



## Stroke Secondary to Fibromuscular Dysplasia

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### Abstract

FMD is a rare systemic vascular disease characterized by abnormal cell proliferation in the artery walls, leading to artery narrowing, twisting, or bulging. It commonly affects the carotid and renal arteries and can result in renal diseases and stroke. This paper presents a case study of a 39-year-old unhoused female patient who experienced a stroke secondary to fibromuscular dysplasia (FMD) and left ventricular thrombus. The patient presented with asymmetric movement of the extremities and psychosis, so intoxication was initially suspected. However, further examination revealed facial asymmetry and motor weakness. CT scans showed a left anterior cerebral artery infarction and irregular contour of the cervical internal carotid arteries, consistent with FMD and carotid thrombus. Management involved stabilizing the patient, providing supportive treatment, and controlling blood pressure. Thrombolytic therapy was not administered due to the time elapsed since symptom onset. The patient was started on anticoagulation for the carotid plaque and left ventricular thrombus. A transthoracic echocardiogram revealed a large left ventricular thrombus and left atrial enlargement, and the patient was started on heart failure medications.

FMD and left ventricular thrombus are not directly related, but FMD can contribute to thrombus development through its impact on the cardiovascular system, including hypertension, arterial wall damage, aneurysm development, or dissection. Treatment for FMD may involve medication, observation, or surgical interventions like angioplasty and stenting. Anticoagulation therapy is essential for managing the left ventricular thrombus.

This case highlights the importance of early diagnosis and treatment of FMD to prevent complications such as stroke. It is important to consider FMD in the diagnosis of younger patients with a stroke, especially those with a history of hypertension, substance abuse, or other risk factors. Further research is needed to better understand the relationship between FMD and stroke and to optimize treatment strategies for these patients.

### Keywords

Fibromuscular Dysplasia, Left Ventricular Thrombosis, Stroke, Renal Disease, Case Report

### Introduction

Fibromuscular dysplasia (FMD) is a rare systemic vascular disease, usually affecting younger women between ages of 20 to 60. This disease is the second most common cause of renovascular hypertension

after atherosclerotic disease, affecting roughly 1% of hypertensive patients. Overall, FMD affects only 7% of the population [1]. However, roughly 40% of the patients with FMD experienced stroke during their lifetime [2]. It involves an idiopathic, non-

inflammatory, and non-atherosclerotic disease process and can affect any artery bed, but often affects the carotid and renal arteries [3]. It is characterized by aberrant cell proliferation inside the artery walls, which causes the blood vessels to bulge, twist, or narrow. About 75% of FMD patients experience renal artery involvement, and >35% of individuals have bilateral renal artery involvement [4].

Although several hormonal and mechanical reasons have been proposed, the exact cause of FMD remains unclear despite substantial research. It has been linked to environmental and genetic causes. 10% of FMD patients have a family relative who has the disease. Risk factors for FMD include smoking, high blood pressure, and certain disorders such as Ehlers-Danlos syndrome, Marfan syndrome, tuberous sclerosis, Alport syndrome, neurofibromatosis type 1, and Williams syndrome [3,4].

FMD can be specifically classified in three ways. This classification is based on the arterial layer that is mainly affected: intimal, medial, or perimedial [5,6]. The intimal type is characterized by subendothelial connective tissue growth and fibrous circumferential intimal thickening. In intimal FMD, the innermost layer of the artery walls, known as the intima, thickens and may develop into a fibrous or fibroelastic plaque. In other words, the intimal type correlates to subendothelial connective tissue growth and fibrous circumferential intimal thickening [5]. The medial type is another classification. The intermediate layer of the artery walls (media), which contains the smooth muscle cells, develops an irregular arrangement, and takes on the appearance of a "string of beads." Fibrosis takes the place of the smooth muscle cells. The renal artery is where medial FMD is most frequently observed; however, it can also affect other arteries [5]. The least frequent form of FMD, accounting for fewer than 10% of cases, is perimedial FMD. In this type of FMD, the aberrant smooth muscle cells are found in the adventitia, the outer layer of the artery wall. This can cause aneurysm development or dissection. Any artery in the body can be affected by perimedial FMD [5,6]. As a result, reduced blood flow to numerous organs and tissues can have a variety of adverse effects.

Many FMD patients have no symptoms at all and are discovered when an imaging examination of the arteries is carried out for another cause. Nonetheless, early diagnosis and treatment are key to a positive outcome. Patients with FMD encounter a variety of symptoms that vary greatly depending on the location of the afflicted arteries. FMD can also result in several serious and perhaps fatal consequences. The most typical symptom of renal artery disease is hypertension [7]. Reduced blood supply to the kidneys because of the renal artery constriction activates the renin-angiotensin-aldosterone pathway, raising blood pressure. Patients with FMD may struggle to control their hypertension, which occasionally may not respond to treatment. Renal failure may result from renal artery stenosis, another FMD consequence. When the renal artery narrows, less blood can reach the kidneys, which can cause ischemia and harm to the renal parenchyma. This can lead to end-stage renal illness or chronic kidney disease, both of which need for dialysis or kidney transplantation [8].

In addition to the renal artery, FMD can also affect other arterial beds, including the carotid, vertebral, coronaries, and mesenteric arteries [5]. This can result in a variety of possible problems, such as aneurysm development, stroke, transient ischemic attack, and mesenteric ischemia. The mesenteric ischemia secondary to FMD can lead to weight loss and postprandial abdominal pain. FMD in the neck's carotid arteries can result in migraines, neck discomfort, tinnitus, vertigo, Horner syndrome, or strokes. FMD can result in claudication or acute limb ischemia in the legs [3].

The relationship between FMD and stroke is complex and not fully understood. However, it is thought that FMD can increase the risk of stroke in several ways. First, FMD has been linked to stenosis, or narrowing, of the carotid or vertebral arteries, which can result in a reduction in blood flow to the brain [1]. This reduced blood flow can increase the risk of a stroke occurring. Second, FMD can result in the development of aneurysms, which are weakened areas in the walls of the arteries. These aneurysms have the potential to burst, resulting in brain hemorrhage and a stroke. Dissections, which are rips in the artery wall,

can also be brought on by FMD. Blood clots that develop because of dissections can obstruct blood flow to the brain and result in a stroke [9].

In conclusion, fibromuscular dysplasia is a rare disorder that affects the arteries and can cause a variety of symptoms, such as hypertension and stroke, depending on which arteries are affected. Although the exact cause of FMD is unknown, it is believed to be due to a variety of factors. Early diagnosis and management are essential to prevent stroke and improve outcomes in patients with FMD. Individuals with FMD should adopt a healthy lifestyle and undergo regular monitoring to prevent complications. In this study, we report a case of a 39-year-old female who presented with a stroke secondary to fibromuscular dysplasia and left ventricular thrombus.

### Case Description

A 39-year-old unhoused, female with several hospitalizations related to illicit drug use was brought in by ambulance after she was found disheveled in a parking lot at 5 pm and not answering questions. There was initial concern for psychosis or intoxication as a cause of her symptoms, but when she arrived at the hospital, she was noted to have asymmetric movement of her extremities. Patient made eye contact but not answering any questions. History was obtained per chart review. The patient has a history of hypertension, psychiatric hospital placement in 2012-2013, schizophrenia, and polysubstance abuse including alcohol and methamphetamines. Review of systems was unable to be performed because the patient was nonverbal. Patient was eventually placed on 5150 for being gravely disabled at 8 pm. CT head done at 4 AM the following day suggest acute infarct of left parietal anterior surrounding the anterior cerebral artery circulation. Neurology was consulted.

Patient's vitals on presentation were stable, besides an elevated blood pressure. She was afebrile, blood pressure was 150/115, pulse was 79 beats/min, respiratory rate was 16 breaths/min, and 97% oxygen saturation on room air. On the physical exam, the patient appeared very disheveled and unkempt, with poor dentition and some reddening on her skin due to picking repeatedly at her face. She made eye contact

but did not answer any questions. She occasionally moaned but was mostly mute and did not provide any meaningful information. Head was normocephalic and atraumatic, and the rest of the exam was benign except the neurological exam. On neurological testing, there was facial asymmetry with labial commissure rising further on the left. There was motor weakness, right lower extremity hemiplegia, and limited motion of the right upper extremity. Achilles reflexes were 4+ on the right side, and 2+ on the left side.

Upon labs, the patient's basic metabolic panel and complete blood count was normal. However, patient had mild transaminitis and hyperbilirubinemia: ALT 55 U/L, AST 52 U/L, alkaline phosphatase 139 U/L, total bilirubin 1.7 mg/dL, and direct bilirubin 0.6 mg/dL. Additionally, the patient had a normal lipase (**Table-1**).

Urine toxicology was positive for cannabinoids on admission, and ethanol and methamphetamine were negative. However, the patient was methamphetamine positive on a hospital admission four months prior. The Hepatitis panel was negative. Abdominal ultrasound was negative for any gallstones or biliary ductal dilatation, and x-ray skull was negative.

CT head without contrast showed a left ACA territory infarction without mass effect or midline shift (**Fig-1**). CT angiogram head and neck with contrast showed irregular contour of cervical ICAs, likely compatible with fibromuscular dysplasia and right carotid mobile plaque/thrombus. X-ray chest demonstrated cardiomegaly without focal consolidation or pleural effusion (**Fig-2**).

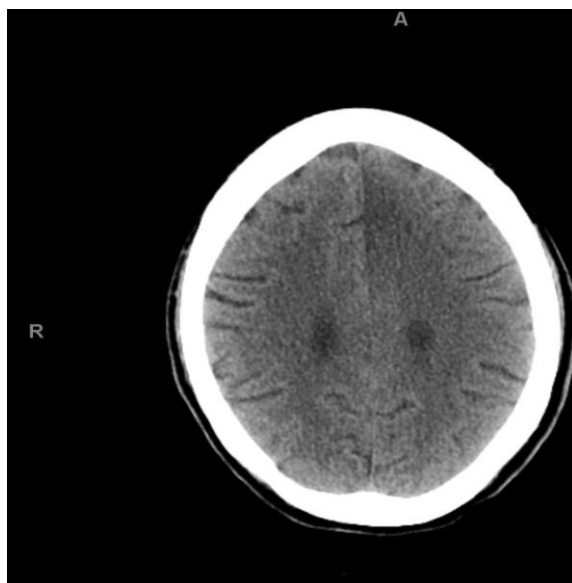
After initial workup, neurology was consulted who recommended no need for thrombolytics because the time frame had passed. Admission to internal medicine was requested, the patient was placed on 1799 hold, and psychiatry was consulted. Per psychiatry, the patient was started on Latuda 40 mg twice daily. Patient was started on aspirin 81 mg daily, lipitor 40 mg daily, and admitted to telemetry with a plan for anticoagulation for right carotid plaque as well as for the left ventricular thrombus.

## Case Report

**Table-1: Patient's Laboratory Results**

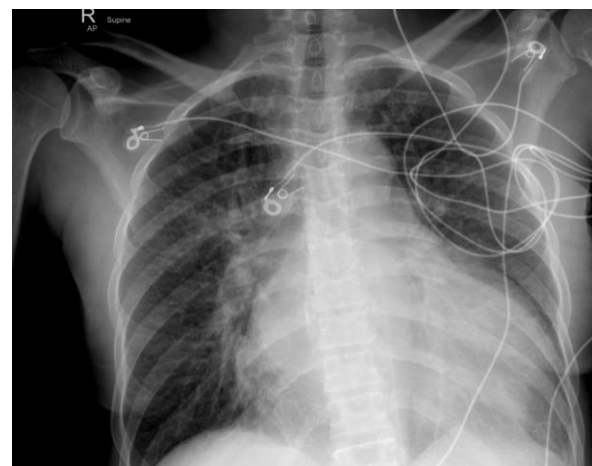
| Test                                         | Patient's Results | Normal Range                      |
|----------------------------------------------|-------------------|-----------------------------------|
| Sodium                                       | 139               | 136-145 mEq/L                     |
| Potassium                                    | 4.4               | 3.5-5.0 mEq/L                     |
| Chloride                                     | 105               | 95-105 mEq/L                      |
| Bicarbonate (HCO <sub>3</sub> <sup>-</sup> ) | 24                | 22-28 mEq/L                       |
| BUN                                          | 16                | 7-18 mg/dL                        |
| Creatinine                                   | 0.88              | 0.6-1.2 mg/dL                     |
| Glucose                                      | 85                | 70-140 mg/dL                      |
| WBC                                          | 7,800             | 4,500 - 11,000/mm <sup>3</sup>    |
| Hemoglobin                                   | 13.5              | 12.0-16.0 g/dL                    |
| Hematocrit                                   | 44.9              | 36% - 46%                         |
| MCV                                          | 81                | 80-100 µm <sup>3</sup>            |
| Platelet Count                               | 261,000           | 150,000 - 400,000/mm <sup>3</sup> |
| ALT                                          | 55 (High)         | 8-20 U/L                          |
| AST                                          | 52 (High)         | 8-20 U/L                          |
| ALP                                          | 139 (High)        | 20-70 U/L                         |
| Total bilirubin                              | 1.7 (High)        | 0.1-1.0 mg/dL                     |
| Direct bilirubin                             | 0.6 (High)        | 0.0-0.3 mg/dL                     |
| Lipase                                       | 37                | 10-140 U/L                        |

BUN: Blood Urea Nitrogen; WBC: White Blood Count; MCV: Mean Corpuscular Volume; ALT: Alanine Transaminase; AST: Aspartate Transaminase; ALP: Alkaline Phosphatase



**Fig-1:**

CT head, taken 4/24/22, showed left parietal parafalcine subcortical white matter hypodensity with no mass effect suggestive of an acute process in the appropriate clinical setting in the ACA distribution.

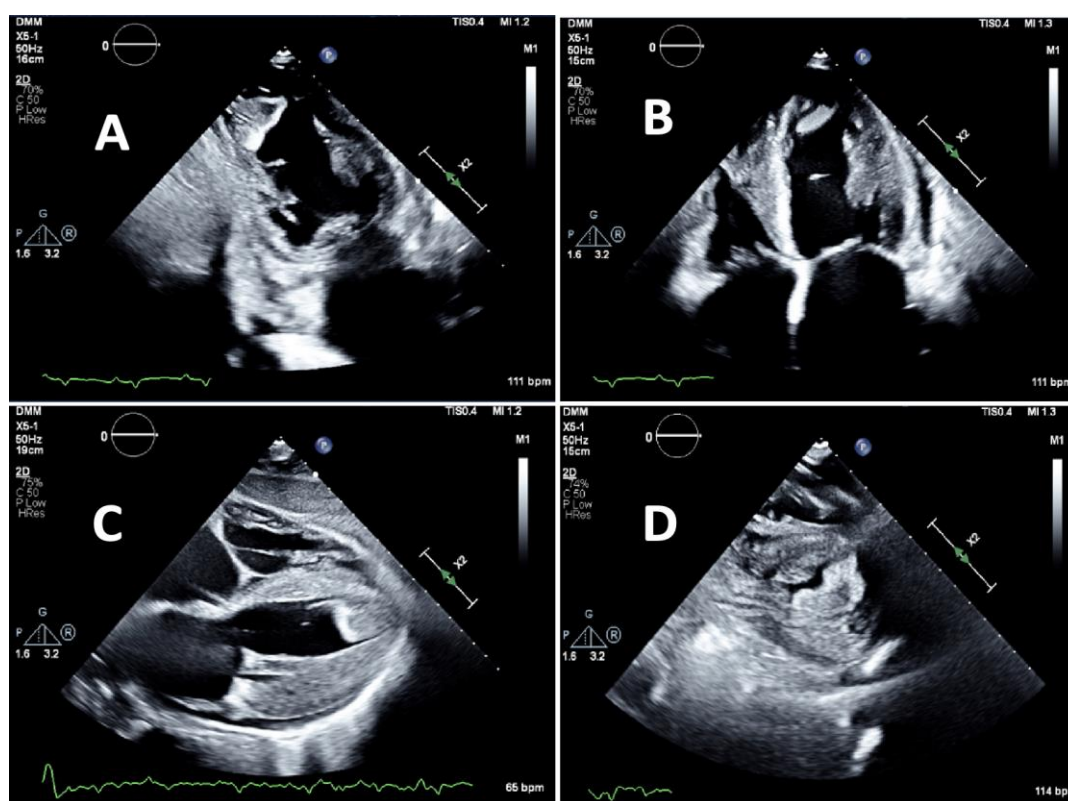


**Fig-2:**

Chest x-ray, taken 4/24/22, showed cardiomegaly without focal consolidation or pleural effusion.

Two days later, a transthoracic echocardiogram (TTE) was completed because of cardiomegaly with a new stroke. Left ventricular ejection fraction was found to be 27%. Left ventricle was mildly dilated in size with mild left ventricular hypertrophy. There was

## Case Report



**Fig-3: Transthoracic Echocardiogram (TTE)**

(A) Two-chamber view. (B) Four-chamber view. (C) Subcostal view. (D) Parasternal short axis.

a large LV thrombus 2.5 cm x 6 cm. Furthermore, TTE showed grade III diastolic dysfunction with elevated LA pressures, severe left atrial enlargement, and mildly elevated pulmonary pressures (**Fig-3**). The aortic root was normal in size and had no hemodynamically significant valvular abnormalities. Patient was started on heart failure medications that included: carvedilol 6.25 mg 2x daily, lisinopril 10 mg once daily and Lasix 20 mg daily.

Repeat CT head six days after admission showed hemorrhagic conversion (**Fig-4**), so it was recommended to hold anticoagulation for at least 7 days per neurology. Patient was eventually started on Xarelto 15 mg oral twice daily for 21 days, with a switch to 20 mg oral daily afterwards.

Patient was discharged a little over a month after admission. The patient was stable throughout the hospitalization and regained most of her strength. Patient was discharged on 20 mg oral daily Xarelto, Latuda 40 mg twice daily, aspirin 81 mg daily, Lipitor 40 mg daily, carvedilol 6.25 mg 2x daily, lisinopril 10 mg once daily, and Lasix 20 mg daily. Patient was also

instructed to follow up for a repeat transthoracic echocardiogram for evaluation for any residual thrombus after 3 months of anticoagulation. Vascular surgery recommended outpatient renal artery doppler ultrasound as part of work-up for fibromuscular dysplasia.



**Fig-4:**

CT head, taken 4/29/22, showed left ACA territory infarct with interim development of subarachnoid hemorrhage in this area.

## Discussion

Stroke caused by left ventricular thrombus with fibromuscular dysplasia (FMD) is a rare but serious illness that needs early and thorough treatment. Early identification and treatment are essential because the combination of these two disorders can considerably raise the risk of morbidity and mortality. Younger people with a cervical bruit, a "swishing" sound coming from one or both ears, transient ischemic attack, stroke, or arterial dissection, as well as people under 35 with newly developed hypertension should all have FMD on their differential diagnosis list [4]. There are various phases involved in managing a patient who has experienced a stroke owing to FMD and a left ventricular thrombus. Stabilizing the patient and administering supportive treatment, such as oxygen therapy, intravenous fluids, and blood pressure control, constitutes the initial phase. Once the patient is stable, attention turns to managing the left ventricular thrombus and FMD specifically.

Imaging techniques like ultrasound, magnetic resonance angiography (MRA), or computed tomography angiography (CTA) are frequently used to diagnose FMD. The initial test of preference for suspected FMD is computed tomographic angiography (CTA), however contrast-enhanced magnetic resonance angiography (MRA) is an alternative if CTA is not appropriate. Additionally, CTA provides a more precise distinction between FMD and atherosclerotic renal artery stenosis by better seeing tiny calcifications [10]. The location, severity, and occurrence of comorbidities all affect how FMD should be treated. FMD may not always need any therapy; instead, the illness can be kept under observation with routine imaging scans. In other situations, a physician could recommend medicine to regulate blood pressure or stop blood clots. Surgery or angioplasty with or without stent implantation are available as treatments for severe cases [11].

There is no connection between left ventricular thrombosis and FMD. However, because of how it affects the circulatory system, FMD can unintentionally contribute to the occurrence of left ventricular thrombosis. Hypertension, a known risk factor for left ventricular thrombosis, can be brought on by FMD.

Blood clots may form as a result of hypertension's damaging effects on the blood vessel walls. Although FMD and left ventricular thrombosis are not directly related, FMD's effects on the cardiovascular system can indirectly cause the development of left ventricular thrombosis [12]. Additionally, FMD might have an impact on the coronary arteries, which feed blood to the heart muscle. A disruption in the blood supply to the heart muscle can harm the muscle and raise the risk of left ventricular thrombosis [13]. Additionally, FMD can result in the development of aneurysms or artery wall dissections, both of which raise the possibility of blood clots developing. In those with FMD, controlling hypertension and other risk factors for cardiovascular disease can help lower the likelihood of left ventricular thrombosis. Treatment of left ventricular thrombosis requires prompt medical intervention.

Anticoagulation therapy is the cornerstone of care for individuals with left ventricular thrombus. Anticoagulation therapy aims to stop the growth and development of thrombi and lower the risk of embolic events. The clinical state, comorbidities, and bleeding risk of the patient all influence the anticoagulant of choice. Warfarin and other direct oral anticoagulants are prevalent oral anticoagulants. Low-molecular-weight heparin or intravenous heparin may be required in some circumstances, though [14].

Stroke caused by FMD can be treated with antiplatelets or anticoagulation. Estimated long-term risk of stroke recurrence ranging in incidence from 1% to 3% [15]. Prognosis for FMD is dependent on early detection. There is no cure for the disease, so a variety of treatments can manage the disease. In general, many people with the disease have a normal life expectancy if they are monitored closely.

Treatment of the underlying vascular abnormalities, which may require angioplasty, stenting, or surgical revascularization, is necessary to manage FMD. For the majority of FMD patients, angioplasty—with or without stent placement—is the preferred method. This entails placing a balloon or stent to enlarge the concerned artery's restricted section after utilizing a catheter to access the affected artery [11].

Rehabilitation following a stroke is an essential component of management in addition to particular treatments for FMD and left ventricular thrombus. Through the enhancement of physical, cognitive, and emotional function, stroke rehabilitation attempts to promote functional independence and quality of life. Physical therapy, occupational therapy, speech therapy, and psychological support are all possible components of rehabilitation [16]. FMD is a chronic illness that needs constant management and observation. FMD patients should adopt a healthy lifestyle that includes consistent exercise, a well-balanced diet, and quitting smoking. Regular imaging checks can help identify any alterations in the arteries and avoid consequences.

Continuous monitoring and follow-up are necessary for the long-term care of patients with stroke secondary to FMD and left ventricular thrombus. This entails ongoing care of risk factors like hypertension, hyperlipidemia, and diabetes as well as routine imaging tests to evaluate the condition of the artery walls and left ventricular function. Patients with carotid artery FMD may potentially benefit from periodic follow-up and surveillance using carotid duplex [10].

In this case, the diagnosis of FMD was a challenge given the patient was not able to communicate on presentation. Since she is unhoused and has not been seen by a primary care physician, the diagnosis of FMD could not be made earlier. Also, the patient is at risk of being lost to follow-up and further outpatient work-up for her disease may not take place.

Overall, treating a patient who has had a stroke caused by FMD and a left ventricular thrombus necessitates a thorough, multidisciplinary approach. Stabilizing the patient, addressing the underlying vascular abnormalities, and averting additional thrombotic episodes should be the main goals. Long-term monitoring and stroke recovery are also crucial aspects of management. For patients with this uncommon but significant illness, early identification and fast treatment are essential for improving outcomes and lowering morbidity and death.

## Conclusion

Stroke caused by left ventricular thrombus with fibromuscular dysplasia is rare. FMD and left ventricular thrombosis are unrelated conditions. However, FMD may unintentionally cause left ventricular thrombosis due to the way it affects the circulatory system. Early diagnosis and management is vital for the best prognosis of the disease. FMD patients should lead a healthy lifestyle and receive routine monitoring to avoid problems. For patients with left ventricular thrombus, anticoagulation medication is the cornerstone of treatment. The goal of anticoagulation medication is to halt thrombi from growing and developing and to reduce the risk of embolic events. Our case further underscores the significance of considering the entire clinical picture in order to make the right diagnosis.

## Conflict of Interest

The authors have read and approved the final version of the manuscript. The authors have no conflicts of interest to declare.

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# Case Report

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