Serial Lipoprotein(a) Measurements in Heart Transplant Recipients

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Introduction: Lipoprotein (a), a genetically determined lipid carrier molecule consists of low-density lipoprotein with apolipoprotein B-100 and apolipoprotein (a), is a well-established biomarker with casual association to development of atherosclerotic cardiovascular disease (ASCVD) events.[1] ACC/AHA guidelines recommend lipid lowering therapy for Lp(a) level >50 mg/dL,[2] as Lp(a) has a positive association with intima thickening leading to coronary allograft vasculopathy (CAV).[3-4] The following case demonstrates the utility in serial Lp(a) level as a critical biomarker for CAV development in conjunction with surveillance cardiac catheterization.

Case: A 49-year-old man with T2DM, Stage 4 CKD, hyperlipidemia, and a history of end stage ischemic cardiomyopathy with HeartMate 3 LVAD device as a bridge to orthotopic heart transplant (OHT, month 0). His A1c was 8.4% at the time of OHT. Lipoprotein (a) level was 113 mg/dL (Month -10) pre-OHT while on high intensity statin. Aggressive management of his baseline risk factors was continued post-transplant. Maximal intimal thickness (MIT) at post-transplant year 1 (Month 12) catheterization demonstrated left main (LM) 0.23mm, proximal LAD (pLAD) 0.25mm, and mid-LAD 0.22mm; at year 2 (Month 24): LM 0.31 mm, pLAD 0.28mm, and mid-LAD 0.24mm. Both surveillance catheterization showed no angiographically significant CAV with ISHLT class 0 and normal LVEDP. Graft function remained intact (Month 23) and post-transplant Lp(a) level was 37 mg/dL (Month 26).

Discussion: Based on ISHLT classification, CAV may be detected with angiography in 8% of HTx recipients within first year post-transplant, 32% in the first 5 years, and 43% in the first 8 years.[5] Current data on role on Lp(a) utility as a surveillance biomarker in CAV remain scarce. One recent study of 150 HTx patients at a tertiary center observed that Lp(a) $\geq$ 30 mg/dL portends a higher risk of early CAV development.[3] Our case demonstrates a significant decrease in post-transplant Lp(a) level with aggressive management of cardiovascular risk factors, and a favorable clinical outcome with MIT < 0.5mm on both surveillance angiography. However, it is crucial to acknowledge that immunosuppressive agents have been observed to decrease serum Lp(a) level.[6-7]

Conclusion: Lp(a) is a highly prognostic biomarker that informs the risk of future cardiovascular events. In addition to surveillance angiography, we support serial assessment of Lp(a) level to assess for CAV development, and risk stratify for further interventions among cardiac transplant recipients.

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