Lipoprotein(a) as a clinical decision-aid tool
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Background

- Lipoprotein(a) (Lp(a)) is an inherited independent risk factor (RF) for cardiovascular disease.
- Its clinical role remains limited, however, given the lack of standardized prospective assays, limited therapeutic agents to lower Lp(a), and lack of outcomes trials for treatment targeting it.

Objective

- We hypothesize that knowledge of an elevated Lp(a) will intensify treatment targets for traditional cardiovascular RFs and increase cascade screening.

Methods

- This is an observational cohort of consecutive patients (n=95) referred for elective percutaneous coronary intervention (PCI) at a single academic hospital seen by the Preventive Cardiology service. Eligibility for Lp(a) screening was adapted as a hybrid of current National Lipid Association recommendations and European Atherosclerosis Society guidelines and required one of the following:
  1. premature coronary disease (M<55, F<65)
  2. family history of premature CAD
  3. requirement for PCI despite:
     a. well-controlled traditional RFs (BP<140/90, LDL<100, A1c<7%; non-smoker)
     b. at least moderate intensity statin (simva 40, atorva 40-80, or rosuva 20-40mg daily).

Results

- Of 95 patients (mean age 57±8, 64% white, 82% male), 57 (60%) were on moderate or high intensity statin therapy.
- Mean LDL-C and non-HDL-C were 87±39 and 111±43mg/dL, respectively.
- Lp(a) was abnormal by reference lab standards (≥30mg/dL) in 46 cases (48%).
- Among those with LDL-C ≤70 and non-HDL-C ≤100mg/dL, Lp(a) was elevated in 14 of 32 subjects (44%), 12/14 (86%) of which were significantly elevated (≥50mg/dL).
- In the known follow-up of subjects with elevated Lp(a) ≥30mg/dL, 16/28 (57%) received intensification of therapy (e.g. increased statin, addition of ezetimibe or PCSK9 inhibitor). Reference values were adapted from a large-scale prescriptive data. [Arch Intern Med. 2008]

Conclusions

- Assessment of the clinical utility of Lp(a) in our cohort of patients shows promise for Lp(a) use above and beyond traditional risk factors for guidance in medical therapy.
- Our findings suggest:
  1. a 48% prevalence of elevated Lp(a) in this high-risk population
  2. that while 34% of patients' LDL and non-HDL were ≤70 and ≤100mg/dL respectively, 44% of those same patients had elevated Lp(a), suggesting residual risk
  3. that knowledge of an elevated Lp(a) may provide information beyond LDL and non-HDL to aid decision-making in lipid-lowering treatment intensification, as was seen in 57% of patients in this series.
- Long-term studies demonstrating that a reduction in Lp(a) improves cardiovascular outcomes are needed. Until then we suggest adoption of Lp(a) screening as a decision-aid tool in intensification of therapy in high-risk patients and as a reason to screen family members when positive.

References

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