


Review Article

Acquired Arteriovenous Fistulas: A Comprehensive Review of Etiology, Anatomical Distribution, Pathophysiology, Clinical Presentation, Diagnosis, and Management

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Abstract

Background: Acquired arteriovenous fistulas (AVFs) are abnormal communications between arteries and veins that most commonly arise following trauma or iatrogenic vascular injury.

Objective: To review the etiology, anatomical distribution, pathophysiology, clinical presentation, diagnosis, and management of acquired arteriovenous fistulas.

Methods: A narrative literature review of articles published between 2019 and 2025 was conducted using PubMed, MEDLINE, EBSCO, Google Scholar, and Dialnet.

Results: Acquired AVFs most frequently result from penetrating trauma or percutaneous vascular procedures, particularly involving the femoral vessels. Clinical manifestations depend on the size and location of the fistula and range from local thrill and bruit to high-output heart failure. Doppler ultrasound and computed tomography angiography are the main diagnostic tools. Management includes conservative monitoring, endovascular therapy, or surgical repair.

Conclusion: Early recognition and appropriate imaging are essential for optimal management. Endovascular techniques have expanded treatment options, although surgical repair remains necessary in selected cases.

Key words

Arteriovenous fistula; Vascular trauma; Endovascular therapy; Vascular surgery; Diagnostic imaging.

Introduction

The first description of an arteriovenous fistula (AVF), defined as an abnormal communication between an artery and a vein, is attributed to William Hunter, who in 1761 reported two cases of brachial arteriovenous fistulas following attempted phlebotomy. He additionally described the associated “whistling” sound (bruit) and the palpable vibratory sensation (thrill).

Arteriovenous fistulas were rarely recognized and diagnosed until the widespread use of firearms during nineteenth-century military conflicts, which significantly altered the magnitude and complexity of battlefield injuries. Subsequent military conflicts, along with the rise in blunt, penetrating, and iatrogenic trauma among civilians, have resulted in an increased incidence of vascular injuries, consequently leading to a greater number of acquired arteriovenous fistulas.

Methodology

A narrative literature review including publications from 2019 to 2025 addressing acquired arteriovenous fistulas in adult populations was conducted. Eligible sources included case reports, case series, observational studies, and review articles published in English or Spanish.

Publications focusing exclusively on congenital arteriovenous malformations or dialysis-related arteriovenous fistulas were excluded.

The literature search was performed using the following electronic databases: PubMed, MEDLINE, EBSCO, Google Scholar, and Dialnet. Relevant articles were selected based on their clinical relevance to the etiology, anatomical distribution, pathophysiology, clinical presentation, diagnosis, and management of acquired arteriovenous fistulas.

The search strategy included combinations of the following keywords: “arteriovenous fistula”, “vascular trauma”, “iatrogenic vascular injury”, and “endovascular therapy”.

Definition

Classically, Rutherford defines an arteriovenous fistula as a direct communication between an artery and a vein that bypasses the capillary network. This definition can be expanded by noting that such a connection results in the diversion of arterial blood directly into the venous circulation, representing a transition from a high-resistance arterial system to a low-resistance venous system and thereby bypassing the capillary beds [1, 2].

Acquired arteriovenous fistulas can be classified into six types:

- **Type 1:** Simple arteriovenous communication.
- **Type 2:** Interposition of an aneurysmal sac between an artery and a vein.
- **Type 3:** Arteriovenous communication associated with an arterial aneurysm.
- **Type 4:** Arterial aneurysm with an aneurysmal sac communicating with the fistula.
- **Type 5:** Presence of an aneurysmal sac located either proximal or distal to the fistula.
- **Type 6:** Arteriovenous fistula located within an aneurysmal sac [3].

Etiology and Incidence

Arteriovenous fistulas may be single or multiple and may be congenital or acquired. Acquired arteriovenous fistulas include those secondary to trauma, whereas a small proportion occur spontaneously, usually due to erosion or rupture of an atherosclerotic or infected aneurysm into an adjacent vein [1].

The different types of arteriovenous fistulas are described below:

Traumatic Arteriovenous Fistulas

In the civilian population, penetrating trauma resulting from surgical injury, gunshot wounds, or stab wounds accounts for the majority (61.7%) of vascular injuries. These injuries involve the upper and lower extremities in approximately 22% and 20% of cases, respectively, whereas thoracic and abdominal arteriovenous fistulas account for about 4%. Stab wounds represent 63% of cases and involve cervicomediastinal arteries in 54% to 75% of patients, most commonly affecting the common carotid artery (21%). Gunshot wounds account for 26% of cases, while blunt trauma represents only 1%. Carotid-cavernous fistulas may occur as a result of basilar skull fractures [2, 4].

Iatrogenic Arteriovenous Fistulas

Iatrogenic AVFs arise as a result of percutaneous cardiac and vascular procedures, central venous catheterization, orthopedic procedures involving the lumbar spine and knee, diagnostic kidney and liver biopsies, and percutaneous insertion of biliary and urinary drainage catheters. The right common femoral artery remains the most common site of iatrogenic injury in the lower extremities [1].

A sharp instrument may penetrate in either direction, that is, from the artery to the vein or from the vein to the artery. Under normal conditions, this communication seals spontaneously; however, in the presence of certain risk factors, spontaneous closure may not occur, leading to the formation of an arteriovenous fistula as a result of the puncturing procedure. Risk factors associated with iatrogenic arteriovenous fistulas include female sex, hypertension, the use of anticoagulants or antifibrinolytic agents, increased body mass index (BMI), and advanced age [2].

Spontaneous Arteriovenous Fistulas

These are caused by rupture or erosion of an atherosclerotic, inflammatory, or mycotic aneurysm of the aorta or iliac artery into the inferior vena cava, iliac vein, or a retroaortic left renal vein. Less frequent cases include syphilitic aortitis, HIV-associated arteritis, and connective tissue disorders such as Marfan syndrome and Ehlers-Danlos syndrome [1].

Arteriovenous Fistulas in Specific Locations

Coronary Arteriovenous Fistulas

Coronary arteriovenous fistulas are anomalous communications between a coronary artery and the great vessels or cardiac chambers. Their prevalence ranges from 0.1% to 0.2%, most commonly detected as an incidental finding on coronary computed tomography angiography. Approximately 50–60% originate from the right coronary artery, while 25–42% arise from the left anterior descending artery. Most patients are asymptomatic; however, symptomatic cases may present with dyspnea, fatigue, or angina, and may develop complications such as aneurysm rupture, ischemic heart disease, pulmonary hypertension, heart failure, an increased risk of endocarditis, and sudden cardiac death. In general, the closer the anatomical location of the fistula to the heart, the greater the hemodynamic impact [5, 6].

Clinically, the classic triad consists of a continuous cardiac murmur (crescendo-decrescendo), a left-to-right shunt, and the presence of a tortuous coronary artery. Acquired coronary fistulas may be either spontaneous - resulting from atherosclerosis, coronary vasculitis, or myocardial infarction - or iatrogenic, such as those occurring after coronary stent placement or coronary artery bypass grafting [7].

Electrocardiographically, these fistulas have been associated with atrial fibrillation and ventricular tachyarrhythmias. Coronary computed tomography angiography is the preferred diagnostic modality because it allows detailed delineation of cardiac anatomy; however,

invasive coronary angiography remains the gold standard. Pharmacologic management is based on the use of beta-blockers to reduce myocardial oxygen demand, along with antiplatelet therapy [5, 7].

Interventional management is indicated for large fistulas regardless of the presence or absence of symptoms, as well as for moderate or small fistulas associated with symptoms such as ischemia, arrhythmias, ventricular dysfunction, or endarteritis. Indications for surgical ligation include large symptomatic high-flow fistulas, multiple communications or drainage sites, tortuous or aneurysmal fistulas, the need for concomitant distal bypass grafting, and sizeable vascular branches that may not be suitable for embolization. In contrast, indications for percutaneous transcatheter closure include fistulas with a proximal origin, a single drainage site, a non-tortuous vessel, an extra-anatomic termination distant from normal coronary arteries, older patients at high surgical risk, and the absence of other significant cardiac pathologies [7].

Oral Cavity Fistulas

These can occur in the tongue, lips, and palate. On examination, a purplish nodular lesion with delayed healing may be observed. The most common areas are the lateral border and the dorsum of the tongue due to intense mechanical contact and vulnerability to trauma such as car accidents, falls, toothpick injuries, or iatrogenic causes. Diascopy can be performed by applying uniform pressure with a glass slide to observe blanching of the lesion. Doppler ultrasound with a linear transducer can confirm the presence of arterial and venous blood flow, and these lesions can be managed by embolization of the responsible artery [8].

Carotid Arteriovenous Fistulas

These lesions consist of a fistulous communication between the carotid artery and the internal jugular vein or its tributaries. They most commonly result from stab wounds,

gunshot injuries, therapeutic or diagnostic catheterization, or blunt trauma to the skull or cervical spine. The main symptoms include tinnitus, a palpable thrill, an audible bruit, a pulsatile mass, and venous dilation, which may progress to ischemia or heart failure [9].

Computed tomography angiography is the diagnostic modality of choice, as it allows precise localization and size assessment prior to intervention. Carotid duplex ultrasonography may be used as an initial diagnostic study; however, its limitations in evaluating distal carotid lesions should be considered. Endovascular intervention is the preferred treatment for these lesions, although a staged hybrid approach may also be performed to achieve initial hemodynamic stabilization through endovascular means followed by definitive surgical repair. Open surgical intervention is generally reserved for fistulas larger than 8 mm [9, 10].

A carotid-cavernous fistula is defined as any abnormal communication between the carotid artery and the cavernous sinus, which may lead to visual impairment and ophthalmoparesis. The principal causes include high-impact trauma (such as basilar skull fractures or severe traumatic brain injury), aneurysms or vascular malformations, and venous thrombosis. The most common symptoms include chemosis (94%), proptosis (87%), increased intraocular pressure (60%), cranial nerve III and VI palsies (54%), diplopia (51%), visual deterioration (28%), headache, and ocular pain. These manifestations are explained by increased pressure within the cavernous sinus, which directly affects cranial nerves III, IV, and V, with cranial nerve VI being the most frequently involved [11].

On physical examination, unilateral pulsatile proptosis may be observed, which in advanced stages can become bilateral. Auscultation may reveal an orbital bruit due to drainage through the superior ophthalmic vein. Increased intraocular pressure may lead to papilledema and

secondary glaucoma, ultimately causing loss of visual acuity due to ischemic damage. Treatment is typically performed through endovascular embolization using covered stents, metallic coils, or other embolic agents. Alternative management strategies include conservative treatment and stereotactic radiosurgery [11, 12].

Carotid–Cavernous Fistulas According to the Barrow Classification:

- **Type A:** Direct (high-flow) communication between the internal carotid artery and the cavernous sinus.
- **Type B:** Indirect communication between the cavernous sinus and the meningeal branches of the internal carotid artery within its cavernous segment.
- **Type C:** Communication between the cavernous sinus and branches of the external carotid artery.
- **Type D:** Communication between the cavernous sinus and meningeal branches of both the internal and external carotid arteries [11].

Vertebral Arteriovenous Fistulas

These typically occur between the vertebral artery, its muscular or radicular branches, and the internal jugular and/or vertebral veins. Iatrogenic injury during cervical spine surgery and internal jugular vein catheterization accounts for approximately 10% of cases. The remaining cases are associated with cervical spine fractures due to blunt trauma, chiropractic manipulation, closed head injuries, gunshot wounds, and stab wounds [1].

Axillary and Subclavian Arteriovenous Fistulas

Penetrating trauma to the neck and thorax is associated with injury to the subclavian vein (44%), the subclavian artery (39%), or both vessels (17%). Penetrating injuries to the axillary artery and fractures of the clavicle or first rib are rare causes of arteriovenous fistulas in this region [1].

Brachial, Radial, and Ulnar Arteriovenous Fistulas

Upper extremity trauma represents a significant proportion of all vascular injuries. In this region, the brachial and forearm arteries are involved in 10% to 22% of cases. The radial and ulnar arteries are frequently used for cardiac interventional access. Additionally, peripherally inserted central catheters (PICCs) should also be considered, as they may similarly be complicated by fistula formation [1].

Femoral Arteriovenous Fistulas

The main causes at this site include gunshot wounds, stab wounds, and long-bone fractures. Although uncommon, sporadic cases of spontaneous AVFs between the superficial femoral artery and vein have been described. Iatrogenically, arteriovenous fistulas occur slightly more frequently after interventional procedures than after diagnostic procedures. Fistulas involving the femoral and external iliac arteries are a rare complication of endovenous laser ablation for varicose veins. Most of these fistulas close spontaneously [1].

Inguinal arteriovenous fistulas can be classified into three categories: those located above the bifurcation of the common femoral artery (accounting for approximately 50% of cases), those located below the bifurcation of the common femoral artery, and indeterminate fistulas. The most common specific connection occurs between the common femoral artery and the superficial circumflex iliac artery [2].

Popliteal Arteriovenous Fistulas

These result from blunt trauma associated with comminuted fractures of the femur and proximal tibia, as well as from orthopedic knee procedures. The incidence of iatrogenic popliteal AVFs is expected to increase due to the more frequent use of popliteal access for interventions aimed at treating deep vein thrombosis and chronic venous insufficiency [1].

Tibial and Peroneal Arteriovenous Fistulas

These are recognized complications of comminuted fractures, balloon catheter thrombectomy, and tibial vessel atherectomy. The increasing use of retrograde tibial access to recanalize occluded lower-extremity arteries in patients with critical limb ischemia may raise the risk of AVF formation in these locations [1].

Aorto-Iliac Arteriovenous Fistulas

Rupture or erosion of an abdominal aortic aneurysm into the inferior vena cava accounts for most acquired aortocaval fistulas. Gunshot and stab wounds constitute the remaining 10% to 20%. Less commonly, iatrogenic injury during peripheral or cardiac arterial catheterization, lumbar disc surgery, and rarely blunt trauma, have been associated with aortocaval fistulas [1].

Iliac fistulas usually result from penetrating trauma or lumbar disc surgery. Anatomical variations, such as a low aortic bifurcation at L4–L5 and degenerative changes of the anterior spinal ligament, may contribute to vascular injury in this region [1].

Vascular injuries at the L3–L4 disc space predominantly affect the aorta and inferior vena cava, whereas most iliac vascular injuries occur during surgery at the L4–L5 and L5–S1 levels. The common iliac arteries, located immediately anterior to the L4–L5 disc space, are the most frequently injured vessels, with right-sided predominance [1].

Renal Arteriovenous Fistulas

Most renal AVFs occur following renal biopsy, but they may also result from trauma, surgery, percutaneous interventions, tumors, inflammation, or direct erosion of an aneurysm into a vein. Additional causes include percutaneous nephrostomy tube placement, laser lithotripsy, and massive ligation of the renal pedicle. Aortic and renal artery aneurysms may also erode into a retroaortic vein or renal vein [1, 13].

Splenic, Hepatic, and Mesenteric Arteriovenous Fistulas

Splenic AVFs may result from blunt or penetrating intra-abdominal trauma, massive ligation of the splenic pedicle, iatrogenic injury during splenoportography, and, rarely, erosion of a pancreatic pseudocyst into the splenic artery and vein [1].

Hepatic arteriovenous fistulas may be intrahepatic or extrahepatic. They most commonly result from penetrating or vehicular trauma but may also occur following percutaneous needle biopsy, transhepatic diagnostic catheterization, biliary drainage, or transjugular intrahepatic portosystemic shunt (TIPS) procedures. Other causes include hepatic artery aneurysms and carcinomas eroding into adjacent veins [1, 14].

Fistulas involving the superior and inferior mesenteric arteries are rare and generally result from penetrating trauma, iatrogenic injury, or massive vascular pedicle ligation during bowel resection [1].

Pathophysiology

The natural history of an arteriovenous fistula (AVF) is determined by the diameter of the involved artery and vein, the size and location of the fistulous communication, the adequacy of collateral circulation, and the competence of the distal venous valves [1].

Fistula Size and Flow

The larger the fistula, the greater the amount of blood “stolen” from the native arterial circulation, resulting in reduced or even reversed antegrade flow distal to the lesion and a marked increase in flow proximal to the fistula. Flow rates exceeding 350 mL/min are associated with failure of spontaneous closure [1].

Chronic Changes

Increased hemodynamic stress leads to attenuation of the arterial wall, accompanied by calcification and lipid deposition in the inflow

artery. These structural alterations result in elongation, tortuosity, and eventual aneurysmal dilation at the site of the fistula. The adjacent vein also becomes dilated, tortuous, and thickened - "arterialized" - with pulsatile flow. Structural changes in both the artery and vein become evident at approximately 2 months and are typically well established by 15 months; however, they appear to be reversible if the AVF is repaired within 2 years [1].

Blood flow is generally antegrade but may reverse (vascular steal phenomenon) in larger defects due to increased collateral flow distal to the fistula, resulting in peripheral arterial ischemia. Elevated venous pressure at the fistula site leads to distal venous dilation and valvular incompetence, which may cause edema, skin hyperpigmentation, varicose veins, and venous ulcers as a consequence of chronic venous insufficiency [4].

Cardiac Effects

The reduction in arterial blood volume and the increase in venous blood volume lead to an increase in venous return to the heart, a decrease in peripheral arterial pressure, and a compensatory increase in cardiac output. This alteration in arterial and venous volumes also activates the renin-angiotensin-aldosterone system, resulting in sodium and water retention and a further increase in circulating blood volume [4, 15].

In patients with large arteriovenous fistulas, the increased preload results in cardiomegaly and dilation of the aorta and inferior vena cava proximal to the fistula. These structural changes may develop over weeks to months or progress gradually over time [1].

If the heart is unable to compensate for the increased blood volume and hemodynamic burden, or if the condition remains untreated, these changes may ultimately lead to high-output congestive heart failure [1, 4].

Clinical Presentation

History

Symptoms in patients with large arteriovenous fistulas may result from increased venous return, diversion of arterial blood flow (ischemia), or venous hypertension (varicosities and edema). Peripheral fistulas typically produce local manifestations, whereas central fistulas are more likely to cause systemic symptoms, such as exertional dyspnea. Most patients with small fistulas remain asymptomatic [1].

The most common clinical findings include a palpable thrill, which may be perceived as a change in local sensation or as a vibrating sensation reported by the patient, a machinery-like bruit, or a pulsatile mass. In aortic and iliac arteriovenous fistulas, the acute onset of abdominal or lumbar pain is often the primary presenting symptom [1, 2].

Physical Examination

General

Patients with high-flow central arteriovenous fistulas may present with widened pulse pressure, tachycardia, hypotension, fatigue, and/or exertional dyspnea. Cutaneous findings may include ulceration, edema, gangrene, hyperpigmentation, and increased warmth of the skin overlying the fistula. A gallop rhythm or jugular venous distention may be present in patients who develop heart failure [4].

Neck and Upper Extremities

The presence of a rapidly expanding or pulsatile hematoma, carotid bruit or thrill, and dilated neck veins suggests a carotid-jugular fistula. Subclavian, axillary, and brachial AVFs may present with pulse deficits, blood pressure discrepancies, arm swelling, dilated venous tributaries, hand ischemia, or a pulsatile mass with an associated thrill and bruit. Radial and ulnar AVFs are usually asymptomatic and manifest as a thrill or bruit over a small pulsatile mass and, rarely, digital embolization [1].

Lower Extremities

The groin and lower extremities should be examined for evidence of a pulsatile or expanding hematoma and/or a thrill or bruit. The Nicoladoni-Branham sign consists of temporary compression of the artery proximal to the fistula, which should result in a decrease in heart rate and a reduction in pulse pressure. Leg swelling with dilated or varicose veins, pigmentation, and ulceration may be present in more chronic cases [16].

Thorax and Abdomen

Evaluation should include assessment for a pulsatile mass or prominent aortic pulsation with an associated abdominal thrill or bruit. Patients with spontaneous aortorenal fistulas may present with abdominal pain, hematuria, proteinuria, varying degrees of renal dysfunction, varicocele, and hemodynamic instability. Clinical findings in patients with visceral AVFs may include jaundice, hepatomegaly, splenomegaly, prominent venous collaterals, and epigastric bruits. Hematochezia and hematuria secondary to rupture of mucosal veins in the rectum and bladder may also occur [1].

Diagnosis

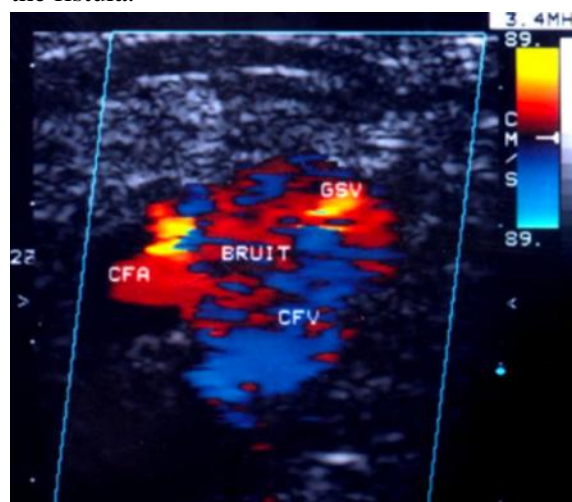
Plain radiographs should be obtained in all patients with suspected arteriovenous fistulas to evaluate for evidence of prior trauma (e.g., bullet or metallic fragments), abnormal calcifications, and signs of congestive heart failure. Subsequently, noninvasive imaging studies should be performed. Depending on their results, additional tests such as ankle-brachial indices or venous or carotid Doppler imaging may be indicated. Endoscopy may be warranted in patients with active gastrointestinal bleeding or a history of esophageal variceal bleeding or gastric ulcers. Differential diagnoses to consider include arteriovenous malformations, hemangiomas, and pseudoaneurysms [1, 4].

Doppler Ultrasound

Doppler ultrasound is the initial diagnostic and follow-up test of choice in lesions outside the

cranium, thorax, and abdomen. Characteristic findings include direct visualization of the fistulous communication and a color mosaic pattern at the level of the fistula as shown in **Figure - 1**. Ultrasound is a noninvasive imaging modality that does not involve ionizing radiation or contrast agents. However, its limitations include a high degree of operator dependence, prolonged examination times when evaluating large areas of interest, limited utility for lesions located in the skull, thorax, or abdomen, and reduced image quality due to factors such as surrounding hematoma, active bleeding at the puncture site, or patient anatomical characteristics [1, 4].

Figure – 1: Color mosaic pattern at the level of the fistula.



Ultrasound findings can be classified into major and minor criteria:

1. Major criteria

- Transition between low- and high-resistance arterial flow patterns.
- Arterialized high-velocity waveform in the venous flow.
- High-velocity turbulent flow at the site of the fistula.

2. Minor criteria

- Direct visualization of the communication between the artery and vein.

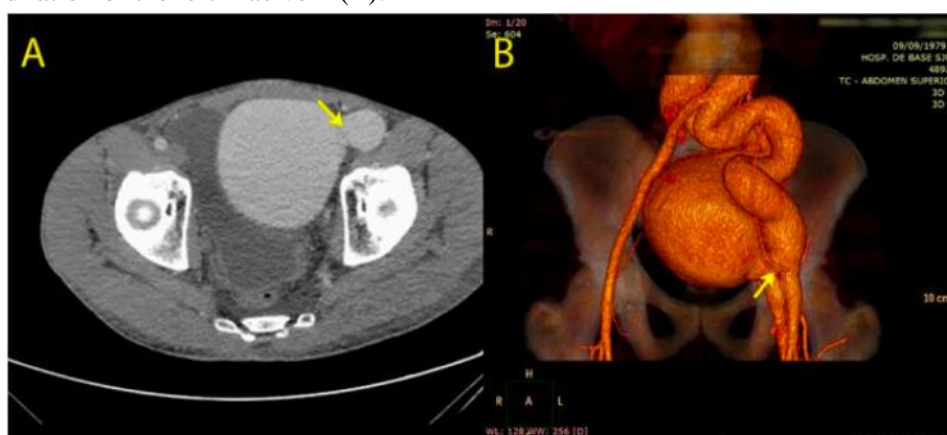
- b. Increased diameter of the arterial vessel.
- c. Focal venous dilation [4].

Computed Tomography Angiography

Computed tomography angiography (CTA) is a minimally invasive, accurate, and operator-independent diagnostic tool that allows for

repeated follow-up examinations. It is comparable to digital subtraction angiography, with a reported sensitivity of 90%–95% and specificity of 99%–100%. The main limitations of CTA include exposure to ionizing radiation and the risk of contrast-induced nephrotoxicity (**Figure - 2**) [1, 17].

Figure – 2: Axial view demonstrating a high-flow arteriovenous fistula arising from the left external iliac artery to the left external iliac vein (A); three-dimensional reconstruction showing aneurysmal dilation of the left iliac vein (B).



Magnetic Resonance Angiography

Magnetic resonance angiography (MRA) facilitates diagnosis by delineating the underlying defect, quantifying flow alterations, and defining the relationship between the fistula and adjacent soft tissue structures. Its principal advantages include the absence of ionizing radiation and a relatively favorable side-effect profile. However, its use is limited by difficulties in monitoring acutely ill patients, longer image acquisition times, and patient intolerance. Contrast-induced toxicity and the potential development of nephrogenic systemic fibrosis in patients with renal insufficiency remain concerns [1].

Digital Subtraction Angiography

Digital subtraction angiography (DSA) provides excellent spatial and temporal image resolution, allows rotational views, and is used for both diagnosis and treatment. Typical angiographic findings include early venous filling and failure to opacify distal arterial segments [1].

Management

The primary goals of therapy are closure of the fistula, restoration of normal hemodynamic flow, and reestablishment and maintenance of vascular continuity. Severe lower-extremity swelling and ulceration are managed with diuretics, limb elevation, and compression bandaging [1].

Conservative Treatment

Patients with small fistulas and normal cardiac function may be monitored with Doppler ultrasound for at least one year. Angiographically, 33%–90% of renal AVFs resolve spontaneously within 6 weeks to 4 years after diagnosis. Stable iatrogenic femoral AVFs demonstrate a 38%–81% probability of spontaneous closure within one month [1].

Ultrasound Guided Compression Therapy

Although proposed as first-line treatment for small iatrogenic AVFs, compression therapy demonstrates limited efficacy, is highly dependent on specific anatomical factors, and

carries a high failure rate and risk of complications. These limitations restrict its role as a definitive therapeutic option [1].

The approximate duration of compression is 10 minutes and is generally more effective during the first weeks following the development of the fistula, particularly in cases in which the onset can be clearly determined, such as after percutaneous catheterization. Both anticoagulation therapy and fistulas present for more than 2–3 weeks reduce the success rate of this technique [2].

Interventional Treatment

Indications for intervention include:

- **Arterial steal** caused by significant diversion of blood flow into the venous system, resulting in claudication or ischemia depending on the location of the fistula.
- **Significant edema or venous insufficiency** secondary to venous hypertension.
- **Heart failure** in cases of high-flow fistulas.
- **Progressive increase in fistula size or flow volume** during ultrasound surveillance.
- **Large fistula following penetrating trauma**, such as stab wounds or gunshot injuries.
- **Persistent iatrogenic arteriovenous fistula** lasting longer than 4 months [2].

Endovascular Therapy

Until recently, open surgery was the only option after failure of conservative management. However, continuous advances in endovascular techniques have significantly expanded their use. Endovascular therapy is the treatment of choice in high surgical risk candidates, stable elderly patients with suitable anatomy, and in cases where the AVF is located in surgically inaccessible regions such as distal carotid, intracranial lesions, or intraparenchymal fistulas. Endovascular management may be performed

through transcatheter embolization, covered stent placement, or a combination of both modalities. Temporary balloon occlusion may serve as a useful adjunct to control bleeding [1, 4].

Embolization

Available embolic agents include autologous clot, gelatin sponge, microfibrillar collagen, polyvinyl alcohol particles, metallic coils, detachable balloons, and liquid embolic agents such as n-butyl cyanoacrylate and ethylene-vinyl alcohol copolymer [1].

Selection of the embolic agent depends on vessel size, delivery catheter caliber, and the anticipated need for repeat embolization. Gelatin sponge (Gelfoam) is preferred for temporary occlusion, whereas metallic coils and particulate agents are used for permanent occlusion. Microcoils and particulate agents delivered through coaxial microcatheters are particularly suitable for small muscular and radicular AVFs involving cervical, renal, visceral, or extremity branches [1, 18].

The principal risk associated with transcatheter embolization is unintended embolization into arteries related to the fistula or into the pulmonary circulation, potentially resulting in parenchymal or limb ischemia, or pulmonary embolism. In high-flow fistulas, larger coils with high radial force are first deployed to provide a scaffold for smaller packing coils, thereby reducing the risk of migration and inadvertent embolization. A venous stent or an inferior vena cava filter may be used before or during embolization to prevent central venous embolism [1, 19].

Covered Stents

Covered stents may be used to exclude the fistula while preserving flow in the main vessel in large-diameter AVFs. Penetrating injuries of the innominate, carotid, and subclavian arteries are anatomically suitable for endovascular therapy [1].

Although technically feasible, covered stent placement for AVFs in the groin and around the knee joint has historically been avoided due to concerns regarding kinking or deformation in these mobile regions. Initially, it was thought that covered stent placement in the common femoral artery would limit future vascular access; however, shorter covered stents (12–28 mm) with expanded diameter ranges now allow precise AVF treatment without compromising femoral branches or precluding subsequent procedures [1].

Abdominal Endografts

Aortic endografts, initially developed for the treatment of thoracic and aortoiliac aneurysmal disease, are now used to manage AVFs involving the aorta, iliac arteries, and branches of the aortic arch. Descending thoracic aortic AVFs are rare but may be amenable to endovascular therapy when present. The main limitations of endograft use include aortic neck diameter, length and angulation, as well as the diameter and tortuosity of access vessels. Renal AVFs are also suitable for endovascular treatment [1].

The risk of air embolism and displacement of thrombotic material leading to pulmonary embolism has led some surgeons to recommend prophylactic placement of a removable inferior vena cava (IVC) filter or insertion of a secondary aortic prosthesis within the IVC to prevent this complication. Amplatzer vascular plug devices have been successfully used to occlude splenic, renal, mesenteric, subclavian, and popliteal AVFs, as well as those occurring after endovascular aneurysm repair in selected patients [1].

Surgical Repair

Surgical repair of arteriovenous fistulas is recommended for young and otherwise healthy patients, individuals with complex anatomy unsuitable for endovascular therapy, or when endovascular treatment has failed [1].

The procedure may be performed under general anesthesia, neuraxial anesthesia, or regional nerve block depending on operator preference. Ultrasound should be performed to confirm vessel patency and to mark the operative site [2].

Superficial veins should be ligated, and the proximal artery and vein are dissected to free them from the surrounding tissue in order to obtain vascular control using vessel loops and to identify the fistula. During fistula dissection, separation of the adventitial layer should be avoided, as this may weaken the arterial wall and complicate repair [2].

Once the fistula is divided, direct pressure is applied to achieve hemostasis, followed by repair with nonabsorbable vascular sutures, such as polypropylene (Prolene). After releasing arterial control, the distal pulse at the repair site should be assessed using Doppler ultrasound [2].

Local and Regional Flaps

Flaps in reconstructive surgery have become a therapeutic alternative when there is exposure of vital structures. Because they have their own blood supply, they represent an effective option for the management of complex wounds [20].

In a case reported at the “*Hermanos Ameijeiras*” *Clinical Surgical Hospital*, a patient with an arteriovenous fistula who experienced failure of endovascular therapy and subsequently developed a hemorrhagic complication with bone involvement of the second finger of the right hand underwent disarticulation with a flap using the McGregor technique (**Figure - 3**). This inguinal flap, vascularized by the superficial iliac artery, is used for lesions of the head, neck, trunk, and extremities (particularly the distal two-thirds of the forearm and the hand). It offers several advantages, including greater coverage in cases of extensive tissue loss, early mobilization, adequate functional recovery, and minimal cosmetic defect [20].

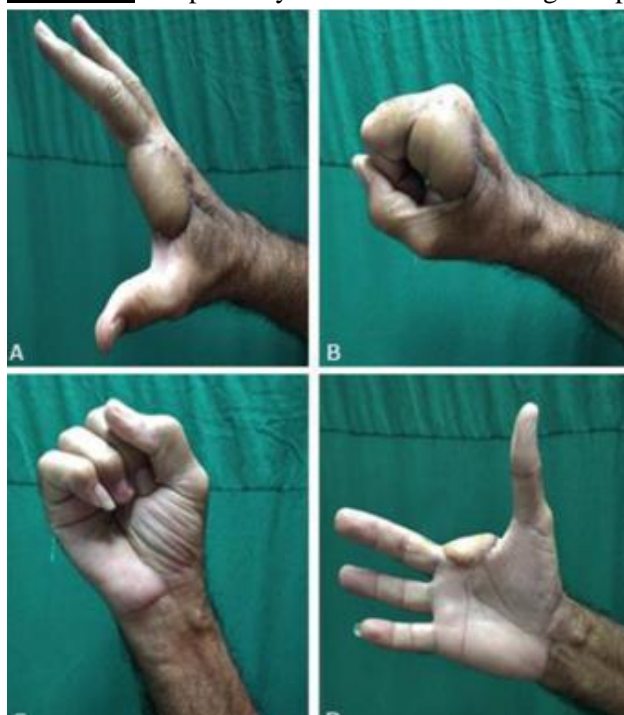
The flap was released after 21 days, and 30 days after release a complete recovery of limb function was observed (**Figure - 4**). These findings suggest that flap reconstruction

represents a valuable therapeutic alternative for the management of complex wounds of vascular origin, such as complicated arteriovenous fistulas [20].

Figure – 3: Flap using the McGregor technique.



Figure – 4: Flap 30 days after release showing complete recovery of limb function.



Conclusion

In symptomatic patients, repair of arteriovenous fistulas is associated with an almost immediate improvement in cardiac and pulmonary function. Normalization of renal function may be delayed by 48 to 72 hours. Younger patients with high-output congestive heart failure are expected to recover completely, whereas recovery in older

individuals with spontaneous arteriovenous fistulas is largely determined by preexisting comorbidities.

At present, no prospective randomized controlled trials compare endovascular therapy with surgical repair in the management of patients with arteriovenous fistulas. Available evidence is

limited to a small number of studies with relatively small patient cohorts. Early outcomes of endovascular therapy appear promising; however, long-term follow-up data remain limited.

Several vascular surgeons advocate endovascular therapy as the preferred treatment modality due to its minimally invasive nature, reduced blood loss, lower hemodynamic instability, applicability in surgically inaccessible locations, and lower morbidity and mortality rates.

Surgical repair, currently reserved for young and otherwise healthy patients, individuals with anatomy unsuitable for endovascular therapy, or cases in which endovascular management has failed, remains curative and durable, with reported patency rates of approximately 90% among surviving patients.

Current literature suggests that, despite its recognized advantages, endovascular therapy cannot yet be considered the universal standard of care for patients with aortic or iliac arteriovenous fistulas.

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