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**A Silent Culprit: The Role of Severe Lipoprotein(a)
Elevation in Thoracic Aortic Aneurysm Progression
and Surgical Outcome in a 61-Year-Old Male**

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Presentation of Case

A 61-year-old African American male with a history of ascending thoracic aortic aneurysm admitted to hospital for aortic aneurysm repair.

Referent is retired military, under the care of VA medical center, however, he was referred secondary to resistant hypertension with systolic blood pressure averaging 160-170. In 2005, he was diagnosed with dyslipidemia, HTN, and an ascending thoracic aortic aneurysm that was being monitored with yearly CT. Past medical history also includes sleep apnea diagnosed in 2014, thyroid goiter diagnosed in 2020, and heavy alcohol use (x4 bourbons daily).

The patient has none of the following cardiovascular risk factors: cigarette smoking, diabetes, and obesity. The patient's LP(a) levels were 600 mg/dL despite the lipid panel revealing the lipids being well controlled with Atorvastatin 80 mg. The results of the lipid panel showed low-density lipoprotein cholesterol (LDL-C) level of 56 mg/dL, Triglyceride level of 134 mg/dL, Total Cholesterol level of 130 mg/dL, and high-density lipoprotein cholesterol (HDL-C) level of 47.

Additional work up included CT thorax/aorta and echocardiogram was ordered. The CT confirmed a 5.2 cm ascending thoracic aortic aneurysm. The echo confirmed mild LVH, mild tricuspid regurgitation, moderate mitral regurgitation, biatrial enlargement, and the 5.2 cm ascending aortic aneurysm. Cardiac Cath was performed and revealed significant CAD but confirmed the thoracic aortic aneurysm

The patient was referred to Cardiothoracic Surgery for Ascending Aortic Aneurysm Repair and a transesophageal echocardiogram (TEE) was ordered. The TEE indicated an enlargement of the aortic aneurysm to 5.8 cm and surgery was scheduled.

An Ascending Aortic Aneurysm Repair with Valve Sparing Root Florida Sleeve procedure was performed. Mitral Valve repair and Ligation of the Left Atrial Appendage was also performed. Post-op complications included small bilateral pleural effusions with dependent consolidation/atelectasis of both lower lobes and an episode of paroxysmal atrial fibrillation that converted to normal sinus rhythm. After 7 days of hospitalization, the patient was discharged with a restarted Apixaban 5 mg BID, Amiodarone 200 mg daily, Carvedilol 12.5 BID, and continued blood pressure medication as before.

Discussion

Lipoprotein(a) (Lp(a)) is likely a causal independent risk factor for cardiovascular disease. The risk of atherosclerotic cardiovascular disease (ASCVD) increases linearly with Lp(a) concentrations. ASCVD risk increases are clinically relevant as Lp(a) concentrations exceed 30 to 50 mg/dL and especially when then exceed 180 mg/dL.

Elevated Lp(a) levels can be a sensitive and specific marker of vascular disease severity. Currently, there are no dedicated treatment methods for treating patients with a history of vascular disease and severely elevated

Lp(a). Despite the lack of treatment currently available, incorporating Lp(a) into treatment plans can help identify high-risk patients who might not be flagged by traditional lipid markers. While current therapies do not directly target Lp(a), emerging treatments currently in Phase 3 trials are showing promise in lowering its levels. As shown in phase 1 & 2. Awareness of a patient's increased risk of ASCVD due to elevated Lp(a) enables more personalized interventions and family screening for potentially reduces the risk of cardiovascular events. Early identification of elevated Lp(a) is essential for proactively informing patients and family about their heightened cardiovascular risk and should be a routine consideration in risk assessment for all individuals.

Conclusion: Elevated LP(a) level is a significant cardiovascular risk factor including risk for thoracic aortic aneurysm. Lp(a) is a genetic inherited risk factor and must be checked in all patients at one time in their life. Family screening must be done for all related patients with elevated Lp(a). Rx for reducing Lp(a) is currently in the pipeline in phase 3 trial, keep tuned.

References

1. "Lipoprotein(a)." UpToDate, Wolters Kluwer, <https://www.uptodate.com/contents/lipoprotein-a>. Accessed 25 Sept. 2024.
2. Lopez-Mattei, Juan-Carlos, et al. "Lipoprotein(a) as a Novel Risk Factor for Cardiovascular Disease: Insights From Emerging Research." *Frontiers in Medicine*, vol. 10, 2023, <https://www.frontiersin.org/articles/10.3389/fmed.2023.1190446/full>. Accessed 25 Sept. 2024.
3. Alhomoud, Mohammed A., et al. "Role of Lipoprotein(a) in Atherosclerotic Cardiovascular Disease: A Review of Current and Emerging Therapies." *Pharmacotherapy: The Journal of Human Pharmacology and Drug Therapy*, vol. 43, no. 7, 2023, <https://doi.org/10.1002/phar.2851>. Accessed 25 Sept. 2024.