"UTILITY OF T2 WEIGHTED AND DIFFUSION WEIGHTED SEQUENCES IN MAGNETIC RESONANCE IMAGING IN DIFFERENTIATION OF BENIGN AND MALIGNANT LIVER LESIONS: BENEFITS OF SINGLE VERSUS COMBINED SEQUENCES- ONE YEAR HOSPITAL BASED OBSERVATIONAL STUDY"

BY

REGISTRATION NO. BS0120007

Dissertation

Submitted to the

KLE Academy of Higher Education and Research,

Belagavi, Karnataka

In partial fulfillment

of the requirements for the degree of

M.D.

IN

RADIO-DIAGNOSIS

DEPARTMENT OF RADIO-DIAGNOSIS,
J. N. MEDICAL COLLEGE,
BELAGAVI -590010. KARNATAKA

JUNE/JULY 2023

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH, BELAGAVI, KARNATAKA

Endorsement by the HOD/Principal/ Head of the Institution

This is to certify that the dissertation entitled **"UTILITY OF T2 WEIGHTED AND DIFFUSION WEIGHTED SEQUENCES IN MAGNETIC RESONANCE IMAGING IN DIFFERENTIATION OF BENIGN AND MALIGNANT LIVER LESIONS: BENEFITS OF SINGLE VERSUS COMBINED SEQUENCES- ONE YEAR HOSPITAL BASED OBSERVATIONAL STUDY"** is a bonafide research work done by **REG. NO. BS0120007.**

Dr. PRADEEPGOUD H. PATIL MD,
Professor and HOD,
Department of Radio Diagnosis,
J. N. Medical College,
Nehru Nagar, Belagavi – 10

Date: 14/01/2023
Place: Belagavi

Dr. N. S. MAHANTASHETTI MD (PAEDS)
Principal,
J. N. Medical College,
Nehru Nagar,
Belagavi – 10

PRINCIPAL
J.N. Medical College,
BELAGAVI- 590 010

Date: 14/01/2023
Place: Belagavi

UNDERTAKING

I, **Reg. No. BS0120007,** hereby declare that the information and the data mentioned in my dissertation entitled **"UTILITY OF T2 WEIGHTED AND DIFFUSION WEIGHTED SEQUENCES IN MAGNETIC RESONANCE IMAGING IN DIFFERENTIATION OF BENIGN AND MALIGNANT LIVER LESIONS: BENEFITS OF SINGLE VERSUS COMBINED SEQUENCES- ONE YEAR HOSPITAL BASED OBSERVATIONAL STUDY"** belongs to me and is original. I am aware of the definition of plagiarism as detailed below:

- An act or instance of using or closely imitating the language and thoughts of another author without authorization and the representation of that author's work as one's own, as by not crediting the original author.
- A piece of writing or other work reflecting such unauthorized use or imitation.
- The deliberate or reckless representation of another's words, thoughts or ideas as one's own without attribution in connection with submission of academic work, whether graded or otherwise.

I hereby declare that the dissertation prepared by me is original one and does not involve plagiarism anywhere. In case at a later stage, it is found that I have indulged in plagiarism, then I am solely responsible for the same and the institution is at liberty to take any disciplinary action against me including cancellation of dissertation or any other penalties imposed by the University.

Date: 6/01/2023

Place: Belagavi

Reg. No. BS0120007

PLAGIARISM ACCEPTED LETTER

KLE

JAWAHARLAL NEHRU MEDICAL COLLEGE

(Recognized by Medical Council of India, New Delhi)

Accredited 'A+' Grade by NAAC (3rd Cycle) Placed in Category 'A' by MHRD (GoI)

Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

0831-2471350 0831-2470759 www.jnmc.edu principal@jnmc.edu

Ref No: MDC/PG/ Date: 07-12-2022.

ACCEPTANCE LETTER

The softcopy of thesis entitled: **"UTILITY OF T2 WEIGHTED AND DIFFUSION WEIGHTED SEQUENCES IN MAGNETIC RESONANCE IMAGING IN DIFFERENTIATION OF BENIGN AND MALIGNANT LIVER LESIONS: BENEFITS OF SINGLE VERSUS COMBINED SEQUENCES- ONE YEAR HOSPITAL BASED OBSERVATIONAL STUDY""** has been submitted for Anti-Plagiarism check through Turnitin software. The scan has been carried out and the scanned output reveals a match percentage of 08% which is within the acceptable limits of 10% as per the guidelines given by UGC.

Guide.

KAHER BELAGAVI J.N. MEDICAL COLLEGE

Dr. (Mrs.) N.S. Mahantashetti.
Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BS0120007,
Postgraduate Student,
2020-21 Batch,
Department of Radiology,
J. N. Medical College, Belagavi.

ETHICAL CLEARANCE LETTER

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : domc@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 145 **Date: 25/01/2021**

To,

PG student in Radio-diagnosis,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"UTILITY OF T2 WEIGHTED AND DIFFUSION WEIGHTED SEQUENCES IN MAGNETIC RESONANCE IMAGING IN DIFFERENTIATION OF BENIGN AND MALIGNANT LIVER LESIONS: BENEFITS OF SINGLE VERSUS COMBINED SEQUENCES – ONE YEAR HOSPITAL BASED OBSERVATIONAL STUDY"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

LIST OF ABBREVIATIONS

GLOSSARY	ABBREVIATIONS
FLL	Focal liver lesions
DWI	Diffusion weighted imaging
ADC	Apparent diffusion coefficient
T2WI	T2 weighted imaging
T1WI	T1 weighted imaging
MRI	Magnetic resonance imaging
HCC	Hepatocellular carcinoma
FNH	Focal nodular hyperplasia
HA	Hepatic adenoma
RN	Regenerative nodules
ECA	Extracellular contrast agents
CEA	Carcinoembryonic antigen
CCA	Cholangiocarcinoma
HAS	Hepatocyte specific agents
TSE	Turbo spin echo
FSE	Fast spine echo
CT	Computed tomography
CLD	Chronic liver disease
IHC	Intrahepatic cholangiocarcinoma

ABSTRACT

Background: The increased use of imaging technology has contributed to an increase in the overall number of focal liver lesions (FLL) that are found incidentally. The vast majority of FLLs that arise in livers that are not cirrhotic are benign and do not progress to malignancy. The most common forms of solid benign tumours are adenomas, focal nodular hyperplasias, and hemangiomas. After metastases, the most frequent malignant lesions that occur in non-cirrhotic liver are hepatocellular carcinoma and intrahepatic cholangiocarcinoma. Accurate characterization of liver masses allows for the avoidance of potentially disastrous outcomes, such as delayed treatment of malignant tumours or treatment that is not essential for benign lesions. In order to reduce the number of needless liver biopsies that are performed, it is of the highest significance to improve the sensitivity and accuracy of cross-sectional imaging for these incidental hepatic lesions. Though detection and characterization of FLLs mainly depends on dynamic enhanced magnetic resonance imaging (MRI), for patients with contraindications such as an allergy to contrast agents, unenhanced MRI sequences, such as T2-WI and DWI have proven crucial in the identification and diagnosis of FLLs. Based on signal intensity of the lesion, T2WI can differentiate benign cystic lesions from malignant solid lesions. Though DWI is beneficial for lesion detection and characterization, it has not yet replaced T2WI in routine use because it has many advantages and limitations simultaneously. This study mainly focuses on the combined ability of both the sequences to improve the diagnostic ability.

Materials and methods: A hospital based observational study was done on 45 patients referred for MRI (Magnetic Resonance Imaging) abdomen and pelvis to a tertiary care teaching institute in Belgaum. All patients were evaluated clinically and

then MRI of the abdomen was done using a 3Tesla MRI scanner. The primary outcome variables in this study were diagnostic accuracy of T2WI, DWI and both the sequences together.

Results: The median age was 56 years in this study. Majority were males (57.7%). Of 45 patients, 9 were known case of chronic liver disease, 2 were known case of hepatitis-b, 2 were known case of carcinoma rectum, 1 had carcinoma stomach, 1 had carcinoma cervix, 1 had carcinoma adrenal gland, 1 had carcinoma tongue and 1 was a known case of wilson's disease. Rest of the 27 patients either showed a liver lesion on routine annual ultrasonography or had either come with other complaints. 37 out of 51 lesions were correctly classified on T2WI (72.5%). 41 out of 51 lesions were correctly classified on DWI (80.3%). Accuracy of correctly classified lesions was increased to 86.2% (44 out 51) when the lesion was categorized using both the sequences.

Conclusion: Accuracy of correctly classified lesions was increased when the lesion was categorized using both the sequences. In this study, DWI was better at categorizing malignant lesions and T2WI was good at categorizing cystic benign lesions.

TABLE OF CONTENT

S. NO	TABLE OF CONTENT	PAGE NO
1	**INTRODUCTION**	**1-3**
2	**AIMS & OBJECTIVES**	**4**
3	**REVIEW OF LITERATURE**	**5-32**
4	**MATERIALS & METHODS**	**33-35**
5	**RESULTS**	**36-55**
6	**DISCUSSION**	**56-60**
7	**CONCLUSIONS**	**61-62**
8	**LIMITATIONS AND RECOMMENDATIONS**	**63**
9	**SUMMARY**	**64-68**
10	**BIBLIOGRAPHY**	**69-77**
11	**ANNEXURES**	**78-93**
	I. INFORMED CONSENT FORM	**78-83**
	II. STUDY PROFORMA	**84-86**
	III. PHOTOGRAPHS OF CASES	**87-91**
	IV. KEY TO MASTER CHART	**92**
	V. MASTER CHART	**93**

LIST OF TABLES

S. NO	TABLE DESCRIPTION	PAGE NO
1.	Age wise distribution of patients	**36**
2.	Gender wise distribution of patients	**37**
3.	Associated comorbidity of the patients	**38**
4.	No of lesions present wise distribution of patients	**39**
5.	Lobe/segment wise distribution	**40**
6.	Status of margin of lesions on T2WI	**41**
7.	Status of signal intensity on T2WI	**42**
8.	Status of lesion character on T2 WI	**43**
9.	Status of lesions at 50 mm/s2	**44**
10.	Status of signal drop of lesions at 400 and 800 mm/s2	**45**
11.	Status of ADC of lesions	**46**
12.	Status of restriction pattern of lesions	**47**
13.	Status of lesion character on DWI	**48**
14.	Status of lesion character on T2WI AND DWI	**49**
15.	Status of BIOPSY/FNAC of lesions	**50**
16.	Status of final lesion character	**51**
17.	Percentage of lesions correctly classified	**52**
18.	Status of diagnosis of all lesions	**53**
19.	Kappa agreement of lesion character	**55**

LIST OF GRAPHS

S. NO	GRAPHS DESCRIPTION	PAGE NO
1.	Age wise distribution of patients	**36**
2.	Gender wise distribution of patients	**37**
3.	Associated comorbidity of the patients	**38**
4.	No of lesions present wise distribution of patients	**39**
5.	Lobe/segment wise distribution	**40**
6.	Status of margin of lesions on T2WI	**41**
7.	Status of signal intensity on T2WI	**42**
8.	Status of lesion character on T2 WI	**43**
9.	Status of lesions at 50 mm/s^2	**44**
10.	Status of signal drop of lesions at 400 and 800 mm/s^2	**45**
11.	Status of ADC of lesions	**46**
12.	Status of restriction pattern of lesions	**47**
13.	Status of lesion character on DWI	**48**
14.	Status of lesion character on T2WI AND DWI	**49**
15.	Status of BIOPSY/FNAC of lesions	**50**
16.	Status of final lesion character	**51**
17.	Percentage of lesions correctly classified	**52**
18.	Status of diagnosis of all lesions	**54**

LIST OF FIGURES

S. NO	FIGURE DESCRIPTION	PAGE NO
1.	Segmental anatomy of the liver	**5**
2.	Segmental numbering of the liver	**6**
3.	CT image showing hepatic cyst	**13**
4.	CT image showing hepatic abscess	**14**
5.	CT image showing intrahepatic cholangiocarcinoma	**19**
6.	CT image of liver metastasis in a known case of carcinoma colon	**22**
7.	T2W image of the liver	**23**
8.	DWI and ADC of hepatocellular carcinoma	**27**

INTRODUCTION

The increased use of imaging technology has contributed to an increase in the overall number of focal liver lesions (FLL) that are found incidentally. The vast majority of FLLs that arise in livers that are not cirrhotic are benign and do not progress to malignancy[1]. The most common forms of solid benign tumours are adenomas, focal nodular hyperplasias, and hemangiomas. After metastases, the most frequent malignant lesions that occur in non-cirrhotic liver are hepatocellular carcinoma and intrahepatic cholangiocarcinoma[2].

Imaging modalities used for liver pathology are ultrasound, CT and MRI. In a systematic review, contrast-enhanced ultrasound, CT, and MRI were all shown to have similar detection effectiveness, with specificities between 82% and 89%, and the summary receiver operating characteristics showed no significant variation. For FLL characterization, magnetic resonance imaging is the standard due to the absence of ionising radiation and inadequate accessibility of ultrasound contrasts.

Accurate characterization of liver masses allows for the avoidance of potentially disastrous outcomes, such as delayed treatment of malignant tumours or treatment that is not essential for benign lesions. In order to reduce the number of needless liver biopsies that are performed, it is of the highest significance to improve the sensitivity and accuracy of cross-sectional imaging for these incidental hepatic lesions. After these biopsies, the post-procedure morbidity rate is between 0.08 and 0.34 percent, and the mortality rate is between 0 and 0.190 percent[3].

Detection and characterization of FLLs mainly depends on dynamic enhanced magnetic resonance imaging (MRI) with several contrast agents. In order to get

complete picture of the liver, it is necessary to look at the parenchyma in addition to the arterial and biliary systems. For patients with contraindications such as allergy to contrast agents, unenhanced MRI sequences, such as T2-WI and DWI have proven crucial in the identification and diagnosis of FLLs.

DWI is more sensitive than the T2-weighted sequences when it comes to the accurate detection of liver lesions. In identifying and accurately defining tiny localised hepatic lesions, DWI works remarkably well. When low b values are used, localised liver lesions that have a high signal-to-noise ratio and a high lesion-to-liver signal intensity ratio are much simpler to visualise. This is due to the fact that these ratios suggest that the lesion is of a higher intensity than the liver itself. In addition to its improved lesion-to-background contrast, DWI also provides physiological information through apparent diffusion coefficient (ADC) values.

The dark blood effect which is seen in DWI is helpful in differentiating between the intra-hepatic arteries and the small localised lesions[4]. Patients who already have cirrhosis of the liver may benefit from the use of DWI since it helps in the early diagnosis of HCCs. On the other hand, compared to conventional T2-weighted images, DWI exhibits a reduced clarity of parenchymal heterogeneity as well as a lower signal intensity associated with regenerating nodules and fibrosis.

Even though DWI is superior at locating and characterizing lesions, it has not yet fully supplanted T2WI in common usage. This is because T2WI is able to differentiate between the two types of lesions without compromising its accuracy (such as cystic benign lesions like cysts and hemangiomas from solid malignant lesions like metastases and hepatocellular carcinoma [HCC]). However, there is a

possibility that the capacity of this approach to differentiate between benign solid lesions and malignant solid lesions is restricted.

At this point in time, we are only aware of two investigations that directly compared DW imaging to T2-weighted imaging with the intention of identifying FLL. Using an MRI scanner with a field strength of 3 Tesla, the primary objective of this research is to determine whether or not combining the two sequences (T2WI and DWI) results in more accurate diagnoses.

OBJECTIVES

AIM: To assess the diagnostic accuracy of diffusion-weighted imaging (DWI), T2 weighted imaging (T2WI), and the combination of the two sequences in differentiating benign from malignant localised liver lesions (FLLs).

OBJECTIVES:

- To perform plain MRI abdomen on patients having symptoms and/or in suspected case of liver lesion.
- To evaluate the diagnostic efficacy of diffusion-weighted imaging (DWI), T2-weighted imaging (T2WI), and a combination of the two sequences in distinguishing benign focal liver lesions from malignant ones.

REVIEW OF LITERATURE

ANATOMY OF LIVER:

Liver is located in the upper right quadrant of the abdomen, just below the right hemi-diaphragm. It has the form of a wedge and is reddish brown. It is divided into two lobes. Liver measures roughly 15 centimetres (about 6 inches) in length and weighs approximately 1.5 kilos (3.3 pounds). Portal vein and hepatic artery supply blood to liver.

Claude Couinaud, a French surgeon and anatomist, was the first person to divide liver into eight segments based on the location of hepatic and portal veins[5]. Each section has a branch that connects to portal vein, hepatic artery, and bile duct respectively. Along central hepatic vein, liver is divided into two lobes: the right and the left. Right lobe is segmented into its anterior and posterior portions along the plane of the right hepatic vein. Along left hepatic vein which extends from the falciform ligament to the inferior vena cava, the left lobe is divided into medial and lateral segments. The horizontal plane along the portal vein divides the liver into superior and inferior segments as follows:

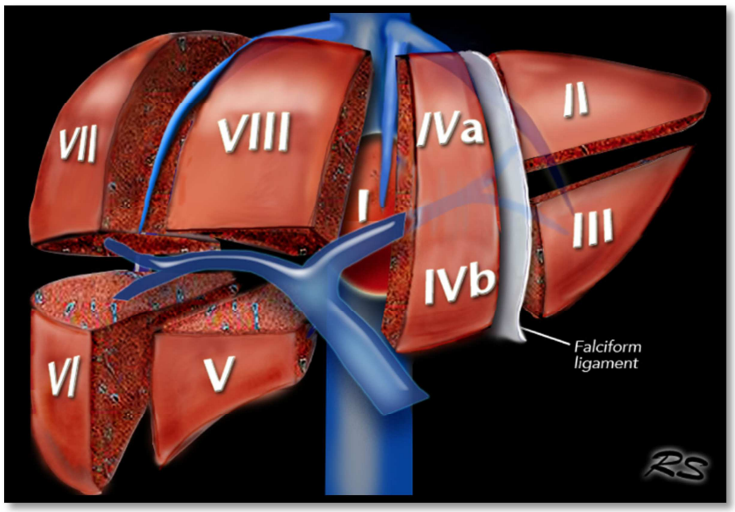


Fig. 1 Segmental anatomy of the liver

The numbering of the eight subdivisions, which are separated according to the Couinaud System, starts at the top left and continues in a path that is clockwise around the subdivisions[5] (as in Fig.2).

1. Caudate: I
2. Superolateral segment of left lobe: II
3. Inferolateral segment of left lobe: III
4. Medial segment left lobe: IV
5. Anteroinferior segment of right lobe: V
6. Posteroinferior segment of right lobe: VI
7. Posterosuperior segment of right lobe: VII
8. Anterosuperior segment of right lobe: VIII

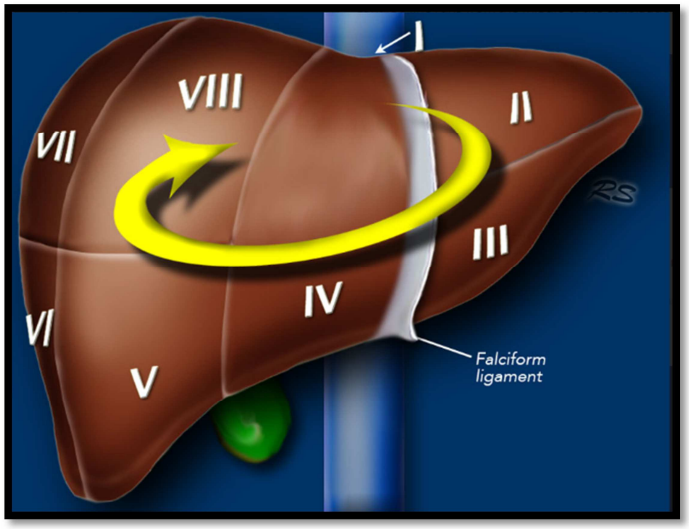


Fig. 2 Segmental numbering of the liver

According to Bismuth's explanation, segment IV is split into two parts: IVa and IVb.

Description of liver tumors:

Tumors of the liver can be basically categorised as either benign or malignant. They can either progress to malignant stage or remain benign for a long time. Benign liver tumours can be subdivided into a number of subgroups depending on their location of origin. These subtypes include hepatocellular, biliary, and stromal liver tumours.

Etiology and risk factors of benign hepatic tumors:

- Hemorrhage or clots in tiny blood vessels in the liver.
- Inflammation in the liver.
- Hormonal imbalance (estrogen).

Types of Benign Tumors:

1. ***Hemangiomas***

The cavernous hemangioma affects three percent to ten percent of people, with a female preference, and most of them are asymptomatic[6]. These are produced when endothelial cells surround blood clots by wrapping themselves around them. They get their supply of blood from the hepatic artery and its branches. Although the root cause of hepatic hemangiomas is unknown.

In the majority of instances, their diameters do not surpass 10 centimeters; nevertheless, a big hemangioma may alter the coagulation properties in addition to pain in the right upper abdomen region. There are a variety of treatment options available, including surgical excision, vascular embolization, and radio-frequency ablation.

A typical hemangioma is well demarcated, has a T1 hypointense appearance, a T2 bright appearance, and enhances in a manner that is comparable to CT postcontrast. In the early stages, the contrast enhancement is nodular and peripheral, but in the late and delayed phases, it gradually fills in the centre from the periphery. The signal strength is similar to that of blood[7]. The ADC value of hemangiomas is noticeably greater than that of solid liver lesions, although it is lower than that of cysts[8].

Giant and flash-filling hemangiomas[9] (small hemangiomas < 2cm), which exhibit quick arterial filling, are two examples of atypical appearances. The distinctive significant T2 hyperintensity will still be visible in both.[10]

Hemangiomas had lower ADC values than normal liver tissue at b-100 and b-600 values and displayed greater ADC values than healthy hepatic tissue on b-1000, according to a research by Bozgeyik et al. However, size of the hemangiomas did not significantly affect the ADC values, since no significant relationship between lesion size and ADC values with various b values was discovered.

2. ***Focal Nodular Hyperplasia (FNH)***

FNH affects approximately 0.2–0.3% of people all over the world with the number of females outnumbering the number of males. FNH has been connected to both women of reproductive age and who use oral contraceptives[10]. It originates as a hepatocyte response to a congenital arteriovenous defect in the liver.

In their early stages, they are often asymptomatic. Ultrasonography and magnetic resonance imaging (MRI) with contrast both play an important role in diagnosis of FNH. The presence of a distinct "central scar" may be seen on contrast-

enhanced imaging. On the other hand, if imaging does not reveal a central scar, it is difficult to differentiate between FNH, hepatic adenoma, and hepatocellular cancer. A biopsy may be performed to assist with the diagnosis.

Because FNH are benign and have low rates of malignant change, they are typically managed conservatively. If it is substantial, however, it can necessitate surgical intervention or vascular embolization.

On T1, FNH seems to vary from iso to hypointense, but on T2, its intensity may range from iso to hyperintense. Fifty percent of these lesions have a central scar that is moderately hyperintense on T2 and has late Gd-contrast enhancement. The efficacy of ADC values in discriminating solid liver masses is severely restricted due to the fact that the ADC values of FNH and adenomas are same as those of metastases and HCC[11].

Following contrast (gadolinium) study, there is a rapid and significant enhancement in 98% of FNH during the arterial phase. It is not feasible to differentiate FNH properly from a hepatic adenoma based only on this imaging. However, an accurate separation is attainable with hepatobiliary contrast enhancement, 1-3 hours after gadobenate dimeglumine enhancement: FNH appear Iso-hyperintense (97%), whereas adenomas are hypointense (100%).

An atypical form of FNH does not have a central scar and it appears hyperintense on both T2 and T1WI but is isointense during the hepatobiliary phase. In addition to this, further investigations such as a biopsy and also follow-up is required for an accurate diagnosis[11].

3. Hepatic Adenoma (HA):

Hepatocellular adenomas are benign liver tumours that are composed of hepatocytes. They account for around 2% of all liver tumours, making them an extremely uncommon form. It presents with nonspecific symptoms such as discomfort in the abdomen, jaundice, and elevated gamma-glutamyl transferase and alkaline phosphatase. Pregnant women, steroid abusers, and those who take contraception or hormone replacement that contains estrogen are the most at risk[12]. Hepatic adenomas may be caused by a number of conditions, including diabetes, obesity, high blood pressure, and abnormal lipid levels. HA's have been demonstrated to be associated with glycogen storage disease in recent study (types I and III).

Since HA's have the potential to evolve into malignant tumours, molecular categorization may be performed by biopsy and pathological investigation. However, a biopsy can make the adenoma bleed or rupture. There are four subtypes under the molecular classification: HNF-1 alpha inactivated mutation, Beta catenin activated mutations, inflammatory type, and unidentified type[13]. Excisional biopsy is the most effective method for obtaining sample of the tumour. There is a 4.2% probability that it will progress to hepatocellular carcinoma. Males are more likely to advance to hepatocellular carcinoma.

It is recommended that HA's can be surgically removed if they meet the following criteria: they must be larger than 5 centimetres in size, they must be growing in size, they must be symptomatic lesions, and they must have molecular markers associated with HCC transformation as well as liver tumour markers such as alpha fetoprotein.

On MRI, Adenomas show T1 and T2 heterogeneous hypointensity. Areas with increased T1 signal intensity is caused by the presence of fat and haemorrhage, whereas necrosis is the cause of areas with lower signal intensity[14]. Adenomas can display a hypointense peripheral rim, which will correspond to a fibrous capsule that has a low signal intensity on both the T1-WI and the T2-WI.

The existence of enormous subcapsular feeding arteries may be attributed to the early arterial enhancement that can be detected on dynamic MRI. On portal-venous phase and equilibrium phase imaging, it typically appears isointense or slightly hypointense.

4. Liver cell Adenomatosis:

There is a strong connection between hepatocellular adenoma and liver cell adenomatosis, which is also referred to as hepatic adenomatosis. Either imaging tests or histological samples may be used to diagnose hepatic adenomas, and the results of either method provide the same conclusion. When there are more than 10 hepatic adenomas in both lobes of the liver in a patient without any pre-existing condition like glycogen storage disorder and is not using any exogenous hormones, a diagnosis of liver cell adenomatosis can be made. They are associated with a greater risk of developing hepatocellular carcinoma[15]. Since liver cell adenomatosis is characterised by multiple lesions that are dispersed over a significant area, surgical removal and transplantation of liver may be an effective form of treatment.

On unenhanced MRI, liver adenomatosis appear hyperintense on T2-WI and hyper- to isointense on T1-WI, while certain lesions can appear hypointense.[16]

Majority of lesions show enhancement during the arterial phase when gadolinium-based contrast media is used. However, the enhancement during the venous and delayed phases is more variable and depends on the histologic nature of the lesion. All peliotic adenomas show enhancement, mixed adenomas show partial enhancement, and no enhancement is seen in steatotic adenomas.

5. Simple cysts:

Cysts in the liver are very prevalent seen in 2.5% of the population[17]. They are encased structures that are filled with fluid. In terms of pathophysiology, they are formed as a reaction to the processes of development, as well as to trauma and inflammation. Few cysts can be hamartomatous in origin.

The average ADC value is highest for hepatic cysts among FLL. Because of the reduction in signal on DWI that occurs in hepatic cysts, with higher b values (more than 500 s/mm2) are able to discriminate hepatic cysts from solid lesions. The presence of fluid inside hepatic cysts enables unfettered diffusion within the lesion, which in turn leads to an increase in ADC values. On unenhanced MRIs, hepatic cysts and hemangiomas may seem to be identical due to the same signal intensities. On the other hand, the ADC values of cysts will be noticeably greater than those of hemangiomas on DW-MRI. As a result, the measurement of ADC values could make it possible to distinguish between simple cysts, hemangiomas and abscesses.

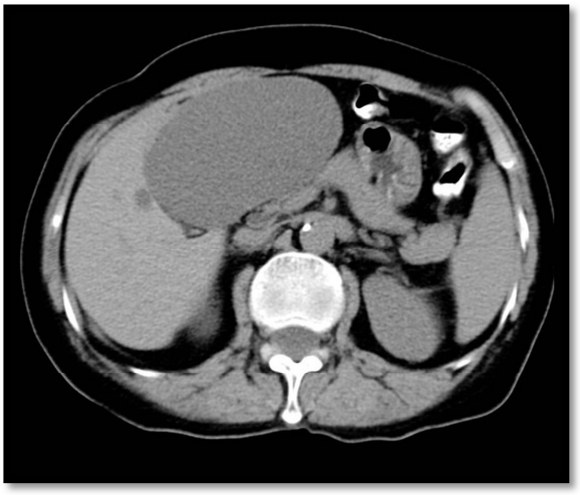


Fig 3: CT axial image shows large hepatic cyst in left lobe of liver.

6. Pseudotumors:

Pseudotumors, on the other hand, are nothing more than "local variations" of the tissue type they originate from. Pseudotumors may take on a wide variety of diverse appearances, including distinct zones of hepatic fibrosis, isolated pockets of fatty liver alterations, and inflammatory pseudotumors[18] etc.

7. Liver abscess

Pyogenic liver abscesses are characterised by the presence of pus and may be either unilocular or multilocular in nature. They are surrounded by capsule made of fibrous tissue. Even though pyogenic abscesses are brought on by a conglomeration of different microorganisms, Escherichia coli and Klebsiella pneumoniae are the ones that are most often found to be the causative agents[19].

There are four primary mechanisms that contribute to the formation of hepatic abscesses:

A. "Hematogenous spread of gastrointestinal tract pathology via the portal vein or disseminated sepsis through the hepatic artery. B). Infection of the bile due to bile duct blockage, which can be caused by a number of different etiologies including stones, neoplasms, and strictures due to ascending cholangitis, pancreatic cancer, or inflammatory bile duct disorders. C). Factors that increase the chance of developing pyogenic liver abscesses after undergoing procedures such as biliary stent placement[20]. D). Other routes of infection for liver abscesses include the direct introduction of bacteria into the liver parenchyma, during cholecystectomy, as well as during hepatic biopsy or surgery, and the superinfection of pre-existing hepatic lesions, such as cysts or necrotic liver lesions".

IMAGING APPEARANCE: On MRI, the central pus is hyperintense on T2WI and hypointense on T1WI, with restricted diffusion on DWI. The inner and outer layers of the wall appear hypo- and hyperintense on T2WI, respectively[21]. Capsular enhancement is seen on contrast study, however this may be absent in immunocompromised patients.

Pyogenic abscesses can also resemble primary or secondary hepatic tumours in appearance, seeming more solid. This is often seen in association with *K. pneumoniae*.

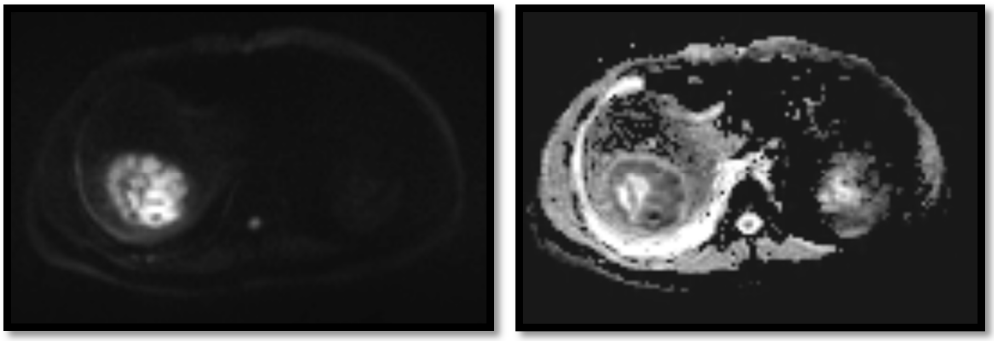


Fig 4: Hepatic abscess showing high signal on DWI with low ADC signal suggestive of restricted diffusion.

8. Nodules in Cirrhotic Liver

Regenerative nodules:

These are well defined, localised proliferations of regular hepatocytes. They typically measure less than 2 cm in size. The progression from RNs to HCC may occur in stages, beginning with low grade dysplastic nodules and progressing to high grade dysplastic nodules and finally HCC.

IMAGING APPEARANCE: On plain CT, they appear hyperdense than normal liver tissue owing to iron deposition, however on portal venous phase, they range from being isodense to hypodense. The RNs are hypointense on T1 and T2, and there is a loss of signal during out of phase gradient echo sequences. On postcontrast imaging, they are either isointense or very slightly hypointense to the liver parenchyma[22].

Dysplastic nodules:

Histopathology reveals that these nodules have abnormal development of the hepatocytes, but there is no indication that they are malignant. There are both high-grade and low-grade nodules.

IMAGING APPEARANCE: Most of them are not seen on ultrasound. On CT, the degree of enhancement seen during the early phase is determined by the severity of dysplasia, which in turn is determined by the degree of hepatic artery neovascularization.

On MRI, low grade nodules are hypointense on T2WI and high grade nodules are slightly hyperintense on T2WI. High-grade dysplastic nodules are hypointense on hepatocyte phase imaging with hepatocellular agents. T2 hyperintensity, restricted diffusion, and microscopic fat are all associated with an increased risk of malignant transformation[23].

Etiology and risk factors of malignant hepatic tumors:

- Hepatitis B and C Virus Infection
- Alcohol abuse
- Cirrhosis
- Smoking
- Higher hepatic DNA synthesis in cirrhosis
- Autoimmune Hepatitis
- Primary Biliary Cirrhosis
- Environmental such as Aflatoxin, Thorotrast, N-Nitrosylated Compound, Pyrrolizidine alkaloids etc.
- Metabolic Diseases such as Type 1 and 3 glycogen storage disease, Wilson's Disease, alpha1-antitrypsin deficiency, Hemochromatosis, Galactosemia, Familial cholestatic cirrhosis etc.

Types of malignant hepatic lesions:

1. Hepatocellular Carcinoma (HCC):

Nearly 75% of all primary liver malignancies are hepatocellular carcinoma, making it the most frequent type of liver cancer. Among those with cancer, it is the fourth leading cause of death[24]. Hepatocellular carcinoma (HCC) is most likely to occur in patients with cirrhosis, chronic viral hepatitis B or C, alcoholism, and/or non-alcoholic steatohepatitis; hence, these patients should undergo routine ultrasonography to reduce their risk of developing HCC. If ultrasound reveals a focal lesion that is larger than one centimetre, either a CT scan or MRI with triple-phase contrast enhancement should be done.

According to the 2010 practice guidelines released by the American Association for the Study of the Liver Diseases (AASLD)[25], the diagnosis of HCC must be based on both histology and imaging. Due to the possibility of cholangiocarcinoma or metastasis from colorectal cancer, a tumour in a cirrhotic liver with a high AFP is no longer usually associated with HCC.

IMAGING APPEARANCE: On plain CT, HCC is hypodense, heterogeneously hypervascular on the arterial phase, with early washout (iso- to hypodense) on the portal phase, and hypodense on the delayed phase. HCC may be distinguished from arterioportal shunts and regenerating nodules by the presence of this early washout.

The characteristic appearance of HCC on extracellular contrast agents (ECA)-enhanced MRI is comparable to that of triple phase CT in most cases[26]. When using ECA, unique characteristics of HCC include the existence of "washout" as well as delayed enhancing pseudocapsule. On T1-WI, the signal strength of HCC may be rather varied, whereas on T2, it seems to be slightly hyperintense. The HCC shows diffusion restriction. Because of the presence of hemorrhage, fat, or necrosis, larger tumours exhibit a signal intensity distribution that is more heterogeneous over all sequences. Macroscopic fat may be recognised by using fat-saturated sequences, whereas microscopic fat can be identified by signal loss on out-of-phase images. Necrosis appears hypointense on T1 and hyperintense on T2WI with the reverse pattern seen with hemorrhage[27]. Other findings include a tumour capsule that is hypointense on both T1 and T2, as well as a tumour thrombus (vascular invasion of the portal or hepatic veins).

2. Intrahepatic cholangiocarcinoma:

Cholangiocarcinoma, often known as CCA, is a kind of epithelial tumour that affects the branches of the intrahepatic biliary system. It is a primary liver cancer, which is the second most frequent[28]. The usual age of diagnosis is between 60 and 70 years old, and men are more likely to be affected than women. Infection with the parasite Opisthorchis viverrini and Clonorchis sinensis, sclerosing cholangitis, choledochal cysts, previous surgery of the biliary tree, exposure to thorotrast[29] & dioxin, and cirrhosis are all considered risk factors for intrahepatic CCA. Patients remain asymptomatic until the latter stages of the disease. The carcinoembryonic antigen (CEA), CA19-9, and CA-125 are all liver markers that have the potential to be increased when intrahepatic CCA is present.

In the majority of instances, they will have a signal that is iso-hypointense on the T1, however the T2-WI may have a variable signal. A typical intrahepatic cholangiocarcinoma will show hyperintensity on T2-WI and will often obstruct both the arteries and the bile ducts, resulting in upstream ductal dilatation. The size of the affected region will decrease in conjunction with the capsular contraction that can be seen there. When contrast is given, the lesion shows delayed enhancement pattern (a hemangioma-like appearance). On the other hand, the enhancement of the cholangiocarcinoma does not match the blood pool. In addition, intrahepatic cholangiocarcinoma may also display homogeneous arterial contrast enhancement[30].

MRI, in combination with magnetic resonance cholangiopancreatography, is often used in the diagnostic process and staging of cholangiocarcinoma at present[31].

In many instances, the early enhancement is continuous around the rim, and it is then followed by increasing enhancement that is heterogeneous over the remaining

portion of the lesion. The cholangiocarcinoma is characterised by a late enhancement, which might be attributed to the fibrotic nature of the tumour. It has been shown that hypervascularity is present in about one third of the cases of cholangiocarcinoma.

According to a study done by Lee et al., the degree of diffusion restriction can be used to establish a treatment plan and optimize the prognosis for patients with intrahepatic cholangiocarcinoma[32].

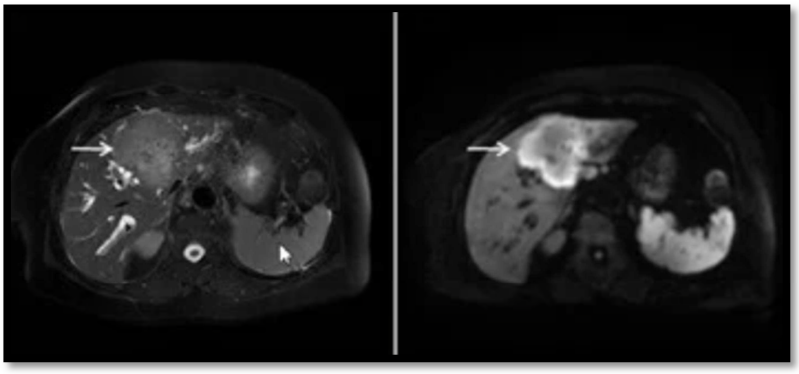


Fig 5 : Case of mass-forming intrahepatic cholangiocarcinoma in the left liver lobe of a 76-year-old man showing lobulated hetrogeneously hyperintense hepatic tumor on axial T2WI FS image and peripheral area of restricted diffusion on diffusion-weighted image (b = 800 s/mm^2)

3. Hemangiosarcoma:

These tumours are extremely rare and aggressive, they only account for 0.2% to 2.0% of all cases of primary liver cancer. Liver hemangiosarcoma can be traced back to the endothelial cells that line arteries as their point of origin. These tumours have poor prognoses because of their propensity for metastasis and their rapid growth rate. These are difficult to diagnose due to the high levels of bleeding, haemorrhage, and necrosis that they present with on CT and MRI. Histological examination of a specimen obtained from a biopsy is the only method that provides 100% accuracy for determining a diagnosis. There is a correlation between exposure to chemicals such as vinyl chloride, arsenic, and thorotrast and the development of these tumours;

however, the underlying cause is often never identified (75 percent of cases are idiopathic) (e.g. occupational exposure). Radiation is also present, which is another source of concern. Majority of patients pass away within the first six months following their diagnosis.

In most cases, the symptoms of hepatic angiosarcoma are non-specific and include fullness in the abdomen, malaise, and fatigue. Changes in laboratory indicators such as bilirubin levels, changed protein-albumin/globulin ratios, or elevated liver enzyme levels are only seen in large sized tumours.

Hepatic angiosarcoma has many presentations ranging from multiple lesions to a single large mass or as a mixed tumor. Non contrast scans may show areas of heterogeneity with hemorrhage. In most cases, they demonstrate early arterial enhancement, which is then followed by gradual filling in of contrast inside the lesion. This is in contrast to the washout that is observed in hepatocellular carcinoma[33]. Angiosarcoma of the liver often has a hypervascular pattern that is distinct from that of other vascular tumours that may occur in the liver, such as hepatoma and hemangioma.

On MRI the lesion shows variable signal intensity on T1 and T2WI along with diffusion hyperintensity and variable ADC values (which are greater than the mean reported in other hepatic malignancies, but lower than those shown in benign cysts and hemangiomas)[34]. A well-known complication that occurs in about 27% of patients is spontaneous rupture of the tumour, which may also be accompanied by peritoneal bleeding[35].

4. Hepatoblastoma:

In children less than 5 years old, hepatoblastoma is the liver tumour that occurs most often. It is very rare in adults[36]. At least ninety percent of patients will have an abnormal AFP, and the AFP may be used as a marker to follow therapy and identify recurrence[37]. The right lobe of the liver is most commonly involved.

IMAGING APPEARANCE: On CT, it is seen as a heterogeneously hypodense mass lesion with calcifications inside it in around 40% of the cases[38].

5. Metastasis

Metastases are the most prevalent liver malignant neoplasms which are forty times more often than primary liver malignant neoplasms. The most common primary malignancy associated with metastasis is colorectal adenocarcinomas[39] and are hypovascular. Neuroendocrine tumors, thyroid malignancies, renal cell, breast carcinoma, melanoma, and sarcoma show hypervascular metastasis[40]. Metastases from other primaries are usually hypovascular.

At autopsy, the liver metastases are found in around 40% of individuals with extrahepatic cancer. As a result, proper characterization of liver metastases is crucial for establishing therapy and prognosis[41]. When it comes to imaging liver abnormalities, including metastases, MR is considered as primary imaging modality at many institutions.

Metastases appear to be hypointense on T1WI and hyperintense on T2WI. On the other hand, metastases that include cystic and necrotic areas demonstrate elevated signal on T2WI. The distinctive T2 hyperintensity of melanotic metastases is caused by the presence of paramagnetic substances. These molecules include melanin,

extracellular methemoglobin, and protein. It is also seen that metastatic lesions resulting from necrotic or mucinous tumours are seen to have high ADC levels. According to the findings of the vast majority of research, the ADC levels of metastasis and HCC were the lowest when compared to those of the other malignant hepatic lesions.

On dynamic post-contrast imaging, metastases are categorised as hypervascular, iso-vascular, or hypovascular based on whether or not they enhance more than the background liver parenchyma during the late arterial hepatic phase.

When it comes to identifying tiny hepatic metastases, hepatocyte-specific agents (HAS) and DWI are more sensitive than CT imaging and give more sensitivity than traditional T2-W MR. Since the metastatic tumours have functional hepatocytes, they appear hypointense during the hepatocellular phase, creating a sharp distinction between enhancing liver tissue and metastases. Hence the detection of small liver metastases is more accurate and sensitive when HSAs and DWI are combined than when single magnetic resonance sequence is used[42 43].

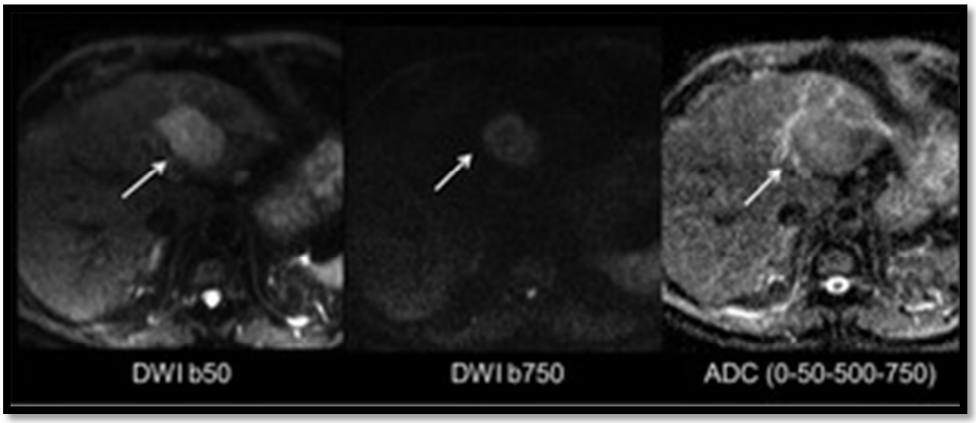


Fig: 6 75-year-old man with metastatic colon cancer. On axial DWI, lesion appears hyperintense on b50 and b750 and intermediate signal on ADC value (1.55×10^{-3} mm/s).

Normal MRI Imaging of the Liver:

The appearance of normal liver on MRI depends on the applied pulse sequence. On T1 weighted images, normal liver appears hyperintense compared to the normal spleen but shows lesser signal intensity than the pancreas. On T2 weighted images, normal liver appears hypointense compared to normal spleen.

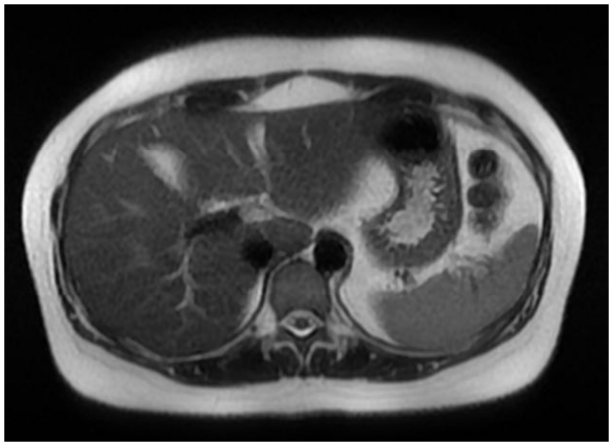


Fig 7. T2WI of the liver

Proposal in clinical setting: Jaundice, hepatomegaly, unexplained fever, abnormal liver function tests can be indicative of a more serious underlying condition. When a mass lesion is seen in a patient with hepatitis B or C infection, associated with chronic liver illness, it is fair to suspect malignancy such as HCC.

Lesions smaller than one centimeter are almost always discovered incidentally. These lesions, which can be cysts, hemangiomas, or biliary hamartomas and are absolutely innocuous.

The major method used to categorise lesions is based on whether they are solid (i.e., benign or malignant tumours) or cystic (cysts, abscesses). Examples of benign tumours with arterial hypervascularization include adenomas and focal nodular hyperplasia (FNH), while examples of malignant tumours with the same

include hepatocellular carcinoma (HCC), metastases from neuroendocrine tumours, and hypernephroma. In most cases, abdominal CT with intravenous (IV) contrast and MRI with vascular (gadolinium) contrast media are sufficient for diagnosis and decision making without the need for histologic confirmation.

Because of the consistent development and improvement in MRI over the past 15–20 years, it is now a reliable imaging modality for the liver. The modifications that have taken place are the result of the development of new technologies as well as the introduction of alternate intravenous contrast media. Phased array coils and systems are able to provide these benefits. It is the only organ for which, in addition to the general, nonspecific Gadolinium-based contrast agents, that are specific to the tissue of interest have been developed.

T2 weighed imaging:

The imaging techniques used for T2WI are the same whether a moderate or strong magnetic field is present. Whether for conventional TSE imaging while the patient is breathing normally or for breath-holding single-shot imaging, turbo or fast SE (TSE or FSE) pulse sequences are nearly always utilised to scan the liver. When utilising the free breathing approach, a picture of the liver can be acquired in approximately two to four minutes, but when using the single-shot method[39], an image of the liver can be obtained in about thirty seconds[44]. It is essential to keep in mind that extremely small lesions that have T2 relaxation durations that are noticeably longer than those of the liver are usually more difficult to recognise on TSE images in comparison to standard T2WI SE images. The echo planar imaging (EPI) technique, which is fundamentally a T2WI method, makes it feasible to obtain an image of the liver in fewer than five

seconds. Another quick sequence for T2W imaging is based on the steady-state concept underlying free precession (e.g., TrueFISP). Even though this technique is utilised for T2W imaging of the liver, it appears to be subpar in comparison to other quick methods (such as HASTE) for imaging the heart and other organs that are constantly in motion.

T2-weighted imaging reveals a lot of detail because the signal decay from mobile protons, like those in a tiny water molecule, takes a long time on the detector plane. T2-weighted imaging reveals a lower signal strength for protons that are coupled fat molecules that are bigger and less moving. This is because the signal for these protons decays more quickly, which is demonstrated by a smaller T2 value. Hemorrhage can change the local magnetic fields and cause susceptibility effects. Some of these alterations have the potential to alter the T1 or T2 relaxation rate of the tissue, making them visible on routine T1- and T2-weighted imaging. Nevertheless, it is not always possible to detect or define the lesion based purely on changes in T1 and T2 relaxation. This is because T1 and T2 relaxations are dependent on one another. Highly T2-weighted conventional spin-echo (SE) MR images can be utilised to distinguish benign from malignant nonsolid lesions such hemangiomas and cysts.

Magnetic resonance imaging's (MRI) ability to visualise variations in the diffusion characteristics of tissue water has evolved as a powerful and multipurpose tool for characterising tissue structure and diagnosing and discriminating disease processes[45].

Each proton in the water molecule experiences a different-sized phase shift as a result of the initial gradient pulse. These changes are sensitive to the position of the molecule in relation to the gradient. The phase shift will be eliminated as long as the

water molecule remains in the same position between the first and second gradient pulses; otherwise, the phase shift will be introduced (which has the opposite effect of the first). It is possible that the signal from a specific location could be lost if the molecule of water moved between the first and second gradient pulses. Additionally, it has been shown that the strength of a signal's attenuation is related to the size of the motion component along the same axis as the diffusion gradient. If the motion is perpendicular to the gradient, there would be no degradation of the signal.

Non-diffusion-related motion artefacts may have a deleterious impact on DW-MRI. Displacements much larger than those to which DW-MRI has been sensitised can be produced by naturally occurring motions within the body, such as respiration, intentional movement, and even arteriole level perfusion. To account for bulk motion, "snap-shot" imaging techniques had to be developed, and echo-planar imaging this far has been the most successful technique. While single-shot systems are robust against motion, they are especially vulnerable to variations in the magnetic field. This causes blurring and artefacts in areas with large fluctuations in magnetic susceptibility, such as the interface between air and tissue or the chemical-shift effect, which is most evident at the fat-water interface. In addition, single-shot methods have a lower resolution than multi-shot methods.

DWI is a method of functional imaging that works by tracking the Brownian motion of individual water molecules. The architecture of a tissue creates obstacles for extracellular water molecules that are aimlessly travelling through the tissue. In densely packed tissue, there is a greater possibility that the cell membranes will act as a barrier to the diffusion of extracellular water. As a result of the reduced length of the ADC's diffusion path, the size of the ADC will be reduced. In necrotic or cystic

tumous, on the other hand, where there are less structural barriers, a high ADC value would be linked with the diffusion channel. This implies that ADC maps, which are created from diffusion-weighted imaging, might offer a method of examining cellularity without the use of any invasive techniques. b-values between 0 and 1000s/mm2 are used to generate diffusion-weighted images and ADCs of superior quality is standard.

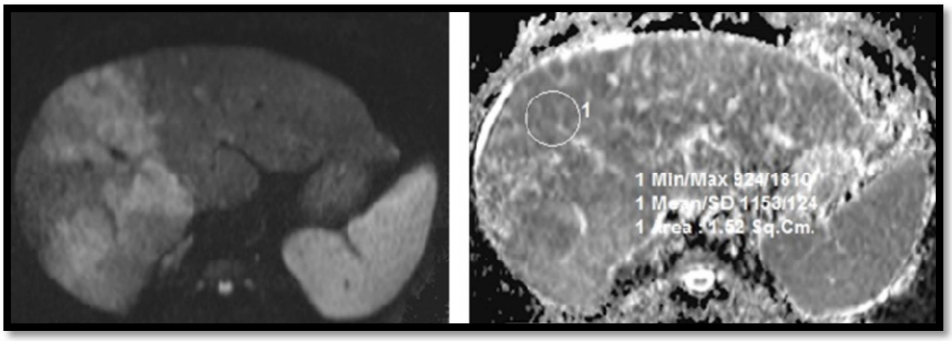


Fig.8 Axial diffusion-weighted (b value=800 s/mm2) image shows hyperintensity. Apparent diffusion coefficients (ADC) shows hypointensity compared with normal parenchyma with ADC value of about 1.15 × 10-3 mm^2/s. Case of hepatocellular carcinoma.

Intravascular space was found to be the source of a significant portion of the signal on the DWI. There is a clear correlation between the degree to which water diffusion is impeded and the cellularity of the tissue as well as the integrity of the cell membranes. Hypercellularity and expanded nuclei are two characteristics that are unique to malignant lesions. These histopathological characteristics lower the ADC value because they reduce the amount of extracellular matrix and the amount of space available for the diffusion of water protons in the regions outside of the cells. Therefore, it is possible to identify benign and malignant liver lesions with the use of the ADC values.

In a study conducted by Filipe et al. the cut- off value used to differentiate benign and malignant lesions was 1.43 x 10^{-3} mm^2 /s. He also concluded that the ADC value of malignant lesions is significantly lower compared to benign lesions[46].

In addition to playing a part in the detection and characterization of tumours, DWI has also shown promise as a tool for predicting and monitoring the efficacy of treatment interventions. Increasing ADC values have been consistently associated with positive treatment outcomes in clinical investigations[47].

In the vast majority of trials, researchers discovered that malignant tumours had ADC values that were much lower than those of benign tumours. This technique does not utilise radiation, does not call for the use of intravenous contrast material, the expediency with which it can be applied, and can provide quantitative information regarding tissue are among the most notable advantages. Patients diagnosed with renal dysfunction might develop nephrogenic systemic fibrosis if gadolinium is given, hence contrast should be avoided[48].

Recently, there has been a lot of interest brought on by the effectiveness of DWI in oncological applications. It has been determined through research that the diagnostic performance of DWI in patients with HCC and metastatic liver disease is comparable to that of contrast MRI.

Standard T2 weighted imaging might make it difficult to locate small lesions surrounding arteries and in the periphery of the liver. However, DWI makes it simple to locate these lesions. It has been shown to be effective in the detection of metastases that are less than 10 millimetres in size. Along with multiphase contrast enhanced-MRI techniques, DWI better depicts metastatic liver lesions, and is more sensitive than gadoxetic acid-enhanced MRI (90.6-95.4%) when used alone.

DWI has also been helpful in facilitating the differentiation of cirrhotic hepatocellular nodules. DW-MRI has also shown to be beneficial in patients with locally progressed HCC by determining whether the venous thrombus is benign or a tumour thrombus caused by HCC suggesting angioinvasion. In comparison to a bland thrombus, tumour thrombus and HCC have been shown to have an ADC ratio that is lower than 2.

Recently, DWI has been utilised in an effort to make a prediction regarding the histopathological grades of HCC. Microvascular invasion and histological sub-type maturation are found to correlate with ADC levels. The ADC of poorly differentiated HCCs is also shown to be much lower compared to that of HCCs with a higher degree of differentiation. Microvascular invasion can be predicted using a cutoff value of 1.1750×10^{-3} mm2/s.

After radiofrequency ablation of hepatic primary tumours and metastases, it may be possible to detect local tumour progression and post-treatment tissue changes unrelated to the tumour by analysing signal variations proximal to the ablation zone using ADC. This would be done in order to determine whether or not the progression is related to the tumour. In the early post-ablation zone, there may be a heterogeneous signal on unenhanced T1 and T2WI as a result of edema, haemorrhage, and the inflammatory response. Four to six months following the ablation, a high T1 and a low T2 signal (coagulation necrosis) continue to be present; however, they eventually revert to normal. It is indicative of a local recurrence if there is a reappearance of nodular enhancing foci inside the ablation zone. Patients who have ADC values (one month after RFA) that are lower than 1.145×10^{-3} mm2/s are at an elevated risk for a local recurrence of HCC.

REVIEW OF STUDIES:

[49]In a large case series that Frank and his colleagues worked on, apparent diffusion coefficient values were used to characterise the various types of focal liver lesions and, in particular, to distinguish solid benign lesions from solid malignant neoplasms. This was done in order to better treat patients with solid malignant neoplasms. The ADC value for cysts was 3.40, while the ADC for hemangiomas was 2.26, the ADC for FNH was 1.79, the ADC for adenomas was 1.49, the ADC for abscesses was 1.97, and the ADC for HCC was 1.53 x $10^{-3}mm^2/s$. The average ADC for benign lesions was 2.50, whereas the average for malignant ones was 1.52. It was quite easy to differentiate cysts from other types of lesions. Tumors of solid tissue, whether benign or malignant, did share several characteristics, though.

[50]According to Demir et al., the average ADC value of a hemangioma was calculated to be 2.46 x $10^{-3}mm^2/s$ when applying a gradient of 1,000 s/mm^2. The average ADC for b-600 among the 61 hemangiomas in their study was 1.98 x $10^{-3}mm^2/s$.

[49]Recent research conducted by Miller and colleagues evaluated ADC levels to characterise a variety of localised liver lesions. Mean ADC values (with b-0 and b-500 s/mm2 values) were 1.53 x 10^{-3} mm^2/s, 1.50 x 10^{-3} mm^2/s, and 1.79 x 10^{-3} mm^2/s, respectively, for hepatocellular carcinoma, metastasis, and focal nodal hyperplasia, respectively. Scientists found that benign tumours had considerably elevated ADC levels than malignant ones. Conversely, a FNH benign lesion's ADC value is not noticeably different from that of a malignant lesion.

[51]Focused MRI of the liver (hepatic MRI) is often used to categorise focal liver lesions (FLLs) as benign or malignant, and Lidia Ciobanu conducted research to evaluate the diagnostic utility of DWI. The average ADC ratio was 1.91 for non-cancerous FLLs and 1.85 for cancerous FLLs, according to the two readers. To know if FLLs are benign or malignant, the ROC analysis with ADC ratio is the best indicator. Liver DWI may help identify FLLs, and the ADC value and ADC ratio can help distinguish between benign and malignant FLLs.

[52]Haradome H et al proved that accuracy was more when both the sequences are combined (80.3%) than individual sequences, T2WI set (68.8%), and DWI set (73.2%). But there was not much difference between the T2WI and DWI sets.

[53]Elbarbary AA et al conducted a study was conducted in 40 patients. The study shows that low ADC values were noted in metastases, cholangiocarcinoma's, hepatocellular carcinomas and the highest values were noted in hemangiomas. However, no significant difference was found amongst the different benign and malignant lesions. The study concluded that DWI alone performs equally well as contrast enhanced MRI in detection and differentiation of different hepatic focal lesions in cases where contrast cannot be given.

[54]Javadrashid R et al conducted a prospective study in 93 patients referred to study the sensitivity and specificity of diffusion-weighted imaging (DWI) along with ADC values in discriminating benign lesions from malignant ones. Mean ADC values for benign and malignant hepatic lesions were found to be 1.58 ± 0.35 (10^{-3} mm^2/s) and 0.87 ± 0.16 (10^{-3} mm^2/s s), respectively. DW imaging had a sensitivity of 97.6% and specificity of 98.7% in detecting malignant hepatic lesions from benign ones.

[55]Paley did this study to determine whether or not differences in signal intensity and lesion-to-liver contrast brought about by ferumoxides-enhanced MRI of localised hepatic lesions may be utilised to differentiate between benign and malignant lesions. Ferumoxides-enhanced T2-WI showed considerable signal reduction in localised nodular hyperplasia. Few hepatic adenomas and hepatocellular carcinomas showed a statistically significant decrease in signal intensity on ferumoxides-enhanced T2-WI. T2-WI enhanced with ferumoxides demonstrated a considerable increase in lesion-to-liver contrast for metastases and hemangiomas, but not for hepatocellular carcinoma. Significant attenuation of signal on ferumoxides-enhanced T2-WI distinguishes focal nodular hyperplasia from other localised hepatic lesions. Reticular endothelial uptake is not unique to focal nodular hyperplasia, even though it is present at lower levels in this condition. Hepatocellular adenoma and carcinoma also share this feature.

[56]Elizabeth MacFarland performed research on a 1.5-T MRI to see if more heavily T2-weighted conventional spin-echo sequences help distinguish between benign and malignant tumours. Malignant hemangiomas had a mean T2 time of 76 msec, whereas hemangiomas had a mean T2 duration of 142 msec 40 and cysts had a mean T2 duration of 341 msec. With a cutoff T2 value of 112 msec, we were able to distinguish hemangiomas from malignant tumours with a 97% accuracy, 100% sensitivity, and 92% specificity. Differentiating between benign lesions like hemangiomas and malignant ones like lymphomas is possible by evaluating T2 relaxation times with a more highly T2-weighted sequence.

MATERIALS AND METHODS

Source of data: The study population includes patients referred to do MRI abdomen study to diagnose and/or to confirm the ultrasonographic or CT findings of focal hepatic lesions, presenting to the Department of Radio-Diagnosis at the KLE's Dr. Prabhakar Kore Hospital & MRC, Belagavi.

Method of collection of data:

a) **STUDY DESIGN:** Hospital based prospective study

b) **SAMPLE SIZE:** The minimum sample size formula based on prevalence rate is

$$n = \frac{z_\alpha{}^2 P(1-P)}{d^2}$$

where, P is the percentage of prevalence and d is the percentage likely difference in the prevalence.

zα is linked with the level of significance. For 5% level of the significance zα = 1.96.

Ref:

With P = 69.88%3 and d = 17.47% of P = 7.50%, the sample size is 26.

To make the study more confirmative, the sample size will be raised to 30.

c) **STATISTICAL ANALYSIS:** Since the study is observational, the plan of analysis will be as follows.

For the continuous quantitative variables mean and standard deviation will be calculated.

Categorical data will be represented in the form of frequency and percentage. The agreement between the variables is assessed using kappa coefficient.

Suitable graphs will be used to depict the comparison.

d) For all the tests the value of p less than 5% (0.05) will be considered significant.

e) SAMPLING METHOD: Universal sampling

f) STUDY DURATION: 1st January 2021 to 31st December 2021 (12 months)

g) INCLUSION CRITERIA:

- All patients referred for MRI abdomen with clinically suspected focal liver lesions and patients with incidental findings detected on Ultrasonography or Computerized tomography (CT).
- Age group: 15-90 years.

h) EXCLUSION CRITERIA:

- Having no FLL.
- Having FLLs with a maximum diameter of less than 5 mm
- Regional therapy (radiofrequency ablation) or systemic chemotherapy before MRI, which could affect signal of the lesions at DWI.

- All patients having cardiac pacemakers, prosthetic heart valves, cochlear implants or any metallic implants.

METHODOLOGY

- All patients referred to the Department of Radio diagnosis at KLE's Dr. Prabhakar Kore Hospital & MRC, Belagavi.

- Patients of age group 15-90 years referred to MRI abdomen clinically suspected of focal liver lesions and with incidental findings detected on USG or CT in a period of 1 year will be subjected for the study.

- Abdomen will be assessed in axial, sagittal and coronal planes.

- Recommended sequences: T1WI, T2WI in axial and coronal plane, DWI and Post contrast dynamic study (whenever indicated).

- Patients are followed with biopsy of the lesion or surgery or follow up after six months at KLE hospital.

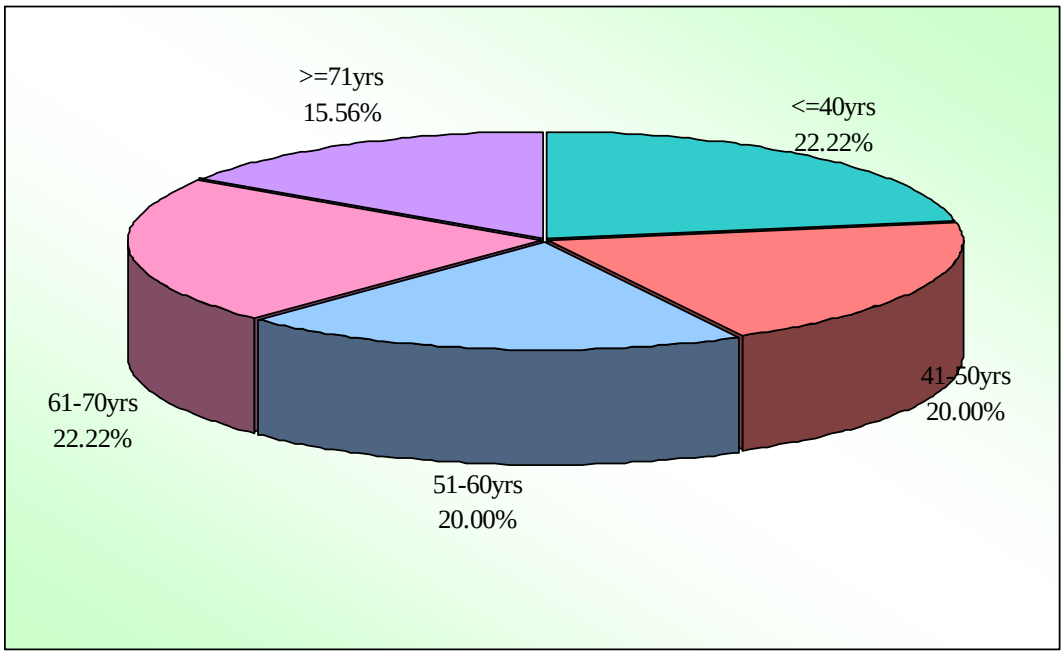
- Informed consent-Written informed consent will be taken from each participant prior to the study.

RESULTS

Table 1: Age wise distribution of patients

Age groups	No of patients	% of patients
<=40yrs	10	22.22
41-50yrs	9	20.00
51-60yrs	9	20.00
61-70yrs	10	22.22
>=71yrs	7	15.56
Total	45	100.00
Mean age	53.57	

Figure 1: Age wise distribution of patients

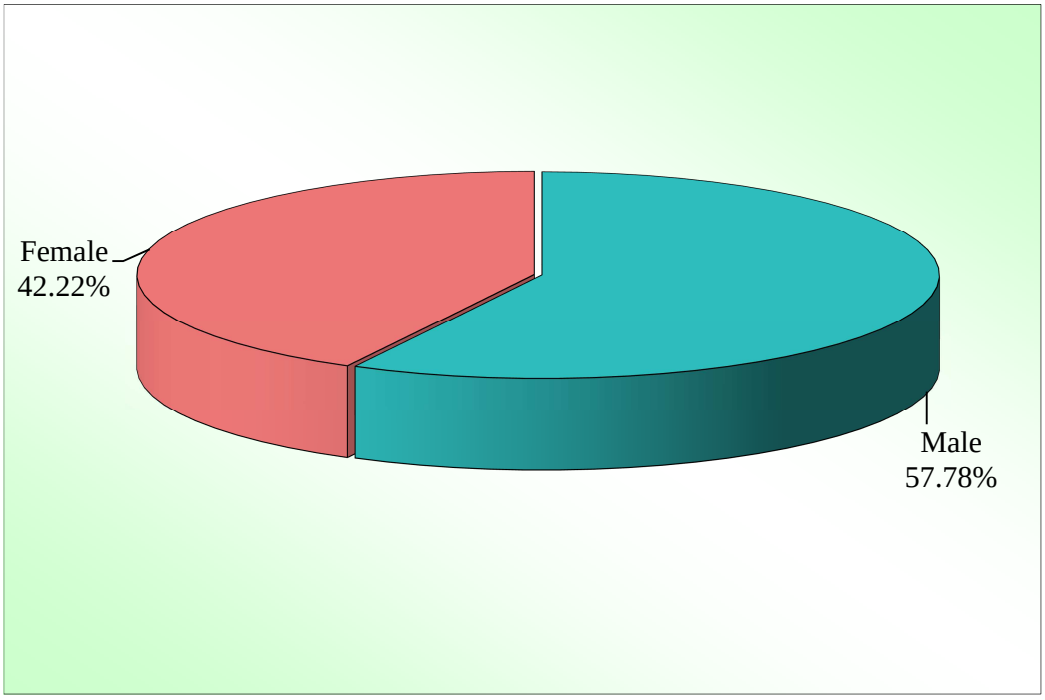


This study included 45 patients of which 78% of the patients are above 40 years of age. The prevalence of focal liver lesions is more in this age group.

Table 2: Gender wise distribution of patients

Gender	No of patients	% of patients
Male	26	57.78
Female	19	42.22
Total	45	100.00

Figure 2: Gender wise distribution of patients

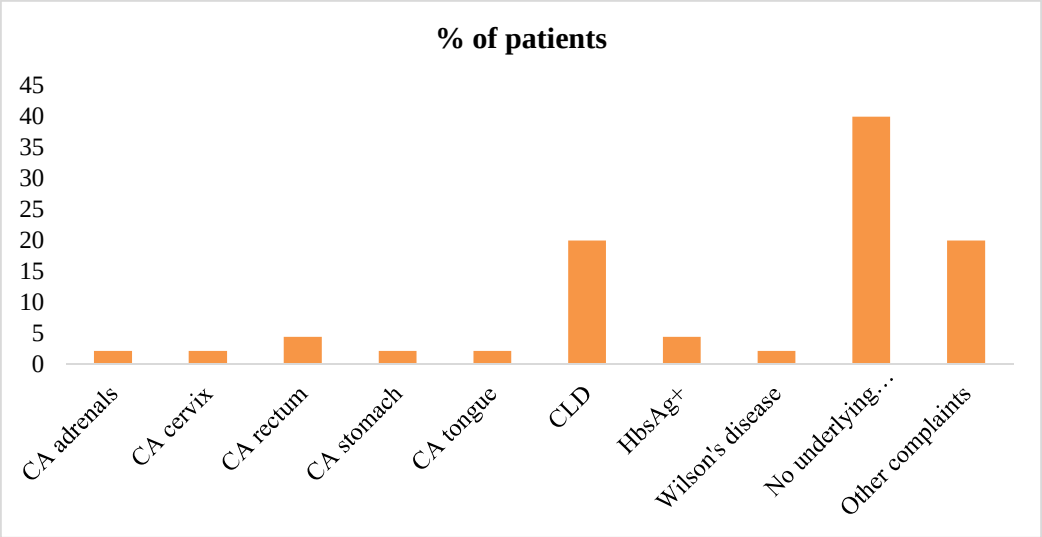


Out of 45 patients with FLL's, 26 are males accounting for 57.7% of the sample and 19 are females accounting for 42.2% of the sample.

Table 3: Associated comorbidity of the patients

Associated comorbidity	No of patients	% of patients
CA adrenals	1	2.2
CA cervix	1	2.2
CA rectum	2	4.4
CA stomach	1	2.2
CA tongue	1	2.2
CLD (chronic liver disease)	9	20.0
HbsAg+	2	4.4
Wilson's disease	1	2.2
No underlying comorbidities	18	40.0
Other complaints	9	20.0
Total	45	100

Figure 3: Associated comorbidity of the patients

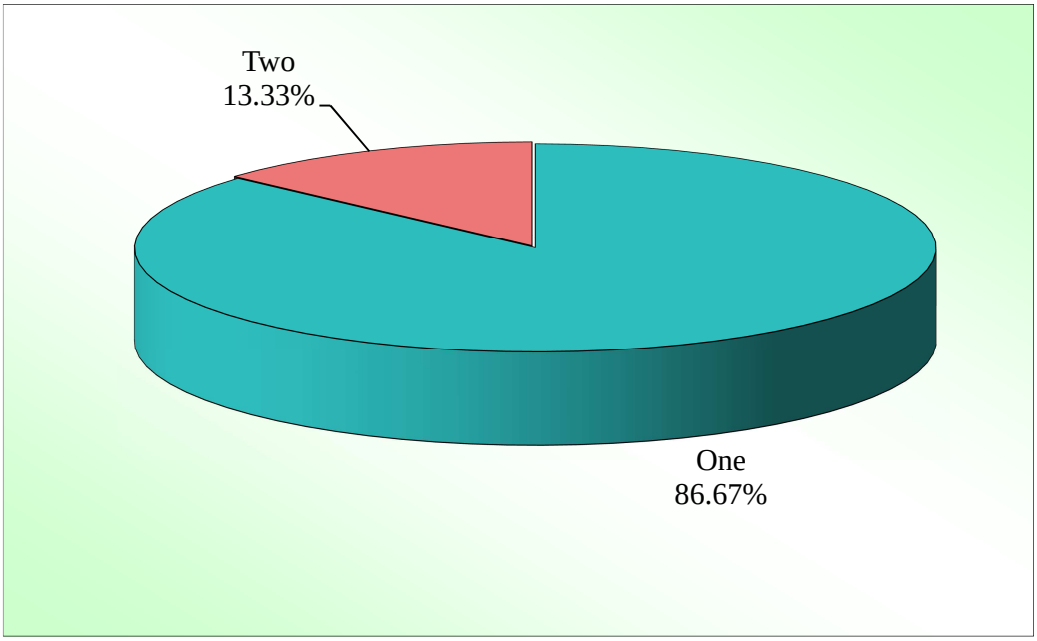


20% of the patients had associated CLD, followed by hepatitis-b, carcinoma rectum etc.

Table 4: No of types lesions wise distribution of patients

No of lesions present	No of patients	% of patients
One	39	86.67
Two	6	13.33
Total	45	100.00

Figure 4: No of types of lesions wise distribution of patients

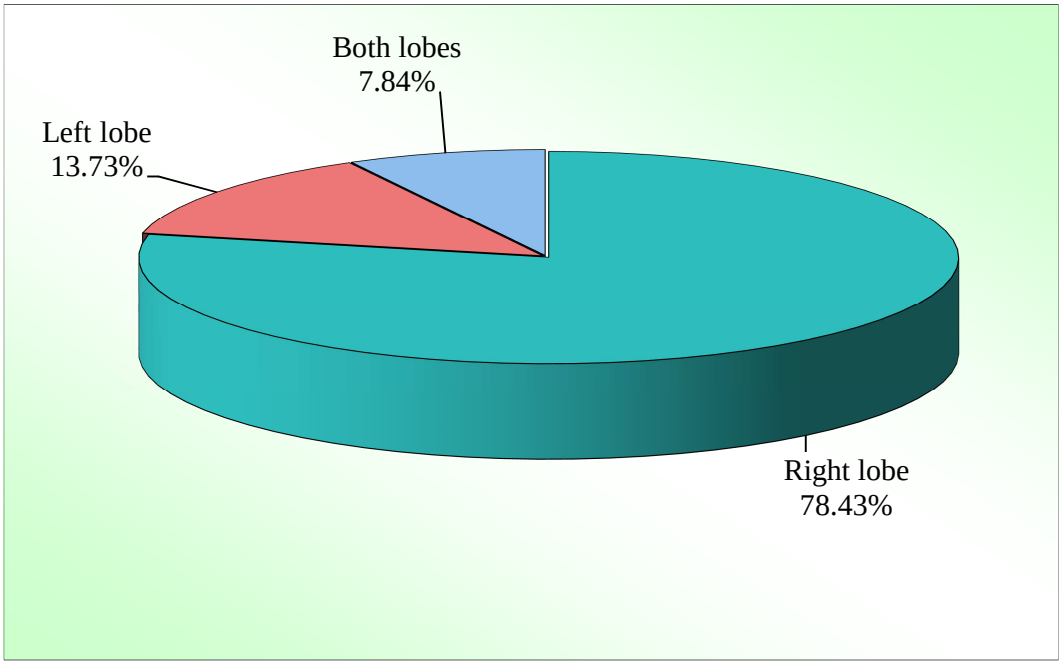


In this study, 39 out of 45 patients had one type of lesion and 6 patients had 2 types of lesions.

Table 5: Lobe/segment wise distribution

Lobe/segment	No of patients	% of patients
Right lobe	40	78.43
Left lobe	7	13.73
Both lobes	4	7.84
Total	51	100.00

Figure 5: Lobe/segment wise distribution



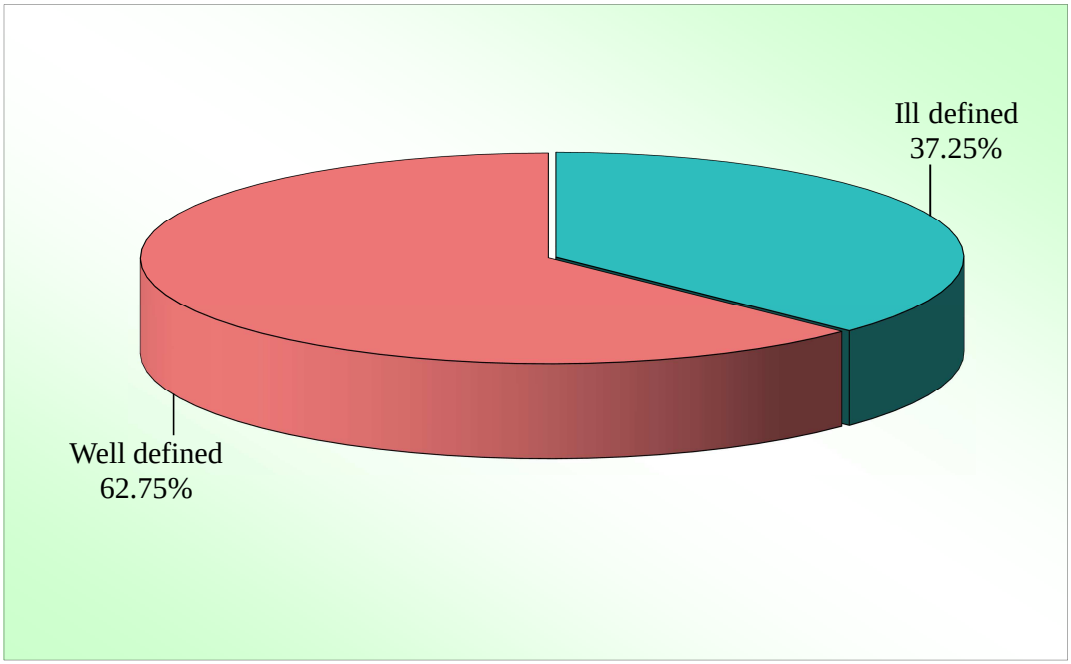
40 out of 51 patients had lesions in the right lobe of the liver, 7 patients had lesions in the left lobe of liver and 4 patients had lesions in both the lobes.

T2 WI findings

Table 6: Status of margin of lesions on T2WI

Margin	No of lesions	% of lesions
Ill defined	19	37.25
Well defined	32	62.75
Total	51	100.00

Figure 6: Status of margin of lesions on T2WI

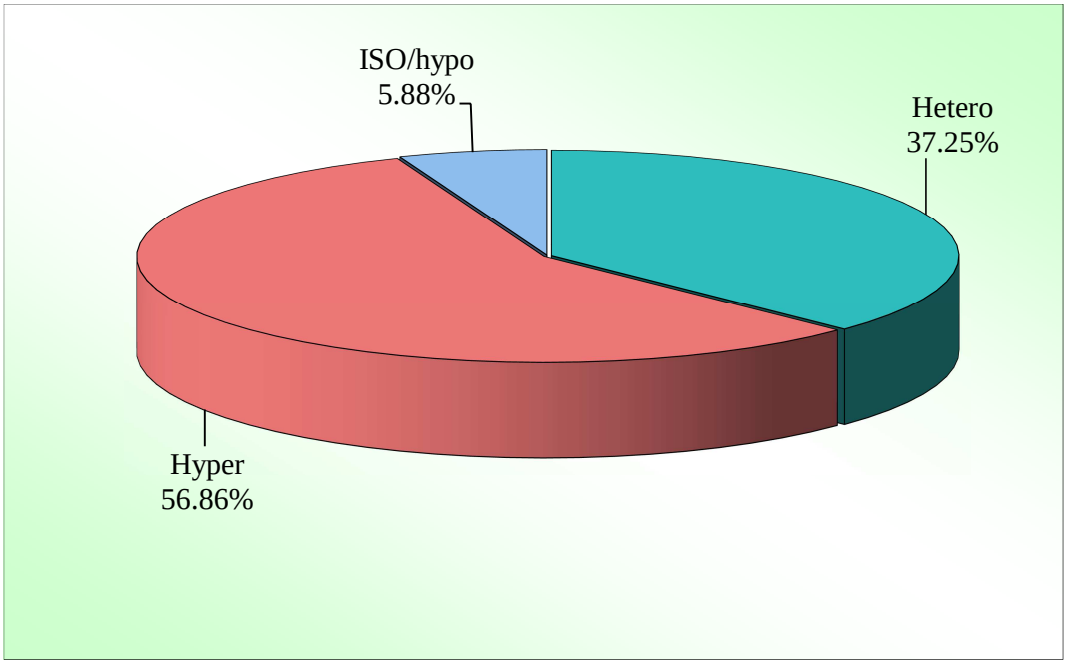


On TW2I, 32 out of 51 lesions had well-defined margins and the rest of the lesions had ill-defined margins.

Table 7: Status of signal intensity on T2WI

Signal intensity	No of lesions	% of lesions
Hetero	19	37.25
Hyper	29	56.86
Iso/hypo	3	5.88
Total	51	100.00

Figure 7: Status of signal intensity on T2WI

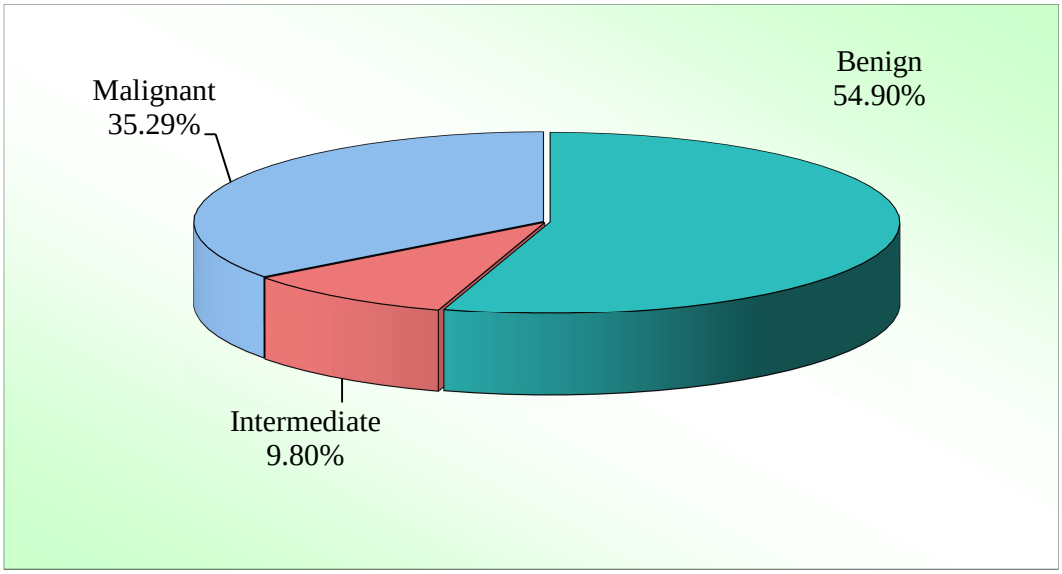


On TW2I, 29 out of 51 lesions were hyperintense, 19 lesions had heterogeneous signal intensity and the rest had iso-hypointensity.

Table 8: Status of lesion character (T2 WI)

Lesion character	No of lesions	% of lesions
Benign	28	54.90
Intermediate	5	9.80
Malignant	18	35.29
Total	51	100.00

Figure 8: Status of lesion character (T2 WI)



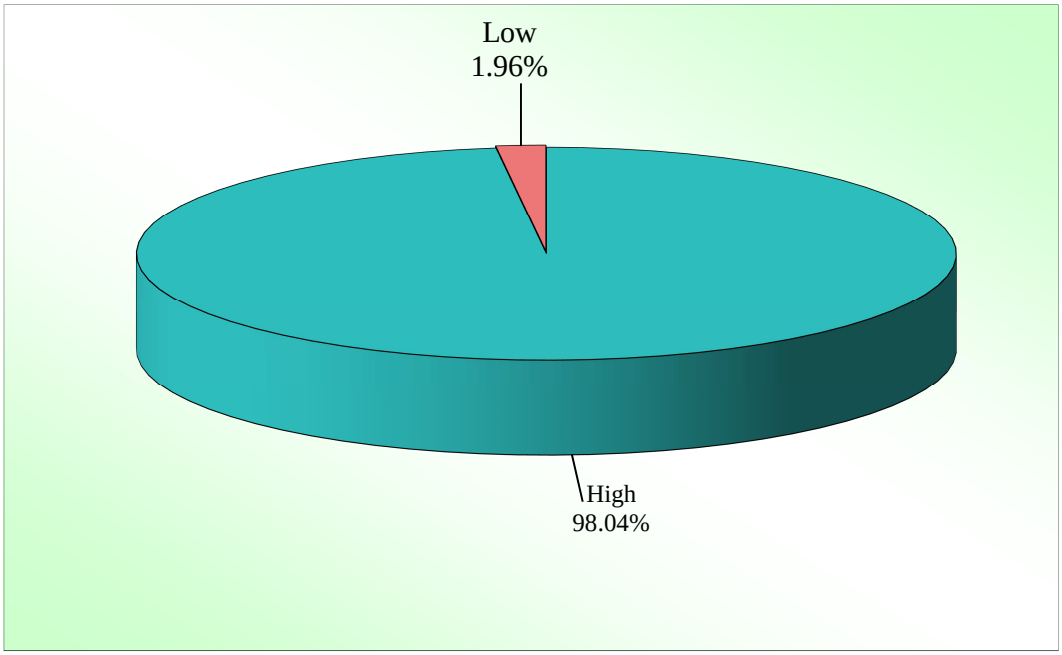
On T2WI, 28 lesions considering benign, 18 lesions are considered malignant and 5 lesions as intermediate based on the lesion margins and signal intensity.

DWI findings

Table 9: Status of lesions at 50 mm/s^2

50 mm/s^2	No of lesions	% of lesions
High signal	50	98.04
Low signal	1	1.96
Total	51	100.00

Figure 9: Status of 50 mm/s^2 of lesions

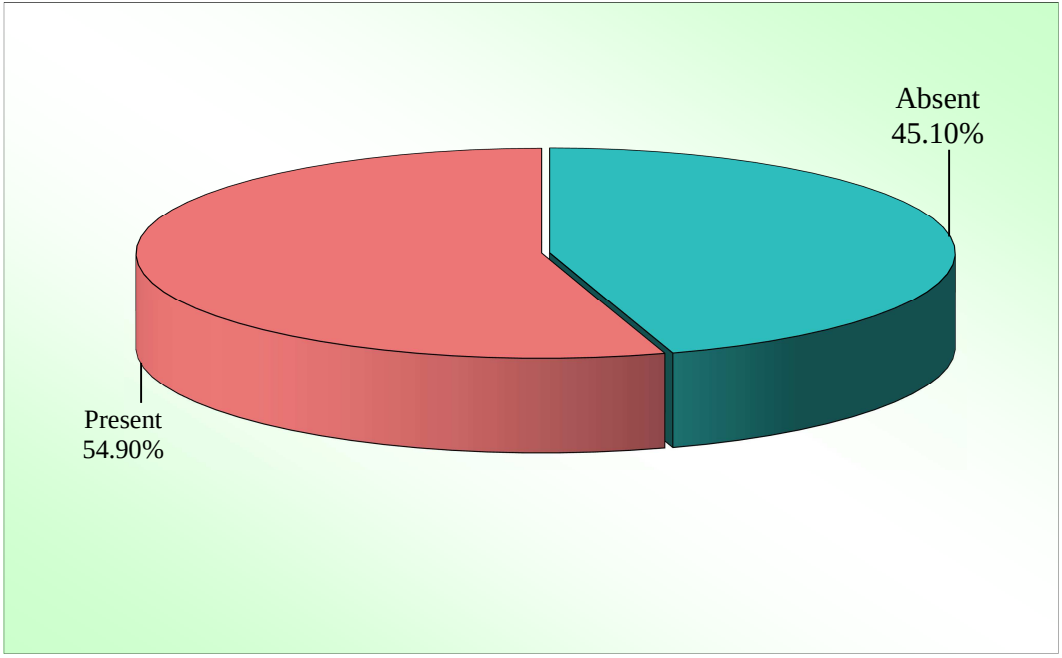


In this study, 50 out of 51 lesions had high signal on DWI at 50 mm/s^2.

Table 10: Status of signal drop at 400 and 800 mm/s^2 of lesions

Signal drop at 400 and 800 mm/s^2	No of lesions	% of lesions
Absent	23	45.10
Present	28	54.90
Total	51	100.00

Figure 10: Status of signal drop at 400 and 800 mm/s^2 of lesions

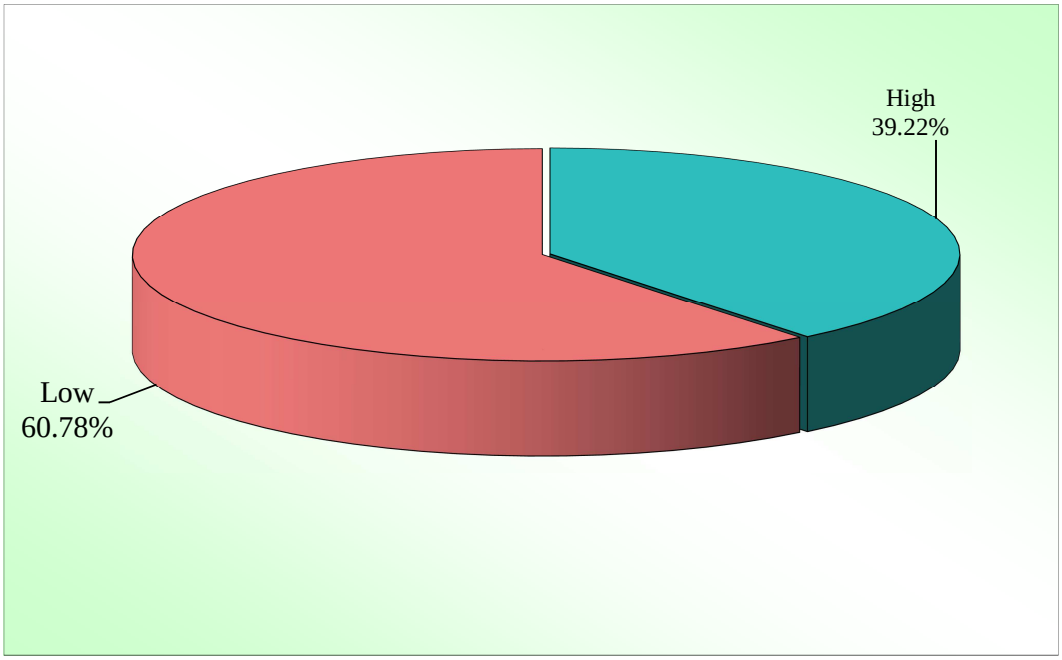


On DWI, signal drop at 400 and 800 mm/s^2 is seen in 28 lesions. Rest of the lesions (23) showed persistent high signal.

Table 11: Status of ADC of lesions

ADC	No of lesions	% of lesions
High signal	20	39.22
Low signal	31	60.78
Total	51	100.00

Figure 11: Status of ADC of lesions

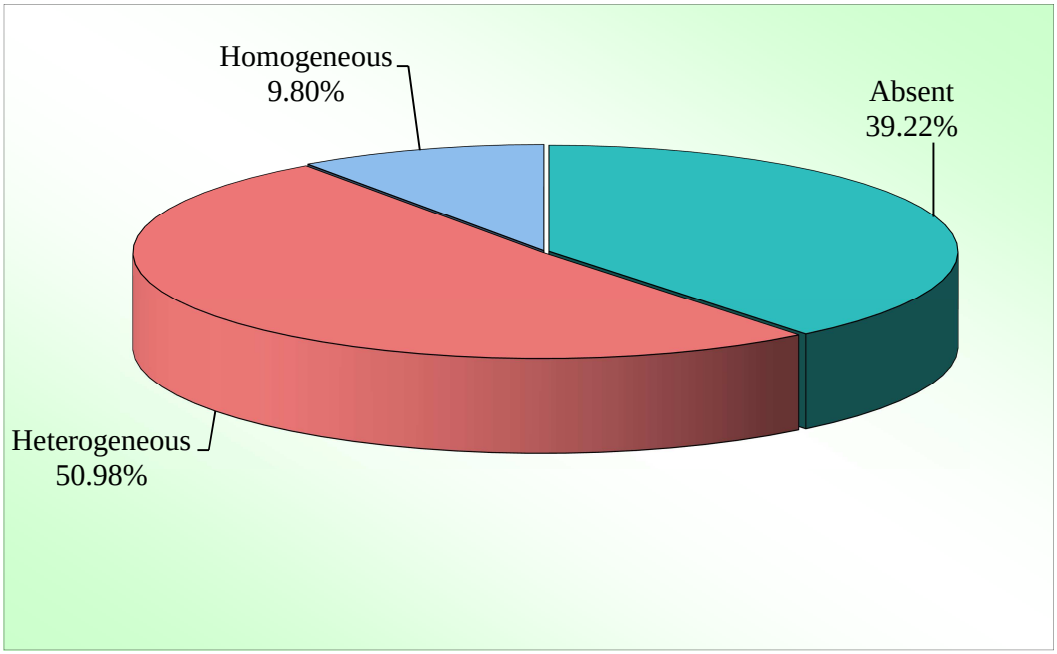


31/51 lesions showed low ADC value and rest of the lesions showed high ADC value

Table 12: Status of restriction pattern of lesions on DWI and ADC

Restriction pattern	No of lesions	% of lesions
Absent	20	39.22
Heterogeneous	26	50.98
Homogeneous	5	9.80
Total	51	100.00

Figure 12: Status of restriction pattern of lesions on DWI and ADC

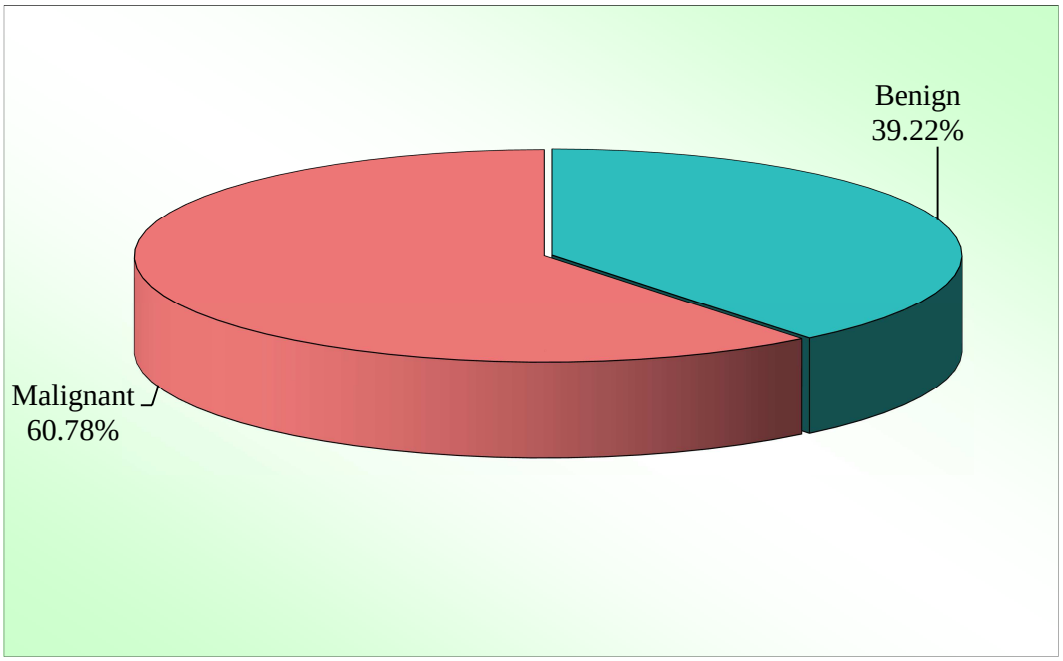


Of the 31 lesions with restricted diffusion, 26 lesions showed heterogeneous pattern of restriction and 5 lesions had homogeneous pattern.

Table 13: Status of Lesion character (DWI)

Lesion character	No of lesions	% of lesions
Benign	20	39.22
Malignant	31	60.78
Total	51	100.00

Figure 13: Status of Lesion character (DWI)

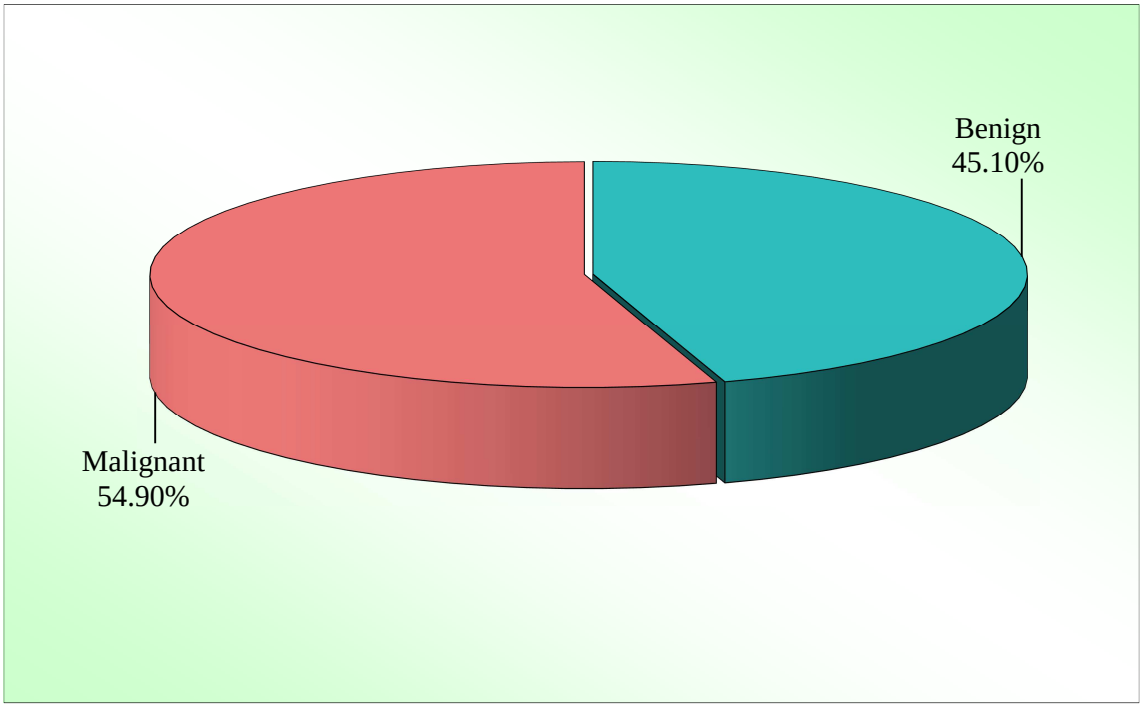


Based on the signal at DWI and ADC, 31 lesions are considered malignant since they had restriction of diffusion and the rest are considered benign.

Table 14: Status of Lesion character (T2WI AND DWI) of lesions

Lesion character	No of lesions	% of lesions
Benign	23	45.10
Malignant	28	54.90
Total	51	100.00

Figure 14: Status of Lesion character (T2WI AND DWI) of lesions

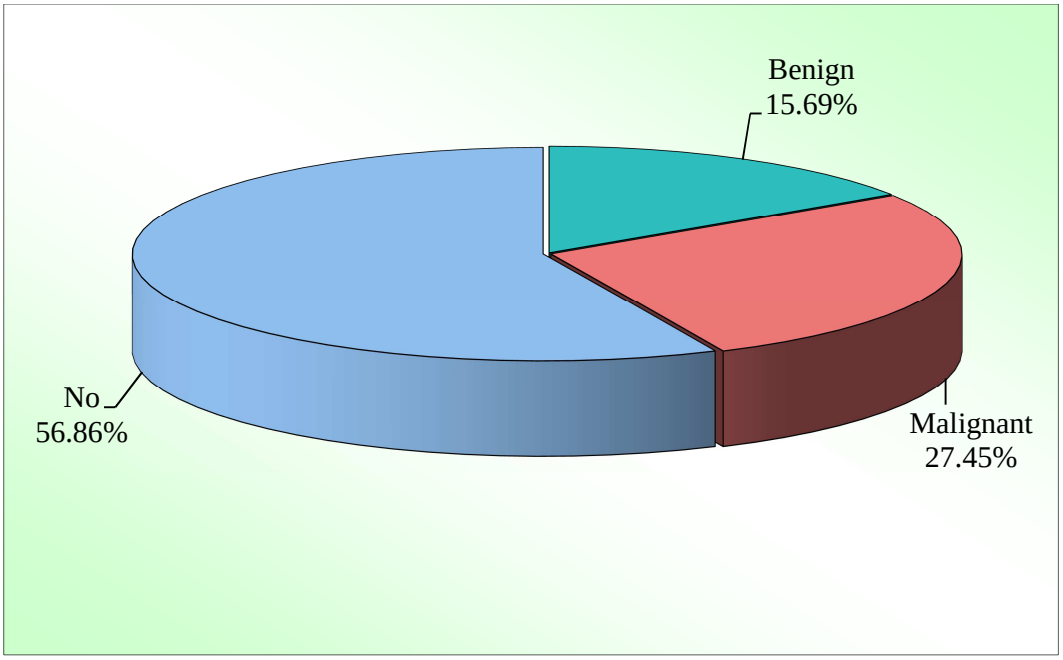


Based on both the sequences, 28 lesions are considered malignant and 23 lesions are considered benign.

Table 15: Status of BIOPSY/FNAC of lesions

BIOPSY/FNAC	No of lesions	% of lesions
Benign	8	15.69
Malignant	14	27.45
No	29	56.86
Total	51	100.00

Figure 15: Status of BIOPSY/FNAC of lesions

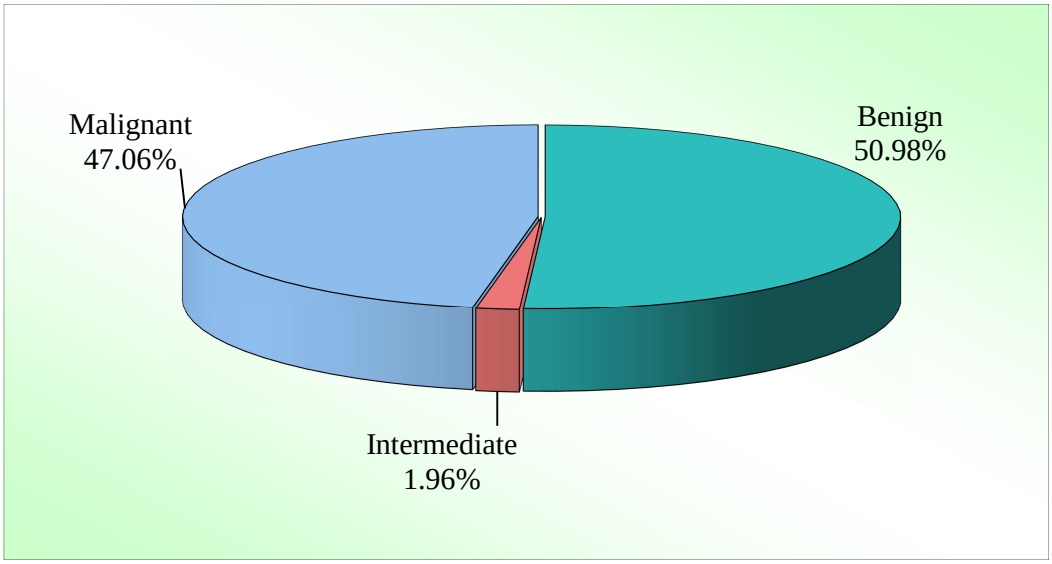


Biopsy was done for 22 lesions of which 14 lesions turned out to be malignant and 8 lesions were benign.

Table 16: Status of final lesion character

Final lesion character	No of lesions	% of lesions
Benign	26	50.98
Intermediate	1	1.96
Malignant	24	47.06
Total	51	100.00

Figure 16: Status of final lesion character

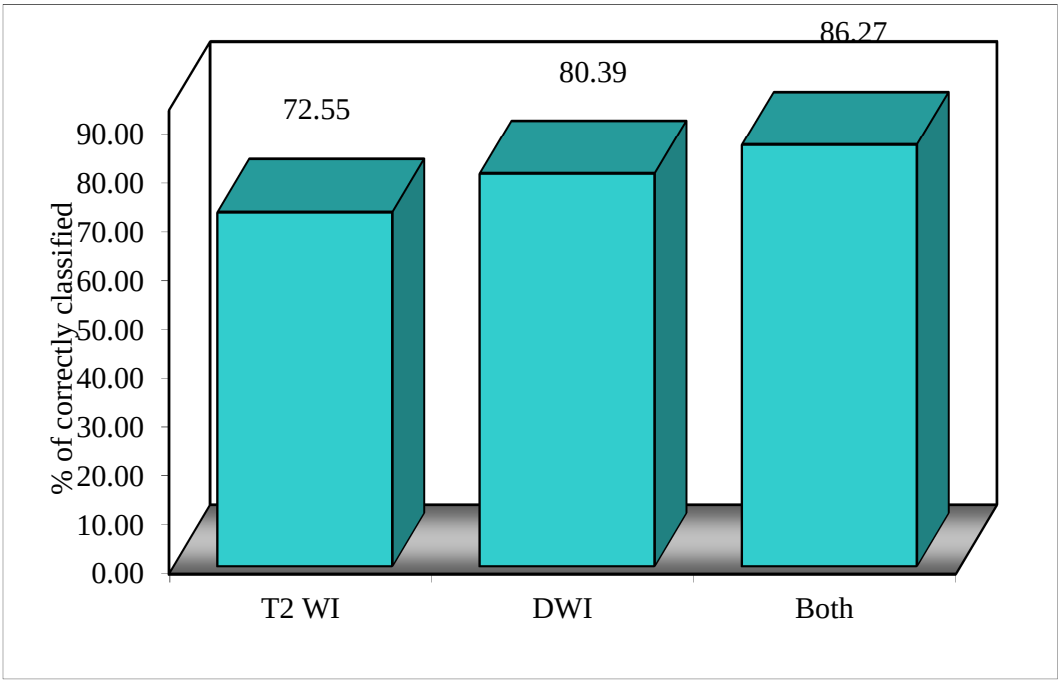


The final lesion character was based on cross-sectional imaging and histopathological correlation. 26 were benign lesions, 24 were malignant lesions and 1 lesion was intermediate.

Table 17: Percentage of lesions correctly classified

Sequences	Correctly classified	% correctly classified
T2 WI	37	72.55
DWI	41	80.39
Both	44	86.27

Figure 17: Percentage of lesions correctly classified



In this study, 37 lesions are correctly classified on T2WI, 41 lesions are correctly classified on DWI and 44 lesions are correctly classified on both the sequences together.

Table 18: Status of diagnosis

Diagnosis	No of lesions	% of lesions
Cholangitic abscess	1	1.96
Dysplatic nodule	1	1.96
Focal nodular hyperplasia	2	3.92
Hemangioma	9	17.65
Hepatic abscess	4	7.84
Hepatic adenoma	1	1.96
Hepatic cyst	8	15.69
Hepatocellular carcinoma	12	23.53
Intrahepatic cholangiocarcinoma	3	5.88
Metastasis	6	11.76
Multicentric HCC	1	1.96
Multifocal HCC	2	3.92
Regenerative nodules	1	1.96
Total	51	100.00

Figure 18: Status of diagnosis

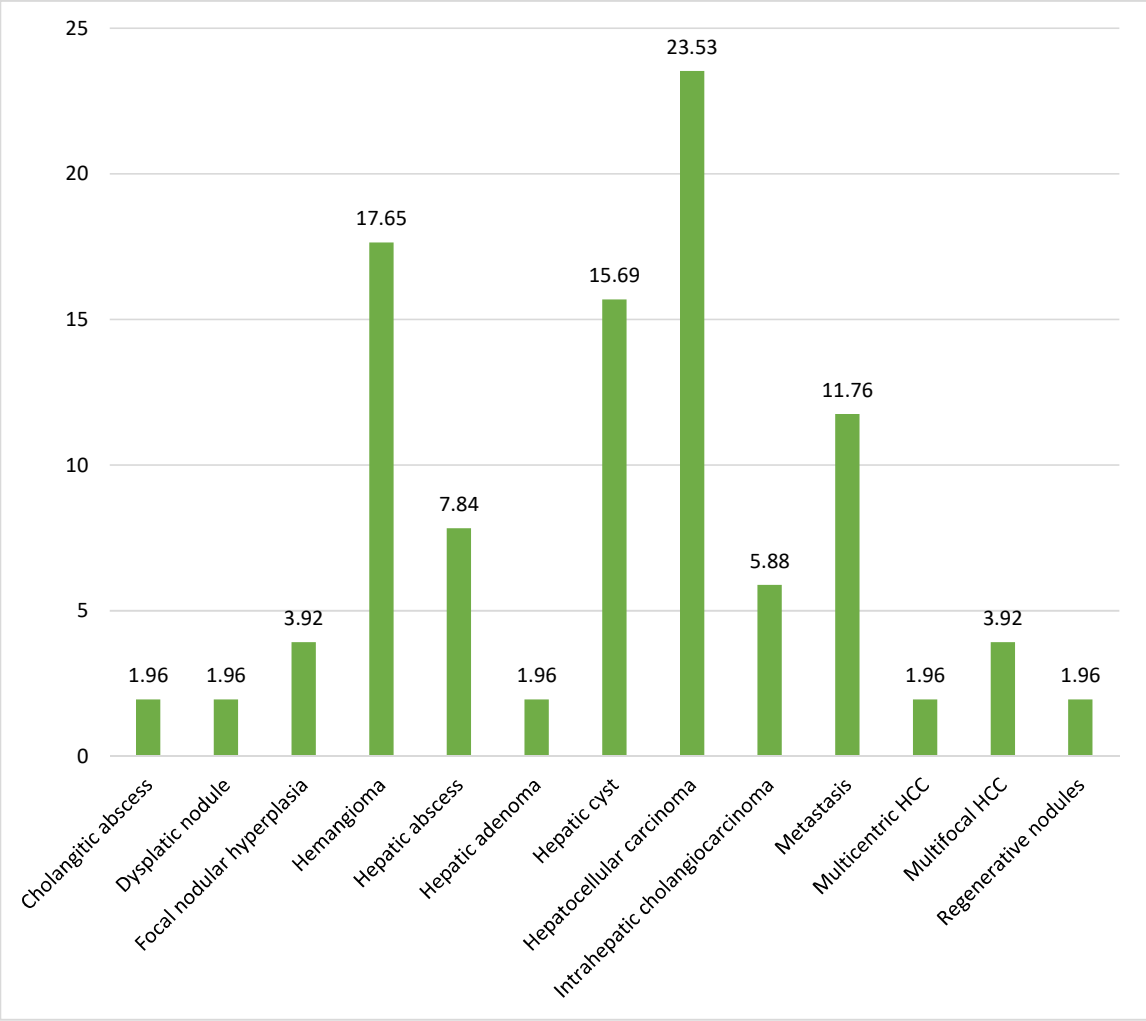


Table 19 a: Kappa agreement between lesion character (T2 WI) and final Lesion character

Agreement	Kappa	Std. Err.	Z-value	p-value
72.55%	0.4479	0.1176	4.2700	0.0001,S

Table 19 b: Kappa agreement between lesion character (DWI) and final Lesion character

Agreement	Kappa	Std. Err.	Z-value	p-value
80.40%	0.6095	0.1361	4.4800	0.0001,S

Table 19 c: Kappa agreement between lesion character (T2 WI and DWI) and final Lesion character

Agreement	Kappa	Std. Err.	Z-value	p-value
86.27%	0.7260	0.1391	5.2200	0.0001,S

DISCUSSION

In this study, the role of T2WI and DWI of liver in plain MRI abdomen is studied in 45 patients presenting with pain abdomen or in asymptomatic patients in whom routine ultrasound abdomen showed a liver lesion, between the age group 15-90 years.

MRI abdomen (plain) is done for 45 patients, of which 9 patients are known case of chronic liver disease, 2 patients are known case of hepatitis-b, 2 patients are known case of carcinoma rectum, 1 patient has carcinoma stomach, 1 patient has carcinoma cervix, 1 patient has carcinoma adrenal gland, 1 patient has carcinoma tongue and 1 patient is a known case of wilson's disease. The most commonly associated condition in this study is chronic liver disease which is seen in 20% of the patients. Rest of the 27 patients either showed a liver lesion on routine annual ultrasonography or had either come with other complaints

The median age in this study is 56. In a similar study conducted by Robert M. Hicks et al. had a median age of 63[57].

Males are more in number than females (26 out of 45 were males) in this study. In a similar study conducted by Yang DM et. al, included 45 patients in their studies in which the 30 were males and 15 were females[58].

A total number of 51 lesions are present in 45 patients, 6 patients have more than one type of lesion. In 78% of the patients right lobe of liver was involved (Of 51 lesions, 40 lesions were present in right lobe, 7 lesions were present in left lobe and 4 lesions were present in both the lobes of liver). In a study conducted by KOTNIS S et.

al, 64% of the patients had lesion in right lobe of the liver (27 out of 38 patients had lesion in right lobe of the liver)[59].

Based on the margins and signal intensity on T2 weighted sequence[52], the status of lesion character is categorized into benign/malignant/intermediate. The lesion is considered benign if the margin is well-defined and showed homogenously high signal intensity on T2, it is considered malignant if it has ill-defined margins with iso or low signal intensity and is considered intermediate if the lesion has ill-defined margins with homogenously high signal intensity or if the lesion has well defined margins with heterogenous low/ isointensity[56].

Of 51 lesions, 29 lesions are homogeneously hyperintense on T2 weighted sequence, 19 lesions are heterogenous and 3 lesions are either iso or hypo intense. 32 lesions had well defined margins on T2 weighted sequence, and 19 lesions had ill-defined margins. Based on these two characteristics, 28 lesions are considered as benign, 18 lesions are considered malignant and 5 lesions are considered intermediate.

On DWI, lesions are assessed at 50 mm/s^2 400 mm/s^2 and 800 mm/s^2. Lesions are categorized into benign and malignant based on the signal on DWI and ADC. Lesion is considered benign if it has high signal on DWI & ADC and it is considered malignant if it has high signal intensity on DWI and low signal intensity on ADC suggesting restriction of diffusion. In a study conducted by Bruegel M et al, Malignant hepatic lesions have demonstrated a significantly lower ADC than benign hepatic lesions[60].

In our study out of 51 lesions, 50 had high signal at 50 mm/s^2. Of the 50 lesions, 28 showed signal drop (low signal) at both 400 and 800 mm/s^2. Based on

DWI and ADC, 31 lesions are considered malignant which showed restriction of diffusion, of which 26 had heterogeneous restriction pattern and 5 lesions had homogeneous restriction pattern. 20 lesions did not show diffusion restriction and are considered benign.

The lesions are again categorized combining both T2WI and DWI sequences. Lesions are considered as malignant if it was malignant one or both the sequences and lesions are considered benign only if it is benign on both the sequences. Of 51, 23 lesions are categorized as benign and 28 lesions are categorized as malignant based on both the sequences.

In our study, biopsy was done for 22 lesions of which 8 lesions are benign and 14 lesions are malignant.

On assessing the accuracy of lesion characterization, correctly classified lesions based on T2WI are 37 (72.5%), based on DWI are 42 (80.3 %) and 44 (86.2 %) based on both the sequences. Hence combination of both the sequences increased the accuracy in discriminating benign and malignant focal liver lesions. In a study conducted by Parikh et al, sensitivity, specificity, and accuracy rates of T2WI and DWI in lesion characterisation were 92%/92%, 80%/83%, and 87%/89%, respectively[4].

Of 51 lesions, Hepatocellular carcinoma (includes multicentric and multifocal HCC) contributed to the highest percentage of lesions found (29.41%), followed by hemangiomas (17.65%), hepatic cysts (15.6%), metastasis (11.7%), hepatic abscess (7.8%), intrahepatic cholangiocarcinoma (5.8%), focal nodular hyperplasia (3.9%),

cholangitic abscess (1.9%), dysplastic nodule (1.9%), hepatic adenoma (1.9%) and regenerative nodule (1.9%).

Misclassified lesions:

On T2WI, 14 lesions were misclassified. 2 metastatic lesions were considered benign since they were well defined and homogenously hyperintense, 2 metastatic lesions were considered intermediate (1 had well defined margins with heterogenous signal intensity and one had ill-defined margins with heterogeneous signal). All the metastatic lesions were correctly classified on DWI, as the lesions showed low ADC values. 3 HCC's were classified as intermediate because the lesions showed ill-defined margins with high signal, 1 HCC was considered benign because of well-defined margins and homogeneously high signal. All the HCC's were correctly classified on DWI, as ADC value was low for these lesions. Hence DWI is more accurate in categorizing malignant lesions than T2WI in accordance with few previous studies.

FNH (n=2) were wrongly classified as malignant on both T2WI and DWI sequences, as the lesions had ill-defined margins and heterogenous signal with low ADC value. One intrahepatic cholangiocarcinoma was misclassified as benign on both the sequences due to well-defined margins, high signal on T2WI and high ADC value.

1 regenerative nodule was wrongly classified on T2WI since it had hypointense signal but was correctly classified as benign on DWI due high ADC value. 1 dysplastic nodule was wrong classified as benign on both T2WI and DWI

since it had well defined margins with hyperintense signal on T2 and high ADC value.

Hepatic abscesses (4) and cholangitic abscess (1) were misclassified as malignant on DWI due to low ADC values. 2 of these lesions were correctly classified as benign on T2WI since the lesions showed well defined margins with high signal intensity.

Hepatic adenoma was misclassified as malignant on DWI due to low ADC value, however the lesion was correctly classified on T2WI as it showed well defined margins with high signal intensity on T2WI.

In agreement with previous similar studies, in this study most of the benign cystic lesions are correctly classified on T2 and DWI sequences[56].

CONCLUSION

- Sample size in the present study was 45.
- 78% of the patients having focal liver lesions were above 40 years of age. Rest 28% were in the age group 20-40 years.
- In the present study, males outnumbered females (57.8% v/s 42.2% respectively).
- The most common associated condition was chronic liver disease (20% of the patients had co-existing CLD), followed by hepatitis-b, carcinoma rectum etc.
- 40 out of 51 (78.4%) patients had lesions in right lobe of liver. Rest of them had either in the left lobe or involving both the lobes (13.7% and 7.8% respectively).
- Lesions were assessed independently using T2WI and DWI sequences and on both the sequences on 3T MRI scanner.
- Histopathological confirmation was done for 22 lesions, of which 14 were malignant and 8 were benign.
- 37 out of 51 lesions were correctly classified on T2WI (72.5%).
- 41 out of 51 lesions were correctly classified on DWI (80.3%).
- Accuracy of correctly classified lesions was increased to 86.2% (44 out 51) when the lesion was categorized using both the sequences.
- Almost all benign cystic lesions were correctly classified on T2WI. Misclassified lesions on T2WI are mostly the malignant ones.
- Most of the malignant lesions were correctly classified on DWI like HCC and metastases. Most misclassified lesions on DWI were hepatic abscesses.

- Solid benign lesions like focal nodular hyperplasia and hepatic adenoma were misclassified as malignant on both the sequences.
- In this study, DWI was better at categorizing malignant lesions and T2WI was good at categorizing cystic benign lesions. However, both the sequences when used together improved the diagnostic accuracy of focal liver lesions.

LIMITATIONS

- Sample size was less in this study.
- Most of the benign lesions were either cysts or hemangioma. Very few number of solid benign lesions like hepatic abscess, focal nodular hyperplasia and hepatic adenomas were included.
- Large number of benign cystic lesions were correctly classified, but solid benign lesions were considered malignant on T2WI and DWI hence decreasing the percentage of correctly classified lesions.
- Biopsy was done for few number of lesions, however the diagnosis was confirmed by cross-sectional imaging and follow up imaging to avoid misclassification.
- Sensitivity, specificity and accuracy can be calculated by single receiver operating characteristic curve (ROC); however, it was not obtained in this study.

SUMMARY

- The overall number of focal liver lesions (FLL) discovered accidentally has increased as a result of the increased usage of imaging technology. The great majority of FLLs that develop in livers without cirrhosis are benign and do not develop into malignancy. Adenomas, focal nodular hyperplasias, and hemangiomas are the three types of solid benign tumours that are most prevalent.

- Ultrasound, CT scan, and MRI are imaging modalities utilised for liver pathology. Since ultrasound contrasts are difficult to obtain and ionising radiation is not present, magnetic resonance imaging is the preferred method for FLL characterisation.

- DWI is useful in distinguishing between the intra-hepatic arteries and the small localised lesions, however it has not yet completely replaced T2WI in everyday practise. This is because T2WI can distinguish between the two types of lesions without reducing its accuracy.

- In this study MRI scanner with a field strength of 3 Tesla is used, to determine whether or not combining the two sequences (T2WI and DWI) results in more accurate diagnoses.

- The study population includes patients referred to do MRI study to diagnose and/or to confirm the ultrasonographic or CT findings of focal hepatic lesions, presenting to the Department of Radio-Diagnosis at the KLE's Dr. Prabhakar Kore Hospital & MRC, Belagavi.

- The role of T2WI and DWI sequences of liver in plain MRI abdomen was studied in 45 patients between the age group 15-90 years.
- MRI abdomen and pelvis was done for 45 patients, of which 9 patients were known case of chronic liver disease, 2 patients were known case of hepatitis b, 2 patients were known case of carcinoma rectum, 1 patient had carcinoma stomach, 1 patient had carcinoma cervix, 1 patient had carcinoma adrenal gland, 1 patient had carcinoma tongue and 1 patient was a known case of wilson's disease. The most commonly associated condition was chronic liver disease which was seen in 20% of the patients. Rest of the 27 patients either showed a liver lesion on routine annual ultrasonography or had either come with other complaints
- The median age in this study was 56. In 78% of the patient's right lobe of liver was involved. Lesions were assessed independently using T2WI and DWI sequences and on both the sequences together on 3T MRI scanner.
- On T2WI, of 51 lesions, 29 lesions were homogeneously hyperintense on T2 weighted sequence, 19 lesions were heterogenous, and 3 lesions were either iso or hypo intense. 32 lesions had well defined margins on T2WI, and 19 lesions had ill- defined margins.
- Lesions were categorized into benign/malignant/intermediate based on margins and signal intensity on T2WI. The lesion was considered benign if the margin is well-defined and showed homogenously high signal intensity on T2, it was considered malignant if it had ill-defined margins and iso or low signal intensity and it was considered intermediate if the lesion had ill-defined margins with homogenously high signal intensity or if the lesion had well defined margins with heterogenous low/ isointensity. Based on these two characteristics, 28

lesions were considered as benign, 18 patients were considered malignant and 5 lesions were considered intermediate.

- Lesions were categorized into benign and malignant based on the signal on DWI and ADC. Lesion was considered benign if it had high signal on DWI & ADC and it was considered malignant if it showed high signal intensity on DWI and low signal intensity on ADC suggesting restriction of diffusion in the lesion.
- Based on DWI and ADC, 31 lesions were considered malignant which showed restriction of diffusion, of which 26 had heterogeneous restriction pattern and 5 lesions had homogeneous restriction pattern. 20 lesions did not show diffusion restriction and were considered benign.
- Based on both T2WI and DWI sequences, lesions were considered as malignant if it was malignant one or both the sequences and lesions were considered benign if it is benign on both the sequences. Of 51, 23 lesions were categorized as benign and 28 lesions were categorized as malignant based on both the sequences.
- Biopsy was done 22 lesions of which 8 lesions had come out as benign and 14 as malignant.
- Correctly classified lesions based on T2WI were 37 (72.5%), based on DWI were 42 (80.3 %) and 44 (86.2 %) based on the both the sequences. Hence combination of both the sequences increased the accuracy in discriminating benign and malignant focal liver lesions.
- 37 out of 51 lesions were correctly classified on T2WI (72.5%).
- 41 out of 51 lesions were correctly classified on DWI (80.3%).
- Accuracy of correctly classified lesions was increased to 86.2% (44 out of 51) when the lesion was categorized using both the sequences.

- Almost all benign cystic lesions were correctly classified on T2WI. Misclassified lesions on T2WI are mostly the malignant ones.
- Most of the malignant lesions were correctly classified on DWI like HCC and metastases. Most misclassified lesions on DWI were hepatic abscesses.
- Solid benign lesions like focal nodular hyperplasia and hepatic adenoma were misclassified as malignant on both the sequences.
- In this study, DWI was better at categorizing malignant lesions and T2WI was good at categorizing cystic benign lesions. However, both the sequences when used together improved the diagnostic accuracy.

- **Limitations of the present study:**

  - Sample size was less in this study.

  - Most of the benign lesions were either cysts or hemangioma. Very few number of solid benign lesions like hepatic abscess, focal nodular hyperplasia and hepatic adenomas were included.

  - Large number of benign cystic lesions were correctly classified, but solid benign lesions were considered malignant on T2WI and DWI hence decreasing the percentage of correctly classified lesions.

  - Biopsy was done for very few number of lesions, however the diagnosis was confoirmed by cross-sectional imaging and follow up imaging to avoid misclassification.

- Sensitivity, specificity and accuracy can be calculated by single receiver operating characteristic curve (ROC), however it was not obtained in this study.

- **Recommendations for future study include:**

  - Use of contrast enhanced sequences for evaluating liver lesions in correlation with CT images.

  - Diagnosis should be confirmed by histopathology correlation

BIBLIOGRAPHY

1) Matos AP, Velloni F, Ramalho M, AlObaidy M, Rajapaksha A, Semelka RC. Focal liver lesions: Practical magnetic resonance imaging approach. World journal of hepatology. 2015 Aug 8;7(16):1987.

2) Galia M, Taibbi A, Marin D, Furlan A, Burgio MD, Agnello F, Cabibbo G, Van Beers BE, Bartolotta TV, Midiri M, Lagalla R. Focal lesions in cirrhotic liver: what else beyond hepatocellular carcinoma?. Diagnostic and Interventional Radiology. 2014 May;20(3):222.

3) Froehlich F, Lamy O, Fried M, Gonvers JJ. Practice and complications of liver biopsy. Results of a nationwide survey in Switzerland. Dig Dis Sci. Aug 1993;38 (8):1480-4

4) Parikh T, Drew SJ, Lee VS, Wong S, Hecht EM, Babb JS, Taouli B. Focal liver lesion detection and characterization with diffusion-weighted MR imaging: comparison with standard breath-hold T2-weighted imaging. Radiology. 2008 Mar;246(3):812-22.

5) Rutkauskas S, Gedrimas VL, Pundzius J, Barauskas G, Basevičius A. Clinical and anatomical basis for the classification of the structural parts of liver. Medicina. 2006;42(2):98-106.

6) Shumacker Jr HB. Hemangioma of the liver. Surgery. 1942 Feb;11(Feb.):209.

7) Miller FH, Hammond N, Siddiqi AJ, Shroff S, Khatri G, Wang Y, Merrick LB, Nikolaidis P. Utility of diffusion-weighted MRI in distinguishing benign and malignant hepatic lesions. Journal of Magnetic Resonance Imaging. 2010 Jul;32(1):138-47.

8) Federle MP, Jeffrey RB, Woodward PJ, Borhani A. Diagnostic imaging: abdomen. 2004.

9) Danet IM, Semelka RC, Braga L, Armao D, Woosley JT. Giant hemangioma of the liver: MR imaging characteristics in 24 patients. Magnetic resonance imaging. 2003 Feb 1;21(2):95-101.

10) Kubota, T., Shimizu, K., Sonoyama, T., Ikeda, E., Kurioka, H., Ouchi, T., Suyama, Y., Hosokawa, Y., & Yamagishi, H. (2001). Enlarged focal nodular hyperplasia of the liver under the influence of oral contraceptives. *Hepato-gastroenterology*, *48*(42), 1736–1739.

11) Holzapfel, K., Reiser-Erkan, C., Fingerle, A. A., Erkan, M., Eiber, M. J., Rummeny, E. J., Friess, H., Kleeff, J., & Gaa, J. (2011). Comparison of diffusion-weighted MR imaging and multidetector-row CT in the detection of liver metastases in patients operated for pancreatic cancer. *Abdominal imaging*, *36*(2), 179–184.

12) Terkivatan T, De Wilt JH, De Man RA, et al. Man- agement of hepatocellular adenoma during preg- nancy. Liver 2000;20:186-187

13) Dharmana, H., Saravana-Bawan, S., Girgis, S., & Low, G. (2017). Hepatocellular adenoma: imaging review of the various molecular subtypes. Clinical radiology, 72(4), 276–285.

14) Paulson EK, McClellan JS, Washington K, et al. Hepatic adenoma: MR characteristics and correla- tion with pathologic findings. AJR Am J Roentgenol 1994;163:113-116.

15) Flejou JF, Barge J, Menu Y, Degott C, Bismuth H, Potet F, Benhamou JP. Liver adenomatosis: an entity distinct from liver adenoma?. Gastroenterology. 1985 Nov 1;89(5):1132-8.

16) Lewin M, Handra-Luca A, Arrivé L, Wendum D, Paradis V, Bridel E, Fléjou JF, Belghiti J, Tubiana JM, Vilgrain V. Liver adenomatosis: classification of MR

imaging features and comparison with pathologic findings. Radiology. 2006 Nov;241(2):433-40.

17) Gaines PA, Sampson MA. The prevalence and characterization of simple hepatic cysts by ultrasound examination. The British Journal of Radiology. 1989 Apr;62(736):335-7.

18) Yoon KH, Ha HK, Lee JS, Suh JH, Kim MH, Kim PN, Lee MG, Yun KJ, Choi SC, Nah YH, Kim CG. Inflammatory pseudotumor of the liver in patients with recurrent pyogenic cholangitis: CT-histopathologic correlation. Radiology. 1999 May;211(2):373-9.

19) Rahimian J, Wilson T, Oram V, Holzman RS. Pyogenic liver abscess: recent trends in etiology and mortality. Clinical infectious diseases. 2004 Dec 1;39(11):1654-

20) Shi SH, Zhai ZL, Zheng SS. Pyogenic liver abscess of biliary origin: the existing problems and their strategies. InSeminars in liver disease 2018 Aug (Vol. 38, No. 03, pp. 270-283). Thieme Medical Publishers.

21) Chan JH, Tsui EY, Luk SH, Fung AS, Yuen MK, Szeto ML, Cheung YK, Wong KP. Diffusion-weighted MR imaging of the liver: distinguishing hepatic abscess from cystic or necrotic tumor. Abdominal imaging. 2001 Mar;26(2):161-5.

22) Hanna RF, Aguirre DA, Kased N, Emery SC, Peterson MR, Sirlin CB. Cirrhosis-associated hepatocellular nodules: correlation of histopathologic and MR imaging features. Radiographics. 2008 May;28(3):747-69.

23) Hussain SM, Zondervan PE, IJzermans JN, Schalm SW, de Man RA, Krestin GP. Benign versus malignant hepatic nodules: MR imaging findings with pathologic correlation. Radiographics. 2002 Sep;22(5):1023-36.

24) Akinyemiju T, Abera S, Ahmed M, Alam N, Alemayohu MA, Allen C, Al-Raddadi R, Alvis-Guzman N, Amoako Y, Artaman A, Ayele TA. The burden of primary liver cancer and underlying etiologies from 1990 to 2015 at the global, regional, and national level: results from the global burden of disease study 2015. JAMA oncology. 2017 Dec 1;3(12):1683-91.

25) Bruix J, Sherman M; American Association for the Study of Liver Diseases. Management of hepatocellular carcinoma: an update. Hepatology. 2011 Mar;53(3):1020-2. doi: 10.1002/hep.24199. PMID: 21374666; PMCID: PMC3084991.

26) Marrero JA, Kulik LM, Sirlin CB, Zhu AX, Finn RS, Abecassis MM, Roberts LR, Heimbach JK. Diagnosis, S taging, and M anagement of H epatocellular C arcinoma: 2018 P ractice G uidance by the A merican A ssociation for the S tudy of L iver D iseases. Hepatology. 2018 Aug;68(2):723-50.

27) Lee JH, Lee JM, Kim SJ, Baek JH, Yun SH, Kim KW, Han JK, Choi BI. Enhancement patterns of hepatocellular carcinomas on multiphasic multidetector row CT: comparison with pathological differentiation. The British journal of radiology. 2012 Sep;85(1017):e573-83.

28) Roth CG, Mitchell DG. Hepatocellular carcinoma and other hepatic malignancies: MR imaging. Radiologic Clinics. 2014 Jul 1;52(4):683-707.

29) Andersson M, Vyberg M, Visfeldt J, Carstensen B, Storm HH. Primary liver tumors among Danish patients exposed to Thorotrast. Radiation Research. 1994 Feb 1;137(2):262-73.

30) Ayuso C, Rimola J, García-Criado Á. Imaging of HCC. Abdominal imaging. 2012 Apr;37(2):215-30.

31) Park HS, Lee JM, Choi JY, Lee MW, Kim HJ, Han JK, Choi BI. Preoperative evaluation of bile duct cancer: MRI combined with MR cholangiopancreatography versus MDCT with direct cholangiography. American Journal of Roentgenology. 2008 Feb;190(2):396-405.

32) Park YS, Kim JH, Kim KW, Lee IS, Yoon HK, Ko GY, Sung KB. Primary hepatic angiosarcoma: imaging findings and palliative treatment with transcatheter arterial chemoembolization or embolization. Clinical radiology. 2009 Aug 1;64(8):779-85.

33) Lee J, Kim SH, Kang TW, Song KD, Choi D, Jang KT. Mass-forming intrahepatic cholangiocarcinoma: diffusion-weighted imaging as a preoperative prognostic marker. Radiology. 2016 Oct;281(1):119-28.

34) Bruegel M, Muenzel D, Waldt S, Specht K, Rummeny EJ. Hepatic angiosarcoma: cross-sectional imaging findings in seven patients with emphasis on dynamic contrast-enhanced and diffusion-weighted MRI. Abdominal imaging. 2013 Aug;38(4):745-54.

35) Chien CY, Hwang CC, Yeh CN, Chen HY, Wu JT, Chan SC, Lin CL, Yen CL, Wang WY, Chiang KC. Liver angiosarcoma, a rare liver malignancy, presented with intraabdominal bleeding due to rupture-a case report. World journal of surgical oncology. 2012 Dec;10(1):1-5.

36) Ishak KG, Goodman ZD, Stocker JT. Tumors of the liver and intrahepatic bile ducts. Amer Registry of Pathology; 2001.

37) Chung EM, Lattin Jr GE, Cube R, Lewis RB, Marichal-Hernández C, Shawhan R, Conran RM. From the archives of the AFIP: pediatric liver masses: radiologic-pathologic correlation part 2. Malignant tumors. Radiographics. 2011 Mar;31(2):483-507.

38) Dachman AH, Pakter RL, Ros PR, Fishman EK, Goodman ZD, Lichtenstein JE. Hepatoblastoma: radiologic-pathologic correlation in 50 cases. Radiology. 1987 Jul;164(1):15-9.

39) Abbruzzese JL, Abbruzzese MC, Lenzi R, Hess KR, Raber MN. Analysis of a diagnostic strategy for patients with suspected tumors of unknown origin. J Clin Oncol. 1995 Aug;13(8):2094-103. doi: 10.1200/JCO.1995.13.8.2094. PMID: 7636553.

40) Roth CG, Mitchell DG. Hepatocellular carcinoma and other hepatic malignancies: MR imaging. Radiologic Clinics. 2014 Jul 1;52(4):683-707.

41) Thian YL, Riddell AM, Koh DM. Liver-specific agents for contrast-enhanced MRI: role in oncological imaging. Cancer imaging. 2013;13(4):567.

42) Donati OF, Fischer MA, Chuck N, Hunziker R, Weishaupt D, Reiner CS. Accuracy and confidence of Gd-EOB-DTPA enhanced MRI and diffusion-weighted imaging alone and in combination for the diagnosis of liver metastases. European Journal of Radiology. 2013 May 1;82(5):822-8.

43) Böttcher J, Hansch A, Pfeil A, Schmidt P, Malich A, Schneeweiss A, Maurer MH, Streitparth F, Teichgräber UK, Renz DM. Detection and classification of different liver lesions: comparison of Gd-EOB-DTPA-enhanced MRI versus multiphasic spiral CT in a clinical single centre investigation. European journal of radiology. 2013 Nov 1;82(11):1860-9.

44) Gaa J, Hatabu H, Jenkins RL, Finn JP, Edelman RR. Liver masses: replacement of conventional T2-weighted spin-echo MR imaging with breath-hold MR imaging. Radiology. 1996 Aug;200(2):459-64.

45) Taouli B, Koh DM. Diffusion-weighted MR imaging of the liver. Radiology. 2010 Jan 1;254(1):47.

46) Filipe JP, Curvo-Semedo L, Casalta-Lopes J, Marques MC, Caseiro-Alves F. Diffusion-weighted imaging of the liver: usefulness of ADC values in the differential diagnosis of focal lesions and effect of ROI methods on ADC measurements. Magnetic Resonance Materials in Physics, Biology and Medicine. 2013 Jun;26(3):303-12.

47) Koh DM, Collins DJ. Diffusion-weighted MRI in the body: applications and challenges in oncology. American Journal of Roentgenology. 2007 Jun;188(6):1622-35.

48) Sadowski EA, Bennett LK, Chan MR, Wentland AL, Garrett AL, Garrett RW, Djamali A. Nephrogenic systemic fibrosis: risk factors and incidence estimation. Radiology. 2007 Apr;243(1):148-57.

49) Miller FH, Hammond N, Siddiqi AJ, Shroff S, Khatri G, Wang Y, Merrick LB, Nikolaidis P. Utility of diffusion-weighted MRI in distinguishing benign and malignant hepatic lesions. Journal of Magnetic Resonance Imaging. 2010 Jul;32(1):138-47.

50) Demir Öİ, Obuz F, Sagol O, Dicle O. Contribution of diffusion-weighted MRI to the differential diagnosis of hepatic masses. Diagnostic and Interventional Radiology. 2007 Jun 1;13(2):81.

51) Caraiani C, Chiorean L, Fenesan DI, Lebovici A, Feier D, Gersak M, Calin AM, Dan M, Militaru C, Mare C, Ciobanu L. Diffusion weighted magnetic resonance imaging for the classification of focal liver lesions as benign or malignant. J Gastrointestin Liver Dis. 2015 Sep 1;24(3):309-17.

52) Haradome H, Grazioli L, Morone M, Gambarini S, Kwee TC, Takahara T, Colagrande S. T2-weighted and diffusion-weighted MRI for discriminating benign from malignant focal liver lesions: Diagnostic abilities of single versus

combined interpretations. Journal of Magnetic Resonance Imaging. 2012 Jun;35(6):1388-96.

53) Elbarbary AA, Elahwal HM, Elashwah ME. Role of diffusion weighted magnetic resonance imaging in evaluation of hepatic focal lesions. The Egyptian Journal of Radiology and Nuclear Medicine. 2015 Jun 1;46(2): 325-34.

54) Javadrashid R, Shakeri Bavil Olyaei A, Tarzamni MK, Razzaghi R, Jalili J, Hashemzadeh S, Mirza-Aghazadeh-Attari M, Kiani Nazarlou A, Zarrintan A. The diagnostic value of diffusion-weighted imaging in differentiating benign from malignant hepatic lesions. Egyptian Liver Journal. 2020 Dec;10(1):1-9.

55) Paley MR, Mergo PJ, Torres GM, Ros PR. Characterization of focal hepatic lesions with ferumoxides-enhanced T2-weighted MR imaging. InPresented at the annual meeting of the Radiological 1999 Dec 8.

56) McFarland EG, Mayo-Smith WW, Saini S, Hahn PF, Goldberg MA, Lee MJ. Hepatic hemangiomas and malignant tumors: improved differentiation with heavily T2-weighted conventional spin-echo MR imaging. Radiology. 1994 Oct;193(1):43-7.

57) Hicks RM, Yee J, Ohliger MA, Weinstein S, Kao J, Ikram NS, Hope TA. Comparison of diffusion-weighted imaging and T2-weighted single shot fast spin-echo: Implications for LI-RADS characterization of hepatocellular carcinoma. Magnetic resonance imaging. 2016 Sep 1;34(7):915-21.

58) Yang DM, Jahng GH, Kim HC, Jin W, Ryu CW, Nam DH, Lee YK, Park SY. The detection and discrimination of malignant and benign focal hepatic lesions: T 2 weighted vs diffusion-weighted MRI. The British Journal of Radiology. 2011 Apr;84(1000):319-26.

59) Lesions: A Cross-sectional Study. Journal of Clinical & Diagnostic Research. 2021 Jan 1;15(1).

60) Bruegel M, Holzapfel K, Gaa J, Woertler K, Waldt S, Kiefer B, Stemmer A, Ganter C, Rummeny EJ. Characterization of focal liver lesions by ADC measurements using a respiratory triggered diffusion-weighted single-shot echo-planar MR imaging technique. European radiology. 2008 Mar;18(3): 477-85.

ANNEXURE – I

INFORMED CONSENT FORM

TITLE: "UTILITY OF T2 WEIGHTED AND DIFFUSION WEIGHTED SEQUENCES IN MAGNETIC RESONANCE IN DIFFERENTIATION OF BENIGN AND MALIGNANT LIVER LESIONS: BENEFITS OF SINGLE VERSUS COMBINED INTERPRETATIONS".

PRINCIPAL INVESTIGATOR:

INTRODUCTION AND PURPOSE: With the advancement in the use of cross-sectional imaging, an increase in rate of incidentally detected focal liver lesions (FLL) has been observed. Despite dynamic enhanced magnetic resonance imaging (MRI) with several contrast agents having a major role in the detection and characterization of FLLs, unenhanced MR sequences such as T2-weighted imaging (T2WI) and diffusion-weighted imaging (DWI) became vital in the characterization of FLLs in patients with contraindications like allergy to contrast agents. Based on signal intensity of the lesion, T2WI can differentiate benign cystic lesions (simple cyst, hemangioma, etc) from malignant solid lesions (metastasis, hepatocellular carcinoma [HCC], etc), but its use in discriminating solid benign and malignant lesions may be limited. Though DWI is beneficial for lesion detection and characterization, it has not yet replaced T2WI in routine use because it has many advantages and limitations simultaneously.

The purpose of this study is to determine the diagnostic accuracies of T2WI and DWI alone and, combined sequences in MRI, in discriminating benign from malignant liver lesions in patients referred to MRI abdomen and in patients with

findings on ultrasonography or computed tomography (CT). Combination of sequences helps in reaching a diagnosis accurately and hence the patients can be treated accordingly without misdiagnosis.

PROCEDURE: We request you to kindly participate in the study titled "UTILITY OF T2 WEIGHTED AND DIFFUSION WEIGHTED SEQUENCES IN MAGNETIC RESONANCE IN DIFFERENTIATION OF BENIGN AND MALIGNANT LIVER LESIONS : BENEFITS OF SINGLE VERSUS COMBINED INTERPRETATIONS- ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY" at Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi is being conducted by Dr. _____ Postgraduate in Radio diagnosis at J.N. Medical College, Belagavi, Karnataka, under the guidance of Dr. _____ Professor & Head, Dept. of Radio-diagnosis, J. N. Medical College, Belagavi.

BENEFITS: No use of surgical equipment/risk associated with it.

COMPLICATIONS: No risk to the patient has been documented from MR imaging of the abdomen conducted earlier.

ALTERNATIVES: If you are not willing to take part in the study, your treatment or any other further investigations the patient wants to undergo, in future, in KLE will not be affected by your decision.

VOLUNTARY PARTICIPATION/WITHDRAWAL: Taking part in this study is voluntary. You may choose not to take part in this study, or if you decide to take part, you can later change my mind and withdraw from the study. Your decision will not change the present or future health care or other services that you receive. The study doctor or the sponsor may stop your participation in this

study. You will tell if any important new findings that may change my willingness to continue to take part. If you choose not to take part in the study, you will receive the standard treatment for patients with your condition.

COSTS: NIL (The study is to be conducted on the participants who are advised MRI abdomen as an investigation by the referring consultant and the participants will bear the charges for it.) Payment for Participation: No incentive will be paid to you for participating in this study.

COMPENSATION: In the event that you become injured as a result of taking part in this study, treatment whatever available at KLE Charitable hospital, Belagavi, will be offered to you. No reimbursement, compensation or free medical care is given.

CONFIDENTIALITY: All information collected about you during the course of the study will be kept confidential to the extent permitted by the law. The code numbers will identify you in this research record. Information from this study may be published but your identity will be kept confidential in any publication/ presentation.

QUESTIONS: If you have any enquiries in the future or in case of research related injury illness, you may contact following persons:

Dr. HARSHA HEGDE.
Chairman, IEC & scientist D, ICMR, National institute of traditional medicine, Belagavi.
Ph. No: 0831-2473777, Ext. 1529

CONSENT TO PARTICIPATE IN RESEARCH STUDY:

1. "I understand that I am participating in the study, which includes MRI abdomen (plain).

2. I confirm that I have read and understood the information in the patient information sheet. Procedure is explained to me in detail along with information about the advantages and disadvantages of taking part in the study. I have been given the opportunity to discuss all aspects of the trial, to ask questions and hereby consent to participation in the trial outlined above.

3. I understand that the decision to take part in this study is completely voluntary and I am aware that I can choose to withdraw from the study at any point of time.

4. I consent to the photographing or recording of the procedure to be performed including appropriate portions of my body, for medical, scientific or educational purposes provided my identity is not revealed in the pictures or by the descriptive texts accompanying them.

5. I understand that there is no significant risk involved in the test that would be done in this study.

6. No guarantee or assurance has been given by anyone as to the results that may be obtained.

7. My signature on this form signifies that I have willingly decided to participate after understanding the above information".

Participant's Name/ legally authorized representative _____

Signature _____

Name and signature of witness _____

Name and signature of interviewer _____

Date:

Place:

ANNEXURE – II

PROFORMA FOR DATA COLLECTION

DATE OF INTERVIEW: _____

NAME OF THE PATIENT: _____

AGE (in years): _____ SEX(M/F):_____ OP/IP NO:_____

MOBILE NUMBER: _____

ADDRESS: HOUSE NO_____ GALLI_____ VILLAGE _____

CITY _____ DISTRICT _____ PIN CODE _____

MRI NUMBER: _____

CHIEF COMPLAINTS: **DURATION:**

1.		
2.		
3.		

HISTORY OF PRESENTING ILLNESS:

1.		
2.		
3.		

PAST HISTORY:

1.		
2.		
3.		

CLINICAL EXAMINATION:

CVS:

RS:

PA:

PROVISIONAL DIAGNOSIS:

ULTRASOUND FINDINGS:

CT FINDINGS:

MRI FINDINGS:

- **T2 weighted sequence:**

- **DWI and ADC:**

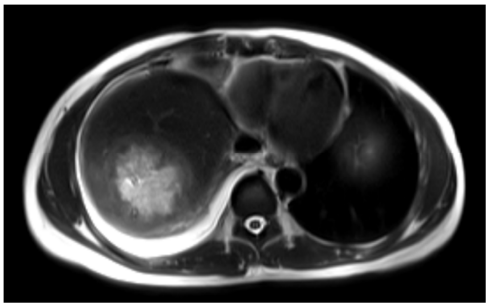
BIOPSY FINDINGS:

FINAL DIAGNOSIS:

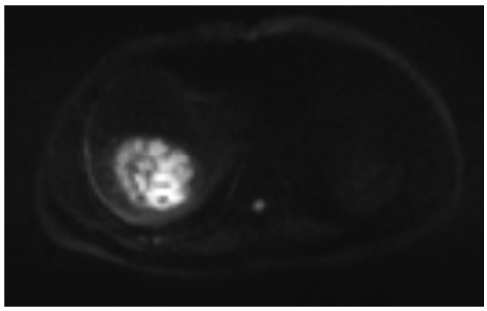
ANNEXURE – III

PHOTOGRAPHS OF CASES

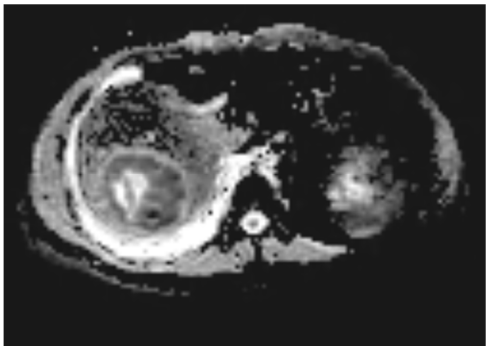
Case 1: A 28-year-old non-alcoholic male, was referred for MRI abdomen in view of pain abdomen and fever. On T2WI, ill-defined hyperintense mass lesion was seen in segment VIII of right lobe of liver. The lesion also showed diffusion restriction on DWI suggesting the possibility of hepatic abscess.



T2WI

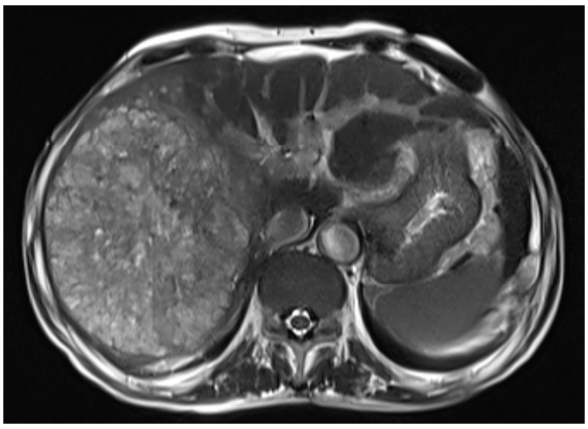


DWI at b value 1000mm^2/sec

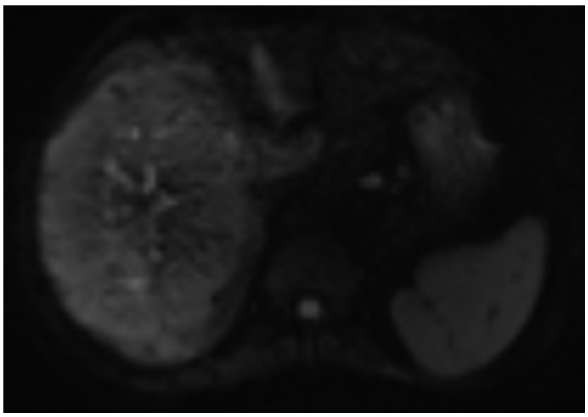


ADC

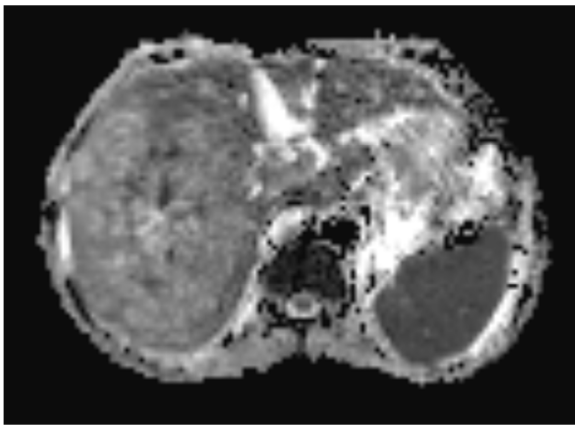
Case 2: A 72-year-old male, was referred for MRI abdomen in view of pain abdomen. He is a known case of hepatitis-b. On T2WI, fairly well defined large lobulated heterogeneous predominantly T2 hyperintense mass lesion in the right lobe of liver with central areas of diffusion restriction on DWI sequence suggesting the possibility of HCC. Biopsy was done and it suggested HCC.



T2WI

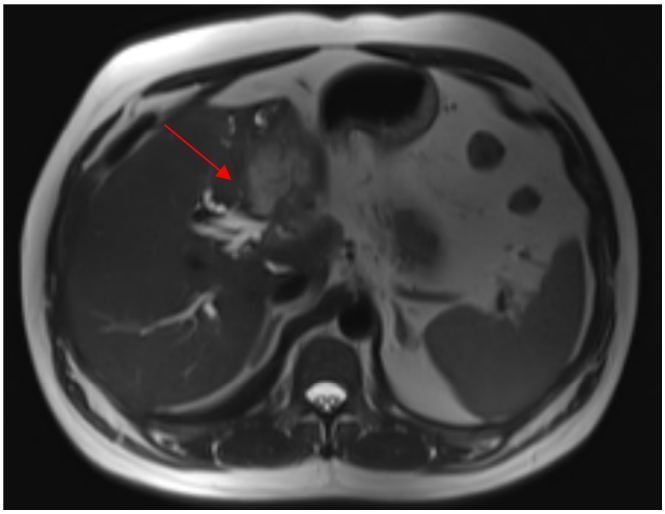


DWI at b value 1000mm2/sec



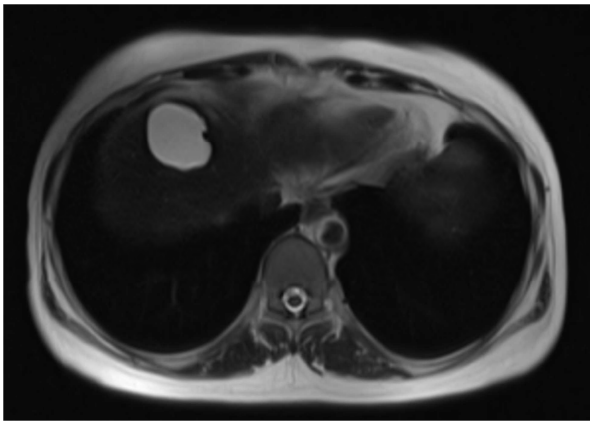
ADC

Case 3: A 46-year-old male, was referred for MRI abdomen in view of pain abdomen and abdominal discomfort. On T2WI, fairly well defined heterogeneous T2 hyperintense mass lesion in the left lobe of liver with no evidence of restricted diffusion on DWI sequence. Biopsy was done for the lesion and it suggested intrahepatic cholangiocarcinoma.

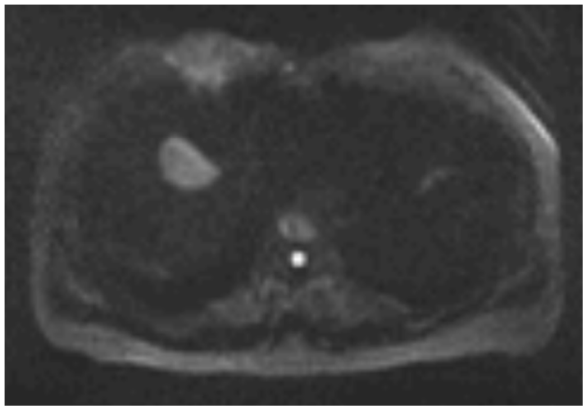


T2WI

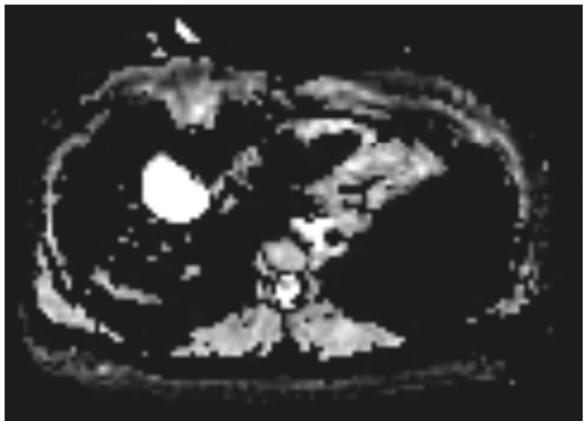
Case 4: A 51-year-old female, was referred for MRI abdomen in view of right upper quadrant pain. Ultrasound abdomen was done and showed cholelithiasis and hypodense cystic area in the right lobe of the liver. On T2WI, well defined homogeneous T2 hyperintense mass lesion in the right lobe of liver with no evidence of restricted diffusion on DWI sequence suggesting the possibility of hepatic cyst.



T2WI

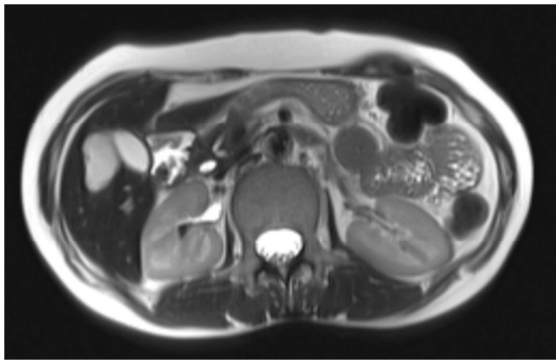


DWI at b value 1000mm2/sec

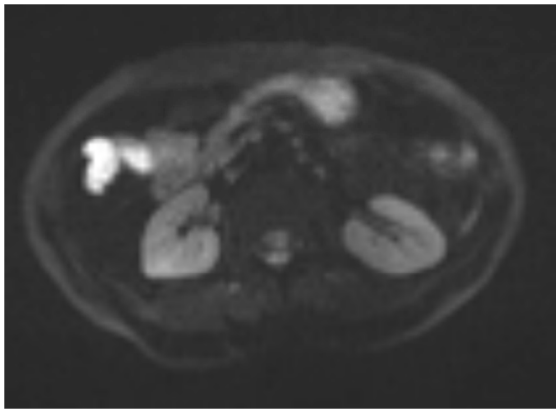


ADC

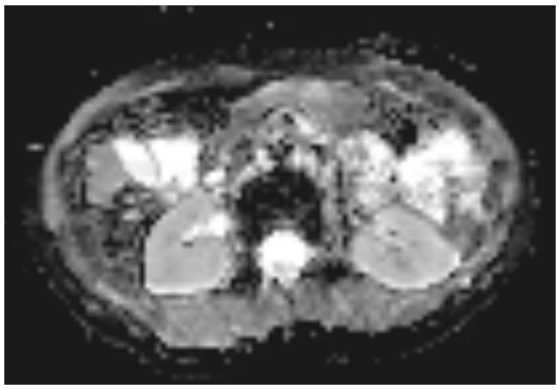
Case 5: A 49-year-old female, was referred for MRI abdomen in view of right upper quadrant pain. Patient is known case of carcinoma cervix. On T2WI, well-defined T2 hyperintense area in segment VI of right lobe with evidence of homogeneous restricted diffusion on DWI sequence suggesting the possibility of metastasis.



T2WI



DWI at b value 1000mm2/sec



ADC

ANNEXURE IV - KEY TO MASTERCHART

T2WI	T2 weighted imaging
DWI	Diffusion weighted imaging
ADC	Apparent diffusion coefficient
ID	Ill-defined
WD	Well-defined
HYPER	Hyperintense
ISO	Isointense
HYPO	Hypointense
HOMO	Homogeneous
HETERO	Heterogeneous

ANNEXURE – V-

MASTER CHART

							T2 WI FINDINGS						DWI						ADC		RESTRICTION					YES (LESION CHARACTER)		NO	FINAL LESION CHARACTER	DIAGNOSIS
							MARGIN		SIGNAL INTENSITY			LESION CHARACTER	50 mm/s2		SIGNAL DROP AT 400 mm/s2		SIGNAL DROP AT 800mm/s2				RESTRICTION PATTERN (PRESENT)		ABSENT	LESION CHARACTER	LESION CHARACTER (T2WI AND DWI)	BENIGN	MALIGNANT			
SR NO:	MRI NO:	AGE	GENDER	ASSOCIATED COMORBIDITY	TYPES OF LESIONS	LOBE/SEGMENT	ILL-DEFINED	WELL-DEFINED	HYPER	HETERO	ISO/HYPO		HIGH	LOW	PRESENT	ABSENT	PRESENT	ABSENT	HIGH	LOW	HOMOGENEOUS	HETEROGENEOUS								
1	24834	45	F	CLD	1	RIGHT LOBE	ID	-	-	HETERO	-	MALIGNANT	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	-	MALIGNANT	-	MALIGNANT	HEPATOCELLULAR CARCINOMA
2	27499	71	M	HbsAg+	2	RIGHT LOBE	ID	-	-	HETERO	-	MALIGNANT	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	-	MALIGNANT	-	MALIGNANT	MULTIIFOCAL HCC
						VII	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	HIGH	-	-	-	ABSENT	BENIGN	BENIGN	-	-	NO	BENIGN	HEPATIC CYST
3	26800	28	M	-	1	VII	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	-	LOW	HOMO	-	-	MALIGNANT	MALIGNANT	BENIGN	-	-	BENIGN	HEPATIC ABSCESS
4	21931	50	F	CLD	1	VI	ID	-	HYPER	HETERO	-	MALIGNANT	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	-	MALIGNANT	-	MALIGNANT	MULTIIFOCAL HCC
5	16548	35	M	-	1	VII	ID	-	-	HETERO	-	MALIGNANT	HIGH	-	PRESENT	-	PRESENT	-	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	-	MALIGNANT	-	MALIGNANT	INTRAHEPATIC CHOLANGIOCARCINOMA
6	26949	52	M	CLD	1	IV b	-	WD	-	HETERO	-	INTERMEDIATE	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	-	-	NO	MALIGNANT	HEPATOCELLULAR CARCINOMA
7	26558	49	F	CA CERVIX	1	VI	-	WD	HYPER	-	-	BENIGN	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	HOMO	-	-	MALIGNANT	MALIGNANT	-	-	NO	MALIGNANT	METASTASIS
8	25160	51	F	CA RECTUM	1	VII & VIII	ID	-	-	HETERO	-	MALIGNANT	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	-	MALIGNANT	-	MALIGNANT	METASTASIS
9	24675	65	F	CA ADRENALS	1	VI	-	WD	-	HETERO	-	INTERMEDIATE	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	-	MALIGNANT	-	MALIGNANT	METASTASIS
10	23530	60	M	CLD	1	VI	ID	-	-	HETERO	-	MALIGNANT	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	-	-	NO	MALIGNANT	HEPATOCELLULAR CARCINOMA
11	22733	38	F	-	2	VII	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	HIGH	-	-	-	ABSENT	BENIGN	BENIGN	-	-	NO	BENIGN	HEPATIC CYST
						VIII	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	HIGH	-	-	-	ABSENT	BENIGN	BENIGN	-	-	NO	BENIGN	HEMANGIOMA
12	22689	70	F	CLD	1	RIGHT LOBE	ID	-	-	-	ISO/HYPO	MALIGNANT	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	-	MALIGNANT	-	MALIGNANT	HEPATOCELLULAR CARCINOMA
13	21841	42	M	-	1	IV b & VIII	-	WD	-	HETERO	-	INTERMEDIATE	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	-	-	NO	MALGNANT	HEPATOCELLULAR CARCINOMA
14	21645	38	M	-	2	VI	-	WD	HYPER	-	-	BENIGN	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	-	MALIGNANT	-	MALGNANT	HEPATOCELLULAR CARCINOMA
						VII	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	HIGH	-	-	-	ABSENT	BENIGN	BENIGN	-	-	NO	BENIGN	HEPATIC CYST
15	21234	75	M	-	1	RIGHT LOBE	-	WD	-	HETERO	-	INTERMEDIATE	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	-	-	NO	MALGNANT	HEPATOCELLULAR CARCINOMA
16	27071	72	M	CLD	2	IV a & VII	ID	-	-	HETERO	-	MALIGNANT	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	-	-	NO	MALIGNANT	HEPATOCELLULAR CARCINOMA
				-		RIGHT LOBE	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	HIGH	-	-	-	ABSENT	BENIGN	BENIGN	-	-	NO	BENIGN	HEPATIC CYST
17	27217	56	M	-	1	VI	ID	-	HYPER	-	-	INTERMEDIATE	HIGH	-	PRESENT	-	PRESENT	-	-	LOW	-	HETERO	-	MALIGNANT	BENIGN	BENIGN	-	-	BENIGN	LIVER ABSCESS
18	23754	23	F	-	1	VII	ID	-	-	HETERO	-	MALIGNANT	HIGH	-	PRESENT	-	PRESENT	-	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	-	-	NO	BENIGN	FOCAL NODULAR HYPERPLASIA
19	41223	65	F	CLD	2	VIII	ID	-	-	HETERO	-	MALIGNANT	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	-	MALIGNANT	-	MALIGNANT	HEPATOCELLULAR CARCINOMA
						V	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	HIGH	-	-	-	ABSENT	BENIGN	BENIGN	-	-	NO	BENIGN	HEMANGIOMA
20	23123	45	F	-	1	V	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	HIGH	-	-	-	ABSENT	BENIGN	BENIGN	-	-	NO	BENIGN	HEMANGIOMA
21	23156	60	M	-	1	VIII	-	WD	-	HETERO	-	MALIGNANT	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	-	-	NO	MALIGNANT	HEPATOCELLULAR CARCINOMA
22	43255	58	F	-	1	III & IV a	ID	-	-	HETERO	-	MALIGNANT	HIGH	-	PRESENT	-	PRESENT	-	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	-	MALIGNANT	-	MALIGNANT	INTRAHEPATIC CHOLANGIOCARCINOMA
23	27865	68	M	-	1	IV a	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	HIGH	-	-	-	ABSENT	BENIGN	BENIGN	-	-	NO	BENIGN	HEMANGIOMA
24	21662	46	F	-	1	VI	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	HIGH	-	-	-	ABSENT	BENIGN	BENIGN	-	-	NO	BENIGN	HEMANGIOMA
25	23419	51	F	-	1	VIII	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	HIGH	-	-	-	ABSENT	BENIGN	BENIGN	-	-	NO	BENIGN	HEPATIC CYST
26	32332	39	F	-	1	III	-	WD	HYPER	-	-	BENIGN	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	HOMO	-	-	MALIGNANT	MALIGNANT	BENIGN	-	-	BENIGN	HEPATIC ADENOMA

27	23140	28	F	WILSON'S DISEASE	1	BOTH LOBES	-	WD	-	-	ISO/HYPO	MALIGNANT	-	LOW	-	-	-	-	HIGH	-	-	-	ABSENT	BENIGN	BENIGN	-	-	NO	BENIGN	REGENERATIVE NODULES
28	23132	71	M	-	1	RIGHT LOBE	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	HIGH	-	-	-	ABSENT	BENIGN	BENIGN	BENIGN	-	-	BENIGN	HEMANGIOMA
29	28760	72	M	HbsAg+	1	RIGHT LOBE	ID	-	-	HETERO	-	MALIGNANT	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	-	MALIGNANT	-	MALIGNANT	HEPATOCELLULAR CARCINOMA
30	28778	56	M	CLD	1	VIII	ID	-	-	HETERO	-	MALIGNANT	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	-	MALIGNANT	-	MALIGNANT	HEPATOCELLULAR CARCINOMA
31	29076	72	M	CLD	1	VII	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	HIGH	-	-	-	ABSENT	BENIGN	BENIGN	-	-	NO	INTERMEDIATE	DYSPLATIC NODULE
32	29854	64	M	-	1	II	ID	-	-	HETERO	-	MALIGNANT	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	-	-	NO	MALIGNANT	MULTICENTRIC HCC
33	23864	67	F	-	1	III	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	HIGH	-	-	-	ABSENT	BENIGN	BENIGN	-	-	NO	BENIGN	HEPATIC CYST
34	24091	46	M	-	1	VIII	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	HIGH	-	-	-	ABSENT	BENIGN	BENIGN	-	-	NO	BENIGN	HEMANGIOMA
35	28154	76	M	CA STOMACH	1	BOTH LOBES	-	WD	HYPER	-	-	BENIGN	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	HOMO	-	-	MALIGNANT	MALIGNANT	-	-	NO	MALIGNANT	METASTASIS
36	28234	65	M	-	1	V	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	HIGH	-	-	-	ABSENT	BENIGN	BENIGN	-	-	NO	BENIGN	HEPATIC CYST
37	26800	28	M	-	1	VII	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	-	LOW	-	HETERO	-	MALIGNANT	BENIGN	BENIGN	-	-	BENIGN	LIVER ASBSCESS
38	22212	46	M	-	1	LEFT LOBE	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	HIGH	-	-	-	ABSENT	BENIGN	BENIGN	-	MALIGNANT	-	MALIGNANT	INTRAHEPATIC CHOLANGIOCARCINOMA
39	44491	38	M	CA TONGUE	1	RIGHT LOBE	-	WD	HYPER	-	-	BENIGN	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	HOMO	-	-	MALIGNANT	MALIGNANT	-	-	NO	MALIGNANT	METASTASIS
40	23781	67	M	-	1	VI	WD	-	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	BENIGN	-	-	BENIGN	CHOLANGITIC ABSCESS
41	26175	28	M	-	1	VIII	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	-	LOW	-	HETERO	-	MALIGNANT	BENIGN	BENIGN	-	-	BEINIGN	LIVER ASBSCESS
42	29001	67	F	-	1	RIGHT LOBE	ID	-	-	-	ISO/HYPO	MALIGNANT	HIGH	-	PRESENT	-	PRESENT	-	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	BENIGN	-	-	BENIGN	FOCAL NODULAR HYPERPLASIA
43	28134	56	F	-	2	VII	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	HIGH	-	-	-	ABSENT	BENIGN	BENIGN	-	-	NO	BENIGN	HEMANGIOMA
						VIII	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	HIGH	-	-	-	ABSENT	BENIGN	BENIGN	-	-	NO	BENIGN	HEPATIC CYST
44	29002	45	F	-	1	VI	-	WD	HYPER	-	-	BENIGN	HIGH	-	PRESENT	-	PRESENT	-	HIGH	-	-	-	ABSENT	BENIGN	BENIGN	-	-	NO	BENIGN	HEMANGIOMA
45	23990	67	M	CA RECTUM	1	VIII	ID	-	-	HETERO	-	MALIGNANT	HIGH	-	-	ABSENT	-	ABSENT	-	LOW	-	HETERO	-	MALIGNANT	MALIGNANT	-	MALIGNANT	-	MALIGNANT	METASTASIS