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MANAGEMENT OF HYPERLIPIDEMIA IN PATIENTS ON PERITONEAL DIALYSIS (PD)

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Abstract

Peritoneal dialysis(PD) is a type of dialysis that uses the peritoneum in a person's abdomen as the membrane through which fluid and dissolved substances are exchanged with the blood. Peritoneal dialysis is a treatment for kidney failure that uses the lining of your abdomen(perytoneum), or belly, to filter your blood inside your body. What are the types of peritoneal dialysis? Continuous ambulatory peritoneal dialysis (CAPD),automated peritoneal dialysis.The main differences between the two types of peritoneal dialysis arethe schedule of exchanges one uses a machine and the other is done by hand.If one type of peritoneal dialysis doesn't suit you, talk with your doctor about trying the other type.CAPD doesn't use a machine. You do the exchanges during the day by hand.You can do exchanges by hand in any clean, well-lit place. Each exchange takes about 30 to 40 minutes. During an exchange, you can read, talk, watch television, or sleep. With CAPD, you keep the solution in your belly for 4 to 6 hours or more. The time that the dialysis solution is in your belly is called the dwell time. Usually, you change the solution at least four times a day and sleep with solution in your belly at night. You do not have to wake up at night to do an exchange.Lipid metabolism disorders in patients with End Stage Renal Disease(ESRD) are first described in 1827 by Dr Bright, especially in patients with nephrotic syndrome (1,2,3).

Key words: peritoneal dialysis,hyperlipidemia,ESRD,CAPD.

Introduction

Peritoneal dialysis is a way to remove waste products from your blood when your kidneys can no longer do the job adequately.A cleansing fluid flows through a tube (catheter) into part of your abdomen and filters waste products from your blood. After a prescribed period of time, the fluid with filtered waste products flows out of your abdomen and is discarded.Peritoneal dialysis differs from hemodialysis, a more commonly used blood-filtering procedure. With peritoneal dialysis, you can give yourself treatments at home, at work or while traveling.Peritoneal dialysis isn't an option for everyone with kidney failure. It is known that patients with ESRD present clinical insight into early atherosclerosis and serious cardiovascular complications, cerebrovascular complications with peripheral arterial arthritis much more frequent with much greater number and older age compared with the healthy population. Many patients reach ESRD with established left ventricular hypertrophy, coronary ischemia, and disseminated atherosclerotic vascular disease. Despite advances in the technology, and analysis of transport kinetics in dialysis, car diovascular morbidity and mortality remain markedly increased in patients on dialysis, and the principal cause of death.The purpose of this document is to address the issue of lipid-lowering therapy in patients with ESRD, particularly patients on peritoneal dialysis (PD).Cardiovascular disease (CVD) remains the most common cause of death in chronic kidney disease, including peritoneal dialysis (PD) patients, and has an incidence that is many times that of the general population(5,6). Dyslipidemia is one such risk factor. Increasing hypertriglyceridemia is a recognised complication of CAPD. To investigate the etiology lipid clearance studies using the intravenous fat tolerance test were performed in control subjects and in uremic patients before and after six months CAPD treatment.While water-soluble substances such as glucose and amino acids are transported in aqueous solution in the blood, transport of the water-insoluble lipids involves the participation of a range of complex molecules. Lipids may be either "simple" (cholesterol and nonesterified fatty acids) or "complex" (cholesterol esters and glycerol esters). Because of their insolubility in plasma, all lipids are transported associated with "apoproteins," forming lipoprotein

complexes. These complexes contain varying propor tions of triglycerides, phospholipids, and cholesterol and its esters. Partial exceptions are the nonesterified fatty acids, of which 99% are transported bound to albumin.Oral carbohydrate intake was restricted and the use of hypertonic dialysate kept to a minimum.This paper reviews the possible etiology of dyslipidemia in PD, its clinical relevance, and current treatment recommendations. The dyslipidemia of pd The dyslipidemia observed in PD(Peritonela Dilaysis) patients has been well characterized (7-11). It is known that patients with End stage renal disease(ESRD) present clinical insight into early atherosclerosis and serious cardiovascular complications, cerebrovascular complications with peripheral arterial arthritis much more frequent with much greater number and older age compared with the healthy population. Patients with ESRD are most likely to have type IV hyperlipoproteinemic type (according to Frederickson classification) dominated by high concentrations of serotonin (hypetroglyceride with a value of 28-100%) (12). The exogenous examination of 220 patients with ESRD verifies the tendency of permanent growth and progression of hypertriglyceridemia (13). It is assumed that the subtle, qualitative changes recorded in the morphology (particle size) of the lipoprotein particles in patients with ESRD exogenously increase their atherogenic effect (increased affinity with the LDL oxidation-LDLox arterial subentotone,small LDL, minor HDL particles) with more frequent atherosclerotic damage to the cardiovascular and cerbrovascular system with deadly effects in hemodialysis centers (14).It is about ischemic heart disease, peripheral vascular disease and cerebrovascular insult. For patients treated with chronic hemodialysis and PD , the activity of Hepatal Trigliceride Lipasae (LTGH) is also reduced by 33-45% . Systemic Lipoprotein Lipasae (LPL) activity has been diminished by the cumulative accumulation of toxins or cytokines - Interleukin-1, Interleukin-6, Interleukin-1α, Interleukin-1β (15,15,17) and is accounted for as causative of disorders (HDL-ch and ApoA-I concentrations are reduced, while the concentrations of N-glycoproteins, LDL-ch, ApoB-100, Apo-E, Apo-C, Lp (a) are increased) followed by increased prevalence of atherosclerotic vascular disease. The most common lipid/lipoprotein (Lp) profile in PD shows elevated total cholesterol, low-density lipoprotein (LDL) cholesterol, triglyceride, Lp (a), and apolipoprotein B (apoB) levels. High-density lipoprotein and apolipoprotein A1 levels are usually low. Not all of these abnormalities are seen in all patients. Compared with hemodialysis patients, the most striking differences are the high apoB protein and LDL cholesterol levels, which are usually normal in hemodialysis patients. Levels of oxidized LDL and antibodies to oxidized LDL are elevated in end-stage renal disease (18). Intermediate-density lipoprotein levels are also elevated. These are the Lp particles that are intermediate in size between very low-density lipoprotein (VLDL) and LDL and an increase in their levels represents the delay in removal of the triglyceride component of the VLDL as it is transformed into the cholesterol–ester-rich LDL.The characteristic lipoprotein abnormalities described in PD patients have recently been thoroughly reviewed by Wheeler (30). Dialysis does not correct uremic dyslipoproteinemia, but may alter its pattern (31). Several studies have shown that, once dialysiscommences, continuous ambulatory peritoneal dialysis (CAPD) patients develop a somewhat different and probably more atherogenic lipoprotein profilethan do hemodialysis (HD) patients. Not all reports agree on the exact differences between the two treatment modalities, but this may not be surprising when one considers that the studies come from ethnically and geographically distinct populations such as northern Europe, southern Europe, and the U.S.A. Furthermore, bias in patient and modality selection will vary from one center to the next. However, compared with uremic patients or those on HD, CAPD patients appear to have higher. LDL and total cholesterol concentrations with similar or l ower HDL levels.Many patients do not have their Lps (apoB and apolipoprotein A_1) measured, as these are not considered routine in some centers. This is unfortunate because the measurement of apoB can give better insight into the risks associated with small dense LDL, a common finding in PD patients. Furthermore, LDL cholesterol measurement is not valid if the triglyceride levels exceed 4 mmol/l and therefore only the measurement of apoB gives a true reflection of the LDL profile. In addition, the measurement of the Lps is valid even in the non-fasting state. As PD patients are effectively never fasting, because of the glucose, carbohydrate, or amino acid loading from the PD solutions, apoB retains its meaning but LDL cholesterol levels may be difficult to interpret. The pathogenesis of the overproduction of LDL particles in PD remains obscure. Hypoalbuminemia secondary to peritoneal protein losses of 1–2 g/l of drained dialysate may at least partly contribute to the abnormality (19). This regard, PD patients could be considered similar to patients with nephrotic syndrome. Studies using hepatomaG2 (HepG2) cell line, a cell line derived from hepatomas, suggested that low amino-acid levels may also contribute to overproduction of the apoB hepatic-derived VLDL, which would account for the high LDL levels as LDL is derived from VLDL(20,21).Glucose might also contribute to these lipid abnormalities. The high level of intermediate-density lipoprotein is attributed to the overproduction of VLDL and to defective function of Lp lipase. The low high-density lipoprotein levels seen in PD patients are also poorly understood, but the loss of high-density lipoprotein across the peritoneum may be a contributing factor. The high Lp(a) levels have been associated with malnutrition and inflammatory states and may simply be part of the overall inflammatory response, similar to the other observed elevations of acute phase reactants. The hypertriglyceridemia, seen in PD, results from the overproduction of VLDL and a deficiency in Lp lipase. There may also be a partial

deficiency of hepatic lipase. The pathogenesis of these abnormalities is also not understood, but the use of glucose-based PD solutions and a variety of drugs such as b-blockers aggravate the problem. The usual level of triglycerides seen in PD patients is 3,8–4.8...5.0 mmol/l). To treat or not to treat? The dilemma for clinicians caring for PD patients with dyslipidemia is that there is an absence of data to guide treatment. The typical lipid profile described above for PD patients is an atherogenic profile. Of course YES, because the treatment of dyslipidemia leads to the slowdown and progression of the ESRD. The implication of the study is that dialysis patients have a risk profile for cardiac death that is distinct from nonuremic atherosclerosis. It may be that their disease is too advanced and complex to benefit from statin therapy, that other risk factors such as inflammation are more important in dialysis, or that the design of the study was inadequate to show benefit. On this latter point, the treatment arm was to receive 20 mg statin. During the study, 18% of the patients had a reduced dose of only 10 mg and 20% in the control group received statins over the course of the study. Whatever the explanation for the results, it is now difficult to advocate for the routine use of statins in patients with ESRD treated with PD.. The question remains as to whether or not all PD patients with dyslipidemia, which would be the vast majority of PD patients, should receive lipid treatment. And what is the applicability of PD patients? PD patients with their more atherogenic lipid profile would have shown benefit from statin therapy. Unfortunately, it is unlikely that another large randomized controlled trial of lipid therapy will be undertaken in PD patients because of the costs involved and the difficulty in patient recruitment. Currently, two sets of guidelines exist that comment on lipid management in PD patients – the International Society for Peritoneal Dialysis (ISPD) guidelines24 and the Kidney Disease Outcomes Quality Initiative (K-DOQI) guidelines (22).All recent studies on the patients with ESRD and dyslipidemia, regardless of the type of HD, suggest treatment of disilidemia with statin. Treatment strategies of dyislipidemi:In the general population, lipid management includes dietary measures, weight optimization, exercise, and medication. PD patients already face dietary restrictions and, in order to achieve recommended caloric intake targets, further dietary manipulation is often not achievable or reasonable. Exercise programs are also challenging for many of this patient population because of other comorbidities. These combined challenges usually make weight reduction programs impractical. Thus, medication and perhaps dialysis solution selection remain the only practical option for the vast majority of PD patients. Statin therapy has proven to be safe and effective (23,24,25). The most common side effects are muscle pain, elevations of creatine kinase, and, rarely, abnormalities of liver enzymes. Severe rhabdomyolysis is a rare but serious complication. It is recommended that the relevant muscle and liver enzymes be followed after a patient is started on a statin. However, only a small percentage of patients have enzyme abnormalities severe enough to warrant discontinuing the drug. Fibrates as a class of drugs should be used with caution in renal failure as the kidneys excrete them and dose adjustment is required. The combination of a statin and a fibrate is not recommended in dialysis patients. It usually lowers LDL cholesterol levels by approximately 20%. The drug has been reported to be safe in renal failure patients and may be a good option for patients who are intolerant of statins or for those who are unsuccessful in achieving the therapeutic target with statins alone. Sevelamer, a non-calcium-based phosphate binder has also been shown to reduce LDL and total cholesterol levels in hemodialysis patients by as much as 20% on average. There are some data to indicate that a glucose-sparing approach to choosing PD solutions can have an impact on lipid profiles. Given that glucose is implicated in the pathogenesis of PD dyslipidemia, it follows that a reduction in glucose loading might be helpful. Maximum glucose avoidance can be achieved through minimizing the need for ultrafiltration, which means management needs to include dietary sodium restriction and diuretics, and angiotensinconverting enzyme inhibitors or angiotensin receptor blockers to help retain urinary volume. With regard to the PD solutions, a prescription that includes 7.5% icodextrin and 1.1% amino acids (Nutrineal, Baxter Healthcare Corporation, McGaw Park, IL, USA) which are glucose free, offers 40% less carbohydrate absorption than a regime that is glucose based with three exchanges of 1.5% dextrose and one exchange of 4.25% dextrose (26). Treatment of Hyperlipidemia in Peritoneal Dialysis Patients While a lipid-lowering diet can be moderately effective in HD patients , no data are available concerning dietary manipulation of plasma lipids in patients on PD (32,33). At this juncture however, it is important to note that effecting good glycemic c ontrol in diabetic patients on PD may be important in improving lipid abnormalities, particularly hypertriglyceridemia. There are several studies that examine the effect of pharmacologic therapy on lipid abnormalities in patients on PD. It is clear that the HMG-CoA reductase inhibitors reduce both total and LDL cholesterol in these patients. A recent retrospective analysis of lipid-lowering therapy in patients with different types of renal disease surveyed 18 interventional studies in patients on CAPD. The analysis demonstrated that the HMG-CoA reductase inhibitors reduced total and LDL cholesterol levels, and significantl y increased HDL levels. Furthermore, the fibric acid analogs led to a significant reduction in plasma triglycerides (32). However, as was noted previously in this paper, the treatment benefits from reducing plasma triglycerides alone with regard to cardio vascular disease remain uncertain. The extent of normalization of serum lipids was similar among the different renal disease groups examined, including CAPD patients, those on HD, patients with chronic renal insufficiency, patients with a functioning rena l transplant, and patients with nephrotic

syndrome. If anything, patients on CAPD or with nephrotic syndrome had a greater reduction in triglycerides and an increase in HDL cholesterol with HMG-CoA reductase inhibitors, compared to those in the other rena l disease categories. The similarity of response in CAPD and nephrotic patients may be the result of a similarity of pathogenesis of lipid abnormalities in these two groups, that is, loss of protein into the dialysis effluent (PD) or the urine (nephrotic syndrome) . The fibric acids should be dose-reduced to avoid myopathy. Low doses of these agents however do not appear to lead to rhabdomyolysis. In the retrospective analysis described, in 18 studies of fibric acid analogs, there were no documented episodes of rhabdomyolysis in 282 patients with decreased renal function, treated for a total of 109 patient-years . Although experience with these agents in PD patients is recent, it appears that the HMG-CoA reductase inhibitors do not have to be dose reduced fo r renal failure. However, even in the absence of overt clinical side effects, creatinine phosphokinase (CPK) levels may increase. In one study of CAPD patients, just 10 mg of simvastatin led to as much as a tenfold increase in CPK levels (33). Similar sid e effects have been reported with other agents such as pravastatin (32). Fish oil supplementation lowers triglycerides in PD patients by approximately 30%. The effect on other lipoprotein fractions has been variable. Some studies have shown a decrease, an increase, or no change in HDL. Most studies showe d no change in LDL , although one study did show an increase in LDL cholesterol with fish oil supplementation(33). Carnitine has been advocated to improve lipids in renal failure patients. Carnitine is involved in mitochondrial transport of fatty acids. In patients on HD, plasma and muscle carnitine levels decrease (36-38). A number of factors may be important in producing a different lipoprotein profile in CAPD patients compared to HD patients. Glucose absorption from the peritoneal cavity of CAPD patients varies between 100 – 200 g/day, and results in increased insulin lev els which are thought to enhance synthesis of triglyceride in the liver (11). In addition, protein loss into the dialysate occurs at a rate of 5 – 15 g/day, along with lipoproteins of all type groups. Sieving results in preferential loss of the smaller mo lecules such as HDL, which is lost at a rate equivalent to 34% of its daily synthetic rate (19). This state has been compared to the nephrotic syndrome, wherein hypoalbuminemia is thought to stimulate hepatic lipoprotein synthesis, although a significant difference is that the kidney is not contributing to albumin catabolism in CAPD patients. It has been suggested that peritoneal protein losses upregulate hepatic VLDL production, but the majority of studies have not demonstrated a correlation between trig lyceride or VLDL levels and protein losses or albumin levels in CAPD patients.This may lead to neuromuscular symptoms an d increased triglycerides, although this theory is controversial. The effects of supplementation on triglyceride levels in HD patients have been mixed. Some studies have shown an improved lipoprotein profile (lower triglycerides and high HDL) (39,40), whi le other studies have shown no change or worsened triglyceride levels. In PD patients, total plasma or muscle carnitine levels do not appear to decrease with time. There are fewer studies of carnitine supplementation done in PD patien ts. Warady et al. did not see an effect on lipids with carnitine supplementation in pediatric PD patients. Wanner et al. found that carnitine supplementation led to an increase in triglyceride levels. Recommendations for the Treatment of Lipid Disorders in Patients on Peritoneal Dialysis As can be noted from the foregoing sections, the following principles are established: 1. Lipoprotein abnormalities are prevalent in patients on peritoneal dialysis. 2. Cardiovascular disease is the most important single cause of death in patients on peritoneal dialysis. 3. Treatment of lipoprotein abnormalities, particularly LDL cholesterol, is associated with reduction in cardiovascular morbidity and mortality in the nondialysis population. 4. Both HMG-CoA reductase inhibitors and fibric acid derivatives effect significant reduction in elevated lipid levels in peritoneal dialysis patients. 5. There is no evidence that improvement in lipid levels leads to a reduction in cardiovascular events in patients on peritoneal dialysis. Three studies have evaluated the impact of replacing a glucose-based exchange with an amino-acid- or icodextrin-based solution. A randomized, crossover study in 22 PD patients replaced a glucose exchange with icodextrin or 1.1% amino acids. Each study period was 8 weeks. In those patients using amino-acid solution, both cholesterol and triglyceride levels decreased significantly and in the icodextrin group, the triglyceride levels decreased significantly (27).In another randomized, crossover study, 21 non-diabetic continuous ambulatory PD patients substituted a hypertonic glucose exchange with icodextrin. This intervention showed a decrease in both LDL cholesterol and triglyceride levels (28). Finally, in a prospective observational study of 12 non-diabetic, anuric PD patients who switched to icodextrin for their long exchange, the triglyceride levels significantly decreased and the high-density lipoprotein levels increased(29). Although these studies are all small, they point toward the importance of reducing glucose load in PD patients as a strategy to improve their lipid profile. Complications of peritoneal dialysis can include:Infections. An infection of the abdominal lining (peritonitis) is a common complication of peritoneal dialysis. An infection can also develop at the site where the catheter is inserted to carry the cleansing fluid (dialysate) into and out of your abdomen. The risk of infection is greater if the person doing the dialysis isn't adequately trained.Weight gain. The dialysate contains sugar (dextrose). Absorbing some of the dialysate might cause you to take in several hundred extra calories a day, leading to weight gain. The extra calories can also cause high blood sugar, especially if you have diabetes.

Hernia. Holding fluid in your abdomen for long periods may strain your muscles.Inadequate dialysis. Peritoneal dialysis can become ineffective after several years. You might need to switch to hemodialysis.

Conclusion

PD patients often have an atherogenic lipid and Lp profile. The importance of this as a contributing factor to the high incidence of cardiovascular disease,insult cerebrovascularis, observed in this population remains uncertain. Pharmacologic intervention is both safe and effective but its utility in reducing cardiovascular events has not been proven. A reduction in the glucose exposure associated with conventional PD fluids, achieved by substituting icodextrin- or amino-acid-based solutions, may improve the dyslipidemia observed in PD patients. For the patient with mildly elevated serum triglycerides, dietary intervention may be helpful, as mentioned above. Attention should be paid to glycemic control in diabetics, and hypertonic dialysate should be avoided, if possible. For more serious hypertriglyceridemia, fibric acid analogs are helpful, since they diminish production of VLDL and stimulate lipoprotein lipase.

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