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PICTORIAL ESSAY

A Pictorial Essay on Fibromuscular Dysplasia Imaging: Beyond the String of Beads

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Abstract

Fibromuscular dysplasia (FMD) is a non-atherosclerotic, non-inflammatory arteriopathy characterized by abnormal cellular proliferation and distorted architecture of the arterial wall. FMD mainly affects the mid-distal segment of renal and extracranial arteries. The clinical phenotype of FMD has recently expanded to include arterial dissection, aneurysm, and tortuosity. FMD is four times more frequent in women than men. The etiology of FMD is unknown, although some theories have been proposed, such as those exploring the role of environmental and genetic factors. Catheter-based angiography, optical coherence tomography, and intravascular ultrasound are some of the most reliable imaging techniques when assessing the morphology of FMD. There is currently no cure for FMD, but patients may take medications for symptomatic management and to prevent complications. If blood pressure is uncontrollable, balloon angioplasty is recommended.

Keywords

Fibromuscular dysplasia, Ultrasound, Intravascular ultrasound (IVUS), Angiography, Angioplasty

Introduction

FMD is an idiopathic arteriopathy usually manifesting in small or medium-sized arteries. FMD mainly affects the carotid, renal and vertebral arteries and could result in subarachnoid hemorrhage, hypoperfusion, aneurysm, dissection, or arterial obstruction [1,2].

FMD is a frequently misunderstood and underdiagnosed disease. It is marked by abnormal growth in arterial walls resulting in stenosis [3]. Due to its rarity and unknown etiology, there is a significant lack of information regarding this condition, and thus

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there exists a considerable gap between symptom onset and its diagnosis [4]. While FMD is not necessarily life-threatening, complications are variable and range from mild to severe [4]. This pictorial essay uses radiological and sonographic images with the objective of familiarizing technologists with different FMD manifestations.

Main Body

FMD has a mean delay to diagnosis of four years [1,4]. Therefore, it is vital to develop a concrete foundation of the clinical manifestations, sonographic findings, and etiological factors associated with FMD. This pictorial essay investigates FMD manifestations in a variety of arteries with embedded case studies.

Coronary artery FMD

Clinical findings of FMD include acute coronary syndrome, left ventricular dysfunction, fatal arrhythmias, and sudden cardiac death. Carefully reviewing the clinical history may allude to indirect signs of FMD that can alter the patient's outcomes and diagnosis.

Pathological manifestations encompass spontaneous coronary artery dissection, intramural hematoma, distal arterial tapering or narrowing, coronary artery spasm, tortuosity, and aneurysm. The images below highlight important lessons pertinent to accurate history collection and thorough vascular assessment [3] (Figure 1, Figure 2, Figure 3 and Figure 4) [5-7].

Cervicocephalic FMD

Figure 5, Figure 6 and Figure 7 [8-10]

Renal artery FMD

Figure 8, Figure 9, Figure 10, Figure 11 and Figure 12 [11-13]

Case study: A young male patient with treatment-resistant hypertension with normal renal ultrasound findings was found to have stenosis in the distal right renal artery [14] (Figure 13).

Iliac artery FMD

Case study: A 71-year-old woman complained of pain and cramping in both legs after a 15-minute walk for the past 6 months. The patient had significant chronic atrial fibrillation and was on Amlodipine (5 mg/day) to control

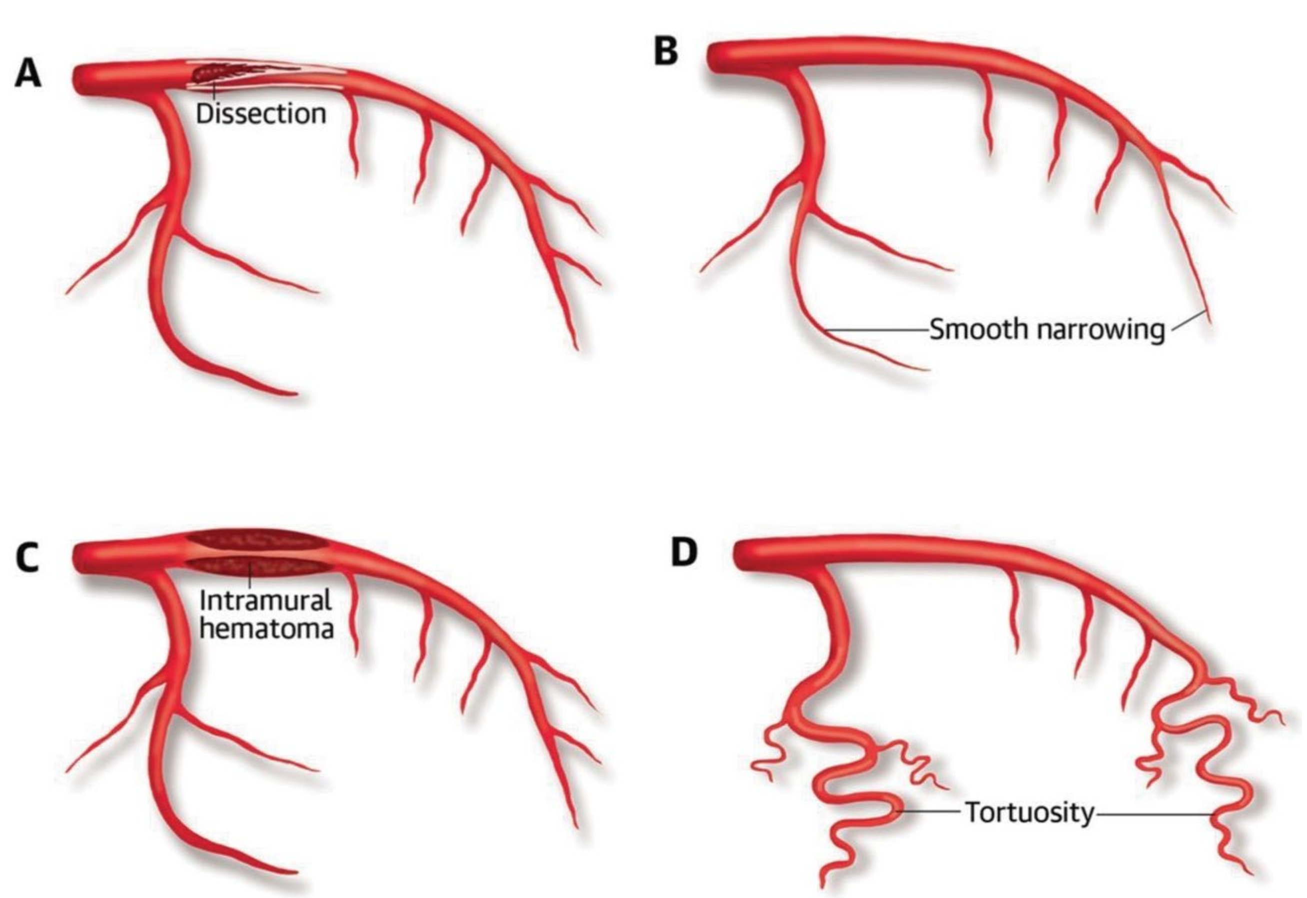

Figure 1: The following diagram demonstrates common pathological findings of coronary artery FMD [5]. **A** represents a Dissection. It occurs when an expanding hematoma leads to a separation of the layers within the coronary arterial wall [5]. **B** depicts *Smooth Narrowing or Distal Tapering*. It may be caused by dissection, intramural hematoma, or even a spasm of the coronary blood vessels [5]; **C** indicates *Intramural Hematoma*. An intramural hematoma is a well-demarcated collection of blood within the arterial walls [5]. **D** represents *Tortuosity* of mid-distal coronary blood vessels. Patients may have tortuous vessels owing to concurrent medial hyperplasia in FMD [5].

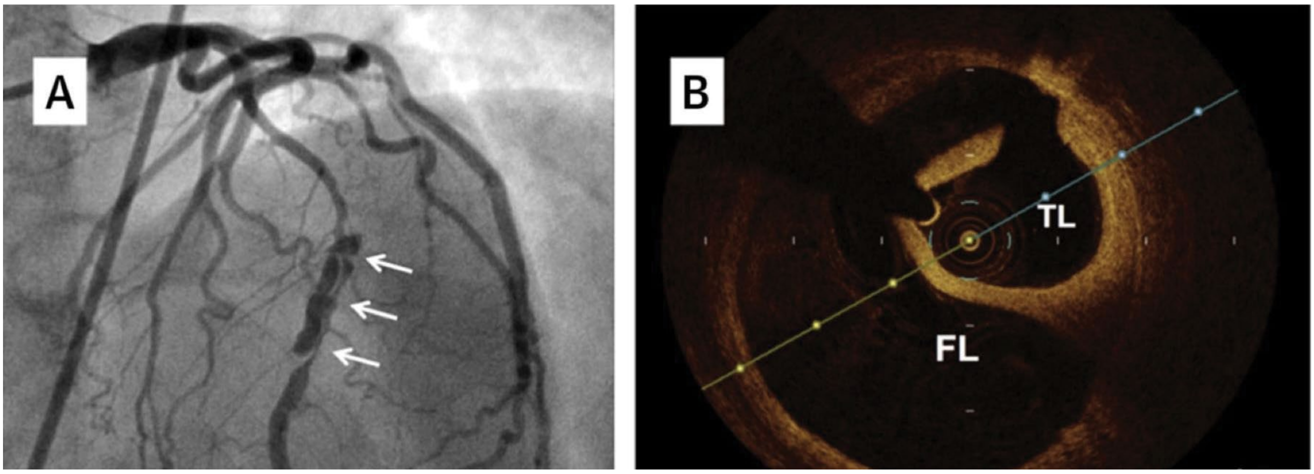

Figure 2: Spontaneous Coronary Artery Dissection (SCAD) is the most common presentation of FMD involving the coronary arteries [5]. It sometimes presents with the characteristic string of beads appearance [5]. There is a strong association of FMD with SCAD - 86% of SCAD cases have concomitant evidence of FMD [6]. The pathophysiology of FMD causing SCAD is not completely understood; however, it can be due to a possible genetic abnormality leading to fibrosis of the vasa vasorum and subsequent ischemia of tunica media [5,6]. The weakened coronary vessel wall, along with the proliferation of myofibroblasts, may result in dissection [5].

Images **A** and **B** demonstrate SCAD in a 34-year-old female patient who presented with ST-segment elevation myocardial infarction (STEMI) seven days after cesarean delivery of a healthy newborn [5]. The pregnancy was complicated by hypertension. **A** is a coronary angiogram in a cranial projection illustrating extensive dissection of the middle Left Anterior Descending (LAD) artery indicated by the arrows [5]. Image **B** utilizes Optical Coherence Tomography (OCT) to depict the dissected LAD artery showing the true lumen (TL) and the false lumen (FL) [5].

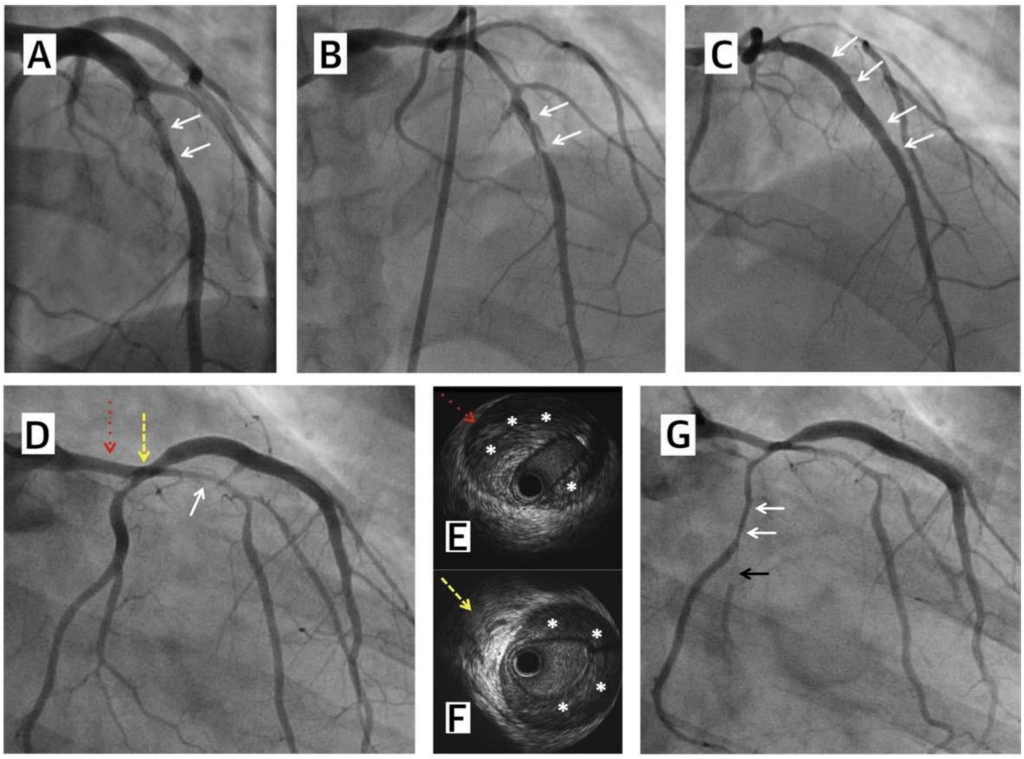

Figure 3: SCAD of the LAD artery with the formation of an intramural hematoma. A 40-year-old woman presented with elevated troponin levels and a Non-ST-segment elevation myocardial infarction (NSTEMI) 10 days after a third-trimester abortion; Image **A** is a coronary angiogram demonstrating SCAD of the middle LAD artery (white arrows). She was managed medically with heparin infusion and tight blood pressure control for 48 hours without percutaneous coronary intervention (PCI). She was stabilized and discharged from the hospital on Aspirin, Clopidogrel, Beta-Blocker, and a Statin. However, she returned within five days after discharge with recurrent NSTEMI and underwent cardiac catheterization; Image **B** is a repeat coronary angiogram illustrating worsening LAD artery dissection (white arrows) [5]; Image **C** is a post-procedure angiogram exhibiting a patent proximal and mid-LAD artery with the placement of stents (white arrows) [5]; Image **D** is a coronary angiogram performed after stenting through PCI. It shows re-expansion of the LAD artery; Images **E** and **F** are intravascular ultrasound images (IVUS) performed immediately after PCI displaying a large intramural hematoma (asterisks) [5]. The patient continued to have intermittent chest pain and rising troponin levels over the next two days and had to undergo a third cardiac catheterization procedure [5].

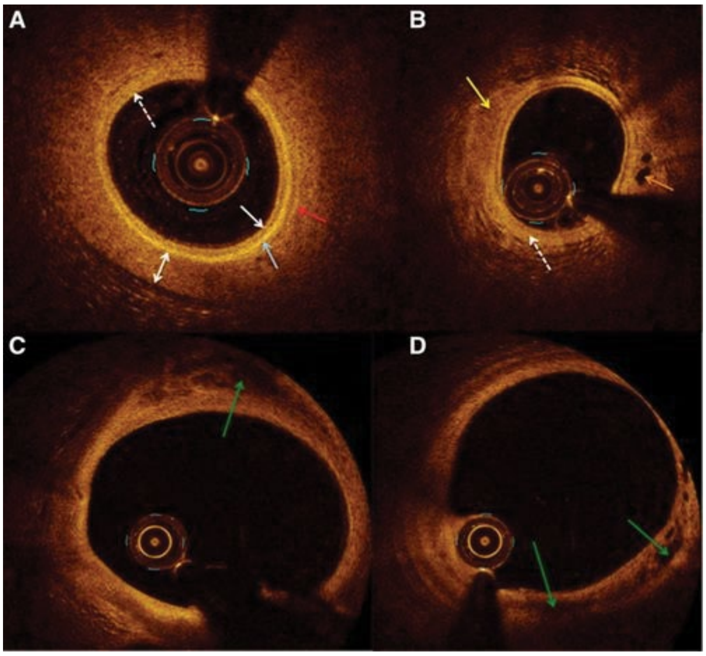

Figure 4: Depicts morphological characteristics of FMD in the coronary arteries using OCT (Optical Coherence Tomography) which can be used to visualize the lumen of the vasculature and obtain information about the composition of the arterial wall. Other imaging modalities are typically employed to detect areas of stenosis, which can be due to several different underlying pathologies. High reflectivity denotes the deposition of a potentially fibrous or collagen matrix. In contrast, low reflectivity denotes smooth muscle hyperplasia or build-up of the proteoglycan-like matrix such as peptidoglycan [7]. **A** shows thickening of the adventitial layer as indicated by the double-ended white arrow [7]. The red arrow shows the formation of an additional layer of the elastic lamina [7]. The white arrow and blue arrow indicate the internal and external elastic laminae, respectively [7]. **B** shows another typical biomarker of FMD, which is the loss of the elastic lamina as depicted using the dashed arrow [7]. The orange arrows indicate the formation of new blood vessels, namely the vasa vasorum [7]. Furthermore, the low reflectivity also suggests the presence of peptidoglycan-like material [7]. The green arrows in **C** and **D** show the formation of cavities surrounding the walls of the coronary arteries [7].

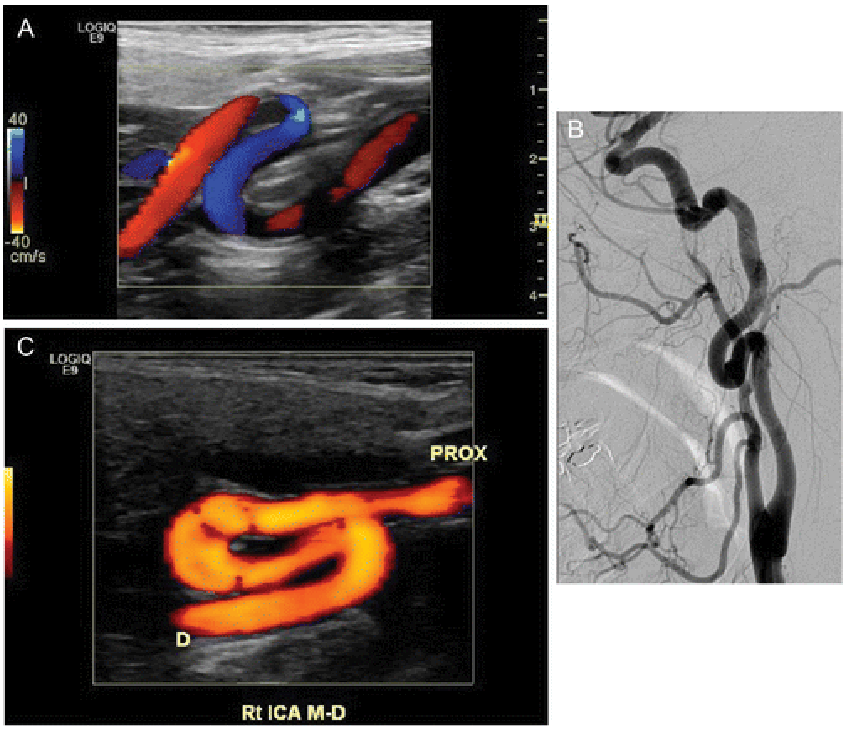

Figure 5: Carotid artery ultrasound images from two distinct patients **A** and **C**. The sonograms were performed on a GE LOGIQ E9 ultrasound machine [8]. The bilateral cervical internal carotid and vertebral arteries were imaged using a 9L-D linear transducer with a frequency of 9MHz in the sagittal plane [8].

(A) Carotid ultrasound showing elongation and tortuosity of the right internal carotid artery with a distinct morphological appearance in the shape of an “S” (S curve) [8]; (B) Catheter angiogram performed in a patient with FMD depicting the S curve in the cervical ICA [8].

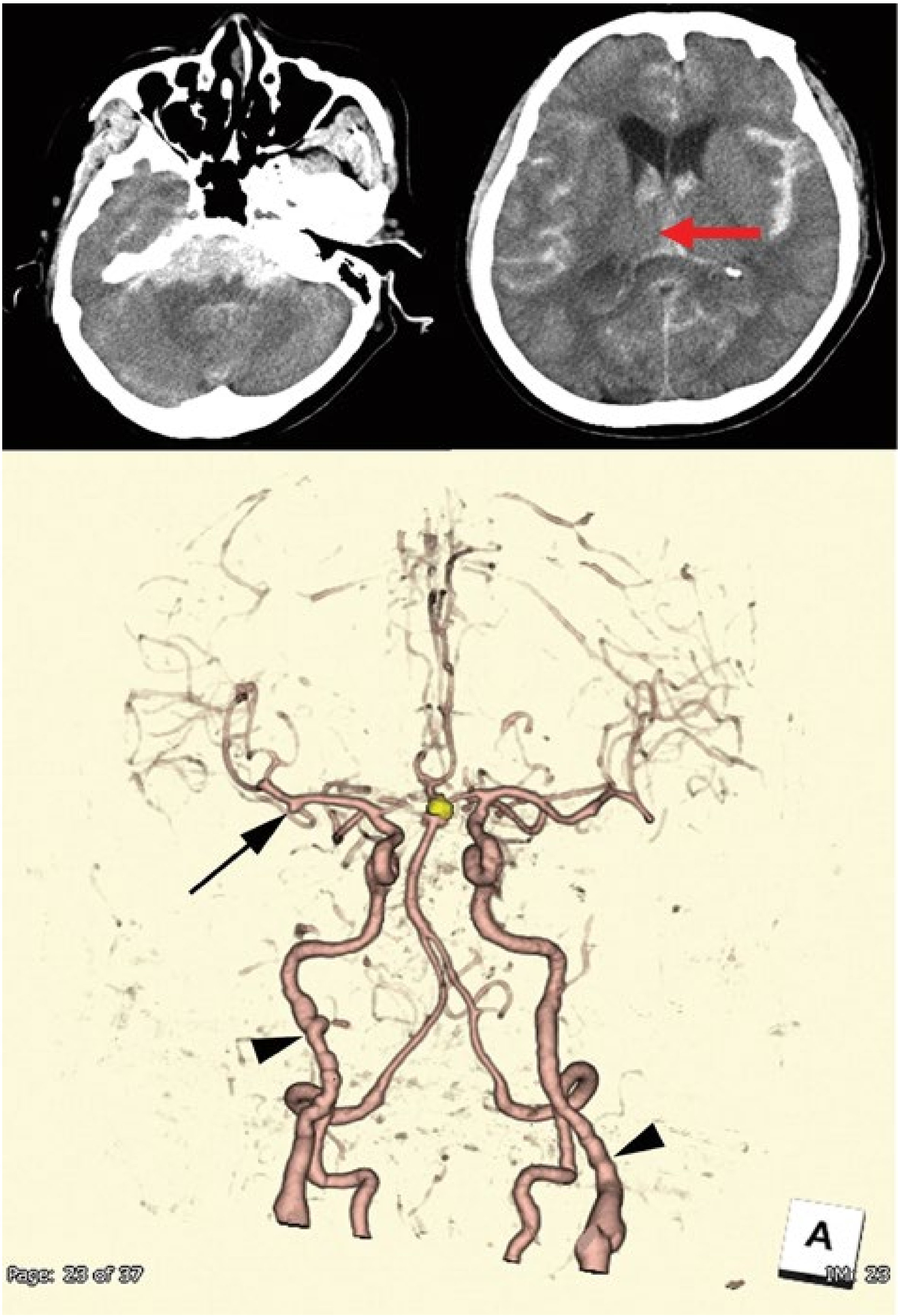

Figure 6: A CT angiogram was performed to assess the cranial vasculature of a patient with subarachnoid hemorrhage in the posterior fossa [9]. The red arrow shows the shift of the midline to the right due to bleeding [9]. The image below shows a head CT angiogram performed on the same patient and the characteristic string of beads appearance, which can be seen bilaterally in the extracranial arteries as indicated by the arrowheads [9]. Furthermore, the basilar artery has a coiled appearance (yellow) [9]. The black arrow indicates the presence of a Berry saccular aneurysm along the middle cerebral artery [9].

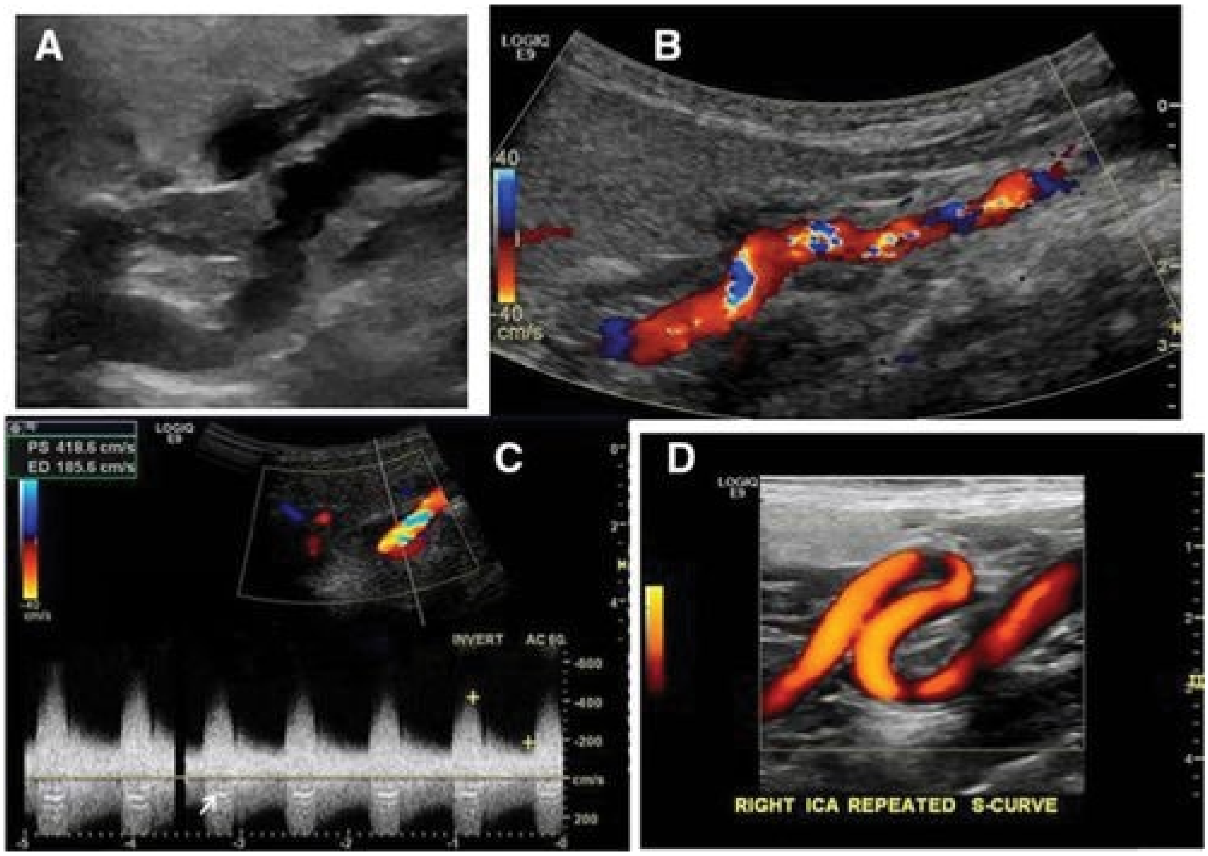

Figure 7: **C** demonstrates the spectral Doppler waveform in the stenosed internal carotid artery [10]. PSV is around 400 cm/s with evident spectral broadening due to the turbulent flow [10]. **A** demonstrates signs of beading and tortuosity in the mid to distal segment of the internal carotid artery [10]; **B** depicts colour Doppler aliasing, and **D** illuminates the tortuous S-shaped of the ICA using power Doppler [10].

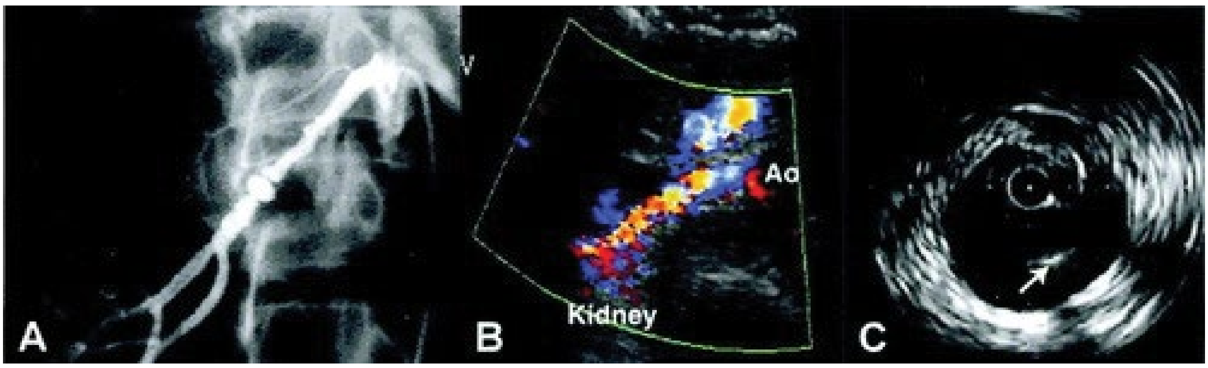

Figure 8: **A** is an angiogram at the level of the renal arteries. **B** color-flow duplex imaging, and **C** illustrates intravascular ultrasound findings imaging of FMD along the right renal artery [11]. The angiogram shows the characteristic string of beads appearance indicative of FMD. The intravascular ultrasound shows the protrusion of a membrane in the lumen of the middle segment of the renal artery affected by FMD, as indicated by the white arrow [11].

Figure 9: Renal angiogram showing the string of beads appearance in the right renal artery in a 53-year-old female, indicative of moderate FMD [12]. Despite FMD manifestation along the main right renal artery, the female donor's right kidney was functional (ERPFs of right kidney- 48.4 mL/min) and a nephrectomy was performed for a kidney recipient [12]. The segment affected by FMD was reconstructed through harvesting an internal iliac artery graft from the patient. The recipient's kidney function recovered post-operatively (normal blood pressure and serum creatinine level < 2 mg/dL over the span of three years after the procedure) [12]. The segment of the renal artery affected by FMD is indicated with the red arrow [12].

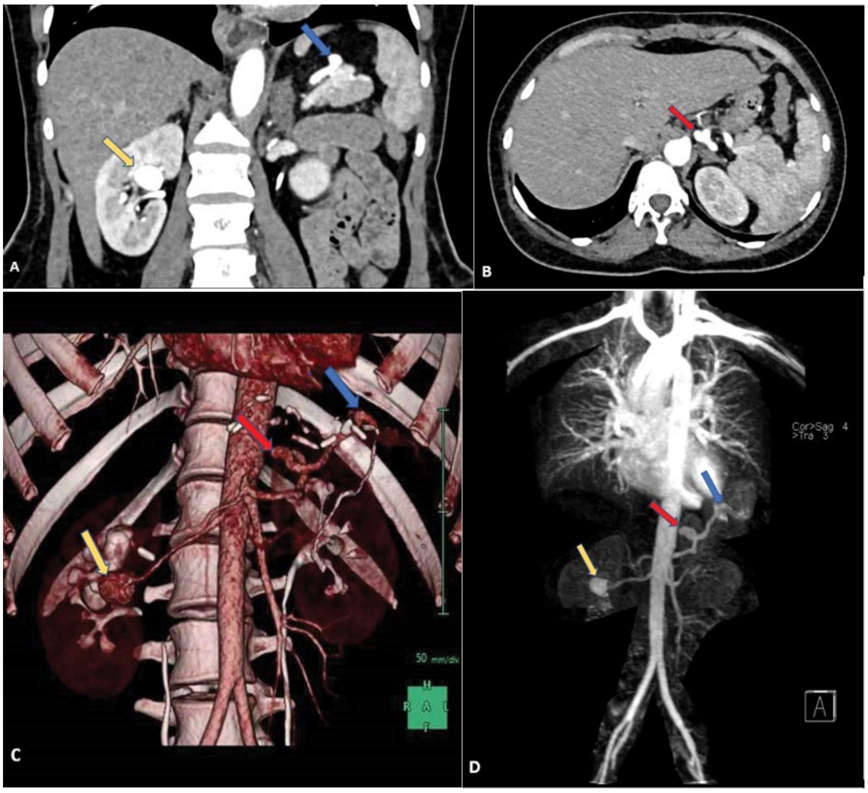

Figure 10: A 40-year-old female with FMD presented with right-sided loin pain and hypertension. A CT with contrast was conducted to assess for vascular anomalies. **A** shows a coronal image, **B** is an axial image, **C** is a 3D reformat C, and **D** is an MRA of the thoracic and abdominal aorta, showing a right renal artery saccular aneurysm in the right renal hilum (yellow arrow), splenic artery saccular aneurysm at the mid-level of the tortuous splenic artery (red arrow) and fusiform aneurysm in the distal splenic artery (blue arrow). This is part of the many cases that are often misdiagnosed simply because FMD does not appear with the classic multifocal string of beads appearance.

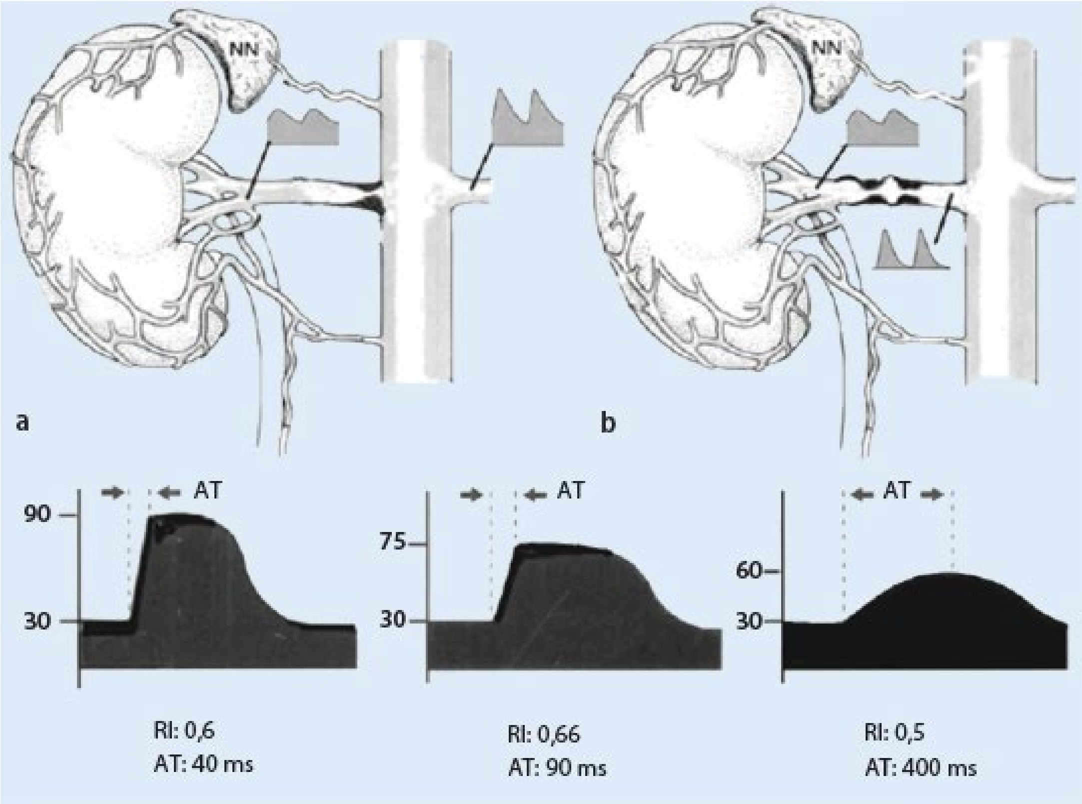

Figure 11: Shows hemodynamic changes associated with renal artery stenosis [13]. The graph furthest on the right illustrates the characteristics of a tardusparvus waveform that encompass a blunted upstroke with a diminished amplitude, increased diastolic flow, prolonged acceleration time (AT) and an overall dampened waveform. In the pre-stenotic region, a sharp upstroke and loss of diastolic flow can be observed. At the site of the stenosis, high blood flow velocities are observed in accordance with the continuity equation.

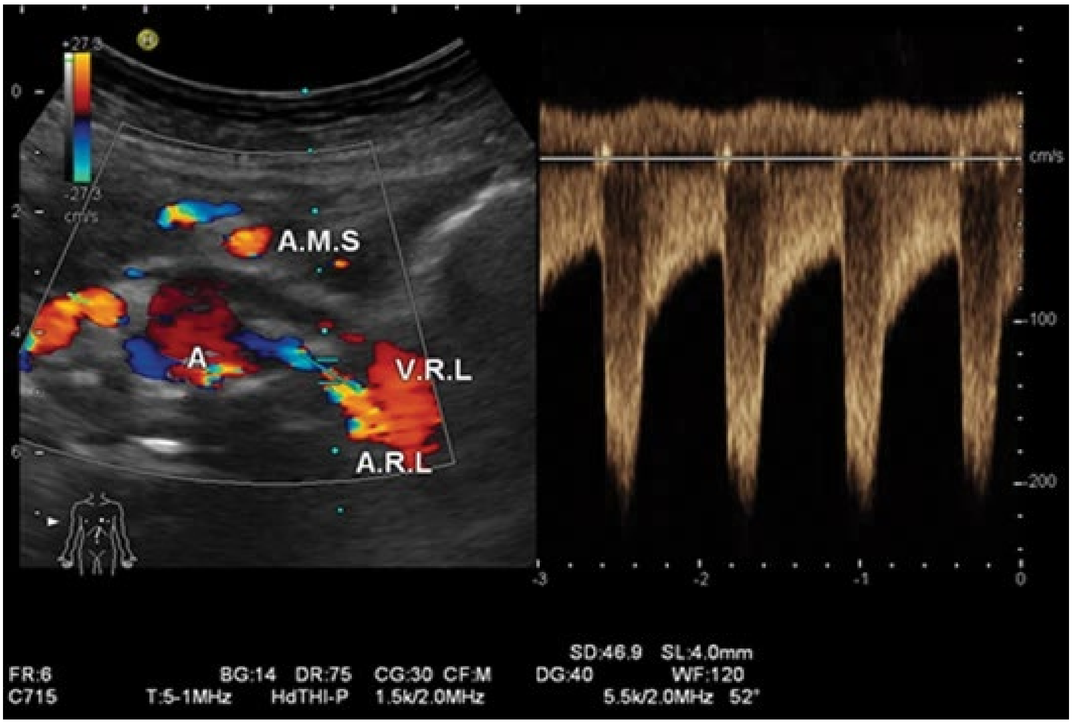

Figure 12: Spectral Doppler was used to complement the findings of colour Doppler in this FMD renal artery case. A peak systolic velocity (PSV) of 220 cm/s was recorded at the site of the stenosis, and a peak systolic velocity of 80 cm/s was recorded at the pre-stenotic segment of the renal artery [13]. At the stenosis, the PSV increased by a factor of 2.7 [13]. This indicates that there is greater than 50% concentric stenosis in the vasculature [13]. Spectral broadening in the ultrasound waveform and aliasing seen on colour Doppler indicate turbulence with a wide range of blood flow velocities being represented.

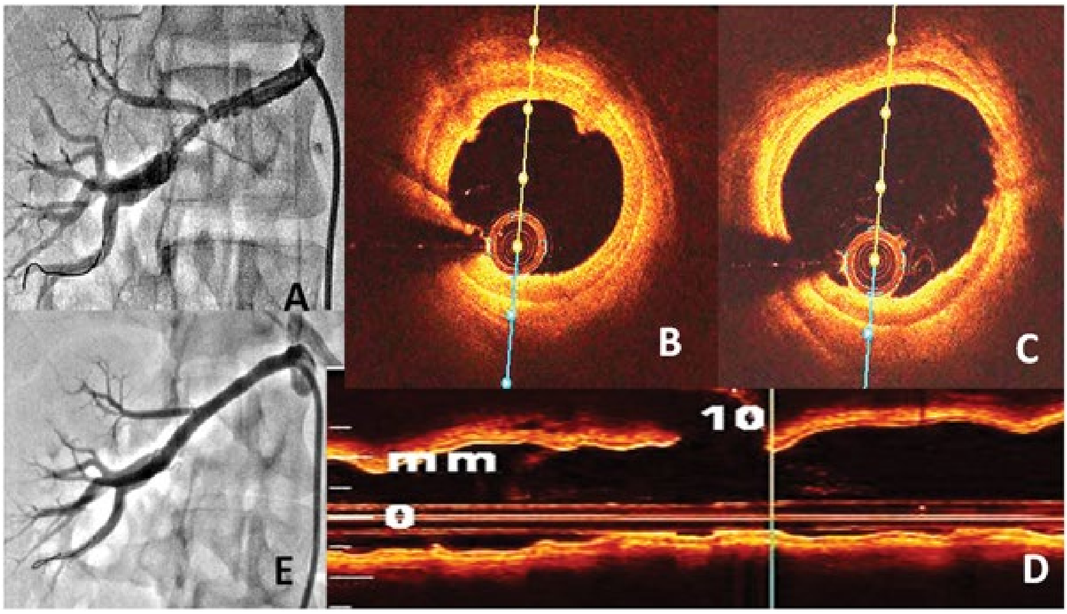

Figure 13: A) Selective renal angiography shows the classic "string of beads" appearance [14]; B,C) OCT revealed thickening of arterial media with regions of low backscatter [14]; D) Regional thickenings resulted in the wavy appearance of the lumen [14]; E) After percutaneous angioplasty, normal flow could be seen on selective angiography of the same vessel [14].

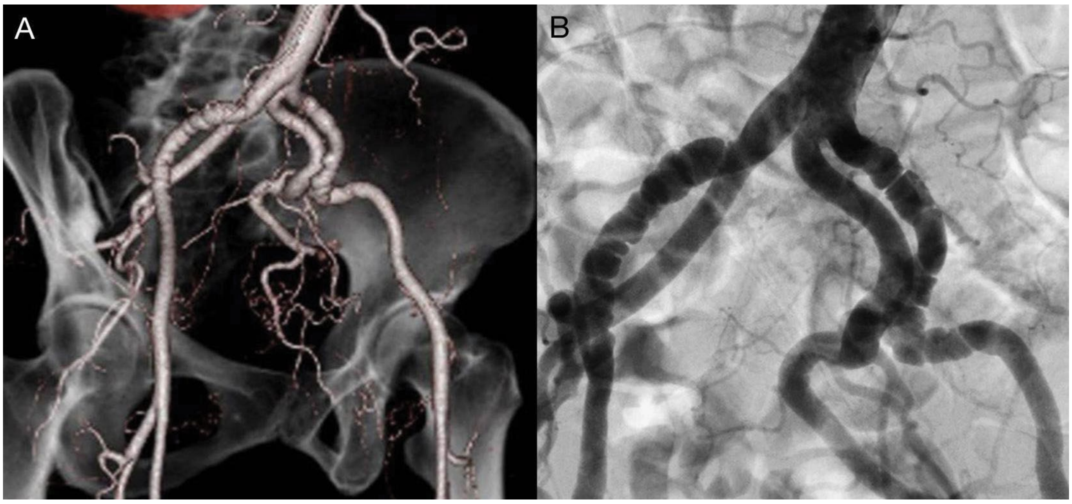

Figure 14: A) Three-dimensional computed tomography angiogram (CTA) of bilateral external iliac arteries showing a typical string of beads pattern [15]; B) Digital subtraction angiography (DSA) of external iliac arteries also shows the string of beads pattern on both sides [15].

her hypertension. She had weak lower extremity pulses on both sides. Her ankle-brachial indices (ABIs) were also reduced to 0.85 on the right side and 0.80 on the left side. Based on these findings, the patient was diagnosed to have mild peripheral artery disease (PAD) with claudication [15] (Figure 14, Figure 15 and Figure 16).

Fibromuscular dysplasia of brachial artery

Figure 17

Case study: While trying to perform transradial coronary angiography in an elderly female with a diagnosis of severe aortic regurgitation as a part of the preoperative workup, the physician faced difficulty passing the guidewire through the left brachial artery [16].

Cerebrovascular FMD

Case study: A 40-year-old woman with no identifiable risk factors presents with symptoms suggestive of Transient Ischaemic Attack (TIA) [17]. The patient participated in a group dance and was involved in moderately vigorous physical activity ten days before the development of symptoms [17] (Figure 18, Figure 19 and Figure 20).

Discussion

Epidemiology

FMD is more common in women among adults. There is no significant sex difference in the prevalence of FMD in children [18]. The renal arteries are involved in ~75%-

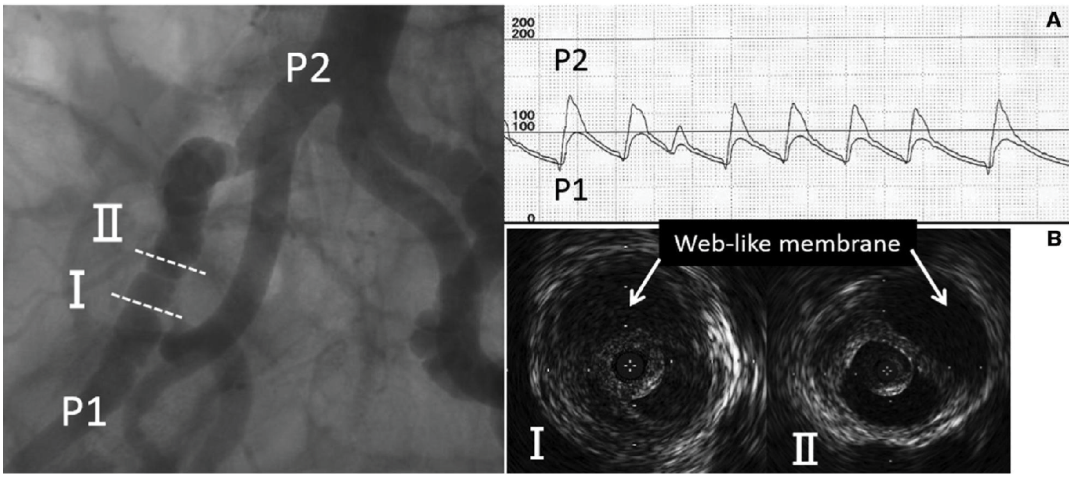

Figure 15: Pressure traces and intravascular ultrasound images (IVUS) of the right external iliac artery before balloon angioplasty were obtained using a 10 cm Radifocus sheath introducer inserted through the left brachial artery. Then, a 90 cm Destination guiding sheath was exchanged and advanced into the right common iliac artery. Once 175 cm of the 0.014 inch Cruise guide wire was positioned proximal to the string of beads pattern of the right iliac artery, IVUS was utilized to locate the web-like membrane obstructing the lumen. Given the multiple sites of stenosis, the pressure gradient across the FMD lesion was measured using a 0.014-inch Aeris pressure wire [15]. A) Pre-angioplasty pressure gradient across the lesion depicting a peak value of 45 mmHg [15]; B) Web-like membrane evident in the lumen before angioplasty [15].

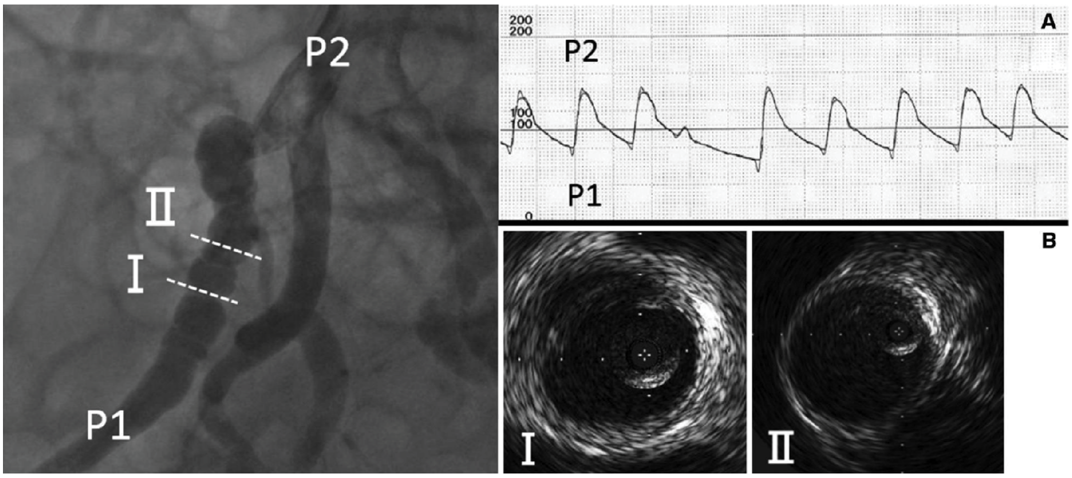

Figure 16: Angioplasty of the right iliac artery was performed using a 7 mm × 40 mm balloon inflated to a pressure of 8 atm. Pressure traces and intravascular ultrasound (IVUS) images of the right external iliac artery were obtained after the procedure. A) There is no post-balloon angioplasty pressure gradient [15]; B) Post-balloon angioplasty image depicting the disrupted web-like luminal membrane [15].

Post-procedure ABIs were 1.02 on the right side and 0.98 on the left side (within normal range). One month following the angioplasty, the patient could walk without any claudication [15].

80% of cases, while the extracranial cerebrovascular arteries are in ~75% of the cases [19].

Etiology

While the cause of FMD remains unknown, various factors are theorized to be involved in its pathogenesis [20]. It is common for this condition to present in several members of the same family - in fact, 7.3% of patients are found to have a relative with this disease [20,21]. Therefore, a genetic influence is a possible explanation - this percentage is not particularly large, and the manifestation or severity of their symptoms can differ [20]. The frequency of non-familial development of the condition complicates research aimed at understanding its genetic basis. Recently, a specific mutation to the COL5A1 gene was explored as a possible monogenic factor of multifocal FMD (mFMD) [21]. It was found that the mutation is correlated with mFMD, which can reveal if it was inherited or if another mechanism was at work [21].

Considering that FMD is more frequently diagnosed in women, it is also popularly speculated that hormones play a role in its development [20]. However, studies that examined the reproductive history or birth control usage found no correlation between hormone levels and the development of the disease [20]. Factors including abnormal arterial growth, blood hemodynamics, or

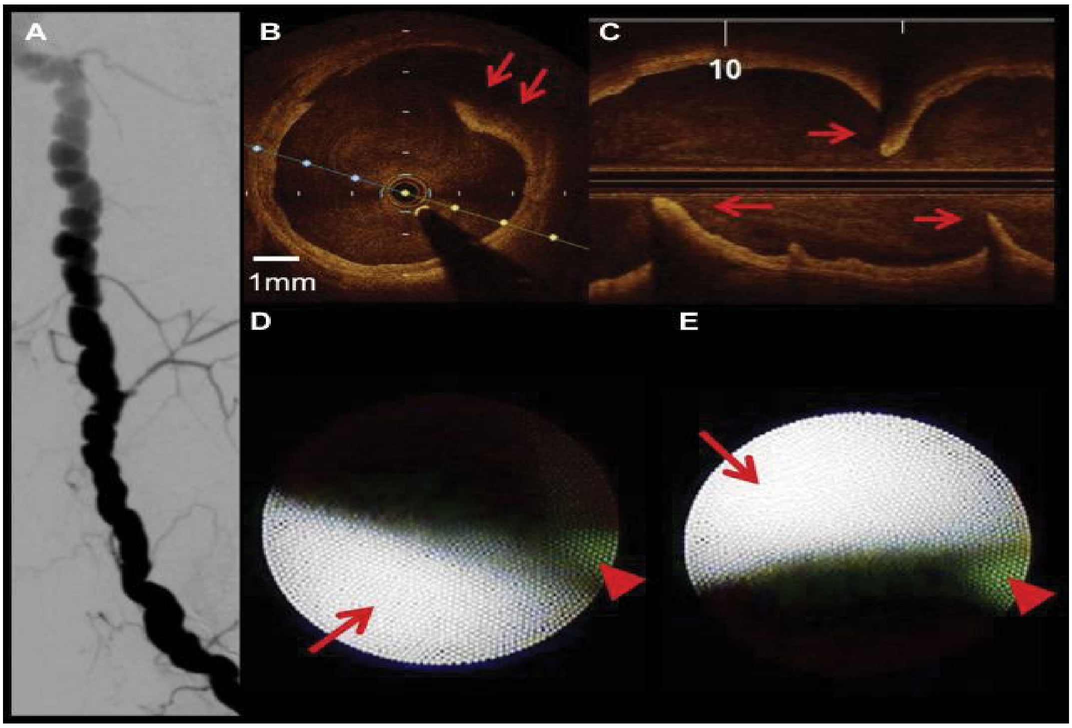

Figure 17: A) Angiography of the left brachial artery revealed the classic "string of beads" appearance, suggestive of fibromuscular dysplasia [16]; B) Thickening of the middle layer of the artery is evident in the OCT scan [16]; C) Intraluminal projections resulting from regional thickenings can be observed [16]; D, E) Angioscopy following the OCT scan demonstrated the presence of fibrous webs in the lumen of the affected arterial segment [16].

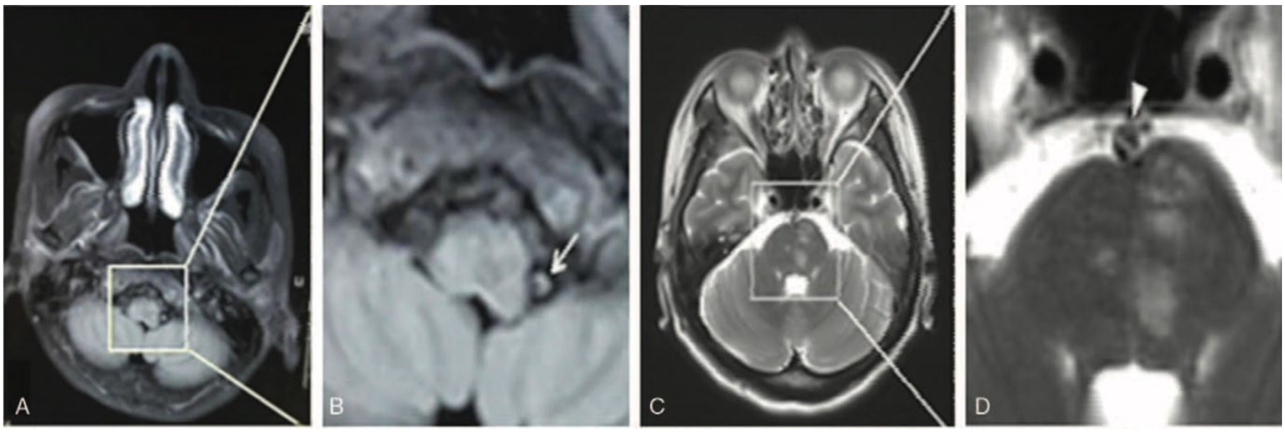

Figure 18: A) T1-Weighted MRI section showed a suspicious hyperintense mural hematoma [17]; B) A magnified image of a showing the mural hematoma; C) The following T2-weighted MRI section indicates an ischaemic stroke in the Pons with a double-lumen sign in the basilar artery; D) Magnified image of c depicting the "double-lumen" sign (arrowhead) [17].

external influences - medications or tobacco use - are also known potential causes of FMD [20].

Classification of FMD

As pathological specimens are not obtained as often as in the past, it is now uncommon to see the histopathologic classification of FMD [22]. Rather, angiography is used to categorize FMD into two types: (1) Multifocal FMD has segmental stenotic and dilated areas, which is called the 'string of beads' sign. The histologic types, medial and perimedial fibroplasia, correlate pathologically to multifocal FMD. (2) Focal FMD appears as a single tubular or circumferential stenotic lesion. It is pathologically equivalent to intimal fibroplasias [23] (Figure 21).

Pathophysiology

FMD is a non-inflammatory and non-atherosclerotic disease characterized by disturbances to the structure of medium-sized arteries [3]. FMD commonly manifests in renal and carotid arteries [24]. However, any vascular bed in the body can be affected, with its cells replaced by fibrous tissue with weak structural integrity [24,25]. As a result, the transformed arteries are more susceptible to damage, leading to hypertension, stenosis, dissection, or aneurysms [25].

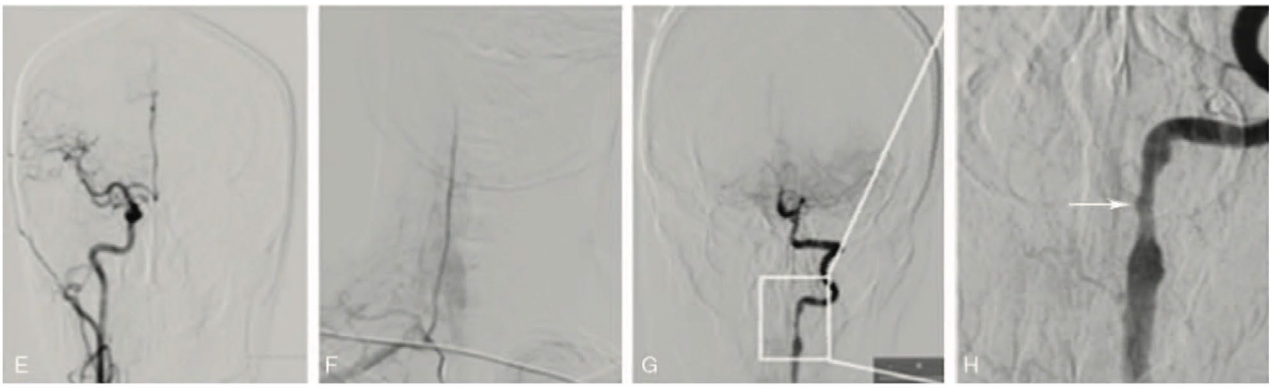

Figure 19: E) Digital subtraction angiography (DSA) revealed a normal right internal carotid artery, and F) A slender right vertebral artery [17]. G) The typical string of beads appearance was found in the left vertebral artery with concomitant stenosis in the second and third segments; H) A clear, magnified image of G (arrowhead) [17].

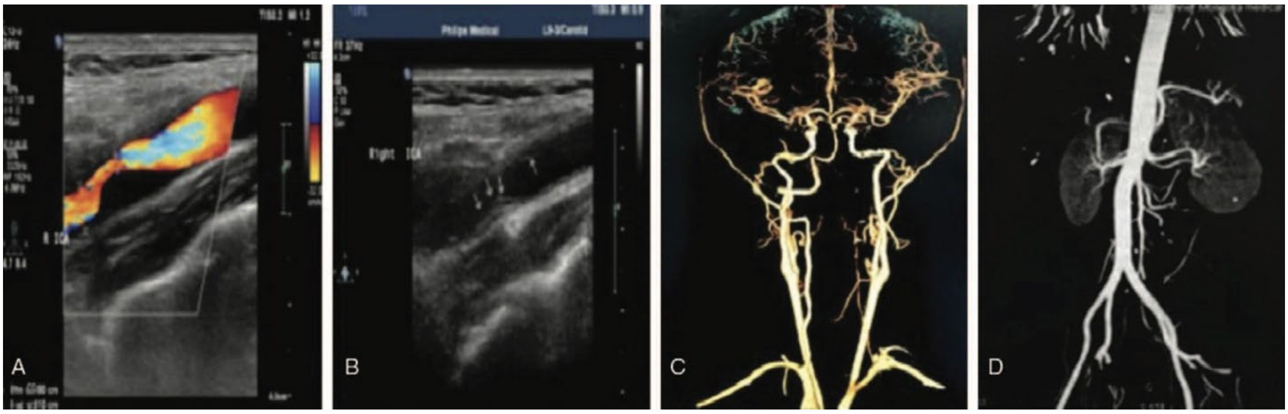

Figure 20: A) Carotid ultrasound revealed high-grade stenosis of the right C2 segment of the Internal Carotid Artery (ICA) [17]; B) The stenosis was suggested by the formation of hematoma after an intimal rupture (arrow); C) CT Angiography image (CTA) demonstrates collateral circulation; D) On follow-up, no further aneurysms were observed in the CTA of the abdominal aorta [17].

Media	Intima	Adventitia
Medial Fibroplasia Perimedial Fibroplasia Medial Hyperplasia	Intimal Fibroplasia	Peri-arterial Fibroplasia
MULTIFOCAL	**FOCAL**	

Figure 21: Previous FMD classification was based on which layer of the artery is affected.

10-20% of renal artery stenosis cases are caused by FMD [24]. The main renal artery is also frequently affected, whereas it is least probable for stenosis to occur in segmental arteries [26]. Secondary hypertension is notably and regularly a complication of renal artery FMD, as well as occasional headaches [24,27].

Treatment

There is no cure for FMD; however, treatments are available and tailored to control risk factors or any associated symptoms [24]. First, it is crucial that prevalent risk factors of FMD - hypertension, diabetes, and high cholesterol - are checked upon diagnosis and regularly tested [3]. Furthermore, environmental factors like tobacco usage should be avoided [3]. Without complications, it is likely that an individual would not require interventional treatment [3]. Antiplatelet therapy is regularly employed to prevent blood clots, with 72.9% of FMD patients adopting this treatment [27]. Other medications to treat headaches or hypertension are usually provided [24].

With FMD, blood vessels tend to narrow and thus affect blood flow [20]. Percutaneous transluminal angioplasties (PTA) are often recommended as a solution to re-establish blood flow in affected arteries [20]. It is unclear whether stent placement in an angioplasty procedure ameliorates cardiovascular problems. Stents may only be utilized in severe cases [3]. Reconstructive surgery is another possibility for patients, depending on the severity of the condition [3]. Typically, the objective is to restore blood flow by eliminating the obstructed area or by creating a bypass [3]. For complications like aneurysms that must be controlled to avoid a potentially fatal rupture, a less invasive angiogram-based procedure is an appropriate option [20].

The rationale for lessons learned

FMD is often under diagnosed and has a delay to diagnosis of around four years. Most patients live a healthy and nearly unaffected life, although this is not guaranteed [28]. Recently, there have been several reports of women who appeared healthy but acquired sudden coronary artery dissection due to their undiagnosed FMD [20]. Furthermore, one study examined the recurrence of this condition with CORAL (Cardiovascular Outcomes in Renal Atherosclerotic Lesions) patients and surprisingly found that it was present in 5.8% of their patients [29]. Overall, ambiguity prevails in the pathogenesis of the disease, and FMD, therefore, remains misunderstood [30].

Summary

Fibromuscular dysplasia is a rare disease defined by more than the string of beads appearance. It can develop in various blood vessels of the body and lead to pseudoaneurysms and dissections [24]. In order to detect FMD, imaging techniques like angiography and intravascular ultrasound are commonly employed [11].

Conflict of Interest

The authors declare that there is no conflict of interest.

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