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Ultrasound evaluation of dialysis access-related distal ischaemia

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Abstract

Dialysis access-related distal ischaemia is a rare yet potentially rather risky complication of haemodialysis angioaccess. Timely diagnosis is crucial to target both the goals of the access team: first of all to preserve the function of the hand ideally along with angioaccess patency. Unfortunately for some patients, urgent access ligation and central vein catheter insertion would be needed to save the hand. After a first clinical examination to determine the diagnostic suspicion, the ultrasound evaluation would provide nearly all the needed information to confirm the diagnosis and to determine the most appropriate procedure to rescue the patient from distal ischaemia. In some cases, photoplethysmography would help in the differential diagnosis of other non-ischaemic causes of similar signs and symptoms. Angiography would complete the preoperative evaluation for some.

Dialysis access-related distal ischaemia would be briefly reviewed, and a deep description of the ultrasound examination tools and findings would be provided for a tailored therapeutic approach.

Keywords

AV fistula, dialysis, ultrasonography–Doppler evaluation, techniques and procedures, dialysis access-related distal ischaemia

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Main text

Definition

Dialysis access-related distal ischaemia is a serious but infrequent complication of arterio-venous (AV) access for haemodialysis (HD). It is known as haemodialysis access-induced distal ischaemia (HAIDI), distal hypoperfusion ischaemic syndrome (DHIS), and arterio venous access ischaemic steal (AVAIS).[1,2]

For simplicity, we would adopt the term HAIDI. In short, it consists of a reduced tissue oxygen delivery to the extremity of the arm where a VA exists because of a reduced capillary perfusion as a consequence of a low inflow pressure or high outflow resistance.[3]

These two aetiologies would configure two variants of hand ischaemia: an 'arterial hypoxic' and a 'venous stagnant' syndrome depending on the aetiology as described. In both cases, function and vitality of the tissues involved (nerves, muscles and skin) would be variably impaired. HAIDI (as well as DHIS and AVAIS) generally refers to the 'arterial hypoxic' distal ischaemia syndrome variant.[4]

Incidence

The HAIDI syndrome occurs in about 1%–8% of the patients undergoing AV fistula (AVF) and AV graft (AVG) creation.[5,6]

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Along with the number of incident end stage renal disease (ESRD) patients, comorbidities also increased in the last decades; as a consequence, more complications have to be managed by the VA team; among those is HAIDI, for which treatment has become more challenging.[7]

Risk factors

Risk factors described for the development of the HAIDI syndrome are the female gender, diabetes mellitus, age greater than 60 years, previous surgery on the same limb and the use of proximal vessels for the creation of the AVF, like in brachiocephalic fistula.[5,6,8–11] Moreover, uremia itself causes the progression of atherosclerosis and the development of medial calcinosis that affect mostly the peripheral arteries.[12]

Medial artery calcinosis as well as diabetic microangiopathy would both interfere with arteriolar dilatation in response to tissue hypoxia caused by a competing proximal AV shunt along the inflow axis.

In the rare variant 'venous stagnant' HAIDI syndrome, a latero-lateral AV anastomosis or an outflow venous stenosis is the cause of hypertension on the venule segment impairing flow and thus causing a 'stagnant' hypoxia.[3,4]

Clinical presentation

The HAIDI syndrome can be divided into an acute and chronic form. The acute classic hypoxic form presents immediately after surgery with a cold white or painful bluish finger. It generally requires urgent intervention, mostly because of the risk of being exposed to monomelic ischaemia. If the symptoms, especially severe pain, persist, angioaccess ligation is mandatory.[13]

The chronic hypoxic form would present later after access creation with a variable degree of signs and symptoms defining four HAIDI stages as shown in Table 1.[14]

Grade 1 to 2a generally do not deserve open or endovascular surgery; often, grade 4b necessitates urgent angioaccess ligation.

Patients who complain of symptoms only during the HD treatment may not require surgical intervention but rather blood pressure (BP) medication modification or dry weight adjustment (to avoid excessive blood volume depletion). The aetiology of this form of steal syndrome is likely related to BP drop during the HD session. Tailoring the therapy may improve the symptoms.[1]

Acute and chronic 'venous stagnant' HAIDI would present with similar functional symptoms associated with the picture of venous congestion.[4,15]

Differential diagnosis is needed to exclude other conditions such as miming HAIDI because of confounding similar signs and symptoms, such as those shown in Table 2.

Table 1. Haemodialysis access-induced distal ischaemia (HAIDI) classification.

Grade 1	No clear symptoms, but discrete signs of mild ischaemia may be observed
Grade 2a	Complaints during a HD session or intense use of the hand: tolerable pain cramps, paraesthesia, numbness, or disturbing coldness in the finger or hand
Grade 2b	Complaints during a HD session or use of the hand. Intolerable pain, cramps, paraesthesia, numbness, or disturbing coldness in the finger or hand
Grade 3	Rest pain or motor dysfunction of fingers or hand, upper limb relevant only
Grade 4°	Limited tissue loss (ulceration, necrosis). Clinically significant hand function (upper limb) is probably maintained if ischaemia is reversed
Grade 4b	Irreversible tissue loss of the hand (upper limb); impossible to preserve clinically significant hand function. Requires amputation

HD: haemodialysis.

Pathophysiology

A pseudo-physiologic blood flow diversion (the so called 'steal') develops after every successful AV shunt creation by a lateral arteriotomy at the forearm. As an example, a retrograde flow is normally observed in the distal radial artery, following a latero-terminal radio-cephalic fistula.[1,4,16,17]

No ischaemia is therefore observed if the distal peripheral vascular bed successfully adapts to the reduced blood flow by small collateral arteries and arteriole dilatation.

Proximal arterial inflow reduction, excess of steal from the AV shunt (wide anastomosis), impaired dilatation of distal arteries, collaterals and arterioles or the variable combination of those are the causes of arterial hypoxic HAIDI, as shown in Table 3.[1,18]

Usually, symptomatic steal tends to be uncommon in distal AVF compared with more proximal accesses when the brachial artery is used as inflow. In this case, the HAIDI syndrome incidence rises up to 5%–8% of cases per new AVF surgery.[11]

Of course, all atherosclerotic risk factors increase the risk of HAIDI development.

Venous hypertension can be caused by a lateral anastomosis on the vein or the existence of an outflow stenosis. As a consequence, capillary flow is significantly impaired, causing the venous stagnant HAIDI syndrome, which is rarer than the hypoxic variant.[4]

Diagnosis

Due to the complexity of the factors involved in the development of the ischaemic syndrome, each case needs particular attention to find the best therapeutic approach.[19]

Table 2. Differential diagnosis of HAIDI.

Miming HAIDI	Clinical features	Diagnostic tool for differential diagnosis
Carpal tunnel syndrome	Numbness, tingling, burning, and pain (primarily in the thumb and index, middle, and ring fingers)	Electrophysiological tests, ultrasonography
Peripheral neuropathy	Sharp, jabbing, throbbing or burning pain, hyperesthesia	Electrophysiological tests, blood tests, nerve biopsy
Central vein occlusion syndrome	Edema of both the upper extremities, face and neck, tenderness, pain and associated erythema which can mimic cellulitis	Digital subtraction central venography, computerised tomography angiography
Monomelic ischaemia	Unilateral severe pain and paraesthesia, handgrip strength deficit	Electrophysiological tests (acute onset of dysfunction involving multiple nerves of the upper extremity after the creation of an AV fistula)
Ungual onychomycosis	Subungual hyperkeratosis and onycholysis, which is usually yellow-white in colour; as the disease progresses, paraesthesia, pain, discomfort, and loss of dexterity	Dermoscopy, histologic examination of the nail

HAIDI: haemodialysis access-induced distal ischaemia; AV: arterio-venous.

Table 3. Pathophysiology of HAIDI.

Pathophysiology	Aetiology
Proximal arterial inflow reduction	Atherosclerotic lesion upstream to the anastomosis
Excess of steal from the shunt	Wide or proximal anastomosis, multiple angioaccess
Impaired patency of distal arteries (radial, ulnar, interosseous)	Atherosclerotic lesion *after* the anastomosis, previous angioaccess
Impaired dilatation of the distal arteries and arterioles	Medial calcinosis, diabetic microangiopathy, uremia
Venous hypertension ("stagnant HAIDI" variant)	Lateral anastomosis on the vein, outflow stenosis

HAIDI: haemodialysis access induced distal ischaemia.

All patients with ischaemic symptoms should undergo a physical examination to first confirm clinically the suspicion of the HAIDI syndrome. In case of a doubt, the direct measurement of finger arterial pressure by photoplethysmography would be helpful to distinguish the hypoperfusion syndrome from other entity mimicking ischaemia by similar signs and symptoms, as shown in Table 2.[1,20–22] Besides complex dedicated devices, plethysmography of the finger oxymetry probe can also be used.

After the confirmation of the diagnosis, a thorough ultrasound examination would provide nearly all the necessary anatomic and haemodynamic information needed for the best management of the ischaemic complication.[23–25]

An angiography could be necessary later to complete the preoperative evaluation in some cases,[1,4] especially when the percutaneous therapy of the arteries is expected after ultrasound examination.

A therapeutic algorithm has been proposed by Bourquelot based on the HAIDI grade and angioaccess blood flow,[4] as shown in Figure 1.

Ultrasound examination: diagnosis and indications for treatment

The minimum equipment required for a complete evaluation for the HAIDI syndrome is a standard linear probe, 7.5 up to 20 MHz for a superficial B-mode and colour-Doppler exploration.

The examination should be performed in a warm environment to prevent influence of room temperature on spasms. The patient may be evaluated in a seated or reclined position, taking care not to place the arm in a forced or uncomfortable position. The entire arm should be exposed and accessible for ultrasound evaluation from the axilla to the fingers. In some cases, supraclavicular fossa could be also investigated. Time from the last dialysis should be conveyed to the examining physician who will then be aware of the blood volumes of the patients at the time of duplex examination. A compressive dress should be removed from the arm to be evaluated; therefore, the time delay since the previous HD is ideally >24 hours to prevent bleeding and flow volume decrease due to excessive ultrafiltration. When an urgent diagnosis is needed, a sterile transparent dressing could be applied.[26]

First of all, the diagnosis of ischaemia should be confirmed by reduced or absent perfusion of digital artery finding. The relief of the symptoms, and the restoration of a normal distal flow, after manual occlusion of the anastomosis is a sign that confirm the diagnosis of HAIDI. Such small arteries could be challenging to explore with a conventional ultrasound machine, and a continuous wave Doppler pencil may be useful, effective and time-sparing (Figure 2). A dynamic manoeuvre could be performed to

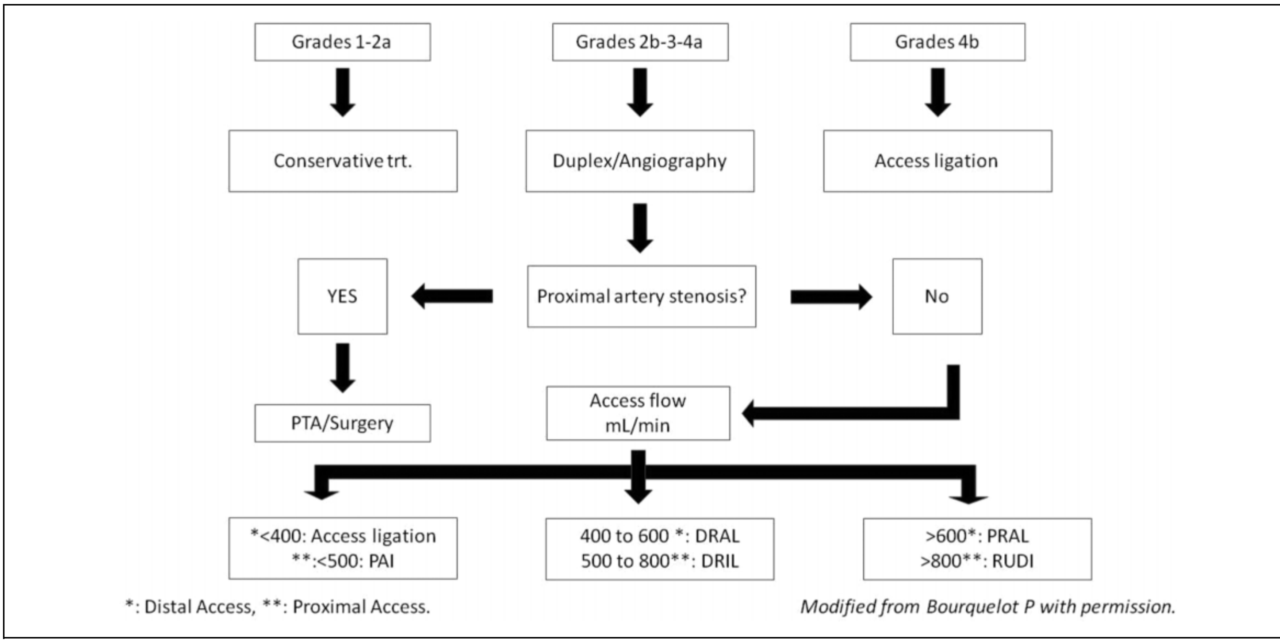

Figure 1. HAIDI treatment algorithm.

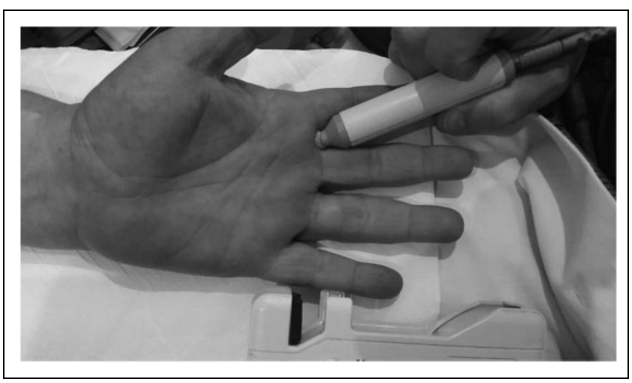

Figure 2. Digital artery flowmetry by a continuous wave Doppler pencil.

Figure 3. Distal blood flow modification after arterio-venous fistula manual compression.

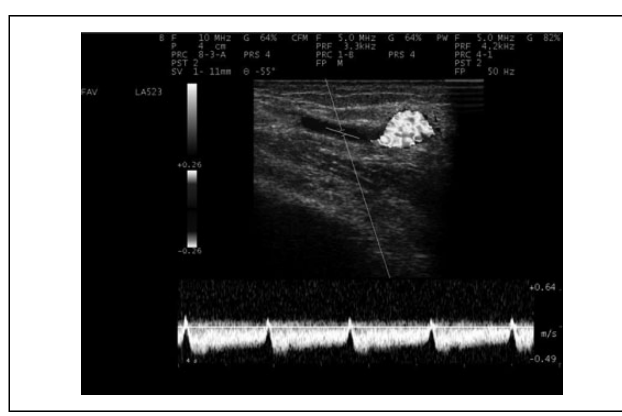

Figure 4. Blood flow distal to the anastomosis: antegrade in systole, retrograde in the diastole pattern.

assess increase of digital perfusion after anastomosis manual occlusion (Figure 3).

Having confirmed the diagnosis of HAIDI, the first step is to estimate the blood flow in order to determine a low, normal, or high flow angioaccess.[4]

The measurement of AVF blood flow should be obtained in a straight segment with laminar flow, at least 5 cm away from aneurysms, stenotic lesions and the anastomosis sites. The brachial artery is therefore the preferred site to perform this measure for both AVF and AVG.[27]

In case of high bifurcation of the brachial artery, the best way is to measure the flow proximally to the bifurcation. If the bifurcation is too high, then the flow volume could be measured in both arteries and summarised. The use of abundant ultrasound gel allows a minimum pressure to be applied on the artery and avoids defective flow

Table 4. Angioaccess flow category.

Site anastomosis	Low flow	Normal flow	High flow
Distal	<400 mL/min	400–600 mL/min	>600 mL/min
Proximal	<500 mL/min	500–800 mL/min	>800 mL/min

calculation. The Doppler spectral curve is used for volume flow calculation. When done properly, the ultrasound determination of fistula's flow correlates well (less than 10% variation) with the thermo-dilution technique.[28] The calculation of flow volume is based on the equation $Q = \pi.r^2 \times TAVM$, where r is the vessel radius and TAVM = time-averaged velocity integral of the mean velocity of the parabolic profile layers. The most critical factor is the determination of the vessel radius.[23,27] Normal blood flow in the brachial artery (healthy subject without AVF) ranges between 30 and 120 mL/min. After the AVF creation, the flow can increase by more than 10-fold and the diameter of the inflow artery and the outflow vein may increase 2–4 times.[29]

Table 4 summarises low, normal and high flow for distal and proximal access to HAIDI complication.

The definition of access flow is crucial to determine the type of intervention needed to rescue patients from ischaemia, trying to preserve patency of the access (Figure 1).[4]

The second step is to define the type and location of the anastomosis and blood flow in the artery distally to the anastomosis (Figure 4). All type of flow can be observed: antegrade, retrograde, antegrade in systole and retrograde in diastole. Generally in distal access, complete retrograde flow is observed in the feeding artery distal to the anastomosis (most of time asymptomatic); when it is associated with HAIDI, the treatment suggested is to interrupt steal by distal radial artery ligation (DRAL).[16,17,30] A dynamic manoeuvre could be performed to preoperatively assess the efficacy of the procedure: digital flow is measured before and after manual compression of distal radial artery to verify the increase of flow after simulation of DRAL.[31]

In proximal access complicated by HAIDI, low blood flow is generally associated with antegrade or bidirectional flow in the artery distal to anastomosis, while bidirectional or retrograde flow is most frequently observed in high flow access.

In the first case, the major cause of ischaemia is insufficient distal perfusion because of either a proximal inflow stenosis or a distal arteriopathy. Subclavian, axillary and brachial artery should therefore be examined to find an atherosclerotic stenosis. An angiography could both complete the inflow evaluation up to the aortic trunk and fix by angioplasty the impaired inflow treating low flow and ischaemia at the same time.[32] Some similar information could be provided alternatively by computerised tomography angiography together with the visualisation of the surrounding structures (thus on eventual thoracic outlet syndrome), yet missing the dynamic and therapeutic tools of angiography.

Distal forearm arteries should then be evaluated if a proximal inflow impairment has been ruled out. Long or segmental stenosis of radial and ulnar artery are not uncommon in HD patients, especially if they are affected by diabetes or when they are haemodialyzed for a long time. Although short arterial stenoses could be easily treated by endovascular intervention, results of this procedure in long stenoses due to medial calcinosis are rather disappointing.[32] The best treatment of the low to normal flow HAIDI is the distal revascularization interval ligation (DRIL) procedure.[33]

Patients with a proximal access with high flow HAIDI mainly suffer from an excessive AV shunt (generally because of a large anastomosis), with the distal vessel not showing any significant stenosis. In these cases, angioaccess flow reduction is the treatment of choice by means of revision using distal inlow[34,35] (RUDI) procedures or banding. Banding solution, however, suffers from scarce reproducibility, given the erratic result available up to now, and depends highly on the surgeońs experience.[36]

Distal access with high flow HAIDI generally does not show at ultrasound any arterial lesion, or proximal or distal. Whenever the ulnar artery would be found stenosed or occluded, endovascular treatment would be quite effective, yet keeping unmodified the high flow of the radiocephalic fistula.[37] After having excluded distal artery stenosis, flow reduction through proximal radial artery ligation (PRAL) or, with the limits of reproducibility, banding is the indicated treatment option.[36,38]

In summary, by ultrasound evaluation, we could 1) confirm diagnosis of HAIDI, 2) establish the HAIDI flow category (= aetiology); 3) find out the existence of an arterial stenosis up- or downstream to the anastomosis in order to determine the right treatment option based on the anatomical and haemodynamic information on the angioaccess..

The last aim of ultrasound evaluation of HAIDI is to check for any remnant unused previous AVF on the same arm that could be ligated to reduce the global AV shunting, thus helping distal perfusion.[4]

Ultrasound examination: HAIDI prevention

Ultrasound may be useful even for HAIDI prevention by preoperative evaluation as well as during angioaccess follow-up.

Preoperatively, ultrasound may prevent HAIDI by discouraging the creation of proximal access in patients with severe distal hypoperfusion because of medial calcinosis or atherosclerotic lesion. In some severe condition of distal arteriopathy, even a distal access creation would be contraindicated. On the other hand, the finding of high bifurcation of the brachial artery ultrasound may be reassuring in high risk patients (i.e. diabetic) regarding a proximal access creation by respect of ischaemia,[4] as the inflow, despite being at the elbow, would be haemodynamically similar to a distal access.

Finally, during the follow-up, the observation of a low subclinical distal perfusion may contraindicate treatment of a concomitant venous stenosis, thus preventing the occurrence of postoperative HAIDI.[4]

Conclusion

Ultrasound evaluation will provide a huge amount of information that could help determine the best treatment options for dialysis access-related distal ischaemia. The anatomical and haemodynamic information on the angioaccess complicated by HAIDI is of great value for determining the intervention needed for a patient.

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Ethical statement

Being a review article, this paper does not need any ethical approval or informed consent.

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