	34	82.9%

[1]Calculations based on forty-one patients: % = No. of patients in category/41 x 100%.

TABLE Va. Inadequate Pre-drug Dietary Therapy

Component of Inadequate Pre-drug Dietary Therapy	No. of Patients	Percentage Of Patients[1]
No Dietary Trial Prior to Drug Treatment	25	61.0%
No Evidence of Dietician Interaction Prior to or at the Initiation of Clofibrate	15	36.6%
Adequate Baseline Data Not Gathered in Cases When a Dietary Trial Occurred	9	22.0%
Effects of Dietary Trial Not Assessed Prior to Initiation of Clofibrate	10	24.4%

[1]Calculations based on forty-one patients: % = No. of patients in category/41 X 100%. A total of thirty-five (85.4%) patients received dietary therapy inadequate in one or more aspects.

TABLE Vb. Inadequate Follow-up Of Therapeutic Response to Clofibrate

Component of Inadequate Follow-up	No. of Patients	Percentage Of Patients[1]
Patient Not Assessed 8 - 12 Weeks After Initiation of Clofibrate	28	68.3%
Periodic Cholesterol and Triglyceride Levels Not Performed	28	68.3%
Dietary Consults Not Ordered During Clofibrate Treatment	29	70.7%
No Evidence in Medical Record of Dietary Counseling by a Physician	24	58.5%

[1]Calculations based on forty-one patients: % = No. of patients in category/41 X 100%. A total of thirty-four patients received follow-up inadequate in one or more categories.

TABLE VI. Rational Therapy Criteria

Listed in successive order are numbers of rational therapy criteria and the respective number of patients whose regimens satisfied that number of criteria.

Number of Criteria Satisfied	No. of Patients	Percentage Of Patients[1]
6	1	2.4%
5	1	2.4%
4	9	22.0%
3	9	22.0%
2	9	22.0%
1	6	14.6%
0	6	14.6%

[1]Calculations based on forty-one patients: % = No. of patients in category/41 X 100%.

Discussion

Meaningful measures of the quality of patient care are becoming increasingly important to health practitioners. Drug utilization review has been rapidly emerging as a practical method of reviewing one aspect of a patient's medical care. The primary goals of drug usage review should be to increase the quality of drug therapy and to assure that medications are prescribed and administered safely. An added goal may be a reduction in cost. Drug usage review is necessary because: 1) There is increasing evidence that the current level of understanding of clinical pharmacology by physicians fall short of optimal standards; 2) The current level of prescribing is very costly, and 3) There is a clear mandate from the public to establish and continually monitor health care performance (21).

When a drug is used in any patient, it's potential benefits and hazards must be carefully weighed. As previously stated, it is not known whether the clinical manifestations of atherosclerosis can be prevented by lowering plasma lipid levels. The use of a drug to lower lipid levels may be considered if a patient cannot, or will not follow a diet, if elevated blood lipid levels are not sufficiently lowered by dietary treatment, or if the patient has severe hyperlipidemia and is at risk of developing complications (2).

Clofibrate has been available as a hypolipidemic agent for more than ten years (2). It is given orally in a daily dose of 1.5 - 2 grams in 2-4 divided doses (1, 2, 6, 10, 12). The drug usually does not have

an effect on plasma cholesterol or triglyceride levels at a dosage of one gram daily or less, and its lipid lowering effects are not enhanced by increasing the dosage above two grams per day (22). In the present review, we found that, although no patients received doses greater than 2 grams per day, nine were initiated on drug therapy with a daily dosage of one gram.

Clofibrate is the drug of choice in the treatment of type III hyperlipidemia. With concurrent dietary therapy, plasma lipids in this type of hyperlipidemia are normalized in practically all patients. Clofibrate is usually the first choice in type IV patients for whom a drug is required. It has unpredictable and usually little efficacy in type V and should not be used in type I hyperlipidemia. Clofibrate may be effective in type II hyperlipidemia, causing a 6 - 10% reduction in serum cholesterol levels. However, several observers have found low density lipoprotein cholesterol to increase in some subjects who are treated with the drug (2).

In this retrospective review of clofibrate utilization, forty-one courses of therapy were fully evaluated. Forty of these patients (97.6%) underwent almost four years of therapy which was inappropriate or inadequate in some respect. An appreciable number of these patients failed to receive adequate pre-drug dietary attention, a serious shortcoming in the viewpoint of the investigators. Fredrickson held diet to be the keystone of hyperlipidemia therapy, stating that often no other treatment is required. Patient understanding of any diet prescribed

is important (14). Levy and colleagues stated that no single diet is effective in all the lipid transport disorders. A dietary prescription for hyperlipidemia must be formulated on an individual basis and is dependent, in part, on the clinical situation. If possible, a patient should receive instructions from a dietician (5).

As patient comprehension is vital to compliance, the investigators considered counseling by a dietician to be a necessary component of therapy. Recently, Gotto et al. (23) demonstrated the effectiveness of dietary counseling in the treatment of patients classified as having type IV hyperlipoproteinemia or endogenous hypertriglyceridemia. Patients receiving a diet without counseling showed little change in their triglyceride levels, while those who received ongoing dietary counseling from physicians and registered dieticians had large significant decreases. Their weights also dropped significantly. Smith et al. also reported similar results (8).

Dietary therapy had not been tried in more than one-half of the patients we evaluated. In those in which therapy by diet had been attempted, it was disappointing that the effects of dietary counseling could not be ascertained because most of the trials were not evaluated before drug was begun. A serious commitment to concomitant dietary therapy was also lacking as evidenced by the sparse number of post-drug dietary consults.

Virtually every author who has discussed hyperlipidemia therapy has stressed the importance of carrying out an adequate number of

cholesterol and triglyceride determinations before any therapy is undertaken. This is important for three reasons: 1) To establish that the patient does indeed have a hyperlipidemia. Hyperlipidemia therapy can mean long-term expense and sacrifice to a patient and should not be approached casually. More than one sample is required, due to the normal variability of the plasma lipids with diet and stress (1). 2) To express the hyperlipidemia in terms of hyperlipoproteinemia. Lipids do not circulate freely in plasma, but are bound to protein as water-soluble lipoproteins (1). Elevated cholesterol or triglyceride levels are associated with abnormally elevated levels of specific lipoproteins (Table I) (6). No single diet or drug can effectively lower all the plasma lipoproteins.(4). Therefore, knowing the hyperlipoproteinemic type provides a rational means of approaching therapy through diet and, if necessary, drugs. 3) To establish a baseline profile. It is highly irrational to prescribe diet or drugs, both of which require quantitative measures of effectiveness, without first gathering baseline information sufficient to serve as a frame of reference. Most workers draw at least three samples at intervals from 1 to 4 weeks (1, 2, 12, 14, 24). The large number of patients in the population we reviewed who were started on diet and/or drug with an incomplete hyperlipidemic workup was disconcerting.

Equally disconcerting was the frequency which clofibrate was prescribed for eulipidemic patients and for patients with hyperlipidemia of unknown type. The use of a potentially toxic drug in patients with

with a normal plasma lipid status cannot be excused. On the other hand, a physician could prescribe a hypolipidemic drug by scrutinizing available data and not formally establishing a hyperlipoproteinemic type. However, the importance of knowing the hyperlipoproteinemic type as a guide to therapy has already been emphasized. It was interesting to observe that all eleven individuals in the "normal" or "not typed" categories each received therapy that satisfied only two or fewer rational therapy criteria.

In the present study, contrary to Fredrickson's recommendations (14), twenty-five patients with cholesterol and triglyceride levels less than 300 mg% were treated with clofibrate. Although twelve of the subjects did achieve at least a 20% decrease in cholesterol and/or triglyceride values, the use of drug was probably not warranted in these patients. Another group of patients was continued on the drug even though they did not sustain at least the minimum acceptable level of response suggested by Fredrickson (14), a 20% decrease in one or both lipids. In twelve patients, the level of one or both lipids actually increased more than 5% above baseline values. A large percentage of patients was started on the drug but not evaluated in 8 to 12 weeks. Some of these subjects did not have blood levels drawn for more than a year after commencing therapy. Another sizable group did not have periodic cholesterol and triglyceride levels performed while taking the drug.

Clofibrate is not the innocuous drug it was once considered to

be (25). The recent Coronary Drug Project (CDP) Research Group (13) evaluation of clofibrate in the secondary prevention of coronary artery disease found evidence of an increased incidence in patients treated with clofibrate of various nonfatal cardiovascular events including pulmonary embolism, thrombophlebitis, arrhythmias, intermittent claudication and angina pectoris. These adverse reactions should be a signal to a physician using the drug in patients with overt coronary artery disease to proceed with extreme caution (22). The CDP also failed to find convincing evidence of a decrease in the incidence of morbidity and mortality from cardiovascular events during the study period. There was no evidence justifying the use of clofibrate as a therapeutic agent in coronary artery disease (13).

It has been shown statistically that there is a positive relationship between medical care recording and medical care quality (26). Lyons and Payne showed that good documentation in the medical care record and good medical care go together. Of the forty-one courses of therapy fully evaluated in the review, only six medical records contained an indication in the narrative section that clofibrate had been started and information regarding the physician's rationale for using it.

The results of this clofibrate utilization review illustrate a pressing need for physician education in the use of hypolipidemic agents. It is the investigators' intent to publish the findings of this study in a journal with a physician audience. The results of a drug utilization

review need to be published so that others may learn from example and be influenced to alter their own prescribing habits. The outcome has already been brought to the attention of the Medical Audit Committee at the VA Hospital. Elsewhere in this institution plans are being made to develop a hyperlipidemia therapy protocol based on Fredrickson's principles (14). Finally, the study will provide further impetus for the addition of an outpatient dietician to the staff and the possible establishment of a hyperlipidemia clinic.

Summary

A retrospective utilization review based on Fredrickson's hyperlipidemia therapy guidelines was conducted on the medical records of patients receiving clofibrate, a widely used hypolipidemic agent. Of the fully evaluated courses of therapy, all but one received therapy which was inadequate in one or more respects. The lack of emphasis on dietary management, a serious therapeutic failing, occurred most frequently. The results of the study point to a need for increased physician education in hyperlipidemia intervention and clofibrate usage.

Name_____ Soc. Sec. No._____ Age____ Diagnosis_____ CAD_____ CAD-Sg_____ Ethanol_____ Diabetes____ Thyroid__

Date	Cholesterol	T.G.	Type	Weight	Dietetic Treatment	Drug Therapy and Dose	Comments

FIGURE II. CLOFIBRATE UTILIZATION ASSESSMENT SHEET

NAME _____ AGE AT THE COMMENCEMENT OF DRUG THERAPY _____

EAL BODY WEIGHT_____ HYPERLIPIDEMIC TYPE _____ DATE OF CLOFIBRATE INITIATION _____

ASSOCIATED RISK FACTORS: SMOKING_____OBESITY_____ALCOHOL USE_____DIABETES_____HYPERTENSION_____
HYPOTHYROIDISM_____FAMILY HISTORY OF CAD_____RENAL DISEASE_____

PRETHERAPY DIAGNOSIS: CORONARY ARTERY DISEASE_____PREVIOUS MI_____CARDIOVASCULAR SURGERY_____
OTHER (SPECIFY) _____

DIETARY THERAPY: EVIDENCE OF DIETICIAN INTERACTION_____LENGTH OF DIETARY TRIAL _____

PREDIETARY WEIGHT_____LBS. PREDIETARY CHOLESTEROL_____mg% PREDIETARY TRIGLYCERIDES_____mg%
POST TRIAL WEIGHT_____LBS. POST TRIAL CHOLESTEROL_____mg% POST TRIAL TRIGLYCERIDES_____mg%
NET CHANGE IN WEIGHT_____LBS. CHANGE IN CHOLESTEROL_____% CHANGE IN TRIGLYCERIDES _____%

CLOFIBRATE THERAPY: INITIAL DOSAGE_____LENGTH OF DRUG THERAPY_____IS PATIENT STILL ON DRUG?_____

PRETREATMENT WEIGHT_____LBS. PRETREATMENT CHOLESTEROL_____mg% PRETREATMENT TRIGLYCERIDES_____
AT FIRST EVALUATION: WEIGHT_____LBS. CHOLESTEROL_____mg% TRIGLYCERIDES_____mg%
NET CHANGE IN WEIGHT_____LBS. CHANGE IN CHOLESTEROL_____% CHANGE IN TRIGLYCERIDES_____%

1. WHEN DID FIRST ASSESSMENT OF DRUG EFFECTIVENESS OCCUR?_____
2. WAS DIETETICS CONSULTED DURING CLOFIBRATE THERAPY?_____
3. WAS THERE DOCUMENTATION IN THE CHART FOR WHY DRUG WAS STARTED?_____

SESSMENT OF CLOFIBRATE USAGE:

1. INADEQUATE PRE-DRUG DIETARY TREATMENT _____
   a. NO DIETARY TRIAL_____ b. NO EVIDENCE OF DIETICIAN INTERACTION_____ c. ADEQUATE
   BASELINE DETERMINATIONS NOT PERFORMED IF A DIETARY TRIAL OCCURRED_____
   d. EFFECTS OF DIET NOT EVALUATED BEFORE CLOFIBRATE _____

2. DRUG INITIATED WITH INADEQUATE BASELINE DATA _____

3. NOT THE DRUG OF CHOICE FOR THE HYPERLIPIDEMIC TYPE _____

4. CHOLESTEROL AND/OR TRIGLYCERIDES LESS THAN 300mg% PRETREATMENT _____
   a. 20% LOWERING IN CHOLESTEROL AND/OR TRIGLYCERIDES ACHIEVED_____
   b. 20% LOWERING IN CHOLESTEROL AND/OR TRIGLYCERIDES NOT ACHIEVED _____

5. DRUG CONTINUED WITH LESS THAN A 20% DECREASE IN CHOLESTEROL AND/OR TRIGLYCERIDES _____

6. INADEQUATE FOLLOW-UP _____
   a. PATIENT NOT ASSESSED 8-12 WEEKS AFTER INITIATION OF DRUG _____
   b. PERIODIC CHOLESTEROL AND TRIGLYCERIDE LEVELS NOT DONE _____
   c. NO POST-DRUG DIETARY CONSULT _____
   d. NO INDICATION THAT PHYSICIAN MAY HAVE GIVEN PATIENT DIETARY COUNSEL _____

7. INADEQUATE DATA _____

8. PATIENT INITIATED ON THERAPY BEFORE THERAPEUTIC GUIDELINES WERE GENERALLY AVAILABLE_____

9. REMARKS:

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