ASDIN white paper: Assessment and management of hemodialysis access-induced distal ischemia by interventional nephrologists

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Abstract
Although not common, hemodialysis access-induced distal ischemia is a serious condition resulting in significant hemodialysis patient morbidity. Patients with signs and symptoms suggestive of hand ischemia frequently present to the general and interventional nephrologist for evaluation. In order to care for these cases, it is necessary to understand this syndrome and its management. Most cases can be managed conservatively without intervention. Some cases requiring intervention may be treated using techniques within the scope of practice of the interventional nephrologists while other cases require vascular surgery. In order for the interventional nephrologists to evaluate and manage these cases in a timely and appropriate manner, practice guidelines are presented.

Keywords
Dialysis access, AV fistula, prosthetic grafts, hand ischemia, steal syndrome, interventional nephrology

Introduction
Hemodialysis access-induced distal ischemia (HAIDI) has been reported to have an incidence of 1%–8%. However, this is based on referred cases requiring surgical treatment. Its true frequency is dependent upon the symptom threshold used to qualify for the diagnosis. Systematic patient evaluation has shown that mild to moderate symptoms occur much more frequently. The reported incidence relates to the upper extremity and is the topic of this review. Access-related distal ischemia can also involve the lower extremity with serious consequences. An understanding of HAIDI and the appropriate role of the interventional nephrologist in its management is important when patients present with symptoms suggestive of HAIDI. Herein, the pathophysiology of the syndrome is discussed followed by recommendations for its diagnosis and management and the basis for the recommendations. These recommendations

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Pathophysiology of dialysis access-related ischemia

An arteriovenous (AV) access represents a non-anatomic communication created between a high-pressure system (artery) and a low-pressure vein with a low outflow resistance. The result is a marked derangement in physiology of the blood flow to the distal extremity. The low resistance of the vein diverts some of the blood destined for the extremity distal to the communication resulting in alteration of perfusion. These high-pressure (proximal) and low-pressure (distal) zones are interconnected by collateral arteries. Because direction of blood flow is dictated by pressure differences in these zones, distal hypoperfusion is prevented primarily by collateral blood flow. This pressure gradient also results in a reversal of blood flow in the inflow artery distal to the access anastomosis, referred to as vascular “steal phenomenon.” This phenomenon may be “complete,” when retrograde flow occurring during both systole and diastole, or “incomplete,” characterized by diastolic retrograde flow only presenting as “to-and-fro” or bidirectional blood flow. In the majority of patients with an arteriovenous fistula (AVF), distal vascular perfusion is maintained and the individual remains asymptomatic. In some cases compensatory collateral flow fails to maintain distal tissue arterial pressure leading to hypoperfusion and ischemia of the hand or fingers, a syndrome referred to as HAIDI. Although closely linked with vascular steal, the volume of flow diversion may not necessarily be the only cause of the syndrome and HAIDI can be seen even with low-volume blood flow diversion.

Five vascular zones are key elements in the hand/vascular access complex and are all important in the pathogenesis of HAIDI. The AV access plays a central role in the pathogenesis of HAIDI through several mechanisms. The low resistance of the AV access outflow tract results in a marked alteration of perfusion (velocity and pressure) in the distal extremity. Some loss of perfusion pressure may be caused by the difference in size and compliance of the AVF venous outflow tract compared to that of the inflow artery. Although there is a direct relationship between the level of access blood flow and HAIDI, it is not possible to predict the occurrence or severity of HAIDI based upon access blood flow. Individual variations in the effectiveness of compensatory mechanisms have a major effect.

When the access is first created, blood flow increases several-fold immediately and 10- to 20-fold over a relatively short period of time. Normal arteries are capable of accommodating this change through the process of remodeling resulting in an increase in diameter (maturational). Arteries with proximal or distal vascular disease may be incapable of undergoing this process, adversely affecting blood flow acutely or over time as arteriopathy progresses or develops. In addition, proximal arterial stenosis has been reported in 20%-30% of patients who present with distal hand ischemia. Collateral branches of major arteries of the upper extremity and hand create a robust compensatory collateral network to these areas, maintaining perfusion when their blood flow is compromised. Because of communications through the palmar arches, the ulnar artery acts as a collateral vessel for a radial artery-based AVF. These collateral vessels act to offset the deleterious effects of the steal phenomenon and prevent hand ischemia.
disease or incomplete palmar arches can adversely affect this function.\textsuperscript{14} The peripheral vascular bed is the high-resistance component of the vascular access complex. With the creation of an AV access there is often an immediate drop in pressure within the digital arteries that may or may not be symptomatic. This can be appreciated by assessing digital pressures.\textsuperscript{26} Existing or progressive microvascular disease involving the peripheral vascular bed results in further increase in resistance to blood flow and exacerbates the hand ischemia.\textsuperscript{1} Perfusion can also decrease if there is a progressive increase in the blood flow in a fistula over time. Thus, HAIDI may appear early following AV access surgery and may either worsen or even subside over time.\textsuperscript{27,28} Because of these differences, the natural history of HAIDI has been classified based upon time of development after AV access creation (Table 1).\textsuperscript{15,27,29}

### Prevention of HAIDI

The development of HAIDI is multifactorial, making prediction of its development in an individual patient uncertain. Nevertheless, an important part of vascular access planning is the application of measures directed toward its prevention. This involves an evaluation of risk factors (Table 2)\textsuperscript{12,14,30,31} and the clinical status of the individual patient. Although risk factors for the development of HAIDI are well-recognized, they have not been consistently validated in all studies.\textsuperscript{2,32–34} In some instances, patients may present with the need for creation of a new dialysis access having already had a prior episode of HAIDI. Certain demographic characteristics have been used to identify a patient as “high-risk”:\textsuperscript{30,35,36}

- Patients with profound atherosclerotic peripheral vascular disease (most often diabetic);
- Patients who have had previous amputations due to peripheral arterial disease;
- Patients who have had HAIDI with a prior dialysis access.

Digital-brachial index (DBI) has been advocated as a tool to predict the likelihood of HAIDI for patients who are at increased risk.\textsuperscript{12,37,38} Many patients can tolerate low finger perfusion and remain asymptomatic. Although clearly useful in risk evaluation, there is no value at which the development of HAIDI is inevitable.\textsuperscript{12,37,38}

Even with a high-risk patient, the creation of an AV access should not be automatically avoided without a thorough clinical evaluation. High-risk patients should be counseled preoperatively, their operative plans should be designed to reduce the risk of hand ischemia,\textsuperscript{37,39–41} and they should be observed closely postoperatively.\textsuperscript{42}

### Diagnosis of HAIDI

HAIDI can generally be diagnosed based upon signs and symptoms.\textsuperscript{6,43,44} Physical examination of the hand will reveal some combination of cold skin, pallor, cyanosis, diminished sensation, and ultimately, ulceration and gangrene. The radial and ulnar pulses are usually diminished.

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**Table 1.** HAIDI classification based upon time of onset.

<table>
<thead>
<tr>
<th>Classification</th>
<th>Time of onset</th>
<th>Clinical features</th>
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<tbody>
<tr>
<td>Acute</td>
<td>Within 24h</td>
<td>Conditions for development of HAIDI are present at time of access creation. Sudden overload of collateral circulation. Least frequent category. Most commonly seen with brachial artery–based AVG. Generally, mild signs and symptoms, but occasionally severe and rapidly progressive.</td>
</tr>
<tr>
<td>Subacute</td>
<td>&gt;24 h, &lt;1 month</td>
<td>Conditions for development of HAIDI are achieved as access flow progressively increases after access creation. Disproportionate maturation of vessels. Most commonly seen with brachial artery–based AVF. Generally, mild signs and symptoms, but occasionally severe and rapidly progressive.</td>
</tr>
<tr>
<td>Chronic</td>
<td>&gt;1 month, often months to years</td>
<td>Development related to comorbidities that appear or progress over time. Disproportionate maturation of vessels. Most common category and most commonly seen with brachial artery–based AVF. Generally, mild signs and symptoms, but with time ulceration and necrosis of tissue can occur.</td>
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HAIDI: hemodialysis access-induced distal ischemia; AVG: arteriovenous graft; AVF: arteriovenous fistula.

**Table 2.** Risk factors for HAIDI.

<table>
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<tr>
<th>Peripheral vascular disease</th>
<th>Macrovascular</th>
<th>Microvascular</th>
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<tbody>
<tr>
<td>Surrogates for peripheral vascular disease</td>
<td>Diabetes mellitus</td>
<td>Older age of patient</td>
</tr>
<tr>
<td>Use of brachial artery for anastomosis</td>
<td>Multiple previous vascular access procedures</td>
<td>Female gender</td>
</tr>
<tr>
<td>High access blood flow</td>
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</tbody>
</table>

HAIDI: hemodialysis access-induced distal ischemia.
or absent, but uncommonly may be normal despite obvious signs and symptoms. Manual occlusion of the access may augment the distal pulse and relieve or significantly decrease the symptoms of HAIDI, a finding which supports the diagnosis.

When HAIDI is associated with a distal radial-cephalic AVF, retrograde distal radial artery flow is a dominant etiological factor. The hand is often warm, and the symptoms of decreased perfusion are limited to the fingers. A prominent thrill may be palpable over the distal radial artery and over the ulnar artery in some cases. HAIDI involving only part of the hand and fingers may be seen when the palmar arch(s) is incomplete or occluded.

Decrease capillary refilling provides evidence of decreased peripheral perfusion. This is tested by pressing firmly on a fingernail to produce blanching and estimating the time required for return of normal color. With good digital perfusion, this should be less than 3 s; greater than 5 s is abnormal. Values between these two extremes are suspicious.

In equivocal cases, digital blood pressure evaluation is valuable both in supporting the diagnosis (Figure 2) and in following the progress of mild cases. This test is especially useful in distinguishing HAIDI from other conditions.

Digital pressure readings have been utilized in three ways—(1) basal digital pressure (BDP), digital pressure under basal conditions; (2) DBI, ratio of the BDP to the contralateral brachial artery systolic pressure; and (3) change in digital blood pressure (CDP) with AV access compression. BDP used in these assessments and measurements should be made with the hand at room temperature (20°C–22°C). A BDP <60 mmHg or a DBI <0.4 in a symptomatic patient supports the diagnosis of HAIDI. Although of considerable value in the diagnosis, it does not distinguish between mild and severe forms of the syndrome. It has been suggested that a return of BDP back toward normal with compression of the access (CDP test) indicates reversibility of the condition. In cases in which HAIDI is due to arterial disease, the BDP and DBI will be consistent with HAIDI, but the CDP will not show a significant change.

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### Differential diagnosis

Pre-existing conditions associated with hand pain such as arthropathy and diabetic neuropathy can generally be ruled out because of their presence prior to AV access surgery. Formulating a differential diagnosis for newly developed hand pain should take into consideration whether symptoms develop early or late after AV access creation. Hand pain, numbness, and tingling that begins immediately after access creation may be related to the surgery, secondary to soft tissue swelling, or the formation of a hematoma and typically resolves within the first 2 to 4 weeks. The differential diagnosis for symptoms that are more severe and/or last longer includes ischemic monomelic neuropathy (IMN) and carpal tunnel syndrome.

IMN occurs acutely after creation of an access and is characterized by excruciating pain in the hand occurring after AV access creation associated with the brachial artery and predominately with an arteriovenous graft (AVG). The hand is warm and shows no evidence of ischemic change. Neurologic hand symptoms and signs predominate, and all three nerves are involved. Nerve conduction studies can be used to confirm the diagnosis. Most physicians recommend immediate access ligation.

Carpal tunnel syndrome is common in dialysis patients. It may be recognized as a pre-existing condition, become manifest or exacerbated by AV access surgery, or develop chronically in relation to AV access creation. When related to vascular access creation, it generally occurs later with gradual onset. Although carpal tunnel syndrome is usually bilateral, it has been reported to be worse in the hand ipsilateral to the AV access. An electromyogram showing reduction of motor conduction can help to establish the diagnosis.

### Clinical evaluation

Once the diagnosis has been made, the patient should undergo a detailed clinical evaluation to determine the appropriate approach for management. The first step
should be to evaluate the patient’s clinical condition including comorbidity burden and degree of frailty. Many dialysis patients have a high comorbidity burden which increases with age. Comorbidities that define mortality risk may also place the patient at risk for distal hypoperfusion. As a result, patients who develop HAIDI tend to have a shortened life expectancy. Patient frailty is also common in the dialysis patient population. When planning for the management of HAIDI, the patient’s long-term prognosis should be considered by the treatment team.

**Clinical stage of syndrome**

The clinical spectrum of ischemia following routine AV access surgery is diverse. While placing patients into defined categories is somewhat subjective, a classification has been adopted for the clinical classification of the syndrome (Table 3). This system has important implications in the clinical management of individual patients (Figure 3).

The threshold required to justify the diagnosis of stage 1 HAIDI is largely subjective since many patients with a new AV access have mild symptoms. In these cases, there are no clear symptoms, only signs of mild ischemia which are often intermittent and frequently limited to the later part of dialysis session. These symptoms generally abate within a few weeks as tissue trauma associated with surgery resolves and compensatory mechanisms take place. Conservative symptomatic treatment may be beneficial—hand warming techniques such as wearing a glove or using blankets, reducing antihypertensive agents if appropriate, modifying the dialysis session, and performing hand exercises.

Frequent follow-up evaluation is important in mild cases, with special attention to subtle neurologic changes and evidence of muscle wasting. If conservative measures fail, the possibility of a stenotic lesion in the inflow feeding artery should be considered. Intervention may optimize arterial inflow in instances of lesions in any of the afferent arteries. Deterioration of nerve conduction, even in the face of symptoms that are mild, should be taken as an indication for intervention or search for another diagnosis.

The presence of pain, mild sensory symptoms, and signs of hypoperfusion of the hand during dialysis or with exercise is indicative of stage 2 HAIDI. Classification as 2a versus 2b is based upon the patient’s subjective experience of pain, tolerable versus intolerable. With stage 2a, HAIDI immediate intervention is not indicated. The first step should be conservative management as outlined above with close follow-up. When symptoms are noted soon (<30 days) after access creation and no neurologic deficits are present, most patients will have spontaneous resolution. Intervention for patients with stage 2a HAIDI is

<table>
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<tr>
<th>Stage</th>
<th>Signs and symptoms</th>
<th>Management</th>
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<tr>
<td>Stage 1</td>
<td>No clear symptoms, only signs&lt;br&gt;Nail beds slightly cyanotic and/or pale, mild coldness of skin of hand, decreased pulse at wrist</td>
<td>Conservative management&lt;br&gt;Close observation</td>
</tr>
<tr>
<td>Stage 2a</td>
<td>Nail beds cyanotic or pale, coldness of skin of hand, decreased pulse at wrist, tolerable pain, cramps, paraesthesia, numbness during dialysis or with exercise of hand</td>
<td>As above plus&lt;br&gt;Access blood flow measurement&lt;br&gt;Low—surgical referral&lt;br&gt;High—consider flow reduction&lt;br&gt;Angiography&lt;br&gt;Treat arterial stenosis</td>
</tr>
<tr>
<td>Stage 2b</td>
<td>Nail beds cyanotic or pale, coldness of skin of hand, decreased pulse at wrist intolerable pain, cramps, paraesthesia, numbness during dialysis or with exercise of hand</td>
<td>Intervention&lt;br&gt;Access blood flow measurement&lt;br&gt;Low—surgical referral&lt;br&gt;High—consider flow reduction&lt;br&gt;Angiography&lt;br&gt;Treat arterial stenosis</td>
</tr>
<tr>
<td>Stage 3</td>
<td>As above plus&lt;br&gt;Rest pain or motor dysfunction of fingers or hand</td>
<td>As above&lt;br&gt;Early treatment is indicated</td>
</tr>
<tr>
<td>Stage 4a</td>
<td>Tissue loss—ulceration, necrosis&lt;br&gt;Motor and/or sensory loss</td>
<td>As above&lt;br&gt;Emergent intervention required</td>
</tr>
<tr>
<td>Stage 4b</td>
<td>Extensive, extensive tissue loss</td>
<td>As above&lt;br&gt;Urgent intervention required&lt;br&gt;Consider closure of access&lt;br&gt;Amputation may be necessary</td>
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HAIDI: hemodialysis access-induced distal ischemia.

If symptoms lasted for more than one month or progress, intervention should be considered.

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**Table 3. Clinical classification of HAIDI.**
indicated only when (1) symptoms fail to ameliorate after 1 month and symptomatic treatment is not effective, (2) neurologic deficits develop, or (3) ischemic changes are advancing. If the pain is intolerable (stage 2b) after conservative management, early intervention is indicated. Either motor impairment or pain at rest (stage 3) occurring any time postoperatively is a clear indication for early intervention. With more severe signs and symptoms of hand ischemia such as ulcers or gangrene (stage 4), there is risk tissue loss. In these cases, emergent evaluation and treatment is essential in order to reverse the ischemia and minimize the degree of disability.

In order to appropriately target the underlying pathology associated with HAIDI, angiographic evaluation of both the proximal and distal arterial circulation is essential. Stenotic lesions occurring in the arterial components of the complex may occur anywhere from the central arteries to the feeding artery and beyond. These studies along with blood flow volume measurements are critical to aid diagnosis and to plan management.

Therapeutic procedure planning and management

The goal of HAIDI management is to correct the underlying pathophysiology by improving distal perfusion while maintaining dialysis access patency. Unfortunately, this is not always possible. Although with good preoperative access planning this is not a common occurrence, in cases in which severe acute ischemic symptoms present early, access ligation should be strongly considered. In later occurring situations where access salvage with resolution of ischemia is not feasible, the decision to ligate the access versus maintaining the access and accept disfigurement, tissue loss, or functional impairment should be an informed patient's decision in concert with the treatment management team. Most cases do not require immediate intervention although in some, prompt intervention is critically important. When intervention is required (depending on the degree of ischemia), an endovascular approach may be appropriate, but in more advanced stages of the syndrome surgical intervention is necessary. The first step in preparing for intervention is the assessment of access blood flow rate. Multiple treatment modalities have been described and their use should be directed by appropriate assessment and investigation of the underlying causative factors. In developing a plan for the management of HAIDI, two issues are important—(1) blood flow volume and (2) location of the anastomosis (Figure 4).

Blood flow volume

A variety of procedures for the treatment of HAIDI are available. The determination of blood flow volume in the AV access using Doppler ultrasound (DUS) is important in choosing the appropriate procedure. Currently, there is no generally accepted definition for normal access blood flow (Qa) or when it is too high. It is significantly higher for a brachial artery–based AVF than for a distal radio-cephalic AVF (RC-AVF). The normal Qa has been reported to range from 785 to 998 mL/min in radial-cephalic AVFs and from 1376 to 1580 mL/min in brachial-cephalic AVFs. However, for intervention planning, levels of 800 mL/min for an AVF and 1000 mL/min for an AVG have been suggested as threshold levels for a patient with a normal blood pressure. Clinical judgment is required to adjust these levels for a case with either low or high blood pressure. Those cases with blood flow less than this classified as low blood flow and those greater as high blood flow. In using DUS to assess blood flow volume, the measurement should be made from the brachial artery at least 5 cm proximal to the
anastomosis regardless of whether or not one is dealing with a radial or a brachial artery–based AVF.68 In cases of high bifurcation of the brachial artery, the measurement should be made in the inflow artery proximal to the bifurcation.

**Location of anastomosis**

The location of the arterial anastomosis, whether it is in the upper arm (brachial or proximal radial artery) or at the wrist (radial or ulnar artery), is an important determinant for the approach to therapy once a decision has been made that intervention is necessary.

**Upper arm vascular access**

This category includes both brachial artery and proximal radial artery–based accesses. However, a brachial artery–associated access is at significantly higher risk for the development of HAIDI. For management purposes, a differentiation is made between (1) low to normal and (2) high categories.

**High access blood flow.** Approximately two-thirds of HAIDI cases presenting for treatment have high access blood flow. These are primarily upper arm AVFs due to a larger artery and outflow vein at the time of surgical construction with higher blood pressure and flow into the access during maturation. Most will be associated with a brachial artery access. It is not unusual to see blood flow rates in excess of 2000 mL/min. Patients with high blood flow–associated HAIDI generally have a later onset of ischemic symptoms.69,69–71 In these patients, HAIDI is the result of the increasing AVF blood flow that occurs over time (increasing the demand) in combination with distal arterial disease, which is also generally progressive.14,22

Blood flow reduction in high flow HAIDI cases results in a significant increase in distal perfusion pressures.69 There are three approaches to blood flow reduction, each appropriate depending on examination, imaging, and anatomic findings—(1) ligation of a large side branch(es) if present, (2) precision banding, and (3) surgical revision using distal inflow (RUDI).72–74

Although uncommon, cases have been reported in which a large venous side branch associated with an AVF contributed significantly to access blood flow volume. Blood flow measurement before and during digital compression of the side branch offers an objective evaluation of its affect. If significant, ligation may be effective in relieving HAIDI.63,75,76

**Precision banding.** AV access banding creates a narrowed zone within the access to reduce blood flow and thereby obtain an increase in distal perfusion.69,77–79 Precision banding refers to banding performed in conjunction with some objective technique for controlling the degree of change in access blood flow. To assure success, accurate intra-operative flow monitoring is mandatory.69 In one report, significant changes were noted with only 0.5-mm variations in lumen diameter.80 The goal is to reduce access blood flow to a level no lower than 500–600 mL/min for AVFs and 600–700 mL/min for AVGs.69,81 The patient’s blood pressure must be relatively stable during the procedure to assure that measurements accurately reflect the effects of banding.

Precision banding can be performed using either a surgical or an endovascular approach. Both techniques are minimally invasive procedures.51,80,82,83 Although successful endovascular banding has been reported in patients with a prosthetic graft, it can be problematic. The walls of a prosthetic graft are not flexible and may fold into the lumen creating a potential problem. These cases and those where endovascular banding fails to relieve the ischemic symptoms should be referred to surgery. In these cases, proximalization of arterial inflow (PAI) has been recommended.39

**Low to normal access blood flow.** The treatment goal in this group of patients is to increase perfusion to the hand without losing the AV access. Primary arterial disease is a major contributor to hand ischemia in a significant number of these cases, especially those at the low end of the blood flow spectrum.5,15 Surgical procedures for these patients include PAI or distal revascularization and interval ligation (DRIL).6 If a patient is not a surgical candidate, precision banding should be considered prior to abandoning the access. In doing this, care must be taken to avoid lowering the Qa in an AVF to less than 400–500 mL/min and less than 600 mL/min and an AVG because of the risk of thrombosis.84 In some cases, the access may not be salvageable.

**Wrist vascular access**

HAIDI is encountered far less frequently with a radial-cephalic AVF than with an upper arm AVF or AVG, especially those associated with the brachial artery. The radial-cephalic AVF is unique in that the radial artery distal to the anastomosis allows retrograde blood flow which may (rarely) facilitate a decrease in hand perfusion pressure resulting in ischemia. Several options exist for treating HAIDI when associated with a distal radial-cephalic AVF. The most common procedure is coil occlusion or ligation of the radial artery distal to the anastomosis (DRAL). In the rare case of a high flow RC-AVF, precision banding or ligation of the radial artery proximal to the anastomosis may be appropriate.73

As long as the ulnar artery is patent and the palmar arch is intact, occluding the distal radial artery and interrupting the retrograde flow from the palmar arch will resolve HAIDI. Hand perfusion pressure is maintained through the
ulnar artery, and the AVF is maintained through the feeding radial artery. Angiographic evaluation in addition to physical and Doppler examination of the arterial components of the hand/vascular access complex is necessary prior to performing this procedure. This is essential not only to detect a stenotic lesion which could be causing or contributing to the problem, but also in order to avoid two possible complications. First, the feeding radial artery must be capable of providing adequate blood flow to maintain access patency in the absence of the retrograde contribution of the ulnar artery–palmar arch. Assessing hand perfusion with and without distal radial artery manual occlusion is helpful in assessing this issue. Second, angiography is necessary to confirm that an intact ulnar artery and palmar arch are present to provide perfusion of the hand prior to occluding the distal radial artery.

The radial artery distal to the RC-AVF is easily interrupted. This can be done surgically or by placing an embolization coil in the vessel. Both procedures are minimally invasive with excellent results when performed following the careful evaluation described above. In cases with a high Qa, blood flow reduction by precision banding should be considered as primary treatment. If primary treatment does not result in resolution of the hand ischemia, the case should be referred to surgery.

**Recommendations for assessment and management of HAIDI by interventional nephrologists**

- Every patient considered for an AV access should have a complete evaluation, including vascular mapping, before access surgery.
- Even with high-risk patients, the creation of an AV access should not be automatically avoided because of the risk of HAIDI, utilizing access procedures mentioned above for such individuals.
- All patients with an AV access should be monitored for the development of signs and symptoms of HAIDI.
- Characteristic signs and symptoms of HAIDI are adequate for the diagnosis in most patients. In equivocal cases, digital blood pressure measurements should be utilized to confirm the diagnosis. Careful neurologic examination should exclude other causes for symptoms.
- Once the diagnosis of HAIDI has been made, the patient should undergo a detailed clinical evaluation to determine the appropriate approach for management.
- The approach to management of HAIDI should be based upon the clinical stage of the syndrome.
- Stages 1 and 2a HAIDI cases should have frequent follow-up evaluation with special attention to subtle evidence of progression. If resolution fails to occur within 1 month or if deterioration is detected, further evaluation should be instituted.
- Stages 2b, 3, and 4 HAIDI cases should be immediately evaluated for intervention.
- If a HAIDI patient’s clinical condition suggests a limited life expectancy, they should be evaluated for the placement of a tunneled dialysis catheter for continuation of palliative dialysis and occlusion or ligation of the access.
- All patients with HAIDI should be evaluated for both proximal and distal arterial stenosis. Significant stenotic lesions should be treated by angioplasty when appropriate.
- HAIDI associated with a radial-cephalic AVF should be evaluated for occlusion of the radial artery distal to the RC-AVF anastomosis in patients with an intact ulnar artery and palmar arch. If this is not successful, the patient should be referred to surgery.
- In cases with a brachial artery or proximal radial artery–associated AVF, brachial artery flow should be assessed as a surrogate for total access flow. Treatment of HAIDI should be determined by blood flow volume.
- Normal/low blood flow volume HAIDI without evidence of arterial stenosis should be referred to surgery.
- High blood flow volume HAIDI should be evaluated for precision banding by endovascular or minimally invasive techniques with targeted brachial artery flow reduction to between 600 and 1000 mL/min.
- If precision banding of a high blood flow HAIDI is not successful, the patient should be referred to surgery.

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References

8. Scheltinga MR and Brujininckx CM. Haemodialysis access-induced distal ischaemia (HAIDI) is caused by locoregional hypotension but not by steal. *Eur J Vasc Endovasc Surg* 2012; 43: 218–223.
30. Beathard et al. https://orcid.org/0000-0002-1745-6851
31. Haimanot Wasse https://orcid.org/0000-0003-4161-7122
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35. https://orcid.org/0000-0003-4161-7122


74. Loh TM, Bennett ME and Peden EK. Revision using distal inflow is a safe and effective treatment for ischemic steal syndrome and pathologic high flow after access creation. *J Vasc Surg* 2016; 63: 441–444.


