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# Recurrent Cardio-embolic Cerebrovascular Accidents due to Lambl's Excrescences?

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**Abstract** Cerebrovascular accidents (strokes) are leading cause of morbidity and mortality in the United States of America. Cardioembolic strokes account for one-third of cerebrovascular accidents. Lambl's excrescences are filiform projections on the native cardiac valves; they are attributed as a rare cause of cardioembolic strokes. They are a result of endothelial damage secondary to valve wear and tear. Lambl's excrescences as a cause of cardioembolic stroke is debated. Lambl's excrescences are a frequent appearance on echocardiography. We here present a case of recurrent cardioembolic strokes in whom lambl's excrescences on aortic valves were detected on transesophageal echocardiography. Previous transthoracic echocardiography in the patient had not revealed lambl's excrescences owing to their low sensitivity. Patient refused aortic valve replacement and medical management of cerebrovascular accident was continued.

Keywords: Lambl's excrescences, aortic valve, transthoracic echocardiography, cardioembolic stroke

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## 1. Introduction

Cerebrovascular accident (CVA) remains one of the leading causes of morbidity and mortality in the United States of America. Cardioembolic stroke accounts for nearly one-third of all ischemic strokes. Lambl's excrescences (LE) are a rare cardiac valvular abnormality whose role in cardio-embolic stroke is still debated. Lambl's excrescences are detected by echocardiography; transesophageal echocardiography being more sensitive to identifying lambl's excrescences when compared to transthoracic echocardiography. We present a patient with multiple episodes of cerebrovascular accidents in whom lambl's excrescences was identified on transesophageal echocardiography.

### 2. Clinical Observation

A 62-year-old lady with a past medical history of hypertension, multiple cerebrovascular accidents with right sided residual weakness, osteoarthritis and chronic obstructive pulmonary disease (COPD) presented with sudden onset left sided weakness that she developed two days before presentation. Here weakness was associated with a headache that got relieved by taking aspirin. Examination revealed a decrease in muscle power of left upper and left lower extremity; findings were consistent with upper motor neuron lesion.

The patient had the first episode of cerebrovascular accident nine months before present presentation, computed tomography of the head obtained during the episode revealed a tiny area of intermediate age infarct in the left frontal region; no stenosis in the carotid or intracranial arteries was noted. The patient had the second episode of cerebrovascular accident seven months before the current presentation during which magnetic resonance imaging of the head revealed subacute ischemic infarction within left middle cerebral artery territory; multiple predominantly cortical based foci within the periphery of left high frontal and posterior parietal lobes was noted. Moderate narrowing of left carotid siphon with normal head and neck arterial circulation was observed on magnetic resonance angiogram. Computed tomography of the head obtained during the present admission revealed an area of hypoattenuation in the right upper parietal lobe, representing acute or subacute ischemia of middle cerebral artery territory. A magnetic resonance imaging of the head revealed several sub-centimeter foci of diffusion restriction consistent in the superior right parietal lobe consistent with acute distal segment right MCA infarcts.

Transthoracic echocardiography (TTE) obtained during the first episode of the cerebrovascular accident revealed trace tricuspid regurgitation, an elevated systolic pulmonary artery pressure (30 mm Hg) and normal ejection fraction; a repeat TTE during the second and present episode of CVA failed to add new findings. Due to recurrent episodes of CVA, a transesophageal echocardiography obtained revealed lambl's excrescences

on the aortic valve (Figure 1 and Figure 2). The patient was referred to rehabilitative medical services with daily

aspirin and high dose statin therapy.

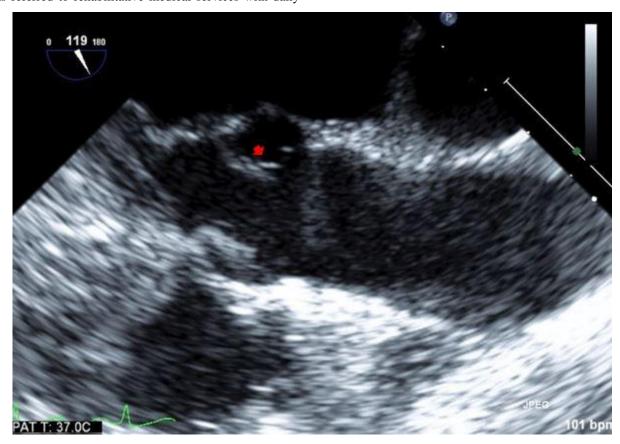


Figure 1. Lambles's Excresences (indicated by red arrow) noted on aortic valve as noted in transesophageal echocardiography

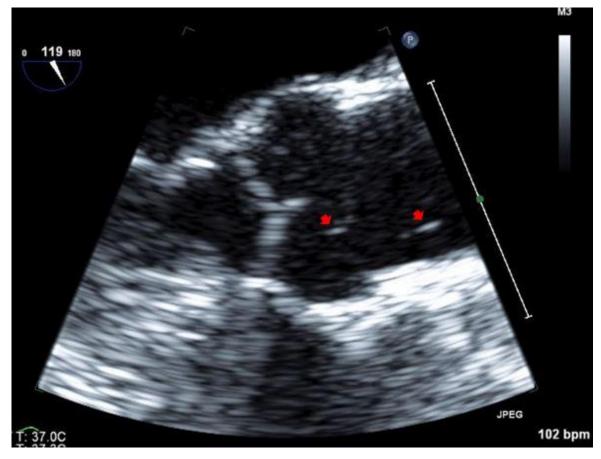


Figure 2. Multiple lambls's excresences (indicated by red arrow) noted on aortic valve of the patient. The filiform nature and the narrow base of lambl's excresences are evident on the Figure

#### 3. Discussion

Cerebrovascular accidents are a leading cause of morbidity and mortality in the United States; 795,000 incidences of new or recurrent stroke (ischemic and hemorrhagic) are reported every year [1]. Recurrent cerebral infarcts have poor outcomes when compared to the first stroke. The 3-year risk of recurrent stroke varies from 6% to 25%. However, it is not clear if the risk of recurrent stroke is different in an ischemic and hemorrhagic stroke [2]. Cardioembolic stroke accounts for 14 to 30% of ischemic stroke, with the in-hospital mortality rate of 27.3%. In the absence contraindications, immediate anticoagulation is strongly advised to prevent recurrent cardioembolic stroke [3]. Etiology of cardio-embolic strokes can be divided into the basic groups i) cardiac valves and chamber abnormalities ii) valve disorders iii) arrhythmias [4,5].

Lambl's excrescences (LE) were first described by Vilem Dusan Lambl, a Bohemian physician in 1856 [6]. LE are 1 mm broad, 4-10 mm long, mobile, filiform projections from native cardiac valves, that show undulating independent motion. They're most commonly seen on mitral valve and aortic valve, especially at the site of valve closure. They usually branch and have a narrow base [7,8]. Endothelial damage secondary to valve wear and tear has been attributed to their location at the site of valve closure [9]. Pathological examination of LE revealed that they are composed of connective tissue lined by a single layer of endothelial cells [10].

LE are reported in 22% of the patients with suspected cardioembolic stroke who undergo transesophageal echocardiography [11]. Despite this high prevalence, the incidence of cardioembolic stroke due to LE per se is not known possibly due to under-recognition of the condition [12]. The exact mechanism of embolism from LE is yet to be understood [19]. Embolization can happen from a thrombus that originated from LE or by a fragment of LE per se. A transesophageal echo is more sensitive in identifying LE when compared to transthoracic echo, mild light computed tomography is another alternate [7,15]. Embolization's from LE have been attributed to causing migraine [12], occlusion of the coronary artery [13], right renal artery occlusion [14] and popliteal artery embolization [16].

The differential diagnosis for flapping structures on cardiac valves includes imaging artifact, vegetation, thrombus, redundant leaflet, flap due to an aortic dissection, papillary fibroelastoma, and LE. It could be particularly difficult to differentiate between LE and papillary fibroelastoma's [8]. Papillary fibroelastoma's are small pedunculated jellylike mass, homogeneous, much demonstrated mobile mass attached by a short stalk; the stalk is often broad-based. Papillary fibroelastoma's are often found on mechanically less affected parts of valves and other areas of the endocardium.

Roldan et al. concluded that valve excrescences are a common finding and did not appear to be the primary source of cardioembolic irrespective of gender and age [18]. Wolf et al. recommend antiplatelet agents after first CVA and anticoagulation or cardiothoracic intervention for recurrent CVA's from LE [19]. Homma et al. also concluded that valvular stands are unlikely sources of

ischemic stroke; further, they did not find the benefit of initiation of aspirin or warfarin therapy for stroke prevention in patients with LE [20]. More recently, Aziz et al. recommend anticoagulation following a stroke and surgical accession of LE following the second stroke. Contrary to the previous recommendations of others Aziz et al. at all recommend close monitoring of asymptomatic LE patients [21].

LE remains a possible cause of cardioembolic stroke. Individualization and characterization of personal risk factors are to be made before initiation of anticoagulation or consideration of surgical options following CVA in a patient with LE. Most patients with the LE are unlikely to benefit from aspirin or warfarin therapy for stroke prevention.

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