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Leriche Syndrome in 2025: what has changed in a century?

Francesco Stilo, Vincenzo Catanese, Nunzio Montelione, Alessandro Ciolli, Francesco Spinelli

INTRODUCTION

Leriche Syndrome represents a significant manifestation of aortoiliac occlusive disease (AIOD), a condition that has long intrigued and challenged medical professionals. The British anatomist and surgeon John Hunter first recognized the implications of occlusion at the aortic bifurcation in the late 18th century, laying the foundation for subsequent explorations in vascular pathology.[1] In the early 20th century, Dr. René Leriche provided a comprehensive understanding of the syndrome that now bears his name,[2] correlating anatomical findings related to atherosclerosis with clinical manifestations, particularly emphasizing their profound impact on limb perfusion and overall quality of life (Figure 1.1).

EPIDEMIOLOGY

Historical overview

Historically, Leriche Syndrome has predominantly affected older male populations, with early studies indicating that it typically presents in individuals over the age of 50.[3, 4] Many remain asymptomatic until they experience significant vascular compromise, underscoring the need for heightened awareness and early detection methods.[5] In the past, the lack of advanced diagnostic tools often led to misdiagnosis, as symptoms were frequently attributed to normal aging rather than to an underlying occlusive disease. Currently, the mortality and morbidity rates associated with Leriche Syndrome are approximately 4.5-5.0% and 18-20%, respectively.[4, 6]

Current trends

Contemporary epidemiological data indicate a notable demographic shift in patients affected by Leriche Syndrome, a rare condition with an incidence of approximately 1 in 12,000 individuals suffering from aortic occlusions, as highlighted in an autopsy study.[7] Recent reports suggest

Figure 1.1. Photograph taken during Michael DeBakey's visit at the clinic of Dr. Leriche in Strasbourg between 1935-1936. René Leriche is in the front row, second from the right, while at his left, in the middle of the front row is Michael DeBakey.

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an increasing incidence among younger populations, particularly those under the age of 50, a trend that correlates directly with rising rates of lifestyle-related conditions such as obesity, diabetes, hypertension, and dyslipidemia.[8-11] Determining the true prevalence of Leriche Syndrome remains challenging, as many patients may be asymptomatic; however, population studies have estimated a prevalence rate of approximately 3%.[12-14]

Sex-related disparities in prevalence have begun to diminish, as increasing recognition of the condition among women—who have historically been underrepresented in clinical studies—has led to more accurate identification and reporting.[15] Research indicates that women may develop more severe clinical manifestations at lower degrees of stenosis compared to their male counterparts.[16, 17]

Geographic variations significantly influence the epidemiology of Leriche Syndrome. In regions with high smoking rates and poor dietary habits, such as parts of Eastern Europe, the incidence of atherosclerotic conditions remains elevated. Conversely, areas with effective public health interventions aimed at reducing smoking prevalence and promoting healthier lifestyles—such as those in Scandinavia—have reported significant declines in the incidence of atherosclerosis.[18]

Socioeconomic status has a marked effect on the prevalence of atherosclerotic diseases. Studies indicate that individuals in lower socioeconomic brackets are at greater risk of developing advanced stages of peripheral artery disease (PAD). The evolving landscape of social

determinants of health has also influenced the epidemiology of Leriche Syndrome, with factors such as income inequality and education level significantly affecting both prevalence and outcomes. Health disparities related to race and ethnicity further shape the epidemiology of AIOD. Research has shown that Black and Hispanic populations in urban areas tend to present with advanced PAD at younger ages compared to their White counterparts, often due to historical inequities in access to healthcare resource.[19-21]

ETIOLOGY

The primary etiology of Leriche Syndrome remains atherosclerosis,[22, 23] which is characterized by pathological changes in arterial walls due to lipid accumulation, inflammation, and fibrous tissue formation. Our understanding of how atherosclerosis drives the pathology of Leriche Syndrome has evolved significantly. In the early 1900s, the mechanisms behind atherosclerotic plaque formation were largely speculative, with a primary focus on lifestyle factors such as diet and smoking.[3] Recent research has shed light on the complex biological processes that drive the development of atherosclerosis, including the role of endothelial dysfunction, which is now recognized as a key factor in the initiation and progression of atherosclerotic lesions. Chronic inflammation, exacerbated by risk factors such as obesity and metabolic syndrome, further aggravates vascular injury and promotes plaque formation.[24]

The risk factor landscape has shifted considerably over the past century. While hypertension, hyperlipidemia, and tobacco use remain significant contributors to atherosclerosis, recent studies have highlighted the impact of diabetes—particularly type 2 diabetes—as a critical risk factor. Emerging factors, such as air pollution and sedentary lifestyles, are also being recognized as contributors to the development of atherosclerosis. Furthermore, non-atherosclerotic etiologies, including large-vessel vasculitis (*e.g.*, Takayasu arteritis), have become increasingly relevant in the differential diagnosis of Leriche Syndrome, particularly among younger patients and specific ethnic groups.[25, 26]

AIOD is primarily caused by atherosclerotic disease; however, in some cases, acute thromboembolism or arteritis may be the predominant pathological issue. This uncommon condition typically begins at the aortic terminus and the origins of the common iliac arteries, progressively advancing both proximally and distally.[27] Table 1.I summarizes the various etiological scenarios.

CLASSIFICATION

AIOD can be classified using several systems. One such classification, based on the anatomical location of the atherosclerotic lesions, is presented in Table 1.II. Approximately 10% of patients with aortoiliac occlusion exhibit type 1 “localized” disease, which is most commonly observed in younger individuals, smokers, and females. Type 2 disease is more extensive, involving the femoral bifurcation. Type 3 disease is characterized by the most diffuse lesion presentation and is typically encountered in older individuals, diabetics, and males. These patients are also likely to present with associated atherosclerosis in the coronary, cerebral, or visceral arteries.[28]

Table 1.I. Etiology of Leriche's Syndrome: atherosclerotic and non-atherosclerotic etiologies.

Atherosclerotic etiologies
Endothelial dysfunction
Lipid accumulation
Inflammation
Fibrous tissue formation
Lifestyle factors
Diet
Smoking
Hypertension
Hyperlipidemia
Hyperglycemia
Diabetes (particularly type 2)
Hyperhomocysteinemia
Modifiable risk factors
Hypertension
Diabetes mellitus
Smoking
Hyperlipidemia
Hyperglycemia
Hyperhomocysteinemia
Non-modifiable risk factors
Age
Gender (men)
Ethnicity (non-Hispanic Black individuals)
Family history
Non-atherosclerotic etiologies
Large-vessel vasculitis (*e.g.*, Takayasu arteritis)
Arteritis
Acute thromboembolism

Table 1.II. Classification system of aortoiliac occlusive disease-based on anatomical location of the lesions.

Class	Vessels of involvement
Type I	Infrarenal abdominal aorta and common iliac arteries
Type II	Infrarenal abdominal aorta, common iliac arteries, external iliac arteries and femoral (common femoral) bifurcation
Type III	Infrarenal abdominal aorta, common iliac arteries, external iliac arteries, femoral (common femoral) bifurcation, popliteal, or tibial arteries

In 2007, the Trans-Atlantic Inter-Society Consensus (TASC) published the TASC II document, which introduced an important classification system for AIOD. As shown in Table 1.III, the TASC II classification categorizes AIOD into four types (A-D). This system differs from previous models by placing less emphasis on anatomical location and instead classifying lesions based on their shape, distribution, and severity.[29, 30] Types A and D represent the two extremes of disease severity. Type A is characterized by the most focal lesions with the shortest lesion length, whereas Type D encompasses the most diffuse, extensive, and complex lesions.

CLINICAL PRESENTATION

The clinical presentation can vary depending on the level of occlusion and the extent of collateral circulation. It ranges from asymptomatic disease to limb-threatening ischemia.[9] Patients with asymptomatic distal abdominal aortic occlusion are often diagnosed incidentally during medical examinations.[31, 32] The clinical spectrum ranges from mild claudication, characterized by muscle pain or cramping during physical activity that resolves with rest, to critical limb ischemia, a severe condition defined by persistent ischemic rest pain, ulcers, or gangrene, which requires urgent medical intervention to prevent irreversible

Table 1.III. TASC II Inter-Society Consensus on Peripheral Arterial Disease.

Type A lesions	Unilateral or bilateral stenoses of CIA
	Unilateral or bilateral single short (≤3 cm) stenosis of EIA
Type B lesions	Unilateral CIA occlusion
	Single or multiple stenosis totaling 3-10 cm involving the EIA not extending into the CFA
	Unilateral EIA occlusion not involving the origins of internal iliac or CFA
Type C lesions	Bilateral CIA occlusions
	Bilateral EIA stenoses 3-10 cm long not extending into the CFA
	Unilateral EIA stenosis extending into the CFA
	Unilateral EIA occlusion that involves the origins of internal iliac and/or CFA
	Heavily calcified unilateral EIA occlusion with or without involvement of origins of internal iliac and/or CFA
Type D lesions	Infra-renal aortoiliac occlusion
	Diffuse disease involving the aorta and both iliac arteries requiring treatment
	Diffuse multiple stenoses involving the unilateral CIA, EIA, and CFA
	Unilateral occlusions of both CIA and EIA
	Bilateral occlusions of EIA
	Iliac stenoses in patients with AAA requiring treatment and not amenable to endograft placement or other lesions re- quiring open aortic or iliac surgery

CIA: common iliac artery; EIA: external iliac artery; CFA: common femoral artery; AAA: abdominal aortic aneurysm.

tissue damage.[33] Acute worsening or onset of symptoms can occur following thrombosis of chronically diseased vessels or an embolic event originating from the aortic or iliac lesions, most often presenting as acute limb ischemia or distal embolization (commonly referred to as "blue toe syndrome").

This progressive disease often manifests in male patients as a classic triad of symptoms: claudication, weak/absent femoral pulses, and erectile dysfunction.[2, 34-37] It is estimated that up to 30% of affected men may experience erectile dysfunction due to inadequate perfusion of the internal pudendal arteries. However, sexual dysfunction—such as hypoactive sexual desire disorder, dyspareunia, and vaginismus—can also affect women. This symptom results from atherosclerosis involving the iliac vessels, which reduces blood flow to the vulval, vaginal, and clitoral regions. Another explanation for reduced sexual function in women with Leriche Syndrome is ischemic damage to the pudendal nerve, which is responsible for innervating the genital organs.[8, 15] The location of the symptoms or pulselessness depends on the extent of the vessels affected and collaterals formed but will typically include hip and buttocks pain accompanied by absent or diminished femoral pulse.[38] Today, most patients presenting with AIOD exhibit diffuse disease that affects multiple levels of the vascular tree, often in conjunction with femoropopliteal or infrageniculate occlusive disease.

Patients with isolated AIOD tend to be younger, with a higher prevalence of smoking and hypercholesterolemia, and typically have a normal life expectancy. In contrast, patients with extensive

multilevel disease are often older, more likely to have diabetes and hypertension, and tend to have increased comorbidities, including cerebrovascular, coronary, and visceral atherosclerosis.[39-41]

COMPENSATORY MECHANISMS

Chronic occlusions significantly impair arterial inflow to the pelvis and lower extremities, progressively reducing blood supply. The chronic nature of AIOD also encourages the development of extensive collateral networks.[42] When these compensatory collaterals develop sufficiently, claudication symptoms remain tolerable, allowing for effective nonoperative management over many years. Unfortunately, undiagnosed and untreated Leriche Syndrome can lead to catastrophic consequences and mortality.[43]

The primary compensatory networks develop from the lumbar and hypogastric feeding vessels and connect to the circumflex iliac, hypogastric, femoral, and profunda arteries. Additional collaterals that arise in more severe cases include the internal mammary artery-to-inferior epigastric connection and the superior mesenteric artery-to-inferior mesenteric artery and hemorrhoidal artery pathway. This latter connection includes the arc of Riolan and the meandering mesenteric artery. The intervisceral system pathway, which involves anastomoses between the superior and inferior mesenteric arteries, connects through the sacral plexus or the inferior rectal artery to the internal iliac artery, compensating for the blood supply to the pelvic cavity and wall. It is important to recognize the presence of such significant collateral networks, as they should be preserved during surgical reconstruction.

A principal collateral pathway is the Winslow pathway, which involves the internal thoracic artery, originating from the subclavian artery (Figure 1.2A). This pathway connects through the upper abdominal arteries to the inferior epigastric artery and eventually links to the external iliac artery to the external iliac artery (Figure 1.2B). The prevalence ranges from 95% to 100%, manifesting either bilaterally or unilaterally and playing a crucial systemic compensatory role in patients with distal occlusions.[44, 45]

Additional collateral formations include branches of the lower thoracic aorta, such as the intercostal and subcostal arteries, or those of the abdominal aorta, such as the lumbar arteries. These vessels connect to the iliac or femoral arteries *via* the deep circumflex iliac artery, enhancing blood supply to the lower limbs. Collateral formation often follows the superior mesenteric artery into the rectal artery supply, reaching the internal iliac or obturator artery and the common femoral artery. Other proximal reconstitution routes include the costal arteries to the superior gluteal arteries.

MORBIDITY AND MORTALITY OF LERICHE SYNDROME

AIOD can manifest in different forms, primarily as acute or chronic Leriche Syndrome. The chronic form is characterized by stenosing atherosclerosis as the underlying pathophysiological condition. Adequate collateral circulation is crucial in preserving blood flow to the abdomen and legs. The rate at which the condition progresses to irreversible ischemia is influenced by the extent of the thrombotic process and the availability of preexisting collateral pathways.[46]

Arterial emboli represent the most prevalent cause of acute aortoiliac occlusion,[47] with approximately 80% originating from cardiac sources and traveling to the extremities.[48] Notably, over 70% of cardiac embolic events can be attributed to atrial fibrillation.[47] Other contributors to cardiac embolism include heart valve abnormalities, endocarditis, atrial myxomas, and dilated cardiomyopathy.[49, 50] Additionally, 10-20% of all peripheral thromboembolic events are classified as noncardiac in origin, which can include conditions such as aortoiliac bifurcation aneurysms, aortic dissection, atherosclerotic plaques, or, on rare occasions, tumors.[51]

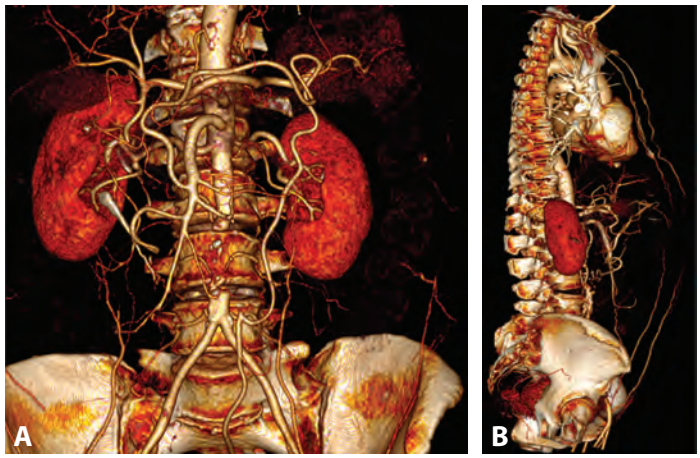

Figure 1.2. The volume rendering of CT-angiography proves Winslow pathway involving the internal thoracic artery (A), and the upper abdominal arteries (B).

CT: computed tomography.

The mortality rate associated with acute limb ischemia is approximately 30% within the first 30 days following diagnosis.[52] The presence of aortoiliac disease, along with peripheral arterial disease at any level, is a strong predictor of future cardiovascular events such as myocardial infarction.[53]

Based on numerous studies, the mortality rate is 30% at 5 years and 50% at 10 years, among such patients, with most deaths due to cardiovascular complications.[30] A large study from the Cleveland Clinic evaluated 1000 patients (381 with lower extremity ischemia) with coronary arteriography before elective vascular surgery and found normal coronary arteries in only 10% of them.[54] Among these patients, 28% had severe triple-vessel coronary artery disease or worse. Cardiology consultation is essential before undertaking any form of open surgical arterial intervention, and most patients should be started on beta-blockers and statins preoperatively. Ultrasound screening for coexisting carotid artery disease is appropriate in patients with AIOD. Smoking is common in this population; therefore, chest radiographs and evaluation for chronic obstructive pulmonary disease are warranted. Additionally, the presence and severity of diabetes, renal dysfunction, and coagulopathy should always be assessed in these patients.

CONCLUSIONS

In conclusion, the clinical presentation of Leriche Syndrome is multifaceted and has evolved significantly over time. Its characteristic symptoms and associated risk factors require a comprehensive understanding by healthcare professionals to ensure timely diagnosis and effective management. As the demographic profile of affected patients continues to shift, ongoing research and increased awareness are essential to improving outcomes in individuals with this complex vascular condition. René Leriche was a pioneer of vascular surgery whose research, teaching, and clinical practice have served as a model for generations of surgeons. His influence endures to this

day. Beyond his surgical skill, Leriche was also a critical thinker and a compassionate physician, qualities that earned him a lasting place in the history of medicine.

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