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RESEARCH ARTICLE

Diagnostic and Therapeutic Challenges of Fibromuscular Dysplasia Affecting Peripheral Arteries in Rare Systemic Presentations

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Abstract: Fibromuscular dysplasia (FMD) is a rare, non-atherosclerotic, and non-inflammatory vascular disorder predominantly affecting medium-sized arteries, most commonly the renal and carotid arteries. However, peripheral arterial involvement, though uncommon, can lead to significant clinical challenges due to its atypical presentations and diagnostic complexity. This review explores the diagnostic and therapeutic challenges associated with FMD in peripheral arteries, particularly in rare systemic presentations. The clinical manifestations of peripheral FMD are often nonspecific, including claudication, limb ischemia, or hypertension, which can mimic other vascular disorders, leading to delayed or misdiagnosis. Imaging remains central to diagnosis, with catheter-based angiography considered the gold standard for detecting the characteristic "string of beads" appearance, although non-invasive modalities such as duplex ultrasonography, computed tomography angiography, and magnetic resonance angiography are increasingly utilized for screening and follow-up. The rarity of peripheral artery involvement further complicates timely recognition and highlights the need for heightened clinical awareness. Therapeutic strategies are largely individualized, balancing symptom severity, lesion location, and risk of complications. Medical management focuses on blood pressure control and antiplatelet therapy, while endovascular interventions such as percutaneous transluminal angioplasty and selective stenting are considered for significant stenoses. Surgical revascularization remains a last-resort option for cases refractory to endovascular therapy. Despite advances, no curative treatment exists, and disease recurrence necessitates long-term surveillance. The absence of large randomized controlled trials limits evidence-based standardization, underscoring the importance of multidisciplinary care and ongoing research. Recognizing the unique diagnostic hurdles and optimizing individualized therapeutic approaches are crucial for improving patient outcomes in peripheral FMD. This review emphasizes the need for further studies on pathophysiology, genetic predisposition, and long-term management strategies to enhance clinical understanding and refine treatment protocols for this rare systemic manifestation.

Keywords: Fibromuscular dysplasia, Peripheral arteries, Vascular disease, Diagnosis, Therapeutic challenges, Endovascular treatment.

INTRODUCTION

Fibromuscular dysplasia (FMD) is a rare, non-atherosclerotic, and non-inflammatory vascular disorder that primarily affects medium-sized arteries. Characterized by abnormal cellular growth within the arterial wall, FMD leads to stenosis, aneurysm formation, dissection, or arterial tortuosity. The disease most commonly involves the renal and carotid arteries, but it can affect almost any arterial bed, including peripheral arteries. Histologically, FMD is classified into medial, intimal, and adventitial types, with medial fibroplasia being the most prevalent. Clinically, patients may remain asymptomatic for years, and when symptoms do appear, they are often nonspecific, complicating early recognition. Advances in imaging techniques, including duplex ultrasonography, computed tomography angiography (CTA), and magnetic resonance angiography (MRA), have improved detection rates, yet challenges remain due to the disease's heterogeneity and subtle presentations [5].

Emerging evidence suggests that FMD may have a higher prevalence in patients with autoimmune conditions, including rheumatoid arthritis (RA). RA, a chronic systemic inflammatory disorder primarily affecting joints, is associated with accelerated vascular remodelling and endothelial dysfunction. These vascular changes may predispose RA patients to secondary arteriopathies, including FMD [6]. Although data are limited, studies have reported an increased incidence of FMD among RA cohorts compared to the general population, highlighting a potential interplay between chronic inflammation and vascular dysplasia.

Understanding this association is essential for clinicians, as early recognition of FMD in RA patients may prevent severe complications such as limb ischemia, aneurysmal rupture, or hypertension secondary to renal artery involvement.

Early diagnosis and management of FMD in peripheral arteries are critical for improving patient outcomes and preventing long-term morbidity. Peripheral FMD can manifest as claudication, ischemic pain, or non-healing ulcers, which often mimic other vascular or musculoskeletal disorders. Delayed diagnosis may lead to progressive arterial stenosis, aneurysm formation, or thromboembolic events [7]. Timely identification using imaging modalities, combined with a high index of clinical suspicion, allows for appropriate intervention. Management strategies range from medical therapy, including blood pressure control and antiplatelet agents, to endovascular procedures like balloon angioplasty or stenting, with surgical revascularization reserved for refractory cases. Early intervention not only alleviates symptoms but also mitigates the risk of life-threatening complications, emphasizing the importance of vigilance in high-risk populations, particularly those with systemic inflammatory disorders such as RA.

DIAGNOSTIC CHALLENGES

Beyond the diagnostic overlap with RA, another challenge in identifying fibromuscular dysplasia (FMD) is its heterogeneous presentation. While the classic "string-of-beads" angiographic finding is well described, not all patients exhibit this pattern. Some may instead have focal stenoses or atypical arterial irregularities, which can be difficult to distinguish from atherosclerotic plaques or inflammatory vasculitis lesions. Moreover, the distribution of FMD is variable although renal and carotid arteries are most commonly affected, peripheral involvement can occur in iliac, mesenteric, and extremity vessels, broadening the spectrum of possible presentations and complicating recognition in patients with pre-existing systemic disease such as RA [8].

Clinical vigilance is particularly important when patients with RA present with atypical vascular symptoms, such as unilateral limb claudication, refractory enema, or neurologic manifestations attributable to impaired perfusion. Unlike musculoskeletal pain from RA, vascular symptoms often follow exertion, have a segmental distribution, and may progress despite adequate control of joint inflammation. Laboratory markers also provide little help in distinguishing these conditions elevated inflammatory markers may reflect RA activity rather than an underlying vascular process, while their absence does not rule out FMD [9].

Imaging therefore remains central to diagnosis, yet even with advanced modalities, pitfalls exist. Duplex ultrasound may detect flow turbulence suggestive of stenosis, but findings can be subtle and dependent on operator expertise.

Table 1: Imaging Modalities for Diagnosing FMD in Peripheral Arteries

Modality	Advantages	Limitations	Role
Duplex Ultrasound	Non-invasive, no radiation/contrast	Operator-dependent, limited in deep/tortuous vessels	First-line screening/monitoring
CTA	High resolution, maps extent, shows "string-of-beads"	Radiation, iodinated contrast risk	Common diagnostic tool
MRA	No radiation, good for young patients	Lower resolution, gadolinium issues in renal failure	Alternative to CTA
DSA	Gold standard, allows treatment	Invasive, radiation, contrast risks	For uncertain cases or planned angioplasty

This table 1 summarizes the strengths and limitations of the main imaging modalities for diagnosing fibromuscular dysplasia (FMD) in peripheral arteries, most commonly affecting the renal and carotid vessels. Duplex ultrasound is often used as the initial test because it is non-invasive, inexpensive, and free of radiation or contrast, making it suitable for both screening and follow-up. However, its accuracy is highly operator-dependent and reduced in patients with obesity, deep-seated vessels, or complex vascular anatomy. CT angiography (CTA) is widely used as a diagnostic tool because it provides excellent spatial resolution and clearly demonstrates the characteristic "string-of-beads" appearance of FMD while also mapping the extent of disease. Its disadvantages include radiation exposure and the need for iodinated contrast, which may cause nephrotoxicity or allergic reactions. MR angiography (MRA) is a valuable alternative in younger patients and those in whom radiation should be avoided, as it uses magnetic fields rather than X-rays. Although it is generally safer, its resolution is lower than CTA, and gadolinium-based contrast agents are contraindicated in patients with advanced renal failure due to the risk of nephrogenic systemic fibrosis. Digital subtraction angiography (DSA) remains the gold standard, offering the highest resolution images and dynamic assessment of blood flow, as well as the unique advantage of enabling simultaneous therapeutic intervention such as percutaneous transluminal angioplasty. Nonetheless, DSA is invasive and carries risks related to arterial puncture, contrast exposure, and radiation. In clinical practice, non-invasive imaging with

CTA or MRA is typically used for diagnosis and disease mapping, while DSA is reserved for cases where uncertainty remains or when endovascular treatment is planned.
CTA and MRA provide detailed anatomic assessment but may still miss early or mild disease, particularly in distal branch vessels. Furthermore, radiologists unfamiliar with the characteristic appearance of FMD may misinterpret findings as atherosclerosis, especially in older patients or those with RA-associated cardiovascular risk factors. For these reasons, digital subtraction angiography retains value not only as a diagnostic reference standard but also as a therapeutic tool, offering the opportunity for immediate balloon angioplasty in appropriately selected cases [10].

Management decisions hinge on accurate differentiation between FMD, atherosclerosis, and vasculitis. In FMD, endovascular therapy often provides durable symptom relief and improved perfusion, whereas immunosuppressive regimens used in vasculitis would be ineffective and potentially harmful. Conversely, mistaking RA-associated vasculitis for FMD could delay necessary systemic treatment, leading to irreversible tissue ischemia or organ damage. This underscores the need for integrated care models in which rheumatologists collaborate closely with vascular medicine specialists and interventional radiologists to tailor diagnostic and therapeutic strategies to individual patients [4]. Future directions in the field include the development of standardized imaging protocols for peripheral FMD, improved non-invasive biomarkers to differentiate vascular disease mechanisms in RA, and increased awareness of FMD as a potential comorbidity in autoimmune populations. Early recognition and intervention remain critical, as timely diagnosis not only reduces morbidity from ischemic complications but also prevents unnecessary escalation of RA therapy in patients whose symptoms are driven by an unrecognized vascular disorder [11].

Distinguishing FMD from other vascular diseases in RA patients adds another layer of complexity. RA is associated with accelerated atherosclerosis, vasculitis, and increased thromboembolic risk, all of which can produce arterial stenosis or irregular vessel contours similar to FMD. Differentiating FMD from these conditions is essential because management strategies differ significantly FMD often responds to targeted endovascular or surgical interventions, whereas atherosclerosis and RA-related vasculitis require systemic therapy. Careful correlation of clinical history, laboratory findings, and multimodal imaging is therefore crucial to accurately identify FMD and avoid unnecessary or inappropriate treatment. Multidisciplinary collaboration between rheumatologists, vascular specialists, and radiologists is often necessary to navigate these diagnostic challenges effectively.

Therapeutic Challenges

When conservative management is insufficient, endovascular and surgical interventions become the mainstay of therapy. Percutaneous transluminal angioplasty (PTA) without stenting is widely regarded as the preferred revascularization technique for FMD because the disease typically involves non-atherosclerotic, elastic vessels that respond well to balloon dilatation. PTA has demonstrated high technical success rates with immediate improvement in arterial flow and symptomatic relief. However, restenosis can occur, particularly in patients with diffuse or multifocal disease, necessitating repeat intervention or long-term surveillance. Stenting is generally reserved for cases with suboptimal angioplasty results, flow-limiting dissection, or recurrent restenosis, but its use remains controversial due to concerns about late thrombosis, fracture, and difficulty with future surgical options [12].

In selected patients with extensive arterial involvement or failure of endovascular approaches, surgical revascularization may be required. Bypass grafting, endarterectomy, or patch angioplasty can restore perfusion, particularly in large-calibre peripheral vessels. Although surgical outcomes are generally favourable in specialized centres, the procedures carry higher perioperative risk, longer recovery times, and potential complications related to both FMD pathology and comorbid rheumatoid arthritis (RA), including impaired wound healing or infection. Careful patient selection and multidisciplinary planning are therefore critical in determining candidacy for surgical intervention.

Long-term management extends beyond procedural intervention and requires vigilant follow-up. Regular vascular imaging often with duplex ultrasonography or CTA allows for monitoring of treated lesions and early detection of new stenoses, aneurysms, or dissections. The risk of recurrence or progression highlights the chronic nature of FMD, emphasizing the importance of patient education regarding symptom recognition and adherence to follow-up schedules. Additionally, aggressive management of cardiovascular risk factors, such as smoking cessation, lipid control, and optimization of RA-related systemic inflammation, contributes significantly to long-term vascular health [1] [13].

The coexistence of RA introduces unique management complexities. Immunosuppressive therapies commonly used in RA, such as glucocorticoids or biologic agents, may influence vascular remodelling, healing capacity, and infection risk after endovascular or surgical procedures. Close collaboration between rheumatologists, vascular surgeons, and interventional radiologists is therefore essential to balance the competing demands of controlling systemic inflammation while minimizing vascular complications.

Emerging areas of research in FMD management include the use of advanced imaging modalities, such as intravascular ultrasound and optical coherence tomography, to better characterize lesion morphology and guide intervention. There is also interest in identifying genetic or molecular markers that may predict disease progression or therapeutic response, which could eventually lead to more targeted medical therapies. Until such advancements are realized, the cornerstone of care remains early recognition, individualized treatment strategies, and coordinated multidisciplinary follow-up to preserve arterial function and improve quality of life in affected patients.

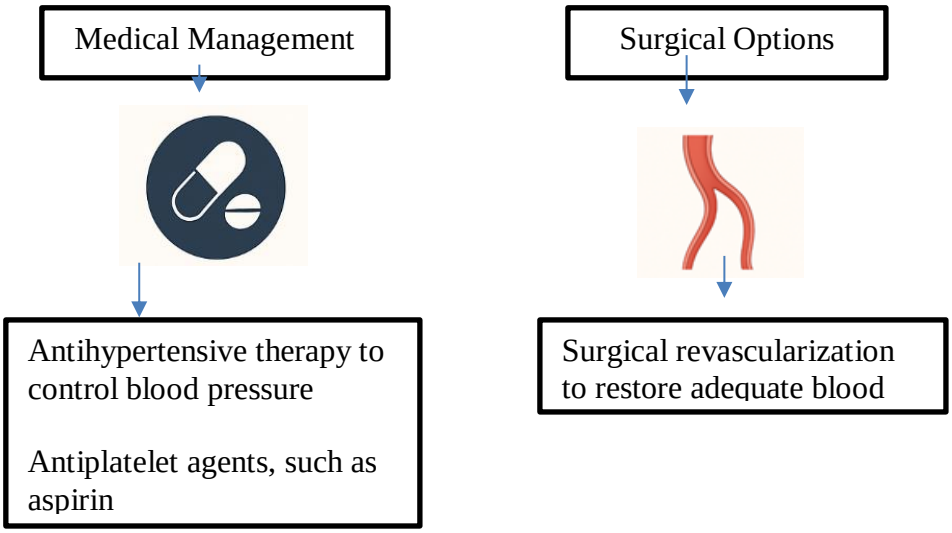

Figure 1: Therapeutic Strategies and Challenges in Managing Fibromuscular Dysplasia of Peripheral Arteries

This figure 1 illustrates the primary therapeutic approaches for managing fibromuscular dysplasia (FMD) in peripheral arteries, highlighting the challenges associated with each method. On the left, **medical management** involves pharmacological strategies, including antihypertensive therapy to maintain controlled blood pressure and antiplatelet agents, such as aspirin, to prevent thrombotic events [14]. In the centre, **endovascular interventions** are depicted with a balloon angioplasty procedure, which aims to dilate stenotic arterial lesions and restore blood flow without open surgery. On the right, **surgical options** are shown, emphasizing revascularization procedures that physically bypass or repair affected arterial segments to ensure adequate perfusion. Together, the figure underscores the spectrum of therapeutic modalities from conservative drug therapy to invasive procedures each tailored to the severity and location of arterial involvement in FMD patients.

The progression of FMD in patients with rheumatoid arthritis (RA) can also be influenced by the medications used to manage RA. Disease-modifying antirheumatic drugs (DMARDs) and biologic therapies, while effective in controlling systemic inflammation, may have indirect effects on vascular health [2] [15]. For example, chronic corticosteroid use is associated with accelerated atherosclerosis and endothelial dysfunction, which could exacerbate arterial stenosis in FMD. Similarly, immunosuppressive therapies may alter vascular remodelling or mask inflammatory markers, complicating the assessment of disease activity and progression. Therefore, clinicians must carefully balance RA treatment regimens with the potential impact on FMD, emphasizing the importance of a multidisciplinary approach.

Surgical and endovascular interventions remain the cornerstone for managing symptomatic or high-risk FMD in RA patients. Percutaneous transluminal angioplasty is the preferred first-line intervention for stenotic lesions in peripheral arteries, offering minimally invasive relief of ischemic symptoms. In select cases, stent placement may be required to maintain vessel patency, particularly in recurrent or complex lesions. Surgical revascularization, such as bypass grafting, is reserved for patients with severe or refractory disease, or when endovascular approaches are not feasible. The presence of RA complicates these interventions due to altered vascular integrity, increased risk of post-procedural complications, and potential interactions with systemic medications. Consequently, individualized treatment planning, careful procedural selection, and long-term follow-up are essential to optimize outcomes in this unique patient population.

CASE STUDIES

The pathophysiology of FMD in the context of rheumatoid arthritis (RA) remains poorly understood, but several mechanisms may explain the clinical overlap and heightened diagnostic complexity. RA is associated with chronic systemic inflammation, accelerated atherosclerosis, and immune-mediated vasculitis, all of which can alter vascular integrity and predispose to arterial pathology. In contrast, FMD is a non-inflammatory, non-atherosclerotic arteriopathy characterized by abnormal cell proliferation within the arterial wall, leading to stenosis, aneurysm formation, or dissection. While the two conditions are mechanistically

distinct, their clinical manifestations ischemic limb pain, swelling, or neurologic symptoms can appear strikingly similar. This overlap increases the likelihood of misclassification, particularly in patients with established RA where musculoskeletal causes are often presumed first [16].

Another diagnostic challenge arises from the rarity of FMD in peripheral arteries compared to renal or carotid involvement. In RA patients, vascular symptoms may be misattributed to peripheral neuropathy, vasculitis, or small-vessel occlusion from thromboembolic disease. Conventional laboratory tests offer little discriminatory power, as inflammatory markers such as ESR or CRP reflect systemic disease activity rather than the presence of FMD. Similarly, autoantibody profiles (e.g., rheumatoid factor, anti-CCP) provide information about RA severity but are not relevant to FMD, emphasizing the reliance on imaging modalities for accurate diagnosis [17].

Therapeutic strategies for patients with RA and FMD must balance vascular intervention with optimal control of systemic inflammation. Conservative management with antiplatelet agents (e.g., aspirin or clopidogrel) is generally recommended to reduce the risk of thromboembolic events, while antihypertensive therapy is crucial for patients with renal artery involvement. Endovascular therapy, most often balloon angioplasty without stenting, is the preferred intervention in symptomatic peripheral FMD, offering high rates of technical success and sustained symptom improvement. However, procedural risks such as vessel dissection, restenosis, or aneurysm formation must be weighed carefully, particularly in RA patients who may be on long-term corticosteroids or biologics that affect wound healing and vascular remodelling.

Long-term surveillance is essential due to the progressive and often multifocal nature of FMD. Serial imaging with duplex ultrasound, CTA, or MRA allows for early detection of new stenoses, dissections, or aneurysms. The presence of RA adds complexity, as disease flares, medication adjustments, and systemic cardiovascular risk all contribute to vascular vulnerability. A multidisciplinary approach ensures that treatment plans are comprehensive—addressing not only local vascular pathology but also systemic inflammation, cardiovascular risk reduction, and rehabilitation to preserve mobility and quality of life [18].

Emerging research suggests that advanced imaging techniques, such as high-resolution vessel wall MRI and intravascular ultrasound, may improve diagnostic precision by differentiating between inflammatory vasculitis, atherosclerosis, and non-inflammatory arteriopathies like FMD. Additionally, there is growing interest in the genetic underpinnings of FMD, with recent studies identifying potential associations with loci involved in extracellular matrix regulation and vascular development. Understanding whether RA patients possess unique susceptibility factors for FMD, or whether the two conditions intersect merely by chance, represents an important area for future investigation.

In summary, FMD in RA patients presents a unique diagnostic and therapeutic challenge due to overlapping clinical features, shared vascular complications, and the influence of systemic inflammation on vascular health [3]. Case-based evidence highlights the value of early recognition, personalized treatment strategies, and vigilant long-term follow-up. Strengthening awareness of this association among rheumatologists and vascular specialists may lead to earlier detection, reduced complications, and improved patient outcomes. Ultimately, collaborative care models and advances in imaging and molecular research hold the promise of refining diagnosis and optimizing management in this complex patient population.

CONCLUSION

Fibromuscular dysplasia (FMD) in the context of rheumatoid arthritis (RA) represents a rare but clinically significant overlap of vascular and rheumatologic pathology. The coexistence of these two conditions poses diagnostic dilemmas because RA itself often presents with vascular manifestations such as vasculitis, accelerated atherosclerosis, and microvascular dysfunction. These overlapping features can obscure the recognition of FMD, particularly in its early stages. Patients may present with intermittent claudication, ischemic pain, or limb swelling, which can easily be mistaken for musculoskeletal pain or neuropathy related to RA. Such diagnostic ambiguity often leads to delays in appropriate imaging, resulting in underestimation of the true prevalence and severity of FMD in RA patients. Therefore, heightened clinical suspicion is critical, especially in patients with atypical vascular symptoms or those unresponsive to conventional RA management. From a diagnostic standpoint, imaging plays a pivotal role, but each modality carries specific strengths and limitations. Duplex ultrasonography is often employed as a first-line, non-invasive test, though it is highly operator-dependent and less effective in visualizing deep or tortuous arterial segments. Computed tomography angiography (CTA) provides high-resolution visualization and can demonstrate the classic "string-of-beads" appearance associated with FMD, yet it involves radiation exposure and iodinated contrast, which may be contraindicated in patients with renal impairment a not infrequent complication in RA. Magnetic resonance angiography (MRA) avoids ionizing radiation but may fail to capture subtle stenoses or fibrotic lesions. Catheter-based angiography, while invasive, remains the gold standard for definitive diagnosis and therapeutic planning, allowing simultaneous intervention when needed. In RA patients, balancing the diagnostic yield against potential risks such as contrast nephropathy or vascular injury requires a tailored, multidisciplinary decision-making process. Therapeutic strategies for

peripheral FMD in RA are equally complex. Unlike atherosclerotic disease, FMD lacks a universally accepted medical regimen, and current management primarily revolves around antiplatelet therapy to reduce thrombotic complications, as well as strict control of modifiable vascular risk factors. In patients with coexisting RA, immunomodulatory drugs further complicate the picture, as some therapies (e.g., corticosteroids) may exacerbate vascular fragility or impair wound healing after interventions. Endovascular treatments, such as percutaneous transluminal angioplasty (PTA), often yield favourable outcomes in renal or carotid FMD, but their success in peripheral artery involvement is more variable, with restenosis and procedural complications posing significant concerns. Surgical reconstruction is reserved for refractory cases, yet it carries high perioperative risks, particularly in patients with systemic inflammation, joint deformities, or compromised functional reserve. Thus, treatment planning requires careful balancing of RA disease activity, vascular lesion characteristics, and overall patient comorbidities. A multidisciplinary approach is essential for optimizing outcomes in this dual disease entity. Rheumatologists, vascular specialists, interventional radiologists, and primary care physicians must collaborate to create individualized care pathways. Coordination ensures that disease-modifying antirheumatic drugs (DMARDs) and biologics are used judiciously alongside vascular interventions, minimizing drug–device interactions and reducing cumulative vascular risk. Regular follow-up with imaging surveillance is critical, given the unpredictable course of both RA and FMD. Beyond monitoring for progression or recurrence of arterial stenosis, clinicians must remain vigilant for complications such as aneurysm formation, thrombosis, or limb ischemia, which can significantly impact quality of life. Patient education also plays a vital role, empowering individuals to recognize early warning signs of vascular compromise and to adhere to lifestyle modifications such as smoking cessation, exercise, and optimal blood pressure control. Looking ahead, research into the pathophysiological links between chronic systemic inflammation and arterial dysplasia is of paramount importance. It has been hypothesized that persistent inflammatory activity in RA may predispose to vascular remodelling and smooth muscle proliferation, thereby potentiating FMD. Identifying biomarkers or genetic signatures that predict susceptibility could revolutionize risk stratification and early detection. Additionally, future studies should evaluate the long-term efficacy and safety of interventional procedures specifically in RA-associated FMD, as current evidence is largely extrapolated from non-RA populations. There is also a pressing need for novel pharmacological approaches, such as targeted agents that address both inflammatory pathways and vascular remodelling mechanisms. Large-scale, multicentre registries and clinical trials will be crucial in establishing standardized guidelines for diagnosis, treatment, and surveillance.

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