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Symptomatic Lambl's Excrescence Presenting as Transient Ischemic Attack

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Abstract

Transient ischemic attack (TIA) is a common neurologic event, often secondary to cardiovascular pathology. Lambl's excrescences (LEs) are rare, filiform, non-neoplastic structures most frequently found on left-sided heart valves and typically identified incidentally. Their causal relationship with embolic phenomena remains controversial due to limited high-quality evidence and the absence of formal management guidelines. We present the case of a 64-year-old woman with symptomatic LE diagnosed by transesophageal echocardiogram, manifesting as TIA and a negative workup for other causes, including a 12-month follow-up with a loop recorder. Also, we discuss diagnostic considerations and management strategies based on current literature.

Keywords

Transient Ischemic Attack (TIA), Lambl's Excrescence, Echocardiography

1. Introduction

Lambl's excrescences (LEs) are delicate, mobile, endothelial-covered filaments arising along the coaptation lines of cardiac valves, most often the aortic and mitral valves [1]. Although they were described by Vilém Dušan Lambl as early as the 19th century, their clinical significance is still a subject of ongoing research [2]. These structures were initially described as incidental findings in echocardiographic studies and are often thought to arise from degenerative changes or mechanical stress on the valve endocardial surfaces, usually at the coaptation margins [3]. Initially thought to be an incidental finding with no clinical significance, LEs have been increasingly recognized in the context of embolic stroke, particularly in patients without other obvious sources of emboli [2] [4]. Their detection has become

more frequent with advances in imaging techniques, particularly trans-esophageal echocardiography (TEE), which allows for detailed visualization of these structures.

2. Case History/Examination

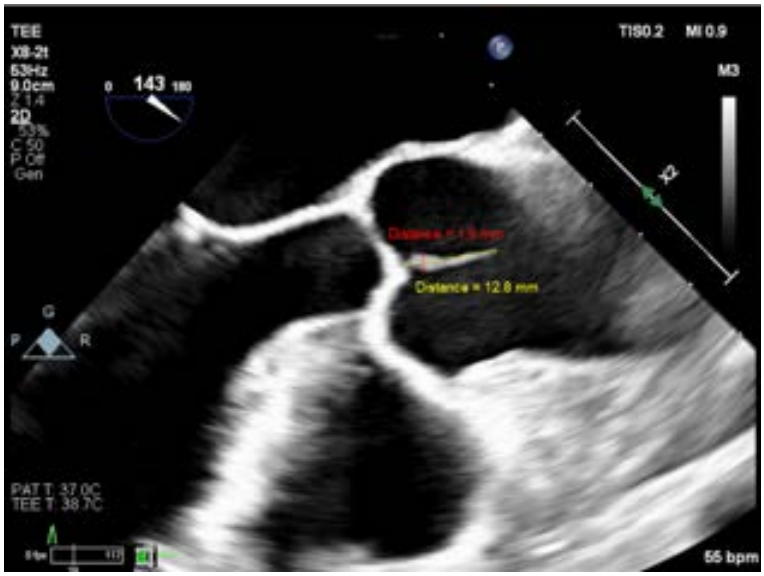
A 64-year-old Caucasian woman with a history significant for hyperlipidemia, hypothyroidism, celiac disease, and smoking presented to the emergency room with a witnessed episode of transient right upper arm paresis and monocular transient loss of vision in the right eye, lasting for approximately 3 minutes. There were no associated dysarthria, vertigo, or auditory symptoms. Her vital signs were within normal limits, and a physical examination in the emergency room was without any abnormality.

2.1. Differential Diagnosis, Investigations, and Treatment

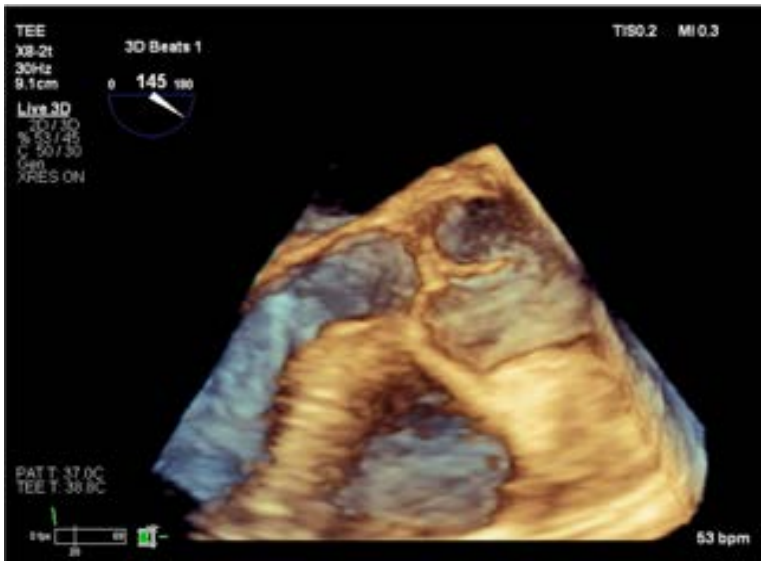
Laboratory findings included elevated total cholesterol at 310 mg/dL, LDL at 235 mg/dL, normal white cell count, and a TSH of 34.4 μIU/mL. Subsequently, she was admitted to the hospital under continuous cardiac monitoring, which was significant for occasional monomorphic premature ventricular contractions (PVCs) but no other arrhythmia. A CT (Computer tomography) angiogram of the head and neck was performed, which revealed the absence of significant stenosis of the carotid arteries and mild chronic small vessel ischemic changes. MRI (Magnetic Resonance Imaging) of the brain was performed and did not show any evidence of infarction. A transthoracic echocardiogram (TTE) revealed preserved left ventricular function, absent vegetations or thrombi, and intracardiac shunts on a bubble study. A TEE was pursued which confirmed the TTE findings with absence of vegetation, intracardiac thrombi or shunts, but in addition revealed the presence of a mobile, thin, serpiginous structure attached to the aortic surface of the aortic valve leaflets near the coaptation line measuring 12.8 × 1.9 mm, which was diagnosed as a LE (**Figure 1(a)**/**Figure 1(b)**). There was no associated valvular dysfunction. Blood cultures and white cell count were normal, and there was no sign of infection to suspect infective endocarditis. The ascending aorta did not reveal any significant atherosclerotic plaque. D-dimer was below the age-appropriate cutoff. During hospitalization, there were no recurrent events, and telemetry did not show atrial flutter or fibrillation. She was discharged on low-dose aspirin, high-intensity statin, and levothyroxine, with an implanted loop recorder.

2.2. Outcome and Follow-Up

A 12-month loop recorder monitoring revealed sinus rhythm with episodes of premature atrial contractions (PACs) and premature ventricular contractions (PVCs) (both less than 1%) and absence of sustained atrial or ventricular arrhythmias. She underwent a hypercoagulable workup and was negative (prothrombin, factor V Leiden, protein C and S, antithrombin III, and homocysteine). She was followed up with cardiology regularly every 6 months to assess her cardiac status



(a)



(b)

Figure 1. (a) Trans-esophageal echocardiography (TEE) showing aortic valve and filamentous structure consistent with Lambl's excrescences; (b) Trans-esophageal echocardiography (TEE) showing aortic valve and Lambl's excrescences in 3D.

and to monitor the effectiveness of the prescribed therapy. Transthoracic echocardiogram was not repeated on follow-up since there was no symptom recurrence.

3. Discussion

LEs are small, filiform projections found on heart valves, typically discovered incidentally during echocardiographic or autopsy studies. They are most commonly seen to arise on the aortic or mitral valve but may be seen on right-sided valves as well [5]. Histologically, they are composed of a core of connective tissue (collagen and elastin), covered by a layer of endothelial cells [6]. Pathophysiologically, their

origin is not well defined but believed to be initiated by endothelial damage to the heart valves from the shear stress due to blood flow, with resultant endothelial tear, followed by fibrin deposition, microthrombi, and endothelial growth [7]. They tend to develop in the Nodulus Arantius and the free margin of the aortic cusp [6]. Embolization of the small fragments or microthrombi can cause TIA or stroke. Another hypothesis is that it can act as a nidus for platelet aggregation and clot formation. Often seen as benign, they have been linked to embolic events by association, although there is a lack of strong evidence to show causality. Retrospective studies showed a higher prevalence of LEs in stroke/TIA patients compared with controls, but prospective data are less supportive [1] [8]. However, in contrast, there are case reports and case series showing an association between LEs and stroke [2] [4] [9].

Differentiation from papillary fibroelastomas (PFEs) and infective vegetations is critical, as management differs. PFEs tend to be bulkier and frond-like morphologically, in contrast to the thin and filamentous structure of LEs, and arise away from the valve leaflet coaptation lines, although there can rarely be giant LEs (>2 cm), making this differentiation difficult solely on TEE. Infective vegetations are irregular non-filamentous masses with variable echogenicity and are usually accompanied by valvular dysfunction, which is in contrast to LE, which does not affect valve function. Echocardiography is the initial test of choice for interrogating cardiac valves due to its low cost, wide availability, and temporal and spatial discrimination. TEE offers superior spatial resolution for detecting these slender lesions, which are often missed on transthoracic studies [10].

There are no consensus guidelines for the management of LEs. The three available options in their management include observation, antiplatelet medications (single or combined antiplatelet therapy)/anticoagulation, and surgical resection [11] [12]. The choice of therapy is dictated by the presence of symptoms, surgical risk, and ongoing treatment. Asymptomatic patients are usually observed. For symptomatic patients without an alternative embolic source, initial single antiplatelet therapy is reasonable. Escalation to dual antiplatelet therapy, anticoagulation, or surgical excision may be considered for recurrent events [13]-[15]. In our patient, single antiplatelet therapy was chosen given the transient, isolated episode, absence of arrhythmia, and lack of recurrent symptoms. In contrast, if the patient had recurrent episodes or a large mobile valvular lesion, we would have considered anticoagulation or surgical intervention.

After conducting a thorough workup and ruling out other possible causes of TIA, including carotid artery disease, atrial fibrillation on monitoring, intracardiac shunt, and significant aortic atheroma, Lambl's excrescence was considered to be the most probable etiology. A definite relationship between TIA and LEs has not been established, and the association is mainly clinical.

4. Conclusion

Lambl's excrescences, while often incidental, may be implicated in embolic events,

particularly when other sources are excluded. In our patient, an extensive diagnostic workup did not identify an alternative embolic source; therefore, Lambl's was considered the most likely etiology by exclusion. Management should be individualized, balancing recurrence risk against the risks of anticoagulation or surgery. Further prospective studies are needed to clarify their clinical significance and inform guidelines.

Authors' Contributions

Each author contributed equally and substantially in the conception, write-up, revision, and final proofreading of this manuscript.

Ethical Statement

This is a case report with no identifiable patient information. Written informed consent was obtained from the patient.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Roldan, C.A., Shively, B.K. and Crawford, M.H. (1997) Valve Excrescences: Prevalence, Evolution and Risk for Cardioembolism. *Journal of the American College of Cardiology*, **30**, 1308-1314. https://doi.org/10.1016/s0735-1097(97)00315-x

[2] Shrestha, B., Pokhrel, A., Oke, I., Paudel, A., Timlisina, B., Parajuli, P., *et al.* (2022) Lambl's Excrescences Associated with Cardioembolic Stroke. *American Journal of Case Reports*, **23**, e934859. https://doi.org/10.12659/ajcr.934859

[3] Aziz, F. and Baciewicz Jr., F.A. (2007) Lambl's Excrescences: Review and Recommendations. *Texas Heart Institute Journal*, **34**, 366-368.

[4] Freedberg, R.S., Goodkin, G.M., Perez, J.L., Tunick, P.A. and Kronzon, I. (1995) Valve Strands Are Strongly Associated with Systemic Embolization: A Transesophageal Echocardiographic Study. *Circulation*, **92**, 495-498.

[5] Voros, S., Nanda, N.C., Thakur, A.C., Narayan, V.K. and Samal, A.K. (1999) Lambl's Excrescences Involving the Pulmonary Valve Detected by Transesophageal Echocardiography. *Echocardiography*, **16**, 35-39. https://doi.org/10.1111/j.1540-8175.1999.tb00783.x

[6] Hurle, J.M., Garcia-Martinez, V. and Sanchez-Quintana, D. (1986) Morphologic Characteristics and Structure of Surface Excrescences (Lambi's Excrescences) in the Normal Aortic Valve. *The American Journal of Cardiology*, **58**, 1223-1227. https://doi.org/10.1016/0002-9149(86)90386-3

[7] Goyal, A., Layman, A.J., Bois, M.C. and Maleszewski, J.J. (2024) Characteristics of Lambl Excrescences of Aortic and Pulmonary Valves in Healthy Hearts. *Cardiovascular Pathology*, **68**, 107588. https://doi.org/10.1016/j.carpath.2023.107588

[8] Cammalleri, V., Idone, G., Cosma, J., Marino, M.M., Mauceri, A., Stifano, G., *et al.* (2022) Lambl's Excrescence in Transcatheter Aortic Valve Implantation: Prevalence and Risk of Embolic Events. *Minerva Cardiology and Angiology*, **70**, 8-15. https://doi.org/10.23736/s2724-5683.20.05482-1

[9] Cohen, A., Tzourio, C., Chauvel, C., Bertrand, B., Crassard, I., Bernard, Y., *et al.* (1997) Mitral Valve Strands and the Risk of Ischemic Stroke in Elderly Patients. *Stroke*, **28**, 1574-1578. https://doi.org/10.1161/01.str.28.8.1574

[10] Zaragosa-Macias, E., Chen, M.A. and Gill, E.A. (2012) Real Time Three-Dimensional Echocardiography Evaluation of Intracardiac Masses. *Echocardiography*, **29**, 207-219. https://doi.org/10.1111/j.1540-8175.2011.01627.x

[11] Alajjuri, M.A., Alajjuri, O.A. and Alani, F.R. (2023) Lambl's Excrescence as an Etiology of Thromboembolism: Case Report and Literature Review. *Clinical Medicine Insights*: *Case Reports*, **16**. https://doi.org/10.1177/11795476231171391

[12] Kalavakunta, J.K., Bantu, V., Tokala, H. and Kodenchery, M. (2010) Sudden Cause of Cardiac Death—Be Aware of Me: A Case Report and Short Review on Brugada Syndrome. *Case Reports in Medicine*, **2010**, Article ID: 823490. https://doi.org/10.1155/2010/823490

[13] Ammannaya, G.K.K. (2019) Lambl's Excrescences: Current Diagnosis and Management. *Cardiology Research*, **10**, 207-210. https://doi.org/10.14740/cr892

[14] Rao, R., Salvatierra, J.F., Nasri, R., Gosal, H. and Ahmad, E. (2024) Lambl's Excrescences in a Patient with Transient Ischemic Attack. *Chest*, **166**, A443. https://doi.org/10.1016/j.chest.2024.06.317

[15] Kondamareddy, D. and Masood, W. (2023) Lambl Excrescences. StatPearls.