

Suboptimal Therapy for Dyslipidaemia in Coronary Bypass Surgical Patients with Premature Ischaemic Heart Disease

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SUMMARY

The incidence of premature multi-vessel coronary artery disease (CAD) is on the rise in Malaysia. The pathogenesis of coronary atherosclerosis is multi-factorial with dyslipidaemia being one such risk factor. Elevated total cholesterol (TC), low density lipoprotein cholesterol (LDL-C) and triglycerides (TG) levels are primarily responsible. We analysed the fasting pre-operative lipid profiles of coronary artery bypass graft (CABG) patients with symptomatic severe premature CAD. A majority of patients had an elevated LDL cholesterol level despite being on a statin. Similarly, no patient with an elevated TG level was prescribed a fibrate.

Pre-operative control of known dyslipidaemia was suboptimal in young adults with angiographically proven severe symptomatic CAD. This is either due to subtherapeutic dose prescribing or failure to commence appropriate anti-lipid drugs. Collectively, general practitioners, cardiologists and cardiac surgeons must be more diligent in monitoring lipid profiles in such patients and be more meticulous in prescribing therapeutic doses to achieve target control.

MAIN PAPER

The incidence of premature multi-vessel coronary artery disease (CAD) is on the rise in Malaysia. Premature CAD is the occurrence of angiographically documented ischaemic heart disease in young adults usually less than 40 years of age. Significant CAD manifests at an earlier age in Asians compared to Western populations as evinced by the INTERHEART study. The median age at first presentation of an acute myocardial infarction (MI) was 53 years (South Asians) versus 63 years (Westerners) and a first document MI occurred in 9.7% of Asian males aged less than 40 years.¹

The pathogenesis of coronary atherosclerosis is multi-factorial and includes classical risk factors such as hypertension, diabetes mellitus, stress, family history (genetics), dyslipidaemia and smoking. With the exception of genetics, all other factors are modifiable. Familial hypercholesterolaemia and lipoprotein disorders have been implicated as a major risk factor for the development of premature CAD.²

We conducted a retrospective observational analysis of lipid profiles on 49 consecutive patients with symptomatic severe

premature CAD who required isolated coronary artery bypass graft (CABG) surgery from January 2008 till September 2014. This represented approximately 3% of our overall CABG workload. The majority were male patients 44/49 (89.7%) with a mean age of 37.9 years. Most patients (67%) had a triple bypass (CABG x 3) procedure for symptomatic and prognostic reasons. All patients were on some form of anti-lipid therapy prior to surgery. We analysed their fasting pre-operative lipid profiles.

Thirty patients (61%) had deranged TC (range <5.17mmol/L), 31(63%) had deranged TG levels (range <1.7mmol/L), 27 (55%) patients had a deranged LDL level (range <3.37mmol/L), and 33 (67%) have a lower HDL level than the normal (range 1.04-1.55mmol/L).

All 27 patients with an elevated LDL were already on a prescribed statin. Statins help reduce hepatic cholesterol biosynthesis via inhibition of the enzyme HMG-CoA reductase and additionally have pleotropic anti-inflammatory properties. None of the 31 CABG patients with an elevated TG level were given a fibrate agent. Fibrates inhibit TG synthesis, increases lipoprotein lipase activity, increase the catabolism of LDL-C and chylomicrons and enhance synthesis of apolipoprotein A-1 (apo AI). Apo AI is protective against atherosclerosis by mediating reverse cholesterol transport and helps with plaque stabilization. Fibrates increase plasma HDL levels via induction of apo AI gene expression.

Our small but contemporary institutional experience demonstrated that a majority of young patients with premature CAD referred for CABG surgery had unacceptably high LDL levels pre-operatively despite prescribed statin therapy suggesting inadequate monitoring of levels and sub-therapeutic prescribing. Furthermore no patient with hypertriglyceridemia (TG) was prescribed any appropriate TG-lowering fibrate medication. In fact had we applied a more stringent criteria of recommended target LDL levels (< 1.8mmol/L) for secondary prevention in high risk individuals,³ then even more of our patients would be deemed to have poor control pre-operatively.

Dyslipidemia plays a significant role in the development of premature severe multi vessel CAD hence primary care physicians, referring cardiologists and even cardiothoracic surgeons must ensure more optimal control of this modifiable

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risk factor through diligent monitoring of lipid profiles. Simply commencing a patient on a statin is inadequate. The patient must be on an appropriate hypolipid agent or combination of drugs, and the dose meticulously titrated to achieve target control whilst monitoring for side effects or intolerance.

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