"ROLE OF DOPPLER EVALUATION IN CREATING ARTERIOVENOUS FISTULA FOR HEMODIALYSIS – AN OBSERVATIONAL STUDY"

By

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In partial fulfillment of the requirements for the degree of

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Under the Guidance of

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Dr. SURAJ H. S.

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LIST OFABBREVIATIONS

Glossary	Abbreviations
ABPM	Ambulatory bp monitoring
ACCOMPLISH	Avoiding cardiovascular events through combination therapy in patients living with systolic hypertension
ACR	Albumin-creatinine ratio
ADPKD	Autosomal dominant polycystic kidney disease
AIUM	American institute of ultrasound in medicine
AKI	Acute kidney injury
ATN	Acute tubular necrosis
AV	Atrioventricular
AVF	Arteriovenous fistula
CDU	Colour doppler ultrasonography
CDUS	Color doppler ultrasonography
CKD	Chronic kidney disease
CKD-EPI	Chronic kidney disease epidemiology collaboration
CTA	Computed tomography angiography
CVC	Central venous catheters
CVD	Cephalic vein diameter
DU	Doppler ultrasound
ECD	Echo color doppler
ECM	Extracellular matrix
EDV	End-diastolic volume

ESRD	End-stage renal disease
FTM	Failure to mature
GFR	Glomerular filtration rate
HD	Hemodialysis
IQR	Interquartile range
KDIGO	Kidney disease improving global outcomes
KDOQI	Kidney disease outcomes quality initiative
MRA	Magnetic resonance angiography
MTHFS	Methylenetetrahydrofolate reductase
NaCl	Sodium chloride
NaHCO3	Sodium bicarbonate
NAVAC	North American consortium of vascular access
ND	No defective access
NSAIDs	Nonsteroidal anti-inflammatory drugs
NYHA	New York heart association
ONTARGET	Ongoing telmisartan alone and in combination with ramipril global end point trial
PKD	Polycystic kidney disease
PRF	Pulse repetition frequency
PSV	Pressure support ventilation
PTFE	Prosthetic fistulas
RAAS	Renin-angiotensin-aldosterone system
RAD	Radial artery diameter
RBC	Red blood cell

RCAVF	Radiocephalic arteriovenous fistula
RI	Resistance index
RPF	Renal plasma flow
RRT	Renal replacement treatment
SCN	Sickle cell nephropathy
SRU	Society of radiologists in ultrasound
TCF7L2	Transcription factor 7 like 2
US	Ultrasound
VA	Vascular access
VAIVTs	Vascular access intervention treatment
VD	Venous distensibility
VM	Vascular mapping
WBCs	White blood cells

ABSTRACT:

Introduction: The utility of Doppler ultrasound (DU) to assess the development and management of arteriovenous fistulas (AVF) for haemodialysis can help to increase the prevalence and patency of AVFs. This study aimed to plan arterio-venous access and map vessels using Doppler ultrasonography.

Material and method: This was an observational study with 33 patients with chronic kidney failure suggested for AV access placement and subjects with previous AV fistula failure scheduled for reconstruction at a different site. Preferred site of access placement was planned by Duplex Doppler sonography by assessment of vessel caliber, wall morphology, peak systolic value of arteries and patency of vessels based on criteria. Venous component, Arterial component and AVF were considered as primary outcome variables. Type of anastomoses was considered as primary explanatory variable. Statistical significance was defined as a P value of less than 0.05. SPSS software; V. 22 was used to analyse the data.

Results: Most of the patients involved in the study were aged between 50-59 years (42.42%) with male predominance. The most common location for AV fistula creation was in Brachio-cephalic site in 45.45% and Radio-cephalic site in 33.33% and lastly Brachial-median cubital site in 21.21% of participants. Out of 33 participants, 5 people had complications, out of which 60% of participants had CV thrombus and 40% of participants had pseudoaneurysm. 30.30% of participants showed successful maturation of AVF and 69.69% showed failure of maturation of AVF. Mean blood flow in matured AVF was > 500 ml/min (1472 ml/min) and in immature AVFs it was less than 500 ml/min (404 ml/min).

Conclusion: All AVFs with an adequate flow volume of more than 500 ml/min, velocity, width & depth from skin were found to be related to the maturation of AVF in all types of AVF.

Keywords: Arteriovenous fistula, Doppler Ultrasound, hemodialysis.

INTRODUCTION

INTRODUCTION:

The adequacy of dialysis via a correctly placed vascular access determines the longevity of life and its quality in patients with end-stage renal failure on hemodialysis (HD).[1] Hemodialysis has an important role in long-term existence of patients with end-stage-renal disease (ESRD). To maintain them on long-term dialysis, vascular access procedures are required.[2] A well-functioning vascular access has a good blood flow, excellent patency, and allows easy and repetitive cannulation with needles. Arteriovenous fistulas (AVFs) provide best access for longevity and the lowest morbidity and mortality; yet, arteriovenous grafts are still useful in elderly patients for whom AVFs are not possible. After surgical creation, the vein gets distended to become a successful arteriovenous fistula (AVF). Fistula undergoes a remodeling process that is referred to as maturation. Although these modifications are diverse, they occur quickly, resulting in a fistula that may be used repeatedly and provides acceptable dialysis treatment.[3] Early discovery of access malfunction or complication allows for follow-up therapies that help to reduce access failure rates.[4] Early or late failure of the AVF is possible. The time between the fistula's formation and the start of its work, or within initial three months of its usage, is accounted for in the early failure of vascular anastomoses for hemodialysis. Late failure of the AVF is defined as occurring after 3 months of hemodialysis use.[5]

AVFs are the preferred initial HD access owing to their longer patency than prosthetic arteriovenous grafts. Arteriovenous grafts are still useful in patients for whom AVFs are not an option, and maybe in particular groups such as the elderly. For nephrologists and vascular surgeons, creating and maintaining a patent and well-functioning AVF has become a significant issue.[4,5]

The most common cause of morbidity in patients with chronic kidney failure is complications connected with HD vascular access. Access failure is usually owing to thrombosis associated with anastomotic or outflow vein stenosis. To restore functionality or create new accesss, many salvage processes are required.[6]

Early detection of anastomotic access dysfunction and remediation may help to reduce the rate of access failure. AVFs are designed to be superficial, and Doppler Ultrasound can easily access them (DU). In a patient-centered VA evaluation, DU is critical.

Other issues linked with fistulas that can be detected with ultrasound include abscess, aneurysm, steal syndrome and hematomas, which can be assessed using a Colour Doppler scan.[9] Preoperative vascular evaluation for AVF generation, assessment of prime puncture time, early detection of problems and selection of appropriate therapeutic methods for correction are all possible with Colour Doppler ultrasonography. The adequacy of dialysis via a correctly placed vascular access is critical for the long-term survival of patients on hemodialysis with ESRD. Duplex Ultrasonography is quickly becoming a standard procedure, and its widespread adoption will have significant resource consequences in the future.[10]

It is mobile, cost effective, and noninvasive, and also, it provides morphologic and functional information of the access flow.[7] It can provide all aspects of vascular access care, including vascular mapping, maturation evaluation, and surveillance.[8] In addition to diagnosing the complications of AVFs, it can be also used to guide intervention procedures to correct the hemodynamic problems and prolong the access patency.[3]

NEED FOR THE STUDY

In hemodialysis (HD) patients, AVF is the preferred vascular access (VA). AVF has a lower rate of infection, complications, and health-care costs than central venous catheters (CVC) and prosthetic fistulas (PTFE), and it has better long-term patency.[9] However, in recent decades, because of aging and comorbidity of HD patients, achieving a functioning and long-lasting AVF is increasingly complex.[10] It is estimated that the early primary failure rate of the AVF, according to the definition of the North American consortium of vascular access (NAVAC),[11]is between 23% and 37% and the primary unassisted permeability at one year is between 40% and 64%. Between 21% to 50 % of the AVFs performed never mature enough to be used in HD.[12,10] Multidisciplinary VA design and follow-up programmes, as recommended in clinical guidelines[17], may help to lower the frequency of primary failure and maintain medium and long term AVF permeability. Use of Doppler ultrasound (DU) in preoperative evaluation and subsequent surveillance of AVF has also demonstrated to be beneficial in terms of primary, aided, and secondary AVF survival.[18,19]

AIMS & OBJECTIVES

AIMS AND OBJECTIVES:

1. To perform Doppler ultrasonography for arterio-venous access planning and vessel mapping.
2. To assess time taken for maturation of AV fistula.
3. To identify and categorise post-operative complications.

REVIEW OF LITERATURE

REVIEW OF LITERATURE:

Chronic renal failure:

Chronic kidney disease (CKD) is defined as kidney damage or an estimated glomerular filtration rate (eGFR) of < 60 ml/min/1.73 m^2 that persists for a duration of more than three months, regardless of the aetiology.[20] It is a gradual loss of kidney function that finally demands the use of dialysis or transplantation (renal replacement therapy). Pathologic abnormalities in imaging investigations or renal biopsies, anomalies in urine sediment or increased urinary albumin excretion rates are all signs of kidney impairment. The 2012 KDIGO CKD classification provides information on the cause of CKD and divides it into six groups depending on glomerular filtration rate (G1 to G5 with G3 split into 3a and 3b). It also includes albuminuria staging (A1, A2, and A3), with each stage of CKD being divided into sub-categories based on the urinary albumin-creatinine ratio in (mg/gm) or (mg/mmol) in an early morning "spot" urine sample.[21]

The six categories include:

• G1: GFR of 90 ml/min per 1.73 m^2 or more

• G2: GFR of 60 to 89 ml/min per 1.73 m^2

• G3a: GFR of 45 to 59 ml/min per 1.73 m^2

• G3b: GFR of 30 to 44 ml/min per 1.73 m^2

• G4: GFR of 15 to 29 ml/min per 1.73 m^2

• G5: GFR < 15 ml/min per 1.73 m^2 or dialysis treatment.

The three levels of albuminuria include an albumin-creatinine ratio (ACR)

- A1: ACR less than 30 mg/gm (less than 3.4 mg/mmol)
- A2: ACR 30 to 299 mg/gm (3.4 to 34 mg/mmol)
- A3: ACR greater than 300 mg/gm (greater than 34 mg/mmol).

The enhanced classification of CKD has aided in the identification of prognostic indicators such as decreased kidney function and increasing albuminuria. However, one disadvantage of using classification systems is the risk of overdiagnosing CKD, particularly in the elderly.[22]

Etiology

The most prevalent primary illnesses that cause CKD and eventually ESRD are as follows:[23]

- Type II diabetes mellitus (30% to 50%)
- Type I diabetes mellitus (3.9%)
- Hypertension (27.2%)
- Primary glomerulonephritis (8.2%)
- Chronic tubulointerstitial nephritis (3.6%)
- Hereditary or cystic diseases (3.1%)
- Secondary glomerulonephritis or vasculitis (2.1%)
- Neoplasm or plasma cell dyscrasias or (2.1 %)
- Sickle Cell Nephropathy (SCN), which affects < 1% of Americans with ESRD. [13]

Causes of CKD maybe can be attributed to prerenal (declined RPF), inherent renal (pathology of the glomeruli, vessels, tubular interstitium), or post-renal (obstructive) etiologies.

Prerenal Ailment

Subjects with chronic heart failure or cirrhosis with consistently diminished renal perfusion develop chronic prerenal illness, which raises the risk of several episodes of an inherent kidney injury, such as ATN. The renal function worsens over time as a result of this.[22]

Inherent Renal Vascular Disease

Nephrosclerosis is the most frequent chronic renal vascular disease, characterized by persistent injury to the tubulointerstitium, glomeruli and blood vessels.

Renal artery stenosis due to fibro-muscular dysplasia or atherosclerosis causes ischemic nephropathy, which is characterised by tubulointerstitial fibrosis and glomerulosclerosis over months or years.[25]

Intrinsic nephrotic or nephritic disease

A nephritic pattern is suggested by abnormal urine microscopy with red blood cell (RBC) casts and dysmorphic red cells, occasionally white blood cells (WBCs), and varying degrees of proteinuria. The most prevalent causes include infective endocarditis, post-streptococcal GN, IgA nephropathy, shunt nephropathy, vasculitis, Goodpasture syndrome and lupus nephropathy.[26]

A nephrotic pattern is defined by proteinuria in the nephrotic range (>3.5 gm / 24 hrs) and urine microscopic examination which is inactive with few cells or casts. The most prevalent causes include focal segmental glomerulosclerosis, minimal change disease, membranoproliferative GN (Type 1 and 2 with cryoglobulinemia), amyloidosis, membranous glomerulosclerosis and diabetic nephropathy. [22]

Inherent Tubular and Interstitial Disease

The most common chronic tubulointerstitial disorder is polycystic kidney disease (PKD). Other etiologies include nephrocalcinosis (most usually caused by hypercalciuria and hypercalcemia), Sjogren syndrome, sarcoidosis, and reflux nephropathy in children and young adults.[27]

Mesoamerican nephropathy, a relatively high frequency of CKD of unclear origin among agricultural labourers from Southeast and Asia Central America, is becoming more widely recognised.[28]

Postrenal (Obstructive Nephropathy)

Prostatic illness, nephrolithiasis or an abdominal/pelvic tumour with a mass effect on the ureter can all cause chronic blockage. Chronic ureteral blockage is caused by retroperitoneal fibrosis, a rare condition.

Epidemiology

Because early to moderate CKD is asymptomatic, determining occurrence and prevalance of the disease is difficult. In the general community, CKD affects between 10% to 14% of the population. Albuminuria (microalbuminuria or A2) and GFR < 60 ml/min/1.73 m^2 have a prevalence of 7% and 3% to 5%, respectively.[29]

In 2012, CKD was responsible for 2,968,600 (1%) of disability-adjusted life-years and 2,546,700 (1%) to 3%) of life-years lost globally.[23]

The Kidney Disease Outcomes Quality Initiative (KDOQI) requires that patients be tested three times over a three-month period for chronicity and CKD, with two of the three outcomes being consistently positive.[30]

Natural History and Development of CKD

When compared to CKD in patients referred to nephrology practices (referred CKD), CKD identified in the over-all population (community CKD) has a markedly different natural history and development path.

CKD in the community is mostly found in the elderly. These people have survived their entire lives with cardiovascular risk factors, hypertension, and diabetes, all of which can harm the kidneys. Post the age of 40 to 50 years, the average frequency of reduction in GFR in this population is around 0.75 to 1 ml/min/year.[31] Renal replacement treatment (RRT) was necessary in just 1% and 20% of individuals with CKD stages G3 and G4, respectively, in a large study of community-based CKD by Kshirsagar et al., but 24 % and 45 % were deceased primarily from CVD, signifying that cardiac events, relatively than progression to ESRD, are the most frequent outcome in community-based CKD.[32]

Referred CKD patients, unlike those with community CKD, present at a young time of life with progressive renal impairment and loss of function due to inherited (autosomal dominant polycystic kidney disease - ADPKD) or acquired nephropathy. The rate of deterioration in GFR in DN has been established to be around 10 ml/min/year. Subjects with chronic proteinuric GN have a higher rate of advancement than those with mild proteinuria in non-diabetic nephropathies. When compared to other nephropathies, subjects with renal impairment and ADPKD, beyond CKD stage G3b, could have a higher degree of progression. In patients with hypertensive nephrosclerosis, good blood pressure control and minimal proteinuria are linked to a very slow progression.[22]

Risk Factors in Progression of CKD

Non-Modifiable CKD Risk Factors

Advanced age, male gender, and non-caucasian race, which includes African Americans, Afro-Caribbeans, Hispanics, and Asians, all impede the course of CKD.

In several kidney disorders, genetic variables that influence CKD development have been discovered. Luttropp et al. found that single nucleotide polymorphisms in the genes TCF7L2

and MTHFS were associated to DN and CKD progression in a population-based cohort study. In the same study, polymorphisms in genes coding for renal scarring mediators and the renin-angiotensin-aldosterone system (RAAS) were found to influence CKD progression.[33]

Modifiable CKD Risk Factors

Proteinuria, Systemic hypertension and metabolic variables are among them.[34] Systemic hypertension, which is the second major cause of ESRD in the United States after diabetes, is one of the most common reasons. Gliosclerosis progression is assumed to be aided by the transmission of systemic hypertension into glomerular capillary beds, as well as the resulting glomerular hypertension.[35] Blood pressure measurements taken at night and during the day (ABPM) appear to be the sturdiest predictors of CKD progression. Systolic blood pressure, somewhat than diastolic blood pressure, appears to be a better predictor of CKD development and has also remained linked to CKD comorbidities.

Significant proteinuria (albuminuria A3) has been related to a higher proportion of CKD development in both diabetic and non-diabetic kidney disease patients in multiple investigations. Furthermore, RAS inhibition or diet-induced reduction in significant proteinuria is linked to a better renal prognosis. However, despite a significant reduction in albuminuria, substantial drops in GFR were observed in intervention studies such as Avoiding Cardiovascular Events Through Combination Therapy in Patients Living with Systolic Hypertension (ACCOMPLISH)[36] and Ongoing Telmisartan alone and in Combination with Ramipril Global End Point Trial (ONTARGET)[37]. As a result, moderate albuminuria (A2) isn't a reliable predictor of CKD progression, and albuminuria reduction has been associated to both better and worsening CKD progression.

Multiple studies have linked the RAAS system to the pathogenesis of renal fibrosis, proteinuria and hypertension in patients with CKD. As a result, RAAS-targeted treatments have proven to be beneficial in delaying the course of CKD. As a result, RAAS inhibitors are now widely used in the treatment of proteinuria and diabetic renal disease.

Obesity and tobacco use have both been linked to the initiation and progression of chronic kidney disease (CKD). Hyperuricemia, lipid abnormalities, and insulin resistance are among metabolic factors that have been linked to the start and progression of CKD.[38]

Recommendations for CKD Screening

Worldwide, screening is being introduced, with a focus on high-risk persons. High-risk populations, such as those with hypertension, diabetes, or who are over 65 years old, should be screened according to the KDOQI criteria. Urinalysis, urine albumin-creatinine ratio (ACR), serum creatinine measurement with GFR estimation, ideally using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation, should all be included. Because there is minimal evidence to support screening asymptomatic patients for CKD in the general population, it is the most cost-effective option.[22]

Pathophysiology

Unlike acute kidney injury (AKI), where the healing process is complete with complete functional kidney recovery, chronic and persistent insults from chronic and progressive nephropathies lead to progressive kidney fibrosis and loss of the natural architecture of the kidney.

All three sections of the kidney, including the glomeruli, tubules, interstitium, and vessels, are affected. Vascular sclerosis, tubulointerstitial fibrosis and Glomerulosclerosis are the histopathological manifestations.

Fibrosis and scarring are multistage phenomena that occur as a result of a complicated, overlapping series of events:

• External inflammatory cells infiltrate injured kidneys.

• Proliferation, activation and injury of intrinsic renal cells (via podocytopenia, mesangiolysis, necrosis and apoptosis)

• Proliferation of extracellular matrix (ECM) generating cells, such as myofibroblasts and fibroblasts.

- ECM deposition in place of regular architecture.

Mechanisms of accentuated Progression of CKD

- Systemic and glomerular hypertension
- Glomerular hypertrophy
- Precipitation of calcium phosphate in the kidney
- Altered prostanoid metabolism

All of these mechanisms lead to a histological disease known as focal segmental glomerulosclerosis. Clinical risk factors for the development of CKD include hypertension, proteinuria, black race and diabetes. Environmental variables such as metabolic syndrome, smoking and possibly some analgesics, as well as obesity, have all been linked to a quicker progression of CKD.[39]

History and Physical examination [14]

Early stages of CKD are asymptomatic, with symptoms appearing in stages 4 and 5. Routine urine or blood tests are used to detect it. The following are few prevalent symptoms and indications of CKD at these stages:

- Nausea
- Vomiting
- Loss of appetite
- Fatigue and weakness
- Sleep disturbance
- Oliguria
- Decreased mental sharpness
- Muscle twitches and cramps
- Swelling of ankles and feet
- Persistent pruritus
- Pain in chest due to uremic pericarditis
- Pulmonary edema from fluid excess causes shortness of breath.
- Hypertension that is hard to control
- Skin pigmentation
- Scratch marks due to pruritus
- Pericardial abrasion rub owing to uremic pericarditis
- High BUN levels with sweat containing urea.
- Hypertensive fundal changes suggesting chronicity

Treatment / Management

General Management

- Changing medicine dosages to account for the predicted glomerular filtration rate (GFR)
- Arteriovenous fistula or graft insertion in preparation for renal replacement therapy

Treating the Reversible Causes of Renal Failure

The possibly reversible causes of acute renal injury, like infection, medicines that reduce GFR, hypotension from shock, and instances that produce hypovolemia, such as vomiting and diarrhea, should all be detected and treated.

Patients with CKD should be thoroughly examined before undergoing intravenous contrast investigations, and any alternatives should be tried first. Other nephrotoxic drugs to avoid include aminoglycoside antibiotics and nonsteroidal anti-inflammatory drugs (NSAIDs).[40]

Slowing the progression of CKD

Hypertension, proteinuria, metabolic acidosis, and hyperlipidemia are all variables that contribute to the advancement of CKD. In CKD, hypertension should be treated by setting blood pressure objectives. Proteinuria should also be achieved.

Smoking has been linked to the development of nephrosclerosis in multiple studies, and quitting smoking slows the progression of CKD.

Restricting protein has also been demonstrated to delay the course of CKD. However, the type of protein consumed and the amount consumed have yet to be identified.[41]

Supplementing with bicarbonate to treat chronic metabolic acidosis has also been found to slow the course of CKD.[42]

Furthermore, in diabetics, strict glucose control has been proven to delay the onset of albuminuria and the progression of albuminuria to overt proteinuria.

Preparation and Initiation of Renal Replacement Therapy

Once the patient's CKD has progressed, he or she should be offered a variety of renal replacement treatment alternatives.

- Hemodialysis (home or in-center)
- Peritoneal dialysis (continuous or intermittent)
- Kidney transplantation (living or deceased donor): It is the treatment of choice for ESRD given better long-term outcomes.
- Patients who do not want renal replacement therapy should be provided with information about conservative and palliative care management.

  • Hemodialysis is done once a non-dominant arm has been given steady vascular access. Intravenous cannulas are avoided in this arm to preserve the veins. AVF is the preferred vascular access. The AV graft and tunnelled hemodialysis catheters are two more hemodialysis access alternatives. The AV fistula has a high percentage of patency, and infections are rare. With an AV fistula, higher flows can be attained with reduced possibility of recirculation. Peritoneal dialysis is performed after placing a peritoneal catheter. [15]

1. **Haemodialysis**

Dialysis is a medical procedure that involves the elimination of toxins and excess water from the blood. The major reason people need dialysis is because their kidneys aren't working properly. The following conditions are required for dialysis: uremic syndrome, hyperkalemia,

extracellular volume expansion, acidosis, failure to respond to medical therapy, creatinine clearance of 10 ml/min/1.73 m^2, and bleeding diathesis (bleeding susceptibility due to coagulation abnormalities).[44,45]

Renal functional capability can be determined by serum creatinine/blood urea nitrogen (BUN) or urea and creatinine clearance. The two types of dialysis techniques are hemodialysis (using a machine/artificial apparatus like the kidney) and peritoneal membrane used as a filter (peritoneal dialysis). Peritoneal dialysis is preferred by younger patients since it is flexible and can be done at home. Hemodialysis is used to treat people who have lost all function of their kidneys.[46]

The hemodialysis mechanism

During hemodialysis, wastes and excess water are removed using a dialyzer, which is an external filter with a semipermeable membrane. The wastes are separated by setting up a counter-current flow gradient, with blood flowing one way and dialyzer fluid flowing the other. Peritoneal dialysis uses the peritoneum as a semipermeable membrane to remove waste and water from the dialysate.

The basic process in dialysis is the movement of solute particles over the dialysis membrane (diffusion). Along the concentration gradient, urea and creatinine, as well as other metabolic waste products, flow from the circulatory system into the dialysate (sodium bicarbonate (NaHCO3), sodium chloride (NaCl), acid concentrate, and deionized water). The speed with which the particles diffuse beyond the membrane into the dialysate is determined by their size. The rate of diffusion through the membrane slows as the size of the solute particle increases. An arteriovenous shunt is formed when arteries conveying oxygenated blood from

the heart are connected to a vein. The vein is strengthened so that it can be pierced several times (by growing muscles surrounding it like an artery), and its pressure is monitored during dialysis.

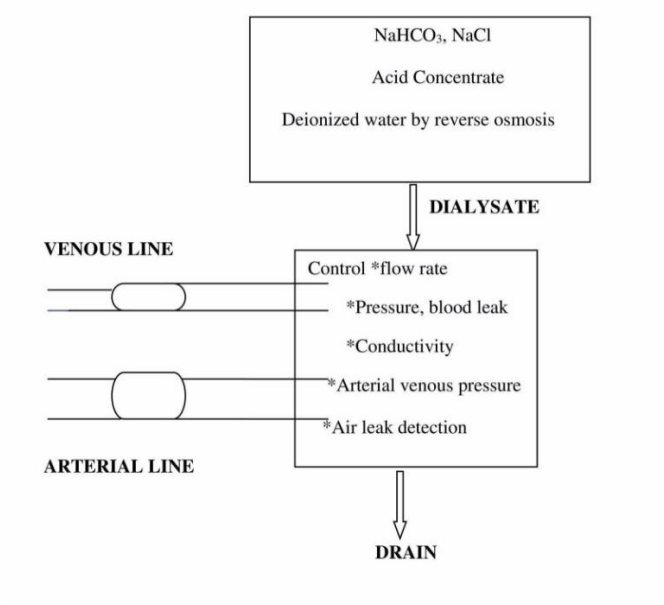


Figure1: Diagram of a Dialyzer [16]

$NaHCO_3$: sodium bicarbonate; NaCl: sodium chloride

Complications

Cardiovascular complications and dialysis

Dialysis has a range of adverse effects, from moderate (hypertension, muscle cramps, allergic reactions) to severe (cardiovascular disease (CVD)). Ongoing inflammation is the main cause of a diseased kidney that does not respond to treatment. Chronic inflammation affects the kidney's natural function, causing metabolic wastes to accumulate in the body. Dialysis aids in the removal of toxins from the body, and the kidney may progressively regain function; however, as shown in Figure 2, this is dependent on the individual's current status of health.

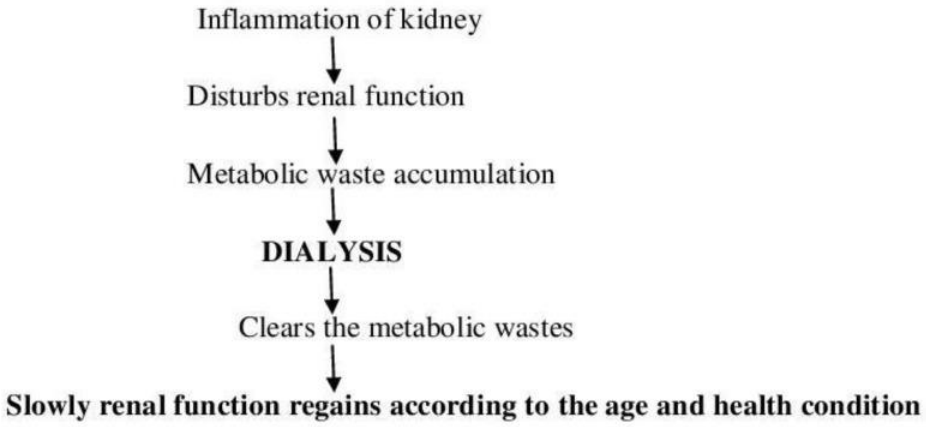


Figure 2: Flow Chart depicting Role of Dialysis and the Stages [16]

Indications

Acute sickness linked with AKI, life-threatening hyperkalemia, refractory acidosis, hypervolemia producing end-organ problems (e.g., pulmonary edema), or any hazardous intake need beginning of hemodialysis. These diseases produce cytokine (immune response modulator) dysregulation and poor clearance, resulting in vasodilation, cardiac depression, and immunosuppression, resulting in end-organ damage, hemodynamic instability or a delay in renal recovery. In high-cytokine situations like sepsis, ARRT improves cytokine elimination. Catheter problems, electrolyte abnormalities, and intradialytic hypotension all have the potential to cause injury.

The Kidney Disease Outcomes Quality Initiative (KDOQI) of the National Kidney Foundation has published hemodialysis adequacy guidelines (2015 update).[47]

It is recommended that people with CKD stage 4 (GFR, 30 mL/min/1.73 m^2) and those who are on the verge of needing maintenance dialysis during the initial assessment be counselled about renal failure and treatment options (kidney transplantation, hemodialysis at home or in

a centre, PD) as well as conservative treatment be counselled about renal failure and treatment options (kidney transplantation, hemodialysis at home or in a centre). It is also necessary to educate family members and caregivers. The decision to start maintenance dialysis should be based on renal failure features (pruritus, acid-base or electrolyte abnormalities, serositis), volume or blood pressure dysregulation, a progressive worsening of nutritional status despite dietary intervention, or cognitive impairment. It should not be based on an asymptomatic person's renal function level.

Arrhythmias caused by electrolyte imbalances, uremic pericarditis, and fluid overload caused by severe congestive heart failure caused by poor kidney function are all conditions that necessitate dialysis. Electrolyte (calcium, magnesium, and potassium) imbalances are the most prevalent arrhythmias after structural heart defects. Acidosis (due to intercellular shift) and decreased renal excretion cause potassium anomalies in patients with chronic kidney failure. In cardiac patients, iatrogenic causes include the misuse of ACE medications, angiotensin-converting enzyme inhibitors, and aldosterone antagonists. Elevated urea levels in renal failure patients can cause uremic pericarditis. Fluid retention happens in patients with CKD and heart failure, leading to worsening heart failure and pulmonary edema.

Shortness of breath, exertional dyspnea, and paroxysmal nocturnal dyspnea are common symptoms. The majority are known cases of CHF with fluid overload. Nausea, bloating, edema, cough, lethargy, and weight gain are some of the other indications and symptoms. Due to diminished brain perfusion, patients with arrhythmia experience anxiety, palpitations, lightheadedness, and syncope. Chest pain can occur, although it's usually caused by ischemia or tachycardia, which causes demand ischemia. Apart from fever, bradycardia, and

hypotension caused by autonomic inhibition, pleuritic chest discomfort with radiation to the trapezius, left shoulder or arm is noted with the onset of pericarditis. In the supine position, the discomfort is the worst, but tilting forward relieves it.

Patients may appear relaxed or dyspneic on physical examination, depending on the quantity of fluid present and NYHA (New York Heart Association) stage of congestive heart failure. After exercise, many patients improve with little rest. Patients may have ascites, wheezing, and frothy red sputum, as well as rales at the base of the lungs. Hepato-jugular reflux can also cause jugular venous distension. Due to the rushing of additional fluid in the left ventricle, a third heart sound (S3) may be heard. It's possible that pulse maybe erratic or non-existent. Hypotension, tachypnea, disorientation, and diaphoresis are all symptoms that might occur.

A transitory pericardial friction rub is always indicative of acute pericarditis. When the patient is leaning forward, auscultation of the apex or left sternal edge during expiration, increases the chances of hearing the pericardial rub.[48]

Contraindications

The inability to secure vascular access is an absolute contraindication to hemodialysis, while poor vascular access, needle phobia, heart failure, and coagulopathy are all relative contraindications.

In patients with significant vascular disease, modern procedures are used to improve the formation and preservation of vascular access. The use of local anaesthetics and nursing encouragement can help overcome relative contraindications like needle aversion. The maintenance of anticoagulation in the extracorporeal circuit is complicated by severe coagulopathy.

The physician has an obligation to accept the patient's decision to refuse dialysis therapy if the patients communicates and ask him to do so. Nonetheless, the nephrologist must ensure that all reversible issues, such as unwarranted anxieties about dialysis or a depressive illness clouding judgement and requiring a mental evaluation, are adequately addressed. In such patients, especially those with many comorbidities, a conservative approach is taken, with all appropriate treatments to avoid dialysis being used.

Patients with a bad quality of life should be spared discomfort of HD, since survival on dialysis may no longer be possible, with the majority of the extra time spent on dialysis treatments and recovery. Medication and nutrition, as well as pain management with analgesics, are used to treat the symptoms of ESRD and associated consequences. Severe itching and sleeplessness can be treated with low dosages of gabapentin or pregabalin.[48]

Complications

The following are the most common hemodialysis complications:

- Intradialytic Hypotension: Due to increased mortality and a higher rate of regional wall motion anomalies during dialysis, known as myocardial stunning, it has poor long-term prognosis. A nadir systolic blood pressure of less than 90 mm-Hg has the strongest link to mortality. Dizziness, light-headedness, nausea, or other minor symptoms are common symptoms. Maintaining the patient in the Trendelenburg position and promptly delivering a 100 mL bolus of normal saline through the blood line are the mainstays of treatment. Reduce the ultrafiltration rate to the bare minimum and monitor the patient until vital signs have returned to normal.
- Muscle Cramps: The cause of the disease is uncertain. Cramps are exacerbated by hypotension, a high ultrafiltration rate, hypovolemia, and a low-sodium dialysis

solution. These variables cause vasoconstriction and muscle hypoperfusion, as well as muscle relaxation impairment. Treatment with 0.9 percent saline is helpful when it occurs in conjunction with hypotension. A forced stretch of the affected muscle may bring relief.

- Some reactions are medical emergencies that require prompt dialysis stoppage, line clamping, and supportive treatment, followed by particular decisive therapy.[49]
- The following are some examples of such complications:
- Dialysis Disequilibrium Syndrome: Patients with this condition are more likely to develop it during or shortly after their first therapy. Neurologic degeneration, restlessness, mental confusion, headache, periodic muscle twitching, and coma are all symptoms of this clinical condition. The transfer of water into the central nervous system (CNS) creates elevated intracranial pressure due to a significant gradient between the urea concentrations in the CSF and blood. Seizures and cerebral edema are more common in patients having rapid dialysis. An acceptable urea concentration reduction goal is 40% in two hours, with a URR of 0.4. An osmotic agent could be added to the blood to prevent the gradient from forming. Sodium, mannitol, high glucose dialysate, and glycerol are commonly used. Increasing the salt concentration of the dialysate throughout the treatment may be advantageous.
- Dialyzer Reactions: Dyspnea, raised body and local temperature at the fistula site, a sense of impending doom, itching, urticaria, coryza, watery eyes, stomach cramping, and diarrhea are all symptoms of anaphylactic type A reactions. Hypersensitivity to the ethylene oxide used to sterilize dialyzers might cause symptoms to appear at any point within the first 30 minutes after dialysis. Intravenous antihistamines, steroids, and epinephrine are used to treat the condition. Dialyzers should be rinsed thoroughly

before use to remove any remaining allergens and to help avoid it. Complement activation causes nonspecific type B dialyzer reactions, which induce chest or back pain 20 to 40 minutes after starting dialysis. Changing the dialyzer membrane could help prevent this.[48]

- Hemolysis: Acute hemolysis during dialysis is characterised by the appearance of port wine in the venous blood line, a significant drop in the hematocrit, and pink-colored plasma centrifuged blood samples. Hematologic tests should be performed on the patient, and he should be monitored for delayed hemolysis. To determine the reason, a dialysate sample must be examined.
- Air Embolism: There is foam in the dialyzer's venous bloodstream, which is a deadly consequence. On chest auscultation, a churning sound might be heard. Place the patient in a left lateral recumbent position, give 100 percent oxygen through a mask, and use a percutaneously inserted needle or cardiac catheterization to remove air from the heart chambers.[48]
- Other nonspecific problems include nausea and vomiting (10%), headache (70%), chest discomfort and back pain (1% to 4%), and itching (1% to 4%). These are most likely connected to hypotension or a first symptom of disequilibrium syndrome. The symptoms are resolved when the accompanying hypotension is treated. A single dose of 5 to 10 mg metoclopramide given prior to dialysis is sufficient. Acetaminophen, which is given during dialysis, can help with headache management. Itching induced by low-grade hypersensitivity to blood circuit components could be reduced by switching to a different type of dialyzer membrane. The most powerful factor of a dialysis patient's quality of life is vascular access failure, most typically stenosis of arteriovenous access. There is a reduction in blood flow and risk of thrombosis.[50]

Blood flow is also hampered by usage of a catheter-related fibro-epithelial sheath. Re-establishment of access is by instilling urokinase, endovascular catheter stripping, or replacement of an indwelling dialysis catheter in a subcutaneous tunnel.[51]

- Precautions for vascular access: Avoid causing extra damage to the arm at the access site by not wearing tight clothing/jewelry, carrying heavy items, or sleeping on it. On the access, rotate the needle insertion site. After removing the needle, use mild pressure to stop the bleeding. Recurrent bleeding can be stopped with light pressure; if the bleeding lasts more than 30 minutes, contact a health care practitioner. Hemorrhage in dialysis patients is caused by the use of heparin and can be effectively treated with protamine sulphate. Detecting a line separation via monitoring venous and arterial pressures, fastening and taping needles, using wetness detectors for blood and dialysate leaks, and using closed connector devices for tubing junctions. Local site examinations for symptoms of infection, such as redness, warmth, and discomfort, must be performed on a regular basis.

2. AV fistula

AVFs are aberrant connections between an a vein and an artery.[17] In certain contexts, these may also be referred to as arteriovenous malformations.[18]AVFs can exist almost anywhere in the body, depending on the etiology. These can be divided into two groups, acquired or congenital. Acquired fistulas can be further subdivided into surgically created, as in for hemodialysis, or secondary to trauma, whether accidental or procedure-related.

The anatomy of the fistula depends on location in the body. AVFs for hemodialysis are typically created in the extremities, with the upper extremity being generally preferred over the lower extremity by vascular surgeons. Surgically, the cephalic and basilic veins are

commonly utilized to produce an AVF. Although the radio-cephalic AVF is the recommended initial access for hemodialysis, the radial artery at the volar wrist and the brachial artery at the antecubital fossa and medial upper arm are common anatomical locations for fistula creation.[19]

Two types of lower extremity surgical AVFs for hemodialysis have been described in the literature. The superficial femoral vein or popliteal vein can be mobilized from the knee to anastomose with the superficial femoral artery; this is called an SFV transposition. The saphenous vein can be anastomosed with the common femoral artery to form a loop AVF on the anterior thigh.[20,21]

Although no type of congenital AV fistula is common, reported locations for congenital AVFs include pulmonary, aorto-caval, dural, carotid-cavernous, coronary, and hepatic.[22,23] While the majority of neck fistulas do occur secondary to trauma, congenital vertebra-vertebral fistulas and carotico-jugular fistulas have been described in children.[24]

AVFs as a result of iatrogenic injury are typically as a result of surgical procedure, invasive line placement, or needle biopsy. The literature demonstrates multiple reports of iatrogenic injury resulting years after surgical procedures. [25,26] Traumatic AVF can essentially occur anywhere there is resulting trauma, and these can also have a late presentation.[27] Greater than 50% of traumatic AVFs happen in the lower extremity, and about one-third occur in the femoral vessels, while 15% take place in the popliteal vessels.[28]

Etiology

Arteriovenous fistulas can be surgically created for hemodialysis access, can occur as a result of a congenital anomaly, or be secondary to iatrogenic injury or trauma. Penetration of any mixed-type vasculature can ultimately result in the healing of arteries and veins together, bypassing downstream arteriole and capillary system.

Iatrogenic fistulas are most commonly reported as a result of percutaneous access of the femoral vein and femoral artery during cardiac catheterization; although, subclavian and carotid fistulas have been reported in association with the placement of central lines.[29] The most common AVFs described as a result of percutaneous biopsy are renal; however, these are typically self-limited, and very few require intervention.[30]

Traumatic fistulas are often associated with direct arterial trauma and long bone fractures, especially where an artery and vein are in close communication. Ninety percent of traumatic AVFs are due to penetrating trauma, the majority of which are gunshot wounds.[31] A small portion of neck AVFs can occur in association with a hyperextension injury or spine surgery. Carotid-cavernous fistulas, typically due to trauma, can be fatal and are most often associated with a basilar skull fracture, penetrating trauma to the area, and ruptured aneurysms.[32] Two-thirds of traumatic AVFs are diagnosed within one week of the injury; however, some may present weeks to years after the event.[33,34]

Congenital fistulas are not well-understood. Central nervous system congenital AVFs can be dural or carotid cavernous. AVFs of the neck are mostly due to trauma; however, fibromuscular dysplasia, neurofibromatosis, and other types of collagen

disorders have been associated. Pulmonary vascular malformations are typically simple and more similar to AVFs than to a true malformation. Other types of AVFs are very rare.

Epidemiology Arteriovenous fistulas were originally described by William Hunter as early as 1757. [28] Much of the experience in managing AVFs originated out of traumatic injuries resulting in AVFs from the Second World War, the Korean War, and Vietnam War. 215 AVFs and aneurysms were reported due to the Korean War. In the civilian population, traumatic AVFs of the abdomen and extremities are equally distributed, unlike the military, where the majority of traumatic AVFs occur in the extremities.[31] This difference is likely on account of body armor worn by the military. Demonstrably, traumatic fistulas are much more common than congenital fistulas and are more frequently discussed. Congenital fistulas are generally rare and found in case reports and small studies.[35]

The National Institute of Diabetes and Digestive and Kidney Diseases reported as of 2013, over 468,000 patients were on hemodialysis. Twenty percent of those patients receive dialysis via a surgically created AVF. In the HEMO (hemodialysis) Study published in 2000, AVF prevalence varied between dialysis centers from 4-77%. The authors discovered a decreased prevalence of AVF compared to other means of HD in females, blacks, obese, elderly, and in patients with peripheral arterial disease. Geographically, the rate of AVF creation varies greatly, with the highest rates being in the Northeast and the lowest in the Southwest of the United States.[36]

History and Physical examination:

The presentation of arteriovenous fistulas can vary depending on the location and etiology. Patients with an AVF for hemodialysis will present with evidence of a surgical incision on the lateral wrist, volar forearm, or upper arm. A working AVF will have a palpable thrill and continuous bruit. A patient with an AVF with an outflow obstruction may present with a pulsatile fistula or prolonged bleeding from a puncture site from hemodialysis. Superficial fistulas have a palpable thrill, a bruit, or even a pulsatile mass. It may be possible to auscultate a machinery-like murmur over the fistula.[37]

Fistulas of the extremities, regardless of etiology, may present with signs of venous hypertension, including varicosities, pain, and swelling. In case of a long-standing fistula, there may be significant size discrepancy between the two limbs. If the patient reports a history of trauma, anywhere from weeks to years after injury, in particular with long bone fractures or ongoing neurologic deficits, index of suspicion for AVF should be raised even if it is a clinically normal exam.[31]

Patients with congenital AVF may not present until later in life, and a history of trauma should be ruled out. Depending upon location, these fistulas may be low-flow at birth and become high-flow lesions in adulthood. Patients with brain arteriovenous malformations can present with headache, neurologic deficits, seizures, or a combination. These patients can also be at risk for hemorrhage or ischemia to the parenchyma surrounding the AVM/AVF due to steal syndrome.

In severe, chronic, or high flow fistulas, patients can present in high output cardiac failure, which results in shunting of oxygenated blood back to the right heart. Due to the shortcut that the arterial blood takes through the venous system, this results in decreased peripheral resistance. The total circulating blood volume is raised to maintain blood pressure, resulting

in heart failure. The Nicoladoni-Israel-Branham sign is a condition characterised by reflex bradycardia and fistula compression due to increased afterload.[24]

Evaluation

Given that AVF is superficial, a duplex ultrasound (US) is a non-invasive and economical technique to confirm the diagnosis of AVF in addition to the clinical evaluation from history and physical examination. A duplex US will demonstrate low resistance flow in the feeding artery. At the anastomosis or fistula, turbulence and high-velocity flow are demonstrated. Thickened walls and high-velocity flow will be seen in the dilated draining veins or venous plexus of the AVF. A pseudoaneurysm, venous aneurysm, or a dilated feeding artery may also be demonstrated on the ultrasound exam.[29]

Computed tomography angiography (CTA) and magnetic resonance angiography (MRA) both show early contrast filling in the arterial phase in the involved vein. While MRA may not be an option in trauma or post-trauma patient due to residual metal, CTA is reliable, non-invasive, and accessible as an initial diagnostic test. Disadvantages of CTA include streak artifact from metallic object, which can be an issue in penetrating trauma, motion artifact, and reliance on contrast timing in the fistula.[38]

Selective angiography is the gold standard and has shown better accuracy over CTA. It is the most intrusive test for an AVF, yet it pinpoints the precise spot of arteriovenous communication, including vascular anatomy, flow dynamics, and treatment mechanisms.[39] The disadvantages of angiography include the cost, the time it takes to

perform the procedure, the requirement for additional arterial or venous access, and a well trained team.

Patients with ESRD who require vascular access should be examined to determine the best location for an AVF. Given the preference of the upper extremity, non-dominant over dominant, forearm over the upper arm, all patients should be evaluated with a duplex ultrasound scan, including patients presenting for evaluation of an existing AVF. For the best results and primary patency rates, criteria have been created. Clinical exam, in addition to vein mapping, helps to decrease primary failure rates and decrease negative surgical exploration in first attempts to create AVFs.[40]

Treatment / Management

In the past, most occurrences of arteriovenous fistulas were treated conservatively during wartime and then surgically if necessary. Early intervention, however, may deter complications of AVFs that can be avoided, and post-traumatic fistulas should be closed as close to diagnosis as possible.[37] The goal of AVF treatment is to isolate and close the fistula while attempting to maintain essential blood flow. Repair may be completed by direct primary repair, reconstruction (autogenous or prosthetic graft, or bypass), or endovascular repair.[28]

Indications for treatment are simple:

- Hemodynamic instability, availability of a surgical team, injury to adjacent tissue, and unsuccessful endovascular repair are all indications for open surgical intervention on a fistula.

- In traumatic fistulas, failure of spontaneous regression within two weeks indicates repair.
- Congenital fistulas are typically repaired when they present due to sequelae in later life, as many may remain asymptomatic until adulthood.
- Hemodialysis fistulas that are no longer functioning, no longer needed, have endured multiple attempts at salvage, or fail to mature should be considered for ligation.[41]

Endovascular management is the preferred method of management for AVFs.

- The first endovascular repair was done in 1992 by Parodi, of a traumatic subclavian AVF; a covered stent was used.
- The advantages of endovascular repair include rapid recovery, less post-operative pain, and less disability compared with the open approach.
- Coils, stent-grafts, covered stents, cyanoacrylate glue, are among the options for closing the fistula.[42]
- Stent grafts are used to exclude the fistula while preserving the essential artery and vein; use of coils is with a low risk of complication to embolizing essential vessels. Endografts designed for larger vessels, such as the aorta or iliac vessels, can be used similarly—stent graft patency rates at a one-year range between 88 and 100%.
- Complications involved with placement of covered stents include arterial dissection or rupture, migration of the device, or embolization. In a review of patients with traumatic AVFs, the most common reason for the failure of the endovascular intervention was the inability to advance the guidewire.
- The patient may also develop intimal hyperplasia at the placement site of the stent or subacute thrombosis, leading to occlusion and distal ischemia. If the patient does

develop venous thrombosis, anticoagulation should be initiated and continued for six months.

- Embolization can be advantageous for complex AVFs with multiple feeding and draining vessels. Deep-seated or intra-organ and adjacent to critical anatomy, AVFs are much more amenable to endovascular management than open surgery.
- Contraindications to endovascular management include contrast allergy.

Open surgery is an option for repair when endovascular management fails.

- In addition to the need for surgical exposure of the fistula and increased risk of bleeding, an open approach is linked to a high rate of morbidity., including venous stasis, limb (or distal) ischemia, limb loss, venous thrombosis, and pulmonary embolism.
- Repairs can be made via autogenous (usually saphenous vein) grafts, synthetic grafts, venous ligation (diameter dependent), bypass, or complex reconstruction involving one or more of the above.[41]
- Limitations for using the saphenous vein autologous graft include needing a vein of 3-8 mm, no lower extremity venous insufficiency, and no thrombophlebitis of the vein.[28]
- Conservative management is an option if no symptoms or complications exist or if the AVF is expected to be self-limited.

Differential Diagnosis

- **Arteriovenous malformation:** These are congenital lesions from failure of fetal vascular differentiation. High flow or complex AVMs usually have high resistance,

similar to a capillary bed, and therefore, tend to be asymptomatic. AVMs can be differentiated from AVFs with high resonance imaging (CTA or MRA).[43]

- **Hemangioma:** Hemangiomas, also known as vascular tumors, may have similar symptoms to AVFs. However, these tend to present with overt bleeding. Hemangiomas have been found to have mitotic activity and can rapidly involute. Similar to AVMs, these can be ruled out with CTA or MRA.[43]
- **Pseudoaneurysm:** These may present as a pulsatile mass associated with a vascular access procedure. Pseudoaneurysms, as well as true aneurysms, may have a thrill due to significant turbulence through the area. Doppler US can differentiate these from a fistula.
- **Malignancy:** Malignant masses can require significant blood flow and present as a previously unnoticed pulsatile mass if close to a larger vessel. Doppler US and/or multiphasic imaging can help to differentiate these.
- **Cyst/Abscess/Hematoma:** These are typically identified as simple hypodense lesions on the duplex US and will not demonstrate flow.[41]
- **Prognosis** While some congenital arteriovenous fistulas can be fatal, leading to failure to survive, the overall prognosis is good. Peripheral arteriovenous fistula does not typically incur systemic hemodynamic effects, and around 15% of all AVFs do. The Schobinger Classification is a clinical staging system that serves to predict the success of treatment. The stage of quiescence (I) is described as cutaneous blush and skin warmth at the fistula site. The second stage (II) demonstrates darkened skin, a pulsatile lesion with a bruit on auscultation, or palpable thrill. This stage is called expansion. Stage III, the destruction phase, is essentially steal syndrome and is characterized by skin changes, ulceration, and distal ischemia. Stage four (IV) is the

decompensated phase, characterized by high output heart failure. While it has been shown that decompensated heart failure can be completely reversed after a high-flow AVF is closed, the prognosis is much better in patients who present earlier.[43]

- **Complications:** AVF has a plethora of complications:
- **Chronic venous insufficiency / venous hypertension:** Venous hypertension can present as swelling of the affected extremity, which can be progressive and limit mobility. Insufficiency manifests itself as skin stasis coloration, varicosities, and ulcerations. This complication is a direct result of arterial pressure against the thinner venous walls and failure of one-way valves in the veins.
- **High cardiac output failure:** In a study of 120 patients who suffered from high-output heart failure, 23% had an AV shunt, which includes congenital, traumatic, and HD fistulas. In all patients with high-output cardiac failure, their increased stroke volume was secondary to decreased vascular resistance, which is a mechanism found in subjects with AVFs, particularly when these become high-flow.[44]
- **Arterial insufficiency:** Steal syndrome can occur in up to 6% of subjects with an AVF or arteriovenous graft for hemodialysis. It is a result of a high-flow fistula causing distal ischemia. These patients can present with severe pain associated with use or during hemodialysis.[43] They may also have swelling or signs of ischemia distal to the fistula, such as cool skin, discolored fingers or toes, diminished distal pulses, hair loss, or atrophy. Increased overall flow to the affected extremity is a typical compensating strategy for the fistula, ensuring adequate blood flow to the higher resistance distal capillary beds; when this mechanism fails, steal syndrome results which draw blood through the lower resistance fistula instead of supplying distal tissue. Inflow stenosis of the arterial supply can be a cause of or worsen steal.

Compression of the fistula will enhance input to the symptomatic distal extremity in the examination for steal syndrome.[44]

- **Hemorrhage:** Hemorrhage is a much rarer complication than those above. Due to the high flow through the venous system, dilatations can occur throughout the venous drainage system. These can become thin-walled and be at risk for rupture. This can be especially problematic with hemodialysis fistulas which are repetitively accessed through the venous side causing ulcerations to the vein and overlying skin. After dialysis, establishing hemostasis can be difficult due increased flow via the fistula as well as outflow stenosis.[45]

3. Role of Doppler ultrasonography for arterio-venous access planning and vessel mapping

Preoperative vascular assessment

AVF vessel selection was exclusively based on a clinical examination of upper limbs, which is of low-cost, bedside operation that requires no special equipment. This method gives sufficient information on the superficial venous circulation (diameter, vascular palpability, course, and patency), but significantly less information on the arterial vessels (based on the results of the Allen test).Additionally, in a significant percentage of patients (25–50 %), a clinical examination is not sufficient alone.[46] DUS takes longer than physical testing and demands the use of a skilled examiner as well as specialized equipment. It does, however, provide valuable information on the arm's superficial and deep veins and a plethora of extra data about arterial circulation. It's also fully reproducible, noninvasive and risk-free.[47]

The only diagnostic imaging technique that can simultaneously observe an area's anatomy and blood supply (B-mode imaging) is DUS (Color Doppler imaging). It is also the only investigation that can be completed by the doctor who will be constructing the vascular anastomoses, which is an undeniable benefit. According to some writers, DUS should not be used as routine preoperative evaluation, but rather when anomalies are discovered during the physical exam. However, international guidelines advocate it as a complementary investigation to the physical examination in all AVF candidates. [48]

Technical necessities and inspection technique

Ultrasonic scanner utilized to map upper-extremity vessels should have a linear probe with frequencies of at least 7 MHz for B-mode and with 5 MHz for Doppler studies. To avoid elbow flexion, patient has to be assessed in supine position with trunk slightly elevated. He or she could possibly be standing in front of operator with his or her forearm propped up on a stand. Supine posture is preferred by most examiners because it allows them to more readily check vascular architecture of arm (subclavicular axillary region) and comforts the patient during examination.[49] Investigation has to be performed in warm environment, with gel warmed as well, to prevent vasoconstriction of vessels being examined.[50] In order to analyze arterial and venous structures, transverse and/or longitudinal scans of arteries and veins should be done. Based on operator's preference and characteristics of individual patient, examination may begin with arterial or venous component. B-mode morphological assessment, as well as Colour and Doppler evaluations of arterial and venous blood flow, are all part of a comprehensive examination of the arm's circulation.[41]

Preoperative arterial mapping

Before surgery, radial, ulnar subclavian, axillary, and brachial arteries should all be examined.[4] Most arterial examinations, however, begin with the distal subclavian artery or even brachial artery in practise. When anomalies are discovered at these levels, the inquiry is moved to proximal subclavian artery.[51]

DUS enables a thorough assessment of the arm's artery circulation based on a variety of variables.[49] All morphological features to evaluate include vessel wall thickness, vessel diameter, vessel course, vessel wall alterations, and any steno-disruptive lesions that may be present. During the functional assessment, blood flow and the ability of the artery to dilate are measured.

Both transverse and longitudinal scans are used to determine an artery's internal diameter, however longitudinal scans allow for a more precise determination of intima–intima distance by imaging intimal layers of superficial and deep vessel walls.[51] To assess accuracy of these data, sonographically calculated vessel diameters were compared to measurements taken straight during operation, and the correlation was good. In Radial-cephalic fistulas, the association between artery diameters and AVF outcomes has been investigated. When arteries with small-caliber (1.5–1.6 mm) were used to build AVF, immediate (during surgery) and early (in the first 8–12 weeks after surgery) AVF failures were shown to be quite common. If the arterial diameters were less than 1.5 mm, Malovrh et al. discovered immediate and early failure rates of 55 % and 64 %, respectively; however, when arterial diameters were higher than 1.5 mm, the rates were much lower (8 % and 17 %, respectively).[52]According to Parmar et al,[53] diameters of arteries which was less than 1.5 mm

had a 46 % early failure rate, while vessels with diameters more than 1.5 mm had no failures. All AVFs produced using arteries with calibre less than 1.6 mm failed prematurely, according to Wong et al.[54] Patent fistulae had radial widths of 2.7 mm preoperatively, compared to AVFs with 1.9 mm that failed in another investigation.[55] In their experience, Silva and colleagues recommended minimum diameter of 2 mm, which was associated with failure rate of 8% and primary patency rate at 1year of 83 %.[56]

Even when the artery diameter is less than 1.5 mm, AVF accomplishment rates of around 50% have been documented.[53] As a result, establishing exact radial artery diameter is improper; instead, keep in mind that diameter of artery used to construct the fistula affects chances of AVF patency and endurance.[49] This also implies the fact that artery diameter is just one of many factors that affect chances of an effective AVF formation. It must be evaluated in concurrence with other clinical and ultrasound criteria that provide information on anatomic and functional status of artery with optimum location for AVF installation. To put it another way, artery's functional quality is a crucial component in AVF success, and it isn't always linked to vessel's internal diameter (as shown, for example, by experience with AVFs shaped in paediatric patients).[57]

There are no criteria for diameter of brachial artery, which is intrinsically larger than radial artery. As a result, its evaluation is less critical to surgical procedure's outcome.[47]

A high-resolution B-mode analysis of changes and thickness of vessel walls can easily detect arteriopathy, which can compromise success of AVF's. In subjects with chronic renal insufficiency, diabetes, and atherosclerosis, arterial wall alterations are prevalent.[49] Scanning the arteries distal wall longitudinally can calculate the intima-media thickness. Increased

thickness appears to be closely connected with fistula failure, and ultrasonographic measurement demonstrates good association with histologic assessment.[58] Calcifications appear in the form of patches of hyperechogenicity with or without posterior shadowing inside artery walls and abnormalities in intimal lamina on sonography. Although these changes are easy to spot, quantifying them is more complicated. Furthermore, they are not contraindications to the progress of a fistula, despite the fact that they can affect the outcome and/or make operative procedures more difficult.[49]

DUS can also detect stenotic and obstructive arterial lesions, and vascular aberrations like brachial artery bifurcation in most proximal area of arm (sensitivity 90% and specificity 99 %).[59]

As previously stated, the functional study evaluates blood flow and the artery's capacity to dilate. On longitudinal scans, mean flow velocity (cm/s) and the vessel diameter can be measured to assess blood flow. However, just a few studies have looked into the usefulness of these measures in predicting AVF outcome. [60] Malovrh et al.[49] discovered that radial artery flow greater than 50 ml/min had successful radial-cephalic AVF construction, and Sato et al. [60] discovered that preoperative radial artery flow of less than 20 ml/min was related with an augmented risk of primary AVF failure within 8 months of surgery.

Following operative procedure, vascular dilatation that feeds AVF is connected to appropriate fistula maturation. Consequently, blood flow inside vascular access rises, and formerly triphasic (high resistance) arterial spectrum becomes biphasic (low resistance). During reactive hyperemia test, changes in radial artery Doppler spectrum can be utilised to

assess artery's ability to widen its calibre (distensibility).[49] During the ischemia phase, the artery's Doppler spectrum is often triphasic, indicating substantial resistance. If the artery dilates during reactive hyperemia phase, arterial spectrum becomes biphasic.

The resistance index (RI) [RI = (PSV - EDV)/PSV] is used to measure this spectrum change; lesser the RI, larger the intensity of reactive hyperemia. According to Malovrh et al, absence of reactive hyperemia (a RI >0.7 when fist is opened) indicates an insufficient increase in arterial flow throughout the test, which is indicative of early postoperative AVF failure. These data imply that the reactive hyperemia test is an excellent predictor of arterial function, and it is especially useful for deciding which artery to use and which surgical location (wrist, forearm, or elbow) to use for AVF construction.[49]

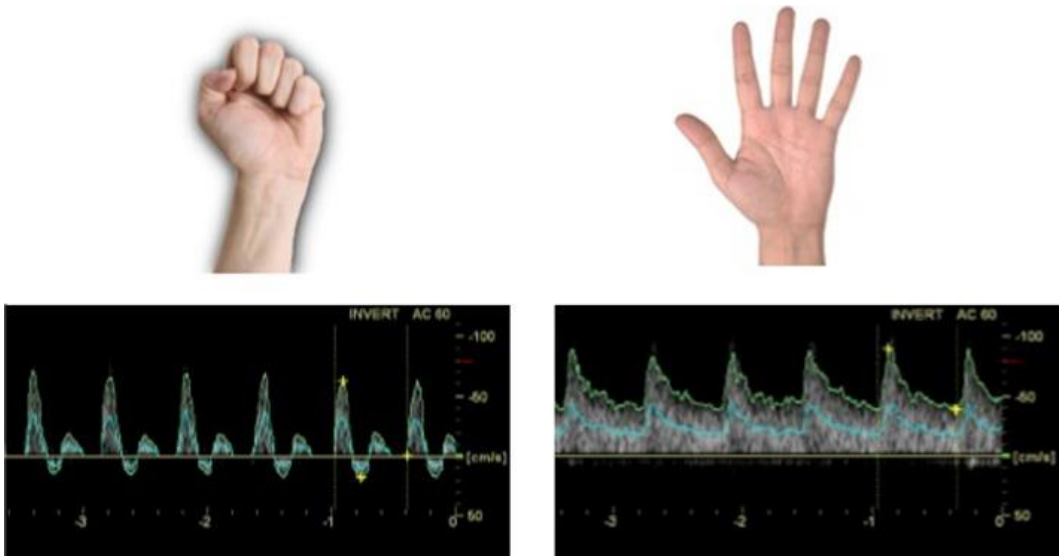


Figure 3: Test for reactive hyperemia. *Right*: Doppler spectrum during the reactive hyperemia phase with the hand opened, *left:* Ischemia phase with fist closed and matching Doppler spectrum. [57]

Preoperative venous mapping

Preoperative venous thrombosis assessment involves a thorough evaluation of the superficial and deep venous networks in the upper limb, from wrist to the central veins by using Doppler Ultrasound. Although direct view of the proximal subclavian vein and innominate vein is not normally possible, the latter veins can be evaluated with ultrasound up to the distal portion of the subclavian vein. DUS cannot be utilized to evaluate the superior vena cava because it is positioned inside the rib cage.[50]

Transverse scans are used to study the superficial venous circulation, starting with the cephalic vein and continuing to the point where it drains into the deep venous system. A tourniquet is placed around the root of the arm, and transverse scans are used to study the superficial venous circulation, starting with the cephalic vein and continuing to the point where it drains into the deep venous system. Basilic vein should be assessed along its whole course, however this is normally only done if the cephalic vein is not suitable for AVF development. This data can be used to construct a map of the superficial venous circulation (Figure 4).[47]

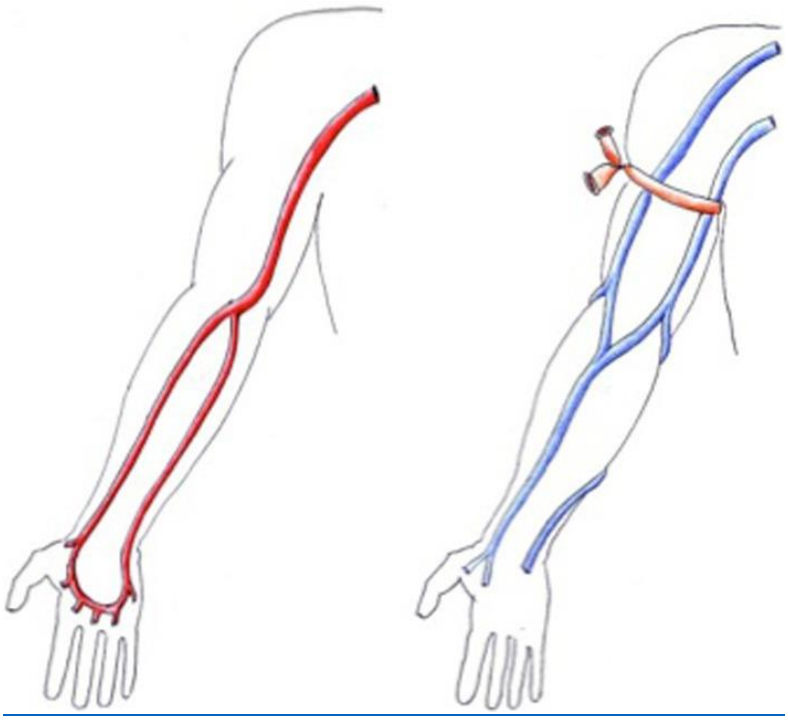


Figure 4: Preoperative vascular mapping examples. *Right*: venous mapping, *left:* arterial mapping [57]

Several ultrasonography criteria can help determine whether a superficial vein can be used to build an AVF. The vein wall's appearance, the vessel's trajectory, patency, diameter and distensibility, and the presence of collateral circuits are all things to think about.[47,50]

A normal vein's walls are thin and regular, and the lumen is completely anechoic. The vein must be sufficiently linear (across a distance of at least 8–10 cm) and located less than 6 mm below the skin surface to allow venipuncture.[61] The transducer is used to apply intermittent pressure to the veins, causing the vessel walls to completely collapse.[47]

Non-compressibility of the vein under transducer pressure implies obstruction and is typically associated with echogenic material in the lumen. If there are any doubts, a Color Doppler module with a low pulse repetition frequency or checking for the Doppler trace in a

longitudinal scan can be used to confirm the vein's patency. A normal venous Doppler spectrum is characterised by continuous, low-velocity flow that becomes increasingly phasic as the test continues towards the central veins; the absence of such flow validates the presence of an obstruction. Doppler spectrum analysis can also be utilised to determine the innominate veins' and superior vena cava's patency.

In fact, at the level of the subclavian and internal jugular veins, the presence of flow that varies in velocity with respiratory and cardiac activity is an indirect indicator of the patency of the ipsilateral innominate vein and the superior vena cava, whereas a monophasic curve indicates steno-occlusion.[49,50] Any suspicions of steno-thrombotic lesions involving a central vein should be confirmed by phlebography. [61]

The vein's diameter should be measured numerous times in the arm. On longitudinal or transverse scans, this can be done.[62] Gel should be used liberally to limit the possibility of underestimate by preventing the transducer from exerting excessive pressure. Despite the fact that fistulas made with small-caliber veins (1.6 mm) are more likely to fail early,[54] there is no consensus on the minimum cephalic vein diameter that will ensure good Radial-cephalic AVF development.[54] When a tourniquet is used, Silva et al.[56] recommend a minimum diameter of 2.5 mm; in the absence of a tourniquet, Mendes et al.[63] recommend a diameter of >2 mm. There are no well-documented guidelines for the min diameter of arm veins, however a value of at smallest 3 mm is recommended.[62]

The vein dilates as a result of the increased blood flow once the AVF is created. The vein's ability to dilate (venous distensibility) can be measured during preoperative mapping. The

vessel's diameter is measured before and after a tourniquet (or a sphygmomanometer cuff inflated to a pressure of 50–60 mm/Hg) is applied for at least 2 minutes, and the percentage increase is determined.[64]

The presence and diameter of auxiliary veins have been linked to the chance of non-maturation, according to certain experts. Wong et al.[54] discovered that supplementary veins < 5 cm from the anastomosis location can mark the fistula's functionality, but Beathard and colleagues emphasised the importance of the vein's diameters and reported greater rates of non-maturation when the AVF was near large collateral veins.[66]

AVF maturation and calculation of blood flow

Role of DUS in assessment of AVF maturation

Maturation is the process of an AVF developing physical qualities that allow it to be used for venipuncture with large-gage needles. The most common reason an AVF can't be used for dialysis is because it hasn't matured.[61] Arteriovenous anastomoses with native vessels have been linked to a high rate of early occlusion and failure to mature (FTM) in the postoperative period, despite the obvious long-term benefits in terms of morbidity and mortality that make them the vascular access of choice for hemodialysis patients. In different case groups, the incidence of FTM for AVFs at Radio-cephalic site ranged from 30 to 60%.[4,67]

But how can you tell if an AVF is mature? In most situations, a physical inspection by a qualified dialysis nurse is sufficient to determine whether the fistula has grown and is thus suitable for puncture. When the fistula does not appear to be fully grown based on visual inspection alone, such as in obese patients or AVFs that take a long time for maturation, the problem arises. An ultrasound examination and assessment of hemodynamic parameters

(AVF blood flow, RI) can assist determine whether the AVF is suitable for cannulation or has failed to mature, resulting in thrombosis or low flow volume.[4]

Even well-developed veins in obese patients can be difficult to see or palpate due to their depth; in these cases, DUS can show whether the fistula is mature, and US mapping of the out-flow veins can make the first cannulation and future punctures easier.[4]

In this context, it's worth remembering Rayner et als. propositions,[68]which was called "the Rule of 6" in the K-DOQI Guidelines.[61] These guidelines shows ultrasound characteristics that indicate a fistula is mature and suitable for use: a flow volume of >600 ml/min, an out-flow vein diameter of 6 mm, and an out-flow vein depth of 6 mm beneath the skin surface.

It's crucial to determine whether the fistula is developing—albeit slowly—or whether AVFs are maturing (i.e., FTM). A DUS examination and periodic computation of the vascular access flow volume at the brachial artery level can be employed to make the diagnosis in these cases. The Doppler spectrum of the brachial artery is usually triphasic (high resistance), with flow rates ranging from 80 to 150 ml/min.[69]

A *locus minoris resistentiae* is formed as soon as the arteriovenous anastomosis is finished, and the velocity/time curve of the brachial artery becomes biphasic (low resistance). Blood flow increases substantially in the first 24 hours following surgery and then progressively thereafter, until the vascular access matures after various periods of time.[69]

Lomonte and colleagues studied 17 Radio-cephalic AVFs, evaluating brachial artery flow volumes before surgery and at 1, 7, 28, and 258 days after the anastomosis was completed. They confirmed that the most significant increase in AVF flow occurred on postoperative day 1, accounting for over half of the flow volume observed on postoperative day.[70] Following that, the rises were more gradual. According to the authors, serial assessments of AVF flow volumes during the first month after surgery can help distinguish fistulas that will mature

appropriately from those that will fail. Blood flow via the fistula should be 250–500 ml/min on postoperative day 1 and 500–900 ml/min one month after the anastomosis is formed, according to the data they acquired.[70] If lower flow rates or, even worse, the brachial artery flow volume diminishes over time, appropriate maturation is dubious, and the fistula will most likely become unsuitable for dialysis due to thrombosis or low flow. As a result, physical examinations and DUS flow volume measurements should be included in all AVF maturation assessments.

The latter enables for the prediction of the vascular access's likely outcome, and if FTM is detected, it can aid in identifying and, in certain situations, rectifying the reason.

Finally, in response to our initial question about the role of DUS in assessing AVF maturation, the data presented above clearly demonstrate that maturation should be sonographically monitored until the fistula is used, particularly when maturation appears to be slow and in patients whose veins are difficult to assess with physical examination alone (e.g., due to obesity). The only imaging technology that can be utilized to monitor the fistula even throughout its maturity is DUS monitoring of AVF flow volumes. Even so, DUS should always be performed before using an AVF for the first time. This test establishes a baseline for vascular access, which can be used in following examinations to assess functional issues.[4]

Calculation of AVF flow volume

When done correctly, calculating the AVF flow volume with DUS is a simple process that takes only a few minutes and is generally reproducible.

Area x Mean velocity x 60 is the formula for computing flow volumes, where area is the cross-sectional area of the vessel in square centimetres. (Because the vessel is cylindrical, its section is a circle with an area equal to the radius squared multiplied by 3.14) (FIG.3). Mean

velocity (in cm/s) is the velocity of red blood cells measured from the Doppler trace recorded at the area measurement location, and 60 is the number of seconds in a minute (because flow volumes are measured in millilitres per minute). To compute flow volume using this technique, a single longitudinal scan of the vessel can be utilised to obtain the vessel diameter and mean flow velocity. The vessel diameter is initially measured on the correctly enlarged B-mode image. After that, the pulsed Doppler module is activated, the PRF is adjusted to eliminate artefacts, and the mean flow velocity is calculated from the time/velocity curve (most scanners provide a time-averaged velocity option) (Fig. 3).When both variables are measured on the same scan, it verifies that they were taken at the same location in the vessel.[50] In fact, once the 2 measurements are taken, no additional computations are required: current ultrasound scanners are equipped with processing algorithms that calculate the AVF flow volume automatically (Fig.).[69]

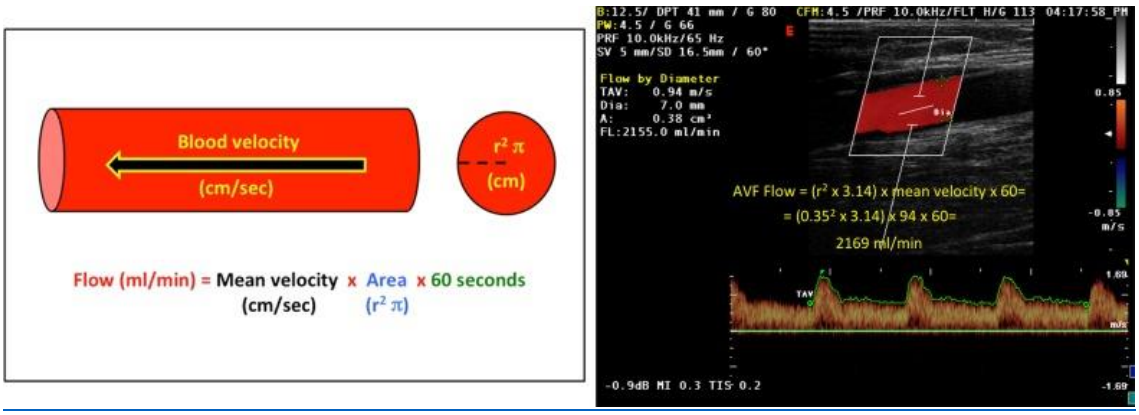


Figure 5: AVF flow volume is calculated. *Left*: theoretical foundation of the formula used to for calculate blood flow volume of a blood vessel. *Right*: example of AVF flow volume calculated manually, with the proposed formula and with scanner software (Flow by diameter) at the level of the brachial artery[57]

The AVF's output vein should be a suitable location for calculating the vascular access flow volume because the arterialized vein is pierced during hemodialysis. However, because the probe can readily compress this vein, readings taken at this level are unreliable. Furthermore, the vessel's diameter varies widely due to its tortuous course and the presence of collateral circuits, making precise measurements of the vessel's cross-sectional area impossible. Furthermore, estimating the mean velocity is difficult due to the turbulent flow that characterizes the venous side of the AVF. Monitoring flow volume at the inflow artery level improves accuracy and reproducibility because of these considerations. However, calculating the flow volume of a distal AVF at the radial artery level might lead to errors since a portion of the fistula flow (approximately 25–30%) may originate from the ulnar artery via the palmar arch.[50,55] It's easy to sample, and it doesn't collapse when subjected to normal transducer pressures. In addition, just above the elbow crease, there is an oblique part of the brachial artery where the sample volume can be simply positioned at an optimal insonation angle. Finally, because of its laminar flow, it is possible to record adequate tracings for precise mean velocity calculation. The brachial artery flow downstream from the AV anastomosis should be subtracted from the value obtained to improve the accuracy of the flow volume measurement in patients with proximal or prosthetic fistulas. Alternatively, the flow volume in prosthetic grafts can be measured directly in the prosthetic conduit, which has a more uniform diameter and is less susceptible to transducer pressure than a native outflow vein.

To avoid the risk of overestimating or underestimating the AVF flow volume, certain considerations should be addressed. First and foremost, make use of the zoom function: the diameter of the vessel must be measured as precisely as possible, because even minor diameter discrepancies result in considerable changes in flow volume (indeed, the formula

used to calculate flow volume entails squaring the vessel radius, which thus becomes an important determinant of flow). The sample volume should always be in the middle of the vessel, but the amplitude should be modified to sample 50–70% of the vessel lumen. This prevents measurements from being limited to red blood cells flowing through the vessel's centre, which travel faster than those flowing near the vessel's walls. The acquisition of velocity data must be as precise as possible; this can be done by carefully controlling the PRF to eliminate any artefacts.

Surveillance of the AVF

The goal of a nephrologist is to prolong life of a healthy AVF by preventing complications, recognising them early, and treating them rapidly. The international guidelines recommend a specific vascular access monitoring (physical examination of the AVF before each dialysis session) and surveillance protocol (assessment of recirculation, venous/arterial pressures, flow volume calculation, and other parameters that give information on AVF function that should be examined monthly). The most effective method of vascular access surveillance is now universally considered as blood flow monitoring.[61]

Reduced flow volumes or values that decrease with time in both native and prosthetic AVFs are indicators of thrombosis.[69,71] There are several ways to define AVF flow (MR angiography, differential conductivity, DUS, Crit-line monitor, glucose infusion, ultrasound dilution technique, ionic dialysance).[48]

DUS has one drawback over the other technologies in that it cannot be utilized during hemodialysis, but it also has an advantage in that it can be used to know status of decreased AVF flow volumes while also looking into the causes.[72,4] To minimize misrepresentation owing to hemodynamic factors (e.g., hypotension), DUS should not be utilized to quantify

AVF flow volumes during immediately in post-dialysis period; values obtained between dialysis sessions or before a dialysis session are suggested.

A well-functioning AVF will have a flow rate of 700 –1,300 ml/min, according to data from the literature on DUS flow volume assessment.[69,70] Access dysfunction and imminent thrombosis are predicted using flow volumes of less than 500 ml/min[50,73]and less than 300 ml/min,[50,69] respectively. Aside from these absolute values, subsequent research has shown that when monthly measurements reveal a decrease in flow volume of > 25% in a vascular access that had previously been stable with flow volumes of >1,000 ml/min over a relatively short period of time (1–4 months), further investigation is warranted, as these findings are predictive of stenosis and vascular access thrombosis.[29,71]

DUS estimations of AVF flow volume can also be used to assess the efficacy of a treatment intervention employed to resolve an issue. The lack of a 20% increase in flow following such an intervention (e.g., percutaneous transluminal angioplasty to eliminate stenosis) indicates that the treatment has failed and that a new solution is necessary.[61]

DUS can be used in AVF monitoring to study the causes of vascular access malfunction, as previously mentioned. While determining flow volume is simple and quick, systematic inspection of an AVF is a complicated and time-consuming procedure that should only be undertaken by trained professionals. As a result, this test should only be utilized when other monitoring/surveillance methods have revealed anomalies or when frequent dialysis is not practicable due to complications (difficult venipuncture, insufficient blood flow, high venous pressure, prolonged bleeding after removal of fistula needles).

The patient's posture and the bare minimum of technological requirements are used to create preoperative mapping. Because vessels involved in AVF are superficial, high-frequency (7.5–13 MHz) linear probes are required to obtain best anatomic vessel wall details and to

accurately assess the superficial wall of the outflow vein, where puncture needle damage likely occurs (approximately 300 punctures per year with 15–16 gauge needle). The outflow vein's wall lies only a few millimeters beyond the skin's surface. This area will invariably be out of focus if a transducer of lower frequency is used, making wall lesions more difficult to see. The vessels of an AVF are almost always parallel to the skin's surface. As a result, the Doppler examination should always be carried out with a beam-steering transducer, which allows the operator to maintain the proper incidence angle (30°–60°) with respect to the flow direction.[57]

These are the steps included in evaluation:

1. The fistula's arterial inflow side is explored (with AVF flow volume).
2. Study of Anastomotic chamber.
3. Assessment venous component of AVF.

A thorough evaluation of the AVF includes a transverse and longitudinal scan of each of these three regions, as well as an investigation of morphological (B-mode) and hemodynamic parameters (with Colour Doppler and Doppler analysis).

Below are the results of a well operating AVF in B-mode, Color Doppler, and Doppler analysis (for purely educational purposes, as in clinical settings the three studies are generally carried out concomitantly). Because discussions of ultrasound findings relevant to the fundamental concerns of AVFs would make this review needlessly lengthy, the reader is directed to expert literature on the subject.[69]

Morphological assessment (B-mode)

The brachial artery is usually the first artery to be evaluated. When viewed longitudinally, a typical artery looks as a completely anechoic strip separated by two three-layered walls. The walls of the arteries, which move in unison with cardiac systole and are noncompressible under probe pressure, distinguish them from the veins on real-time imaging.

The brachial artery is tracked till antecubital fossa, where it splits into the radial and ulnar arteries, which travel down the lateral and medial forearms, respectively. The radial artery, which is commonly the inflow artery of an AVF, displays a continuous, regular increase in diameter and mild tortuosity, which is particularly noticeable in high-flow AVFs. Another common finding is pulsatility that is significantly higher than that of the same vessel in the contralateral arm (more comonly near the anastomosis).[69]

The arterial or inflow component of fistula is investigated first, followed by surgical anastomosis, which frequently follows a winding path. The turbulence of the flow generates a "thrill" in the anastomotic part of the vessel because of its high pulsatility. It is characterised by fine, rapid, palpable, and sonographically documented vibrations that influence the tissues around the vessel. The sonographic image of the anastomotic region can aid in determining the kind of AVF: end-to-end (E-E), side-to-end (S-E), or side-to-side (S-S).[69]

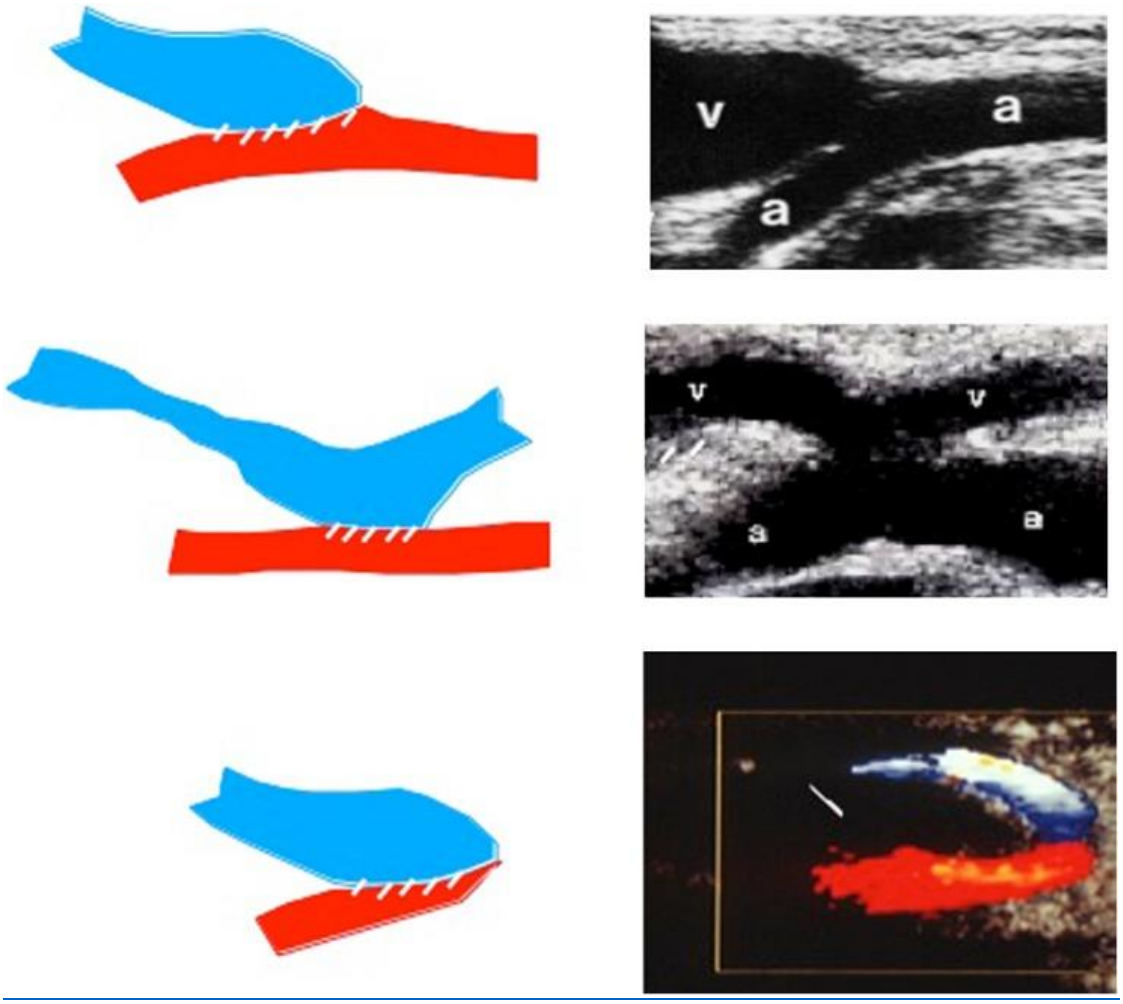


Figure 6: Longitudinal ultrasound scans of the anastomotic region of various AVF types. *Top* side-to-end AVF (S-E), *Center* side-to-side AVF (S-S), *Bottom* end-to-end AVF (E-E) [57]

If the non-linearity of the anastomotic chamber prevents such scans, the presence or absence of the arterial and distal venous segments in reference to the anastomosis site can be used to determine the kind of anastomosis.

Tortuosity, ectasia, and variations in segmental calibre characterise the outflow vein, which are induced by repetitive venipuncture wall damage. The vessel walls seem swollen as a

result of intimal hyperplasia, a condition that allows the vessel to withstand multiple venipunctures with large-caliber needles.[69]

Color Doppler examination[69]

The presence of colour implies that the vessels under examination are in good health. In general, the scanner is set up to "map" arterial flow in red (flow that is going towards the transducer) and venous flow in blue (flow moving away from the transducer).

The most critical elements to adjust for a thorough flow metric analysis is the pulse repetition frequency (PRF). Due to higher flow velocities, the PRF utilized to analyze AVFs is usually higher (1,000–6,000 Hz) than those used to examine the upper limbs. It is important to remember that this is a "dynamic" setting that will need to be adjusted several times throughout the examination to avoid aliasing (especially near the anastomosis, where the flow is faster) and non-coding of the venous flow, which can occur, for example, during exploration of the outflow vein following a high PRF assessment of the AVF. Lower PRFs (1,000 Hz) can be used when a vessel appears to be patent based on morphological observations and reactions to compression operations, but intraluminal flow indications are missing. This can happen in big venous aneurysms, for example, when the lumen's vast size forces it to rupture.[69]

The AVF's input artery displays somewhat homogeneous, laminar flow on Color Doppler imaging (with maximum velocity at the centre of the lumen and the lowest values near the vessel walls). There is an increase in flow velocity (reflected by lighter colours, even white) and turbulence at the anastomosis (reflected by a disorderly alternation of reds and blues within the same luminal segment). Areas of vortex flow are usually recognized at this level,

especially at the anastomosis, because to the large flow volumes and calibre abnormalities that define the outflow vein. They are reflected by intraluminal colour signals that alternate in a spiroidal pattern. As you go away from the AVF, the channel diameter lowers, the vortexing decreases, and the venous flow becomes more uniform and regular.

Doppler ultrasound assessment[69]

On Doppler ultrasound imaging, a healthy AVF displays a significantly lower peripheral resistance than the contralateral limb, as well as plenty of anterograde flow throughout the diastolic phase. As the transducer approaches the anastomosis, the flow through the afferent artery tends to increase in velocity in both the systolic and diastolic phases, and spectral broadening is observed, which is maximal near the anastomosis and returns to baseline with the disappearance of the acoustic window. At the anastomosis level, pure turbulent flow profiles are identified, with loss of arterial phasicity, a broad spectrum extending above and below the baseline, and high peak systolic velocities that vary greatly between moments. On the venous side, near the anastomosis, flow is "arterialized," with apparent systolic–diastolic phasicity and a wide range. As the distance from the AVF increases, the arterial phasicity is gradually reduced, and the mean flow velocity increases.

4. MOST RELEVANT STUDIES:

A retrospective study by **MR, Behera et. al.,**[74] **(2021)** involved 43 subjects with problematic cannulation and underwent doppler examination. The study population were grouped into 2: group 1 consisted of initial difficult cannulation and group 2 secondary difficult cannulation. Group 1 had 60% of subjects where 23.3% had stenosis, 20.9% had immature fistula, 18.6% has outflow stenosis, 11.6% had inflow stenosis, 11.6% had anatomical abnormalities, 9.3%

had outflow + CZ stenosis and 4.7% had inflow + CZ stenosis. In group 1 immature fistula was most frequent and CZ stenosis was common in group 2. This study found lesser diagnostic value of Doppler Ultrasound comparative to fistulogram.

A study by **Abd-Elmageed et. al.,**[75] **(2020)**, performed a color Doppler ultrasonography (CDU) in 100 subjects to test the blood flow for AV fistulas. They found 82 mature AVF and 18 failing AVFs. Flow of blood was considerably less in the failure group than in the mature group in the early postoperative phase (P = 0.001). With an AUC of 0.952, the cutoff value was 200.5 ml/min. For predicting fistula failure, CDU has a sensitivity of 99 percent, a negative predictive value of 94 percent, and a precision of 97 percent, and also a specificity of 89 % and a positive predictive value of 98 percent. During the postoperative phase (days 7–14), the flow of blood in AV fistula, failure and the probability of failure, and blood flow less than 200 ml/min were all assessed.

A study by **Mohammed H. et al.,**[76] **(2019)** involved 60 subjects with age ranging of 18-50 years having complicated AVF. The most common complication was thrombosis in 13.3% followed by stenosis in 3.3%, aneurysmal formation in 6.6%, hematoma in 6.6%, infection in 3.3%, venous hypertension in 3.3%, thrombosis and hematoma in 16.6%, aneurysmal formation and infection in 13.3% and stenosis of 10%.[108]

A retrospective study by **Cho, S et al.,**[77] **(2019)** involved 127 patients. According to logistic regression analysis, the need for PTA was noted with brachial artery flow volume (FV) of 612.9 mL/min or a brachial artery resistance index (RI) of > 0.63. If brachial artery FV was less than 612.9 mL/min or the RI was greater than 0.63, there was severe stenosis. Despite

greater access intervention rates, by adding USG to establish the necessity for angiography after identifying a PE anomaly results in decreased rates of access thrombosis, catheter placement, and access loss when compared to clinical surveillance.

In a retrospective study by **Timmy et al,**[78] **(2018)** examined 2 issued ultrasonography maturation criteria used to foresee unassisted AVF usage for hemodialysis. At 6 and 12 weeks after surgery, 105 patients had standardised postoperative ultrasounds to assess AVF diameter and blood flow. The following ultrasound criteria were assessed: I National Kidney Foundation (NKF) Kidney Disease Outcome Quality Initiative criteria: AVF outflow vein lumen diameter of 6 mm and blood flow of 600 mL/min; and (ii) University of Alabama at Birmingham (UAB) criteria: AVF outflow vein lumen diameter of 4 mm and blood flow of 500 mL/min. Performance characteristics were obtained for both criterion. When compared to the NKF criteria, the UAB criteria had a higher sensitivity (89 vs. 68%) but a lower specificity (42 vs. 70%) for unassisted AVF use. For radiocephalic AVFs, the UAB criteria showed higher sensitivity (86 % vs. 46 %) but lower specificity (58 % vs. 83 %).

The impact of a new multidisciplinary vascular access (VA) clinic with routine DU was evaluated by **Sauco, I Aragoncillo, et al., (2018).**[79] There were 345 and 364 patients in this study, respectively. The number of surgical treatments was identical in both groups of patients (p =.289), with a trend toward more preventative surgical correction of AVF (17 vs. 29, p =.098). The number of new AVFs was 155 vs. 169, with a lesser primary failure rate (26.4 vs. 15.3%, p =.015) and a non-significant increase in radiocephalic AVF, 25.8 vs. 33.2 percent (n = 40 vs. 56), p =.159. The degree of agreement between the clinic's indication and the procedure performed also improved (81.3 vs. 93.5 percent, p =.001). However, effective

AVF treatment, as well as lower radiological test costs and routine DU, can enhance VA results, resulting in a lower initial failure rate and a greater chance of radiocephalic AVF.

F. Nalesso et al,[80] **(2018)** did a study on Color Doppler technique, which is a useful tool for detecting vascular access (VA) concerns in hemodialysis patients. It is also highly inexpensive, quick, non-invasive, and reproducible. Currently, there is no conventional or widely accepted Echo Color Doppler (ECD) approach. It consists of an ECD research strategy. The methodology includes calculating brachial artery flow, describing arterio-venous and/or graft-vascular anastomosis, and describing the efferent vessel and/or graft. The method allows for the preparation of a medical report that takes into account both anatomic and hemodynamic characteristics at the VA. Complications were decreased, chronic complications were avoided, and acute issues were detected early.

Abdelaziz O et al,[81]**(2018)** investigated 25 patients with clinically suspected AVF problems were assessed utilising CDUS of upper limbs over the course of a 9 month study. There were 11 (44%) males and 14 (56%) females, with a range of ages. Shunt problems plagued all 25 individuals. Venous thrombosis (n =12, 48 %) was most common occurrence, followed by stenosis (n =11, 44 %), aneurysm and pseudoaneurysm development (n = 5, 20%), and infection (n =1, 4%). Four patients experienced several complications. Sixteen (64%) of the patients had difficult fistulas that required surgical intervention [reconstruction of new fistula (n = 3), ligation (n =1), graft (n =1), or superficialization (n =2)]. As a result, CDUS is a noninvasive diagnostic method.[81]

Castro, A et al.,[82] (2018), included 221 people with arteriovenous fistulas (AVFs), and found 58.8% having SS, 18.6% having BS, and 22.6 % having no defective access (ND). SS had a much higher rate of thrombotic events than BS and ND (13.1 vs. 4.4 percent, p = 0.018). The annual thrombosis rate was 0.007, 0.037, and 0.004 in the ND, SS, and BS, respectively. At 5 years, AVF cumulative survival was significantly less in SS (89.5%) compared to BS (100%) and ND (97.4%) (p = 0.03).

Kahraman, N. et al.,[83] (2018) included 103 individuals and were categorized into two groups: control, DUS (-), and study, DUS (+). The variations between 20 patients in the DUS (+) group (50 % male) and 20 patients in the DUS (-) group (45 % male) were noted. For a period of 12 months, the primary patency rates in the DUS (+) and DUS (-) groups were 95 % and 65 %, respectively. As per the study's results AVF was formed between the most convenient vessels which reduced procedure failure.

The observational cohort study conducted by **Robbin, ML et. al.,[84] (2017)** assessed ultrasonography characteristics correlated with AVF clinical maturation in newly produced AVF. AVF blood flow, depth, diameter, upper arm artery diameter, presence of stenosis, presence of accessory veins, seven case-mix characteristics (age, sex, black race, AVF location, diabetes, dialysis status, and body mass index), and clinical centre were all examined in the study of 602 patients. AVF blood flow, diameter, and depth each predicted both unaided and total clinical maturation in a statistically significant manner at each ultrasound measurement period. The cross validated area under the receiver operating characteristic curve for models constructed using these three ultrasound parameters for unassisted AVF clinical maturation was 0.69, 0.74, and 0.79 at day 1, 2 weeks, and 6 weeks,

respectively, for unassisted AVF clinical maturation, and 0.69, 0.71, and 0.76 for overall AVF maturation. The other criteria, such as AVF parameters and clinical maturation, did not improve maturation prediction.

Mudoni, Anna, et al.,[85] **(2016**) employed ultrasonography to design a study technique for AVF in hemodialysis patients. They came to the conclusion that Doppler ultrasound plays a significant role in interdisciplinary collaboration in AVF monitoring and suggested that it be included in management framework of AVF.

In a study conducted by **Zhu, Y et al., (2016),**[86] included 132 patients. After a 6-month follow-up, 113 fistulas were classified as mature, while 19 fistulas were classified as failure. Within 2 weeks after surgery, the brachial and radial artery diameters gradually grew in both groups (p0.05), and the diameter and blood flow of the cephalic vein gradually increased within 4 weeks after surgery (p0.05). In failure group, blood flow was significantly lower than that of the mature fistulas from the first day after formation. The area under the receiver operating characteristic (ROC) curve for predicting fistula maturity was 0.95 (529 ml/min was the ideal cut-off value), cephalic vein diameter was 0.83 (5.2 mm), and the two factors together yielded an area under the ROC curve of 0.96.

In a prospective observational study, **Tamara K. Jemcov et al,**[87]**(2013)** studied and identified cut-off values of morphologic and functional vascular characteristics impacting effective RCAVF development using ultrasound. A total of 122 patients (66 men) underwent primary RCAVF construction in this study. Before AVF insertion, ultrasonography examination was used to evaluate the internal diameters of the cephalic vein (CVd) and radial

artery (ARd), as well as venous distensibility (VD), resistance index (RI), and endothelial function by flow mediated dilatation (FMD). In 53 % of patients, AVF maturation was successful, and in 36% of patients, maturation was prolonged. The limits of factors important for RCAVF performance were determined using ROC analysis (CVd >1.8 mm, ARd >1.6 mm, VD >0.4 mm). Female sex was linked to a longer maturation time (OR 0.35, 95 percent CI=0.17-0.72; P=0.005), and a smaller ARd (1.83 vs. 2.01 mm, P=0.01) but a better FMD (2981.5 vs 2689.5, P=0.02) compared to males. [87]

A **Matsui, Satoshi, et al**.,[88] (2012) conducted a single-center observational design study to evaluate the efficacy of CDUS for vascular access evaluation. 57 VAIVTs (vascular access intervention treatment) were performed during the first period, with 37 instances (65 percent) being emergent. During the second, 42 VAIVTs were performed, with 11 cases (25%) being emergent. Period 2 had a lower frequency of emergent intervention therapy than period 1 (P0.001). As a result, the duration of X-ray exposure time per patient was reduced in individuals who got VAIVT during both periods (P0.03). The use of CDUS to evaluate vascular access reduced the number of emergent VAIVTs and the amount of time spent in the X-ray machine.

Ilhan, G. et al.,[89](2012) studied 63 patients who underwent PE and vascular mapping (VM) using Color Doppler ultrasonography (CDUS), as well as 76 patients who were examined solely by physical examination to determine when vascular access surgery should be performed. With pre-operative ultrasonographic vascular mapping, the rate of effectively created AVF increased from 75% to 97 % (P=.001). The approach planned with physical examination was modified in 22 cases (34.9%) based on CDUS evaluation. Based on the

CDUS results, the surgical site for AVF formation and the kind of surgical technique were changed in 12 patients. The rate of permanent access installation was substantially higher in CDUS patients (P=.001). All patients who had vascular mapping had successful VA construction, but the PE group had a negative surgical exploration rate of 18.4 %. The patency rate for fistulas was 80.7 percent for the physical examination (PE) group and 93.4 % for the vascular mapping (VM) group at six months.

LACUNAE IN LITERATURE:

With a decreased probability of primary failure of freshly produced AVF, more alternatives for performing native distal AVF, and higher efficacy in the management of the prevailing dysfunctional AVF, a multidisciplinary approach in the consultation of VA with routine DU has significant benefits for the patient. Furthermore, it provides for the reduction of health costs in fistulogram and phlebography by both minimizing and optimizing complementary examinations. Although the evidence is positive and aligns with the bulk of published studies, additional research is needed to understand the true impact that multidisciplinary teams can have on the construction and function of AVF, patient quality of life, and health-care costs.

MATERIAL & METHODS

MATERIALS & METHODS

Study site: This study was conducted in Department of Radio-Diagnosis at Sri Devaraj URS Academy of Higher Education and Research, Tamaka, Kolar-563103.

Study population: All the eligible 33 patients referred to Department of Radio-Diagnosis at R.L. Jalappa Hospital and Research Center attached to Sri Devraj Urs Medical College, Tamaka, Kolar were considered as study population.

Study design: The current study was an observational study

Sample size: Sample size estimated by using the maturation rate of AV fistulas detected by Doppler ultrasonography from the study by Muray Cases S et al,[90] by using the formula

$$\text{Sample size} = \frac{Z_{1-\alpha/2}{}^2 p(1-p)}{d^2}$$

Here

Z1-α/2 = 1.96 at 5 % error alpha. As in majority of studies *p* values are considered significant below 0.05 hence 1.96 is used in formula.

p = Expected proportion in population based on previous studies or pilot studies.

d = Absolute error or precision – Has to be decided by researcher.

p = 0.75

1 – p = 0.25

d = 15%

Using the above values at 95% Confidence level a sample size of minimum 33 subjects will be involved in the study and evaluated.

Sampling method: All the eligible subjects were recruited into the study consecutively by convenient sampling till the sample size is reached.

Study duration: The data collection for the study was done by the period of 18 months.

Inclusion Criteria:

1) All patients with chronic renal failure planned for arterio-venous access placement.

2) Patients with previous AV fistula failure scheduled for reconstruction at a different site.

Exclusion criteria:

1) Patients with upper limb arterial & venous occlusive disorders.

2) Patients with deformed or scarred upper limbs.

Ethical considerations: Study was approved by institutional human ethics committee. Informed written consent was obtained from all the study participants and only those participants willing to sign the informed consent were included in the study. The risks and benefits involved in the study and voluntary nature of participation were explained to the participants before obtaining consent. Confidentiality of the study participants was maintained.

Data collection tools: All the relevant parameters were documented in a structured study proforma.

Methodology:

Hospital based observational study of patients was done. Preferred site of access placement was planned by Duplex Doppler Sonography by assessment of vessel caliber, wall morphology, peak systolic value of arteries; patency of vessels based on criteria; specifications declared for access placement in fistula formation by the American Institute of Ultrasound in Medicine (AIUM), American College of radiology (ACR); Society of Radiologists in Ultrasound (SRU).

Tourniquet was placed around the arm and superficial venous system namely cephalic, basilic and median cubital vein was assessed first in transverse plane followed by longitudinal planes. Venous diameter and distensibility and diameters were recorded at distal and mid-

forearm, cubital fossa, mid-arm followed by assessment of axillary and subclavian veins. Distal non-dominant limb is preferred first, followed by proximal non-dominant limb and dominant limb.

Subsequently arterial diameter, flow velocities of radial and brachial arteries were assessed. Radial artery was assessed at the wrist up to mid forearm and brachial artery from its bifurcation superiorly up to mid-arm region. Post-operative follow up was performed between 4^{th} to 6^{th} week for all the patients.

STATISTICAL METHODS

Venous component, Arterial component and AVF were considered as primary outcome variables. Type of anastomoses was considered as primary explanatory variable. Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency and proportion for categorical variables. Non normally distributed quantitative variables were summarized by median and interquartile range (IQR). Data was also represented using appropriate diagrams like bar diagram, pie diagram. All Quantitative variables were checked for normal distribution within each category of explanatory variable by using visual inspection of histograms and normality Q-Q plots. Shapiro- wilk test was also conducted to assess normal distribution. Shapiro wilk test p value of >0.05 was considered as normal distribution. For non-normally distributed Quantitative parameters, Medians and Interquartile range (IQR) were compared between study groups using Kruskal Wallis test (> 2 groups).

Categorical outcomes were compared between study groups using Chi square test /Fisher's Exact test (If the overall sample size was < 20 or if the expected number in any one of the cells is < 5, Fisher's exact test was used.). P value < 0.05 was considered statistically significant. Data was analyzed by using SPSS software, V.22.

RESULTS

RESULTS:

A total of 33 subjects were included in the final analysis.

Table 1: Descriptive analysis of age group in the study population (N=33)

Age Group	Frequency	Percentages
40 to 49	9	27.27%
50 to 59	14	42.42%
60 to 69	10	30.30%

Among the study population, 9 (27.27%) participants were aged between 40 to 49 years, 14 (42.42%) participants were aged between 50 to 59 years and 10 (30.30%) participants were aged between 60 to 69 years. (Table 1 & Figure 1)

Figure 7: Bar chart of age group in the study population (N=33)

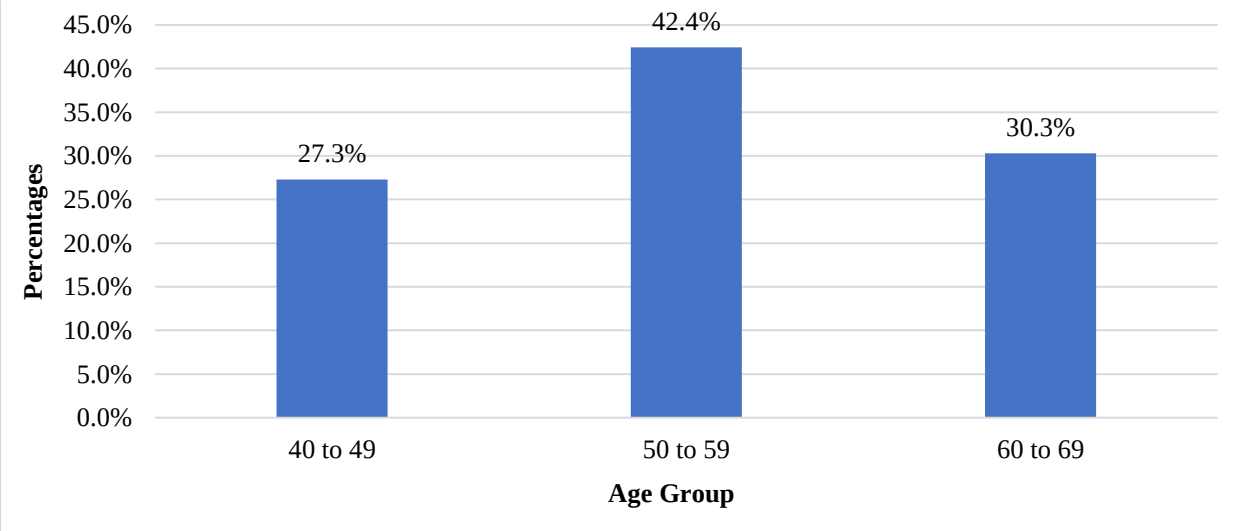


Table 2: Descriptive analysis of gender in the study population (N=33)

Gender	Frequency	Percentages
Male	24	72.7%
Female	9	27.3%

Among the study population, 24 (72.73%) participants were male and 9 (27.27%) participants were female. (Table 2 & Figure 2)

Figure 8: Pie chart of gender in the study population (N=33)

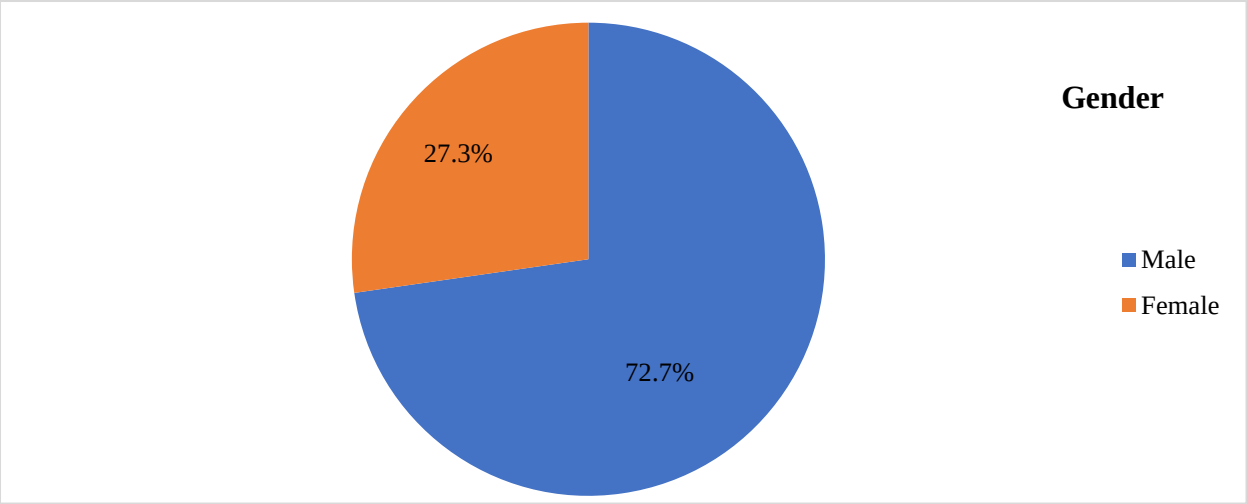


Table 3: Add the comorbidities present in the study population (N=33)

Clinical history	Frequency	Percentages
Diabetes mellitus	16	47%
hypertension	8	24%
Diabetes mellitus+ hypertension	10	29%

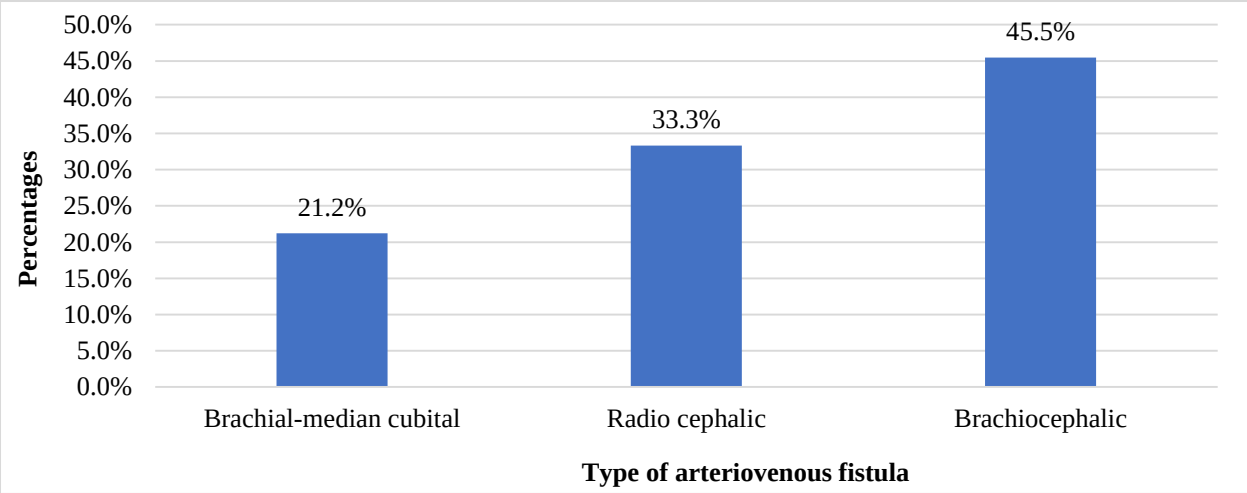
The clinical history of the study population included diabetes mellitus in 47%, hypertension in 24%, diabetes and hypertension combined in 29%.

Table 4: Descriptive analysis of type of arteriovenous fistula in the study population (N=33)

Type of arteriovenous fistula	Frequency	Percentages
Brachial-median cubital	7	21.21%
Radio-cephalic	11	33.33%
Brachio-cephalic	15	45.45%

Out of 33 patients studied, 7 (21.21%) underwent brachial-median cubital fistula, 11 (33.33%) underwent Radio-cephalic fistula and 15 (45.45%) underwent Brachio-cephalic fistula. (Table 4 & Figure 3)

Figure 9: Bar chart of type of arteriovenous fistula in the study population (N=33)



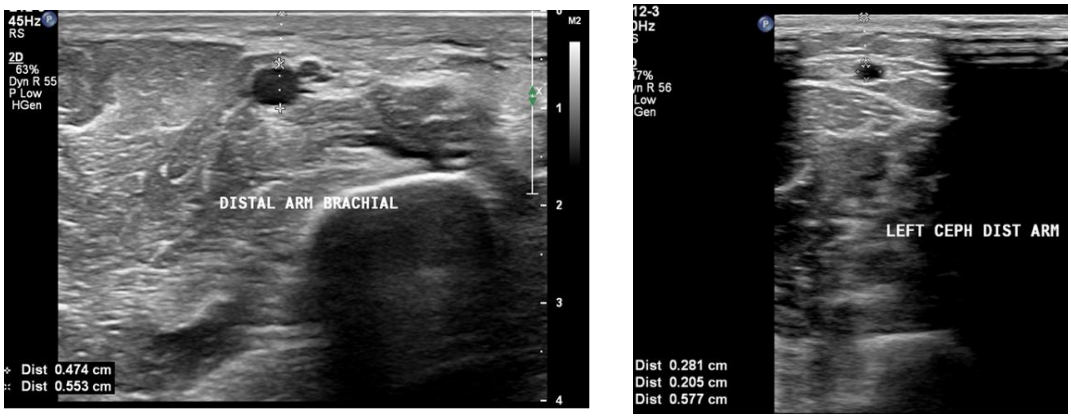


Figure 10: Ultrasound Grey Scale Images Showing Preoperative Arterial And Venous Mapping

Table 5: Summary of ultrasound assessed parameters at venous component, site of arteriovenous fistula & arterial component

Parameter	Median (IQR)
Diameter - Venous Component (N=33)	4.9 (4.55,5.6)
PSV - Venous Component (N=30)	111 (91,145)
FV - Venous Component (N=30)	466 (364,1024)
Diameter - Arterial Component (N=33)	5.4 (4.8,5.75)
PSV - Arterial Component (N=33)	286 (246.5,436)
Width at AVF (N=33)	6.1 (5.5,6.5)
Depth of AVF (N=33)	6.7 (6.05,7.45)
PSV At AVF (N=33)	375 (320,589)

In the venous component, the median diameter was 4.9 mm (IQR 4.55,5.6), median PSV was 111 cm/sec (IQR 91,145) and the median FV was 466 ml/min (IQR 364,1024). In the arterial component, the median diameter was 5.4 mm (IQR 4.8,5.75) and median PSV was 286 cm/sec (IQR 246.5,436). The median width at AVF was 6.1 mm, depth of AVF was 6.7 mm and PSV at AVF was 375 cm/sec in the study population. (Table 5)

Table 6: Comparison of mean increase in the diameter of the cephalic and median cubital vein among the types of AVF at pre AVF and post AVF

Types of anastomoses	Pre AVF	Post AVF
Brachiocephalic	2.9 mm	4.8mm
Radio cephalic	2.1mm	5.4 mm
Branchia-median cubital	2.2 mm	5.3 mm

Mean venous diameters of cephalic vein with respect to the three types of AVF constructed were as follows: Mean cephalic venous diameters increased from 2.9 to 5.1 mm after 4 weeks in brachiocephalic type of anastomoses. Mean cephalic venous diameter increased from 2.1 mm to 4.1 mm after 4 weeks in radio-cephalic fistula. Mean median cubital vein diameter increased from 2.2 to 4.6 mm after 4 weeks in brachial-median cubital fistula.(table 6)

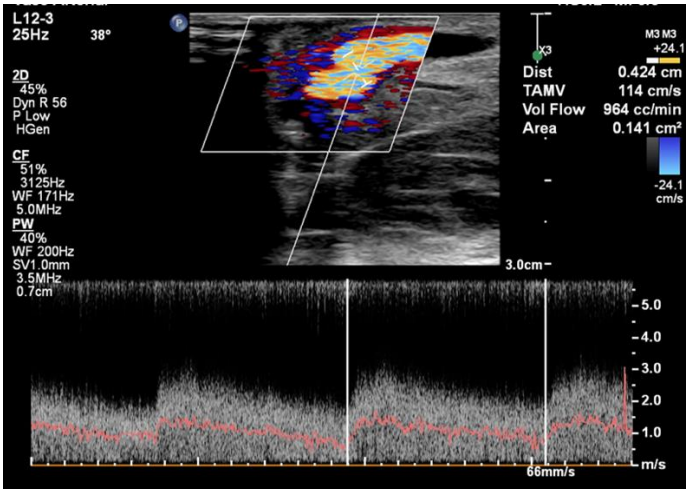


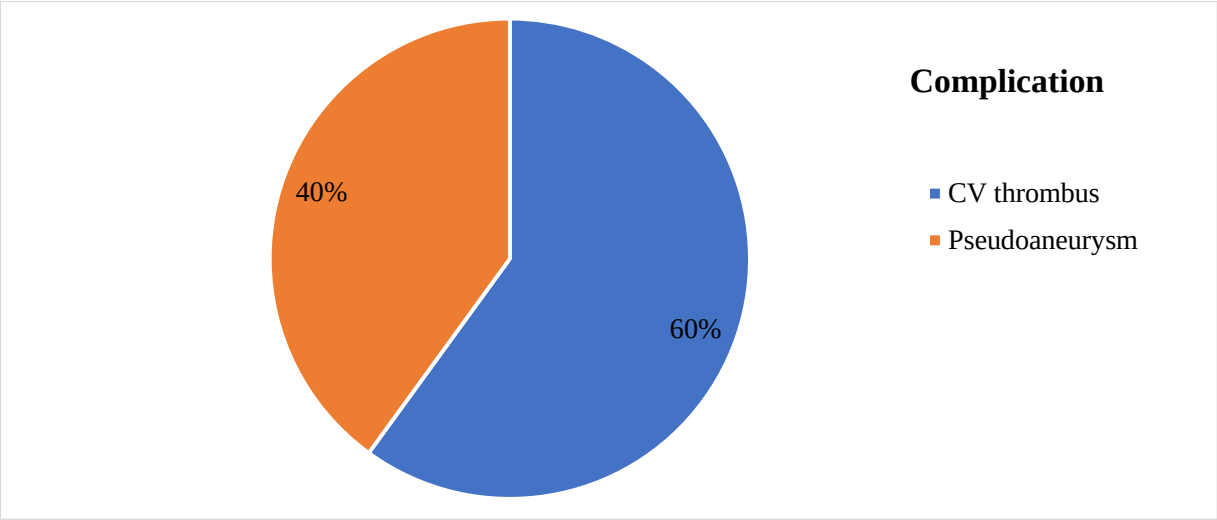
Figure 11: Colour Doppler ultrasound image depicting the flow volume (ml/min) of the venous component

Table 7: Descriptive analysis of complications in the arteriovenous fistula (N=5)

Complication	Frequency	Percentages
CV thrombus	3	60%
Pseudoaneurysm	2	40%

Out of 33 participants, only 5 people had complications, 60% of participants had CV thrombus and 40% of participants had pseudoaneurysm. (Table 7)

Figure 12 : Pie chart of complications in the arteriovenous fistula (N=5)



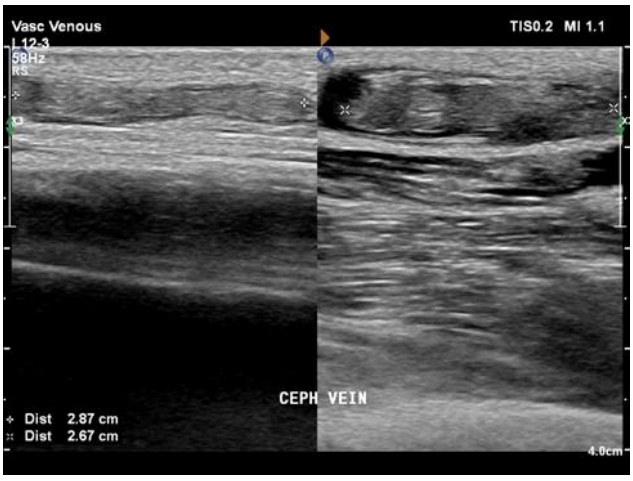


Figure13: Ultrasound grey scale image showing cephalic vein thrombosis

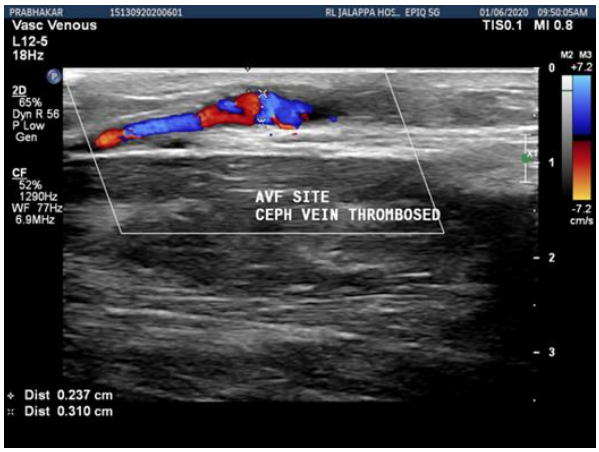


Figure 14: Colour Doppler ultrasound image showing venous thrombosis close to the site of AVF

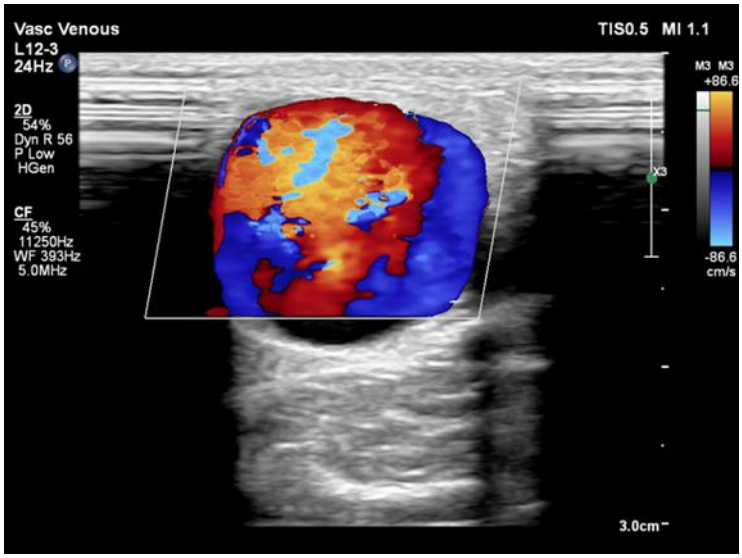


Figure 15: Colour Doppler ultrasound showing pseudoaneurysm formation with a classic "YIN YANG" pattern.

Table 8: Descriptive analysis of status of AVF in the study population (N=33)

Status of AVF	Frequency	Percentages
Failure to mature	23	69.69%
Successful	10	30.30%

Out of 33 participants, 69.69% of fistulas failed to mature and 30.30% matured successfully. (Table 8 & figure 10)

Figure 16: Bar chart of status of AVF in the study population (N=33)

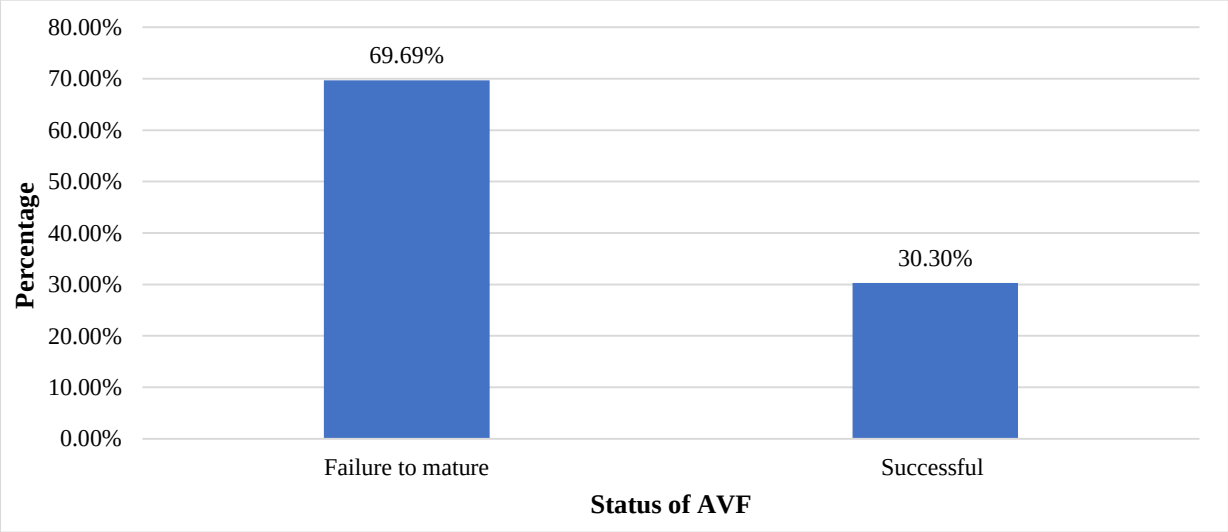


Table 9: Comparison of age group across type of arteriovenous fistula (N=33)

Age Group	Type of arteriovenous fistula			Chi square	P value
	Brachial-Median Cubital (N=7)	Brachiocephalic (N=15)	Radio-cephalic (N=11)		
40 To 49	2 (28.57%)	4 (26.67%)	3 (27.27%)	1.686	0.793
50 To 59	3 (42.86%)	5 (33.33%)	6 (54.55%)		
60 To 69	2 (28.57%)	6 (40%)	2 (18.18%)		

The difference in age groups across the type of arteriovenous fistula is found to be insignificant with a P- value of 0.790.

Figure 17: Cluster bar chart of comparison of age group across type of anastomoses (N=33)

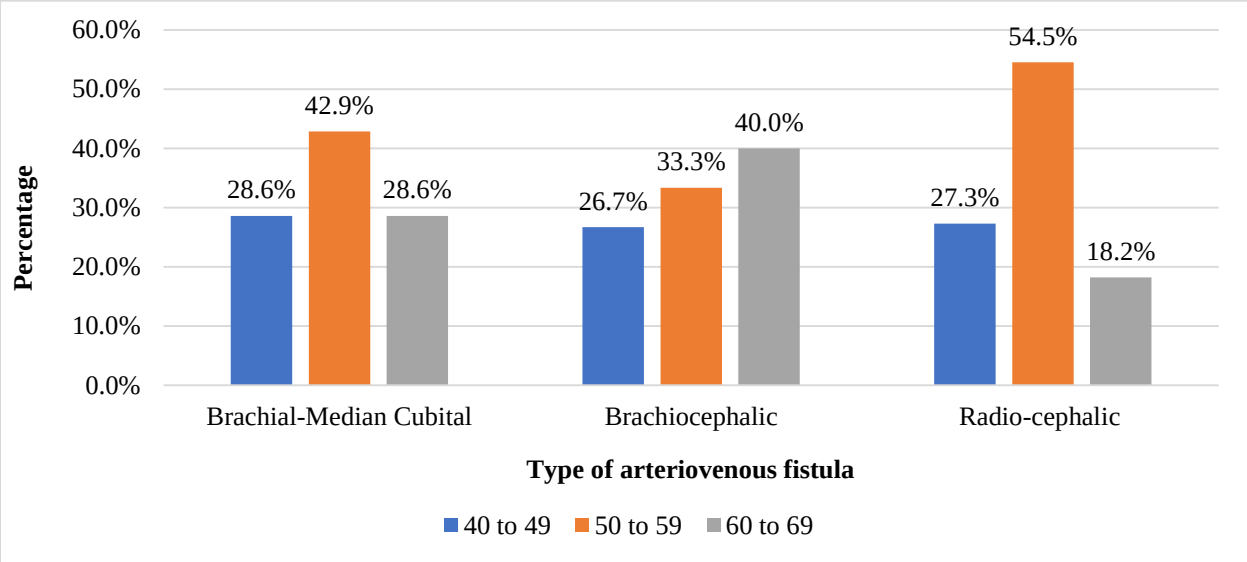


Table 9: Comparison of status of AVF across type of arteriovenous fistula (N=33)

Status of AVF	Type of arteriovenous fistula		
	Brachial-Median Cubital (N=7)	**Brachio-cephalic (N=15)**	**Radio-cephalic (N=11)**
CV Thrombus	1 (14.29%)	1 (6.67%)	1 (9.09%)
Failure to mature	4 (57.14%)	7 (46.67%)	7 (63.64%)
Pseudoaneurysm	0 (0%)	1 (6.67%)	1 (9.09%)
Successful	2 (28.57%)	6 (40%)	2 (18.18%)

No statistical test was applied- due to 0 subjects in the cells

Out of 7 participants with Brachial-median cubital fistula, majority of 57.14% fistulas failed to mature and 28.57% of fistulas matured successfully. Out of 7 participants with Brachio-cephalic fistula, majority of 46.67% fistulas failed to mature and 40% of fistulas matured successfully. Out of 7 participants with Radio-cephalic fistula, majority of 63.64% fistulas failed to mature and 18.18% of fistulas matured successfully. (table 9)

Table 10: Comparison of ultrasound assessed parameters at venous component, site of arteriovenous fistula & arterial component across type of arteriovenous fistula

	Type of arteriovenous fistula			Kruskal Wallis Test (P value)
	Brachial-Median Cubital	Brachiocephalic	Radio cephalic	
Diameter - Venous Component (N=33) (mm)	5.4(4.9,5.5)	5.2 (4.7,5.7)	4.7 (4.3,5.1)	0.054
PSV - Venous Component (N=30) (cm/sec)	108.5 (83,146.75)	114 (94.75,146.75)	114 (89,144.75)	0.895
FV - Venous Component (N=30) (ml/min)	471 (347.25, 1135.75)	441 (362.25,689.25)	475.5 (367,1740.5)	0.587
Diameter - Arterial Component (N=33) (mm)	5.2 (4.4,5.6)	5.6 (5.1,5.8)	5.2 (4.8,5.8)	0.385
PSV - Arterial Component (N=33) (cm/sec)	267 (264,423)	287 (246,424)	286 (241,457)	0.978
Width at AVF (N=33) (mm)	6 (5.4,6.3)	6.1 (5.9,6.6)	5.9 (5.5,6.6)	0.477
Depth of AVF (N=33) (mm)	7.5 (6.4,8.5)	6.8 (5.8,8.1)	6.5 (6,6.9)	0.158
PSV At AVF (N=33) (cm/sec)	347 (314,601)	375 (322,645)	399 (318,584)	0.788

Among the people with brachial-median cubital fistula, the median diameter of the venous component was 5.4 mm (IQR 4.9 to 5.5). It was 5.2 mm (IQR 4.7 to 5.7) and 4.7 mm (IQR 4.3 to 5.1) among people with Brachio-cephalic and Radio-cephalic fistula. The difference in the diameter of venous component across type of arteriovenous fistula was statistically not significant (P Value 0.054). Among the people with brachial-median cubital fistula, the median PSV of venous component was 108.5 cm/sec (IQR 83 to 146.75). It was 114 cm/sec (IQR 94.75 to 146.75) and 114 cm/sec (IQR 89 to 144.75) among people with Brachio-cephalic and Radio- cephalic fistula. The difference in the PSV at venous component across

type of arteriovenous fistula was statistically not significant (P Value 0.895). Among the people with brachial-median cubital fistula, the median FV at venous component was 471 ml/min (IQR 347.25 to 1135.75). It was 441 ml/min (IQR 362.25 to 689.25) and 475.5 ml/min (IQR 367 to 1740.5) among people with Brachio-cephalic and Radio-cephalic fistula. The difference in the FV at venous component across type of arteriovenous fistula was statistically not significant (P Value 0.587). Among the people with brachial-median cubital fistula, the median diameter at arterial component was 5.2 mm (IQR 4.4 to 5.6). It was 5.6 mm (IQR 5.1 to 5.8) and 5.2 mm (IQR 4.8 to 5.8) among people with brachio-cephalic and radio-cephalic fistula. The difference in the diameter at arterial component across type of arteriovenous fistula was statistically not significant (P Value 0.385). Among the people with brachial-median cubital fistula, the median PSV at arterial component was 267 cm/sec (IQR 264 to 423). It was 287 cm/sec (IQR 246 to 424) and 286 cm/sec (IQR 241 to 457) among people with Brachio-cephalic and Radio-cephalic fistula. The difference in the PSV at arterial component across type of arteriovenous fistula was statistically not significant (P Value 0.978). Among the people with brachial-median cubital fistula, the median width at AVF was 6 mm (IQR 5.4 to 6.3). It was 6.1 mm (IQR 5.9 to 6.6) and 5.9 mm (IQR 5.5 to 6.6) among people with Brachio-cephalic and Radio-cephalic fistula. The difference in the width at AVF across type of arteriovenous fistula was statistically not significant (P Value 0.477). Among the people with brachial-median cubital fistula, the median depth at AVF was 7.5 mm (IQR 6.4 to 8.5). It was 6.8 mm (IQR 5.8 to 8.1) and 6.5 mm (IQR 6 to 6.9) among people with Brachio-cephalic and Radio-cephalic fistula. The difference in the width at AVF across type of arteriovenous fistula was statistically not significant (P Value 0.158). Among the people with brachial-median cubital fistula, the median PSV at AVF was 347 cm/sec (IQR 314 to 601). It was 375 cm/sec (IQR 322 to 645) and 399 cm/sec (IQR 318 to 584) among people

with Brachio-cephalic and Radio-cephalic fistula. The difference in the width at AVF across type of arteriovenous fistula was statistically not significant (P Value 0.788).(table10)

DISCUSSION

DISCUSSION

The hemodialysis patient's lifeline is access, yet obtaining and maintaining it is a challenging task. The arteriovenous fistula (AVF) has long been considered the best access method.[91] Preoperative duplex ultrasound evaluation of upper extremity veins and arteries is a useful adjunct to physical examination, particularly for obese patients, those who have had multiple previous access surgeries, or those who are otherwise difficult to examine well, or for those with suspected arterial or venous disease.[47] Following the construction of the access, long-term functional patency may be difficult to achieve due to the occurrence of stenotic lesions that result in thrombosis or failure to mature. It is still unknown what role duplex ultrasonography serves in a surveillance programme.[91]

The present study aimed to perform Doppler ultrasonography for arterio-venous access planning and vessel mapping where adequate diameters of > 2 mm obtained by preoperative ultrasound measurements of the veins and arteries were chosen for AVF construction. Follow up to check for maturation of the AVF after 4th week was done.

This study was an observational study involving 33 patients with chronic renal failure suggested for AV access placement and subjects with previous AV fistula failure scheduled for reconstruction at a different site. Most of the study subjects were aged between 50-59 years (42.42%) followed by 60-69 years were 30.30% and 40-49 years were 27.27%. Male predominance was found in our study (M-73%, F-27%). A prospective study by Abdelaziz, O et al, [1] with 25 subjects found female predominance in their study (M:F-44%, 56%) with mean age of 51.56 ± 15.524 years (range 8-70years). Another study by Abd-Elmageed, M et

al,[75] involved 100 subjects with age ranging from 32-73 years with mean age of 59.8 ± 8.10 years and female predominance (M-47% F-53%).

The clinical history of study subjects included diabetes mellitus in 47%, hypertension in 24%, diabetes and hypertension combined in 29%.

Most common type of AVF was Brachio-cephalic in 45.45% followed by Radio-cephalic in 33.33% and Brachial-median cubital in 21.21%. In a similar study by Abdelaziz, O et al,[1] found brachiocephalic fistula in 40%, Radiocephalic fistula was in 36% and Brachiobasilic fistula in 24%.

The present study's observations on US measures are as follows: the median diameter of the venous component was 4.9 mm (IQR 4.55,5.6), median PSV was 111 cm/sec (IQR 91,145) and the median FV was 466 ml/min (IQR 364,1024). The median diameter of arterial components was 5.4 mm (IQR 4.8,5.75) and median PSV was 286 cm/sec (IQR 246.5,436). The median width at AVF was 6.1 mm, depth of AVF was 6.7 mm and PSV at AVF was 375 cm/sec in the study population. The study by Robbin et al,[92] recommended a blood flow of 350-450 ml/min which aided in maturation if AVF whereas, as less than 350 ml/min resulted in failure and difficulty in dialysis. Further, Zhu et al,[93] suggested to have blood flow greater than 200 ml/min for a minimum of 6 dialysis sittings in Chinese population may be because of their dietary habits and body type. The study by Abd-Elmageed, M et al,[75] followed their study subjects for 6 months and found 18% to have failure of maturation and 82% had successful maturation and found the blood flow to be greater than 500 ml/min in matured AVF subjects (median 559.5 ml/min) whereas in failed subjects showed median 271 ml/min of blood flow. Hence, they observed 88% of increased blood flow in matured AVF and it was

just 38.2% increase in blood flow in the failed maturation of AVF. Similarly, in our study we found 30.30% to have successful maturation of AVF and 69.69% showed failure to mature. The mean blood flow in matured AVF showed greater than 500 ml/min (1472 ml/min) and mean blood flow in immature was less than 500 ml/min (404 ml/min).

A multicenter observational study by Robbin, M et al,[94] observed that there AVF diameter showed a slight increase and the overall median vein depth decreased by approximately 0.1 cm from 1 day to 6 weeks suggesting remodeling of the overlying soft tissues. They found that accounting for vein depth in addition to AVF blood flow and vein diameter significantly improved prediction of subsequent clinical maturation. Similar results were found in our study.

According to the guidelines on hemodialysis vascular access, the arteriovenous fistula is the preferred method of dialysis access, and its malfunction is a leading cause of morbidity and hospitalisation in hemodialysis patients.[95] The need of timely intervention in case of early fistula failure cannot be overstated. Clinically, however, nephrologists may need 3–4 months to confirm an immature fistula. As a result, well-defined criteria employed in the early post-operative period after fistula construction for identifying fistulas that may fail would be extremely beneficial.[96] Blood flow increases rapidly following fistula construction, peaking in 4 to 12 weeks.[97] Approximately 40 to 60 % of the overall blood flow increment occurs within 24 hours of fistula creation. We monitored the flow of blood at the fistula site in the early post-operative period (after 4th week) to see if it could predict fistula complications. In 33 subjects, 5 of them recorded complications where, CV thrombus was seen in 60% and pseudoaneurysm in 40%. In a study by Abdelaziz, O et al,[1] (n=25) found 8% with failure of

maturation and remaining 92% had successful maturation. They observed venous thrombosis to be most common complication (48%) and stenosis in 44%, aneurysmal dilatation in 8%, pseudoaneurysm formation in 12% and infection in 4% and 6 subjects have more than one complication. A study by Abd-Elmageed, M et al,[98] (n=100) found 82% mature and 12% failed AVF maturation. The result of the fistula was found to have a highly significant relationship with age in this investigation. The failed group had a greater mean age than the mature group, 68.3 ± 3.08 vs 58.0 ± 7.67 (P = 0.001). However, in our study the difference in the age groups across the type of arteriovenous fistula is found to be insignificant with a P-value of 0.790.

In the present study, the failure of AVF maturation was more common to Radio-cephalic fistula (63.64%) followed by Brachial-median cubital fistula (57.14%) and Brachio-cephalic fistula (46.67%). The most common location which showed successful maturation was Brachio-cephalic fistula (40%), followed by Brachial-median cubital fistula (28.57%) and Radio-cephalic fistula (18.18%). Diabetes, female sex, patient age, and incident dialysis by CVC have all been linked to early fistula failure.[99,100,101] In a prospective research by Lok et al,[102] age >65 years, peripheral vascular disease, and coronary artery disease were all predictive of maturation failure. However, in our study the reasons for failure could have been due to comorbidities and age.

In our study the people with brachial-median cubital AVF, the median diameter of venous component was 5.4 mm (IQR 4.9 to 5.5). It was 5.2 mm (IQR 4.7 to 5.7) and 4.7 mm (IQR 4.3 to 5.1) among people with Brachio-cephalic and Radio-cephalic fistulas. The difference

in the diameter of venous component across type of arteriovenous fistula was statistically not significant (P Value 0.054).

The present study found the median PSV of venous component at Brachial-median cubital fistula to be 108.5 cm/sec (IQR 83 to 146.75) and Brachio-cephalic and Radio-cephalic fistula was 114 cm/sec (IQR 94.75 to 146.75) and 114 cm/sec (IQR 89 to 144.75) respectively. The difference in the PSV of venous component across type of arteriovenous fistula was statistically not significant (P Value 0.895).

The median flow volume of venous components observed in brachial media cubital fistula was 471 ml/min (IQR 347.25 to 1135.75), for brachiocephalic and radio cephalic was 441 ml/min (IQR 362.25 to 689.25) and 475.5 ml/min (IQR 367 to 1740.5) respectively. The difference in the FV of venous component across type of arteriovenous fistula was statistically not significant (P Value 0.587). We observed that the venous volume in all types of AVF was greater than 500 ml/min in all the types of AVF expecting a good outcome.

Among the people with Brachial-median cubital fistula, the median diameter of arterial component was 5.2 mm (IQR 4.4 to 5.6). It was 5.6 mm (IQR 5.1 to 5.8) and 5.2 mm (IQR 4.8 to 5.8) among people with Brachio-cephalic and Radio-cephalic fistula. The difference in the diameter of arterial component across type of arteriovenous fistula was statistically not significant (P Value 0.385).

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The difference in the PSV at arterial component across type of arteriovenous fistula was statistically not significant (P Value 0.978). Among the people with Brachial-median cubital fistula, the median width at AVF was 6 mm (IQR 5.4 to 6.3). It was 6.1 mm (IQR 5.9 to 6.6) and 5.9 mm (IQR 5.5 to 6.6) Among people with Brachio-cephalic and Radio-cephalic fistula. The difference in the width at AVF across type of arteriovenous fistula was statistically not significant (P Value 0.477). Among the people with brachial-median cubital fistula, the median depth of AVF was 7.5 mm (IQR 6.4 to 8.5). It was 6.8 mm (IQR 5.8 to 8.1) and 6.5 mm (IQR 6 to 6.9) among people with Brachiocephalic and radio cephalic fistula. The difference in the width of AVF across type of arteriovenous fistula was statistically not significant (P Value 0.158). Among the people with brachial-median cubital fistula, the median PSV at AVF was 347 cm/sec (IQR 314 to 601). It was 375 cm/sec (IQR 322 to 645) and 399 cm/sec (IQR 318 to 584) among people with Brachiocephalic and Radio cephalic fistula. The difference in the width at AVF across type of arteriovenous fistula was statistically not significant (P Value 0.788).

The diameters of the cephalic vein, radial artery, and brachial artery, as well as peak systolic velocity of the radial artery and brachial artery, were considerably lower in the failure group than in the mature group, according to Zhu Y et al,[93] and Niyyar VD et al,[103]. In our study the mean diameter of the cephalic vein at pre AVF increased from 2.9 to 5.1 mm following 2.1 mm to 4.1 mm in radio cephalic fistula and 2.2 to 4.6 mm post 4 weeks in brachiocephalic and brachial median cubital anastomoses indicating that the patency of the vessels is maintained in all types of AVF after 4th week of follow up.

CONCLUSION

CONCLUSIONS:

- This study was an observational study involving 33 patients with chronic kidney failure suggested for AV access placement and subjects with previous AV fistula failure scheduled for reconstruction at a different site.
- Majority of our study subjects were aged between 50-59 years
- Male predominance was found in our study.
- The most common location for AV fistula creation was in Brachio-cephalic site in 45.45% and Radio-cephalic site in 33.33% and Brachial-median cubital site in 21.21%.
- Out of 33 participants, 5 people had complications, out of which 60% participants had CV thrombus and 40% participants had pseudoaneurysm.
- Out of 33 participants, 69.69% arteriovenous fistulas did not mature and 30.30% matured successfully.
- The failure of AVF maturation was more common in radio cephalic site (63.64%) followed by brachial-median cubital fistula (57.14%) and Brachiocephalic fistula (46.67%). The most common location which showed successful maturation was Brachiocephalic (40%), followed by Brachial-median cubital (28.57%) and Radio-cephalic fistula (18.18%).
- In the 33 patients that have been included in the study, 9 arteriovenous fistulas with adequate flow volume (> 500 ml/min), velocity, width & depth from skin were associated with good outcome.
- 30.30% of AVFs showed successful maturation and 69.69% of AVFs failed to mature. Mean value of blood flow in matured AVF was greater than 500 ml/min (1472

ml/min) and mean value of blood flow in immature fistulas was less than 500 ml/min (404 ml/min).

Arteriovenous fistulas with an adequate flow volume of > 500 ml/min, velocity, width & depth from skin were found to be related to the maturation of AVF in all types of AVF.

Limitations and recommendations

- While the study's limited sample size may be regarded a drawback, the abundance of exclusion criteria has decreased the risks.
- Follow-up time is limited.
- An intraoperative CDU assessment of the arteriovenous anastomoses would be more useful in predicting maturity, however this was not available at our facility.
- More prospective studies with a broader patient population are needed.

SUMMARY

SUMMARY

For hemodialysis patients, the native AVF is the preferred vascular access since it lasts longer and has less complications than other types of vascular access; these benefits translate to a higher quality of life and a longer lifetime for hemodialysis patients. This study was an observational study involving 33 patients suffering from chronic kidney disease suggested for AV access placement and subjects with previous AV fistula failure scheduled for reconstruction at a different site. Most of the patients aged between 50-59 years (42.42%) with male predominance. The most common location for AV fistula creation was in Brachio-cephalic site in 45.45% and then in Radio-cephalic site in 33.33% and lastly in Brachial-median cubital site in 21.21% patients. Out of 33 participants, 5 people had complications, out of which 60% participants had CV thrombus and 40% participants had pseudoaneurysm. Out of 33 participants, 69.69% arteriovenous fistulas did not mature and 30.30% were reported successfully matured after 4 weeks. The failure of AVF maturation was more common in radio cephalic fistula (63.64%) followed by brachial-median cubital fistula (57.14%) and Brachiocephalic fistula (46.67%). The most common location which showed successful maturation was Brachiocephalic (40%), followed by brachial-median cubital (28.57%) and radio cephalic fistula (18.18%). The blood flow greater than 500 ml/min determined to be successful in maturation of all types of AVF.

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ANNEXURE

ARTERIAL & VENOUS MAPPING OF UPPER LIMB

FINDINGS:

- **BRACHIAL, RADIAL AND ULNAR ARTERIES:**

  Type of flow -

  PSV -.

- **STENOSIS / NARROWING / WALL CALCIFICATIONS** :
- **SUPERFICIAL VENOUS SYSTEM INCLUDING CEPHALIC VEIN , MEDIAN CUBITAL VEIN & BASILIC VEIN** :

  Calibre & compressibilty-

  Thrombus -

	LEFT	RIGHT
	CEPHALIC VEIN	BASILIC VEIN
Distal arm	mm	mm
Cubital fossa	mm	mm
Proximal forearm	mm	mm
	LEFT	
	RADIAL ARTERY	ULNAR ARTERY
Proximal forearm	mm	mm
Mid forearm	mm	mm
Distal forearm	mm	mm
	LEFT	
	BRACHIAL ARTERY	
Mid arm	mm	mm
Cubital fossa	mm	mm

IMPRESSION:

POST AVF EVALUATION

CLINICAL DATA:

FINDINGS & IMPRESSION:

- INFLOW COMPONENT :

  calibre :

  PSV :.

- OUTFLOW COMPONENT :

  calibre :

  PSV :

  Flow volume :

- AT THE SITE OF FISTULA :

  calibre :

  PSV :

- WIDTH OF ANASTOMOTIC SITE :
- DEPTH OF THE FISTULA :

INFORMED CONSENT FORM

STUDY TITLE: ROLE OF DOPPLER EVALUATION IN CREATING ARTERIOVENOUS FISTULA FOR HEMODIALYSIS – AN OBSERVATIONAL STUDY.

Chief researcher/ PG guide's name: Dr. N. RACHEGOWDA.
Principal investigator: Dr. SURAJ H. S.

Name of the Patient:
Age :
Gender :

a. I have been informed in my own language that this study involves Doppler ultrasonography as part of procedure. I have been explained thoroughly and understand the procedure.
b. I understand that the medical information produced by this study will become part of institutional record and will be kept confidential by the said institute.
c. I understand that my participation is voluntary and may refuse to participate or may withdraw my consent and discontinue participation at any time without prejudice to my present or future care at this institution.
d. I understand that I will not be provided with any monetary incentives for participating in this study.
e. I agree not to restrict the use of any data or results that arise from this study, provided such a use is only for scientific purpose(s).
f. I confirm that Dr. SURAJ H. S / Dr. N. RACHEGOWDA. (chief researcher/ name of PG guide) has explained to me the purpose of research and the study procedure that I will undergo and the possible risks and discomforts that I may experience, in my own language. I hereby agree to give valid consent to participate as a subject in this research project.

Participant's signature/thumb impression
Signature of the witness: Date:
1)
2)

I have explained to _____ (subject) the purpose of the research, the possible risk and benefits to the best of my ability.

Chief Researcher/ Guide signature Date:

PATIENT INFORMATION SHEET

Principal Investigator: Dr. SURAJ H. S. / Dr. N. RACHEGOWDA

I, Dr. SURAJ H. S. post-graduate student in Department of Radio-Diagnosis at Sri Devaraj Urs Medical College. I will be conducting a study titled "Role Of Doppler Evaluation In Creating Arteriovenous Fistula For Hemodialysis- An observational study" for my dissertation under the guidance of Dr. N. Rachegowda., Professor, Department of Radio-Diagnosis. In this study, we will assess the role of Doppler ultrasonography in creating arteriovenous fistula for hemodialysis with subsequent follow up for its maturity and complications. You will not be paid any financial compensation for participating in this research project.

All of your personal data will be kept confidential and will be used only for research purpose by this institution. You are free to participate in the study. You can also withdraw from the study at any point of time without giving any reasons whatsoever. Your refusal to participate will not prejudice you to any present or future care at this institution

Name and Signature of the Principal Investigator

Date

MASTER CHART

MASTERSHEET

Name	UHID	Age	Gender	Type	Diameter venous component	PSV venous component	FV venous component	Diameter arterial component	PSV arterial component	Width at AVF	Depth of AVF	PSV at AVF	Complication
1	931234	57	Male	Brachiocephalic	4.8	136	960	5.1	502	6.3	6.9	682	successful
2	923460	41	Female	Brachiocephalic	4.3	91	421	5.2	241	5.5	6.0	315	Failure to mature
3	923468	58	Male	Brachiocephalic	4.2	99	478	5.9	247	5.4	6.5	397	Failure to mature
4	956710	52	Male	Brachiocephalic	5.1	141	1,024	4.9	457	6.6	6.2	594	succesful
5	987613	51	Male	Radio cephalic	6.3	152	1,341	4.6	591	5.9	6.8	723	succesful
6	931412	47	Male	Radio cephalic	5.7	57	326	5.8	298	6.1	7.1	384	Failure to mature
7	912356	52	Male	Brachial-Median Cubital	5.4	68	364	5.7	267	6.3	7.6	357	Failure to mature
8	912376	61	Male	Brachiocephalic	5.9	73	387	5.9	286	5.8	6.7	364	Failure to mature
9	914765	41	Male	Brachiocephalic	5.6	139	1,820	5.7	464	6.8	6.0	584	succesful
10	918765	59	Male	Brachial-Median Cubital	5.4	88	297	5.2	233	5.4	7.5	345	Failure to mature
11	915631	55	Male	Radio cephalic	4.8	91	472	5.6	246	6.1	6.8	364	Failure to mature
12	917654	58	Male	Radio cephalic	4.1	96	431	5.7	288	6.5	6.7	333	Failure to mature
13	915781	66	Male	Brachiocephalic	4.5	110	347	5.8	279	6.6	6.9	399	Failure to mature
14	918789	53	Female	Brachial-Median Cubital	4.9	121	466	5.6	264	5.1	6.4	314	Failure to mature
15	917613	60	Female	Brachiocephalic	4.3	118	473	5.7	237	5.5	7.5	318	Failure to mature
16	913243	52	Female	Radio cephalic	4.7	97	377	5.9	201	6.1	7.4	398	Failure to mature
17	917654	55	Male	Radio cephalic	6.4	154	1,441	5.1	691	6.2	5.8	848	succesful
18	910865	52	Male	Brachiocephalic	4.7	106	368	5.4	209	6.3	7.1	366	pseudoaneurysm
19	910453	48	Male	Brachiocephalic	4.8	156	1,714	4.2	409	5.8	5.5	521	succesful
20	918765	41	Male	Radio cephalic	5.6	111	451	5.8	287	5.7	8.3	375	pseudoaneurysm
21	910876	69	Male	Brachial-Median Cubital	5.8	96	476	5.4	264	6.2	8.6	347	Failure to mature
22	910876	57	Female	Radio cephalic	5.7	117	364	5.8	279	6.7	8.1	311	Failure to mature
23	910567	67	Female	Brachial-Median Cubital	5.5	146	964	4.4	423	6.5	6.1	601	succesful
24	910843	51	Male	Radio cephalic	5.2	126	357	5.1	245	6.6	8.8	322	Failure to mature
25	910896	42	Male	Brachial-Median Cubital	4.9			4.8	312	5.5	8.5	122	cv thrombus
26	910813	49	Male	Brachial-Median Cubital	5.2	149	1,651	4.2	679	6.0	6.4	734	succesful
27	911784	49	Male	Brachiocephalic	4.8	162	1,920	4.1	448	6.3	5.8	641	succesful
28	910856	67	Male	Brachiocephalic	4.3	83	92	5.8	387	6.6	6.7	575	Failure to mature
29	915324	60	Male	Radio cephalic	4.6			5.5	278	6.8	5.7	66	cv thrombus
30	90348	68	Male	Brachiocephalic	3.8			4.3	68	4.4	7.1	21	cv thrombus
31	910764	60	Female	Brachiocephalic	5.8	167	1,890	4.8	486	5.3	5.5	512	succesful
32	910918	57	Female	Brachiocephalic	4.2	76	364	5.2	247	5.9	6.1	293	Failure to mature
33	910983	48	Female	Radio cephalic	5.2	145	468	4.6	424	5.3	5.4	645	Failure to mature