



Multiple cardioembolic attacks with a huge ventricular thrombus in dilated cardiomyopathy: A case report and comprehensive review

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Abstract

Left ventricular thrombus is one of the major complications of dilated cardiomyopathy. Although the presence of a cardiac thrombus is a major risk factor for embolization, several probable conditions, the connection of which is not thoroughly studied, such as gout disease and methamphetamine abuse, are also possible causes. We present the case report of a male in his early 40s with a history of alcohol and methamphetamine abuse, gout, and dilated cardiomyopathy, experiencing multiple ischemic attacks, including acute limb ischemia, dysarthria, and renal infarct. Echocardiography revealed a large clot in the left ventricle. The patient received heparin at a rate of 1000 units/hour, followed by warfarin at 10 mg/day, and probenecid at 500 mg/day. The thrombus resolved upon anticoagulant therapy, with improvement in the patient's symptoms.

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Left ventricular thrombus formation in dilated cardiomyopathy is influenced by many factors, including drug toxicity (for instance, methamphetamine), uric acid levels, and N-terminal precursor B-type natriuretic peptide. The thrombus can cause cardioembolic attacks in multiple sites, and the choice for emergent therapy is an oral anticoagulant, which may improve the ejection fraction. However, certain aspects related to cardiac thrombosis and embolization risk still warrant more comprehensive studies.

Keywords

Dilated cardiomyopathy, thrombosis, transient ischemic attack, limb ischemia, methamphetamine, gout

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Introduction

Dilated cardiomyopathy (DCM), also known as non-ischemic cardiomyopathy, includes a range of myocardial complications with various etiologies resulting in the dilation of the left ventricle or both ventricles of the heart with systolic dysfunction, usually defined as a left ventricle ejection fraction (LVEF) of less than 40%. Most of the DCM cases are idiopathic, while the remaining can be a consequence of an infiltrative disease, coronary artery disease, alcohol abuse, medications, Takotsubo syndrome, and pregnancy. DCM affects men more than women, and its prevalence in the general population is 40 cases per 100,000. DCM has a high mortality rate and is a leading cause of heart transplantation.^{1,2}

In DCM, left ventricular (LV) thrombus formation occurs owing to stasis, with a reported prevalence of up to 36% for LV thrombus and a risk of 11% for systemic embolization.³ We report a case of a male with DCM who experienced multiple simultaneous ischemic attacks in the brain, lower limb, and kidney due to a large LV thrombus. The patient had a history of alcohol and drug abuse and uncontrolled gout. This study aimed to discuss the associated risk factors, including drug abuse or high uric acid levels, locations, characteristics

of the cardiac thrombus and emboli, and the chosen therapy. We also review the existing literature.

Methods

We reviewed the clinical features of a patient with multiple ischemic attacks and LV thrombus who had DCM, with a history of drug abuse and uncontrolled gout. The study was conducted in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. Moreover, the patient provided written informed consent for treatment. We adhered to the CARE reporting guidelines⁴ and de-identified all patient details.

We also performed a review of literature that was conducted using PubMed database searches with keywords that included the MeSH terms “Thrombosis” and “Dilated Cardiomyopathy.” The time frame was limited to all English articles from 2000 to the present year. After screening the titles and abstracts of case report studies, we included nine case reports documenting LV thrombus in adults with non-ischemic DCM. The exclusion criteria included venous thrombosis, coagulation disorders, and a history of cardiac procedures (Table 1).

Table 1. Summary of the left ventricular thrombus in dilated cardiomyopathy cases presented in the literature.

Author, year	Age, sex	Medical history	Main symptoms	Imaging	Thrombus location, size, number	Ejection fraction	Treatment	The result of the treatment
Nishi et al. ¹⁰ 2002	23, F	Arterial septal defect treated by surgery at age 5 years	Palpitations, nocturnal dyspnea, and orthopnea 6 weeks after a normal pregnancy	1. CXR: cardiomegaly. 2. Echocardiography: left ventricular dilation 3. ECG: right axis deviation, negative T wave changes in leads I, aVL, and V2–6, and QT prolongation (QTc 0.47 s)	Multiple thrombi in the apex of both ventricles, 10 mm in diameter. N/A	33%	Furosemide and dobutamine for HF IV heparin for biventricular thrombi	Thrombi disappeared 4 days after heparin therapy
Manikkan et al. ¹¹ 2008	26, F	Smoker, pituitary macroadenoma, Miscarriage, Pregnancy 5 months before symptoms	Epigastric and left upper quadrant pain, nausea, and vomiting	1. CXR: cardiomegaly, bilateral pleural effusion 2. ECG: non-specific T wave changes, sinus tachycardia 3. CT scan of the abdomen and pelvis: 3 × 1.8 cm splenic infarct 4. CT angiogram of the chest: no sign of pulmonary embolism 5. Echocardiography: dilated left ventricle	Left ventricle, The biggest one was 1 cm, 4	10%–15%	Diuretics, beta-blockers, angiotensin-converting enzyme inhibitors, warfarin	After 24 hours, the symptoms disappeared
Lo et al. ¹³ 2007	34, M	Alcohol and amphetamine abuse	Abdominal pain, right leg numbness	1. CXR: cardiomegaly 2. CT: multiple infarcts in the spleen and both kidneys, a filling defect in the right femoral artery 3. 2-dimensional echocardiography: 3 thrombi in the apex of the left ventricle 4. Real-time 3-dimensional echocardiography: 4 separate thrombi, 3 of which were connected	The apex of the left ventricle, N/A, 4	17%	Emergency embolectomy on the right leg, IV heparin and oral warfarin	After 8 weeks, the thrombi disappeared, and left ventricular function improved (EF = 46%)
Ho et al. ²⁷ 2010	34, M	There was no medical history except flu-like symptoms 2 months before the visit	General tonic colonic seizure, confusion, and somnolence	1. CXR: cardiomegaly 2. ECG: Left ventricle hypertrophy 3. Non-enhanced CT: focal edema over the right parietal-temporal lobe 4. Brain MRI: multiple acute brain	The apex of the left ventricle, 1 × 4 cm, 1	15%–20%	IV heparin, aspirin 100 mg daily, and oral warfarin	Improved EF and resolution of the thrombus (EF = 40%) During the 6-month follow-up, EF reached 46%

(continued)

Table 1. Continued.

Author, year	Age, sex	Medical history	Main symptoms	Imaging	Thrombus location, size, number	Ejection fraction	Treatment	The result of the treatment
Jeon et al. ²⁶ 2011	40, M	Orthopedic surgery on both legs a year before presentation	Dyspnea, right facial and limb weakness, and both lower limb pain	<p>infarctions over the right frontal and temporal lobes; bilateral parietal and occipital, as well as the right cerebellum</p> <p>4. Magnetic resonance angiography: the following signal distal to the left P3 was not seen</p> <p>5. TTE: dilated LV and left atrium, a mass measured 1 × 4 cm in the apex of the left ventricle</p> <p>1. CXR: mild cardiomegaly</p> <p>2. ECG: non-specific ST segmental changes</p> <p>3. TTE and TEE: left ventricle dilation and mobile hyperchoic pedunculated mass in the apex of the left ventricle</p> <p>4. Brain MRI: spotty ischemic brain lesions</p> <p>5. CT angiography: occlusion of right femoral artery, right popliteal artery, and left popliteal artery</p> <p>6. Coronary angiography: insignificant stenosis (5%) on the right coronary artery</p>	The apex of the left ventricle, 4.3 × 4.2 cm, 1	19%	Surgical removal of the cardiac mass via mitral valve approach, Low molecular weight heparin, embolectomy for the lower limbs	At 1-year follow-up, there was no sign of thrombus, and left ventricle EF reached 59%
				<p>1. CXR: cardiomegaly and pulmonary venous congestion</p> <p>2. ECG: sinus tachycardia</p> <p>3. TTE: biventricular dilation, biatrial enlargement, global hypokinesis of the left ventricle, multiple echo-dense masses in the left ventricle</p>				
Altuwaijri et al. ⁹ 2012	24, F	Worsening dyspnea, orthopnea, and paroxysmal nocturnal dyspnea 7 months post-partum	Dyspnea, limb edema		The lateral wall of the left ventricle, N/A, N/A	Less than 20%	Diuretic, beta blocker, angiotensin-converting enzyme inhibitor, and IV heparin	Thrombi disappeared on the cardiac magnetic resonance 4 days after treatment

(continued)

Table 1. Continued.

Author, year	Age, sex	Medical history	Main symptoms	Imaging	Thrombus location, size, number	Ejection fraction	Treatment	The result of the treatment
Kim et al. ²⁴ 2014	20, M	No medical history was reported. During the current admission, a pituitary adenoma leading to Cushing syndrome was found	Acute shortness of breath	1. CXR: cardiomegaly 2. TTE: enlargement of the left ventricle with global hypokinesia, two mobile masses located in the apex of the left ventricle 3. Brain MRI: 1-cm pituitary adenoma	Apex of the LV, N/A, N/A	20%	Diuretic agents, inotropic agents, an angiotensin-converting enzyme inhibitor, and aspirin, IV heparin, trans-sphenoidal adenectomy	The resolution of thrombi after 8 days was obvious in the TTE
Mine et al. ⁸ 2014	67, M	Mild diabetes mellitus and heart failure due to dilated cardiomyopathy, which the therapy was started a day before presentation	Left-limb paresis and left visual field defect	1. CXR: cardiomegaly 2. ECG: LV hypertrophy, premature ventricular contractions 3. Brain MRI: multiple cerebral infarctions in the occipital lobe, corona radiata and left frontal lobe 4. Echocardiography: LVED diameter of 66 mm and two mobile thrombi at the basal septum and apical inferior portions of the left ventricle	Basal septum and apical inferior portions of the left ventricle, N/A, 2	27%	Edaravone and heparin, warfarin	Paralysis improved, and thrombi disappeared 6 days after 6 days of heparin therapy
Elkowski et al. ²⁵ 2020	53, M	Dilated cardiomyopathy was diagnosed a few months earlier	Exacerbation of heart failure, palpitations	1. CXR: severe pulmonary congestion with bilateral pleural effusion 2. ECG: sinus tachycardia 3. Echocardiography: typical features of dilated cardiomyopathy and three thrombi connected with the false tendons	The apex of the left ventricle, The largest one was 2.5 × 1.8 cm, 2	25%	Anticoagulant and apixaban	Thrombi disappearance without EF improvement

CRTD: cardiac resynchronization therapy defibrillator; CT: computed tomography; CXR: chest radiography; ECG: electrocardiography; EF: ejection fraction; F: female; HF: heart failure; ICD: implantable cardiac defibrillator; IV: intravenous; LV: left ventricle; LVED: left ventricular end diastolic; M: male; MRI: magnetic resonance imaging; N/A: not available; TEE: transesophageal echocardiography; TTE: transthoracic echocardiography.

Case presentation

A Persian male in his early 40s presented with a chief complaint of severe left lower limb pain, which started two hours before the presentation, and was brought into the emergency department by ambulance. The patient reported a self-limited transient episode of dysarthria lasting for 2 hours the night before presentation, and a stage 2A claudication a month ago.

Considering the patient's medical history, he reported having poorly controlled gout for the past 15 years. The patient was a heavy smoker (50 packs/year), heavy drinker, and methamphetamine user. Additionally, he was hospitalized 6 months ago due to exertional dyspnea. Since that previous admission for exertional dyspnea, the patient has been prescribed sacubitril/valsartan 26/24mg and furosemide 40mg twice daily, eplerenone 25mg as half a tablet, and bisoprolol 2.5mg daily. He was also taking allopurinol 100mg each day. Furthermore, he quit smoking, drinking, and using drugs. He also reported a family history of myocardial infarction in his father. The LVEF was reported to be 20%–25% upon admission. Medical documents indicated no signs of LV clots but confirmed a diagnosis of non-ischemic DCM through cardiac magnetic resonance imaging (MRI).

During the physical examination, the patient was found to be afebrile. The heart rate, blood pressure, and jugular venous pressure were 119 bpm, 129/80 mm Hg, and 12 cm H₂O, respectively. Grade 2 mitral murmurs and fine crackles were detected at the base of both lungs upon examination. Upon examining the extremities, the affected limb appeared pale, cold, and tender, with a weak pulse, which was recorded as +1. Motor and sensory examinations, as well as reflexes, were normal. The patient's knee and interphalangeal joints were swollen, tender, erythematous,

and warm, exhibiting a decreased range of motion, with many visible dysmorphic tophi (Figure 1(a) and (b)).

The patient was admitted, and we immediately requested routine laboratory tests, plasma uric acid level, and synovial fluid analysis (Table 2). Electrocardiography revealed incomplete left bundle branch block, left axis deviation, intraventricular conduction delays in the inferior leads, and biatrial abnormalities.

Brain computed tomography (CT) revealed no abnormalities. Left lower limb arterial Doppler ultrasonography was also performed, revealing a monophasic waveform in the lower extremity. This pattern may indicate reduced arterial compliance or increased peripheral resistance, which is often associated with arterial disease or stenosis. For the left lower extremity, iliac artery, and abdominal aorta, CT angiography was requested but postponed due to the patient's elevated creatinine plasma levels. A transthoracic echocardiography (TTE) indicated very severe LV enlargement (LV end-diastolic volume index: 208 cc/m²) with severe systolic and moderate to severe diastolic dysfunction, LVEF: 5%–10%, global severe hypokinesia, and no LV hypertrophy. A massive (10 cm × 7 cm, 25.5 cm³) protruding LV clot was attached to the apex, anteroapical, and mid-anterior segments of the LV, with a semi-mobile portion and intrinsic liquefaction (high-risk LV thrombus). Severe left atrial enlargement, mild right atrial enlargement (LAVi: 69 cc/m², RAVi: 34 cc/m³), thickened and tethered mitral valves, and moderate mitral regurgitation were noted (Figure 1(c)). Following the TTE, anticoagulant therapy was initiated; the patient received 1000 units of heparin per hour, followed by warfarin 10 mg daily after 5 days. Additionally, owing to the elevated uric acid levels and severe arthritis, probenecid 500 mg daily was added to allopurinol. A follow-up echocardiography conducted two weeks



Figure 1. (a) Gout arthritis and multiple dysmorphic tophi in the knee and; (b) interphalangeal joints; (c) a large thrombus ($10\text{ cm} \times 7\text{ cm}$, 25.5 cm^2) pointed by the red arrow, is visible attaching to the apex of the left ventricle; (d) a decrease in the size of the left ventricular (LV) thrombus 2 weeks after the initiation of anticoagulant therapy is obvious on the follow-up transthoracic echocardiography. The arrow reveals a clot measuring $7\text{ cm} \times 3\text{ cm}$, 17.2 cm^2 ; (e, f) abdominal aorta and lower extremity computed tomography angiogram using maximum intensity projection and volume rendering techniques. Red arrows demonstrate a wedge-shaped defect in the right renal artery, and white arrows point to a significant stenosis of the left common iliac artery and (g) on the 3-month follow-up transthoracic echocardiography, the severe LV enlargement was improved, and the LV thrombus was resolved.

after the initiation of anticoagulants demonstrated a protruding LV clot ($7\text{ cm} \times 3\text{ cm}$, 17.2 cm^2), which is attached to the apex, anteroapical, and mid-anterior segments of the LV, with a semi-mobile part and multiple mobile particles (Figure 1(d)).

CT angiography was performed 3 days later, following a decrease in the creatinine levels. CT angiography revealed a thrombus involving the left common iliac artery, leading to a cut-off of the origin of the common iliac artery on the left side with

Table 2. Patient's laboratory results during hospitalization.

Variables	On admission	7 th day of admission	On discharge
White blood cell count	9800/mm ³	8700/mm ³	8800/mm ³
Red blood cell count	4.76 million/mm ³	4.9 million/mm ³	4.88 million/mm ³
Hemoglobin	12.4 g/dL	13 g/dL	13 g/dL
Creatinine	2.6 mg/dL	1.4 mg/dL	1.2 mg/dL
Sodium	144 mEq/L	144 mEq/L	142 mEq/L
Potassium	4.3 mEq/L	4.1 mEq/L	4.3 mEq/L
Uric acid	9.1 mg/dL	8.3 mg/dL	7.1 mg/dL
Erythrocyte sedimentation rate 1 hour	36 mm/h	31 mm/h	27 mm/h
C-reactive protein	52 mg/L	46 mg/L	38 mg/L
NT-proBNP	3700 pg/mL	2600 pg/mL	1700 pg/mL
Synovial fluid analysis on admission			
Color yellow			
Clarity cloudy			
Viscosity moderate			
White blood cell count 6100/mm ³			
PMN 66%			
Glucose 60 mg/dL			
Total protein 14 g/L			
Lactate 13 mmol/L			
Gram stains negative			
Culture negative			
Crystal monosodium urate crystals			

NT-proBNP: N-terminal pro-B-type natriuretic peptide; PMN: polymorphonuclear.

good runoff. An extensive wedge-shaped heterogeneous enhancement of the right kidney was also discernible, likely indicating extensive thrombosis resulting in infarction (Figures 1(e) and (f)).

The patient's leg pain diminished upon treatment. A nephrology consult was requested, and conservative treatment was recommended. Fifteen days later, the patient was discharged once he reached the therapeutic range of warfarin; he was advised to consult a vascular surgeon for iliac stenosis. At the monthly follow-up, after 3 months of warfarin therapy, the clot was resolved, and the N-terminal pro-B-type natriuretic peptide (NT-proBNP) decreased to 950 pg/mL, but the ejection fraction (EF) remained at 10%–15% (Figure 1(g)).

Heart failure treatment and warfarin therapy were recommended to be continued

for 6 months. The patient had both lower limb and coronary angiography after discharge. Coronary angiography was normal. Stenting of the left iliac artery was performed, and the patient's claudication improved completely.

Follow-up

During the monthly visit and evaluation of LV function and thrombosis with cardiac MRI, we noticed that LVEF was 35%, and no further evidence of cardiac thrombosis.

After cessation of addictive drugs and alcohol and the complete elimination of LV thrombosis and upon consultation with a neurologist and vascular surgeon, we decided to discontinue warfarin and evaluate him every month in our clinic. All symptoms improved, and the patient continued with heart failure treatment.

Discussion

DCM presenting with shortness of breath, cough, fatigue, and swelling in the legs can cause complications, such as heart valve problems, stroke, and arrhythmia. The ischemic stroke in idiopathic DCM is most commonly attributed to cardioembolism; however, other factors, namely hypertension, hyperlipidemia, atrial fibrillation, lower glomerular filtration rate, and intracardiac thrombus can also increase the risk of the ischemic attack in patients with DCM.⁵

A previous study introduced high D-dimer levels of more than 444 ng/mL and decreased EF as factors increasing the risk for LV thrombus in patients with DCM.⁶ In contrast, the risk factors for systemic embolization of the cardiac thrombus are still inconsistent. Characteristics associated with a higher tendency for embolization include protrusion of the thrombus into the LV cavity, large thrombus size, diffuse LV dilatation, and impaired systolic function.⁷ The patient at the time of the presentation had an LVEF as low as 5%–10%, a large protruding LV thrombus attached to the apex, and a severely dilated LV. All these factors together can explain the multiple ischemic attacks our patient experienced.

LV thrombuses can develop at various sites, but it is commonly observed in the apex of the left ventricle, as reported in the literature. The second most common location is the anterior-apical wall. However, thrombi were also observed in the basal⁸ and lateral walls of the LV.⁹ The biventricular thrombi¹⁰ are usually seen in DCM cases with EF less than 30%.⁷

To find the accompanied factors with LV thrombus in adults with non-ischemic dilated cardiomyopathy, among the reported cases in Table 1, 3 of 9 had peripartum cardiomyopathy, described as new onset heart failure occurring during the last

month of pregnancy and five months after delivery. Peripartum cardiomyopathy is evaluated with EF less than 45% and a left ventricle end-diastolic diameter of more than 2.7 cm/m.^{2,9–12} Other factors responsible for DCM and thrombus in the cases included alcohol consumption, amphetamine abuse, and human immunodeficiency virus, of which the first two were present in our case as well. Amphetamine-induced cardiomyopathy, although rare, can cause severe DCM.^{13,14} Catecholamine-mediated signaling causes the cardiovascular system to react to the stimulants by causing vasoconstriction, vasospasm, tachycardia, and hypertension. Myocardial fibrosis and alterations in myocardial contractility can also be brought on by stimulants, leading to the development of dilated or hypertrophic cardiomyopathy. The core of the treatment of amphetamine-induced cardiomyopathy is the discontinuation of drug usage, with valsartan and sacubitril administration.¹⁵

An interesting point in the medical history of our case was the uncontrolled gout and elevated uric acid levels as high as 9.1 mg/dL. Although the effect of elevated uric acid levels on DCM in adults is still not well elucidated and warrants further research, some studies showed that the elevated uric acid level is a risk factor for ischemic heart disease and can be a marker to evaluate the severity of DCM in children.^{16,17} It highlights the role of high uric acid levels in endothelial dysfunction, inflammation, and prothrombotic state by increasing factors such as interleukin-6.¹⁸ Krishnan¹⁹ demonstrated that heart failure, subclinical LV systolic dysfunction, and mortality increased in patients with gout. Mouradjian et al.²⁰ proved in their study that hyperuricemia and gout are independent risk factors for cardiovascular disease and worsening of heart failure. Treatment of these issues with uric acid lowering therapy is recommended for significantly

reducing the risk of cardiac disease during gout flare-up. Moreover, Cipolletta et al.²¹ reported that high uric acid levels and gout flare were related to venous thromboembolism.

Li X-L et al.²² and Tigen et al.²³ developed a nomogram to predict the likelihood of ventricular thrombosis in DCM. Their model supports the influence of age, LVEF, uric acid, NT-proBNP, and D-dimer on ventricular thrombosis. Additionally, the increased NT-proBNP levels in our case indicates a poor prognosis for DCM with sinus rhythm.^{22,23} A review of the literature revealed that LV thrombus in a setting of DCM can present as heart failure exacerbation,^{9,10,24,25} or ischemic attacks. Additionally, embolisms were reported in the lower limb,^{7,8,26} brain,^{8,26,27} kidney,¹³ and spleen.^{11,13} Upon comparing our case to previous studies, it is important to note that simultaneous infarcts have been reported in the left common iliac artery and right renal artery, along with a transient ischemic attack characterized by dysarthria. This underscores the need for prompt initiation of emergent therapy due to the features of the LV clot, such as protrusion and its size, which were more significant than those of the thrombi reported in the literature. Notably, in one of the cases, symptoms began immediately after the administration of vasodilators and diuretics for heart failure treatment.⁸ The exact indications for preventing LV thrombus in DCM remain unknown to date. However, Takotsubo syndrome, LV noncompaction, eosinophilic myocarditis, peripartum cardiomyopathy, and cardiac amyloidosis are considered risk factors for LV thrombus, and oral anticoagulants can be prescribed for its management.⁸ At present, surgical removal of LV thrombus is not recommended except when anticoagulant therapy is not well tolerated; moreover, a high risk for embolization and occurrence of thromboembolic events exists despite

anticoagulation therapy.^{26,28} The valuable point in the literature review is that after the resolution of thrombi, the EF improved in some cases.^{13,26,27} The reviewed cases did not mention any recurrence of LV thrombus; however, two studies report a recurrence of LV thrombus or an increase in the size of the thrombus in the patients with inappropriate medical adherence and those with hematologic malignancies, chronic renal failure, and inflammatory disease.^{27,28}

Conclusion

We emphasize the critical impact of LV thrombosis in patients with DCM, leading to multiple ischemic attacks affecting various organs. Our case report underscores the importance of recognizing the associated risk factors for LV thrombus formation and embolic events, characteristics of the thrombus, and potential complications, while also highlighting the need for urgent therapy initiation and the potential for improvement in EF after the resolution of the thrombus. Additionally, the presence of high uric acid levels in our case report warrants for further research to understand better the impact of elevated uric acid levels on DCM and to optimize management strategies for preventing and treating LV thrombus in patients with DCM.

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Author contributions

Maziar. M. contributed to conceptualization, methodology, project administration, supervision, writing, review and editing. **Mani. M.** contributed to data curation, investigation, resources, software, visualization, writing, review and editing. **M.G.** contributed to

conceptualization, data curation, investigation, resources, validation, writing, original draft. **P.S.** contributed to formal analysis, methodology, writing original draft. **S.P.** contributed to data curation, software, writing. **G.G.** contributed to methodology, writing, original draft, review and editing. **M.R.** contributed to investigation, formal analysis, writing, review and editing.

Consent to participate

Patient provided written informed consent prior to participating.

Consent for publication

Informed consent for publication was provided by the participant.

Data availability

The data sets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declaration of conflicting interest

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethical considerations

Ethical approval to report this case was obtained from local ethics committee of Guilan University of Medical Sciences (approval number: IR.GUMS.REC.1402.607).

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Patient perspective

My overall health has improved since starting the treatment, and I have not experienced any further episodes of dysarthria. The claudication has also improved following the stenting procedure.

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