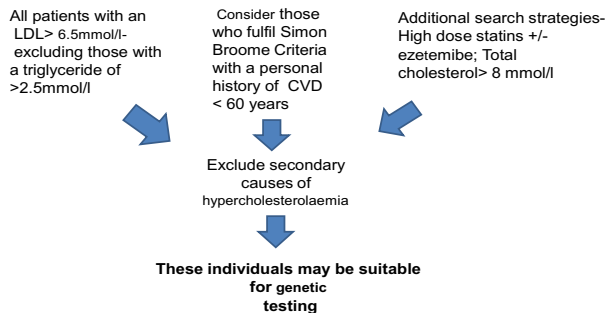


Familial Hypercholesterolaemia Genetic Search Strategy



This has negated the need for initial referral to secondary care to access genotyping and had a subsequent impact on waiting time for new patients into lipid clinic.

LIPOPROTEIN APHERESIS – THE CARDIFF EXPERIENCE

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Introduction: Cardiff Lipid Unit has performed lipoprotein apheresis for 22 years and has completed over 6000 treatments. The unit has 18 patients and utilizes 4 different lipoprotein apheresis techniques. We assessed our treatment efficacy against published standards for 2012 as follows, LDL (low density lipoprotein) cholesterol reductions following treatment of 60% with a post treatment LDL of <1.8mmols/l, total cholesterol (TC) reduction of 50% and lipoprotein(a) (Lp(a)) reduction of 50%.(Thompson et al 2010) A database was created to facilitate data collection and analysis

Method: During 2012 Cardiff Lipid Unit performed 432 lipoprotein apheresis treatments in 18 patients. Each patient received apheresis treatments once per fortnight. Blood samples were taken before and after each treatment and the results were collated. Percentage reduction and mean total and LDL cholesterol values were calculated and averaged over the course of the year for each patient and thereafter for the whole unit.

Results: Unit averaged data were as follows. Mean post treatment LDL cholesterol values of 1.55mmol/l with a reduction of 67%, Lp(a) reduction of 66% and TC reductions of 55%

Conclusion: Analysis of data from the database demonstrates that all efficacy targets were met following lipoprotein apheresis treatment. Periodic analysis of data ensures that targets are met with the aim of optimising cardiovascular outcomes.

EXTENDED RELEASE NIACIN LOWERS MEDIATORS OF VASCULAR INFLAMMATION BUT DOES NOT IMPROVE *IN VITRO* HDL ANTIOXIDANT FUNCTION IN STATIN TREATED DYSLIPIDEMIC PATIENTS

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Aims: Several randomised controlled trials have previously examined the effects of extended release niacin/laropiprant (ERN/LRP) combined with low dose statins in dyslipidaemic patients. I investigated the influence of ERN/LRP versus placebo in patients who had persistent dyslipidaemia despite receiving high doses of potent statins as the latter more accurately reflected actual clinical practice. I assessed the effect of ERN/LRP on mediators of vascular inflammation and HDL's *in vitro* anti-oxidant function.

Methods: This was a randomised double blind cross over trial. I studied the effect of ERN/LRP compared to placebo in 27 patients who were receiving maximum tolerated doses of statins and gauged compliance. I measured lipid profile, apolipoproteins, cholesteryl ester transport protein (CETP) activity, glycated apolipoprotein B100 (gly apoB), paraoxonase 1 activity (PON1), oxidised LDL (oxLDL), lipoprotein phospholipase A2 (Lp-PLA2), lysophosphatidyl choline (lyso-PC), macrophage chemoattractant protein (MCP1), serum amyloid A (SAA) and myeloperoxidase (MPO). I also examined the capacity of HDL to protect LDL from *in vitro* oxidation.

Results: ERN/LRP treatment was associated with a significant improvement in HDL cholesterol and significant reduction in total cholesterol, triglycerides, LDL cholesterol, non-HDL-C, total apoB, lipoprotein (a), CETP activity, oxLDL, Lp-PLA2, lyso-PC, MCP1 and SAA. HDL's capacity to protect LDL against *in vitro* oxidation did not improve on treatment with ERN/LRP compared with placebo.

Conclusions: Treatment with ERN/LRP results in a significant improvement in HDL-C and reduction in pro-atherogenic lipoproteins/apolipoproteins in patients who have persistent dyslipidaemia despite high doses of potent statins. For the first time I have shown that ERN/LRP reduces mediators of vascular inflammation but does not affect HDL's *in vitro* anti-oxidant function in these patients. Withdrawal rates due to flushing were 5% as a result of combining laropiprant with niacin.