"ROLE OF DOPPLER ULTRASONOGRAPHY OF THE SPLENO-PORTAL SYSTEM IN DIAGNOSING EARLY HEPATIC DYSFUNCTION IN NON- ALCOHOLIC FATTY LIVER DISEASE PATIENTS – A CASE CONTROL STUDY"

By

Dr. VIMAL CHAUDHARY

DISSERTATION SUBMITTED TO SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, KOLAR, KARNATAKA

In partial fulfilment of the requirements for the degree of

DOCTOR OF MEDICINE

IN
RADIODIAGNOSIS

Under the Guidance of:

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PROFESSOR,
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2025

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Abstract

Background:

"Non-alcoholic fatty liver disease (NAFLD) is an increasingly prevalent condition associated with metabolic syndrome and characterized by hepatic steatosis in the absence of secondary causes such as alcohol intake or hepatotoxic drugs. Early detection and monitoring of hepatic dysfunction in NAFLD remain challenging due to the limitations of invasive biopsy and the underutilization of advanced imaging modalities in routine clinical practice. Doppler ultrasonography offers a non-invasive, accessible, and cost-effective alternative for assessing hepatic vascular dynamics.

Aim:

"To assess the role of Doppler ultrasonography of the spleno-portal system in diagnosing early hepatic dysfunction in NAFLD patients."

Materials and Methods:

This was a cross-sectional comparative study conducted over 18 months (June 2023–December 2024) in the Department of Radio-Diagnosis at R.L. Jalappa Hospital, Kolar. A total of 80 adults were included: 44 NAFLD patients (diagnosed based on metabolic syndrome) and 36 healthy age- and gender-matched controls. All subjects underwent abdominal color Doppler ultrasonography using the Philips EPIQ 5 system. Parameters measured included liver echotexture, spleen size, portal vein and hepatic artery diameter, peak systolic velocity (PSV), end-diastolic velocity (EDV), resistive index (RI), and pulsatility index (PI).

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"ROLE OF DOPPLER ULTRASONOGRAPHY OF THE SPLENO-PORTAL SYSTEM IN DIAGNOSING EARLY HEPATIC DYSFUNCTION IN NON ALCOHOLIC FATTY LIVER DISEASE PATIENTS". Abstract Background: "Non-alcoholic fatty liver disease (NAFLD) is an increasingly prevalent condition associated with metabolic syndrome and characterized by hepatic steatosis in the absence of secondary causes such as alcohol intake or hepatotoxic drugs. Early detection and monitoring of hepatic dysfunction in NAFLD remain challenging due to the limitations of invasive biopsy and the underutilization of advanced imaging modalities in routine clinical practice. Doppler ultrasonography offers a non-invasive, accessible, and cost-effective alternative for assessing hepatic vascular dynamics. Aim: "To assess the role of Doppler ultrasonography of the spleno-portal system in diagnosing early hepatic dysfunction in NAFLD patients." Materials and Methods: This was a cross-sectional comparative study conducted over 18 months (June 2023– December 2024) in the Department of Radio-Diagnosis at R.L. Jalappa Hospital, Kolar. A total of 80 adults were included: 44 NAFLD patients (diagnosed based on metabolic syndrome) and 36 healthy age- and gender-matched controls. All subjects underwent abdominal color Doppler ultrasonography using the Philips EPIQ 5 system. Parameters measured included liver echotexture, spleen size, portal vein and hepatic artery diameter, peak systolic velocity (PSV), end-diastolic velocity (EDV), resistive index (RI), and pulsatility index (PI). Results: NAFLD patients demonstrated significantly lower hepatic artery PSV and RI compared to controls ($p < 0.05$). Portal venous flow and splenic artery PSV were also consistently reduced in the NAFLD group. Abnormal Doppler findings were associated with deranged liver function tests. These results suggest hemodynamic alterations in the spleno-portal system even in early stages of NAFLD. Conclusion: Doppler ultrasonography is a valuable, non-invasive tool for early detection of hepatic vascular changes in NAFLD. Incorporating hepatic and splenic Doppler evaluation into routine assessment may enhance diagnosis and monitoring. Future studies with larger cohorts, histological correlation, and MRI validation are recommended. Keywords: NAFLD, Doppler ultrasonography, portal vein, hepatic artery, splenic artery, hepatic dysfunction, non-invasive imaging 1. INTRODUCTION "Non-alcoholic fatty liver disease (NAFLD) encompasses a spectrum of liver conditions" marked by the accumulation of fat in the liver (macrovesicular steatosis), detectable through imaging or biopsy, in the absence of other contributing factors such as significant alcohol intake or prolonged use of medications known to cause fatty liver, or genetic disorders, are absent. Potential contributing variables include the prevalence of childhood obesity, the consumption of unhealthy fast food, rising obesity rates, longer lifespans, and sedentary lifestyles.1Because as fatty liver disease is diagnosed by ultrasonography, the incidence and prevalence of NAFLD remain underreported. Three to ten percent of teenagers, thirty to fifty percent of people with diabetes mellitus, ninety percent or more of patients with hyperlipidemia, eighty to ninety percent of obese adults, and forty to seventy percent of obese babies have NAFLD.2 Before being identified, people with non-alcoholic fatty liver disease (NAFLD) may have a range of vague symptoms, even though the majority of them are asymptomatic. One of the most common early symptoms is fatigue. Acute to moderate upper gastric pain, polydipsia, sleep problems, and abdominal distension.3 Although liver biopsies are considered the most reliable technique for diagnosing non-alcoholic fatty liver disease (NAFLD), they are invasive procedures that carry a significant risk of sample mistakes if performed by inexperienced professionals.4 Ultrasound (US) is the most common method used to make a clinical diagnosis of fatty liver disease globally, and it is often discovered by accident. With a sensitivity of 77% to 100%, liver echogenicity performs better than the renal cortex and spleen5. Moreover, a lack of diaphragm definition and inadequate intrahepatic vascular

ABBREVIATIONS

ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
CAP	Controlled attenuation parameter
CT	Computed tomography
HA	Hepatic artery
HARI	Hepatic artery resistance index
INR	International Normalized Ratio
LGV	Left gastric vein
MRI	Magnetic resonance imaging
NAFLD	Non-alcoholic fatty liver disease
NASH	Non-alcoholic steatohepatitis
PV	Portal vein
RAPV	Right anterior pulmonary vein
RGV	Right gastric vein
RI	Resistance index
RPPV	Right posterior pulmonary vein
SMV	Superior mesenteric vein
SV	Splenic vein
SWE	Shear Wave Elastography
US	Ultrasound
VCTE	Vibration-controlled transient elastography
VPI	Vein pulsatility index

ABSTRACT

Background:

Non-alcoholic fatty liver disease (NAFLD) is an increasingly prevalent condition associated with metabolic syndrome and characterized by hepatic steatosis in the absence of secondary causes such as alcohol intake or hepatotoxic drugs. Early detection and monitoring of hepatic dysfunction in NAFLD remain challenging due to the limitations of invasive biopsy and the underutilization of advanced imaging modalities in routine clinical practice. Doppler ultrasonography offers a non-invasive, accessible, and cost-effective alternative for assessing hepatic vascular dynamics.

Aim:

To assess the role of Doppler ultrasonography of the spleno-portal system in diagnosing early hepatic dysfunction in NAFLD patients.

Materials and Methods:

This was a cross-sectional comparative study conducted over 18 months (June 2023–December 2024) in the Department of Radio-Diagnosis at R.L. Jalappa Hospital, Kolar. A total of 80 adults were included: 44 NAFLD patients (diagnosed based on metabolic syndrome) and 36 healthy age- and gender-matched controls. All subjects underwent abdominal color Doppler ultrasonography using the Philips EPIQ 5 system. Parameters measured included liver echotexture, spleen size, portal vein and hepatic artery diameter, peak systolic velocity (PSV), end-diastolic velocity (EDV), resistive index (RI), and pulsatility index (PI).

Results:

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Conclusion:

Doppler ultrasonography is a valuable, non-invasive tool for early detection of hepatic vascular changes in NAFLD. Incorporating hepatic and splenic Doppler evaluation into routine assessment may enhance diagnosis and monitoring. Future studies with larger cohorts, histological correlation, and MRI validation are recommended.

Keywords: NAFLD, Doppler ultrasonography, portal vein, hepatic artery, splenic artery, hepatic dysfunction, non-invasive imaging

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INTRODUCTION

1. INTRODUCTION

"Non-alcoholic fatty liver disease (NAFLD) encompasses a spectrum of liver conditions" marked by the accumulation of fat in the liver (macrovesicular steatosis), detectable through imaging or biopsy, in the absence of other contributing factors such as significant alcohol intake or prolonged use of medications known to cause fatty liver, or genetic disorders, are absent. Potential contributing variables include the prevalence of childhood obesity, the consumption of unhealthy fast food, rising obesity rates, longer lifespans, and sedentary lifestyles.[1]Because as fatty liver disease is diagnosed by ultrasonography, the incidence and prevalence of NAFLD remain underreported. Three to ten percent of teenagers, thirty to fifty percent of people with diabetes mellitus, ninety percent or more of patients with hyperlipidemia, eighty to ninety percent of obese adults, and forty to seventy percent of obese babies have NAFLD.[2]

Before being identified, people with non-alcoholic fatty liver disease (NAFLD) may have a range of vague symptoms, even though the majority of them are asymptomatic. One of the most common early symptoms is fatigue. Acute to moderate upper gastric pain, polydipsia, sleep problems, and abdominal distension.[3]

Although liver biopsies are considered the most reliable technique for diagnosing non-alcoholic fatty liver disease (NAFLD), they are invasive procedures that carry a significant risk of sample mistakes if performed by inexperienced professionals.[4] Ultrasound (US) is the most common method used to make a clinical diagnosis of fatty liver disease globally, and it is often discovered by accident. With a sensitivity of 77% to 100%, liver echogenicity performs better than the renal cortex and spleen[5]. Moreover, a lack of diaphragm definition and inadequate intrahepatic vascular visibility are recognized as markers of an obese liver on ultrasonography.

Extensive research on a non-invasive approach for the longitudinal monitoring of NAFLD is being conducted in order to meet a large unmet need in the treatment of this disease. Serum biomarkers and a physical method that uses a range of imaging modalities, including computed tomography (CT) scans, magnetic resonance imaging (MRI), ultrasound (US), and color Doppler are the two non-invasive techniques used.

Liver tissue stiffness is assessed using vibration-controlled transient elastography and ultrasound-based elastographic methods, including strain imaging, shear wave imaging, and acoustic radiation force impulse.[6] Magnetic resonance elastography is a far more accurate method of detecting liver fibrosis. Apart from fibrosis,[7] MRI methods including frequency selective imaging, out-of-phase/in-phase chemical shift imaging, and proton MR spectroscopy may be used to measure the estimated amount of fat accumulation in the liver parenchyma.[8] A CT volumetric scan can identify the fibrosis stages in people with NAFLD. Most of these approaches, however, are mainly used in research settings and are not appropriate for use in daily life.

Color Doppler imaging, which can be performed with any commercial ultrasound equipment, is now the main and most accessible method for evaluating the liver's intrinsic veins. Numerous research have examined the hepatic vascular dynamics of individuals with NAFLD and NASH, and they have discovered that the distinct waveforms and velocities of the portal vein, hepatic artery, and hepatic vessels are affected.[9] Right lobe atrophy and left lobe hypertrophy are hallmarks of advanced liver disease, which may be brought on by a number of different conditions, including NASH.[10] It has been proposed that as NAFLD progresses, changes in volume may either occur concurrently with or precede the segmental distribution of portal venous flow. There is a dearth of Indian literature on this subject.[11]

In order to determine the importance of color Doppler ultrasonography results in the diagnosis of NAFLD, the study's objective was to assess the color Doppler characteristics of the portal vein system in patients with NAFLD and compare them to those of healthy persons.

AIM & OBJECTIVE

2. AIMS AND OBJECTIVES

Aim

To assess the role of doppler ultrasonography of the spleno-portal system in diagnosing early hepatic dysfunction in non-alcoholic fatty liver disease patients.

Objectives

1. To describe the ultrasonographic features of liver and spleen echotexture in NAFLD patients.
2. To describe the color doppler ultrasonographic features of portal vein, hepatic artery" and splenic artery in NAFLD patients.
3. To compare color doppler ultrasonographic parameters of portal vein and hepatic artery between patients with NAFLD and healthy subjects.

REVIEW OF LITERATURE

3. REVIEW OF LITERATURE

EPIDEMIOLOGY OF NON-ALCOHOLIC FATTY LIVER DISEASE (NAFLD)

"Non-alcoholic fatty liver disease (NAFLD) is more common in men than in women"; its estimated worldwide prevalence is 30% (40% vs. 26%). Furthermore, the prevalence depends on the geographic location and diagnostic technique. A meta-analysis of 92 studies using ultrasonic screening found that the prevalence of NAFLD was lowest in Western Europe at 25% and highest in South America at 44%.[12] Using vibration-controlled transient elastography (VCTE) with controlled attenuation settings, a research of more than 4,000 participants in the United States found that liver steatosis was common at 57%.

The prevalence of NAFLD seems to be increasing. The prevalence of NAFLD rose from around 20 cases per 1000 person-years in 2000 to 70 cases per 1000 person-years in 2015, according to a meta-analysis of 63 studies including over a million people.[13]

ANATOMY OF THE PORTAL VENOUS SYSTEM

Anatomy in Gross

Portal vein (PV) circulation has no valves and a restricted wall structure. Adults are 6–8 millimeters long and 13–14 millimeters wide. Spleen, gallbladder, and pancreas provide portal vein blood. The L1-L2 vertebral level marks the point where the splenic and superior mesenteric arteries converge behind the neck of the pancreas. The portal vein supplies approximately 72% of the liver's oxygen, delivering it at a rate of 40 ml per minute. Human portal blood flow averages 1000–1200 mL/min. Normal portal pressure is 7 mmHg.[14]

Main pancreatic vein (MPV) climbs toward spine at 40°–90° via hepatoduodenal ligament. The liver enters via the porta hepatis. Hepatic hilum divides left & right portal veins from portal trunk.

The RPV's anterior and posterior branches follow. The RPPV supplies Couinaud segments VI and VII with blood, whereas the RAPV supplies V and VIII. Segment I of the caudate lobe and segments II, III, and IV of the liver get blood from the left portal vein.[15]

Hepatic segmental branches bifurcate to create smaller venous branches that become portal venules.

Portal venules finish at hepatic sinusoids. Hepatocytes and endothelial cells transfer blood to central arteries in hepatic sinusoids. Then the hepatic arteries deliver blood to the inferior vena cava.

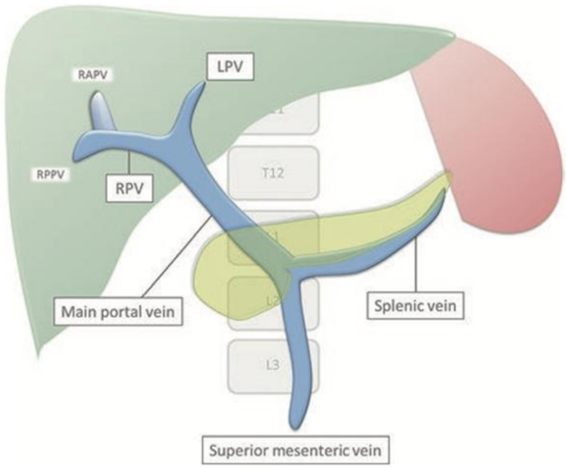


Figure 1. The normal anatomy of the portal venous system. RPPV stands for right posterior portal vein, RAPV for right anterior portal vein, LPV for left portal vein branch, and RPV for right portal vein.

Tributaries

<u>Portal Vein</u>

The MPV is divided into the left, right, and superior pancreaticoduodenal veins, as well as the splenic & superior mesenteric veins. The portal vein (PV) is the result of numerous veins connecting together. The cystic vein drains into the right portal vein, whereas the umbilical vein empties into the left.

The coronary vein, also referred to as the left gastric vein, is a major tributary of the main-portal vein (MPV).Portal hypertension is a substantial risk factor for developing fundal varices in the stomach and esophagus.[16] LGV originates in the stomach's anterior and posterior walls as well as the lower esophageal branches.

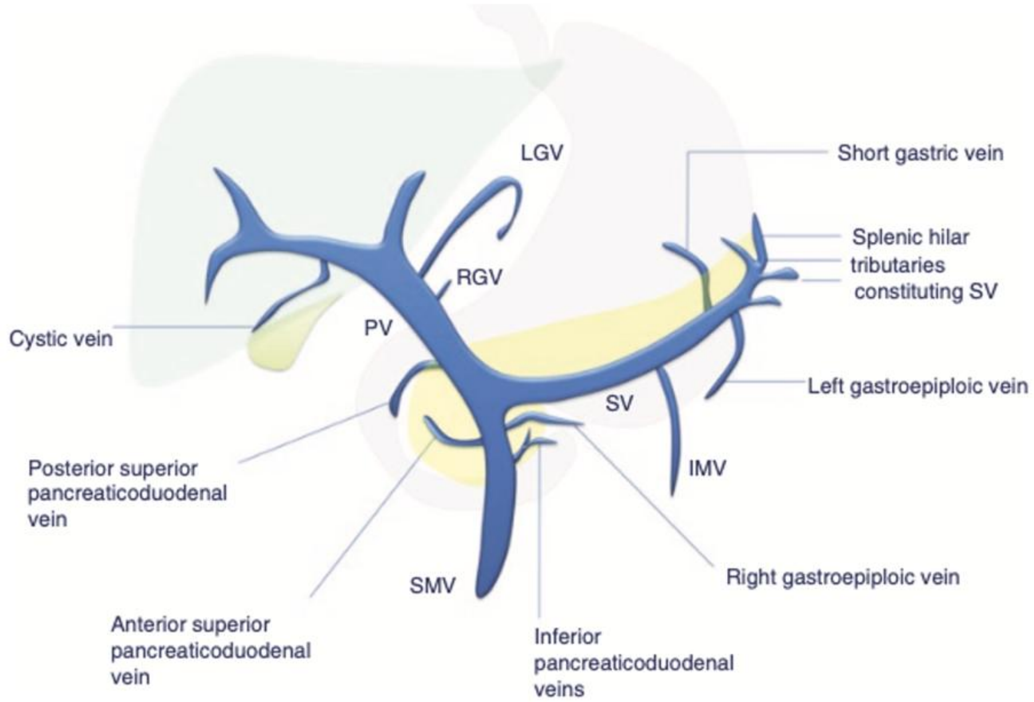


Figure 2. Principal venous tributaries of the portal system. SMV: superior mesenteric vein, IMV: inferior mesenteric vein, LGV: left gastric vein, RGV: right gastric vein, PV: portal vein, and SV: splenic vein

The minor curvature frequently leads to the main portal vein (30%) or the spleno-portal junction (33%). Approximately 37% of lymphatic vaginal veins (LGVs) reach the systemic circulation.

The right gastric vein (RGV) travels via a slight bend adjacent to the stomach pylorus. Beyond the duodenal closure, it establishes a distinct connection to the MPV. The stomach's curvature was eliminated. It receives the prepyloric vein, which empties the duodenum.

Superior Mesenteric Vein

The superior mesenteric vein transports the majority of blood from the small intestine and the colon up to the splenic flexure. The structure climbs the mesenteric base to reach the superior vena cava behind the pancreatic stalk.[17] The SMV receives tributaries from the SMA. In this context, the tributaries of the venous system are the ileal, ileocolic, right colic, and jejunal veins.

The splenic vein

The splenic vein is formed by multiple small veins that originate in the hilum of the spleen. The inferior mesenteric vein (IMV), the short gastric veins, which drain the gastric fundus, the left gastroepiploic vein, which drains the top curve of the stomach, and the pancreatic veins, which drain the pancreatic neck, body, and tail, all contribute blood to the splenic vein (SV).

Inferior Mesenteric Vein

The IMV joins the SV in 38% of cases. It subsequently reaches the first jejunal vein, spleno-portal confluence (32.7%), or superior mesenteric vein (29.3%). The inferior mesenteric vein draws blood from the left colic, sigmoid, and superior rectal veins.[18] It cleans the left colon between the splenic flexure and the midrectum.

Portal Vein Anatomical Variants

Multidetector CT demonstrates normal portal vein architecture 65–80% of the time, according to case studies. The main portal vein (MPV) divides into the left and right portal veins near the hepatic hilum. The right portal vein (RPV) further branches into the right anterior and right posterior portal veins. Any deviation from the norm is anatomical. With liver transplants, hepatic resections, and interventional treatments on the rise, understanding the portal vasculature and its alterations is crucial.

Four main variations of photovoltaic branching are recognized in the literature.

- Type 1: This variation has a documented incidence rate of 9–11% and is often called the trifurcation pattern. The left pulmonary vein (LPV), right anterior pulmonary vein (RAPV), and right posterior pulmonary vein (RPPV) are the three branches of the main pulmonary vein (MPV).
- Type 2: In around 9.7–23% of cases, RPPV is present in the first branch of the portal vein (PV).
- Type 3: As direct derivatives, RAPV and LPV are related.
- With less than 2% of occurrences, type 4 is the rarest variety and is identified by the lack of portal vein bifurcation. The trunk of the main portal vein (MPV) passes through both the left and right lobes of the liver in one continuous intrahepatic arch.

PATHOPHYSIOLOGY OF NON-ALCOHOLIC FATTY LIVER DISEASE (NAFLD)

Insulin resistance and hepatic and systemic inflammation are common features of non-alcoholic fatty liver disease (NAFLD), a systemic metabolic disease. Although the most common symptom of the illness is basic hepatic steatosis, 10–20% of patients have non-alcoholic steatohepatitis, a more advanced form marked by inflammation and liver fibrosis. Hepatocellular carcinoma and liver cirrhosis occur in a considerable percentage of people with NAFLD. Approximately 25% of people worldwide now suffer with NAFLD, with a greater incidence among those who are obese and have type 2 diabetes.[19] Normal weight people are also affected. This syndrome is an example of a systemic metabolic illness because of its pathophysiology, which involves hepatic immune dysfunction, gastrointestinal dysbiosis, lipotoxicity, and resulting hepatic insulin resistance. It also often shows signs of both systemic and hepatic insulin resistance.[20]

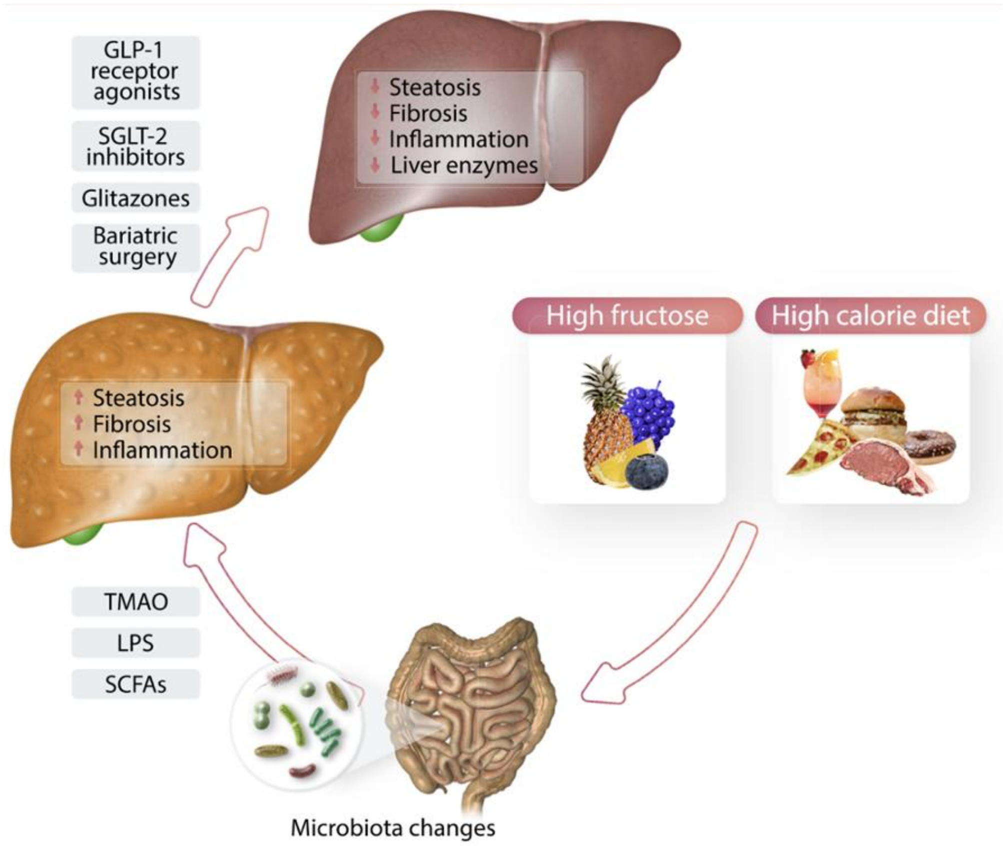


Figure 3. Non-alcoholic fatty liver disease (NAFLD) has many different causes

CLINICAL FEATURES OF NAFLD

The patient's presentation

The majority of NAFLD patients may not have any symptoms, while a considerable proportion of MASH patients may have lethargy, malaise, and nebulous upper abdomen pain. Laboratory tests that show increased aminotransferases or incidental hepatic steatosis on abdominal imaging are often used to identify asymptomatic individuals. People with decompensated cirrhosis are an example of the other extreme, however they are uncommon.

On a physical examination, most people with NAFLD show no signs of liver abnormalities. In certain instances, fatty infiltration of the liver may result in hepatomegaly.

Five to eighteen percent of those with non-alcoholic fatty liver disease have hepatomegaly.[21] Depending on body size and sex, liver sizes of less than 16 cm are often seen on transabdominal ultrasonography.

Ascites, spider angiomata, and palmar erythema are signs of chronic liver disease that people with cirrhosis may experience.

Among individuals diagnosed with non-alcoholic fatty liver disease, laboratory results include:

- Liver enzymes: While some individuals with non-alcoholic fatty liver disease (NAFLD) may show normal aminotransferase levels, others can exhibit mildly to significantly elevated levels of aspartate aminotransferase (AST) and alanine aminotransferase (ALT). When increased, the AST to ALT ratio is less than one, and AST & ALT are normally two to five times the upper range of normality. A normal ALT level does not rule out histologic damage, and the degree of inflammation or liver fibrosis does not always indicate the degree of aminotransferase increase.
- Additionally, while the diagnosis is often derived from increased aminotransferases (or incidental imaging findings) rather than from population-based screening, the incidence of abnormal liver enzymes in NAFLD patients is still unclear.
- Other liver tests, including alkaline phosphatase, may show a rise of two to three times the upper range of normal. Patients with cirrhosis may have aberrant blood albumin and bilirubin levels, which often lie within the normal range.
- Additional test results: Hematologic abnormalities, such as thrombocytopenia and neutropenia, may be seen in people with cirrhosis. People with non-alcoholic fatty liver disease (NAFLD) may have elevated blood ferritin or transferrin saturation. Furthermore, a higher risk of steatohepatitis and fibrosis has been associated with blood ferritin levels in NAFLD patients that are surpassing the upper limit of normal by over 50%.[22]

DIAGNOSTIC EVALUATION OF NAFLD

The purpose of the diagnostic evaluation in patients of suspected NAFLD serves to verify the diagnosis of NAFLD and rule out any further liver disease reasons.

Physical examination and medical history

The history comprises a description of symptoms, pharmacological interventions (including over-the-counter medications and herbal supplements), pre-existing medical conditions (like type 2 diabetes mellitus, dyslipidemia, and obesity), familial medical history (especially cirrhosis, non-alcoholic fatty liver disease, or hepatocellular carcinoma), and patterns of alcohol consumption (including the amount, frequency, and duration of use).

One condition linked to the use of amiodarone, glucocorticoids, methotrexate, and tamoxifen is hepatic steatosis.[23]

Physical examination comprises BMI evaluation, palpation for liver enlargement, and detection of clinical indicators of chronic liver disease such as splenomegaly, ascites, and hepatitis.

Laboratory studies

In patients with suspected NAFLD, the following laboratory tests are performed to assess hepatic function and related conditions:

- Aminotransferases – Alanine aminotransferase (ALT) & aspartate aminotransferase (AST)
- Alkaline phosphatase
- Bilirubin total
- Albumin in serum
- Prothrombin Time/International Normalized Ratio (INR)
- A thorough examination of platelets
- Fasting Blood glucose levels
- High-density lipoprotein cholesterol, triglycerides, and total serum cholesterol

The following tests are needed to look into the cause of liver disease in more detail:

- Hepatitis B surface antigen (HBsAg), hepatitis B surface antibody (anti-HBs), and total hepatitis B core antibody (anti-HBc).
- Hepatitis C viral RNA & anti-hepatitis C virus antibodies were found to be positive.
- Ferritin, total iron-binding capacity, & plasma iron levels
- Anti-mitochondrial antibodies
- Antinuclear antibody, antismooth muscle antibody, and immunoglobulin G (IgG) levels (for female patients and/or those with a history of autoimmune illnesses or aminotransferases more than five times the range of normal)
- Ceruloplasmin (for those with neurocognitive problems or those under 50)
- Concentration of alpha-1 antitrypsin (AAT)
- Tissue transglutaminase (tTG-IgA)-targeting antibody

Diagnostic imaging

The growing clinical relevance of NAFLD, coupled with the disadvantages of liver biopsy, has led to a rising interest in accurate, non-invasive imaging approaches. To date, several imaging methods have been utilized to evaluate patients with NAFLD.

Conventional ultrasonography

- Ultrasonography using Doppler
- CT scan
- Magnetic resonance imaging's proton density fat fraction (MRI-PDFF)
- Controlled attenuation parameter (CAP) parameter
- Vibration-controlled transient elastography is referred to as VCTE.

- Impulse of Acoustic Radiation Force (ARFI)
- Elastography using Shear Waves (SWE)

Every imaging method has pros and cons when it comes to measuring hepatic fat. Recent studies have examined the effectiveness of many imaging modalities that assess hepatic stiffness to quantify inflammation & fibrosis in patients with NAFLD.[24]

Biopsy of the liver

When the proportion of steatotic hepatocytes in liver tissue surpasses 5%, NAFLD is histologically identified. Patients with steatosis may accumulate iron in their livers.[25]

Any one of the following histologic abnormalities may be present in people with NAFLD:

- Only steatosis.
- Steatosis, which is linked to portal or lobular inflammation, lacks hepatocyte ballooning.
- Hepatocytes expand as a result of steatosis, although inflammation is absent.

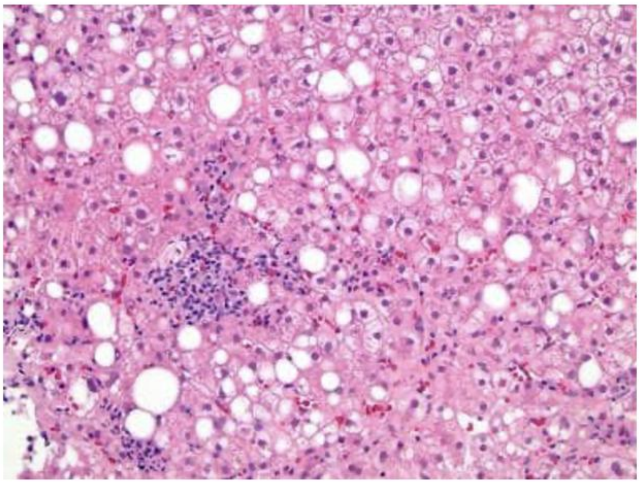


Figure 4. Steatosis and inflammatory foci, which are mostly macrovesicular in form and include Kupffer cells and lymphocytes inside the hepatic lobules, are characteristics of steatohepatitis. This is seen in the following figure.

PROGNOSIS

Patients with non-alcoholic fatty liver disease (NAFLD) have higher death rates than the overall population. These people have a higher chance of dying from cardiovascular causes because of metabolic abnormalities. Compared to hepatic disorders, cardiovascular problems are linked to a greater death rate in humans. Non-alcoholic steatohepatitis (NASH) may develop into cirrhosis, however simple steatosis is reversible and non-progressive. NAFLD is a condition that worsens with time.[26] A study by Ekstedt et al. found that 41% of patients developed cirrhosis during a 13-year follow-up.[27] In cohorts with "non-alcoholic fatty liver disease (NAFLD) or non-alcoholic steatohepatitis (NASH) and minimal or no cirrhosis", the risk of developing hepatocellular carcinoma (HCC) over 20 years was low, ranging from 0% to 3%.In contrast, the risk was significant in NASH groups with cirrhosis, with a range of 2.4% over a seven-year period. White et al. found this using a meta-analysis.[28]

COMPLICATIONS

In decreasing order, the following are the most serious side effects of non-alcoholic fatty liver disease (NAFLD):

- Cardiovascular disorders
- The hepatocellular cancer
- End stage liver disease

There is a strong correlation between the severity of these events and the grade and histological stage of liver disease.

TREATMENT OF NAFLD

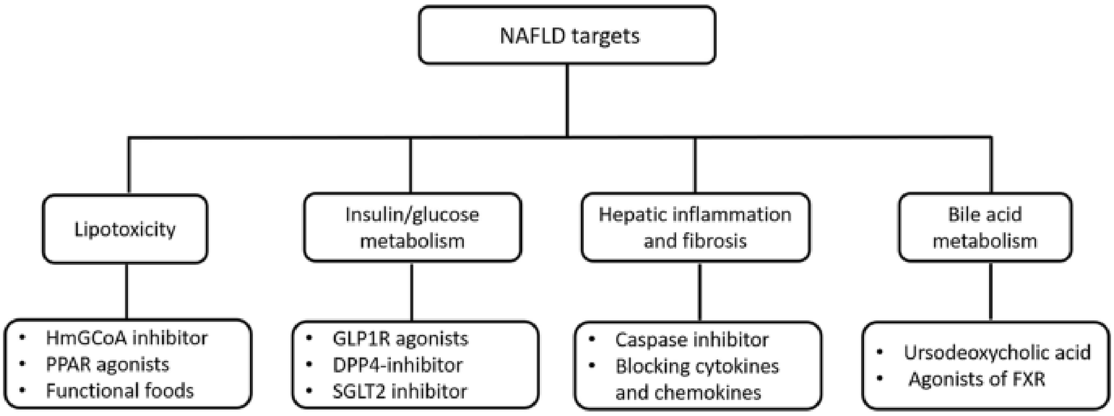


Figure 5. Goals for treating non-alcoholic fatty liver disease

DOPPLER ULTRASOUND PHYSICS

Basic principles

Motion observation is the main source of ultrasonic flow images, which include both color and spectral Doppler images. Ultrasound scanners use wave sequence analysis to detect blood flow. From pulse to pulse, the stationary tissue's reflections don't change. Due to reflections from moving objects, the time it takes for a signal to reach the receiver might vary. One way to measure these changes is to use a direct temporal difference or, more often, a phase shift that produces the Doppler frequency. They are then processed using the Doppler effect to create a sonogram, which is a visual depiction of color flow.

The movement has to match the beam's direction, as seen in the picture below. When the flow is perpendicular to the beam, no relative motion between pulses can be seen.

The following variables affect the Doppler signal's strength:

- An increase in blood velocity causes a corresponding rise in the Doppler frequency.
- The Doppler frequency rises with increasing ultrasonic frequency.
- As the ultrasonic beam gets closer to the flow direction, the angle between the two decreases, increasing the Doppler frequency.

- B-mode, which exhibits better penetration at lower ultrasonic frequencies, is analogous to this.
- The right frequency must be chosen by striking a compromise between improved penetration and higher flow sensitivity. The use of Doppler ultrasonography is rather widespread.

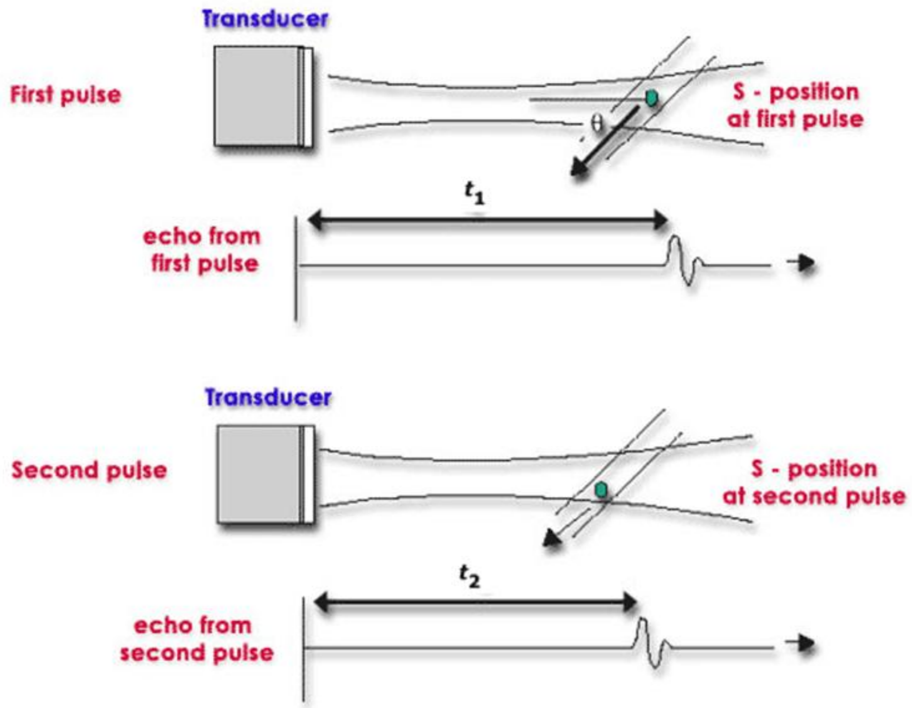


Figure 6. Ultrasonic velocity evaluation

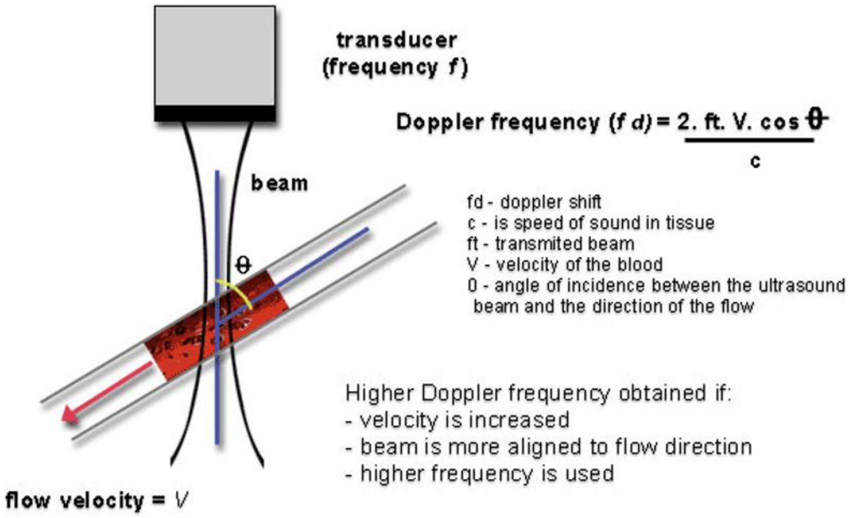


Figure 7. Doppler ultrasonography uses a phase shift in the received signal to assess the movement of scatterers across the beam

Every one of the three basic levels of ultrasonic imaging provides more detailed information than the one before it. At the first level, a standard grayscale brightness mode (B-mode) assessment is performed without using Doppler. The second level includes a designated area specifically targeted for color Doppler analysis. At this level, the flow of blood via blood arteries is graphically shown. An infinitesimal zone that contains a vessel of interest at the third level is called a sample volume, and it acts as a confined region for research. A spectral Doppler waveform is created when the focus is pointed at the vessel.

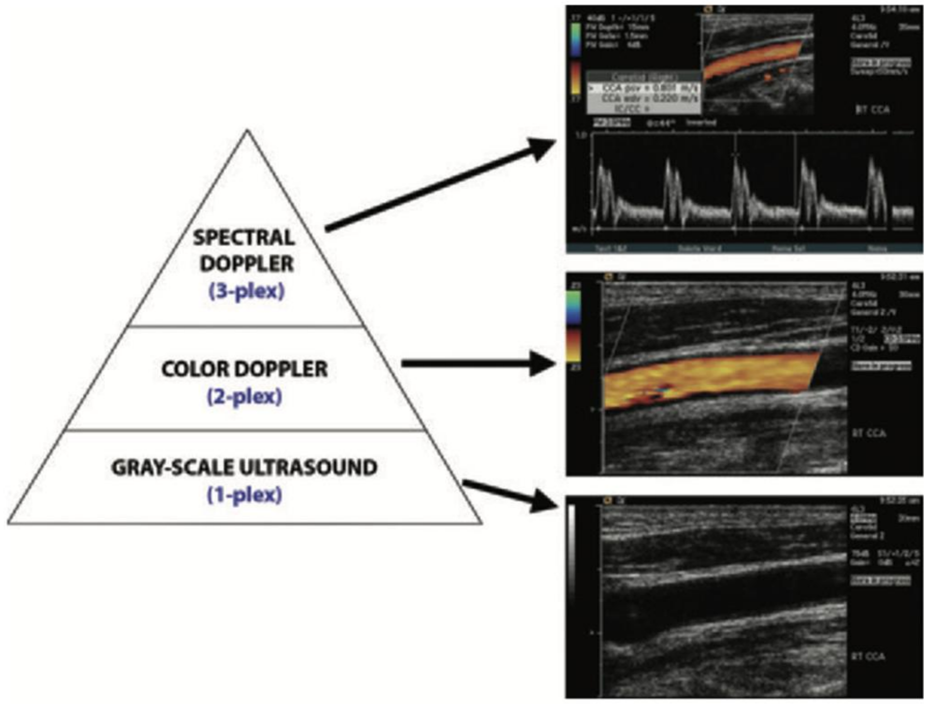


Figure 8. Doppler examination terminology

The Spectral Doppler's components

The spectrum waveform is shown in the bottom portion of the screen, and the color Doppler picture is shown in the top portion. This is how the spectral Doppler ultrasound results are shown. The waveform is made up of information from a tiny sample volume, usually 2-4 mm in size and often found close to the vessel's core. The position of this sample volume is the responsibility of the sonographer, also known as the ultrasound technician.

Waveform Features and Attributes

Each spectrum waveform's morphological characteristics include details on direction, acceleration, and velocity. The waveform's closeness to the baseline provides directional information.[29] The distance between the baseline and any point on the trajectory is what determines velocity. The gradient of the curve, which shows the rate of velocity change, is analyzed to produce acceleration data. Acceleration fluctuations are indicated by peaks or inflection points in the waveform.

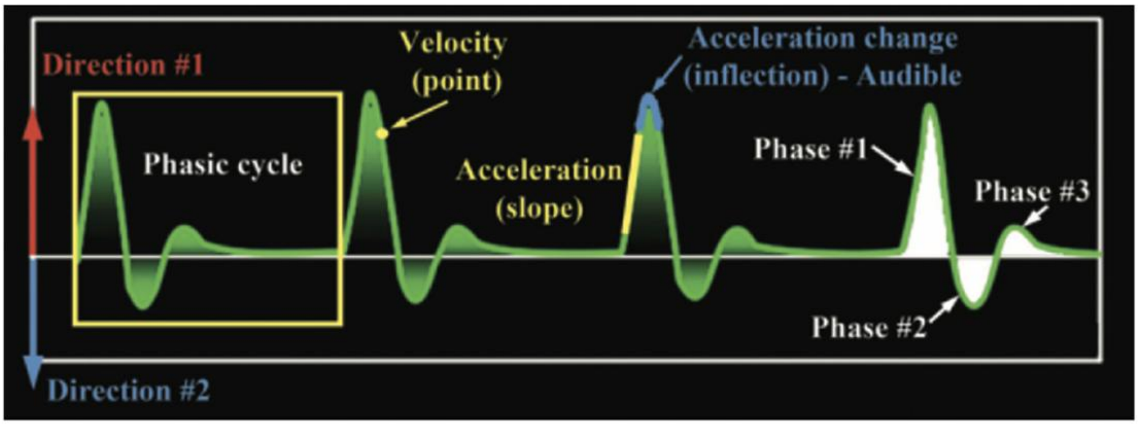


Figure 9. The features of a spectral waveform are shown in a magnified perspective

<u>**Antegrade and retrograde directions of blood circulation**</u>

There are two main definitions for blood flow orientation. The goal is to characterize the vascular flow exactly. To indicate the direction of passage, the phrases antegrade and retrograde are used. Optimizing the flow of transducers from the United States is the second goal. A flow is the movement that is directed either toward or away from the transducer. In the absence of a standard, color Doppler technology indicates that blood flowing toward the transducer is red and blood flowing away from the transducer is blue.[30] Blood flow toward the transducer is indicated by positive spectral Doppler imaging values that are higher than the baseline, while blood flow away from the transducer is indicated by negative values that are lower than the baseline.

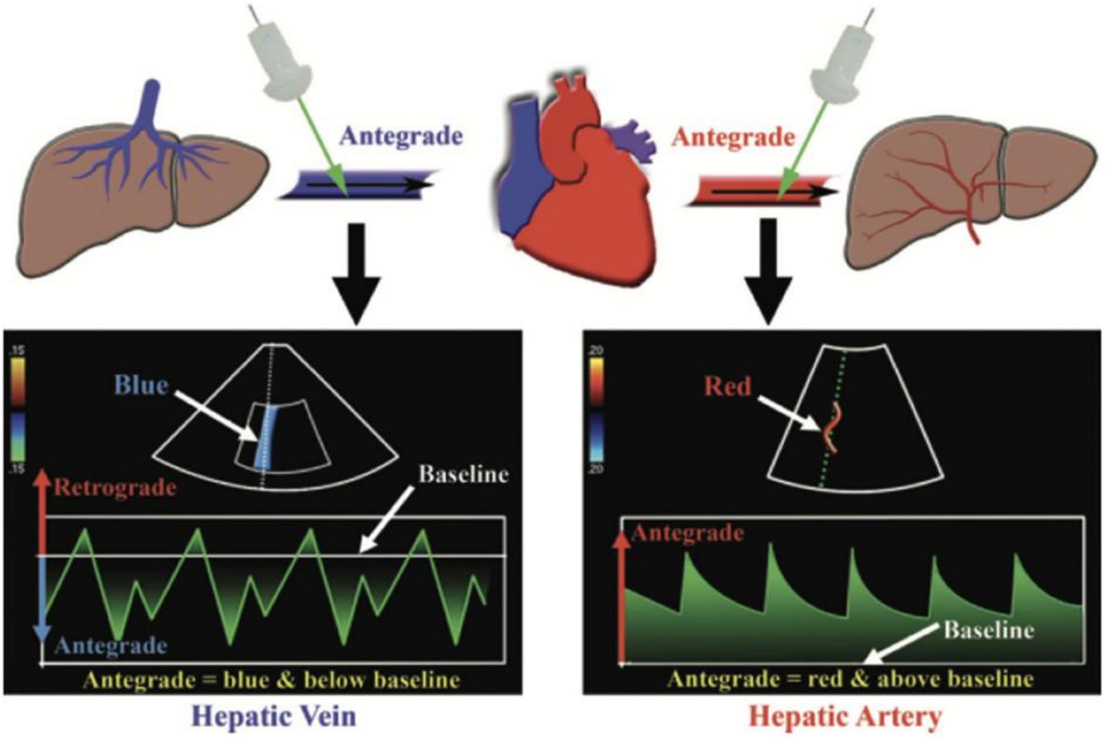


Figure 10. the difference between antegrade and retrograde flow.

Phase Quantification vs. Phasicity

The intricacy of the liver is highlighted by the Doppler terminology, which highlights the subtle differences between phase & phasic, as well as the many interpretations of what defines a phase. When referring to their intensity and the existence or absence of components, the phrases phasic and cyclic may be used interchangeably.[31] Any process exhibiting phasic or cyclical features may be referred to as phasic or phasicity interchangeably. Conversely, a phase refers to a distinct and quantifiable component of a phasic process.

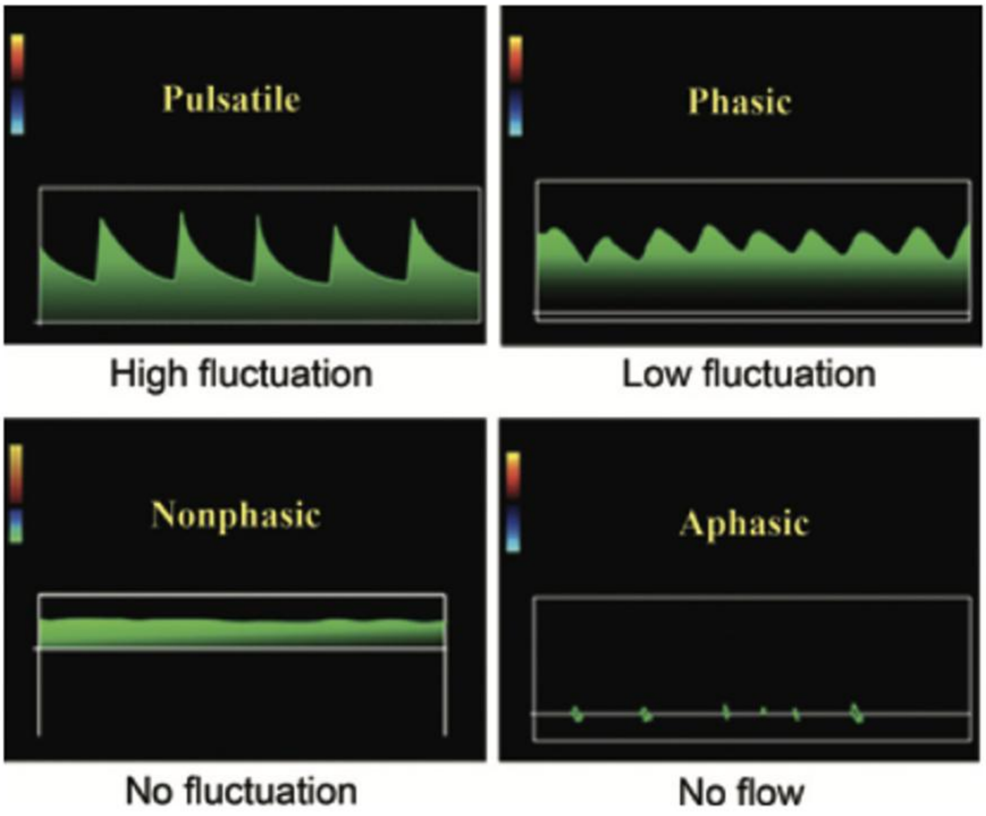


Figure 11. The various waveforms are shown in diagrams

Resistance of the Arteries

Under normal physiological conditions, arteries can adjust their resistance to prioritize blood flow to organs with the greatest demand. When an organ becomes active, its arteriolar network typically dilates, resulting in a low-resistance flow pattern that ensures adequate blood delivery. The waveform changes to increased resistance in "power save" mode, which causes the arterioles of an organ to contract. Blood flow is thus diverted to other organs.

The three traditional indices used to assess vascular resistance are the pulsatility index (PI), the systolic/diastolic ratio, and the resistive index (RI). The portal vein and arteries use different methods to determine the pulsatility index (PI).[32]

In the field of Doppler ultrasound imaging of the liver, the following points outline a clear and efficient method for grasping the factors that affect the hepatic artery's resistance index (RI).Since a high RI does not primarily indicate hepatic illness, it is less relevant as a single observation than a low RI.

1. The high renal index (RI) may be caused by the patient's advanced age, severe distal microvascular disease, or postprandial state. Numerous variables, including chronic cirrhosis-induced liver disease or chronic hepatitis, may contribute to the latter condition.
2. Proximal blockage or distal blood flow diversion, such as via arteriovenous or arterioportal connections, might cause a lower resistance index. These symptoms are often seen in Osler-Weber-Rendu syndrome, trauma (including injuries from medical procedures), and severe cirrhosis.

INDICATIONS AND ROLE OF DOPPLER IN HEPATIC DISEASES

Equipment

Modern ultrasound systems that are used to scan the adult abdomen are distinguished by transducers with a wide frequency range. These transducers may be used for a wide range of tasks, including spectral Doppler waveform capture, color & power-Doppler imaging, and the creation of real-time grayscale pictures. For liver examination, harmonic imaging is sometimes used in combination with transducer frequency parameters ranging from 3 to 5 MHz. Hepatic artery color and spectral Doppler imaging often requires frequencies in the range of 2 to 4 MHz. Regardless of the frequency used for the other function, Doppler and imaging may be used at different frequencies in a range of ultrasonic systems and transducers with wide bandwidths. When employing color Doppler for liver imaging, the grayscale setting may be changed to a frequency of 5 MHz and its harmonics. Furthermore, the color Doppler optimization frequency might be changed to 3 MHz.

The Doppler evaluation of hepatic arteries may be performed using the same methods as grayscale imaging. Because low-frequency transducers can enter capillaries and do a thorough examination of blood flow inside them, they are essential. Lower frequency transducers may have reduced sensitivity to slow-flowing blood compared to higher frequency transducers. It is often advised to adjust the Doppler setting to the maximum frequency in order to produce a distinctive Doppler waveform and preserve flow sensitivity by reducing beam attenuation.

Indications for doing a Doppler liver examination:

- Portal hypertension
- Assessments of aberrant liver function
- Hepatitis
- Hepatitis
- Metastases in the liver
- Trauma
- Hypercoagulable state
- Bleeding throughout the digestive system

NORMAL DOPPLER FLOW IN THE HEPATOPORTAL CIRCULATION

Hepatic artery

In healthy people, 25–30% of the liver's blood supply comes from the hepatic artery or arteries. The celiac axis in the upper abdomen, which comes just after the aorta, is where the major hepatic artery begins. The artery splits into left and right branches when it enters the liver via the porta hepatis. In almost 25% of people, there are several hepatic arteries. It is common to observe variations in the left and right hepatic arteries. Occasionally, “the right hepatic artery is replaced by an accessory artery that arises from the superior mesenteric artery and supplies the right lobe of the liver. Similarly, when the left hepatic artery is replaced, it is often substituted by the left gastric artery, a branch of the celiac artery. Within the liver, the hepatic artery runs alongside the portal vein and its branches. Although hepatic arteries are relatively small, the main hepatic artery can usually be seen near the porta hepatis on grayscale imaging, while its smaller “branches are typically not visible.

In the hepatic artery, the Doppler waveform displays low resistance, unidirectional flow, and pulsatile traits. A prominent peak is seen during systole, followed by continuous forward flow throughout the entire cardiac cycle. The resistive index (RI) generally ranges from 0.55 to 0.70, and it is calculated by dividing the difference between peak systolic and end-diastolic velocities by the peak systolic velocity.

Portal veins

In healthy individuals, approximately 70% to 75% of the liver’s blood supply is delivered through the portal veins. The primary portal veins in the upper abdomen—the splenic vein and the superior mesenteric vein—are situated behind the pancreas. Blood enters the liver through the porta hepatis, where it divides into the left and right portal veins. The primary portal vein splits, causing the right portal vein to split into anterior and posterior branches. In 35% of instances, this structural alteration takes place.

Currently, the main portal vein branches into the “right posterior”, right anterior, and left portal veins. According to other theories, the left and right portal veins merge into a trunk, while the main portal vein bifurcates to generate the right posterior branch.[33] The hepatic arteries are small compared to the portal vein and its tributaries. In grayscale imaging, the hepatic arteries are less apparent than the principal portal vein and its deeper tributaries inside the liver parenchyma.

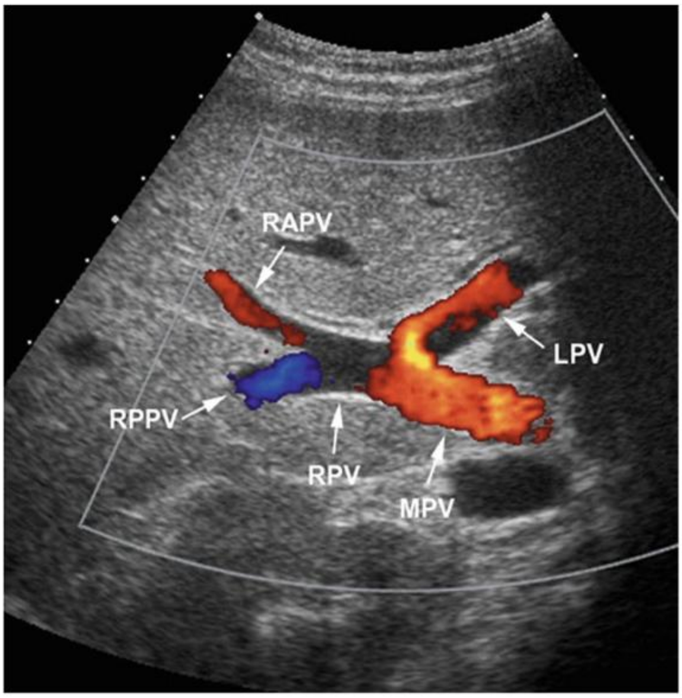


Figure 13. Vein of the main portal and all of its branches. The main portal vein (MPV), which is separated into the left and right portal veins (LPV and RPV), as well as the right portal vein's bifurcation into anterior (RAPV) and posterior (RPPV) branches, are visible on the porta hepatis color Doppler ultrasound

There is little change in the portal veins Doppler waveform, which shows a steady antegrade flow into the liver. Heart pulse and breathing have very little effect on flow. The portal vein's maximum velocity usually ranges from 15 to 30 cm/s, with a normal range of 15 to 40 cm/s. The lowest velocity of the principal portal vein is more than half of the highest velocity, and it almost ever fluctuates. From the lowest to the greatest, the velocity ratio is, in essence, less than "0.5".

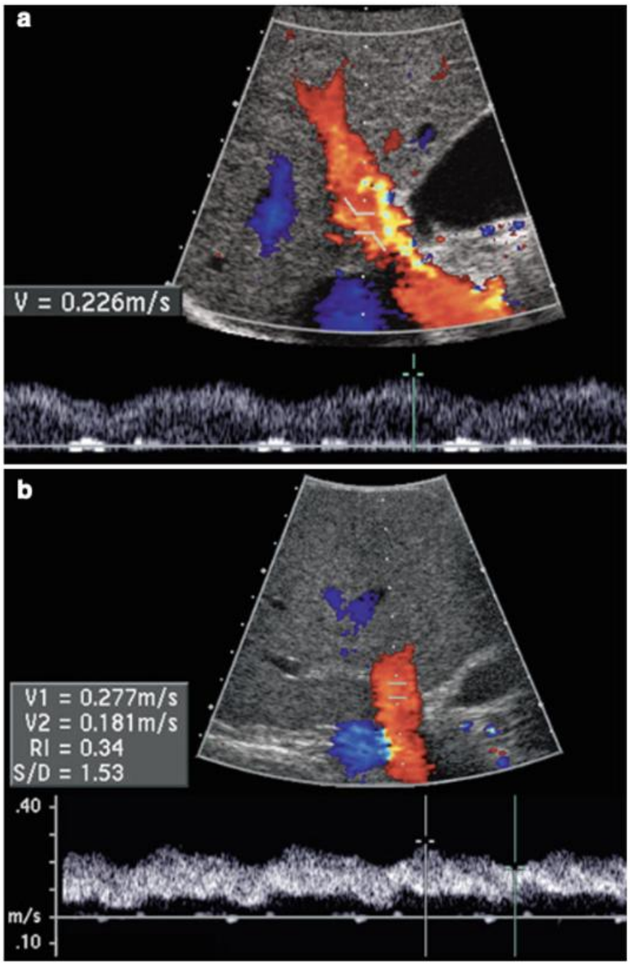


Figure 14. The portal vein waveform. With a maximum velocity of 22.6 cm/s, the color and spectral Doppler ultrasonography of the major portal vein show a continuous, antegrade, slightly undulating flow into the liver. (b) There is considerable pulsatility in the Portal Doppler waveform, with the lowest velocity (18.1 cm/s) exceeding half of the highest velocity (27.7 cm/s).

Hepatic veins

All hepatic blood must be collected from the hepatic arteries via the inferior vena cava. All blood entering the liver passes via the portal vein and hepatic arteries before leaving the body through the inferior vena cava. The left, right, and center veins are the most common hepatic veins. The interstitial area between the left liver lobe's medial and lateral portions is where the left hepatic vein is situated. While the right hepatic vein passes through the front and posterior regions of the right lobe, the middle hepatic vein links the left and right liver lobes.[34] The left and center veins are joined into a single artery in certain hepatic venous layouts, but others feature an extra right inferior hepatic vein.

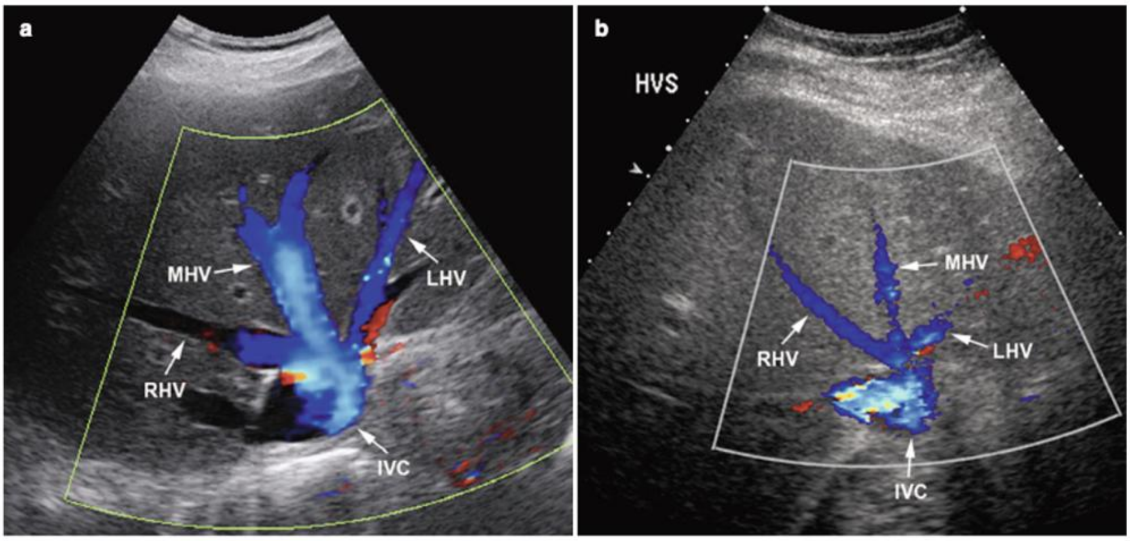


Figure 15. Hepatic veins. (a) and (b) Color Doppler sonograms of the liver showing the convergence of the liver's inferior vena cava (IVC) with the left, middle, and right hepatic veins (LHV, MHV, and RHV)

With a few brief exceptions, hepatic veins mostly transport blood to the heart and away from the liver throughout the cardiac cycle. Due to increased right atrial pressure during atrial contraction, the first hepatic vein waveform shows retrograde flow into the liver, which starts with atrial systole. A-wave is the word used to characterize this event.[35] Instead of a weakened reverse flow, some people may have a decreased forward flow in the A-wave. Following atrial systole, which includes the release of blood into the major arteries, comes ventriculoventricular systole. When "the tricuspid valve is drawn into the right ventricle", there is an increase in both atrial pressure and backward blood flow. During the heart's contraction phase, the S-wave—also referred to as the hepatic vein waveform—reflects the movement of blood into the inferior vena cava. This phase represents the peak blood flow within the heart during the cardiac cycle. As systole comes to an end, the tricuspid valve begins to separate from the right ventricle.

According to the hepatic waveform, the V-wave occurs when blood flow reverses or decreases away from the heart. During diastole, the tricuspid valve opens, allowing blood to enter the ventricles and atria. The D-wave now serves as a representation of the hepatic waveform. The velocities of forward flow are almost the same as those of cardiac systole.[36]

Occasionally, the hepatic venous flow can be categorized into four phases: the A-wave, V-wave, S-wave, and D-wave, although it may also present as a triphasic pattern at times. Because some people see the V-wave as a period of transition rather than a wave, it is referred to as the latter wave. The hepatic venous waveform pattern must be recognized in order to detect abnormal channel flow, regardless of the nomenclature used.

DOPPLER US IN NAFLD

Duplex Doppler ultrasonography (US) is recognized as a vital diagnostic technique for the noninvasive evaluation of the hepatic vasculature and certain hepatic parenchymal disorders.[37] According to a recent research, the substantial adipose infiltration of the liver may have an impact on the hepatic vein hemodynamics and the hepatic artery resistance index (HARI), as measured by Doppler ultrasonography.[38]

A. Portal vein pulsatility-index in non-alcoholic fatty liver disease (NAFLD)

Retroperitoneal and intraperitoneal adipose tissues exist. This regional adiposity is important because intraperitoneal adipose tissue drains into the liver via the portal vein, whereas retroperitoneal empties into the systemic circulation. Thus, free fatty acids, glycerol, and other adipocytokines from intraperitoneal adipose tissue may impact hepatic glucose, triglyceride, insulin, and other substrates and hormone metabolism. Unique venous outflow pattern supports portal fat hypothesis 38. Portal vein pulsatility index is an imaging biomarker obtained from duplex Doppler. Pulsatility of the portal vein is measured. In pulsed-wave Doppler ultrasonography, Vmax is the maximum blood velocity and Vmin the lowest. The portal vein pulsatility index (VPI) is (Vmax – Vmin) / Vmax. In a rabbit model of steatosis, mild fatty liver infiltration drastically decreases portal and total hepatic blood flow and microcirculation and increases hepatic artery flow and portal pressure.[39]

Numerous studies have explored VPI distribution in non-alcoholic fatty liver disease (NAFLD), although metformin, dietary modifications, & increased physical activity have cast doubt on these results".[40]

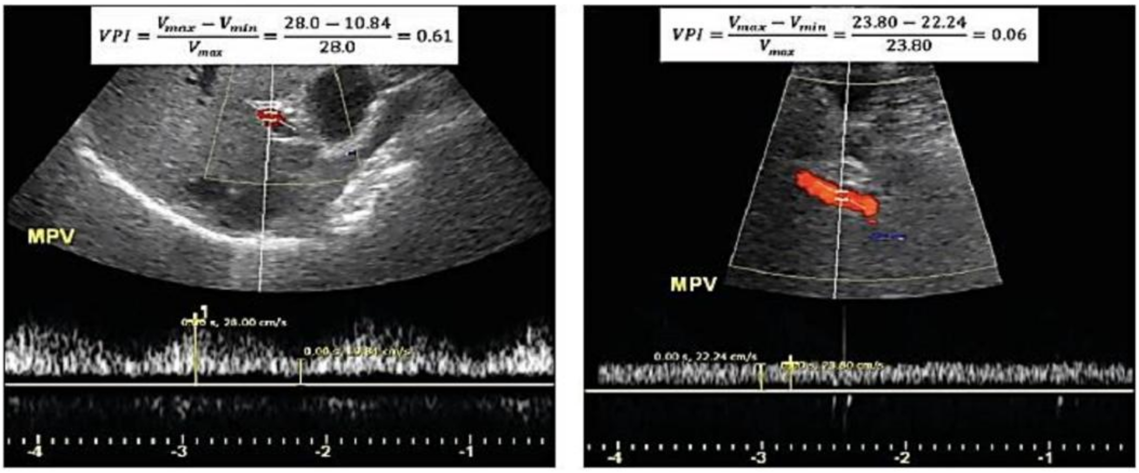


Figure 16. It calculates the portal venous pulsatility index (VPI). Non-alcoholic fatty liver disease (NAFLD) at fibrosis stage F0 in a 47-year-old man. The main portal vein (MPV) level B-mode sonographic picture shows how to determine the maximum (Vmax) and lowest (Vmin) velocity from the spectral waveform, with color Doppler and spectral Doppler areas of interest (ROIs) overlay. Despite being high, the calculated VPI of 0.61 is unlikely to indicate NAFLD. B) A male patient aged 59 with non-alcoholic fatty liver disease (NAFLD) at stage F4 of fibrosis. There is little temporal fluctuation in the spectral Doppler waveform recorded in MPV. An estimated VPI of 0.06 indicates a high risk of NAFLD.

B. Assessing hepatic veins with Doppler in non-alcoholic fatty liver disease (NAFLD)

A healthy person's hepatic veins have three peaks: antegrade diastolic flow, antegrade systolic flow, and a transient retrograde flow during right atrial systole. The flow pattern is influenced by factors such as right atrial pressure, the compliance of the hepatic parenchyma, and the pressures within the thoracic and abdominal cavities that change with breathing. Hepatic veins exhibiting biphasic or monophasic patterns waveforms are connected to cirrhosis, fibrosis, hepatitis, transplant rejection, hepatic veno-occlusive disease, Budd-Chiari syndrome, and obesity.

Abnormal HV Doppler waveforms and obesity livers have been studied seldom. Small patient cohorts are used to compare outcomes with parenchymal liver diseases. Over the last decade, the HV Doppler waveform has been studied in connection to chronic parenchymal liver disease. A enlarged liver increases arterial blood flow and decreases portal venous flow. Hypertrophied hepatocytes may compress the hepatic vein, lowering venous blood flow phasicity and altering waveforms, causing aberrant blood flow in steatotic livers. Fatty liver patients may benefit from a Doppler and regular ultrasonography to monitor fat accumulation and hepatic perfusion.[11] Hepatic venous flow patterns and Doppler indices may vary with considerable liver adipose infiltration, according to several studies.

C. Index of Hepatic Artery Resistivity

Before transient elastography (TE) was used in clinical practice, a number of studies indicated that the assessment of certain hemodynamic parameters obtained from Doppler ultrasonography of the hepatic arteries might indirectly represent histological abnormalities, such as liver fibrosis. It has been shown that cirrhosis raises the-resistance index in the splenic-artery (SARI) and the hepatic-artery (HARI), in contrast to chronic viral hepatitis.[41] The hepatic artery resistive index (HARI) is used to evaluate microcirculatory resistance in individuals with chronic hepatitis, alcoholic liver disease in adults, liver disease following transplantation, and liver conditions associated with obesity. In contrast to their slim counterparts, obese people with fatty livers had significantly higher levels of ALT, TG, TC, and HARI, according to study by Hizli et al.[42] An increase in HARI level was shown to be significantly correlated with an increase in BMI. In order to detect insulin-resistant hepatic arterial perfusion abnormalities early on, they recommend using HARI as the main treatment. An Italian research found a strong negative-association between HARI and the severity of diffuse fatty liver disease in individuals with NAFLD.[43] This implies that when the severity of fatty liver disease rises, HARI falls. Nearly all of these results are in agreement with previously published data.[44]

Additionally, the degree of fibrosis as determined by the NAFLD fibrosis score has shown a substantial positive link with the assessment of HARI. Accordingly, the build-up of fibrous tissue may cause arterial stiffness to rise, which in turn may raise flow resistance. On the other hand, HARI may be impacted in different ways by the liver's varied tissue makeup (adipose vs fibrous).[45] The gray US index (liver echogenicity) may stay the same or decline more slowly than the RI index in some individuals who responded to treatment. This highlights the critical need for early diagnosis and the identification of hepatic artery flow anomalies in the NAFLD group. More accurate techniques for assessing the development of patients with fat livers include Doppler ultrasonography and HARI.

PREVIOUS SIMILAR STUDIES

1. The impact of hepatic lipid infiltration on the portal vein's Doppler flow hemodynamics was examined by Erdogmus et al. (2008).[46] Sixty NAFLD patients and twenty healthy people (control group) underwent liver and portal vein Doppler sonography. Based on the sonographic features of hepatosteatosis, patients were divided into three groups: mild (grade 1), moderate (grade 2), and severe (grade 3). Each cohort consisted of twenty individuals. The portal vein pulsatility index (VPI), mean flow velocity (MFV), peak maximum velocity (Vmax), and peak minimum velocity (Vmin), were lower in NAFLD patients-than in the-controls ($p < 0.001$). The victims' VPI was 0.20”, while the control group's was 0.31. The patients MFV was 12.3 cm/sec, while the control group's was 16.5 cm/sec. As portal vein

flow dropped, the degree of lipid infiltration rose for both MFV ($r = -0.951$, $p < 0.001$) and VPI ($r = -0.946$, $p < 0.001$).

2. In patients with nonalcoholic fatty liver disease, Mohammadinia et al. (2010) evaluated the impact of varying levels of fatty infiltration on the hepatic vein waveform patterns and the hepatic artery resistance index.[47] In group B, there were two (10%) monophasic and biphasic hepatic vein waveforms; in group C, there were eleven (55%) and sixteen (80%); and in group A, there were none. There were significant differences ($p < 0.001$) in the distribution of the triphasic Doppler waveform pattern between the patient cohort and the control group. There were statistically significant differences ($p < 0.001$) in the hepatic artery resistance indices of Groups A, B, C, and D, which were 0.81, 0.78, 0.73, and 0.68, respectively.
3. Ulusan et al. (2011) investigated the correlation between portal venous velocity and hepatic-abdominal fat in NAFLD patients using MRI and spectral Doppler ultrasonography.[48] This prospective study used portal Doppler ultrasonography on 35 NAFLD patients and 29 healthy controls. There was a substantial, There is a significant difference in portal venous velocity between patients with NAFLD and healthy controls ($p < 0.0001$). However, "abdominal or hepatic fat did not affect portal vein velocity ($p > 0.05$). Significant associations were seen between hepatic fat fraction and subcutaneous adiposity ($p < 0.0001$), intraperitoneal fat accumulation ($p = 0.017$), and retroperitoneal fat accumulation ($p < 0.0001$)."
4. Solhjoo et al. (2011) studied the relationship between portal vein Doppler indices and hepatic vein waveform patterns in NAFLD.[49] In this case-control study, 31 healthy persons and 31 NAFLD patients were controls. Based on B-mode ultrasonography, the patients exhibited hyperechoic livers and elevated ALT/AST values. Eleven patients underwent liver biopsies. After an eight-hour fast, B-mode and duplex Doppler were done. Patients with NAFLD had VPI and MFV values of 0.25 and 12.82 cm/second ($P < 0.01$), while the control group had values of 0.42 and 17.27 cm/second". NAFLD patients also exhibited

significantly higher rates of abnormal hepatic vein Doppler waveform patterns (55.2%) compared to controls (3.2%; P < 0.001). No correlation was seen between adipose infiltration and hepatic vein waveform pattern, MFV, or VPI (P = 0.197, 0.911, and 0.714). No correlation was discovered between MFV or VPI and liver enzymes. More aberrant hepatic capillaries were seen in those with enzyme levels above twice the normal level (P = 0.05).

5. Balasubramanian et al. (2016) examined how hemodynamic changes affect NAFLD severity.[50] Patients with NAFLD showed mean portal vein Vmax, Vmin, MFV, and VPI values of 12.23, 9.31, 10.76, and 0.24 cm/sec. The control group achieved 0.3, 10.01, 12.23, and 14.05 cm/sec equivalent values. All changes were statistically significant except for Vmin. Patients with hepatic steatosis had an average HARI of 0.65 (p=0.001), while the control group had a mean of 0.75. The severity of NAFLD showed a negative correlation with HARI (r = -0.517), VPI (r = -0.44), and Vmax (r = -0.293).. MFV correlated negatively with NAFLD severity (r=-0.182).
6. Sehgal et al. (2017) use Doppler sonography to assess NAFLD portal vein hemodynamics. A Mylab-40 ultrasound equipment with a low-frequency, 3-5 MHz convex transducer was used to examine 100 fasting subjects for 4-6 hours by experienced abdominal sonography and Doppler radiologists. The SSG Hospital, Vadodara Department of Radiodiagnosis performed this test as part of a prospective case-control outpatient study from April to November 2016. Participants were divided into test patients (64 with varied hepatic fatty infiltration) and controls (36 healthy). Grayscale liver lipid infiltration images divided the test group into three categories. The control group had an MFV of 16.8 cm/second and a VPI of 0.32. The grade 1 fatty infiltration group had values of 0.27 and 14.2 cm/second, whereas the grade 2 group had 0.22 and 12.2 cm/second, and the grade 3 group had 0.18 and 10.8 cm/second. Both MFV (f - 43.9, p <0.001) and VPI (f - 55.3, p <0.001) show a negative correlation with lipid infiltration.

7. Investigating hemodynamic variations in the portal vein and hepatic arteries of non-alcoholic fatty liver and establishing a relationship between these variations and the severity of the illness were the goals of Sabry et al. (2021).[51] In this case-control research, the 80 participants were split into four groups: patients with non-alcoholic fatty liver disease in grades 1, 2, and 3, healthy volunteers, and healthy volunteers. Doppler studies showed that patients with non-alcoholic fatty liver disease had significantly lower mean flow velocity, portal vein pulsatility index, hepatic artery resistivity index, peak maximum velocity, and peak minimum velocity compared to healthy persons. The severity of the condition was indirectly associated with other indices, except from the peak minimum velocity, which was significantly lower in grade 3 patients compared to grade 1 and 2 patients.
8. In the prospective cross-sectional study conducted by Basnet et al. (2022) over-the period of one-year from September 2020 to August 2021, a total of 70 participants aged between 30 and 60 years were assessed to investigate the association between fatty liver disease and portal vein hemodynamics.[52] All ultrasonographic evaluations were carried out using the Toshiba Aplio 500 equipped with a 3.5 MHz convex array deep probe, ensuring consistent imaging quality. Out of the total cohort, 50 participants were diagnosed with fatty liver disease based on ultrasonographic findings, while 20 exhibited normal liver parenchyma and served as the control group. The patients with fatty liver demonstrated a significantly higher mean BMI of 30.6, as compared to 23.55 in the control group, indicating a strong correlation between obesity and hepatic steatosis. Hemodynamically, those with fatty liver showed a considerable reduction in portal vein velocities, with a mean minimum velocity (V_{min}) of 20.0 cm/s and maximum velocity (V_{max}) of 24.6 cm/s. In contrast, individuals in the control group had significantly higher values, with a mean V_{min} of 30.05 cm/s and V_{max} of 34 cm/s. These differences in both V_{min} and V_{max} were statistically significant ($p < 0.001$), suggesting that fatty infiltration of the liver leads to measurable reductions in portal venous flow. Furthermore, although the mean age of participants with fatty liver (42.1 years)

was slightly higher than that of the control group (40.6 years), this difference was less pronounced compared to the disparity in BMI and portal vein velocities. Overall, the findings highlight a clear association between increased BMI, fatty liver disease, and reduced portal venous flow, thereby reinforcing the role of obesity-related hepatic steatosis in altering intrahepatic hemodynamics.

9. In 2023, Safeeq et al. assessed the use of splenoportal Doppler ultrasonography in NAFLD patients.[53] There was just one hospital and one center where this cross-sectional research was carried out. Patients who satisfied the inclusion requirements provided the data. NAFLD will be diagnosed by the ultrasonographic examination and clinical evaluation. Doppler Ultrasonography was then used to assess the selected patient's abdomen, which was assessed based on a number of parameters. As fatty liver disease worsened, the hepatic artery resistive index and pulsatility index dropped, while the portal vein velocity and its pulsatility index dropped considerably. Additionally, hepatic vein phasicity reduced while liver size and portal vein diameter rose. In addition to assessing hemodynamic alterations in the portal circulation, it has been shown that early portal hypertension and hepatic fibrosis may be predicted.

10. Verma et al. (2023) compared patients with mild to severe non-alcoholic fatty liver disease with healthy controls to evaluate the segmental differences in portal venous pulsed wave color Doppler flow velocity.[54] In this prospective, observational case-control study, color Doppler was employed to assess the peak velocity of each segmental branch of the portal vein in patients with mild to severe non-alcoholic fatty liver disease.. Three cohorts the non-alcoholic fatty liver disease group (n = 32), the non-alcoholic steatohepatitis-portal hypertension group” (n = 13), and the healthy controls (n = 30) were used to examine the data. The cohort with non-alcoholic fatty liver disease showed reduced velocity in all eight liver areas when compared to the control group. The non-alcoholic fatty liver disease cohort showed a substantially higher “ratio of segment 2 to segment 7 peak portal vein maximum

velocity" (1.03) than the control group (0.90). However, with a p-value of 0.003, the non-alcoholic steatohepatitis-portal hypertension group showed an even higher ratio (1.83).

MATERIAL & METHODS

4. MATERIAL AND METHODS

4.1 STUDY DESIGN

It was a “cross-sectional comparative study”.

4.2 STUDY DURATION

It was 18 months (June 2023 till Dec 2024)

4.3 STUDY SITE

Department of Radio-Diagnosis in R.L. Jalappa Hospital and Research Center attached to SDUMC, Kolar

4.4 STUDY POPULATION

Subjects undergoing abdomen color doppler ultrasound.

4.5 INCLUSION CRITERIA

1. Adults (aged 18 years more more) males and females.
2. Cases: those diagnosed with NAFLD on the basis of metabolic syndrome.
3. Healthy controls: age and gender matched subjects undergoing ultrasound abdomen for a cause unrelated to fatty liver disease.

4.6 EXCLUSION CRITERIA

1. Those consuming > 20 mg alcohol per day.
2. Those diagnosed with viral liver disease.

4.7 SAMPLE SIZE AND SAMPLING TECHNIQUE

“The sample size was calculated using following formula”:

“$n= (Z\alpha/2 + Z\beta)^2 * (SD * 2) / d^2$”

n- Sample size

$Z\alpha/2$ – “Z value at 5% error (1.96)”

$Z\beta$ – “Z value at 10% (1.28)”

SD- "average standard deviation of the character" = (SD1+SD2)/ 2

d – "clinically relevant effect" (taken as 0.2)

Difference in Mean Hepatic artery resistivity index between controls and cases from the study Balasubramanian et al was 0.75 ± 0.06 and 0.65 ± 0.06.

SD – 0.06

$$n = \frac{(1.96+1.28)^2 * (0.06 * 2)}{(0.2)^2}$$

n- 31.5 (minimum sample size)

During the study period, consecutive subjects fulfilling the study criteria were included in the study as follows

NAFLD group = 44 patients

Non-NAFLD group = 36 subjects

4.8 COLOR DOPPLER IMAGING ULTRASOUND PROTOCOL

- The Philips EPIQ 5 ultrasound instrument, which has a curvilinear broadband transducer C5-1MHz, was used for all tests in this study.
- A recumbent posture was adopted for the patient. On an initial premise, traditional grayscale sonography was used.
- The patients' liver sonographic results were categorized as either having coarse echotexture, lipid infiltration, or normal echotexture.
- The transducer was positioned using a para-median or slightly oblique plan along the longitudinal axis of the principal portal vein in order to measure the portal vein. The measuring site was located halfway between the bifurcation of the portal vein during calm inspiration and the junction of the splenic and superior mesenteric veins.

- The hepatic artery parameters were measured at the porta hepatis level while the patient was in the supine posture.
- Because of its dual supply to the hepatic arteries and its visibility at the portal triad, the major hepatic artery was selected for study. During suspended respiration, the patient's hepatic artery resistance index, peak systolic velocity (PSV), and acceleration time (AT) were recorded.

4.9 DATA COLLECTION

A semi-structured research proforma that had been previously created was used to gather data. The following data was acquired:

- Demographics: age and sex
- Clinical information includes the patient's personal history, medical history, and results of a localized physical examination.
- The hepatic ultrasonography examination's findings:
  - The size, shape, and texture of the liver
  - Measurements of the spleen in centimeters
- Doppler results for the porto-venous system's hepatic artery and portal vein:
- Millimeters are used to measure the diameter.
- Average speed (Vm) (cm/s)
- Spectral Doppler ultrasonography is used to measure peak systolic velocity (PSV). A measurement in centimeters per second (cm/s) is used. Each observable peak in a Doppler waveform's spectral window represents the peak systolic velocity.
- Blood flow velocity during diastole is measured using the end-diastolic velocity (EDV) spectral Doppler ultrasonography metric, which is measured in centimeters per second (cm/s). On a Doppler waveform, the point at the conclusion of the cardiac cycle, which comes just before the peak of systole, is the end-diastolic volume (EDV).

- A hemodynamic statistic used in ultrasonography, the pulsatility index (PI), often referred to as the Gosling index, is computed using the mean, minimum, and maximum Doppler frequency shifts. Typically, the ultrasound machine calculated the mean velocity (vmean) while the operator recorded the maximum (vmax) and lowest (vmin) speeds. The mean velocity is divided by the difference between the peak systolic and lowest diastolic velocities to get the PI.
- One flow metric used in ultrasonic measurements is the resistance index (RI), also referred to as the Pourcelot index. It is calculated using the mean, minimum, and maximum Doppler frequency fluctuations.
- Respiratory phasicity

4.9 STATISTICAL ANALYSIS

Patient profiles that were created using a range of clinical, laboratory, and demographic factors were included in the research. “The mean and standard deviation were used to describe the quantitative parameters. Ordinal data were represented using absolute values and percentages. Cross tables were created in order to examine the relationships between NAFLD patients and non-NAFLD persons using the chi-square test. The Student's t-test was used to compare quantitative data. A P-value of less than 0.05 indicates statistical significance. All analyses were performed using SPSS software, version 24.0.

4.10 STATEMENT OF ETHICS

The Institutional Ethics Committee authorized the research process before it started, and it complies with the Declaration of Helsinki. Every patient completed an informed consent form. It is not meant to do the subjects any harm. Before giving their permission, participants were briefed on the study's methodology. The inquiry did not result in any extra costs for the participants.

RESULTS

5. RESULTS

Table 1. Comparison of gender distribution between case (NAFLD) and controls (no NAFLD)

Gender		Study groups Case (NAFLD)	Control (no NAFLD)	Total
Female	N	18	15	33
	%	41%	42%	41%
Male	N	26	21	47
	%	59%	58%	59%
Total	N	44	36	80
	%	100%	100%	100%
		"p value* = 0.41"		

"""*analyzed using chi-square test"""

Overall, 59% were males and rest 41% were females. The gender distribution is similar between the two groups with no significant difference (p = 0.41). Among the NAFLD cases, 18 were female (41%) and 26 were male (59%), while in the control group, 15 were female (42%) and 21 were male (58%). The total cohort included 33 females (41%) and 45 males (56%).

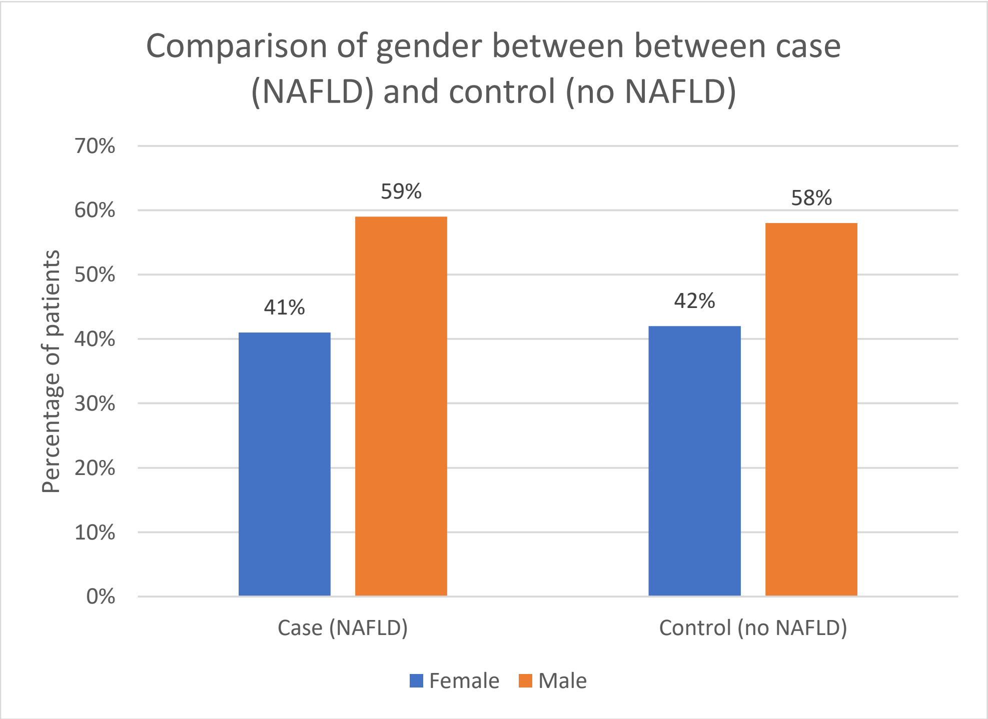


Table 2. Comparison of liver echotexture between case (NAFLD) and controls (no NAFLD)

Liver echotexture		**Study groups** **Case (NAFLD)**	**Control (no NAFLD)**	**Total**
Coarse	N	41	1	42
	%	93%	3%	53%
Normal	N	3	35	38
	%	7%	97%	47%
Total	N	44	36	80
	%	100%	100%	100%
		“p value* < 0.01”		

“*analyzed using chi-square test”

A vast majority of NAFLD cases (93%, n=41) had coarse liver echotexture, while only 7% (n=3) showed a normal texture. In contrast, nearly all control subjects (97%, n=35) exhibited normal liver echotexture, with only one individual (3%) displaying a coarse texture.

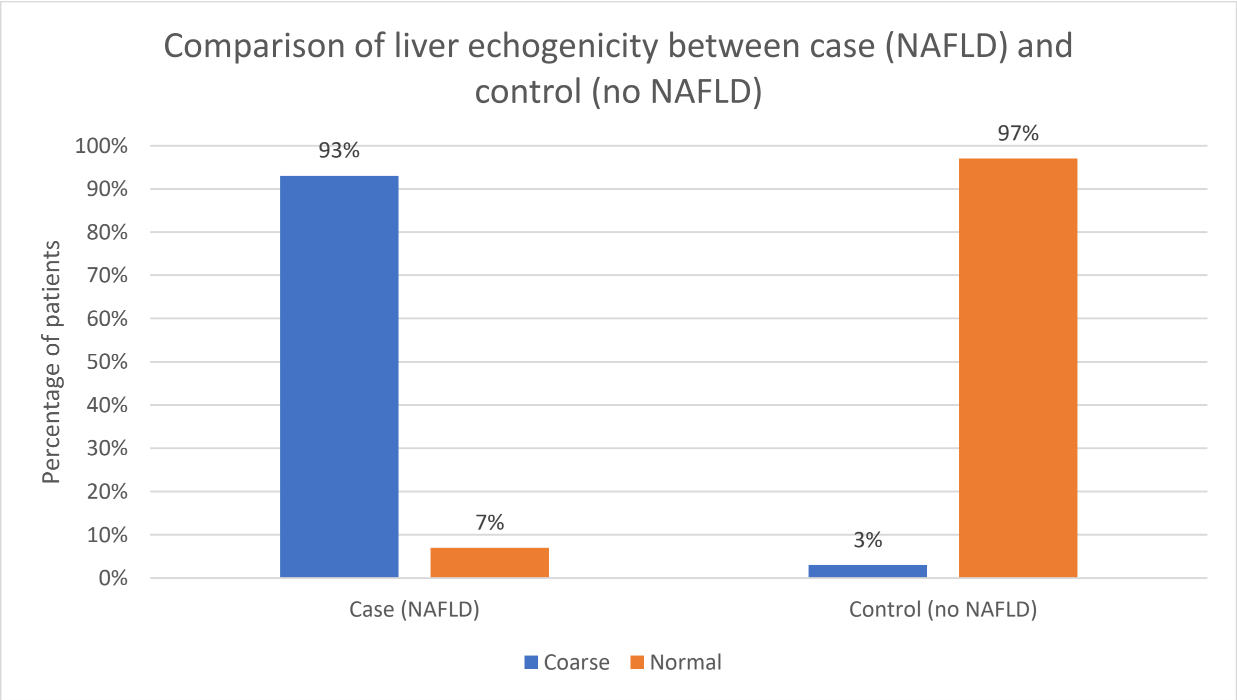


Table 3. Comparison of hepatomegaly between case (NAFLD) and controls (no NAFLD)

Hepatomegaly		Study groups		
		Case (NAFLD)	**Control (no NAFLD)**	**Total**
Normal	N	25	36	61
	%	57%	100%	76%
Increased	N	19	0	19
	%	43%	0%	24%
Total	N	44	36	80
	%	100%	100%	100%
		"p value* < 0.01"		

"*analyzed using chi-square test"

Hepatomegaly was present in a large portion of NAFLD patients but absent in controls. This significant difference ($p < 0.01$) highlights liver enlargement as a feature of NAFLD.

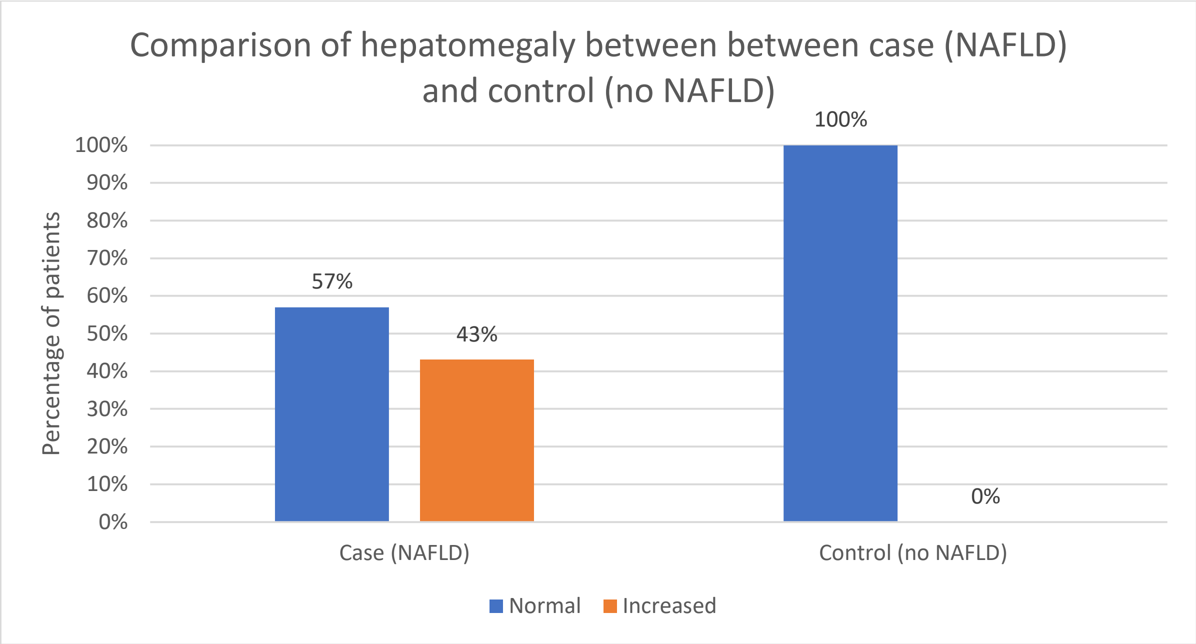


Table 4. Comparison of hepatic artery PSV between case (NAFLD) and controls (no NAFLD)

Hepatic artery PSV (cm/s)		Study groups: Case (NAFLD)	Control (no NAFLD)	Total
Decreased (<60)	N	44	0	44
	%	100 %	0 %	55 %
Normal (60 to 80)	N	0	30	30
	%	0 %	83 %	38 %
Raised (>80)	N	0	6	6
	%	0 %	17 %	8 %
Total	N	44	36	80
	%	100 %	100 %	100 %
		"p value* < 0.01"		
Mean HA PSV (cm/s)		43.1 ± 7.1	72.6 ± 6.6	
		"p value** < 0.01"		

"*analyzed using chi-square test; **analyzed using Mann Whitney test"

All NAFLD patients (100%, n=44) exhibited decreased hepatic artery PSV values (<60 cm/s), while none of the controls did. In the control group, 30 subjects (83%) had normal PSV values (60–80 cm/s), and the remaining 6 (17%) had elevated values (>80 cm/s). The mean PSV was significantly lower in NAFLD patients (43.1 ± 7.1 cm/s) compared to controls (72.6 ± 6.6 cm/s), with a p-value < 0.01.

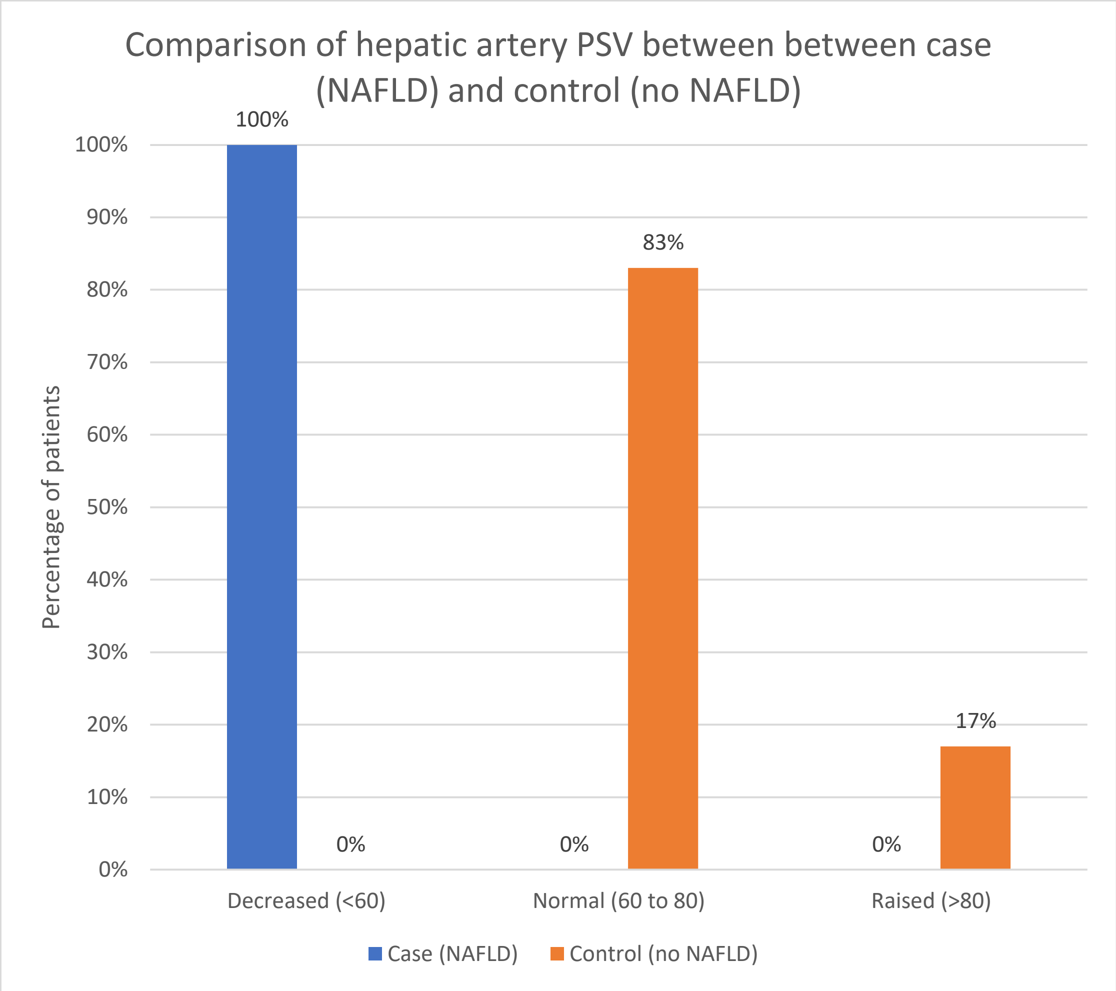

Comparison of hepatic artery PSV between between case (NAFLD) and control (no NAFLD)
Percentage of patients
100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
100%
0%
0%
83%
0%
17%
Decreased (<60)
Normal (60 to 80)
Raised (>80)
Case (NAFLD)
Control (no NAFLD)

Table 5. Comparison of hepatic artery RI between case (NAFLD) and controls (no NAFLD)

Hepatic artery RI		Study groups Case (NAFLD)	Control (no NAFLD)	Total
Decreased (<0.55)	N	30	0	30
	%	68 %	0 %	38 %
Normal (0.55 to 0.75)	N	14	31	45
	%	32 %	86 %	56 %
Raised (>0.75)	N	0	5	5
	%	0 %	14 %	6 %
Total	N	44	36	80
	%	100 %	100 %	100 %
		"p value* < 0.01"		
Mean hepatic artery RI		0.5 ± 0.1	0.7 ± 0.05	
		"p value** < 0.01"		

"*analyzed using chi-square test; **analyzed using Mann Whitney test"

A decreased RI (<0.55) was found in 68% (n=30) of NAFLD cases, while none of the controls had a low RI. Most control participants (86%, n=31) had normal RI values (0.55–0.75), and 14% (n=5) showed elevated RI (>0.75). Among the NAFLD group, 32% (n=14) had normal RI, and none had elevated values. The mean RI was significantly lower in NAFLD patients (0.5 ± 0.1) than in controls (0.7 ± 0.05), with a highly significant p-value (< 0.01).

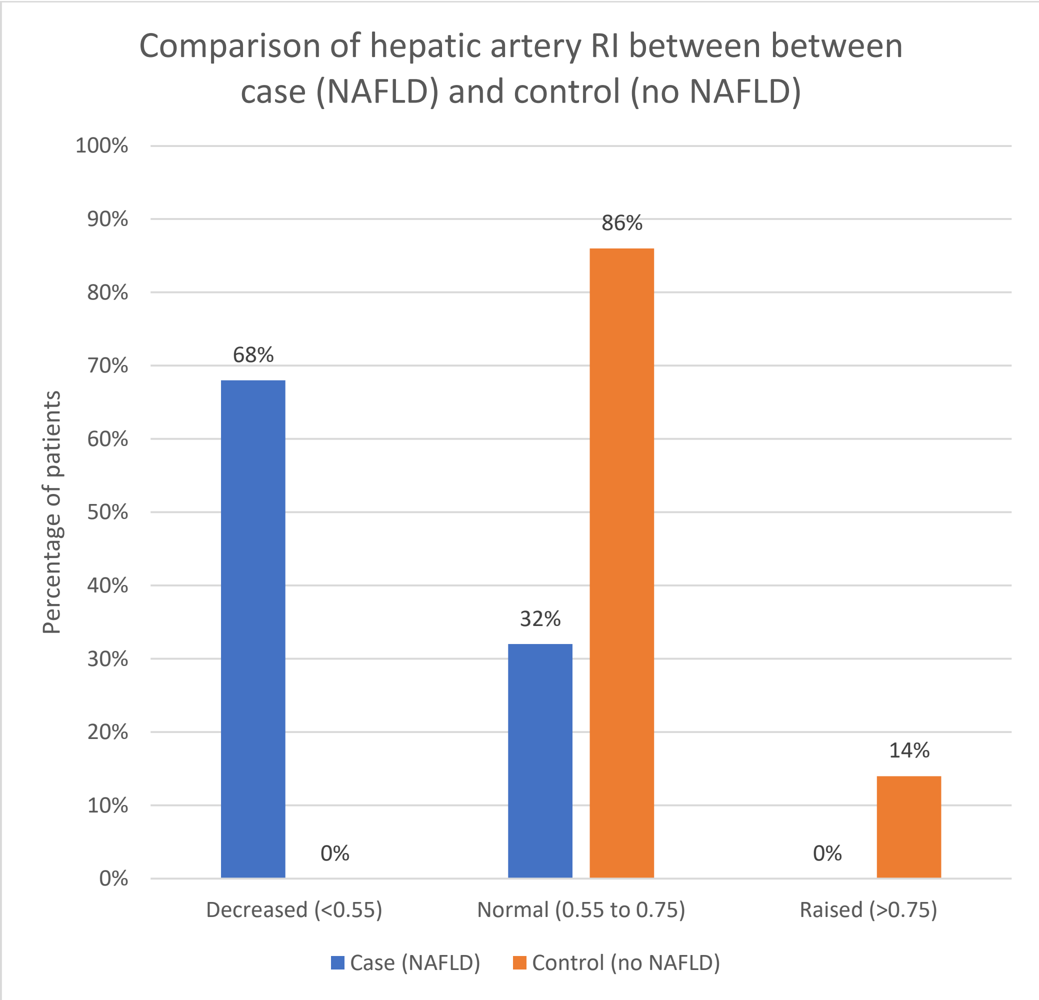
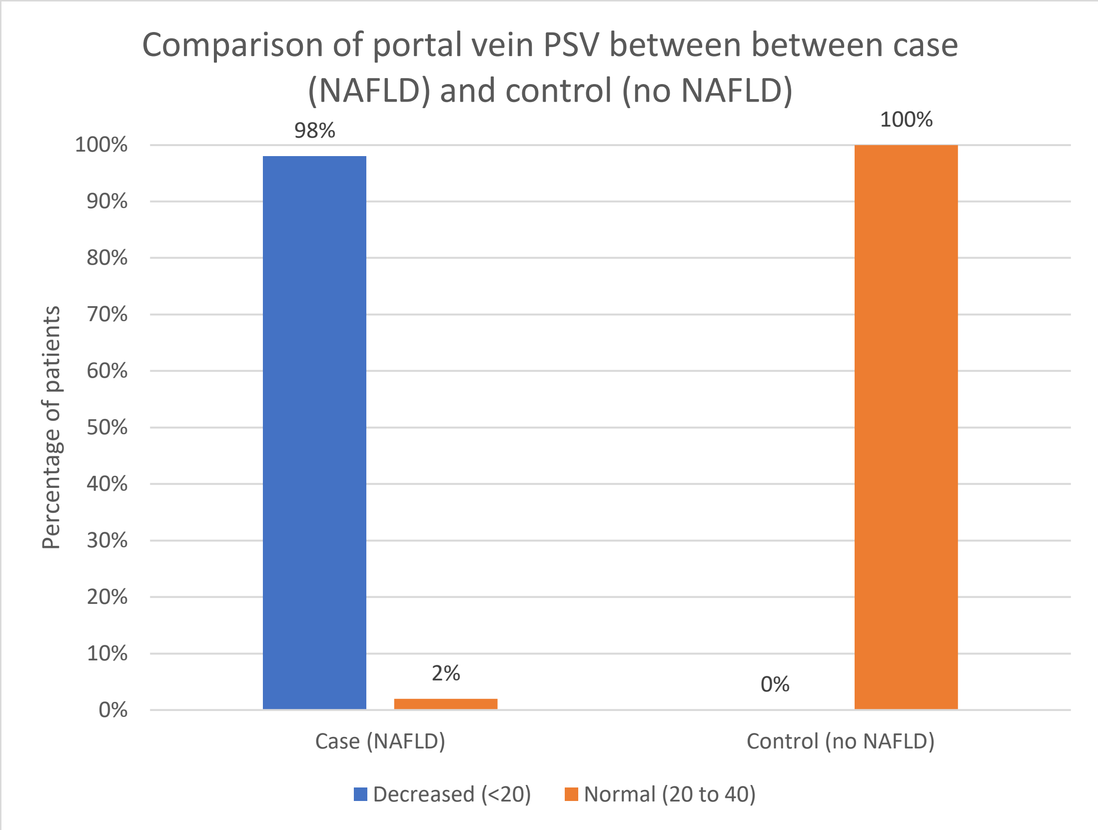

Comparison of hepatic artery RI between between case (NAFLD) and control (no NAFLD)
Percentage of patients
100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
68%
0%
32%
86%
0%
14%
Decreased (<0.55)
Normal (0.55 to 0.75)
Raised (>0.75)
Case (NAFLD)
Control (no NAFLD)

Table 6. Comparison of portal vein PSV between case (NAFLD) and controls (no NAFLD)

Portal vein PSV (cm/s)		**Study groups**		
		Case (NAFLD)	**Control (no NAFLD)**	**Total**
Decreased (<20)	N	43	0	43
	%	98 %	0 %	54 %
Normal (20 to 40)	N	1	36	37
	%	2 %	100 %	46 %
Total	N	44	36	80
	%	100 %	100 %	100 %
		"p value* <0.01"		
Mean portal vein PSV (cm/s)		18.2 ± 2.1	25.7 ± 3.6	
		"p value** < 0.01"		

"*analyzed using chi-square test; **analyzed using Mann Whitney test"

In NAFLD patients, 98% (n=43) had decreased portal vein velocities (<20 cm/s), while only one case (2%) had a normal velocity. All control participants (100%) had normal portal vein PSV (20–40 cm/s). The mean portal vein PSV was markedly lower in the NAFLD group (18.2 ± 2.1 cm/s) compared to controls (25.7 ± 3.6 cm/s), with a p-value < 0.01.



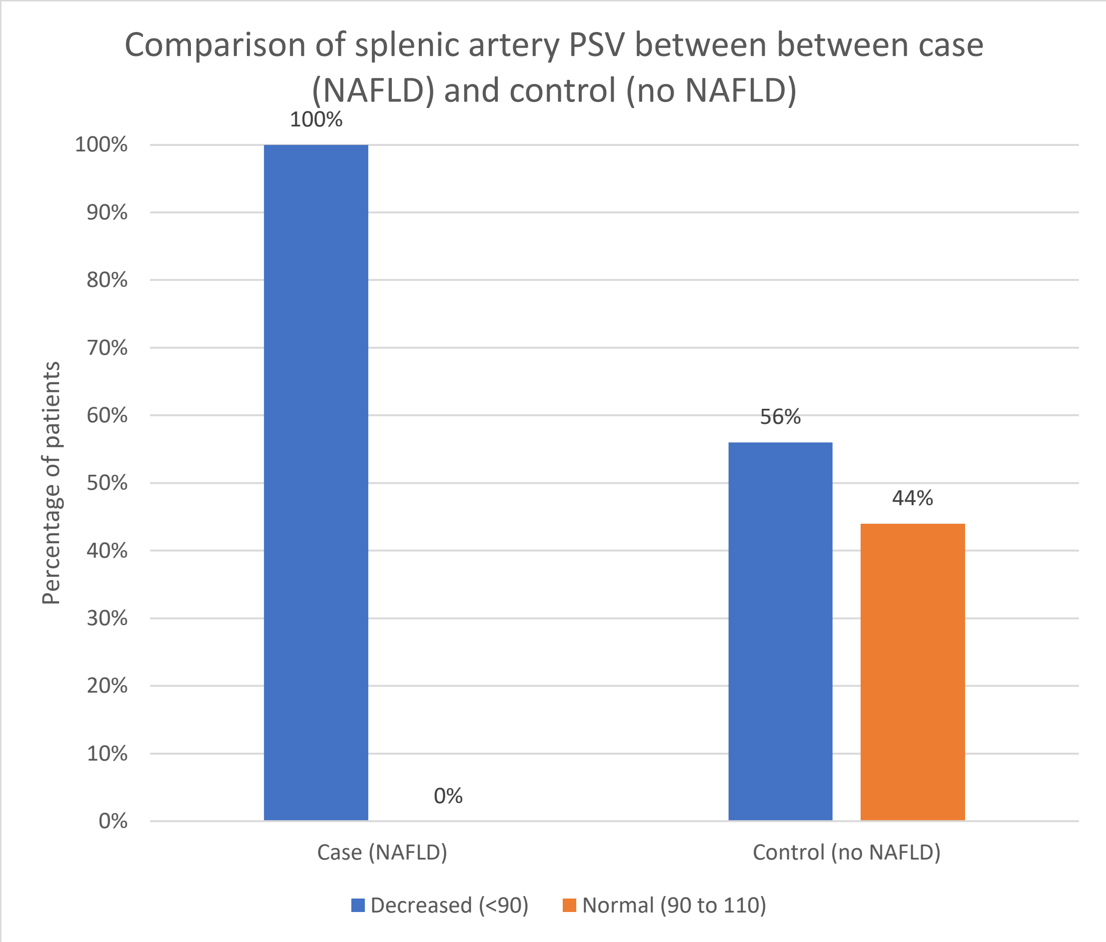
Comparison of portal vein PSV between between case (NAFLD) and control (no NAFLD)
Percentage of patients
100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
98%
2%
0%
100%
Case (NAFLD)
Control (no NAFLD)
Decreased (<20)
Normal (20 to 40)

Table 7. Comparison of splenic vein PSV between case (NAFLD) and controls (no NAFLD)

		Study groups		
Splenic artery PSV (cm/s)		**Case (NAFLD)**	**Control (no NAFLD)**	**Total**
Decreased (<90)	N	44	20	64
	%	100 %	56 %	80 %
Normal (90 to 110)	N	0	16	16
	%	0 %	44 %	20 %
Total	N	44	36	80
	%	100 %	100 %	100 %
		"p value* < 0.01"		
Mean splenic artery PSV (cm/s)		50.2 ± 8.5	88.1 ± 9.3	
		"p value** < 0.01"		

"*analyzed using chi-square test; **analyzed using Mann Whitney test"

All NAFLD patients (100%) had decreased splenic artery PSV values (<90 cm/s), whereas only 56% (n=20) of the controls had similar findings. The remaining 44% (n=16) of controls showed normal velocities (90–110 cm/s). In the NAFLD group, the mean splenic artery PSV (50.2 ± 8.5 cm/s) was considerably lower than that in the control group (88.1 ± 9.3 cm/s), with a p-value below 0.01.



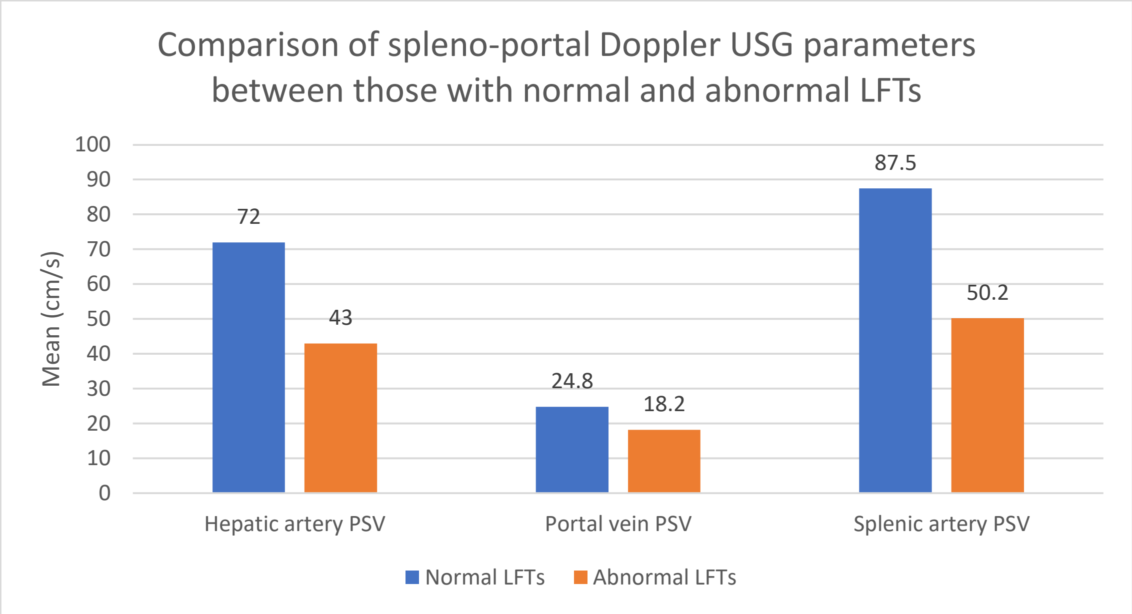
Comparison of splenic artery PSV between between case (NAFLD) and control (no NAFLD)
Percentage of patients
100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
100%
0%
56%
44%
Case (NAFLD)
Control (no NAFLD)
Decreased (<90)
Normal (90 to 110)

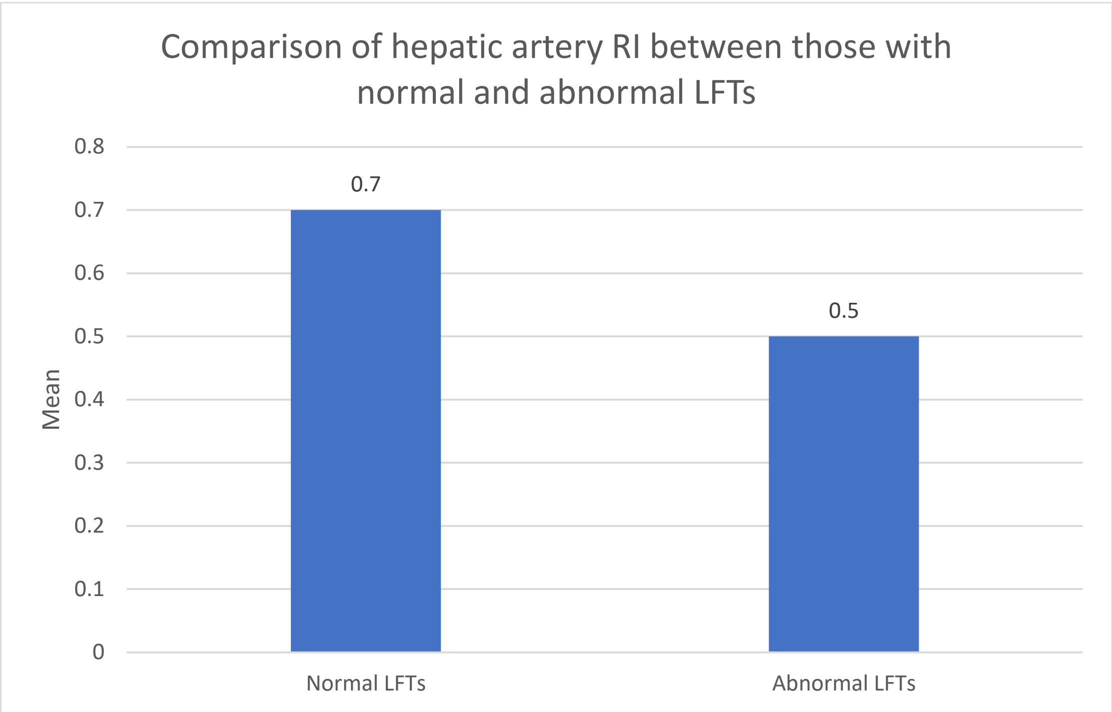
Table 8. Comparison of spleno-portal doppler parameters between patients with normal and abnormal LFTs

	LFTs	N	Mean	SD	p value*
Hepatic artery PSV	Abnormal	42	43.0	7.7	< 0.01
	Normal	38	72.0	8.0	
Hepatic artery RI	Abnormal	42	0.5	0.1	< 0.01
	Normal	38	0.7	0.1	
Portal vein PSV	Abnormal	42	18.2	2.6	< 0.01
	Normal	38	24.8	4.2	
Splenic artery PSV	Abnormal	42	50.2	9.4	< 0.01
	Normal	38	87.5	12.4	

“*analyzed using Mann Whitney test”

This table evaluates the relationship between liver function test (LFT) abnormalities and various Doppler parameters. Among individuals with abnormal LFTs (n=42), the mean hepatic artery PSV was significantly lower (43.0 ± 7.7 cm/s) compared to those with normal LFTs (72.0 ± 8.0 cm/s). Similarly, hepatic artery RI was lower in the abnormal LFT group (0.5 ± 0.1) than in the normal LFT group (0.7 ± 0.1). Portal vein PSV and splenic artery PSV were also reduced in patients with abnormal LFTs (18.2 ± 2.6-cm / s and 50.2 ± 9.4 cm/s, respectively) compared to their counterparts with normal LFTs (24.8 ± 4.2 cm / s and 87.5 ± 12.4 cm / s, respectively). All comparisons yielded p-values < 0.01, indicating strong statistical significance.


Comparison of spleno-portal Doppler USG parameters between those with normal and abnormal LFTs
Mean (cm/s)
100
90
80
70
60
50
40
30
20
10
0
72
43
24.8
18.2
87.5
50.2
Hepatic artery PSV
Portal vein PSV
Splenic artery PSV
Normal LFTs
Abnormal LFTs


Comparison of hepatic artery RI between those with normal and abnormal LFTs
Mean
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0.7
0.5
Normal LFTs
Abnormal LFTs

DISCUSSION

6. DISCUSSION

The Department of Radio-Diagnosis at R.L. Jalappa Hospital and Research Center carried out this cross-sectional comparative investigation in Kolar, connected to SDUMC. Assessing the effectiveness of spleno-portal system Doppler ultrasonography in identifying early hepatic dysfunction in individuals with non-alcoholic fatty liver disease was the aim of the research. Doppler ultrasonography of the spleno-portal system was performed on 36 non-NAFLD patients and 44 NAFLD patients. This is a discussion of the current investigation's findings.

Features of ultrasound of the liver

In the present study, 93% (n=41) of NAFLD patients exhibited a coarse liver echotexture, compared to only 7% (n=3) who had a normal liver echotexture. In contrast, almost every control person (97%, n=35) had a normal liver echotexture. Hepatomegaly was seen in a significant percentage of NAFLD patients but not in the control group.

According to Safeeq et al., there is a statistically significant rise in liver size that corresponds with the advancement of the illness and is linked to higher echogenicity in later stages of nonalcoholic fatty liver disease.

Although Basnet et al. evaluated liver parenchymal echogenicity using ultrasonography, they did not give priority to echotexture statistics. They used ultrasound grading (Grades 1–3) based on echogenicity to assess the degree of fatty liver.

In their diagnostic approach, Sabry et al. used echogenicity in addition to transient elastography and the CAP score for determining the severity of NAFLD. They admitted that hepatic echogenicity changed as grades increased.

Sehgal et al. used grayscale echogenicity in relation to the renal cortex to classify the degree of steatosis. Based on the finding that increasing echogenicity was associated with deteriorating fatty infiltration, patients were divided into three groups.

For categorization, Verma et al. combined structural alterations with echogenicity, such as surface abnormalities and hidden intrahepatic vasculature. They emphasized the left lobe's growth and the right lobe's atrophy as markers of the progression of the illness.

Balasubramanian et al. used echogenicity-based grading (Grades 1–3) to support our findings of hepatomegaly and discovered that liver span increased considerably with the severity of NAFLD. They warned that a shorter liver span in Grade 3 might indicate early cirrhosis, a little but important detail that was missed in our study. There was no discernible difference in group diameter, even though Solhjoo et al. utilized a similar echogenicity-based grading system and included portal vein diameter. They found no association between portal vein indices and the degree of steatosis, which is contrary to our results.

Ulusan et al. used MRI and ultrasonography to establish that individuals with NAFLD had increased hepatic fat. They saw hyperechogenic livers on ultrasonography with NAFLD, which is in line with our results, even if they did not highlight volume alterations.

The biopsy-based grading system developed by Mohammadinia et al., which showed a rising liver span from control to severe NAFLD, was consistent with our hepatomegaly results.

Erdogmus et al. graded using ultrasonography echogenicity and found that increased hepatic echogenicity was associated with worsening fat infiltration. Liver span was not emphasized, although overall parenchymal brightness and vascular clarity were similar diagnostic criteria across investigations.

Hepatic arteries, PSV and RI

All NAFLD patients in our study (100%, n=44) showed reduced hepatic artery PSV values (<60 cm/s), whereas none of the controls did. Thirty respondents (83%) had normal PSV levels (60–80 cm/s), whereas six respondents (17%) in the control group had excessive values (>80 cm/s). With a p-value less than 0.01, the mean PSV of NAFLD patients was substantially lower (43.1 ± 7.1 cm/s) than that of controls (72.6 ± 6.6 cm/s). A reduced RI (<0.55) was seen in 68% (n=30) of NAFLD patients, whereas all controls had a normal RI. While 86% of the control group (n=31) had normal RI values (0.55–0.75), 14% (n=5) had excessive RI (>0.75). While 32% (n=14) of the NAFLD group had normal RI values, none of them were higher. Those with abnormal LFTs (n=42) had a substantially lower mean hepatic artery PSV (43.0 ± 7.7 cm/s) than those with normal LFTs (72.0 ± 8.0 cm/s). The hepatic artery RI was lower in the aberrant LFT group (0.5 ± 0.1) than in the normal LFT group (0.7 ± 0.1).

Additionally, Safeeq et al. observed that hepatic artery PSV and RI reduced as the severity of NAFLD increased. Their results reflected the trend seen in our investigation and confirmed the effectiveness of these markers for early identification.

Basnet et al. primarily examined portal vein characteristics rather than assessing the hepatic artery resistance index or peak systolic velocity.

Although Sabry et al. validated lower HARI values in NAFLD overall compared to controls (0.74 ± 0.04 vs. 0.82 ± 0.02), their more complex findings showed that HARI increased with the severity of NAFLD (from 0.70 in grade 3 to 0.79 in grade 1), which is contrary to the trend we saw in our study. Different measuring techniques or sample compositions might be the cause of the variation.

Rather of assessing hepatic artery characteristics directly, Sehgal et al. focused on portal vein hemodynamics.

Verma et al. found that those with NAFLD and portal hypertension had a substantially greater hepatic artery resistance index (RI) of 0.88 ± 0.11 than people with NAFLD alone (0.67 ± 0.11) and the control group (0.65 ± 0.04), which is in contradiction to our findings. This suggests that as the illness progresses, RI increases.

A strong agreement was reached by Balasubramanian et al., who discovered that HARI was inversely connected with NAFLD stage and that it dramatically declined as NAFLD severity increased (from 0.69 in grade 1 to 0.61 in grade 3). This validates RI as a severity measure and supports the direction of our investigation. Instead of measuring the hepatic artery resistance index (RI) directly, Solhjoo et al. observed elevated anomalous hepatic venous waveforms, which are often linked to portal hypertension and hence suggest altered hepatic hemodynamics.

Ulusan et al. did not assess peak systolic velocity or the hepatic artery resistance index; they only looked at portal vein flow.

Mohammadinia et al. observed that RI reduced as the severity of NAFLD increased, which clearly contradicted the results of our investigation.Their control group, however, had a higher RI (0.81) than those with fatty infiltration, which is consistent with Balasubramanian's findings but contradicts Verma and Sabry's findings, which show that RI increases with advanced disease.

Erdogmus et al. did not assess any hepatic artery characteristics; they only looked at portal flow indices.

Peak Systolic Velocity of the Portal Vein and Splenic Artery

While, in our study, 98% (n=43) of NAFLD patients exhibited reduced portal vein velocities (<20 cm/s), just one instance (2%) had a normal portal vein velocity. All control subjects (100%) had normal portal vein peak systolic velocities (20–40 cm/s). In comparison to the controls (25.7 ± 3.6 cm/s), the NAFLD group's mean portal vein PSV was significantly lower (18.2 ± 2.1 cm/s), with a p-value < 0.01. Compared to 100% of NAFLD patients, only 56%

(n=20) of the control group exhibited lower splenic artery PSV values (<90 cm/s). Normal velocities ranged from 90 to 110 cm/s for 16 controls, or 44% of the total. The mean peak systolic velocity (PSV) of the splenic artery was significantly lower in the NAFLD group (50.2 ± 8.5 cm/s) compared to the control group (88.1 ± 9.3 cm/s), with a p-value of less than 0.01. Additionally, individuals with abnormal liver function tests (LFTs) had lower portal vein and splenic artery PSVs (24.8 ± 4.2 cm/s and 87.5 ± 12.4 cm/s, respectively) than those with normal LFTs (18.2 ± 2.6 cm/s and 50.2 ± 9.4 cm/s, respectively).According to Safeeq et al., when NAFLD worsens, portal vein velocity, diameter, and pulsatility index (VPI) all significantly decline. They also suggested that the portal vein was wider, which was not a component that our research looked at.

According to Basnet et al., portal vein V_min and V_max were much lower in those with fatty livers. Their V_max was 24.6 ± 7.4 cm/s, which was somewhat higher than our group's mean PSV. This might be the result of a less severe illness or different assessment techniques.

According to Sabry et al., NAFLD dramatically decreased all portal vein parameters, with Vmax measuring 23.18 ± 3.49 cm/s and Vmin being 17.46 ± 1.45 cm/s. They demonstrated a constant decline in Vmax, Vmin, and VPI in tandem with the rise in NAFLD grade, which supported the results of our investigation.

Sehgal et al. verified that MFV and VPI gradually decreased at different phases of fatty infiltration. Compared to the control value of 16.8 ± 2.6 cm/s, the mean flow velocity (MFV) dropped to 10.8 ± 1.5 cm/s in instances with severe NAFLD.

Verma et al. discovered that segmental portal vein velocities were globally decreased in all liver segments in individuals with nonalcoholic fatty liver disease. Additionally, they saw portal flow redistribution, which is characterized by increased flow in the left lobe, particularly in those with portal hypertension

A significant decrease in portal vein Vmax, VPI, and MFV in NAFLD was confirmed by Balasubramanian et al. They found a significant negative connection (r = -0.44) between VPI and the degree of illness, which supported the results of our study.

NAFLD dramatically reduced both MFV and VPI, according to Solhjoo et al. (MFV: 12.82 vs. 17.27 cm/s; VPI: 0.25 vs. 0.42). They may not have been able to find a relationship between Doppler measures and steatosis grade because of the small sample size.

Ulusan et al. discovered that those with NAFLD had a considerably lower portal vein velocity (Vmax ~18 cm/s in NAFLD compared to ~43 cm/s in controls) using Doppler and MRI. This supports their diagnostic threshold (less than 20 cm/s) and is consistent with our study's portal vein PSV findings.

Rather than reporting portal vein peak systolic velocity, Mohammadinia et al. concentrated on hepatic vein waveform and hepatic artery resistance index. They did, however, find that hepatic perfusion patterns decreased as the severity of NAFLD increased.

In a thorough portal Doppler research, Erdogmus et al. showed that as NAFLD grade increased, there was a notable decrease in Vmax (from 34 to 23.5 cm/s), Vmin, MFV, and VPI. Their correlation values (e.g., Vmax r = -0.969) were significantly significant and reflected the tendencies in our analysis.

LIMITATIONS

7. LIMITATIONS

There are several limitations of the current study:

1. Based only on liver function tests and ultrasound results, NAFLD was diagnosed and advanced without histological confirmation.
2. Because the research was cross-sectional and observational, it was unable to determine causation or monitor changes in hemodynamics over time.
3. The significant operator dependence of Doppler ultrasonography may affect the repeatability of the findings of our investigation.

SUMMARY

8.SUMMARY

“The Department of Radio-Diagnosis at R.L. carried out this cross-sectional comparative investigation. In Kolar, Jalappa Hospital and Research Center” is connected to SDUMC. Assessing the effectiveness of spleno-portal system Doppler ultrasonography in identifying early hepatic impairment in individuals with “non-alcoholic fatty liver disease was the goal of the research”. Doppler ultrasonography of the spleno-portal system was performed on 36 non-NAFLD patients and 44 NAFLD patients.

93% (n=41) of NAFLD patients exhibited a coarse liver echotexture, compared to only 7% (n=3) who had a normal liver echotexture. In contrast, almost every control person (97%, n=35) had a normal liver echotexture. Hepatomegaly was seen in a significant percentage of NAFLD patients but not in the control group.

All NAFLD patients (100%, n=44) showed reduced hepatic artery PSV values (<60 cm/s), whereas none of the controls did. Thirty patients (83%) had normal PSV values (60–80 cm/s), whereas six patients (17%) in the control group had excessive values (>80 cm/s). With a p-value less than 0.01, the mean PSV of NAFLD patients (43.1 ± 7.1 cm/s) was much lower than that of controls (72.6 ± 6.6 cm/s).

While the RI was normal for all controls, 68% (n=30) of NAFLD patients showed a decreased RI (<0.55). Eighty-six percent (n=31) of the control group had normal RI values (0.55–0.75), whereas fourteen percent (n=5) had abnormal RI (>0.75). While 32% (n=14) of the NAFLD group had normal RI values, none of them were higher.

Those with aberrant LFTs (n=42) had a considerably lower mean hepatic artery PSV (43.0 ± 7.7 cm/s) than those with normal LFTs (72.0 ± 8.0 cm/s). The hepatic artery RI was lower in the aberrant LFT group (0.5 ± 0.1) than in the normal LFT group (0.7 ± 0.1).

While 98% (n=43) of NAFLD patients exhibited reduced portal vein velocities (<20 cm/s), just one instance (2%) had a normal portal vein velocity. All control subjects (100%) had normal portal vein peak systolic velocities (20–40 cm/s). In comparison to the controls (25.7 ± 3.6 cm/s), the NAFLD group's mean portal vein PSV was significantly lower (18.2 ± 2.1 cm/s), with a p-value < 0.01.

Compared to 100% of NAFLD patients, only 56% (n=20) of the controls exhibited lower splenic artery PSV values (<90 cm/s). Of the total, 16 controls, or 44%, exhibited normal velocities between 90 and 110 cm/s. "The mean splenic artery PSV was considerably lower in the NAFLD group (50.2 ± 8.5 cm/s) than in the control group (88.1 ± 9.3 cm/s), as shown by the p-value < 0.01."

Additionally, the portal vein peak systolic velocity (PSV) and splenic artery PSV were lower in individuals with abnormal liver function tests (LFTs) than in those with normal LFTs (24.8 ± 4.2 cm/s and 87.5 ± 12.4 cm/s, respectively) (18.2 ± 2.6 cm/s and 50.2 ± 9.4 cm/s, respectively).

CONCLUSION

9. CONCLUSION

The following conclusions are drawn from the current investigation's findings:

1. Nonalcoholic fatty liver disease (NAFLD) patients have significantly altered hepatic and splenic circulation.
2. Hepatic artery peak systolic velocity (PSV) and resistive index (RI) were much lower in those with nonalcoholic fatty liver disease (NAFLD).
3. Portal venous flow is consistently reduced in NAFLD patients.
4. The splenic artery peak systolic velocity (PSV) is lower in those with non-alcoholic fatty liver disease (NAFLD).
5. Abnormal splenic-porto system Doppler findings are linked to abnormal liver function tests.

Consequently, it is recommended that:

1. Doppler ultrasonography of the hepatic artery, portal vein, and splenic artery may be used to evaluate NAFLD.
2. For a more precise diagnosis and ongoing follow-up with NAFLD patients, Doppler ultrasonography need to be included into regular evaluations.
3. Larger patient cohorts with NAFLD that are categorized by severity, verified by biopsy, and validated by MRI or elastography should be the goal of future research.

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ANNEXURE

“ROLE OF DOPPLER ULTRASONOGRAPHY OF THE SPLENO-PORTAL SYSYTEM IN DIAGNOSING EARLY HEPATIC DYSFUNCTION IN NON-ALCOHOLIC FATTY LIVER DISEASE PATIENTS – A CASE CONTROL STUDY”

PATIENT PROFORMA

Name :

Age :

Sex :

UHID :

CLINICAL HISTORY :

History of presenting illness:

Past History:

Personal History:

History of Type II DM:

LOCAL EXAMINATION:

ULTRASOUND PAREMETERS ASSESSED	
1.Size	
2. Echogenicity	
3. PORTAL VEIN ,PSV	
4. HEPATIC ARTERY PSV, RI	
5. Splenic artery PSV	

Ultrasound Impression:

LAB INVESTIGATION:
1. LFTs:

For further information/ clarification please contact:

DR. VIMAL CHAUDHARY

CONTACT NO: +919911981010

<u>STUDY TITLE:“ROLE OF DOPPLER ULTRASONOGRAPHY OF THE SPLENOPORTAL SYSYTEM IN DIAGNOSING EARLY HEPATIC DYSFUNCTION IN NON-ALCOHOLIC FATTY LIVER DISEASE PATIENTS – A CASE CONTROL STUDY ”</u>

<u>Principal investigator:</u> Dr. VIMAL CHAUHDHARY

<u>Under guidance of:</u> Dr. Adarsh AD

Name of the subject:

Age:

Gender:

UHID No:

- I have been informed in my own language that this study involves ultrasound as a part of procedure. I have been explained thoroughly and understand the procedure.
- 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.
- 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.
- 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).
- I confirm that Dr. VIMAL CHAUDHARY/ Dr. Adarsh AD (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:

ಸಮ್ಮತಿಪತ್ರ:

ಈಕೆಳಗೆಸಹಿಮಾಡಿರುವ ------------------

ಆದನಾನುಈಅಧ್ಯಯನದಲ್ಲಿಪಾಲ್ಗೊಳ್ಳುವಸಲುವಾಗಿವೈದ್ಯಕೀಯಪ್ರಯೋಗಪರೀಕ್ಷೆಗೆಒಳಪಡಲುನನ್ನವೈಯ್ಯಕ್ತಿಕ ವಿವರಗಳನ್ನುನೀಡಲುಸಮ್ಮತಿಸಿರುತ್ತೇನೆ.

ಈಅಧ್ಯಯನದಉದ್ದೇಶ, ಅಧ್ಯಯನದಸಂದರ್ಭದಲ್ಲಿನೀಡುವಮತ್ತುಸಂಗ್ರಹಿಸುವಮಾಹಿತಿಯಗೋಪ್ಯತೆಯಬಗ್ಗೆನನಗೆನನ್ನಸ್ಥಳೀಯಭಾಷೆ ಯಲ್ಲಿಓದಿಹೇಳಲಾಗಿದೆ/ವಿವರಿಸಲಾಗಿದೆಮತ್ತುನಾನುಇದನ್ನುಅರ್ಥಮಾಡಿಕೊಂಡಿರುತೇನೆ. ಈಅಧ್ಯಯನದವಿವಿಧಅಂಶಗಳಬಗ್ಗೆಪ್ರಶ್ನೆಗಳನ್ನುಕೇಳುವಅವಕಾಶವನ್ನುನನಗೆನೀಡಲಾಗಿದೆಮತ್ತುನನ್ನಪ್ರಶ್ನೆಗಳಿಗೆ ತೃಪ್ತಿಕರವಾದಉತ್ತರಗಳುದೊರೆತಿರುತವೆ. ಈಅಧ್ಯಯನದಮೂಲಕಸಂಗ್ರಹಿಸಿರುವಮಾಹಿತಿಯನ್ನುಸಂಶೋಧನೆಯಉದ್ದೇಶಕ್ಕೆಮಾತ್ರಬಳಸತಕ್ಕದ್ದು.

ಈಅಧ್ಯಯನದಿಂದಯಾವುದೇಸಂದರ್ಭದಲ್ಲಿಹಿಂದೆಸರಿಯುವಸ್ವಾತಂತ್ರ್ಯನನಗಿದೆಎಂಬುದನ್ನೂ, ಈಅಧ್ಯಯನದಲ್ಲಿಪಾಲ್ಗೊಳ್ಳುವುದರಿಂದನನಗೆಯಾವುದೇಹೆಚ್ಚುವರಿವೆಚ್ಚತಗಲುವುದಿಲ್ಲವೆಂಬುದನ್ನುತಿಳಿದಿರುತ್ತೇನೆ.

ಪರೀಕ್ಷಾರ್ಥಿಯಹೆಸರುಮತ್ತುಸಹಿ/ಹೆಬ್ಬೆಟ್ಟಿನಗುರುತು

ಸಾಕ್ಷಿಗಳಹೆಸರುಮತ್ತುಸಹಿ

1. ದಿನಾಂಕ:
2. ದಿನಾಂಕ:

ಸಂದರ್ಶಕರಹೆಸರುಮತ್ತುಸಹಿ ಪ್ರಧಾನಪರೀಕ್ಷಕರಹೆಸರುಮತ್ತುಸಹಿ

ದಿನಾಂಕ: ದಿನಾಂಕ:

“ROLE OF DOPPLER ULTRASONOGRAPHY OF THE SPLENOPORTAL SYSYTEM IN DIAGNOSING EARLY HEPATIC DYSFUNCTION IN NON-ALCOHOLIC FATTY LIVER DISEASE PATIENTS – A CASE CONTROL STUDY”

PATIENT INFORMATION SHEET

Principal Investigator: Dr. VIMAL CHAUDHARY

I, Dr. VIMAL CHAUDHARY, post-graduate student in Department of Radio-Diagnosis at Sri Devaraj URS Medical College. I will be conducting a study titled *“ROLE OF DOPPLER ULTRASONOGRAPHY OF THE SPLENOPORTAL SYSYTEM IN DIAGNOSING EARLY HEPATIC DYSFUNCTION IN NON-ALCOHOLIC FATTY LIVER DISEASE PATIENTS – A CASE CONTROL STUDY”* for my dissertation under the guidance of Dr. Adarsh AD, Professor, Department of Radio-Diagnosis. In this study, we will assess the role of Doppler ultrasound in diagnosing early hepatic dysfunction in non-alcoholic fatty liver. You would have to undergo ultrasound before entering the study. The study will not add any risk or financial burden to you if you are a part of the study. 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.

Dr. VIMAL CHAUDHARY

Mobile no: 9911981010

E-mail id: drvimaldavid@gmail.com

"ಆಲ್ಕೊಹಾಲಿಕ್ ಅಲ್ಲದ ಕೊಬ್ಬಿನ ಪಿತ್ತಜನಕಾಂಗದ ಕಾಯಿಲೆಯ ರೋಗಿಗಳಲ್ಲಿ ಆರಂಭಿಕ ಯಕೃತ್ತಿನ ಅಪಸಾಮಾನ್ಯ ರೋಗನಿರ್ಣಯದಲ್ಲಿ ಸ್ಪ್ಲೆನೋಪೋರ್ಟಲ್ ಸಿಸ್ಟಮ್ನ ಡಾಪ್ಲರ್ ಅಲ್ಟ್ರಾಸೋನೋಗ್ರಫಿಯ ಪಾತ್ರ - ಒಂದು ಪ್ರಕರಣ ನಿಯಂತ್ರಣ"

<u>ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ</u>

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ: ಡಾ. ವಿಮಲ್ ಚೌಧರಿ

ನಾನು, ಡಾ. ವಿಮಲ್ ಚೌಧರಿ, ಶ್ರೀ ದೇವರಾಜ್ ಅರಸ್ ವೈದ್ಯಕೀಯ ಕಾಲೇಜಿನಲ್ಲಿ ರೇಡಿಯೋ ರೋಗನಿರ್ಣಯ ವಿಭಾಗದಲ್ಲಿ ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿ. "ಆಲ್ಕೊಹಾಲಿಕ್ ಅಲ್ಲದ ಕೊಬ್ಬಿನ ಪಿತ್ತಜನಕಾಂಗದ ಕಾಯಿಲೆಯ ರೋಗಿಗಳಲ್ಲಿನ ರೋಗಿಗಳಲ್ಲಿನ ರೋಗಿಗಳಲ್ಲಿನ ಆರಂಭಿಕ ಯಕೃತ್ತಿನ ಅಪಸಾಮಾನ್ಯ ಕ್ರಿಯೆಯನ್ನು ಪತ್ತೆಹಚ್ಚುವಲ್ಲಿ ಸ್ಪ್ಲೆನೋಪೋರ್ಟಲ್ ಸಿಸ್ಟಮ್ನ ಡಾಪ್ಲರ್ ಅಲ್ಟ್ರಾಸೋನೋಗ್ರಫಿಯ ಪಾತ್ರ" ಎಂಬ ಶೀರ್ಷಿಕೆಯ ಅಧ್ಯಯನವನ್ನು ನಾನು ನಡೆಸುತ್ತಿದ್ದೇನೆ. ರೇಡಿಯೋ-ರೋಗನಿರ್ಣಯದ ಈ ಅಧ್ಯಯನದಲ್ಲಿ, ಆಲ್ಕೊಹಾಲ್ಯುಕ್ತವಲ್ಲದ ಕೊಬ್ಬಿನ ಪಿತ್ತಜನಕಾಂಗದಲ್ಲಿ ಆರಂಭಿಕ ಯಕೃತ್ತಿನ ಅಪಸಾಮಾನ್ಯ ಕ್ರಿಯೆಯನ್ನು ಪತ್ತೆಹಚ್ಚುವಲ್ಲಿ ಡಾಪ್ಲರ್ ಅಲ್ಟ್ರಾಸೌಂಡ್‌ನ ಪಾತ್ರವನ್ನು ನಾವು ನಿರ್ಣಯಿಸುತ್ತೇವೆ. ಅಧ್ಯಯನಕ್ಕೆ ಪ್ರವೇಶಿಸುವ ಮೊದಲು ನೀವು ಅಲ್ಟ್ರಾಸೌಂಡ್ಗೆ ಒಳಗಾಗಬೇಕಾಗುತ್ತದೆ. ನಿಮ್ಮ ದಿನನಿತ್ಯದ ಕೆಲಸದ ಭಾಗವಾಗಿ ಅಲ್ಟ್ರಾಸೌಂಡ್‌ನ ವೆಚ್ಚವನ್ನು ನೀವು ಭರಿಸುತ್ತೀರಿ. ಈ ಸಂಶೋಧನಾ ಯೋಜನೆಯಲ್ಲಿ ಭಾಗವಹಿಸಿದ್ದಕ್ಕಾಗಿ ನಿಮಗೆ ಯಾವುದೇ ಹಣಕಾಸಿನ ಪರಿಹಾರವನ್ನು ನೀಡಲಾಗುವುದಿಲ್ಲ.

ನಿಮ್ಮ ಎಲ್ಲಾ ವೈಯಕ್ತಿಕ ಡೇಟಾವನ್ನು ಗೌಪ್ಯವಾಗಿ ಇರಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಈ ಸಂಸ್ಥೆಯಿಂದ ಸಂಶೋಧನಾ ಉದ್ದೇಶಕ್ಕಾಗಿ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ. ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಸ್ವತಂತ್ರರು. ಯಾವುದೇ ಕಾರಣಗಳನ್ನು ನೀಡದೆ ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು. ಭಾಗವಹಿಸಲು ನಿಮ್ಮ ನಿರಾಕರಣೆಯು ಈ ಸಂಸ್ಥೆಯಲ್ಲಿ ಯಾವುದೇ ಪ್ರಸ್ತುತ ಅಥವಾ ಭವಿಷ್ಯದ ಕಾಳಜಿಗೆ ನಿಮ್ಮನ್ನು ಪೂರ್ವಾಗ್ರಹ ಮಾಡುವುದಿಲ್ಲ

ಡಾ. ವಿಮಲ್ ಚೌಧರಿ

ಮೊಬೈಲ್ ಸಂಖ್ಯೆ: 9911981010

ಇ-ಮೇಲ್ ಐಡಿ: drvimaldavid@gmail.com

S. No.	Gender	Case/Control	Hepatic artery PSV	Hepatic artery RI	Portal vein PSV	Splenic artery PSV	Liver echotexture	Raised LFT Values	Hepatomegaly
1	Male	Control	83.2	0.8	28	92.2	Normal	Normal	normal
2	Female	Case	47.5	0.5	18.6	62.5	Coarse	Abnormal	normal
3	Male	Case	48	0.5	19.5	57.2	Coarse	Abnormal	normal
4	Male	Control	72	0.7	28	82.3	Normal	Normal	normal
5	Male	Case	32.4	0.4	16	42.8	Coarse	Abnormal	increased
6	Female	Case	40.4	0.5	17.9	48	Coarse	Abnormal	increased
7	Female	Control	76	0.7	30.1	73.4	Normal	Normal	normal
8	Male	Control	88.7	0.8	29.8	102	Normal	Normal	normal
9	Male	Case	48	0.5	15.6	52.3	Coarse	Abnormal	normal
10	Female	Control	82.6	0.8	28.7	98.8	Normal	Normal	normal
11	Female	Case	52	0.6	19.3	60.8	Coarse	Abnormal	normal
12	Female	Case	51.2	0.6	19.2	56.2	Coarse	Abnormal	normal
13	Male	Case	49.2	0.5	15.6	61.3	Coarse	Abnormal	normal
14	Male	Control	76.5	0.7	25.3	82.2	Normal	Normal	normal
15	Male	Case	38.9	0.3	14.5	44.5	Coarse	Abnormal	increased
16	Male	Control	66.7	0.7	24.2	68.3	Normal	Normal	normal
17	Male	Case	53.4	0.6	16.4	48.3	Coarse	Abnormal	increased
18	Male	Control	62.8	0.6	31.2	85.3	Normal	Normal	normal
19	Female	Control	68.3	0.7	28.8	92.8	Normal	Normal	normal
20	Female	Case	47.2	0.5	18.8	58.2	Coarse	Abnormal	normal
21	Female	Control	71.4	0.7	33.1	86.9	Normal	Normal	normal
22	Female	Case	34.5	0.4	18.2	40.3	Coarse	Abnormal	increased
23	Female	Control	79.8	0.8	36.4	94.5	Normal	Normal	normal
24	Female	Case	58.8	0.7	16.7	73.5	Coarse	Abnormal	normal
25	Male	Control	71.6	0.7	29.2	106.4	Normal	Normal	normal
26	Male	Case	42.1	0.6	19.8	65.4	Coarse	Abnormal	normal
27	Male	Control	63.8	0.6	24.1	91.2	Normal	Normal	normal
28	Male	Control	68.7	0.7	25.2	93.4	Normal	Normal	normal
29	Male	Control	77.6	0.7	34.2	88	Normal	Normal	normal
30	Female	Case	58.8	0.7	24.3	60.2	Coarse	Abnormal	normal
31	Female	Case	42.1	0.6	19.4	52.5	Coarse	Abnormal	normal
32	Male	Case	50.2	0.6	17.4	54	Coarse	Abnormal	Increased
33	Male	Case	43.4	0.5	18.7	51.3	Coarse	Abnormal	increased
34	Male	Case	42.4	0.5	19.1	53.4	Coarse	Abnormal	Normal
35	Male	Control	72	0.7	28.2	84.3	Normal	Abnormal	Normal
36	Female	Control	68	0.7	26.3	85.4	Normal	Normal	Normal
37	Male	Case	36.2	0.5	15.8	41.2	Coarse	Abnormal	Increased
38	Female	Case	41.5	0.5	16	42.3	Coarse	Abnormal	normal
39	Male	Case	38.4	0.5	15.3	46.1	Coarse	Abnormal	Increased
40	Female	Case	59.5	0.6	16.2	74.9	Normal	Normal	Normal
41	Female	Control	62	0.6	24.3	72.7	Normal	Normal	Normal
42	Male	Control	66	0.6	22.2	78.4	Normal	Normal	Normal
43	Female	Case	41.3	0.5	18.9	52.1	Coarse	Abnormal	Increased
44	Male	Case	43	0.5	19.5	52.8	Coarse	Abnormal	increased
45	Male	Case	39.3	0.5	18.5	44.2	Coarse	Abnormal	Normal
46	Male	Case	40.1	0.5	19.2	41.2	Coarse	Abnormal	Increased
47	Male	Control	80	0.7	22.2	97.2	Normal	Normal	Normal
48	Female	Control	82.3	0.7	26.1	92.1	Normal	Normal	Normal
49	Male	Case	48	0.6	17.2	56	Coarse	Abnormal	Normal
50	Female	Control	78	0.7	23	86.2	Normal	Normal	Normal
51	Male	Case	45.2	0.6	18.2	52	Normal	Abnormal	normal
52	Male	Control	72	0.7	22.1	81.3	Normal	Normal	normal
53	Male	Case	43	0.6	19.2	49.1	Coarse	Abnormal	increased
54	Female	Case	41.6	0.5	19	44.3	Coarse	Abnormal	increased
55	Male	Case	33.1	0.4	15.2	40.1	Coarse	Abnormal	increased
56	Male	Control	64.8	0.7	26.2	67.2	Normal	Normal	normal
57	Female	Control	69.7	0.7	24.3	75.3	Coarse	Normal	normal
58	Female	Case	43.6	0.6	16.2	43.2	Coarse	Abnormal	normal
59	Male	Case	49.5	0.6	19.3	52.1	Coarse	Abnormal	increased
60	Male	Control	71.3	0.7	26.2	92.1	Normal	Normal	normal

S. No.	Gender	Case/Control	Hepatic artery PSV	Hepatic artery RI	Portal vein PSV	Splenic artery PSV	Liver echotexture	Raised LFT Values	Hepatomegaly
61	Male	Case	41.3	0.4	16	42.1	Coarse	Abnormal	normal
62	Male	Case	52.1	0.5	19	44.4	Coarse	Normal	normal
63	Male	Case	39.2	0.5	18.2	48.3	Normal	Abnormal	normal
64	Male	Control	70.2	0.7	22.2	72.2	Normal	Normal	normal
65	Male	Control	67.4	0.7	24.2	89.3	Normal	Normal	normal
66	Male	Control	78	0.8	26.2	90.1	Normal	Normal	normal
67	Female	Control	83.3	0.7	27.2	92.2	Normal	Normal	normal
68	Female	Case	40.4	0.5	19.3	54.1	Coarse	Abnormal	increased
69	Male	Case	38.2	0.4	14.2	42.2	Coarse	Abnormal	increased
70	Female	Control	77.2	0.6	24.2	82.2	Normal	Normal	normal
71	Female	Control	75.2	0.7	22.1	90.1	Normal	Normal	normal
72	Female	Case	43.2	0.5	16.4	46.2	Coarse	Abnormal	increased
73	Male	Control	78.2	0.7	23.2	88.2	Normal	Normal	normal
74	Male	Case	58.2	0.4	18.9	56.2	Coarse	Normal	normal
75	Female	Control	73.2	0.7	24.2	96.2	Normal	Normal	normal
76	Female	Case	59.2	0.4	13.2	41.2	Coarse	Abnormal	normal
77	Female	Control	78.6	0.7	22.1	94.3	Normal	Normal	normal
78	Female	Case	40.1	0.4	14.2	42.3	Coarse	Abnormal	normal
79	Male	Case	45.2	0.5	16.9	48.3	Coarse	Abnormal	normal
80	Male	Control	80.2	0.7	21.1	76.4	Normal	Normal	normal